



Swedish National Institute
of **Public Health**

Physical Activity in the Prevention and Treatment of Disease

PROFESSIONAL ASSOCIATIONS
FOR PHYSICAL ACTIVITY (SWEDEN)



Physical Activity in the Prevention and Treatment of Disease

PROFESSIONAL ASSOCIATIONS
FOR PHYSICAL ACTIVITY (SWEDEN)

© PROFESSIONAL ASSOCIATIONS FOR PHYSICAL ACTIVITY, SWEDEN (YRKESFÖRENINGAR FÖR FYSISK AKTIVITET, YFA), 2010
THIS IS A TRANSLATED AND UPDATED VERSION OF THE SECOND EDITION OF THE BOOK IN SWEDISH.

SWEDISH NATIONAL INSTITUTE OF PUBLIC HEALTH R 2010:14

ISSN: 1651-8624

ISBN: 978-91-7257-715-2

EDITORIAL COMMITTEE: PROFESSIONAL ASSOCIATIONS FOR PHYSICAL ACTIVITY – CARL JOHAN SUNDBERG, CHAIR

FOR SNIPH: ANNA JANSSON AND CARINA EDLING – PUBLIC HEALTH PLANNING OFFICERS,

MIA WADMAN – PRODUCTION MANAGER

TRANSLATION: SEMANTIX AB

LAYOUT AND GRAPHIC PRODUCTION: AB TYPOFORM

COVER PHOTOGRAPHY: LEIF R JANSSON/SCANPIX

PRINTING: ELANDERS, 2010

SWEDISH TITLE: FYSS, FYSISK AKTIVITET I SJUKDOMSPREVENTION OCH SJUKDOMSBEHANDLING

ALL RIGHTS RESERVED. COPYING THE CONTENTS OF THIS BOOK, IN PART OR IN WHOLE, IS PROHIBITED UNDER THE COPYRIGHT ACT WITHOUT WRITTEN CONSENT FROM PROFESSIONAL ASSOCIATIONS FOR PHYSICAL ACTIVITY, SWEDEN (YRKESFÖRENINGAR FÖR FYSISK AKTIVITET, YFA). THIS PROHIBITION ALSO APPLIES TO EDUCATIONAL USE AND INCLUDES EVERY TYPE OF COPYING THROUGH PRINTING, COPYING, AUDIBLE RECORDING ETC.

Contents

Preface	5
Background	7
PART I	9
1. General effects of physical activity	11
2. General recommendations regarding physical activity	41
3. Promoting physical activity	50
4. Becoming physically active	68
5. Motivational interviewing about physical activity	83
6. Assessing and controlling physical activity	97
7. Various types of physical activity and exercise	116
8. Health aspects of strength training	122
9. Infections and sports	134
10. Sports and sudden death	150
11. Children and young people	157
12. Pregnancy	176
13. Menopause	185
14. Elderly	200
PART II	209
15. Alcohol dependence/abuse	211
16. Anxiety	221
17. Asthma	232
18. Back problems (chronic)	242
19. Cancer	256
20. Chronic obstructive pulmonary disease (COPD)	271
21. Coronary artery disease	283
22. Cystic fibrosis	300
23. Dementia	316
24. Depression	325
25. Diabetes mellitus – type 1 diabetes	336

26. Diabetes mellitus – type 2 diabetes _____	345
27. Dizziness and balance disorders _____	356
28. Gastrointestinal diseases _____	367
29. Heart failure _____	379
30. Heart rhythm disturbances _____	397
31. Hypertension _____	410
32. Kidney disease (chronic) and kidney transplant _____	426
33. Lipids _____	439
34. Metabolic syndrome _____	447
35. Multiple sclerosis _____	464
36. Obesity _____	481
37. Osteoarthritis _____	498
38. Osteoporosis _____	510
39. Pain _____	523
40. Parkinson’s disease _____	541
41. Peripheral arterial disease _____	548
42. Post-polio _____	554
43. Rheumatoid arthritis _____	567
44. Schizophrenia _____	579
45. Spinal cord injury _____	589
46. Stress _____	602
47. Stroke _____	611

Preface

Östersund, November 2010

Physical activity has both health promoting and disease prevention properties. An increase in physical activity is one of the measures that would have the greatest positive impact on the health of the population. If everyone followed the recommendation of being physically active on a daily basis, the health of the population would improve considerably and healthcare costs would drop dramatically.

Regular exercise has well-documented preventative and/or curative effects on a number of different diseases, such as diabetes, cardiovascular disease, colon cancer and depression. The burden of illness and disease related to physical inactivity costs society a great deal in terms of increased healthcare costs and production losses. The European Union Public Health Information System (EUPhix) estimates that physical inactivity might cost a country about EUR 150–300 per citizen and year, and the medical cost of physical inactivity in the US alone was estimated at USD 75 billion in 2000. According to the World Health Report 2000, physical inactivity was estimated to cause 1.9 million deaths worldwide every year.

The healthcare system is in a strong position to work on increasing physical activity in the population. On one hand, people often come into contact with the healthcare system on a regular basis. On the other, people often trust their health and medical care providers in matters regarding their health. Healthcare providers also reach the groups in society that are the most sedentary, such as the elderly and the ill. One advantage of physical activity as a treatment compared to medication is that physical activity makes patients feel actively involved in their own treatment and encourages them to take personal responsibility for their own health. Physical activity on prescription (FaR®) is a method used in Swedish healthcare to increase physical activity in the population. With this method, the patient receives an individualized prescription for physical activity in a group or individual setting. Use of this method has grown in recent years and was applied by all county councils in Sweden in 2008.

The English version of this text has been partly updated with additional beneficial correlations between physical activity and metabolic syndrome, stress, dementia, schizophrenia and other conditions. In some cases, physical activity can also replace pharmaceuticals and, in others, can reduce the need for medication. Prescribing physical activity should be just as natural as other proven treatments and methods. This text aims to provide the knowledge necessary to do so.

We would like to express our gratitude to all of the authors for their excellent work. I would especially like to thank Professor Jon Karlsson and Associate Professor Carl J Sundberg and the other members of YFA – Yrkesföreningar för Fysisk Aktivitet (Professional Associations for Physical Activity) for the strong collaboration that made this publication possible.

Sarah Wamala

DIRECTOR-GENERAL

SWEDISH NATIONAL INSTITUTE OF PUBLIC HEALTH

Background

This first version in English is a translation based on the second edition of Physical Activity in the Prevention and Treatment of Disease (FYSS). The first edition of FYSS was published 2003, and a great deal has happened since then concerning the knowledge of physical activity in disease prevention and disease treatment. The first edition felt mostly like an inciter of interest in the area, but those of us who worked with the second Swedish and now the first English edition still feel that the area is just as current and exciting as then, while at the same time noting that the handbook is now perceived as an established concept in Swedish healthcare.

The book includes no less than 47 chapters. A great deal has happened in the field over the past eight years, and new facts from several well-done studies have now completed the knowledge base. The objective of the book is to increase the knowledge of the value of promoting physical activity in the population. This can be done in a number of different ways and through different actors such as healthcare services, NGOs, municipalities and county councils. The book is intended to be a knowledge base, easily available and practical, for all who work with promoting physical activity, but also to function as a textbook for various educational programmes.

As before, the project was run by Professional Associations for Physical Activity (Yrkesföreningar för Fysisk Aktivitet – YFA), which is an independent sub-association of the Swedish Society of Sports Medicine (Svensk Idrottsmedicinsk Förening – SIMF) which in turn is a section within the Swedish Society of Medicine. YFA comprises various professional categories, all of which have the objective of promoting physical activity in the prevention and treatment of disease. Since the work with the first Swedish edition, YFA has cooperated with the Swedish National Institute of Public Health (SNIPH), a cooperation that made the publication of this handbook possible. We would hereby like to express a huge debt of gratitude to the Swedish National Institute of Public Health and especially former Director General Gunnar Ågren and current Director General Sarah Wamala and their co-workers for their whole-hearted cooperation and support.

To further deepen the knowledge in this book, we have chosen to cooperate with the Norwegian Directorate of Health and the National Council for Physical Activity in Norway. Accordingly, both Swedish and Norwegian authors have contributed to a broader knowledge base and more in-depth analysis of the evidence. We would like to direct a major thanks to all of the Norwegian co-authors and editors, especially Anita Andaas Aadland, Department of Physical Activity, Norwegian Directorate of Health, and Professor Roald Bahr, Norwegian School of Sports Sciences and National Council for Physical Activity, for a smooth and well-functioning cooperation. This cooperation has made it possible to publish a Norwegian edition of FYSS.

Compiling a book of this extent has proven to be a gigantic project, which with the help of many, particularly the members of YFA's Board, has now become a reality. Many thanks to YFA administrator Peter Lamming for his dedicated work. Finally, we would like to thank the Board of the Swedish Society for Sports Medicine for fruitful interaction and the society's chancery officer Ann-Kristin Andersson for continuing support.

It is our hope that FYSS will contribute to increased physical activity in patients and in society at large, and thereby contribute to improving the health of the entire population.

The editorial committee, Stockholm, September 2010

MATS BÖRJESSON

MAI-LIS HELLENIUS

EVA JANSSON

JON KARLSSON, PREVIOUS CHAIR, YFA

MATTI LEIJON

AGNETA STÅHLE, DEPUTY CHAIR, YFA AND EDITOR OF FYSS 2008 (SWEDISH EDITION)

CARL JOHAN SUNDBERG, CHAIR, YFA

JILL TAUBE

1. General effects of physical activity

Authors

Jan Henriksson, MD, PhD, Professor, Department of Physiology and Pharmacology, Karolinska Institutet, Stockholm, Sweden

Carl Johan Sundberg, MD, PhD, Associate Professor, Department of Physiology and Pharmacology, Karolinska Institutet, Stockholm, Sweden

Introduction

All of the body's tissues and our genetic material generally look like they did in our ancestors 10,000 years ago. The human body is built for movement. Body and mind benefit from physical activity. Most organs and tissues are affected by physical activity and adapt to regular exercise. Regular physical activity significantly reduces the risk of premature death.

This chapter focuses on the immediate effects of physical exertion and the long-term effects of regular physical activity/fitness training (aerobic training). Physical activity refers to all bodily movement that results from the contraction of the skeletal muscles and results in increased energy expenditure (1). For greater detail and references, we refer to textbooks and overview articles in the exercise physiology literature (2–6) or the more focused references provided in each section. Aspects of strength training are discussed in a separate chapter.

Physical activity can be carried out at different levels of intensity. The more intense, the greater the immediate impact on various bodily functions. Oxygen consumption, which is directly linked to energy expenditure, increases from 0.25 litres per minute at rest to slightly more than 1 litre per minute during a relaxed walk. During maximum exertion, it increases to 2–7 litres per minute, i.e. up to 10–25 times the resting rate.

During physical exertion, the pulse rises and cardiac output increases. Ventilation multiplies, blood pressure increases, body temperature rises, perfusion in the heart and muscles increases, more lactic acid is formed and the secretion of hormones such as adrenaline, growth hormone and cortisol increases.

Maximum oxygen uptake capacity depends on body size, gender, age, fitness level, genetics and more. The factors that limit performance capacity in full-body exertion differ depending on the length of the session. The durations stated in the following text shall only be viewed as approximate for an “average”, middle-aged person – major differences

exist. In maximal exertion that lasts 5–15 minutes, central circulation (the heart) is generally considered to comprise the most important limitation of the performance capacity (by limiting the maximum oxygen uptake capacity). The longer the exertion continues, the more performance capacity is limited by properties of the engaged skeletal muscles (mitochondria, capillaries, some transport molecules, buffer capacity, etc.), which affect the so-called anaerobic threshold (see below for definition). In terms of long-term endurance (more than 30–60 minutes), the muscles' carbohydrate deposits (glycogen) also comprise a limitation.

What determines the response to exercise training?

Several factors determine how much a person improves if the degree of physical activity increases. One important factor is the fitness/performance level when the period of exercise training begins. A person who is inactive and in poor shape improves more in relative terms than a person who is well trained. The effects of exercise are specific to the organs and tissues that are exercised – only the muscles that are used adapt and only the parts of the skeleton that are loaded are strengthened. The length of the period of exercise training also plays a major role. Although some effects from exercise can be seen after a surprisingly short time of one to a few weeks, the effects are considerably greater if training continues for several months to years. Of course, the effect of exercise gradually “levels off” and eventually a considerable amount of training is required just to maintain the prevailing level of fitness.

Three other important factors are frequency (how often the person exercises), duration (how long a session is) and intensity (how hard/intense the session is). These three factors determine the combined “exercise dose”. In other words, the higher the dose, the greater the effect. It should be pointed out that low doses also have an effect, although to a more limited extent.

Frequency

For physical activity to have the maximum performance and health effects, it must be pursued often and regularly. The effect that an exercise session has can affect the body for several days, and then subside. Consequently, for low-intensity physical activity, a daily “dose” is recommended.

Duration

As a rule, the longer the activity continues, the greater the effect it has. In many cases, the daily activity session can be divided up into several separate 10–15 minute periods, as long as the total time is sufficient. One common recommendation with regard to time is 30 minutes of physical activity per day.

Intensity

The harder an exercise session is, the greater its performance and health effects usually are, although excessively intense exercise can lead to deteriorations. Good health-related effects

often seem to be achieved at a lower intensity, although a higher intensity is important to be able to improve fitness and to maintain an improvement in condition.

Moreover, there are of course a large number of factors that affect the outcome of the exercise training. For example, exercise can be conducted with relatively constant or with varying intensity (interval training) and with varying size of the engaged muscle mass (arm, abdomen and leg muscles compared with just leg muscles, for example). Genetics also seem to play a relatively large role in how large the response to exercise training is, perhaps accounting for around a third to one half of the variation between people. There is some evidence in the literature that individuals who increase their performance capacity at a certain exercise dose more than others appear to activate key genes in a stronger way (7, 8). It has not been established whether differences in exercise response are only due to genetic mechanisms (9). Age can be of significance, although older persons do not generally appear to have a worse ability to increase their relative performance. The composition of the diet may also play a role; a deficient diet lessens the response to exercise training. Dietary supplements provide no proven effect, however.

Effects of acute exertion and regular exercise

When discussing the effects of physical activity on the bodily organs and organ systems, it is necessary to differentiate 1) what happens in the body during (and after) a session of physical activity compared with the situation at rest, and 2) what differences are achieved (at rest or under exertion) after a certain period of exercise training compared with an untrained condition. In this text, the former is referred to as “Effects of acute exertion” and the latter as “Effects of exercise training”. The effects of acute exertion are due to a number of factors and differ between different tissues. The time for achieving different effects from exercise training varies from function to function, some processes start immediately in connection with the first exercise session, others take weeks to months before they are noticeable.

From a physiological perspective, a physical activity is called either aerobic or anaerobic, depending on which form of metabolism is dominant. One rule of thumb is that physical activity is aerobic (dependent on oxygen) if the maximum time one can perform the activity exceeds two minutes (3). Then the muscles mainly obtain their energy from the oxygen-dependent degradation of carbohydrates or fat. If one has the energy to carry out the activity for two minutes, but no longer, the metabolism is probably approximately 50 per cent aerobic and 50 per cent anaerobic (not oxygen-dependent). In short-term, intense physical activity, the muscles work without a sufficient oxygen supply (anaerobic metabolism) and the dominant energy-providing process is the splitting of glycogen and glucose into the degradation product lactic acid. Consequently, it is natural that aerobic exercise and anaerobic exercise provide different effects of exercise training. Aerobic exercise performed for a sufficient period of time stimulates the adaptation of the heart and the aerobic systems of the skeletal muscles, which is why regular exercise leads to the heart increasing its capacity accompanied by an increased mitochondrial volume in the

engaged skeletal muscle cells. The exercise time in pure anaerobic exercise (such as sprint training) is too short to provide these exercise responses in the heart and muscles. Such anaerobic training instead leads to improved conditions for greater lactic acid production and lactic acid tolerance.

Daily physical activity often has elements of both aerobic and anaerobic activities, such as walking in hilly terrain. Strength training, especially with heavy weights, is an extreme form of anaerobic exercise. In interval training (such as interspersed 10–15 second periods of hard exertion and equally long periods of rest), aerobic and normally anaerobic exercises are combined so that the total period of exercise at heavy loads can be kept sufficiently long to provide an exercise effect on both the heart and the muscles' aerobic systems (81).

Measurement of exercise dose and exercise effects

Measurement of heart rate or perceived exertion (10) are methods to adjust the exercise dose to the person's own capacity. This is discussed in depth in a separate chapter. Pedometers, or step counters, that measure vertical movements, are good aids for measuring the total number of steps when walking and running, but are relatively insensitive to many other movements. To obtain objective measurements of an individual's physical activity during a certain period of time, tri-axis accelerometers are used instead, measuring frequency, intensity and duration of movements on horizontal, sagittal and vertical planes (11).

Training effects in aerobic exercise are often measured as the change in the maximum oxygen uptake capacity. This is the highest oxygen consumption a person can achieve and is measured when the individual works with a maximum pulse during e.g. running. In short-term exercise (5–15 minutes), the maximum performance capacity is largely dependent on the maximum oxygen uptake capacity (12). This can be improved by 20–50 per cent in 2–6 months if exercise is of sufficient intensity. However, there are very large individual differences in the response to exercise. From the maximum oxygen uptake capacity, the individual's maximum energy expenditure can be calculated, since every litre of consumed oxygen corresponds to an amount of energy of approximately 20 kJ (5 kCal). Since direct measurement of the maximum oxygen uptake capacity is relatively difficult, and requires both special equipment and nearly maximum exertion by the individual, an indirect approach is often used where the maximum oxygen uptake capacity is calculated based on heart rate measurements at lower levels of exertion (3). The reliability of the indirect methods is limited, however.

Determination of the anaerobic threshold (lactic acid threshold test) can be achieved in a reliable manner from blood lactic acid samples taken during non-maximum exertion (13). The anaerobic threshold is defined as “the highest sustained exertion intensity at which lactic acid appearance in the blood will be equal to the rate of its disappearance”. In terms of percentages, it increases more than the maximum oxygen uptake capacity after a period of endurance training. The economy of motion can also be improved with exercise. Accordingly, less energy is consumed for the same amount of work performed (measured as lower oxygen consumption), efficiency is improved. This may be very noticeable for many activities such as running while for others, such as cycling, efficiency is gener-

ally identical for all individuals. Besides the aforementioned effects of exercise training, which are significant to performance capacity, important exercise effects can be measured that are primarily of significance to one's metabolism (and risk of disease). Among these are glycosylated hemoglobin (HbA1C) – an integrated measure of blood sugar increases over an extended period; glucose tolerance – a measure of plasma glucose two hours after the consumption of 75 grams of glucose, inverse measure of insulin sensitivity and blood glucose control; and fat tolerance (14) – a measure of blood lipids in the hours after a fat-rich meal, inversely related to insulin sensitivity. Other methods to measure the effects of regular exercise include underwater weighing, skin-fold, DXA, BOD POD (15) – methods to measure the body's fat content; and immunoglobulin A in saliva (16) – method of measuring immunity in connection with physical activity.

Heart

Acute exertion

During exercise, which involves large muscle groups, there is a high demand on the heart and blood circulation. The skeletal muscles' requirement of a supply of oxygen and nutrients and the need for greater removal of carbon dioxide and other waste products as well as the need for temperature and acid-base balance demands greater blood circulation. With help from the autonomic nervous system, this increased need can be felt, which leads to an increase in pulse and the contractile force of the heart. An increase from a resting heart rate, usually 60–80 beats per minute, up to a pulse of around 120 beats per minute is accompanied by an increase in the heart's stroke volume, meaning the amount of blood pumped out with each heartbeat. Under exertion, heart frequency increases from a resting heart rate of approximately 60–80 beats per minute to the maximum pulse, which can be 150–230 beats per minute depending mainly on age, but also on individual factors. The greater contractile force of the heart increases pressure in both chambers of the heart. Pulse rate and contractile force increase in proportion to the work load and the amount of blood the heart pumps out increases from 4–5 litres per minute at rest to 20–40 litres per minute at maximum exertion intensity. The greater cardiac exertion increases demand on the heart's own circulation (coronary circulation), which increases 5–8 fold.

Effects of exercise training

The effects of exercise training on the heart, like the majority of organs and tissues, depends on the frequency, intensity and duration of the exercise sessions. After a few months of sufficiently intensive exercise, it is typical for the resting heart rate and the pulse during submaximum exertion to be 5–20 beats per minute lower, for the stroke volume to increase by 20 per cent or more, and for the heart's contractility to improve, which is reflected in a greater ejection fraction (the percentage of the heart's blood volume that is pumped out in one heartbeat) at maximum exertion intensity. The maximum heart rate is unaffected or only slightly decreased. Structurally, the heart's internal volume increases significantly and its wall thickness increases somewhat, which altogether means greater cardiac muscle

mass, primarily due to the individual cardiac muscle cells increasing in size. In addition, the number of capillaries and mitochondria increases. The expansion capacity of the coronary vessels is improved with exercise training (17). The extent to which the occurrence of and sensitivity of receptors to signal substances and hormones in the cardiac muscles change with regular exercise is not fully established (18).

Skeletal muscles

Acute exertion

Acute muscle exertion demands greater activation of individual motor units and the recruitment of more motor units. In low intensity exertion, mainly motor units with slow-twitch muscle fibres (type I) are recruited and in more intense exertion, fibres in fast-twitch motor units (type IIa and IIx) are also recruited.

Muscle exertion demands a great deal of energy in the form of adenosine triphosphate (ATP). The primary sources of energy for ATP production are carbohydrates and fatty acids. Carbohydrates are stored in the form of glycogen in the liver and skeletal muscles. These stores are limited and must be refilled daily. Fatty acids are mostly stored in adipose tissue in virtually unlimited amounts.

Several factors affect the choice of energy sources during exertion, such as:

Exertion intensity

Energy expenditure is proportional to the exercise intensity. At rest, 60 per cent of the energy needed is provided by fats and, in low-intensity exertion, roughly the same proportion of the energy is extracted from fat as from carbohydrates. In more intense exertion, relatively more carbohydrates are used. This is due to several factors, including the fact that the fast, less oxidative and more glycolytic muscle fibres are involved to a greater extent and that the muscle cells' access to oxygen (oxygen pressure) gradually decreases. With higher work loads, the need for carbohydrates per unit of time increases sharply and can reach above 200 grams per hour. In maximum aerobic exertion, virtually only carbohydrates are considered to be burned and in even more strenuous, so-called supra-maximum exertion, large additional amounts of carbohydrates are split to lactic acid. The higher the load, the more lactic acid is formed, which makes both muscle tissue and blood more acidic (lower pH). However, the fatty acid need levels out with increasing work load and rarely exceeds 20–30 grams per hour. If exertion continues for several hours, 50 grams per hour may be burned.

The highest level of fat burning in skeletal muscles (grams per minute) is achieved at exercise intensities approximately corresponding to 50 per cent of the maximum oxygen-uptake capacity in the general population and to slightly more than 60 per cent of the maximum oxygen-uptake capacity in very fit individuals (19). However, it should be added that the total fat expenditure (during and after the exertion) is primarily dependent on the total energy expenditure, which is why the fat expenditure in high-intensity exertion is greater overall than in low-intensity exertion lasting the same amount of time. However,

the duration of high-intensity exercise is often very short and, consequently, fat expenditure is limited.

Fitness level

A well-trained person uses more fat for energy extraction and thereby saves carbohydrates with each work load, which means that it is possible to maintain a higher intensity of exertion for a longer period of time.

Duration of exercise

The longer a session of exercise of sub-maximal intensity lasts, the greater is the proportion of fat used. This is partially related to the gradual emptying of the body's carbohydrate deposits.

Diet

The composition of the diet also affects which energy sources are used. When fasting or on a fat-rich/carbohydrate-poor diet, fatty acids are used to a greater extent. After so-called carbohydrate loading, carbohydrates are used to a greater extent during exercise, but in spite of this glycogen (the storage form of carbohydrates) also lasts longer.

Body temperature

In strong hypothermia or heat loading, relatively more carbohydrates are used.

Oxygen supply

When oxygen availability is limited, such as at high altitudes, and when the blood flow to the working arm or leg is restricted, carbohydrates are used to a greater extent. An example of the latter situation is exercise with the arms when held above the heart. In acute exercise, the blood flow increases sharply (by 50–100 times) in the working muscles. This improves the oxygen supply and is mainly due to the vasodilatation (expanding of the vessels) brought about by various factors in the muscles.

Muscle fatigue

Fatigue upon exertion can be due to many different factors in multiple tissues. The local fatigue in the muscles can, for example, be caused by the accumulation of products from ATP decomposition or a lack of glycogen. Dehydration affects the circulating blood volume and can cause fatigue.

Effects of exercise training

The skeletal muscles comprise a very adaptable tissue. Endurance training affects its structure and function significantly. In terms of the muscles' contractile function, some studies have found increased activation and increased recruitment of motor units after a longer period of exercise training (20). Local fatigue in the working muscles decreases after a period of exercise training.

Structure

The size of the muscle fibres changes only slightly in endurance training, while it can of course increase sharply if the endurance training has elements of strength training. The slow-twitch fibres (type I) can become somewhat larger. In terms of the distribution between fibre types within the type II group (fast-twitch fibres), the proportion of type IIx decreases after around one week at the same time that the proportion of type IIa increases. The transition from type II to type I (slow-twitch) fibres is very limited in the short term, although the proportion of slow protein increases in many fibres.

The amount of mitochondria increases markedly (see below). The small capillaries also increase in number. This improves the blood flow and extends the perfusion time of the tissue, which facilitates the exchange of oxygen and nutrients. However, in contrast to some animals, it appears to be difficult to affect myoglobin content with exercise in humans.

Transport capacity

The occurrence of so-called glucose transporters (GLUT-4) in the skeletal muscle cell membrane increases immediately in connection with an exercise session and even more after a longer period of exercise training. This increases sensitivity to insulin and tolerance to sugar (glucose). The occurrence of fatty acid-binding proteins increases on the capillary wall and in the muscle cell. Exercise training also improves the occurrence of special transport molecules for fatty acids into the muscle cell and its mitochondria, and transport molecules for lactic acid out of the muscle cell. This raises the transport capacity considerably. Furthermore, as early as within a few days, the ATPase activity of the sodium-potassium pumps (enzyme rate) increases, which probably improves the ability to restore the ion balance (contractility) after the end of muscle exertion (21).

Nutrient deposits

The amount of deposited carbohydrates (in the form of glycogen) and fatty acids (in the form of triglycerides) in skeletal muscle cells can be more than doubled (glycogen is tripled or quadrupled) with exercise training.

Fat and carbohydrate use

The amount of enzymes that break down fatty acids increases very rapidly after exercise training, which facilitates fatty acid use. The mitochondrial density, and consequently the muscle's fat and carbohydrate burning capacity, increases relatively quickly with exercise training. Within just 4–6 weeks, a 30–40 per cent increase can be noted. The very well-trained have 3–4 times higher mitochondrial densities in trained muscles than those that do not exercise.

After just a few weeks' training, a higher "metabolic fitness" is achieved, meaning that carbohydrates are "saved" and fat is used to a greater extent for energy extraction under exertion at a given, sub-maximal load level. These tangible differences in substrate selection are due in part to higher mitochondrial density and a greater transport capacity for fatty acids. The production and concentration of lactic acid is lower at a given load. The

buffer capacity for lactic acid is also improved (22). At maximum exertion (higher exertion intensity possible after exercise training), the lactic acid concentration is significantly higher.

Mechanisms

During acute exertion, the external and internal environments of the skeletal muscle cells change. Hormones and growth factors surround and are bound to the cells to a greater extent. One example is Vascular Endothelial Growth Factor (VEGF), which affects the new formation of blood vessels (23). Inside the cells, the temperature, calcium content and occurrence of the ATP molecule's decomposition products increase. At the same time, pH and oxygen pressure decrease. These and other factors directly and/or indirectly affect a number of proteins in the skeletal muscles. For example, the degree of phosphorylation of so-called mitogen-activated protein kinases (24) and mitochondrial factors (25, 82) is affected, which in turn affects processes that control the adjustment to exercise training, such as the activity degree of some genes. It should also be noted that only the muscles used/exercised adapt, hence, the exercise is specific, which is of crucial significance.

Blood

Acute exertion

Upon exertion, the working muscles swell up somewhat, which is due to all of the metabolites formed in the muscle cells osmotically "extracting" fluid from the blood. This fluid withdrawal to the working muscles directly decreases blood volume somewhat, which means that the concentration of hemoglobin (blood value) increases by 5–15 per cent. After the exertion, this increase subsides.

The occurrence of leukocytes also rises sharply under exertion, largely because leukocytes are released from lymphatic tissue, such as lymph nodes and the spleen.

Effects of exercise training

Regular endurance training entails a significant increase in both plasma and blood cells, which together increase blood volume by 10–15 per cent or more (26). The plasma expansion begins as early as the first week after the first exercise session. A change in the number of red blood cells, which is relatively less, can first be observed after a few weeks. Because the plasma volume expands more, the percentage of the blood comprised by red blood cells will drop due to dilution. This is why, paradoxically, a person often has a lower Hb value (blood count) after a period of exercise training despite a higher total amount of red blood cells. The increased blood volume increases the venous return of blood to the heart and consequently the end-systolic volume of the heart, which contributes to increasing the stroke volume and lowering the heart rate at rest and in sub-maximal exertion.

Mechanisms

The increased plasma volume is probably due to hormonal factors that increase fluid retention and to an increased synthesis of albumin that binds more fluid in the plasma. The increased formation of red blood cells is probably due to an increase in the erythropoietin (EPO) concentration.

Lipoproteins

Acute exertion

Significant changes in blood lipid levels (lipoproteins) are seen after individual exercise sessions, with higher levels of HDL cholesterol (4–43%), especially subfractions 2 and 3, associated with a reduction of the levels of triglycerides and very low density lipoproteins (VLDL). These changes can last 24 hours after the end of exertion (27). A work load corresponding to five kilometres of running at an intensity corresponding to the anaerobic threshold has also been given as a threshold value for achieving these changes (28).

An exercise session, such as a one-hour long brisk walk, done within 24 hours before a fat-rich meal means that the increase in lipoproteins in connection with the meal will be significantly lower than if no exercise session had been done (29). This can probably in part be explained by the enzyme lipoprotein lipase in the skeletal muscles' capillaries being activated by the exertion and increasing its decomposition of the fat molecules (triglycerides) that flow through the capillaries, and in part by a decreased secretion of triglycerides from the liver. Both of these effects are probably connected to the lack of energy (reduced levels of energy substrate) that occurs in the skeletal muscles and liver after an exercise session of a sufficient length (30).

Effects of exercise training

Changes in the composition of lipoproteins with exercise are among the changes that are thought to underlie the reduced risk of cardiopulmonary diseases among fit individuals. Physically fit individuals normally have higher levels of HDL cholesterol (high-density lipoproteins) and lower levels of triglycerides than untrained individuals. The increase in HDL cholesterol is considered to be especially important due to its role in the process (reverse cholesterol transport) whereby the body extracts cholesterol from peripheral tissues for transport to the liver and excretion.

Other changes in prolonged training, although not as constantly occurring, include lower overall cholesterol and LDL cholesterol (low-density lipoproteins) (83) as well as lower concentrations of Apolipoprotein B. The approximate exercise volumes required to obtain these positive effects from exercise have been given as the equivalent of 25–30 km jogging or fast walking per week, or in other words an exercise-related energy expenditure of 1,200–2,200 kcal per week. Higher exercise volumes entail additional positive effects. With this level of exercise, people of both genders can expect the HDL cholesterol level to rise by 10–20 per cent and the triglyceride level to decrease by 10 to 30 per cent (31).

The significance of relatively extensive physical exercise to influence the composition and concentrations of lipoproteins is clearly exemplified by a large U.S. study (32) in which overweight men and women were divided into four groups, one control group and three exercise groups, that were monitored for eight months. Group A exercised (ergometer cycling, jogging) with an energy expenditure that corresponded to 32 km jogging per week and with a strenuous load (65–80% of maximum oxygen uptake capacity), group B exercised at the same intensity, but with a shorter distance (corresponding to an energy expenditure of 19 km jogging per week), while group C underwent the same amount of exercise as group B, but at a lower intensity (corresponding to 40–55% of the maximum oxygen uptake capacity). After eight months of exercise, the concentration of HDL cholesterol had only increased in group A (+ 9%), together with several other beneficial lipoprotein changes (such as lower LDL concentrations with an increase in the LDL particles' size). Some changes were also noted in groups B and C (primarily an increase in the size of the LDL and VLDL particles), but to a much lower extent (32).

Blood coagulation factors and platelet characteristics

Acute exertion

An exercise session leads to a significant increase in the number of platelets in the blood. This cannot be explained by the decrease in the plasma volume that occurs in acute exertion (see above), but rather is probably due to a release of blood platelets from various organs, such as the spleen, bone marrow and from pulmonary circulation. An activation of blood platelets has also been observed during acute exercise sessions, primarily among untrained individuals. Such an activation is reflected in an increased occurrence of the protein, P-selectin, on the platelets' surface, with greater aggregation tendency of platelets and greater formation of thrombin and fibrin leading to a shortened coagulation time. It is mainly higher intensity exertion that provides these potentially negative effects of physical exercise. Studies of individuals with coronary disease provide clear support for the theory that platelet aggregation and activation increase through physical activity. Interestingly, this increase does not appear to be inhibited by acetylsalicylic acid, which is normally an effective treatment for conditions of increased blood clot tendency (33).

These results can be compared with what is known about the risk of being afflicted by a cardiac infarction in connection with physical exertion. It should be noted that it is relatively uncommon for a heart attack to be triggered by physical exertion; only around 5 per cent of cardiac infarctions occur during or within one hour of physical exertion. Of these cases, 70 per cent can be related to coronary occlusion by a platelet-rich blood clot. The definition of physical exertion that is commonly used in these contexts is an energy expenditure that is six times higher than the energy expenditure at sitting rest (six metabolic equivalents or 6 MET), which can normally be said to correspond to light jogging or shovelling snow, for example.

In various studies, the risk of being struck by a heart attack during a randomly selected hour was compared with the same risk during and within one hour after physical exertion.

In several studies, large increases in risk were reported in connection with physical exertion (34, 35). However, the results only apply to untrained men who rarely (less than once a week) subjected themselves to this degree of exertion. Regular exercise constitutes strong protection against the increased risk of cardiac infarction in connection with physical exertion, and the risk has been estimated to only be 2.5 times (34) and 1.3 times (35) greater, respectively, than at rest for men who exercise regularly (>6 MET at least 4–5 days per week). For women, the risk of being struck by a heart attack during and in connection with physical exertion is very small (compared with the risk during a randomly selected hour without physical exertion), and the small risk that has been reported appears to vanish with regular exercise. For both men and women who exercise regularly, the risk of having a heart attack at all (that is at any hour of the day) is less than half of that among untrained individuals (36).

Effects of exercise training

The sharply reduced risk of sudden death or acute cardiac infarction during an exercise session among people who exercise regularly indicates that prolonged exercise gives rise to changes that counteract the increased aggregation tendency of platelets under exertion. This has also been shown (37). Another important explanation is that the mechanism for the dissolution of blood clots, fibrinolysis, is enhanced among individuals who exercise regularly (38). Other important explanations of the reduced tendency for blood clots among fit individuals may be that regular exercise leads to higher levels of prostaglandin, in part as a result of higher levels of HDL cholesterol, and a higher nitrogen oxide content, both of which inhibit blood clot formation.

Vessels, blood pressure and blood flow distribution

Acute exertion

Arterial blood pressure depends on the cardiac output (volume of blood per minute) and the peripheral resistance in the vessel tree. During a session of acute exercise, the cardiac output increases significantly at the same time that peripheral resistance decreases sharply, although not to the same extent that the cardiac output increases. This means that the mean blood pressure increases, almost entirely dependent on a systolic blood pressure increase in proportion to the exertion intensity. At maximum intensity, the blood pressure, measured over the brachial artery, is 180–240 mm Hg. The change in resistance is not uniform in the various tissues of the body.

In the vessels of the working muscles and the heart, a marked vasodilation occurs with a resulting decrease in resistance. This results in the skeletal muscles' share of the blood flow at rest increasing from approximately one fifth (of 4–5 litres per minute) to approximately four fifths (of 20–30 litres per minute) under exertion. In other groups of vessels, such as in the digestive tract, kidneys and resting skeletal muscles, there is a constriction of the resistance vessels, which decreases the relative perfusion in these areas. Under strenuous exertion, particularly if the exertion is prolonged and occurs in a high ambient

temperature, the skin receives a relatively larger share of the blood flow. In the hours after a session of acute exertion, blood pressure drops by 5–20 mm Hg below the normal resting blood pressure.

Effects of exercise training

Up to just two decades ago, researchers believed that the vessels, except the capillaries, were relatively passive tubes that could not change with exercise. However, it turns out that regular exercise improves the function and structure of vessels that supply the engaged skeletal muscles and those in the heart muscle.

The dilation capacity of arterioles is improved and the inner volume of large arteries increases. Thanks to this and the increased capillarisation, an even larger proportion of the blood flow (84) can be guided to the working skeletal muscles. These and other mechanisms, such as increased parasympathetic activity and decreased release of adrenaline and noradrenaline (see the section on the hormone system), contributes somewhat to lowering resting blood pressure among those with normal blood pressure despite an increased blood volume.

Mechanisms

Through the “friction” exerted by the flow of blood against the vessel wall (shear stress), nitric oxide synthase, the enzyme that catalyses the formation of nitric oxide (NO), is induced. NO makes the smooth muscles relax and the vessels open. Capillary formation is probably induced through the formation of so-called angiogenic factors, of which vascular endothelial growth factor (VEGF) is the most studied. Together with anti-inflammatory mechanisms and better antioxidant activity, the increased NO formation contributes to counteracting arteriosclerosis/atherogenesis (hardening of the arteries/plaque formation).

Immune system

Acute exertion

The prevalent view is that regular physical activity of moderate to average intensity improves the function of the immune system and can reduce sensitivity to infections. However, hard or prolonged exercise in endurance sports can lead to immunosuppression and greater infection sensitivity (85).

During sessions of acute exertion, there is an increase in the majority of leukocyte populations in the blood, primarily neutrophil granulocytes and natural killer (NK) cells, and there is also a marked lymphocyte mobilisation to the blood. These changes have been related to the immune-stimulating function of moderate to intense physical exertion (39). However, in the process after a session of intense exertion, there is often a period of weakened immune function with reduced NK-cell activity and reduced lymphocyte proliferation (39). This immunosuppression is also clear in several organs, such as the skin, mucous membranes of the upper respiratory passages, lungs, blood and muscles, and appears to comprise an “open window” of diminished immunity through which viruses and bacteria

can enter and gain a foothold in the body. This immunosuppression can last from 3 to 72 hours after an intense exercise session, depending on which immunological markers are measured (40). Among athletes, the risk of developing a clinical infection in this situation can be particularly large upon exposure to new pathogenic flora when travelling, and to a lack of sleep, mental stress, poor food, etc. It has been shown that the concentrations of immunoglobulin A (IgA) and M (IgM) in saliva decrease immediately after an intense exercise session, after which they return to normal levels within 24 hours (41). Prolonged intense exercise can, however, result in a chronic reduction of these levels. This increases the risk of respiratory infections and it has been proposed that measurements of IgA and IgM in saliva during periods of intense training may be a way of keeping the risk of infection somewhat under control.

Macrophages are important cells in the immune system for phagocytosis, the elimination of microorganisms and tumour cells, and for T-lymphocyte-mediated immunity. There are preliminary indications that physical exertion can stimulate the macrophages' function in the former two areas, and possibly reduce T-lymphocyte-mediated immunity. Hard exercise has also been shown to give rise to raised levels of several both pro-inflammatory and anti-inflammatory cytokines, cytokine inhibitors and chemokines. The increase in the IL-6 cytokine after an exercise session is particularly sharp, but the significance of this increase is still unknown. It is well-known that physical activity leads to an activation of endogenous opioid peptides (86), which probably also plays a role in the immune system (42).

Effects of exercise training

Research regarding the relationship between the degree of physical exercise, the immune system and sensitivity to infections and other diseases is still in an early phase, and it is often difficult to draw definite conclusions from the results available in the literature. At rest, trained and untrained individuals appear to show relatively small differences in their immune system, with the exception of the activity of NK cells, which are usually higher among well-trained individuals. Besides these changes, reduced function of neutrophil granulocytes and reduced concentrations of NK cells have also been observed among over-trained individuals as a result of prolonged intense exercise (40).

Skeleton

Maximum bone mass is reached at the age of 20–30 years for both men and women, and then slowly decreases with rising age. Besides women having relatively greater bone-mass loss with age, there is also a sharp drop in connection with menopause. This means that post-menopausal women have significantly less bone mass than men (43). In the majority of studies of the significance of physical activity, bone mass refers to the bone's mineral density or mineral content. Mineral density can be measured through quantitative computed tomography, ultrasound densitometry or dual-energy x-ray absorptiometry (DXA) on selected parts of the skeleton or the entire body. It is also known that the bone's

size (volume), structure and protein content are affected by mechanical loading (44, 45), and it has been observed that these variables can often be more informative, since bone density measurements can underestimate the strengthening of the bone structure as a result of exercise (46).

Bone tissue is continuously reformed through resorption and synthesis, whereby the balance between these two processes determines if a net formation or decomposition occurs. How large the bone mass ultimately becomes is also dependent on the initial conditions and time. The common perception is that the balance is determined by the mechanical load the bone cells are subjected to (micro-strain), and that deficient loading leads to decreased bone mass and excess loading to increased bone mass. Important factors include how often the load is repeated and the direction, duration, speed, etc. of the load. For example, dynamic loading with higher speed is more significant than static loading or loads with a low speed. There is also data that indicates that varying types of loading can be more effective than repeating the same loading (47, 48). What in a given situation constitutes the threshold load for bone synthesis to exceed resorption depends on multiple factors, such as the levels of calcium, vitamin D and hormones. When mechanical loading is combined with an increase in oestrogen or androgen levels, the effect on new bone formation is greater than if each factor is allowed to act separately (49). For women, normal ovarian function is therefore very important to the development of bone.

In light of this, it can be expected that it is the intensity of exercise rather than its duration that is important to achieving greater bone density, and that types of exercise with high, temporary loading of the bone (high impact) are particularly effective. The greatest mineral density among athletically active men and women is also found in sports that involve impact loading, such as weightlifting, aerobics, squash, volleyball and football. The difference in bone density among trained and untrained persons, or between the hitting arm and the non-hitting arm among tennis and squash players, is usually on the magnitude of 10–20 per cent, while changes in bone tissue volume and strength may be larger (44).

It is known that intense physical exercise in adolescence, meaning mechanical loading on the skeleton, results in larger, stronger and more mineral-dense bones and that this effect is more pronounced if the exercise is begun early (50). If the exercise starts at an adult age, only small improvements in bone density are achieved. In spite of this, it has been clearly shown that the risk of a hip fracture is lower among trained individuals, while the proof that exercise at an adult age would reduce other types of fractures related to osteoporosis is not currently as strong (51). On the other hand, monotonous repetitions of the same load over time can cause microscopic damage that weakens the bone and eventually gives rise to so-called stress fractures (52). An interesting finding is that veteran cyclists, with many years of training behind them, have significantly lower bone density than control persons of the same age and, although very physically fit, they therefore have a higher risk of being affected by brittle bones with increasing age (53). Among women, intense exercise training such as long-distance running can also lead to diminished bone density, probably due to hormonal changes, possibly in combination with low energy consumption (49). The state of proof is relatively weak with regard to training with low to moderate intensity exercise also providing a positive effect on the skeleton. In

terms of non-weight-bearing activity, such as swimming, such activities do not normally lead to greater bone density.

In light of the fact that half of all women and one third of all men will be affected by a brittle bone fracture in their lifetime (51), it is of great interest to know if it is possible to build up a strong skeleton during adolescence that can protect against fractures later in life. However, available data does not conclusively indicate the existence of any such lasting protective effect (51) and it has been reported that the risk of fractures among former athletes is not lower than among those previously not athletically active (51). On the other hand, in recent years, studies have been published that support the hypothesis that physical activity in the teenage years and as young adults really is linked to higher bone density late in life, such as for lumbar vertebra (54) and proximal femurs after menopause among women (54, 55), as well as for lumbar vertebra and femurs among older men (56), which in turn could reduce the risk for a fracture in a long-term perspective.

Cartilage

Isolated cartilage cells respond to mechanical loading and an increased strain of a cyclically varying type leads to greater net synthesis of extracellular matrix in cartilage in organ baths. However, static loading commonly leads to decreased matrix production. In animal experiments, both intense physical activity on one hand and total immobilisation on the other have proven to lead to osteoarthritis-like changes. Intense and prolonged physical activity in human beings is also probably associated with osteoarthritis in hips and knees (57). The function of cartilage tissue is linked to the interaction between tissue matrix and the extra-cellular fluid that is bound to proteoglycan molecules in the cartilage tissue. Loading leads to deformation of the cartilage with fluid outflow from the tissue matrix to the surroundings, which normalises in the hours after the exertion. After 100 knee bends in people, this normalisation is reported to take more than 90 minutes (58). Consequently, it can be assumed that the balance between deformation and restitution is an important factor, and if this is kept at an appropriate level, damage to the cartilage in connection with physical activity may be avoided (87). What constitutes an appropriate level can probably vary significantly depending on different joint anatomy, joint mobility, etc. (59). There are research results that indicate that kinesiotherapy and passive motion training have positive effects on cartilage tissue by speeding the restitution phase (57).

Connective tissue

Connective tissue responds to strain with increased collagen synthesis, while immobilisation has the opposite effect. It is believed that a session of physical activity may in fact lead to increased decomposition of connective tissue as a result of the activation of protease enzymes. Analogous to what was reported above for cartilage, it therefore appears as if the actual exercise session leads to a degradation, meaning a reduction of the

synthesis rate, while synthesis markedly increases in the restitution phase in the ensuing days. Consequently, it is the balance between the effect on synthesis and decomposition that determines if a certain training programme leads to improved ligament strength or to a degradation with ruptures or damage as a result (60). It has also been shown that several hormonal growth factors and inflammatory mediators play a role in this balance. An interesting observation is that a considerable net synthesis of new connective tissue often requires several weeks or months of exercise, because the enhanced decomposition is most pronounced at the beginning of a period of exercise and can counteract the increased new formation of connective tissue (60). The strong ligaments that characterise well-trained individuals provide greater sustainability because the load per cross-sectional area decreases.

Lungs and gas exchange

Acute exertion

In low-intensity exertion, it is mainly the size of each breath (tidal volume) that increases. In more high-intensity exertion, the respiratory rate increasingly rises. Altogether, this means that the pulmonary ventilation increases from 6–8 litres per minute at rest to up to 150 litres per minute among the untrained and up to 200 litres per minute among well-trained persons under maximum exertion.

Under exertion, large amounts of oxygen is consumed and roughly the same amount of carbon dioxide is formed. Despite the sharply increased carbon dioxide formation, the content in arterial blood and exhalation air decreases at maximum exertion. This is due to the pulmonary ventilation increasing by 15–30 times at the same time that carbon dioxide formation increases by only 10–15 times. The extraction of oxygen from arterial blood increases from around 25 per cent at rest to more than 75 per cent under strenuous exertion.

Effects of exercise training

The pulmonary ventilation under maximum exertion increases. Under sub-maximum exertion, the respiratory rate, tidal volume and consequently pulmonary ventilation is significantly lower after exercise training. Exercise training improves the endurance of the respiratory muscles. This occurs by adaptations in the same way as in other skeletal muscles that are regularly exercised (see above). The lungs' blood flow distribution changes and there is a lesser degree of mismatch between perfusion and air ventilation; the upper parts of the lungs in particular receive a greater blood flow. The lungs' gas diffusion improves.

Mechanisms

The probable underlying causes behind the training-induced changes in the respiratory muscles are the same as for other muscles (see above). In terms of the improved blood flow distribution in the lungs, it may be due to the increased blood volume combined with changes in the vessels of the lungs.

Body composition and adipose tissue

The sharp increase in overweight and obesity that has occurred in the Western World in the past 15–20 years has been associated with growing inactivity, although the relative significance of decreased physical activity compared with altered caloric intake and meal-time patterns is unknown (61).

The energy expenditure when running on level ground is on a magnitude of 1 kcal per kg of body weight and kilometre, while the corresponding value for walking is 20–25 per cent lower. Accordingly, one hour of walking corresponds to 1/10 of the energy expenditure per day of a standard man (2,800 kcal per day) or woman (2,100 kcal per day). It being difficult and nearly impossible to predict on an individual level how more physical activity will affect body weight and body composition is illustrated by the fact that three glasses (of 2dl each) of a soft drink that may be consumed in connection with training also corresponds to 10 per cent of the daily energy needs. It has been said that the increase in the average weight of 20–40 year-olds in the U.S. in the 1990s (approximately 7–8 kg in eight years) could have been avoided if 100 kcal more of energy on average had been expended (or 100 kcal less food consumed) per day. This corresponds to just 15–20 minutes of walking or one glass of a soft drink (62).

The appetite can also be affected by physical activity in various ways. Low energy levels and low levels of insulin in plasma, which is often observed after an exercise session, stimulates the appetite through neuropeptide Y-releasing neurons in the central nervous system. On the other hand, intense exertion can lower the appetite through the release of corticotropin-releasing hormone (CRH) from the hypothalamus with an anorectic effect. At the population level, knowledge about how regular physical activity affects body composition is more certain, and several major studies with observation times of approximately 3–4 months show that various exercise programmes can be expected to provide a decrease in fat weight of an average of 0.1 kg per week. As a rule, the decrease in fat weight is always larger than the decrease in body weight, and body weight often does not change at all due to increased muscle mass (63). The decrease in fat weight is seen in both genders. Although a tendency of larger decreases are seen in men, it cannot be said for certain that any gender difference exists.

There is support from studies of rats that exercise training-induced changes occur in adipose tissue similar to those seen in skeletal muscles, with increases in both mitochondrial enzyme activity and in the level of glucose transport protein (GLUT-4). One difference from the skeletal muscles (where exercise training provides a decrease) is that there is an increase in adipose tissue of the hormone-sensitive lipase enzyme (HSL) with training, that is to say the enzyme responsible for the release of fatty acids (lipolysis) into the blood. This agrees with studies in organ baths of fat cells from humans and rats, where it could be shown that adrenaline (which stimulates HSL) gives rise to significantly greater release of fatty acids in fat cells that were taken from trained individuals than in fat cells from untrained individuals. It is known that being overweight leads to lower HSL concentrations in the adipose tissues, but that the concentration increases in connection with periods of fasting. The increased adrenaline effect on the release of fatty acids among trained individ-

uals can, however, also be due to a higher level of the adenylate cyclase enzyme in the fat cells. Adenylate cyclase conveys the effects of adrenaline by giving rise to the messenger molecule cyclic adenosine monophosphate, cAMP. The number of receptors for adrenaline on the surface of the fat cells is probably not affected by exercise, however. To some extent, the increased fat degradation activity in adipose tissue from trained individuals can be seen as a compensation for a lower overall adipose tissue mass in a trained individual (64).

In the past decade, it has been discovered that adipose tissue is significantly more metabolically active than was previously known. Today, it is known that several potent peptides are released from adipose tissue and have important effects on other organs in the body. Two such peptides are leptin, which has an anorectic effect on the energy balance and also affects sugar metabolism, and adiponectin, which stimulates fat burning. Adipose tissue also releases pro-inflammatory proteins such as tumour-necrosis factor alpha (TNF- α) and other cytokines and acute phase proteins. Angiotensinogen (AGT) formed in adipose tissue affects blood pressure, and can play a role in the blood pressure increase seen in overweight individuals. It has not been established how physical activity and exercise training affect these factors, but the decreased fat mass seen with exercise training can be expected to decrease the significance of these factors (88). Leptin has been examined in several studies, but there does not appear to be any unambiguous effect of exertion or exercise training on leptin levels. However, lower plasma levels of TNF- α have been observed among well-trained people, which is of interest because TNF- α formed by adipose tissue is considered to result in diminished tissue sensitivity to insulin, primarily in skeletal muscles. A decreased level of TNF- α could therefore contribute to the greater insulin sensitivity that exercise training entails (65).

Nervous system

Much of the knowledge that applies to the effects of acute exertion and exercise training on the nervous system is gathered from studies of animals, but growing numbers of human studies of cognition and learning are being published.

Acute exertion

During exertion, the brain has a total metabolism and total blood flow that do not significantly differ from that at bodily rest. However, during exertion, the activity, metabolism and blood flow in the areas that take care of motor activity increase measurably. The glucose concentration increases interstitially in the central nervous system (CNS) regardless of the blood sugar concentration. Besides glucose, the brain uses lactic acid as an energy substrate under intense exertion. The release of neurotransmitters (signal substances) such as dopamine, serotonin and glutamate in various parts of the brain are also affected during physical exertion.

Effects of exercise training

Regular physical activity affects several different functions in the human nervous system (89). Functions connected more directly to physical activity improve, such as coordination,

balance and reaction ability. This increases the functional ability, which can contribute to the increased well-being which is tied to regular physical activity. Moreover, cognitive ability (especially planning and coordination of tasks) is retained better, sleep quality is improved, depression symptoms decrease and self-esteem improves. Experiments in animals have shown that growth factors significant to cells in the central nervous system are affected by physical activity (66). In the hippocampus (important to memory formation), the gene expression of a large number of factors increases. For example, the occurrence of IGF-1, a very important growth factor, increases. The occurrence of noradrenaline increases in the brain. There are also studies that indicate that the new formation of brain cells increases in animals that are allowed to run (67). These animals also show better learning ability. Other studies have shown that the new formation of vessels increases in the cerebral cortex after exercise training, which can be of significance to the supply of nutrients. In cells in the peripheral nervous system, studies in animals have shown that markers for oxidative capacity/aerobic capacity increase. In addition, there are findings that indicate that cell size can increase with regular physical activity.

Mechanisms

The increased metabolism associated with more activity in parts of the cells of the brain, spinal cord and peripheral nervous system entails an effect on gene activity, in part caused by increased production of growth factors such as brain-derived neurotrophic factor (BDNF), nerve growth factor (NGF) and galanin (68). Local hypoxia may potentially drive the formation of new blood vessels around the brain cells.

Skin

Acute exertion

Under acute exertion, especially prolonged exertion in heat, perfusion of the skin increases sharply and the degree of sweating can be multiplied many times over. A well-trained person can excrete 2–3 litres of sweat per hour under extreme conditions. Various hormones affect the sweat glands so that salt is largely saved.

Effects of exercise training

Exercise improves sweating function and thereby heat-regulation capacity. Therefore, a well-trained person has better heat tolerance at rest and under exertion. Among other factors, this is due to altered perfusion and changed gene expression in the cells of the sweat glands. Regular physical activity reduces the amount of subcutaneous fat.

Gastrointestinal tract and liver

Acute exertion

The gastrointestinal tract is affected in many ways during and after acute exertion (69). Under strenuous exertion, symptoms occur such as stomach aches, diarrhoea, etc. It is not easy to determine the degree to which such symptoms are related to stress, dietary and fluid intake, or the physical exertion. The frequency at which the stomach empties decreases, at most during strenuous exertion. Besides motility, digestion and absorption, the gastrointestinal tract's blood flow, its secretion of hormones and other factors are affected. The stomach's lymphoid tissue and the mucous membranes' immunological functions, such as IgA, are also affected. Under exertion, the liver increases its glycogenolysis, which contributes to maintaining blood sugar (see below under "Hormone system").

Effects of exercise training

A well-trained person has a higher gastric emptying rate. The risk of the formation of gall stones is also reduced.

Hormone system

Acute exertion

Several hormone systems are activated under exertion, and physical activity entails increased plasma concentrations of multiple hormones, such as adrenaline/noradrenaline, adrenocorticotrophic hormone (ACTH), cortisol, beta endorphin, growth hormone, renin, testosterone, thyroid hormone and several gastrointestinal hormones. The levels of glucagon in arterial blood is only affected to a small degree by physical exertion, while the concentration of insulin decreases (70). The decrease in the insulin level in plasma during exertion, which can be very sharp (a drop to half or less of the resting level), is probably mediated by the increased activity in sympathetic nerves and by small reductions in the blood glucose level during the exertion. The latter explains why the insulin decrease under exertion is counteracted or even converted to an insulin increase upon sugar intake during the exercise session. Because glucagon, like insulin, exerts a significant part of its effect on the liver, one is at risk of underestimating the significance of glucagon under exertion when measuring arterial concentrations since the concentration in the portal vein, which is the concentration "seen" by the liver, is significantly higher and probably significantly raised under exertion (71).

The catecholamines adrenaline and noradrenaline increase sharply and exponentially with increasing work load. The source of the circulating adrenaline is the adrenal medulla and the increase in plasma adrenaline in physical exertion is due to increased sympathetic nerve activation of this organ. Although the blood's noradrenaline also comes in part from the adrenal medulla, the most important cause of the sharp rise in the plasma content of noradrenaline under exertion is a "flood" of noradrenaline from the sympathetic nerves. The most important sympathetic nerves in this respect are those stimulating a higher heart

rate and cardiac contractile force during exertion, as well as those which innervate the liver and adipose tissue. It is believed that a lower glucose concentration in the portal vein is an important cause of the strong activation of the sympathetic nervous system under exertion. The increase in noradrenaline starts at a lower work load than the increase in adrenaline, and noradrenaline also increases more sharply when exertion intensity increases. These hormones can increase 10–20 fold in strenuous or prolonged exertion. The noradrenaline content of the blood is often raised for several hours after the end of exertion, while the adrenaline concentration goes back to resting values within a few minutes (72).

The liver's greater release of glucose is one of the most important metabolic changes under exertion and compensates for the muscles' increased glucose uptake without the blood glucose level dropping too much. It is practically entirely caused by the changes in insulin and glucagon (73). The reduction of the plasma insulin level that takes place with exertion is believed to make the liver more sensitive to the glycogen-degrading effect of glucagon. The increased activation of the sympathetic nervous system during physical exertion appears to lack any direct significance to the liver's increased glucose release. However, under prolonged exertion, when the adrenaline levels are at their highest, adrenaline can have some stimulatory effect on the liver's glucose release in addition to glucagon. Adrenaline and noradrenaline are mainly of significance to the carbohydrate metabolism at the muscle level by making the muscle's glycogen degradation process sensitive to the stimulatory effect that the contraction process (actually the calcium ions that are released) has. However, if prolonged exercise leads one to "hit the wall" due to a blood glucose reduction, a crisis reaction is triggered, whereby adrenaline is released, which leads to an increase in the liver's glucose release. The liver's limited glycogen deposits mean that new synthesis of glycogen in the liver (so-called gluconeogenesis) becomes important in prolonged exertion (in addition to the sugar consumed by drinking). Here, the hormone cortisol plays an indirect role by increasing the capacity of the enzymatic machinery that takes care of this process.

Another crucial enzymatic process during physical exertion is the release of free fatty acids from the body's fat deposits, since free fatty acids are the body's other important nutrient during exertion. Here, noradrenaline, released by the sympathetic nerves that innervate adipose tissue, plays the most important role. Insulin has an inhibitory effect on the release of fatty acids, although this effect is diminished by its plasma concentration dropping sharply during exertion.

Increased levels of beta endorphines during prolonged exercise can be of significance to well-being and blood pressure reduction in connection with an exercise session (74).

Effects of exercise training

Naturally, lower hormone responses at a given work load are observed among well-trained than among untrained individuals. This applies to the increases in noradrenaline, adrenaline, growth hormone, ACTH and glucagon as well as the reduction in insulin. The reduced hormonal activation during exertion among well-trained persons is particularly notable with regard to the sympathetic nervous system, where the change occurs rapidly, normally during the first two weeks of exercise (75). The physiological mechanism behind

this rapid change is unknown, but the activation of stress hormones that occurs with other stress stimuli is not reduced among fit individuals. It is also well-known that the adrenal medulla's capacity to excrete adrenaline is greater among well-trained individuals (sports adrenal medulla).

The so-called hypothalamus-pituitary-adrenal (HPA) axis is a messenger for the body's responses to various states of stress. The resting state of the HPA system is affected by regular endurance training so that the daily rhythm is shifted (the morning peak comes earlier) and the release of the pituitary gland's control hormone ACTH is increased. Although this can be interpreted as a hormonal stress state in the trained body, the effector hormone of the HPA axis, cortisol, does not change its resting level as a result of regular exercise, however. This apparent paradox seems to be explained by the fact that cortisol provides less effective feedback inhibition of the pituitary and possibly of the hypothalamus in well-trained individuals, which leads to an increased level of ACTH (76, 77). This is suspected to be one of several different explanations of the menstruation disruptions that occur in female athletes. Disturbances of the reproductive system in male athletes are rarely discussed, but may also exist (90).

Well-trained individuals have lowered insulin concentrations in plasma, both basally and after sugar intake, due to both a reduced release of insulin from the islets of Langerhans (78) and an increased tissue sensitivity to insulin (79). The increased insulin sensitivity is strongly linked to the reduced risk of having cardiovascular disease that is characteristic of physically trained individuals. As described above in the section on adipose tissues, regular exercise leads to an increased capacity for lipolysis in the adipose tissue. This contributes to a well-trained person being able to maintain a sufficient fat release during physical exertion even though the activation of the sympathetic nervous system, which controls lipolysis, is sharply reduced. Regular exercise has a carbohydrate-saving effect by a large part of the energy need being met with the burning of fat. This is registered by the liver and, after just 10 days of exercise, the liver's glucose release during a two-hour exercise bout can be reduced by 25 per cent (80). In spite of this, regular physical exercise leads to a greater capacity for gluconeogenesis in the liver.

Table 1. The effect of endurance training on a few physiological reactions during sub-maximum and maximum exertion. The direction of the arrows or ± 0 indicate the change compared with an untrained state.

	Submax.	Max.
VO ₂ *	± 0	↑
Blood pressure	↓	± 0
Heart rate	↓	± 0
Pulmonary ventilation	↓	
Muscle metabolism		
Glycogen metabolism	↓	only glycogen
Fatty acid metabolism	↑	none
Lactic acid formation	↓	
Sweating	↑	↑
Body temperature	↓	± 0
Adrenaline concentration in blood	↓	↑

* VO₂ = oxygen uptake capacity.

Acknowledgement

The authors are grateful to Sigmund B. Strømme, Professor emeritus, Norwegian School of Sport Sciences, Oslo, for constructive points of view and updates.

References

1. Shephard RJ, Balady GJ. Exercise as cardiovascular therapy. *Circulation* 1999;99:963-72.
2. Saltin B, Gollnick PD. Skeletal muscle adaptability. Significance for metabolism and performance. In: Peachey L, Adrian R, Gaiger S, eds. *Handbook of physiology*. Section 10. Skeletal muscle. Baltimore: Williams & Wilkins Company; 1983. pp. 555-631.
3. Åstrand P-O, Rodahl K, Dahl HA, Strömme SB. *Textbook of work physiology. Physiological bases of exercise*. 4. edn. Champaign (IL): Human Kinetics; 2003.
4. Wilmore JH, Costill DL. *Physiology of sport and exercise*. 3. edn. Champaign (IL): Human Kinetics; 2004.
5. McArdle WD, Katch FI, Katch VL. *Exercise physiology. Energy, nutrition, and human performance*. 5. edn. Philadelphia: Lippincott Williams & Wilkins; 2001.
6. Booth FW, Chakravarthy MV, Gordon SE, Spangenburg EE. Waging war on physical inactivity. Using modern molecular ammunition against an ancient enemy. *J Appl Physiol* 2002;93:3-30.
7. Timmons JA, Jansson E, Fischer H, Gustafsson T, Greenhaff PL, Riddén J, et al. Modulation of extracellular matrix genes reflects the magnitude of physiological adaptation to aerobic exercise training in humans. *BMC Biol* 2005;3:19.
8. Timmons JA, Larsson O, Jansson E, Fischer H, Gustafsson T, Greenhaff PL, et al. Human muscle gene expression responses to endurance exercise provide a novel perspective on Duchenne muscular dystrophy. *FASEB J* 2005;19:750-60.
9. Rankinen T, Bray MS, Hagberg JM, Pérusse L, Roth SM, Wolfarth B, et al. The human gene map for performance and health-related fitness phenotypes. The 2005 update. *Med Sci Sports Exerc* 2006;38:1863-88.
10. Borg GA. Psychophysical bases of perceived exertion. *Med Sci Sports Exerc* 1982;14:377-81.
11. Crouter SE, Churilla JR, Bassett Jr DR. Estimating energy expenditure using accelerometers. *Eur J Appl Physiol* 2006;98:601-12.
12. Bassett Jr DR, Howley ET. Limiting factors for maximum oxygen uptake and determinants of endurance performance. *Med Sci Sports Exerc* 2000;32:70-84.
13. Svedahl K, MacIntosh BR. Anaerobic threshold. The concept and methods of measurement. *Can J Appl Physiol* 2003;28:299-323.
14. Herd SL, Kiens B, Boobis LH, Hardman AE. Moderate exercise, postprandial lipidemia, and skeletal muscle lipoprotein lipase activity. *Metabolism* 2001;50:756-62.
15. Fields DA, Goran MI, McCrory MA. Body-composition assessment via air-displacement plethysmography in adults and children. A review. *Am J Clin Nutr* 2002;75:453-67.
16. Gleeson M, Pyne DB, Callister R. The missing link in exercise effects on mucosal immunity. *Exerc Immunol Rev* 2004;10:107-28.
17. Bowles DK, Woodman CR, Laughlin MH. Coronary smooth muscle and endothelial adaptations to exercise training. *Exerc Sport Sci Rev* 2000;28:57-62.

18. Zanesco A, Antunes E. Effects of exercise training on the cardiovascular system. Pharmacological approaches. *Pharmacol Ther* 2007;114:307-17.
19. Achten J, Jeukendrup AE. Optimizing fat oxidation through exercise and diet. *Nutrition* 2004;20:716-27.
20. Doherty TJ. Effects of short-term training on physiologic properties of human motor units. *Can J Appl Physiol* 2000;25:194-203.
21. Green HJ. Adaptations in the muscle cell to training. Role of the Na⁺-K⁺-ATPase. *Can J Appl Physiol* 2000;25:204-16.
22. Hawley JA. Adaptations of skeletal muscle to prolonged, intense endurance training. *Clin Exp Pharmacol Physiol* 2002;29:218-22.
23. Gustafsson T, Rundqvist H, Norrbom J, Rullman E, Jansson E, Sundberg CJ. The influence of physical training on the angiotensin and VEGF-A systems in human skeletal muscle. *J Appl Physiol* 2007;103:1012-20.
24. Widegren U, Wretman C, Lionikas A, Hedin G, Henriksson J. Influence of exercise intensity on ERK/MAP kinase signalling in human skeletal muscle. *Pflugers Arch* 2000; 441:317-22.
25. Bengtsson J, Gustafsson T, Widegren U, Jansson E, Sundberg CJ. Mitochondrial transcription factor A and respiratory complex IV increase in response to exercise training in humans. *Pflügers Arch – Eur J of Physiol* 2001;443:61-6.
26. Sawka MN, Convertino VA, Eichner ER, Schnieder SM, Young AJ. Blood volume. Importance and adaptations to exercise training, environmental stresses, and trauma/sickness. *Med Sci Sports Exerc* 2000;32:332-48.
27. Thompson PD, Crouse SF, Goodpaster B, Kelley D, Moyna N, Pescatello L. The acute versus the chronic response to exercise. *Med Sci Sports Exerc* 2001;33:S438-45, discussion S452-3.
28. Park DH, Ransone JW. Effects of submaximal exercise on high-density lipoprotein-cholesterol subfractions. *Int J Sports Med* 2003;24:245-51.
29. Petitt DS, Cureton KJ. Effects of prior exercise on postprandial lipemia. A quantitative review. *Metabolism* 2003;52:418-24.
30. Gill JM, Hardman AE. Exercise and postprandial lipid metabolism. An update on potential mechanisms and interactions with high-carbohydrate diets. Review. *J Nutr Biochem* 2003;14:122-32.
31. Durstine JL, Grandjean PW, Davis PG, Ferguson MA, Alderson NL, DuBose KD. Blood lipid and lipoprotein adaptations to exercise. A quantitative analysis. *Sports Med* 2001;31:1033-62.
32. Kraus WE, Houmard JA, Duscha BD, Knetzger KJ, Wharton MB, McCartney JS, et al. Effects of the amount and intensity of exercise on plasma lipoproteins. *N Engl J Med* 2002;347:1483-92.
33. El-Sayed MS. Exercise and training effects on platelets in health and disease. *Platelets* 2002;13:261-6.

34. Mittleman MA, Maclure M, Tofler GH, Sherwood JB, Goldberg RJ, Muller JE. Triggering of acute myocardial infarction by heavy physical exertion. Protection against triggering by regular exertion. Determinants of Myocardial Infarction Onset Study Investigators. *N Engl J Med* 1993;329:1677-83.
35. Willich SN, Lewis M, Lowel H, Arntz HR, Schubert F, Schroder R. Physical exertion as a trigger of acute myocardial infarction. Triggers and Mechanisms of Myocardial Infarction Study Group. *N Engl J Med* 1993;329:1684-90.
36. Sundberg CJ, Jansson E. Reduced morbidity and the risk of premature death. Regular physical exercise is beneficial for health at all ages. *Läkartidningen* 1998;95:4062-7.
37. Wang JS, Jen CJ, Chen HI. Effects of exercise training and deconditioning on platelet function in men. *Arterioscler Thromb Vasc Biol* 1995;15:1668-74.
38. Rauramaa R, Li G, Vaisanen SB. Dose-response and coagulation and hemostatic factors. *Med Sci Sports Exerc* 2001;33:S516-20, discussion S528-9.
39. Malm C, Celsing F, Friman G. Fysisk aktivitet både stimulerar och hämmar immunförsvaret. [Physical activity both stimulates and inhibits the immune defence.] *Läkartidningen* 2005;102:867-73.
40. Nieman DC. Special feature for the olympics. Effects of exercise on the immune system. Exercise effects on systemic immunity. *Immunol Cell Biol* 2000;78:496-501.
41. Gleeson M, Pyne DB. Special feature for the olympics. Effects of exercise on the immune system. Exercise effects on mucosal immunity. *Immunol Cell Biol* 2000;78:536-44.
42. Gleeson M. Special feature for the olympics. Effects of exercise on the immune system. Overview. Exercise immunology. *Immunol Cell Biol* 2000;78:483-4.
43. Russo CR, Lauretani F, Bandinelli S, Bartali B, Di Iorio A, Volpato S, et al. Aging bone in men and women. Beyond changes in bone mineral density. *Osteoporos Int* 2003;14:531-8.
44. Heinonen A, Sievanen H, Kannus P, Oja P, Vuori I. Site-specific skeletal response to long-term weight training seems to be attributable to principal loading modality. A pQCT study of female weightlifters. *Calcif Tissue Int* 2002;70:469-74.
45. Saino H, Luther F, Carter DH, Natali AJ, Turner DL, Shahtaheri SM, et al. Evidence for an extensive collagen type III proximal domain in the rat femur. II. Expansion with exercise. *Bone* 2003;32:660-8.
46. Kontulainen S, Sievanen H, Kannus P, Pasanen M, Vuori I. Effect of long-term impactloading on mass, size, and estimated strength of humerus and radius of female racquetsports players. A peripheral quantitative computed tomography study between young and old starters and controls. *J Bone Miner Res* 2003;18:352-9.
47. Turner CH, Takano Y, Owan I. Aging changes mechanical loading thresholds for bone formation in rats. *J Bone Miner Res* 1995;10:1544-9.
48. Lanyon LE. Functional strain in bone tissue as an objective, and controlling stimulus for adaptive bone remodelling. *J Biomech* 1987;20:1083-93.
49. Balasch J. Sex steroids and bone. Current perspectives. *Hum Reprod Update* 2003;9:207-22.

50. Kannus P, Haapasalo H, Sankelo M, Sievanen H, Pasanen M, Heinonen A, et al. Effect of starting age of physical activity on bone mass in the dominant arm of tennis and squash players. *Ann Intern Med* 1995;123:27-31.
51. Karlsson M. Does exercise reduce the burden of fractures? A review. *Acta Orthop Scand* 2002;73:691-705.
52. Midtby M, Magnus JH. Normal bone remodelling. What can go wrong in osteoporosis? *Tidsskr Nor Laegeforen* 1998;118:552-7.
53. Nichols JF, Palmer JE, Levy SS. Low bone mineral density in highly trained male master cyclists. *Osteoporos Int* 2003;14:644-9.
54. Rideout CA, McKay HA, Barr SI. Self-reported lifetime physical activity and areal bone mineral density in healthy postmenopausal women. The importance of teenage activity. *Calcif Tissue Int* 2006;79:214-22.
55. Rikkinen T, Tuppurainen M, Kröger H, Jurvelin J, Honkanen R. Distance of walking in childhood and femoral bone density in perimenopausal women. *Eur J Appl Physiol* 2006;97:509-15.
56. Lynch NA, Ryan AS, Evans J, Katzell LI, Goldberg AP. Older elite football players have reduced cardiac and osteoporosis risk factors. *Med Sci Sports Exerc* 2007;39:1124-30.
57. Karlsson MK, Nordqvist A, Karlsson C. Physical activity, muscle function, falls and fractures. *Food Nutr Res.* 2008; 52: Published online 2008 December 30.
58. Eckstein F, Tieschky M, Faber S, Englmeier KH, Reiser M. Functional analysis of articular cartilage deformation, recovery, and fluid flow following dynamic exercise in vivo. *Anat Embryol (Berl)* 1999;200:419-24.
59. Saxon L, Finch C, Bass S. Sports participation, sports injuries and osteoarthritis. Implications for prevention. *Sports Med* 1999;28:123-35.
60. Kjaer M, Langberg H, Magnusson P. Overuse injuries in tendon tissue. Insight into adaptation mechanisms. *Ugeskr Laeger* 2003;165:1438-43.
61. Eisenmann JC, Bartee RT, Wang MQ. Physical activity, TV viewing, and weight in U.S. youth. 1999 Youth Risk Behavior Survey. *Obes Res* 2002;10:379-85.
62. Hill JO, Wyatt HR, Reed GW, Peters JC. Obesity and the environment. Where do we go from here? *Science* 2003;299:853-5.
63. Ballor DL, Keesey RE. A meta-analysis of the factors affecting exercise-induced changes in body mass, fat mass and fat-free mass in males and females. *Int J Obes* 1991;15:717-26.
64. Enevoldsen LH, Stallknecht B, Langfort J, Petersen LN, Holm C, Ploug T, et al. The effect of exercise training on hormone-sensitive lipase in rat intra-abdominal adipose tissue and muscle. *J Physiol* 2001;536:871-7.
65. Straczkowski M, Kowalska I, Dzienis-Straczkowska S, Stepień A, Skibinska E, Szelachowska M, et al. Changes in tumor necrosis factor-alpha system and insulin sensitivity during an exercise training program in obese women with normal and impaired glucose tolerance. *Eur J Endocrinol* 2001;145:273-80.
66. Cotman CW, Engesser-Cesar C. Exercise enhances and protects brain function. *Exerc Sport Sci Rev* 2002;30:75-9.

67. van Praag H, Christie BR, Sejnowski TJ, Gage FH. Running enhances neurogenesis, learning, and long-term potentiation in mice. *Proc Natl Acad Sci USA* 1999;96:13427-31.
68. Dishman RK, Berthoud HR, Booth FW, Cotman CW, Edgerton VR, Fleshner MR, et al. Neurobiology of exercise. *Obesity* 2006;14:345-56.
69. Moses FM. The effect of exercise on the gastrointestinal tract. *Sports Med* 1990;9:159-72.
70. Galbo H. The hormonal response to exercise. *Diabetes Metab Rev* 1986;1:385-408.
71. Wasserman DH, Lacy DB, Bracy DP. Relationship between arterial and portal vein immunoreactive glucagon during exercise. *J Appl Physiol* 1993;75:724-9.
72. Christensen NJ, Galbo H, Hansen JF, Hesse B, Richter EA, Trap-Jensen J. Catecholamines and exercise. *Diabetes* 1979;28:58-62.
73. Wasserman DH, Lickley HL, Vranic M. Interactions between glucagon and other counterregulatory hormones during normoglycemic and hypoglycemic exercise in dogs. *J Clin Invest* 1984;74:1404-13.
74. Jonsdottir IH, Hoffmann P, Thoren P. Physical exercise, endogenous opioids and immune function. *Acta Physiol Scand Suppl* 1997;640:47-50.
75. Winder WW, Hagberg JM, Hickson RC, Ehsani AA, McLane JA. Time course of sympathoadrenal adaptation to endurance exercise training in man. *J Appl Physiol* 1978;45:370-4.
76. Wittert GA, Livesey JH, Espiner EA, Donald RA. Adaptation of the hypothalamo-pituitary adrenal axis to chronic exercise stress in humans. *Med Sci Sports Exerc* 1996;28:1015-9.
77. Duclos M, Corcuff JB, Arsac L, Moreau-Gaudry F, Rashedi M, Roger P, et al. Corticotroph axis sensitivity after exercise in endurance-trained athletes. *Clin Endocrinol (Oxf)* 1998;48:493-501.
78. Wasserman DH. Regulation of glucose fluxes during exercise in the postabsorptive state. *Ann Rev Physiol* 1995;57:191-218.
79. Henriksson J. Influence of exercise on insulin sensitivity. *J Cardiovasc Risk* 1995;2:303-9.
80. Mendenhall LA, Swanson SC, Habash DL, Coggan AR. Ten days of exercise training reduces glucose production and utilization during moderate-intensity exercise. *Am J Physiol* 1994;266:E136-43.
81. Gibala M. Molecular responses to high intensity interval exercise. *Appl Physiol Nutr Metab* 2009;34:428-32.
82. Hood DA. Mechanisms of exercise-induced mitochondrial biogenesis in skeletal muscle. *Appl Physiol Nutr Metab* 2009;34:465-72.
83. Vislocky LM, Pikosky MA, Herron Rubin K, Vega-Lopez S, Courtney Gaine P, Martin WF, Zern TL, Lofgren IE, Luz Fernandez M, Rodriguez NR. Habitual consumption of eggs does not alter the beneficial effects of endurance training on plasma lipids and lipoprotein metabolism in untrained men and women. *Journal of Nutritional Biochemistry* 2009; 20:26-34.

84. Laughlin MH, Roseguini B. Mechanisms for exercise training-induced increases in skeletal muscle blood flow capacity: differences with interval sprint training versus aerobic endurance training. *J Physiol Pharmacol* 2008;59 Suppl 7:71-88.
85. Moreira A, Delgado L, Moreira P, Haahtela T. Does exercise increase the risk of upper respiratory tract infections? *Br Med Bull.* 2009;90:111-31.
86. Mathur N, Pedersen BK. Exercise as a mean to control low-grade systemic inflammation. *Mediators Inflamm.* Epub 2009.
87. Hunter DJ, Eckstein F. Exercise and osteoarthritis. *J Anat.* 2009;214(2):197-207.
88. van Praag H. Exercise and the brain: something to chew on. *Trends Neurosci.* 2009 May;32(5):283-90.
89. van Praag H. Exercise and the brain: something to chew on. *Trends Neurosci.* 2009 May;32(5):283-90.
90. Hackney AC. Effects of endurance exercise on the reproductive system of men: the "exercise-hypogonadal male condition". *J Endocrinol Invest.* 2008;31:932-8.

2. General recommendations regarding physical activity

Authors

Eva Jansson, MD, PhD, Professor, Department of Laboratory Medicine, Division of Clinical Physiology, Karolinska Institutet, Karolinska University Hospital, Stockholm, Sweden

Sigmund A Anderssen, PhD, Professor, Norwegian School of Sports Sciences, Oslo, Norway

Summary

The total amount of physical activity, a combination of intensity, duration and frequency, is related to various health variables in a so-called dose-response relationship. This chapter describes recommendations on physical activity, both in general and in relation to aerobic fitness, strength and flexibility. The link between physical activity, health and physical capacity is also described, as well as the scientific background of the current recommendations in brief. To facilitate the prescription of physical activity, a strategy is also outlined for the application of the recommendations through the activity pyramid.

A summary of the health-enhancing recommendations:

All individuals should be physically active for a combined minimum of 30 minutes, preferably every day. The intensity should at least be moderate, such as a brisk walk. Additional health effects can be achieved if the daily amount or intensity is increased beyond this. In addition, an updated recommendation from the U.S. also includes strength and flexibility training.

Activity pyramid

To make it easier to prescribe physical activity, the activity pyramid can be an aid (see figure 1). The principle is that the activities further down in the pyramid are done more often and at a lower intensity than the activities higher up. One should accordingly proceed from the pyramid's base to plan which daily activities can be done. Thereafter, an assessment is made of if and when it would be appropriate to expand the activities, or move up the pyramid to achieve additional health benefits, based on the individual's needs,

interests and previous and current experience of physical activity, physical capacity and state of health. Activities higher up in the pyramid also provide improvements in aerobic fitness, strength and flexibility.

Note that in some cases, one must begin further up in the pyramid. Chronic disease and/or an advanced age may have led to such weak muscles that strength training, for instance, may be the type of exercise that must precede other exercise to make walking, one of the pyramid’s “base activities”, possible at all.

Flexibility training should be performed at least 2–3 times per week, such as stretching (10–30 seconds), 4 times per muscle group.

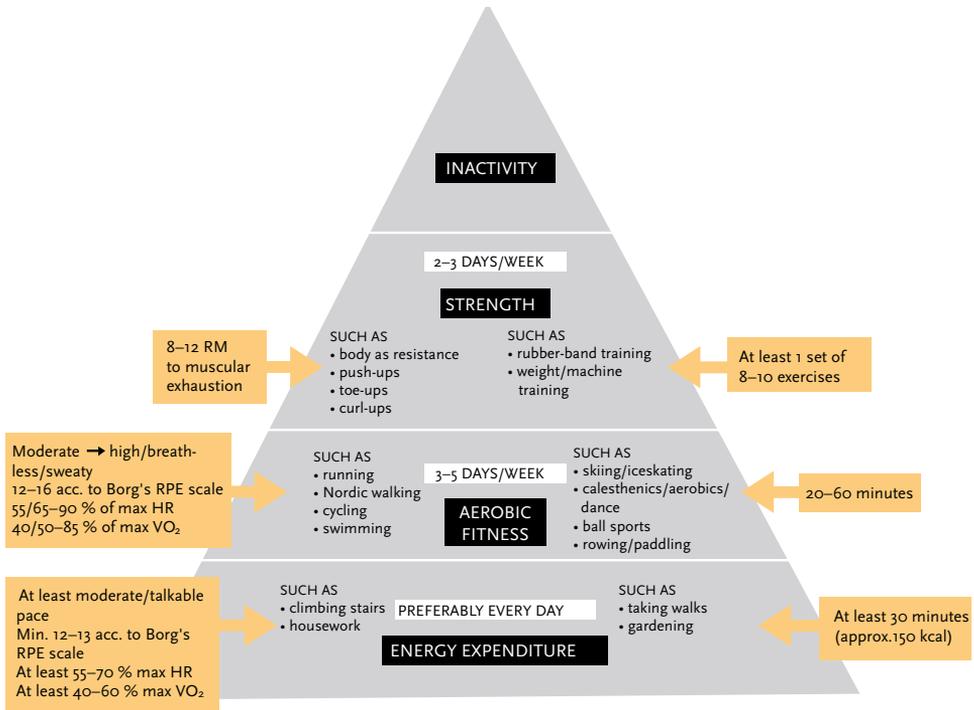


Figure 1. The activity pyramid.

Proceed from the pyramid’s base to plan which daily activities can be done. Thereafter, an assessment should be made of if and when it would be appropriate to expand the activities, or move up the pyramid to achieve additional health benefits, based on the individual’s/patient’s needs, interests and previous and current experience of physical activity, physical capacity and state of health. Activities higher up in the pyramid also provide improvements in aerobic fitness, strength and flexibility.

Note that in some cases, one must begin further up in the pyramid. Chronic disease and/or an advanced age may have led to such weak muscles that strength training, for instance, may be the type of exercise that must precede other exercise to make walking, one of the pyramid’s “base activities”, possible at all. Flexibility training should be performed at least 2–3 times per week, such as stretching (10–30 seconds), 4 times per muscle group. 8–12 RM = the highest load that can be lifted through the entire range of motion 8–12 times.

Health-enhancing recommendations (table 1)

Recommendations for physical activity are based on the knowledge that exists regarding the relationship between physical activity and health. Physical activity has various dimensions such as intensity, duration and frequency. Overall physical activity (a combination of the aforementioned factors) is related to various health variables in a so-called dose-response relationship. This relationship between physical activity and health benefits (risk reduction) appears as a continuum, which does not appear to have any lower boundary. The various health parameters (such as those with regard to osteoporosis, mental illness, obesity and risk factors for cardiovascular disease) probably have different dose-response relationships, but this has not been sufficiently studied. The recommendations for physical activity and expected health effects are also reliant on the starting point, both with regard to activity level and risk profile. This means that the lower the activity level and the worse the risk profile is, the greater the effect can be expected to be if the activity level increases. It has accordingly been shown that the largest health difference is between people that are physically inactive and those that are a little physically active. This means that significant health benefits can be achieved through regular, moderate physical activity.

On behalf of the Swedish National Institute of Public Health, Professional Associations for Physical Activity (YFA) has prepared recommendations on physical activity. The Swedish Society of Medicine's Board adopted the recommendation in September 2000, which is worded as follows:

All individuals should be physically active for a combined minimum of 30 minutes, preferably every day. The intensity should at least be moderate, such as a brisk walk. Additional health effects can be achieved if the daily amount or intensity is increased beyond this. Physically active individuals run half the risk of dying from cardiovascular disease as sedentary persons of the same age. Physical activity also decreases the risk of having high blood pressure, age-related diabetes and colon cancer. Quality of life is also improved by physical activity due to greater mental well-being and better physical health. There is also strong support for physically active individuals having a lower risk of being affected by brittle bones, bone fractures caused by falls, blood clots, obesity and mental disorders. In light of this, all clinically active physicians should advise their patients regarding physical activity adjusted to their state of health and personal lifestyle.

This recommendation is based on a U.S. recommendation published in 1995 (1) by 20 experts in the areas of epidemiology, physiology and medicine. The scientific support structure was further deepened in the report *Physical activity and health. A report of the Surgeon General* (2) and has recently been updated and clarified by the American College of Sports Medicine and the American Heart Association (3). In 2008, this resulted the Physical Activity Guidelines for Americans (27).

In summary, the Physical Activity Guidelines for Americans from 2008 emphasizes that:

1. The intensity should be at least moderate, which means that one is able to talk, but not to sing, i.e. heart rate and breathing will be increased. This type of exercise is denoted as aerobic physical activity in these guidelines.
2. The duration of each exercise session should not be less than 10 minutes. It is not enough to just walk a few minutes back and forth to the parking lot.
3. Exercise of moderate intensity can be replaced by exercise of higher intensity (vigorous intensity). For instance, walking 30 minutes per day can be replaced by running 20–30 minutes, 3–4 times per week. The same amount of energy is expended in these two examples, but in a shorter period of time in the alternative with higher intensity. Consequently, the expected health benefits are considered to be the same.
4. The goals are set per week instead of per day – 150 minutes of brisk walking per week (moderate intensity) or 75 minutes of running (vigorous intensity). It is also possible to mix these two intensity levels over the week. The activity should be spread throughout the week.
5. Everyone is recommended to do strength training and flexibility exercises at least two times per week according to table 2.
6. Balance training is important for the elderly.
7. Children need at least 60 minutes per day of moderate to vigorous activity.
8. More health benefits are achieved if the amount of physical activity is increased from 150 to 300 minutes if the intensity is moderate and from 75 to 150 minutes if the intensity is vigorous.

As previously mentioned, the health-enhancing recommendations are based on a dose-response relationship between the amount of physical activity (the product of intensity, duration and frequency) on the one hand and morbidity and death from cardiovascular disease or diabetes on the other (1, 2, 5–7). The amount of physical activity can be expressed with the help of energy measurements, such as kilocalories (kcal) or kilojoules (kJ). These relationships are based on epidemiological studies, meaning studies at a population level that often include thousands of individuals. The physical activity is, however, self-selected, which may be a weakness in scientific contexts, but there are growing numbers of randomised studies that unequivocally support these epidemiological studies and thereby strengthen the causal relationship between physical activity and disease (8–11). For in-depth reading, see references 12–15.

Recommendations for aerobic fitness, strength and flexibility (table 2)

The American College of Sports Medicine (ACSM) published the first version concerning aerobic fitness training in 1978. In the second version from 1990, strength and flexibility training were also included. The latest and third version from 1998 (16) regarding aerobic fitness, strength and flexibility is somewhat modified and compares the

recommendations concerning aerobic fitness, strength and flexibility with the “health-enhancing” recommendations.

In contrast to the health-enhancing recommendations, recommendations for aerobic fitness and strength are based on a dose-response relationship between the exercise intensity, duration or frequency on one hand and measurements of aerobic fitness, such as maximal oxygen uptake or measurements of strength, on the other. These relationships are most often obtained through experimental studies on significantly fewer individuals than in the epidemiological studies. *Note that the training of aerobic fitness and strength also leads to improved health in addition to providing specific effects in the form of improved physical capacity.*

What distinguishes and merges the current recommendations?

1. Target group

The health-enhancing recommendations address *everyone* while the ACSM recommendations regarding the training of physical capacity are limited to *healthy adult individuals*. This is because the intensity requirement is higher for the recommendations regarding physical capacity and consequently, the risks of negative effects also increase, particularly for individuals with chronic diseases. Special recommendations are therefore needed for these groups and individuals, which are presented in the various sections of FYSS.

2. Intensity

According to the *health-enhancing recommendations*, *intensity can be either moderate or more intense* to achieve positive health effects. Consequently, intensity is not directly decisive to the health effect, but rather the total energy expenditure seems to be more significant to the effect. However, with moderate intensity, the duration must be longer than with a higher intensity to achieve similar health effects (3, 5–7). However, at higher intensity, one should keep in mind that the body needs to recover for the optimal effect of the exercise, meaning that days of rest should be included.

In terms of *improving aerobic fitness and strength*, *a certain intensity must be achieved* to obtain optimal effects, an intensity that is higher than moderate for the majority of individuals (16).

3. Duration

According to the health-enhancing recommendations, the *duration* is given as “a combined minimum of 30 minutes” and the frequency as “preferably every day”. Combined means that one can accumulate activity over the day, such as three bouts of 10 minutes each. This is based on the fact that the activities mapped out in the major epidemiological studies, on

which the recommendations are based, may have been carried out intermittently during the day. Examples of such activities include climbing stairs, walking to and from work, and household and gardening work. However, in the latest U.S. recommendations, it has been established that the activity sessions should not be shorter than 10 minutes each (3, 4, 27).

The minimum amount given as 30 minutes of daily physical activity corresponds to a daily energy expenditure of approximately 150 kcal per day or approximately 1,000 kcal per week. The selection of 30 minutes (150 kcal) is based on studies in which it was found that the risk of premature death was already reduced at 70 kcal per day, but that the risk decreased further if energy expenditure amounted to approximately 150 kcal per day (17–19). The high frequency, meaning “preferably every day”, is significant to a high accumulated energy expenditure over time and to be able to “use” everyday activities at the same time. From a practical perspective, it is easier to incorporate physical activity in daily activities if everyday activities in particular can be used.

In the recommendations for aerobic fitness and strength in the latest version from 1998, the modification was introduced that aerobic exercise (20–60 minutes, 3–5 times per week) can be divided into several sessions during the day, although a minimum of 10 minutes per session. Experimental studies regarding the effect on maximal oxygen uptake support this modification (20–22).

The lower recommended frequency for the training of aerobic fitness and strength than in the health-enhancing recommendations, 3–5 times per week for aerobic fitness and 2–3 times per week for strength, is due to the body requiring periods of recovery for the exercise to have an optimal effect in exercise with higher intensity. In addition, the risk for strain injuries increases if the frequency is too high in aerobic and strength exercise.

4. The recommendations are “merged”

As previously mentioned, the intensity is different between both of the recommendations. The health-enhancing recommendations say that the intensity should be “at least moderate” to achieve health effects, an intensity that is too low to effectively improve aerobic fitness and strength for most individuals (table 2). However, the health-enhancing recommendations state that “additional health effects” beyond those achieved with, for example, a 30-minute brisk daily walk (moderate intensity), can be achieved if the amount and/or intensity are/is increased. If one opts to increase intensity, the two recommendations are “merged”, meaning that a more *high-intensity activity can provide both health benefits and greater aerobic fitness/strength* (3, 4, 27). This is on condition that the chosen intensity is not so high that the duration is extremely short and the energy expenditure is thereby below that which corresponds to a 30-minute brisk daily walk, or approximately 150 kcal per day. However, at higher intensity, the risks of cardiovascular complications increase (23). It should be noted that in regular exercise, the total risk across the day is reduced in terms of the risk of having a cardiovascular complication, although the risk during the actual exercise session is elevated (23). However, the increased risk in connection with a single exercise session appears to be lower for women than for men (24).

Table 1. Health-enhancing recommendation (1-4, Swedish Society of Medicine).

	Frequency	Intensity/load	Duration
Energy expenditure	Preferably every day	At least 55–70% of max HR* At least 40–60% of max VO ₂ ** At least 12–13 as per Borg's RPE scale*** At least a "talkable" pace	At least 30 min.

* Max HR = maximal heart rate.

** Max VO₂ = maximal oxygen uptake.

*** RPE = Borg's ratings of perceived exertion, scale 6–20 (25).

Table 2. Recommendations for aerobic fitness, strength and flexibility (3, 4, 16, 26).

	Frequency (days/week)	Intensity/load	Duration
Aerobic fitness training	3–5	55/65–90% of max HR* (40/50–85% max VO ₂ **) 12–16 according to Borg's RPE scale*** moderate → high/breathless/sweaty	20–60 min.
Strength training	2–3	8–12 RM**** (75% of 1 RM) Borg's RPE scale >16	At least 1 set of 8–10 exercises
Flexibility	2–3		(10–30 seconds) 4 times/muscle group

* Max HR = maximal heart rate.

** Max VO₂ = maximal oxygen uptake.

*** RPE = Borg's ratings of perceived exertion, scale 6–20 (25).

**** RM = repetition maximum. 1 RM corresponds to the highest load that can be lifted through the entire range of motion just once.

References

1. Pate RR, Pratt M, Blair SN, Haskell WL, Macera CA, Bouchard C, et al. Physical activity and public health. A recommendation from the Centers for Disease Control and Prevention and the American College of Sports Medicine. *JAMA* 1995;273:402-7.
2. U.S. Department of Health and Human Services. Physical activity and health. A report of the Surgeon General. Atlanta (GA): U.S. Department of Health and Human Services, Centers for Disease Control and Prevention, National Center for Chronic Disease. Prevention and Health Promotion; 1996.
3. Haskell WL, Lee IM, Pate RR, Powell KE, Blair SN, Franklin BA, et al. Physical activity and public health. Updated recommendation for adults from the American College of Sports Medicine and the American Heart Association. *Med Sci Sports Exerc* 2007;39:1423-34.
4. Nelson ME, Rejeski WJ, Blair SN, Duncan PW, King AC, Macera CA, et al. Physical activity and public health in older adults. Recommendation from the American College of Sports Medicine and the American Heart Association. *Med Sci Sports Med* 2007;39:1435-45.
5. Hu FB, Segal RJ, Rich-Edwards JW, Colditz GA, Solomon CG, Speizer FE, et al. Walking compared with vigorous physical activity and risk of type 2 diabetes in women. A prospective study. *JAMA* 1999;282:1433-9.
6. Manson J, Greenland P, La Croix AZ, Stefanick ML, Mouton CP, Oberman A, et al. Walking compared with vigorous exercise for the prevention of cardiovascular events in women. *New Engl J Med* 2002;347:716-25.
7. Manson JE, Hu FB, Rich-Edwards JW, Colditz GA, Stampfer MJ, Willett WC, et al. A prospective study of walking as compared with vigorous exercise in the prevention of coronary heart disease in women. *New Engl J Med* 1999;341:650-8.
8. Hambrecht R, Walther C, Mobius-Winkler S, Gielen S, Linke A, Conradi K, et al. Percutaneous coronary angioplasty compared with exercise training in patients with stable coronary artery disease. A randomized trial. *Circulation* 2004;109:1371-8.
9. Taylor RS, Brown A, Ebrahim S, Jolliffe J, Noorani H, Rees K, et al. Exercise-based rehabilitation for patients with coronary heart disease. Systematic review and meta-analysis of randomized controlled trials. *Am J Med* 2004;116:682-92.
10. Pan XR, Li GW, Hu YH, Wang JX, Yang WY, An ZX, et al. Effects of diet and exercise in preventing NIDDM in people with impaired glucose tolerance. The Da Qing IGT and diabetes study. *Diabetes Care* 1997;20:537-44.
11. Tuomilehto J, Lindstrom J, Eriksson JG, Valle TT, Hämäläinen H, Ilanne-Parikka P, et al. Prevention of type 2 diabetes mellitus by changes in lifestyle among subjects with impaired glucose tolerance. *New Engl J Med* 2001;344:1343-50.
12. Sutor CW, Kraak VI. Adequacy of evidence for physical activity guidelines development. Workshop summary. The National Academies 2007. ISBN 0-309-66777-1. www.nap.edu/catalog/11819.html.

13. Kiens B, Beyer N, Brage S, Hyldstrup L, Ottesen LS, Overgaard K, et al. Fysisk inaktivitet. Konsekvenser og sammenhaenge. En rapport fra Motions- og ernæringsrådet [Physical inactivity. Consequences and contexts. A report from the Danish Exercise and Nutrition Council.]; Publ. No. 3. 2007.
14. Pedersen BK, Saltin B. Fysisk aktivitet. Håndbog om forebyggelse og behandling. [Physical activity. Handbook on prevention and treatment.] National Danish Board of Health, National Centre for Health Promotion and Disease Prevention; 2003. GraPhia og revidering. ISBN elektronisk utgåva 87-91232-78-3. www.sst.dk/publikationer.
15. Pedersen BK, Saltin B. Evidence for prescribing exercise as therapy in chronic disease. *Scand J Med Sci Sports* 2006;16:3-63.
16. American College of Sports Medicine. The recommended quantity and quality of exercise for developing and maintaining cardiorespiratory and muscular fitness and flexibility in healthy adults. *Med Sci Sports Exerc* 1998;30:975-91.
17. Leon A, Connett J, Jacobs DR Jr, Rauramaa R. Leisure-time physical activity levels and risk of coronary heart disease and death. The Multiple Risk Factor Intervention Trial. *JAMA* 1987;258:2388-95.
18. Paffenbarger RS Jr, Hyde RT, Wing AL, Lee I, Jung DL, Kampert JB. The association of changes in physical activity level and other lifestyle characteristics with mortality among men. *N Engl J Med* 1993;328:538-45.
19. Slattery M, Jacobs DR, Nichama MZ. Leisure time physical activity and coronary heart disease death. The US Railroad Study. *Circulation* 1989;79:304-11.
20. Jakicic JM, Wing RR, Butler BA, Robertson RJ. Prescribing exercise in multiple short bouts versus one continuous bout. Effects on adherence, cardiorespiratory fitness, and weight loss in overweight women. *Int J Obes* 1995;19:893-901.
21. DeBusk RF, Stenestrand U, Sheehan M, Haskell WL. Training effects of long versus short bouts of exercise in healthy subjects. *Am J Cardiol* 1990;5:1010-3.
22. Ebisu T. Splitting the distance of endurance running. On cardiovascular endurance and blood lipids. *Japanese J Phys Education* 1985;30:37-43.
23. Thompson PD, Franklin BA, Balady GJ, Blair SN, Corrado D, Estes NA 3rd, et al. Exercise and acute cardiovascular events. Placing the risks into perspective. *Circulation* 2007;115:2358-68.
24. Whang W, Manson JE, Hu FB, Chae CU, Rexrode KM, Willett WC, et al. Physical exertion, exercise, and sudden cardiac death in women. *JAMA* 2006;295:1399-403.
25. Borg GA. Psychophysical bases of perceived exertion. *Med Sci Sports Exerc* 1982;14:377-81.
26. William MA, Haskell WL, Ades PA, Amsterdam EA, Bittner V, Franklin BA, et al. Resistance exercise in individuals with and without cardiovascular disease. 2007 Update. A scientific statement from the American Heart Association Council on Clinical Cardiology and Council on Nutrition, Physical Activity and Metabolism. *Circulation* 2007;116:572-84.
27. Physical Activity Guidelines Advisory Committee. Physical Activity Guidelines Advisory Committee Report, 2008. Washington, DC:U.S. Department of Health and Human Services, 2008.

3. Promoting physical activity

Authors

Matti E. Leijon, PhD, MPH, Center for Primary Health Care Research, Lund University/Region Skåne, Malmö, Sweden

Lena Kallings, PhD, Public Health Scientist, Department of Public Health and Caring Sciences, Uppsala University, Uppsala, Swedish National Institute of Public Health, Östersund, Sweden

Johan Faskunger, PhD, Center for Family and Community Medicine, Karolinska Institutet, Stockholm County Council, Stockholm, Sweden

Geir Lærum, Counsellor, PT, Public Health Scientist, Nordland County Municipality, Bodø, Norway

Mats Börjesson, MD, PhD, Associate Professor, Department of Medicine, Sahlgrenska University Hospital, Gothenburg, Sweden

Agneta Ståhle, PT, PhD, Associate Professor, Department of Neurobiology, Care Sciences and Society, Division of Physiotherapy, Karolinska Institutet, Stockholm, Sweden

Introduction

The lack of physical activity, unhealthy eating habits, tobacco use and alcohol consumption are still the largest causes of death and disease in modern society. A sedentary lifestyle entails a sharply increased risk of disease and premature death (1, 2). Lifestyle-related illness entails large costs and is consequently a major burden on healthcare services. Individuals of all ages, both men and women, achieve health benefits by exercising, and greater physical activity is also important to improve well-being and quality of life (1, 3, 4).

Increasing physical activity is therefore an important task for society in general and for healthcare in particular. Physical activity is now included as an important component in the treatment and prevention of various diseases.

Physical activity in disease prevention and treatment (FYsisk aktivitet i Sjukdoms-prevention och Sjukdomsbehandling – FYSS) is a tool for those who prescribe physical activity. Here, a number of different experts in various areas have compiled the latest evidence of the connection between physical activity and health.

What is physical activity?

Definitions (5)

In this initial chapter, we have consciously chosen use the term physical activity ahead of terms such as exercise and training:

- In general, physical activity means all types of movement that increases energy expenditure. This means all types of muscle activity such as walking, household and gardening work, and physical strain in work, outdoor life, exercise and training.
- Health-enhancing physical activity means all physical activity that improves health and physical capacity without constituting a risk of injury.
- Exercise is planned physical activity with a certain objective, such as to provide well-being or to improve future health or the equivalent, and most often involves changing into appropriate clothing.
- Training involves a clear goal of increasing performance capacity in various types of physical activity, such as in athletics.

What affects our physical activity?

It is important to emphasize that the main part of our weekly expenditure of energy in the form of movement can be associated with physical activity not tied to scheduled exercise and sports. This primarily concerns everyday physical activity, active transport, physical activity at work, physical activity during leisure time in the home or as a hobby, exercise, training and sports. The major health potential lies in increasing our overall level of physical activity, both in connection with work and during free time (5).

From theory to practice

Physical activity by prescription

Interest in promoting physical activity has grown in healthcare. Both professional organisations and healthcare personnel have a positive view of prescribing physical activity to patients (6, 7). However, a major challenge remains in converting the knowledge to practical reality – going from the evidence that currently exists regarding the health benefits of physical activity to effectively and naturally integrating physical activity into daily activities (8). One way of more systematically promoting physical activity is to use a written prescription for physical activity. This approach has become increasingly more common in several countries in the past decade (7, 9, 10), and in Sweden, exercise by prescription, proactive healthcare by referral, prescriptions of physical activity or Physical Activity on Prescription, FaR[®] have been used (5). Physical Activity on Prescription (FaR[®]) is currently a commonly accepted concept and involves a prescription adjusted to the individual corresponding to conventional treatment. The prescription can comprise everything from a simple, written suggestion of an activity to a comprehensive solution with a supportive structure from a prescriber, activity organiser or leader. At the time of the prescription, the prescriber can find recommendations and instructions adapted to disease in FYSS (11, 12).

FaR[®] on various levels¹

Prescribing FaR[®] is only one of many possible means of promoting physical activity that can be used in healthcare. Other examples are referrals to other actors in healthcare and verbal advice to patients. Figure 1 places FaR[®] in a context to clarify the roles and commitments for various actors or settings.

Level	State of health	→ Prescription form	→ Activity type	→ Arena/activity organiser
1	Ill individual /high risk	Referral (not FaR [®])	Physiotherapy	Healthcare services
2	Ill individual /high risk		Specially adapted FaR [®] activities. Most often low intensity exercise	Volunteer organisations
3	Physically inactive person or person who needs to increase physical activity. Capable of normal training. Preference for group activities	FaR [®] . In writing with or without referral	Regular offering of activities	Volunteer and sports organisations
4	Physically inactive person or person who needs to increase physical activity. Capable of normal training. Preference for own activities	In writing (FaR [®]) or orally (not FaR [®]) Without referral	Own activities. Everyday exercise, e.g. walking, cycling, outdoor life, gardening	Local society, residential area, home

Figure 1. Schematic model describing various levels of promoting physical activity, including FaR[®], in healthcare and NGOs.

Level 1 is relevant to patients with already developed disease/injury, has very elevated risk of disease or is otherwise assessed to be unfit to participate in FaR[®] activities not under the direction of healthcare services. These patients are not included in FaR[®], but rather are referred elsewhere within healthcare such as physiotherapy, although a prescription for physical activity can be written out to “formalise” the advice. Oral advice on everyday physical activity such as walking can also beneficially be included in level 1.

1. Excerpt from the report *Fysisk aktivitet på recept (FaR[®]) – en vägledning för implementering*. [Physical activity on prescription (FaR[®]) – a guide for implementation.] (in Swedish, summary in English), Swedish National Institute of Public Health, R 2007:1.

Level 2 means that patients are issued FaR[®] and referred to participation in specially adapted FaR[®] programmes or activities, such as low-intensity group activities. Activity organisers are primarily volunteer and sports organisations, but may also be actors within healthcare, such as occupational healthcare or special physiotherapist units.

Levels 3 and 4 are appropriate if the patient is considered to be capable of participating in the ordinary range of activities in association life or is assessed to manage maintaining his or her own activity. The selection of level should primarily be controlled by the patient's own preferences. Oral or written advice on own activity (level 4) is appropriate if:

- The patient is assessed to be receptive to, motivated and capable of following the recommendation on his/her own or with the help of social support.
- The patient commands sufficient strategies to safely and effectively increase his/her physical activity on his/her own.
- The patient's state of health and needs (social, emotional, medical) do not require a customised programme with coaching.

Just as the patient's state of health can vary between the various levels, the selection of adapted activities varies, as well as the prescriber's and the recipient organisation's expertise in the FaR[®] work. To take care of patients on levels 1 and 2, the knowledge requirements are higher than for those who work with patients on levels 3 and 4, both for healthcare actors and for activity organisers.

For patients to be motivated to change at all and interested in FaR[®] or oral advice, the health counsellor's role is very important in the prescription process. Patients in need of change, but who have a low level of motivation should be referred to a health counsellor or similar person with knowledge of motivational discussions and the principles of behavioural change. At levels 2 and 3, the prescribing unit actively cooperates with various activity organisers, which means a joint responsibility for developing the FaR[®]-related efforts in terms of coordination, offering, quality and role distribution. This approach often includes some form of sluice or coordination function that can exist either within healthcare, the activity organiser or both, through which the patient or his/her prescription passes. Here, it is important that there be a very clear division of responsibility for the various stages in the FaR[®] model at the local level, for instance via a detailed flow chart.

Regardless of whether the advice is oral or in writing, with or without a referral, or recommends group or individual activities, the ultimate goal should be to improve the possibilities for the patient to be able to maintain regular physical activity on his or her own on the long term.

Individually adapted advice

A prescription of physical activity should be individually adapted with regard to the dosage (intensity, duration and frequency) and type of activity. The point of departure is that *healthy people* benefit from being physically active. The greatest impact on public health, in the form of reduced morbidity (incidence of disease), is achieved if the physical activity of the *least physically* active groups is increased, such as the socioeconomically disadvantaged and the elderly. *Individuals with risk factors* who have not yet developed disease are also a potential target group, such as those with a hereditary predisposition for cardiovascular disease, the obese or smokers.

Persons with various diseases comprise another important target group. However, the prescription must be individualised in consideration of the patient's actual disease, functional capacity, medicine interactions and potential contraindications for a certain activity.

Case history of physical activity

To be able to prescribe a suitable physical activity level/activity for a patient, the *case history of physical activity* can be used to assess the current level of activity. This should include information on current activity (if any), how much the patient exercises, what the patient thinks he or she has time for and the extent to which he or she was previously physically active. Appropriate questions may include:

1. What are you doing right now?	→	Activity
2. How often?	→	Frequency
3. How long?	→	Duration
4. How hard?	→	Intensity

It is important that the advice is individual, that encouragement is given, that one agrees with the patient on how everything should be carried out and when a follow-up should take place. It is also important that the level of exercise is set at an adequate level, especially for those completely unaccustomed to exercise, to avoid failure and for increased flexibility.

What should the prescription include?

The prescription should be specific with clear lines about:

- *The type of physical activity* that is suitable for the individual patient.
- Dose, in other words the *intensity* of and *duration* of each activity session, and the *frequency* (number of times per week).

Form for the prescription of physical activity

Today, there are several different variants of forms for prescribing physical activity. The original Swedish FaR[®] prescription (see figure 2) was a reworked version of the common pharmaceutical prescription. The majority of county councils currently have specially adapted forms that also include suggestions of activities and actors. Some of these are also integrated with the existing patient record systems.

RECEPT/FYSISK AKTIVITET	
Receptutfärdaren anger genom signum vilka speciella försiktighetsmått som kan behöva iaktas vid genomförandet av de fysiska aktiviteterna F = Försiktig start U = Undvik vissa aktiviteter O = Inga speciella försiktighetsmått	Patient (namn, födelseid, adress och telefon)
① Träningsform <input type="checkbox"/> Kondition <input type="checkbox"/> Styrka <input type="checkbox"/> Uthållighet <input type="checkbox"/> Underhåll Aktivitet:	
Intensitet: <input type="checkbox"/> Hög <input type="checkbox"/> Måttlig <input type="checkbox"/> Lätt Antal ggr/behandlingstid: Dosering, användning, ändamål D.S Minst minuter ggr/vecka för/vid/mot undvik	
② Träningsform <input type="checkbox"/> Kondition <input type="checkbox"/> Styrka <input type="checkbox"/> Uthållighet <input type="checkbox"/> Underhåll Aktivitet:	
Intensitet: <input type="checkbox"/> Hög <input type="checkbox"/> Måttlig <input type="checkbox"/> Lätt Antal ggr/behandlingstid: Dosering, användning, ändamål D.S Minst minuter ggr/vecka för/vid/mot undvik	
Receptutfärdarens namnförtydligande, yrke, adress, telefonnr. Sjukvårdsinrättning, klinik	
Ort: Datum: Receptutfärdarens namnteckning: Gäller mån. från detta datum.	
Receptet löses lämpligen in hos (Namn och telefon)	

Figure 2. The original Swedish prescription form for physical activity on prescription (FaR®). The form resembled the ordinary drug prescription form in terms of content, layout and colour. Two different physical activities can be prescribed on the form with information on the type of physical activity, dose (i.e. intensity, duration, frequency) and reason for prescription. The name of the patient, the name of the prescriber and the date of the prescription should also be noted.

How can FYSS be used in practice?

The following is an example that describes how FYSS can be used by a doctor in the meeting with a patient.

As the prescribing doctor, you have a patient in front of you with recently arisen hypertension. The patient's blood pressure is at 150/95 and in an assessment of the patient's total risk profile (age, gender, smoking and other risk factors) he or she is assessed to not be sufficiently physically active. You decide on a non-pharmacological initial treatment once the diagnosis has been confirmed with repeated blood pressure measurements. You look in FYSS under "Hypertension". You may not be really certain which type of activity is most appropriate to achieve a reduction in blood pressure, what effects can be expected and how long it takes to achieve a maximum effect. In FYSS, you read that:

"The latest international recommendations for the treatment of hypertension emphasize an individual prescription of treatment based on the patient's unique 'risk profile' (13). Physical activity, and the impact on other lifestyle factors, is considered to be the primary treatment in mild hypertension (blood pressure, BP, < 160/90) (13) and is considered to be a supplemental treatment for hypertension otherwise (13). However, physical activity is considered to be contraindicated upon an uncontrolled BP over 180/105 until an adequate reduction in blood pressure has been achieved with pharmaceutical treatment (14).

In hypertension, mainly activities of moderate intensity that improve condition, at least 30 minutes a day, five times a week, are recommended (13–15). Suitable activities could be jogging, Nordic walking, swimming and/or cycling. Physical activity is a perishable good and needs to be continuous to maintain its effect. Meta-analyses of studies have shown that dynamic activity at this intensity can reduce systolic blood pressure by approximately 7 mm Hg and diastolic pressure by 5 mm Hg (15), although with low weights and many repetitions (circuit training), which can reduce systolic blood pressure by approximately 4 mm Hg and diastolic pressure by 3 mm Hg. However, strenuous strength training, such as weightlifting and power lifting, should be avoided. The maximum effect on the blood pressure is achieved after up to six months of physical activity. Consideration of any current diseases must, of course, be taken when the prescription is written."

With the help of FYSS, a patient with newly arisen hypertension with a low overall risk profile could be given clear recommendations about, when and what form of physical activity is suitable as treatment for him or her.

Success factors

A prescription for physical activity does not automatically lead to changed behaviour and a higher physical activity level in the patient on the long term. Several other factors affect the result. A major challenge is that our modern social structure often even counteracts a physically active lifestyle and encourages being sedentary. Methods for behavioural change are therefore considered to be important for successful change efforts, but to-date, few programmes have systematically used evidence-based methods for behavioural change (16), even though this has been encouraged to increase the effectiveness of

health-promoting programmes. For example, this could involve health counselling focusing on raising the patient’s motivation, objectives, handling perceived obstacles, strengthening self-confidence and social support, encouraging daily activities and measuring one’s own activity, such as through step-counters or a journal (17). The programme set-up should be based on a tried model for behavioural change and support for its maintenance, such as through follow-up phone conversations.

Various professional trade organisations and national institutions have emphasized the promotion of physical activity as an important area of development in recommendations and management documents. The American College of Preventive Medicine argues for primary care routinely involving advice regarding physical activity in patient meetings (6).

A number of factors considered to be success factors in successful interventions have been identified, including:

- the staff having been given qualitative continued education
- goal-oriented measurement of the patient’s activity level
- the use of behavioural-change models
- advice tailored to vulnerable sub-groups
- a focus on changing one lifestyle element at a time
- involving teams of healthcare staff who help each other to carry out different parts of the intervention
- good support systems and clear procedures at each respective unit
- including individually adapted advice in writing based on the “stages of change”
- involving some form of follow-up of patients.

In addition, the *5A structure* is considered to be very promising as an effective model that can relatively easily be introduced in primary care (6).

Counselling with the 5As

One commonly occurring obstacle to all lifestyle advice in healthcare is a lack of time. Today, there are a large number of conversational techniques that can be used in a short consultation of a few minutes or as a series of longer conversations on multiple occasions. The 5As (Assess, Advise, Agree, Assist, Arrange) aim at quickly and easily structuring a lifestyle discussion (18).

1. **Assess** – Ask about the physical activity level and assess the patient’s proclivity to change.
2. **Advise** – Provide information and individually adapted advice based on the patient’s needs and motivation to change. Consideration should be taken of age, gender, ethnicity, BMI, disease history, activity experience, prior attempt to change lifestyle etcetera. The advice may be either oral or written.
3. **Agree** – Communicate in a spirit of partnership by using patient-centred techniques and finding activities that the patient is interested in and has the possibility of pursuing – set up realistic goals.

4. **Assist** – Use various techniques to increase the patient’s motivation and self-confidence and to address the surrounding environment.
5. **Arrange** – Plan the follow-up, provide ongoing assistance/support and potential return visits to adjust the treatment plan as needed, including referral to more intensive or specialised treatment and sluice the patient on to an activity organiser if this is of interest.

These five steps need not necessarily be followed at the same visit, or even by the same person. The measurement and assessment of the patient’s activity level can be carried out by the patient or by a suitable staff group with various test instruments (19, 20). For example, the physician can assess the patient’s level of activity and desire to change and give oral advice with a referral to other personnel who have more knowledge in motivational work and who complete the remaining stages of the 5As. The concluding stages, assist and arrange, can take place both within healthcare and at the healthcare services’ cooperative partners within the FaR[®] work. One such comprehensive solution enables the healthcare staff, the healthcare system and its cooperation partners to be able to carry out supplementary tasks (20).

Competency, responsibility, follow-up and confidentiality in Sweden²

Prescribed physical activity is a complement to or replacement for pharmaceuticals. The person prescribing physical activity should have an adequate level of competency and sufficient knowledge of the current patient’s health and illness status, since there can be some activities that are directly unsuitable for certain disease conditions (also refer to the respective chapters in FYSS). The prescriber should sign off on any special cautionary measures that may need to be observed or not.

Which professional groups may prescribe physical activity?

The prescription of physical activity is not a rule-controlled task (in Sweden) and can consequently in principle be carried out by any of the licensed personnel, on condition that the person in question commands the knowledge necessary to carry out the task correctly. It is the responsibility of every operational manager to lead the work in the scope of the human resources and other resources the caregiver makes available and to distribute duties in line with their type and level of difficulty and in consideration of the need for expertise and professional experience. The operational manager is responsible for ensuring that the personnel that is to carry out various tasks has the expertise required for this.

2. Excerpt from the report *Fysisk aktivitet på recept (FaR[®]) – en vägledning för implementering*. [Physical activity on prescription (FaR[®]) – a guide for implementation.] (in Swedish, summary in English), Swedish National Institute of Public Health, R 2007:1.

Liability

In accordance with the Swedish National Board of Health and Welfare's regulations (SOSFS 2005:12) regarding management systems for quality and patient safety in health and medical care (in Sweden), the *caregiver* shall ensure that the management system includes procedures that indicate the personnel's responsibilities and authority. *The operational manager* is responsible for appointing and registering which qualified healthcare personnel will perform the task of independently prescribing physical activity. A member of the *health and medical care personnel* that prescribes physical activity bears responsibility for the measure in accordance with Chapter 2 Section 5 of the Act (1998:531) on professional activity in health and medical services. A prescription of physical activity shall be documented in the patient's records, in accordance with Section 3 of the Patient Records Act (1985:562). Oral advice regarding physical activity shall also be noted in the patient's records.

Patient insurance applies to activity within county council care. Therefore, adequate record notes regarding physical activity as a treatment are just as important as notes about examinations or pharmaceutical treatments. However, if a patient who was prescribed physical activity injures him or herself in activities outside the county council's facilities, the insurance only applies if the injuries can be directly related to the prescription in question. If, for example, the patient stumbles on a tree root on the exercise course and sprains an ankle, the insurance does not apply. However, if the patient should be struck by a heart attack while conducting the activity, this should be reported to the patient insurance agency for further investigation. For more information, see www.psr.se. For example, if the patient injures him or herself at a gym after being prescribed physical activity, the owner of the gym can be liable under civil law if it is a matter of deficient safety procedures at the gym. Otherwise, the owner of the gym cannot be held liable for injuries the patient may potentially suffer.

Follow-up

Within healthcare, the quality of the operation shall systematically and continuously be developed and ensured pursuant to Section 31 of the Health and Medical Services Act (1982:763). Section 2 a, paragraph one of the Health and Medical Services Act contains fundamental stipulations regarding patient safety and the quality of care. The follow-up of a prescription of physical activity does not differ from the follow-up of other treatments within healthcare. Pursuant to the Swedish National Board of Health and Welfare's regulations (SOSFS 2005:12) regarding management systems for quality and patient safety in health and medical care, the management system shall ensure that procedures are in place for how new methods of treatment shall be developed, tried and introduced so as to ensure patient safety. Furthermore, procedures shall be in place for how established methods are to be applied, continuously followed up and, where necessary, revised, as well as procedures for the measures that shall be undertaken when the application of the the methods needs to be changed.

Confidentiality

As per Chapter 7 Section 1 of the Secrecy Act (1980:100), confidentiality applies in healthcare for information regarding the individual's state of health or other personal circumstances, unless it is clear that the information can be disclosed without the individual or any party related to the individual suffering harm. As per Chapter 14 Section 4 paragraph one of the aforementioned act, confidentiality for the protection of the individual does not apply to the individual him or herself and can otherwise be granted exemption by him or her in part or in whole.

For private healthcare, a corresponding stipulation regarding confidentiality is set forth in Chapter 2 Section 8 of the Act (1998:531) regarding professional activity in health and medical services.

If, for example, prescribed physical activity is sent to an organisation outside healthcare for information, the patient's consent must consequently be obtained first. The Secrecy Act does not apply outside public operations. When, for instance, a private company receives prescribed physical activity, the company should prepare a contract regarding confidentiality.

Methods to promote physical activity

To evaluate effects and cost impacts of various methods for healthcare to promote physical activity, the Swedish Council on Technology Assessment in Health Care (SBU) conducted a systematic review of the scientific literature (4). The main objective of the report was to review the effectiveness of the measures with regard to the impact on the level of physical activity. All studies reviewed in the report had a follow-up time of at least six months, a relevant control group and the outcome measurement was an increase in the level of physical activity.

Advice, theory-based behavioural interventions, supervised training in groups and individually adapted training programmes as well as children and young people were the methods and important areas that were identified (4).

With regard to advice, the conclusions were that:

- Advice and counselling of patients in everyday clinical practice increases physical activity by 12–50 per cent for at least six months after the counselling session.
- More frequent, intensive counselling by means of repeated sessions for several months additionally boosts physical activity.
- Counselling supplemented by prescribed physical activity, diaries, pedometers, informational brochures, etc., increases activity by another 15–50%.

With regard to supervised training in groups and individually adapted training programmes, the conclusions were that:

- A six-month group exercise programme for patients with coronary artery disease promotes physical activity.
- A six-month supervised exercise programme for patients with peripheral arterial disease increases physical activity in terms of walking distance and/or time.

With regard to theory-based behavioural interventions, the conclusions were that:

- Theory-based behavioural intervention increases physical activity 10–15% more than usual care and as much as structured exercise programmes.
- More extensive behavioural interventions further boost physical activity, though with a diminishing marginal effect.
- Interventions that include a person's entire lifestyle, focusing on diet and stress management as well as physical activity, reinforce the increase in activity.

With regard to methods directed at children and young people, it was determined that:

- Devoting greater resources to school curricula in areas such as health education, textbooks, study materials and teacher training increases activity by 5–25% during physical education classes – even more so for boys than for girls.
- School-based interventions that include multiple components – such as teacher training, curriculum modifications, extra activity sessions during class periods and/or recess, support for behaviour changes, improved health education and the involvement of parents – favourably impact the physical activity of children and adolescents during the school day and sometimes during after-school hours and weekends as well.
- School-based interventions for groups at greater risk of cardiovascular disease increase physical activity by approximately 10%.

The supply of health-economy studies that illustrated the project's questions was so limited that no conclusions could be made regarding the cost-effectiveness of the methods studied.

Description of an implementation model for FaR®³

A fundamental starting point for all intervention work is that it is important to take the local conditions into account. This means that the work on FaR®, for example, can look differently in different parts of the country or within the same county or municipality. A summary is presented below of the model for implementation of FaR® as described in the report *Fysisk aktivitet på Recept (FaR®) – en vägledning för implementering [Physical Activity on Prescription (FaR®) – a guide for implementation]* (11).

Work on promoting physical activity in healthcare should be based on a broad cooperation with other actors and be rooted in the operation, preferably in a policy decision. The so-called PRECEDE-PROCEED model developed by Green and Kreuter (21) is an accepted and useful model for developing, implementing and evaluating health-promoting programmes and efforts in accordance with such an approach, and has been further developed for the work on promoting physical activity in primary care and how this can be systematised (22).

3. Excerpt from the report *Fysisk aktivitet på recept (FaR®) – en vägledning för implementering. [Physical activity on prescription (FaR®) – a guide for implementation.]* (in Swedish, summary in English), Swedish National Institute of Public Health, R 2007:1.

Here, a modified version of this model is presented, adjusted to Swedish conditions, which treats how all healthcare services can work to promote physical activity. The model is divided into nine phases that include a mapping of needs, diagnoses, policy, coordination, implementation and evaluation of a FaR[®] programme (see figure 3).

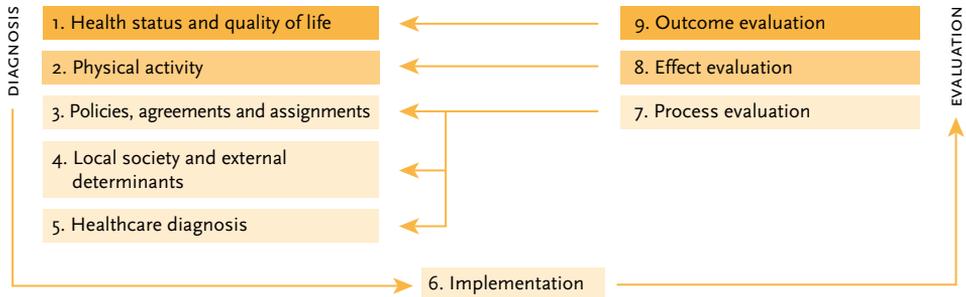


Figure 3. Framework for the diagnosis, implementation and evaluation of an FaR[®] programme.

Phase 1 concerns mapping and understanding the health needs in the local society, the health status of the residents and the patients' state of ill-health, such as the occurrence of chronic disease, quality of life, age structure, socioeconomic differences, etc. Investigating health status and quality of life are important since FaR[®] programmes should ultimately lead to improved health on an individual level and improve markers for health on a local society level.

Phase 2 brings up the need to map the patients' physical activity patterns, establish goals for increasing physical activity and establish what types of physical activity are needed. This stage in the planning can also bring up potential interaction with other lifestyle habits (such as smoking, diet, alcohol).

Phase 3 maps whether or not there is support for the FaR[®] efforts in current management documents/agreements/assignments to work with promoting physical activity in both one's own organisation as well as in cooperation with other actors.

Phase 4 concerns healthcare as an actor in the local society and the impact of the local society's other determinants for physical activity and health, such as climate, availability of activities, access to bicycle paths, exercise trails, parks, the local sports offering and other association life. Even if it is not healthcare's primary task to map physical and social conditions, obstacles and qualities in the local society, there are a number of important aspects to take into account when starting an FaR[®] programme.

Phase 5 maps factors that affect the patient's meeting with healthcare and is divided into three overall areas: healthcare, personnel issues and patient issues that somehow affect the health-promoting work with FaR[®] (see table 1).

Table 1. The impacts of healthcare, personnel and patients on the prescription of physical activity.

Level	Definition	Example
Patient	Factors that the patient brings with into the meeting with the caregiver.	Knowledge, reason for the visit, expectations, motivation, obstacles, conditions, experiences, history of activity.
Personnel	Factors that the licensed personnel brings with to the meeting with the patient.	Knowledge, interest, motivation, skills, expectations of themselves to be able to influence patients, the personnel's own level of activity, role models (positive: bicycle helmet in the treatment room, or negative: smell of cigarette smoke), empathy.
Healthcare services	External factors that affect the FaR [®] work.	Prescription form, information materials, personnel training and sufficient expertise at an organisation level, bicycle rack outside the unit, attractive stairwells, other personnel, exercise possibilities during or in connection with working hours for the personnel.

Phase 6 emphasizes the importance of working systematically. A systematic approach is more effective in promoting physical activity and in creating better conditions for long-term efforts at the programme level (16). The experiences from the Swedish FaR[®] efforts is that the implementation process in particular is the most difficult phase and the one that takes the longest (12). Also refer to the success factors above.

It is important that every FaR[®] programme establishes its own approaches and models as well as selection criteria for the participants. This includes a good internal structure for the FaR[®] work (who, what, how?) and good coordination with the activity organisers (with whom, which activities, in what way?). Supportive factor include the local society's range of facilities and other external conditions for exercise and physical activity, as well as the expertise of relevant professional groups to work with FaR[®]. It is in healthcare that the prescription process begins and it is the prescriber/prescribing unit that decides what the first steps look like.

It is also important that both healthcare and other involved actors respect each other's knowledge, underlying conditions and possibilities in the FaR[®] work.

Phase 7–9 – evaluation

Evaluation efforts have been divided into three phases: process evaluation, effect evaluation and outcome evaluation.

Phase 7 – the process evaluation focuses on achieved intermediary measurements in phases 3, 4 and 5, such as knowledge, attitudes, motivation and inclination to change of the patient and the personnel, changes in factors concerning the prescribing unit and activity organisers as well as policy issues. The process evaluation can also investigate the implementation phase. The objective of the process evaluation is to better understand the processes and factors that facilitate or obstruct goal fulfilment in phases 1 and 2.

Phase 8 – the effect evaluation investigates the FaR[®] programme’s affect on physical activity, quality of life or accepted physiological evaluation measurements such as oxygen uptake capacity or work capacity, and should indicate whether the goals set in phase 2 were achieved or not. The follow-up of the prescription and feedback to the prescriber is a very important dimension to improve maintenance and to drive the entire work with prescribed physical activity further. Follow-up should be carried out at the end of the activity programme/activity prescription to follow-up the outcome of the behavioural change to a more physically active lifestyle.

Phase 9 finally investigates the FaR[®] programme’s impact on various health outcomes and the goal-fulfilment of goals established in phase 1. This phase does not usually belong to the individual unit’s main tasks, but can be carried out in cooperation with the public health unit in the county council/region or the equivalent.

FaR[®] in the Nordic region

There is also a growing interest in promoting physical activity within healthcare outside Sweden. In the Nordic region, there are several variants of prescribing physical activity: *Physical Activity on Prescription* (FaR[®]) in Sweden, *Grønn resept* (*Green prescription*) in Norway, *Motion som lægemiddel* (*Exercise as a remedy*) in Denmark, and *Prescription for physical activity* in Finland. In contrast to Sweden (the majority of Sweden’s county councils), where all licensed healthcare personnel may prescribe physical activity, the work in the other Nordic countries is more based on only physicians being able to do so. In Norway, work on physical activity is conducted in parallel with dietary efforts. There is also a national compensation system and limits to the diagnosis groups in which the Green Prescription is an option. The Norwegian government decided that the entire country would work with Green Prescriptions. In Denmark, efforts have taken place on a national level to develop a method for the entire country. In 2008, the Danish National Board of Health will draft guidelines for using exercise as a remedy. Since 2003, there has been an equivalent to FYSS in Denmark, *Fysisk aktivitet – håndbog om forebyggelse og behandling* [*Physical activity – handbook on prevention and treatment*]. In Finland, two models exist for the prescribing of physical activity and in both only physicians may prescribe physical activity. The national method is the Prescription for Physical Activity, which is a cooperation project between the Finnish Rheumatism Association, the “I form för livet” [In shape for life] programme, the Finnish Medical Association, the Finnish Heart Association and the UKK Institute. There is also the “Fysiotek” conducted by Folkhälsan (the largest Swedish-speaking social and healthcare NGO that is directed at the Swedish-Finnish population). An important reason that the work in developing prescribed physical activity differs between the Nordic countries lies in differences in social structures and that the county councils in Sweden have greater autonomy (23). Links to and a more detailed description of the on-going efforts in the Nordic region are available on the FYSS website, www.fyss.se.

The following are a number of useful Internet addresses for each country:

Sweden

Swedish National Institute of Public Health www.fhi.se
FYSS www.fyss.se

Norway

Norwegian Directorate of Health www.shdir.no

Denmark

National Board of Health www.sst.dk

Finland

Folkhälsan Finland www.folkhalsan.fi
(Swedish-speaking part of Finland)
Liikkumisresepti www.liikkumisresepti.net
(in Finnish with summaries in
Swedish and English)

References

1. U.S. Department of Health and Human Services, Physical Activity and Health. A report of the Surgeon General. Atlanta (GA): U.S. Department of Health and Human Services, Centers for Disease Control and Prevention, National Center for Chronic Disease Prevention and Health Promotion; 1996.
2. World Health Organization. The World Health Report 2002. Reducing risks, promoting healthy life. Geneva: World Health Organization; 2002.
3. Department of Health. At least five a week. Evidence on the impact of physical activity and its relation to health. Report from the Chief Medical Officer. London: Department of Health; 2004.
4. SBU (Swedish Council on Technology Assessment in Health Care). Metoder för att främja fysisk aktivitet. En systematisk litteraturoversikt. [Methods for promoting physical activity. A systematic literature review.] SBU Report no. 181. Stockholm: SBU; 2007.
5. Schäfer Elinder L, Faskunger J. Fysisk aktivitet och folkhälsa. [Physical activity and public health.] R 2006:13. Stockholm: Swedish National Institute of Public Health; 2006.
6. Jacobson DM, Strohecker L, Compton MT, Katz DL. Physical activity counseling in the adult primary care setting. Position statement of the American College of Preventive Medicine. *Am J Prev Med* 2005;29:158-62.
7. Harrison RA, McNair F, Dugdill L. Access to exercise referral schemes. A population based analysis. *J Public Health (Oxf)* 2005;27:326-30.
8. Huang N. Motivating patients to move. *Aust Fam Physician* 2005;34:413-7.
9. Elley CR, Kerse N, Arroll B, Robinson E. Effectiveness of counseling patients on physical activity in general practice. Cluster randomised controlled trial. *BMJ* 2003; 326:793.
10. Sorensen JB, Skovgaard T, Puggaard L. Exercise on prescription in general practice. A systematic review. *Scand J Prim Health Care* 2006;24:69-74.
11. Faskunger J, Leijon M, Ståhle A, Lamming P. Fysisk aktivitet på Recept (FaR[®]). En vägledning för implementering. [Physical activity on prescription (FaR[®]). An implementation guide.] R 2007:1. Stockholm: Swedish National Institute of Public Health; 2007.
12. Kallings LV, Leijon M. Erfarenheter av Fysisk aktivitet på Recept – FaR[®]. [Experiences from Physical activity on prescription – FaR[®].] R 2003:53. Stockholm: Swedish National Institute of Public Health; 2003.
13. Mancia G, De Backer G, Dominiczak A, Cifkova R, Fagard R, Germano G, et al. Guidelines for the management of arterial hypertension. The task force for the management of arterial hypertension of the European Society of Hypertension (ESH) and of the European Society of Cardiology (ESC). *J Hypertens* 2007;25:1105-87.
14. American College of Sports Medicine Position Stand. Physical activity, physical fitness and hypertension. *Med Sci Sports Exerc* 1993;25:i-x.

15. Fagard R, Cornelissen VA. Effect of exercise on blood pressure control in hypertensive patients. *Eur J Cardiovasc Prev Rehabil* 2007;14:12-7.
16. Riddoch C, Puig-Ribera A, Cooper A. Effectiveness of physical activity promotion schemes in primary care. A review. London: Health Education Authority; 1998.
17. Eden KB, Orleans CT, Mulrow CD, Pender NJ, Teutsch SM. Does counseling by clinicians improve physical activity? A summary of the evidence for the U.S. Preventive Services Task Force. *Ann Intern Med* 2002;137:208-15.
18. Whitlock EP, Orleans CT, Pender N, Allan J. Evaluating primary care behavioral counseling interventions. An evidence-based approach. *Am J Prev Med* 2002;22:267-84.
19. Flocke SA, Gordon LE, Pomiecko GL. Evaluation of a community health promotion resource for primary care practices. *Am J Prev Med* 2006;30:243-51.
20. Stange KC, Woolf SH, Gjeltema K. One minute for prevention. The power of leveraging to fulfill the promise of health behavior counseling. *Am J Prev Med* 2002;22:320-3.
21. Green L, Kreuter M. Health program planning. An educational and ecological approach. Boston: McGraw Hill; 1991.
22. Taylor A. The role of primary care in promoting physical activity. In: McKenna J, Riddoch C, eds. *Perspectives on health and exercise*. London: Palgrave Macmillan; 2003.
23. Kallings L, Ståhle A. Motion på recept i de nordiska länderna. [Exercise by prescription in the Nordic countries.] *Svensk Idrottsmedicin* 2005;3:32-5.

4. *Becoming physically active*

Authors

Anita Wester, PhD, Psychologist, National Agency for Education, Stockholm, Sweden

Lina Wahlgren, MSc, Swedish School of Sport and Health Sciences, Stockholm, and Department of Health Sciences, Örebro University, Örebro, Sweden

*Ingemar Wedman, PhD, Professor, Swedish School of Sport and Health Sciences, Stockholm, Växjö University, Växjö, and University of Gävle, Gävle, Sweden**

Introduction

Regular physical activity increasing health and well-being for the individual and saving society considerable cost are known facts today. It is estimated that the cost resulting from insufficient physical activity amounts to approximately SEK 6 billion per year (1). Efforts are being pursued in society as a whole and in schools to increase the level of physical activity (2).

Many are aware of the benefit of physical activity and recommend regular physical activity, but are nonetheless passive in their daily lives. The research that concerns going from being physically passive to being active show that this process is more difficult than what can initially be believed. Behavioural change is a difficult process to grasp in many regards (3–6).

With the recommendations for physical activity that exist today (a minimum of 30 minutes a day) (7), a majority of the adult population is inactive (8). Inactivity results in a lack of energy and excess weight. Daily life also places increasingly fewer demands on natural physical activity. We can change tyres on the car at a garage. We can take the lift instead of stairs, etc. The excess weight sneaks in and, consequently, the weight gain is not as striking – circumstances “cushion” a loss of energy and weight gain.

Physical activity in a time perspective

In a study from Västerbotten, a health follow-up has long taken place among persons who turn 40, 50 and 60 years old. The study is being carried out by the Västerbotten County Council and comprises diverse data, including a few questions regarding physical activity. As a part of the study, a follow-up was carried out of approximately 16,000 people after ten years (9, 10). See table 1.

Table 1. Responses to the question of how often the person had exercised after first having changed into training clothes. The same question was answered by 16,000 people with ten years in between.

First year Tenth year	Never	Now and then	1 time/week	2–3 times/ week	More than 3 times/week	Total
Never	4 022	933	503	354	112	6 004
Now and then	1 679	1 336	685	522	136	4 358
Once weekly	988	814	824	642	153	3 421
2–3 times/week	287	272	286	469	162	1 476
More than 3 times/week	133	111	88	169	171	672
Total	7 109	3 526	2 386	2 158	754	15 931

The questions asked in this context pertain to exercise where one changes into training clothes. It then turns out that virtually everyone is classed as physically inactive, and approximately 4,000 have never changed into training clothes, not even over a period of ten years.

Thoughts about changing lifestyles

Riksbankens Jubileumsfond (Jubilee Fund of the Swedish Central Bank) carried out a conference 25 years ago with the title “Changing lifestyles”, which also became the title of a book published after the conference (11). At this conference, a number of researchers participated with various areas of interest, including antidotal smoking treatments, physical activity, diabetes follow-up, etc. The common message from these scientists was that it is difficult to change behaviour, regardless of which behaviour is in question.

Behavioural change models in the area of physical activity

A number of models have been used and are currently used to try to understand, explain and change behaviour in various health-related areas (12). In the area of physical activity, the following theories and models have been used or are used in research on behavioural change: *Classic learning theories*, *Health belief model*, *Transtheoretical model*, *Relapse prevention*, *Social cognitive theory*, *Theory of planned behaviour*, *Social support* and *Ecological perspective* (7, 13). One example where various models are combined is the *Groningen Active Living Model* (GALM), which focuses on the elderly (14). The most common today are the *Transtheoretical model* and *Social cognitive theory*. It appears as if theory-based behavioural interventions increase physical activity. For example, improvements in physical activity can be seen in the state of ill-health, such as cardiovascular disease, when behavioural change programmes have been used in treatment. Furthermore, interventions that include lifestyle changes, or changes in multiple areas such as physical activity and diet, appear to increase physical activity (15).

Transtheoretical model

One of the most popular and most used models to describe and change behaviour is the transtheoretical model. The transtheoretical model was developed, and refined, by the U.S. researchers Prochaska, DiClemente and Norcross at the beginning of the 1980s. Work on the model began with an analysis of theories that have been used for behavioural change in psychotherapy. The objective of the analysis was to harmonize the various theories with each other into a model. The transtheoretical model can therefore definitely be said to be *transtheoretical*, since it harmonizes behavioural change principles and approaches for change from a number of different intervention theories (5, 16).

After the work on harmonizing the various theories, it was investigated how often various approaches were used by people in for instance antidotal smoking treatments. Then it turned out that people use different approaches at different points in time in their efforts to stop smoking. Consequently, behavioural change is considered to occur by moving through different stages (5, 16).

Hence, the transtheoretical model had its origins in smoke cessation, but has also been used in other health-related areas, such as alcohol abuse, obesity and physical inactivity (5, 16). Although the transtheoretical model is not directly developed for the area of physical activity, its use appears to be promising in the area (17). The model's use also appears to be promising in work on physical activity in rehabilitation (18). A popular science and practically applicable example of the transtheoretical model tied to the area of physical activity is J. Faskunger's book *Motivation för motion* [Motivation for exercise] (6).

The transtheoretical model comprises several different components. One of the components is called the *stages of change* and contains various stages based on people's inclination for change, in other words where a person is in the behavioural change process. Another component is called the *processes of change*¹ and includes various approaches through which people move between various stages of change. The third component focuses on why people change and consists of activity-specific *self-efficacy* and motivation balance (5, 16). In working with behavioural change, it is important to use all of the components of the model to achieve a successful result, meaning to change a behaviour (17).

1. Faskunger uses the term "process", but the interpretation made here is that the English word "process" rather entails a form of an approach.

Stages of change

According to the transtheoretical model, behavioural change is seen as a process over time and there are six stages of change: *Precontemplation, contemplation, preparation, action, maintenance* and *termination* (5, 6, 16).

People who are not regularly physically active nor are interested in or intend to change their inactive behaviour are in the **precontemplation stage**. One reason they are in the precontemplation stage may be too little knowledge or information about the risks of being physically inactive. Another reason may be that they have tried to change their behaviour a number of times, but have failed and thereby lost faith in their ability to change. Regardless of reason, people in the precontemplation (or denial) stage avoid reading about, talking about and thinking about their risky inactive behaviour. They deny that physical inactivity is a problem for them (5, 6, 16).

People who are not regularly physically active, but who intend to change their inactive behaviour in the next six months are in the **contemplation stage**. They understand that physical inactivity is a problem for them and honestly contemplate how to go about becoming physically active. However, some people do not make it any farther, but rather get stuck at the contemplation stage for a prolonged period. They become “chronic contemplators”. People at this stage are not ready for traditional activity-oriented interventions where the participant is expected to become active immediately (5, 6, 16).

People who are not regularly physically active, but have plans of becoming physically active in the near future, most often within one month, are in the **preparation stage**. Usually, people in the preparation stage have tried some form of physical activity in the past year and also have a concrete plan for implementation. For people in the preparation stage, activity-oriented interventions are suitable since they are ready to become physically active (5, 6, 16).

People who are regularly physically active and have been so for six months are in the **action stage**. Changes in the action stage are more visible to the surroundings than in the other stages of change. It is therefore easy to believe that people in the action stage have achieved a change in behaviour, but the action stage should only be considered a part of the behavioural change process. Regular physically active behaviour requires time to be established (5, 6, 16).

People who are regularly physically active and have been so for more than six months are in the **maintenance stage**. As in the other stages, there are also challenges in the maintenance stage. People in the maintenance stage should focus on the work of consolidating and strengthening the gains of being physically active, based on lessons from the other stages of change. They should also work on preventing relapse (5, 6, 16). People who have fully assumed a behaviour are in the **termination stage**. They have full faith in their

behaviour and that they will not return to their previous behaviour, regardless of the situation. Behaviour takes place out of habit and automatically (5, 6, 16). One example is that one puts on the seat belt without thinking about it when getting into the car (19). The termination stage has been debated. It may be so that the termination stage is too strict and the realistic goal for areas such as physical activity is to be in a lifelong maintenance stage (5, 16, 20). The termination stage could possibly be associated with the areas of everyday exercise, such as always spontaneously choosing to take the stairs instead of the lift.

The behavioural change process should not be seen as a linear process, but rather as spiral shaped. The shift between the stages can take place both forwards and backwards. Relapses should be viewed as a natural part of the process. It is therefore important to work with relapse prevention so that the relapses do not become more than sidesteps. People often need to go through both success and setbacks to succeed in making a change (5, 16).

In working with behavioural change, it is important to know the stage of change that people are in so that the efforts agree with people’s receptivity. If efforts and stages of change do not agree, the number who drop out may increase. In the area of physical activity, it can be easy to think about activity-oriented interventions as an opportunity for behavioural change. However, succeeding with activity-oriented interventions presuppose that people are in the preparatory stage or further. However, this does not appear to be the case. A majority appear instead to be in the earlier stages of change of the precontemplative and contemplative stages (5, 16, 21).

To obtain information about which stage of change people are in, the following statements, presented in table 2, can be used (5).

Table 2. Statements that can be used to gather information about which stage of change a person is in.

	Precon- templation	Contemp- lation	Preparation	Action	Maintenance
1. I am regularly physically active and have been so for more than six months.	No	No	No	No	Yes
2. I am regularly physically active and have been so for six months.	No	No	No	Yes	
3. I am not regularly physically active, but I have plans of becoming physically active in the near future; within one month.	No	No	Yes		
4. I am not regularly physically active, but I intend to change my physically inactive behaviour in the next six months.	No	Yes			

If the answer is no to all of the statements, the person is in the precontemplative stage. If the answer is no to the first three statements and yes to the last, the person is in the contemplative stage. If the answer is no to the first two statements and yes to the third, the person is in the

preparatory stage. If the answer is no to the first statement and yes to the second, the person is in the action stage. Lastly, if the answer is yes to the first statement, the person is in the maintenance stage (5). A yes to the first statement could also mean that the person is in the termination stage.

Processes of change

Activities or processes that people use in the respective stages of change to move to another stage can provide guidelines for interventions, in other words they can be a good guide for the person changing behaviour. There are ten processes that have proven to have the strongest empirical support. The following five approaches can be viewed as experiential or contemplative: *consciousness raising*, *dramatic relief*, *environmental re-evaluation*, *self re-evaluation* and *social liberation*, and the following five can be viewed as behaviourally or activity oriented: *counter-conditioning*, *helping relationships*, *reinforcement management*, *self-liberation* and *stimulus control* (5, 6, 16).

Consciousness raising involves seek new knowledge and information about physical activity.

Dramatic relief involves negative feelings associated with physical inactivity, such as breathlessness and excess weight. The negative feelings can decrease and can even become positive when the physical activity increases in scope.

Environmental re-evaluation involves re-evaluating how physical activity and physical inactivity affect one's surroundings. This can include the person's assessment of how a physically inactive lifestyle affects family and friends.

Self re-evaluation involves intellectually and emotionally re-evaluating the value of physical activity to one personally; for example, physical activity makes me stronger and more energetic.

Social liberation includes becoming aware, accepting and finding possible alternatives for physical activity in society, including everyday exercise.

Counter-conditioning involves handling situations that entail everyday physical inactivity. One example is taking the stairs instead of the lift.

Helping relationships involve getting help from others to be able to increase the physically active behaviour.

Reinforcement management involves changing physically inactive behaviour by rewarding physically active behaviour.

Self-liberation involves believing in, choosing and committing to greater physical activity, in other words feeling that one will manage to be regularly physically active.

Stimulus control involves taking control over the situations and causes that lead to physical inactivity, such as avoiding situations involving a great deal of sitting still.

Different processes of change are used in the different stages of change. Based on the empirical evidence, it is appropriate in the first stages of change to use cognitive, emotional and self-evaluation approaches to move forward in the stages of change. In the later stages, it appears as if people use more self-liberation, counter-conditioning, reinforcement management, stimulus control and helping relationships to move to the maintenance or termination stages. To succeed in a behavioural change, it is important to use the right process of change at the right time, in other words at the right stage of change (5, 16).

The processes of change should not be confused with techniques for behavioural change. The various processes can include many strategies or techniques to achieve change and these can also vary between people (5). For example, techniques that are used in the reinforcement management change process vary. One person rewards himself with some time in the sauna at one of three completed physical activity sessions a week. Another rewards herself with cinema tickets after a month of regular physical activity. The sauna and cinema tickets can be said to constitute different techniques for the reinforcement management process.

Intervening processes

The processes of change that were touched upon above have ties to certain intervening variables, including decisional balance and self-efficacy. The latter are the actual drive or the engine that means that a shift from one stage of change to another takes place through various processes of change. These intervening processes can be viewed as a type of spiral that moves between a certain stage or certain process, and then on to the next stage and (in part) other approaches and constantly drives the change forward, although with some relapses at times. *Decisional balance* reflects a person's inclination to change based on personal advantages and disadvantages of the new behaviour. It appears as if it is more important to work on increasing the advantages than on reducing the disadvantages (5, 16).

The concept of *self-efficacy* comes from social cognitive theory (22). To describe the concept of self-efficacy in Swedish, situation or activity-specific self-confidence or self-esteem is used, in other words faith in one's own ability (6, 15). This is a type of self-confidence that people have with regard to their personal capacity of being able to handle various situations. Consequently, self-efficacy varies depending on the activity, situation and requirements of skills in a specific area (5, 6, 22). For example, the same person can have a strong faith in her ability to lead groups, but a weak faith in her ability to lose weight. Another example is that the same person can have a strong faith in his ability to be physically active when the sun is shining, but a weak faith in his ability to be physically active when it is raining.

People's self-efficacy can be positively developed through the following four areas. The first area concerns acquiring positive experiences. Succeeding leads to a stronger faith in one's own ability. The second area concerns being surrounded by positive role models. Seeing other people overcome similar problems strengthens confidence in one's own ability. The third area concerns acquiring support from the surroundings. Positive and realistic feedback can strengthen self-efficacy. The fourth area concerns focusing on positive emotional and physical states. In the area of physical activity, it is important to keep in mind that the activity itself can generate negative feelings, such as fatigue, aches and pains. Being able to handle these negative feelings requires a high level of self-efficacy (22). It has been shown that self-efficacy plays a significant role in behavioural change in the area of physical activity (15).

Establishing regular exercise habits

The study

A dissertation by Wester-Wedman (3, 4) presents the results of a project in which 44 physically inactive persons, equal numbers of men and women, were monitored for 24 months in their attempts to become regular exercisers. The persons included in the study were examined with the help of surveys, self-evaluations, interviews, journals and condition measurements. The types of exercise studied were jogging or walking briskly. The participants were recruited through advertisements and were in the ages of 30–50 with varying educational and professional backgrounds.

In the terminology of the transtheoretical model, these people were in the preparation stage or maybe even in the contemplation stage. One year after the start of the study, a follow-up telephone interview was conducted with the people who registered for, but were not included in the experiment. There, it came forth that only a small number of the 115 (of a total of 120) persons that could be reached had changed their exercise habits in a positive direction in the past year, in other words the project comprised a helping relationship, which is one of the transtheoretical model's processes of change.

Data was gathered every three months during the first 18 months and then after 24 months, which meant that the process in the changed exercise habits² could be studied in a somewhat in-depth manner.

2. The term exercise/exercise habits refers to jogging or briskly walking the in the following.

Obstacles and their change over time

From the analyses, it came forth that the prospective exercisers met with obstacles and problems of various types in the first months. It was not just a question of putting on one's trainers and heading out for a jog or walk to suddenly discover that one was a regular exerciser. Many decisions must be made and many practical details must be resolved. Where should I jog? How far? What should I wear? Shoes? Do training clothes need to be washed every time? Where are they stored in between exercise sessions? My hair gets sweaty! What will the neighbours say? Other obstacles that are often mentioned were related to certain practical arrangements having to be in place to be able to exercise, primarily the creation of new routines and, for instance, arranging a baby-sitter. Disruptive traffic, bad weather, bad road conditions, a fear of dogs, a fear of being molested, a fear of the dark, a lack of motivation, a lack of time, injuries and illness were other obstacles that showed up among the novice exercisers. To support and help the participants with the aforementioned issues and questions, in the first three months they had access to coaches who were regular exercisers themselves (3, 4).

Eventually, answers were found to the questions and solutions to the various practical problems, such as a hook in the cellar where the training clothes can hang in between exercise sessions. Of course, this phase takes varying amounts of time for the different individuals, but after a few months most matters were resolved and it was relatively easy to realise the exercise plans. However, it is important to remember that things happen in people's lives all the time that force planned exercise sessions to be cancelled and sometimes it can be a question of "beginning all over again". This is nothing unique, but rather happens to most people (3, 4).

Experiences and their change over time

From the very beginning, starting to exercise provides different experiences that change in scope and content over time. At the beginning, there are quite a few negative experiences connected with a lack of condition and muscle strength, as well as feelings of monotony and boredom during the actual exercise. At the same time, there are also positive feelings of liberation, harmony, relaxation, a wonderful environment and pleasure at "getting the body moving". After the exercise, there is a relatively brief positive feeling – "it is nice to have done the exercise" (3, 4).

The change over time can be summarised by it initially being a matter of both positive and negative feelings in connection with the exercise, but once one is an accustomed exerciser, there are almost only positive feelings and experiences left during the exercise.

Effects and their change over time

There is considerable agreement that exercise provides positive effects. From Wester-Wedman's study (3), it is clear that both the type of effect as well as its scope changes over time. The effects that the exercisers talk about are categorised as mental or physical, in both cases either short-term or long-term, and cognitive – a clear head, clears the mind – and social – common interest, new subject of conversation.

With regard to the mental and physical effects, they change over time from being highly short-term/specific to becoming increasingly long-term in both cases. In addition, the balance between both types of effects changes over time so that the physical, which are more prominent in the beginning, after a long time of regular exercise, also make way for mental effects. Examples of short-term/specific physical effects include having the energy to jog a longer distance and becoming less out-of-breath during the exercise, while long-term effects can include better condition, weight loss or acquiring leg muscles. Short-term mental effects can include feeling relaxed after jogging or being energised by the exercise, while long-term effects can include feeling calmer, having better self-confidence, a stronger psyche and becoming happier and more stable (3). The change in the effects over time are illustrated in figure 1.

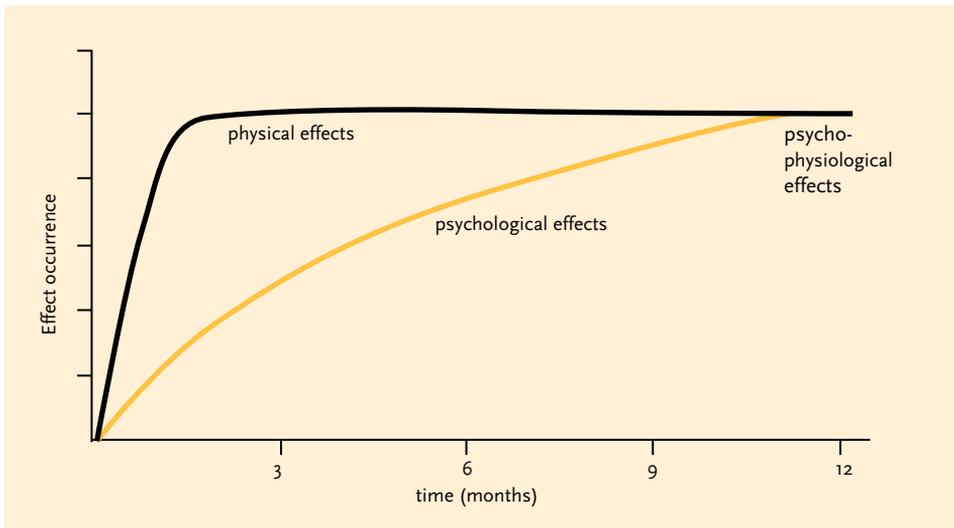


Figure 1. Development of physical and mental effects over time.

Motivational change

As described above, there is a development and change in the obstacles, experiences and effects as exercise continues. The novice exerciser has no experiences or perceptions to rely on and in the beginning, “external” rewards are needed, such as encouraging shouts from family and friends, weight loss or improvements in jogging time per kilometre (24). In this phase, it is a matter of repeating the behaviour as often as possible with the help of these rewards from without. In transtheoretical terms, one can talk about the processes of helping relationships or reinforcement management. At this time, one has not yet become a regular exercise in the sense that the habits have not been established.

Gradually as time goes by and the behaviour is repeated, the exerciser gains more and more positive experiences and more perceived effects that gain a more long-term nature. These experiences are incorporated in the motivations and gain increasing significance as motives for continuation of the exercise, while the external motives decrease in importance. There is a change of the motives from “externally motivated” to “internally motivated” behaviour. The motives have been internalised and the exercise habits are established (3, 4). The individual has gone from the action stage via the maintenance stage and in some cases to the termination stage.

The changes over time in experiences and effects of exercising described above can be graphically illustrated with the help of a free interpretation of Solomon’s opponent-process theory of acquired motivation (23).

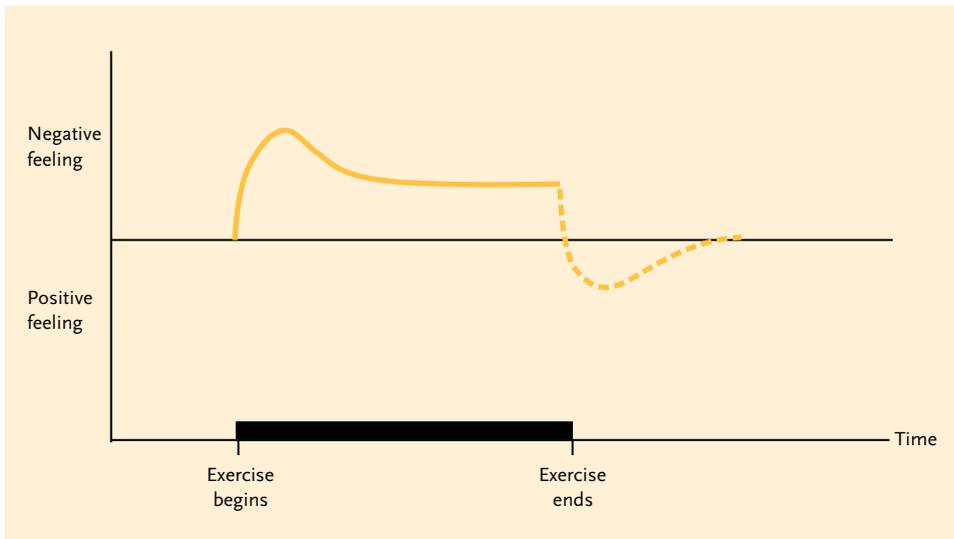


Figure 2. Perceptions of physical activity at an initial phase.

From figure 2, it can be seen that the perception of the actual exercise session in the initial phase is overwhelmingly negative, while the immediate after-effect is positive and relatively brief. The negative perceptions during the exercise session are primarily comprised of physical feelings of discomfort – heavy legs, heavy breathing, as well as boredom and monotony.

The positive after-effect that occurs immediately after the end of the exercise can be seen as a contrast effect that most often means that “it is nice that it is over”.

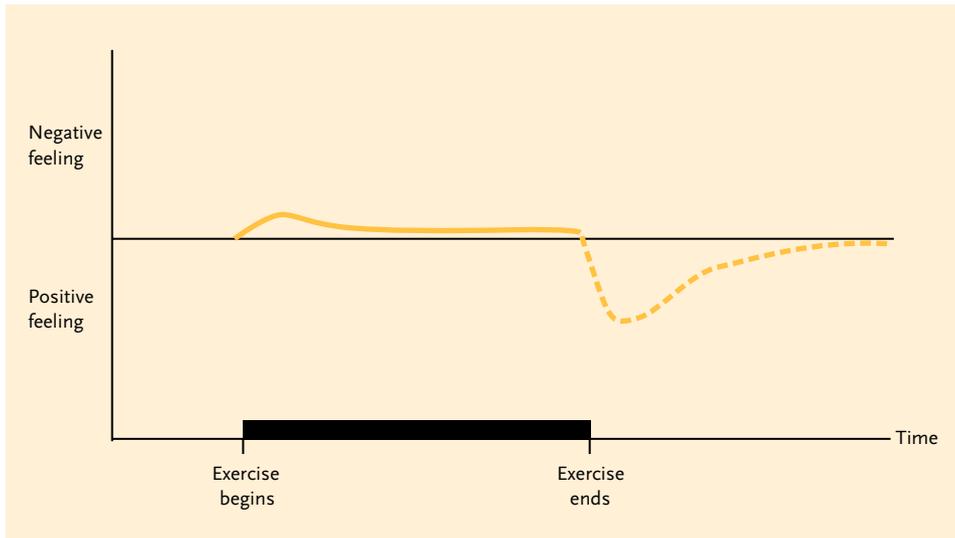


Figure 3. Perceptions of physical activity after having exercised for some time.

Figure 3, which describes the process in the experienced exerciser, shows that the perception during the actual exercise is nearly neutral. The physical exertion that the exercise nonetheless entails is perceived not at all as negatively, because condition and muscle strength has improved, something that often makes space for positive feelings during exercise.

The positive feeling after exercising is both stronger and more extensive in time in the experienced exerciser. According to the exercisers themselves, the content of this feeling is also qualitatively different than the novice exerciser’s immediate after-effect.

In summary, it can be confirmed that it takes time to go from being passive to being regularly active, at least six months or more. This is very individual and depends on the individual’s life situation otherwise, such as his/her age, gender, family and work situation. During this time, a great deal can happen – seasons change, a holiday can occur, the weather can occasionally be poor and work and family may demand extra attention. In addition to this, temporary illness or injury may also occur and there are also a great many other things that one is to have time to do during one’s free time (3, 4).

Women and men

In this chapter, the completely neutral term of “exerciser” was used without problemising any differences between men and women. From Wester-Wedman’s study (3), it is clear that men and women perceive different types of obstacles and their extent. Perceptions and effects are also of different types and scopes and the time that it takes to go from being a novice to a regular exerciser differs, due in part to the aforementioned factors. However, the process progresses in the same manner with the same factors involved, although at a different pace, for men and women. A follow-up study shows that many women prefer less physically demanding types of exercise, such as walking instead of jogging, when they themselves choose exercise activity (25).

The physical activity must be adapted to the individual’s conditions

An important part of the many messages given in connection with performing regular physical activity is that the conditions vary between people. Age, gender and religion are some conditions. Being a parent or not and having certain disabilities, etc. also belong to these. Some of these conditions cannot be changed and affect the possibilities of performing regular physical activity. Instead, the physical activity must be adapted to these conditions and look differently for a single parent than for a person who lives under other conditions, for example.

In addition to this, is the central aspect of achieving an impact towards becoming physically active. The transtheoretical model provides conditions for this to be able to occur (5). According to this model, it is accepted that the change means moving from being entirely uninterested in physical activity to being a regular exerciser. This move is about the same for everyone, but the change can look differently for separate individuals. In principle, this is a matter of going from a high level of external motivation to a high level of internal motivation (3).

Consequently, it takes significantly more time to adapt mentally than to do so physiologically. It is only when the two curves (see figure 1) meet that the exercise functions on its own – meaning as a result of internal motivation. Change takes time (3, 5, 14). Consequently, it must be a central component of the change process that it be allowed to take time and that the external support remains for a considerable period of time, in one way or other. Within healthcare, the local healthcare centres can, for example, be given a special role in this respect.

Acknowledgement

Huge thanks to Professor Yngvar Ommundsen, Norwegian School of Sports Sciences, Oslo, for constructive points of view and updates.

References

1. Bolin K, Lindgren B. Fysisk inaktivitet. Produktionsbortfall och sjukvårdskostnader. [Physical inactivity. Production losses and healthcare costs.] Stockholm: Friluftsförbundet i samverkan (FRISAM); 2006.
2. Bunkefloprojektet. [The Bunkeflo Project.] <http://www.bunkeflomodellen.com/>.
3. Wester-Wedman A. Den svårfångande motionären. En studie avseende etablerandet av regelbundna motionsvanor. [The elusive exerciser. A study of the establishment of regular exercise habits.] Diss. Umeå: Umeå University; 1988.
4. Wester A. Den svårfångade motionären. [The elusive exerciser.] In: Kindeberg T, Svederberg E, Svensson L, eds. Pedagogik i hälsofrämjande arbete. [Pedagogy in health promotion work.] Lund: Studentlitteratur; 2001. pp. 185-206.
5. Prochaska JO, Norcross JC, DiClemente CC. Changing for good. A revolutionary six-stage program for overcoming bad habits and moving your life positively forward. New York: Quill; 2002.
6. Faskunger J. Motivation för motion. En handbok för hälsovägledning steg för steg. [Motivation for exercise. A handbook for health counselling step-by-step.] Farsta: SISU Idrottsböcker; 2002.
7. U.S. Department of Health and Human Services. Physical activity and health. A report of the Surgeon General. Atlanta (GA): U.S. Department of Health and Human Services, Centers for Disease Control and Prevention, National Center for Chronic Disease. Prevention and Health Promotion; 1996.
8. Boström G. Levnadsvanor och hälsa. [Living habits and health.] In: Ardbo C, ed. Folkhälsorapport 2005. [Public Health Report 2005.] Stockholm: Swedish National Board of Health and Welfare; 2005. pp. 292-332.
9. Västerbotten County Council. Manual 2004 för Västerbottens hälsoundersökning. [Manual 2004 for Västerbotten health survey.] Umeå: Samhällsmedicin, Landstingskontoret, Västerbotten; 2004.
10. Weinehall L. Personal communication on 7 June 2007.
11. Arvidsson O, ed. Att förändra levnadssätt. Rapport från ett symposium om metoder att förbättra folkhälsa, 1 och 2 oktober 1981. [Changing lifestyle. Report from a symposium on methods to improve public health, 1-2 October 1981.] Riksbankens Jubileumsfond 1982:3. Stockholm: Liber/Allmänna förlag; 1982.
12. Glanz K, Rimer BK, Lewis FM, eds. Health behavior and health education. Theory, research and practice. 3. edn. San Francisco: Jossey-Bass; 2002.
13. Sallis JF, Owen N. Physical activity & behavioral medicine. Thousand Oaks (CA and London): Sage; 1999.
14. Stevens M. Groningen Active Living Model. Development and initial validation. Diss. Groningen: Rijksuniversiteit Groningen; 2001.
15. SBU (Swedish Council on Technology Assessment in Health Care). Metoder för att främja fysisk aktivitet. En systematisk litteraturoversikt. [Methods for promoting physical activity. A systematic literature review.] March 2007. Report 18. Stockholm: SBU; 2007.

16. Prochaska JO, Redding CA, Evers KE. The transtheoretical model and stages of change. In: Glanz K, Rimer BK, Lewis FM, eds. *Health behavior and health education. Theory, research and practice*. 3. edn. San Francisco: Jossey-Bass; 2002. pp. 99-120.
17. Spencer L, Admans TB, Malone S, Roy L, Yost E. Applying the transtheoretical model to exercise. A systematic and comprehensive review of the literature. *Health Promotion Practice* 2006;7:428-43.
18. Guillot MA, Kilpatrick M, Hebert E, Hollander D. Applying the transtheoretical model to exercise adherence in clinical settings. *American Journal of Health Studies* 2004;19:1-10.
19. Prochaska JO. Staging. A revolution in helping people change. *Manag Care* 2003;12:6-9.
20. Fallon EA, Hausenblas HA. Transtheoretical model. Is termination applicable to exercise? *American Journal of Health Studies* 2004;19:35-44.
21. Weinberg RS, Gould, D. *Foundations of sport and exercise psychology*. 2. edn. Champaign (IL): Human Kinetics; 1999.
22. Bandura A. *Self-efficacy. The exercise of control*. New York: W.H. Freeman and Company; 1997.
23. Solomon RL. The opponent-process theory of acquired motivation. The costs of pleasure and the benefits of pain. *American Psychologist* 1980;35:691-712.
24. Jung J. *Understanding human motivation. A cognitive approach*. New York: MacMillan; 1978.
25. Wester-Wedman A. Hur gick det sen för motionärerna? En uppföljning av motionsprojektet. [How did it go for the exercisers afterwards? A follow-up of the exercise project.] In: Patriksson G, ed. *SVEBI:s årsbok 1990*. [SVEBI Annual 1990.] Svensk förening för Beteende-vetenskaplig Idrottsforskning; 1990.

* Deceased

5. *Motivational interviewing about physical activity*

Authors

Barbro Holm Ivarsson, Psychologist, Stockholm, Sweden

Peter Prescott, Psychologist, Bergen, Norway

Introduction

Unfortunately, there is no simple medicine you can take to become more physically active. You have to do the job yourself and as most well know, it is not always easy. One's attitude to becoming more physically active also varies over time. You can be determined and make concrete plans just to feel your motivation disappear, your good plans seem unrealistic and your desire disappears.

Motivational interviewing (MI) is currently mainly used to help people change lifestyles and was originally developed in counselling work with people addicted to alcohol. Motivational interviewing is used, for example, as a method in professional counselling regarding the harmful use of alcohol, illegal drugs, tobacco use, harmful gambling, risky sexual behaviour and to promote greater physical activity and good eating habits and to combat obesity. This method fits in all contexts when it is desirable to stimulate another person to change behaviour without inciting resistance, and is therefore very well suited to discussions of physical activity in, for example, healthcare, preventative healthcare, schools and the sports world.

Motivational interviewing can be used as an independent intervention to generate interest and motivation, prepare and help a patient/client to commence with change. Motivational interviewing can also be integrated into other treatment, which appears to improve the result of the treatment in question.

This chapter includes a description of how motivational interviewing can be applied to physical activity. To illustrate this, we can follow a motivational interview between Per, who is a keep-fit coach, and Eva, an unmarried 35-year-old social worker, who has recurring, diffuse back problems (lumbago).

In continuation, the abbreviation MI is used for motivational interviewing as well as the designations interviewer and client.

Motivational interviewing

Motivational interviewing is a client-centric empathetic conversational method. This means that the interviewer listens, tries to understand the client's perspective and emphasizes that the client shall contemplate his/her values, find his/her own answers and him/herself decide regarding change. At the same time, MI is a guiding method in the way that the interviewer chooses to investigate and focus on some of the topics the client presents, but at the same time chooses to not follow-up other topics. The interviewer thereby "guides" the client towards change.

Motivational interviewing requires good listening skills. The interviewer listens with an accepting, non-moralising interest and tries to understand the client's problems, rather than convince or present his/her own solutions to them. This does not mean that the interviewer does not provide information or practical advice, but it is formulated as suggestions that the client can choose to accept or not: "Would it be OK if I say something about what has helped other people in the same situation?"

MI appears to have the greatest effect on the least motivated clients, both due to the client-centred style and the manner of addressing resistance to change. This method is not used to try to get a client to do something that he or she does not want to do, but rather has the objective of stimulating the client to change his/her own conditions in a respectful manner

Lifestyle change and change talk

In MI, the interviewer tries to bring out the client's own desire for change and resources to change, including cognitive and emotional resources and concrete actions. The interviewer also strengthens the client's confidence by showing faith in the client being able to make a change, and emphasizing that change is the client's own choice; the client is responsible for his/her own life and knows best how the change shall be made.

We can recognise clients that work constructively with change in the way they speak. They express motivation, have ideas about practical methods, believe in themselves, make decisions and pledge to follow concrete plans. It is typical for MI that the interviewer tries to bring out, specifically listen for and reinforce the client's talk of change. Talk of change comprises statements that indicate that the client is actively working on his or her own change. When the client talks, he or she shapes and strengthens his/her own values, and the interviewer tries to actively stimulate this.

Change talk can be divided into two main categories: preparatory change talk and commitment to a concrete plan. Preparatory change talk lays the foundation and leads to commitments/decisions, and research indicates that making a decision or commitment is associated with lifestyle change. This can be illustrated in the following manner:

MI → preparatory change talk → commitment/decision → lifestyle change

Preparatory change talk can also be divided into two main categories: motivational statements and statements of faith in one's own ability to be able to make the change.

Examples of motivational statements:

- Desire for change: "I want to get into better shape. I actually like to exercise. I would like to change this."
- Concrete reasons for change: "I am tired of having a wardrobe with clothes sized XXL. I feel tired, am in poor condition, am not at all in shape."
- Necessity: "I just have to begin to exercise. Things will go bad otherwise."

Examples of statements of faith in one's own ability to be able to make the change:

- "I have managed harder things in life. It will go well if I take one step at a time and watch out for the inner-saboteur – the thoughts that can ruin things for me."

Examples of statements of commitment/decisions:

- "I have thought this through and have made a decision. This is actually one of the most important things in my life right now. I plan on already getting started tonight."

In MI, the interviewer needs to be able to identify change talk, because it is what the interviewer tries to listen for, evoke and reinforce. Active listening in the form of *open questions* and *reflection* is used to evoke, keep focus on, add nuance to and reinforce change talk. *Summaries* are used to gather together the change talk expressed by the client.

Opening of the interview

As a rule, a motivational interview begins with the interviewer investigating the background of the contact. The client needs to feel safe and a climate of cooperation must be established. If the client has been pressured by someone else to come to counselling, it is important that the interviewer express understanding for it not always being so easy to come to counselling under such conditions, but that the counselling would still hopefully be able to benefit the client.

Sometimes, one must *set an agenda* for the conversation, if there are several topics that the client has difficulties with and wants to talk about. If so, it is wise to begin by investigating what the client is most concerned with and is motivated to talk about. If the topic is a given, one can begin by asking the client to tell about the problem.

Per: "First, I want to ask you to tell me about your back, how your back problems affect your life and what you have done to address the situation. And then I would like to hear a little about how you view exercising, the good and the bad. Is that OK?"

Active listening

The basic skill in MI is known as active listening, which contributes to a good climate of cooperation. Through active listening, the interviewer can gain an understanding of the client's view of his/her situation and thoughts of problems and solutions, at the same time that the client understands him/herself better. Besides listening, the interviewer uses *open questions*, *reflection* and *summaries* to actively show his/her understanding of the client's perceptions. *Affirming* the client and his or her efforts is also fundamental.

A. Open questions

An open question begins with a question word such as "When?", "Where?", "How?" or "What?". These are questions that grammatically cannot be answered with a "yes" or "no", but evoke the client to explain, to talk. The question "Why" should be used with caution, because it is so charged that it can easily make people feel accused. Of course, this is particularly true if asking about why the client does something that seems foolish.

A few examples of relevant open questions:

"Can you tell/say a little about...?"

"How did you come to think that it might be necessary to change this?"

"How does your back pain affect your everyday?"

"What could you do if your back were better?"

"What have you tried before to make your back better?"

"What experiences have you had in terms of exercise, for good and bad?"

"What are your thoughts with regard to becoming more physically active if you think about the future?"

"What can be the next step towards change?"

Per: "Tell me about your back problems and how they affect your everyday?"

Eva responds: "Well, it goes a bit up and down. Sometimes, my back is good, but other times ... well, it's mostly just a painful struggle. When it's really bad, I can't handle lying down or sitting. I can barely go to work. Sometimes, I almost can't manage to put on pants and socks. The pain is so great that I don't have the strength to be active. It also affects my sleep. Everything is so tough, stressing and burdensome. It's always hectic at work and my job is to support people who have an even more difficult time in life than I do. It's tough enough as it is, but when I'm in pain myself, well ... sometimes I call in sick. In addition to this, I don't have the strength to be social. I just feel annoyed and can't bother caring about others."

Notice Eva's change talk in the form of motivational statements about the negative consequences of her back pain. Also notice that she has no change talk that concerns the desire to change or it being necessary to change. Nor does she express any faith in her ability to be able to make a change or says anything about commitments or decisions.

B. Reflection

Reflecting means re-stating, being a mirror for the client. Interviewers who have reflections as a natural part of their communication style are perceived as empathetic. One can reflect by re-stating a sentence, part of a sentence or just a word. It is important to emphasize that a reflection is not a question.

The interviewer can reflect back without changing anything particular in what the client has said:

- "Parrot": "It's always hectic at work", "Hard to get dressed".
- Synonym: "Your back pain varies", "You have a hard time handling everyday things".

The interviewer can also choose to reflect an underlying statement or emotion:

- Underlying sentence: "Having back pain ruins a lot for you".
- Underlying sentence: "You're frustrated", "You feel dejected".

The interviewer selects reflection to guide the conversation so that it focuses on the right topic. The interviewer can thereby choose one topic over another. If Per reflects "tough and burdensome", Eva will probably continue talking about what it is like to be worn out and to lack energy. If Per reflects "it goes a bit up and down", the conversation will probably continue on this topic and if Per reflects "always hectic at work", Eva continues to talk about how things are at work.

Per: "When your back hurts, your day is different." (Reflection)

Eva: "Yeah, everything is so much harder and more strenuous. It's a lot about the pain, how I can get through the day. And that's what it's like until my back gets better."

Per: "Everything is just one big struggle while it's going on, and then it gradually passes. How has it been to try physical activity to make your back better?" (Reflection and open question)

Eva: "When I'm in pain, I don't have the strength to do much. When my back is better, I don't exercise much anyway. I try to get motivated, but don't get very far. Now and then, I pull myself together and keep at it a while. I know I should exercise more."

Notice Eva's change talk. There are examples of motivational statements, "harder and more strenuous", intent to change, "I should exercise more," decisions for short periods, "Pull myself together". "Keep at it a while" also says something about her having concrete ways of being physically active.

C. Summaries

Summaries and reflections are actually two sides of the same thing. The difference lies in how much is included. In a reflection, one gives back one or two elements of what the client has said. A summary includes multiple elements. Summaries work like small résumés and contribute to these parts of the conversation being remembered and also potentially reinforced. An underlying message in summaries is empathy: "I hear what you

are saying, I am trying to understand and remember it because what you say is important and I want to check whether I have understood you correctly.”

MI functions simply in theory, but is more difficult in practice. Even if one is accustomed to communicating and listening, the systematic and goal-oriented active use of active listening demands quite a bit of practice before becoming an automatic skill. What proves to be most difficult is to learn to make reflections in a systematic, yet natural manner. At the same time, reflections are the most fundamental element of MI. In contrast to open questions and reflection, summaries do not occur as often in daily speech, but are rather more reserved for professional communication.

Per: “Can I make a little summary and see if I have understood you correctly?” (Ask for permission)

Eva: “Yes.”

Per: “Your back problems come and go. Sometimes, your back is good and sometimes bad. When your back is bad, you are plagued by pain and sometimes it’s just a matter of getting through the day. It affects your mood, energy and what you get done both privately and at work. You succeed in exercising for short periods, but it doesn’t sound as if you are completely satisfied with your own effort.” (Summary)

Eva: “Yes, that’s right.”

Per: “You mentioned that you should exercise more. How do you view physical activity for preventing and alleviating your back pain?” (Open question)

Eva: “I know I should exercise, but it’s also a struggle.”

Per: “What have you tried that has helped?” (Open question)

Eva: “Going to the gym and doing exercises is nothing for me. Taking walks works best. The problem is getting it in as a part of my daily routine. It takes time to go for walks. Work takes so much time and is so stressful.”

Per: “Work takes a great deal of time and is stressful and, so far, it’s been hard to make exercise a part of your daily activities, at the same time that you know that it helps. Taking walks works best, in your experience.” (Summary)

Scale questions to evoke change talk

We will look more closely at two so-called scale questions that can be used to evoke change talk about motivation in the form of the importance of change and a belief in the ability to succeed.

Scale questions about weight

Per: “Is it OK if I ask a few questions about how you view physical activity?” (Ask for permission)

Eva: “Yes.”

Per: “Think of a scale from 0 to 10, where 0 means that it’s not important at all and 10 means that it’s very important. How would you rate your view of physical activity on such a scale?”

What figure would you give yourself?"

Eva: "5."

○ _____ 5 _____ 10
 Not important at all Very important

The interviewer can use a follow-up question downwards on the scale to evoke change talk:

Per: "What is it that makes you say 5 and not 3, for example?" (Open question)

Eva: "Well, it's important to exercise to get a better back. I know that it helps and my back is killing me."

A follow-up question upwards on the scale also works well:

Per: "You rate yourself as a five because you have a lot of back pain periodically. What would have to happen for you to rank yourself higher on the scale?" (Reflection and open question)

Eva: "Yeah, you tell me ... it would be if it gets even worse. And maybe if I start walking more and get better. Then it will be important to continue."

Per: "So both worse pain and exercise that works could result in a higher rating." (Reflection)

Scale questions about faith in the ability to succeed if one is determined

Per: "If you really decided to exercise regularly, how much faith do you have in your ability to succeed? On a scale from 0 to 10, where 0 means that you have no faith at all in your ability to succeed and 10 means that you could do it without a doubt?"

Eva: "As it is now, 2."

○ _____ 2 _____ 10
 No faith in the ability Strong faith in the ability
 to succeed to succeed

Follow-up question downwards on the scale:

Per: "You still say 2, but not 0." (Reflection)

Eva: "I know that I could do it if I really want to, but I know what it's like when I'm going out after a hard day at work or when it's raining or I'm tired. After all, I've managed it before periodically. It's a question of self-discipline."

Follow-up question upwards on the scale:

Per: "You have some self-discipline that can help you and you have managed it periodically before. What would you need to go a little higher on the scale, like a 3 or higher?" (Reflection and open question)

Eva: “It’s a question of making a decision. Getting exercise into the day-to-day and getting started. I actually value exercising, I feel better both physically and mentally.”

Per: “You think it is pretty important to start to exercise. You’ve mentioned several reasons. On the other hand, you don’t have very much faith in your ability to really get it done. But self-discipline and strong determination would help. It seems as if you have some self-sabotaging thoughts that make your self-discipline fail.” (Summary)

Readiness for change

Clients have different levels of readiness for change, which can roughly be divided into three stages.

Table 1. Stages of change.

Not ready – uninterested	Uncertain – ambivalent	Ready to act
Low readiness or disinterest in change	Shifting readiness for change, ambivalence	High readiness for change
Focus:	Focus:	Focus:
<ul style="list-style-type: none"> • Create discrepancy • Evoke ambivalence 	<ul style="list-style-type: none"> • Investigate ambivalence • Decision 	<ul style="list-style-type: none"> • Practical methods • Commitment to follow a plan
What one can talk about:	What one can talk about:	What one can talk about:
<ul style="list-style-type: none"> • View of the situation now • Negative consequences • View of physical activity 	<ul style="list-style-type: none"> • Advantages/disadvantages of the situation/with change • Obstacles and solutions • Small steps to try change 	<ul style="list-style-type: none"> • Practical planning • Ways of achieving success • Point in time

The transition between stages is sliding and they can also overlap one another. The client’s attitude to change often varies over time and it can sometimes seem as if it shifts between disinterest, ambivalence and readiness to act in the course of a conversation. Ideally, one (or more) motivational interviews lead to the client deciding to make a commitment to change, but in terms of clients that are early in the change process, the objective is instead to activate motivation thoughts. Changing one’s way of life is a process that takes time. The interviewer should not expect major changes on the short term among those who are at an early stage in the process, but if the client gets help in thinking about the habit and its consequences, a change can come about earlier than it would have otherwise.

Not ready – uninterested

Clients who are not ready for change as a rule do not voluntarily seek counselling in life-style issues. They are often pressured by others into therapy or the interviewer brings up the topic when the client seeks help for another problem. Consequently, the client can have a resistance to talking about the problematic habit, in this case physical activity. The style of the interviewer can therefore be crucial to whether it is a constructive conversation about change. When clients are pressured into counselling, it is important that the interviewer show respect and understanding for the client’s aversion to talking about the matter.

Initially, one should discuss the background of the contact and talk about what the conversation is about to create trust: “It seems as if you’ve been sent here by your doctor. What do you think? Now that you’re here anyway – is it OK if we nonetheless talk a little about your back pain and about physical activity and see if I can help you somehow?”

The interviewer should ask the client for permission to talk about the subject: “There are several things that can affect back pain. Is it OK if we talk a little about it?”

Clients with little motivation tend to have more resistance to attempts to make a difference. The interviewer’s ability to listen respectfully is therefore absolutely crucial to whether the client can investigate problems in a constructive manner or react with resistance and defence. If the interviewer reflects and summarises change talk and overlooks the client’s counter arguments, one can counteract negative reactions. It is a matter of sowing small seeds of change and evoking curiosity. We call it creating a discrepancy – a difference – between how it is and how the client thinks it should be.

For a summary of the interview focus in this stage, see table 1.

Uncertain – ambivalent

The objective of this stage is to investigate the ambivalence that exists towards the life habit and to potential change, and to help the client strengthen a desire for change. Most of all, the interviewer wants to stimulate the client to make a decision or take a step in the direction of change, although he or she remains uncertain.

Eva’s ambivalence to taking walks is illustrated in table 2:

Table 2. Examples of arguments for and against change.

—	+
<p>Arguments against change (for status quo)</p> <ul style="list-style-type: none"> • Hard to find space for it in pressured daily life • Hard to find energy to go out • Self-discipline is a challenge • Takes time • Causes more pain in the beginning • Uncomfortable when the weather is bad 	<p>Arguments for change (against status quo)</p> <ul style="list-style-type: none"> • Feel better physically and mentally • My back is killing me • Less pain on the long term • Less annoyed • Sleep better • Have more energy at work and in private, more social • Better self-confidence

Eva is ambivalent to becoming more physically active. Ambivalence is actually a motivational conflict between “want/don’t want”. However, it also looks as if Eva finds herself in another common dilemma; her faith in her ability to be able to manage it is lower than her motivation, which can be derived from the responses to the scale questions.

An obligating decision can help Eva increase her motivation. Then, she needs to work out a plan that is realistic. Eva also probably has self-sabotaging thoughts, that is to say automatically negative thoughts that pop up and cause determination to wane.

Per: “What speaks against taking regular walks is that it takes time, sometimes it is difficult to find the energy and it demands self-discipline. You also feel that it can also cause more pain in the beginning and is hard. Sometimes the weather keeps you from walking. At the same time, you think that you have to do something because your back is killing you. You feel worn, tired and annoyed and are in a great deal of pain. You think that regular exercise will have a good impact on both your body and mood, you can get more energy at work and in your private life, and become more social. Additionally, deep down, you actually like to take walks (Summary) ... Eva, what are you going to do now? What will be the next step for you?” (Open question, key question)

Eva: “Well, when you look at it like that, there’s only one thing I can do. I just have to pull myself together and get started for real, but it is difficult. I have to really decide this time.”

In his summary, *Per* includes some of what has come forth in the previous conversation that reinforces *Eva*’s thoughts in the direction of change. *Per* concludes with the key question about how *Eva* can take a step further.

For a summary of the conversational focus in this stage, see table 1.

Ready to act

When the client is ready to get started with the change, focus will be on strengthening the commitment to change and working out a concrete and realistic plan for change. In addition, at this stage the client will be more set on cooperating to find solutions to difficulties and obstacles. Although the client’s own ideas are best, the client is also receptive to practical advice.

Per: “Can I ask you an important question?”

Eva: “Yes.”

Per: “Do you really mean that you plan to start to exercise?” Are you serious?”

Eva: “Yes, I have to. I have no choice.” (Commitment)

Per: “OK. Then I would like to go further and look at two things. First: How are you going to go about it in concrete terms? What does your plan look like? Secondly: Self-sabotaging thoughts – are there any that can ruin it for you now?”

For a summary of the interview focus in this stage, see table 1.

Offer information

Although the main strategy in MI is to activate the client’s own ideas about solutions, the interviewer also offers information and gives advice and suggestions where necessary. When the interviewer does so, it is important to activate the client. It is often difficult for the client to accept, remember and convert the interviewer’s ideas and suggestions into practice. Information and advice that does not feel relevant and is not desired, easily incites resistance, particularly in clients with low motivation. It is therefore important to ask for permission to give information and check what the client already knows so that the information can be tailored to the client’s needs.

1. Prepare

Inform the client that information is coming and ask for permission:

Per: “I would like to tell a little about my experiences of what often happens with one’s thoughts when one does not take a daily walk as decided. Is it OK if I talk a little about this?”

Eva: “Yes, that would be good.”

2. Adapt

The interviewer investigates what the client already knows about the subject to avoid giving information that the client already has. The client’s answers are confirmed and the significance of the information is reinforced by follow-up questions and reflections:

Per: “Before I tell a little, I want to ask you what you have noticed yourself. What thoughts keep you from getting out and going for a walk?”

Eva: “It’s hard to say. I sort of lose my spark. Sometimes, I don’t have the energy or it’s something with the weather. I find excuses to stay home.”

3. Reflect and provide information

Per: “It’s not always easy to put what happens into words. When one has to change habits, it’s not uncommon to have what we call self-sabotaging thoughts. They are thoughts that come on their own and that one does not notice. They can cause self-discipline and motivation to wane. For example: ‘Not today.’, ‘It’s too cold, rainy, hot, etc.’, ‘It’s been a hard day at work, I deserve to rest.’, ‘I’m too tired.’ ‘It’s no use anyway, I’ll never be able to manage it’.”

4. Give the client the opportunity to process the information

Per: “What do you think about this?”

Eva: “I recognise that. It’s like what I would have said myself, but I don’t know what I’ll do to not give in to those thoughts.”

Per: “Self-sabotaging thoughts are something that nearly everyone has when they’re going to change habits. It helps to be aware of these thoughts and meet them with constructive counter arguments. Two questions can be of help to understand more about this: 1. What happens when you do not take the walk you planned? 2. What happens if you follow the plan and take your walk anyway, the times that you don’t feel like it? The answer to the latter question says something about what you already do that helps. It is important to use such strategies more determinedly to keep your motivation up. Shall we look at this more closely?”

After having given information, Per goes on to try to evoke Eva’s own resources.

Meet resistance with respect

As previously mentioned, clients can feel resistance to both counselling and change and it is important to not give this resistance too much space in the conversation. The way the

interviewer meets the client's scepticism and counter reactions is crucial. The interviewer can create resistance by trying to push harder than what the client is prepared for and by arguing, confronting, provoking, convincing or using other strong attempts to influence. A counter reaction can also be evoked in the client when the interviewer adds something new. This is often expressed in the form of the client saying: "Yes, but ..."

One way resistance is expressed is through the client's so-called expressions of resistance. These often go in the opposite direction from change talk, and concern for example change not being necessary or desirable, the habit having positive effects, the time not being right ("Not right now") or helplessness. A great deal of resistance in counselling conversations is associated with lower client and counsellor satisfaction and worse effect of the treatment.

As a method, MI prevents resistance by the interviewer using as respectful and accepting a style as possible. Meeting the client where he or she is in the change process and showing understanding of ambivalence also reduce resistance. In addition, a fundamental principle in MI is that the client chooses if he or she wants to change living habits or not, the way to do it and the time, and the interviewer shows this.

In that the MI interviewer tries to affect the client in the direction of lifestyle change, resistance will most likely be activated, however. A common strategy for addressing resistance in MI is to take a step back and reflect the client's opinions. This is called "rolling with resistance". With this, one shows respect for what the client says and tries to understand the message behind the resistance.

Per: "It would be good for you to start exercising regularly." (Advice)

Eva: "Yes, but with the way my weekdays look, I simply don't have the chance of making it work, but of course it would be good for my back."

Per: "Weekdays are tough and it is difficult to find time to exercise, at the same time that it would have been good for your back." (Double-sided reflection)

Eva: "Yes, I know that I should, but I can't see how I can manage it ... maybe on the weekends."

Per: "Before we look at how you can arrange it, maybe we can look more closely as what would be good for your back."

Per meets the resistance with a so-called double-sided reflection that reflects both sides of the matter, takes a step back and tries to investigate the motivation.

Follow-up

When possible, one should offer the client follow-up conversations.

Per: "Changes in lifestyle are generally not made over night, they often take a while before one gets it to work. Shall we meet again so I can hear how it's going for you?"

Eva: "Gladly, I think that is a good idea."

If the client says “No, thank you,” the interviewer can answer: “OK, then I would like to wish you good luck with what you’ve decided. It will certainly go well (show optimism and faith in the client’s ability). You are always welcome to get in touch if you would like to talk a little more.”

Evidence

In a metaanalysis, MI’s promising effects and potential are summarised with regard to increasing flexibility in programmes for diet and physical activity (1). However, there are still relatively few studies in the area, but more studies are under publication.

References

1. Hettema J, Steele J, Miller WR. Motivational interviewing. *Annu Rev Clin Psychol* 2005;(1):91-111.

Websites

www.fhi.se/mi – Swedish website about MI.
www.motivationalinterview.org – international website about MI.
www.somra.se – website about MI and alcohol abuse.

Books and manuals

Barth T, Børtveit T, Prescott P. Förändringsfokuserat rådgivning. [Change-focused counselling.] Oslo: Gyldendal; 2001. Stockholm: Liber; 2004.

Barth T, Näsholm C. Motiverande samtal – MI. Endring på egne vilkår. [Motivational interviewing – MI. Helping people to change on their terms.] Lund: Studentlitteratur; 2006. Bergen: Fagbokforlaget; 2007.

Holm Ivarsson B. Motiverande samtal MI. [Motivational interviewing MI.] In: Fossum B, ed. Kommunikation. Samtal och bemötande i vården. [Communication. Conversations and personal treatment in healthcare.] Lund: Studentlitteratur; 2007.

Holm Ivarsson B, Pantzar M. Introduktion till motiverande samtal. En handledning för skolhälsovården. [Introduction to motivational interviewing. A guide for school healthcare.] Östersund: Swedish National Institute of Public Health; 2007.

Miller WR, Rollnick S. Motivational interviewing. Preparing people for change. New York: Guilford Press; 2002. Norrköping: Kriminalvårdens förlag; 2003.

Prescott P, Bortveit T. Helse og atferdsendring. [Health and behaviour change.] Oslo: Gyldendal Oslo 2004.

Rollnick S, Mason P, Butler C. Health behaviour change. A guide for practitioners. New York: Churchill Livingstone; 1999.

6. Assessing and controlling physical activity

Authors

Maria Hagströmer, PT, PhD, Associate Professor, Department of Neurobiology, Care Sciences and Society, Division of Physiotherapy, Karolinska Institutet, Stockholm, Sweden

Peter Hassmén, PhD, Professor, Department of Psychology, Umeå University, Umeå, Sweden

Summary

Physical activity has many healthy effects, both physical and mental, and is therefore used both for the prevention and treatment of disease. To make the right prescription of physical activity possible, and to help individuals find the right load and evaluate prescriptions issued, reliable methods and measurement instruments are necessary. This chapter describes various measurement methods, their reliability and limitations, and how they can practically be used in connection with the prescription of physical activity.

Assessment of physical activity

The outcome of a physically active lifestyle is that different bodily functions are improved, such as aerobic fitness and strength. Other functions and parameters can also be affected, such as body weight, waist circumference, body composition, blood pressure and lipoproteins. The same applies to mental health, where conditions of depression and anxiety can be reduced through physical activity. Besides these effects, the actual physical activity or frequency of exercise can be measured or assessed with different instruments. In this chapter, the concept of assessment is consistently used instead of measurement since certain measurements are direct while others are indirect and are based on the participant's own assumptions (1, 2).

Physical activity is another word for bodily movement that results in increased energy expenditure. It is also a complex behaviour. Accordingly, physical activity can be assessed in the form of energy expenditure or as a behaviour. The components of the activity that have shown a correlation with health are intensity, duration and frequency. For health-enhancing effects (3, 4), the activity is recommended to be carried out at an *intensity* that is at least moderate, for a combined time (*duration*) of at least 30 minutes and preferably

every day (regular *frequency*). A few different methods are described below that can be used to assess the degree of physical activity.

Questionnaires

Questionnaires for assessment of physical activity are the most common method and there are currently hundred of variants available (2, 5). The most basic only ask about the individual's exercise habits and offer pre-determined responses on a 3–5 degree scale. The more advanced ask exactly what has been done and for how long, and maybe even how often the individual has been physically active during a certain period of time (the past week, month, or the like). Most questionnaires ask about the degree of exertion, which is affected by the individual's capacity. It is likely that the better aerobic fitness and strength the individual has, the easier the activity is perceived to be. In addition, the individual's body weight is of significance since it costs more energy to carry around more weight and the activity is then perceived as more strenuous.

To calculate the energy expenditure from questionnaires, the given activities are weighted with an energy expenditure measure for the activity. Metabolic equivalent (MET), or a multiple of the oxygen uptake at rest, is often used (6, 7). Resting corresponds to 1 MET and calm activities 1–3 MET. Activities of moderate intensity can vary between 3–6 MET and activities that entail a high level of exertion are over 6 MET.

On prescription forms for physical activity, there is a question where the prescriber can obtain a rapid view of health-enhancing physical activity. It asks: *How many days in the past week have you been physically active with at least moderate intensity during a total of 30 minutes per day?* It is followed by the same question, although with a time perspective of “a regular week”. The question has been method-tested in a project at the Karolinska Institutet (8).

However, if exercise or training habits are asked about, it should be noted that the respondent only assesses parts of the total physical activity completed. These questions most often show a high degree of reliability and validity, since it is easier to remember what is done regularly and with a higher intensity (1, 2, 9). It is also exercise that has shown the strongest association to achieved health effects. If exercise is prescribed, then it is also exercise that should be evaluated. However, if everyday activities are prescribed, they cannot be assessed with questions about exercise.

As presented by many studies, it has often been difficult to compare physical activity levels within a country, but especially between countries since different methods have been used. This has led a group of international researchers to develop a method that measures all health-enhancing activity and is standardised and can be used internationally. The International Physical Activity Questionnaire (IPAQ) was developed and method-tested at the beginning of the 2000s (10, 11) and is now a national and international standard in several countries and organisations (WHO, EU). This instrument has also been method-tested in Sweden, where the results indicated that its reliability and validity is on a par with other subjective instruments (12, 13).

Diaries

To determine the total energy expenditure and also obtain a measure of how the activity is divided over the day, diaries can be used (2, 14, 15). The journal should include what has been done based on given examples with a certain time interval (every 5th or 15th minute). These have shown a high degree of concordance with the total energy expenditure, but are time-consuming for the participants, which means that they are seldom useful in large-scale studies.

Movement sensors

To escape from the systematic errors that self-reports of physical activity entail (it is difficult to remember the degree of exertion, over-reporting is common, etc.), objective methods are used. The instruments that can assess activity directly are *step-counters* and *accelerometers*.

Step-counters provide a rough measure of the activity and their use can be beneficial in interventions so the persons themselves can follow their activity development since direct feedback to the individual is possible. It should be noted that there are many different brands of varying quality. Depending on sensitivity and so on, the variation in the number of steps can be more than 20 per cent. A good step-counter should be method-tested in terms of reliability and validity, have a cap, not have a filter function and should be robust. The sensitivity should be 0.35 G, which means that it is sensitive to natural human movement (16). The disadvantage of step-counters primarily lies in the fact that they say nothing about intensity. This means that if a person walks 100 meters, the step-counter will register approximately 110 steps, while it only registers approximately 70 steps if the person runs.



Figure 1. Example of an accelerometer and how it is attached to the body.

Accelerometers are more advanced instruments, which also means that they are more precise. They measure acceleration in one or more orthogonal planes with the help of either a mechanical pendulum or a digital function. Acceleration is a direct measurement of body movement and the higher the acceleration, the greater the intensity. Besides total physical activity, accelerometers can also provide a measure of intensity, duration and frequency, that is to say the

pattern of the activity. Another strength that the accelerometer has is that it can assess inactivity and sedentary behaviour. However, accelerometers are more costly than step-counters, but they are preferable if greater precision is desired. A good accelerometer should be method-tested and easy to wear (17, 18).

With accelerometer technology, a time period can also be set over which the activity should be summarised (a so-called epoch). The shorter the time period, the more precision is possible. For adults, the time period of one minute is most often used and for children 10–15 seconds. In addition, newer models of accelerometers manage to store data for a longer time, which means that measurements can be carried out for months if desired, but the individual's activity is usually measured for a week. Accordingly, an accelerometer produces enormous amounts of data. If a 15 second time period/epoch is used, there will be four points per minute, times 1,440 minutes per day, times seven days per week, resulting in approximately 40,000 data points per individual. An extensive post-treatment of collected raw data is needed before a comprehensible description of an individual's physical activity can be made. The advantage of using accelerometers often outweighs the disadvantages, however.

Both step counters and accelerometers are insensitive to activities that take place with the upper-body or activities such as swimming and cycling. In spite of this, they provide a good view of overall activity and for accelerometers also of how the activity is divided over the day. Studies have shown that approximately 90 per cent of the time is spent sitting, standing and walking, that is to say that the persons studied carry out activities that the movement sensors can register.

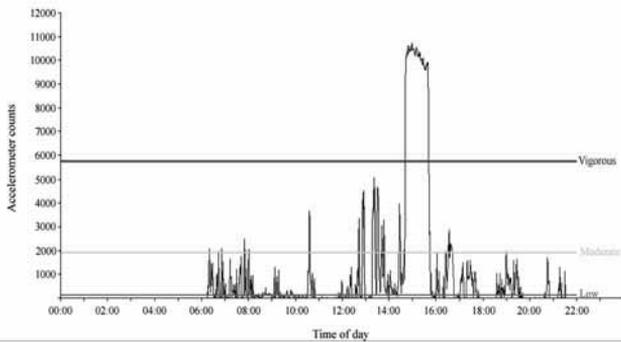


Figure 2. Example of how a day can look, registered with an accelerometer.

Heart rate monitoring

One way of indirectly measuring physical activity is to use heart rate monitoring, such as with a heart-rate monitor. With the help of a sensor around the chest and a receiver in the form of a watch, the pulse can be continuously monitored. The pulse has a virtually linear relationship to exercise intensity (primarily aerobic work – with oxygen). Several models of heart-rate monitors have the possibility of storing data and can be connected

to a computer for processing. This method makes it possible to measure intensity, duration and frequency. It also provides a good measurement of the total energy expenditure (19). Heart-rate monitors are frequently used at the individual level to find the individual's optimal exercise intensity based on current aerobic capacity.

Combination of methods

New instruments are constantly being developed to assess physical activity. The most modern, which are also more advanced and expensive than the aforementioned, combine several methods and technologies. ActiReg is an instrument that combines body position and movement separately or in combination with heart rate. ActiReg classifies the activity's energy expenditure in the categories easy, moderate or very strenuous. ActiHeart is another instrument that combines accelerometry and heart rate. In this method, the accelerometry weighs the heaviest at low intensity, while heart rate weighs heavier at high intensity. This way, the measurements are weighted to make the calculation of the completed physical activity more precise. New products combine accelerometry and GPS data (Global Positioning System) to also weigh in movements/distance in the calculations.

Determination of sedentary behaviour

A person who follows the health-enhancing recommendations, or the recommendations for strength and aerobic fitness, can also be sedentary for a significant part of the day. In other words, it is possible to periodically both be highly active and sedentary "at the same time" (20). Physical inactivity (can be defined as not fulfilling the recommendation) and being sedentary can thereby be viewed as two risk factors, which both need to be studied together and independent of one another.

To determine the degree of sedentary behaviour, several different types of questions have been used, such as about the time that children and young people spend in front of the TV or computer. These questions are misleading unless the total activity is also taken into account. Of the objective instruments, accelerometers and heart rate monitoring can provide an illustration of all so-called sedentary time, as well as active time. Step-counters, however, cannot say anything about time spent sitting still. Questionnaires like the IPAQ can also provide an illustration of this behaviour.

Assessment of aerobic fitness

Aerobic fitness can be evaluated with maximal or submaximal tests on ergometer cycles, stair machines or treadmills (21). Maximal tests should not be carried out on risk individuals other than under controlled forms, such as in a physiological laboratory. However, submaximal tests are very well suited to clinics and in prevention and promotion work. All submaximal tests build on the same principle, that is to say that there is a linear relationship between exercise intensity and pulse. With the help of the maximal heart rate, which can be calculated by decreasing the value 220 (for men, 225 for women) by the individual's age, and a set exertion, such as through a standardised resistance on a test cycle, the maximal oxygen uptake can be calculated. The most common submaximal method in Sweden is Åstrand's cycle test (22).

All submaximal aerobic fitness tests have a minimum of 10–15 per cent method error and can be used on the individual level before and after an intervention if the conditions are standardised. The systematic errors are largely due to the assumption made about the individual's maximal heart rate (220/225 – age only gives a rough estimate of the maximal heart rate), but also to full stroke volume not being achieved and handling error, such as the cycle not being calibrated or the pulse watch not being applied properly. It should also be mentioned that submaximal aerobic fitness tests have low reproducibility compared with maximal tests.

Another way of estimating the aerobic capacity is to use the RPC scale (Rating of Perceived Capacity) (23, 24). The scale should be seen as a complement to aerobic fitness tests and be used to provide a fast, approximate estimate of fitness in clinics where neither time nor equipment is available for aerobic fitness tests. The scale is based on various activities connected to metabolic equivalents (MET). The scale starts at 1 MET (which corresponds to the oxygen uptake at rest) and ends at 20 MET for men (which corresponds to an aerobic fitness value of 70 ml/kg/min) and 18 MET for women (which corresponds to an aerobic fitness value of 63 ml/kg/min) (figure 3). A qualified estimate of aerobic fitness is obtained by the individual assessing the most strenuous activity and corresponding MET value that the person in question believes he or she can maintain for 30 minutes. Based on the estimate, the aerobic fitness figure (maximal oxygen uptake in ml/kg/min) can then be calculated by multiplying the number of MET that the individual has given by 3.5. In addition, oxygen uptake is obtained in litres per minute by multiplying the aerobic fitness figure by the individual's body weight. To further increase the estimate's precision in research, for example, an age correction can be made. However, this is not necessary in clinical practice.

Estimation of perceived capacity – RPC (Rating of Perceived Capacity)**Can you do the following for half an hour or more**

1	Sit
2	
3	Walk slowly
4	
5	Walk at a normal pace/cycle slowly
6	
7	
8	Jog/cycle
9	
10	Run
11	
12	Run fast/cycle fast
13	
14	
15	Run very fast
16	
17	
18	Do aerobic exercise at an elite level (women)
19	
20	Do aerobic exercise at an elite level (men)

Figure 3. RPC scale.

There is strong evidence that performance capacity in the form of maximal oxygen uptake has a dose-response relationship with health and that oxygen uptake can rapidly improve in an untrained individual when he/she begins exercising. But determining performance capacity does not provide an illustration of whether the individual is active or not overall, since genes also control performance capacity. The individual can have a high capacity without being physically active, vis-a-vis a low capacity and be physically active. The higher the capacity, the more space there is to maintain a high level of energy expenditure. Therefore, it is also important to measure capacity (in the assessment and control of physical activity). If an intervention aims to increase the total energy expenditure, a method that can measure it must be used, however.

Assessment of strength

Strength can be assessed with both standardised methods for static or dynamic muscular endurance and strength such as with the so-called 1 RM method (one repetition maximum, as described below), which gives a measure of dynamic strength. The assessment can take place based on normal values, if they exist, but also with the help of the “healthy side”, the quality of the movement and the Borg rating (which is explained in detail under the heading “Assessment and control” further down in the chapter). Regardless of the test method, the experimenter must be aware that different factors affect the test result. Among these are a standardised test procedure as well as anatomical, neurological, psychological, mechanical and muscular factors.

Among the standardised methods are Sörensen’s test for static muscular endurance in back stretches, Svantesson’s test for dynamic muscular endurance in the calf muscles and UKK’s (Uhro K Kekkonen Institute of Sports Medicine) test battery for health-related fitness. The UKK tests include strength, endurance, balance, motor control, flexibility and aerobic fitness (25).

With the objective of optimising the load in strength training and to be able to evaluate if performed strength training has had the desired effect, dynamic strength can be measured by finding the load that the individual can only manage to lift once – 1 RM. To test the weight that corresponds to 1 RM, various approaches can be used. The most common procedure is to take a suitable weight and test how many times the individual can manage to lift it through the full range of motion at the same speed and under control (preferably not more than 10 times since the table is only reliable up to approximately 10–15 repetitions). Then, 1 RM is calculated based on figure 4. This approach is the safest from an injury perspective. Another alternative is to try to find the 1 RM weight, that is to say the weight the individual can lift once. However, this entails some risks of both overloading and improper execution. The measured strength then forms the basis to guide the strength training towards the desired objective, considering load, sets, repetitions and frequency.

Number of repetitions	Per cent of max.
1	100
3	90
5	85
7	80
10	70
≈ 15	60
≈ 25	50
≈ 50	30

Figure 4. Repetition maximum.

Assessment of body composition

An outcome of regular physical activity and good eating habits is that the body measurements return to the normal variation. Body measurements can thereby function as an indicator and an evaluation instrument in the prescription of physical activity. A few basic body measurements are presented below.

Height, weight and BMI

Correctly measuring the height of children and adults is seldom problematic. At adult age, height is also relatively stable, although aging entails some height reduction. Of course, the yardstick used to measure height should be checked and no measurement taken with shoes. In studies of the truthfulness of self-stated height in surveys and interviews, quite a few

errors have been shown to exist. For example, the height given by short men is often taller than the true height, and the elderly are often unaware of their reduced height.

The weight indicated or measured can also be encumbered by errors. Scales used should be calibrated and be of good quality. The person to be weighed should be sparsely clad. In self-stated weight, a number of problems also arise, where overweight people indicate a lower weight than their true weight, underweight people state a higher weight and so on. The differences between self-stated weight in surveys and in interviews and the true weight is larger for teenagers, those with little education and the overweight.

When the body measure of BMI (Body Mass Index) is to be calculated, that is to say the weight in kilograms divided by the square of the height in metres, a number of problems arise if data is based on self-reported height and weight. This means that BMI data from this type of study cannot at all be compared with BMI data based on measured height and weight. It is notable that BMI does not differentiate weight from muscle and from body fat. Many muscular elite athletes would therefore be classed as overweight if only BMI were used at the individual level.

For adults, there are well-defined limits for what is considered to be overweight and obese ($BMI \geq 25-29.9 =$ overweight, $BMI \geq 30 =$ obesity). For children, there are a few different limits set to define overweight and obesity at different ages where the most commonly used are Cole's cutoff points (26).

Waist circumference

The waist circumference is measured with the help of a tape measure in a standardised manner. It is measured after a relaxed exhale, approximately two centimetres above the navel, which is just under the lowest rib. The individual can learn how to measure and follow his or her own development. In addition, there are some recommended guidelines where women who have waist circumferences over 80 cm are at greater risk and over 88 cm much greater risk for cardiovascular disease. For men the limits are at waist circumferences of 94 and 102 cm, respectively. This applies to ethnic Caucasians. For other ethnic groups, such as persons from Asia, lower cutoff points apply. Hip measurement is also of major interest; in recent years, it has been shown that stout hips can function as a protection against hip fractures and have a correlation to less risk of cardiovascular disease among women (27). To calculate the waist/hip ratio, the waist measurement is taken as above and the seat measurement measured at the broadest point. The waist circumference divided by the hip circumference should not exceed 0.85 for women and 1.0 for men.

Body composition

To find out the distribution between fat and non-fat (which can be muscles, bones, fluid), more advanced methods can be used. Some of these are used at exercise facilities in consultations and for research purposes.

Skin-fold measurement is a relatively simple method where the experimenter measures subcutaneous fat at standardised locations with the help of a calliper. With the help of tables or formulae for age group and gender, an approximate fat percent can then be calculated for the particular individual (28).

Bioimpedance is a method based on muscles conducting an electric impulse better than fat due to the higher water content. The most valid bioimpedance equipment measures through the entire body, leg to arm. The most used methods, but with the worst reliability, are those that only measure arm to arm or leg to leg (28).

More sophisticated methods are found in research contexts and are often used to validate basic methods or to evaluate research projects. Among these are the water method, underwater weighing and air displacement technology as well as DXA (Dual Energy Xray Absorptiometry). These methods are often expensive and require costly equipment and training (28).

To keep in mind in an assessment of physical activity

The elderly

For the healthy elderly, the same principles for assessment and evaluation apply as described above. For example, in national living habit studies, the same question about exercise habits in leisure time is used for all adults ages 18 to 84 (29). To more specifically assess the degree of physical activity among the elderly, the method-tested Activity Scale is often used and is recommended (30).

Persons with obesity

Among overweight persons (BMI 25–29.9) and the obese (BMI 30–35), the instruments described above can be used. Those with severe obesity (BMI over 35) have difficulty moving at all and everything they do costs a great deal of energy since they carry a great deal of weight (31). Studies of energy expenditure among the severely obese have shown that some use up to 90 per cent of their maximal capacity when walking at a self-chosen speed (32). There can also be other obstacles in the form of joint problems and incontinence that affects the perceived exertion and degree of activity.

If objective instruments are used such as step-counters, they must be set in such a way that allows the registration of vertical movement. Otherwise, a risk exists for them becoming stuck in the “folds” and not being exposed to any vertical acceleration. Moreover, the obese often perceive it as unpleasant and warm to wear the activity monitor.

An outcome of physical activity and exercise can be that daily functions improve. This means that functional tests, such as standing up unassisted and being able to tie one’s shoes, can function as an indicator and evaluation instrument in the prescription of physical activity (33).

To keep in mind among persons on medication

Certain drugs, such as beta-2 stimulators, which are common for asthma, and beta blockers, which are common for cardiovascular problems, affect systems (such as heart rate) in the body, which in turn can affect the assessment of aerobic fitness and physical activity. For these individuals, movement sensors (step-counters and accelerometers) are recommended ahead of heart rate monitoring. In aerobic fitness tests, perceived exertion (34) should always be used in combination with heart rate.

Children

Children have an entirely different movement pattern than adults, and in one minute, can be active with a high level of intensity, have time to rest and then be just as active again. Nor do children think or remember physical activity in the way that adults do, which makes it nearly impossible to ask children how physically active they are. Only assessing how often children participate in some sport or physical education gives a restricted view of the total activity.

At a national level in Sweden, the WHO instrument HBSC (Health Behaviour in School Children) has been used to measure the health habits of children and young people (35). In the instrument, young people are asked if they have been physically active for at least an hour five times a week or more often. The responses do not give any information about which activity was done or how strenuous it was, but a pretty good view of dose and regularity. The question is method-tested by the WHO (36), although not specifically in Sweden. Other method-tests of more specific questions like IPAQ have shown that children and young people do not understand the concepts, do not perceive time in the same way as an adult and therefore have difficulty in answering them.

To avoid children's and young people's difficulty in remembering activities, which is largely due to the activity pattern being irregular and more play-oriented, objective assessment instruments are recommended such as step-counters or accelerometers.

Assessment and control of intensity

When prescribing physical activity, it is relatively unproblematic to give and take instructions regarding the physical activity's frequency (how often) and duration (how long). In terms of intensity, it is not as simple. As previously presented in the chapter, a number of methods have been developed to assess how intense work done is and, respectively, many recommendations with regard to how intense the physical activity should be to lead to health effects. For example, it is said that aerobic activity should be carried out at least at a *moderate level*, with an *intensity that gives rise to light breathlessness and sweating*, or be of *average intensity* or a level that *allows conversation*. If a physiologist is asked, intensity is often expressed in terms of a *percentage of the maximal oxygen uptake* (50–65 %) or in a *percentage of the maximal heart rate* (60–75 %) or the age-predicated maximal heart rate. For strength-oriented activities, the recommended intensity is often given in relation to the repetition maximum (1 RM) and in per cent, for example 80 per cent of 1 RM in strength training and 50 per cent of 1 RM in muscular endurance training.

These ways of describing intensity can in practice be difficult to explain (for the prescriber) and follow (for the patient). From a pedagogic perspective, a method that is easy to explain and easy to understand is therefore valuable. A method that has proven to work well both from a scientific research perspective and out in reality is the rating scales designed by the Swedish psychologist Gunnar Borg. In terms of ratings of perceived exertion, Borg's RPE scale (Ratings of Perceived Exertion) is common while strength estimates are preferably made with the help of the CR10 scale (Category Ratio). Both scales are based on verbal expressions that are rooted in a numerical scale between 6–20 (RPE

scale) or 0–10 (CR10 scale). The advantage of using rating scales is that the responses reflect a total appraisal of signals from many different parts of the body (37).

A number of different physiological reactions such as pulse, respiratory frequency, sweating and pain from joints and exerted muscles contribute to the total perceived exertion. Exactly how these physiological reactions co-vary and contribute to the perceived exertion is not known, but it can be assumed that some are more generally applicable (such as pulse) while others are more individually related (such as signals from joints and muscles). However, it is known that ratings on the RPE scale grow linearly towards the load increase both in cycle ergometer work and running on a treadmill, just like the heart rate and oxygen expenditure increases when the load increases. A correlation coefficient between 0.85 and 0.99 has also been reported with regard to load and perception increase, as well as subjective perceptions and heart rate or oxygen uptake (37).

The exertion rating is also affected by a number of factors such as age, training status and personality. Although growth against load remains linear from low to high intensity regardless of age, the absolute relationship between heart rate and RPE rating will change. The RPE scale's number variation between 6–20 corresponds to an approximate heart rate variation for a young person between 60 and 200 beats/minute (based on the maximal heart rate corresponding to approximately 220 minus the age for men and 225 minus the age for women). By the maximal heart rate decreasing with increasing age, the relationship to ratings on the RPE scale will change. While a rating of 15 roughly corresponds to a pulse of 150 beats/minute for a young person during cycle ergometer exertion, the same rating for a middle-aged person would correspond to around 130 beats/minute or 110 beats/minute for an elderly person. The advantage of ratings of the degree of exertion are consequently clear since the variation range is maintained to a significantly greater extent than is the case with the heart rate at an increased age.

At the same time, it is known that well-trained persons often underestimate their degree of exertion while untrained persons overestimate it (38). The individual's personality has also proven to affect the exertion ratings. For example, persons with distinct type A behaviour (which is considered to increase the risk of cardiovascular disease) have proven to underestimate their exertion compared with individuals with a lesser degree of this behavioural pattern (39).

Another personality trait that appears to affect the perception of exertion is the individual's locus of control¹, in that those with an internal locus of control show a more accurate rating behaviour compared with those with an external locus of control (40, 41). Within health-psychology research, it is well known that persons with an internal locus of control, who themselves consider that they can affect their health to a high degree, both follow

1. The individual's feeling that it is possible or impossible to influence and control his or her own performance. Athletes with a high level of internal control (internal locus of control) feel that successful performance is most often due to their own ability, such as good preparations and good training, in other words factors that can be influenced. People with a high level of external control (external locus of control) instead more often feel that good performance is due to happenstance, randomness or luck.

prescriptions better and regain health faster than those with an external locus of control (42). However, none of this reduces the RPE scale's reliability, but in the same way that a measured heart rate has to be related to the individual's degree of training and age, the exertion rating must be assessed in terms of likelihood and credibility. If the scale is used to compare the ratings for the same person at different test times (intra-individual comparisons), an effect from the individual's personality plays a smaller role than if comparisons are made between individuals (inter-individual comparisons). Naturally, this is also true of heart rate and oxygen consumption, since we assume that heart rate, oxygen uptake and personality are relatively constant over time (with a reservation for the unavoidable age change with regard to maximal heart rate and any exercise effects).

In the following section, both the RPE scale and the CR10 scale are described as well as how they can be used both for ratings in connection with physical exertion and to control intensity. The latter is particularly useful when physical activity is prescribed and the patient needs to know how intense the activity in question should be.

Borg RPE scale®

6	No exertion at all
7	Extremely light
8	
9	Very light
10	
11	Light
12	
13	Somewhat hard
14	
15	Hard
16	
17	Very hard
18	
19	Extremely hard
20	Maximal exertion

© Gunnar Borg, 1970, 1985, 1994, 1998, 2006

© 2000

Figure 5. Borg RPE scale®.

For the ratings to show a high degree of reliability, detailed instructions are required so that the patient rates his/her degree of exertion and nothing else. Oral instructions can be worded as follows (34):

While working, we want you to rate your feeling of exertion, how hard and strenuous it is and how tired you feel. The perception of exertion is mainly felt as fatigue in your muscles, and in your chest in the form of breathlessness or possible pain. All types and levels of physical activity require some exertion, even if only minimal. This also applies if one does light exercise, such as walking slowly.

Use this scale from 6, "No exertion at all", to 20, "Maximal exertion".

- 6 "No exertion at all" means that you do not feel any strain at all, for example, no muscle fatigue, no breathlessness or difficulty breathing.
- 9 "Very light". Like taking a short walk at your own pace.
- 13 "Somewhat hard". You can continue without great difficulty.
- 15 It is "strenuous" and laborious. You are tired, but can continue anyway.
- 17 "Very hard". A very strong strain. You can continue, but have to work very hard and you feel very tired.
- 19 An "extremely" high level. For most people, this corresponds to the highest level of exertion they ever felt.

Try to be as honest and spontaneous as possible and do not think about what the load actually is. Try not to underestimate or overestimate it. It is important that it is your own feeling of exertion and not what you believe others think. Look at the scale and base your rating on the words, but then choose a number. Use whichever numbers you want on the scale, not just those marking the expressions.

Any questions?

Central and local exertion

In some contexts, it can be valuable to differentiate central exertion (breathing, pulse) and local exertion (the working muscles). This can be the case if the individual suffers from cardiopulmonary problems when the central exertion is probably higher than the exertion in the entire body (total). If the difficulty is located in muscles and/or joints, a local rating may say more than an overall rating. The instructions above can then be modified so that the person is instructed to especially notice the exertion centrally or locally. When untrained, but healthy individuals work on a cycle ergometer, the exertion in the legs is often considerably higher than the central exertion. However, if the work is on a treadmill (walking, jogging, running), the central and local exertion track each other relatively well and it most often suffices to ask the person to rate their overall degree of exertion.

Borg CR10 scale®

In contrast to the RPE scale, which is specially designed for ratings of exertion, the CR10 scale is a general scale. The CR10 scale can be used in the majority of areas where it is of interest to make use of the individual's subjective perceptions. This can include ratings of aches and pain both locally in the legs as well as centrally, such as chest pain or breathlessness (dyspnoea). In healthcare, VAS (Visual Analogue Scale) is often used. Here, the CR10 scale is an alternative that has proven to be very reliable (43).

0	Nothing at all	
0.3		
0.5	Extremely weak	Just noticeable
0.7		
1	Very weak	
1.5		
2	Weak	Light
2.5		
3	Moderate	
4		
5	Strong	Heavy
6		
7	Very strong	
8		
9		
10	Extremely strong	“Maximal”
11		
↓		
●	Absolute maximum	Highest possible

Gunnar Borg CR10 scale
© Gunnar Borg, 1982, 1998

Figure 6. Borg CR10 scale®.

Correct instructions are also necessary in the use of the CR10 scale.

Give the person the scale to look at. With the help of this scale, you should say how strongly you feel the aches (the pain or any other current sensation). “Nothing at all” corresponds to 0 and means that you do not feel any aches at all (pain, etc.). “Extremely strong” (maximal) corresponds to a 10. For most people, this is the greatest pain (ache, etc.) they have ever experienced. A pain that is even stronger than what you have ever experienced before is conceivable, which is why the absolute maximum value (the highest possible) is a bit higher. If you feel that your experience is stronger than “Extremely strong” – that is stronger than you have ever expe-

rienced before – you can respond with a number somewhat higher than 10, such as 11.3 or 12.5 or higher. “Extremely weak”, which lies at 0.5 on the scale is something barely noticeable, in other words, the feeling is right on the edge of what is possible to feel. You use the scale in such a manner that you start by looking at the verbal expressions, then choose a number. If your perception (ache or the like) corresponds to “Very weak”, you give a 1. If it is “Moderate”, you give it a 3 and so on. You can use any numbers on the scale whatsoever, including half values, such as 1.5 or decimals such as 0.8 or 8.3. It is very important that you say what you feel and not what you think you should say. Rate as honestly and frankly as possible and try to not underestimate or overestimate it. Remember to base your rating on the verbal expressions in front of each rating. Then say a number.

Control of intensity

Both the RPE and the CR10 scales can be used to control physical activity carried out for rehabilitative purposes. After the patient has gotten to know the scale(s) and received proper instructions on how to rate exertion (RPE scale) or aches, pain or the like (CR10 scale), it is possible to prescribe appropriate intensity levels that can then be used in rehabilitation. The intensity levels must of course be based on the person’s situation, illness and conditions. In the respective chapters, recommended intensity levels are given, which is why we refer to these with regard to suitable levels for rehabilitation or preventative exercise activity.

If perception ratings are to control the intensity in rehabilitation, it is important to subject the patient to the activity in question under controlled circumstances. When the patient rates his/her exertion (pain or the factor in question) at the same time that pulse, blood pressure and other current physiological parameters are registered, it is possible to determine if the person tends to over or underestimate his/her perceived exertion/pain. An appropriate level of intensity can thereby be “calibrated” for the unique conditions of each individual so that the risk of over-exertion in association with rehabilitation is minimised.

Lastly, a word of warning. It has been shown that people perceive cycling, walking, jogging, running and so forth as less strenuous outdoors than indoors (in a laboratory). This means that the prescribed level of exertion must be adjusted downwards. If the person rates his or her exertion as a “15” (Hard) on the RPE scale in the lab and this is deemed to be an adequate level, the recommendation should be that the person not work harder than to a “13” (Somewhat hard). In fact, research has shown that the difference in perceived exertion is approximately two scale steps when the same type of exertion is done indoors and outdoors, respectively (37).

References

1. Ainsworth BE, Levy S. Assessment of health-enhancing physical activity. Methodological issues. In: Oja P, Borms J, ed. *Health enhancing physical activity*. Oxford: Meyer & Meyer Sport; 2004.
2. Welk G. *Physical assessment in health-related research*. Leeds (UK): Human Kinetics; 2002. p. 269.
3. Pate RR, Pratt M, Blair SN, Haskell WL, Macera CA, Bouchard C, et al. Physical activity and public health. A recommendation from the Centers for Disease Control and Prevention and the American College of Sports Medicine. *JAMA* 1995;273:402-7.
4. Haskell WI, Lee I-M, Pate RR, Powell KE, Blair SN, Franklin BA, et al. Physical activity and public health: Updated recommendation for adults from the American College of Sports Medicine and the American Heart Association. *Med Sci Sports Exerc* 2007;39:1,423-34.
5. Kriska A, Caspersen C. Introduction to a collection of physical activity questionnaires. *Med Sci Sports Exerc* 1997;Suppl:5-205.
6. Ainsworth BE, Haskell WL, Leon AS, Jacobs Jr DR, Montoye HJ, Sallis JF, et al. Compendium of physical activities. Classification of energy costs of human physical activities. *Med Sci Sports Exerc* 1993;25:71-80.
7. Ainsworth BE, Haskell WL, Whitt MC, Irwin ML, Swartz AM, Strath SJ, et al. Compendium of physical activities. An update of activity codes and MET intensities. *Med Sci Sports Exerc* 2000;32:498-504.
8. Bergman-Boden K, Johansson C. Hur väl överensstämmer självskattad och objektivt mätt hälsofrämjande fysisk aktivitet? En metodprovning av fysisk aktivitet på recept (FaR) enkäten. C-uppsats i sjukgymnastik. [How well do self-assessed and objectively measured health-enhancing physical activities agree? A method test of the prescribed physical activity (FaR) survey. Level C essay in physiotherapy.] Stockholm: Department of Neurobiology, Care Sciences and Society, Karolinska Institutet; 2007.
9. Klesges RC, Eck LH, Mellon MW, Fulliton W, Somes GW, Hanson CL. The accuracy of self-reports of physical activity. *Med Sci Sports Exerc* 1990;22:690-7.
10. Craig C, Marshall AL, Sjostrom M, Bauman AE, Booth ML, Ainsworth BE, et al. International physical activity questionnaire. 12-country reliability and validity. *Med Sci Sports Exerc* 2003;35:1381-95.
11. IPAQ, The International Physical Activity Questionnaire. www.ipaq.ki.se.
12. Ekelund U, Sepp H, Brage S, Becker W, Jakes R, Hennings M, et al. Criterion-related validity of the last 7-day, short form of the International Physical Activity Questionnaire in Swedish adults. *Public Health Nutr* 2006;9:258-65.
13. Hagstromer M, Oja P, Sjostrom M. The International Physical Activity Questionnaire (IPAQ). A study of concurrent and construct validity. *Public Health Nutr* 2006;9:755-62.
14. Bouchard C, Tremblay A, LeBlanc C, Lortie G, Sauard R, Theriault G. A method to assess energy expenditure in children and adults. *Am J Clin Nutr* 1983;37:461-7.

15. Westerstahl M. Physical activity and fitness among adolescents in Sweden with a 20-year trend perspective. Stockholm: Department of Laboratory Medicine, Division of Clinical Physiology, Karolinska Institute; 2003.
16. Schneider PL, Crouter SE, Bassett DR. Pedometer measures of free-living physical activity. Comparison of 13 models. *Med Sci Sports Exerc* 2004;36:331-5.
17. Freedson P, Melanson E, Sirard, J. Calibration of the Computer Science and Applications, Inc. accelerometer. *Med Sci Sports Exerc* 1998;30:777-81.
18. Ward DS, Evenson KR, Vaughn A, Rodgers AB, Troiano RP. Accelerometer use in physical activity. Best practices and research recommendations. *Med Sci Sports Exerc* 2005;37:S582-8.
19. Ceesay SM, Prentice AM, Day KC, Murgatroyd PR, Goldberg GR, Scott W, et al. The use of heart rate monitoring in the estimation of energy expenditure. A validation study using indirect whole-body calorimetry. *Br J Nutr* 1989;61:175-86.
20. Ekelund U, Brage S, Froberg K, Harro M, Anderssen SA, Sardinha LB, et al. TV viewing and physical activity are independently associated with metabolic risk in children. The European Youth Heart Study. *PLoS Med* 2006;3:e488.
21. American College of Sports Medicine. Physical fitness testing. In: ACSM's Guidelines for Exercise Testing and Prescription. London: Williams & Wilkins; 2005.
22. Andersson G, Forsberg A, Malmgren S. Konditionstest på cykel. Testledarutbildning. [Ergometer bicycle testing. Test manager training.] Farsta: SISU Idrottsböcker; 2005.
23. Wisen AG, Farazdaghi RG, Wohlfart B. A novel rating scale to predict maximal exercise capacity. *Eur J Appl Physiol* 2002;87:350-7.
24. Wisen AG, Wohlfart B. Aerobic and functional capacity in a group of healthy women. Reference values and repeatability. *Clin Physiol Funct Imaging* 2004;24:341-51.
25. UKK-Institute. UKK fitness test batteri. <http://www.ukkinstituutti.fi/en/ukk-tests/295>.
26. Cole TJ, Bellizzi MC, Flegal KM, Dietz WH. Establishing a standard definition for child overweight and obesity worldwide. International survey. *BMJ* 2000;320:1240-3.
27. Heitmann BL, Frederiksen P, Lissner L. Hip circumference and cardiovascular morbidity and mortality in men and women. *Obes Res* 2004;12:482-7.
28. Norton K, Olds T. Anthropometrica. A textbook of body measurement for sports and health courses. Marrickville (NSW): Southwood Press, UNSW PRESS; 1996.
29. Swedish National Board of Health and Welfare. Health in Sweden. Sweden's Public Health Report 2005. Stockholm: Swedish National Board of Health and Welfare; 2005.
30. Grimby G. Physical activity and muscle training in the elderly. *Acta Med Scand* 1986;S711:233-7.
31. Ekelund U, Aman J, Yngve A, Renman C, Westerterp K, Sjöström M. Physical activity but not energy expenditure is reduced in obese adolescents. A case-control study. *Am J Clin Nutr* 2002;76:935-41.
32. Mattsson E, Larsson UE, Rössner S. Is walking for exercise too exhausting for obese women? *Int J Obes Relat Metab Disord* 1997;21:380-6.

33. Evers Larsson U, Mattsson E. Functional limitations linked to high body mass index, age and current pain in obese women. *Int J Obes Relat Metab Disord* 2001;25:893-9.
34. Borg G. Borg's perceived exertion and pain scales. Champaign (IL): Human Kinetics; 1998.
35. HBSC. Skolbarns hälsovanor. [The health habits of school children.] http://www.fhi.se/templates/Page____649.aspx.
36. Booth ML, Okely AD, Chey T, Bauman A. The reliability and validity of the physical activity questions in the WHO health behaviour in schoolchildren (HBSC) survey. A population study. *Br J Sports Med* 2001;35:263-7.
37. Hassmén P. Perceived exertion. Applications in sports and exercise. Stockholm: Department of Psychology, Stockholm University; 1991, pp. 1-42.
38. Hassmén P. Perceptual and physiological responses to cycling and running in groups of trained and untrained subjects. *Eur J Appl Phys Occup Phys* 1990;60:445-51.
39. Hassmén P, Ståhl R, Borg G. Psychophysiological responses to exercise in Type A/B men. *Psychosomatic Medicine* 1993;55:178-84.
40. Hassmén P, Koivula N. Ratings of perceived exertion by women with internal or external locus of control. *J General Psych* 1996;123:297-307.
41. Koivula N, Hassmén P. Central, local, and overall ratings of perceived exertion during cycling and running by women with an external or internal locus of control. *J General Psych* 1998;125:17-29.
42. Rydén O, Stenström U. Hälsopsykologi. Psykologiska aspekter på hälsa och sjukdom. (Psychological aspects of health and disease.] 2. edn. Stockholm: Bonnier Utbildning AB; 2000.
43. Neely G, Ljunggren G, Sylvén C, Borg G. Comparison between the visual analogue scale (VAS) and the category ration scale (CR-10) for the evaluation of leg exertion. *Int J Sports Med* 1992;13:133-6.

7. Various types of physical activity and exercise

Authors

Monica Klungland Torstveit, PhD, Assistant Professor, Agder University, Faculty of Sport and Health, Kristiansand, Norway

Kari Bø, PT, PhD, Professor, Norwegian School of Sports Sciences, Department of Sports Medicine, Oslo, Norway

Types of activity

There are a number of activities that can contribute to improving the individual's physical capacity. The emphasis of this chapter is on the most common types of activities offered at various fitness centres, by sports associations, private actors or that can be done on one's own. The activities are presented in alphabetical order.

Aerobics

Aerobics is group exercise with step combinations, running and jumping. This form of exercise can also include strength and flexibility exercises, but this is most often not the case. Mainly, coordination and aerobic fitness are trained comprehensively. Aerobics is sometimes divided into low impact, that is to say without running and jumping, and high impact, which includes these components. A combination of low and high impact is most common. Several different types exist, such as step-aerobics, which is group training to music where one climbs up and down on a step-up board during the session. This type of aerobics involves aerobic and coordination training, that also provides a strengthening effect on the leg and gluteal muscles.

Aquarobics

In aquarobics, the water acts as resistance and one can regulate the resistance oneself by decreasing or increasing the intensity. Aquarobics is a good alternative for those who are untrained or overweight since the risk of injury is minimal. Both aerobic fitness and strength are trained in a low-impact manner.

Aqua running

Running in a pool with a vest or belt for greater lift and an optimal position in the water is a low-impact form of training. Aqua running is excellent for rehabilitation after injury, as well as an effective form of condition training at all levels. Greater frequency and stride provide an increased load on both muscles and the oxygen-transporting organs.

Ball, netball and racquet sports

There are a number of ball/netball games and racquet sports that can affect physical fitness in various ways. Most have a positive effect on aerobic fitness, muscle strength and coordination. Activity examples include football (soccer), handball, basketball, volleyball, floorball, tennis, squash, badminton and table tennis.

Callisthenics

The Nordic countries have a strong tradition with regard to various forms of group callisthenics. These have often been organised by callisthenics associations and athletic associations, as well as various private organisers. Men and women most often participate in separate training groups with different emphases. Nordic callisthenics were not originally focused on aerobic fitness and consequently differ from aerobics. The main emphasis was dynamic flexibility training, stature, balance and strength training as well as rhythm and aesthetic movements.

With a background in the blossoming fitness and aerobics wave in the U.S. in the 1970s and 1980s, new callisthenic models were developed in Scandinavia with a focus on the training of aerobic fitness. Friskis&Svettis was developed in Sweden (1) and Gymnastikk i tiden in Norway (2). Both models are based on the training of all components of physical fitness, such as aerobic fitness, muscle strength, flexibility and relaxation during an exercise session of approximately 60 minutes. In Gymnastikk i tiden, continuous aerobic training is used in accordance with the U.S. aerobics model. Friskis&Svettis uses the interval training principle with alternating aerobic, strength and flexibility exercises. Both types of exercise use music and step combinations inspired by various forms of dance and exercise.

Cross-country skiing

Cross-country skiing is an activity that requires a high endurance capacity, muscle strength and balance. Cross-country skiing involves all of the major muscle groups in the body and at the same time also involves many small muscle groups in a gentle manner. Cross-country skiing requires snow, while roller skis or roller blades can be used on snowless ground. The latter two have the same movement patterns as cross-country skiing and have the same physical requirements. All three forms are technically demanding and the practitioner needs some time to learn the right technique and movement pattern.

Cycling

Cycling primarily involves all major muscle groups of the legs and has a good aerobic fitness-enhancing effect. Compared with running, cycling is significantly milder on joints and muscles, which makes it a suitable conditioning activity for the untrained to begin with. In cycling, one does not need to bear one's own weight, which can make it easier for overweight persons. By cycling with higher resistance, one can also achieve good muscular training mainly of the thighs and calves. Cycling can be done outdoors on a regular bicycle or indoors on an exercise bike. Outdoor cycling requires good balance.

Spinning is indoor-cycling to music on exercise bikes in a group session. The instructor provides encouragement, but the individual determines how hard he or she wants to exercise by regulating the resistance on the wheel. Spinning can be effective aerobic training that also provides a strength-training effect, primarily on the legs and the gluteus muscles, but does not demand a great deal of coordination. Spinning can therefore be an alternative for those who do not like difficult movements, and the training is also non-weight bearing.

Dance

Dance is a type of activity that places high demands on several components of physical fitness, such as coordination, balance, flexibility, aerobic fitness and muscle strength. Dance is most often done to music or some other form of rhythm accompaniment. There are a number of different forms of dance, such as boogie-woogie, disco/freestyle, hiphop/break, rock'n'roll, salsa, sport dance, street jazz/funk, capoeira, cheerleading, country and line dance, drill dance, flamenco, folk dance, old-time dance and ballroom dance.

Nordic walking

Compared with regular walking, Nordic walking involves more muscles in the body because the upper body is used more actively. This way, the heart rate, oxygen consumption and energy expenditure can be increased, which provides a good effect both on aerobic and muscular fitness. In addition, the hip, knee and ankle joints are unloaded to some degree when walking in hilly terrain if poles are used. Use the poles rhythmically, and walk with a skiing stride (right foot-left arm, left foot-right arm). Choose a pole length that provides a good hold and a pleasant pendulum movement. To obtain the right pole length, the body height can be multiplied by 0.7. Preferably choose poles of an adjustable length.

Pilates

Pilates is a type of activity that consists of a system of exercises developed by Joseph Pilates in the early 20th century. In pilates, muscle strength and flexibility are combined with a focus on concentration, balance, breathing and relaxation. Pilates training has the objective of building up strength in the entire body with an emphasis on the abdominal and the back muscles. The aim is to achieve bodily control by working with calm and precision. Great

importance is placed on active breathing and the execution of the movements. Pilates can be done as group training on mats with various types of preps, such as balls, rings and weights, or individually with or without special equipment.

Qigong

Qigong is an old Chinese form of therapeutics that is practiced by millions of Chinese for health-prevention purposes in accordance with the philosophy of traditional Chinese medicine. Qigong combines soft and slow movements with relaxation, concentration and breathing exercises to strengthen and balance the entire body. Qigong is a tool for relaxation and to reduce stress.

Rowing/canoeing

Rowing and canoeing provide a good exercise effect, especially in terms of aerobic fitness, but also in terms of muscle strength. Both types of activity involve most major muscle groups in the entire body. However, rowing also involves the leg muscles to a greater extent than canoeing, and can also be practiced indoors on a rowing machine.

Running

Running is an activity that places great demands on aerobic fitness due to the involvement of major muscle groups. Running can be done year-round both indoors and outdoors, which makes the activity easily accessible. The disadvantage of running is that it impacts the muscles, ligaments and joints relatively hard, which can lead to overload and attrition injuries. Those beginning to run should remember to proceed cautiously, in other words begin with shorter distances and gradually increase the distance of the exercise route and the number of exercise sessions per week.

Step-machine/cross-training

Training on a step-machine is based on the same movement pattern as walking up stairs, while cross-training creates a movement pattern that is a mixture between running, cycling and cross-country skiing. Cross-training involves a softer movement pattern without the high impact that regular running provides. The strength training effect is somewhat less than in cycling, but with the active addition of the muscles in the upper part of the body. In step-up and cross-training, it is primarily aerobic fitness that is improved. The continuous, soft movements provide a moderate muscle load and low impact on, for example, joints. Thus, this form of exercise can be valuable in rehabilitative training after injury.

Strength training in a group session

Strength training can be practiced individually or in a group, with or without equipment. Fitness centres often offer group training with weight bars, barbells or special rubber-bands to music. The participants each work with their own equipment and if one wants to increase the intensity, additional weights or heavier barbells/rubber bands are added. This is a comprehensive form of strength training where large parts of the body are activated with an emphasis on muscular endurance and a unique alternative for those who want to become thoroughly trained without building large muscles. Everyone works according to their own ability. However, correct lifting technique is important.

Swimming

Swimming can be done both indoors in a pool or outdoors. The most common disciplines are the breast stroke, back stroke and crawl. Swimming is a non-weight bearing activity and a good alternative for overweight people, in part because there is little risk of injury.

Yoga

Yoga means union, and is an old mental and physical form of activity with roots in Asian culture. The objective of yoga is to find inner peace. There are several branches in yoga and the one that has become most renown in the Western World is the physical yoga that is based on body positions (*asanas*), breathing (*pranayama*) and meditation (*dhyana*). By using physical exercises/positions, breathing techniques, deep relaxation and meditation, one tries to strengthen the body and mind in a balanced and natural way. An exercise session consists of a number of different exercises. Some exercises are easy and others a little more difficult, but the exercises can always be individualised. The exercises are combined with breathing, by inhaling and exhaling through the nose. The desired effect of yoga is less stress and tension physically, mentally and emotionally, as well as greater flexibility and strength.

Walking

Walking is often viewed as the simplest and most natural form of exercise. Walking is mild on muscles and joints, and at the same time an activity that most people can do and requires a minimum of equipment – only a pair of good shoes. When walking, large muscle groups, mainly in the legs, are involved. For the untrained, it can be an advantage to begin with a brisk walk on a flat surface, while the slightly more trained should choose more hilly terrain. Walking on a steep incline involves the gluteal, thigh and calf muscles to a greater extent, which places considerable demand on the oxygen transporting organs. For those who cannot run, but still want to have real aerobic training, walking on a treadmill with an incline is an alternative. Also see the section on Nordic walking.

References

1. Johannesson I, Holmsäter J, Johansson A, Gustafsson L. Friskis & Svettis motionsbok. [Friskis & Svettis exercise book.] Stockholm: Prisma; 1984.
2. Bø K, Kamhaug EL. Gymnastikk i tiden. [Callisthenics in time.] Oslo: Universitetsforlaget; 1989.

8. Health aspects of strength training

Authors

Eva Jansson, MD, PhD, Professor, Department of Laboratory Medicine, Division of Clinical Physiology, Karolinska Institutet, Stockholm, Karolinska University Hospital, Stockholm, Sweden

Ulrik Wisløff, MD, PhD, Professor, Department of Circulation and Medical Imaging, Norwegian University of Science and Technology, Trondheim, Norway

Dorthe Stensvold, MSc, Department of Circulation and Medical Imaging, Norwegian University of Science and Technology, Trondheim, Norway

Summary

The traditional view of strength training is that it provides greater strength and endurance, and is primarily used as a tool in rehabilitation of musculoskeletal injuries. In more recent years, however, interest has grown in the health-enhancing effects of strength training. The diseases where strength training's possible preventive and soothing effects are discussed include diabetes, obesity, metabolic syndrome (increased lipoproteins, blood sugar, blood pressure and waist measurement), cardiovascular disease, osteoporosis, joint and back pain as well as anxiety and depression. It has also been shown that there is a relationship between muscle strength and increased risk of premature death (1).

Strength training for the elderly has received particular attention (2). Diminished muscle function can significantly limit daily activity and increase the risk for falls and fractures among the elderly. Strength training can therefore be the type of training that must precede other exercise, such as walking, to make other physical activity possible at all. Two decades' loss of strength and muscle mass in elderly can, however, be regained within two months with strength training (3).

Recommendations

In 1990, the American College of Sports Medicine (ACSM) published one of the first general recommendations to the population regarding strength training as a part of a comprehensive exercise programme comprising aerobic fitness, strength and flexibility. This type of recommendation (guidelines/position stands) is based on scientific documentation. In association with the ACSM document, strength training achieved acceptance

and the recommendation was followed by several similar documents from other health organisations.

The recommendations from ACSM and from several other organisations such as the American Heart Association, the US Department of Health and Human Services and the American Diabetes Association (2, 4–9) prescribe that strength training should be carried out at least twice a week. An exercise session comprises 8–10 different exercises for various muscle groups. Every exercise is done at least once (one set) with a load of 8–12 RM (table 1). Individuals with a chronic disease are recommended a load of 10–15 RM, in other words somewhat lighter weights with more repetitions. The weight that one can lift only once is called the repetition maximum (1 RM).

Table 1. Recommendations for strength training (indicates minimum levels) (2, 4–9).

	Set/RM*	Number of exercises	Frequency (times/week)
Health adults (beginners/inactive)	1/8–12	8–10	2
Elderly	1/10–15	8–10	2
Cardiovascular/ diabetic patients	1/10–15	8–10	2

At least one set of each exercise should be done.

* RM = Repetition Maximum. 1 RM corresponds to the highest load that can be lifted through the entire range of motion once.

The recommendations are directed at individuals with no or very little experience of strength training. The strength gain for exercise programmes with more sets compared with one set is only marginal once training begins (7, 8, 10), but the training rapidly reaches a plateau where more sets are required to increase strength capacity further. The reason for initially only recommending one set is that it is more likely that more individuals will do the strength training programme if it is less time consuming. These factors combined form the basis of the recommendation – at least one set. A similar reasoning lies behind the recommendation; at least twice a week instead of at least three times a week.

For healthy adults with prior experience of strength training, ACSM has recently issued scientific documentation regarding more advanced strength training (6, 11), where a more varied load profile is recommended in the area of 1–12 RM and up to 4–5 exercise sessions per week.

Is strength training healthy?

Both strength (table 2) and aerobic exercise can lead to a significant improvement in health, and the two variants provide some similar and some dissimilar exercise responses over time. An important aspect is that a certain degree of muscle strength is necessary to be able to be physically active. For persons who have been physically inactive and bedridden due to illness, and who have thereby lost both strength and aerobic fitness, it is especially important to train for strength to manage the strain they meet in daily life.

1. *Muscle strength, muscle mass*

A few months of strength training often provides very large changes in measured muscle strength, ranging from 20–30 per cent up to several hundred per cent (12) depending on the type of exercise and evaluation methods as well as on the initial degree of fitness. Most studies indicate an increase in the muscle fibres' cross-sectional area in the magnitude of 10–60 per cent, usually around 20 per cent. Measurements of the entire muscle group's cross-sectional area with magnetic resonance imaging (MRI) or computerised tomography (CT) most often indicate an increase of around 10 per cent (13), which can depend on the extra-cellular space decreasing with exercise and consequently, the actual increase in muscle mass being underestimated with MRI and CT. It is particularly important to remember with regard to strength training that the capacity to increase strength and grow muscle is retained over the years, and muscle growth and strength improvements have even been described among 90–100 year-olds (14, 15).

Besides strength training's positive effects on muscle mass, the research of recent years has shown that food and protein intake in connection with exercise (before and especially after) are important to optimise muscle growth (16).

2. *Maximal oxygen uptake and endurance*

The majority of studies show that strength training does not appreciably increase maximal oxygen uptake. In spite of this, strength training can increase aerobic endurance both on an ergometer cycle and treadmill (17, 18). It has also been found that strength training decreases cardiovascular stress in the form of reduced heart rate and blood pressure when walking with weights (19). If this can be attributed to the adaptation that occurs as a result of the actual strength training, or if it is a result of one being in a condition to be more physically active in daily life due to greater strength capacity and thereby obtaining better aerobic fitness has not been established. Regardless of the cause, this is an important cardiovascular result of strength training on an individual level.

3. *Metabolism and body composition*

Strength training can be a significant aid for the control of body weight, body composition and energy expenditure. A prerequisite for reduced body weight and fat mass is that the daily energy expenditure increases in relation to the daily energy intake. The daily energy expenditure's two most important components are the basal metabolic rate (BMR) and energy expenditure in connection with physical activity, both spontaneous daily activity and structured physical training. The increase in the energy expenditure in direct connection with strength training is moderate. In relative numbers, the load during a session is approximately 20–50 per cent of maximal oxygen uptake (20), corresponding to 100–200 kcal (rough estimate) for a 30–40 minute session, which is approximately the same as on a walk.

The most important determinant for BMR is the body's fat-free mass, of which 60–75 per cent is muscle. Among the inactive, BMR comprises the largest component (60–75%) of the daily energy expenditure. Strength training studies show that BMR can

increase by approximately 5 per cent or 100 kcal per day (3, 21) during the course of an exercise period of 3–5 months. Hunter and colleagues showed that the fat-free mass increased by 2 kg and BMR by 90 kcal per day among 70-year-old women and men after 26 weeks of strength training (45 minutes training 3 times per week) (3). A BMR increase of 90 kcal per day entails increased burning of fat by 15g per day at rest (burning 1g of fat releases 6–7 kcal), compared with expenditure before the period of training. This involves a major change over one year and illustrates the importance of just a moderate increase in BMR after a period of exercise.

The reason for this increase is believed to depend on a combination of greater muscle mass in itself (1 kg muscle has a basal metabolic rate of 10 kcal per day), greater protein synthesis and breakdown (22) and greater sympathoadrenergic activation (21). The fact that the muscle mass in itself does not appear to be able to explain the entire increase is supported by studies that show that BMR can be increased for up to 48 hours after an individual exercise session (23), that is to say BMR can increase without the muscle mass changing. However, it should be pointed out that the studies that show that BMR increases in connection with strength training, the training has been relatively comprehensive and intense (at least 3 sets per exercise session, 3 times per week). A recently published strength training study of young women (2 sets per exercise session, 3 times per week) showed no increase in BMR (25).

The most interesting finding in Hunter's study (3) was, however, that the average total daily energy expenditure increased by 240 kcal, in other words by approximately 10 per cent. This probably means that the physical activity outside the training programme increased, since the total of the BMR increase (90 kcal per day) and the energy expenditure in connection with strength training (60 kcal per day) amounted to 150 kcal per day. Another study showed a similar find among young men (24). Theoretically, one month's strength training could consequently reduce fat mass by 1 kg if the energy intake is kept constant. However, it is not self-evident that physical training increases daily energy expenditure. In fact, another study showed that daily energy expenditure was unchanged in connection with an intensive aerobic exercise programme among elderly men and women, which is likely due to spontaneous physical activity outside the exercise programme decreasing (26).

4. Insulin sensitivity

Strength training can entail improved insulin sensitivity (17, 27–30) and in some cases improved glucose tolerance and glycemic control as well (17, 29–33). The effects of strength training on glucose metabolism can probably be explained by its effects on body weight, body composition and metabolism. Quality changes in the muscles probably also contribute. For example, strength training leads to a higher proportion of type IIA fibres at the expense of type IIB fibres, in other words a change towards higher oxidative capacity and slower contraction speed (20).

5. Lipoproteins

Studies have shown that there is a connection between muscle strength and an improved lipid profile (33, 34). It has also been shown that individual strength training sessions can lead to an increase in HDL (High Density Lipoprotein, the good cholesterol) among young, untrained men (35).

6. Blood pressure

Multiple studies show that strength training can lower blood pressure (17, 29, 37, 38). A meta-analysis from 2007 shows that strength training reduces diastolic blood pressure, but not systolic blood pressure, among hypertensive persons, in other words those with high blood pressure (39). However, the results are not unambiguous. Therefore, the ACSM discourages individuals with manifest increased blood pressure from only doing strength training (40). Aerobic training is primarily recommended for blood-pressure treatment purposes or a comprehensive programme that includes both aerobic training and strength training.

7. Bone density, risk of falling, balance and flexibility

A large number of studies show that strength training increases bone density or reduces the age-related decrease and that the effect is relatively specific to the muscles and parts of the skeleton to which muscles attach (41–43). Significantly more studies are done on women, due to osteoporosis (brittle bones) being much more commonly occurring particularly among elderly women. The risk of fractures of the neck of the femur doubles every five years after the age of 50 and one out of three women aged 80 break the neck of the femur (17). The increases in bone density observed after both strength training and, for example, aerobic training are, however, most often less than 5 per cent, and it is claimed that the increase in bone density should be greater to prevent fractures from falling (17). On the other hand, an important effect of strength training might be to prevent falling accidents. However, there is limited evidence that falling accidents become less common after strength training, but it has been shown that risk factors for falls, such as muscle strength, walking ability and balance, are affected in a positive direction (17, 41).

There is no clear-cut evidence that strength training increases flexibility, but rather it can decrease with strength training. A comprehensive programme is therefore recommended to include flexibility training as well as aerobic training and strength training (4).

8. Joint pain/back pain

The wear and breakdown of cartilage in the knees leads to pronounced pain and disability. Strength training has been shown to decrease pain and improve function (17, 44–46). After cardiovascular disease, chronic pain in the lumbar region is one of our largest health problems. It has been shown that a specific training programme for the lumbar region, comprising only one set of 8–12 repetitions once per week, can provide reduced pain and increased strength and flexibility (47). In this context, it should, however, be mentioned

that many other types of strength training, as well as other types of exercise, are effective for the treatment of chronic lower back pain (48). Strength training was applied as early as the 19th century and early 20th century for rehabilitative purposes, but then “fell into oblivion” for nearly 100 years, a period during which treatment methods such as ultrasound, electric stimulation and massage have been dominant (47).

9. Mental health

Both aerobic training and strength training can alleviate the symptoms of depression and anxiety. Ten weeks of strength training among depressed elderly persons proved to significantly reduce all measures of depression (49). However, it has not been shown that training can prevent the onset of these symptoms (50). Interesting finds include that the length of an individual exercise session appears to be of significance to the effect on the state of mind. According to these studies, the training session should exceed 20 minutes and more optimally amount to 30–40 minutes (50).

Table 2. Effects of strength training.

1.	Muscle strength	↑↑↑
	Muscle mass	↑↑
2.	Maximal oxygen uptake	↔ ↑
	Endurance	↑
3.	Basal metabolic rate	↑
	Fat mass	↓
4.	Insulin sensitivity	↑
5.	Blood pressure	↓ ↔
6.	Lipoproteins	↓ ↔
7.	Bone density	↑ ↔
	Fall risk	↓
	Balance	↑
	Flexibility	↔ ↓
8.	Joint pain	↓
	Back pain	↓
9.	Mental health	↑

Modified from Hurley and Roth (17) and Pollock and Evans (12).

↑↑↑ = very large increase, ↑↑ = large increase, ↑ = increase, ↓ = decrease, ↔ = small or no change or varying findings.

Is strength training dangerous?

If strength training is done as per the recommendations, the collective assessment is that strength training is at least as safe as aerobic training if not safer (9, 51–53), but like other physical training, there is some risk, although very little, of cardiovascular and musculoskeletal complications in strength training.

In a study of elderly men and women, only two minor musculoskeletal injuries were found per 1,000 hours of training, and in most cases, training could be resumed after a period of rest (54).

Very few cardiovascular complications have been reported in connection with strength training among both the young and the old, including individuals with cardiovascular disease, for example. Among 57 heart attack patients, who trained both strength and aerobic fitness for 12 weeks, only one patient had an uncomplicated imbalance of the heart rhythm during a strength training session, while a total of 45 patients had chest pain or an ECG change indicating a lack of oxygen in the heart or a rhythm disturbance during the training or tests of aerobic fitness (53).

The fear of strength training that sometimes occurs is based in the hemodynamic response that has been shown in some forms of strength training. For example, studies show that young men who carry out repeated concentric and eccentric contractions with both legs to maximum fatigue with a load of 90 per cent of 1 RM can reach blood pressures in the magnitude of 300–400 mm Hg (38, 55). Research subjects in these studies were permitted to hold their breath (so-called Valsalva's manoeuvre) in connection with the lifts. However, later studies show that the hemodynamic response is more moderate, approximately as in aerobic exercise, if abdominal pressure is avoided (56–59). To reduce the risk of sharp increases in blood pressure, regular breathing is recommended in connection with strength training and to exhale when the exertion is at its greatest (the lift phase and/or contraction phase) and to inhale when the exertion is less (return and/or the extension phase) (9, 52). For patients with increased risk of cardiovascular complications, such as early after a heart attack, it is also recommended to not exceed 15–16 (hard) on Borg's RPE scale (60).

Another discussed risk is that strength training, through major blood pressure increases, could lead to heart enlargement of a concentric type, but most studies indicate that this concern is exaggerated. Among body builders who abuse anabolic steroids, however, heart enlargement and degraded diastolic heart function have been found (61).

Contraindications to strength training

Absolute:

- Unstable coronary-artery disease
- Uncompensated congestive heart failure
- Uncontrolled cardiac arrhythmia
- Pulmonary hypertension (severe, > 55 mm Hg)
- Aortic stenosis (serious)
- Acute myocarditis, endocarditis or pericarditis
- Uncontrolled high blood pressure (> 180/110 mm Hg)
- Aorta dissection
- Marfan syndrome
- High intensity strength training (80–100% of 1 RM) among people with serious kidney failure due to diabetes (diabetes nephropathy)
- Diabetes retinopathy

Relative contraindications (consult physician):

- High risk of cardiovascular disease
- Uncontrolled diabetes
- Uncontrolled high blood pressure (> 160/> 110 mm Hg)
- Severely impaired aerobic fitness (< 4 MET)
- Limitations in muscles or skeleton
- Those with a pacemaker or defibrillator

Williams et al (8).

References

1. Katzmarzyk PT, Craig CL. Musculoskeletal fitness and risk of mortality. *Med Sci Sports Exerc* 2002;4:740-4.
2. Nelson ME, Rejeski WJ, Blair SN, Duncan PW, King AC, Macera CA, et al. Physical activity and public health in older adults. Recommendation from the American College of Sports Medicine and the American Heart Association. *Med Sci Sports Med* 2007;39:1435-45.
3. Hunter GR, Wetzstein CJ, Fields DA, Brown A, Bamman MM. Resistance training increases total energy expenditure and free-living physical activity in older adults. *J Appl Physiol* 2000;89:977-84.
4. American College of Sports Medicine. The recommended quantity and quality of exercise for developing and maintaining cardiorespiratory and muscular fitness and flexibility in healthy adults. *Med Sci Sports Exerc* 1998;30:975-91.
5. U.S. Department of Health and Human Services, Physical Activity and Health. A Report of the Surgeon General. Atlanta (GA): U.S. Department of Health and Human Services, Centers for Disease Control and Prevention, National Center for Chronic Disease Prevention and Health Promotion; 1996, pp. 22-9.
6. American College of Sports Medicine. Progression models in resistance training for healthy adults. *Med Sci Sports Exerc* 2009;41:687-708.
7. Haskell WL, Lee IM, Pate RR, Powell KE, Blair SN, Franklin BA, et al. Physical activity and public health. Updated recommendation for adults from the American College of Sports Medicine and the American Heart Association. *Med Sci Sports Exerc* 2007;39:1423-34.
8. Williams MA, Haskell WL, Ades PA, Amsterdam EA, Bittner V, Franklin BA, et al. Resistance exercise in individuals with and without cardiovascular disease. 2007 update. A scientific statement from the American Heart Association Council on Clinical Cardiology and Council on Nutrition, Physical Activity, and Metabolism. *Circulation* 2007;116:572-84.
9. Sigal RJ, Kenny GP, Wasserman DH, Castaneda-Sceppa C, White RD. Physical activity/exercise and type 2 diabetes. *Diabetes Care* 2006;29:1433-8.
10. Hass CJ, Garzarella L, de Hoyos D, Pollock ML. Single versus multiple sets in long-term recreational weightlifters. *Med Sci Sports Exerc* 2000;32:235-42.
11. Hoff, J. Helgerud, J. and Wisløff, U. Endurance training into the next millenium; muscular strength training effects on aerobic endurance performance. Invited review. *Am J Med Sports* 4: 58-67, 2002).
12. Pollock ML, Evans WJ. Resistance training for health and disease. Introduction. *Med Sci Sports Exerc* 1999;31:10-1.
13. Porter MM. The effects of strength training on sarcopenia. *Can J Appl Physiol* 2001;26:123-41.

14. Fiatarone Singh MA, Ding W, Manfredi TJ, Solares GS, O'Neill EF, Clements KM, et al. Insulin-like growth factor I in skeletal muscle after weight-lifting exercise in frail elders. *Am J Physiol* 1999;277:E135-43.
15. Kryger AI, Andersen JL. Resistance training in the oldest old. Consequences for muscle strength, fiber types, fiber size, and MHC isoforms. *Scand J Med Sci Sports* 2007; 17:422-30.
16. Koopman R, Saris WHM, Wagenmakers AJM, van Loon LJC. Nutritional interventions to promote post-exercise muscle protein synthesis. *Sports Med* 2007;37:895-906.
17. Hurley BF, Roth SM. Strength training in the elderly. Effects on risk factors for age-related diseases. *Sports Med* 2000;30:249-68.
18. McCartney N, McKelvie RS, Haslam DRS, Jones NL. Usefulness of weight-lifting training in improving strength and maximal power output in coronary artery disease. *Am J Cardiol* 1991;67:939-45.
19. Parker N, Hunter G, Treuth M. Effects of strength training on cardiovascular responses during a submaximal walk on a weight-loaded walking test in older females. *J Card Rehab* 1996;16:56-62.
20. Tesch PA. Short- and long-term histochemical and biochemical adaptations in muscle. In: Komi PV ed. *Strength and power in sport*. Blackwell Science; 1992.
21. Pratley R, Nicklas B, Rubin M, Miller J, Smith A, Smith M, et al. Strength training increases resting metabolic rate and norepinephrine levels in healthy 50- to 65-year-old men. *J Appl Physiol* 1994;76:133-7.
22. Phillips SM, Tipton KD, Aarsland A, Wolf SE, Wolfe RR. Mixed muscle protein synthesis and breakdown after resistance exercise in humans. *Am J Physiol Endocrinol Metab* 1997;273:E99-107.
23. Williamson DL, Kirwan JP. A single bout of concentric resistance exercise increases basal metabolic rate 48 hours after exercise in healthy 59–77-year-old men. *J Geront Med Sci* 1997;52A:M352-5.
24. van Etten, LM, Westerterp KR, Verstappen FT, Boon BJ, Saris WH. Effect of an 18-week weight-training program on energy expenditure and physical activity. *J Appl Physiol* 1997;82:298-304.
25. Hunter GR, Byrne NM, Gower BA, Sirikul B, Hills AP. Increased resting energy expenditure after 40 minutes of aerobic but not resistance exercise. *Obesity* 2006;14: 2018-25.
26. Goran MI, Poehlman ET. Endurance training does not enhance total energy expenditure in healthy elderly persons. *Am J Physiol* 1992;263:E950-7.
27. Miller JP, Pratley RE, Goldberg AP, Gordon P, Rubin M, Treuth MS, et al. Strength training increases insulin action in healthy 50- to 65-year-old men. *J Appl Physiol* 1994;77:1122-7.
28. Ryan AS, Pratley RE, Goldberg AP, Elahi D. Resistive training increases insulin action in postmenopausal women. *J Geront Med Sci* 1996;51A:M199-205.
29. Braith RW, Stewart KJ. Resistance exercise training. Its role in the prevention of cardiovascular disease. *Circulation* 2006;113:2642-50.

30. Ishii T, Yamakita T, Sato T, Tanakas S, Fujii S. Resistance training improves insulin sensitivity in NIDDM subjects without altering maximal oxygen uptake. *Diabetes Care* 1998;21:1353-5.
31. Eriksson JG. Exercise and the treatment of type 2 diabetes mellitus. An update. *Sports J Med* 1999;27:381-91.
32. Sigal RJ, Kenny GP, Boulé NG, Wells GA, Prud'homme D, Fortier M, et al. Effects of aerobic training, resistance training, or both on glycemic control in type 2 diabetes. *Ann Intern Med* 2007;147:357-69.
33. Cauza E, Hanusch-Enserer U, Strasser B, Ludvik B, Metz-Schimmerl S, Pacini G, et al. The relative benefits of endurance and strength training on the metabolic factors and muscle function of people with type 2 diabetes mellitus. *Arch Phys Med Rehabil* 2005;86:1527-33.
34. Tucker LA, Silvester LJ. Strength training and hypercholesterolemia. An epidemiologic study of 8,499 employed men. *Am J Health Promot* 1996;11:35-41.
35. Kohl HW 3rd, Gordon NF, Scott CB, Vaandrager H, Blair SN. Musculoskeletal strength and serum lipid levels in men and women. *Med Sci Sports Exerc* 1992;24:1080-7.
36. Hill S, Bermingham MA, Knight PK. Lipid metabolism in young men after acute resistance exercise at two different intensities. *J Sci Med Sport* 2005;8:441-5.
37. Fagard RH, Cornelissen VA. Effect of exercise on blood pressure control in hypertensive patients. *Eur J Cardiovasc Prev Rehabil* 2007;14:12-7.
38. MagDougall JD, McKelvie RS, Moroz DE, Sale DG, McCartney N, Buick F. Factors affecting blood pressure during heavy weightlifting and static contractions. *J Appl Physiol* 1992;73:1590-7.
39. Kelley G. Dynamic resistance exercise and resting blood pressure in adults. A meta-analysis. *J Appl Physiol* 1997;82:1559-65.
40. American College of Sports Medicine. Physical activity, physical fitness, and hypertension. *Med Sci Sports Exerc* 1993;25:i-x.
41. Layne JE, Nelson ME. The effects of progressive resistance training on bone density. A review. *Med Sci Sports Exerc* 1999;31:25-30.
42. Rutherford OM. Is there a role for exercise in the prevention of osteoporotic fractures? *Br J Sports Med* 1999;33:378-86.
43. Vouri IM. Dose-response of physical activity and low back pain, osteoarthritis, and osteoporosis. *Med Sci Sports Exerc* 2001;33:S551-86.
44. Ettinger WH, Burns R, Meissner SP, Applegate W, Rejeski WJ, Morgan T, et al. A randomized trial comparing aerobic exercise and resistance exercise with a health education program in older adults with osteoarthritis. *JAMA* 1997;297:25-31.
45. Roddy E, Zhang W, Doherty M, Arden NK, Barlow J, Birrell F, et al. Evidence-based recommendations for the role of exercise in the management of osteoarthritis of the hip or knee. The MOVE consensus. *Rheumatology* 2005;44:67-73.
46. Bennell K, Hinman R. Exercise as a treatment for osteoarthritis. *Curr Opin Rheumatol* 2005;17:634-40.

47. Carpenter DM, Nelson BW. Low back strengthening for the prevention and treatment of low back pain. *Med Sci Sports Exerc* 1999;31:18-24.
48. van Tulder M, Malmivaara A, Esmail R, Koes B. Exercise therapy for low back pain. A systematic review within the framework of the Cochrane collaboration back review group. *Spine* 2000;25:2784-96.
49. Singh NA, Clements KM, Fiatarone MA. A randomized controlled trial of progressive resistance training in depressed elders. *J Gerontol A Biol Sci Med Sci* 1997;52:M27-35.
50. Paluska S, Schwenk TL. Physical activity and mental health. *Sports Med* 2000;29:167-80.
51. McCartney N. Role of resistance training in heart disease. *Med Sci Sports Exerc* 1998;30:S396-402.
52. McCartney N. Acute responses to resistance training and safety. *Med Sci Sports Exerc* 1999;31:31-7.
53. Daub WD, Knapik GP, Black WR. Strength training early after myocardial infarction. *J Cardiopulm Rehabil* 1996;16:100-8.
54. Pollock ML, Carroll JF, Graves JE, Leggett SH, Braith RW, Limacher M, et al. Injuries and adherence to walk/jog and resistance training programs in the elderly. *Med Sci Sports Exerc* 1991;23:1194-200.
55. MacDougall JD, Tuxen D, Sale DG, Moroz JR, Sutton JR. Arterial blood pressure response to heavy resistance exercise. *J Appl Physiol* 1985;58:785-90.
56. McKelvie RS, McCartney N, Tomlinson C, Bauer R, MacDougall JD. Comparison of hemodynamic responses to cycling and resistance exercise in congestive heart failure secondary to ischemic cardiomyopathy. *Am J Cardiol* 1995;76:977-99.
57. Fleck SJ. Cardiovascular response to strength training. In: Komi PV, ed. *Strength and power in sport*. Blackwell Science; 1992.
58. Fleck SJ, Dean LS. Resistance-training experience and the pressor response during resistance exercise. *J Appl Physiol* 1987;63:116-20.
59. Haslam DRS, McCartney N, McKelvie RS, MacDougall JD. Direct measurements of arterial blood pressure during formal weightlifting in cardiac patients. *J Cardiopulmonary Rehabil* 1988;8:213-25.
60. Borg GA. Psychophysical bases of perceived exertion. *Med Sci Sports Exerc* 1982;14:377-81.
61. Urhause A, Kindermann W. Sports-specific adaptations and differentiation of the athlete's heart. *Sports Med* 1999;28:237-44.

9. Infections and sports

Authors

Göran Friman, MD, PhD, Professor, Department of Infectious Diseases, Uppsala University Hospital, Uppsala, Sweden

Lars Wesslén, MD, Department of Infectious Diseases, Gävle Hospital, Gävle, Sweden

Ola Rønsen, MD, Chief Medical Officer for Norway's Olympic Team, Olympiatoppen, Oslo, Norway

Summary

In general, physical activity in association with infections is accompanied by certain medical risks, in part for the infected individual, in part for other athletic practitioners who can be infected. The latter is primarily relevant to team sports, but also other sports where the athletes have close physical contact, before, during or after training and competition. Both of these aspects are discussed in this chapter. Furthermore, suggestions on guidelines are provided, which primarily pertain to adult individuals, to counselling for healthcare personnel as well as others in the management and counselling of athletes affected by acute infections. Lastly, concrete retraining programmes are proposed after mononucleosis, which can also be used after pneumonia and other powerful infections.

Definitions, occurrence and causes

The infections that are in question in sports medicine in the Nordic region are caused by viruses or bacteria. Infections are very common, especially upper respiratory infections. Among these, the common cold is dominant. They are caused by viruses and are largely complication-free and self-healing, but sometimes bacteria can “take over” and give rise to complications such as sinus infections, ear infections and, in worst case, pneumonia. Sore throats (tonsillopharyngitis) are also most often caused by different viruses, but sometimes it is a matter of an infection by beta-streptococci, which requires antibiotic treatment. Mononucleosis is a special form of sore throat. It is viral, often has an extended course of disease and requires extra attention with careful follow-up and special advice to athletes. Infections of the heart muscle (myocarditis) can be caused by multiple viruses and bacteria, and like mononucleosis, constitute a special problem area in sports medicine,

requiring specialist treatment. Mononucleosis and myocarditis are therefore discussed in separate sections. Acute diarrhoea diseases (gastroenteritis) always give rise to fluid loss, which more or less affects performance capacity.

Infections of the skin and soft tissue requiring treatment are more common among athletes than the average population, although it is most often a matter of skin damage, such as scrapes, that seldom obstruct training and competition. Epidermophytosis (athlete's foot) is also common. The herpes virus causes small blisters on the skin, most often around the mouth, but also in other places. Herpes is not more common among athletes than others, but in contact sports can spread to other athletes. Infection to opponents and teammates occurs through small cracks in the skin. Herpes gladiatorum is classic, where blisters develop at several different places where the skin has been damaged.

A borrelia infection in the skin (erythema migrans) is seen among athletes exposed to tick bites. Borrelia can sometimes spread to the inner organs. Ticks can also transmit viral encephalitis (tick-borne encephalitis, TBE) within certain geographical areas. Borrelia can be cured with antibiotics and an effective vaccine exists to prevent TBE.

Symptoms, diagnostics, treatment and complications

The symptoms of infection are primarily due to the immune system's reaction to viruses and bacteria, and are signs that the body reacts normally. The symptoms of the common cold are known to all. Irritation of the nasal mucous membrane with a running nose and nasal congestion with or without a sore throat are most common as well as bronchitis with coughing and hoarseness in worse colds. Body temperature is normal or only slightly raised. In these cases, antibiotic treatment should be avoided.

Viruses that accumulate in the surface of the mucous membrane (epithel layer) damage it. Bacteria can then more easily gain a foothold and cause complications to the cold such as an **ear infection** (otitis media) and a **sinus infection** (sinusitis). A feeling of having an ear blockage, reduced hearing and pain in the ear and, respectively, thick green-yellow mucous and pressure at the base of the nose and cheeks with or without a fever are then common symptoms. With these conditions, anti-inflammatory preparations for the mucous membranes and, as a rule, antibiotic treatment are administered.

Bacterial tonsillitis (tonsillopharyngitis caused by beta-streptococci) usually starts abruptly with a fever, often up to 39 degrees, and painful swallowing in contrast to viral throat infections. There is no cough or rhinitis. The tonsils are swollen and red, often with yellow pus clots. As a rule, the soft palate and palatal arches are intensively red, usually with elements of skin bleeding (petechiae). There are often distinctly tender lymph nodes in the mandibular angles. Bacteria can be directly indicated with the help of a rapid test or culture from the throat. A more or less pronounced polymorphonuclear leukocytosis develops in the blood and the C-reactive protein (CRP) increases (an increase is a sign of inflammation or infection). Treatment is given with penicillin V (Kåvepenin[®]) for 10 days. With a shorter period of treatment, the risk of relapse is greater. For those allergic to penicillin, clindamycin (Dalacin[®]) is suggested instead.

With **viral tonsillopharyngitis**, onset is less sudden and the throat symptoms not quite as marked as a rule. Rhinitis/nasal congestion and/or a cough are also often present. Fever may or may not be present. The reddening of the mucous membrane is paler than with streptococcal tonsillitis. Grey-white clots occur in the tonsils. Several respiratory viruses can be the cause, such as an adenovirus. Tests to establish this, as well as some other respiratory viruses that can have similar symptoms, exist but are not used, since treatment against these viruses does not exist and the infection is self-healing. In tonsillopharyngitis among athletes, particularly when there is no simultaneous rhinitis, hoarseness or cough, it is wise to test for beta-streptococci, since the clinical presentation is not always typical and a streptococcus infection needs to be treated with antibiotics.

Acute diarrhoea (gastroenteritis) can be caused by a virus, bacteria or parasites and is associated with more or less fluid loss. The ensuing dehydration, which can give rise to reduced plasma volume, as well as the general impact of the infection reduces performance capacity. Additional fluid loss through sweating in connection with physical exertion can lead to a collapse. Undiagnosed heart disease can also manifest itself. Myocarditis can occasionally occur as a complication of infectious gastroenteritis.

Herpes blisters (herpes simplex-virus) are the most common form of “mouth ulcers”, but also occur at other places on the skin. They are self-healing, but have a tendency to return often among certain people. Early, local treatment with virus-inhibiting cream/ointment can be tried. Specific antiviral substances in tablet form or as an injection exist against herpes simplex, but are only used in special cases, for example, for persons with sharply reduced infection defence or in serious herpes illness such as encephalitis. In sports, special measures are required in outbreaks of herpes blisters in the skin among practitioners of contact sports.

Borrelia of the skin (skin borreliosis, erythema migrans) presents itself as a growing redness at the site of a tick bite. Redness caused by borrelia will, as a rule, present 4–5 days after the bite at the earliest, but sometimes first becomes visible after up to four weeks afterwards. It then grows in a ring shape for a few weeks and at the same time fades in the centre. On the arms and legs, it often appears more as red garlands than as a ring. Early redness within a few days at a tick bite is, however, a general bite reaction, which does not need to be treated unless it continues to grow in extent.

Most people who have skin borreliosis do not know that they have been bitten by a tick. A person that has a slowly growing redness somewhere on the body can have borreliosis, but it can also be due to many other conditions such as a fungal infection, eczema or other special skin diseases. Serologic tests are unreliable for skin borreliosis, which is therefore a clinical diagnosis. Skin borreliosis is indeed generally self-healing, but should still be treated with regular penicillin, because otherwise there is a risk that the bacteria spread to other organs, such as the central nervous system (neuroborreliosis), joints or the heart muscle. In neuroborreliosis, peripheral facial paralysis, in other words a paralysis of the muscles of one side of the face (Bell’s palsy), is a common manifestation. In other cases,

meningoencephalitis develops with fever, neck pain and sometimes radiating pain (radiculitis pains). Then, the spinal fluid (liquor) shows an increase in the number of leukocytes (pleocytosis) with a preponderance of monocytes, that is to say a clinical presentation similar to that of viral meningitis. The diagnosis of neuroborreliosis is established with an antibody test in blood and liquor. Neuroborreliosis is now treated with high doses of doxycyclin in tablet form as a rule. There is not yet a vaccine against borreliosis. Ticks should be removed with tweezers or special tick removers: pull straight out without rotating! Any remaining jaw sections eventually loosen without any special action.

Tick-borne encephalitis (TBE) is a virus infection that spreads through tick bites. It often has a two-phase course that begins with a few days' of fever and general malaise, after which a powerful headache arises and the fever rises again. In the spinal fluid, both pleocytosis with an excess of monocytes and an increase in albumin (barrier damage) are seen as signs of encephalitis. Palsies, often in an extremity (monoplegia), and cramps are not uncommon. There is often a long convalescence and residual symptoms such as concentration difficulties and focal muscle weakness are more common than previously believed. The virus diagnosis is made with the help of an antibody test (specific TBE-IgM antibody test in blood serum is positive five days after the first symptom and is always positive if encephalitis has had time to develop). No special treatment is available, but there is an effective vaccine and persons who are at risk of tick bites in the geographic areas in question should be vaccinated.

Mononucleosis (EBV infection)

Mononucleosis is a prolonged virus infection that gives rise to a sore throat, but also causes symptoms from several other organs. The cause is the Epstein-Barr virus (EBV), which has the throat as an entry point and comes from infected saliva from other persons. The majority are already infected as small children without developing the typical clinical presentation. However, this presentation arises if the infection first occurs in the teenage years or later ("kissing disease"). In mononucleosis, onset occurs more slowly than with streptococcal tonsillopharyngitis. Typical symptoms include swallowing pain that increases over several days, increasingly swollen lymph glands in the neck, as well as in the armpits, and a fever that reaches 39 degrees. Headache, muddled speech and acetone-smelling breath is common.

The virus spreads through the blood vessels to the entire reticulo-endothelial system (RES), where mainly the T-lymphocytes proliferate. The spleen swells to varying degrees, but is soft and therefore sensitive and fragile. In rare cases, the spleen can rupture spontaneously, for example, due to the increase in pressure that occurs in the abdomen in heavy lifts, which can result in life-threatening bleeding. The risk of the spleen rupturing increases if it is subjected to blows or pressure, common in contact sports for example. A ruptured spleen requires an immediate operation with the removal of the spleen to prevent the person from bleeding to death. Also refer to the section "Suggestions for guidelines for management and counselling" (1).

The course of the disease in mononucleosis often takes several weeks and blood tests provide good guidance for diagnosis. Accordingly, the total number of leukocytes in the blood increases at the same time that the white blood count is shifted in the first week of illness towards a dominance of mononuclear cells. Some of these have leaking cytoplasm, so-called atypical lymphocytes (McKinley-cells). Liver function tests in serum (for example ALAT) are slightly to moderately increased as a rule. Among older children, adolescents and adults, mononucleosis can in most cases be diagnosed with rapid testing with blood tests that indicate heterophilic antibodies. It is positive within 5–7 days after the onset of symptoms. In the cases where the rapid test has a negative outcome, samples can be sent for conventional analysis of specific IgM and IgG antibodies against EBV, which confirms or rules out the diagnosis. There is no specific treatment.

Myocarditis

Through the years, myocarditis has probably been the infection complication discussed most in connection with sports and training. This is because myocarditis can in exceptional cases have a serious development at the same time that the symptoms are often diffuse. In the majority of cases, myocarditis heals without residual symptoms and sports and training can be resumed. Sudden death or heart failure is rare (2, 3).

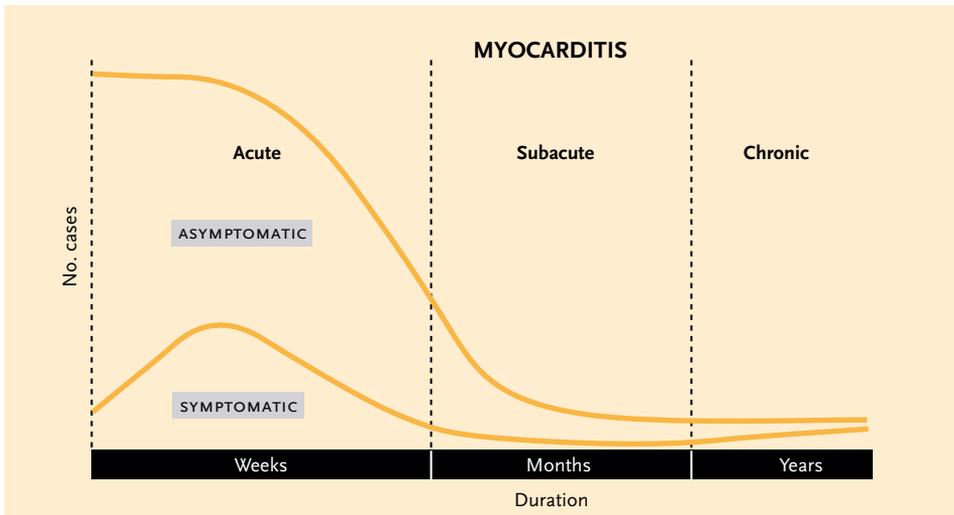


Figure 1. The main characteristics of the epidemiology of myocarditis. The knowledge of the large proportion of symptom-free (sub-clinical) cases is based on findings among persons that have died from other causes. From the original diagram by L Wesslén, 1997 (4).

The accumulation of sudden deaths among young Swedish elite orienteers during the 1980s and beginning of the 1990s was caused by an uncommon variant of bacterial myocarditis with no or very few symptoms until serious, acute dysrhythmias occurred. In these cases, the myocarditis had been prolonged (subacute). Despite the lack of symptoms in the majority of cases, similar advanced changes were seen in all of the hearts after death. Only one such death is known to have occurred among young orienteers (under the age of 35) at the elite level after 1992, when a six-month suspension of competition was introduced and antibiotic treatments were given to the Swedish elite ranked athletes.

Because of the special disease mechanisms, myocarditis is a condition of central interest in sports medicine. This is related to the infection being located in an organ that is activated and strained considerably in sports, particularly in oxygen-intensive (aerobic) endurance work. The metabolism of the heart muscle is then significantly increased. In increased activity and metabolism, the heart muscle is more receptive to infection than at rest. Among other things, this is due to an increased number of protein receptors that capture microorganisms then being exposed on the surface of the heart muscle cells. Microorganisms often come out in the blood during an early phase of a viral infection and can then more easily than at rest “attach” to and penetrate the heart muscle cells. Many different viruses and bacteria can infect the heart and give rise to myocarditis. Some microorganisms have a relatively large tendency to attack the heart, while others very seldom do so. Among the former are enteroviruses (primarily Coxsackie viruses) as well as many others, such as the Epstein-Barr virus (EBV, mononucleosis) and adenoviruses. Among the latter are the common cold viruses (rhinovirus and coronavirus). It is also well-established in experimental studies that increasing the work load of an individual with ongoing myocarditis results in an increase of the amount of microorganisms and the tissue damage in the heart muscle.

The majority of myocarditis patients have or have recently had a respiratory infection when the myocarditis becomes symptomatic. However, myocarditis can sometimes appear without the patient feeling any prior symptoms. Unfortunately, there are currently no rapid tests for all of the different viruses and bacteria that are of interest in the context of myocarditis (with the exception of beta-streptococci, EBV and a few more viruses). In addition, in respiratory infections, it is often difficult or impossible based on the symptoms and signs of the patient to determine which virus or bacteria is the cause and consequently, whether there is a risk of myocarditis. Consequently, the general recommendation is to avoid heavy physical loads and strain with acute symptoms of infection.

The symptoms of acute myocarditis most often have a rapid onset, ranging from diffuse sensations in the chest to sharp, often breathing-correlated pain in the area of the heart. As a rule, pain presupposes that the pericardium is inflamed, since the actual heart muscle (myocardium) does not clearly signal pain upon inflammation except in severe oxygen deficiency such as in a heart attack. With viral myocarditis, the pain-sensitive pericardium is generally more or less involved in the infection, but this is often not the case with bacterial myocarditis. Rapidly occurring dizziness, unexplained shortness of breath and fatigue are other common symptoms in acute myocarditis. Irregular heart activity, a racing heart, dizziness or fainting during ongoing exertion are always serious signs. All of the symptoms

mentioned here and the signs shall always lead to an emergency physician consultation. Occasional extra heartbeats at rest directly after completed exertion are, however, common in the healthy heart, but if there are several extra beats in an uninterrupted sequence, a doctor should be consulted. Under a microscope (histopathologically), randomly spread inflammatory sites (accumulations of leukocytes) are as a rule seen in the heart with myocarditis in the occasional cases that lead to death and are autopsied. If such an inflammatory site strikes the heart's impulse relay system, sudden death can occur even without prior symptoms due to electric instability that leads to serious cardiac arrhythmias.

The prognosis in acute myocarditis, with or without pericarditis, is good in the majority of cases, in other words the myocarditis heals without residual symptoms and sports can gradually be resumed. The follow-up should be individualised. In recent years, the diagnosis of myocarditis has been expanded with the concept of inflammatory cardiomyopathy, which means both that the common signs of heart muscle inflammation have been observed and that a functional disruption of the heart has also been established. In these cases, as a physician, one should be very careful with the investigation and follow-up to ensure a desirable healing process. In the uncomplicated cases, it most often suffices with an exertion ECG being done prior to giving a clean bill of health together with a doctor's visit. A large Finnish study of myocarditis among military conscripts indicated that the majority of the recruits with myocarditis could return to military duty within 2–3 months of the onset of the disease (2); in a minority of cases, longer convalescence was required.

There are still no vaccines for the majority of the viruses and bacteria that can give rise to myocarditis. Therefore, general precautions must instead be taken to avoid infection to the furthest extent possible.

How is physical capacity affected by infections?

Infections with fever (with or without myocarditis) are accompanied by a change in metabolism with the aim of mobilising the infection defences (3, 4). The healing of an infectious disease does not occur automatically, but instead requires that the body succeeds in defending against the microorganisms (viruses and bacteria), sometimes with the help of antibiotics. This effort affects various organs and tissues. Amino acids are expended for the increased synthesis of immunoglobulins, immune cells, etc. and as a source of energy. Fever further increases the energy needs since the metabolic processes then go faster. In addition, a loss of appetite (anorexia) is common in fever, and the body is largely relegated to using its own deposits for the energy supply. The fat deposits cannot be effectively utilised in fever and amino acids are instead collected from the striated muscles. A negative nitrogen balance is therefore quickly established. A study of a cross section of young men indicated that muscle strength had fallen by 15 per cent upon *recovery* after a week-long infection with fever. The individual variations were somewhat large, however. The relative inactivity or confinement to bed that can be seen as a part of the treatment of febrile infections contributed insignificantly to the drop in muscle strength. This was instead primarily due to the infection-caused adjustment of the metabolism when muscle

is decomposed to become energy for the immune system (muscle catabolism). The aerobic (oxygen-dependent) capacity had, however, fallen by 25 per cent and inactivity/being bedridden significantly contributed to this. Besides “muscle condition”, aerobic capacity is also determined by the blood volume and blood circulation’s autonomic controls (the sympathetic and parasympathetic nervous system), which were both disadvantaged by both the infection and being bedridden. During an *ongoing* infection and fever, aerobic capacity, muscle strength and muscle stamina are reduced as is the coordination of muscle activities. An athlete that has to perform in connection with an infection can therefore expect reduced muscle strength, reduced aerobic and muscular endurance and degraded coordination ability, which has an impact in elite contexts where competition is tough.

Physical activity both stimulates and inhibits the immune system

In general, physical exertion stimulates the immune system and consequently the defence against infection. An untrained individual that begins to exercise regularly gradually strengthens his or her immune function and thereby decreases his or her receptiveness to infections. Intensive aerobic endurance work (such as middle and long distance running, skiing, cycling, rowing, orienteering) of a duration of at least one hour initially gives a strong stimulation of the immune system, which afterwards turns to the opposite; a period of temporary weakening of the immune function occurs after the exercise session/competition. In other words, the immune system “recovers” after the strong stimulation that the exercise/competition incited, and then the susceptibility to infections is temporarily greater (figure 2).

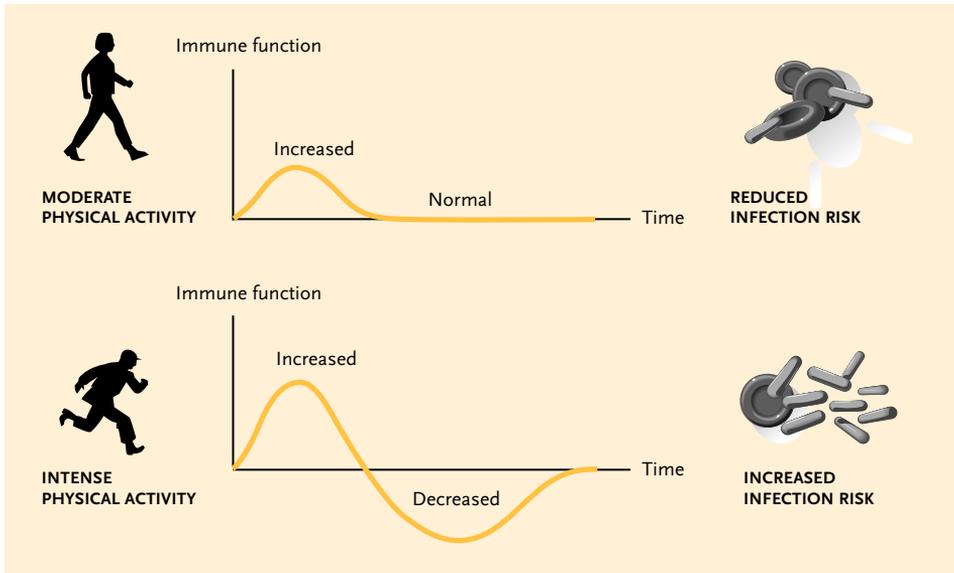


Figure 2. During moderate to intense physical exertion, the immune function is stimulated through the mobilisation of lymphocytes to the blood, among other effects. Intense exertion is followed by a period of degraded immune function with reduced NK cell activity, subdued lymphocyte proliferation and reduced levels of IgA antibodies in the saliva. Susceptibility to infection is then increased (5).

This effect is seen in both untrained and very well-trained individuals. The duration of this drop in immune function partially depends on the exertion’s intensity and duration and is partially individual. One usually expects the drop to be able to last from a few hours up to a day (even longer after a marathon, for example). This type of repeated exertion sessions done with too little time in between is at risk of leading to a prolonged increase in susceptibility to infection and greater risk of complications if one comes down with an infection (figure 3). The planning of the frequency of training and competition as well as resting periods is therefore important.

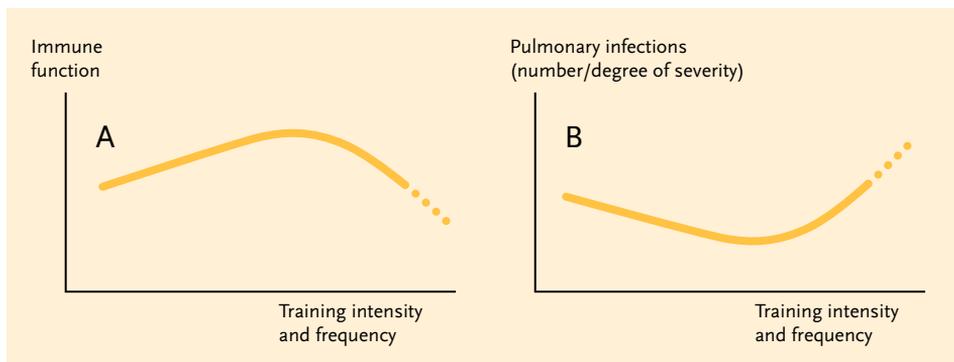


Figure 3. A. The immune function in relation to physical activity and exercise. B. Susceptibility to respiratory infections in relation to physical activity and exercise (6).

Infections and physical activity – medical risks

One perception that is often met with is that one can “run the rubbish out of the body”, in other words, get rid of an infection that has begun to show symptoms through an intense exercise session. However, there is no scientific support for this working. On the contrary, it can be risky. The infection can take a more serious turn and complications can occur. In other words, if an infection has already been established, the immune stimulation of such an exercise session provides no benefit. On the contrary, it can worsen the infection. For example, an upper respiratory infection can spread to the bronchi and lungs and, if one is unfortunate, myocarditis can occur. This is also true even if the individual has no fever.

It is at the very first symptoms of an infection such as a general feeling of malaise, an irritation in the throat, etc., with or without a fever, that it is very difficult to judge whether the symptoms constitute the start of a serious condition or not. Besides the risk of myocarditis, this is an important reason to apply the general recommendation to refrain from intense physical exertion while awaiting the continued development (3).

The risks of physical activity to those who are infected vary strongly depending on the location of the infection, its degree and microbial cause, as well as the intensity and type of physical activity. Intense/prolonged physical exertion, and even mental stress, can reduce the defence against infection and worsen the infection, as mentioned above. Furthermore, a subclinical (without symptoms) infection complication, such as myocarditis, is made worse by heavy exercise.

The risk level is generally higher for a trained and competing athlete, particularly at the elite level, than for the regular exerciser. A physician’s advice to individual patients must therefore be individualised.

Muscular and cardiopulmonary performance capacity is reduced by the majority of infections, especially if the infection is associated with fever. This temporarily reduced performance capacity cannot generally be prevented by continuing to exercise during the infection. On the contrary, exercise during an infection can lead to additional reductions in performance capacity, infection complications and other injuries. This is particularly true with mononucleosis, which has a special immunological situation (1).

The nervous system is generally affected in infection and fever so that coordination capacity (“motor precision”) is degraded. This condition can affect performance capacity, especially in sports that require a high degree of precision. At the same time, the risk of injuries in joints, ligaments and tendons increases (3).

Physical exertion with a fever entails an increased hemodynamic load on the heart compared with exertion in a healthy individual. This can lead to the manifestation of another, perhaps as yet undiagnosed, heart disease such as coronary sclerosis (obstructed coronary arteries), hypertrophic cardiomyopathy (pathological thickening of parts of the heart muscle) or myocarditis, sometimes in the form of a fatal arrhythmia.

In general, the physician should therefore always assume a cautious attitude to physical activity in his or her advice to infected individuals. This is particularly important when it concerns trained and competing athletes, who have greater “pressure” of their own and from their surroundings to perform than regular exercisers. Extra attention must be devoted to elite athletes, where the requirements and expectations of participation and success are extra large. Mental stress can always weaken the immune system. In some sports, the reduction of performance capacity associated with infection can be compensated by the athlete’s routine and skill, which can incite greater risk-taking. The elite athlete must sometimes take certain risks to win, but they should not be unreasonably high and active individuals must be aware of them. Here, the physician has a duty of contributing to a reasonable risk assessment of the individual case.

The following proposal of concrete guidelines for management and counselling in cases of infection in elite athletes, primarily intended for general practitioner physicians, was published in connection with the 2000 Sydney Olympics (3).

Suggestion for guidelines for management and counselling

Risks to the individual

In people with fever (38 degrees Celsius or more), rest should always be recommended.

People who know their normal temperature and pulse curves should rest, if their resting temperature has increased by 0.5–1 degree or more and at the same time their resting heart rate has risen by 10 beats/minute or more, in combination with general symptoms (malaise, muscle pains, muscle tenderness, diffuse joint pains, headache).

In general malaise, alone or in combination with one or more of the symptoms muscle pains, muscle tenderness, diffuse joint pains and headache, should give reason to recommend rest, until these symptoms have disappeared.

In all infections, caution should be observed during the first 1–3 days of an infection, even with a normal body temperature, until the body’s defence against infection has had time to become mobilized and until the further development of the infection becomes clear. Serious infections often have prodromal symptoms and in such cases it often takes 1–3 days before the serious nature of the infection becomes evident.

In people with nasal catarrh without a sore throat, cough or general symptoms, caution is recommended during the first 1–3 days, after which training can gradually be resumed if the symptoms do not become worse. (Note! Differential diagnosis: allergy.)

If additional manifestations exist at the same time as nasal catarrh (such as sore throat, hoarseness or cough), one should be more restrictive, depending on the degree and development of the symptoms.

In people with a sore throat without any other manifestations, caution is advised until the symptoms have begun to improve. In cases of beta-streptococcal tonsillopharyngitis,

which should be treated with penicillin for 10 days, rest is recommended until the symptoms have disappeared and caution is recommended during the first week of treatment even in the absence of symptoms, due to the risk of residual bacterial toxins that can affect the heart. (The objective of the last three days of antibiotic treatment is to reduce the risk of relapse.)

In mononucleosis, the situation is special. See “Advice regarding the start of training and training progression in athletes after mononucleosis” below (1).

Here, it shall only be mentioned that persons who pursue contact sports such as football, wrestling, weightlifting, etc., should wait 4–6 weeks after the onset of symptoms before resuming these sports, because it often takes such a long time before the spleen has regained its normal size and consistency. An enlarged spleen in mononucleosis is fragile and can rupture if it is subjected to a blow or increased pressure, and weightlifting can cause a spontaneous rupture.

In cystitis, a urinary tract infection without fever which mainly affects women, strenuous physical exertion should be avoided until the symptoms have subsided.

In gastroenteritis, heavy physical exercise should be avoided.

In skin infections, the recommendations need to be based on an individual assessment. All athletes should observe caution in episodes of herpes accompanied by regional lymphadenitis or general symptoms. Minor, surface skin infections seldom constitute contraindications to training and competing. An exception is a dermal herpes infection among wrestlers and other practitioners of contact sports. They should refrain from practicing the sport even with minor herpes lesions until the vesicles have dried. Erythema migrans should be treated with penicillin for 10 days and rest is recommended during the first week.

People with ongoing genital infections should avoid strenuous physical exertion. In asymptomatic genital chlamydial infection, it seems reasonable to restrict the physical activity during the period of antibiotic therapy, after which the infection can be considered to have healed.

Asymptomatic HIV infection constitutes no hindrance to exercise and sports. There are no indications that physical activity and sports have an unbeneficial effect on the health of HIV infected individuals. However, it has been documented that exercise and competition have an important promotional effect on the quality of life of many HIV patients.

Risks to the heart

In most cases of febrile infectious diseases, training can be resumed as soon as the fever has abated (3). This should be done gradually and it is important to pay attention to the “body’s signals” at the same time. If unexpected symptoms suspected of coming from the heart should appear, for example dizziness/fainting under exertion (exertional syncope), pain, a sense of pressure or discomfort in the chest, irregular heart beats, abnormal breathlessness or fatigue, the training should be discontinued and a physician consulted, because myocarditis can occur in connection with a number of different infections. Fainting under exertion is a serious symptom that should always lead to an emergency physician’s examination of the heart. It is important to point out that myocarditis can develop even without prior symptoms of infection. In middle aged people, the possibility of acute coronary disease (obstructed

coronary arteries), in other words acute myocardial infarction or angina pectoris, should also be considered with symptoms of this type. This is particularly true of chest pain brought on by exertion. Those intending to resume training after an acute myocarditis should seek individual consultation by a physician. A European expert group suggest competitive sports may be resumed within six months of the acute disease, provided that the individual has no symptoms, normal left ventricular function and no arrhythmias (10).

In general, it may be said that in infections, as in other situations, it is important to “listen to the body’s signals”.

Antibiotic treatment constitutes no inherent obstacle to physical activity and sports. It is the infection that “decides”.

Risks to the environment – epidemiological aspects

Plantar warts are readily spread via shower floors and changing rooms. These warts should therefore be treated quickly in athletes.

Wrestling is probably the sport where the athletes have the closest physical contact. Besides air and droplet borne infections from the air passages, there is a significant risk of transmitting disease through contact. “Mat herpes” (herpes gladiatorum) is a classic example of this, where the herpes virus from one individual is inoculated through visible or invisible skin lesions to the other. This often occurs through small surface burns that arise from the friction when the wrestler lands on the mat. Epidemics of herpes gladiatorum among wrestlers have been described many times.

Respiratory tract infections can readily be transmitted both by droplet infection and by contact (direct or indirect contact via objects) among sportspeople who are in close proximity before, during or after a training or competitive event. Examples of this are countless. In addition, the fact that strenuous or prolonged physical exertion can reduce the defence against infection increases the susceptibility to respiratory tract infection.

Because prevention of exposure is the only prophylactic measure available, the risks of infection and the mechanisms of infection should be known by the individual athlete, as well as by trainers and sports leaders, before an infected individual allows himself or is allowed to meet his fellow participants prior to important training and competitive events. Annual immunization against influenza should be recommended for elite athletes. Vaccination against tick-borne encephalitis (TBE) is important for those spending time in the forest or land in areas of exposure (see above under “Symptoms, diagnostics, treatment and complications”).

Athletes with HIV infection should be allowed to participate in sports just like any others. Physicians of HIV patients who are engaged in sports associated with a risk of exposure of blood, such as wrestling, boxing, football and so on, should inform the patients concerned of the theoretical risk that the infection can be transmitted further. In the U.S., doctors are additionally recommended to strongly advise against their HIV patients’ continued participation in sports of this kind. It is important to consider the anonymity aspects and to make sure that the infection status of the person concerned does not come to the knowledge of the leaders or teammates unless the individual has given his or her consent.

Advice regarding the start of training and training progression

– after mononucleosis and other infectious diseases with significant reduction of the physical functional capacity

In mononucleosis, the immune system is the site of the infection, in other words the virus is located in the immune cells. Consequently, a particularly strong immunological activation occurs in mononucleosis. Because physical exercise is inherently immune stimulating, disease symptoms can therefore readily return when exercise is resumed (1). There is no simple test that indicates the activity level of the immune system to use as a guide. It is therefore important that elite athletes affected by mononucleosis consult with a physician who has experience of infection and sports medicine when the symptoms are on the way to subsiding and training begins to come into question again. The patient should have been symptom-free and managed the daily activities, in other words have had a clean bill of health, for at least one week before the physician makes his or her clinical assessment and potentially approves the resumption of training. Sometimes, fatigue after mononucleosis can last for many weeks or even months and consequently, the appropriate time to resume training must be assessed individually. The following factors can serve as guides (1):

1. The patient's general state of health, freedom from symptoms such as fever, fatigue, muscle pain, muscle tenderness, etc.
2. Normalisation/reduction of increased counts of leukocytes, lymphocytosis and liver enzymes.
3. Normalisation of any spleen enlargement, especially important among those active in contact sports such as wrestling, football, hockey, etc., as well as weightlifting (since Valsalva's manoeuvre can cause a ruptured spleen).

The physician must make a comprehensive assessment since no single test predicts the suitable point in time for the start of training. It often takes 4–6 weeks from the onset of symptoms for an enlarged spleen to regain its normal size and consistency and thereby its protected place under the fifth rib (1, 7–9). An ultrasound examination can be recommended for contact sports practitioners who are free from symptoms and ready to resume training before then.

How much training is appropriate at the beginning and how quickly can one return to normal training after mononucleosis?

For ethical reasons, there are no controlled, scientific studies that can provide a conclusive answer to this question, since it would require at least one test group with training at a potentially harmful level of exertion. There are not even good studies with moderate exertion regarding the “return-to-play” problems. Consequently, the advice given is based on the individual physician's collective experience.

As a general recommendation, the following advice can be given to apply to the first month (1):

1. Train so cautiously and lightly that the pulse does not exceed approximately 120 beats per minute and you do not become especially out of breath.
2. Begin with 20–30 minute long training sessions, preferably alternating light strength and endurance training, and increase the training time by 5 minutes every training session.
3. Include one day of recovery and rest between every day of training, in other words train every other day the first week.
4. Carefully note how you tolerate the training and ensure that you recover during the day of rest before you train again the next day.
5. Take a break of 2–3 days, and possibly consult with your doctor, if you should feel that the disease symptoms return or other problems occur.
6. As long as the first 3–4 training sessions (6–8 days) could be completed without problem, you can continue with a cautious increase in the intensity of the training by increasing the number and length of the training sessions per week.
7. Use at least as much time to train up to your normal training amount and intensity (condition level) as the time the infection symptoms lasted when you were ill. Listen to your body's signals – consult with your physician when necessary – more time may be needed to come back!

References

1. Rønsen O. Prevention and management of respiratory tract infections in athletes. *New Studies in Athletes* 2005;20:49-56.
2. Karjalainen J, Heikkilä J. Incidence of three presentations of acute myocarditis in young men in military service. *Eur Heart J* 1999;20:1120-5.
3. Friman G, Wesslén L. Special feature. Infections and exercise in high-performance athletes. *Immunology and Cell Biology* 2000;78:510-22.
4. Friman G, Ilbäck NG. Acute infection. Metabolic responses, effects on performance, interaction with exercise, and myocarditis. *Int J Sports Med* 1998;19(suppl. 3):pp.172-82.
5. Klarlund-Pedersen B, Friman G, Wesslén L. Exercise and infectious diseases. In: Kjaer M, Krogsgaard M, Magnusson P, Engebretsen L, Roos H, Takala T, et al. *Textbook of sports medicine*. London. Blackwell Science Ltd; 2003, pp. 410-21.
6. Friman G, Wesslén L, Karjalainen J, Rolf C. Infectious and lymphocytic myocarditis epidemiology and factors relevant to sports medicine. *Scand J Med Sci Sports* 1995;5: 269-78.
7. Eichner ER. Sports medicine pearls and pitfalls. Defending the spleen. Return to play after infectious mononucleosis. *Curr Sports Med Rep* 2007;6:68-9.
8. Waninger KN, Harcke HT. Determination of safe return to play for athletes recovering from infectious mononucleosis. A review of the literature. *Clin J Sport Med* 2005;15: 410-6.
9. Auwaerter PG. Infectious mononucleosis. Return to play. *Clin Sports Med* 2004;23:485-97.
10. Corrado D, Pelliccia A, Bjørnstad HH, Vanhees L, Biffi A, Borjesson M, Panhuyzen-Goedkoop N, Deligiannis A, Solberg E, Dugmore D, Mellwig KP, Assanelli D, Delise P, van-Buuren F, Anastasakis A, Heidbuchel H, Hoffmann E, Fagard R, Priori SG, Basso C, Arbustini E, Blomstrom-Lundqvist C, McKenna WJ, Thiene G; Study Group of Myocardial and Pericardial Diseases of the European Society of Cardiology. Cardiovascular pre-participation screening of young competitive athletes for prevention of sudden death: proposal for a common European protocol. Consensus Statement of the Study Group of Sport Cardiology of the Working Group of Cardiac Rehabilitation and Exercise Physiology and the Working Group of Myocardial and Pericardial Diseases of the European Society of Cardiology. *Eur Heart J*. 2005;26(5):516-24.

10. Sports and sudden death

Authors

Mats Börjesson, MD, PhD, Associate Professor, Department of Medicine, Sahlgrenska University Hospital, Gothenburg, Sweden

Eva Nylander, MD, PhD, Professor, Division of Cardiovascular Medicine, Linköping University Hospital, Linköping, Sweden

Erik Ekker Solberg, MD, PhD, Diakonhemmet Hospital, Oslo, Norway

Summary

Cases of sudden death (SCD) among young athletes are usually due to rare previously undiagnosed hereditary and/or congenital cardiovascular diseases. The Swedish National Board of Health and Welfare recommends cardiac screening of risk groups, such as those with known cardiovascular diseases, or those with a family history of sudden cardiac death, or people who have alarming symptoms in connection with physical exertion. However, tracing individuals with hidden cardiovascular disease through cardiac screening of all adolescents or athletes or those who are routinely physically active is not recommended. This is because the diseases are rare and the diagnostics are not sufficiently accurate to identify all of those with the diseases, which leads to problems with both false negatives and false positives.

However, the Swedish Sports Confederation as well as the Swedish National Board of Health and Welfare recommend targeted heart check-ups of elite athletes.

Definition

Sudden cardiac death is commonly defined as “death occurring within one hour of the onset of symptoms in a person with previously known or unknown heart disease”, if death was witnessed (1), or “death within 24 hours of the person having been seen alive and well with no other known cause” if death is not witnessed.

Causes

The cause of sudden death related to sports or physical activity, not related to trauma or accident, is almost always heart disease. In this context, athletes are divided into younger

(< 35 years-old) and older (> 35 years-old), which is of importance regarding the specific disease that causes death (2).

In athletes over the age of 35, the cause is almost exclusively coronary artery disease/myocardial infarction. In persons with an underlying coronary disease, there is an elevated risk of sudden death in connection with intense physical activity. However, this risk decreases in persons who pursue regular physical activity. Regular, individually tailored physical activity is also of considerable health benefit for those with established coronary disease, in part due to the positive effects on classical risk factors and endothelial function. Sudden death resulting from coronary artery disease among people over the age of 35 will not be discussed in more detail in this chapter.

In those under the age of 35, virtually all cases of SCD involve people who have an undiagnosed heart condition that predisposes them to a life-threatening arrhythmia triggered by strenuous physical activity. There are several relatively uncommon diseases that can cause sudden cardiac death among young people who have lived unaware of the underlying condition. The most common is a heart muscle disease (so-called cardiomyopathy, the most common of which is hypertrophic cardiomyopathy, which has a prevalence of approximately 1/500), malformations of the coronary arteries and diseases that affect the heart rhythm and the conducting system, so-called ion channel diseases.

Prevalence/Incidence

The prevalence of sudden cardiac death in persons under the age of 35 is approximated to 1–2/100,000 individuals per year. In total, around 0.3 per cent of young people suffer from congenital/hereditary heart defects with an elevated risk of sudden death during intense physical activity. The causes of sudden cardiac death in this group may be population specific, but hypertrophic cardiomyopathy is most common, followed by coronary artery anomaly, arrhythmogenic right ventricular cardiomyopathy, which causes malignant arrhythmia, followed by ion channel diseases, Wolff-Parkinson-White (WPW) syndrome, valvular heart disease, Marfan syndrome and myocarditis.

Diagnostics

Examinations intended to find individuals with the diseases associated with an elevated risk of sudden cardiac death in connection with sports have been frequently discussed in recent years. This identification is possible, since a large part of the diseases are hereditary and many are also accompanied by findings on a 12-lead-resting electrocardiogram (ECG). Moreover, the bearer of the disease can often have symptoms that may incite suspicion of illness, but that sometimes could be ignored by the athlete himself and by those in their surroundings.

These facts constitute the background of the recommendations from the Swedish National Board of Health and Welfare (3) that people who have:

1. relatives that have died suddenly and unexpectedly before the age of 40,
2. first-degree relatives with diagnosed hypertrophic cardiomyopathy or another cardiovascular disease associated with an elevated risk,
3. “alarming symptoms” during sports activities, such as chest pain, dizziness, a feeling of fainting (pre-syncope), distinct tachycardia or abnormal breathlessness (dyspnoea),
4. demonstrated abnormal findings on ECG, shall be examined with regard to the diseases in question (see below).

Targeted cardiac screening of athletes

The European Society of Cardiology (ESC) recommendations

Should all athletes have a health exam to identify persons who have a hidden heart disease? This has been discussed and has been handled differently in different countries. In the U.S., recommendations for “preparticipation screening” of athletes have existed for many years, consisting of questions about family history, symptoms and a physical examination. No ECGs are recommended routinely in the U.S. at the present time (4). Since the beginning of the 1980s, Italy has introduced mandatory screening of all “competitive athletes” consisting of medical history, physical examination and resting ECG (5).

In 2005, the European Society of Cardiology (ESC) presented a proposal for a common European protocol concerning cardiac screening of individuals who participate in competitive sports (6). The objective is to prevent sudden cardiac death by finding potential cardiovascular abnormalities that could convey an elevated risk of sudden death during intense physical exertion.

The European expert group recommends a systematic cardiovascular evaluation of everyone who is to participate in organised competitive sports. The recommended protocol comprises the following:

1. personal and family medical history (symptoms and heredity),
2. clinical examination,
3. 12-lead resting ECG.

If no relevant findings are made on this screening, the person is judged to be eligible for competitive sports. Upon abnormal findings, further examinations are needed by a physician experienced in sports cardiology. Examinations such as echocardiography, maximum exercise tests, 24-hour ECG, MRI, angiography/heart muscle biopsy and electrophysiological examinations may be advocated.

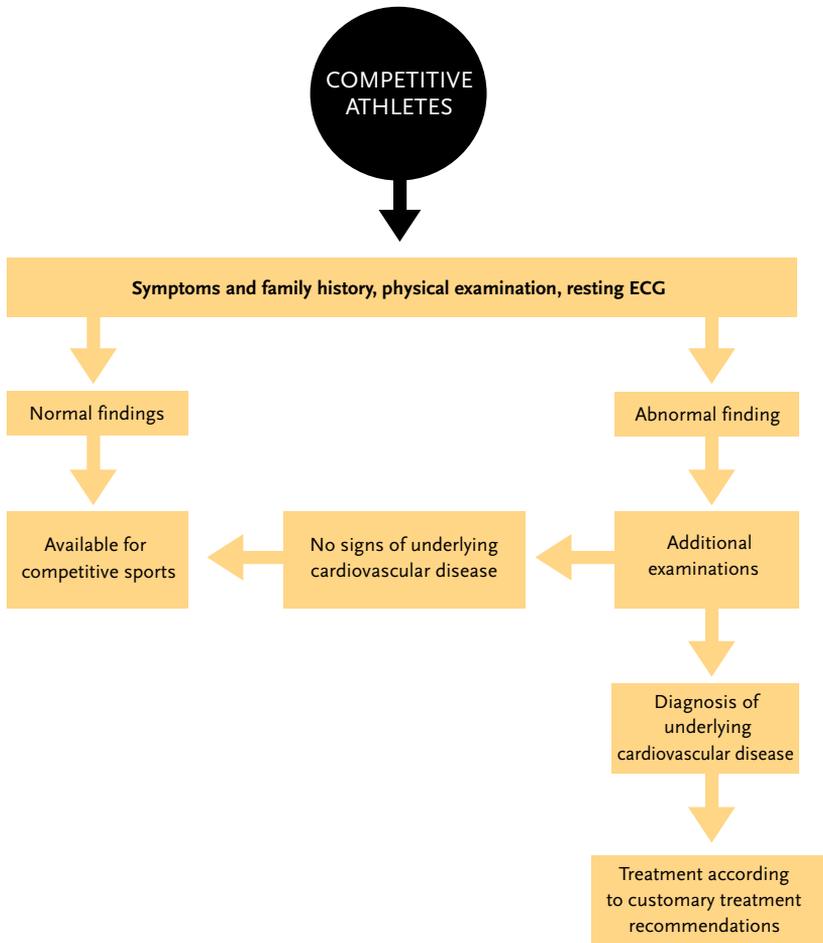


Figure 1. European Society of Cardiology's screening recommendations. Adapted from: Corrado et al. *Eur Heart J* 2005.

Nordic recommendations

Previously, no screening was recommended in the Nordic countries. They were historically even able to compete internationally without having been subjected to targeted cardiac screening, although regular pre-season examinations were conducted, focusing on the musculoskeletal system.

The ESC recommends that every country adapt the existing recommendations to their specific healthcare systems, including making practical adjustments with regard to access to sports medicine expertise.

The discussion in the Nordic countries in recent years has focused on the problems versus the benefits of screening. Screening as the term is commonly used, meaning looking for disease in large population groups without elevated risk, does not meet the WHO criteria. In addition, the diseases are too rare and the diagnostic methods do not have sufficient sensitivity or specificity. The expression screening may therefore be somewhat inappropriate, since most advocate a more targeted examination limited to specified risk groups. Consequently, the expression “targeted cardiovascular examination” may be more relevant.

In Sweden, a review of existing procedures has been conducted and, since 2005, the Swedish Sports Confederation has recommended that the examinations including medical history, physical examination and resting ECG should be confined to elite athletes (7). “Elite” is defined by the respective sporting associations and includes students at national sports gymnasiums. Since 2006, the Swedish National Board of Health and Welfare recommends (3) screening of risk groups as well as elite athletes.

The debate continues in the other Nordic countries, but no formal recommendations from the authorities have yet been issued (8). The Danish Society of Cardiology emphasizes the importance of targeted screening of certain risk groups with symptoms and a positive family history, but does not propose any general screening. Authors from Finland and Sweden have recently proposed a “Nordic approach” where cardiac screening is limited to elite athletes in sports with a considerable strain on the cardiovascular system (most). Education is also proposed to provide better knowledge about risk groups in both sports and among non-athletes (9).

Does screening save lives?

In Italy, where cardiac screening of competitive athletes has been conducted since the 1980s, a decrease in the incidence of sudden cardiac death from 4.2/100,000 per year to 0.4/100,000 per year (10) during the period 1979–2004 has been shown. However, this was not a controlled study and factors other than screening may have played a role.

A number of international sports associations also currently recommend, or plan to introduce, mandatory cardiac screening for participating athletes in international elite contexts (UEFA, FIFA).

Recommendation summary

- Greater preparedness in healthcare and the school system is proposed to find first-degree relatives of individuals with heart disease that is associated with an elevated risk of sudden death, regardless of whether they participate in sports or not. Similarly, greater awareness of these problems in the sports community is of value.
- Awareness of exertion-related symptoms, in the form of chest pains, dizziness, syncope, tachycardia attacks or abnormal breathlessness (dyspnea) is recommended, and athletes with such symptoms should be examined at the appropriate level of the healthcare system.
- Elite athletes are recommended targeted cardiac screening with medical history, physical examination and resting ECG. This is the responsibility of organised sports (clubs, sports schools), primarily through their respective team physicians/school doctors. However, abnormalities found should lead to a referral for further examination in a healthcare facility with good knowledge in sports cardiology, sports physiology and diagnostics.

References

1. Myerburg RJ, Kessler KM, Bassett AL, Castellanos A. A biological approach to sudden cardiac death. Structure, function and cause. *Am J Cardiol* 1989;63:1512-6.
2. Maron BJ, Thompson PD, Puffer JC, McGrew CA, Strong WB, Douglas PS, et al. Cardiovascular preparticipation screening of competitive athletes. A statement for health professionals from the Sudden Death Committee (clinical cardiology) and Congenital Cardiac Defects Committee (cardiovascular disease in the young), American Heart Association. *Circulation* 1996;94:850-6.
3. Swedish National Board of Health and Welfare. Plötslig hjärtdöd bland barn, ungdomar och unga vuxna vid idrott och fysisk ansträngning. Komplettering av Socialstyrelsens riktlinjer för hjärtsjukvård 2004. [Sudden cardiac death among children, adolescents and young adults in sports and physical exertion. Supplement to the Swedish National Board of Health and Welfare's guidelines for cardiac care 2004.] Stockholm: Swedish National Board of Health and Welfare; 2006.
4. Maron BJ, Thompson PD, Ackerman MJ, Balady G, Berger S, Cohen D, et al. Recommendations and considerations related to preparticipation screening for cardiovascular abnormalities in competitive athletes. 2007 update. A scientific statement from the American Heart Association Council on Nutrition, Physical Activity, and Metabolism, endorsed by the American College of Cardiology Foundation. *Circulation* 2007;115:1643-55.
5. Pelliccia A, Maron BJ. Preparticipation cardiovascular evaluation of the competitive athlete. Perspectives from the 30-year Italian experience. *Am J Cardiol* 1995;75:827-9.
6. Corrado D, Pelliccia A, Bjornstad HH, Vanhees L, Biffi A, Borjesson M, et al. Cardiovascular pre-participation screening of young competitive athletes for prevention of sudden death. Proposal for a common European protocol. Consensus Statement of the Study Group of Sport Cardiology of the Working Group of Cardiac Rehabilitation and Exercise Physiology and the Working Group of Myocardial and Pericardial Diseases of the European Society of Cardiology. *Eur Heart J* 2005;26:516-24.
7. Swedish Sports Confederation. Plötsliga dödsfall i samband med idrottsutövning. Bakgrund och rekommendationer. [Sudden death in association with practicing sports. Background and recommendations.] Stockholm: Swedish Sports Confederation; Sept. 2005.
8. European Heart Rhythm Association, Heart Rhythm Society, Zipes DP, Camm AJ, Borggrefe M, Buxton AE, et al. The report of the task force on sudden death. Reducing the risk. A strategic approach. *J Am Coll Cardiol* 2006 Sep 5;48:e247-346.
9. Hernelahti M, Heinonen OJ, Karjalainen J, Nylander E, Börjesson M. Sudden cardiac death in young athletes. Time for a Nordic approach in screening? *Scand J Med Sci Sports* in press.
10. Corrado D, Basso C, Pavei A, Michieli P, Schiavon M, Thiene G. Trends in sudden cardiovascular death in young competitive athletes after implementation of a preparticipation screening program. *JAMA* 2006;296:1593-601.

11. Children and young people

Author

Ulrika Berg, MD, PhD, Department of Woman and Child Health, Paediatric Endocrinology Division, Karolinska Institutet and Astrid Lindgren's Children's Hospital, Stockholm, Sweden

Summary

Our society demands less and less everyday physical activity, such as that in connection with transportation to and from work or school. Some places, where children previously played freely are now judged to be dangerous in various ways. There is also a concern that children and young people, just like adults, are becoming less physically active, which could lead to health problems as early as their childhood years and/or as adults.

This chapter comprises a summary of what we currently know about children's physical activity level and what effects various types of physical activity can have during the childhood years and/or at an adult age. In conclusion, a summary is provided and the Swedish and international recommendations that currently exist for physical activity among children are presented. Evidence exists that the physical activity level of children can be influenced. This area is to some extent controversial and there is a lack of longitudinal studies.

Background

Physical activity is defined as "all types of physical movement done by the skeletal muscles that increases energy expenditure", in other words, virtually all the time that the child is not sleeping or completely still. The physical activity can be spontaneous, planned and/or organised. The spontaneous physical activity can be comprised of play or transportation. Our society demands less physical activity from people in connection with transportation, for example. Some places, where children previously played freely are judged to be dangerous in various ways. There is a concern that children and young people, just like adults, are becoming less physically active, which could lead to health problems as early as their childhood year and/or as adults. In this context, it can also be mentioned that Sweden is among the European countries (of a total of 18) that have the least guaranteed time dedicated to the school subject of sports and health per week.

How much do children and young people exercise today?

Surveys, pedometers (step counters), accelerometers, the doubly labelled water method and heart-rate monitoring are some methods used for activity registration among children (1). Validity, reproducibility and practical implementability are discussed and a combination of various methods is recommended. For example, surveys have the advantage of being able to be implemented in large groups, but the results should be interpreted with some caution, particularly among children under the age of 12 (2).

Within the scope of the European Youth Heart Study (EYHS), a total of 800 Swedish children (ages 9–10 and 15–16, respectively) were studied with accelerometers during the 1998–1999 school year (3). The “health-enhancing level” of physical activity was defined based on energy expenditure and was rooted in the adult recommendations current at the time (30–60 minutes of daily activity, corresponding to a brisk walk or more strenuous). For four days, all physical activity during the waking hours was registered. The group of 9–10 year-olds were active at a health-enhancing level an average of 200 minutes a day. Those achieving 60 minutes of such activity per day comprised 85% of the boys and 65% of the girls. A decreasing level of physical activity in the older group confirms data from several other studies. The girls were less active at a health-enhancing level than the boys in both age groups, which also agrees with previous studies conducted with various types of methodology. However, there was no gender difference in terms of sedentary time (not including night-time sleep), which on average was 284 minutes per day (younger group) and 432 minutes per day (older group). Differences in the physical activity level only explained the differences in maximal oxygen uptake (aerobic fitness) to a limited extent, which confirms several previous studies that these two variables do not always correlate to one another (4).

In another study (5, 6), where step-counters were used as a measure of the physical activity level, Swedish 7–14 year-olds ($n = 892$) and 15–18 year-olds ($n = 375$) were studied. There was a wide spread of the degree of physical activity in all age groups. The majority of the 7–14 year-olds achieved a number of steps (8,000 per day) that corresponds to slightly more than 30 minute’s of moderately strenuous activity/day. The degree of activity was lower among the 15–18 year-olds and this was especially clear among the boys.

Measurement of physical variables can be an indirect measure of how much children and young people exercise although it does not provide any detailed knowledge about the intensity. The maximal oxygen uptake has proven to have a correlation with the overall degree of physical activity among children and young people in some studies, but not in others (4). Consequently, it cannot be considered to always constitute a certain indirect measure of degree of physical activity (particularly if consideration is not made of the activity’s intensity). It is difficult to draw any conclusions as to whether or not Swedish children’s/adolescents’ aerobic fitness has diminished over the years, but there are indications of this (3, 7, 8).

In one study, where the physical capacity of Swedish 16-year-olds was compared in 1995 and 1974, an increased body weight but not height and a greater bone strength probably related to this was reported. However, a lower aerobic fitness (running test) and decreased arm strength (8) were also observed. The young people also completed a questionnaire where they said that they were more active in their free time and members of various sports associations to a greater extent than the young people of 1974. At the same time, 70 per cent of the young people said that they were physically active less than one hour a day at moderate intensity (9). In a study of a total of 2,000 Swedish children in grades 3, 6 and 9 in 2001 (“the school project”), a somewhat complex view of children’s performance capacity appeared (7). Here, a relatively wide spread is seen within the age groups. Clearly not all children are unfit and weak. However, there is a group of children of both genders and in all age groups that markedly distinguishes themselves from their contemporaries in a negative sense. A comparison was made with children studied in 1987 and it was found that strength and stamina in the upper body muscles had decreased and that aerobic fitness (maximal oxygen uptake) had decreased among the boys (the 16-year-olds were studied in this regard).

In a survey of children in 2001, the total time the children devoted to some form of physical activity was summed (10). A low level of activity was classified as such that “the child participates (and exercises extensively) during the school’s physical education (PE) lessons twice per week, and also pursues some sport once a week and cycles/walks a maximum of 10 minutes a day”. The level of exertion is not presented in detail. On average, many children in grades 6 and 9 said that they spent a somewhat extensive period of time pursuing physical activity. However, there was a high level of variation between the children in each age group. In grades 6 and 9, 2–4 children out of 10 reported a low or very low level of physical activity. In 2001, the Swedish National Institute of Public Health (SNIPH) and the Swedish National Agency for Education conducted a survey of 905 compulsory school and upper secondary school students from grade 8 up (11). In summary, 63 per cent indicated that they were physically active (defined as an activity causing one to become out of breath and sweaty, such as a fast walk) at least 30 minutes a day during a normal week. Those virtually completely physically inactive comprised 15 per cent (in other words, exercised less than two hours per week). In a study of 301 upper secondary school students in 1996, 26 per cent of the girls and 35 per cent of the boys said that they “seldom or never” performed any physical activity that made them sweaty and out of breath (12). The physical capacity was assessed through seven physical tests that were compiled into a physical index. The lowest physical index was in the young people in the practical programmes, especially the girls. This can be considered to be particularly serious since the young people who were training for professions with heavy physical loads had the lowest physical capacity (13).

There are few studies of the activity habits of preschool children. A Scottish study of 78 children (studied with accelerometers and the doubly labelled water method) showed that 5 year-olds were more active than 3 year-olds and that 5-year-old boys were more active than girls of the same age (14). Studies in the U.S. have shown that the degree of physical activity of 3–5 year-olds differs significantly between various day-care centres/preschools (15, 16). This was particularly clear for high intensity physical activity. The

physical activity at day-care centres comprised approximately 50 per cent of the children's total physical activity. In one of the U.S. studies, boys were more active at a high intensity than girls (16).

Studies have shown that children of physically active parents are more physically active than children of inactive parents (17). However, no certain correlation between the degree of physical activity during childhood and the degree of activity at an adult age has been shown (18, 19). Whether or not there is such a correlation for inactivity should be more closely investigated.

In summary, there are consequently indications that certain groups of Swedish children and young people are not physically active enough, which can at least partially explain decreases in physical activity. Girls are less active than boys and the degree of physical activity decreases from around the age of 11–12.

Effects of physical activity

Functional physical capacity, such as muscle strength and aerobic fitness, can be improved with strength and aerobic training, respectively. From a health perspective, the effects on bone mass, body fat and future risk factors (“risk indicators”) such as cardiovascular disease are important to observe. Effects on the growing individual's motor, cognitive, emotional and social development should also be observed.

Studies of effects of physical activity on growing individuals demand special considerations. A capacity increase can sometimes be more due to natural maturation and growth than to physical exercise. For children with the same chronological age (13 years for example), the “biological age” (measured with variables such as growth rate, skeletal maturity, puberty development) can differ by several years, which naturally affects factors such as muscle strength. Adequate control groups are important. “Dose-response” is often difficult to comment on, since most studies have only studied a selected dose of physical activity and cannot comment on whether a lower dose would suffice to achieve health effects.

Effects on aerobic fitness

Aerobic fitness is based on several building blocks, both the central circulatory capacity (maximal cardiac output/oxygen uptake) and local muscle endurance (adaptation). Altogether, studies show that both of these components develop with increasing age, but are already trainable during the growth years. Maturity appears to be of significance to the effect on the maximal oxygen uptake (greater effect in older children and adolescents), but it has not been established exactly how (20). The magnitude of the dose required at various ages has not been studied in detail, but in several studies an increase has been observed after strenuous to moderately strenuous activity (such as aerobics, football/soccer) 30–60 minutes at a time, at least three times a week (20).

Many of the studies done with regard to the effects of aerobic fitness training in children and adolescents are relatively difficult to assess. Has the intensity of the training programme been adequate? What was the maximal oxygen uptake when the study was initiated? Age? Level of maturity? Longitudinal or cross-sectional study? Control groups? Moreover, the effect of training has been evaluated in different ways, such as by measuring the maximal oxygen uptake, the reaction to submaximal exertion and in some cases muscle biopsies or heart volume, making the results difficult to compare (20). As previously mentioned, the maximal oxygen uptake cannot be considered to comprise a certain, indirect measure of the overall level of physical activity, especially if the exertion intensity is not monitored (see the section on “How much do children and young people exercise today?”).

Effects on muscle strength

Children are trainable and can increase their muscle strength by 14–30 per cent through training designed to train strength. The strength increase is considered to be an adaptation in the nervous system of the younger, prepubescent children where no effects on muscle mass could be seen. In addition to this, there is evidence of an increase in the muscle mass as a result of training during puberty. A training dose of two times per week appears to be adequate to achieve a strength increase. No heavy loads are needed; studies indicate that it is most effective to have a low load that is repeated many times (13–15 repetitions). Such training can be carried out with one’s own body as resistance such as in various games or with light weights. There is little risk of injury with adequate guidance/instruction. Today, there are no official Swedish recommendations with regard to strength training for children (21–23).

Effects on cognitive function

Motor development is important to the child’s overall maturity. In addition to the effects on healthy, normally developed children in terms of maturity, there is interest in motor exercise as a means to help children who have difficulties with language, perception, concentration and learning. Most children with considerable concentration difficulties have immature motor skills (24). In the “Bunkeflo project”, the effect of daily, scheduled physical activity (one hour per weekday) was studied in children in grades 1–3. Children who had immature or late development of motor skills were also offered one extra hour of motor skill training per week. The children in the intervention group had improved gross motor skills compared with the control group, which only participated in the school’s regular two PE lessons per week. Existing motor deficiencies did not subside on their own and the school’s two regular PE lessons were not sufficient to stimulate the motor development of these students. In addition, the children in the intervention group performed better in mathematics and Swedish than the children who only had the school’s regular two PE lessons per week. This was particularly

pronounced in the group of children assessed to have motor deficiencies that received extra motor skill training. Although the study can be criticised for not having been randomised, the results indicate interesting ties between physical activity, motor skill training and school performance.

For detailed reading, refer to Ericsson's intervention study concerning motor skills, concentration ability and school performance (25). There, three possible explanatory models are also described for the connection between motor skills/physical activity and cognition. The perceptual motor perspective focuses on the significance of the child's motor experiences to perceptual and sensory development, which is considered to be a prerequisite for cognitive processes. The neurophysiological explanation perspective is based on motor skill training/physical activity being able to entail changes in the nervous system's structure and function (such as neural connections, degree of alertness) that facilitate learning and memory processes. The psychological perspective is based on motor/physical activity inherently providing psychological changes (motivation, communication, social skills), which in turn facilitate learning.

In an article (26), Påske refers to a systematic analysis of the value of special, so-called perceptual training programmes (27): Sometimes, but not always, a modest, positive effect can be observed on the trained areas while "academic areas" (language, reading, mathematics) are unaffected by the training. The training has a moderate or no effect on the intellectual level of the normally gifted, but some (although moderate) effect on the mentally disabled in various stages. In the analysis, inclusion criteria, age distribution and frequency or the exact design of training were not presented. More well-described studies are desirable.

Effects on mood and mental health

In a Cochrane overview from 2004 (28), eight studies were included in a meta-analysis (children and young people ages 3–20). The results showed that regular physical activity had positive effects on self-esteem. The studies are heterogeneous. No certain conclusions can be drawn regarding the most effective type of physical activity, its intensity, duration or the context in which it was carried out. In a more recent Cochrane overview (29) that included 16 studies (children and young people up to age 20, but the majority ages 16 and up), the conclusion was drawn that regular physical activity has certain effects on both depression and anxiety, and that the intensity of the physical activity does not appear to have any significance. It is also noted that the studies have deficiencies and that there is particularly a need for more studies of children under age 16. Both studies in which physical activity was a part of a larger intervention and studies in which only physical activity was used were included in both of these overviews. Consequently, there are indications that regular physical activity has effects on self-esteem and the occurrence of problems of depression and anxiety, but it is too early to precisely describe the "dose".

Effects on the skeleton

High Bone Mineral Density (BMD) and skeletal girth reduce the risk of fractures in adults (30). Weight-bearing physical activity, such as callisthenics, running, tennis and ice hockey, can affect BMD and skeletal girth during the growth years. Peak bone mass is the highest amount of bone mass the individual stores in a lifetime. The greatest effects of physical training on the skeleton arise before puberty (30, 31). Since girls have an earlier puberty than boys (approximately two years earlier), training should be begun as early as age 7. The girls who did all-round physical education (PE) five days a week from 7 years of age had a higher bone mass at age 13 than those who had PE 1–2 times a week. In addition, data from this study indicates that continued PE maintained the higher bone mass all the way up to 18 years of age (32). In studies of former athletes, it is noted that the beneficial skeletal changes indeed remain for an extended period, but that they do not appear to still exist 30–60 years after the activity in question was finished. Continued activity at a lower level may possibly maintain the gain in BMD for at least approximately 15 years (30, 33). One Swedish study investigated whether or not the fracture risk later in life is lower in individuals with a high degree of physical activity during the adolescent years. Four hundred former male football and ice-hockey players (ages 60 and up, average age 71) and 800 age-matched controls were studied. The men in the former group, who had trained regularly between the ages of 13 to 36, indicated a lower number of fractures after age 35 than the men in the group that had not trained. Otherwise, the men indicated no differences in lifestyle when the study was carried out (34).

Girls intensely involved in sports who combine a high amount of exercise with insufficient nutritional intake and low body weight during the period up to peak bone mass can develop a delayed menarche or amenorrhea due to a disturbed hormonal balance and as a result of this also show various degrees of bone brittleness. This is one of many important reasons to pay attention to eating disorders in athletically active girls early on, including those who do not participate in organised sports (35). In this context, it should also be emphasized that insufficient energy intake relative to expenditure is not always due to eating disorders. It may be a matter of difficulties in combining long school days with frequent training sessions and adequate mealtimes.

Effects on obesity and risk factors for cardiovascular disease

Obesity and excess weight are becoming increasingly more common among Swedish children and young people (36) and increase the risk of developing cardiovascular disease at an adult age, regardless of one's adult weight (17). Obesity and excess weight are difficult to treat once established. Prevention is therefore of central importance in this context (36, 37). In weight-reduction programmes among already overweight children, dietary changes combined with increased physical activity provide better weight reduction results than dietary changes alone (38). Of course, the family should be involved in both the

prevention and treatment of excess weight. Correlations between physical activity and excess weight have been investigated with varying methodology and are difficult to assess on a whole (4, 17, 39, 40). In one of these studies (step-counter), the number of steps/day was linked to BMI in Swedish 6–12 year-olds and the interpretation of data was made such that a normal BMI required a minimum of 15,000 steps per day in boys and 12,000 steps per day in girls. (39).

Physical inactivity, such as watching television, is more clearly tied to excess weight in children (41). Whether or not this is a matter of selection, in other words that already overweight children choose inactivity or if watching television in itself causes excess weight, is somewhat unclear. “Screen time” of various types leaves less time left over for other activities, including spontaneous physical activity. Interestingly, a correlation between computer use and excess weight has not, however, been proven. The difference could possibly be due to watching television more often being associated with simultaneous energy intake, but this has not been proven in Sweden. Interventions in preschool/school have succeeded in reducing television watching (42, 43). In one of the studies (43), the reduction in the intervention group led to a lower BMI increase and less subcutaneous fat.

Atherosclerotic processes (causing fat deposits in the arteries) begin early in life (44). Excess weight during the growth years increases the risk of an accumulation of risk factors, which in adults has been shown to be of significance to the risk of developing cardiovascular disease (45). Such risk factors in children are usually called risk indicators and include, for example, increased blood pressure, hyperinsulinism (sign of lower insulin sensitivity) and elevated lipid levels. In the 9–10 year-olds and 15–16 year-olds studied in the European Youth Heart Study (EYHS), physical capacity (here, the maximal oxygen uptake) was more important than overall level of physical activity in preventing the occurrence of risk indicators for cardiovascular disease in these age groups (3). This indicates that physical activity of a moderate/high intensity may be important, because it is more likely to lead to a high aerobic capacity than activity of a lower intensity (see the section on aerobic fitness).

International studies indicate that a high level of physical activity during the growth years reduces the risk of developing cardiovascular disease at an adult age (46). Children/young people with a low aerobic capacity and other risk indicators for future cardiovascular disease have the most to gain from such training. One compilation (six prospective studies, only two of which included controls) has shown that aerobic training for 30 minutes three times a week for a minimum of three months can provide a blood pressure reduction in hypertensive children and young people 11–21 years of age (47). A training programme of 15 weeks, with an intensity of approximately 65 per cent of the maximal heart rate and an increasing duration from 20–45 minute per session and three times a week, was conducted on seven obese 13-year-old boys. The programme resulted in higher insulin sensitivity despite a maintained body weight and proportion of body fat (48). Effects of various training programmes on lipids in children are difficult to evaluate and no reliable conclusions can be drawn.

Are there risks of strenuous physical exercise during growth and maturation?

Discussions are currently ongoing with regard to whether or not very strenuous training without adequate recuperation can have negative effects on the growth and maturity of children and young people, including skeletal maturity and bone density, achieved final height and sexual maturity. Excessively strenuous training straining the back before the child is completely grown has also been called into question. Discussions concerning strenuous training are not included in this chapter. For more reading, refer to references 49 to 52 (49–52).

What do children and young people themselves say?

In the previously mentioned survey (53) covering 905 compulsory and upper secondary school students, some causes came forth as to why as much as 13–15 per cent of the students (15% of the girls in compulsory school!) seldom or never participated in physical education. This involved, for example, physical reasons, such as menstruation, problems with fainting and so on, but two thirds of the causes were of a psychological or social nature (boring, shy, disgusting). In addition, the young people's attitudes and evaluation of physical activity were investigated and they were asked what could get them to be more physically active in general. Here, several of the boys gave suggestions such that the school should support the students with access to the school's sports hall and more activities in the schoolyard during breaks. School teams were another suggestion – training buddies are important. The girls' motives for physical activity were most often items such as to feel good, become healthy, quit smoking and become thin faster. A varying content of the lessons in the subject of sports and health is noted as being important as well as support, encouragement and inspiration. It was mainly the upper secondary students who said that the economy could be crucial to the possibility of pursuing the form of physical activity that one wished. They also said that a lack of time due to studies can be an obstacle. The significance of cultural and ethnic factors to attitudes to physical activity can be investigated more closely.

Recommendations

Sweden supports the Nordic nutritional recommendations (54):

- A minimum of 60 minutes of physical activity is recommended every day. Both moderate and strenuous activity should be included.
- The activity can probably be divided up into several shorter sessions during the day.
- The activities should be as varied as possible to provide aerobic fitness, muscle strength, flexibility, speed, shorter reaction times and coordination.

This volume of physical activity agrees with international recommendations (55–58, 73). The U.S. recommendations from 2008 (73) recommend children and adolescents to do 60 minutes or more of physical activity daily. Most of the 60 or more minutes a day should be either moderate or vigorous aerobic physical activity, and should include vigorous physical activity at least three days a week. As a part of their 60 or more minutes of daily physical activity, children and adolescents should include muscle-strengthening and bone strengthening physical activity at least three days of the week. According to recent Finnish recommendations, children should be physically active for at least one to two hours per day. It is also emphasized that continued periods of sitting for more than two hours at a time should be avoided. Screen time with entertainment media should be limited to two hours per day (74).

Criticism has been expressed of currently making specific recommendations regarding physical activity in children and young people. As stated above, the available scientific data probably does not provide sufficient support to be able to specify an exact “dose” of physical activity that should provide all of the positive effects in all children and young people. The dose required to provide several positive effects later in adult life has also not been established and the existing level of physical activity is not known in various groups of children (59).

How can physical activity be promoted in children and young people?

- **Involve the family.** Children of physically active parents are more physically active than children of inactive parents (17).
- **Individualise the “prescription”.** It is desirable that there be several different alternatives to choose from. The choices/possibilities of following the recommendation are determined by individual preferences, gender, ethnicity, age, habits, family background, personality and so on. Contacts between sports/outdoor organisations and schools/healthcare are one possible solution (see “Idrottslyftet” or Sports Lift at www.rf.se for ideas).
- **Take the gender aspect into consideration.** Girls are generally less physically active than boys. The choice of activities can be affected by gender.
- **Increase the conditions for daily physical activity, such as in transportation.** Physically active “school commuting” can be of major significance to overall daily physical activity (60). “Walking school buses” mean that the parents arrange walking groups to school so that an adult always accompanies the children. Political efforts that affect the local environment so that it makes physically active transports possible, becomes attractive and encourages physical activity (61–63) are important here. The Government has charged the Swedish National Institute of Public Health to commence development efforts in this area (see www.fhi.se).
- **Pre-school/school-based interventions.** Utilise a combination of various methods. For ideas, see (63–65).

- **Healthcare-based measures.** There is a current lack of documented studies. Utilise a combination of various methods. For ideas see (64). Involving the family early on, such as in connection with health check-ups, may possibly be a way forward.

Children/young people and sports

In 2005, the Council of the Swedish Sports Confederation revised the sporting movement's conceptual programme, which is available in its entirety at www.rf.se (66). Some points concerning children and young people are briefly summarised below:

Children (ages 0–12)

- Sports should be playful, multifaceted and adjusted to the child's growth rate.
- Sports should be led by a leader with fundamental knowledge of children's physical, mental and social development.
- Children benefit from practicing several different sports and should have the right to do so in various associations.

Young people (ages 13–20)

General sports

- Consideration should be made of the individual's needs and circumstances.
- The transition between general sports and elite-focused sports should be gradual.
- The sporting movement should develop types of training and competition that meets the needs of boys and girls for general sports.

Elite sports

- Elite endeavour for those interested in doing so in socially safe ways.
- Competition activities should counteract exclusion.
- Leaders should be given the possibility of acquiring in-depth knowledge of physical, mental and social development.
- Ethical boundaries should be actively discussed (doping for example).

Special groups

Children with immature or late-development of motor skills

As yet, there are no official Swedish recommendations with regard to the identification and/or treatment of gross motor difficulties in children with immature or late development of motor skills without other anomalies (such as more or less visible motor disabilities). Proposals of measures in this area have been presented (25, 67).

Overweight and obese children

As previously mentioned, prevention is of central importance in this context. A combination of measures is necessary. An increase in the degree of physical activity is not enough on its own. A “decrease of physical inactivity” is a prescription that may be easier to follow than an “increase of physical activity” (68).

A guide in this context in terms of prevention comprises the national Swedish action plan for good eating habits and increased physical activity in the population that was prepared on behalf of the Government in 2005 (www.fhi.se). The objective in terms of physical activity in children is stated to be to:

- ”1. Increase the proportion of healthy children who are physically active for at least 60 minutes every day at a minimum of a moderate level , or a total of at least seven hours per week.
2. Reduce the proportion of children with a sedentary lifestyle.”

In more intense organised “training sessions” for obese children, consideration should be taken to the child’s current degree of excess weight. In a one-year programme, the Moderate Intensity Progressive Exercise Program (MPEP), the frequency, duration and intensity is gradually increased over time (every 10th to 15th week) (69). For an obese child, this can initially involve participating in one training session twice a week for 25 minutes with an intensity that is 50 per cent of the maximum oxygen uptake capacity. It should be emphasized that training programmes for obese and severely obese children should be administered in consultation with people knowledgeable in the area.

Children with asthma

The Swedish Paediatric Society’s section for children and youth allergology emphasizes the importance of encouraging children with asthmatic symptoms to participate in physical activity. Detailed advice for the prevention, investigation and treatment of exercise-induced asthma is provided on the website (70). Increased physical fitness may contribute to psychological well-being and improved asthma control. In addition to the children with an allergic asthma, there are also children who have asthma symptoms in connection with exercise. Children with these symptoms quickly adapt and tend to avoid physical exertion. With greater knowledge and guidance, these children can be helped to break this pattern. Good basic medication is important. Symptoms are easily triggered by running, less so by cycling and least by swimming. Children who already have been diagnosed with asthma, who try to participate but cannot do as much as others and have coughing attacks upon exertion are recommended to consult a school nurse or doctor for evaluation. For known asthma, the following are recommended:

- A. If preventive bronchial dilation medication is prescribed, it should be taken approximately 15 minutes before the sports lesson/training session.
- B. Warming up is important. Let the participants warm up slowly for 10–15 minutes so that the pulse is gradually increased.

- C. The actual training session, or parts of it, may preferably consist of so-called interval training, in other words short (a few minutes) intense sessions interspersed with less strenuous exercises. This can then be followed by more continuously strenuous exercises as long as symptoms do not appear.
- D. Winding down is also important. Conclude the session over approximately 5–10 with exercises that slowly lower the pulse.
- E. If the boy/girl has symptoms, make sure that he/she winds down properly.
- F. For more severe symptoms, it is important as under E that the bronchial dilation medicine be taken and that he/she can rest, preferably sitting with the arms resting on the knees. If the symptoms do not subside within a few minutes, see to it that he/she is given a new medicine dose and that medical help is summoned.

For additional recommendations regarding physical exercise see (70).

Children with diabetes mellitus type 1

It is desirable to create conditions for better and safer physical activity for children/young people with diabetes. In an updated care programme for diabetes, which according to the plans should be presented in 2007, a special section on sports and diabetes will be included (Peter Adolfsson, Paediatrician, Queen Silvia Children's Hospital, Personal Communication).

Have methods of promoting physical activity been evaluated?

A systematic review of the scientific literature on various methods to promote physical activity was published by the Swedish Council on Technology Assessment in Health Care (SBU) in 2007 (64). The review included 24 studies of children and young people, of which 21 were school-based and three were healthcare-based. Eleven of the studies were considered to have sufficient scientific evidence to form the basis of the conclusions regarding children and young people:

1. The development of the school subject of sports and health, such as through greater investment in health education, educational materials and teacher training, leads to 5–25 per cent more physical activity during sports classes. This applies to boys to a higher extent than to girls (strong scientific evidence).
2. School-based interventions that comprise multiple components, such as teacher training, changes in curricula, extra activity sessions during class time and/or breaks, support in behavioural change, strengthened health education and the involvement of parents, have a positive effect on the physical activity of children and young people during the school day and in some cases also during free time (moderately strong scientific evidence).

3. School-based interventions directed at groups with an elevated risk of cardiovascular disease lead to approximately 10 per cent more physical activity (limited scientific evidence).”

For details and references, refer to (64) where the entire report is available. One can also refer to a compilation of systematic overviews concerning school-based methods to promote health and prevent disease in children and young people, which shows that school-based methods can be effective, particularly to promote mental health, good eating habits and greater physical activity (71). According to the Swedish curriculum, “schools should strive to offer all students daily physical activity in the scope of the entire school day” (72).

Accordingly, it was confirmed that children’s physical activity level can be affected and that there is a need for more longitudinal studies in the area, particularly with regard to healthcare-based interventions.

Conclusion

There is scientific evidence of several positive effects of physical activity among children and young people. It is also possible to influence the physical activity level in children. There is a need for more studies in the area. Feel free to contact the author to report non-published interventions/experiences (ulrika.berg@ki.se).

Acknowledgement

Thanks to Ingunn Fjørtoft, M.D., Telemark University College, Notodden, Norway, for constructive observations and advice.

References

1. Sirard JR, Pate RR. Physical activity assessment in children and adolescents. *Sports medicine (Auckland, NZ)* 2001;31:439-54.
2. Brettschneider W, Naul R. Study on young people's lifestyles and sedentariness and the role of sport in the context of education and as a means of restoring the balance. Final report. Paderborn: University of Paderborn; 2004.
3. Hurtig Wennlöf A. Cardiovascular risk factors in children. Stockholm: Karolinska Institutet; 2005.
4. Ekblom . Physical fitness and overweight in Swedish youths. Dissertation. Stockholm: Karolinska Institute and Idrottshögskolan; 2005.
5. Raustorp A, Mattsson E, Svensson K, Stahle A. Physical activity, body composition and physical self-esteem. A 3-year follow-up study among adolescents in Sweden. *Scandinavian Journal of Medicine & Science in Sports* 2006;16:258-66.
6. Raustorp A, Pangrazi RP, Stahle A. Physical activity level and body mass index among schoolchildren in south-eastern Sweden. *Acta Paediatr* 2004;93:400-4.
7. Ekblom O, Oddsson K, Ekblom B. Health-related fitness in Swedish adolescents between 1987 and 2001. *Acta Paediatr* 2004;93:681-6.
8. Westerstahl M, Barnekow-Bergkvist M, Hedberg G, Jansson E. Secular trends in body dimensions and physical fitness among adolescents in Sweden from 1974 to 1995. *Scandinavian Journal of Medicine & Science in Sports* 2003;13:128-37.
9. Westerstahl M, Barnekow-Bergkvist M, Hedberg G, Jansson E. Secular trends in sports. Participation and attitudes among adolescents in Sweden from 1974 to 1995. *Acta Paediatr* 2003;92:602-9.
10. Engström L. Hur fysiskt aktiva är barn och ungdomar? [How physically active are children and adolescents?] *Svensk Idrottsforskning* 2002;11.
11. Strandell A, Bergendahl L, Kallings L. Sätt Sverige i rörelse 2001. Förskolan/skolan. [Get Sweden moving 2001. Preschool/school.] Report 2002;10. Stockholm: Swedish National Institute of Public Health; 2002.
12. Sollerhed AC, Ejlertsson G. Low physical capacity among adolescents in practical education. *Scandinavian Journal of Medicine & Science in Sports* 1999;9:249-56.
13. Sollerhed AC. Young today – adult tomorrow! Studies on physical status, physical activity, attitudes, and self-perception in children and adolescents. Malmö: Lund University; 2006.
14. Reilly JJ, Jackson DM, Montgomery C, Kelly LA, Slater C, Grant S, et al. Total energy expenditure and physical activity in young Scottish children. Mixed longitudinal study. *Lancet* 2004;363:211-2.
15. Pate RR, Pfeiffer KA, Trost SG, Ziegler P, Dowda M. Physical activity among children attending preschools. *Pediatrics* 2004;114:1258-63.
16. Finn K, Johannsen N, Specker B. Factors associated with physical activity in preschool children. *The Journal of Pediatrics* 2002;140:81-5.
17. Steinbeck KS. The importance of physical activity in the prevention of overweight and obesity in childhood. A review and an opinion. *Obes Rev* 2001;2:117-30.

18. Twisk JW, Kemper HC, van Mechelen W. The relationship between physical fitness and physical activity during adolescence and cardiovascular disease risk factors at adult age. The Amsterdam Growth and Health Longitudinal Study. *International Journal of Sports Medicine* 2002;23 Suppl 1:pp.8-14.
19. McMurray RG, Harrell JS, Bangdiwala SI, Hu J. Tracking of physical activity and aerobic power from childhood through adolescence. *Med Sci Sports Exerc* 2003;35:1914-22.
20. Mahon A. Exercise training. In: Armstrong N, Van Mechelen W (eds.). *Pediatric exercise and science in medicine*. 1. edn. New York: Oxford University Press; 2000.
21. Augustsson J, Wernbom M. Muskelstyrkeutveckling hos barn och ungdomar. [Muscle strength development in children and adolescents.] *Svensk Idrottsforskning* 2007;1:44-47.
22. Tonkonogi M. Styrketräning för barn – bu eller bä? [Strength training for children - good or bad?] *Svensk Idrottsforskning* 2007;1:38-43.
23. Malina RM. Weight training in youth-growth, maturation, and safety. An evidence-based review. *Clin J Sport Med* 2006;16:478-87.
24. Kadesjö B. Barn med koncentrationssvårigheter. [Children with concentration difficulties.] Stockholm: Liber utbildning AB; 1992.
25. Ericsson I. Motorik, koncentrationsförmåga och skolprestationer. En interventionsstudie i skolår 1–3. [Motor skills, concentration ability and school performance. An intervention study in school years 1-3.] Malmö: Malmö University; 2003.
26. Påske W. Motorik, perception och inläring. [Motor skills, perception and learning.] *Tidskrift i gymnastik och idrott* 1989;116: 15-24.
27. Kavale K, Mattson P. "One jumped off the balance beam". Meta-analysis of perceptual-motor training. *Journ of Learn Disabil* 1983;16:165-73.
28. Ekeland E, Heian F, Hagen KB, Abbott J, Nordheim L. Exercise to improve self-esteem in children and young people. *The Cochrane Library* 2005;2. (Downlade 22 Oct. 2007: www.thecochranelibrary.com).
29. Larun L, Nordheim LV, Ekeland E, Hagen KB, Heian F. Exercise in prevention and treatment of anxiety and depression among children and young people. *The Cochrane Library* 2007; 2. (Downlade 22 Oct. 2007: www.thecochranelibrary.com).
30. Karlsson M. Fysisk träning under tillväxtåren ökar benmassan. [Physical training during the growth years increases bone mass.] *Läkartidningen* 2002;99:3400-5.
31. Karlsson KM, Stenevi-Lundgren H, Linden C, Gärdsell P. Daglig gymnastik stärker skellet. [Daily callisthenics strengthen the skeleton.] *Läkartidningen* 2006;103:2979-80.
32. Valdimarsson O, Sigurdsson G, Steingrimsdottir L, Karlsson MK. Physical activity in the post-pubertal period is associated with maintenance of pre-pubertal high bone density. A 5-year follow-up. *Scandinavian Journal of Medicine & Science in Sports* 2005;15:280-6.
33. Nordstrom A, Olsson T, Nordstrom P. Sustained benefits from previous physical activity on bone mineral density in males. *The Journal of Clinical Endocrinology and Metabolism* 2006;91:2600-4.

34. Nordstrom A, Karlsson C, Nyquist F, Olsson T, Nordstrom P, Karlsson M. Bone loss and fracture risk after reduced physical activity. *J Bone Miner Res* 2005;20:202-7.
35. Carlsson C. Ätstörningar. En kunskapsöversikt. FoU-rapport. [Eating disorders. A knowledge overview. R&D report.] Stockholm: Swedish Sports Confederation 2004;1. (Downloaded 22 Oct. 2007 www.rf.se).
36. J Perlhagen, Flodmark CE, Hernell O. Fetma hos barn. Prevention enda realistiska lösningen på problemet. [Obesity in children. Prevention only realistic solution to the problem.] *Läkartidningen* 2007;104:138-41.
37. SBU (Swedish Council on Technology Assessment in Health Care). Förebyggande åtgärder mot fetma. En systematisk litteraturoversikt. [Preventative measures against obesity. A systematic literature review.] Stockholm: SBU; 2004.
38. Epstein LH, Wing R, Koeske R, Vaoski A. A comparison of lifestyle exercise, aerobic exercise and calisthenics on weight loss in obese children. *Behav Ther* 1985;16:345-56.
39. Tudor-Locke C, Pangrazi RP, Corbin CB, Rutherford WJ, Vincent SD, Raustorp A, et al. BMI-referenced standards for recommended pedometer-determined steps/day in children. *Prev Med* 2004;38:857-64.
40. Ekelund U, Aman J, Yngve A, Renman C, Westerterp K, Sjostrom M. Physical activity but not energy expenditure is reduced in obese adolescents. A case-control study. *Am J Clin Nutr* 2002;76:935-41.
41. Rydell A, Brennerberg S. TV-konsumtion och barns hälsa och anpassning. [TV consumption and children's health and adaptation.] R 2004:24. Stockholm: Swedish National Institute of Public Health; 2004.
42. Dennison BA, Russo TJ, Burdick PA, Jenkins PL. An intervention to reduce television viewing by preschool children. *Arch Pediatr Adolesc Med* 2004;158:170-6.
43. Robinson TN. Reducing children's television viewing to prevent obesity. A randomized controlled trial. *JAMA* 1999;282:1561-7.
44. Berenson GS, Srinivasan SR, Bao W, Newman WP 3rd, Tracy RE, Wattigney WA. Association between multiple cardiovascular risk factors and atherosclerosis in children and young adults. The Bogalusa Heart Study. *The New England Journal of Medicine* 1998;338:1650-6.
45. Williams CL, Hayman LL, Daniels SR, Robinson TN, Steinberger J, Paridon S, et al. Cardiovascular health in childhood. A statement for health professionals from the Committee on Atherosclerosis, Hypertension, and Obesity in the Young (AHOY) of the Council on Cardiovascular Disease in the Young, American Heart Association. *Circulation* 2002;106:143-60.
46. Eisenmann JC. Physical activity and cardiovascular disease risk factors in children and adolescents. An overview. *The Canadian Journal of Cardiology* 2004;20:295-301.
47. Alpert B, Wilmore J. Physical activity and blood pressure in adolescents. Physical activity guidelines for adolescents. *Pediatric Exercise Science* 1994;6:361-80.
48. Kahle EB, Zipf WB, Lamb DR, Horswill CA, Ward KM. Association between mild, routine exercise and improved insulin dynamics and glucose control in obese adolescents. *International Journal of Sports Medicine* 1996;17:1-6.

49. Lundin O, Swärd L. Ryggens disk – elitidrottarens achilleshäla? [Disks of the back - the elite athlete's Achilles's heel?] *Svensk Idrottsforskning* 1999;3:15-7.
50. Malina RM. Physical growth and biological maturation of young athletes. *Exerc Sport Sci Rev* 1994;22:389-433.
51. Caine D, Lewis R, O'Connor P, Howe W, Bass S. Does gymnastics training inhibit growth of females? *Clin J Sport Med* 2001;11:260-70.
52. Lundin O. Ryggproblem vid elitträning i unga år. [Back problems in elite training at a young age.] *Svensk Idrottsforskning* 2007;1:51-3.
53. Strandell A, Bergendahl L, Kallings L. Sätt Sverige i rörelse 2001. Förskolan/skolan. [Get Sweden moving 2001. Preschool/school.] Report 2002;10. Stockholm: Swedish National Institute of Public Health; 2002.
54. NNR. Nordic Nutrition Recommendations. Integrating nutrition and physical activity. 4. edn. Report 2004:013. Copenhagen: Nordic Council; 2004.
55. American Alliance for Health PE, Recreation and Dance. Physical activity for children. A statement of guidelines. American Alliance for Health PE, Recreation and Dance (AAHPERD) Publications; 1998.
56. Cavill N, Biddle S, Sallis J. Health-enhancing physical activity for young people. Statement of the United Kingdom Expert Consensus Conference. *Pediatric Exercise Science* 2001;13:12-25.
57. WHO [World Health Organization] Annual global "Move for health" initiative. A concept paper. Geneva: World Health Organization; 2003.
58. Strong WB, Malina RM, Blimkie CJ, Daniels SR, Dishman RK, Gutin B, et al. Evidence based physical activity for school-age youth. *The Journal of Pediatrics* 2005;146:732-7.
59. Twisk JW. Physical activity guidelines for children and adolescents. A critical review. *Sports Medicine (Auckland, NZ)* 2001;31:617-27.
60. Dang P, Lundwall S, Engstrom L-M, Schantz P. Tiden talar för fysiskt aktiv skolpendling. [Time supports physically active school commuting.] *Svensk Idrottsforskning* 2006;3:14-5.
61. Giles-Corti B, Broomhall MH, Knuiaman M, Collins C, Douglas K, Ng K, et al. Increasing walking. How important is distance to, attractiveness, and size of public open space? *Am J Prev Med* 2005;28(2 Suppl 2):169-76.
62. Hume C, Salmon J, Ball K. Associations of children's perceived neighborhood environments with walking and physical activity. *Am J Health Promot* 2007;21:201-7.
63. Fjortoft I. The natural environment as a playground for children. The impact of outdoor play activities in pre-primary school children. *Early Childhood Education Journal* 2000;29:111-7.
64. SBU (Swedish Council on Technology Assessment in Health Care). Metoder för att främja fysisk aktivitet. [Methods of promoting physical activity.] Stockholm: SBU; 2007.
65. Fjortoft I. Landscape as playscape. Learning effects from playing in natural environment on motor development in children. Dissertation. Oslo: Norwegian University of Sport and Physical Education; 2000.

66. Swedish Sports Confederation. Idrotten vill. [Sports want to.] Stockholm: Swedish Sports Confederation; 2005.
67. Ericsson I. Rör dig – lär dig. Motorik och inläring. [Exercise - learn. Motor skills and learning.] Stockholm: SISU Idrottsböcker; 2005.
68. Thompson NS, Smolak L. Body image, eating disorders and obesity in youth. Assessment, prevention and treatment. Washington: American Psychological Association; 2001.
69. Sothorn MS. Exercise as a modality in the treatment of childhood obesity. *Pediatr Clin North Am* 2001;48:995-1015.
70. Swedish Paediatric Society. Ansträngningsutlöst astma. Utredning och behandling [Exertion-triggered asthma. Examination and treatment.]; 2006.
71. Stewart-Brown S. What is the evidence on school health promotion in improving health or preventing disease and, specifically, what is the effectiveness of the health promoting school approach? Copenhagen: WHO Regional Office for Europe's Health Evidence Network (HEN); 2006.
72. Swedish National Agency for Education. Läroplan för det obligatoriska skolväsendet, förskoleklassen och fritidshemmet Lpo 94. [Curriculum for compulsory schools, preschools and recreation centres Lpo 94.] 2006:23. Stockholm: Swedish National Agency for Education; 1994.

12. Pregnancy

Authors

Ann Josefsson, MD, PhD, Department of Obstetrics and Gynaecology, Linköping University Hospital, Linköping, Sweden

Kari Bø, PT, PhD, Professor, Norwegian School of Sports Sciences, Department of Sports Medicine, Oslo, Norway

Summary

In the majority of cases, physical activity during pregnancy is safe for mother and foetus and entails no elevated risk of abnormal pregnancy or delivery outcomes. All pregnant women should therefore be recommended to participate in condition-enhancing exercise as a part of a healthy lifestyle. Regular exercise also improves and maintains good general fitness during pregnancy and can be a good way of preparing the body for the actual delivery. Physical activity should take place at a moderate level of exertion during a total of approximately 30 minutes per day. Activities should be chosen such that they entail a minimal risk of falling and damage to the foetus. From a public health perspective, one of the advantages of women exercising regularly during pregnancy is that they are most often more inclined to continue with some form of physical activity after they have given birth as well.

Physiological changes during pregnancy

During pregnancy, the need for oxygen increases, which entails increased respiratory depth and an increased respiratory rate. The blood volume increases by approximately two litres, which leads to a higher heart rate and greater stroke volume. The resting heart rate increases by approximately 10–15 beats per minute, although with large individual differences. The heart's capacity is affected by the pregnant woman's position. From around the fourth month of pregnancy, the venous reflux is obstructed by the growing uterus in the supine position, called vena cava compression. This leads to an elevated risk of a reduced stroke volume and a drop in blood pressure, which is why physical exercise lying on the back should be avoided after the 16th week of pregnancy. The same is true of exercises while standing still for prolonged periods, which can create the same effect (1).

Hormonal changes caused by pregnancy entail increased flexibility in the joints. The pregnancy-related weight gain leads to the skeleton, muscles, joints and ligaments being placed under greater strain. At the same time, there is a forward shift in the body's centre of balance, the back muscles are strained more, which creates a higher compression on the rear segments of the lumbar region. The curve of the back increases and, consequently, it becomes more difficult to maintain one's balance.

In pace with the growth of the belly, the distance between the rectus abdominis muscles may also increase, called diastasis recti. It is assumed that diastasis recti combined with the altered centre of balance forward can lead to reduced torso stability.

Both the pregnancy in itself and physical activity increase the metabolism and raise the body temperature. A body temperature of more than 39.2 degrees Celsius is believed to be teratogenic (in other words, can cause foetal damage) during the first three months. However, a higher respiratory rate and higher skin perfusion help to reduce the risk of an abnormally high body temperature (hyperthermia). However, a good fluid supply and avoiding physical exercise in a hot and humid climate are important. The risk of low blood sugar (hypoglycaemia) can be avoided with an adequate caloric intake and by limiting the exercise session's length to a maximum of 45 minutes.

Back and pelvic pain

The altered posture during pregnancy, with a greater lumbar lordosis (back curvature) and thoracic kyphosis (backwards arching of the upper spine), is considered to be one of the reasons that back problems can arise. The prevalence of back and pelvic related pain during pregnancy is approximately 45 per cent (2).

Urinary incontinence

Pregnancy and, above all, delivery are risk factors for the development of urinary incontinence with potential damage to the muscles, connective tissues and peripheral nerves (3). More frequent emptying of the bladder and pressure urges are common during pregnancy due to the pressure of the foetus. The incidence of incontinence during pregnancy varies between 32 and 64 per cent (3).

Obesity

Throughout the Western World, there is a rapidly increasing proportion of pregnant women with excess weight and obesity. Today, nearly 40 per cent of pregnant women are overweight (BMI > 25) and approximately 10–12 per cent are obese (BMI > 30) upon registration with maternity care in Sweden. Excess weight and mainly obesity in the mother are associated with elevated risks of serious complications during both pregnancy and birth, which is why this is a problem that must be taken very seriously.

Effects of physical activity

Besides a retained or increased physical well-being, the benefits of physical activity for pregnant women include less fatigue and a reduced risk of swelling in the extremities and varicose veins. In addition, physically active pregnant women often experience less stress, anxiety, depression and sleeping disorders. It has been shown that adverse pregnancy and neonatal outcomes are not increased in women who have exercised during pregnancy (4–5). Some data indicates that regular physical exercise during pregnancy decreases the total time the birth takes and reduces the risk of obstetric complications.

Preeclampsia/pregnancy toxemia

Regular physical activity among those not pregnant has been shown to be able to reduce the risk of hypertension. Consequently, in two randomised studies, the effect of regular exercise of moderate intensity was studied in relation to the risk of developing preeclampsia. The results indicate that a reduction of risk can be achieved, although further studies are needed for clear evidence (6).

Gestational diabetes

In a Cochrane overview comprising four randomised studies involving 114 pregnant women with gestational diabetes, the effect of physical activity was studied during months 6–9 of pregnancy with regard to blood sugar levels, decreased insulin requirements and a potential decrease in perinatal and maternal morbidity. No differences were found between the groups and the authors are of the opinion that larger studies are needed to be able to answer the question of whether physical activity leads to an improvement in terms of diabetes (7).

Obesity

The effect of regular physical activity among obese pregnant women is sparsely studied. In one Swedish case-control study, it was found that obese pregnant women who participated in a programme with motivational talks and aquarobics 1–2 times a week had a smaller weight gain during pregnancy than obese pregnant women who received customary maternal healthcare (8).

Back and/or pelvic pain

In a randomised study by Kihlstrand et al (9), it was found that women with back pain during months 3–6 of the pregnancy who were offered aquarobics achieved a significant reduction in both pain and sick listing during the remainder of the pregnancy. In another randomised study, pregnant women were offered aerobic exercise for 60 minutes three times a week during 12 weeks from month 3–6 of the pregnancy. It was found that the

exercise group had significantly less back pain and restricted mobility compared with the control group (10).

Elden et al (11) showed that a reduction of pelvic pain could be achieved both in a group of pregnant women who received acupuncture and in a group that received stabilising exercise compared with pregnant control subjects. In a primary prevention study among first-time pregnant women, Mørkved et al (12) found significantly fewer pregnant women with pelvic related pain among the women who were randomised to a group training programme consisting of abdominal, back and pelvic floor training and relaxation compared with the controls. In summary, in a Cochrane overview it has been concluded that pregnancy-specific exercise programmes, stabilising exercises, acupuncture and aquarobics appear to reduce back and pelvic pain during pregnancy compared with customary maternity healthcare and obstetric monitoring (13), but that more and more well designed studies are needed. Studies that aim to clarify whether or not physical activity begun early in pregnancy can prevent back and/or pelvic pain during pregnancy would be particularly valuable.

Urinary incontinence

Four randomised controlled studies have investigated the effect of pelvic floor exercise during pregnancy as a method to prevent/reduce incontinence. Three of these show a significant effect from exercise, while one study could not prove any such effect (the latter study is published as a summary and consisted only of a consultation with a physiotherapist, however). The three studies that showed a positive correlation included first-time pregnant women. Sampsel et al (14) found that those who did pelvic floor exercises had significantly fewer symptoms than those who had not exercised in week 35, 6 weeks and 6 months after the birth, but that there were no differences between the groups after 12 months. However, the study had a high dropout rate. Reilly et al (15) found that women who exercised during pregnancy reduced the prevalence of incontinence three months after birth compared with control groups (19 or 33% with urinary incontinence). Mørkved et al (16) found significantly fewer women with urinary incontinence in the exercise group both at week 36 and three months after the birth. The muscle strength in the pelvic floor muscles was significantly higher at both measurement occasions in the exercise group. Several randomised controlled studies have shown an effect from pelvic floor exercises after the birth (17). The effect appears to be better with more frequent follow-up and more intense exercise.

Counselling on physical activity during pregnancy

Every woman is unique and an individual assessment is needed based on the current condition status and the type of physical activity, intensity, duration and frequency. A reasonable objective should be to strive for retained fitness during pregnancy, but not to strive for peak performance. Every exercise session should include a warm-up and a wind-down

phase (1, 18). Pregnant women who were not previously physically active should gradually begin with a short exercise session three times a week. Then, a gradual increase can be made to 30–45 minutes, three times a week, supplemented with virtually daily physical activity for 30 minutes in total.

Aerobic exercise

With regard to aerobic exercise, normal recommendations can be followed as long as one avoids excessively high body temperature (see above) in the first three months. Pregnant women should exercise in light clothing and remember to drink fluids during and after exercising. Activities of a high intensity in a warm and humid climate should be avoided (18). All activities that include rhythmic and dynamic exertion with major muscle groups are recommended, such as brisk walks, Nordic walking, jogging, cycling, aerobics, step-up training, callisthenics and dance. Swimming is also an excellent activity. Aquarobics is suitable for women with pelvic and back problems.

Since the heart rate is elevated during pregnancy, the pulse level is not always a good way of measuring the intensity of an exercise session. Instead, the recommendation is to assess the physical strain with either a so-called talk test or with Borg's Rating of Perceived Exertion (RPE) scale. A talk test is based on the exercise session's intensity not being greater than what permits normal conversation. Borg's scale measures subjective exertion between 6 and 20. The recommended level during pregnancy is 12–14, in other words somewhat hard (19).

Strength training

Strength training during pregnancy should focus on the pelvic floor muscles and the back and abdominal muscles, but training of the lower and upper extremities can advantageously be included. Since there is a risk of vena cava compression, in other words that the venous reflux to the heart is obstructed by the growing uterus, which can lead to an elevated risk of a reduced stroke volume and a drop in blood pressure, it is recommended that strength exercises for the abdomen be done sitting, lying on one's side or standing after the 16th week of pregnancy (20). In general, 7–8 exercises are recommended for the body's most important muscle groups, with 8–12 repetitions in three sets (19). Pregnant women should avoid such high loads that the strain reflex arises. Many have problems to "find" the pelvic floor muscles when exercising and in such a case it may be appropriate to contact a physiotherapist to check that the exercises are done correctly before the birth. After the birth, it may be even more difficult depending on what damage has been done to the muscles, supportive tissues and potential nerve damage. Consequently, it is always an advantage to have learned the technique ahead of time. For beginners, it is also important to learn the right technique for the general strength exercises. The physiotherapist can provide advice on both technique and the scope of the exercises.

Flexibility training

Due to generally increased flexibility during pregnancy, it is important to ensure that stretching and extension exercises are done with a certain degree of caution, so that imbalances do not arise. The muscle groups that have been used in the aerobic and strength training should be extended/stretched, but specific flexibility exercises are not necessary. In general, stretching and flexibility training aims to retain normal joint flexibility. The exercises should be done at a relaxed pace and the extended position should be maintained for 10–30 seconds. The training should be done at least 2–3 times a week (19, 20). Flexibility training for the untrained can advantageously be done under the guidance of an instructor.

Functional tests/need for health check-ups

The following conditions require a professional medical assessment and consultation regarding whether or not physical exercise is appropriate during pregnancy, the type of exercise, the load and the extent of the training (4):

- Heart disease
- Undiagnosed cardiac arrhythmia in the mother
- Restrictive lung disease
- Chronic bronchitis
- Poorly controlled hypertension, thyroid disease, diabetes mellitus or epilepsy
- Anaemia
- Bleeding in month 4–9
- Preeclampsia or pregnancy-induced hypertension
- Preterm labour
- Intrauterine growth retardation
- Cervical weakness/cerlage
- Preterm prelabour rupture of membranes
- Twin pregnancy
- Smoking > 20 cigarettes/day
- Orthopaedic disease that limits motor capacity
- Morbid obesity (BMI > 40)
- Malnutrition or eating disorders.

Warning signals when physical activity should be stopped and the pregnant woman should contact women's healthcare for a medical consultation (4):

- Pronounced breathlessness
- Breathlessness before exercising
- Pronounced fatigue
- Headaches

- Chest pains/pressure on the chest
- Dizziness
- Pronounced abdominal or pelvic pain
- Painful contractions or preterm labour
- Leakage of amniotic fluid
- Vaginal bleeding
- Reduced foetal movement
- Muscle weakness
- Swelling or pain in the calves.

Contraindications

Pregnant women should not go diving since the foetus is not protected from decompression sickness and gas embolism. Contact sports should be avoided from month 4–6.

Risks

Pregnant women who pursue sports with a risk of falling, such as downhill skiing, skating, ice hockey, apparatus gymnastics or riding, should be made aware of degraded balance and the risk of foetal injury in a potential fall.

Physical exercise at a high altitude (> 2,500 metres) may not be common among pregnant women, but has been shown to entail a redirection of blood from the placenta to the muscles. Theoretically, this can entail a risk that the foetus will receive too little oxygen. Therefore, at least 4–5 days of acclimation is needed to reset the metabolism.

Competition sports during pregnancy require careful joint assessment by the obstetrician in charge and the sports physician in charge. Regular check-ups during pregnancy are recommended as well as possible ultrasound check-ups concerning the foetus' growth. It is particularly important to ensure that there is an adequate fluid and nutritional intake and the risk of an elevated body temperature be avoided. Competitive athletes should also be informed that the pregnancy will entail diminished physical performance capacity.

References

1. Artal R, O'Toole M. Guidelines of the American College of Obstetricians and Gynecologists for exercise during pregnancy and the postpartum period. *Br J Sports Med* 2003;37:6-12.
2. Wu WH, Meijer OG, Uegaki K, Mens JMA, van Dieen JH, Wuisman PIJM, et al. Pregnancy-related pelvic girdle pain (PPP)! Terminology, clinical presentation, and prevalence. *Eur Spine J* 2004;13:575-89.
3. Hunkskaar S, Burgio K, Clark A, Lapitan MC, Nelson R, Sillen U, et al. Epidemiology of urinary (UI) and faecal (FI) incontinence and pelvic organ prolapse (POP). In: Abrams P, Cardozo L, Khoury S, Wein A, eds. *Incontinence. Vol 1. Basic and evaluation*. Plymouth (UK): Health Publication Ltd; 2005. Chapter 5, pp. 255-312.
4. Royal College of Obstetricians and Gynaecologists. *Exercise in pregnancy. Statement 2006 January*;4.
5. Kramer MS, McDonald SW. Aerobic exercise for women during pregnancy. *Cochrane Database of Systematic Reviews* 2006;3.
6. Meher S, Duley L. Exercise or other physical activity for preventing pre-eclampsia and its complications. *Cochrane Database of Systematic Reviews* 2006;2.
7. Ceysens G, Rouiller D, Boulvain M. Exercise for diabetic pregnant women. *Cochrane Database of Systematic Reviews* 2006;3.
8. Claesson I-M, Sydsjö G, Brynhildsen J, Cedergren M, Jeppsson A, Nyström F, et al. Weight gain restriction for obese pregnant women. A case-control intervention study. *BJOG* 2008;115:44-50.
9. Kihlstrand M, Stenman B, Nilsson S, Axelsson O. Water-gymnastics reduced the intensity of back/low back pain in pregnant women. *Acta Obstet Gynecol Scand* 1999;78:180-5.
10. Garshasbi A, Zadeh SF. The effect of exercise on the intensity of low back pain in pregnant women. *Int J Gynecol Obstet* 2005;88:271-5.
11. Elden H, Ladfors L, Olsen MF, Ostgaard HC, Hagberg H. Effects of acupuncture and stabilising exercises as adjunct to standard treatment in pregnant women with pelvic girdle pain. Randomised single blind controlled trial. *BMJ* 2005;331:249-50.
12. Mørkved S, Salvesen KÅ, Schei B, Lydersen S, Bø K. Does group training during pregnancy prevent lumbopelvic pain? A randomized clinical trial. *Acta Obstet Gynecol* 2007;86:276-82.
13. Pennick VE, Young G. Interventions for preventing and treating pelvic and back pain in pregnancy. *Cochrane Database of Systematic Reviews* 2007;2.
14. Sampsel CM, Miller JM, Mims BL, DeLancey JOL, Ashton-Miller J, Antonakos C. Effect of pelvic muscle exercise on transient incontinence during pregnancy and after birth. *Obstet Gynecol* 1998;91:406-12.
15. Reilly ETC, Freeman RM, Waterfield MR, Waterfield AE, Steggles P, Pedlar F. Prevention of postpartum stress incontinence in primigravidae with increased bladder neck mobility. A randomised controlled trial of antenatal pelvic floor exercises. *BJOG* 2002;109:68-76.

16. Mørkved S, Bø K, Schei B, Salvesen KÅ. Pelvic floor muscle training during pregnancy to prevent urinary incontinence. A single-blind randomized controlled trial. *Obstet Gynecol* 2003;101:313-9.
17. Mørkved S. Does pelvic muscle training decrease postpartum stress incontinence? *ISMJ* Vol. 4 No 6. 2003.
18. Wolfe LA, Davies GAL. Canadian guidelines for exercise in pregnancy. *Clin Obstet Gynecol* 2003;46:488-95.
19. ACSM's Guidelines for Exercise Testing and Prescription. American College of Sports Medicine, 7. edn. Philadelphia (PA): Lippincott Williams & Wilkins; 2006.
20. Bø K, Thune C, Winther B. *Sprek, slank & sunn mamma!* Oslo: Boksenteret Erik Pettersen & Co AS; 2004.

13. Menopause

Authors

Mats Hammar, MD, PhD, Professor, Department of Obstetrics and Gynecology, Women's Clinic, Faculty of Health Sciences, Linköping University, Linköping, Sweden

Kari Bø, PT, PhD, Professor, Norwegian School of Sports Sciences, Department of Sports Medicine, Oslo, Norway

Summary

Menopause is the woman's last menstruation and occurs when the production of oestrogen from the ovaries has decreased so much that the mucous membrane (endometrium) of the uterus is no longer stimulated. The transition period is a period that covers approximately 5–10 years before and after menopause and is characterised in many women by certain physical and psychological changes. These are often associated with reduced oestrogen levels in the body. Oestrogen replacement therapy mitigates many, but not all of these changes and works well in most women, but can sometimes result in undesired effects or side-effects. Consequently, not all women can receive oestrogen replacement therapy, such as those with a tendency for blood clots or who have had breast cancer.

Physical activity also mitigates most of the changes that arise in the transition age, sometimes just as distinctly as oestrogen, in some cases more. Problems from fragile vaginal mucosa are not at all affected by physical activity, but can in principle always be easily treated with low dose oestrogen applied locally in the vagina.

Menopausal women can follow general exercise recommendations and exercise principles. Suitable activities include brisk walks, Nordic walking, dance, exercise callisthenics, cycling, jogging, skiing and so on.

Definition

Prevalence/Incidence

All women experience reduced ovarian hormone production in their 50s, which leads to the cessation of menstruation, but far from all women have difficulties in association with this. Up to 75 per cent of women in the Western World have hot flushes and sweats, and

nearly half of all women have symptoms of fragile vaginal mucosa. As described below, not all of these women, however, need medical help for these symptoms.

Causes and symptoms

Menopause is the woman's last spontaneous menstruation and in the Western World occurs on average around the age of 51–52, and a few years earlier in smokers. This is caused by the ovarian oestrogen production decreasing so much that the endometrium of the uterus is no longer stimulated and therefore does not need to be flushed out. Oestrogen is produced in the ovarian follicles, which are already formed in the fetus and do not regenerate after that. Approximately 5–10 years before menopause, the remaining follicles already produce less oestrogen. In this stage, some women already note some symptoms with *irregular menstruation* and, sometimes, early effects on their moods (1).

Some 50–75 per cent of all women report *vegetative symptoms* with hot flushes and sweats around menopause, often with negative effects on night-time sleep and the ability to work (1). The symptoms can already arise in women with irregular menstruation cycles, but are most common right after the last menstruation – menopause (1–3). These symptoms are considered to be due to the body's thermostat (located in the brain) becoming less stable and suddenly changing its setting to a lower level, whereby the body needs to get rid of energy in the form of heat and by sweating. The thermostat's reduced stability is probably due to the oestrogen reduction leading to altered formation of substances in the brain – such as beta-endorphins – which normally stabilise the thermostat (4).

In a German study, 500 men were compared with 153 women. No differences were then found between men and women in their 50s with regard to the frequency of sweating attacks (5). Both genders had more sweating attacks than younger people, which was particularly true of sudden night sweats, but also those occurring in the daytime. The conclusion drawn was that sweating attacks are a phenomenon that arises due to an altered temperature regulation in the age group of 50–55 year-olds, and the study raised the question of oestrogen's significance in the context.

The *mood problems* reported by many women around menopause are considered to mainly be linked to the vegetative symptoms with associated sleep disorders, reduced quality of life and increased psychological vulnerability (6–8). The lower oestrogen levels have been suggested to directly incite mood effects, through altered formation of signal substances in the brain (6).

After menopause, up to half of all women have symptoms of *fragile vaginal mucosa* with soreness during intercourse, burning upon urination and an increased prevalence of urinary tract infections (2, 3). These symptoms usually first arise a few years after menopause once the oestrogen levels have become really low, because the vaginal mucosa is normally also stimulated by low levels of oestrogen. When this stimulation subsides, the mucous membrane becomes thin and fragile and its blood supply is reduced.

Urinary incontinence is more widespread among women than men, and is a symptom that can have many different causes. There are several types of incontinence and the most common are stress and urge incontinence as well as mixed-type incontinence. Stress

incontinence is involuntary leakage upon physical exertion, sneezing or coughing. Urge incontinence is a sudden pressure urge that leads to involuntary leakage. Mixed-type incontinence is a combination of both types (9).

The prevalence statistics for urinary incontinence based on the definition of “occasionally” or “at least once in the past 12 months” varies in most studies between 25 and 45 per cent (10). Stress incontinence is more widespread among young and middle-aged women, while urge incontinence and mixed-type incontinence is dominant in older women. High prevalence figures have also been found in athletically active women who have not given birth (11).

It has long been believed that menopause was an important factor in the development of urinary incontinence, since atrophy changes can result in diminished closure of the urethra and an elevated risk of urinary tract infection. Urinary tract infections can entail problems in holding water (repetitive urination and pressure). The research results concerning a lack of oestrogen as a cause or risk in this context are not unambiguous, however. Some studies have shown a lower prevalence among post-menopausal women than pre-menopausal women, and some studies show that there are more women with urinary incontinence among those who take oestrogen than among those that do not (10). Since urinary incontinence is a problem for women of all ages, a high prevalence has also been found among pre-menopausal and perimenopausal women.

Another widespread dysfunction in the pelvic floor is prolapse, which is defined as a drop in one or more of the following areas: front vaginal wall, rear vaginal wall, vaginal vault, portio or the larger part of the uterus (9). The condition can be clinically rated on a scale from 1–4 where the absence of prolapse is defined as stage 0. Utero-vaginal prolapse is commonly occurring and prevalence statistics vary between 5 and 94 per cent depending on the definition, population and classification system (10). It has been assumed that as many as 50 per cent have one type of prolapse or another after a vaginal birth (12), but the prevalence based on symptoms is lower (7–23%) (10). The symptoms are fatigue, a feeling of weight and discomfort in the genitals, a feeling that something is “falling out” and sometimes difficulties emptying the bladder and urinary incontinence. Prolapse can, like urinary incontinence, lead to discomfort during physical activity and can be an important factor in reduced physical activity among women.

A reduced production of oestrogen involves changes in several systems in the body. These include an acceleration in skeletal bone loss and the risk of *osteoporosis* increases. Bone tissue is produced the entire time, at the same time that it is broken down in other areas. The cells that build up and break down are somewhat in balance, but when the oestrogen levels decrease, the decomposing cells’ activity begins to dominate. Consequently, the woman can be affected by osteoporosis with a risk of fractures, mainly in the wrist, neck of the femur and vertebra.

Furthermore, oestrogen probably has a positive effect on balance. According to this theory, when oestrogen levels drop, balance degrades, the risk of falling increases and thereby also the risk for osteoporotic fractures (13). These effects of oestrogen are probably exerted on substances in the cerebellum where the balance is controlled.

Oestrogen also has positive effects on *lipids* by affecting the liver. Oestrogen has a direct relaxing effect on the walls of the blood vessels, and also has a damping effect on

how some lipids are stored in the vessel wall as a part of the actual arteriosclerosis process. These phenomena have been tied to women having a relatively rapidly increasing risk of cardiac infarction after menopause, when the effect of oestrogen rapidly subsides.

Diagnostics of menopausal symptoms

If in close connection with menstruation becoming irregular or ending, a woman experiences sudden hot flushes or sweats night and day, the diagnosis is usually simple. If the woman had previously had a hysterectomy or uses some type of contraceptive that has ended menstruation (such as a hormone spiral or injection with a contraceptive injection), a blood test (to analyse follicle-stimulating hormone – FSH) is taken to secure the diagnosis of “hormonal” menopause, since altered menstruation does not provide any help in making a diagnosis. In some cases, the typical symptoms of sudden hot flushes and sweats are caused by factors other than reduced oestrogen levels. Then, other functions are studied, such as the functioning of the thyroid.

If the woman does not have distinct hot flushes and sweats, but is rather mostly troubled by dejection and sleeping disorders, other diagnoses, such as depression, must be considered. Sometimes one may choose to try oestrogen replacement therapy for a brief period, even against mild depression, to see how the symptoms are affected, particularly if the woman has hot flushes and sweats with night-time sleep disruptions at the same time.

Treatment of menopausal symptoms

Hot flushes and sweats are the most common cause of women in Sweden seeking medical help around menopause. Most women are helped by oestrogen replacement therapy. Oestrogen alleviates these symptoms very distinctly (approximately a 90% reduction in the number of hot flushes per day) and thereby improves night-time sleep and well-being (14, 15). Since oestrogen also stimulates a number of different target organs and tissues, the treatment will reduce symptoms of fragile mucous membranes in the vagina and bladder, stimulate bone tissue so as to prevent osteoporosis, possibly also affect balance positively and have a good effect on lipid levels. Oestrogen replacement therapy also possibly reduces the risk of colon cancer and the risk of Alzheimer’s disease (16). It has long been believed that oestrogen replacement therapy reduces the risk of arteriosclerosis and heart attack, since women treated with hormones more seldomly have heart attacks than women who do not receive hormone therapy. However, it has been shown that this may partially be due to women who receive hormone therapy being more health aware and healthier from the beginning than those who do not receive hormone therapy and more recent studies have shown that oestrogen-progestagen given to women above age 60–65 may rather increase their risk of myocardial infarctions.

Disadvantages of oestrogen replacement therapy

Because oestrogen also stimulates the mucous membrane of the uterus, it can lead to a risk of increased thickness of the endometrium and also sometimes atypia of the endometrium on the long term. Therefore, treatment is usually always given with a hormone that has the same effect as the body's progesterone, which, if used for about 10–14 days per month, usually leads to the woman having regular menstruation. After a few years of treatment, progesterone can be given in a small daily dose and the woman can thereby avoid menstruation. A disadvantage is that some women have undesirable bleeding similar to menstruation, while others have negative mood effects from progesterone (17, 18). Nausea and breast pain are other, not uncommon, but often rapidly transient symptoms of oestrogen.

The most serious side-effects of oestrogen replacement therapy are blood clots and breast cancer. The risk of blood clots is doubled with the use of oestrogen taken orally, but since the risk is small from the beginning, the absolute risk is still very small during hormone treatment as well. Breast cancer affects nearly one tenth (10 of 100) of Swedish women at sometime in life. If all Swedish women were to use oestrogen against menopausal symptoms for 10 years, approximately another three women out of 100 would be affected.

It was long believed that oestrogen replacement therapy would reduce the risk of arteriosclerosis and heart attack, but research of recent years has not been able to prove this. Rather, the risk of heart attack is somewhat elevated during the first years of treatment, at least if treatment is applied among somewhat older women after the age of 60–65. This is probably due to the oestrogen's effect on the coagulation system with a somewhat elevated propensity for blood clots, which can then affect the heart's coronary artery (19, 20). Today, one speaks of a "window of opportunity", which means that treatment applied soon after the last menstruation probably entails mainly advantages, also with regard to the cardiovascular system.

Effects of physical activity

The effects of physical activity are the same in menopausal women as in people in general, but are more distinct at this age due to certain specific effects.

Effects on various functions affected during menopause

Vegetative symptoms with hot flushes and sweats have been shown to occur less often in women who exercise regularly than in inactive women. Studies from Linköping, Sweden have shown that women who exercise regularly, report fewer menopausal symptoms than women who do not exercise (21, 22). In a later study, a group of inactive women were randomly assigned to begin exercising regularly and maintain a journal over the number of hot flushes and sweats, complete quality of life forms, etc. (23). It turned out that the number of hot flushes decreased and quality of life improved in the women who exercised three sessions a week. A U.S. longitudinal study was able to confirm these findings in

women with prior depression, but not in women in general (24). This can be explained by physical activity leading to increased production of beta-endorphins in the brain, which are of significance to maintaining the stability of the brain's thermostat. If this is the cause of the effect, the woman should be recommended regular exercise that activates large muscle groups at least 30 minutes at a time, at least three times a week. Examples of such exercise can be Nordic walking, exercise callisthenics or strength training. It should be noted that women who exercise regularly can also have hot flushes and exercise is no guarantee that these symptoms will subside. However, exercise gives several other valuable effects at the same time.

The mood effect is often improved when hot flushes and sweats decrease, but exercise has been shown to be valuable by relieving stress and reducing depression. This has been shown in several scientific studies of women (and men) in general (25–29). Women who were randomly assigned physical exercise or yoga for four months experienced improved quality of life, primarily women who had improved fitness at the same time (30). Another randomised study found that as little as six weeks of regular walking on a treadmill improved quality of life, but that the effect only lasted among those who continued for another six weeks and declined in those who stopped exercising (31).

Symptoms of *fragile vaginal mucosa* are not at all affected by physical activity, but can be easily treated locally with low dose oestrogen. Such local low-dose therapy provides no other valuable oestrogen effects or side-effects and can be used by all women who need it without risk.

Oestrogen replacement therapy has little effect on *stress incontinence* (32). Randomised, controlled studies have shown that between 44 and 70 per cent of women with stress incontinence are completely healed after pelvic floor exercises, which is equivalent to < 2 grams of leakage in a pad weighing test after training (33). There have been no specific studies of post-menopausal women, but the majority of studies include women ages 40–50 or older. Pelvic floor exercises are effective and have no side-effects. Internationally, it is recommended that pelvic floor exercises should be the primary choice in terms of treatment alternatives for stress or mixed-type incontinence (34). Several studies also draw the conclusion that more intensive exercise is more effective than training without follow-up. Therefore, it is recommended that women with stress incontinence be given information about exercise methods and frequent follow-up by a physiotherapist. Strength training can advantageously be done in a group where one also focuses on other supplemental physical exercises.

One U.S. study has shown that if one learns to find and activate the pelvic floor muscles before and during coughing it leads to an average of 73 per cent less leakage after a coughing attack (35). Actively tensing the muscles (pinching) to prevent leakage when coughing, sneezing and heavy lifting can therefore entail rapid improvements. To increase muscle volume and improve the nerve-muscle function so that the muscles automatically contract when one runs, jumps or dances, requires regular training for 5–6 months, however (36).

A Norwegian study showed that 70 per cent remained satisfied with their condition and had no leakage when coughing five years after the organised training had been concluded.

In one Belgian study, two thirds of those who were satisfied upon completed training said that they remained satisfied after 10 years (33). How much training is required to retain muscle strength is individual, but 8–12 pinches/contractions of the muscles as hard as possible 1–2 times a week are recommended (37).

That pelvic floor training has an effect on *urge incontinence* has not been shown in randomised, controlled studies.

Treatment of *utero-vaginal* prolapse includes surgery, a prolapse ring and pelvic floor training. Surgery is most often the only alternative when the prolapse has an advanced progression, but has a high frequency of relapse (38). Most women are operated in their 50s, with an average age of 55 at the first operation (38). There are no studies that have compared the use of a prolapse ring with pelvic floor training or no treatment, and little is known of what kind of ring is most effective (39). Today, there are only two randomised controlled studies of pelvic floor training, of which one has a low methodological quality and the other is only reported in summary and is a pilot study (40). However, both studies indicate a positive effect of strength training of the pelvic floor muscles. This is assumed to be an effect of the pelvic floor being lifted, and of a tightening of the connective tissue and muscles, but there are no studies that have investigated this (41). Today, it is not known if pelvic floor training can be primarily preventative or which stage it may potentially be possible to alleviate or slow a prolapse.

Osteoporosis is a condition that is affected by hereditary disposition, smoking, physical inactivity, oestrogen deficiency and by some diseases. Regular physical activity decreases the bone loss that usually begins as early as age 30–35 and accelerates after menopause. A number of studies have shown a positive effect of regular exercise, and then primarily of exercise that strains the skeleton, such as apparatus training with resistance, walks, exercise callisthenics, etc. (42–46). However, bone tissue is the tissue that adapts the fastest to activity, and variation and increased loading are therefore important to the effect. The guidelines for better bone health emphasize activity with a high weight load, in other words running and exercises where jumps occur and strength training with heavy weights (47). Various types of ball sports, for example, can be excellent activities to stimulate bone density. However, it should be noted that such exercise can increase the risk of strain injuries and it is therefore important that the training be individually adapted. A Cochrane overview draws the conclusion that aerobics, weight-bearing exercise and strength training are effective for increasing bone density in the backs of post-menopausal women and that walking has an effect on the bone density of the back and hips (48). Some, but not all, studies have shown that the effects of oestrogen and exercise strengthen each other (44, 45, 49). The effect is greater, the longer and the more often one exercises. In addition, it has been observed that regular physical activity improves balance and the risk of falling thereby decreases (46).

The risk of *cardiovascular disease* decreases with regular exercise and the effect is caused by a number of mechanisms, such as a positive impact on lipids, improved insulin effect, weight loss, decreased stiffness of the artery walls, etc. (50–58). This has been shown for both men and women and in various ages, as well as in women around menopause. With the new knowledge that indicates that oestrogen replacement therapy does not

reliably protect against heart attack (19, 20), this knowledge about the effects of exercise becomes even more important. In addition, exercise improves aerobic fitness and endurance, which hormone treatment does not do (59). Thirty minutes of physical activity at a moderate intensity (sweaty or breathless) reduces the risk of premature death in otherwise inactive persons (60). Aerobic exercise at an intensity of 70–80 per cent of maximal heart rate is recommended to improve the maximal oxygen uptake (37). All activities that involve major muscle groups and are dynamic and rhythmic by nature (cycling, brisk walks, aerobics, etc.) are recommended to increase aerobic fitness (37).

Epidemiological data has shown that people who have been and are physically active have a reduced risk of having some *forms of cancer* such as breast and uterine cancer (61–64). The studies claim that this was not only due to the physically active women being generally more aware of health. The cause of these findings can possibly be that more intensive exercise can thin out or completely prevent ovulation, which means that oestrogen production is lower and the risk of breast and uterine cancer thereby decreases. Other proposed causes of a reduced risk of breast cancer in physically active women is that exercise also activates antioxidative systems (65), reduces breast density measured by mammography (66) and decreases oestrogen levels (67). A randomised study was also recently published that found that women who were randomly assigned 45 minutes' exercise five times a week for one year had fewer colds than the control group (68).

In summary, regular physical activity entails many advantages for menopausal women. The majority of the effects do not differ from those also observed in people of other ages, but the effects are clear and affect several of the phenomena that otherwise usually become prevalent in menopause in particular. One should choose varied types of exercise to avoid overload problems from excessively one-sided exercise. It is also important that the activities chosen are perceived as pleasant and enjoyable and that one gladly exercises and trains in a group. Group exercises can often involve a certain "social pressure" that means that one continues. It is necessary that these activities continue regularly and are maintained for an extended period of time.

Indications

Physical activity during menopause can serve as both primary and secondary prevention, in other words can both prevent problems from arising and function as treatment once something has happened (such as an osteoporotic fracture). The treatment is probably nonetheless most effective as primary prevention, since the problem developed can in itself reduce the possibilities of pursuing regular exercise. The effect of physical activity in many cases reinforces the effect of hormone therapy and there is absolutely no obstacle to combining these measures. The need is most clear among women who do not choose hormone therapy (primarily with regard to the effect on bone density) and physical activity can also continue for an unlimited period, which is not true of hormone therapy.

Prescription

Physical activity during menopause should contain both condition and strength elements to prevent osteoporosis, cardiovascular disease and reduce the risk of hot flashes and sweats, urinary incontinence and improve mood.

Menopausal women can follow general exercise principles (also see Chapter 2) for adults that mean that one should do at least 30 minutes of moderate physical activity per day (one will be able to talk but not sing, i.e. breathing and heart rate will be increased) to achieve health benefits. A combination of moderate and intense activity can also be utilised to achieve these effects. To improve or retain aerobic fitness, exercise is recommended with an intensity of up to 70–80 per cent of the maximal heart rate three times a week (37). Moderate activity can also be divided up into multiple sessions per day, such as 3 x 10 minutes (69). To retain or increase muscle strength, strength training of the most important muscle groups (abdomen, back, pelvic floor, gluteal, thigh and arm muscles) should be done 2–3 times a week with one to three series of 8–12 repetitions close to maximum capacity. To retain or increase flexibility, flexibility training is recommended for the body's joints 2–3 times a week, with 2–4 repetitions and with a duration of 15 seconds for each extension (37, 69).

The intensity of the exercise, the number of sessions per week and the time for every session should be gradually increased for a tentative minimum of three months to not lead to overload symptoms. The exercise can gladly be performed as group training and with varying content to increase the chances of the activity becoming permanent.

Suggestions of suitable activities

Brisk walks, Nordic walking, dance, aerobics, step-up training, exercise callisthenics, strength training, cycling, jogging and skiing are excellent activities. Swimming is a good activity that stimulates the muscles and fitness, but does not have any effect on osteoporosis. Varying the activities during the week is stimulating. Strength training can be done at home, under guidance at a training centre or as general group training to music.

Functional tests/need for health check-ups

- The effect of physical activity in menopausal women can be evaluated at the earliest after three months with regard to well-being and the effect on hot flashes/sweats.
- The effect on bone density can hardly be measured until after at least one year or more.
- The effect on weight, lipid levels and aerobic fitness can (if so desired) be measured for the first time after approximately six months.
- The effect on the pelvic floor muscles and incontinence can be seen after 3–4 months' regular training.

Interactions with pharmaceutical treatment

Hormone therapy and physical activity can advantageously be combined without any problem.

Contraindications

There are no contraindications except in acute illness with a diminished general state of health.

Risks

The risk of injury in excessively intense and rapidly increasing training must be observed, which is why intensity, frequency and duration should not be increased too rapidly, but rather gradually and with caution. An excessively rapid increase could cause a risk of overload symptoms, which can take a long time to heal and thereby make exercise difficult for a long period of time and, which is perhaps most important, could mean that the woman would not dare to continue or resume her training.

References

1. McKinley S, Brambilla D, Posner J. The normal menopause transition. *Maturitas* 1992;14:103-15.
2. Berg G, Gottvall T, Hammar M, Lindgren R. Climacteric symptoms among women aged 62–64 in Linköping, Sweden in 1986. *Maturitas* 1988;10:193-9.
3. Hammar M, Berg G, Fåhraeus L, Larsson-Cohn U. Climacteric symptoms in an unselected sample of Swedish women. *Maturitas* 1984;6:345-50.
4. Wyon Y, Spetz AC, Theodorsson E, Hammar M. Concentrations of calcitonin gene related peptide and neuropeptide Y in plasma increase during flushes in postmenopausal women. *Menopause* 2000;7:25-30.
5. Heinemann K, Saad F. Sweating attacks. Key symptom in menopausal transition only for women? *European Urology* 2003;44:583-7.
6. Ditkoff EC, Crary WG, Cristo M, Lobo RA. Estrogen improves psychological function in asymptomatic postmenopausal women. *Obstet Gynecol* 1991;78:991-5.
7. Oldenhave A, Jaszmann L, Haspels AA, Everaerd W. Impact of climacteric on well-being. A survey based on 5 213 women 39 to 60 years old. *Am j Obstet Gynecol* 1993;168:772-80.
8. Skarsgård C, Berg G, Ekblad S, Wiklund I, Hammar M. Effects of estrogen therapy on well being in postmenopausal women without vasomotor symptoms. *Maturitas* 2000;36:123-30.
9. Abrams P, Cardozo L, Fall M, Griffiths D, Rosier P, Ulmsten U, et al. The standardisation of terminology of lower urinary tract function. Report from the Standardisation Sub-committee of the International Continence Society. *Neurourol Urodyn* 2002;21:167-78.
10. Hunskaar S, Burgio K, Clark A, Lapitan MC, Nelson R, Sillen U, et al. Epidemiology of urinary (UI) and faecal (FI) incontinence and pelvic organ prolapse (POP). In: Abrams P, Cardozo L, Khoury S, Wein A. *Incontinence. Vol 1. Basic and evaluation.* Plymouth: Health Publication Ltd; 2005. Chapter 5, pp. 255-312.
11. Bø K. Urinary incontinence, pelvic floor dysfunction, exercise and sport. *Sports Med* 2004;7:451-64.
12. Swift SE. The distribution of pelvic organ support in a population of female subjects seen for routine gynecologic health care. *Am J Obstet Gynecol* 2000;183:277-85.
13. Ekblad S, Bergendahl A, Enler P, Ledin T, Möller C, Hammar M. Disturbances in postural balance are common in postmenopausal women with vasomotor symptoms. *Climacteric* 2000;3:192-8.
14. MacLennan A, Lester S, Moore V. Oral oestrogen replacement therapy versus placebo for hot flushes (Cochrane Review). In: *The Cochrane Library* 2001;2. Oxford Update Software.
15. Polo-Kantola P, Erkkola R, Helenius H, Irjala K, Polo O. When does estrogen replacement therapy improve sleep quality? *Am J Obstet Gynecol* 1998;178:1002-9.

16. Werkö L, Bergkvist L, Bixo M, Björkelund C, Hammar M, Hellgren Wängdahl M, et al. *Behandling med östrogen. [Treatment with oestrogen.] Report 159.* Stockholm: SBU; 2002.
17. Holst J, Bäckström T, Hammarbäck S, von Schoultz B. Progestogen addition during oestrogen replacement therapy. Effects on vasomotor symptoms and mood. *Maturitas* 1989;11:13-20.
18. Björn I, Bixo M, Nojd K, Nyberg S, Bäckström T. Negative mood changes during hormone replacement therapy. A comparison between two progestogens. *Am J Obstet Gynecol* 2000;183:19-26.
19. Writing Group for the Women's Health Initiative Investigators. Risks and benefits of estrogen plus progestin in healthy postmenopausal women. Principal results from the Women's Health Initiative Randomized Controlled Trial. *JAMA* 2002;288:321-33.
20. Herrington DM, Vittinghoff E, Lin F, et al., for the HERS Study Group. Statin therapy, cardiovascular events and total mortality in the Heart and Estrogen/Progestin Replacement Study (HERS). *Circulation* 2002;105:2962-7.
21. Hammar M, Berg G, Lindgren R. Does physical exercise influence the frequency of postmenopausal hot flushes? *Acta Obstet Gynecol Scand* 1990;69:409-12.
22. Ivarsson T, Spetz A-C, Hammar M. Physical exercise and vasomotor symptoms in postmenopausal women. *Maturitas* 1998;29:139-46.
23. Lindh-Astrand L, Nedstrand E, Wyon Y, Hammar M. Vasomotor symptoms and quality of life in previously sedentary postmenopausal women randomised to physical activity or estrogen therapy. *Maturitas* 2004;48:97-105.
24. Thurston RC, Joffe H, Soares CN, Halow BL. Physical activity and risk of vasomotor symptoms in women with and without a history of depression. Results from the Harvard Study of Moods and Cycles. *Menopause* 2006;13:553-60.
25. Petruzello SJ, Landers DM, Hatfield BD, Kubitz KA, Salazar W. A meta-analysis on the anxiety-reducing effects of acute and chronic exercise. *Sports Medicine* 1991;1:143-82.
26. Martinsen AW. Benefits of exercise for the treatment of depression. *Sports Medicine* 1990;9:380-9.
27. LaFontaine TP, DiLorenzo TM, Frensch PA, Stucky-Ropp RC, Bargman EP, McDonald DG. Aerobic exercise and mood. A brief review, 1985-1990. Leading article. *Sports Medicine* 1992;13:160-70.
28. Morgan WP, O'Connor PJ. Psychological effects of exercise and sports. In: Ryan AJ, Allman FL Jr, eds. *Sports medicine*. 2. edn. San Diego: Academic Press; 1989, pp. 671-89.
29. Raglin JS. Exercise and mental health. Beneficial and detrimental effects. *Sports Medicine* 1990;9:323-9.
30. Elavsky S, McAuley E. Physical activity and mental health outcomes during menopause. A randomized controlled trial. *Ann Behav Med* 2007;33:132-42.

31. Asbury EA, Chandruangphen P, Collins P. The importance of continued exercise participation in quality of life and psychological well-being in previously inactive postmenopausal women. A pilot study. *Menopause* 2006;13:561-7.
32. Andersson KE, Appell R, Cardozo L, Chapple C, Drutz H, Fourcroy J, et al. Pharmacological treatment of urinary incontinence. In: Abrams P, Cardozo L, Khoury S, Wein A. *Incontinence. Vol 2. Management*. Plymouth: Health Publication Ltd; 2005. Chapter 14, pp. 811-54.
33. Bø K. Is there still a place for physiotherapy in the treatment of female incontinence? *EAU Update Series* 2003;1:145-53.
34. Wilson PD, Berghmans B, Hagen S, Hay-Smith J, Moore K, Nygaard I, et al. Adult conservative treatment. In: Abrams P, Cardozo L, Khoury S, Wein A. *Incontinence. Vol 2. Management*. Plymouth: Health Publication Ltd; 2005. Chapter 15, pp. 855-964.
35. Miller JM, Ashton-Miller JA, DeLancey JOL. A pelvic floor muscle pre-contraction can reduce cough-related urine loss in selected women with mild SUI. *J Am Geriatr Soc* 1998;46:870-4.
36. Bø K. Pelvic floor muscle training is effective in treatment of female stress urinary incontinence, but how does it work? *Int Urogynecol J* 2004;15:76-84.
37. American College of Sports Medicine. *ACSM's Guidelines for exercise testing and prescription*. 7. edn. Philadelphia: Lippincott Williams & Wilkins; 2006.
38. Brubaker L, Bump R, Fynes M, Jacuetin B, Karram M, Kreder K, et al. Surgery for pelvic organ prolapse. In: Abrams P, Cardozo L, Khoury S, Wein A. *Incontinence. Vol 2. Management*. Plymouth: Health Publication Ltd; 2005. Chapter 17, pp. 1371-1401.
39. Adams EJ, Hagen S, Maher C, Thomson AJM. Mechanical devices for pelvic organ prolapse in women. *Cochrane Database for Systematic Reviews* 2004;2.
40. Hagen S, Stark D, Mher C, Adams EA. Conservative management of pelvic organ prolapse in women. *Cochrane Database of Systematic Reviews* 2007;2.
41. Bø K. Can pelvic floor muscle training prevent and treat pelvic organ prolapse? *Acta Obstet Gynecol* 2006;85:263- 8.
42. Martin D, Notelovitz M. Effects of aerobic training on bone mineral density of postmenopausal women. *J Bone Miner Res* 1993;8:931-6.
43. Rikli RE, McManis BG. Effects of exercise on bone mineral content in postmenopausal women. *Res Q Exerc Sport* 1990;61:243-9.
44. Heikkinen J, Kurttila-Matero E, Kyllonen E, Vuori J, Takala T, Vaananen HK. Moderate exercise does not enhance the positive effect of estrogen on bone mineral density in postmenopausal women. *Calcif Tissue Int* 1991;49:83-4.
45. Notelovitz M, Martin D, Tesar R, Khan FY, Probart C, Fields C, et al. Estrogen therapy and variable-resistance weight training increase bone mineral in surgically menopausal women. *J Bone Miner Res* 1991;6:583-90.
46. Hammar M, Brynhildsen J, Wyon Y, Nedstrand E, Notelovitz M. The effects of physical activity on menopausal symptoms and metabolic changes around menopause. *Menopause* 1995;2:201-9.

47. Kohrt WM, Bloomfield SA, Little KD, Nelson ME, Yingling VR. American College of Sports Medicine. Physical activity and bone health. Position stand. *Med Sci Sports Exerc* 2004;11:1985-96.
48. Bonaiuti D, Shea B, Iovine S, Robinson V, Kemper HC, Wells G, et al. Exercise for preventing and treating osteoporosis in postmenopausal women. *Cochrane Database for Systematic Reviews* 2002;2.
49. Maddalozzo GF, Widrick JJ, Cardinal BJ, Winters-Stone KM, Hoffman MA, Snow CM. The effects of hormone replacement therapy and resistance training on spine bone mineral density in early postmenopausal women. *Bone* 2007;40:1244-51.
50. Ledin T, Kronhed A, Möller C, Möller M, dkvist L, Olsson B. Effects of balance training in elderly evaluated by clinical tests and dynamic posturography. *J Vest Research* 1991;1:129-38.
51. Lindheim S, Notelovitz M, Feldman E, Larsen S, Khan F, Lobo R. The independent effects of exercise and estrogen on lipids and lipoproteins in postmenopausal women. *Obstetrics & Gynecology* 1994;83:167-72.
52. Hardman AE, Hudson A, Jones PR, Norgan NG. Brisk walking and plasma high density lipoprotein concentration in previously sedentary women. *BMJ* 1989;299:1204-5.
53. Owens JF, Mathews KA, Wing RR, Kuller LH. Can physical activity mitigate the effects of aging in middle aged women? *Circulation* 1992;85:1265-70.
54. Lobo RA, Notelovitz M, Bernstein L, Khan FY, Ross RK, Paul WL. Lp(a) lipoprotein. Relationship to cardiovascular disease risk factors, exercise and estrogen. *Am J Obstet Gynaecol* 1992;166:1182-8.
55. Blair SN, Goodyear NN, Gibbons LW, Cooper KH. Physical fitness and incidence of hypertension in healthy normotensive men and women. *JAMA* 1984;252:487-90.
56. Lamarche B, Deprés JP, Pouliot MC, Moorjani S, Lupien PJ, Thériault G, et al. Is the body fat loss a determinant factor in the improvement of carbohydrate and lipid metabolism following aerobic exercise training in obese women? *Metabolism* 1992;41:1249-56.
57. van Dam S, Gillespy M, Notelovitz M, Martin AD. Effect of exercise on glucose metabolism in postmenopausal women. *Am J Obstet Gynecol* 1988;159:82-6.
58. Sugawara J, Otsuki T, Tanabe T, Hayashi K, Maeda S, Matsuda M. Physical activity duration, intensity, and arterial stiffness in postmenopausal women. *Am J Hypertens* 2006;19:1032-6.
59. Church TS, Earnest CP, Skinner JS, Blair SN. Effects of different doses of physical activity on cardiorespiratory fitness among sedentary, overweight or obese postmenopausal women with elevated blood pressure. A randomized controlled study. *JAMA* 2007;297:2081-91.
60. Statens råd for ernæring og fysisk aktivitet. [Norwegian National Council on Nutrition and Physical Activity.] *Fysisk aktivitet og helse. Anbefalinger. [Physical activity and health. Recommendations.] Report 2.* Oslo: Statens råd for ernæring og fysisk aktivitet, Norwegian Ministry of Health and Social Affairs; 2000.

61. Sternfeld B. Cancer and protective effect of physical activity. The epidemiological evidence. *Med Sci Sports Med* 1992;24:1195-209.
62. Levi F, La Vecchia C, Negri E. Selected physical activities and the risk of endometria cancer. *Br J Cancer* 1993;67:846-51.
63. Vihko VJ, Apter DL, Pukkala EI. Risk of breast cancer among female teachers of physical education and languages. *Acta Oncol* 1992;31:201-4.
64. Bernstein L, Henderson B, Hanisch R, Sullivan-Lalley J, Ross R. Physical exercise and reduced risk of breast cancer in young women. *J Natl Cancer Inst* 1994;89:1403-8.
65. Gogo-Dominguez M, Jiang X, Esteban Castelao J. Lipid peroxidation and the protective effect of physical exercise on breast cancer. *Med Hypotheses* 2007;68:1138-43.
66. Irwin ML, Aiello EJ, McTiernan A, Bernstein L, Gilliland FD, Baumgartner RN, et al. Physical activity, body mass index, and mammographic density in postmenopausal breast cancer survivors. *J Clin Oncol* 2007;25:1061-6.
67. McTiernan A, Wu L, Chen C, Chlerbowski R, Mossavar-Rahmani Y, Modugno F, et al. Relation of BMI and physical activity to sex hormones in postmenopausal women. *Obesity (Silver Spring)* 2006;14:1662-77.
68. Chubak J, McTiernan A, Sorensen B, Wener MH, Yasui Y, Velasquez M, et al. Moderate-intensity exercise reduces the incidence of colds among postmenopausal women. *Am J Med* 2006;119:937-42.
69. Haskell WL, Lee I-M, Pate RP, Powell KE, Blair SN, Franklin BA, et al. Physical activity and public health. Updated recommendation for adults from the American College of Sports Medicine and the American Heart Association. *Med Sci Sports Exer* 2007;39:1423-34.

14. Elderly

Authors

Jan Lexell, MD, PhD, Professor, Department of Rehabilitation Medicine, Lund University Hospital, Division of Rehabilitation Medicine, Department of Clinical Sciences, Lund University, Lund, Sweden

Kerstin Frändin, PhD, PT, Associate Professor, Department of Neurobiology, Care Sciences and Society, Division of Physiotherapy, Karolinska Institutet, Stockholm, Sweden

Jorunn L Helbostad, PhD, PT, Department of Neuroscience, Faculty of Medicine, Norwegian University of Science and Technology (NTNU), Trondheim, Norway

Summary

Although factors such as heredity and illness affect how we age, it is absolutely clear that regular physical activity and exercise, as a significant lifestyle factor, can both prevent and reduce various age-related physical and mental changes. Elderly men and women can improve their aerobic fitness and endurance as well as balance, strength and flexibility up to a very advanced age. Aerobic exercise can affect risk factors for cardiovascular disease. Strength training leads to an increased muscle mass and muscle strength and improved functional capacity. Physical activity and training also affects bone mass as well as balance, coordination and flexibility, which together reduce the risk of falling accidents and fractures. Physical activity and training also appear to be able to have a positive effect on various psychological factors and quality of life among the elderly. Altogether, available data indicate that physical activity and exercise, as a significant lifestyle factor, are an effective way of maintaining a high level of activity and high degree of independence among elderly men and women. However, the elderly are a very heterogeneous group and individualised exercise programmes are most often preferable to general recommendations.

In terms of *aerobic exercise*, mainly low to moderate intensity activity is recommended to affect risk factors of cardiovascular disease, while moderate to high intensity activity can be needed to be able to achieve improvements in cardiovascular function. Training should be done at least 2–3 times a week, at least 20 minutes at a time depending on intensity. In terms of *strength training*, exercises should be done 1–2 times a week and include the body's major muscle groups in both the upper and lower extremities. Suitable activities with regard to both aerobic exercise and strength training include brisk walks, dancing, callisthenics, swimming, skiing, jogging, cycling and weight training.

Background

In the past 10 years, our knowledge has increased markedly with regard to the effects of physical activity and exercise among men and women over the age of 70 and the significance this has to ageing (1–3). Worldwide, the elderly comprise a larger part of the population and the most rapidly growing proportion are those over the age of 85. For many of these people, physical activity and exercise comprise an important part of preventing illness, improving physical and mental capacity and thereby retaining a high degree of personal independence and quality of life. The health benefits of physical activity are largely the same for the elderly as for other age groups. People who remain active have more years without disability and there are many indications that chronic diseases in association with ageing are partially due to physical inactivity rather than ageing itself. The functional level of the elderly is affected by heredity, illness and lifestyle, and the variation in the level of function between different people also increases with increasing age. However, research has been able to show that it is also possible to enhance health and prevent functional impairment through physical activity and exercise among people with a low functional level and a complex clinical picture.

Effects of ageing and physical activity

Cardiovascular function and aerobic exercise

Maximal oxygen uptake (VO_2 max) decreases by between 5 and 10 per cent per decade after the age of 30 (1, 4). This is caused by a decreased maximal heart rate, reduced heart-minute volume, decreased arteriovenous oxygen difference and reduced stroke volume. In the elderly, the heart muscle also reacts differently to maximum physical exertion than in younger people, including with regard to the ejection fraction (in other words, the stroke volume in relation to the total diastolic volume) and contractility (capacity to contract). However, the effects in terms of cardiovascular function are qualitatively and quantitatively relatively similar in the elderly compared with younger people at submaximum physical exertion.

The effects of aerobic exercise on cardiovascular function are the same in healthy elderly persons as in younger people (1). Several studies of aerobic exercise in the form of cycling, swimming and running have shown increases between 10 to 30 per cent of VO_2 max. The increase in VO_2 max among the elderly is related to the exercise intensity, as in younger people. The improvement of VO_2 max is explained by both changes in the heart muscle's function, with an impact on maximal stroke volume, heart-minute volume, left-chamber function and increased arteriovenous oxygen difference.

Several exercise studies have unanimously shown positive effects on various risk factors for cardiovascular disease among the elderly (1). Light to moderate aerobic exercise in the elderly has led to improved glucose metabolism, greater glucose tolerance and insulin sensitivity, decreased blood pressure and improvements in lipids, effects that can

be completely comparable to those obtained from pharmaceutical treatment. Exercise has also had a positive effect on body composition with a 1–4 per cent decrease in body fat and up to 25 per cent decrease in intraabdominal fat in men. In the majority of studies, these changes have been fully comparable to those observed in younger persons.

Exercise among the elderly with cardiovascular disease has, in several studies, led to positive effects of the same extent as in younger people with corresponding diseases. The changes comprise a decreased heart rate at rest and during submaximum exertion, which together lead to improved physical performance capacity and a decrease in exertion-triggered cardiac symptoms.

Muscle function and strength training

With increasing age, the body's muscle mass decreases, which gradually leads to a reduction in muscle strength (5). A healthy 80-year-old man or woman may have lost half of his or her original muscle mass in some muscles, leading to a halving of muscle strength. At the same time as the decrease in muscle mass, there is an increased storage of fat and connective tissue in the skeletal muscles. The decrease in the muscle mass is caused by a loss of muscle fibres, with a reduction of the size of the remaining muscle fibres, which is in turn due to a reduction in motor neurons of the ventral horns of the spinal cord. Other factors, such as hormonal changes and altered protein synthesis, also contribute to this regression. The reduced muscle mass and muscle strength also lead to changes in activity capacity, such as the ability to walk. In parallel with the decreased muscle mass, there is also a decrease in bone mass, which increases the risk of osteoporosis and fractures from falling accidents.

In a large number of studies, strength training – defined as training with weights or against a gradually increasing load – has proven to provide increases in muscle strength in the elderly, even over the age of 90, of between 50 to 200 per cent (6, 7). There is a strong correlation between the intensity of strength training, in other words the load, and the improvements achieved. In the studies that achieved major and practically significant results, the load during training has been high, often more than 80 per cent of the maximum strength (80% of a repetitive maximum). Training was done with weights and no more than three times a week. Every or every other week, the load was adjusted in pace with the increasing strength, to thereby always maintain a constant load.

In the majority of the studies, the strength increase has been of the same magnitude as in younger people. The main part of the strength increase, in both young and old, primarily comprises an adaptation in the nervous system at the beginning of training. Evaluation of the strength increase, through muscle biopsies, computed tomography or magnetic resonance imaging, has also shown that the muscle mass increased (5–10%) and that the muscle fibres became larger (10–30%) (6–8). When strength training continued for extended periods, up to one year, the increase in muscle mass and muscle fibre size was even larger. Some studies have also mapped the possibility of retaining the achieved strength increase. As in younger people, one exercise session per week can mean that the achieved strength increase is maintained.

In addition to effects on muscle strength, strength training also leads to positive effects on body composition in general, protein metabolism and bone mass (primarily among elderly women) (9). Studies of the addition of various dietary supplements and hormones (growth hormone, oestrogen, testosterone) in the elderly have not, however, shown any significant effects on muscle mass or muscle strength beyond that achieved by the actual strength training.

In recent years, interest has grown in the training of explosive strength, or power, and it has been shown that it is of equal or greater significance to functional ability than traditional strength training (10). An important observation is that power tends to decrease to a greater extent than muscle strength with increasing age. Compared with pure muscle strength, climbing stairs, standing up from a chair and walking speed are therefore affected to a greater extent by both increased and decreased power than by muscle strength alone.

Balance, flexibility and walking ability

Balance, flexibility and walking ability are also affected by increasing age (4). Changes in these functions and the relationship to mobility and falling accidents have led to a greater interest in the effects of physical activity and exercise. Balance is a compound function and is dependent on the coordination of information from sensory and motor systems in different parts of the peripheral and the central nervous system (including basal ganglia, the cerebellum, the vestibular system, vision, muscle and joint sensitivity as well as tactile sensibility).

Flexibility comprises the joints' ability to maintain a range of motion, which is not only dependent on the function of the joint, but the function of the surrounding structures as well (muscles, tendons, ligaments). The ability to walk is dependent on several factors such as balance and joint flexibility, as well as aerobic fitness, muscle strength and power.

A number of studies have shown that balance changes with increasing age. It has long been believed that there is a correlation between balance and falling accidents, but recent studies have shown that the cause of the majority of falling accidents is multi-factoral and that changes in balance are only one cause (11).

Individually adapted exercises to improve muscle strength and balance, combined with one walk a week, have been shown to be able to reduce the propensity to falling among elderly with diminished function living at home (12). For people with a complex clinical picture and major disability, several other efforts are needed in addition to training to prevent falling accidents.

Studies of physical activity and exercise, often including several different types of exercise, have indicated both improved balance and a reduced risk of falling and proportion of falls (13). Large U.S. multicentre studies have included aerobic exercise, strength training, tai chi and flexibility training and indicated effects of various balance elements, but it has not been established what type of training has the greatest single effect (14). With the objective of improving and maintaining balance, general exercise programmes are therefore recommended that include both strength training and aerobic exercise as well as training for balance, flexibility and coordination.

An important factor that has received growing attention is confidence in one's own ability, often called self-efficacy (15). Those who have a low self-efficacy and a fear of

falling avoid activities with which they feel unsafe, and then receive less training and can end up in a vicious cycle of gradually decreasing activity and function. The feeling of safety and greater self-efficacy can, however, be affected by both exercise and information.

Increasing age affects the structures (bones, muscles, connective tissue) needed for retained joint flexibility. Diminished joint flexibility is also a risk factor for diminished functional capacity. With increasing age, the range of motion of several joints of the body is reduced in many people, both proximally and distally. In spite of this, there are few controlled studies of the effects of physical activity and exercise on joint flexibility. The studies that exist have been relatively small and several have lacked a control group. In some cases, the results have not indicated any effects, while others have shown significant effects on joint flexibility among the elderly. The intervention in these studies has consisted of both indirect exercises, such as walking, dance and callisthenics, and direct exercises such as stretching with the aim of increasing the range of motion. In light of this, there are no specific recommendations regarding programmes for the elderly with the aim of increasing joint flexibility and range of motion. Instead, general aerobic exercise programmes are recommended, such as aerobic training, callisthenics, walks and swimming, where flexibility is trained indirectly. Further studies are also needed to establish the intensity and duration of the training, as well as the significance of increased joint flexibility with regard to balance, mobility and the reduction of falling accidents.

The ability to walk is also affected positively by all-round training, but sometimes also needs to be trained specifically. In terms of walking speed, a correlation with muscle strength has only been able to be shown in persons with diminished strength (16, 17). It can therefore be assumed that strength training of the legs in particular has the best effect on the ability to walk in people with diminished functional capacity, such as fragile individuals.

Psychological function and quality of life

It is well known that physical activity has significant effects on various psychological functions and this has also been noted with regard to the elderly (18). Mainly cognitive function and depression, two areas in which the elderly can be affected, and the effects of physical activity and exercise have attracted interest.

A large number of studies have shown possible correlations between physical activity and cognitive function, such as memory, concentration, attention and reaction time (18). Several studies have also indicated large differences in these capacities in physically active elderly persons compared with inactive elderly persons. However, the design of these studies and the lack of descriptions of the test subjects' performance capacity in other respects make these results difficult to interpret. Several exercise studies in recent years have, however, been able to indicate a possible link between increased physical performance capacity and increased cognitive function among the elderly (19). It has also been shown that physically active persons run less risk of developing age-related dementia compared with persons who are less active (20, 21). However, more controlled studies are needed to establish the significance of physical activity and training to an improved cognitive function among the elderly.

Depression is relatively commonly occurring among the elderly. Symptoms of depression have been reported in up to 15 per cent of the elderly population. Physical activity and exercise are currently prescribed as a type of treatment for mild depression and increasing numbers of studies support the correlation between the degree of physical activity and depression, but the proportion of scientific studies that support this treatment is still low (22, 23). It is concluded here as well that more controlled studies are needed to establish the correlation between physical activity, exercise and depression among the elderly.

Health-related quality of life concerns how a person rates his or her own health. Several studies have found that elderly persons who are physically active report a higher health-related quality of life than less active persons (24, 25). There are also signs that indicate that health-related quality of life increases as a result of physical activity and exercise. However, there is a lack of knowledge regarding the correlation between the amount and type of physical activity and exercise and the improvement in health-related quality of life.

Prescription

In general, the individual is encouraged to find activities and types of exercise that he or she is comfortable with and finds enjoyable (26–28). The chance thereby increases that he or she will continue to be physically active over the years. It is important to build up a lifestyle that includes regular physical activity and exercise and that the activities recommended, in one form or another, already comprise a part of the elderly person's life.

Aerobic exercise

Mainly activities that involve major muscle groups are recommended, such as cycling, swimming, walking, jogging and skiing. The intensity and duration of the activity are crucial to the degree of change in cardiovascular function achieved with aerobic exercise. Mainly low to moderate intensity activity is recommended to affect risk factors of cardiovascular disease, while moderate to high intensity activity may be needed to be able to achieve improvements in cardiovascular function. The recommendation regarding intensity should therefore be guided by a total appraisal of multiple factors. Training should be done at least 2–3 times a week, at least 20 minutes at a time.

The contraindications for testing and aerobic exercise are the same for the elderly as for younger people. The most common absolute contraindications are ECG changes that have recently arisen or a recent heart attack, unstable angina, uncontrolled arrhythmia, total atrioventricular (AV) block and acute cardiac insufficiency. Relative contraindications comprise cardiomyopathies (heart muscle diseases), cardiac valve disease and uncontrolled metabolic diseases. These and other conditions, which are significantly more common among the elderly, mean that testing and consultation regarding participating in physical activity and exercise should be done based on set guidelines.

Muscle function and strength training

Based on the positive effects of strength training in the elderly, this type of exercise should be included as a significant part of the recommendations regarding physical activity and exercise for the elderly. Strength training should always be individualised and be progressive, in other words the load should be gradually adjusted in pace with increasing strength. Exercises should be done 1–2 times a week and include the body's major muscle groups in both the upper and lower extremities. The number of repetitions can be 10–12 for the elderly, although fewer repetitions, 8–10, with a higher load provide a greater effect. The previous recommendation regarding the number of sets to achieve a maximum effect was three, but more recent studies show that positive effects can also be achieved with fewer sets. The same contraindications can be observed for strength training as for aerobic exercise. Progressive strength training often presupposes access to weights or various machines and apparatus that make possible an adjustable resistance, which is why training can advantageously take place at a specially equipped gym. For many elderly persons, particularly those with some level of disability, training should, however, also be done in the form of various functional elements, such as rising from a chair and climbing stairs.

Balance, flexibility and walking ability

Training of balance, flexibility and walking ability is best done through all-round exercise, individually or in a group. Balance can be trained by challenging one's stability and control, such as by standing on one leg or walking in circles, sideways or over obstacles. It is crucial that the balance training be customised so that it provides the optimum effect based on the individual's needs. Different activities place different requirements on balance and training should therefore be done in body positions and movements that are important for the person to be able to function in everyday life.

Flexibility is best maintained by using the entire scale of exercise opportunities, in other words keeping the body going in an all-round way, both with physical activity and exercise, but just as much through various everyday and free-time activities. Regular walks, preferably in varying terrain and on different surfaces, contribute to good balance, flexibility and walking ability.

Psychological function and quality of life

Available data indicates a positive correlation with regard to aerobic fitness and strength training and psychological function, but clear guidelines are lacking with regard to intensity and duration of various types of exercise. Consequently, the recommendations are to stimulate various types of physical activity and exercise, where the individual should be encouraged to find activities and types of exercise that he or she is comfortable with and finds enjoyable. The social environment, in other words where and with whom the activity or exercise is done, is probably also very important to positively affecting memory, cognitive capacity, power of initiative, mood and perceived health.

References

1. American College of Sports Medicine Position Stand. Exercise and physical activity for older adults. *Med Sci Sports Exerc* 1998;30:992-1008.
2. Fiatarone Singh MA. Exercise comes of age. Rationale and recommendations for a geriatric exercise prescription. *J Gerontol Med Sci* 2002;57A:M262-82.
3. Frankel JE, Bean JF, Frontera WR. Exercise in the elderly. Research and clinical practice. *Clin Geriatr Med* 2006;22:239-56.
4. Spirduso WW, Francis KL, MacRae PG. Physical dimensions of aging. 2. edn. Champaign (IL): Human Kinetics; 2005.
5. Porter MM, Vandervoort AA, Lexell J. Ageing of human muscle. Structure, function and adaptability. *Scand J Med Sci Sports* 1995;5:129-42.
6. Latham N, Anderson C, Bennet D, Stretton C. Progressive resistance strength training for physical disability in older people. *Cochrane Database Syst Rev* 2003;2: CD002759.
7. Hunter GR, McCarthy JP, Bamman MM. Effects of resistance training on older adults. *Sports Med* 2004;34:329-48.
8. Lexell J, Downham DY, Larsson Y, Bruhn E, Morsing B. Heavy-resistance training for Scandinavian men and women over seventy. Short- and long-term effects on arm and leg muscles. *Scand J Med Sci Sports* 1995;5:329-41.
9. Suominen H. Muscle training for bone strength. *Aging Clin Exp Res* 2006;18:85-93.
10. Porter MM. Power training for older adults. *Appl Physiol Nutr Metab* 2006;31:87-94.
11. Simpson JM, ed. Postural instability and falling in old age. *Physiotherapy Theory and Practice*. Special Issue 1999;15:60-140.
12. Helbostad J, Sletvold O, Moe-Nilssen R. Effects of home exercises and group training on functional abilities in home-dwelling older persons with mobility- and balance problems. A randomized study. *Aging Clin Exp* 2004;85:993-9.
13. Gillespie LD, Gillespie WJ, Robertson MC, Lamb SE, Cumming RG, Roew BH. Interventions for preventing falls in elderly people. *Cochrane Database Syst Rev* 2003;4:CD000340.
14. Tinetti ME, Baker DI, McAvay G, Claus EB, Garret G, Gottschalk M, et al. A multifactorial intervention to reduce the risk of falling among elderly living in the community. *N Engl J Med* 1994;331:821-7.
15. Tinetti ME, Richman D, Powell L. Falls efficacy as a measure of fear of falling. *J Gerontol Psych Sci* 1991;45:239-42.
16. Buchner DM, Larson EB, Wagner EH, Koepsell TD, deLateur BJ. Evidence for a non-linear relationship between leg strength and gait speed. *Age Ageing* 1996;25:386-91.
17. Bean JF, Kiely DK, Herman S, Leveille SG, Mizer K, Frontera WR, et al. The relationship between leg power and physical performance in mobility-limited older people. *J Am Geriatr Soc* 2002;50:461-7.
18. Spirduso WW, Poon LW, Chodzko-Zajko WJ. Exercise and its mediating effects on cognition. Champaign (IL): Human Kinetics; 2007.

19. Heyn P, Abreu BC, Ottenbacher KJ. The effects of exercise training on elderly persons with cognitive impairment and dementia. A meta-analysis. *Arch Phys Med Rehabil* 2004;85:1694-704.
20. Laurin D, Verreault R, Lindsay J, MacPherson K, Rockwood K. Physical activity and risk of cognitive impairment and dementia in elderly persons. *Arch Neurol* 2001; 58:498-504.
21. Larson EB, Wang L, Bowen JD, McCormick WC, Teri L, Crane P, et al. Exercise is associated with reduced risk for incident dementia among persons 65 years of age and older. *Ann Intern Med* 2006;144:73-81.
22. Strawbridge WJ, Deleger S, Roberts RE, Kaplan GA. Physical activity reduces the risk of subsequent depression for older adults. *Am J Epidemiol* 2002;156:328-34.
23. Lindwall M, Rennemark M, Halling A, Berglund J, Hassmén P. Depression and exercise in elderly men and women. Findings from the Swedish national study on aging and care. *J Aging Phys Act* 2006;15:41-55.
24. Brown DW, Brown DR, Heath GW, Balluz L, Giles WH, Ford ES, et al. Associations between physical activity dose and health-related quality of life. *Med Sci Sports Exerc* 2004;36:890-96.
25. Acree SL, Longfors J, Fjeldstad AS, Fjeldstad C, Schank B, Nickel KJ, et al. Physical activity is related to quality of life in older adults. *Health Qual Life Outcomes* 2006;4:37-41.
26. Christmas C, Andersen RA. Exercise and older patients. Guidelines for the clinicians. *J Am Geriatr Soc* 2000;48:318-24.
27. Mazzeo RS, Tanaka H. Exercise prescription for the elderly. Current recommendations. *Sports Med* 2001;31:809-18.
28. American College of Sports Medicine Position Stand. Physical activity programs and behavior counseling in older adult populations. *Med Sci Sports Exerc* 2004;36:1997-2003.

15. Alcohol dependence/abuse

Author

Helena Prochazka, MD, PhD, Swedish Armed Forces Centre for Defence Medicine, Gothenburg, Sweden

Summary

Alcohol dependence and abuse is often a chronic condition, even if there may be long periods of sobriety. Excess consumption of alcohol is a known risk factor in the development and aggravation of many other diseases, and treatment must therefore be oriented to both the addictive state itself and the complications of alcohol abuse. Structured physical training constitutes an important addition to therapy in the withdrawal phase and in the following treatment of anxiety and depression, as well as in the treatment of secondary diseases such as diabetes and cardiovascular disease. The training should include fitness training starting at low intensity and gradually working up to higher intensity, strength training and coordination training. Suggested prescription:

	Type of training	Intensity	Frequency (times/week)	Duration (min./session)
Withdrawal treatment	Low intensity aerobic fitness training	40–60% of APM*	4	15
	Strength training	40–70% of 1 RM**	3	20
Subsequent treatment	High intensity aerobic fitness training	50–75% of APM*	3	30
	Strength training	50–70% of 1 RM**	2	30
	Ball sports	50–85% of APM*	2	40

* APM = Age Predicted Maximal Heart Rate (220 – age)

** RM = Repetition Maximum. 1 RM corresponds to the maximum weight load that can be lifted through the entire exercise movement one time.

Definition

The diagnoses of alcohol *dependence* and *abuse* are made based on patient history with the aid of the Diagnostic and Statistical Manual of Mental Disorders (DSM) IV (1). *Abuse* exists when a person meets one of the following criteria within a 12-month period:

1. Recurrent use of alcohol resulting in a failure to fulfil major obligations at work, school, or home.
2. Recurrent use of alcohol in hazardous situations, such as at work or driving while intoxicated.
3. Recurring legal problems resulting from the substance use.
4. Continued use despite recurring problems.

The diagnosis of *dependence* requires that the person meet three of the following seven criteria within a 12-month period:

1. Needs increasing amounts of alcohol to achieve the same effect (increase in tolerance).
2. Experiences withdrawal symptoms when not drinking.
3. Drinks more or for longer periods than intended.
4. Persistent desire or unsuccessful efforts to cut down on drinking.
5. Spends a significant amount of time drinking, planning to drink, or recovering from drinking alcohol.
6. Important social, occupational or recreational activities are neglected.
7. Continued use despite physical or psychological problems.

In conclusion, *dependence* and *abuse* are characterised by the individual losing control over his or her consumption, which has led to significant disability or suffering for the individual and/or his or her surroundings.

Prevalence/Incidence

Despite the fact that the consensus in various studies on alcohol use is only 50–70 per cent, there is agreement that alcohol problems, in the form of hazardous consumption, abuse and dependence, are a common occurrence and growing problem in Sweden. The incidence of alcoholism in men is approximately 2.5 per 1000 per year. The prevalence is estimated at 5–9 per cent for men and about 1.5 per cent for women. The prevalence in Europe is between 0.6 and 20 per cent, as varying definitions are used (2). In the USA, an estimated 10–15 per cent of men and 5 per cent of women suffer from chronic alcoholism, of whom one quarter are actively using (3).

Cause and risk factors

Alcohol dependence has a multifactorial genesis. Individuals with so-called type 2 alcoholism (4) have a characteristically clear genetic predisposition and other characteristics have been found in the form of early onset of alcoholism, “novelty seeking” behaviour, aggression, a tendency towards hypoglycemia, low levels of serotonin metabolite 5-HIAA (5-hydroxyindoleacetic acid) in cerebrospinal fluid and low MAO-B (monoaminooxidase B) activity in the thrombocytes. This type of alcohol dependence is also called *primary alcoholism* and occurs to a larger extent in men. *Secondary alcoholism* or type 1 alcoholism traditionally occurs more in women, and develops secondarily to other psychological disorders such as depression and anxiety, which in turn can present in conjunction as a response to a crisis and adjustment disorders (5).

Symptoms and diagnosis

A diagnosis is made in the first instance according to the criteria listed above under *Definition*. When a diagnosis cannot be made with the aid of a specific patient history, a somatic examination and blood analysis can serve as a guide. Swelling of the parotid glands, an enlarged liver, elevated blood pressure, elevated blood levels of AST (Aspartate aminotransferase), ALAT (Alanine aminotransferase), gamma GT (Glutamyl transpeptidase), MCV (mean erythrocyte volume), CDT (Carbohydrate deficient transferrin), urate and IgA (Immunoglobulin A) confirm suspicions of problems with alcohol.

Current treatment principles

With the exception of treatment of withdrawal symptoms, all other pharmacological therapies should be given in combination with psychosocial interventions.

Pharmacological treatment

Withdrawal treatment

The primary goal is treatment and prevention of life-threatening alcoholic delirium, epileptic seizures and other withdrawal symptoms. The most common treatment in Sweden is either a benzodiazepine or chlome-thiazole (Hemineurin) programme, often supplemented with anti-spasmodic drugs and vitamin B.

Alcohol dependence and relapse prevention therapy

There are two approved substances for pharmacological treatment of alcohol dependence in Sweden: acamprosate (Campral) and naltrexone (Revia). The former increases the number of completely sober individuals by reducing the craving for alcohol, and the latter reduces alcohol consumption. A third pharmaceutical, disulfiram (Antabuse), a so-called “aversion” drug, does not have the anti-craving effect and is only used under controlled conditions.

Psychosocial treatment

The Swedish Council on Technology Assessment in Health Care's (Statens beredning för medicinsk utvärdering, SBU) compiled knowledge base (3) compares a host of psychosocial treatment methods. The most effective of these share a number of features, such as a clear structure and well-defined measures with detailed guidelines. Included in such methods are cognitive behavioural therapy and a 12-step programme that follows the Minnesota Model, often combined with self-help programmes such as Alcoholics Anonymous (AA) and other motivation-boosting measures.

In people with psychological disorders, alcoholism is treated parallel to the mental disorder, and in homeless persons a combination of behavioural therapy methods with structured and coordinated support is given.

Effects of physical activity

Organised physical activity and training, a part of several complex treatment therapies, have proven positive effects in both the treatment of the acute withdrawal phase and in treatment of alcohol craving, anxiety and depression. Continued regular physical activity then has an anxiety-alleviating and depression-prevention effect, as well as a significant psychosocial function in that it fills the emptiness following alcoholism and builds up the individual's self-confidence (6).

Withdrawal phase

One English intervention study using a cross-over design (7) showed that brief sessions of exercise (10 minutes of cycling) in the withdrawal phase led to a cautiously mild, but temporary, effect on alcohol craving. Otherwise, clinical experience suggests that physical training in the acute stage alleviates above all somatic withdrawal symptoms. Several studies have shown a significant decrease of the shakes, sweating and reduction of anxiety following organised low-intensity fitness training (8, 9). Another important effect of physical activity in the acute detoxification stage is better sleep (10).

Long-term effects

Restoration/improvement of physical fitness is the most important long-term effect of physical activity and training, and serves as a basis for other physical improvements in heart function, peripheral circulation, blood sugar levels and body image, accompanied by a reduction in neurological symptoms. Regular physical activity has a positive impact on mood (11–13) and relieves anxiety. A study on movement therapy (14) covering three basic movement elements (running, jumping and ball sports) measured improvement in somatic parameters (increased strength and reduced neurological symptoms) as well as improved stress and anxiety management. The long-term effects of physical activity and exercise are, however, completely dependent on regularity.

Indications

There is a lack of controlled epidemiological studies on the relationship between the effects of physical activity and development of alcoholism. Regarding the indications for secondary prevention, individually adapted physical activities are recommended for all patients regardless of age.

Prescription

Light physical training begins after the initial withdrawal problems have subsided, which generally occurs in the second week of detoxification. After somatic examination and a functional test, the first step is low-intensity aerobic fitness training, and, after a few days, combining this with strength training. Subsequent exercise programmes span several months time and aim to establish a permanent change in the way of life. They can therefore be started at a day unit and then followed up in out-patient care, with evaluation of somatic parameters and functional tests (Table 1).

Table 1. Types of physical training for alcohol abuse.

	Purpose	Type of exercise	Intensity	Duration (min./session)	Frequency (times/week)	
Withdrawal treatment	Relieve withdrawal symptoms	LFT*	40–60% of APM***	15	4	
	Improve sleep quality	LFT: Walking, strength training	35–70% of APM	20	3	
Subsequent treatment	Improve aerobic fitness	HFT**:	50–75% of APM	40–70% of 1 RM****	30	3–5
				Cycling, walking, running		
	Increase strength	Strength training	50–70% of 1 RM	30	2–4	
	Increase aerobic endurance	Moderate intensity, exercise of choice	50–70% of APM	45–60	2–4	
	Improve coordination	Ball sports	50–85% of APM	40	2–4	

* LFT = Low Intensity Aerobic Fitness Training.

** HFT = High Intensity Aerobic Fitness Training.

*** APM = Age Predicted Maximal Heart Rate (220 – age).

**** RM = Repetition Maximum. 1 RM corresponds to the maximum weight that can be lifted through the entire exercise movement one time.

Functional tests

Somatic examination and function testing should always be used as a guide for structured physical training. Follow-up and ongoing evaluation of measurable parameters are of great importance even as a motivation-boosting and supporting component of the treatment. Before training begins and at evaluation, a standardised 6-minute walk test (6MW test) (15) is most suitable. The test can be conducted either on a treadmill or in a hallway with a straight stretch of at least 30 metres. See the proposed reporting sheet in Figure 1. The Borg CR10 scale (16) is used for assessment of subjective symptoms of fatigue and breathing difficulties (for more on this, see the chapter on “Assessment and management of physical activity”). The scale is shown to the patient, who responds with a perceived level of difficulty/discomfort between 1–10. The instructions “rate your shortness of breath” and “rate your level of fatigue” are repeated in the beginning and at the end of the test.

Figure 1. Proposed Reporting Sheet for 6MW Test.

Patient name _____

National ID no. _____ Date _____

6MWD no. _____ Monitored by _____

Age _____ Sex _____

Weight (kg) _____ Height (m) _____ BMI (kg/m²) _____

Blood pressure _____ Resting heart rate _____

Medications before test (drug, dose and time) _____

	Beginning of 6MW test	End of 6MW test
Time		
Heart rate (beats/min)		
Dyspnoea (Borg scale)		
Fatigue (Borg scale)		

Stopped or rested before end of test? No _____ Yes, reason _____

Symptoms during test: Chest pain, nausea, dyspnoea, cramps, dizziness, other

Number of lengths: _____ (x 60 m) + last partial length (m): _____ = _____ m

Total distance in 6 minutes: _____ metres

Percentage difference compared to first 6MW test: +/- _____ %

Comments _____

Contraindications and risks

Contraindications for physical training in alcohol dependence are dependent on the patient's cardiovascular, pulmonary and neurological status. *Absolute contraindications* include severe cardiac insufficiency, uncontrolled arrhythmia, high blood pressure, unstable angina, severe obstruction, recent heart infarction and severe neuropathy. The *relative contraindications* include cardiomyopathy, severe diabetes and other metabolic diseases, as well as complications such as acute pancreatitis, acute hepatitis and portal hypertension.

Interactions with drug therapy

The approved medications for alcohol treatment, acamprosate (Campral), naltrexone (Revia) and disulfiram (Antabuse), do not constitute contraindications to physical activity. To the contrary, regular exercise can relieve drug side-effects such as headache and fatigue.

Proposed intervention programme based on fitness treatment

At Charter Hospital in Texas, Fridinger and colleagues (17) have come up with a treatment programme for chemical substance abusers, which can be applied in most abuser/addiction groups. The programme's philosophy is based on a combination of mental and physical education aimed at increasing the chances of total recovery, and builds on four phases.

1. Somatic/psychiatric examination
2. Screening of fitness and nutritional status
3. Organised exercise activities
4. Education sessions

Somatic and psychiatric examination forms the basis for planning the patient's participation in common physical activities. The individual need for detoxification method and time frame is planned and a contract is written with the patient.

Within 72 hours after admission, the patient goes through the screening phase, which includes functional tests and a self-assessment of his or her motivation according to step 4 of the AA 12-step programme. An individually based nutrition plan with the consumption of fats, cholesterol, carbohydrates and fibre is created with the aid of computer software.

The next phase contains daily exercise and scheduled activities. Every morning starts with 15 minutes of stretching, followed by a 15-minute walk outside or an indoor activity. In addition to these daily activities, the programme includes 20–30 minutes of exercise training, 3–4 times per week, comprising either ergometer cycling, walking, jogging on

a treadmill, or taking part in an exercise class. In addition to these activities, are three compulsory occasions focusing on muscle strength (e.g. strength training with weights).

The final phase is education in six key areas.

1. The importance of physical activity for well-being
2. Education of the risk factors for relapse
3. Stress management
4. The significance of smoking in relapse to abuse of other substances
5. Nutrition
6. The psychological benefits of physical activity

References

1. American Psychiatric Association. Diagnostic and statistical manual of mental disorders. Washington DC. American Psychiatric Association; 1994.
2. Prytz H. Alkoholismens epidemiologi [The epidemiology of alcoholism]. In: Nordén Å, Ed. Alkohol som sjukdomsorsak [Alcohol as a cause of disease]. Stockholm: Norstedts förlag; 1988. p. 64-70.
3. SBU (Swedish Council on Technology Assessment in Health Care). Behandling av alkohol- och narkotika-problem. En evidensbaserad kunskapssammanställning. [Treatment of alcohol and drug problems. A compilation of evidence-based knowledge.] Report no. 156/1. Stockholm: SBU; 2001.
4. Virkkunen M, Linnoila M. Serotonin in early onset, male alcoholics with violent behaviour. *Annals in Medicine* 1990;22:327-31.
5. Ottosson J. Alkoholism [Alcoholism]. In: Psykiatri [Psychiatry]. 5th edn. Stockholm: Liber; 2000. p. 99-134.
6. Martinsen EW. Fysisk aktivitet for sinnets helse [Physical activity for mental health]. *Tidsskr Nor Laegeforen* 2000;120: 3054-6.
7. Ussher M, Sampuran AK, Doshi R, West R, Drummond DC. Acute effect of a brief bout of exercise on alcohol urges. *Addiction* 2004;99:1542-7.
8. Palmer J, Vacc N, Epstein J. Adult inpatient alcoholics. Physical exercise as a treatment intervention. *J Stud Alcohol* 1988;49:418-21.
9. Sinyor D, Brown T, Rostand L, Sereganian P. The role of physical fitness program in the treatment of alcoholism. *J Stud Alcohol* 1982;43:380-6.
10. King AC, Oman RF, Brassington GS, Bliwise DL, Haskell WL. Moderate-intensity exercise and self-rated quality of sleep in older adults. *JAMA* 1997;277:32-7.
11. Meyer T, Broocks A. Therapeutic impact of exercise on psychiatric diseases. Guidelines for exercise testing and prescription. *Sports Med* 2000;30:269-79.
12. Byrne A, Byrne DG. The effect of exercise on depression, anxiety and other mood states. A review. *J Psychosom Res* 1993;37:565-74.
13. Sexton H, Maere Å, Dahl NH. Exercise intensity and reduction in neurotic symptoms. *Acta Psychiatr Scand* 1989;80:231-5.
14. Tsukue I, Shohoji T. Movement therapy for alcoholic patients. *J Studies Alcohol* 1981; 42:144-9.
15. ATS statement. Guidelines for the Six-Minute Walk Test. *Am J Respir Crit Care Med* 2002;166:111-7.
16. Borg G. Psycho-physical bases of perceived exertion. *Med Sci Sports Exerc* 1982; 14:377-81.
17. Fridinger F, Dehart B. A model for the inclusion of a physical fitness and health promotion component in a chemical abuse treatment program. *J Drug Education* 1993;23:215-22.

16. Anxiety

Authors

Egil W. Martinsen, MD, PhD, Professor, Aker University Hospital, Oslo University, Oslo, Norway

Jill Taube, MD, Centre for Family and Community Medicine, Karolinska Institutet, Stockholm, Sweden

Summary

There is limited scientific documentation on the effects of physical activity on anxiety disorders. Anxiety syndromes are more prevalent among physically inactive than active people. The alleviating effect of physical activity on state anxiety is well-documented, while its effect on trait anxiety is less well defined. The documentation available on physical activity as an alternative or complement to other treatment methods relates mainly to panic disorder and agoraphobia, but it is probably useful in generalised anxiety disorder as well. Various forms of physical activity appear to have the same effect.

Definition

Anxiety encompasses a broad spectrum of feelings, from apprehension and worry to fear and panic. The anxiety often results in physical symptoms, indicating overactivity of the sympathetic nervous system: breathing difficulties, palpitations, sweating, dizziness, nausea and symptoms of muscle tension such as shaking and restlessness. Many people with anxiety experience a feeling of panic and their behaviour is often characterised by a tendency toward withdrawal and avoidance.

Anxiety is basically a survival instinct, but can become dysfunctional as a result of improper learning or an inherited biological vulnerability. The concept of anxiety is also used to describe normal emotional reactions in conjunction with severe stress, and there is a substantial grey zone between normal anxiety and anxiety disorders.

The diagnostic categories are generally consistent with DSM-IV and ICD-10. According to DSM-IV (1), anxiety disorders can be divided into the following diagnostic categories:

- **Panic disorder** is characterised by a sudden attack of rapidly increasing anxiety and palpitations, breathing difficulties, chest pain, dizziness and other physical symptoms. These physical symptoms are often interpreted as indications of a serious illness. During a panic attack, the person develops a severe fear of fainting, dying, losing their mind or losing control.
- **Agoraphobia** literally translates as a fear of open places, but this does not cover the full meaning of the concept. In ancient Greece, “agora” was the place where young men would meet to discuss social matters and politics. Modern equivalents to an “agora” are shopping centres, shops, cafés, restaurants, public meeting places or public transportation. The reason that agoraphobic individuals avoid these places is the fear of having a panic attack or panic-like symptoms.
- **Social phobia or social anxiety disorder** is characterised by a fear of making a fool of oneself or doing something embarrassing in social situations where it would attract attention. Patients with social phobia believe that other people are watching and judging them, and look down on them. Most patients with social phobia are worried that others will see how anxious they are, i.e. that they are shaking, sweating and embarrassed. Other patients have a fear of not being clever enough, for example, that they will forget things or lose their voice when speaking. These patients will either endure social situations with distress or avoid them altogether, limiting their prospects of functioning at work or in social situations.
- **Specific phobias** are characterised by anxiety in connection with situations or objects that most people would not be afraid of. For example, an animal such as a mouse, spider, bird, cat or dog or in a special situation, i.e. a fear of heights, thunder or the sight of blood. Individuals with specific phobias usually lessen their suffering by avoiding situations where they are likely to be exposed to their fears.
- **Obsessive compulsive disorder or obsessional neurosis** are characterised by compulsive actions and/or obsessive thoughts. A compulsion is when something is done for too long and as a ritual, often to neutralize an obsessional thought and the distress it involves. Common compulsions include excessive cleaning and repeated checking of doors, on-off switches, etc. An obsession is a persistent, unwanted and intrusive thought or impulse that the patient is unable to ignore. Such thoughts often entail a fear of hurting other people, catching a contagious disease, or being the cause of negative events.
- **Post-traumatic stress disorder** affects people who have been exposed to a disastrous event, whereupon they relive the trauma either while awake or as a nightmare. The traumatic event could be a traffic accident or a rape. These patients are in a constant state of hypervigilance and avoid situations or objects that remind them of the traumatic event.

- In the case of **generalised anxiety disorder**, the anxiety is not limited to a specific situation but is general and persistent. Patients with generalised anxiety disorder have unrealistic concerns that something bad will happen to them or their close family and friends. Common complaints include a constant feeling of worrying, apprehension, shaking, muscle tension, sweating, dizziness, palpitations and upset stomach.

The scientific literature usually differentiates between state anxiety, i.e. anxiety relating to the current situation, and trait anxiety, which relates to the character and personality of an individual (2).

Diagnosis is based on clinical talks using structured clinical interview guides developed to increase the reliability of the diagnosis, but no objective tests exist. Anxiety disorders often occur in connection with other mental disorders such as depression and drug abuse. Anxiety involves subjective feelings of distress and avoidance behaviours that often lead to underperformance at school or in the workplace.

Prevalence/Incidence

Nearly 20 per cent of all people will experience an anxiety disorder at some time in their lives, and approximately 10 per cent suffer from anxiety at any given point in time. Social phobia and specific phobias are the most common anxiety disorders (3, 4).

Causes and risk factors

There is a certain genetic disposition, which is most evident in obsessive compulsive disorder. Other risk factors include having experienced separation, physical assault, absence of loving care in childhood, and psychosocial stress later in life.

Prognosis

The severity of an anxiety disorder varies over time, with occasional spontaneous improvements. However, without any form of treatment, many patients become chronically disabled.

Treatment

Today's treatments for anxiety consist mainly of psychotherapy and medications. The benefits of exposure therapy have been known since the 1960s and cognitive behavioural therapy (CBT) is the most effective and best documented method for all anxiety disorders. Antidepressants are the most widely used drug therapy for anxiety disorders. The effects of both tricyclic antidepressants and selective serotonin reuptake inhibitors are well-documented (5).

Minor tranquilizers like benzodiazepines (Valium, Sobril and similar) are fast-acting and have an anxiety-inhibiting effect. The disadvantage with these drugs is that they may become addictive and impede the learning process and, hence, very limited use is recommended. Antidepressants, on the other hand, are not addictive and do not impede the learning process. It is common for patients to relapse after a complete course of medication, and long-term treatment is often necessary to achieve the desired effect. However, the effects of cognitive behavioural therapy usually remain after a completed treatment (5).

A number of patients report short-term effects from alcohol, and self-medication with alcohol is not uncommon. The disadvantage of using alcohol to relieve anxiety is that the symptoms often become worse when the level of alcohol in the blood decreases, leading to the added risk of alcohol misuse and dependence.

Effects of physical activity

Prevention

A major epidemiological study on a representative sample of people in the USA showed that those who were physically active suffered less anxiety: panic disorder, social phobia, specific phobias and agoraphobia (6). As this is a cross-sectional study, it does not establish a cause-effect relationship: Does physical inactivity cause anxiety or does anxiety, and especially the tendency to avoid, cause physical inactivity? Longitudinal studies are needed to examine the cause-effect relationship and no such studies have been published to date.

A German study looked at the question from a different angle (7). The objective of the study was to see whether light physical activity could prevent panic attacks in 15 healthy study subjects. The subjects performed 30 minutes of fitness training or were asked to rest before being injected with a panic-inducing substance. 12 of the 15 subjects in the resting group had a panic attack compared to only 6 after physical activity. This indicates that physical activity may prevent panic attacks in healthy individuals, but it is not yet clear whether it also applies to patients with panic disorder.

Treatment

A number of randomised control trials involving subjects with normal or elevated anxiety levels, who do not fulfil the criteria for a psychiatric diagnosis, have shown physical activity to reduce the level of anxiety and tension (state anxiety). The effect usually occurs 5–15 minutes after the training is finished and lasts on average 2–4 hours. How physical activity affects the more chronic character-related, trait anxiety remains uncertain (2).

Few studies have been carried out on patients with anxiety disorders. There are a number of studies on panic disorder and agoraphobia, but only one study on generalised anxiety disorder and social phobia.

Panic disorder and agoraphobia

A British psychiatrist by the name of Orwin (8, 9) conducted some interesting studies at beginning of the 1970s. He studied patients with agoraphobia who had a fear of travelling by bus. Every time the patients got on a bus, they experienced severe anxiety, with palpitations, increased heart rate, sweating and a feeling of not being able to breathe. As a result, they had been avoiding using buses and similar situations for a long time. Orwin asked the patients to approach the bus in a different way. He asked them to run to the bus stop, so that they would have an increased heart rate and palpitations, and be sweating and out of breath before they got on the bus. This meant that the patients' physical reactions were already activated to a maximum, eliminating the possibility of further reactions. The anxiety of riding the bus thus subsided as the physical symptoms were ascribed to the running and not the bus.

In a Norwegian study without a control group, patients with panic disorder and agoraphobia at a psychiatric hospital took part in an 8-week treatment programme. The main part of the treatment consisted of physical activity, with 1 hour of fitness training 5 days a week, and dynamic group therapy 3 times a week. While anxiety levels decreased significantly during the treatment period, at the 1-year follow-up most patients were found to have relapsed (10).

A group of German researchers carried out a randomised controlled trial on patients with panic disorder (11). The patients were divided into three groups. The first group participated in regular physical activity in the form of fitness training. The second group received antidepressants (clomipramine), and the third group was given placebo tablets, thereby constituting a control group. The study went on for a period of 8 weeks. The findings showed that physical training and antidepressants had a greater effect than the placebo tablets, and that the drug therapy was slightly more effective than the physical training. The drop-out rate for the training group tended to be higher than for the patients who received clomipramine. The results of this study are consistent with the Norwegian study. However, the German researchers did not follow up their patients, and it is therefore not known whether the progress made by the patients remained once the treatment had finished.

In another study, patients with panic disorder and agoraphobia were randomly assigned either conventional treatment by a general practitioner, or asked to participate in a 16-week lifestyle programme led by an occupational therapist. The lifestyle programme involved a review of each patient's fluid intake, diet and eating habits, physical activity, and the use of caffeine, alcohol and nicotine. Lifestyle changes were discussed, recorded and followed up (12). After 20 weeks, the patients in the lifestyle programme had significantly reduced anxiety levels and fewer panic attacks. After 10 months, the lifestyle programme group still showed better results, though the difference between the two groups was no longer significant. Although not solely a study on training, physical activity was an important part of the intervention.

Generalised anxiety disorder

A group of patients with generalised anxiety disorder also participated in the Norwegian study referred to above (10). The reduced levels of anxiety experienced by these patients throughout the treatment period persisted at the 12-month follow-up. No other studies on generalised anxiety disorder have been found. Before a randomised controlled trial on the effects of different treatments is carried out, it is difficult to determine the therapeutical value of physical training.

Other anxiety disorders

The Norwegian study also included a group of patients with social phobia. This group did not show any change either during the treatment period or at follow-up. No other studies on patients with social phobia and physical activity have been published to date. Neither have the effects of physical activity on specific phobias, obsessive compulsive disorder and post-traumatic stress disorder been studied. However, physical activity is unlikely to have any greater effect on specific phobias and obsessive compulsive disorder. There are many similarities between post-traumatic stress disorder and panic disorder, and it is therefore theoretically possible that physical activity may have a beneficial effect in post-traumatic stress disorder.

To sum up, a number of the studies appear to indicate that physical activity can prevent anxiety. A transient decrease in the level of anxiety after physical activity has been shown in a number of studies in healthy individuals with and without elevated anxiety levels. Physical activity can be used as a treatment alternative for panic disorder and agoraphobia, and perhaps even for generalised anxiety disorder. The other anxiety disorders have been studied only to a limited extent.

Patients with anxiety disorders can do normal physical training, and have a normal physiological response to the training.

Potential mechanisms

There are various hypotheses about how physical activity affects anxiety levels, with physiological, neurobiological and psychological hypotheses having been put forward.

- **Improved physical condition.** People in good physical condition are generally in better health and have greater resistance to disease and other pressures. Well-trained individuals are able to cope with the everyday challenges of life, by using a lower percentage of their maximal heart rate, whereupon the heart rate normalises more rapidly after a stressful situation.
- **Effects of neurotransmitters on the brain.** Dopamine, serotonin, noradrenaline and gamma-aminobutyric acid (GABA) levels may be affected. This is the basis of medical treatment of panic disorder, and the notion that physical activity has an impact on these systems is to some extent supported by animal experiments.

- **An increased release of beta-endorphins** may have a calming effect.
- **Reduced activation of the hypothalamic-pituitary-adrenal (HPA) axis** plays an important role in stress response regulation.
- **The thermogenic hypothesis.** Body temperature rises by 1–2 degrees during vigorous physical exertion. It is possible that the active increase in body temperature through exercise may have the same calming effect as a passive rise in temperature, e.g. in a sauna.
- **The distraction hypothesis** is based on trials conducted by Bahrke and Morgan (13). They found that the level of anxiety decreased after 30 minutes on an exercise bike, but that the reduction in anxiety was just as great after subjects had rested in a soundproof room. The result can be explained by distraction. Physical activity has been shown to divert negative thoughts and, hence, anxiety and distressing thoughts.
- **Cognitive reinterpretation** is yet another psychological hypothesis. According to the cognitive theory of panic disorder, the reason for patients developing and maintaining a panic disorder is that they interpret physical symptoms as a “disaster”. Palpitations, for example, are seen as a sign of heart disease and impending death (14). The physical reactions experienced during a panic attack and physical activity are quite similar in character, and both are due to acute activation of the sympathetic nervous system. However, when patients get used to the physical reactions experienced during physical activity, they learn to interpret them as less catastrophic.

Indications

Primary prevention

Everything considered, physically active individuals appear to run less risk of developing an anxiety disorder.

Secondary prevention

Physical activity can be recommended as an alternative or supplement to the ordinary treatment of panic disorder, with or without agoraphobia, and perhaps also for generalised anxiety disorder and for increased anxiety levels in healthy individuals.

Prescription

One randomised controlled trial compared fitness training (mainly jogging and brisk walking) with the training of flexibility, coordination and relaxation in patients with anxiety disorders, such as panic disorder and agoraphobia (15). After 8 weeks, significantly decreased levels of anxiety and avoidance were noted in both groups. There was not much difference between the training methods used. Sexton, Maere and Dahl (16) compared 8 weeks of walking versus jogging in hospital patients admitted for anxiety and depression treatment. At the end of the 8-week programme, both groups showed the same improvement in anxiety levels. At the 6-month follow-up, however, most joggers had stopped jogging, while the walkers had continued walking. The patients with the highest aerobic fitness had the lowest levels of anxiety.

Consequently, there does not appear to be a difference between different types of physical activity with regard to psychological value. Physical training should therefore be planned according to the interests and enjoyment of the patient.

The fitness training can, for example, be made up of brisk, 30-minute daily walks, or 30 minutes of jogging three times a week, at an intensity of 60–80 per cent of maximal oxygen uptake (talking speed). Other alternatives include strength, mobility or flexibility training of the same duration and frequency. Patients should start with low-intensity training, to facilitate tolerance.

Special considerations

One important consideration is that, paradoxically, many patients experience increased anxiety when they start training. This is explained by the activation of the sympathetic nervous system during physical activity producing symptoms like increased heart rate, palpitations, sweating and breathlessness. The same physical reactions occur in patients with severe anxiety. Thus many patients with an anxiety disorder avoid physical activity, as they feel it leads to an increase in anxiety. If informed about this phenomenon in advance, however, most patients tend to overcome these symptoms, with the majority able to carry on with their physical activity. Physical activity combined with education is a good way of getting to know one's bodily symptoms, which are less frightening when they occur under normal circumstances, such as training. Learning that the anxiety decreases when the patient persists rather than avoids a certain situation is also an important experience (17).

At one time, physical activity was generally thought to trigger panic attacks in the same way as lactic acid. Two out of three patients with panic disorder do suffer panic attacks when injected with lactic acid, but, although exposed to lactic acid during physical activity, panic attacks during exercise are very rare. In a literature review, O'Connor, Raglin and Martinsen (18) found only five panic attacks reported in connection with 444 training sessions in 420 patients with panic disorder. This yields a panic frequency of about 1 per cent, which is much lower than the 67 per cent reported by infusion studies. Consequently, an intravenous injection of lactic acid appears to produce a different effect than the lactic acid released naturally during intense physical activity.

Some patients who train regularly may experience increased anxiety when forced to stop or cut down on their training, for example, because of an injury. This can be unpleasant, but usually disappears after a time. This can be explained by the patients having become physically dependent on the training (17).

Functional test/Need for health check-ups

For some individuals with an anxiety disorder, in particular panic disorder, the physical symptoms can be painful – palpitations and chest pains are common. These symptoms are similar to those seen in patients with heart disease. Patients displaying symptoms such as these should undergo a physical examination before training is commenced. A standard medical examination and an ECG and thyroid hormone test (T4) is usually sufficient. If in doubt, the patient should be referred to a heart specialist. It is wise to conduct a thorough examination on one occasion. Patients often become more anxious when repeated examinations are carried out by uncertain doctors.

There are a number of patient questionnaires that can be used to record anxiety levels, to determine whether the training is beneficial or not.

Interactions with drug therapy

The most important group of drugs used to treat anxiety disorders are antidepressants, and the use of antidepressants or benzodiazepines in no way hinder the patient from being physically active. A possible adverse effect of some antidepressants is a fall or rise in blood pressure. In such cases, the blood pressure should be monitored, but this seldom stops the patient from training. Some patients may need a limited supply of beta blockers for social phobias, such as stage fright. Beta blockers can sometimes restrict physical capacity somewhat, mainly symptoms like tired legs, though this is not associated with any risk (19).

Contraindications

There are no contraindications to physical activity in physically healthy persons with anxiety disorders.

Risks

A small number of patients feel that their anxiety disorder deteriorates when they first begin to train. This may be unpleasant but poses no danger. Unless the patient has concurrent physical illnesses that render physical activity impossible, there is no risk involved with physical activity and training.

References

1. American Psychiatric Association. Diagnostic and Statistical Manual of Mental Disorders 4th edn. Washington (DC): American Psychiatric Association; 1994.
2. Raglin JS. Anxiolytic effects of physical activity. In: Morgan WP, Ed. Physical activity and mental health. Washington (DC): Taylor & Francis; 1997. p.107-26.
3. Kessler RC, McGonagle KA, Zhao S, Nelson CB, Hughes M, Eshleman S. Lifetime and 12-months prevalence of DSM-III-R psychiatric disorder in United States. *Archives of General Psychiatry* 1994;51:8-19.
4. Kringlen E, Torgersen S, Cramer V. A Norwegian psychiatric epidemiological study. *American Journal of Psychiatry* 2001;158:1091-8.
5. Nathan PE, Gorman JM. *A Guide to Treatments that Work*. Oxford: Oxford University Press; 2007.
6. Goodwin RD. Association between physical activity and mental disorders among adults in the United States. *Journal of Preventive Medicine* 2003;36:698-703.
7. Strohle A, Feller C, Onken M, Godemann F, Heinz A, Dimeo F. The acute antipanic activity of aerobic exercise. *American Journal of Psychiatry* 2005;162:2376-8.
8. Orwin A. "The running treatment." A preliminary communication on a new use of an old therapy (physical activity) in the agoraphobic syndrome. *British Journal of Psychiatry* 1973;122:175-9.
9. Orwin A. Treatment of situational phobia. A case for running. *British Journal of Psychiatry* 1974;125:95-8.
10. Martinsen EW, Sandvik L, Kolbjørnsrud OB. Aerobic exercise in the treatment of non-psychotic mental disorder. An exploratory study. *Nordisk Psykiatrisk Tidsskrift* 1989;43:521-9. [Nordic Journal of Psychiatry].
11. Broocks A, Bandelow B, Pekrun G, George A, Meyer T, Bartmann U, et al. Comparison of aerobic exercise, clomipramine and placebo in the treatment of panic disorder. *American Journal of Psychiatry* 1998;155:603-9.
12. Lambert RA, Harvey I, Poland F. A Pragmatic, unblended randomised controlled trial comparing an occupational therapy-led lifestyle approach and routine GP care for panic disorder treatment in primary care. *J Affect Disord*. 2007 Apr;99(1-3):63-71. Epub 2006 Oct 2.
13. Bahrke MS, Morgan WP. Anxiety reduction following exercise and meditation. *Cognitive Therapy and Research* 1978;4:323-33.
14. Wells A. *Cognitive therapy of anxiety disorders. A practice manual and conceptual guide*. John Wiley & Sons: Chichester; 1997.
15. Martinsen EW, Hoffart A, Solberg T. Aerobic and non-aerobic exercise in the treatment of anxiety disorders. *Stress Medicine* 1989;5:115-20.
16. Sexton M, Maere Å, Dahl NH. Exercise intensity and reduction in neurotic symptoms. *Acta Psychiatrica Scandinavica* 1989;80:231-5.
17. Martinsen EW. *Kropp og sinn. [Body and Mind] Fysisk aktivitet og psykisk helse. [Physical activity and mental health]* Bergen: Fagbokforlaget; 2004.

18. O'Connor PJ, Raglin JS, Martinsen EW. Physical activity, anxiety and anxiety disorders. *International Journal of Sport Psychology* 2000;312:136-55.
19. Martinsen EW, Stanghelle JK. Drug therapy and physical activity. In: Morgan WP, Ed. *Physical Activity and Mental Health*. Washington (DC): Taylor & Francis; 1997. p. 81-90.

17. Asthma

Author

Margareta Emtner, PT, PhD, Associate Professor, Uppsala University and Uppsala University Hospital, Uppsala, Sweden

Summary

Reduced physical performance is common in both adults and children with asthma. Chronic obstruction of the airways and an increased sensitivity to different stimuli (e.g. physical exertion) contributes to the reduction in performance. Physical activity is valuable and necessary for all persons with asthma. Physical training improves physical capacity, reduces dyspnoea (shortness of breath), and improves exercise-induced breathing difficulties. People with a low to moderate degree of obstruction can take part in physical training on the same terms as healthy individuals. The training should comprise aerobic exercise (fitness training), strength training and flexibility training (see table below). Suitable activities include swimming, ball sports, cycling, walking and aerobic exercise on land and in the water. The recommendation for people with severe obstruction should be strength training, flexibility training and light physical activity.

Type of training	Intensity	Frequency (times/week)	Duration
Aerobic fitness training	Low intensity: > 55% of max HR* > 40% of VO ₂ max**	≥ 5	≥ 30 min
	High intensity: > 70% of max HR* > 60% of VO ₂ max**	≥ 3	≥ 20 min
Strength training	70% of 1 RM***	≥ 2	8–12 reps, 2–3 sets

* Max HR = Maximal Heart Rate.

** VO₂ max = Maximal Oxygen Uptake.

*** RM = Repetition Maximum. 1 RM corresponds to the maximum weight that can be lifted through the entire exercise movement one time.

Definition

According to the Global Initiative for Asthma (GINA), asthma can be briefly described as follows: Asthma is a chronic inflammatory disorder of the airways, in which the chronic inflammation is associated with airway hyperresponsiveness. The airflow obstruction is often reversible either spontaneously or with treatment (1).

Cause and risk factors

Asthma is a multifactorial and heterogeneous disease. Risk factors include genetic predisposition, atopy (tendency toward allergy) and hyperresponsiveness. In recent years, a relation between overweight and asthma has also been reported in both children and adults (2). Environmental factors such as tobacco smoke and mould and mildew can contribute to development of the disease in children. Approximately 5–15 per cent of people who became asthmatic as adults are classified as occupational asthmatics (3). Among these, are bakery, industry and agricultural workers, hairdressers, and people who have been exposed to welding fumes and solvents.

Prevalence/Incidence

During the past decades, the prevalence of asthma has increased all over the world. According to current Swedish studies, the prevalence in Sweden is approximately 10 per cent (4). The increase is highest in young adults, then drops and is lowest in upper middle age, to increase once again. In adults, asthma is considered a chronic disease, while children often grow out of it (5).

Pathophysiology

Asthma is a disease characterised by an inflammation of the bronchial tubes. A large number of inflammatory cells, including the mast cells, eosinophils, T lymphocytes, macrophages and neutrophils, are involved (1). Inflammation can occur after exposure to things such as allergens (allergy-causing substances) and obstruction is due to a constricting of the smooth muscle, oedema, remodelling (structural changes in the mucous membranes of the airways) and an increase in mucus production (1).

Exercise-induced bronchial obstruction

Most people who have asthma experience difficulty breathing during physical exercise, which is due to exertion causing the airways to constrict (6). This is called exercise-induced airway obstruction. Exercise-induced airway obstruction is defined as a case of a PEF (peak expiratory flow) greater than or equal to 15 per cent, or a FEV₁ (forced expiratory volume in one second) greater than or equal to 10 per cent, in connection with physical exertion (6). The problems arise during physical exertion, or commonly 5–15 minutes

afterwards, and persist for 30–60 minutes. Often symptoms disappear by themselves. The degree of exercise-induced breathing difficulty varies with the intensity of the exercise, the type of activity, and the surroundings (environment) in which the training occurs. Running, for example, produces more problems than jogging or walking. The problems are greatest when training in cold, dry air, and least when training in warm, humid environments. Air pollutants have been shown to increase the level of exercise-induced breathing difficulties. In approximately 20–50 per cent of people with asthma, exercise-induced breathing difficulties can also be observed several hours after the exertion. This is called late-phase reaction.

There are two theories on exercise-induced asthma, the hyperosmolar theory and the airway rewarming theory. The hyperosmolar theory is where the high ventilation during physical exercise leads to drying of the bronchial mucous membrane (the air taken in must be humidified and the airways give off water), which in turn entails a hyperosmolar stimulus. The increase in osmolarity activates surrounding cells, such as mast cells, and bronchial constriction occurs. The airway rewarming theory is where an increased ventilation of air that is colder than body temperature leads to vasoconstriction of the bronchial mucous membrane. After the exertion, vasodilation occurs, and the dilated vessels fill with blood, swelling and obstructing the airways (7).

Symptoms and diagnosis

The diagnosis of asthma is made after a careful case history is taken, where attacks on the airways such as hissing and wheezing, respiratory distress and cough are present (8). The obstruction of the airways should be examined with a reversibility test (a comparison before and after administering drugs to dilate the airway). An increase in FEV₁ of at least 15 per cent (at least 200 ml) or a PEF increase of at least 20 per cent indicates the presence of asthma. A negative reversibility test does not rule out asthma. Continued investigation includes daily registration of PEF in an aim to determine whether a variable obstruction of the airway is present. In the case of asthma, it is typical that the airway obstruction does vary over time. The next step is to perform a steroid test. The patient is given a high dose of oral steroids for 2–3 weeks. During this time, the patient also keeps a PEF diary. Allergy tests and in some cases also a lung x-ray can be included in an asthma investigation.

In exercise stress tests for diagnosing asthma, the exercise should be conducted at a high load (80% of maximal aerobic capacity) (8). The exercise should not be preceded by a warm-up, but should begin at approximately 60 per cent of maximal aerobic capacity and then be increased every minute until the patient is unable to continue. The patient should use a nose clamp to avoid breathing through the nose. A drop in FEV₁ of more than 10 per cent compared to the baseline value should be considered pathological.

Prognosis

The introduction of inhaled steroids has had a big impact on asthma morbidity, prognosis and mortality (9). People with asthma have a larger annual decrease in FEV₁ than

non-asthmatics and asthmatics who smoke have a larger decrease than non-smoking asthmatics (10). Few people with asthma die of asthma. In Sweden, approximately 300 people per year die as a result of asthma.

Treatment principles

Pharmacological treatment

The pharmacological treatment of asthma is effective. Most asthmatics should use inhaled steroids regularly. Treatment with inhaled steroids yields reduced asthma symptoms, better lung function, reduced bronchial response, fewer asthma attacks, improved health-related quality of life, and a reduced risk for death from asthma (4). Maintenance therapy should combine beta-2 agonists with inhaled glucocorticosteroids. Leukotriene inhibitors (Singulair), which are given orally, are an alternative for patients who are unable to inhale. An additive effect is achieved if Singulair is used with inhaled steroid treatment. Singulair also has a protective effect against exercise-induced asthma. In patients who suffer from asthma only periodically, inhalation treatment with short-acting beta-2 agonists should be sufficient. In severe cases of asthma, oral steroids can also be used. In the case of acute deterioration of the disease, repeated doses of beta-2 agonists should be taken and, if symptoms do not subside, the inhaled glucocorticosteroid dose quadrupled. Systemic treatment with steroids can also have a dramatic effect (4).

Pharmacological treatment of exercise-induced obstruction of the airways

Exercise-induced breathing difficulties can be alleviated or even prevented by pre-medicating with beta-2 agonists and/or sodium cromoglycate (Lomudal), 10–20 minutes before the physical exercise (7). Leukotriene inhibitors (Singulair) can reduce or prevent constriction of the airways up to 24 hours after medication (11). Regular treatment with inhaled steroids also reduces the severity of exercise-induced symptoms.

Effects of physical activity

Physical training and physical activity have positive effects, from both a physiological and psychological standpoint, and in both the short- and long term (3 years) (12–14). Patients who have taken part in exercise are less afraid to exert themselves and dare to be more physically active in their daily life.

Acute effects of fitness training

Aerobic exercise improves cardiovascular capacity, measured as maximal oxygen uptake and maximal ventilation output per minute (15). Lung function and hyperresponsiveness are not changed by aerobic exercise. Improvement is seen in asthma and exercise-induced symptoms, and in limitations in daily life, number of visits to the emergency department, and the number of sick days (12, 14). Quality of life can also improve after a period of training (16).

Long-term effects

In the long-term, physical performance can be retained at a moderate level, even in people who have only been physically active in daily life. In people who perform physical exercise regularly, the amount of inhaled steroids has been able to be reduced (13). The number of emergency visits and sick days is also reduced (13).

Indications

Physical training should only be carried out under optimal conditions, that is, when no- or only a low level of obstruction is present. Precaution must also be taken with training in the case of exercise-induced problems. In the case of exercise-induced breathing difficulties, metabolic and circulatory changes occur in the body and we do not know how these changes affect the disease in the long term.

Prescription

Several international studies have shown that physical capacity in children and adults with asthma is lowered (17, 18). Children with asthma choose to a greater extent to participate in physical activities of moderate and low intensity, that is, they avoid high intensity physical activities (19). Many patients also feel physically limited in daily life as a result of their breathing difficulties (20). Breathing problems can also contribute to a strong sense of uncertainty in connection with physical activity, and it is of utmost importance that people unaccustomed to exercising receive advice, information and knowledge on how physical training can be carried out. The training should include aerobic training, and strength, flexibility and relaxation training, as well as breathing exercises.

The recommendation for people with a mild form of asthma, who experience bronchial obstruction only with infections and who are able to manage their exercise-induced breathing difficulties by taking beta-2 agonists before training, is to be physically active or take part in regular physical training to the same extent as healthy individuals (12, 14, 17). Training can occur outside the direction of medical care. For these people, their physical training should only be under the direction of medical care during periods when their asthma is getting worse or when a boost in motivation is needed.

People with variable airway obstruction require the assistance of a physiotherapist to get started with low intensity aerobic training and/or strength training.

People with chronic bronchial obstruction who, despite optimal medication, have significant limitations need the help of a physiotherapist to exercise at the level at which they are able. Training should begin with flexibility training, strength training and light physical activity.

Aerobic training can be carried out at low intensity or high intensity (see Table 1) and either continually or in interval form (21). All activities involving large muscle groups, and thereby loading the oxygen-supplying organs, are beneficial. Suitable activities include swimming, ball sports, cycling, walking and aerobic exercise training on land or in the water. In interval training, 2–3 minutes of high intensity training should be alternated with low intensity training or active rest of 1–2-minute intervals. The training should continue for at least 6–10 weeks. The greatest effect (measured as oxygen uptake) is achieved through high intensity training. Training in a heated pool or indoors should be recommended initially to those who are unaccustomed to exercising, to minimise the degree of exercise-induced breathing difficulties.

Strength training should include dynamic endurance training (see Table 1), above all for the arm, leg, shoulder and core muscles. Each exercise should be performed 8–12 times and repeated in 2–3 sets (21). A rest period of 1–3 minutes should be added between each set. The training should continue for at least 8–10 weeks. With low intensity training (40–50% of 1 RM), the training can occur daily; for higher intensity (60–80% of 1 RM), however, training should occur 2–3 times per week.

Flexibility training should cover flexibility exercises for the neck, shoulders, thorax, thigh and calf muscles, and be included in every training session.

Table 1. Description of different types of training.

Type of training	Intensity	Frequency (times/week)	Duration
Aerobic fitness training	Low intensity: > 55% of max HR* > 40% of VO ₂ max**	≥ 5	≥ 30 min.
	High intensity: > 70% of max HR > 60% of VO ₂ max	≥ 3	≥ 20 min.
Strength training	70% of 1 RM***	≥ 2	8–12 reps, 2–3 sets

* Max HR = Maximal Heart Rate.

** VO₂ max = Maximal Oxygen Uptake.

*** RM = Repetition Maximum. 1 RM corresponds to the maximum weight that can be lifted through the entire exercise movement one time.

Special considerations for the training

In order to reduce exercise-induced symptoms, the training should be preceded by pre-medication with beta-2 agonists, 15 minutes before exercising (7). A long warm-up (approx. 20 min), gradual intensification of the warm-up and using interval training has been shown to be highly effective in alleviating or completely preventing breathing difficulties (7). Every training session should end with a 5–15 minute cool-down. A heat

exchanger can be used when training in cold temperatures. People with asthma who experience exercise-induced breathing difficulties can have a refractory response to further exertion, that is, their breathing problems seem less severe in new physical exertions if they occur within 30 minutes to 3 hours (22).

Functional mechanisms

People with asthma with no obstruction achieve the same cardiovascular improvements as healthy individuals achieve after a period of aerobic training (17). The ventilation improvements seen after a training period are likely due to metabolic changes, which would also occur in healthy individuals who perform aerobic exercise. The improvement in exercise-induced difficulties can likely be explained in that the minute ventilation for the same exertion decreases after a period of training (14). Physical training, which involves a loading of the body, can probably prevent the development of osteoporosis (brittle bones), a risk that is especially high for steroid-dependent people.

Functional tests

A functional test should be conducted before physical training begins, in part to facilitate planning of adequate training, and in part to facilitate evaluation of the training. In all testing, measurement of PEF and oxygen saturation should be carried out before, during and up to 15 minutes after the test.

Cycling test and treadmill test

Standardised maximal or submaximal tests are carried out to investigate the patient's tolerance and limitations with respect to physical exertion. PEF, heart rate, oxygen saturation, shortness of breath, exertion and chest pain should be recorded both during and for a short time after the test. This type of test can also be used to evaluate the effects of physical training.

Walking test

Standardised walking tests are often used in clinical contexts to assess physical capacity in relation to activities of daily life. In a 6- or 12-minute walk test, the patient is encouraged to walk as far as possible in 6 or 12 minutes, respectively, on a measured stretch of hallway (23, 24). In all of the walking tests, the length of gait, heart rate, oxygen saturation and perceived exertion and shortness of breath are measured on a Borg scale (25). Note that a 6-minute walk test may not be sensitive enough to record changes in relatively healthy people with asthma.

Muscle function

Both dynamic muscle strength and endurance can be measured with isokinetic devices. Dynamic muscle strength can in addition be measured by the repetition maximum (RM), that is, the heaviest weight that can be lifted through an entire movement exercise one time. Dynamic endurance strength can be measured by the person performing a maximum number of repetitions at a given load. After a period of training, the test is repeated with the same load. An increase in the number of repetitions is an indication of an increase in muscle endurance.

Perception of quality of life and symptoms

A person's overall health-related quality of life can be measured with the Short-Form Health Survey (SF-36) (26), while the St. George's Respiratory Questionnaire (27) is often used to measure disease-specific quality of life. The severity of symptoms can be measured with a visual analogue scale (VAS) or the Borg scale.

Risks

No serious events need occur if the patient has undergone a functional test before commencing training, so that the physical limitations the patient demonstrates are known to the person in charge or instructing the training. No intensive training should occur if the disease is deteriorating.

References

1. GINA. From the global strategy for asthma management and prevention, Global Initiative for Asthma (GINA); 2006. Retrieved 2006-10-22 from <http://www.ginasthma.org>.
2. Ronmark E, Andersson C, Nystrom L, Forsberg B, Jarvholm B, Lundback B. Obesity increases the risk of incident asthma among adults. *Eur Respir J* 2005;25:282-8.
3. Blanc PD, Toren K. How much adult asthma can be attributed to occupational factors? *American Journal of Medicine* 1999;107:580-7.
4. Swedish Medical Products Agency. Farmakologisk behandling vid astma. Behandlingsrekommendation. [Pharmacological treatment for asthma. Treatment recommendation.] Uppsala: Swedish Medical Products Agency; 2007. www.lakemedelsverket.se.
5. Norrman E, Nystrom L, Jonsson E, Stjernberg N. Prevalence and incidence of asthma and rhinoconjunctivitis in Swedish teenagers. *Allergy* 1998;53:28-35.
6. Anderson S. Exercise-induced asthma. In: Carlsen K, Ibsen T, Eds. *Exercise-induced Asthma and Sports in Asthma*. Copenhagen: Munksgaard; 1999, pp. 11-8.
7. Storms WW. Review of exercise-induced asthma. *Medicine and Science in Sports and Exercise* 2003;35:1464-70.
8. Larsson K. Astma hos vuxna. Förekomst, sjukdomsbild, diagnostik och behandling. [Asthma in adults. Incidence, clinical picture, diagnostics and treatment.] Södertälje: AstraZeneca Sverige AB; 2005.
9. Suissa S, Ernst P. Inhaled corticosteroids. Impact on asthma morbidity and mortality. *Journal of Allergy and Clinical Immunology* 2001;107:937-44.
10. Lange P, Parner J, Vestbo J, Schnohr P, Jensen G. A 15-year follow-up study of ventilatory function in adults with asthma. *New England Journal of Medicine* 1998;339:1194-200.
11. Philip G, Villaran C, Pearlman DS, Loeys T, Dass SB, Reiss TF. Protection against exercise-induced bronchoconstriction two hours after a single oral dose of montelukast. *J Asthma* 2007;44:213-7.
12. Emtner M, Finne M, Stålenheim G. High-intensity physical training in adults with asthma. A comparison between training on land and in water. *Scand J Rehab Med* 1998;30:201-9.
13. Emtner M, Finne M, Stålenheim G. A 3-year follow-up of asthmatic patients participating in a 10-week rehabilitation program with emphasis on physical training. *Arch Phys Med Rehab* 1998;78:539-44.
14. Emtner M, Herala M, Stålenheim G. High-intensity physical training in adults with asthma. A 10-week rehabilitation program. *Chest* 1996;109:323-30.
15. Ram FS, Robinson SM, Black PN, Picot J. Physical training for asthma. *Cochrane database of systematic reviews (Online)* 2005:CD001116.
16. Basaran S, Guler-Uysal F, Ergen N, Seydaoglu G, Bingol-Karakoc G, Ufuk Altintas D. Effects of physical exercise on quality of life, exercise capacity and pulmonary function in children with asthma. *J Rehabil Med* 2006;38:130-5.

17. Clark C, Cochrane L. Assessment of work performance in asthma for determination of cardiorespiratory fitness and training capacity. *Thorax* 1988;43:745-9.
18. Ludwick S, Jones J, Jones T. Normalisation of cardiopulmonary endurance in severely asthmatic children with asthma. *Journal Pediatr* 1985;106:556-60.
19. Chiang LC, Huang JL, Fu LS. Physical activity and physical self-concept. Comparison between children with and without asthma. *Journal of Advanced Nursing* 2006;54: 653-62.
20. Mancuso CA, Sayles W, Robbins L, Phillips EG, Ravenell K, Duffy C, et al. Barriers and facilitators to healthy physical activity in asthma patients. *J Asthma* 2006;43:137-43.
21. Haskell WL, Lee IM, Pate RR, Powell KE, Blair SN, Franklin BA, et al. Physical activity and public health. Updated recommendation for adults from the American College of Sports Medicine and the American Heart Association. *Circulation* 2007 Aug 28;116:1081-93.
22. Clark T, Godfrey S, Lee T. *Asthma*. 3rd edn. London: Chapman & Hall; 1992.
23. Guyatt G, Sullivan M, Thompson P, Fallen E. The 6-minute walk. A new measure of exercise capacity in patients with chronic heart failure. *Can Med Assoc J* 1985;132: 919-32.
24. McGavin C, Groupta S, McHarty G. 12-minute walking test for assessing disability in chronic bronchitis. *Br Med J* 1976;1:822-3.
25. Borg GA. Psychophysical bases of perceived exertion. *Medicine and Science in Sports and Exercise* 1982;14:377-81.
26. Ware J, Scherbourne C. The MOS 36-Item Short-Form Health Survey (SF-36). Conceptual framework and item selection. *Med Care* 1992;30:473-83.
27. Jones P, Quirk F, Baveystock C, Littlejohns P. A self-complete measure of health status for chronic airflow limitation. *The American Review of Respiratory Disease* 1992;145: 1321-7.

18. Back problems (chronic)

Author

Tommy Hansson, MD, PhD, Professor, Department of Orthopaedics, Sahlgrenska University Hospital, Gothenburg, Sweden

Summary

Many people are afflicted one or more times in their life by back problems that remain for an extended period of time. A large majority of all prolonged or chronic back problems are non-specific. This means that the cause of the problems cannot be determined, not even with advanced magnetic resonance imaging (MR) technology. The physical inactivity often resulting from chronic back problems has not been shown to improve pain or back function. On the contrary, inactivity has been shown to have harmful physical and psychological effects. There are growing indications that the best way in which to alleviate pain and improve function if there is no definitive cause of the chronic back problems that can be diagnosed with traditional methods is to return to as normal physical activity as possible despite the problems. Attempting to normalise physical activity is also sufficient for the majority of specific back problems. An increase in physical activity may involve a return to daily activities such as walking and domestic chores and have the objective of mitigating a “fear of movement”. Normalisation can then be followed by gradually intensifying strength and aerobic fitness training.

Definition

Diagnosis – based on symptom duration

Lumbar spine problems can be defined in many different ways. Based on the duration of the symptoms, back problems are described as acute, subacute or chronic (1, 2). Since “chronic” suggests an incurable condition, which is seldom the case with back problems, the term is directly misleading and tends to stigmatize the patient (3). The boundary between acute, subacute and chronic problems has to-date been considered to be around 3 months (1). However, there are growing indications that the term “chronic” can and should be applied considerably earlier. One reason for this is that the changes that were previously considered to constitute, and respectively define, the transition to chronic back

problems can be observed earlier than previously expected. Major population studies carried out in recent years have shown that back and neck problems are characterised by frequent relapses (4–7). It appears to be increasingly clear that back and neck problems often tend to become chronic. Most people have mild to moderate symptoms and a typical progression appears to involve symptoms that fluctuate between periods of few symptoms and periodic deterioration where pronounced problems are relatively rare (4).

Diagnosis – based on symptoms

Regardless of duration, problems in the lumbar region can be divided into three symptom groups. The various symptoms may occur separately or in combination (2, 8, 9). The three main symptoms are:

1. **Lumbago.** Symptoms are called lumbago when back pain is located somewhere between the lower ribs and gluteal folds on the back of the thighs. The symptoms are still called lumbago even in the somewhat common case that the pain radiates down along the back of the thigh, as far down as the knee joint.
2. **Sciatica.** Symptoms are called sciatica when the pain extends along the innervation area of the sciatic nerve, i.e. the area served by the sciatic nerve. Sciatica pain is often accompanied by an impact on both sensitivity and motor functions. By definition, sciatica means that one or more of the L5, S1 and/or sometimes S2 nerve roots signal(s) symptoms along its or their distribution area.
3. **Neurogenic claudication.** Symptoms of neurogenic claudication include pain and motor and/or sensory effects, which typically present themselves with a certain physical activity, usually walking a certain distance or assuming a certain body position (10). Symptoms most often occur along the distribution area of the sciatic or femoral nerve. The pain typically subsides when the individual rests or changes body position.

All three symptoms may occur with both acute and chronic back problems.

Diagnosis – based on causes

When the cause of back problems can be determined, such as by means of a clinical examination and/or radiographs or magnetic resonance imaging, the problems are classified as *specific*, regardless of the type of symptoms. Consequently, back problems without a diagnosable cause are called *non-specific* (2, 9, 11, 12).

Of acute back problems, 90–95 per cent are considered to be non-specific. The percentage of specific back problems increases with the duration of the problems. After around three months, one third of all back problems are estimated to be specific. The existence of specific problems is considered to have a negative effect on the prognosis for improvement on the short term. It is also relatively typical that the onset of non-specific symptoms is most often more acute, such as the onset of lumbago, than specific symptoms, where a more prolonged onset is considered to be more common.

Regardless of whether symptoms are specific or non-specific by nature, they can be the same, i.e. lumbago, sciatica or claudication (9). However, symptoms of sciatica or claudication occur significantly more often in specific back problems. It can generally be said that a specific treatment most often exists for specific symptoms, which is not the case with non-specific symptoms.

Causes of specific chronic back problems

There are a number of causes of specific back problems. The most common causes of specific chronic back problems are:

1. **Disc hernia.** A lumbar disc hernia arises when tissue from the disc's core (nucleus pulposus) protrudes between or through the peripheral part of the disc (annulus fibrosus) and causes pressure on one or more of the nerve roots in the spinal cavity. The pressure on the nerve root leads to pain along the compressed nerve root and also usually in the lumbar region, i.e. lumbago. The occurrence of symptom-inducing disc hernia is difficult to determine. It has also been shown that nearly 2/3 of the normal adult population have asymptomatic disc hernias that are visible with magnetic resonance imaging (13). The prevalence of sciatica, most likely caused by a disc hernia, varies between 3 to 5 per cent (1).
2. **Spinal stenosis** and disc hernia are the most common causes of specific problems in the lumbar region. Classic symptoms include neurogenic claudication, i.e. activity or position-induced pain along the sciatic nerve. Spinal stenosis is caused by a "narrowing" of the spinal canal or the so-called spinal nerve root canals. The narrowing of the actual spinal canal is called central spinal stenosis while the narrowing of the nerve root canals is called lateral spinal stenosis. Typical symptoms arise in connection with e.g. walking or special body positions, usually those that lead to an extension of the lumbar region, and in connection with activities or positions that aggravate the compression of the nerves in the spinal canal. The narrowing or compression of the cauda equina is in turn caused by a degeneration of the tissues that make up the joint systems between adjacent vertebrae (10). These degenerative changes usually develop gradually over several years and can eventually lead to a compression of the nerve roots in the cauda equina (14). When walking, for example, the pressure rises and characteristic symptoms, neurogenic claudication, appear. Symptoms of compression become apparent when the nerve roots in cauda equina have less space than 0.75 cm² (15).
3. **Spondylolisthesis.** Also referred to as isthmic spondylolisthesis, i.e. a defect usually found in the fifth vertebral arch of the lumbar vertebra. Spondylolisthesis is a forward slippage of a vertebral column usually above the most proximal end-plate of the sacral vertebra. The slippage is considered to be of clinical relevance, meaning that it causes symptoms, only when it amounts to at least 25 per cent of the subadjacent vertebral end-plate. Characteristic symptoms include pain in the lower lumbar region, which often radiates along the back of the thighs down towards the knees, affecting the walking pattern (16, 17).

4. **Pronounced instability.** Often characterised by a pronounced degeneration of the relevant intervertebral joint. The term intervertebral joint refers to the joints between two vertebrae comprised of the disc and the two facet joints. A marked degeneration leads to a lower disc height and usually pronounced changes in the form of facet joint osteoarthritis. This can cause an abnormal forward or backward slippage between adjacent vertebrae. Pronounced instability is usually suspected if distinct pain is induced by movements of the back, at certain positions or a sudden movement (18). This diagnosis is still controversial and, consequently, so is its correlation to back problems. In what is presumed to be pronounced instability, examinations with special technology have not been able to show any increased flexibility, but rather back muscle activity that has been interpreted as a defensive response by a painful intervertebral joint that is more indicative of a segmental disorder rather than instability (19, 20).
5. **Inflammatory back disorders.** Back problems can be caused by various types of inflammatory diseases such as Mb Bechterew. Pain is most often felt in and along the entire spine. In typical cases, pain arises both under strain and at rest, such as at night time.
6. **Osteoporotic fractures.** Osteoporotic fractures occur in the spine in the form of vertebral compressions. Osteoporotic fractures are usually found between the thoracic spine and lumbar spine, i.e. in vertebrae Th12 and L1. To-date, the progression has been considered to be benign, i.e. a good prognosis in terms of subsiding pain and improvement in back function. However, recent studies of the natural progression of fractures appear to contradict this anticipated good progression. A very large proportion of patients with a fresh vertebral compression have proven to continue to have pronounced pain, poor back function and, as a result, a greatly reduced quality of life for at least 12 months after the fracture (21).

It is important to remember that specific changes may occur without giving rise to symptoms. As previously mentioned, this is particularly true of herniated discs (13). The spinal changes must correspond with the symptoms for the symptoms to be described as specific.

A large number of changes visible on radiographs or deviations from the “norm” have long been considered to possibly be the cause of both acute and chronic back problems. Most of these changes and abnormalities have been able to be dismissed as clinically insignificant, meaning that they have no correlation to the occurrence of back problems (22, 23).

Back pain

Pain, particularly chronic pain, is often very complicated and remains a reaction that is difficult to explain, in which emotional and psychosocial factors play a role, creating large differences both within each individual and between individuals (11, 24).

In order for the pain of lumbago to arise, peripheral nerve endings or nociceptors must be activated by mechanical, chemical, thermal or inflammatory stimulation or what is otherwise referred to as nociceptive stimuli. Nociceptors normally have a high stimulation threshold, which means that they are not triggered to signal pain by normal stimuli,

such as light pressure from touch, etc. Nociceptors are found in essentially every tissue that surrounds or forms the human spine (25). In terms of the actual disc, nociceptors have been observed in the peripheral parts of annulus fibrosus. The core parts of the disc constitute an exception where no nociceptors have been found. Although still subject to debate, it has been reported that nerve endings, and thereby the possibility of nociception or pain signal transduction, can be developed in a degenerated, aged disc. Nerve endings have been detected in severely degenerated discs (26). These nerve endings have been assumed to have “entered” the disc in connection with the formation of scar/connective tissue as a result of degeneration (27, 28). This “immigration” may come from the vertebral body end-plates and underlying spongy (porous) bone which are both full of nociceptors (29). Nerve endings are also found in blood vessel walls, as well as all of the muscles surrounding the spinal column.

Experimental studies have shown that there is feedback from the pain transduction neuron to the motor nerves, i.e. that a nociceptive stimuli in the annulus fibrosus triggers a contraction of the the back muscles (30, 31). This would very likely explain the active contraction of back muscles so often observed with acute back problems. It is not unlikely that essentially the same mechanism would also explain the prolonged back muscle contractions seen in individuals with chronic back problems (20). However, recent experiments have also shown that a damaged annulus fibrosus has a segmental muscle influence that implies a reflex inactivation of, for example, the multifidus muscles (31).

A prolonged muscle contraction could be facilitated by fear, not least the fear of more pain as a consequence of moving. Consequently, the contraction itself could be the main cause of the back pain, even long after the initial pain-inducing nociceptive stimulation has healed (usually within 2–3 days) (4, 7).

In addition to mechanisms that promote the transmission of pain impulses, there are also dampening or inhibiting mechanisms in the spinal cord and brain stem (32). Among the latter are endogenous opiates or endorphins, as well as transmission substances such as noradrenalin and serotonin. It is via the endorphins, among others, that a positive correlation has been presumed to exist between physical activity and its influence on pain, such as in chronic back problems.

Because all tissue components are equipped with nociceptors, with the exception of the disc’s core, all of them at least have the prerequisites to signal pain, either separately or collectively. Hypothetically, non-specific back problems can thereby conceivably be caused by nociceptive stimuli (pain due to damaged or irritated tissue) generated by all of the tissue components of the back, i.e. the tissue of bones, ligaments, tendons, discs, muscles, vessels and/or nerves in and around the vertebral column. Accordingly, conceivable damage or mechanisms that can generate a nociceptive stimuli could, for example, be:

1. Internal tears or ruptures in the annulus fibrosus with penetration of the nucleus material into the annulus (33).
2. Microfractures in vertebral end-plates and/or surrounding spongy bone with inflammation/oedema in the spongy bone (34–36).
3. Tears or ruptures of vertebral column tendons (37).

4. Tears or ruptures of facet joint capsules.
5. Inflammatory reactions in facet joints and synovial membranes.
6. Tears and other changes in the spinal muscles.

Common to all of these conceivable changes or damage as causes of back problems is that they cannot be diagnosed using the methods and technology available at the present time (2).

Disc degeneration

Disc degeneration is the name for the degenerative changes that occur in the disc with increasing age. The first signs of degenerative changes in the disc appear as early as adolescence (38). The degree of degeneration varies greatly from individual to individual (39–41). Early signs of disc degeneration are a loss of water in the tissue and tears of the annulus fibrosus (29). Over time, pronounced degenerative disc changes will lead to a decreased disc height. These changes affect the mechanical characteristics of the disc.

In a review of evidence on the correlation between physical load and degree of disc degeneration, several extensive post-mortem studies confirmed that between 72 and 90 per cent of all 70-year-olds have degenerative changes, but that those in women are less pronounced (42). Another clinical radiology study involving more than 15,000 patients confirmed that men have degenerative changes more frequently than women and that the men's changes were also more pronounced. However, no correlation was found between heavy physical work and the occurrence of these degenerative changes (8). Another radiology study confirmed that a reduction in disc height was not correlated with occupational category or the occurrence of lifting in the work or professional exposure to full-body vibrations (22). In two Finnish studies, there was an increased prevalence of disc degeneration among elite athletes who were exposed to extreme physical loads. However, the studies also report that, in spite of being subjected to an extreme load for more than 20 years, sometimes with injuries, only 10 per cent of the degeneration could be explained by the extreme physical load. In contrast, a 5-year follow-up study using magnetic resonance imaging showed that low physical activity was one of the risk factors for accelerated development of disc degeneration (39). In another Finnish study of identical twins, no statistically significant correlation could be established between the degree of occupational physical exposure and the prevalence of disc degeneration (41, 43, 44). Heredity was by far the most important factor that could explain the degree of degeneration in these identical twins.

Effects of chronic back problems

Chronic back problems and the physical inactivity that they cause not only negatively affect the back itself, but also the entire body. Early on, it was possible to use measurements of bone mineral density in the vertebral column to show that the bone mineral content in the vertebrae was lower, the longer the back problems had been present (45–47). Because bone mineral content is without comparison the factor that means the most to the

strength of a vertebra, a lower bone mineral content means weaker vertebrae. Besides the actual bone, a low bone mineral content in the spine has also been shown to reflect weaker tendons, ligaments and other soft tissues of the back (37, 48).

Chronic back problems also have a negative effect on the back muscles. This has been shown in repeated studies of the back muscles. Using computed tomography or magnetic resonance imaging, it has been possible to identify spinal muscular atrophy in individuals with chronic back problems (49–53). Experimental studies have found back muscles to be negatively affected only a few days after minor damage to e.g. the annulus fibrosus (31). The muscle changes observed suggest that reflex inhibition may be one of several conceivable causes of the inactivity changes resulting from back problems. Worse functional characteristics of the muscles have also been indirectly found in the form of reduced strength, endurance, etc. (54, 55).

Besides the direct negative effects on the various tissues of the spine, chronic back problems have been shown to have general effects in the form of reduced fitness, overweight, etc. (54) In this context, it deserves mentioning that although the negative physical effects of chronic back problems are considerable, the negative psychosocial effects, which often cause e.g. a lack of self-confidence, social isolation and exclusion, are usually at least just as serious (1, 56–59).

Effects of physical activity

Several randomised controlled studies have shown that the advice to patients with acute or chronic back problems to “be as normally physically active as possible” despite the problems is a treatment that improves pain, functional ability and, where relevant, the capacity to work as well (60–63).

When the scientific evidence for or against the effect of back and/or aerobic fitness training was evaluated a few years ago in the SBU report “Back pain, neck pain”, it was confirmed that there was strong evidence that back training has a positive effect on chronic back problems (1). The most commonly studied back training methods are flexion and extension exercises, and the training of back, neck and abdominal muscles. The muscle training methods have commonly been dynamic, static or focused on increasing endurance (64–66).

For most people, walking or jogging with varying intensity is the most common and most readily available way of improving their aerobic fitness on their own (67). For others, swimming or cycling might be preferable or even a requirement to be able to exercise at all. The latter may, for example, apply to people with spinal stenosis or other specific back problems, where the space in the spinal canal is restricted, such as in spondylolisthesis. Cycling, which usually involves bending forward, is often a symptom-free exercise for people with spinal canal compression. Preparation studies have shown that the space in the spinal canal increases by around 40 cm² when the lumbar spine is moved from maximum backward to maximum forward position, which would probably explain the positive effects of cycling (68).

The direct effects of aerobic training on back problems have not been completely established. One conceivable pain-inhibiting effect may be through the effect of exercise on the body's endorphin production. The movement of the spine when walking or jogging may also contribute to the essential metabolic transport into and out of the relatively avascular middle vertebral plates (discs) (69). Metabolites that could affect the induction of pain in the periphery of the disc could thereby be transported away (33). A generally positive effect on both pain and function of physical activity compared to inactivity has been confirmed in many studies of chronic back problems (70, 71).

By drilling a metal pin into the spinous process and then attaching an accelerometer on test subjects and asking them to walk or run in place, it was confirmed that there is very little effect on the lumbar spine from the impact generated by walking or running (72). The conclusion from these studies is that the load on the lumbar spine when jogging at a moderate pace is only marginally elevated compared with standing still or walking. Consequently, walking or jogging at a moderate pace should not cause any harmfully elevated load on the spinal column.

Perhaps the most important effect of various training methods and exercises is probably the activating effect that they have on contracted and probably painful back muscles, as well as counteracting or breaking down the fear of movement that individuals with chronic back problems have (20, 60).

The old expression "everything in moderation" may also be applicable to back problems since there appears to be an elevated risk of back problems among those who are very physically active, at least among young people.

Indications

Non-specific and most specific chronic back problems.

Prevention

The vast majority of people suffer from back pain at some point in their lives. The degree of discomfort can vary from slight discomfort to distinctly severe pain. One or more episodes of slight or moderate back problems are usually forgotten before long. Consequently, determining whether a preventive measure should be classified as primary or secondary is often both impossible and not very meaningful. There are few studies that clearly show a preventive effect from various physical activities and a reduced incidence of back problems (50, 73). At least the number of studies that have not been able to indicate any preventive effect or even a negative, aggravating effect are not fewer in number than those with a positive effect (74, 75).

Prescription

The principal advice to most patients regardless of whether the problems are acute or chronic is to be “as normally physically active as possible” despite the problems. The severity of the symptoms should determine the pace that the normalisation of the physical activity should and can have. If the symptoms are of a non-specific nature, there are, by definition, no tissue-related changes that would constitute a contraindication of a rapid normalisation of physical activities. In such cases, the only limitation may possibly be the hypotrophy or atrophy caused by the inactivity. Strongly emphasizing the fact that no tissue damage could be located and that, consequently, no “dangerous” injury or change exists can be a strong motivating factor for the individual to achieve a normalisation of his or her level of physical activity. In the event of a pronounced fear of movement, a gradual normalisation of physical activity can be tried, possibly with well-defined intermediate objectives.

In case of specific problems such as spinal stenosis or symptom-inducing disc hernia, the activity level must be adapted to the presence of symptoms, especially nerve root damage. In many cases, perhaps especially with spinal stenosis where normal walking is more difficult or impossible, cycling may be a good alternative in order to maintain a certain level of physical activity.

Examples of suitable early activities to return to normal physical activity include light household work, walking, cycling, etc. For most people that do not have an extremely physically challenging job, early occupational training or a partial return to work is a very important step towards a normalisation of physical and, perhaps just as important, social activity. Once symptoms have subsided, individual training advice is particularly important for those who have not previously trained e.g. their back and abdominal muscles.

Contraindications

Absolute contraindications include certain specific types of chronic back problems, such as those caused by a tumour, metastasis or fracture and where the stability of the spine is endangered by a normal load.

Relative contraindications may exist with certain types of specific chronic back problems, such as spinal stenosis, a herniated disc or spondylolisthesis. Intensifying nerve root effects in the form of pain or another nerve effect radiating down the leg indicates that the pressure on the nerve root(s) is increasing and that there may be a risk of more pronounced nerve damage.

Risks

See above.

References

1. Jonsson EN, Nachemson A. Ont i ryggen, ont i nacken [Back pain, neck pain] SBU-rapport [SBU-Report] Volume 145. Stockholm: SBU; 2000.
2. Hansson T, Westerholm P. Arbete och besvär i rörelseapparaten. [Work-related musculoskeletal disorders] Arbete och Hälsa [Work and Health] Volume 2001:12 Stockholm: National Institute for Working Life; 2001.
3. Holloway I, Sofaer-Bennett B, Walker J. The stigmatisation of people with chronic back pain. *Disabil Rehabil* 2007;29:1456-64.
4. Cassidy JD, Cote P, Carrol LJ, Kristman V. Incidence and course of low back pain episodes in the general population. *Spine* 2005;30:2817-23.
5. Walker BF, Muller R, Grant WD. Low back pain in Australian adults. Prevalence and associated disability. *J Manipulative Physiol Ther* 2004;27:238-44.
6. Ihlebaek C, Hansson T, Laerum E, Indahl A, Holm S. Prevalence of low back pain and sickness absence. A "borderline" study in Norway and Sweden. *Scand J Public Health* 2006;34:555-8.
7. Carroll LJ, Cassidy JD, Cote P. The Saskatchewan Health and Back Pain Survey. The prevalence and factors associated with depressive symptomatology in Saskatchewan adults. *Can J Public Health* 2000;91:459-64.
8. Friberg S, Hirsch C. Anatomical and clinical studies on lumbar disc degeneration. 1950. *Clin Orthop Relat Res* 1992;279:3-7.
9. Deyo RA, Rainville J, Kent DL. What can the history and physical examination tell us about low back pain? *JAMA* 1992;268:760-5.
10. Schonstrom NS, Bolender NF, Spengler DM. The pathomorphology of spinal stenosis as seen on CT scans of the lumbar spine. *Spine* 1985;10:806-11.
11. Waddell G. Subgroups within "nonspecific" low back pain. *J Rheumatol* 2005;32:395-6.
12. Krismer M, van Tulder M. Strategies for prevention and management of musculoskeletal conditions. Low back pain (non-specific). *Best Pract Res Clin Rheumatol* 2007;21:77-91.
13. Boos N, Rieder R, Schade V. 1995 Volvo Award in clinical sciences. The diagnostic accuracy of magnetic resonance imaging, work perception and psychosocial factors in identifying symptomatic disc herniations. *Spine* 1995;20:2613-25.
14. Schonstrom N, Bolender N, Spengler D, Hansson T. Pressure changes within the cauda equina following constriction of the dural sac. An in vitro experimental study. *Spine* 1984;9:604-7.
15. Ogikubo O, Forsberg L, Hansson T. The relationship between the cross-sectional area of the cauda equina and the preoperative symptoms in central lumbar spinal stenosis. *Spine* 2007;32:1423-8, discussion 1429.
16. Frennered AK, Danielson BI, Nachemson AL. Natural history of symptomatic isthmic low-grade spondylolisthesis in children and adolescents. A seven-year follow-up study. *J Pediatr Orthop* 1991;11:209-13.
17. Danielson B, Frennered K, Irstam L. Roentgenologic assessment of spondylolisthesis. I. A study of measurement variations. *Acta Radiol* 1988;29:345-51.

18. Pitkanen MT, Manninen HI, Lindgren KA, Sihvonen TA, Airaksinen O, Soimakallio S. Segmental lumbar spine instability at flexion-extension radiography can be predicted by conventional radiography. *Clin Radiol* 2002;57:632-9.
19. Kaigle AM, Holm SH, Hansson TH. Experimental instability in the lumbar spine. *Spine* 1995;20:421-30.
20. Kaigle AM, Wessberg P, Hansson TH. Muscular and kinematic behavior of the lumbar spine during flexion-extension. *J Spinal Disord* 1998;11:163-74.
21. Suzuki NO, Ogikubo O, Hansson T. The prognosis for pain, back function and QoL after an acute osteoporotic vertebral fracture. ISSLS 34th annual meeting. Hongkong 2007.
22. Frymoyer JW, Newberg A, Pope MH, Wilder DG, Clements J, MacPherson B. Spine radiographs in patients with low-back pain. An epidemiological study in men. *J Bone Joint Surg Am* 1984;66:1048-55.
23. Boos N, Semmer N, Elfering A, Schade V, Gal I, Zanetti M, et al. Natural history of individuals with asymptomatic disc abnormalities in magnetic resonance imaging. Predictors of low back pain-related medical consultation and work incapacity. *Spine* 2000;25:1484-92.
24. Linton SJ. Do psychological factors increase the risk for back pain in the general population in both a cross-sectional and prospective analysis? *Eur J Pain* 2005;9:355-61.
25. Brown MF, Hukkanen MV, McCarthy ID, Redfern DR, Batten JJ, Crock HV, et al. Sensory and sympathetic innervation of the vertebral endplate in patients with degenerative disc disease. *J Bone Joint Surg Br* 1997;79:147-53.
26. Johnson WE, Evans H, Menage J, Eisenstein SM, El Haj A, Roberts S. Immunohistochemical detection of Schwann cells in innervated and vascularized human intervertebral discs. *Spine* 2001;26:2550-7.
27. Freemont AJ, Peacock TE, Goupille P, Hoyland JA, O'Brien J, Jayson M. Nerve ingrowth into diseased intervertebral disc in chronic back pain. *Lancet* 1997;350:178-81.
28. Roberts S, Eisenstein SM, Menage J, Evans EH, Ashton IK. Mechanoreceptors in intervertebral discs. Morphology, distribution and neuropeptides. *Spine* 1995;20:2645-51.
29. Roberts S, Evans EH, Kletsas D, Jaffray DC, Eisenstein SM. Senescence in human intervertebral discs. *Eur Spine J* 2006;15 Suppl 3:S312-6.
30. Indahl A, Kaigle AM, Reikeras O, Holm SH. Interaction between the porcine lumbar intervertebral disc, zygapophysial joints and paraspinal muscles. *Spine* 1997;22:2834-40.
31. Hodges P, Kaigle AM, Holm SH, Hansson T, Holm S. Rapid atrophy of the lumbar multifidus follows experimental disc or nerve root injury. *Spine* 2006;31:2926-33.
32. Dahl JB, Erichsen CJ, Fuglsang-Frederiksen A, Kehlet H. Pain sensation and nociceptive reflex excitability in surgical patients and human volunteers. *Br J Anaesth* 1992;69:117-21.
33. Holm S, Nachemson A. Variations in the nutrition of the canine intervertebral disc induced by motion. *Spine* 1983;8:866-74.
34. Hansson T, Roos B, Nachemson A. The bone mineral content and ultimate compressive strength of lumbar vertebrae. *Spine* 1980;5:46-55.

35. Kjaer P, Korsholm L, Bendix T, Sorensen JS, Leboeuf-Y de C. Modic changes and their associations with clinical findings. *Eur Spine J* 2006;15:1312-9.
36. Albert HB, Kjaer P, Jensen TS, Sorensen JS, Bendix T, Manniche C. Modic changes, possible causes and relation to low back pain. *Med Hypotheses* 2007;9 [Epub ahead of print].
37. Neumann P, Keller T, Ekström L, Hult E, Hansson T. Structural properties of the anterior longitudinal ligament. Correlation with lumbar bone mineral content. *Spine* 1993;18:637-45.
38. Coventry M, Ghormley R, Kernohan J. The intervertebral disc. Its microscopic anatomy and pathology. Part III. *J Bone and Joint Surg* 1945;3:460-74.
39. Elfering A, Semmer N, Birkhofer D, Zanetti M, Hodler J, Boos N. Risk factors for lumbar disc degeneration. A 5-year prospective MRI study in asymptomatic individuals. *Spine* 2002;27:125-34.
40. Videman T, Battie MC, Ripatti S, Gill K, Manninen H, Kaprio J. Determinants of the progression in lumbar degeneration. A 5-year follow-up study of adult male monozygotic twins. *Spine* 2006;31:671-8.
41. Battie MC, Videman T, Levalahti E, Gill K, Kaprio J. Heritability of low back pain and the role of disc degeneration. *Pain* 2007;131:272-80.
42. Miller JA, Schmatz C, Schultz AB. Lumbar disc degeneration. Correlation with age, sex and spine level in 600 autopsy specimens. *Spine* 1988;13:173-8.
43. Videman T, Battie MC, Gibbons LE, Manninen H, Gill K, Fisher LD, et al. Lifetime exercise and disk degeneration. An MRI study of monozygotic twins. *Med Sci Sports Exerc* 1997;29:1350-6.
44. Battie MC, Videman T, Gibbons LE, Fisher LD, Manninen H, Gill K. 1995 Volvo Award in clinical sciences. Determinants of lumbar disc degeneration. A study relating lifetime exposures and magnetic resonance imaging findings in identical twins. *Spine* 1995;20:2601-12.
45. Hansson T, Sandstrom J, Roos B, Jonson R, Andersson GB. The bone mineral content of the lumbar spine in patients with chronic low-back pain. *Spine* 1985;10:158-60.
46. Vuori IM. Dose-response of physical activity and low back pain, osteoarthritis, and osteoporosis. *Med Sci Sports Exerc* 2001;33 Suppl:S551-86, discussion 609-10.
47. Uusi-Rasi K, Sievanen H, Pasanen M, Oja P, Vuori I. Association of physical activity and calcium intake with the maintenance of bone mass in premenopausal women. *Osteoporosis Int* 2002;13:211-7.
48. Neumann P, Ekstrom LA, Keller TS, Perry L, Hansson TH. Aging, vertebral density and disc degeneration alter the tensile stress-strain characteristics of the human anterior longitudinal ligament. *J Orthop Res* 1994;12:103-12.
49. Mannon RJ, Woolf CJ. Pain mechanisms and management. A central perspective. *Clin J Pain* 2000;16 Suppl:S144-56.
50. Moreau CE, Green BN, Johnson CD, Moreau SR. Isometric back extension endurance tests. A review of the literature. *J Manipulative Physiol Ther* 2001;24:110-22.
51. Reinsel TE, Grobler LJ, Meriam C. Progressive paraspinal muscle atrophy presenting as low-back pain. Case report. *J Spinal Disord* 1995;8:249-51.

52. Jorgensen MJ, Marras WS, Gupta P. Cross-sectional area of the lumbar back muscles as a function of torso flexion. *Clin Biomech (Bristol, Avon)* 2003;18:280-6.
53. Danneels LA, Vanderstraeten GG, Cambier DC, Witvrouw EE, De Cuyper HJ. CT imaging of trunk muscles in chronic low back pain patients and healthy control subjects. *Eur Spine J* 2000;9:266-72.
54. Moffroid MT. Endurance of trunk muscles in persons with chronic low back pain. Assessment, performance, training. *J Rehabil Res Dev* 1997;34:440-7.
55. Ng JK, Richardson CA, Kippers V, Parnianpour M. Relationship between muscle fiber composition and functional capacity of back muscles in healthy subjects and patients with back pain. *J Orthop Sports Phys Ther* 1998;27:389-402.
56. Crombez G, Vlaeyen JW, Heuts PH, Lysens R. Pain-related fear is more disabling than pain itself. Evidence on the role of pain-related fear in chronic back pain disability. *Pain* 1999;80:329-39.
57. Crombez G, Vervaeke L, Lysens R, Baeyens F, Eelen P. Avoidance and confrontation of painful, back-straining movements in chronic back pain patients. *Behav Modif* 1998;22:62-77.
58. Buer N, Linton SJ. Fear-avoidance beliefs and catastrophizing. Occurrence and risk factor in back pain and ADL in the general population. *Pain* 2002;99:485-91.
59. Grotle M, Vollestad NK, Veierod MB, Brox JI. Fear-avoidance beliefs and distress in relation to disability in acute and chronic low back pain. *Pain* 2004;112:343-52.
60. Liddle SD, Gracey JH, Baxter GD. Advice for the management of low back pain. A systematic review of randomised controlled trials. *Man Ther* 2007;12:310-27.
61. Malmivaara A, Hakkinen U, Aro T, Heinrichs ML, Koskeniemi L, Kuosma E. et al. The treatment of acute low back pain. Bed rest, exercises, or ordinary activity? *N Engl J Med* 1995;332:351-5.
62. Indahl A, Velund L, Reikeraas O. Good prognosis for low back pain when left untampered. A randomized clinical trial. *Spine* 1995;20:473-7.
63. Indahl A, Haldorsen EH, Holm S, Reikeras O, Ursin H. Five-year follow-up study of a controlled clinical trial using light mobilization and an informative approach to low back pain. *Spine* 1998;23:2625-30.
64. Lindgren KA, Sihvonen T, Leino E, Pitkanen M, Manninen H. Exercise therapy effects on functional radiographic findings and segmental electromyographic activity in lumbar spine instability. *Arch Phys Med Rehabil* 1993;74:933-9.
65. Kellett KM, Kellett DA, Nordholm LA. Effects of an exercise program on sick leave due to back pain. *Phys Ther* 1991;71:283-91, discussion 291-3.
66. Mannion AF, Muntener M, Taimela S, Dvorak J. Comparison of three active therapies for chronic low back pain. Results of a randomized clinical trial with one-year follow-up. *Rheumatology (Oxford)* 2001;40:772-8.
67. Jacob T, Baras M, Zeev A, Epstein L. Physical activities and low back pain. A community-based study. *Med Sci Sports Exerc* 2004;36:9-15.
68. Schonstrom N, Lindahl S, Willen J, Hansson T. Dynamic changes in the dimensions of the lumbar spinal canal. An experimental study in vitro. *J Orthop Res* 1989;7:115-21.

69. Sjolie AN. Access to pedestrian roads, daily activities and physical performance of adolescents. *Spine* 2000;25:1965-72.
70. Hartvigsen J, Christensen K. Active lifestyle protects against incident low back pain in seniors. A population-based 2-year prospective study of 1 387 Danish twins aged 70–100 years. *Spine* 2007;32:76-81.
71. Hasenbring MI, Plaas H, Fischbein B, Willburger R. The relationship between activity and pain in patients 6 months after lumbar disc surgery. Do pain-related coping modes act as moderator variables? *Eur J Pain* 2006;10:701-9.
72. Ray JK, Keller T, Magnusson M, Hansson T. In-vivo measurements of lumbar transmissibility in the upright human subject. Heidelberg: ISSLS; 1991.
73. Hilde G, Hagen KB, Jamtvedt G, Winnem M. Advice to stay active as a single treatment for low back pain and sciatica. *Cochrane Database Syst Rev* 2002;2:CD003632.
74. Taimela S, Diederich C, Hubsch M, Heinrich M. The role of physical exercise and inactivity in pain recurrence and absenteeism from work after active outpatient rehabilitation for recurrent or chronic low back pain. A follow-up study. *Spine* 2000;25:1809-16.
75. Hansson TH, Hansson EK. The effects of common medical interventions on pain, back function and work resumption in patients with chronic low back pain. A prospective 2-year cohort study in six countries. *Spine* 2000;25:3055-64.

19. Cancer

Author

Inger Thune, MD, PhD, Oslo University Hospital, Ullevål, Norway

Summary

A sedentary lifestyle and overweight causes 25 per cent of all new cancer cases in the world. Increased knowledge of the impact of overweight and physical inactivity on prevention, treatment and rehabilitation of cancer diseases is therefore important. Cancer develops in a complex interaction between genetics, environment and lifestyle, in which a number of biological mechanisms affect one another. Physical activity affects a series of biological mechanisms, such as metabolism of energy, levels of sex hormones, insulin resistance, leptin, prostaglandins and C-reactive protein, and immune function. It also enables DNA repair. Increased knowledge of these effects on concrete biological mechanisms has laid the groundwork for understanding the observed relations between physical activity and individual cancer diseases, and made it possible to recommend physical activity as a protective factor against cancer.

We know today that regular physical activity is one of the key factors that a person is able to influence to prevent becoming ill with cancer. Existing knowledge shows that, depending on the frequency, intensity and duration, regular activity at work or as recreation can reduce cancer of the colon by 20–60 per cent. Corresponding knowledge on physical activity in relation to breast cancer, shows that 4 hours of activity per week, at an intensity corresponding to 6 METs (MET = metabolic equivalent, i.e. the uptake of oxygen at rest; 6 METs corresponds to a light jog), reduces breast cancer by 30–50 per cent. Physical activity also protects against uterine cancer and may also be significant with respect to prostate and lung cancer.

As a result of the growing incidence of cancer as well as increased survival of patients, an increasing number of people are living longer with cancer. In recent years, research has shown that physical activity not only protects against a number of types of cancers, but is also valuable for patients undergoing cancer treatment and during the rehabilitation phase, as well as for improving function and quality of life. Regular physical activity is an effective way to reduce the side-effects of cancer, resulting in part from physical inactivity and in part from the disease itself. Too much rest can lead to a decrease in aerobic fitness, strength, mobility and unwanted weight gain in the patient.

With respect to physical activity as treatment and rehabilitation for cancer diseases, an effort should be made to include at least 15–60 minutes of daily activities that involve the large muscle groups, such as walking, cycling or skiing, at moderate to high intensity adapted to the particular individual. Avoid heavy loading.

Incidence, causes and risk factors

Historically speaking, cancer has always existed. It was a more uncommon disease in our forefathers who lived as hunters and gatherers some 10,000 years ago (1), however, both because average life expectancy was lower and because the disease itself was less common. Since then, our ways of life have undergone dramatic change, while our genetic make-up has changed by only 0.003 per cent (2). Thus, our modern living habits characterised by physical inactivity subject human cells and biological processes to much more strain than in the past.

The word “cancer” comes from the Greek *karkinos* (carcinoma), meaning crab. Cancer can manifest as a solid growth or “tumour”, or as non-tumorous cancers such as leukaemia. “Cancer” is a collective term used to describe over 100 different cancerous diseases that can attack all types of cells in the body. Each cell type can produce completely different forms of cancer, and several different cancer diseases can develop from the same cell type.

Although there are many different types of cancer, they share a number of common characteristics, for example, an uncontrollable cell proliferation (cell division), cell growth and cell death, as well as interaction with other cells and the spread of growth into other organs.

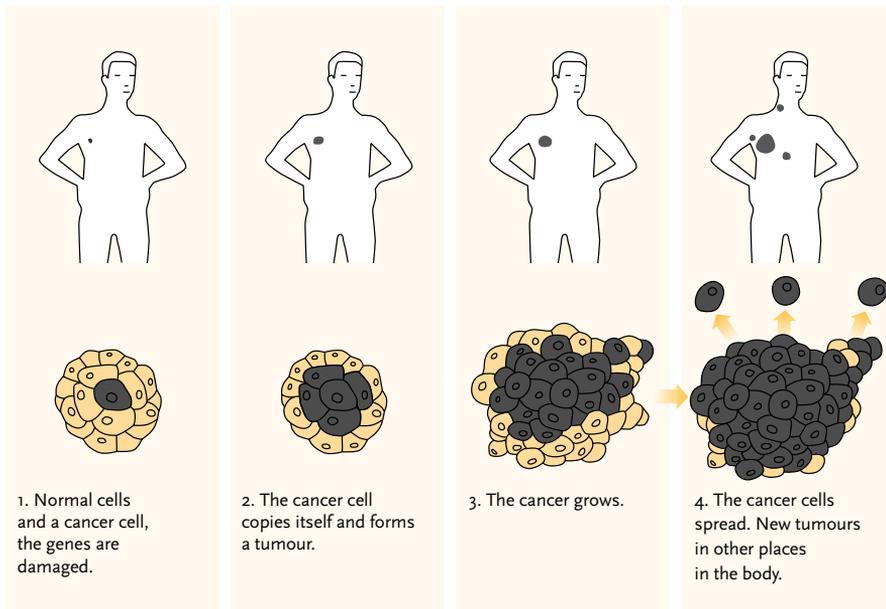


Figure 1. Development of cancer from a normal cell to a cancerous growth (1–20 years).

Cancer develops in a complex interaction between genetics, environment and lifestyle, in which a number of biological mechanisms influence one another. The time from when an uncontrolled process begins until the cancer can be diagnosed varies greatly and it can take up to 20 years from initial development of a cancer cell until a tumour is able to be detected in an organ. Who gets cancer and whether it can be prevented is dependent on whether the preventive factor or the factor to be used in treatment specific to the cancer in question, for example, physical activity, is able to affect the interaction between all the biological mechanisms involved.

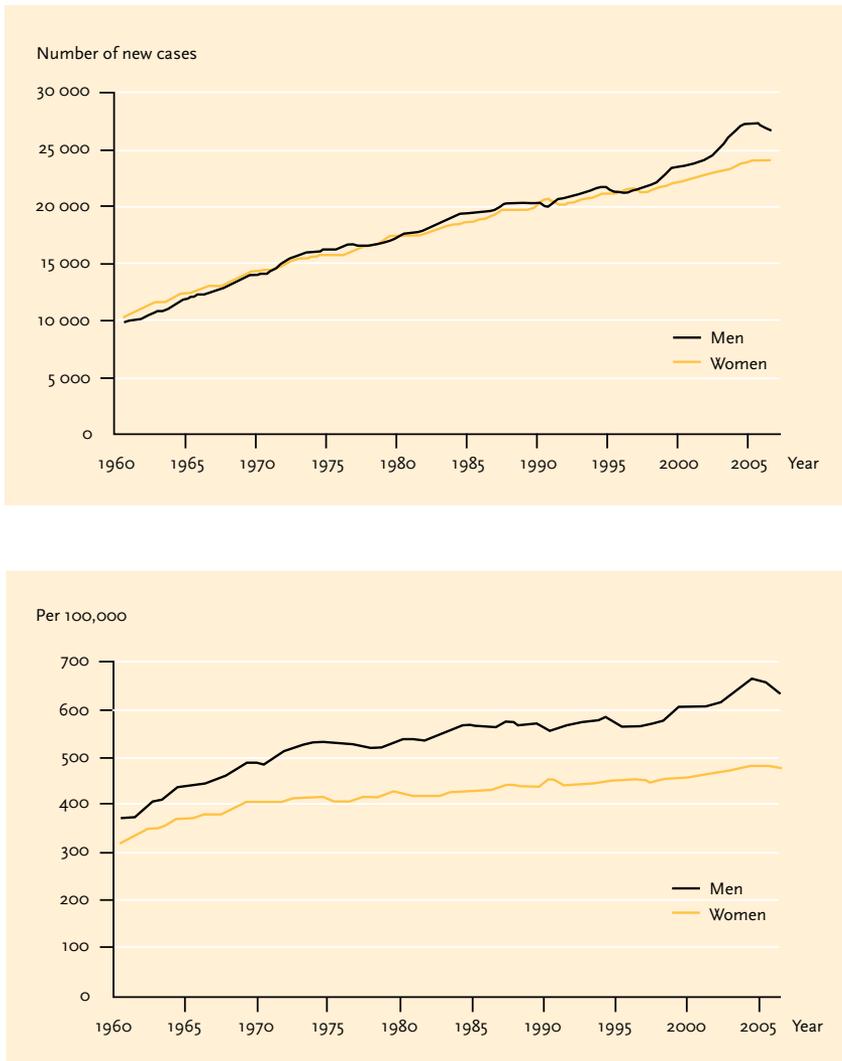


Figure 2. Cancer development in Sweden. Source: Swedish National Board of Health and Welfare, 2006 (4).

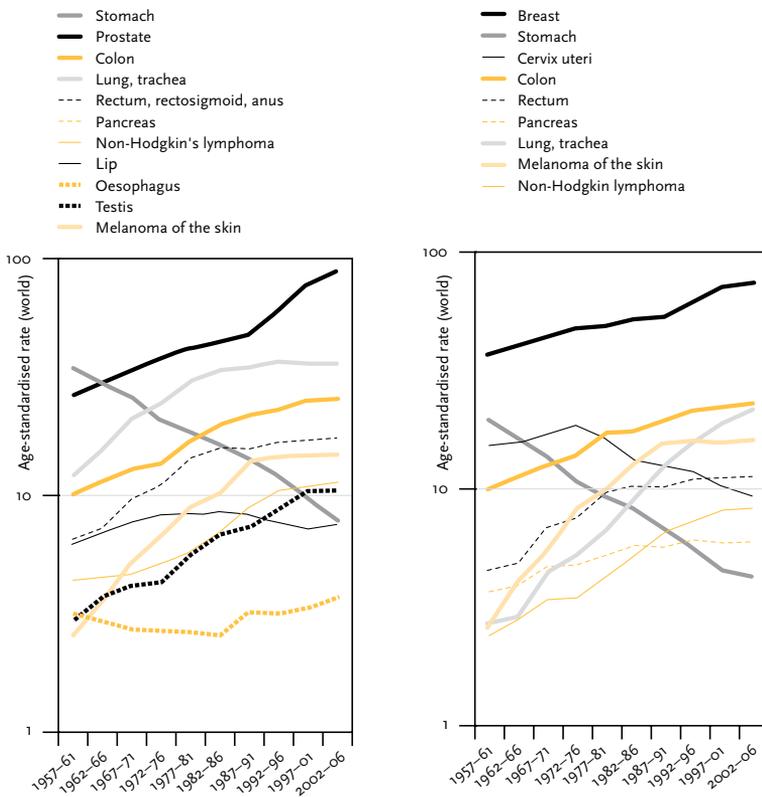


Figure 3. Cancer development in Norway. Source: Cancer Registry, Norway, 2007 (3).

The number of new cancer cases in the world has grown considerably. In Sweden and Norway, an increase of an entire 80 per cent has been noted over the past 50 years (3, 4). In the past few decades, the number of cancer cases has increased at an annual average of 1.7 per cent in men and 1.1 per cent in women. Prostate cancer is the most common type of cancer in men, while breast cancer dominates in women. The fact that we are living longer is partly responsible for the increase, but there is also an actual increase in the number of cancer cases. The chances of survival now are virtually double what they were 40 years ago. The survival rate has increased steadily since the 1950s. The reason for this is earlier diagnosis and better surgical treatment in the large diagnosis groups. Today's cytotoxins, radiation and hormone treatments have also helped. Of those who get breast cancer, 84 per cent now live longer than 5 years with the disease. Even the survival rate for patients with colorectal cancers has continually increased in recent years. In the late 1980s, three of ten patients with rectal cancer suffered a relapse, while the corresponding figure at the turn of the millennium was one in ten (3, 4).

Physical activity – biological mechanisms

Both the increased incidence of cancer and the increased percentage of people living with the disease are connected to our sedentary lifestyles. A growing number of studies show a relationship between physical activity and several types of cancer and there is discussion of whether physical activity may also have a role to play in treatment and rehabilitation (1, 5).

Physical activity is an important factor for good physical health and its positive relation to things such as intestinal function, immune status, energy balance and reduced menstrual pain have been known for several hundred years (6). Physical activity also affects a number of biological mechanisms that in turn affect cancer development and the risk of reoccurrence.

Demonstrating the effects of physical activity on concrete biological mechanisms of importance in cancer development has established the plausibility of the connection observed between physical inactivity and certain cancer diseases (7). We also know that cancer develops through an interplay of genetic predisposition/vulnerability and environment and lifestyle.

Early studies on the importance of physical activity in cancer development addressed energy metabolism (6, 8), sex hormone levels (6, 9), and animal studies the ability to influence DNA repair (1, 5). Other biological processes and factors have since been studied, for example, hyperinsulinemia, insulin resistance and other hormones (leptin), prostaglandins and C-reactive protein. Physical activity and energy balance have been shown, separately and in interaction, to influence these potentially cancer-related factors.

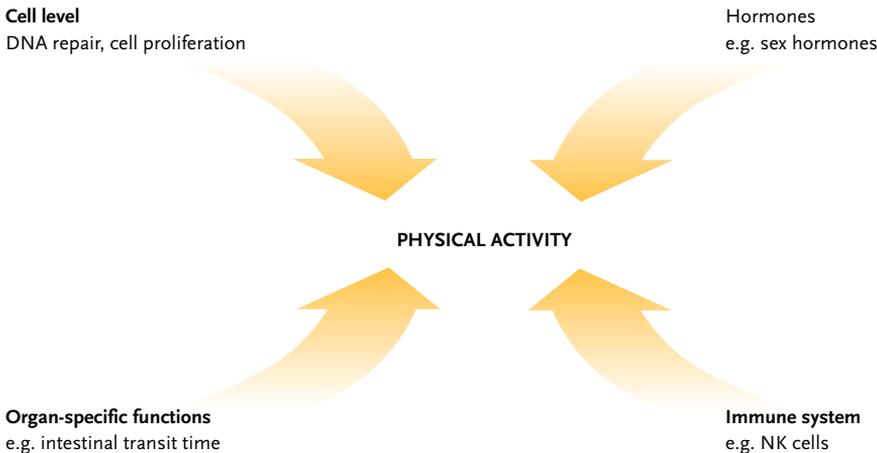


Figure 4. Physical activity and biological mechanisms.

The impact of physical activity on the metabolism of sex hormones is one of the factors that has been shown to have the strongest association to the protective effect against cancer in women. Women with elevated levels of oestrogen and androgen have an increased risk

of developing breast cancer, and anti-oestrogens are used both preventively and in treatment (1, 5). Elevated levels of oestrogen also increase the risk of uterine cancer.

Anti-androgen therapy is used to treat prostate cancer and has also been shown to prevent its development (5). Physical activity reduces the cumulative oestrogen dose that women are subjected to throughout life in a number of ways: it increases the age of menstrual onset, leads to changes in the hormonal environment of each menstrual cycle, and reduces levels of oestrogen and binding proteins in postmenopausal women. It has also been clearly demonstrated that physical activity affects testosterone levels in men, which has a potential effect on prostate cancer.

There are also other hormones related to variations in physical activity, for example, insulin. Physical activity affects insulin sensitivity and glucose uptake, shown in recent years to be linked to various types of cancer, for example, cancer of the colon, breast, uterus, prostate, pancreas and stomach. Insulin stimulates cell proliferation (cell division), inhibits apoptosis (cell death), and impacts the synthesis and availability of sex hormones.

An insulin-like growth factor, IGF-1, and its binding proteins, are believed to be associated with a higher risk for several forms of cancers, and variations in physical activity have been shown to affect the levels of this hormone.

The close relationship between physical activity and energy balance thus shows that physical activity levels are related to weight development in a population, and weight gain constitutes a risk factor for a number of cancer diseases (of the colon, breast, kidney, uterus). Physical activity also affects the availability of energy, which again plays a role in the overall ability to repair and control cells.

Variation in physical activity is associated with systemic inflammation, which in turn is related to a number of chronic diseases, including cancer. Pro-inflammatory factors such as C-reactive protein, serum amyloid A, interleukin-1 and TNF-alpha (tumour necrotic factor), and anti-inflammatory factors like adiponectin, are associated with cancer risk and prognosis. The importance of physical activity for these factors in relation to cancer risk is not clear.

The direct effect of physical activity on DNA repair and normal cell growth is studied as physical activity appears to affect a cell's ability to grow normally, for example, in the intestinal epithelium.

The immune system plays a role in the development of cancer with respect to identifying and eliminating unknown components. People with inherited immune diseases and/or congenital immune defects have a higher risk of cancer. Physical activity can both improve the quality and number of specific components in the immune system, for example, natural killer (NK) cells. An increase in physical activity results, in addition, in an increase to a number of immune system components (monocytes, neutrophils), followed by a reduction of these same factors to below-initial levels that lasts from 1–3 hours. In the case of continuous physical activity, there is a reverse dose-response relationship between these factors in the immune system and physical activity. The actual importance of physical activity and its effect on the immune system in relation to cancer development has, however, not been established.

There is great variation between individuals with respect to how long it takes food to pass through the intestine. Physical activity reduces this transit time and thus also the time that intestinal cells are subjected to potentially carcinogenic substances. One randomised controlled intervention study shows that physical activity reduces cell proliferation (cell division) in the colon (10). Physical activity can also affect the secretion of bile salts.

Physical activity also affects lung function, and improved lung capacity reduces the time that lung cells are in contact with carcinogenic elements in the air.

Table 1. Biological mechanisms – physical activity and cancer (prevention and prognosis).

Mechanisms	Effect	Type of cancer
Energy metabolism	Fat deposits that store/metabolise carcinogenic elements are reduced, carcinogens are reduced.	All types of cancer
Blood flow	Local and general blood flow increase and carcinogenic elements are reduced.	All types of cancer
Mechanical transit time stomach-intestine	Passage time for food and potentially carcinogenic elements is shortened.	Stomach-intestinal cancer
Respiration	Potential particle deposition in the lungs is reduced.	Lung cancer
Heat/trauma	Cell division/regeneration can increase.	All types of cancer
Sex hormones	Reduction of the cumulative levels of hormones that affect the growth of all cell types.	Breast, uterine and prostate cancer
Insulin and glucose	Insulin levels are reduced, sensitivity to insulin is improved.	Colon, breast, pancreatic, oesophageal, kidney, thyroid and uterine cancer
Inflammation	Reduces the ability of cells to repair themselves.	All types of cancer
Immune function	Optimisation of the number and activity of macrophages and lymphokine-activated NK cells.	All types of cancer

Measuring physical activity in relation to cancer

Different methods are used to measure physical activity in studies relating to cancer (1, 11, 12), which can make comparison difficult. Self-reported measurements like questionnaires and recorded data are often used, though in recent years direct observation and more objective measurements such as heart rate and fitness tests have also been used (1). This data is often linked to validation associated to energy metabolism and metabolic profile, and researchers have later attempted to gain knowledge of the total daily physical activity.

The most accurate self-reported measures of physical activity provide information about the type, intensity, frequency, duration and reason for the activity (e.g. cycling to work). Physical activity is thus quantified as minutes per day or metabolic equivalent (MET), or MET minutes per day (11). Calculations like these are important in order to study the dose-response relationship, a critical value related to specific cancer risk and survival. Another important factor is that the level of physical activity differs in different phases of life and varies over time, which appears to be significant for specific types of cancer.

Unfortunately there is no consensus on what level of activity constitutes “inactivity”. These levels vary between studies and are therefore often related to country, social group, age and gender. Often studies use a percentage of the study sample (e.g. one quarter) as the definition of “most inactive”. The levels of physical activity that form the basis for who is classified as inactive therefore vary between studies.

Primary prevention factors

Colorectal cancers

The relationship between physical activity and the risk for colorectal cancers has been investigated in many observation studies, epidemiological studies in several countries, in both men and women of different ages and ethnic groups. In healthy men and women who engage in regular physical exercise, the risk of colon cancer is reduced by 10–70 per cent (1, 5, 13–15). A threshold value for physical activity has not been able to be established, but studies indicate that the dose-response relationship is such that the longer the duration and the higher the intensity of physical activity, the higher the protective effect found for colon cancer. Men and women who reported high intensity during three periods of life, and men who burned more than 2500 kcal per week in high intensity physical activity, were shown to be able to cut their risk of developing colon cancer later in life by half (5).

Important biological mechanisms that reduce intestinal transit time, insulin sensitivity and cell proliferation in the intestinal epithelium have been studied and support the connection between physical activity and colon cancer (1, 5).

It has, however, not been established whether physical activity protects against rectal cancer. Even here, many studies have been conducted but have not yielded a similarly uniform picture. The existing biological mechanisms are not as convincing for rectal cancer either (1, 5).

Breast cancer

Oestrogen and progesterone levels are important factors in the development of breast cancer, and physical activity affects the production, metabolism and excretion of these hormones. It was thus not surprising that Frisch and colleagues reported early on, in 1985, that physical activity among college students in the US protected against later development of breast cancer (9). A number of studies and reports conclude that 4 hours of physical activity per week, at work and during leisure time, at an intensity corresponding to 6 METs (i.e. 6 times the oxygen uptake at rest, corresponding to a light jog), yields a 30–50 per cent reduction of breast cancer in postmenopausal women (16–18). Physical activity also protects younger, premenopausal women, but in this case genetic factors play a larger role than lifestyle and the protective effect is lower and can be associated to specific subgroups, for example, women with a particular genetic predisposition.

A study of women who carry the inherited BRCA1 and BRCA2 genes showed much earlier development of breast cancer in women who were not physically active compared to physically active women who carried the BRCA1/BRCA2 gene (19). This emphasises

the importance of the interaction between heredity and environment in the prevention of cancer, and that a factor such as physical activity can also be important for people with a congenital vulnerability to developing different types of cancer.

Another important aspect of physical activity as a protective factor against breast cancer is that it appears to have a particular effect during so-called “sensitive” periods, when the breast is especially susceptible to carcinogenic agents, for example, during puberty (1, 5, 18).

Uterine cancer

Due to its biological effect on energy balance, sex hormones and insulin sensitivity, physical activity may be of importance in cancer of the uterus. A number of studies have shown that physical activity gives a 20–40 per cent reduction in uterine cancer (1, 5, 20). Researchers have, however, been unable to identify especially critical periods or thresholds for physical activity.

Prostate cancer

Even if prostate cancer is a common and increasing form of cancer in men, we know very little about the underlying mechanisms for developing prostate cancer. Few of the risk factors are known. Physical activity as a potential effect on endogenous hormone levels, like testosterone and insulin, is equally important to consider. A series of studies have investigated the link between physical activity and prostate cancer (1, 21). Most of these studies, especially those that looked at the most aggressive and advanced types, have found that physical activity protects against prostate cancer (1, 5).

Lung cancer

Because physical activity can affect the time that potential carcinogenic agents are in contact with lung cells, both through improved lung capacity and overall blood flow, it is of interest to note that the majority of published studies (1, 22, 23) in this field show that physical activity protects against lung cancer. The problem of adjusting for so strong a risk factor as smoking, however, makes it difficult to draw final conclusions (1, 5).

Other types of cancer

A number of studies have been done on ovarian and testicular cancer, cancer of the kidneys and lymph nodes, etc. (though the number of studies in each individual disease group is limited). However, researchers have not arrived at a uniform picture of the importance of physical activity in these diseases.

Physical activity as treatment and rehabilitation in cancer disease

There used to be a perception that cancer was difficult to prevent and that patients treated for cancer should rest and reduce their physical activity. This is partly true in that physical activity can lead to pain, heart palpitations and breathing difficulties. Research has shown, however, that physical activity not only protects against a number of cancer diseases, but

is also valuable during both the treatment and rehabilitation phases (5, 24, 25). Physical activity also improves cancer patients' quality of life (25–27). Regular physical activity is an effective way of reducing side-effects resulting in part from the inactivity (13, 28) and in part from the disease itself. Inactivity leads to a loss of fitness, strength and mobility, unfavourable weight gain (29–31) and insulin sensitivity (32) in the patient. It is also known from a number of smaller and larger studies that cancer patients generally reduce their level of activity when the disease breaks out. Physical activity is therefore being recommended for more and more patients.

In recent years, there have also been a number of studies that looked at the relation between physical activity and survival, above all regarding breast cancer and colon cancer (33). After renewed review of the existing literature, international specialist groups nevertheless conclude that it is too early to determine whether physical activity can affect the survival of cancer patients (5, 24).

Possible benefits of regular physical activity

- Maintained and improved fitness
- Better balance and reduced risk of falls and broken bones
- Reduced risk of heart disease
- Prevents osteoporosis
- Improves blood flow and prevents blood clots
- Improves ability to manage on one's own and reduces dependence on others
- Better self-esteem
- Reduced anxiety and depression
- Reduced nausea
- Increased ability to maintain social network
- Reduced fatigue
- Better ability to maintain a stable weight
- Better quality of life
- Encourages a healthy and varied diet
- Possible improved survival

A person's level of physical activity is impacted by the ongoing treatment, the time that has passed since the previous treatment, medications, and the patient's fitness and stress levels. On the other hand, we know that physical activity in itself strengthens the muscles, improves fitness and the ability to tolerate medicines, and increases the chances of recovery. Physical activity can also lead to improved self-esteem and self-control. It is, however, important to seek the advice of the responsible physician and physiotherapist, so that the level of physical activity can be adapted to the individual's needs. In cases where the patient has no pain, 30–60 minutes of daily physical activity, adapted to the patient's situation and previous experience, is recommended.

Goals for physical activity

- Maintain good physical and social function
- Optimise the ability to provide an individually adapted treatment
- Reduce symptoms, with respect to nausea and fatigue in particular
- Attain an optimal weight and avoid unfavourable weight gain or weight loss

It is important to be aware that different types of cancer in themselves, as well as intensive treatment in the form of chemotherapy and radiation therapy, can produce very different pain symptoms. If a patient experiences pronounced fatigue after a difficult operation or intensive chemotherapy, it can be hard to motivate them to be physically active. Intensive radiation therapy can result in stomach problems in the form of powerful bowel movements, which may make activity impossible.

Type of activity, frequency, intensity and duration*Type of activity*

Activities that involve large muscle groups, for example, walking, cycling and skiing. Avoid heavy loading.

Frequency

Daily

Intensity

Moderate to high intensity adapted to the particular individual

Duration

A minimum of 15–60 minutes

The time must be adapted to the individual's situation, age and previous experience of physical activity and exercise.

Other considerations

During ongoing treatment it is important that the physical activity be discussed with the responsible physician, preferably in cooperation with the physiotherapist, and adjusted as necessary. It is important to remember that the cancer disease, ongoing treatment and blood profile, for example, low haemoglobin levels, affect the type of activity that should and can be performed. During ongoing treatment, the following special precautionary measures should be taken:

- Always consult the physician responsible for treatment before beginning an activity. This is of particular importance if the patient is taking special medications or has some form of heart or lung disease.

- Avoid activities that:
 - require high intensity in patients with low haemoglobin levels, < 8.0 g/dl
 - entail an increased risk for bacterial infection in patients with a low white blood cell count, $< 0.5 \times 10^9/l$
 - can lead to an increased risk of bleeding in cases where thrombocyte levels are $< 50 \times 10^9$, such as contact sports.
- In the case of shortness of breath – investigate cause, activity and tolerance.
- In the case of leg pain – avoid activities that can lead to increased risk of fracture, such as contact sports.
- In the case of pronounced fatigue – plan daily activities of a low to moderate level, preferably outdoors, balanced with rest.

References

1. International Agency Research Against Cancer. Weight control, physical activity and cancer. Chemoprevention. International Agency Research Against Cancer (IARC), IARC Press; 2002.
2. Trevathan WR, Smith EO, McKenna JJ. Evolutionary Medicine. Oxford University Press; 1999.
3. Norwegian Cancer Registry. Cancer in Norway 2006. Oslo: Norwegian Cancer Registry; 2007.
4. Swedish National Board of Health and Welfare. Cancer Incidence in Sweden 2006. Official Statistics of Sweden. Stockholm: Swedish National Board of Health and Welfare; 2006.
5. World Cancer Research Fund, American Institute for Cancer Research. Food, nutrition, physical activity, and the prevention of cancer. A global perspective. Washington (DC): American Institute for Cancer Research (AICR); 2007.
6. Rammazzini B. Diseases of the Workers (*De morbis artificum diatriba*, 1713). Wright WC, translator. New York: Hafner; 1964.
7. Batty D, Thune I. Does physical activity prevent cancer? Evidence suggests protection against colon cancer and probably breast cancer. *BMJ* 2000;321:1424-5.
8. Rabiagliatti A. Air, food, and exercises. In: An essay on the predisposing causes of disease, 3rd edn. London: Bailliere, Tindall and Cox; 1903, pp. 31-434.
9. Frisch RE, Wyshak G, Albright NL, Schiff I, Jones KP, Witschi J, et al. Lower prevalence of breast cancer and cancers of the reproductive system among former college athletes compared to non-athletes. *Br J Cancer* 1985;52:885-91.
10. Campbell KL, McTiernan A, Li SS, Sorensen BE, Yasui Y, Lampe JW, et al. Effect of a 12-month exercise intervention on the apoptotic regulating proteins Bax and Bcl-2 in colon crypts. A randomized controlled trial. *Cancer Epidemiol Biomarkers Prev* 2007;16:1767-74.
11. Ainsworth BE, Sternfeld B, Slattery ML, Daguisé V, Zahm SH. Physical activity and breast cancer. Evaluation of physical activity assessment methods. *Cancer* 1998;83: 611-20.
12. Thune I, Smeland S. Is physical activity important in the treatment and rehabilitation of cancer patients? *Tidsskr Nor Lægeforen* 2000;120:3302-4.
13. Gerhardsson de Verdier M, Steineck G, Hagman U, Rieger Å, Norell SE. Physical activity and colon cancer. A case-referent study in Stockholm. *Int J Cancer* 1990; 46:54;2390-7.
14. Slattery ML, Edwards SL, Ma KN, Friedman GD, Potter JD. Physical activity and colon cancer. A public health perspective. *Ann Epidemiol* 1997;7:137-45.
15. Nilsen TI, Romundstad PR, Petersen H, Gunnell D, Vatten LJ. Recreational physical activity and cancer risk in subsites of the colon. Nord-Trøndelag Health Study. *Cancer Epidemiol Biomarkers Prev* 2008;17:183-8.
16. Thune I, Brenn T, Lund E, Gaard M. Physical activity and risk of breast cancer. *N Engl J Med* 1997;336:1269-75.

17. Moradi T, Nyrén O, Zack M, Magnusson C, Persson I, Adami HO. Breast cancer risk and lifetime leisure-time and occupational physical activity (Sweden). *Cancer Causes Control* 2000;11:523-31.
18. Bernstein L, Patel AV, Ursin G, Sullivan-Halley J, Press MF, Deapen D, et al. Lifetime recreational exercise activity and breast cancer risk among black women and white women. *J Natl Cancer Inst* 2005;97:1671-9.
19. King MC, Marks JH, Mandell JB. Breast and ovarian cancer risk due to inherited mutations in BRCA1 and BRCA2. *Science* 2003;302(5645):643-6.
20. Furberg AS, Thune I. Metabolic abnormalities (hypertension, hyperglycemia and overweight), lifestyle (high energy intake and physical inactivity) and endometrial cancer risk in a Norwegian cohort. *Int J Cancer* 2003;104:669-76.
21. Friedenreich CM, Thune I. A review of physical activity and prostate cancer. *Cancer Causes Control* 2001;12:461-75.
22. Thune I, Lund E. The influence of physical activity on lung cancer risk. A prospective study of 81,516 men and women. *Int J Cancer* 1997;70:57-62.
23. Steindorf K, Friedenreich C, Linseisen J, Rohrmann S, Rundle A, Veglia F, et al. Physical activity and lung cancer risk in the European Prospective Investigation into Cancer and Nutrition Cohort. *Int J Cancer* 2006;119:2389-97.
24. Doyle C, Kushi LH, Byers T, Courneya KS, Demark-Wahnefried W, Grant B, et al. Nutrition and physical activity during and after cancer treatment. An American cancer society guide for informed choices. *CA Cancer J Clin* 2006;56:323-53.
25. Thorsen L, Skovlund E, Strømme SB, Hornslien K, Dahl AA, Fosså SD. Effectiveness of physical activity on cardiorespiratory fitness and health-related quality of life in young and middle-aged cancer patients shortly after chemotherapy. *J Clin Oncol* 2005;23:2378-88.
26. Thune I, Smeland S. Can physical activity prevent cancer? *Tidsskr Nor Lægeforen* 2000;120:3296-301.
27. Courneya KS, Mackey JR, Bell GJ, Jones LW, Field CJ, Fairey AS. Randomized controlled trial of exercise training in postmenopausal breast cancer survivors. Cardiopulmonary and quality of life outcomes. *J Clin Oncol* 2003;21:1660-8.
28. Abrahamson PE, Gammon MD, Lund MJ, Flagg EW, Porter PL, Stevens J, et al. General and abdominal obesity and survival among young women with breast cancer. *Cancer Epidemiol Biomarkers Prev* 2006;15:1871-7.
29. Irwin ML, Yasui Y, Ulrich CM, Bowen D, Rudolph RE, Schwartz RS, et al. Effect of exercise on total and intra-abdominal body fat in postmenopausal women. A randomized controlled trial. *JAMA* 2003;289:323-30.
30. Holmes MD, Chen WY, Feskanich D, Kroenke CH, Colditz GA. Physical activity and survival after breast cancer diagnosis. *JAMA* 2005;293:2479-86.
31. McTiernan A, Rajan KB, Tworoger SS, Irwin M, Bernstein L, Baumgartner R, et al. Adiposity and sex hormones in postmenopausal breast cancer survivors. *J Clin Oncol* 2003;21:1961-6.

32. Irwin ML, McTiernan A, Bernstein L, Gilliland FD, Baumgartner R, Baumgartner K, et al. Relationship of obesity and physical activity with C-peptide, leptin, and insulin growth factors in breast cancer survivors. *Cancer Epidemiol Biomarkers Prev* 2005;14:2881-8.
33. Pierce JP, Stefanick ML, Flatt SW, Natarajan L, Sternfeld B, Madlensky L, et al. Greater survival after breast cancer in physically active women with high vegetable-fruit intake regardless of obesity. *J Clin Oncol* 2007;25:2345-51.

20. Chronic obstructive pulmonary disease (COPD)

Author

Margareta Emtner, PT, PhD, Associate Professor, Uppsala University and Uppsala University Hospital, Uppsala, Sweden

Summary

Reduced physical performance capacity is common in people with chronic obstructive pulmonary disease. Destruction of the small airways and alveoli, inflammation of the bronchi and deterioration of skeletal muscle strength contribute to the reduced performance capacity. Exercise training improves physical capacity and reduces dyspnoea (shortness of breath). Everyone should be recommended to engage in 30 minutes of physical activity in daily life, 5–7 days per week. Participation in exercise training should also be encouraged. The training should comprise aerobic exercise (fitness training), dynamic strength training and flexibility training (see table below). Suitable activities include cycling, walking, and fitness training on land or in the water. No training should occur at saturation levels (oxygen saturation) below 88–90 per cent.

Type of training	Intensity	Frequency (times/week)	Duration
Aerobic fitness training	Low intensity: 55–70% of max HR* 40–60% of VO ₂ max**	2–5	≥ 30 min.
	High intensity: > 70% of max HR > 60% of VO ₂ max 60–80% W max***	2–3	≥ 30 min.
Dynamic strength training Leg, hip, core and shoulder muscles	70% of 1 RM****	2	8–12 reps, 2–3 sets

* Max HR = Maximal Heart Rate.

** VO₂ max = Maximal Oxygen Uptake.

*** W max = Maximal workload.

**** RM = Repetition Maximum. 1 RM corresponds to the maximum weight that can be lifted through the entire exercise movement one time.

Definition

Chronic obstructive pulmonary disease (COPD) is defined as follows: “Chronic obstructive pulmonary disease (COPD) is a preventable and treatable disease with some significant extrapulmonary effects that may contribute to the severity in individual patients. Its pulmonary component is characterised by airflow limitation that is not fully reversible. The airflow limitation is usually progressive and associated with an abnormal inflammatory response of the lung to noxious particles or gases” (1).

Cause and risk factors

COPD is primarily caused by smoking (80–95%), but the disease can also occur in non-smokers. Increasing age, heredity, low socioeconomic group, occupational exposure to industrial pollutants and urban environments increase the risk of developing the disease (2). Despite the fact that the risk of developing COPD is not influenced by gender, we know that the effect of smoking is larger for women. People born with a deficiency of the enzyme alpha1-trypsin can develop COPD, above all if they smoke. When it comes to smoking, there is a clear dose-response relation, that is, the more years of smoking the greater the risk of developing COPD (2).

Prevalence/Incidence

COPD is a public health disease that occurs mostly in older persons. In the Nordic countries, 4–6 per cent of the adult population have COPD (2). The prevalence among 45-year-old smokers is 5 per cent, and rises thereafter to 25% for 60-year-old smokers, and 50% of smokers who are 75 years old (2). Approximately 50 per cent have mild COPD, just over a third moderate COPD, and the remainder severe COPD. Mortality has increased during the 1990s and 2000s. According to the cause of death registry, in 2003, 2558 people with COPD died in Sweden. Mortality is related to the severity of the disease.

Pathophysiology

COPD is a disease characterised by airflow limitation, which is only reversible in certain cases. The disease is progressive and characterised by an inflammatory process in the airways and lung tissue. Consequently, there is a loss of elastic recoil and increased airway resistance, which limits both inhalation and exhalation capacity. At a later stage of the disease, a thickening of the vessel walls occurs, which has a negative effect on the gas exchange, and can lead to both hypoxia (low oxygen levels) and hypercapnia (high carbon dioxide levels). In severe cases, elevated blood pressure in the pulmonary circulation can develop, i.e., pulmonary hypertension. This affects the right side of the heart with right-sided heart failure, cor pulmonale, which in turn can lead to the development of oedema in the body. Dynamic hyperinflation can also occur, where an increased amount of air remains in the lungs (3). The dynamic hyperinflation results in a deterioration of the

length-tension relationship of the diaphragm muscle, which leads to increased respiratory effort.

COPD is not only a lung disease but also a systemic disease, that is, other organs and systems in the body are also affected (4). People with COPD often have reduced cardiovascular capacity, impaired peripheral skeletal muscle strength, hormonal changes (reduced levels of anabolic steroids), systemic inflammation, and an increased energy expenditure at rest. This limits their ability to be physically active (5, 6). The peripheral skeletal muscle shows both structural and biochemical changes: decreased number of type I fibres (oxidative) and high number of type II fibres (glycolytic), decreased muscle mass, decreased capillary density, and reduced number of aerobic enzymes (7).

Symptoms and diagnosis

The diagnosis is made on the basis of symptoms such as chronic cough, coughed up mucous, dyspnoea, increased respiratory work, increased production of secretions, hyper-reactivity, and a long history of smoking and symptom development. The diagnosis is confirmed with a lung function test, where FEV% is less than 0.70. FEV% is the ratio of FEV₁ (forced expiratory volume in one second) to (F)VC (FVC = forced vital capacity and VC = vital capacity, where the highest value of FVC and VC is used) ($FEV\% = FEV_1 / (F)VC$). Establishing a COPD diagnosis requires also a reversibility test, that is, a spirometric examination after inhalation of beta2 agonists. COPD is classified into four stages based on the FEV₁ value. People with an FEV% less than 0.70 and FEV₁ equal to or greater than 80 per cent of expected value have pre-clinical COPD, an FEV₁ between 50–79 per cent – mild COPD, an FEV₁ between 30–49 per cent – moderate COPD, and an FEV₁ under 30 per cent of expected value – severe COPD (1).

Prognosis

People with reduced lung function ($FEV_1 < 50\%$ of expected value) have a higher mortality (8). If they also present with pulmonary hypertension, the prognosis is even worse (9). People with hypoxia (reduced oxygen level in the blood) and hypercapnia (elevated level of carbon dioxide in the blood) also have poorer survival, as do those with impaired nutritional status and functional status (e.g., shorter walking distance) (10, 11). Middle-aged people with moderate or severe disease who quit smoking live an average of seven years longer than those who continue to smoke.

Treatment principles

Quitting smoking is the most effective treatment and results in reduced mortality as well as reduced symptoms (cough and production of secretions). Rehabilitation that includes exercise training, education, a review of diet, etc., is important and improves physical capacity, quality of life and dyspnoea (12). The pharmacological treatment includes bronchodilators like tiotropium, ipratropium and beta2 agonists. Inhaled steroids are recommended for

people with an FEV₁ lower than 50 per cent of expected value (13). Continuous oxygen treatment for people with respiratory failure is necessary and increases longevity (14). People with COPD should also be vaccinated against influenza and pneumococci prophylactically.

Effects of physical activity

Exercise training and physical activity have been shown to have positive effects both physiologically and psychologically. In addition to improvement in physical capacity after a period of training, patients with COPD who have taken part in training are less afraid of exerting themselves and become more physically active in their daily lives (15, 16). The quality of life (disease control and dyspnoea) improves, the sense of well-being increases (17), and morbidity decreases (18). On the other hand, no training study has shown a change in lung function (16).

Effects of aerobic fitness training

Oxygen uptake capacity (VO₂ max), which is reduced in people with COPD, increases significantly after a period of training (19, 20). Endurance capacity also increases significantly (21). Minute ventilation (VE), heart rate, dyspnoea, blood lactate levels and hyperinflation are decreased for the same exercise (19, 20, 22, 23). The oxidative enzymes (24) and oxygen extraction (25) of skeletal muscle are improved as a result of a period of training. The maximal workload increases 13–24 per cent, while endurance capacity increases by an average of 87 per cent, that is, the greatest effect is achieved in endurance capacity (26).

Effects of strength training

Resistance training for the legs improves muscle strength and muscle endurance (27, 28). Aerobic capacity can also improve (29, 30).

Effects of aerobic training combined with strength training

A combination of strength training and aerobic training improves both muscle strength and aerobic capacity (31, 32).

Long-term effects

Studies that looked at long-term effects of exercise training have shown that training must be kept up, even if at a somewhat lower level, in order to maintain the positive effects achieved (16).

Indications

All people with COPD who have a lower quality of life and/or lower physical capacity should be offered rehabilitation that includes exercise training (1). The training can occur when the patients are in a stable phase of the disease, but also in close connection to a period of exacerbation (33). Everyone with COPD can take part in exercise training, regardless of age and severity of the disease.

Prescription

The physical performance capacity of people with COPD is reduced and engaging in some form of exercise training is of great value, both physically and psychologically to everyone. All exercise training should be balanced, that is, include aerobic training (fitness training), strength training (endurance strength) and flexibility training (see Table 1) (34). The training should start with a warm-up component and end with cool-down and stretching.

To begin with, the training should occur under controlled forms and under the direction of a physiotherapist. In connection with training, it is important to measure oxygen saturation. Saturation should not fall below 88–90 per cent. If the saturation decreases, in the first place the workload (intensity) and/or the duration of the training should be reduced. Pursed-lip breathing can be used during training to maintain the saturation at an acceptable level, that is, ≥ 90 per cent. For hypoxemic people and people who desaturate during training ($\text{SaO}_2 < 88\%$), oxygen supplementation should be given during training (14). It has also been shown that normoxemic people with COPD can exercise at a higher intensity and thereby see more improvement if they receive supplemental oxygen during training (21). However, patients who require supplemental oxygen during training should strive to train without oxygen supplementation so that, if possible, they can move to training outside medical care. People with a low body mass index ($\text{BMI} < 22$) should be recommended to take nutritional supplements to help increase peripheral muscle strength and aerobic capacity. Pre-medicating with bronchodilators can be recommended for patients who usually have the assistance of these medications.

Patients with mild and severe COPD can perform aerobic exercise at a high intensity (22, 35). For untrained individuals, however, it can be good to begin at a low intensity. Patients with severely limited ventilation can be recommended to begin with strength training or only flexibility training.

Table 1. Description of different types of training.

Type of training	Intensity	Frequency (times/week)	Duration
Aerobic fitness training	Low intensity: 55–70% of max HR* 40–60% of VO ₂ max**	2–5	≥ 30 min.
	High intensity: > 70% of max HR > 60% of VO ₂ max 60–80% W max***	2–3	≥ 30 min.
Dynamic strength training Leg, hip, core and shoulder muscles	70% of 1 RM****	2	8–12 reps, 2–3 sets

* Max HR = Maximal Heart Rate.

** VO₂ max = Maximal Oxygen Uptake.

*** W max = Maximal workload.

**** RM = Repetition Maximum. 1 RM corresponds to the maximum weight that can be lifted through the entire exercise movement one time.

Aerobic training can be carried out at low intensity or high intensity and either as continuous or interval training. The effects of the two types of training are equivalent (36). All activities involving large muscle groups, and thereby loading the oxygen-transporting organs, are beneficial. Suitable activities include cycling, walking, and fitness training on land or in the water (26). For interval training, 2–3 minutes of high intensity training should be alternated with 1–2-minute intervals of low intensity training or active rest. The training should continue for at least 8–10 weeks (26). The greatest effect of training (measured as oxygen uptake) is achieved through high intensity training (22, 37).

Strength training should include endurance strength training and above all target the muscles used for movement (38). Training for core and shoulder muscles should also be included. Each exercise should be performed 8–12 times in 2–3 repetitions at an intensity of 70 per cent of 1 RM (RM = repetition maximum, 1 RM corresponds to the maximum weight that can be lifted through the entire exercise movement one time) (26). To begin with, however, it is completely sufficient to perform only one set. A rest period of 1–3 minutes should be added between sets. The training should continue for at least 8–10 weeks. For low intensity training (40–50% of 1 RM), the training can occur daily; for higher intensity (70–80% of 1 RM), however, training should occur 2 times per week (37, 39, 40).

Flexibility training should cover mobility exercises for the neck, shoulders, thorax, thigh and calf muscles, and be included in every training session.

Functional mechanisms

In aerobic training, there is an increase in the skeletal muscle of the enzymes that stimulate oxidative metabolism and oxygen extraction improves (7). The number of mitochondria increases and blood lactate levels fall for the same degree of workload, that is, oxygen can be metabolised better and the aerobic capacity therefore improves (20). Minute ventilation decreases and oxygen uptake capacity (VO_2) increases (19, 20, 22). In strength training, the cross-section surface area of type I and type IIa fibres increases. Quality of life and symptoms improve through exercise training. This is probably an effect of both a physical and psychological nature.

Functional tests

A functional test should always be conducted before physical training begins, in part to facilitate planning of an appropriate training programme, and in part to facilitate evaluation of the training. All testing should include measurement of saturation.

Cycle test and treadmill test

Standardised maximal or submaximal tests are carried out to investigate the patient's tolerance and limitations with respect to physical exertion. Heart rate, ECG, blood pressure, oxygen saturation, shortness of breath, exertion and chest pain should be recorded both during and for a short time after the test. Note: If heart disease is suspected, an ECG and blood pressure should be measured up to 5 minutes after the loading stops.

Walking test

Standardised walking tests are often used in clinical contexts to assess physical capacity in relation to activities of daily life. The Incremental Shuttle Walking Test (ISWT) (41) is a maximal test in which walking speed is increased every minute. The Endurance Shuttle Walking Test (ESWT) (42) is an endurance test that uses the same speed throughout the test. In both of these tests, the patient walks round two cones placed 9 metres apart. In a 6- or 12-minute walk test, the patient is encouraged to walk as far as possible in 6 or 12 minutes, respectively, on a measured stretch of hallway (43, 44). In all of the walking tests, the walking distance, heart rate, oxygen saturation, and perceived exertion and shortness of breath are measured on the Borg scale (45).

Muscle function

Both dynamic muscle strength and endurance can be measured with isokinetic devices. Dynamic muscle strength can in addition be measured by repetition maximum (RM). A suitable way to measure dynamic endurance strength is to have the person perform

a maximum number of repetitions at a given load. After a period of training, the test is repeated with the same load. An increase in the number of repetitions is an indication of an increase in muscle endurance.

Perception of quality of life and symptoms

General health-related quality of life can be measured with the Short-Form Health Survey (SF-36) (46), while disease-specific quality of life is often measured with the Chronic Respiratory Questionnaire (CRQ) or St. George's Respiratory Questionnaire (48). The severity of symptoms can be measured with a visual analogue scale (VAS) or the Borg scale.

Risks

No serious events need occur if the patient has undergone a functional test with ECG recording before commencing training, so that the physical limitations the patient demonstrates are known to the person in charge of or instructing the training. No strenuous training should occur if the disease is deteriorating. Many patients with COPD also have decreased cardiac function and high blood pressure. Blood pressure should therefore be monitored during the training.

Acknowledgement

I would like to thank Olav Kåre Refvem, Licensed Physician, Pulmonary Disease Specialist, and Carl C. Christensen, MD, Glittreklubben, Hakadal, Norway, for constructive views and updates.

References

1. Rabe KF, Hurd S, Anzueto A, Barnes PJ, Buist SA, Calverley P, et al. Global strategy for the diagnosis, management, and prevention of chronic obstructive pulmonary disease: GOLD Executive Summary. *American Journal of Respiratory and Critical Care Medicine*. 2007 Sep 15;176:532-55.
2. Lundbäck B. KOL-prevalens, incidens och riskfaktorer [COPD prevalence, incidence and risk factors]. In: Larsson K, Ed. KOL. Kroniskt obstruktiv lungsjukdom [Chronic Obstructive Pulmonary Disease]. Stockholm: Boehringer Ingelheim AB; 2006.
3. O'Donnell DE, Revill SM, Webb KA. Dynamic hyperinflation and exercise intolerance in chronic obstructive pulmonary disease. *American Journal of Respiratory and Critical Care Medicine* 2001;164:770-7.
4. Agustí AG, Noguera A, Sauleda J, Sala E, Pons J, Busquets X. Systemic effects of chronic obstructive pulmonary disease. *Eur Respir J* 2003;21:347-60.
5. Bernard S, LeBlanc P, Whittom F, Carrier G, Jobin J, Belleau R, et al. Peripheral muscle weakness in patients with chronic obstructive pulmonary disease. *American Journal of Respiratory and Critical Care Medicine* 1998;158:629-34.
6. Gosselink R, Troosters T, Decramer M. Peripheral muscle weakness contributes to exercise limitation in COPD. *American Journal of Respiratory and Critical Care Medicine* 1996;153:976-80.
7. Mador MJ, Bozkanat E. Skeletal muscle dysfunction in chronic obstructive pulmonary disease. *Respiratory Research* 2001;2:216-24.
8. Siafakas NM, Vermeire P, Pride NB, Paoletti P, Gibson J, Howard P, et al. Optimal assessment and management of chronic obstructive pulmonary disease (COPD). European Respiratory Society Task Force. *Eur Respir J* 1995;8:1398-420.
9. Barbera JA, Peinado VI, Santos S. Pulmonary hypertension in chronic obstructive pulmonary disease. *Eur Respir J* 2003;21:892-905.
10. Bowen JB, Votto JJ, Thrall RS, Haggerty MC, Stockdale-Woolley R, Bandyopadhyay T, et al. Functional status and survival following pulmonary rehabilitation. *Chest* 2000;118:697-703.
11. Soriano JB, Maier WC, Egger P, Visick G, Thakrar B, Sykes J, et al. Recent trends in physician diagnosed COPD in women and men in the UK. *Thorax* 2000;55:789-94.
12. Rabe KF, Beghe B, Luppi F, Fabbri LM. Update in chronic obstructive pulmonary disease 2006. *American Journal of Respiratory and Critical Care Medicine* 2007;175:1222-32.
13. Wise RA, Tashkin DP. Optimizing treatment of chronic obstructive pulmonary disease. An assessment of current therapies. *American Journal of Medicine* 2007;120:S4-13.
14. Cranston JM, Crockett AJ, Moss JR, Alpers JH. Domiciliary oxygen for chronic obstructive pulmonary disease. *Cochrane database of systematic reviews (Online)* 2005;4:CD001744.
15. Bendstrup KE, Ingemann Jensen J, Holm S, Bengtsson B. Out-patient rehabilitation improves activities of daily living, quality of life and exercise tolerance in chronic obstructive pulmonary disease. *Eur Respir J* 1997;10:2801-6.

16. Hill NS. Pulmonary Rehabilitation. *Proceedings of the American Thoracic Society* 2006;3:66-74.
17. Lacasse Y, Brosseau L, Milne S, Martin S, Wong E, Guyatt GH, et al. Pulmonary rehabilitation for chronic obstructive pulmonary disease. *Cochrane database of systematic reviews (Online)* 2002:CD003793.
18. Griffiths TL, Burr ML, Campbell IA, Lewis-Jenkins V, Mullins J, Shiels K, et al. Results at 1 year of outpatient multidisciplinary pulmonary rehabilitation. A randomised controlled trial. *Lancet* 2000;355:362-8.
19. Casaburi R, Porszasz J, Burns MR, Carithers ER, Chang RS, Cooper CB. Physiologic benefits of exercise training in rehabilitation of patients with severe chronic obstructive pulmonary disease. *American Journal of Respiratory and Critical Care Medicine* 1997;155:1541-51.
20. Maltais F, LeBlanc P, Jobin J, Bérubé C, Bruneau J, Carrier L, et al. Intensity of training and physiologic adaptation in patients with chronic obstructive pulmonary disease. *Am J Crit Care Med* 1997;155:555-61.
21. Emtner M, Porszasz J, Burns M, Somfay A, Casaburi R. Benefits of supplemental oxygen in exercise training in nonhypoxemic chronic obstructive pulmonary disease patients. *American Journal of Respiratory and Critical Care Medicine* 2003;168:1034-42.
22. Casaburi R, Patessio A, Ioli F, Zanaboni S, Donner CF, Wasserman K. Reductions in exercise lactic acidosis and ventilation as a result of exercise training in patients with obstructive lung disease. *The American Review of Respiratory Disease* 1991;143:9-18.
23. Porszasz J, Emtner M, Goto S, Somfay A, Whipp BJ, Casaburi R. Exercise training decreases ventilatory requirements and exercise-induced hyperinflation at submaximal intensities in patients with COPD. *Chest* 2005;128:2025-34.
24. Maltais F, Simard AA, Simard C, Jobin J, Desgagnes P, LeBlanc P. Oxidative capacity of the skeletal muscle and lactic acid kinetics during exercise in normal subjects and in patients with COPD. *American Journal of Respiratory and Critical Care Medicine* 1996;153:288-93.
25. Sala E, Roca J, Marrades RM, Alonso J, Gonzalez De Suso JM, Moreno A, et al. Effects of endurance training on skeletal muscle bioenergetics in chronic obstructive pulmonary disease. *American Journal of Respiratory and Critical Care Medicine* 1999;159:1726-34.
26. Troosters T, Casaburi R, Gosselink R, Decramer M. Pulmonary rehabilitation in chronic obstructive pulmonary disease. *American Journal of Respiratory and Critical Care Medicine* 2005;172:19-38.
27. Spruit MA, Gosselink R, Troosters T, De Paepe K, Decramer M. Resistance versus endurance training in patients with COPD and peripheral muscle weakness. *Eur Respir J* 2002;19:1072-8.
28. Casaburi R, Bhasin S, Cosentino L, Porszasz J, Somfay A, Lewis MI, et al. Effects of testosterone and resistance training in men with chronic obstructive pulmonary disease. *American Journal of Respiratory and Critical Care Medicine* 2004;170:870-8.

29. Clark CJ, Cochrane L, Mackay E. Low intensity peripheral muscle conditioning improves exercise tolerance and breathlessness in COPD. *Eur Respir J* 1996;9:2590-6.
30. Simpson K, Killian K, McCartney N, Stubbing DG, Jones NL. Randomised controlled trial of weightlifting exercise in patients with chronic airflow limitation. *Thorax* 1992;47:70-5.
31. Bernard S, Whittom F, LeBlanc P, Jobin J, Belleau R, Berube C, et al. Aerobic and strength training in patients with chronic obstructive pulmonary disease. *American Journal of Respiratory and Critical Care Medicine* 1999;159:896-901.
32. Ortega F, Toral J, Cejudo P, Villagomez R, Sanchez H, Castillo J, et al. Comparison of effects of strength and endurance training in patients with chronic obstructive pulmonary disease. *American Journal of Respiratory and Critical Care Medicine* 2002;166:669-74.
33. Puhan MA, Scharplatz M, Troosters T, Steurer J. Respiratory rehabilitation after acute exacerbation of COPD may reduce risk for readmission and mortality. A systematic review. *Respiratory Research* 2005;6:54.
34. Haskell WL, Lee IM, Pate RR, Powell KE, Blair SN, Franklin BA, et al. Physical activity and public health. Updated recommendation for adults from the American College of Sports Medicine and the American Heart Association. *Circulation* 2007;116:1081-93.
35. Punzal PA, Ries AL, Kaplan RM, Prewitt LM. Maximum intensity exercise training in patients with chronic obstructive pulmonary disease. *Chest* 1991;100:618-23.
36. Arnardottir RH, Boman G, Larsson K, Hedenstrom H, Emtner M. Interval training compared with continuous training in patients with COPD. *Respiratory Medicine* 2007;101:196-204.
37. ATS. Pulmonary rehabilitation-1999. American Thoracic Society. *American Journal of Respiratory and Critical Care Medicine* 1999;159:1666-82.
38. Hodgkin J, Celli BR, Connors GL, Eds. Pulmonary Rehabilitation. Guidelines to success. 3. edn. Baltimore: Lippincott Williams and Wilkins; 2000.
39. British Thoracic Society. Pulmonary Rehabilitation. *Thorax* 2001;56:827-34.
40. Storer T. Exercise in chronic pulmonary disease. Resistance exercise prescription. *Med Sci Sports Exercise* 2001;33:S680-S6.
41. Singh SJ, Morgan MD, Scott S, Walters D, Hardman AE. Development of a shuttle walking test of disability in patients with chronic airways obstruction. *Thorax* 1992;47:1019-24.
42. Revill SM, Morgan MD, Singh SJ, Williams J, Hardman AE. The endurance shuttle walk. A new field test for the assessment of endurance capacity in chronic obstructive pulmonary disease. *Thorax* 1999;54:213-22.
43. Guyatt G, Sullivan M, Thompson P, Fallen E. The 6-minute walk. A new measure of exercise capacity in patients with chronic heart failure. *Can Med Assoc J* 1985;132:919-32.
44. McGavin C, Groupta S, McHarty G. 12-minute walking test for assessing disability in chronic bronchitis. *Br Med J* 1976;1:822-3.

45. Borg GA. Psychophysical bases of perceived exertion. *Medicine and Science in Sports and Exercise* 1982;14:377-81.
46. Ware J, Scherbourne C. The MOS 36-Item Short-Form Health Survey (SF-36). Conceptual framework and item selection. *Med Care* 1992;30:473-83.
47. Guyatt GH, Berman LB, Townsend M, Pugsley SO, Chambers LW. A measure of quality of life for clinical trials in chronic lung disease. *Thorax* 1987;42:773-8.
48. Jones P, Quirk F, Baveystock C, Littlejohns P. A self-complete measure of health status for chronic airflow limitation. *The American Review of Respiratory Disease* 1992; 145:1321-7.

21. Coronary artery disease

Authors

Agneta Ståhle, PT, PhD, Associate Professor, Department of Neurobiology, Care Sciences and Society, Division of Physiotherapy, Karolinska Institutet, Stockholm, Sweden

Åsa Cider, PT, PhD, Physiotherapy Department, Sahlgrenska University Hospital, Gothenburg, Sweden

Summary

Coronary artery disease, that is, angina pectoris or myocardial infarction, is one of our most widespread public diseases. Coronary artery disease involves the presence of pathological changes, arteriosclerosis, in the walls of one or more of the coronary vessels. Physical inactivity is a potent risk factor for coronary artery disease, but old age, male gender and heredity, as well as smoking, high blood pressure, blood lipid disorders, diabetes and overweight also increase the risk of developing the disease. Prescribing a minimum of 30 minutes per day of regular physical activity constitutes excellent primary prevention against coronary artery disease, and regular exercise, aerobic exercise 3–5 times per week and resistance exercise 2–3 times per week, is a powerful treatment for already established coronary artery disease.

A recommendation to increase physical activity can be given generally in a primary preventive aim, but in order to plan optimal exercise as secondary prevention requires that the patient be tested with respect to aerobic fitness and muscle function.

The assessment begins with a stress test/fitness test with ECG monitoring, a muscle function test, and assessment of the current level of physical activity. Based on these tests and the patient's general condition, a risk assessment is made, and thereafter an appropriate exercise programme and physical activity level is drawn up for the patient.

It is essential that the initial rehabilitation is carried out under supervision, preferably that of a specialised physiotherapist and access to emergency care equipment. Most patients exercise for 3–6 months under the direction of cardiac rehabilitation, and most often the exercise can then continue outside the hospital's management when the condition has been properly stabilised.

Table 1. Description of training methods investigated in different scientific studies in patients with coronary artery disease.

Training method	Intensity	RPE***	Frequency (times/week)	Duration
Central circulation aerobic training, distance or interval	50–80% of VO ₂ max*	12–15 central	3–5	40–60 min./ session
Resistance training	1–3 sets of 10–15 RM** (65–75% of 1 RM)	13–16 local	2–3	8–10 exercises

* VO₂ max = Maximal Oxygen Uptake.

** RM = Repetition Maximum. 1 RM corresponds to the maximum weight that can be lifted through the entire exercise movement only one time.

*** RPE = Rate of Perceived Exertion (Borg scale 6–20).

Definition

Prevalence/Incidence

Coronary artery disease is one of the most widespread public diseases with a prevalence in the population in Sweden of just under 200,000 cases. Two thirds of the patients are men. Women develop the disease somewhat later than men. Every year, there are an estimated 10,000 new cases of angina pectoris (1). Both mortality and development of ischaemic heart disease have decreased since 2004, and preliminary data also suggests that the lower numbers remain for 2005. According to the Swedish National Board of Health and Welfare registry for cause of death, 17,971 people died of ischaemic heart disease in 2004, whereof approximately 9800 of myocardial infarction (2). In recent decades, medical treatment and intervention (bypass surgery and percutaneous coronary intervention [PCI]) have yielded better results, which has led to more patients surviving acute development of the disease, meaning in turn a successive increase in the number of patients in need of cardiac rehabilitation with exercise.

Cause

Coronary artery disease involves the formation of pathological changes in the wall of one of more of the coronary arteries, so-called hardening of the arteries or arteriosclerosis, and is the most common cause of acute coronary events, that is, acute myocardial infarction or unstable angina pectoris (3).

Risk factors

Old age, male gender and hereditary factors for cardiovascular disease, as well as risk factors such as physical inactivity, smoking, high blood pressure, blood fat disorders, overweight/obesity and diabetes, increase the risk of developing coronary artery disease (3).

Pathophysiological mechanisms, symptoms and diagnostics

Hardening of the arteries (atherosclerosis) is the predominant cause of acute coronary artery disease. Atherosclerosis primarily attacks the innermost layer of the artery wall, the intima, which is made up of endothelial cells. Initially, a storing of blood fats (lipids) occurs between the endothelial cells, where inflammatory cells, macrophages, ingest the fat. The macrophages ingest the lipids until they burst and become so-called “foam cells”. A fibrous mass then develops around the foam cell, forming a plaque. These atherosclerotic plaques do not attack the entire vessel but appear in patches. The area around the arterial branch points are particularly susceptible (4, 5).

Symptoms

The dominating symptom in acute coronary artery disease is usually *central chest pain* and includes both unstable angina and acute myocardial infarction. However, at onset women often present with more unspecific symptoms like breathing difficulties, nausea or other forms of pain. *Angina pectoris* (constriction of the arteries) is described as stable when symptoms have been present for at least a few weeks without obvious signs of worsening. Effort angina is angina induced by physical or mental stress that ceases quickly once the exertion has stopped (1), whereas variant (spasm) angina is considered to be induced by contraction of (constriction in) an artery that lasts so long that the heart muscles are subjected to a symptom-producing shortage of oxygen. Variant angina can occur during rest and mixed types are not uncommon (6). The course in people with stable angina has improved with the introduction of effective treatment options, such as anti-ischaemic, antithrombotic, antihypertensive and blood lipid-lowering drugs, as well as surgical and catheter-based coronary artery interventions, percutaneous coronary interventions (PCI). The prognosis for individuals with stable angina is now relatively good. It is important, however, to be alert of any destabilisation of the angina, characterised by a rapid worsening, which in most cases requires emergency hospital care (5). In most individuals, fissuring or rupture of an atherosclerotic plaque in a coronary artery is the precipitating cause of the acute element of coronary artery disease (4). The subsequent course, with activation of thrombocytes and plasma coagulation, leads to the formation of a blood clot (thrombosis) that completely or partially blocks the artery. Ischaemia occurs when the artery that supplies blood is partially or completely blocked and leads to reduced availability of oxygen and nutrients as well as removal of debris. When an artery is blocked, a gradual change toward cardiac cell death occurs, that is dependent on the degree of ischaemia and how long it lasts (7). The structural changes that the heart undergoes after a *myocardial infarction* are not restricted to the infarct zone, but also spread to the “healthy myocardium”, which must compensate for the loss of function in the damaged area by, among other things, hypertrophy of the heart muscle, capillary growth, and storing of collagen in the healthy areas, resulting in a more rigid heart with poorer energy balance (8). These adverse effects can be reduced by medicating with beta blockers and ACE inhibitors (5).

Diagnostics and assessment of prognosis should be commenced as early as possible parallel to introducing treatment. With a patient history, ECG and biochemical markers of myocardial damage, the diagnosis can be made and prognosis determined within the first few hours in most patients. Sometimes additional examinations are needed, such as echocardiography, coronary radiology and/or a stress test with ECG monitoring. If acute coronary artery disease is suspected, the patient should always be admitted to hospital and treated, with PCI for example, as soon as possible (9).

Treatment principles

The treatment of acute coronary artery disease, which should be initiated as soon as possible after symptom onset, comprises reperfusion therapy and/or antithrombotic therapy. Reperfusion is usually achieved using PCI and/or in combination with insertion of a stent, the purpose of which is to prevent the vessel from closing up again (reocclusion) or pharmacological treatment (thrombolysis) or a combination of these (facilitated PCI). PCI is also used for unstable angina, and in this case after a coronary x-ray to verify actual coronary artery changes. In some cases, coronary artery bypass graft surgery (CABG) is necessary.

In other cases, pharmacological anti-ischaemic treatment to stabilise the condition is offered, and then primarily with the aid of pharmaceuticals such as acetylsalicylic acid (ASA), beta blocker, nitrates and calcium channel blockers. In some cases, primarily in the case of reduced pumping capacity such as heart failure, ACE inhibitors are used (5).

Effects of physical activity

The positive effects of being physically active when suffering from coronary artery disease was shown as early as the end of the 1700s (10, 11) These findings were unfortunately lost to memory and it was not until the mid-1960s that exercise was used as therapy for coronary artery disease (12, 13). The first cardiac rehabilitation programmes based on physical training were then started, and the first Swedish recommendations issued in 1980 (14).

Acute effects

Acute physiological effect of exercise in ischaemic heart disease

Heart rate, stroke volume and cardiac output

The cardiovascular system's immediate response to exercise is an increase in heart rate due to reduced activity in the parasympathetic nervous system (vagal slowing). This is followed by increased activity in the sympathetic nervous supply to the heart and the body's blood vessels. Relatively rapid heart rate during submaximal exertion or post-exercise recovery is often seen soon after a myocardial infarction or heart surgery. An unusually low heart rate during submaximal exertion can be due to beta blocker medications or an increase in stroke volume from the exercise. The use of beta blockers, which lower the heart rate, limit the interpretation of heart rate response in exercise.

At an early stage during exercise, cardiac output increases through an increase in stroke volume due to an improved length-tension relationship in the heart muscle. This is called the Frank Starling mechanism, and involves an enhancement of force when the muscle fibre lengthens. The lengthening of the heart muscle fibre is due to increased venous return flow. The increase in cardiac output occurs mainly through an increase in heart rate, meaning that when beta blocker therapy is given the maximal cardiac output becomes lower.

Arrhythmias

The presence of different rhythm disturbances (arrhythmias) is not uncommon in ischaemic heart disease. If the arrhythmias present at rest and disappear during exertion, they are usually benign. If, however, the arrhythmias increase during exertion, there is cause to stop the exercise and discuss further medical investigation (15).

Blood pressure

Systolic blood pressure rises with increased dynamic exercise as a result of the increased cardiac output. Diastolic blood pressure usually remains unchanged or is somewhat higher. It is important to note in the clinical work that, during auscultation, diastolic pressure can be heard right down to zero during exertion, and can thereby be a false reading.

An insufficient fall in blood pressure or elevation of blood pressure can occur during exercise. During ongoing exertion, a poor rise in blood pressure or a drop in blood pressure is due to impeded outflow in the aorta, severe left ventricle dysfunction, angina in the heart or beta blocker medication. In certain individuals with heart disease, however, the blood pressure can increase (over and above the measured maximal exertion value) in the recovery phase.

If the exercise is stopped abruptly, some individuals may experience a substantial fall in systolic blood pressure. This drop in blood pressure is due to accumulation of venous blood and a delayed increase in peripheral resistance adjusted to the decrease in cardiac output.

Oxygen uptake in the heart

The oxygen uptake of the heart during exercise can be calculated using the so-called double product (rate pressure product = RPP), defined as the systolic blood pressure times the heart rate divided by 100. There is a linear relation between the heart's oxygen uptake and the heart's blood supply that occurs primarily in the diastolic phase. During exercise, the blood flow to the heart muscle can increase up to five times the resting value. A person with heart disease is usually not able to maintain adequate blood flow to the ischaemia-affected part of the heart and the heart's metabolic requirements during exercise can thereby not be met, resulting in acute oxygen deficiency in the heart muscle, angina pectoris.

Skeletal muscle blood flow and peripheral resistance

The skeletal muscle blood flow can increase three-fold during exercise and the total peripheral resistance decreases due to increased vasodilatation in working skeletal muscle during exercise. In the case of beta blocker therapy, a somewhat smaller increase in blood flow in the working muscle occurs, which is why the feeling of fatigue in peripheral musculature is greater in patients receiving beta blockers (15).

Long-term effects of exercise in ischaemic heart disease

Regular exercise training in people with coronary artery disease results in similar specific changes, for example, in skeletal muscle- and cardiovascular capacity, as in healthy individuals. Generally speaking, the effect is dependent on the type of exercise. In the case of more aerobic-oriented training, there is improvement in above all oxygen uptake capacity (VO_2), while strength training results in increased muscle function of specifically trained muscles. The training effects obtained allow an individual to exercise at a higher exertion level and/or at a lower heart rate for each submaximal level. Studies have shown that medium intensity aerobic training of both cardiac patients and healthy individuals for 8–12 weeks, in 45 minute sessions 3–5 times per week, leads to marked improvements in both maximal and submaximal exertion levels (16).

Lower resting heart rate (resting bradycardia)

A lower resting heart rate is perhaps the most obvious effect of regular exercise. The underlying mechanisms include an altered autonomic balance and increased stroke volume. This effect is seen in both healthy individuals and those with heart disease, with or without beta blocker (15).

Lower blood pressure

Resting blood pressure and blood pressure at a given level of exertion are lower in well-trained individuals. Blood pressure is cardiac output times peripheral resistance. Because peripheral resistance decreases during exercise, this results in reduced expulsion resistance for the left ventricle and an increased ejection fraction (i.e. the percentage of the blood that is pumped out of the heart with each heart beat, or the heart's ability to pump out blood) and stroke volume are obtained. For every given level of submaximal exertion, a lower systolic pressure results in a corresponding lowering of the double product, and this in turn leads to a reduced risk for ischaemia in the heart. During vigorous exercise, high pressure builds within the working muscle group, which can lead to a reduction or even blocking (occlusion) of muscle blood flow, which is why afterload (i.e. the muscle contraction or tension that develops in the ventricular wall during systole) in turn increases. The consequence of this is a limitation of stroke volume and ejection fraction (15). Occlusion of intramuscular vessels begins when the muscles contract at 15 per cent of their maximal voluntary contraction (MVC) and becomes complete at approximately 70 per cent of MVC. In patients with heart disease and impaired muscle strength, appropriate regular resistance training can lead to better heart function because a stronger skel-

etal muscle results in the vascular contraction (constriction) not occurring until a higher per cent of MVC (17).

Increased peripheral venous tone

Exercise results in an increase in vein tone (tension), which increases central blood volume and thereby the filling pressure of the heart (ventricular preload). Cardiac output thus increases and the risk for a pronounced drop in blood pressure (hypotension) after exercise training decreases (15).

Increased stroke volume and contractility of the heart muscle

Exercise results in a certain increase in the heart muscle's (myocardial) contractility. This helps to increase stroke volume and benefits oxygen uptake. The increased stroke volume leads to about the same corresponding increase in functional activity of the heart, and a given physical exertion can then be performed at a lower level of the individual's VO_2 max. The lower heart rate reduces the double product and therefore the oxygen required by the heart muscle is reduced and the risk for oxygen deficiency (angina) decreases. Aerobic exercise can also increase the blood flow in the coronary vessels by improving the vessel's elasticity and increasing endothelium-dependent vasodilatation of the arteries. Aerobic exercise also leads to regeneration of vessels, which increases the surface of the coronary vessel bed and the density of the heart capillaries. The above-mentioned effects help to increase the ischaemic threshold, such that the exertion level at which angina is precipitated is higher (15).

Endothelial function and the blood coagulation system

Studies of patients with myocardial infarction have shown that exercise has a positive effect on fibrinolytic enzymes. Exercise is also important to reduce the blood platelets' ability to stick together. Together with an increase in plasma volume and decrease in blood viscosity, these changes reduce the risk for blood clots in the coronary vessels. The vascular endothelium plays an important role in the regulation of arterial vessel tone, blood pressure and local thrombocyte aggregation, that is, the ability of the blood platelets to stick together, through the release of endothelium-dependent relaxing factors. One such factor is nitric oxide (NO) that is released via the increased pressure (shear stress) placed on the endothelial cell wall with increased blood flow. Endothelium-dependent vasodilatation is impaired in patients with ischaemic heart disease. There is convincing evidence that exercise improves endothelial function in both healthy individuals and those with heart disease through increasing the endothelium-dependent vasodilatory capacity, mainly by increased release of NO (18–22). Convincing evidence also exists for the positive impact of exercise on the blood coagulation (fibrinolytic) system (23, 24).

Chronic inflammation

Inflammation has been shown to be closely linked to the development of arteriosclerosis. Studies have shown that aerobic exercise lowers the level of C-reactive protein (CRP), which could indicate that regular exercise has an anti-inflammatory effect. However, there are at present no studies on patients with ischaemic heart disease (25–27).

Autonomic function

Aerobic exercise can increase the threshold for ventricular tachycardia (rapid, extra heart beats initiated by the ventricle). This effect reduces the risk for sudden death by reducing the activity in the sympathetic nervous system and raising parasympathetic activity. It has also been shown that exercise raises VO_2 max in both atrial fibrillation and ventricular arrhythmias (28–30).

Exercise also has a positive effect on a number of factors important in the development of cardiovascular disease. Examples of these include blood lipids, cholesterol and insulin sensitivity. Even other lifestyle changes are significant and through adding regular physical activity into one's new lifestyle other lifestyle factors, such as diet and smoking, are also positively impacted. This can further reduce the risk for cardiovascular morbidity and mortality.

Exercise and effect on mortality

Exercise in cardiac rehabilitation, as compared to regular care, lowers both total mortality (20%) and mortality specifically related to heart disease (cardiac mortality) (26%) (31). The specific mechanisms that can contribute to reduced mortality in connection with exercise training are not yet fully established and probably relate to several factors (17). Table 2 describes possible biological mechanisms for the reduced mortality.

Table 2. Long-term regular exercise training affects many of the factors that contribute to a reduction of mortality related to heart disease (cardiac mortality).

Cardiovascular effect

- Lower heart rate at rest and during exercise.
- Lower blood pressure at rest and during exercise.
- Lower oxygen demand in the heart at submaximal levels of exercise training.
- Increase in plasma volume.
- Increased myocardial contractility.
- Increased peripheral venous tone.
- Positive changes in fibrinolytic (blood coagulation) system.
- Increased endothelium-dependent vasodilatation.
- Increased gene expression for production of an enzyme (NO synthase) that helps to produce nitric oxide (NO).
- Increased parasympathetic activity.
- Increase in coronary blood flow, coronary collateral vessels and myocardial capillary density.

Metabolic effect

- Reduced obesity.
- Increased glucose tolerance.
- Improved blood lipid profile.

Lifestyle effect

- Reduced likelihood of smoking.
- Possible reduction of stress physiological responses.
- Possible short-term reduction of appetite.

Indication

All physically inactive healthy people and patients with established coronary artery disease.

Primary prevention

A number of scientific studies in the last decade have shown regular physical activity to be health-promoting in all age groups (32). Increasing one's capacity for physical activity also reduces the risk of dying of coronary artery disease (33). Physical inactivity is now considered a primary risk factor for developing coronary artery disease (34) and is just as potent a risk factor as smoking, elevated blood lipids and high blood pressure (35). There is a dose-response relation between the level of physical activity and cardiovascular illness and death, which means that every increase in activity level is an improvement! "A little is better than none, more is better than a little" (36).

Epidemiological studies that have looked at the impact of physical activity level on developing and dying from cardiovascular disease have found that if the total amount of energy used for physical activity exceeds 4200 kJ per week (\approx 1000 kcal/week), for example, regular brisk walking for more than three hours per week, complemented by more vigorous activities/exercise, the risk for developing coronary artery disease decreases by 20 per cent for men (37) and 30–40 per cent for women (38). It is perfectly fine to divide the physical activity into shorter sessions (39); the main thing is that one burns energy through physical activity.

Secondary prevention

Secondary prevention of coronary artery disease means that after a manifest cardiac event, such as myocardial infarction, coronary intervention (coronary artery surgery or PCI) or in the case of persistent angina that cannot be further corrected medically (so-called refractory angina), measures should be taken that in both the short- and long-term can prevent death, relapse and progression of the underlying disease (16). In already established heart disease, regular, adapted exercise is required in order to achieve a reduction in mortality. This means that the exercise must be drawn up according to the current physical capacity. As an individual's clinical picture and performance capacity may vary from occasion to occasion, especially in the acute phase, special care is needed for these patients. A stress test/fitness test with ECG monitoring, and a muscle function test should be carried out before exercise commences. Based on the outcomes of these tests and a patient history (anamnesis) aimed at identifying individual risk factors (physical inactivity, smoking, high blood lipids, high blood pressure, overweight, diabetes), a risk profile assessment is made, in which consideration is given to the current physical capacity and possible symptoms during exertion.

Regular exercise in cardiac rehabilitation is a potent measure that reduces mortality by 26 per cent. Training entails aerobic exercise 3–5 times per week and resistance training 2 times per week (see Table 3).

Functional tests

All exercise at the physiotherapist should be preceded by some form of loading test, where general aerobic fitness and functional capacity is evaluated before a level of exercise is selected. *A stress test/fitness test* with ECG monitoring *is a requirement* and should be conducted with current medication. Assessment of muscle function entails testing of 10 RM (repetition maximum) before drawing up an individually tailored programme with resistance exercise.

Physical activity level is assessed with the aid of a questionnaire survey and pedometer. These tests can be performed again after the exercise period is complete to evaluate the results achieved from the exercise programme and continued prescription of exercise (40).

Prescription

Type of activity

The general goal of exercise in cardiovascular disease is to improve aerobic capacity through loading the central circulatory system. When it comes to the central circulation, large muscle groups should be used. Exercise can be conducted as intervals or as distance training. One Norwegian study shows that interval training led to higher VO_2 max compared to distance training (41). There is nevertheless a need for more and bigger studies on interval versus distance training before we are able to state that the one type of exercise is superior to the other in patients with coronary artery disease (42).

Each exercise session should always begin with a warm-up phase and end with a cool-down phase of similar length, regardless of the activity being done. Interval training means alternating between harder and easier intervals while distance training maintains the same level of intensity throughout the entire session (43). If there is a tendency to exertion-induced chest pain, the warm-up should be a bit longer than normal.

All exercise should begin with a successive warm-up of 6–10 minutes at an intensity of up to 40–60 per cent of VO_2 max and an exertion level of 10–12 according to the Borg RPE scale (15, 44). A proposed interval training is three loading exercise sessions of 4–5 minutes at an intensity of up to 60–80 per cent of VO_2 max and with an exertion level of “somewhat hard” to “hard”, corresponding to 13–15 on the RPE scale. Between the loading intervals follow 4–5-minute sessions lighter exercise at an intensity of up to 40–60 per cent of maximal capacity and an exertion level of 10–12 on the RPE scale.

Distance training means exercising at the same level for approximately 20–40 minutes. The load can then lie at 13–14 on the Borg exertion scale. All exercise should finish off with successive cool-down and stretching of at least 6–10 minutes.

Table 3. Description of exercise training methods investigated in different scientific studies in patients with coronary artery disease.

Training method	Intensity	RPE***	Frequency (times/week)	Duration
Aerobic central circulatory training, distance or interval	50–80% of VO ₂ max*	12–15 central	3–5	40–60 min./session
Resistance training	1–3 sets of 10–15 RM** (65–75% of 1 RM)	13–16 local	2–3	8–10 exercises

* VO₂ max = Maximal oxygen uptake.

** RM = Repetition Maximum. 1 RM corresponds to the maximum weight that can be lifted through the entire exercise movement only one time.

*** RPE = Rate of Perceived Exertion (Borg scale 6–20).

The choice of activity should always be preceded by a history of the patient's physical activity where consideration is given to current aerobic fitness level, interests and requirements. Aerobic exercise can be carried out in the form of brisk walks, jogging, cycling, swimming, exercise or aquafit classes, skiing, skating, dance or ball sports, depending on the individual's interests, and should include 30–45 minutes of exercise, 3–5 times per week. This should be complemented with at least 30 minutes of daily physical activity, which need not be strenuous nor performed all at once and can include everything from regular moving about to walks and climbing stairs (36, 39). The goal is to achieve a daily energy expenditure of at least 660 KJ (² 150 kcal). When the extra weekly energy expenditure from exercise is added to the daily physical activity, the energy expenditure will exceed what is considered sufficient to achieve health effects (39).

Resistance training, which used to be considered contraindicated for cardiovascular disease, has in recent studies shown to be both a safe and effective way to exercise (45, 46). The patient should perform 1–3 sets of 8–10 resistance exercises, 10–15 RM, 2–3 times per week (15). If the patient has a very low physical capacity, peripheral muscle training may in certain cases be the type of exercise needed before other exercise, in order to enable other forms of activity. For a more detailed description of this type of exercise, see the chapter on Heart failure.

Women, the elderly and certain immigrant groups and cardiac rehabilitation

Studies have shown that rehabilitation for cardiovascular disease is underused by women, the elderly and certain immigrant groups (47) despite these patients having great benefit from cardiac rehabilitation (48, 49). It is therefore especially important to offer and encourage these patient groups to take part in exercise for cardiac rehabilitation.

Interactions with drug therapy

Beta receptor blockers

Beta blockers have a well-documented effect for coronary artery disease. They reduce the myocardial oxygen demand primarily through a reduction of heart rate, but also by lowering blood pressure as well as some reduction in myocardial contractility. These effects are seen both at rest and during exertion. The effect is similar for all drugs in this group and is dose-dependent (50). A certain local fatigue, above all in the leg muscles, can present during exertion, however, and can be attributed to a reduced blood flow with subsequent shortage of oxygen in the working muscles (51). In spite of the metabolic and circulatory changes reported for beta blockers, the oxygen uptake capacity increases after fitness training similarly in people with coronary artery disease and concurrent beta blocker therapy and in people without beta blockers (52). The effects of exercise are independent of age and resemble the effects attained in healthy individuals (53).

Calcium channel blockers

Certain calcium channel blockers (verapamil, diltiazem) are negative chronotropes, that is, they lead to a lowering of resting heart rate and reduced maximal heart rate. As a rule, this limits VO_2 max, though the drugs themselves do not present a particular risk in connection with exertion.

Diuretics

Diuretics do not affect heart rate and cardiac contractility to any great extent, but lead to a decrease in plasma volume, peripheral resistance and blood pressure. Diuretics can also produce hypokalaemia, which leads to muscle weakness and extra ventricular beats.

In warm weather, diuretics can have potentially negative effects through an increased risk of dehydration and electrolyte disturbances (15).

ACE inhibitors

ACE inhibitors have a secondary preventive effect after myocardial infarction and here especially in people with concurrent heart failure (50). From a haemodynamic standpoint, these drugs have similar effects both at rest and during exertion, and lower the blood pressure by reducing peripheral resistance. None of the drugs have a negative effect on the haemodynamic response in exercise.

Nitrates

The oldest drug still being used for coronary artery disease is nitroglycerin. Nitrates come in short-acting forms, which counteract individual attacks, and in long-acting, preventive form. None of these affect physical performance capacity negatively, and can sometimes be taken before exercise with a preventive aim (50).

Contraindications

Absolute contraindications for physical activity and exercise are unstable angina and/or recent symptom onset that are severely debilitating. These people should be treated in hospital, with medical and/or invasive therapies. Serious heart rhythm disturbances (e.g. ventricular tachycardias, total atrioventricular block) constitute an obstacle, as do insufficiently regulated hypertension and ongoing infection that affects the patient's general condition.

Relative contraindications. Tolerance for arrhythmias is generally reduced if the patient is hypoglycemic (low blood sugar level) and/or dehydrated. It is therefore important to monitor these factors in all types of training and especially in people with heart disease.

Risks

The relative safety of supervised exercise in cardiac rehabilitation is well documented. The incidence of cardiovascular events during supervised exercise is low and ranges from 1/50,000 to 1/120,000 person hours of exercise for non-fatal cardiac events and 1 death/750,000 person hours of exercise. Cardiac rehabilitation always contains risk stratification in order to identify patients with an increased risk of cardiovascular events in connection with exercise (15).

It is important to note, however, that approximately half of all cardiac complications occur during the first month after suffering an acute coronary event. At 1-year follow-up, a high-risk patient runs three times the risk of myocardial infarction compared to a low-risk patient. It is therefore essential that the initial rehabilitation is carried out under supervision, and under the direction of a physical therapy specialist with access to emergency care equipment. An stress test with ECG monitoring before commencement of exercise is an important tool for determining the level of exercise, as well as for ruling out possible exertion-related symptoms that can have a negative effect on one's ability to train (54).

References

1. Swedish National Board of Health and Welfare. Socialstyrelsens riktlinjer för hjärtsjukvård 2004. Det medicinska 1. [Swedish National Board of Health and Welfare Guidelines for Cardiac Medical Care 2004. Medical Aspects 1.] Swedish National Board of Health and Welfare. Socialstyrelsens riktlinjer för hjärtsjukvård 2004. Det medicinska faktdokumentet. [Swedish National Board of Health and Welfare Guidelines for Cardiac Medical Care 2004. Medical Facts Document.] Stockholm: Swedish National Board of Health and Welfare; 2004.
2. Swedish National Board of Health and Welfare. Statistik. Hälsa och sjukdomar 2007:4. Hjärtinfarkter 1987–2004 samt utskrivna för vård av akut hjärtinfarkt 1987–2005. [Statistics. Health and Diseases 2007:4. Myocardial infarctions 1987-2004 and discharged after acute myocardial infarction care 1987-2005.] Stockholm: Swedish National Board of Health and Welfare; 2007.
3. Libby P, Bonow RO, Zipes DP, Mann DL. Braunwald's Heart Disease. London: Saunders; 2007.
4. Davies MJ. The composition of coronary-artery plaques. *N Engl J Med* 1997;336:1312-4.
5. Wallentin L. Akut kranskärlssjukdom. [Acute Coronary Artery Disease] Stockholm: Liber; 2005.
6. Lanza GA. Cardiac syndrome X. A critical overview and future perspectives. *Heart* 2007;93:159-66.
7. Roque M, Badimon L, Badimon JJ. Pathophysiology of unstable angina. *Thromb Res* 1999;95:V5-14.
8. Willems IE, Arends JW, Daemen MJ. Tenascin and fibronectin expression in healing human myocardial scars. *J Pathol* 1996;179:321-5.
9. Libby P, Bonow R, Braunwald E, Zipes D. Pathophysiology of heart failure. Amsterdam: Elsevier; 2004.
10. Heberden W. Pectoris dolor. In: Payne T, Ed. Commentaries on the history and cure of diseases. London; 1807.
11. Parry C. An inquiry into the symptoms and causes of syncope angionosa, commonly called angina pectoris. Illustrated by dissections. London: R Cutwell for Cadell and Davies; 1799.
12. Sanne H, Selander S. Mobilization and rehabilitation in cases of myocardial infarction. *Läkartidningen* 1967;64:1539-45.
13. Hellerstein HK. Exercise therapy in coronary disease. *Bull N Y Acad Med* 1968;44:1028-47.
14. Ekelund C, Ekelund L-G, Kinnman A, Rydén L, Sanne H, man-Rydberg A. Återanpassning efter hjärtinfarkt [Readjustment after Myocardial Infarction]. Stockholm: SPRI; 1980.
15. Fletcher GF, Balady GJ, Amsterdam EA, Chaitman B, Eckel R, Fleg J, et al. Exercise standards for testing and training. A statement for healthcare professionals from the American Heart Association. *Circulation* 2001;104:1694-740.

16. Balady GJ, Williams MA, Ades PA, Bittner V, Comoss P, Foody JM, et al. Core components of cardiac rehabilitation/secondary prevention programs. 2007 Update. A scientific statement from the American Heart Association Exercise, Cardiac Rehabilitation, and Prevention Committee, the Council on Clinical Cardiology; the Councils on Cardiovascular Nursing, Epidemiology and Prevention, and Nutrition, Physical Activity, and Metabolism; and the American Association of Cardiovascular and Pulmonary Rehabilitation. *Circulation* 2007;115:2675-82.
17. Shephard RJ, Balady GJ. Exercise as cardiovascular therapy. *Circulation* 1999;99:963-72.
18. Edwards DG, Schofield RS, Lennon SL, Pierce GL, Nichols WW, Braith RW. Effect of exercise training on endothelial function in men with coronary artery disease. *Am J Cardiol* 2004;93:617-20.
19. Farsidfard F, Kasikcioglu E, Oflaz H, Kasikcioglu D, Meric M, Umman S. Effects of different intensities of acute exercise on flow-mediated dilatation in patients with coronary heart disease. *Int J Cardiol* 2007 Mar 16. (Epub ahead of print).
20. Gielen S, Adams V, Niebauer J, Schuler G, Hambrecht R. Aging and heart failure. Similar syndromes of exercise intolerance? Implications for exercise-based interventions. *Heart Fail Monit* 2005;4:130-6.
21. Linke A, Erbs S, Hambrecht R. Exercise and the coronary circulation—alterations and adaptations in coronary artery disease. *Prog Cardiovasc Dis* 2006;48:270-84.
22. McAllister RM, Laughlin MH. Vascular nitric oxide. Effects of physical activity, importance for health. *Essays Biochem* 2006;42:119-31.
23. deJong AT, Womack CJ, Perrine JA, Franklin BA. Hemostatic responses to resistance training in patients with coronary artery disease. *J Cardiopulm Rehabil* 2006;26:80-3.
24. Paramo JA, Olavide I, Barba J, Montes R, Panizo C, Munoz MC, et al. Long-term cardiac rehabilitation program favorably influences fibrinolysis and lipid concentrations in acute myocardial infarction. *Haematologica* 1998;83:519-24.
25. Caulin-Glaser T, Falko J, Hindman L, La Londe M, Snow R. Cardiac rehabilitation is associated with an improvement in C-reactive protein levels in both men and women with cardiovascular disease. *J Cardiopulm Rehabil* 2005;25:332-6, Quiz 337-8.
26. Gielen S, Walther C, Schuler G, Hambrecht R. Anti-inflammatory effects of physical exercise. A new mechanism to explain the benefits of cardiac rehabilitation? *J Cardiopulm Rehabil* 2005;25:339-42.
27. Goldhammer E, Tanchilevitch A, Maor I, Beniamini Y, Rosenschein U, Sagiv M. Exercise training modulates cytokines activity in coronary heart disease patients. *Int J Cardiol* 2005;100:93-9.
28. Hautala AJ, Makikallio TH, Kiviniemi A, Laukkanen RT, Nissila S, Huikuri HV, et al. Heart rate dynamics after controlled training followed by a home-based exercise program. *Eur J Appl Physiol* 2004;92:289-97.
29. Tsai MW, Chie WC, Kuo TB, Chen MF, Liu JP, Chen TT, et al. Effects of exercise training on heart rate variability after coronary angioplasty. *Phys Ther* 2006;86:626-35.

30. Wu SK, Lin YW, Chen CL, Tsai SW. Cardiac rehabilitation vs. home exercise after coronary artery bypass graft surgery. A comparison of heart rate recovery. *Am J Phys Med Rehabil* 2006;85:711-7.
31. Taylor RS, Brown A, Ebrahim S, Jolliffe J, Noorani H, Rees K, et al. Exercise-based rehabilitation for patients with coronary heart disease. Systematic review and meta-analysis of randomized controlled trials. *Am J Med* 2004;116:682-92.
32. Blair SN, LaMonte MJ, Nichaman MZ. The evolution of physical activity recommendations. How much is enough? *Am J Clin Nutr* 2004;79:913S-20.
33. Pate RR, Pratt M, Blair SN, Haskell WL, Macera CA, Bouchard C, et al. Physical activity and public health. A recommendation from the Centers for Disease Control and Prevention and the American College of Sports Medicine. *JAMA* 1995;273:402-7.
34. Ades PA, Savage PD, Poehlman ET, Brochu M, Fragnoli-Munn K, Carhart RL Jr. Lipid lowering in the cardiac rehabilitation setting. *J Cardiopulm Rehabil* 1999;19:255-60.
35. Sundberg CJ, Jansson E. Reduced morbidity and the risk of premature death. Regular physical exercise is beneficial for health at all ages. *Läkartidningen* 1998;95:4062-7.
36. Le Masurier GC, Sidman CL, Corbin CB. Accumulating 10,000 steps. Does this meet current physical activity guidelines? *Res Q Exerc Sport* 2003;74:389-94.
37. Lee IM, Sesso HD, Paffenbarger RS Jr. Physical activity and coronary heart disease risk in men. Does the duration of exercise episodes predict risk? *Circulation* 2000;102:981-6.
38. Manson JE, Hu FB, Rich-Edwards JW, Colditz GA, Stampfer MJ, Willett WC, et al. A prospective study of walking as compared with vigorous exercise in the prevention of coronary heart disease in women. *N Engl J Med* 1999;341:650-8.
39. Lee IM, Paffenbarger RS Jr. Associations of light, moderate, and vigorous intensity physical activity with longevity. The Harvard Alumni Health Study. *Am J Epidemiol* 2000;151:293-9.
40. Ades PA. Cardiac rehabilitation and secondary prevention of coronary heart disease. *New Engl J Med* 2001;345:892-902.
41. Rognmo O, Hetland E, Helgerud J, Hoff J, Slordahl SA. High intensity aerobic interval exercise is superior to moderate intensity exercise for increasing aerobic capacity in patients with coronary artery disease. *Eur J Cardiovasc Prev Rehabil* 2004;11:216-22.
42. Swain DP, Franklin BA. Is there a threshold intensity for aerobic training in cardiac patients? *Med Sci Sports Exerc* 2002;34:1071-5.
43. Wilmore JH, Costill DL. *Physiology of sport and exercise*. Illinois: Champaign; 2005.
44. Borg G. Perceived exertion as an indicator of somatic stress. *Scand J Rehab Med* 1970;2:92-8.
45. McCartney N. Role of resistance training in heart disease. *Med Sci Sports Exerc* 1998;30:S396-402.
46. McCartney N. Acute responses to resistance training and safety. *Med Sci Sports Exerc* 1999;31:31-7.
47. Jeger RV, Jorg L, Rickenbacher P, Pfisterer ME, Hoffmann A. Benefit of outpatient cardiac rehabilitation in under-represented patient subgroups. *J Rehabil Med* 2007;39:246-51.

48. Mochari H, Lee JR, Kligfield P, Mosca L. Ethnic differences in barriers and referral to cardiac rehabilitation among women hospitalized with coronary heart disease. *Prev Cardiol* 2006;9:8-13.
49. Allen JK, Scott LB, Stewart KJ, Young DR. Disparities in women's referral to and enrollment in outpatient cardiac rehabilitation. *J Gen Intern Med* 2004;19:747-53.
50. Nordlander R, Schwahn Å, Lindström B. Ishemisk hjärtsjukdom. Läkemedelsboken 2007/2008. [Ischaemic heart disease. The Drug Book.] Stockholm: Apoteket AB; 2007.
51. Eston R, Connolly D. The use of ratings of perceived exertion for exercise prescription in patients receiving beta-blocker therapy. *Sports Med* 1996;21:176-90.
52. Wenger NK. Quality of life in chronic cardiovascular illness. *Ann Acad Med Singapore* 1992;21:137-40.
53. Gordon NF, Duncan JJ. Effect of beta-blockers on exercise physiology. Implications for exercise training. *Med Sci Sports Exerc* 1991;23:668-76.
54. Brauer K, Jorfeldt L, Pahlm, O. Det kliniska arbetsprovet [The Clinical Stress Test]. Lund: Studentlitteratur; 2003.

22. Cystic fibrosis

Authors

Louise Lannefors, PT, Heart and Lung Division, Lund University Hospital, Lund, Sweden

Ulrika Dennersten, PT, Heart and Lung Division, Lund University Hospital, Lund, Sweden

Sandra Gursli, PT, Norwegian Centre for Cystic Fibrosis, Oslo, Norway

Johan Stanghelle, MD, PhD, Professor, Sunnaas Hospital, Nesoddtangen, and University of Oslo, Oslo, Norway

Summary

The clinical picture of cystic fibrosis (CF) can vary considerably. The most common symptoms are malnutrition and rapidly progressive obstructive lung disease, which for most CF sufferers can entail respiratory insufficiency, secondary heart disease (cor pulmonale) and the need for lung transplantation. At present, treatment is only symptomatic and is aimed at slowing the rapid advancement of the disease. With optimal treatment, individuals with CF can live well into their adult years. Half of all patients with CF in Sweden and Norway are now older than 18 years, meaning that CF is no longer only a childhood disease. Since the beginning of the 1980s, fitness, strength and flexibility training have become an important part of the basic treatment. The choice of exercises, intensity, duration and frequency must be adapted to the individual's requirements, conditions and current situation. For most patients, it is the continuing deterioration of lung function that gradually becomes the limiting factor for physical capacity, but all patients with CF can perform some kind of physical activity and training.

Definition

Cystic fibrosis (CF) is the most common hereditary (autosomal recessive) and potentially deadly disease in the white population (1–3), but occurs in all races. A recent estimate of the incidence in Sweden was approximately 1/5600 infants (4) and the incidence in Norway is estimated to be about the same. Cystic fibrosis is a disease that attacks several organs in the body and is due to disorders in salt transport across cell membranes. CF affects the body's exocrine glands (mucous and pancreatic glands), affecting the transport of sodium

and chloride through the cell membrane, which in turn leads to very thick, sticky mucus (1–3). Disorders in the transport system of salt also affect the patient's sweat, which contains high levels of salt (3). The diagnosis is made on the basis of clinical symptoms with the aid of a sweat test (1) and can now often be confirmed with gene analysis.

Symptoms

Symptoms present primarily in the lungs and gastrointestinal tract but may also occur in other parts of the body (5). The changed environment in the airways of the lungs leads to the mucociliary clearance system not working properly. Peripherally, that is, behind the “mucus plugs”, this creates a deoxygenated environment, which serves as a breeding ground for the bacteria chronically found in colonised CF lungs. Studies have shown that seemingly asymptomatic infants have signs of infection and inflammation already at 4–6 weeks of age (6). Most of these children become chronic carriers of one or more types of bacteria found in our environment that do not affect healthy individuals. Stagnated secretions, inflammation and chronic bacterial infections of the pulmonary airways are the most common symptoms (1, 3).

Without treatment, the disease leads to malnutrition, chronic obstructive bronchitis, repeated cases of pneumonia and destruction of the lung tissue in the form of bronchiectasis, fibrosis and emphysema (1). This leads to escalating impairment of lung function, which in time can lead to respiratory insufficiency and cor pulmonale. At this point, lung transplantation is the only possible treatment option. The chronic obstruction can be caused by a number of different factors such as bronchial spasms, swelling of the mucous membrane, a collection of mucus and instability of the airways. In some patients there may also be an element of bronchial hyperresponsiveness or an asthmatic component (3). The risk of losing fitness, mobility and muscle strength increases as lung function deteriorates. Some patients also suffer from chronic infections and sinusitis. Spontaneous rib fractures can occur secondary to frequent coughing, as can problems with incontinence, especially in women, even in younger years. Herniation of the abdominal muscle wall or the groin can also occur. The obstructive respiratory pattern and pulmonary hyperinflation can lead to a stiff thorax, straining of the muscles used for inspiration and coughing, and rupturing of the intercostal muscles. Spontaneous pneumothorax can occur, as can haemoptysis, ranging from small harmless streaks of blood in the sputum to severe bleedings that require acute treatment.

In the gastrointestinal tract, the viscous secretion of the pancreas inhibits normal secretion of digestive enzymes, resulting in malabsorption of fat and fat-soluble vitamins (3), which also leads to vitamin- and mineral deficiencies. Left untreated, malnutrition in the childhood years leads to stunted growth and in adults to increasing weight loss. An obstructive respiratory pattern and increased respiratory exertion, chronically activated immune defenses and constant inflammation of the mucous membrane of the airways causes great expenditure of energy (7–9). The increased consumption of energy combined with malnutrition leads to increasing muscle atrophy (10). Osteopenia (diminished bone density) occurs as early as the late teens, with some individuals also developing osteoporosis (11). With age, CF-related diabetes may develop (3).

The clinical picture varies considerably. The disease is progressive in nature and treatment is symptomatic but primarily preventive. The rate of progression is also individual and varies between different periods of life in the same individual.

Treatment and its goals

There is at present no treatment that will cure CF, but symptomatic treatment is being developed continually (2). The goal of treatment is to prevent destruction of the lung tissue and to slow the disease's rate of progression by controlling symptoms and maintaining good physical function of the patient (12). Treatment includes both short- and long-term goals and involves active daily intervention. Achieving good compliance with treatment requires active support and ongoing education of patients and their families.

The physiotherapist must be able to define immediate and long-range problems and needs, and be able to present these in a positive manner. In order to maintain lung function and physical capacity in the long term, a practical and motivated treatment therapy must be the goal for every individual. To achieve good compliance, the agreed-upon treatment must be followed up, reviewed and evaluated frequently. The patient and physiotherapist always arrive at such agreements together, with both parties equal participants and willing to compromise. This is an important requirement to be able to achieve a high level of compliance with daily treatment (13–16).

The basic treatment aims at the following:

- **Nutritional status**

The impaired ability to absorb nutrients (malabsorption) is treated by adding digestive enzymes, energy-rich food, vitamins and minerals. Active supervision of nutritional status is crucial, as are different types of nutritional supplements where needed (12).

- **Lung function**

Inhalation of bronchodilators, mucolytic and anti-inflammatory drugs are often part of the treatment. Treatment to mobilise and clear the mucus from the airways helps to prevent stagnation of secreted mucus and mucus plugs, to keep all airways ventilated. The bacteria of chronically colonised airways cannot be eliminated, but the numbers can be minimised and the chronic inflammation caused by the infection held to a minimum. The bacteria growth is controlled in part by mucus mobilising treatment/physical exercise and in part with antibiotics. CF treatment incorporates a generous amount of antibiotics, given in tablet form, intravenously or via inhalation, as decided by using subjective and objective parameters (12).

The mucus mobilising portion of the treatment is very time-consuming. There are many different techniques today to loosen, transport and evacuate the viscous sputum from the airways (17). It is important to find a technique or combination of techniques that suits the particular individual. It is also important for people with CF to learn to control their cough, both to avoid urinary incontinence as well as for social purposes. In order to achieve optimal effect, the inhalation and mucus evacuation treatment for each individual should be planned strategically. The goal is for the treatment to be as gentle and effective as possible, from both a short- and long-term standpoint, in addition to encouraging the independence of the patient (13).

- **Fitness, mobility and strength**

Physical training is carried out to maintain good functional status and counteract loss of fitness, poor posture, and to reduce the risk of a stiff chest (12, 13, 17). How the training is carried out varies according to the individual's age, symptoms, personality and interests.

Treatment outcomes and prognosis

Treatment concentrated to CF centres has shown good (2, 4, 12). Breathing exercises and physical training are considered the cornerstones of the treatment, along with medical treatment and nutritional supplements (5, 12, 17–23). Treatment outcomes have improved markedly in recent decades (2, 4). In Sweden, there are currently some 535 people between the ages of 0–65 years living with CF, half of whom are over 18 years. The corresponding figure for Norway is 260 people, where similarly more than half are over the age of 18 years. Recent estimates regarding the prognosis for children with CF born in 1991 or later is that 95 per cent will live to be more than 25 years old (4). Thus, CF is no longer only a childhood disease, but also a concern for adult medicine. With adequate treatment and good support, most people with CF can live a fulfilling life of a good quality well into their adult years. Many manage to maintain a good functional capacity and lung function. Despite poor lung function, others still have a good physical capacity. A survey study from 1998 showed that 75 per cent of adult CF patients who had finished school were working, and 39 (26 women and 13 men) had children (4).

Effects of physical activity

The objective of physical training for individuals with CF is to:

- Stimulate the respiratory apparatus and intervene with resting respiratory patterns to increase the ventilation volume and/or distribution of the ventilation, and to stimulate mucociliary clearance and mobilise the mucus.
- Maintain normal working capacity. A high level of fitness reduces the risk of worsening in connection with exacerbations (deterioration), and makes recovery easier. Despite poor lung function, fitness may be good.

- Maintain good mobility, primarily of the thorax (24). Mobility of the thorax, back and shoulders must be maintained in order to perform effective mucus evacuation therapy (16). Stretching tense structures is time-consuming, painful and often unpleasant – preventing stiffness is easier and much more pleasant.
- Maintain good muscle strength. Strength training for the postural muscles helps to preserve mobility and avoid thoracic kyphosis. Good posture also helps patients to maintain the image of looking like everyone else, despite their advanced lung disease.
- Avoid osteopenia and osteoporosis.
- Improve/maintain good body awareness.
- Learn to coordinate muscle contractions to avoid urinary incontinence in connection with coughing or other physical exertion.
- Learn to distinguish between acceptable shortness of breath and abnormal dyspnoea and be able to manage these conditions.
- Increase self-confidence (25).

Strength and endurance of the peripheral skeletal muscles can be impaired in patients with lung disease (10). Both oxygen transport and energy metabolism in the muscle cells are worse than in healthy individuals for many reasons, including a change in the distribution of different types of muscle cells, a low capillary density, and biomechanical changes. Possible causes are the effects of chronic inflammation, malnutrition, hypoxia (decreased concentration of oxygen in the body's tissues), hypercapnia (increased concentration of carbon dioxide in the blood), use of corticosteroids and low level of physical activity (10, 26). Strength training that focuses on peripheral skeletal muscles has, however, shown to be effective (27, 28). Improved oxidative capacity reduces the production of carbon dioxide, which in turn reduces respiratory need, dyspnoea and muscular fatigability (27).

Physical activity affects both circulation and ventilation (28). Many individuals experience a mucus-mobilising effect in connection with activity. This effect can likely be attributed to the increased ventilation, both general and regional, increased tidal volume, increased rate of air flow and a temporary elevation of functional residual capacity (FRC) during physical exertion in individuals with obstructive pulmonary disease (28). Blocked airways are thus opened, and mucus dislodged and transported to larger airways. An increase in mucociliary clearance and positive biochemical factors such as less viscous mucus also likely play a role (29). During regular breaks in the physical activity, for example, in interval or circuit training, or after an exercise session, the loosened mucus may be evacuated. The combination must be stressed, however, in order to achieve mucus evacuation (13). This method of managing the mucus-mobilising part of treatment has been shown to be equally effective (18), and in certain cases more effective than other respiratory exercises, and is associated with the following advantages:

- It is effective from a time standpoint as well, also providing fitness training, mobility training and training of muscle strength.
- Anyone can take part as long as the objectives are maintained – not only CF patients benefit from physical exercise, which can improve compliance with the treatment.

- It can easily be changed and adapted according to the severity of the disease, the individual's interests and moods, location, weather, etc.
- It is easy to “take with you” to school, work, on holidays, etc.
- It can be done on one's own and thereby gives independence.
- It is, for the most part, stimulating and fun.

A high level of fitness impacts both survival and quality of life, helps individuals with CF to function like others, and enables them to function at work and have a family (21–23, 30, 31). Patients with a well-functioning basic therapy can, however, not expect to see further improvement in lung function from the increase in physical exercise. For these individuals, unchanged lung function values in the long term are seen as a positive outcome. However, if the current “treatment package” is insufficient, improvements in lung function can be achieved when treatment is optimised. Improved work capacity thus depends on the frequency, intensity and duration of the exercise training, similarly as in healthy individuals.

Prescription

Physical activity and training is an established and important part of the daily treatment of CF today. Physical activity/training should be carried out during antibiotic treatment despite the presence of chronic infection. Physical activity/training can serve as a part in mucus-mobilising treatment to increase ventilation and loosen secretions (13, 18) and/or as a supplement to other therapies (13, 16, 31). Treatment plans are holistic and include different types of strength training, for the core muscles as well as large and small muscle groups in both the upper and lower extremities, and exercises for the pelvic floor.

Individual adaptation and dosage

Physical activity/exercise must be adapted to the individual. Factors of importance for the type and dosage relate firstly to age, nutritional and functional status, lung condition, with special regard to the degree of obstruction, amount of secretion, and presence of hyper-responsiveness or instability of the airways. Exercise training can have an impact on the acceptable intensity level and perceived dyspnoea, while these are also dependent on daily condition and personality. Finding an exercise regime that can be tolerated in the patient's current state and is perceived as positive is essential to achieve a high level of compliance (16, 31). The need for pre-medicating with inhaled bronchodilation therapy should be evaluated, as well as the warm-up before exercise sessions, whenever treatment or the requirements and conditions for treatment change. For patients who desaturate (oxygen saturation decreases) during physical exercise, the need for providing oxygen during training should be evaluated in order to maintain a saturation of more than 90 per cent in the blood. This helps to reduce ventilatory and cardiovascular demands during training. An alternative can be to control the exercise intensity to maintain oxygen saturation over

90 per cent (32). Many patients benefit from “pursed-lip” breathing to lower the respiratory level, increase the size of each breath, and thereby improve gas exchange in the lungs. Constant optimisation of the treatment in cooperation with the patient strengthens the daily routines. Close follow-up and evaluation is required to motivate the patient to comply with the treatment.

Options for using physical activity/exercise as a part of mucus-mobilising therapy

There are four main ways of using physical activity/exercise for patients with CF to mobilise mucus with a loose delineation between them (13, 14). The factors that determine the option chosen for a particular individual are mainly age, amount of mucus in the airways, lung function, possible complications, and what subsequently proves to be the most effective (15).

The choices are:

- *Alternate dislodging, moving and evacuating of mucus with physical activity/exercise*
This option involves short intervals of physical activity/exercise to loosen the mucus and breaks between the intervals to assess the amount of secretion/expectorate the mucus. The intensity of the intervals should be tailored to the individual, with high intensity activities having proven to be effective. The breaks can include careful chest compression and manual coughing support for the very young, followed by specific coughing technique, huffing (17) and coughing.
- *Dislodge the mucus during physical/exercise and move and evacuate it afterwards*
This option involves 30 minutes of individually tailored physical activity/exercise to loosen the mucus, followed by cycles of individually tested mucus-mobilising techniques to evacuate the mucus using specific coughing technique, huffing and coughing.
- *Dislodge, move and evacuate the mucus before physical activity/exercise*
This option is for patients with large amounts of mucus who have a need for individually tested mucus-mobilising treatment before physical activity/exercise.
- *Dislodge, move and expectorate the mucus while conducting endurance training*
This option involves patients with small amounts of mucus and slightly reduced lung function being able to take short breaks to assess and expectorate possible mucus. The short breaks need not necessarily affect the intensity.

Physical activity/exercise can affect mucus-mobilisation by, for example, opening blocked airways and getting air in “behind” the mucus as well as increasing the breathing movements (respiratory pump) of the thorax. This helps to loosen and transport the mucus from the small airways into the larger ones. Physical activity/exercise combined with a specific coughing technique, huffing and coughing, is then used as a mucus-mobilising treatment option. This treatment option is often the first choice for children since it can be perceived as a natural approach when it comes to treatment.

One or more test treatments should be carried out to evaluate the individual effect of the physical activity/exercise. Evaluation of the response and effect determines whether physical activity/exercise can be used as part of the mucus-mobilising treatment for that individual. The trial treatment should provide an answer regarding the level and type of physical activity/exercise that will contribute to the treatment and, based on this, needs, possibilities/limitations and dosage can be determined (13).

Patients with CF perform inhalation and mucus-mobilising therapy 1–3 times per day according to their individual needs. Seemingly symptom-free patients are generally treated once a day. Physical activity/exercise is part of the main therapy. For patients with more pronounced symptoms, additional treatment sequences on the same day can comprise inhalation combined with other mucus-mobilising techniques.

Age-related treatment plans

Physical activity for very young children, age 0 to 1 year, comprise motor stimulation according to the child's motor development and activation of motor reflexes. Positive stimulation and activation of reflexes is done in different body positions with the aim of influencing the breathing pattern, increasing the amount of inspired air, affecting the ventilation distribution, and increasing the demands on the respiratory apparatus. The flow of exhalation can be increased with careful chest compressions to loosen and transport the mucus to the central airways. The compressions must be carried out with appropriate force during exhalation with the aim of increasing the expiratory flow and enabling the child to prolong exhalation. The compressions must also follow the breathing pattern, frequency and exhalation movement. Mobilised mucus induces a coughing reflex and the force of the cough can be enhanced manually. All of these techniques require education and training as the dosage of force must be such that it does not give the opposite effect (13, 16, 33).

From the age of 1 to about 4 years, the physical activity/training comprises chasing games and other active play. These games should also include fun "exercises" for strength and mobility. Those conducting the physical activity and exercise training with the children must learn what games are suitable. At 2–3 years of age many children can begin to lengthen exhalation and hold obstructed airways open by playing "blowing" games. The children are made aware of coughing and coughing technique. "Steaming up the mirror" can be used as a starting point for later learning the huffing technique. The chest compressions can then be replaced by specific coughing technique, huffing and coughing (14). In time, most 4- and 5-year-olds will be able to control their breathing technique, huff effectively, control the strength of their cough and achieve peak expiratory flow (PEF).

At 5–10 years old, the physical activity/training can be scheduled as various gym games or as relays and obstacle courses. The training should include fun exercises for fitness, strength and mobility. Breaks in the training are used for cycles of specific coughing technique, huffing and coughing to move and evacuate the loosened mucus. Those who began physical activity early are now well-developed from a motor standpoint and win over their peers, siblings, parents, the physiotherapist and physician, which as a rule creates self-confidence and is a good investment for future treatment.

After the age of 10 years, the physical training can be planned as circuit training with various content. A combination of low and high intensity exercises is recommended, often in the form of interval training. This training includes exercises to maintain mobility and strengthen the muscles of the thorax. Breaks in the training are used for cycles of specific coughing technique, huffing and coughing to move and evacuate the loosened mucus. This type of exercise can be alternated with running with an adult. Running gradually becomes popular with some people since it is perceived as the most time-efficient and “normal”. Running can be complemented with simple mobility and strength exercises. Specific coughing, huffing and coughing are done at the end when the exercising is finished.

Physical training as a complement to mucus-mobilising therapy

All individuals with CF can perform physical training of some type regardless of their symptoms. For those with normal or slightly reduced lung function, training schedules, including intensity, are the same as for healthy individuals. In order to achieve as wide an effect as possible, a combination of different types of training should be used. Both high and low intensity training should be used, see Table 1. An effective way to exercise oxidative capacity is to perform high intensity training in intervals of 30 seconds at maximal exertion and 30 seconds at rest, for 30 minutes, or perhaps 3 minutes of intensive exertion and 3 minutes of rest, for 3–5 repetitions (34–36).

All-round strength training and mobility training should also be included. A good starting point for many people is to find, early on, a type of physical training that is also socially stimulating and that can be done with friends, such as playing football, field or ice hockey, bandy, horseback riding, jogging, Nordic walking, swimming, spinning, etc. This activity can then be complemented with strength and mobility exercises. Many patients choose to go with their friends, spouse or partner to fitness, aerobics or other exercise classes that offer aerobic, strength and flexibility training. For others, in-home exercise programmes using simple aids such as an exercise ball, Bobath ball, trampoline, exercise bike, weights, Thera-bands, wall bars, etc., may be a better option. The programme is planned by the physiotherapist in cooperation with the patient/parents.

Table 1. Specialised training/physical activity for different stages of cystic fibrosis.

Status	Training
Normal lung function/strength/flexibility.	No restrictions. Regular aerobic fitness and strength training principles. Enjoyable sports activities. CF-specific mobility and strength training.
Normal or slightly reduced lung function – FEV ₁ * > 70% of expected value – oxygen saturation does not decrease during exertion.	As above. Close follow-up.
Moderately reduced lung function – FEV ₁ * 40–70% of expected value – risk for desaturation at night and during exertion – possibly dependent on supplemental O ₂ during sleep	High intensity interval training with long breaks, and low intensity training. Flexibility training, above all for back, chest and shoulders. Strength training, above all for postural muscles and pelvic floor. Evaluate need for supplemental O ₂ during training.
Severely reduced lung function – FEV ₁ * < 40% of expected value – high risk for desaturation at rest – evaluate 24-hour dependency on supplemental O ₂ .	High intensity interval training with shorter training intervals and longer breaks, and low intensity training. Flexibility training, above all for back, chest and shoulders. Strength training, above all for postural muscles and pelvic floor. Need for supplemental O ₂ during training.
Respiratory insufficiency while awaiting lung transplantation.	Light physical exercise. Flexibility training, above all for back, chest and shoulders. Adequate strength training, above all for postural muscles and pelvic floor. Requires supplemental O ₂ during training.

* FEV₁ = Forced Expiratory Volume in one second.

There are many examples of adults with CF who have been able to take part in sports at a high level. It has also been shown that patients with CF can run a marathon with normal biochemical, metabolic and endocrinological response (36, 37).

Special considerations

Pronounced dyspnoea

Patients must be trained to distinguish between acceptable shortness of breath and abnormal dyspnoea, and to manage their shortness of breath and to recognise dyspnoea that can lead to panic and anxiety early. Training intensity, equipment and aids should be adapted to the individual's level of function and ability.

Acute infection and fever

Temporarily stop physical exercise and strength training that give rise to an increased heart rate. Flexibility training can still be carried out.

Nutritional status and energy balance

In the case of malnutrition, physical activity/training contributes to further weight loss and muscle atrophy. The need for proper nutritional support combined with dosage of physical activity/training is assessed in cooperation with a dietitian/nutritional physiologist and physician, in order to build up muscle mass and muscle function (38).

Asthma or bronchial hyperresponsiveness

The need for pre-medication is assessed with a reversibility test, both at work and in connection with exertion. The test should be repeated when the symptom picture changes.

Diabetes

Patients with CF-related diabetes can experience a substantial drop in blood sugar during physical activity/training, which they must learn to manage in cooperation with the dietitian/nutritional physiologist and physician.

Over-exertion

All-round training is recommended to avoid over-exertion and to enable optimal function in day-to-day life.

Decrease in oxygen saturation of the blood

The need for supplementation is determined with the help of an oxygen saturation meter (SpO₂).

Oxygen saturation < 90 per cent, measured as SpO₂, should be avoided.

Training intensity and/or oxygen supplementation during training is determined in relation to SpO₂.

Joint problems and arthritis (joint inflammation)

The need for alternative forms of training and relief is assessed.

Reduced spleen or liver function

Avoid physical activity/training that can lead to trauma to the abdomen/back.

Salt and mineral deficiencies

Excess sweating can result in symptoms of extensive loss of fluids and salts (39). Ample fluids and salt tablets should be administered for long sessions of high-intensity physical training.

Haemoptysis

In the case of minor symptoms (streaks of blood in the sputum or small bloody expectorations), stop the training session. In the case of massive haemoptysis (large amounts of coughed up blood), seek emergency medical attention.

Pneumothorax

In the case of sudden, increased dyspnoea and chest pain, pneumothorax may be suspected. Stop the training session and seek medical attention immediately.

Functional tests

Patients usually visit the clinic every six weeks, and every visit includes contact with the physiotherapist. In Sweden, meeting with the physiotherapist always includes at least one treatment session, where evaluation of the prescribed inhalation therapy, mucus-expectoration treatment and compliance occurs. A spirometric examination and functional tests are also conducted, in which chest flexibility, muscle strength and work capacity are followed up. Many also have an out-patient visit to the physiotherapist in between. Once a year an extensive lung function test is carried out, at a clinical physiology laboratory, which includes both static and dynamic volumes, as well as maximal exercise test (12, 40, 41). The treatment is continually adjusted to the measured outcomes and compliance.

The testing programme in Norway includes spirometry at every visit to the clinic. The physiotherapist evaluates and follows up the different parts of the pulmonary physiotherapy, that is, mucus evacuation therapy and physical function, posture, work capacity and work tolerance. When necessary, the patient is referred to a specialist in manual therapy. Every or every second year, an extensive 3-day cross-disciplinary review is conducted, covering lung function exams and maximal exercise tests.

Interactions with drug therapy

Many patients use inhaled beta-2 agonists, which have a heart rate-increasing effect. This seldom has significance for the planning of physical training or its outcomes, but be known for the evaluations. Insulin has a blood glucose-lowering effect as does physical training. Consideration should be given to the balance between blood glucose-lowering effect and food intake, especially in intensive and/or extended training.

In connection with lung transplantation

CF is a chronic destructive disease whose progression cannot always be slowed despite intensive treatment. Lung transplantation may ultimately be the only remaining treatment option. In this case, physical training is of utmost importance so that the patient will be in optimal physical condition before this big operation. The training does not differ from that described earlier however (see Table 1). Even patients being treated with non-invasive ventilation should engage in physical exercise.

For the period immediately post-lung transplantation, the physical training is different than for other intensive care patients. Even patients who need extended assisted ventilation

should perform physical training. The goal is to successively regain normal physical function. The physical training can then be carried out according to the usual principles. Maximal oxygen uptake (> 30 ml/kg/min) is seldom attained, however, despite normal lung function. Many patients are limited by accumulation of lactic acid, experienced as tiredness in the legs, due to changes in muscle metabolism. A few individuals have taken part in a marathon race (37). The lungs are large organs and therefore require large doses of immune-suppressing drugs. Despite the lungs being extremely vulnerable to the environment, the immune defense against bacteria remain intact. Patients may, however, be more susceptible to occasional infections.

References

1. Davies PB, Drumm M, Konstan MW. Cystic fibrosis. *Am J Crit Care Med* 1996; 154:1229-56.
2. Varlotta L. Management and care of the newly diagnosed patient with cystic fibrosis. *Current Opinion Pulm Med* 1998;4:311-8.
3. Dodge JA, Brock DJH, Widdicombe JH. Cystic fibrosis. Current topics. Chichester (UK): John Wiley & Sons Ltd; 1994.
4. Lannefors L, Lindgren A. Demographic transition of the Swedish cystic fibrosis community. Results of modern care. *Resp Med* 2002;96:681-5.
5. Kerem K, Conway S, Elborn S, Heijerman H. Standards of care for patients with cystic fibrosis. A European consensus. *J Cystic Fibrosis* 2005;4:7-26.
6. Nixon GM, Armstrong DS, Carzino R, Carlin JB, Olinsky A, Robertson CF, et al. Early airway infection, inflammation, and lung function in cystic fibrosis. *Arch Dis Child* 2002;87:306-11.
7. Coates AL, Boyce P, Muller D. The role of nutritional status, airway obstruction, hypoxemia and abnormalities in serum lipid composition in limiting exercise tolerance in children with cystic fibrosis. *Acta Paed Scand* 1980;69:353-8.
8. Turck D, Michaud L. Cystic fibrosis. Nutritional consequences and management. *Baillieres Clin Gastroenterol* 1998;12:805-22.
9. Dorlöchter L, Helgheim V, Røksund OD, Rosendahl K, Fluge G. Shwachman-Kulczycki score and resting energy expenditure in cystic fibrosis. *J Cystic Fibrosis* 2003;2:148-51.
10. Skeletal muscle dysfunction in chronic obstructive pulmonary disease. A statement of the American Thoracic Society and European Respiratory Society. *Am J Respir Crit Care Med* 1999;159:S1-40.
11. Haslam RH, Borovnicar DJ, Stroud DP, Strauss BJ, Bines JE. Correlates of prepubertal bone mineral density in cystic fibrosis. *Arch Dis Child* 2001;85:166-71.
12. Cystisk fibros. Vårdprogram för Sverige av Arbetsgruppen för Cystisk Fibros 1994. [Cystic Fibrosis. Care programs for Sweden by the Working Group for Cystic Fibrosis, 1994] Swedish Medical Association's Working Group for Cystic Fibrosis.
13. Gursli S. Lungefysioterapi. En dynamisk prosess. [Lung Physiotherapy. A dynamic process.] Oslo: Unipub forlag; 2005.
14. Gursli S. Training programmes in Scandinavia. Proceedings of 25th European Cystic Fibrosis Conference, Genoa, Italy. Monuzzi Editore 2002;C622R9033:159-63.
15. Gursli S. Chest physiotherapy. Practical course as follow up model emphasizing individual adaptation in the use of physical exercise in airway clearance. *J Cystic Fibrosis* 2003;A253:S66.
16. Lannefors L, Button BM, McIlwaine M. Physiotherapy in infants and young children with cystic fibrosis. Current practice and future developments. *J R Soc Med* 2004;97: 8-25.
17. Physiotherapy in the treatment of cystic fibrosis. 3rd edn. International Physiotherapy Group for Cystic Fibrosis; 2002. www.cfww.org/pub/Physiotherapy.pdf.

18. Andreasson B, Jonson B, Kornfält R, Nordmark E, Sandström S. Long-term effects of physical exercise on working capacity and pulmonary function in cystic fibrosis. *Acta Paediatr Scand* 1987;76:70-5.
19. Desmond KJ, Schwenk WF, Thomas E, Beaudry PH, Coates AL. Immediate and long term effects of chest physiotherapy in patients with cystic fibrosis. *J Pediatr* 1983;103:538-42.
20. Blomquist M, Freyschuss U, Wiman LG, Strandvik B. Physical activity and self treatment in cystic fibrosis. *Arch Dis Child* 1986;61:362-7.
21. Webb AK, Dodd ME, Moorcroft J. Exercise and cystic fibrosis. *J R Soc Med* 1995;88:30-6.
22. Nixon PA. Role of exercise in the evaluation and management of pulmonary disease in children and youth. *Med Sci Sports Exerc* 1996;28:414-20.
23. Moorcroft AJ, Dodd ME, Webb AK. Exercise testing and prognosis in adult cystic fibrosis. *Thorax* 1997;52:291-3.
24. Vibekk P. Chest mobilization and respiratory function. In: Pryor JA, Ed. *Respiratory Care*. Edinburgh: Churchill Livingstone, Medical Division of Longman Group UK Limited; 1991. pp. 103-19.
25. Ekland E, Heian F, Hagen KS, Abbott J, Nordheim L. Exercise to improve self-esteem in children and young people. *Cochrane Database of Systematic Reviews* 2007;2.
26. de Meer K, Jeneson JA, Gulmans VA, van der Laag J, Berger R. Efficiency of oxidative work performance of skeletal muscle in patients with cystic fibrosis. *Thorax* 1995; 50:980-3.
27. Maltais F, LeBlanc P, Simard C, Jobin J, Berube C, Bruneau J, et al. Skeletal muscle adaptation to endurance training in patients with chronic obstructive pulmonary disease. *Am J Respir Crit Care Med* 1996;154:442-7.
28. Kruhlak RT, Jones RL, Brown NE. Regional air trapping before and after exercise in young adults with CF. *West J Med* 1986;145:196-9.
29. Hebestreit A, Kersting U, Basler B, Jeschke R, Hebestreit H. Exercise inhibits epithelial sodium channels in patients with cystic fibrosis. *Am J Respir Crit Care Med* 2001; 164:443-6.
30. Nixon PA, Orenstein DM, Kelsey SF, Doershuk CF. The prognostic value of exercise testing in patients with cystic fibrosis. *N Engl J Med* 1992;327:1785-8.
31. Gulmans VA, de Meer K, Brackel HJ, Faber JA, Berger R, Helders PJ. Outpatient exercise training in children with CF. Physiological effects, perceived competence and acceptability. *Pediatr Pulmonol* 1999;28:39-46.
32. Heijerman HG, Bakker W, Sterk PJ, Dijkman JH. Oxygen-assisted exercise training in adult cystic fibrosis patients with pulmonary limitation to exercise. *Int J Rehabil Res* 1991;14:101-15.
33. Gursli S. Lungefysioterapi til barn [Lung physiotherapy for children]. *Barnestafetten* 2006;47.
34. Vogiatzis I, Nanas S, Roussos C. Interval training as an alternative modality to continuous exercise in patients with COPD. *Eur Respir J* 2002;20:12-9.

35. Shah AR, Gozal D, Keens TG. Determinants of aerobic and anaerobic exercise performance in cystic fibrosis. *Am J Respir Crit Care Med* 1998;157:1145-50.
36. Stanghelle JK. Physical exercise in the management of CF patients. Thesis. Oslo: University of Oslo; 1993.
37. Stanghelle J, Koss JO, Bjørtuft ^ˆ, Geiran O. Marathon with cystic fibrosis and bilateral lung transplant. *Scand J Med Sci Sports* 2000;10:42-6.
38. Bakker W. Nutritional state and lung disease in cystic fibrosis. *Neth J Med* 1992; 41:130-6.
39. Montain SJ, Sawka MN, Wenger CB. Hyponatremia associated with exercise. Risk factors and pathogenesis. *Exerc Sport Sci Rev* 2001;29:113-7.
40. Barker M, Hebestreit A, Gruber W, Hebestreit H. Exercise testing and training in German CF centres. *Pediatr Pulmonol* 2004; 37:351-5.
41. Decramer M, Gosselink R. Physical activity in patients with cystic fibrosis. A new variable in the health-status equation unravelled? *Eur Respir J* 2006;28:678-9.

23. Dementia

Authors

Jorunn L Helbostad, PT, PhD, Department of Neuroscience, Faculty of Medicine, Norwegian University of Science and Technology (NTNU), Trondheim, Norway

Kristin Taraldsen, PT, MSc, Department of Neuroscience, Faculty of Medicine, NTNU, Trondheim, Norway

Ingvild Saltvedt, MD, Department of Geriatric Medicine, St. Olav's Hospital and Department of Neuroscience, Faculty of Medicine, NTNU, Trondheim, Norway

Summary

Dementia is a serious health concern among the elderly and as yet there are few interventions that have proven to be able to prevent development of dementia. Recent research suggests that activity in general and physical activity in particular can be beneficial in this context. The relation with the amount and type of physical activity has, however, not been established. Advice regarding activities for people with dementia should be the same as for the population as a whole. People with established dementia have the same need for physical activity as healthy people, but are often unable to carry it out on their own. Individually tailored physical activity for persons with dementia requires carefully selected environmental conditions and activities.

Introduction

Physical activity has a known disease-preventing effect and is important in preventing physical disability in people with chronic diseases. Large numbers of the population are, however, less physically active than what is likely required to maintain good physical function into old age. In the context of dementia, physical activity is important for preventing dementia and secondarily to prevent disability in people who have already developed dementia.

What is dementia and how does it express itself?

Dementia is a collective term to describe a number of pathological conditions in the brain characterised by decline in mental function, emotional problems, and difficulty managing practical tasks in daily life (1). Dementia mainly affects people over the age of 65 years (95% of cases) and is then called senile dementia. However, even younger people can develop dementia.

In Sweden, there are currently approximately 140,000 people with some form of dementia disease. The prevalence of dementia increases with age, from about 1 per cent at 65 years to over 50 per cent at 90 years of age (2). Because of the increasing number of elderly in the population, we can expect that the number of people with dementia will rise considerably in years to come.

Dementia is characterised by decreased mental functions that begin insidiously and develop over time. Common symptoms are impaired short-term memory, impaired linguistic ability, and difficulty writing, counting, recognising people and things, orienting oneself and carrying out practical tasks such as getting dressed and meal situations. Social function and personality also change, which can be seen in that the person becomes isolated and passive. Emotional problems, and loss of inhibitions and judgement occurs, as does aggression. The diagnosis of dementia requires that the symptoms are serious enough, that they impact the daily way of life and have lasted more than 6 months. In certain dementia states, an impairment of motor ability can be an early sign and, in later stages, difficulty walking, a tendency toward falling, difficulty feeding oneself and incontinence can occur. In the majority of the cases, the dementia exhibits a progressive course and leads to a reduced life expectancy.

There are a number of diseases that can lead to dementia. Alzheimer's disease is the most common dementia disease and is responsible for over 50 per cent of cases, but poor blood circulation in parts of the brain is also a common cause. The risk of developing dementia is greatest for the oldest in the population, and for people with dementia in the family, people with high blood pressure, high cholesterol, people who smoke, or in cases where diabetes is part of the disease picture. It has also been shown that people with a sedentary lifestyle have an increased risk in relation to those with active lifestyles, socially and mentally.

Investigation of dementia should comprise a general medical examination by a physician, including blood samples and a CT or MRI scan of the brain. This is complemented by establishing the patient's mental status and overall level of function through clinical examination and a battery of tests, in addition to interviews with the next of kin or other care providers.

The treatment of dementia involves different measures that should preferably be carried out parallel to one another and can be aimed at both the patient and next of kin. Many elderly persons benefit greatly from regular follow-up through home care services, possibly in combination with day centres. The next of kin of people with dementia often carry a great burden and need both education and guidance, as well as the opportunity to take part in dementia support groups. Getting relief at day centres or short-term stays at

nursing homes can be needed and later the disease requires constant supervision of the patient.

The goal should be to give people with dementia optimal treatment for any other existing diseases since we often observe that mental function deteriorates if the patient is also physically ill, for example, with pain, urinary tract infection or constipation. Some people with dementia become depressed and acquire behaviour problems that can lead to a need for medication. Many are sensitive to drug side-effects and a review of total drug intake is therefore important. In recent years, drugs have been developed that delay the disease's progression and in the odd case even improve the condition. These drugs are used primarily for patients with Alzheimer's disease, but can also be effective in certain other dementia conditions.

The importance of physical activity in preventing development of dementia

Several studies conducted in recent years suggest that regular physical activity has a protective effect against the risk of developing senile dementia (3–5). Findings have shown that people who are generally active run a smaller risk of developing dementia than those who take part in fewer activities. Whether the activity is energy-intensive or not plays a smaller part in this context (6). Even in the oldest subjects (over 85 years), there are indications that regular physical activity protects against the development of dementia (7).

Physical activity is often performed together with social and mental activities, and being involved in a number of different activities likely stimulate one's ability to organise things and remember, which can be one of the reasons that physical activity appears to have a protective effect. Physical activity can also reflect a sound lifestyle, something that in itself protects against exposure to factors that can have a negative impact on cognitive function.

Even genetic differences play a role in whether physical activity protects against dementia. It has been found, for example, that physical activity protects against the development of dementia in non-carriers of a particular gene, the apolipo-protein E (APOE genotype), while the relationship is small in those who carry the gene (6).

The importance of physical activity in people with dementia

Older, acutely ill persons may be at risk for developing dementia due to physical and mental effects of the condition (8). Preventing inactivity resulting from illness can therefore be a factor in preventing a decrease in function in general, and mental function in particular. There is insufficient evidence to be able to say whether or not physical activity programs are beneficial for mental function in people with dementia. However, older people with dementia have the same need for physical activity as other older persons (23).

The importance of the activity in physical, social and emotional function

There are studies that have shown that structured exercise training leads to an increase in physical fitness and function in daily life in people with dementia (9). For older people still living at home, an exercise programme along with training for the next of kin regarding how to handle the person's behaviour has been reported to improve walking capacity and self-reported health at the same time as reducing depression (10). Other studies have not shown as obvious effects from exercise (11). The difference in conclusions can be due to the fact that different studies have investigated different criteria and measured effects in different ways. A general feature of the studies conducted is that only persons with a mild or moderate degree of dementia were included.

Most people who stay at a nursing home/care facility have mild cognitive decline or dementia. Studies from USA and Europe show that structured physical exercise for older people in nursing homes (70+) with multiple diagnoses yields positive effects on muscle strength and mobility. There is documentation that supports to a reasonable extent the effect on mobility, and contradictory results when it comes to walking function, activities of daily life (ADL), balance and endurance. Although low mental function was not a criterion for inclusion in the study, there is reason to believe that many of the participants had mild cognitive decline and that the findings can only be transferred to people with milder dementia (12).

It has also been reported that tailored physical activity affects the intellectual functions in older people with dementia, especially aspects such as attention, memory, the ability to communicate and to perform practical tasks, as well as overall mental function (13, 14).

Physical activity combined with techniques to reduce negative behaviours have been shown to improve physical health and reduce depression in people with Alzheimer's disease (15). The findings suggest that involvement is an important success factor when it comes to achieving gains in physical function.

Together these studies indicate that older persons with different degrees of declining mental health, both those still living at home and those in some kind of institution, can benefit from exercise training. The exercise has several physical and psychological effects, and helps to preserve health, mental functions, the ability to communicate, as well as function in daily life as the most important goal.

Preventing falls

Due to impaired motor and mental function, people with dementia are at increased risk of falling, and those who do fall run the risk of further injuries. While physical exercise that focuses on muscle strength and balance has been shown to be effective in preventing falls in general in elderly still living in their homes, the same benefits are not seen in people with dementia (11, 16). This can indicate that factors other than balance and muscle strength, and here probably factors that can be linked to cognitive decline, are important risk factors for people with dementia. Yet, there are few studies to confirm this, meaning that we must be careful when interpreting the conclusions.

Recommendations

Physical activity to prevent dementia

Studies have been unable to show the exact amount of activity that has a beneficial preventive effect on the development of dementia, other than that repetition is more important than high intensity (17). Neither has the type of activity that is beneficial been established. Recommendations regarding physical activity should therefore follow general guidelines that generally apply for achieving positive health effects in the population.

Physical activity to prevent decline in function in people with dementia

People can live up to 20 years with progressive dementia. The goal of physical activity should be to prevent a decline in function and help to maintain function in daily life. Taking part in activities can also be an angle of approach to communication and general stimulation. The need for physical activity in patients with dementia is often the same as that in other patients, but adaptation and design of the activity must be done differently. Being physically active in itself creates a sense of well-being, but many people with dementia need help getting started, help to adjust, and supervised follow-up in order to maintain a good activity level.

Some people may have a reduced ability to take initiative and therefore easily become inactive. These people may feel that their mobility is rapidly worsening, not only as a result of the dementia condition, but also as a result of the inactivity, and thus find themselves in danger of losing certain basic functions, for example, the ability to walk. Measures to prevent this type of decline in function are therefore important.

Another group of patients may be restless and prone to wandering. Others are described as demonstrating negative behaviour, which can be linked not only to the dementia condition, but more to the person's lack of ability to understand his or her surroundings (1). It is therefore possible that activities tailored for and better coordinated to the immediate environment may serve these people well.

Amount and type of physical activity

Physical activity in people with dementia can yield both physiological, and psychological and emotional effects. There is no basis for assuming that the dose and intensity of exercise components, such as muscle strength, flexibility and balance, should be any different than in people without dementia. When it comes to the impact of activities on cognition and emotion, there is little documented knowledge regarding the amount and type of activity. Knowledge from the field of motor learning (18) indicates that the total amount performed and that the task is perceived as meaningful and motivating are the most important factors for learning.

When it comes to the type of activity, it seems that different physical activities can have a positive impact. Many studies are based on a general mobility programs, with a focus on walking ability. Everything from chair exercises, aerobics, strength training using weights,

exercise programs on the exercise bike, to training of other skill-based functions have been reported (9). In order to prevent loss of function in patients with multi-functional decline, exercise that affects several components of fitness is recommended, for example, strength, endurance, balance and mobility, as well as specific training of skills required in daily life (19).

For people with complex conditions, it appears that physical training combined with techniques to influence their condition is especially beneficial (10).

Adapted physical activity

Dementia affects a person's level of function and leads to a gradual reduction of intellectual ability and affects one's memory and the ability to perform everyday activities. The memory affects performance of activities such that the person may forget what he or she is doing. Problems with orientation, impaired judgement, and failing motivation and emotional control can make activities even more difficult. The physical activity must therefore be adapted to the particular individual, according to mental and physical status (20).

Communication can be a big problem in this context (21). Factors that make communication more difficult can include a patient's difficulties in localising the source of sounds, understanding what is said, impaired memory and slow response, as well as difficulty expressing oneself clearly. Many older people have impaired hearing, which is especially important to check in patients with dementia as many of them refuse to use a hearing aid. In such cases, a speech amplifier may help to improve verbal communication. Repeating what is said is also important, as is using words that are familiar to the patient. Furthermore, it is easier to understand positive instructions than negative ones. For example, it is better to say "stand there" instead of "don't sit down". Goal-related tasks are simpler than using pure movement instructions, which can be illustrated by the following: If you want a patient who is sitting down to get up, it can be easier to ask the person to fetch a tea cup on the table a little ways from the chair.

Some key points for good verbal communication

- Give the person time to respond.
- Express yourself clearly and simply – one instruction or piece of information at a time.
- Use words and expressions that the patient is very familiar with.
- Repeat things often, and paraphrase if the patient does not understand.
- Avoid giving instruction for movements, have the patient solve the tasks instead.

Non-verbal communication is an important complement to speech but must occur in a way that does not confuse the patient. Examples of methods that can be used are gestures, physical contact, visual reinforcement, audio amplification, or demonstrating the activity.

Certain activities may frighten some people with dementia, for example, due to spatial perception problems. Patients may feel unsafe when getting up from a chair or bed, or walking down steps. In situations such as these, it is important to "block" perceived threats, for example, by placing the back of the chair in front of the person getting up or walking backwards in front of the person down the steps.

For patients with severe dementia, it can be difficult to function in a “moving” environment (people who are moving about, varying light conditions, different sounds, floors with different colours and sharp contrasts) or if the usual environment suddenly changes. During training, it is therefore important to reduce all interfering information in the environment or consciously select which challenges are needed to make the training realistic.

Using activities that the person is already familiar with facilitates involvement better than new activities, and it is therefore necessary to identify and adapt such activities.

Applicable tests

In people suspected of cognitive impairments, a screening test such as the Mini Mental Status (MMS) (22) can be used to investigate which aspects of mental function are impaired. Physical function can be mapped with tests specifically designed for the elderly, to evaluate functions relevant to the requirements of everyday life with respect to muscle strength, balance, mobility and endurance. A well-tailored testing situation and simple instructions are, however, decisive in obtaining correct test results.

References

1. Engedal K. Demens og demenssykdommer [Dementia and dementia diseases]. In: Engedal K, Wyller TB, Eds. Aldring og hjernesykdommer [Aging and Brain Diseases]. Oslo: AKRIBE; 2003, pp. 125-48.
2. SBU (Swedish Council on Technology Assessment in Health Care) Demenssjukdomar [Dementia diseases]. Report no. 2006:172. Stockholm: SBU; 2006.
3. Larson EB, Wang L, Bowen JD, McCormick WC, Teri L, Crane P, et al. Exercise is associated with reduced risk for incident dementia among persons 65 years of age and older. *Ann Intern Med* 2006;144:73-81.
4. Laurin D, Verreault R, Lindsay J, MacPherson K, Rockwood K. Physical activity and risk of cognitive impairment and dementia in elderly persons. *Arch Neurol* 2001; 58:498-504.
5. Rovio S, Kareholt I, Helkala EL, Viitanen M, Winblad B, Tuomilehto J, et al. Leisure-time physical activity at midlife and the risk of dementia and Alzheimer's disease. *Lancet Neurol* 2005;4:705-11.
6. Podewils LJ, Guallar E, Kuller LH, Fried LP, Lopez OL, Carlson M, et al. Physical activity, APOE genotype, and dementia risk. Findings from the Cardiovascular Health Cognition Study. *Am J Epidemiol* 2005;161:639-51.
7. Sumic A, Michael YL, Carlson NE, Howieson DB, Kaye JA. Physical activity and the risk of dementia in oldest old. *J Aging Health* 2007;19:242-59.
8. Blocker WP, Jr. Maintaining functional independence by mobilizing the aged. *Geriatrics* 1992;47:42, 48-50, 53.
9. Heyn P, Abreu BC, Ottenbacher KJ. The effects of exercise training on elderly persons with cognitive impairment and dementia. A meta-analysis. *Arch Phys Med Rehabil* 2004;85:1694-704.
10. Teri L, Gibbons LE, McCurry SM, Logsdon RG, Buchner DM, Barlow WE, et al. Exercise plus behavioral management in patients with Alzheimer's disease. A randomized controlled trial. *JAMA* 2003;290:2015-22.
11. Hauer K, Becker C, Lindemann U, Beyer N. Effectiveness of physical training on motor performance and fall prevention in cognitively impaired older persons. A systematic review. *Am J Phys Med Rehabil* 2006;85:847-57.
12. Rydwick E, Frandin K, Akner G. Effects of physical training on physical performance in institutionalised elderly patients (70+) with multiple diagnoses. *Age Ageing* 2004;33:13-23.
13. Eggermont L, Swaab D, Luiten P, Scherder E. Exercise, cognition and Alzheimer's disease. More is not necessarily better. *Neurosci Biobehav Rev* 2006;30:562-75.
14. Heyn P. The effect of a multisensory exercise program on engagement, behavior, and selected physiological indexes in persons with dementia. *Am J Alzheimers Dis Other Demen* 2003;18:247-51.
15. Woodhead EL, Zarit SH, Braungart ER, Rovine MR, Femia EE. Behavioral and psychological symptoms of dementia. The effects of physical activity at adult day service centers. *Am J Alzheimers Dis Other Demen* 2005;20:171-9.

16. Shaw FE, Bond J, Richardson DA, Dawson P, Steen IN, McKeith IG, et al. Multifactorial intervention after a fall in older people with cognitive impairment and dementia presenting to the accident and emergency department. Randomised controlled trial. *BMJ* 2003;326:73.
17. Podewils LJ, Guallar E, Beauchamp N, Lyketsos CG, Kuller LH, Scheltens P. Physical activity and white matter lesion progression. Assessment using MRI. *Neurology* 2007; 68:1223-6.
18. Schmidt RA, Lee TD. Motor control and learning. A behavioral emphasis. 4. edn. Champaign: Human Kinetics; 2005.
19. Helbostad JL. Physical training for nursing home residents – has it any effect? *Tidsskr Nor Laegeforen* 2005;125:1195-7.
20. Helbostad JL. Fysioterapi i geriatrien [Physiotherapy in geriatric medicine]. In: Helbostad JL, Granbo R, Tøsterås H, Eds. Aldring og bevegelse. Fysioterapi for eldre. [Ageing and Movement. Physiotherapy for the elderly.] Oslo: Gyldendal Akademiske Forlag; 2007, pp. 366-94.
21. Oddy R. Promoting mobility for people with dementia. 2. edn. London: Age Concerns England; 2003.
22. Braekhus A, Laake K, Engedal K. The Mini-Mental State Examination. Identifying the most efficient variables for detecting cognitive impairment in the elderly. *J Am Geriatr Soc* 1992;40:1139-45.
23. Forbes D, Forbes S, Morgan DG, Markle-Reid M, Wood J, Culum I. Physical activity programs for persons with dementia. *Cochrane Database Syst Rev* 2008;(3):CD006489.

24. Depression

Authors

Bengt Kjellman, MD, PhD, Associate Professor, Stockholm, Sweden

Egil W. Martinsen, MD, PhD, Professor, Aker University Hospital, University of Oslo, Oslo, Norway

Jill Taube, MD, Center for Family and Community Medicine, Karolinska Institutet, Stockholm, Sweden

Eva Andersson, MD, PhD, Swedish School of Sport and Health Sciences, Stockholm, Sweden

Summary

Physical activity has a positive effect in depression with respect to both preventing depressive episodes, and acute and long-term treatment of episodes that do occur. The physical training is beneficial when conducted parallel to customary treatment with drugs and therapy. Although many studies have methodological problems and there is still a shortage of long-term studies, there is clear scientific support for using physical training in the acute treatment of mild and moderate depression and as a means to reduce the risk of relapse. A preventive effect has been shown in epidemiological studies and long-term studies followed up to 10 years. Other health effects of physical activity are also of importance, as depression often covariates with physical diseases. Physical activity holds great benefit both for the individual and for society.

Definition

Major depression is common and becoming more prevalent. The lifetime prevalence in different countries and different studies ranges between 6 and 20 per cent. One Norwegian study, published by Kringlen and colleagues in 2001, found a lifetime prevalence of 17.8 per cent (1). In 2000, WHO ranked depressive disorders as the fourth largest health problem in the world. A significant difference exists between the genders, with depression in women ranking fourth in total disease burden, and only seventh in men (2).

Diagnostic criteria

From a diagnostic standpoint, depression belongs to the group of mental illnesses called mood disorders and is divided into two main groups: unipolar affective disorders with only depressive episodes, and bipolar affective disorders with both depressive episodes and hypomanic and/or manic episodes. In comparison to unipolar disease, bipolar disease is rare and will not be discussed here. Research is in general lacking when it comes to physical activity and bipolar disease. Dysthymia is a milder form of depression but the condition is often chronic, lasting two years or more, and is not addressed here either.

Depression demonstrates a significant comorbidity with other psychiatric disorders, above all anxiety disorders, as well as physical diseases where cardiovascular disease is prominent.

According to the Diagnostic and Statistical Manual of Mental Disorders (DSM) IV, which is used world-wide, the following criteria apply to *major depression*:

A: Five or more of the following symptoms have been present during the same 2-week period and represent a change in the person's condition. At least one of the symptoms, 1) depressed mood or 2) loss of interest, must be present.

1. Depressed mood most of the day, nearly every day, as indicated by either subjective report (e.g. feel sad or empty) or observation made by others (e.g. appears tearful).
2. Markedly diminished interest or pleasure in all, or almost all, activities most of the day, nearly every day, as indicated by either subjective account or observation made by others.
3. Significant weight loss (when not dieting) or weight gain (e.g. a change of more than 5% of body weight in a month), or decrease or increase in appetite nearly every day.
4. Sleeping disturbance (insomnia or hypersomnia nearly every night).
5. Psychomotor agitation or retardation nearly every day (observable by others, not merely subjective feelings of restlessness or being slowed down).
6. Feeling of fatigue or loss of energy nearly every day.
7. Feelings of worthlessness or excessive or inappropriate guilt (which may be delusional) nearly every day (not merely self-reproach or guilt about being sick).
8. Diminished ability to think or concentrate, or indecisiveness, nearly every day (as indicated either by subjective account or as observed by others).
9. Recurrent thoughts of death (not just fear of dying), recurrent suicidal ideation without a specific plan, or have a specific plan for committing suicide.

The symptoms are not better explained by other psychiatric conditions and must cause clinically significant suffering or impaired function at work, in social situations or in other important respects.

Diagnostic procedure

DSM IV and ICD 10, with similar criteria are used in diagnostics in Norway and Sweden. The diagnosis is based on these criteria, which are descriptive. The rating scales are used mainly to measure the severity of the depression. There are no biomarkers for the disease.

Cause and risk factors

There is no simple causal relationship and genetic predisposition is not particularly strong. One frequently used model is that of stress and susceptibility. For the individual, negative stress causes depression, but individual susceptibility varies. Risk factors that can produce increased vulnerability include separation in childhood, psychological trauma, abuse and a number of somatic factors. Losses of varying nature are common as a precipitating factor. How lifestyle, for example, physical inactivity, affects depression is discussed in studies that are presented below.

Pathophysiology

There is no single pathophysiology. In some cases, a pathological increase in hypothalamic-pituitary-adrenocortical axis activity can be found as a sign of stress in ongoing depression. A normalising usually occurs with recovery. Because antidepressant drugs improve the function of neurotransmitters such as serotonin, noradrenaline and dopamine, one theory is that depression is caused by disturbances in these systems. New research has shown cell death in certain parts of the brain, especially the hippocampus, in depression.

What does the disease lead to?

Depression produces considerable suffering for the person affected and others close to him or her. It also leads to reduced levels of function at work and socially. Depression is a dominating diagnosis when it comes to sick leave and sick pay for mental illness, and is the major cause of completed suicide.

Treatment principles

Pharmacological treatment with modern antidepressant drugs and several forms of psychotherapy, including cognitive therapy and interpersonal therapy, have a documented effect. To prevent repeat episodes, antidepressant drugs and treatment with lithium are used. Improvement of the patient's condition is not always complete and a combination of different drugs as well as the use of electroconvulsive therapy (ECT) may be needed in severe cases. The use of light therapy for winter depression is widespread in Sweden and Norway but there are different opinions on the scientific basis for this.

As with other disease conditions, non-specific treatment options such as psychosocial support and rehabilitation measures are often needed.

Effects of physical activity

Acute effects

As early as 1905, Franz and Hamilton published the first report describing how moderate physical activity led to significant improvement in the cognitive, physical and emotional conditions of two patients with severe depression (3). In 1984, McCann and Holmes (4) showed that running had a significantly better effect than relaxation and no treatment at all in a group of students with mild to moderate depression. A few years later, Martinsen and colleagues at the Modum Bad centre in Norway published results in which they found that depressed patients admitted to hospital had significantly better effects from physical training three times a week for nine weeks, than from occupational therapy for the same amount of time (5, 6).

In recent years, a number of important studies have verified earlier findings of a positive acute effect from physical training in depression. In 1999, Blumenthal and colleagues published a study in which 156 patients aged 50–77 years were randomly divided into three treatment groups. The trial lasted for four months. Group 1 was treated with sertraline (Zoloft), 50–200 mg per day. The second group received exercise training in the form of 30 minutes of walking and jogging, 3 times a week. The third group received both sertraline and exercise training. They found no significant difference in treatment effect between the three groups, and all showed a good effect from the treatment (7).

In 2005, Dunn and colleagues published a long-awaited dose-response study (8). Eight patients were assigned to one of four groups. Group 1 received exercise treatment with an energy expenditure of 7 kcal/kg body weight/week, 3 times a week; Group 2 – an expenditure of 7 kcal/kg body weight/week, 5 times a week; Group 3 – 17.5 kcal/kg body weight/week, 3 times a week; Group 4 – 17.5 kcal/kg body weight/week, 5 times a week; and Group 5 – stretching, 3 times a week, as a placebo. Groups 3 and 4 received the exercise intensity commonly given in public health recommendations, while groups 1 and 2 received low intensity exercise treatment. The treatment lasted for 12 weeks.

They found that the depression score (according to the Hamilton Depression Rating Scale, HDRS) fell by 47 per cent in groups 3 and 4, but by only 30 per cent in groups 1 and 2, and by 29 per cent in Group 5. The conclusion was that the exercise intensity usually recommended had an obvious therapeutic effect in mild to moderate depression, while lower intensity was equivalent in effect to the placebo (8). In 2006, Trivedi and colleagues published an article in which 17 patients who had not become well with antidepressant drugs received exercise training for 12 weeks, continuing with the same medication during that time. The patients included in the study showed a strong positive effect with greatly reduced depression scores (9). This pilot study shows the possibility of using exercise to increase the effect of antidepressant drugs in depression. In a randomised controlled study the same year, Knubben and colleagues showed that relatively strenuous daily jogging for 10 days, up to 80 per cent of maximal heart rate, yielded significantly better therapeutic effect than placebo (stretching and relaxation) in a group of admitted patients with moderate to deep depression (10).

Long-term effects

When following up the above-mentioned patients from Blumenthal and colleagues' study, Babyak and colleagues found that, after an additional 6 months, those who received only exercise training had a significantly lower risk of further depressive episodes than those who received only sertraline. A significantly higher percentage of those who received the exercise training were also in full remission at 10 months after the study start (11).

There are also a number of studies with longer follow-up periods. Three recently published prospective studies show a link between physical activity and depression. Harris and colleagues followed 424 depressed patients for a period of 10 years. They found that more physical activity was associated with less depression and counteracted the effect of physical illnesses and negative stress factors on the depressive symptoms (12).

Meta-analyses

In a meta-analysis from 1998, Craft and Landers looked at 37 articles and found that exercise was better than no treatment at all for depression. No difference was found between different types of exercising. The effect was, however, better if the treatment lasted more than 9 weeks, compared to less than 8 weeks. According to the authors, the best effect was found for moderate to severe depression (13). In a meta-analysis published in 2001, Lawlor and Hopker found 14 studies whose methodologies merited inclusion, though, according to the authors, all of which also did have methodological weaknesses. However, they found a total effect of 1.1, which is satisfactory. The conclusion was nevertheless that it was not possible to say with certainty whether exercise had an antidepressant effect (14). In an article from the same year, Dunn and colleagues conducted a review of 18 studies. Eight of these found a 50 per cent reduction in depressive symptoms during the acute phase. In seven studies with follow-up periods ranging from 3 to 21 months, they were able to demonstrate that the effect was retained with continued exercise. These authors also draw attention to methodological problems, and call for a controlled dose-response study, which they later conduct themselves (see above) (15).

Meta-analysis makes it possible to draw conclusions from a larger group of patients than provided by individual studies. A disadvantage is that, if demands with respect to the quality of the included studies are high, a lot of data from somewhat smaller, well-conducted studies gets left out.

Epidemiological studies

As early as 1988, using data from Canada and the US, Stephens found that a high level of physical activity correlated to a low level of anxiety and depressive symptoms (16). In a German population group, Weyerer found that those who reported no physical activity ran three times the risk of developing moderate to severe depression as those who reported being physically active (17). Three Finnish studies also found similar results (18–20). A well-known study is that of Paffenbarger and colleagues from 1994, in which they followed Harvard students for a period of 23–27 years, retrospectively, and compared the amount

of physical activity and exercise to depressive illness. The conclusion was that those who engaged in regular physical exercise had a lower risk of developing depression. The effect was clearly dose-dependent (21). There are also studies that show no link between physical activity and reduced depression. One such study is a study published by Cooper-Patrick and colleagues with a 15-year follow-up (22).

Wiles and colleagues show in a 2007 report from the Caerphilly Study in Wales that there is a relation between a high level of physical activity in leisure time and at work, and reduced incidence of mental disorders (mainly depression and anxiety), at a 5-year follow-up but not after 10 years (23). An interesting study from Japan by Yoshiuchi and colleagues measured the physical activity of 184 individuals, aged 65–85 years, with an accelerometer and pedometer, for one year. They found a significant negative relation between physical activity and depressive symptoms (24).

There are obvious weaknesses in these types of studies, both regarding the validity of the data and the causal relationships, especially in cross-section studies. Is it the physical inactivity that leads to depressive symptoms or is it the depression that leads to inactivity. There may also be a third factor that explains the relationship. In longitudinal studies, a population is followed over time. In this case, one can start with healthy individuals, identify those who become ill, and observe whether there is a link between the level of physical activity and disease tendency. In these studies, the causal relationships are clearer. A weakness of such studies, however, is the selection phenomenon. It is possible that people who are physically active have more resources and that they would fare best regardless of whether or not they were physically active. In order to control this phenomenon, randomised controlled intervention studies are required.

Plausible hypotheses of functional mechanisms

Physical activity involves a change in behaviour, a behaviour modification. In depression, a person's behaviour is often characterised by passiveness, withdrawal and isolation. Changing behaviour can affect thoughts and emotions, and in doing so contribute to reversing depression (25). Cognitive behaviour therapy has also been shown to produce a positive effect in the treatment of depression. Physical exercise has been shown to encourage positive thoughts and emotions, increased confidence in coping, and increased self-confidence and capacity for self-control. Salmon (26) discusses another possible psychological mechanism. His hypothesis is that one becomes more resistant to stress through physical exercise. This could be linked to reduced activity in the hypothalamus-pituitary-adrenocortical axis, whose function is often pathologically increased in depression.

Another possibility is that it is the improved physical functional capacity gained through exercise that is the mechanism of action. However, there does not appear to be clear connection between the improvement in physical capacity and reduction of depression in depressed patients (27). Physical activity improves synthesis and metabolism of the neurotransmitters noradrenaline, serotonin and dopamine in test animals (28). Definite proof that this is the case in humans is not available yet, however, though it is a plausible hypothesis of an important underlying mechanism. A popular hypothesis is that the effect

of exercise is due to an increased concentration of endorphins, that is, the body's own morphine. Both rat and human trials support this hypothesis (29, 30), but more research is needed on the effect of endorphins in the brain of patients being treated with physical exercise.

An exciting possibility is also that exercise dramatically helps in cell regeneration in some parts of the brain, especially the hippocampus, which is important for learning and memory. Researchers have found a lower hippocampal volume in depressed individuals (31), and that treatment with antidepressant drugs yields regeneration of cells there (32). A research group at Karolinska Institutet have recently found that the antidepressive effect seen in depressed rats that were allowed to run is linked to hippocampal cell proliferation, and that the cell proliferation when they run is as high as when being treated with antidepressant drugs (33).

Indications

1. Physical activity and exercise can be used to reduce the risk of developing depression.
2. Exercise training can be used as a treatment for clinical depressive disorders. The training is conducted parallel to other antidepressant treatment such as medication and/or psychotherapy.
3. Exercise training can be used to reduce the risk of further depressive episodes both during the first year and later on.

Prescription

Type of training	Intensity	Frequency (times/week)	Duration (min.)
Aerobic training Minimum of 9 weeks	Moderate to high (13–15; Borg RPE scale)	2–3	30–45
Strength training Minimum of 9 weeks	8–10 exercises 1–3 sets of 8–12 reps	2–3	30–60

* RM = Repetition Maximum. 1 RM corresponds to the maximum weight that can be lifted through the entire exercise movement one time.

Functional tests/Need for health check-ups

A pilot project on exercising against depression (in Swedish “Motion mot depression”) has been conducted for a total of four semesters in cooperation with the Psychiatric Care Team from northwest and central Stockholm and the Swedish School of Sport and Health Sciences (Gymnastik och idrottshögskolan, GIH).

The following tests and instruments were used:

- Depression-rating using MADRS (34) or PHQ-9 questionnaire (35).
- Fitness/endurance tests using Åstrand's exercise bike test (36) or the 6-minute walk test (37).
- Abdominal and hip flexor muscle test (38) and back-lift endurance test (39).
- Leg strength test and shoulder musculature test.
- Health survey from GIH on perceived health and lifestyle habits such as physical activity, diet, smoking, alcohol, sleep, and time for recreation.

The severity of the depression should be rated before and after treatment using an appropriate rating scale. If possible, the and endurance tests should be performed before and after completed treatment.

Interactions with drug therapy

One experience from treatment with older antidepressant drugs, so-called tricyclic antidepressants, is that they can make exercising more difficult due to their side-effects, above all in the form of increased heart rate, dry mouth and sweating. Modern antidepressant drugs have a lot fewer side-effects and are judged to affect exercise to a very small degree.

Contraindications

Underweight patients with diagnosed eating disorders should not be prescribed exercise for depression.

Risks

As with other indications, there are risks with certain somatic disorders.

References

1. Kringlen E, Torgersen S, Cramer V. A Norwegian psychiatric epidemiological study. *American Journal of Psychiatry* 2001;158:1091-8.
2. Ustun TB, Ayuso-Mateos JL, Chatterji S, Mathers C, Murray CJL. Global burden of depressive disorders in the year 2000. *British Journal of Psychiatry* 2004;184:386-92.
3. Franz SL, Hamilton GV. Effects of exercise upon the retardation in condition of depression. *American Journal of Insanity* 1905;62:239-56.
4. McCann IL, Holmes DS. Influence of aerobic exercise on depression. *Journal of Personality and Social Psychology* 1984;46:1142-7.
5. Martinsen EW, Medhus A, Sandvik L. Effects of aerobic exercise on depression. A controlled study. *BMJ* 1985;291:109.
6. Martinsen EW, Medhus A. Adherence to exercise and patient evaluation of physical exercise in comprehensive treatment programme for depression. *Nordisk Psychiatrist Tidsskrift* 1989;43:411-5.
7. Blumenthal JA, Babyak MA, Moore KA, Craighead WE, Herman S, Khathri P, et al. Effects of exercise training on older patients with major depression. *Archives of Internal Medicine* 1999;159:2349-56.
8. Dunn AL, Trivedi MH, Kampert JB, Clark CG, Chambliss HO. Exercise treatment for depression. Efficacy and dose response. *American Journal of Preventive Medicine* 2005;28:1-8.
9. Trivedi MH, Greer TL, Granneman BD, Chambliss HO, Alexander J. Exercise as an augmentation strategy for treatment of major depression. *Journal of Psychiatric Practice* 2006;12:205-13.
10. Knubben K, Reischies FM, Adli M, Schlattman P, Bauer M, Dimeo F. A randomised controlled study on the effects of a short-term endurance training programme in patients with major depression. *British Journal of Sports Medicine* 2007;41:29-33.
11. Babyak M, Blumenthal JA, Herman S, Khatri P, Doraiswamy M, Moore K, et al. Exercise treatment for major depression. Maintenance of therapeutic benefit at 10 months. *Psychosomatic Medicine* 2000;62:633-8.
12. Harris AHS, Cronkite R, Moos R. Physical activity, exercise coping and depression in a 10-year cohort study of depressed patients. *Journal of Affective Disorders* 2006;93:79-85.
13. Craft LL, Landers DM. The effect of exercise on clinical depression and depression resulting from mental illness. A meta-analysis. *Journal of Sport & Exercise Psychology* 1998;20:339-57.
14. Lawlor DA, Hopker SW. The effectiveness of exercise as an intervention in the management of depression. Systematic review and meta-regression analysis of randomised controlled trials. *BMJ* 2001;322:1-8.
15. Dunn AL, Trivedi MH, O'Neal HA. Physical activity dose response effects on outcomes of depression and anxiety. *Medicine and Science in Sports and Exercise* 2001;33:587-97.
16. Stephens T. Physical activity and mental health in the United States and Canada. Evidence from four population surveys. *Preventive Medicine* 1988;17:35-47.

17. Weyerer S. Physical inactivity and depression in the community. Evidence from the Upper Bavarian Field Study. *International Journal of Sports Medicine* 1992;13:492-6.
18. Ruuskanen JM, Ruoppila I. Physical activity and psychological well-being among people aged 65 to 84 years. *Age Ageing* 1995;24:192-6.
19. Lampinen P, Heikkinen R-L, Ruoppila I. Changes of intensity of physical exercise as predictors of depressive symptoms among older adults. An eight-year follow-up. *Preventive Medicine* 2000;30:371-80.
20. Hassmén P, Koivula N, Uutela A. Physical exercise and psychological well-being. A population study in Finland. *Preventive Medicine* 2000;30:17-25.
21. Paffenbarger RS Jr, Lee I-M, Leung R. Physical activity and personal characteristics associated with depression and suicide in American college men. *Acta Psychiatrica Scand (Suppl)* 1994;377:16-22.
22. Cooper-Patrick L, Ford DE, Mead L. Exercise and depression in midlife. A prospective study. *American Journal of Public Health* 1997;87:670-3.
23. Wiles JN, Haase MA, Gallacher J, Lawlor DA, Lewis G. Physical activity and common mental disorders. Results from the Caerphilly Study. *American Journal of Epidemiology* 2007;165:946-54.
24. Yoshiuchi K, Nakahara R, Kumano H, Kuboki T, Togo F, Watanabe E, et al. Yearlong physical activity and depressive symptoms in older Japanese adults. Cross-sectional data from the Nakojana study. *American Journal of Geriatrics and Psychiatry* 2006;14:621-4.
25. Beck JS. *Cognitive Therapy. Basics and beyond*. New York: Guilford Press; 1995.
26. Salmon P. Effects of physical exercise on anxiety, depression and sensitivity of stress. A unifying theory. *Clinical Psychology Review* 2001;21:35-61.
27. Martinsen EW. Physical activity and depression. Clinical experience. *Acta Psychiatrica Scand (Suppl)* 1994;377:23-7.
28. Meeusen R. Exercise and the brain. Insight in new therapeutic modalities. *Annual Transplant* 2005;10:49-51.
29. Pert CB, Bowie DL. Behavioral manipulation of rats causes alterations in opiate receptor occupancy. In: Usdin E, Bunney WE, Kline NS, Ed. *Endorphins in Mental Health*. New York: Oxford University Press; 1979. pp. 93-105.
30. Farrel PA, Gates WK, Maksud MG, Morgan WP. Increase in plasma beta endorphin/betalipotropin immunoreactivity after treadmill running in humans. *Journal of Applied Physiology* 1982;52:1245-9.
31. Campbell S, Marriott M, Nahmias C, MacQueen GM. Lower hippocampal volume in patients suffering from depression. A meta-analysis. *American Journal of Psychiatry* 2004;161:598-607.
32. Manji HK, Moore GJ, Chen G. Clinical and preclinical evidence for the neurotrophic effects of mood stabilizers. Implications for the pathophysiology and treatment of manic-depressive illness. *Biological Psychiatry* 2000;48:740-54.

33. Björnebekk A, Mathé AA, Brené S. The antidepressant effect of running is associated with increased hippocampal cell proliferation. *International Journal of Neuropsychopharmacology* 2005;8:357-68.
34. Montgomery SA, Asberg M. A new depression scale designed to be sensitive to change. *British Journal of Psychiatry* 1979;134:382-9.
35. Kroenke K, Spitzer RL, Williams JB. The PHQ-9: validity of a brief depression severity measure. *Journal of General Internal Medicine* 2001;16:606-13.
36. Åstrand P-O, Ryhming I. A nomogram for calculation of aerobic capacity (physical fitness) from pulse rate during submaximal work. *Journal of Applied Physiology* 1954;7:218-21.
37. Guyatt GH, Sullivan MJ, Thompson PJ, Fallen EL, Pugsley SO, Taylor DW, et al. The 6-minute walk. A new measure of exercise capacity in patients with chronic heart failure. *Canadian Medical Association Journal* 1985;132:919-23.
38. Ekblom, Oddsson K, Ekblom B. Barns fysiska prestationsförmåga. Skolprojektet 2001. [Children's physical performance capacity. The School Project.] *Svensk Idrottsforskning* 2002;3:27-31.
39. Biering-Sørensen F. Physical measurements as risk indicators for low-back trouble over a one-year period. *Spine* 1984;2:106-19.

25. *Diabetes mellitus – type 1 diabetes*

Authors

Claes-Göran Östenson, MD, PhD, Professor, Department of Endocrinology, Metabolism and Diabetes, Karolinska University Hospital and Karolinska Institutet, Stockholm, Sweden

Kåre Birkeland, MD, PhD, Professor, Department of Endocrinology, Aker University Hospital and Faculty of Medicine, University of Oslo, Oslo, Norway

Jan Henriksson, MD, PhD, Professor, Department of Physiology and Pharmacology, Karolinska Institutet, Stockholm, Sweden

Summary

Type 1 diabetes is a chronic disease with hyperglycemia (elevated or abnormally high blood sugar levels) due to insulin deficiency. Type 1 diabetes is treated with insulin, often with multiple injections daily.

In patients with type 1 diabetes, blood glucose levels change during physical activity, to a large extent because of insulin levels in the blood. Because insulin levels during exercise are most often higher in diabetics than in non-diabetic individuals, an effect of the treatment, hypoglycemia can easily arise during exercise. The tendency to hypoglycemia can persist for many hours after an exercise session. However, this risk can be avoided by planning diet and insulin doses in connection with physical exercise, and can likely also be reduced through physical training.

Regular physical activity increases the sensitivity to insulin, primarily in the skeletal muscle, which leads to a reduced need for insulin. Because diabetes is associated with a substantially increased risk for developing cardiovascular disease, physical training is very important for this patient group, as with non-diabetics, to influence risk factors such as elevated blood lipid levels and high blood pressure. Control of blood sugar, measured as glycosylated haemoglobin (HbA1c), has not shown to be significantly affected by physical training in the studies reported in the literature. It cannot be ruled out, however, that one may be able to achieve improved glucose control in individual cases by combining physical activity with other measures.

As is true for most people, patients with type 1 diabetes should engage in physical activity for a minimum of 30 minutes per day, at an intensity of at least moderate level, such as a brisk walk, cycling, etc. To attain further health and aerobic fitness effects, this

should be combined with physical activity/exercise/training of somewhat higher intensity at least 3 times per week, for example, a fitness class, ball sports, skiing, or similar activity depending on the person's interests.

Definition

Type 1 diabetes is a chronic disease that involves hyperglycemia (increased or abnormally high blood sugar levels) due to insulin deficiency. The disease was earlier called childhood- or juvenile- or insulin-dependent diabetes, but these descriptions should no longer be used.

Prevalence/Incidence

The onset of type 1 diabetes can be at any age, but is most common in children and young adults, with an increased risk during the preschool years and during puberty. The disease is found in most countries, but the annual incidence varies widely: in Europe, between 3/100,000 in the Balkan states to 30–40/100,000 in Scandinavia and Sardinia. In Sweden and Norway, it is estimated that 4/1000 develop type 1 diabetes before the age of 15 years, and a total of 7/1000 before the age of 35 years. Type 1 diabetes has long been considered responsible for 10–15 per cent of all diabetes, but it is possible that this number is larger in reality due to overlooked autoimmune diabetes that develops in the elderly.

Cause

In most cases (> 90%), the disease is caused by an autoimmune process that gradually destroys the insulin-producing beta cells in the islets of Langerhans. The exact cause of the disease has not been established but appears to be complex. Surrounding factors likely trigger type 1 diabetes in individuals with a genetic predisposition. Twin studies have shown concordance for the disease in 30–50 per cent of monozygote (identical) and 5–15 per cent in dizygote (fraternal) twins. Several genes are believed to be responsible for the inherited background, with the strongest link found in genes that code for HLA (DR-3 and DR-4) in the major histocompatibility complex (MHC) region on chromosome 6. There is also an increased co-occurrence of type 1 diabetes and other autoimmune diseases such as struma lymphomatosa (Hashimoto's thyroiditis), pernicious anaemia and Addison's disease.

Risk factors

Among the external factors that could initiate the autoimmune process in endocrine pancreas, discussion has concentrated on primarily viral infections. An increased development of type 1 diabetes during the fall and winter could be related to viral epidemics, but also to the cold (which can lead to decreased glucose tolerance and increased need for

insulin). Other risk factors that have been put forward, though not indisputably proven to play a role, are nitrosamines in food and early exposure to the protein in cow's milk. And finally, it is likely that the autoimmune process in the pancreas can accelerate in individuals who are growing rapidly (as in puberty), in infections, and in connection with stressful events.

Pathophysiological mechanisms

Even before the onset of clinical diabetes in the islets of Langerhans, signs of chronic inflammation can be seen, so-called insulinitis, with infiltration of macrophages, T- and B-lymphocytes, and ongoing destruction of beta cells. In most patients with established type 1 diabetes, the beta cells have completely disappeared. At the clinical onset, patients have elevated titres of autoantibodies to glutamic acid decarboxylase (GAD) in almost 80 per cent, and tyrosine-phosphatase IA-2 in over 55 per cent of cases. Over 90 per cent have a positive titre if GAD and IA-2 are combined. Insulin antibodies have also been shown. Studies have shown that elevated antibody titres are present before onset of the clinical symptoms. It is unclear, however, whether these antibodies play an active role in beta cell destruction or rather appear secondary to this.

Symptoms and prognosis

Untreated type 1 diabetes leads to severe insulin deficiency with hyperglycemia and ketoacidosis. Symptoms of this condition include abnormally high secretion of urine (polyuria), excessive thirst (polydipsia), emaciation and fatigue. Despite as optimal insulin treatment as possible, there is a risk for developing both acute complications in the form of hypo- and hyperglycemia, and late complications related to the eyes, nervous system, kidneys and cardiovascular system. These complications can have an impact on the patient's ability to engage in physical activity.

Diagnostics

Diabetes is defined as a fasting plasma glucose of 7.0 mmol/l or higher, symptoms of diabetes and random plasma glucose measurements of over 11.0 mmol/l, of plasma glucose over 11.0 mmol/l two hours after intake of 75 g glucose (oral glucose tolerance test). At onset of type 1 diabetes, plasma glucose levels are typically over 20 mmol/l, most often accompanied by an increased ketone body production and sometimes acidosis. C-peptide levels in the blood can be low and as a rule the patient has positive titres for GAD and IA-2 antibodies.

Treatment

The treatment for type 1 diabetes is insulin. This often occurs in multiple injections daily, for example, with rapid-acting insulin before meals and medium-acting insulin at night,

or a mixed insulin (rapid and intermediate-acting) before breakfast and the evening meal. Insulin can also be administered via an insulin pump.

Effects of physical activity

Effect of acute exercise

In patients with type 1 diabetes, blood glucose levels change during physical activity, to a large extent because of insulin levels in the blood. It is thus important to consider the type of insulin (rapid- or long-acting) that the patient takes and the time interval between insulin injection and activity. Blood glucose levels fall in hyperinsulinemia during the physical activity if the latter is long-lasting (more than 30–60 minutes) or intensive, if more than three hours has passed since the last meal, and if the patient does not eat more before and during the activity (1, 2). Blood glucose levels can be lower than normal for up to 24 hours after an exercise session. On the other hand, blood glucose levels can rise in hypoinsulinemia, during vigorous exercise, or with the intake of large amounts of carbohydrates before and during the activity.

Effect of regular exercise training

Physical activity increases the sensitivity to insulin, primarily in the skeletal muscles, which leads to a reduced need for insulin (2–4). Control of blood sugar, measured as glycosylated haemoglobin (HbA1c), has not shown to be significantly affected by physical training in the studies reported in the literature (2, 4). It should, however, be noted that these results come from groups of study subjects that performed standardised training programmes and it cannot be ruled out that, some individuals may be able to achieved improved glucose control by combining physical activity with other measures. Because diabetes is associated with a substantially increased risk for developing cardiovascular disease, it is also important that physical training in this patient group leads to a reduction of the risk factors for heart disease, for example, improved blood lipid profile and lower blood pressure (4–6). Otherwise, in type 1 diabetes, the body appears to adapt to exercise training with normal increases of maximal oxygen uptake and of the muscles' capacity to burn energy (mitochondrial oxidative capacity), while the increase in the degree of capillarisation of muscles with training can be somewhat decreased in type 1 diabetes (8, 9). The increased oxidative capacity in a given exercise leads to a larger part of the energy requirements of the musculature being met by burning fat. This should mean that the risk for hypoglycemia is reduced, at least during moderately intensive exertion, in physically fit persons with type 1 diabetes, but the research data in this area is still insufficient (10).

Indications

Primary prevention

There is nothing to suggest that physical activity can prevent the development of type 1 diabetes.

Secondary prevention

Regular physical activity is recommended for people with type 1 diabetes, but special care must be taken to avoid hypoglycemia (1). It is not certain that physical activity leads to better control of blood glucose, but there is nevertheless improvement in blood lipids and other risk factors for cardiovascular disease (4–6). No studies at present suggest that physical activity prevents diabetes' late complications. It cannot be ruled out, however, that one may be able to achieve this goal on an individual basis via improved glucose control through a combination of physical activity and other measures.

Prescription

As is true for most people, patients with type 1 diabetes should engage in a minimum of 30 minutes of physical activity per day, at an intensity of at least moderate level, such as a brisk walk, cycling, etc. To attain further health and aerobic fitness effects, this should be combined with physical activity/exercise/training of a somewhat higher intensity at least 2–3 times per week, for example, a fitness class, ball sports, skiing, or similar activity depending on the person's interests (see also Table 1). Strength training is also recommended (Table 1). If cardiovascular symptoms exist, the strength training should be less strenuous than shown in the table, for example 12–15 repetitions instead of recommended 8–12. In the case of eye symptoms, even lighter weights should be used and 15–20 repetitions of each exercise be performed. In order to avoid a rise in blood pressure, lifts should be done on exhalation and the muscles relaxed during inhalation. For aerobic fitness and strength training, each session should begin with a warm-up and end with a cool-down period of 5–10 minutes each, including careful stretching of tight muscles and soft tissues.

The physical activity should be planned carefully, with measurement of blood glucose before and after (and sometimes during) the activity session. A suitable blood glucose interval before exercising is 6–15 mmol/l. If the blood glucose level is over 15 mmol/l and/or ketosis occurs, the person should not exercise, and if the blood glucose level is under 6 mmol/l, he or she should definitely eat more before exercising. If possible, schedule the physical activity one to two hours after a meal, and more than one hour after insulin injection. Avoid injecting insulin in parts of the body that are active during the exercise. If necessary, reduce the preceding dose of insulin by 30–50 per cent, especially before extensive training or in the case of regular exercising. Even the dose of insulin after the physical activity may need to be reduced (5).

Take along some “fast carbohydrates” in the form of dextrose tablets, energy drinks or fruit during the training. During long exercise sessions, it can be necessary to take 15–30 g dextrose every 30 minutes.

Table 1. General recommendations for physical activity in diabetes mellitus – type 1 diabetes (11).

Type of training	Examples of activities	Frequency	Intensity	Duration
Basic activity	Walking, climbing stairs, gardening. It is also desirable to increase standing/walking time at work and in the home.	Daily	So talking is still possible, 30–50% of maximal oxygen uptake; 12–13 acc. to Borg's scale	> 30 min.
Aerobic fitness training	Nordic walking, jogging, cycling, swimming, skiing, skating, fitness class/aerobics/dance, ball sports, rowing	3–5 days/week	Until out of breath Begin slowly and gradually increase to 40–70% of maximal oxygen uptake; 13–16 acc. to Borg's scale*	20–60 min.
Strength training	Movements using the body as resistance, resistance bands, weights, weight/resistance equipment	2–3 days/week	Until or near muscular exhaustion for each exercise**	8–10 exercises, with 8–12 reps of each exercise

* Level of exertion may need to be reduced in the case of retinal, renal or cardiovascular complications as well as autonomic dysfunction.

** Replace with easier exercises in the case of retinal, renal or cardiovascular complications.

Functional mechanisms

Blood sugar concentration is a result of the balance between the liver's ability to release glucose into the blood stream and the uptake of glucose by the tissues. At rest, the liver releases approximately 7.5 g sugar per hour, most of which (about 6 g/hour) is consumed by the central nervous system and the tissues that lack the capacity of aerobic metabolism, above all the red blood cells. During physical exercise, the picture changes in that the working muscles' glucose uptake increases dramatically. For example, a person burns about 30 g of carbohydrates per hour of walking, whereof 15–20 g is covered by blood sugar. In moderate running, carbohydrate utilisation can increase to 90–100 g per hour, whereof about a quarter is covered by blood sugar (7).

The normally precisely regulated balance between glucose released by the liver and taken up by the tissues is easily disrupted in connection with exercise in people with type 1 diabetes. Normally, physical exertion leads to a sharp drop in the concentration of insulin in the blood. This is a result of the powerful activation of the sympathetic nervous system that occurs in connection with physical exertion, which leads to impairment of the insulin-producing cells. The fact that the sugar uptake of the muscles increases sharply in connection with exertion, despite the low insulin level in the blood is due to a non-insulin-dependent increase in the permeability of sugar to the muscle cells. In type 1 diabetes, this delicate

regulation can be disrupted by the insulin left in the blood from the previous dose of insulin, which tends to both increase the skeletal muscle's glucose uptake and decrease the glucose released by the liver (2). The result is that hypoglycemia easily arises during exertion, and can persist for many hours after an exercise session (1). The skeletal muscle's increased sensitivity to insulin that is present for 1–2 days after an exercise session, largely caused by a drop in muscle glycogen levels also contributes to the persistence of the hypoglycemia (2, 3). Exercising leads to a significant increase in the muscles' metabolic capacity (mitochondrial oxidative capacity) (8). In a given exercise, this leads to a larger part of the energy requirements of the muscle being met by burning fat. The reduced use of carbohydrates during exertion can be considerable and can lower the needs of the musculature for blood sugar during exertion by 20 per cent or more, thereby reducing the risk for exercise-induced hypoglycemia.

In poor diabetes regulation with high blood sugar and a tendency to ketosis, an exercise session can yield the opposite effect, that is, hyperglycemia. The reason for this is unclear, but likely has to do with insulin deficiency that produces high blood sugar, at the same time as the increase in the skeletal muscle's sensitivity to insulin, as a result the exercise, can be reduced, for example, as a result of high levels of fatty acids and ketone bodies in the blood (2).

Functional tests/Need for health check-ups

In some cases, especially in older patients or in the case of long diabetes duration, it is appropriate to conduct a stress test or other examination to assess heart status. The presence of peripheral and autonomic neuropathy, impaired sensation, impaired joint function and proliferative retinopathy should also be assessed, as should kidney disease. The latter are necessary because elevated blood pressure during activity may aggravate eye problems and the development of kidney disease. And finally, one should examine the feet with respect to loss of sensation, uneven loading, pressure sores and hyperkeratoses, as well as the presence of sores.

Interactions with drug therapy

Physical activity increases both insulin sensitivity and insulin-independent glucose uptake in muscle, and thus enhances the insulin effect.

Contraindications

Absolute

- Hyperglycemia and/or ketosis.

Relative

- Caution with concurrent heart disease.
- In the case of peripheral neuropathy, there is a risk for injuries to the feet and joints.
- In the case of proliferative retinopathy, there is a risk for exacerbation of eye status (uncommon).
- In the case of autonomic neuropathy, physical activity that is too intensive can be associated with risks (hypotension and lack of early warning signs for cardiac ischaemia).
- In the case of kidney disease, high blood pressure (systolic pressure of 180–200 mmHg) can aggravate the development of the disease.

References

1. Cryer PE, Davis SN, Shamon H. Hypoglycemia in diabetes. *Diabetes Care* 2003;26:1902-12.
2. Wallberg-Henriksson H. Acute exercise. Fuel homeostasis and glucose transport in insulin-dependent diabetes mellitus. *Med Sci Sports Exerc* 1989;21:356-61.
3. Borghouts LB, Keizer HA. Exercise and insulin sensitivity. A review. *Int J Sports Med* 2000;21:1-12.
4. Peirce NS. Diabetes and exercise. *Br J Sports Med* 1999;33:161-72; Quiz 172-3, 222.
5. Wallberg-Henriksson H, Krook A. Motion. In: Agardh C-D, Berne C, Stman J, Eds. *Diabetes*, 3rd edn. Stockholm: Liber AB; 2005, pp. 97-108.
6. Rigla M, Sanchez-Quesada JL, Ordonez-Llanos J, Prat T, Caixas A, Jorba O, et al. Effect of physical exercise on lipoprotein(a) and low-density lipoprotein modifications in type 1 and type 2 diabetic patients. *Metabolism* 2000;49:640-7.
7. Henriksson J, Sahlin K. Metabolism during exercise. Energy expenditure, hormonal changes. In: Kjaer M, Magnusson P, Roos H, Eds. *Textbook of Sports Medicine*. Oxford: Blackwell Science; 2003.
8. Henriksson J. Effects of physical training on the metabolism of skeletal muscle. *Diabetes Care* 1992;15:1701-11.
9. Kivelä R, Silvennoinen M, Touvra AM, Lehti TM, Kainulainen H, Vihko, V. Effects of experimental type 1 diabetes and exercise training on angiogenic gene expression and capillarization in skeletal muscle. *FASEB J* 2006;20:1570-2.
10. Ramchandani N, Cantey-Kiser JM, Alter CA, Brink SJ, Yeager SD, Tamborlane WV, et al. Self-reported factors that affect glycemic control in college students with type 1 diabetes. *Diabetes Educ* 2000;26:656-66.
11. Standards of medical care in diabetes 2007. *Diabetes Care* 2007;30 (Suppl 1):S4-41.

26. *Diabetes mellitus – type 2 diabetes*

Authors

Claes-Göran Östenson, MD, PhD, Professor, Department of Endocrinology, Metabolism and Diabetes, Karolinska University Hospital and Karolinska Institutet, Stockholm, Sweden

Kåre Birkeland, MD, PhD, Professor, Department of Endocrinology, Aker University Hospital and Faculty of Medicine, University of Oslo, Oslo, Norway

Jan Henriksson, MD, PhD, Professor, Department of Physiology and Pharmacology, Karolinska Institutet, Stockholm, Sweden

Summary

Type 2 diabetes represents 80–90 per cent of all diabetes and is a chronic disease characterised by hyperglycemia and other metabolic disorders. The basis of treatment is measures that reduce insulin resistance, above all increased physical activity, weight loss in the case of overweight, and stopping the use of tobacco. If these lifestyle measures are not enough to properly control the disease, there are a number of different medications in tablet form, and insulin.

Generally speaking, it can be said that, by leading to a decreased sensitivity to insulin, physical inactivity is a significant risk factor for type 2 diabetes, and several studies have shown that the development of type 2 diabetes can be prevented through exercise training combined with dietary guidelines. Regular exercise for type 2 diabetes has a positive effect on both insulin sensitivity and other risk factors for cardiovascular disease, for example, blood lipid profile and blood pressure. This is of great importance since the risk for developing cardiovascular disease is three to four times higher in diabetes. Several studies also report improved glucose control. It is recommended that the patient perform a minimum of 30 minutes of medium intensity physical activity daily, such as a brisk walk, cycling or similar activity adapted to his or her overall physical condition and lifestyle. Further health effects and aerobic fitness can be achieved if this is combined with somewhat more intensive exercise at least 2–3 times per week, such as a fitness class, tennis, swimming, skiing or similar, depending on the individual's interests.

Definition

Type 2 diabetes is a chronic disease characterised by hyperglycemia (elevated or abnormally high blood sugar levels) and other metabolic disturbances, including metabolism of lipids and haemostasis. The disease was earlier called adult-onset or non-insulin-dependent diabetes, but these descriptions should no longer be used.

Prevalence/Incidence

Type 2 diabetes represents 80–90 per cent of all diabetes. The total prevalence in the Nordic countries is 4–5 per cent of the population over 20 years of age, but rises sharply after the age of 50–60 years. Approximately 20 per cent of people over the age of 70 years are affected. In recent years, the disease has been shown in children with a genetic predisposition and who also have other risk factors, for example, overweight and physical inactivity. Globally, the incidence of type 2 diabetes is increasing dramatically, above all in India, the Middle East, China, USA and parts of Latin America. The actual prevalence is often unknown, as the disease can develop quite insidiously and not be detected until a health check-up is performed. It has been estimated that the number of undiagnosed patients with type 2 diabetes make up at least half or even the same number of patients already known to have diabetes. In a small portion, approximately 5 per cent, of patients earlier counted as type 2 diabetics, the genetic background has been established. These people carry different types of mutations in transcription factors or glucokinase (Maturity Onset Diabetes of the Young, MODY), which is inherited as autosomal dominant and leads to diabetes in the early years (to be distinguished, however, from type 2 diabetes in children with the risk factors mentioned above).

Cause

Hyperglycemia in type 2 diabetes most often develops due to insufficient insulin secretion and reduced insulin sensitivity (insulin resistance). Insulin resistance presents, at least in pronounced disease, both in the liver and in extrahepatic (outside the liver) tissues, principally in the skeletal muscle (1, 2). This leads to a pathologically increased glucose production from the liver and reduced glucose uptake in the muscles. However, insulin resistance cannot, on its own, lead to hyperglycemia/diabetes, but a concurrent defective secretion of insulin from the beta cells in the islets of Langerhans is also required. Type 2 diabetes develops in genetically predisposed individuals via a stage of reduced glucose tolerance. Heredity is considered polygenous, but which genes are responsible for defects in insulin secretion and/or insulin sensitivity is not yet fully established. Of the known candidate genes associated to an increased risk for type 2 diabetes, most appear to have more significance for insulin secretion than for insulin resistance (3–7).

Risk factors

Most of the lifestyle factors that are known to increase the risk for type 2 diabetes reduce insulin sensitivity (8–14). These include overweight, physical inactivity and the use of tobacco. There is also evidence that a fat-rich and fibre-poor diet, as well as psychosocial stress, independent of body weight, can lead to an increased risk for developing type 2 diabetes (10).

Pathophysiological mechanisms

The molecular mechanisms behind defective insulin secretion and insulin resistance are still unclear. Insulin release is reduced primarily when stimulated with glucose, but also when stimulated with other agents, such as certain amino acids. The beta cell defect is likely primary, but some studies have shown that the defect may arise as a result of “exhaustion” (high demand on secretion and concurrent insulin resistance). Even the toxic effect of hyperglycemia (glucotoxicity) and dyslipidemia (lipotoxicity) can aggravate beta cell function as well as insulin sensitivity, though these toxic effects can largely be reversed by good metabolic control.

Symptoms and prognosis

In most patient with type 2 diabetes, the disease develops insidiously and with few symptoms. The diagnosis can be made at health check-ups or when more symptoms appear due to higher blood glucose levels, for example, in the case of a serious infection or other concurrent disease. One can then observe increased urination and increased thirst, but rarely significant weight loss. Other symptoms that should lead one to think of type 2 diabetes are skin and urinary tract infections, polyneuropathy (disease of the peripheral nerves), impotence and cardiovascular disease. As in other diabetes, there is a risk in type 2 diabetes for developing late complications in the eyes, nerves, kidneys and cardiovascular system. The risk of myocardial infarction or stroke is 3–4 higher, and it is not uncommon that type 2 diabetes is detected in patients with acute cardiovascular disease.

Diagnostics

Diabetes is defined as a fasting plasma glucose of 7.0 mmol/l or higher, symptoms of diabetes and random plasma glucose measurements of over 11.0 mmol/l, or plasma glucose over 11.0 mmol/l two hours after intake of 75 g glucose (oral glucose tolerance test).

Treatment

The basis of treatment on measures that reduce insulin resistance, mainly increased physical activity (8, 9, 12–20) and a fibre-rich diet containing a maximum of 30 per cent fat (primarily mono- and polyunsaturated) and 50–60 per cent complex carbohydrates. Any use of tobacco should be stopped. If these lifestyle measures are not enough to properly control the disease, there are a number of different kinds of oral medications and insulin (21).

Metformin is the drug of choice, the main effect of which is the reduction of glucose production in the liver, while sulfonylureas or glinides are used to stimulate insulin secretion. Glitazones (thiazolidinediones) can be used in combination with either metformin or insulin-stimulating drugs to increase insulin sensitivity, above all in muscle. Newer drugs are analogues to the intestinal hormone GLP-1 (glucagon-like peptide), such as exenatide, or enzyme inhibitors that increase the endogenous level of GLP-1, such as sitagliptin. These drugs improve plasma glucose levels by, among other things, increasing the endogenous insulin secretion and inhibiting glucagon secretion. Acarbose inhibits the breakdown of disaccharides in the intestine. Type 2 diabetes is a progressive disease, however, and after 5–10 years of treatment a large majority of patients fail on these peroral drugs. Insulin treatment can then be required in order to maintain acceptable control, especially if weight loss occurs. Today, insulin treatment is most often given in combination, for example, with metformin. Primary insulin treatment can be necessary if the patient with type 2 diabetes has high blood glucose levels at onset. In many cases a transition to peroral therapy can occur later, however.

Effects of physical activity

Effect of acute exercise

In healthy individuals, physical exercise does not normally lead to changes in the blood sugar concentration, even if maximal exertion can lead to increased blood sugar levels. This generally also applies to type 2 diabetics with only dietary treatment, and it is unusual that physical exercise leads to hypoglycemia in this patient group. These individuals therefore do not normally need to think about eating more in connection with an exercise session, as long as the physical exercise is not strenuous or long-lasting (e.g. a marathon race) (22). In people with type 2 diabetes who receive insulin treatment, sulfonylurea drugs or glinides, however, moderate to strenuous exercise leads to a fall in blood sugar concentration during the exertion itself, a change that can remain up 12 hours after the exercise is finished. In the course following very strenuous exercise, on the other hand, hyperglycemia can arise due to increased plasma hormone levels, which stimulate the liver's glucose production, together with a reduction in the sugar uptake of the skeletal muscle post-exercise (23, 24).

Effects of regular exercise training

Regular exercise training in type 2 diabetics leads to an increase in the insulin sensitivity of the tissues even at rest. An increased insulin sensitivity with training is also seen in non-diabetics, but is of particular importance in type 2 diabetics and other patient groups that normally already have a reduced insulin sensitivity at rest (12, 13, 15–20, 25). It can generally be said that, by leading to a lower sensitivity to insulin, physical inactivity is a risk factor for type 2 diabetes, and several studies have shown that the development of type 2 diabetes can be prevented through exercise training.

An interesting finding is that there is a reversed relationship between the amount of exercise training and the risk of developing type 2 diabetes (10). Because type 2 diabetes is associated with a 3–4 times higher risk for myocardial infarction and stroke, it is also important that exercise training in this patient group has an impact on the risk factors for heart disease, in addition to through the increased insulin sensitivity, also through leading to an improved blood lipid profile and lower blood pressure (16–19, 22, 26). An interesting question is also whether blood sugar control is affected by exercise training in type 2 diabetics, since good blood sugar control reduces serious late complications in diabetes. The research data in this area was negative for a long time, but recent studies have shown that improved blood sugar control can be achieved with exercise training, especially for younger age groups (27). A recently published study, in which non-insulin-treated type 2 diabetics (39–70 years) were randomly assigned to endurance training, strength training, combined training or to a control group without training, for 6 months, showed improved blood sugar control, measured as glucosylated haemoglobin (HbA1c), in all training groups. Significant best outcomes were shown in the sample group where fitness and strength training were combined (28). In a randomised study, researchers were able to show that lifestyle treatment (physical activity 2–3 times per week and dietary counseling) were as effective at reducing HbA1c in a group of tablet-treated type 2 diabetics with poor blood sugar control as starting insulin treatment (29).

Indications

Primary prevention

A number of randomised prospective studies and epidemiological observation studies suggest good primary prevention effect from physical activity in individuals with reduced glucose tolerance (11–14, 30–33). In some of these studies, regular exercise was combined with weight loss (approx. 5%) (30, 31), whereas other large prospective studies have shown a diabetes-preventive effect with physical activity as the only intervention (32–34). An example of the latter that can be noted is the study in the Chinese city of Da Qing (32), in which 577 individuals with impaired glucose tolerance were divided into four groups, among them a group with only exercise training as treatment. After 6 years, 68 per cent of the individuals in the untreated control group had developed type 2 diabetes, compared to only 41 per cent in the exercise group.

Secondary prevention

Regular physical activity is an important part of the treatment of type 2 diabetes (12, 13, 16–20, 34). By contributing to good metabolic control (see above), it is likely that also the development of late diabetic complications can be reduced.

Prescription

Medium intensity physical activity, a minimum of 30 minutes per day of brisk walking, cycling or similar activity, adapted to the individual’s overall physical condition and lifestyle. It is important to include warm-up and cool-down components of a lower intensity. Further health and aerobic fitness effects can be achieved if this is combined with somewhat more intensive exercise at least 2–3 times per week, for example, a fitness class, tennis, swimming, or skiing. Strength training is also recommended (Table 1). If cardiovascular symptoms are present the strength training should be less strenuous than shown in the table, for example 12–15 repetitions instead of recommended 8–12. In the case of eye symptoms, even lighter weights should be used, for example, 15–20 repetitions of each exercise. In order to avoid a rise in blood pressure, lifts should be done on exhalation and the muscles relaxed during inhalation. For aerobic fitness and strength training, each session should begin with a warm-up and end with a cool-down period of 5–10 minutes each, including careful stretching of tight muscles and soft tissues.

Physical activity aimed at weight loss should be combined with a reduced calorie intake. Hypoglycemia seldom occurs in connection with exercise and extra intake of carbohydrates is therefore not necessary. Patients receiving insulin treatment or taking insulin-stimulating peroral drugs may become hypoglycemic, however, especially if they do not have pronounced insulin resistance (hypoglycemia in connection with physical activity is also discussed under the heading of Effect of acute exercise in this chapter and the chapter on type 1 diabetes).

Table 1. General recommendations for physical activity in type 2 diabetes mellitus (35).

Type of training	Examples of activities	Frequency	Intensity	Duration
Basic activity	Walking, climbing stairs, gardening. It is also desirable to increase standing/walking time at work and at home.	Daily	So talking is still possible, 30–50% of maximal oxygen uptake; 12–13 acc. to Borg’s scale.	> 30 min.
Aerobic fitness training	Nordic walking, jogging, cycling, swimming, skiing, skating, fitness class/aerobics/dance, ball sports, rowing.	3–5 times/week	Until out of breath Begin slowly and gradually increase to 40–70% of maximal oxygen uptake; 13–16 acc. to Borg’s scale*.	20–60 min.
Strength training	Movements using the body as resistance, resistance bands, weights, weight/resistance equipment.	2–3 days/week	Until or near muscular exhaustion for each exercise**.	8–10 exercises, with 8–12 reps of each exercise

* Level of exertion may need to be reduced in the case of retinal, renal or cardiovascular complications as well as autonomic dysfunction.

** Replace with easier exercises in the case of retinal, renal or cardiovascular complications.

Functional mechanisms

Even though it is unusual for physical exercise to lead to hypoglycemia in people with type 2 diabetes, the blood sugar concentration changes in connection with exercise more often in type 2 diabetics than in non-diabetics. This has to do with the marked increase in sugar uptake for the skeletal muscle during exertion due to a non-insulin-dependent increased permeability of sugar to the muscle cells. In healthy non-diabetic individuals, the increased sugar uptake of the skeletal muscle is compensated for during exertion by an increased release of glucose from the liver due to an increase in the hormone glucagon as a result of the exertion, while insulin levels fall. In people with type 2 diabetes, this compensation is sometimes not sufficient and leads to lower blood sugar, possibly due to the fact that the insulin concentration in the blood is often elevated in these people, which can result in insufficient release of glucose from the liver (23, 24). The increased blood sugar concentration seen in type 2 diabetics in the course following a vigorous exercise session is considered to be connected to remaining high concentrations of so-called counter-regulating hormones post-exercise (24).

The increased insulin sensitivity with regular exercise can be explained by changes at different levels, for example, a changed body composition, with less fat and more muscle, and cellular changes in the skeletal muscle, such as increased concentrations of glucose transporter proteins and glycogen synthase (13, 25).

Functional tests/Need for health check-ups

In some cases, especially in older patients or in the case of long diabetes duration, it is appropriate to conduct a stress test or other examination to assess heart status. The presence of peripheral neuropathy, impaired sensation, impaired joint function, eye complications (proliferative retinopathy) and kidney disease should also be assessed. The latter are necessary because elevated blood pressure during activity may aggravate eye problems and the development of kidney disease. And finally, one should examine the feet with respect to loss of feeling, uneven loading, pressure sores and corns and calluses, as well as the possible presence of sores.

Interactions with drug therapy

Physical activity increases both insulin sensitivity and insulin-independent glucose uptake in muscle, and thus amplifies the insulin effect. This can be of practical importance in certain patients receiving insulin treatment or insulin-stimulating peroral drugs.

Contraindications

Relative

Caution with concurrent heart disease. In the case of peripheral neuropathy there is a risk for injuries to the feet and joints. In the case of eye complications (proliferative retinopathy) there is a risk for exacerbation of eye status (uncommon). In the case of autonomic neuropathy, physical activity that is too intensive can be associated with risks (hypotension and lack of early warning signs for cardiac ischaemia). In the case of kidney disease, high blood pressure (systolic pressure of 180–200 mmHg) can aggravate the development of the disease.

References

1. Boden G. Pathogenesis of type 2 diabetes. Insulin resistance. *Endocrinol Metab Clin North Am* 2001;30:801-15.
2. Gerich JE. Is insulin resistance the principal cause of type 2 diabetes? *Diabetes Obes Metab* 1999;1:257-63.
3. Saxena R, Voight BF, Lyssenko V, Burt NP, de Bakker PI, Chen H et al. Genome-wide association analysis identifies loci for type 2 diabetes and triglyceride levels. *Science* 2007;316:1331-6.
4. Scott LJ, Mohlke KL, Bonnycastle LL, Willer CJ, Li Y, Duren WL, et al. A genome-wide association study of type 2 diabetes in Finns detects multiple susceptibility variants. *Science* 2007;316:1341-5.
5. Sladek R, Rocheleau G, Rung J, Dina C, Shen L, Serre D, et al. A genomewide association study identifies novel risk loci for type 2 diabetes. *Nature* 2007;445:881-5.
6. Zeggini E, Weedon MN, Lindgren CM, Frayling TM, Elliott KS, Lango H, et al. Replication of genome-wide association signals in UK samples reveals risk loci for type 2 diabetes. *Science* 2007;316:1336-41.
7. Steinthorsdottir V, Thorleifsson G, Reynisdottir I, Benediktsson R, Jonsdottir T, Walters GB, et al. A variant in CDKAL1 influences insulin response and risk of type 2 diabetes. *Nat Genet* 2007;39:770-5.
8. Sheard NF. Moderate changes in weight and physical activity can prevent or delay the development of type 2 diabetes mellitus in susceptible individuals. *Nutr Rev* 2003;61:76-9.
9. Ryan AS. Insulin resistance with aging. Effects of diet and exercise. *Sports Med* 2000;30:327-46.
10. Perry IJ. Healthy diet and lifestyle clustering and glucose intolerance. *Proc Nutr Soc* 2002;61:543-51.
11. Kriska AM, Blair SN, Pereira MA. The potential role of physical activity in the prevention of non-insulin-dependent diabetes mellitus. The epidemiological evidence. *Exerc Sport Sci Rev* 1994;22:121-43.
12. Ivy JL, Zderic TW, Fogt DL. Prevention and treatment of non-insulin-dependent diabetes mellitus. *Exerc Sport Sci Rev* 1999;27:1-35.
13. Ivy JL. Role of exercise training in the prevention and treatment of insulin resistance and non-insulin-dependent diabetes mellitus. *Sports Med* 1997;24:321-36.
14. Helmrich SP, Ragland DR, Paffenbarger Jr RS. Prevention of non-insulin-dependent diabetes mellitus with physical activity. *Med Sci Sports Exerc* 1994;26:824-30.
15. Young JC. Exercise prescription for individuals with metabolic disorders. Practical considerations. *Sports Med* 1995;19:43-54.
16. Wallberg-Henriksson H, Rincon J, Zierath JR. Exercise in the management of noninsulin-dependent diabetes mellitus. *Sports Med* 1998;25:25-35.
17. Tudor-Locke CE, Bell RC, Meyers AM. Revisiting the role of physical activity and exercise in the treatment of type 2 diabetes. *Can J Appl Physiol* 2000;25:466-92.

18. Hamdy O, Goodyear LJ, Horton ES. Diet and exercise in type 2 diabetes mellitus. *Endocrinol Metab Clin North Am* 2001;30:883-907.
19. Eriksson JG. Exercise and the treatment of type 2 diabetes mellitus. An update. *Sports Med* 1999;27:381-91.
20. Creviston T, Quinn L. Exercise and physical activity in the treatment of type 2 diabetes. *Nurs Clin North Am* 2001;36:243-71.
21. Nathan DM, Buse JB, Davidson MB, Heine RJ, Holman RR, Sherwin R, et al. Management of hyperglycemia in type 2 diabetes. A consensus algorithm for the initiation and adjustment of therapy. A consensus statement from the American Diabetes Association and the European Association for the Study of Diabetes. *Diabetes Care* 2006;29:1963-72.
22. Peirce NS. Diabetes and exercise. *Br J Sports Med* 1999;33:161-72; Quiz 172-3, 222.
23. Cryer PE, Davis SN, Shamon H. Hypoglycemia in diabetes. *Diabetes Care* 2003;26:1902-12.
24. Kelley DE, Goodpaster BH. Effects of exercise on glucose homeostasis in Type 2 diabetes mellitus. *Med Sci Sports Exerc* 2001;33:S516-501, Discussion S528-9.
25. Borghouts LB, Keizer HA. Exercise and insulin sensitivity. A review. *Int J Sports Med* 2000;21:1-12.
26. Rigla M, Sanchez-Quesada JL, Ordonez-Llanos J, Prat T, Caixas A, Jorba O, et al. Effect of physical exercise on lipoprotein(a) and low-density lipoprotein modifications in type 1 and type 2 diabetic patients. *Metabolism* 2000;49:640-7.
27. Boule NG, Haddad E, Kenny GP, Wells GA, Sigal RJ. Effects of exercise on glycemic control and body mass in type 2 diabetes mellitus. A meta-analysis of controlled clinical trials. *JAMA* 2001;286:1218-27.
28. Sigal RJ, Kenny GP, Boulé NG, Wells GA, Prud'homme D, Fortier M, et al. Effects of aerobic training, resistance training, or both on glycemic control in type 2 diabetes. A randomized trial. *Ann Intern Med* 2007;147:357-69.
29. Aas AM, Bergstad I, Thorsby PM, Johannesen O, Solberg M, Birkeland KI. An intensified lifestyle intervention programme may be superior to insulin treatment in poorly controlled type 2 diabetic patients on oral hypoglycaemic agents. Results of a feasibility study. *Diabet Med* 2005;22:316-22.
30. Knowler WC, Barrett-Connor E, Fowler SE, Hamman RF, Lachin JM, Walker EA, et al. Diabetes Prevention Program Research Group. Reduction in the incidence of type 2 diabetes with lifestyle intervention or metformin. *New Engl J Med* 2002;346:393-403.
31. Tuomilehto J, Lindstrom J, Eriksson JG, Valle TT, Hämäläinen H, Ilanne-Parikka P, et al. Prevention of type 2 diabetes mellitus by changes in lifestyle among subjects with impaired glucose tolerance. *N Engl J Med* 2001;344:1343-50.
32. Pan XR, Li GW, Hu YH, Wang JX, Yang WY, An ZX, et al. Effects of diet and exercise in preventing NIDDM in people with impaired glucose tolerance. The Da Qing IGT and Diabetes Study. *Diabetes Care* 1997;20:537-44.
33. Eriksson K-F, Lindgärde F. Prevention of type 2 (non-insulin-dependent) diabetes mellitus by diet and physical exercise. *Diabetologia* 1991;34:891-8.

34. Östenson C-G, Båvenholm P, Efendic S. Motion effektivt vapen i kampen mot typ 2-diabetes [Exercise an effective weapon in the fight against type 2 diabetes]. *Läkartidningen* 2004;101:4011-5.
35. Standards of medical care in diabetes 2007. *Diabetes Care* 2007;30 (Suppl 1):S4-41.

27. Dizziness and balance disorders

Authors

Torbjörn Ledin, MD, PhD, Associate Professor, Ear, Nose and Throat Clinic, Linköping University Hospital, and Division of Neuroscience – Otorhinolaryngology, Department of Clinical and Experimental Medicine (IKE), Faculty of Health Sciences, Linköping, Sweden

Ann-Sofi Kammerlind, PT, PhD, Linköping University Hospital, Division of Physiotherapy, Department of Medical and Health Sciences, Faculty of Health Sciences, Linköping, Sweden

Summary

Dizziness is a common complaint, the prevalence of which increases with age. Reduced, lost or impaired function in one or more of the components of our balance system may be caused either by aging or by a number of illnesses and injuries resulting in a disturbed balance and motion illusions (dizziness). Diagnostics include a detailed anamnesis, vestibular function testing, positional testing and assessment of postural control. This chapter describes some diagnoses of dizziness where mobility training is essential to recovery.

An acute loss of peripheral vestibular function on one side leads to a sudden onset of severe dizziness and disequilibrium. Recovery can be accelerated through stimulation of central compensation by gradually increasing the intensity of eye and head movements and balance exercises. Benign paroxysmal positional vertigo (BPPV) is when the otoconia become dislodged from their usual position and migrate into one of the semicircular canals of the inner ear causing dizziness with head movements or postural changes. BPPV is treated according to two treatment principles: habituation training and maneuver treatment. Damage to the central nervous system and age-related changes in the balance system can also lead to dizziness and disequilibrium. In older people, dizziness and disequilibrium constitute a serious risk factor for falls and subsequent fractures. In the case of damage to the central nervous system and age-related dizziness, the aim of physical training is to improve balance, coordination and strength, to reduce the fear of moving, and to increase the level of activity.

Definition

Prevalence/Incidence

In Sweden, approximately 20 per cent of younger women and 15 per cent of younger men report having experienced dizziness at some point. The prevalence of dizziness increases with age and, at the age of 75, about 40 per cent of women and 30 per cent of men report that they suffer from dizziness or disequilibrium (1). In a Norwegian population-based health study (HUBRO 2000–2001), approximately 30 per cent of women and 20 per cent of men aged 30–60 years stated that they had suffered dizziness in the past 14 days. The incidence in 75–76-year-olds was 10 per cent higher for both genders (2).

Among patients seeking medical help for dizziness, 44 per cent were found to have a peripheral vestibular disorder, 11 per cent a central vestibular disorder, 16 per cent a psychiatric condition, and 26 per cent a different cause of dizziness (e.g. medical treatment). In 13 per cent of the patients, the cause of dizziness was unknown (3). Approximately one in three people aged 65 years or older report a fall in the past 12 months (4), and about 10 per cent report having fallen as a result of dizziness or problem with their balance (5).

Cause

The function and movement of humans are dependent on balance and postural control. The information provided by three receptor organs, i.e. the vestibular system, vision and proprioception, is integrated in the central nervous system, producing movements in the musculoskeletal system. Receptors in the vestibular part of the inner ear register the position and movements of the head. The visual system signals the positioning and movement of the body in relation to its surroundings, at the same time as the proprioceptive receptors provide information about the position and movements of body parts in relation to each other (6).

Reduced, lost or disturbed function in one or more of the components of our balance system may be caused either by aging or by a number of illnesses and injuries, resulting in a disturbed balance function and motion illusions (dizziness). The following describes some of the most common diagnoses of dizziness and disequilibrium where physical activity plays an important role. Other diagnoses not covered here are, for example, cervical vertigo, disequilibrium due to acoustic neurinoma, migraine-related dizziness, Ménière's disease, bilateral peripheral vestibular loss and psychogenic dizziness.

Pathophysiological mechanisms

An acute loss of peripheral vestibular function on one side can be either partial or total, and may be caused by, for example, a virus (7). In the case of benign paroxysmal positional vertigo (BPPV), otoconia (calcium carbonate crystals) have become dislodged from the usual position and migrated into one of the semicircular canals of the inner ear (often the posterior canal), leading to incorrect registration of movements with changes in body

position (8). Benign paroxysmal positional vertigo is usually classified as degenerative or idiopathic, but can also occur as a result of head trauma, acute peripheral vestibular loss, or a long period of confinement to bed (9). Injuries to the central nervous system (predominantly the medulla oblongata, pons and cerebellum) are sometimes caused by disturbed blood circulation, leading to dizziness and disequilibrium due to impaired central processing (10). In the case of age-related dizziness and disequilibrium, gradual age-related deterioration of balance system function, disease and inactivity may negatively effect postural control (11, 12).

Symptoms

Patients with an acute loss of balance nerve function, for example, in connection with vestibular neuritis (inflammation of the balance nerve), may suffer sudden onset nystagmus (pathological movements of the eye with a quick and a slow phase), rotational dizziness, nausea and disequilibrium (13). A certain degree of recovery takes place without treatment within a period of a few weeks or months, facilitated by various compensation mechanisms of the central nervous system (7).

As for BPPV, after a brief latency period, rotational dizziness and nystagmus are provoked in the affected semicircular canal for approx. 10–30 seconds with head movements (8). These periods of dizziness and nystagmus are not long enough for the patient to experience any severe feelings of nausea in isolated cases of provocation.

The character and course of the dizziness and disequilibrium caused by an injury to the central nervous system vary depending on the localisation and extent of the injury. The natural course following central disequilibrium is often more prolonged and the outcome often worse than that of a peripheral vestibular injury (10), likely due to inferior central compensation of the damaged mechanism.

The onset of age-related dizziness and disequilibrium is often slow and inconstant. Inactivity is often a secondary consequence. Dizziness and disequilibrium in older people constitute a significant risk factor for falls and fractures (14), with the number of falls, fractures and other fall-related injuries increasing with age (15).

Diagnosis

Anamnesis plays an important role in the diagnosis of dizziness and disequilibrium. A description of the nature and duration of the dizziness experienced, along with triggering factors and other concurrent symptoms, is very useful and often essential when making a diagnosis, as many patients are examined subsequent to experiencing acute symptoms.

Nystagmography (16) is used to assess vestibular function and the presence of nystagmus in diagnosing acute peripheral vestibular loss, where findings should include reduced function in one of the lateral semicircular canals, and the absence of central function disturbance (slow visual tracking movement, positioning testing and visual suppression of caloric nystagmus).

However, the diagnosis of BPPV cannot be based on an anamnesis alone. A Dix-Hallpike maneuver test using Frenzel glasses is also required to detect nystagmus (17). The patient is quickly laid back into a supine position with the head lower than the body and suspected ear turned 45 degrees to the side so that the posterior canal is affected by movement when changing position.

Nystagmography can also be used to detect damage to the central nervous system (16). To make the diagnosis of central infarction or bleeding, computed tomography or magnetic imaging is needed (18). Often other associated central neurological findings are also made (18).

Treatment

In acute peripheral vestibular loss, recovery is stimulated with eye and head movements and balance exercises of a gradually increased intensity. Patients with acute onset of symptoms should be instructed on how to perform these movements and exercises in order to quickly activate the central compensation mechanisms, i.e. adaptation and substitution (9, 19). A doctor or physiotherapist should then monitor the patient's progress for the following month, and the patient be offered intensive outpatient vestibular rehabilitation training if recovery and return to work do not progress as planned.

There are two treatment principles for BPPV: habituation training and maneuver treatment (20). Habituation training involves the patient practicing typical position changes that provoke dizziness at least twice a day (21). Maneuver treatment involves repositioning the otoconia from the affected semicircular canal through a series of body position changes and specific head movements, such as the Epley maneuver (22). The Epley maneuver can be performed directly after a positive Dix-Hallpike test as it starts in the latter's end position. Patients with BPPV do not have as good balance as healthy people when performing static or dynamic balance tests (23), and many continue to have symptoms of unsteadiness after successful treatment of BPPV (24). Some patients may therefore require balance training as a complement to the maneuver treatment or habituation training.

In the case of injury to the central nervous system, balance and coordination training is necessary and a certain degree of compensation may be achieved through mobility training.

The purpose of training for patients with age-related dizziness is to improve balance, coordination and strength, to reduce the fear of moving, and to increase activity levels.

Effect of physical activity

Research on patients with acute vestibular loss following surgery has shown that it is possible to stimulate compensation with mobility and balance training (20, 25–27). A number of studies indicate that vestibular rehabilitation aids central compensation mechanisms in patients with reduced vestibular function (20, 28–31). A systematic review of the quality of studies on vestibular dysfunctions is currently being carried out (32).

In the case of BPPV, it has long been known that habituation training accelerates an otherwise slow spontaneous recovery (21, 27). However, with its documented beneficial effect, the maneuver treatment is used more often these days (22, 33–35).

The effects of mobility training in patients with injuries to the central nervous system are still insufficiently described and evaluated (10). In a small randomised study of patients aged 65 years and over with central dizziness and/or disequilibrium, improved balance function and subjective rating of symptoms were noted following balance training in group (36).

In the case of age-related dizziness and disequilibrium, balance training performed by healthy seniors (37) and seniors who have suffered a fall (38) has shown to yield positive short-term effects in the form of improved balance. Balance training also reduces the risk of falls in elderly people (39).

Indications

As mentioned above, the functional mechanisms and evaluated effects of physical activity for several dizziness diagnoses are well-documented. Mobility training and physical activity are generally recommended for patients suffering from dizziness. Regardless of the cause, dizziness and disequilibrium often lead to a fear of moving and inactivity and, consequently, less stimulation of the balance system, resulting in a vicious circle of increased dizziness and disequilibrium. Muscular symptoms are also common, for example, around the neck, owing to increased muscle tension and avoidance of head movements during dizzy spells (40).

Prescription

Table 1. Guidelines for prescribing physical activity to patients with dizziness.

Type of activity	Intensity	Duration (min.)	Frequency	Recommended activities
Compensation training (for example in case of acute peripheral vestibular deficits)	To cause some dizziness during training	10–20	Every other hour in the first week, then at least 2 times/day	Quick eye and head movements in reclining, sitting and standing positions and appropriate forms of walking that gradually increase in level of intensity.
Habituation training (e.g. BPPV)*	To cause some dizziness during training	10–20	At least 2 times/day, more if possible	Quick change of positions that causes dizziness, e.g. from sitting to transverse position in bed.
Balance training (in case of reduced balance whatever the cause)	Balance exercises should be challenging	20–60	At least 2 times/week	Standing and walking on different surfaces and for example combined with ball exercises. Group training. Outdoor walks on an uneven ground.
General fitness, strength and mobility training (for example in case of secondary inactivity)	Exercises should be fairly low-intensity	20–60	At least 2 times/week	Group training. Outdoor activities.

* BPPV = Benign Paroxysmal Positional Vertigo.

It is important to remember that mobility training does not rule out the importance of a careful diagnostic evaluation.

Functional mechanisms

In the case of a loss of function in the balance system, the function can be partly recovered through central compensation (7, 41), and the peripheral semicircular canal function relatively often returns within a couple of months (42). Habituation reduces the sensation of dizziness through central adaptive mechanisms, as the patient repeatedly performs movements and position changes that provoke dizziness (20). Training of balance, strength and mobility, etc., results in improved coordination of the sensory, central and musculoskeletal parts of the balance system, facilitating good balance function.

Functional tests

In the case of acute peripheral vestibular loss and injury to the central nervous system, it should be checked whether eye and head movements provoke dizziness, which is a sign that training is needed to achieve compensation and increase the tolerance of movement.

Standing and walking balance tests are used to assess the severity of disequilibrium and decide on a suitable training intensity. Static clinical balance tests measure the patient's ability to maintain his/her balance in different positions with the eyes open and closed. Examples of positions are the Romberg Test, the Tandem Romberg Test, standing on foam rubber, and standing on one leg. These tests have good reliability (43, 44) and are sensitive to age-related changes (45). Examples of a dynamic clinical balance tests are walking forwards and backwards on a line. These tests have been shown in earlier studies to have good reliability (44) and to be sensitive to changes in the assessment of balance (37). The number of incorrect steps is recorded as the study subject walks between two lines in a figure eight (46). This test also has good reliability (44).

BPPV patients prescribed habituation training are asked to perform a body position test according to a maneuver diagram. Since only movements that provoke dizziness yield the desired habituation effects, each patient must be tested and provided with an individually adapted body position training programme. The nature, intensity and duration of the dizziness should be noted and the most provoking movements chosen for the training programme (21).

For age-related dizziness and disequilibrium, functional balance tests such as the Berg Balance Scale (47) and the Timed Get Up and Go Test (48) can be recommended. The Berg Balance Scale together with a description of symptoms provided by the patient have been shown to predict the risk of falls (14).

After training, the dizziness and balance should be re-assessed in line with the above.

Drug therapy

Medications for dizziness usually work to suppress central balance function. It is very seldom that medication is indicated for anything other than for acute treatment of acute peripheral and central vestibular injury, and then only to alleviate nausea due to rotational dizziness. When used, then medications should be discontinued as soon as possible as they have a negative effect on central compensation, which attempts to reduce the degree of dysfunction in the central vestibular neural pathways. Caffeine and amphetamines do, in theory, have a certain positive effect on the compensation mechanisms, but this is not a practical solution to the problem. There is no place for medications in the treatment of BPPV or age-related dizziness. Medications can naturally affect many types of dizziness, for example, through poorly optimised blood pressure or other central circulatory mechanisms. There is currently no medication registered in Sweden that improves the central blood flow. However, drug therapy is very beneficial in the case of Ménière's Disease.

Contraindications (relative)

Certain disabilities or pain situations render it impossible to make head movements or to change body positions with the speed and range of motion needed to achieve the best possible training effect. This requires a careful and individual assessment of the extent to which exercises can be modified and performed.

Risks

In patients with dizziness and disequilibrium, training must always be carried out under safe and secure conditions to avoid injuries from falls, etc.

Acknowledgement

The authors would like to thank Anne-Lise Tamber, Physiotherapist, University Lecturer, PhD Student, Department for Health Professions, Oslo University College, Faculty of Medicine, Oslo University, Norway, for her constructive views and updates.

References

1. Sixt E, Landahl S. Postural disturbances in a 75-year-old population. I. Prevalence and functional consequences. *Age Ageing* 1987;16:393-8.
2. Tamber A-L, Bruusgaard, D. Unpublished results from The Oslo Health Study (HUBRO) (<http://www.fhi.no>).
3. Kroenke K, Hoffman RM, Einstadter D. How common are various causes of dizziness? A critical review. *South Med J* 2000;93:160-7.
4. Tinetti ME, Speechley M, Ginter SF. Risk factors for falls among elderly persons living in the community. *N Engl J Med* 1988;319:1701-7.
5. Jönsson R, Sixt E, Landahl S, Rosenhall U. Balance symptoms in an elderly population. *J Vest Res* 2004;14:47-52.
6. Shumway-Cook A, Wollacott MH. *Motor Control. Theory and practical applications*. 2nd edn. Baltimore: Lippincott Williams & Wilkins; 2001.
7. Curthoys IS, Halmagyi GM. Vestibular compensation. A review of the oculomotor, neural and clinical consequences of unilateral vestibular loss. *J Vestib Res* 1995;5: 67-107.
8. Furman JM, Cass SP. Benign paroxysmal positional vertigo. *N Engl J Med* 1999;341: 1590-6.
9. Brandt T, Dieterich M, Strupp M. *Vertigo and dizziness. Common complaints*. Munich: Springer; 2005.
10. Furman JM, Whitney SL. Central causes of dizziness. *Phys Ther* 2000;80:179-87.
11. Matheson AJ, Darlington CL, Smith PF. Further evidence for age-related deficits in human postural function. *J Vestib Res* 1999;9:261-4.
12. Konrad HR, Girardi M, Helfert R. Balance and aging. *Laryngoscope* 1999;109: 1454-60.
13. Strupp M, Arbusow V. Acute vestibulopathy. *Curr Opin Neurol* 2001;14:11-20.
14. Shumway-Cook A, Baldwin M, Polissar NL. Predicting the probability for falls in community-dwelling older adults. *Phys Ther* 1997;77:812-9.
15. Sattin RW, Lambert Huber DA, DeVito CA, Rodriguez JG, Ros A, Bacchelli S, et al. The incidence of fall injury events among the elderly in a defined population. *Am J Epidemiol* 1990;131:1028-37.
16. Fife TD, Tusa RJ, Furman JM, Zee DS, Frohman E, Baloh RW, et al. Assessment: Vestibular testing techniques in adults and children. Report of the Therapeutics and technology assessment subcommittee of the American Academy of Neurology. *Neurology* 2000;55:1431-41.
17. Norré ME. Diagnostic problems in patients with benign paroxysmal positional vertigo. *Laryngoscope* 1994;104:1385-8.
18. Baloh RW. Differentiating between peripheral and central causes of vertigo. *Otolaryngol Head Neck Surg* 1998;119:55-9.
19. Herdman SJ. *Vestibular rehabilitation*. 2nd edn. Philadelphia: FA Davis Co; 2000.

20. Herdman SJ, Blatt PJ, Schubert MC. Vestibular rehabilitation of patients with vestibular hypofunction or with benign paroxysmal positional vertigo. *Curr Opin Neurol* 2000;13:39-43.
21. Norré ME, Beckers AM. Vestibular habituation training. *Arch Otolaryngol Head Neck Surg* 1988;114:883-6.
22. Epley JM. Particle repositioning for benign paroxysmal positional vertigo. *Otolaryngol Clin North Am* 1996;29:323-31.
23. Chang WC, Hsu LC, Yang YR, Wang RY. Balance ability in patients with benign paroxysmal positional vertigo. *Otolaryngol Head Neck Surg* 2006;135:534-40.
24. Kollén L, Bjerlemo B, Möller C. Evaluation of treatment in benign paroxysmal positional vertigo. *Advances in Physiotherapy* 2006;8:106-15.
25. Herdman SJ, Clendaniel RA, Mattox DE, Holliday MJ, Niparko JK. Vestibular adaptation exercises and recovery. Acute stage after acoustic neuroma resection. *Otolaryngol Head Neck Surg* 1995;113:77-87.
26. Mruzek M, Barin K, Nichols DS, Burnett CN, Welling DB. Effects of vestibular rehabilitation and social reinforcement on recovery following ablative vestibular surgery. *Laryngoscope* 1995;105:686-92.
27. Whitney SL, Metzinger Rossi MM. Efficacy of vestibular rehabilitation. *Otolaryngol Clin North Am* 2000;33:659-72.
28. Krebs DE, Gill-Body KM, Parker SW, Ramirez JV, Wernick-Robinson M. Vestibular rehabilitation. Useful but not universally so. *Otolaryngol Head Neck Surg* 2003;128:240-50.
29. McGibbon CA, Krebs DE, Wolf SL, Wayne PM, Scarborough DM, Parker SW. Tai Chi and vestibular rehabilitation effects on gaze and whole body stability. *J Vest Res* 2004;14:467-78.
30. Topuz O, Topuz B, Ardic FN, Sarhus M, gmen G, Ardic F. Efficacy of vestibular rehabilitation on chronic unilateral vestibular dysfunction. *Clin Rehabil* 2004;18:76-83.
31. Meli A, Zimatore G, Badaracco C, De Angelis E, Tufarelli D. Vestibular rehabilitation and 6-month follow-up using objective and subjective measures. *Acta Otolaryngol* 2006;126:259-66.
32. Hillier SL, Hollohan V, Hilton MP. Vestibular rehabilitation for unilateral peripheral vestibular dysfunction. Protocol. *Cochrane Database of Systematic Reviews* 2005, Issue 3. Art No CD005397. DOI10.1002/14651858.CD005397.
33. White J, Savvides P, Cherian N, Oas J. Canalith repositioning for benign paroxysmal positional vertigo. *Otol Neurotol* 2005;26:704-10.
34. Woodworth BA, Gillespie MB, Lambert PR. The canalith repositioning procedure for benign positional vertigo. A meta-analysis. *Laryngoscope* 2004;114:1143-6.
35. Hilton M, Pinder D. The Epley (canalith repositioning) manoeuvre for benign paroxysmal positional vertigo. *Cochrane Database of Systematic Reviews* 2004, Issue 2. Art No CD003162. DOI 10.1002/14651858.CD003162.
36. Kammerlind AC, Håkansson JK, Skogsberg MC. Effects of balance training in elderly people with nonperipheral vertigo and unsteadiness. *Clin Rehabil* 2001;15:463-70.

37. Ledin T, Kronhed AC, Möller C, Möller M, dkvist LM, Olsson B. Effects of balance training in elderly evaluated by clinical tests and dynamic posturography. *J Vestib Res* 1991;1:129-38.
38. Shumway-Cook A, Gruber W, Baldwin M, Liao S. The effect of multidimensional exercises on balance, mobility, and fall risk in community-dwelling older adults. *Phys Ther* 1997;77:46-57.
39. Gillespie LD, Gillespie WJ, Robertson MC, Lamb SE, Cumming RG, Rowe BH. Interventions for preventing falls in elderly people. *Cochrane Database of Systematic Reviews* 2003, Issue 4. Art No CD000340. DOI 10.1002/14651858.CD000340.
40. Luxon LM. Vertigo. New approaches to diagnosis and management. *Br J Hosp Med* 1996;56:519-20, 537-41.
41. Brandt T, Strupp M, Arbusow V, Dieringer N. Plasticity of the vestibular system. Central compensation and sensory substitution for vestibular deficits. In: Freund HJ, Sabel BA, Witte OW, red. *Brain plasticity. Adv Neurol* 1997;73:297-309.
42. Allum JHJ, Ledin T. Recovery of vestibulo-ocular reflex-function in subjects with an acute unilateral peripheral vestibular deficit. *J Vest Res* 1999;9:135-44.
43. Franchignoni F, Tesio L, Martino MT, Ricupero C. Reliability of four simple, quantitative tests of balance and mobility in healthy elderly females. *Aging (Milano)* 1998;10:26-31.
44. Kammerlind A, Bergquist Larsson P, Ledin T, Skargren E. Reliability of clinical tests and subjective ratings in dizziness and disequilibrium. *Advances in Physiotherapy* 2005;7:96-107.
45. Briggs RC, Gossman MR, Birch R, Drews JE, Shaddeau SA. Balance performance among noninstitutionalized elderly women. *Phys Ther* 1989;69:748-56.
46. Frändin K, Sonn U, Svantesson U, Grimby G. Functional balance tests in 76-year-olds in relation to performance, activities of daily living and platform tests. *Scand J Rehabil Med* 1995;27:231-41.
47. Lundin Olsson L, Jensen J, Waling K. Bergs balansskala, den svenska versionen av The Balance Scale [The Berg Balance Scale, the Swedish version of The Balance Scale] *Vetenskapligt Supplement* 1996;1:16-9. [Scientific Research Supplement]
48. Mathias S, Nayak US, Isaacs B. Balance in elderly patients. The "Get-Up-and-Go" Test. *Arch Phys Med Rehabil* 1986;67:387-9.

28. *Gastrointestinal diseases*

Author

Per M. Hellström, MD, PhD, Professor, Department of Medical Sciences, Gastroenterology/Hepatology, Uppsala University, Uppsala, Sweden

Introduction

Symptoms from the gastrointestinal tract are common with vigorous physical activity and training. It is estimated that approximately half of everyone who participated in endurance sports experience symptoms in the form of nausea, vomiting, abdominal pain, bloating, diarrhoea, stomach cramps, or even blood in the stool.

The gastrointestinal tract is thus not an “athletic organ” that adapts to increased levels of physical and mental stress. The intestine’s most important task is to take up nutrients during the body’s resting periods. During physical activity, the intestine can be thrown off course and lose its normal function. For this reason, proper nutrition and a nutritional balance during rest is considered fundamental for effective exercise training and intense competition. Balanced nutrition (nutrient intake) is fundamental for preventing symptoms from the gastrointestinal tract. It can also prevent unexpected gastrointestinal symptoms caused by vigorous training, which can reduce physical capacity.

There are several conditions of the gastrointestinal tract that can be exacerbated by exercise training, and where the symptoms of gastrointestinal disease may be expressed. This applies in particular to the inflammatory intestinal diseases of Crohn’s disease and ulcerative colitis, in which the catabolic disease with increased demands for extra energy, tissue healing and immunological defense mechanisms does not permit further load on the organism through an increased exercise-induced physiological demand on metabolism. A summary of these conditions is given at the end of the chapter.

The collective knowledge today suggests that a small number of functional conditions of the gastrointestinal tract can be improved through exercise training. This applies above all to diseases of the colon (large intestine) and in particular to the concept of constipation (obstipation). The symptom picture for such diseases is not only affected by the organism’s physical fitness, but also by psychological and mental factors, which complicate the picture for diseases of the gastrointestinal tract.

Constipation (obstipation)

Summary

Running and power walking stimulate the intestinal motor function in constipation. The body's movements thereby provide mechanical stimulation of the gastrointestinal tract, which increases passage of the contents from the large intestine to the rectum during motion. When the rectum becomes full, the defecation reflex is induced. During physical work, blood flow is steered away from the intestine to the working muscles. The reduced blood flow leads to a deficiency of oxygen in the tissue, which can lead to the release of several different hormones from the gastrointestinal tract, which in turn stimulate functions of the gastrointestinal tract. A cornerstone in the treatment of constipation is regular exercise together with a high fibre content in the diet (20–30 g per day) and, naturally, going to the toilet when the urge to empty the bowel occurs.

Definition of the concept

The concept of constipation/obstipation means different things to different individuals. The stool can be too small, too hard, too difficult to pass, occur too seldom, and some also experience incomplete emptying of the rectum after a bowel movement. An estimated 2 per cent of the population of working age have problems with constipation and the condition is six times more common in women than in men. In old age, it is estimated that constipation occurs in a third of the population.

Normal stool weight is 35–225 g per bowel movement. Low stool weight indicates constipation. There are also significant geographical differences. In Northern Europe and the USA, stool weight is normally 100–200 g per bowel movement, while in India, the average is 311 g, and in Uganda 470 g.

The frequency of bowel movements provides the most practical measure of the degree of constipation. Several studies show that people in Northern Europe and the USA have at least three bowel movements per week. Even here there are large geographical variations, with higher bowel movement frequency with higher stool weights.

The cause of chronic constipation is unknown. The condition is most common in women and, as a rule, onset begins in puberty and problems become worse after childbirth. Investigations of the intestinal motor function have shown a reduced response of motor stimulating gastrointestinal hormones (gastrin, cholecystokinin, motilin) released after meals. The tissue in the large intestine also shows low levels of the neurotransmitters, substance P (SubP), vasoactive intestinal peptide (VIP) and motilin.

The symptoms, mainly abdominal pain and distension, develop steadily between bowel movements. The intervals between bowel movements also become longer and can at worst be up to about a month. Upon examination, one finds no deviating pathological findings, and the abdomen is soft and not tender. The rectum is, paradoxically enough, often empty.

The condition is diagnosed through the patient giving his or her disease history, where fewer than three bowel movements per week is considered to be diagnostic. The diagnosis

can be further confirmed by a transit examination with the aid of radiopaque markers. The markers then collect in the large intestine and the transit time can be calculated.

Most patients can usually manage the treatment on their own by taking large amounts of laxatives or daily enemas. Temporary stimulation of increased bowel movements can be achieved with stimulant laxatives (Pursennid, Dulcolax, Microlax) and also water enemas. In the long term, however, these drugs make the constipation worse. For long-term use, lactulose combined with a fibre supplement in the diet is recommended, which is considered to yield the best results. The fibre supplement can in some cases exacerbate the condition by increasing the intestinal volume load and production of gas. Fibre supplementation should therefore occur successively, to achieve a target level of 20–30 g per day.

Effect of physical activity

Acute effects

Past experience suggests that exercise training stimulates colon motor function and improves the condition in constipation. Urges to empty the bowel that arise in connection with intensive physical activity and training are likely a direct effect on the colon. Animal trials have shown that stimulation of the sciatic nerve gives rise to increased blood flow and increased motor function of the colon (1). The sympathicotonic stimulation during exercise yields a relaxation and inhibition of the gastrointestinal tract's functions in humans (2). Experience also suggests that long-distance runners experience urges relatively late during training (after approx. 30–40 minutes), which suggests that nerve activity has significance for the intestinal effect. Running, and to a lesser extent cycling and swimming, lead to dramatic pressure changes in the abdominal cavity (3). During high intensity work, the diaphragm presses down against the abdominal organs, which is believed to be the cause of the sensation of “stitches” (4)

Exercise training has large effects on the blood flow in the intestine. The blood flow is also affected considerably more during aerobic (submaximal) training in untrained than in well-trained individuals (5). This also has effects on the release of several hormones and neurotransmitters in the intestine. VIP and the neurotransmitter-like peptide histidine-methionine (PHM) is released in large amounts from intestinal tissue when blood flow to the organ is cut off. Mechanical stimulation of the gastrointestinal tract can also cause a release of VIP. These neurotransmitters reduce the sodium uptake in the intestine and increase secretion, which can lead to watery diarrhoea (6). Other hormones, such as secretin, glucagon, gastric inhibitory peptide (GIP) and prostaglandines, are also released during intensive, long-lasting training, and all of which can lead to fluid secretion from the small intestine and thereby also influence the colon. During exercise training, insulin levels in the blood stream drop, while another hormone, pancreatic peptide (PP), rises, which helps to relax the colon (7, 8). The internal anal sphincter can also be relaxed by both VIP and PHM, which can rise to very high levels during physical work combined with lack of fluid and dehydration (9).

Together, these effects explain why, above all, running, which entails a combination of blood flow effects and mechanical stress on the gastrointestinal tract, leads to a greater

release of neurotransmitters and more gastrointestinal symptoms than cycling and swimming. Long-distance walking can give symptoms such as increased intestinal motor function and flatulence, but yields fewer symptoms in general due to the lower work intensity (10). To this can be added that mental stress in itself can increase intestinal motor function (11), but this should be limited to the actual competition situation.

Long-term effects

Many long-distance runners experience symptoms from the colon, with diarrhoea, cramp-like abdominal pain or increased problems with gas, as well as the urge to empty the bowel during or immediately after running. Our knowledge on the exact relations of this are limited. In one study, it was shown that 30 minutes of aerobic training, 3 times a week for a total of 6 weeks, at a level corresponding to 70–80 per cent of maximum exertion, shortened intestinal transit time from 35 to 24 hours, while the transit time for the control group remained unchanged (2). Experience from treatment of patients with constipation also suggests that physical activity, above all power walking and running, had a good effect on constipation. To a lesser extent other sports such as cycling and swimming stimulate intestinal function even in the long term (compare the “after-dinner stroll”).

Experience from long-distance running also suggests that, above all, young women suffer from gastrointestinal symptoms with diarrhoea and the urge to empty their bowel (12). This has been observed in a number of studies and must be considered an established concept today in connection with strenuous physical exertion and competition.

A determining factor in this context is that increased exercise is often tantamount to a complete change in lifestyle and should therefore be seen as a change over a very long time.

When the treatment is to begin, the patient must be prepared for some increase in abdominal symptoms, particularly an increased sense of pressure, which may even be experienced as painful. This can nevertheless be seen as a first sign of a successful treatment outcome and should lead to further encouragement in efforts to a more movement-oriented lifestyle.

Indications

Constipation as a condition can be treated primarily with increased physical activity.

Prescription

In the first place, the recommendation is running at a submaximal level (pulse just over 110) for 30 minutes every other day. This should not be seen as a one-time treatment, but as a change in lifestyle pattern for a very long time to come. In the second place, power walking at a corresponding level of exertion can be recommended, but is evidently not as effective.

The first step in the treatment of constipation thus involves regular exercise and a diet high in fibre (20–30 g per day) along with the individual’s own sensitivity to natural signals and the urge to empty the bowel.

Functional mechanisms

Increased release of neurotransmitters in the intestine, which can stimulate a reduced transit time and emptying of the bowel.

Functional tests

The simplest measure of effects on constipation is to keep a record of bowel movement habits. No further follow-up besides this is needed.

Interactions with drug therapy

No known interactions.

Contraindications

Absolute contraindications include acute myocardial infarction and ongoing asthma attack. Relative contraindications include exercise-induced asthma.

Risks

None. If bleeding from the bowel should occur, the patient should contact a physician.

The gastrointestinal tract and metabolic diseases

Absorption of nutrients from the gastrointestinal tract occurs in the small intestine first. The gastric emptying speed is therefore decisive in metabolic control and blood sugar balance. Absorption of sugar and fat must be counter-regulated by insulin in order to not reach injuriously high levels. In cases of a reduced capacity to release insulin in sufficient amounts (adult onset diabetes) and insulin resistance (overweight, pre-diabetes, adult onset diabetes), the gastric emptying speed can be decisive for sugar control. Already during physiological conditions, cycling at a submaximal level (70% of maximal capacity) can affect the pressure relations in the stomach and increase emptying (13), which increases the absorption of nutrients from the small intestine.

In diabetes, gastrointestinal tract activity and emptying of the stomach have obvious significance for metabolic control, above all through the lack of insulin to keep the blood sugar down. In diabetes, emptying of the stomach can be slow. It has been found that 30 minutes of walking after meals improves gastric emptying and metabolic control in many diabetics (14). One should, however, keep in mind that intensive physical activity can dramatically lower the blood sugar and even lead to hypoglycemia (blood sugar that is so low that it impacts general health) in these individuals.

Other gastrointestinal diseases

Diseases of the gastrointestinal tract often involve catabolic conditions where the patient is not able to take advantage of the nutrients needed to maintain a normal nutritional balance. In such circumstances, it is reasonable to recommend exercise training for the particular individual. There are several conditions where physical activity is considered to exacerbate and even induce symptoms of disease. The following gives a presentation of which diseases this applies to and the underlying causes behind such recommendations.

Gastro-oesophageal reflux

Chest pain from the oesophagus can be induced in connection with exertion. Gastro-oesophageal reflux, that is, inflammation of the oesophagus caused by a backward flow (reflux) of the contents of the stomach, is a common symptom that can arise with motility disturbances in the oesophagus. Gastro-oesophageal reflux often presents in connection with physical activity, particularly with symptoms in the form of “heartburn” (acid reflux), but also with chest pain (12, 13). This applies to running, cycling, strength training and various fighting sports. In the first case, the cause of this is likely constant dislocations of the gastrointestinal tract during the impact of running, which can lead to mechanical leakage. In the second case, increased abdominal pressure likely occurs, which pushes the contents of the stomach up into the oesophagus thus giving reflux symptoms. The symptoms are amplified in physical activity after meals (15–17).

Distinguishing between symptoms that precipitate from the oesophagus and angina pectoris is difficult. A suspicion of coronary insufficiency (insufficient blood flow in the coronary arteries) during physical exertion is very important, because angina pectoris directly affects the heart’s vital functions. Here, the attending physician must always keep in mind that chest pain coming from the oesophagus can also occur with coronary insufficiency and that reflux can exacerbate myocardial ischaemia. Patients with known coronary insufficiency have been shown to give a positive Bernstein test with acid in the oesophagus and in certain patients even ECG changes as with myocardial ischaemia (18, 19). It has also been described that gastro-oesophageal reflux is more common during physical exertion in individuals with known coronary insufficiency (20, 21). The clinical importance of symptoms from the oesophagus in connection with exertion lies in the danger zone for incorrect assessment of a clinically significant coronary insufficiency with myocardial ischaemia during exertion. Reflux symptoms can, however, be prevented through temporary drug therapy with an H₂-receptor antagonist (ranitidine) (12), or, in more pronounced cases, with a proton pump inhibitor (omeprazole).

Gastrointestinal bleeding

Bleeding from the gastrointestinal tract, usually in the form of occult bleeding, is seen in 8–22 per cent of all marathon runners (22). Usually the source of bleeding is localised to the stomach or more seldomly to the colon. The background to this finding is a

dramatically reduced blood flow to the area that supplies blood to the gastrointestinal tract (splanchnicus). During running, the blood flow to the gastrointestinal tract decreases by 80 per cent and has been considered a local state of shock (23). This affects the perfusion of blood in the mucous lining in particular, since 90 per cent of the blood flow to the gastrointestinal tract during rest goes to the mucous membrane (24). It then ceases to function with a normal exchange of salts and fluid between the intestine and the body's inner environment. An interesting finding is that the source of bleeding in the stomach is usually localised to the mid corpus region where the gastric acid is produced, while the lower antrum is rarely affected. In these cases, gastroscopy reveals a haemorrhagic gastritis. It has been speculated that there is a similar originating mechanism for these membrane injuries as for "stress ulcers", which are seen in intensive care cases with multiple trauma. Exercise-induced gastrointestinal bleeding appears to be related to the degree of exertion. It occurs more frequently in young athletes upon vigorous exertion than at lower levels of exertion (25–28). The bleeding can be prevented through treatment with H₂-receptor antagonists (22, 29) or proton pump inhibitors.

Strenuous training often leads to low blood haemoglobin levels. The reason for this is in part a dilution of the blood through the increase in blood volume in athletes, and in part that blood cells are destroyed, something seen primarily in long-distance runners. This has been assumed to be due to trauma to the blood cells in the soles of the feet.

Bleeding from the gastrointestinal tract is thus not an uncommon finding after a strenuous physical exertion. It must in the first place be seen as a transient natural reaction and does not require special investigation if the condition subsides within about a week. In the case of known ulcer disease, however, strenuous physical training, especially long-distance running or marathon races, are not recommended because this can theoretically lead to a bleeding episode, which always involves a significant medical risk.

Medication in the form of non-steroidal anti-inflammatory drugs (NSAIDs) should absolutely be avoided if bleeding occurs, since all drugs of this type (ibuprofen, naproxen, diclofenac, sulindac, ketoprofen, etc.) as well as acetylsalicylic acid increase the risk of bleeding.

Inflammatory bowel disease

The extreme metabolic stress that an individual undergoes when developing inflammatory bowel disease is significant and places demands on increased nutritional supplementation for all of the body's cellular energy processes, healing and immunological defense functions. If the organism is unable to take advantage of these demands, a situation of reduced physical work capacity, increased glycogen and protein breakdown and impaired immune defenses arises (30, 31).

Strenuous exercise and a general inflammatory response demonstrate many similarities and in many cases activate the same biological mediators (32). In the case of cell disintegration of the intestinal lining with biochemical and mechanical stress, disturbances in the gastrointestinal tract's barrier functions occur, which can lead to an increased uptake of bacterial endotoxins and other toxic contaminants. In athletes, one sees a similar increase

in the uptake of endotoxins in the intestine after strenuous exercise (33, 34). Secretion of IgA in the intestine also decreases during physical activity, which can weaken the immune system. (35).

In strenuous exercise, the uptake of endotoxins from bacteria can lead to a classic inflammatory response, with an increase in the number of white blood cells in the blood (leukocytosis) and increased levels of cytokines, such as tumour necrosis factor α (TNF α), interleukin-1 and interleukin-6 in the blood stream (36–38). It has been shown that 80 per cent of runners with endotoxins in the blood have symptoms of nausea, vomiting and diarrhoea, which can be compared to 18 per cent in the control group without endotoxins (32). An interesting observation here is that long-distance runners develop high levels of the protective blood lipid fraction HDL, which can bind endotoxins in the blood stream and thereby serve as protection (39, 40). A new study has also found that the quality of life in Crohn's disease improves if the patients engage in physical activity. Exercise can reduce the disease activity and the psychological stress of the disease. Low intensity exercise gives no deterioration of the symptom picture or activation of the disease and can, from a psychological perspective, be good for certain patient groups with inflammatory bowel disease (41).

Our knowledge thus indicates primarily a negative effect from strenuous exercise for inflammatory conditions in the gastrointestinal tract. This conclusion is based primarily on the fundamental negative metabolic consequences resulting from the combination of inflammation and exercise training. Low intensity training does not exacerbate the condition and can be beneficial from a psychological standpoint.

A special case in this context is the well-known fact that, through travelling to competitions around the world, athletes are often exposed to the risk of gastrointestinal infection. Similarly to the case of inflammatory bowel disease, this means that the body is not able to take advantage of the nutrition absolutely required during the building up phase, making top performance impossible (42).

Stitch pain

The symptom of stitch pain is usually located on the left side (compare “side stitch” or “side ache”). This condition is clearly related to exercise and occurs more often at the beginning of a training period and in intensive training. The condition has no defined cause. Usually it subsides with a successive increase in the amount of exercise and is not a reason to avoid exercise training (12).

Final remarks

Despite lacking “athletic” characteristics, the function of the gastrointestinal tract has decisive importance for how an athletic activity should be planned and carried out. Gastrointestinal tract functions at rest play a role in building up the body and thereby the optimisation of nutritional status leading up to athletic performance. This also applies in disease conditions, where rest is important for achieving the best conditions for healing

and health. Our knowledge about these relations can often be utilised to optimise nutritional conditions in disease, for example, in diabetes. In reasonable amounts, physical activity has a positive effect on many gastrointestinal tract conditions by helping to normalise physiological control.

References

1. Hultén L. Extrinsic nervous control of colonic motility and blood flow. An experimental study in the cat. *Acta Physiol Scand Suppl* 1969;335:1-116.
2. Cordain L, Latin RW, Behnke JJ. The effects of an aerobic running program on bowel transit time. *J Sports Med Phys Fitness* 1986;26:101-4.
3. Rehrer NJ, Meijer GA. Biomechanical vibration of the abdominal region during running and bicycling. *J Sports Med Phys Fitness* 1991;31:231-4.
4. Porter AMW. Marathon running and the cecal slap syndrome. *Br J Sports Med* 1982;16:178.
5. Clausen JP. Effective physical training on cardiovascular adjustment to exercise in man. *Physiol Rev* 1977;57:779-815.
6. Rehrer NJ, Beckers EJ, Brouns F, Saris WH, Ten Hoor F. Effects of electrolytes in carbohydrate beverages on gastric emptying and secretion. *Med Sci Sports Exerc* 1993;25:42-51.
7. Galbo H. Gastro-entero-pancreatic hormones. In: Galbo H, Ed. *Hormones and Metabolic Adaptation to Exercise*. New York: Thieme; 1983, pp. 59-61.
8. Tach Y. Nature and biological actions of gastrointestinal peptides. Current status. *Clin Biochem* 1984;17:77-81.
9. Brouns F. Etiology of gastrointestinal disturbances during endurance events. *Scand J Med Sci Sports* 1991;1:66-77.
10. Peters HP, Zweers M, Backx FJ, Bol E, Hendriks ER, Mosterd WL, et al. Gastrointestinal symptoms during long-distance walking. *Med Sci Sports Exerc* 1999; 31:767-73.
11. Dill JE. CNS and gut responses to stress. Which comes first? *Am J Gastroenterol* 1996;91:1292.
12. Moses FM. The effect of exercise on the gastrointestinal tract. *Sports Med* 1990;9: 159-72.
13. van Nieuwenhoven MA, Brouns F, Brummer RJ. The effect of physical exercise on parameters of gastrointestinal function. *Neurogastroenterol Motil* 1999;11:431-9.
14. Lipp RW, Schnedl WJ, Hammer HF, Kotanko P, Leb G, Krejs GJ. Effects of postprandial walking on delayed gastric emptying and intragastric meal distribution in long-standing diabetics. *Am J Gastroenterol* 2000;95:419-24.
15. Neuffer PD, Young AJ, Sawka MN. Gastric emptying during exercise. Effects of heat stress and hypohydration. *Eur J Appl Physiol* 1989;58:433-9.
16. Rehrer NJ. Aspects of dehydration and rehydration during exercise. In: Brouns F, Ed. *Advances in Nutrition and Top Sport. Medicine and sport science*. Basel: Karger; 1991, pp. 128-46.
17. Rehrer NJ, Beckers EJ, Brouns F, Saris WH, Ten Hoor F. Effects of electrolytes in carbohydrate beverages on gastric emptying and secretion. *Med Sci Sports Exerc* 1993;25:42-51.

18. Mellow MH, Simpson AG, Watt L, Schoolmeester L, Haye O. Esophageal acid perfusion in coronary artery disease. *Gastroenterology* 1983;85:306-12.
19. Schofield PM, Bennett DH, Whorwell PJ, Brooks NH, Bray CL, Ward C, et al. Exertional gastro-oesophageal reflux. A mechanism for symptoms in patients with angina pectoris and normal coronary angiograms. *Br Med J* 1987;294:1459-61.
20. Johnston P, O'Connor B, Lennon JR, Crowe J. A comparative evaluation of bicycled exercise testing versus endoscopy plus 24 hour oesophageal pH monitoring in the diagnosis of gastrooesophageal reflux. *Gastroenterology* 1987;92:A1457.
21. O'Connor BJ, Johnston P, Lennon JR, Crowe J. The effect of exercise on post prandial acid reflux in oesophagitis and coronary artery disease. *Gastroenterology* 1987;92:A1457.
22. Moses FM. Gastrointestinal bleeding and the athlete. *Am J Gastroenterol* 1993;88:1157-9.
23. Sanchez LD, Corwell B, Berkoff D. Medical problems of marathon runners. *Am J Emerg Med* 2006;24:608-15.
24. Granger DN, Richardson PD, Kvietyts PR, Mortillaro NA. Intestinal blood flow. *Gastroenterology* 1980;78:837-63.
25. McMahon LF Jr, Ryan MJ, Larson D, Fisher RL. Occult gastrointestinal blood loss in marathon runners. *Ann Intern Med* 1984;101:847-7.
26. Buckman MT. Gastrointestinal bleeding in long-distance runners. *Ann Intern Med* 1984;101:127-8.
27. Cantwell JD. Gastrointestinal disorders in runners. *JAMA* 1981;246:1404-5.
28. Fogoros RN. Runner's trots. Gastrointestinal disturbances in runners. *JAMA* 1908;243:1743-4.
29. Cooper BT, Douglas SA, Firth LA, Hannagan JA, Chadwick VS. Erosive gastritis and gastrointestinal bleeding in a female runner. Prevention of the bleeding and healing of the gastritis with H₂-receptor antagonists. *Gastroenterology* 1987;92:2019-23.
30. Mainous MR, Block EF, Deitch EA. Nutritional support of the gut. How and why. *New horiz* 1994;2:193-201.
31. Mainous MR, Ertel W, Chaudry IH, Deitch EA. The gut. A cytokine-generating organ in systemic inflammation? *Shock* 1995;4:193-9.
32. Camus G, Deby-Dupont G, Duchateau J, Deby C, Pincemail J, Lamy M. Are similar inflammatory factors involved in strenuous exercise and sepsis? *Int Care Med* 1994;20:602-10.
33. Bosenberg AT, Brock-Utne LG, Gaffin SL, Wells MTB, Blake GTW. Strenuous exercise causes systemic endotoxemia. *J Appl Physiol* 1988;65:106-8.
34. Brock-Utne JG, Gaffin SL, Wells MT, Gathiram P, Sohar E, James MF, et al. Endotoxaemia in exhausted runners after long-distance race. *S Afr Med J* 1988;73:533-6.
35. Mestecky J, Russell MW, Elson CO. Intestinal IgA. Novel views on its function in the defence of the largest mucosal surface. *Gut* 1999;44:2-5.
36. Northoff H, Berg A. Immunologic mediators as parameters of the reaction to strenuous exercise. *Int J Sports Med* 1991;14:S9-15.

37. Northoff H, Weinstock C, Berg A. The cytokine response to strenuous exercise. *Int J Sports Med* 1994;15:S167-71.
38. Sprenger H, Jacobs C, Nain M, Gressner AM, Prinz H, Wesemann W, et al. Enhanced release of cytokines, interleukin-2-receptors, and neopterin after long distance running. *Clin Immunol Immunopathol* 1992;63:188-95.
39. Flegel WA, Baumstark MW, Weinstock C, Berg A, Northoff H. Prevention of endotoxin-induced monokine release by human low- and high-density lipoproteins and by apolipoprotein A-1. *Infect Immun* 1993;61:5140-6.
40. Maier RV. Interactions of bacterial lipopolysaccharides with tissue macrophages and plasma lipoproteins. In: Maier RV, Ed. *Pathophysiological Effects of Endotoxins at the Cellular Level*. New York: Alan R Liss; 1981, pp. 135-55.
41. Ng V, Millard W, Lebrun C, Howard J. Exercise and Crohn's disease. Speculations on potential benefits. *Can J Gastroenterol* 2006;20:657-60.
42. Karageanes SJ. Gastrointestinal infections in the athlete. *Clin Sports Med* 2007;26:433-48.

29. Heart failure

Authors

Åsa Cider, PT, PhD, Physiotherapy Department, Sahlgrenska University Hospital, Gothenburg, Sweden

Rajja Tyni-Lenné, PT, PhD, Associate Professor, Department of Physical Therapy, Karolinska University Hospital, Stockholm, Sweden

Maria Schaufelberger, MD, PhD, Associate Professor, Sahlgrenska University Hospital, Gothenburg, Sweden

Summary

Chronic heart failure has a multifactorial origin and reduced physical performance capacity is common in those who suffer from it. Changes in the peripheral blood circulation are likely just as important as left ventricular function in the reduction of physical performance capacity. Research from the past decade has shown that exercise clearly improves function in patients with heart failure, which is why exercise should be offered to these patients today as a part of treatment.

The table below gives a description of training methods with the levels of intensity, frequency and duration that have been scientifically studied. In day-to-day clinical work, we strive for a combination of aerobic central circulatory training and peripheral muscle training, 2–3 times per week, together with home training.

Table 1. Description of training methods investigated in different scientific studies in patients with chronic heart failure.

Training method	Intensity	RPE*	Frequency	Duration (min./session)
Aerobic central circulatory training	(40) 60–80% of VO ₂ max**	11–15 central	1 time/week to several times/day	1–60
Peripheral muscle training	35–80% of 1 RM***	13–15 local	2 times/week to 1 time/day	15–60
Combination of aerobic central circulatory training and peripheral muscle training	60–80% of VO ₂ max** 60–80% of 1 RM	13–15 central and local	3 times/week	45–60
Hydrotherapy	40–80% of HRR****	11–15	3 times/week	45
Respiratory muscle training	30% of max. inspiratory pressure		3 times/week to daily	30–60

* RPE = Rating of Perceived Exertion (Borg scale 6–20).
 ** VO₂ max = Maximal Oxygen Uptake.
 *** RM = Repetition Maximum. 1 RM corresponds to the maximum weight that can be lifted through the entire exercise movement one time.
 **** HRR = Heart Rate Reserve.

Suitable activities are aerobic exercise, for example, walking, exercising on land or in water, and cycling combined with peripheral muscle training with, for example, resistance bands (Thera-Band®) or weights and exercise pulleys. The latter form of training is especially suited in cases of pronounced impairment of physical performance capacity. During the exercise session, light to moderate exertion and light to moderate breathlessness are suitable levels of load.

Patients with stable heart failure with predominant systolic dysfunction, New York Heart Association (NYHA) class (I)-II-III, are best suited for exercise. Before training begins, there should be contact with a knowledgeable physiotherapist so that the patient can undergo aerobic and muscular physical function testing. Based on the outcomes of this, a suitable training programme is then drawn up.

Definition

The syndrome of heart failure has been defined in a number of ways, but no one definition is complete. A common definition is: Heart failure is a condition where the heart’s pumping capacity is unable to meet the metabolic needs of the peripheral tissues (1).

Cause

In the Western world, ischaemic heart disease and hypertension (elevated blood pressure) are predominant causes of heart failure (2). Other etiological factors include diabetes, valvular disease, endocrine disorders, toxic effect, arrhythmia and systemic disease. There is also an idiopathic type, that is, where the cause is not known (1).

Prevalence/Incidence

The prevalence of heart failure varies between 0.3–2 per cent according to different sources and rises with increasing age, to approximately 10 per cent at 80 years of age (3, 4). The incidence of heart failure is also age-dependent and varies between 1–5 new cases per 1000 individuals per year (2). A Swedish registry study indicates that the incidence of heart failure is decreasing, in part due to improved medical care (5).

Pathophysiology

Loss of myocardial cells, changes in heart muscle tissue and contractility reduce the resting stroke volume. This is compensated for by activation of the neuroendocrine system through increased sympathetic system activity and activation of the renin-angiotensin system. This leads to a pathological remodeling in both the heart and the peripheral vascular system, progressive weakening of arterial and cardiopulmonary baroreflexes and a down-regulation of beta receptors in the heart. The parasympathetic heart activity is also reduced (1). The cause of the reduced exercise capacity in heart failure is complex. Haemodynamic variables at rest correlate poorly to maximal exercise capacity (6), while the correlation during exercise is better (7). It has been proposed that diastolic dysfunction may be more important for exercise capacity than systolic dysfunction (8). However, it is likely factors in the skeletal muscle that have the most significance for the reduction in exercise capacity (9). Peripheral blood flow is decreased due to reduced cardiac output and increased peripheral vascular resistance, which is mediated via changes in endothelial function and directly via vasoactive agents (10–12). Insulin resistance is reduced (13) in chronic heart failure and peripheral skeletal muscle mass is reduced (14, 15). Skeletal muscle strength and endurance are also reduced (16, 17). A change in muscle fibre composition and reduced oxidative capacity in skeletal muscle have also been reported. It moreover appears that inflammatory cytokines play an important role in loss of skeletal muscle mass and fatigue in chronic heart failure (18, 19). Skeletal muscle receptors that are sensitive to exercise, that is, ergoreceptors, are overactive. One of the tasks of these receptors is to control vascular constriction, which can be affected by exercise (20, 21). Whether these changes are contingent on physical inactivity or are a consequence of the heart failure syndrome itself is a topic of discussion (9, 22).

Symptoms and diagnosis

The diagnostic criteria recommended by the Task Force on Heart Failure of the European Society of Cardiology (23) are as follows:

1. Symptoms consistent with heart failure at rest or during exercise.
2. Objective evidence of cardiac dysfunction at rest.
3. Improvement in symptoms in response to treatment directed towards heart failure.

Points 1 and 2 should be met, while point 3 substantiates the diagnosis in cases where there is doubt. Fatigue, shortness of breath and reduced physical performance capacity are cardinal symptoms of heart failure. Functional capacity is often classified with NYHA criteria (24). Typical signs of heart failure are crackling sounds from the lungs, tachycardia, ventricular gallop, peripheral oedema, enlarged liver and stasis of the neck veins.

Heart failure can be caused by systolic and/or diastolic dysfunction. In systolic dysfunction, the ventricular pumping capacity is impaired. Systolic function is usually measured as an ejection fraction (i.e. the stroke volume in relation to the total diastolic volume). Diastolic dysfunction is characterised by typical symptoms or signs of heart failure despite a normal ejection fraction, with concurrent signs of decrease in myocardial compliance. The diagnosis for systolic and/or diastolic dysfunction is made with the aid of a patient history, clinical examination, echocardiogram, x-ray and laboratory tests. Stress tests have limited diagnostic value in heart failure, but a normal stress test in an untreated patient rules out the diagnosis (25).

Prognosis

Despite the fact that the prognosis has improved in recent years (5), it is still poor, with a 5-year survival rate of approximately 50 per cent. The more symptoms a patient has, the worse the prognosis (2).

Treatment principles

The drugs most commonly used in the treatment of heart failure are diuretics, ACE inhibitors and beta blockers. Diuretics are used primarily to relieve symptoms. In addition to relieving symptoms, ACE inhibitors also have a prognostic effect. Beta blockers have a prognostic and symptomatic effect in moderate and severe heart failure. In patients with severe heart failure, spironolactone has a favourable prognostic effect. Angiotensin receptor blockers have been shown to reduce mortality in moderate and severe heart failure.

Effects of physical activity

Acute effects of aerobic exercise

The maximal oxygen uptake (VO_2 max) during arm exercise, cycling and walking in people with heart failure can be 30–40 per cent lower than in healthy individuals (26, 27). Maximal cardiac output is also decreased due to a decrease in maximal stroke volume and insufficient capacity to increase heart rate normally (28, 29). Systolic blood pressure does not increase normally (30), while the sympathetic activity increases more during exercise in patients with heart failure than in healthy individuals (31).

The respiratory minute volume (VE) is increased in patients with heart failure at every given load compared with healthy individuals. At maximal exertion, however, VE is approximately 50 per cent of predicted value, suggesting that this does not limit the exercise capacity (32). The ratio between minute ventilation and carbon dioxide production (VE/VCO_2) increases, and it is most often shortness of breath that is the limiting factor for physical activity in these patients.

The peripheral resistance during exercise decreases in patients with heart failure but is still higher than in healthy individuals. Normally, the blood flow in the skeletal muscle increases during aerobic exercise. In heart failure, the blood flow to the working muscles is reduced when large muscle mass is involved, which is the case with aerobic exercise (11). This results in anaerobic metabolism early on in the physical activity (33). Normal blood flow can be maintained, however, if the exercise is performed using a sufficiently small muscle mass (34).

Acute effects of muscle exercise

Isometric muscle exercise (static exercise)

In healthy individuals, the blood flow in the skeletal muscle in an isometric contraction is inhibited due to increased intramuscular pressure, which compresses the blood vessels (35). During isometric exercise, heart rate and systolic and diastolic blood pressure increase more in patients with heart failure than in healthy individuals (36). The increased afterload (the pressure that the ventricle is subjected to during the ejection phase, i.e. the expulsion resistance) that isometric exercise leads to could be harmful to patients with heart failure. However, in two Swedish studies patients with heart failure performed isometric exercise without any complications (16, 37).

Isotonic muscle exercise (dynamic exercise)

Exercise in the form of leg press at 60–80 per cent of maximum exertion does not yield a larger effect on haemodynamic variables than cycling. Neither does it yield a negative effect on left ventricular function (38, 39).

Acute effects of hydrotherapy

When the body is immersed in water, significant physiological changes occur. The physiological changes are due in part to the temperature and depth of the water. The pressure the body is subjected to is 0.74 mm Hg/1 cm H₂O. Immersion in thermoneutral water, approximately 35°C, to the upper edge of the sternum (suprasternal fossa) results in an increase in venous return that gives an increased blood volume in the heart. The increased preload results in a haemodynamic improvement with increased stroke volume and ejection fraction, while the heart rate remains unchanged or is somewhat lower. The result is an increase in cardiac output, while the mean arterial pressure is unchanged and the total peripheral resistance decreases due to vasodilatation in the peripheral tissues and abdominal organs (40).

The increased volume in the heart also stimulates cardiopulmonary receptors, which in turn signal the kidneys (via the brain) to reduce sympathetic stimulation and increase urine secretion (diuresis) and reduce the neurohormones renin, aldosterone and angiotensin II (41). This means that warm baths have a similar effect as that sought in modern drug treatment of heart failure. Even an increase in cardiac output, stroke volume and ejection fraction together with a decrease in peripheral resistance has been shown after warm baths. There is at present scientific debate regarding the benefits of hydrotherapy in heart failure. A group of German researchers (42) suggests that certain patients with severe mitral insufficiency (poorly functioning valve between the left atrium and ventricle) should not exercise in water, as they have measured a marked increase in preload in these patients. Studies conducted in Sweden have not been able to confirm this, so further research in this area is needed.

Long-term effects

Aerobic exercise, muscle exercise and hydrotherapy

The long-term effects of exercise in patients with heart failure (Table 2) are in many respects in line with those measured in healthy individuals and patients with heart disease without heart failure (43, 44). The exercise methods studied include central circulatory training, cycling in particular but also exercise classes, walking and peripheral muscle training. Hydrotherapy, tai chi and respiratory muscle training have also been studied.

Table 2. Long-term effects of aerobic central circulatory training and peripheral muscle endurance training in patients with chronic heart failure.

Effect variable	Result	Effect variable	Result
VO ₂ max	↑	Sympathetic activity	↓
Workload		Muscle blood flow	↑
watt	↑	Oxidative capacity	↑
exercise time	↑	Capillarisation	↑
		Muscle fibre area	↑
Heart rate		Muscle strength and endurance	↑
resting	↓		
	↔		
maximal	↑		
Stroke volume		Endotheliumindependent and endothelium-dependent vascular function	↑
resting	↑		
exercise	↑		↑
Cardiac output		Dyspnoea	↓
exercise	↑	Fatigue	↓
Ejection fraction*	↔	NYHA class	↓
	↑		
Systolic blood pressure		6-minute walk test	↑
resting	↔		
exercise	↓		
Respiratory minute volume	↓	Quality of life	↑
			↔
Inflammatory cytokines	↓	Mortality	↓

↑ = Increase, ↔ = No change, ↓ = Decrease.

* Ejection fraction = Ratio of stroke volume to total end-diastolic volume.

Indications

Patients with stable heart failure with predominant systolic dysfunction in NYHA class (I)-II-III. Experience from exercise training of patients in NYHA Class IV and patients with diastolic dysfunction is limited (43, 44).

Primary prevention

For patients with asymptomatic left ventricular dysfunction or myocardial infarction, training increases exercise capacity and cardiac output (45, 46). Whether training in asymptomatic patients with reduced left ventricular function is able to prevent the development of heart failure is not known.

Secondary prevention

Exercise training improves several of the pathophysiological findings found in heart failure disease. Two meta-analyses have shown that longer periods of exercise can have an impact on mortality and morbidity (44, 47). Exercise also has positive effects on underlying factors such as diabetes and hypertension (48, 49).

Prescription

Different types of exercise

Central circulatory training/aerobic exercise, peripheral/local muscle training on land and in water, and respiratory muscle training, are the most evaluated techniques (see Table 3).

Central circulatory training, which has been studied the most, involves the engagement of large muscle groups at the same time, for example, cycling, walking and/or exercise classes (44).

Table 3. Description of training methods investigated in different scientific studies in patients with chronic heart failure.

Training method	Intensity	RPE*	Frequency	Duration (min./session)
Aerobic central circulatory training	(40) 60–80% of VO_2 max**	11–15 central	1 time/week to several times/day	10–60
Peripheral muscle training	35–70% of 1 RM***	13–15 local	2 times/week to 1 time/day	15–60
Combination of aerobic central circulatory training and peripheral muscle training	60–80% of VO_2 max** 60–80% of 1 RM	13–15 central and local	3 times/week	45–60
Hydrotherapy	40–80% of HRR****	11–15	3 times/week	45
Respiratory muscle training	30% of max. inspiratory pressure		3 times/week to daily	30–60

* RPE = Rating of Perceived Exertion (Borg scale 6–20).

** VO_2 max = Maximal Oxygen Uptake.

*** RM = Repetition Maximum. 1 RM corresponds to the maximum weight that can be lifted through the entire exercise movement one time.

**** HRR = Heart Rate Reserve

In *peripheral muscle training*, the skeletal muscle is exercised locally at a relatively high load. Techniques for peripheral/local muscle training include endurance training with different types of weights and bands, for example, Thera-Band® (HCM, Hygenic Corporation, Malaysia) (50).

Respiratory training comprises both inspiratory and expiratory resistance training (51, 52).

In the studies of exercise training conducted until now, there is large variation when it comes to intensity, duration, frequency and exercise model. Empirically, patients with more severe heart failure (NYHA III) are considered to need a longer rehabilitation period than patients with ischaemic heart disease without heart failure, for example, in order to develop the habit of incorporating exercise into daily life. Individually adapted exercise programs where consideration is given to functional capacity and recovery time are very important for this patient group. Considering the disease's pathology, both central circulatory training and peripheral muscle training are probably important. Peripheral muscle training performed in sequences is probably of value to patients with more advanced heart failure, in order to regain lost muscle mass. This is an important step before commencing an exercise programme that engages the central circulation (53). It is essential for the patient's functional capacity that not only the leg muscles but also the arm and trunk muscles are exercised. Patients with chronic heart failure should always be referred to a physiotherapist for an individually adapted training programme. An intensity corresponding to between 60–80 per cent of VO_2 max, that is, 11–15 on the Borg RPE scale, is effective (54, 55). Two to three sessions per week are suitable for out-patient exercise training, while training at home and in hospital can be conducted more frequently.

Functional mechanisms

Oxygen uptake (VO_2)

Oxygen uptake improves by an average of about 15 per cent (2–34%), which is explained by the impact on several interacting factors such as improved heart muscle function, reduced vasoconstriction in the arterioles of the active skeletal muscle, along with an increased oxygen extraction and metabolic function in the active skeletal muscles. The measuring methods and measures of effect in the different studies vary, however, which can make it difficult to compare results between the studies. The effect of exercise training reported in the studies enable the patients to increase their activities at home, during leisure time and at work if applicable (43, 44).

Cardiac function

Cardiac function may be improved by exercise training due to better stroke volume and reduced chronotropic response during acute exercise (44).

Peripheral blood circulation and skeletal muscle function

Exercise normalises the peripheral blood circulation, probably mediated in part by improved endothelial function (56). Increased muscle strength gained through exercise is a result of both increased blood circulation to the muscles, increased muscle volume, and improved metabolic function in the muscle cells themselves. The oxidative capacity improves due to an increase in the amount of oxidative enzymes, mitochondrial volume and the number of type I fibres. Occlusion of intramuscular vessels begins already at 15 per cent of maximal voluntary contraction. The improved muscle function means that the patient is able to perform his or her daily activities at a lower percentage of maximal voluntary contraction, which theoretically results in reduced load on the heart (50).

Respiratory function

Respiratory minute volume (VE) and the ratio between minute ventilation and carbon dioxide production (VE/VCO₂) decreases, blood flow to the respiratory muscles increases, and the metabolic situation improves after exercise training (57). These could possibly be some of the reasons for the reduced breathlessness (dyspnoea) the patient experiences after exercise.

Neuroendocrine and autonomic function, and inflammatory cytokines

Exercise reduces the neuroendocrine activation and the number of inflammatory cytokines (58). The autonomic function also improves (59).

Mortality and hospitalisation

Two meta-analyses suggest that exercise can reduce mortality and hospitalisations in chronic heart failure. As in every case, the results from a meta-analysis must be interpreted with caution (60, 61).

Quality of life and symptoms

Quality of life and symptoms improve through training (44, 62). The underlying cause of this phenomenon may be of both a physical and psychological nature.

Functional tests

A functional test should always be performed to determine the appropriate individual exercise level before an exercise period begins. The same test should in addition always be conducted at the end of an exercise period to evaluate the effect of the exercise programme and for continued prescription.

Cycle/Treadmill test

A standardised maximal or submaximal cycle/treadmill test, preferably with analysis of maximal oxygen uptake, forms the basis for an appropriate design of an exercise programme (55, 63). It is also a way of ascertaining whether the patient tolerates increased physical exertion.

6-minute walk test

A standardised 6-minute walk test (64, 65) is often used to assess exercise capacity in relation to activities of daily life (ADL). The patient is encouraged to walk as far as possible for six minutes on a measured stretch of hallway. Measurement variables are the distance walked, perceived exertion measured on the Borg scale (66), and heart rate.

Muscle function

Strength and endurance, both isometric and isotonic, can be measured with an isokinetic instrument such as KINCOM (KINetic COMmunicator) (37). Clinical endurance tests can also be performed, for example, through one-legged toe raises or shoulder flexion test with weights (67).

Rating of symptoms, quality of life and physical activity

General health-related quality of life can be measured with the SF-36 questionnaire (68), while disease-specific quality of life is often measured with the Minnesota Living with

Heart Failure Questionnaire (69). An example of a survey that measures the level of physical activity is Frändin and Grimby's activity scale (76).

Symptom severity can be assessed with the visual analogue scale (VAS) (70) or the Borg scale (66). Level of function may be evaluated using, for example, NYHA classification criteria (24).

Interactions with drugs

Beta blockers

Maximal heart rate is lower in patients with heart failure treated with beta blockers than in patients not receiving beta blockers (71). Exercise capacity increases and the ejection fraction improves both at rest and during exertion after chronic treatment with beta blockers. No effect on the skeletal muscle has been found (72).

ACE inhibitors

ACE inhibitors have a moderate effect on exercise capacity. Cardiac output increases and the left ventricular filling pressure falls during exertion (73). The effect of ACE inhibitor therapy on skeletal muscle is not unambiguous. Increased muscle fibre area and changes in myosin composition in skeletal muscle fibres have been described earlier (71, 72).

Digitalis

Digitalis increases contractility in the myocardium and thereby the stroke volume, while heart rate decreases. Exercise capacity increases under digitalis therapy (74, 75).

Diuretics

An open study of severely symptomatic patients showed increased oxygen uptake during exertion after eight days of treatment with diuretics (76). Diuretics can have potentially negative effects through an increased risk of dehydration and electrolyte disturbances in warm weather.

Contraindications

Uncompensated heart failure, obstructive hypertrophic cardiomyopathy, significant valve disease (above all aortic stenosis), active myocarditis, a drop in blood pressure, serious arrhythmia or severe ischaemia during exertion. Other serious diseases such as ongoing infection, uncontrolled diabetes, uncontrolled hypertension or recent pulmonary embolism (54) are also contraindications.

Risks

No serious incidents have occurred during or after training of patients with heart failure (44, 62). However, the heart failure patients who have participated in exercise studies to date have been selected. The mortality is in general high in this patient group, which is why it is important that exercise initially should be implemented in a medical setting in case a serious complication should occur.

References

1. Libby PB, Mann RO, Zipes DL. *Brunwald's heart disease*. London: Saunders; 2007.
2. Cowie MR, Mosterd A, Wood DA, Deckers JW, Poole-Wilson PA, Sutton GC, et al. The epidemiology of heart failure. *Eur Heart J* 1997;18:208-25.
3. Eriksson H. Heart failure. A growing public health problem. *J Intern Med* 1995 Feb;237:135-41.
4. Ho KK, Pinsky JL, Kannel WB, Levy D. The epidemiology of heart failure. The Framingham Study. *J Am Coll Cardiol* 1993;22:6A-13A.
5. Schaufelberger M, Swedberg K, Koster M, Rosen M, Rosengren A. Decreasing one-year mortality and hospitalization rates for heart failure in Sweden. Data from the Swedish Hospital Discharge Registry 1988 to 2000. *Eur Heart J* 2004;25:300-7.
6. Franciosa JA, Park M, Levine TB. Lack of correlation between exercise capacity and indexes of resting left ventricular performance in heart failure. *Am J Cardiol* 1981;47:33-9.
7. Metra M, Raddino R, Dei Cas L, Visioli O. Assessment of peak oxygen consumption, lactate and ventilatory thresholds and correlation with resting and exercise hemodynamic data in chronic congestive heart failure. *Am J Cardiol* 1990;65:1127-33.
8. Miyashita T, Okano Y, Takaki H, Satoh T, Kobayashi Y, Goto Y. Relation between exercise capacity and left ventricular systolic versus diastolic function during exercise in patients after myocardial infarction. *Coron Artery Dis* 2001;12:217-25.
9. Clark AL, Poole-Wilson PA, Coats AJ. Exercise limitation in chronic heart failure. Central role of the periphery. *J Am Coll Cardiol* 1996;28:1092-102.
10. Drexler H. Reduced exercise tolerance in chronic heart failure and its relationship to neurohumoral factors. *Eur Heart J* 1991;12:21-8.
11. Sullivan MJ, Knight JD, Higginbotham MB, Cobb FR. Relation between central and peripheral hemodynamics during exercise in patients with chronic heart failure. Muscle blood flow is reduced with maintenance of arterial perfusion pressure. *Circulation* 1989;80:769-81.
12. Zelis R, Longhurst J, Capone RJ, Mason DT. A comparison of regional blood flow and oxygen utilization during dynamic forearm exercise in normal subjects and patients with congestive heart failure. *Circulation* 1974;50:137-43.
13. Swan JW, Walton C, Godsland IF, Clark AL, Coats AJ, Oliver MF. Insulin resistance in chronic heart failure. *Eur Heart J* 1994;15:1528-32.
14. Schaufelberger M, Eriksson BO, Grimby G, Held P, Swedberg K. Skeletal muscle fiber composition and capillarization in patients with chronic heart failure. Relation to exercise capacity and central hemodynamics. *J Card Fail* 1995;1:267-72.
15. Volterrani M, Clark AL, Ludman PF, Swan JW, Adamopoulos S, Piepoli M, et al. Predictors of exercise capacity in chronic heart failure. *Eur Heart J* 1994;15:801-9.
16. Magnusson G, Isberg B, Karlberg KE, Sylven C. Skeletal muscle strength and endurance in chronic congestive heart failure secondary to idiopathic dilated cardiomyopathy. *Am J Cardiol* 1994;73:307-9.

17. Stibrant Sunnerhagen K, Cider A, Schaufelberger M, Hedberg M, Grimby G. Muscular performance in heart failure. *J Card Fail* 1998;4:97-104.
18. Tejero-Taldo MI, Kramer JH, Mak Iu T, Komarov AM, Weglicki WB. The nerve–heart connection in the pro-oxidant response to Mg-deficiency. *Heart Fail Rev* 2006;11:35-44.
19. von Haehling S, Doehner W, Anker SD. Nutrition, metabolism, and the complex pathophysiology of cachexia in chronic heart failure. *Cardiovasc Res* 2007;73:298-309.
20. Ponikowski PP, Chua TP, Francis DP, Capucci A, Coats AJ, Piepoli MF. Muscle ergoreceptor overactivity reflects deterioration in clinical status and cardiorespiratory reflex control in chronic heart failure. *Circulation* 2001;104:2324-30.
21. Witte KK, Clark AL. Why does chronic heart failure cause breathlessness and fatigue? *Prog Cardiovasc Dis* 2007;49:366-84.
22. Piepoli MF, Scott AC, Capucci A, Coats AJ. Skeletal muscle training in chronic heart failure. *Acta Physiol Scand* 2001;171:295-303.
23. Swedberg K, Cleland J, Dargie H, Drexler H, Follath F, Komajda M, et al. Guidelines for the diagnosis and treatment of chronic heart failure. Executive summary (update 2005). Task Force for the Diagnosis and Treatment of Chronic Heart Failure of the European Society of Cardiology. *Eur Heart J* 2005;26:1115-40.
24. New York Heart Association. Nomenclature and criteria of the heart and great vessels. New York: Little and Brown Company; 1973.
25. Remme WJ, Swedberg K. Guidelines for the diagnosis and treatment of chronic heart failure. *Eur Heart J* 2001;22:1527-60.
26. Keteyian SJ, Marks CR, Brawner CA, Levine AB, Kataoka T, Levine TB. Responses to arm exercise in patients with compensated heart failure. *J Cardiopulm Rehabil* 1996;16:366-71.
27. Clark AL, Davies LC, Francis DP, Coats AJ. Ventilatory capacity and exercise tolerance in patients with chronic stable heart failure. *Eur J Heart Fail* 2000;2:47-51.
28. Feigenbaum M. Cardiopulmonary, hemodynamic and neurohormonal responses to acute exercise in patients with chronic heart failure. Florida: University of Florida; 1997.
29. Higginbotham MB, Morris KG, Conn EH, Coleman RE, Cobb FR. Determinants of variable exercise performance among patients with severe left ventricular dysfunction. *Am J Cardiol* 1983;51:52-60.
30. Stone GW, Kubo SH, Cody RJ. Adverse influence of baroreceptor dysfunction on upright exercise in congestive heart failure. *Am J Med* 1986;80:799-802.
31. Colucci WS, Ribeiro JP, Rocco MB, Quigg RJ, Creager MA, Marsh JD, et al. Impaired chronotropic response to exercise in patients with congestive heart failure. Role of postsynaptic beta-adrenergic desensitization. *Circulation* 1989;80:314-23.
32. Weber KT, Kinasevitz GT, Janicki JS, Fishman AP. Oxygen utilization and ventilation during exercise in patients with chronic cardiac failure. *Circulation* 1982;65:1213-23.
33. Massie B, Conway M, Yonge R, Frostick S, Ledingham J, Sleight P, et al. Skeletal muscle metabolism in patients with congestive heart failure. Relation to clinical severity and blood flow. *Circulation* 1987;76:1009-19.

34. Magnusson G, Kaijser L, Sylven C, Karlberg KE, Isberg B, Saltin B. Peak skeletal muscle perfusion is maintained in patients with chronic heart failure when only a small muscle mass is exercised. *Cardiovasc Res* 1997;33:297-306.
35. Shephard RJ, Balady GJ. Exercise as cardiovascular therapy. *Circulation* 1999;99:963-72.
36. Mangieri E, Tanzilli G, Barilla F, Ciavolella M, Serafini G, Nardi M, et al. Isometric handgrip exercise increases endothelin-1 plasma levels in patients with chronic congestive heart failure. *Am J Cardiol* 1997;79:1261-3.
37. Cider A, Tygesson H, Hedberg M, Seligman L, Wennerblom B, Sunnerhagen KS. Peripheral muscle training in patients with clinical signs of heart failure. *Scand J Rehabil Med* 1997;29:121-7.
38. McKelvie RS, McCartney N, Tomlinson C, Bauer R, MacDougall JD. Comparison of hemodynamic responses to cycling and resistance exercise in congestive heart failure secondary to ischemic cardiomyopathy. *Am J Cardiol* 1995;76:977-9.
39. Meyer K, Hajric R, Westbrook S, Haag-Wildi S, Holtkamp R, Leyk D, et al. Hemodynamic responses during leg press exercise in patients with chronic congestive heart failure. *Am J Cardiol* 1999;83:1537-43.
40. Cider A, Svealv BG, Tang MS, Schaufelberger M, Andersson B. Immersion in warm water induces improvement in cardiac function in patients with chronic heart failure. *Eur J Heart Fail* 2006;8:308-13.
41. Gabrielsen A, Bie P, Holstein-Rathlou NH, Christensen NJ, Warberg J, Dige-Petersen H, et al. Neuroendocrine and renal effects of intravascular volume expansion in compensated heart failure. *Am J Physiol Regul Integr Comp Physiol* 2001;281:R459-67.
42. Meyer K, Bucking J. Exercise in heart failure. Should aqua therapy and swimming be allowed? *Med Sci Sports Exerc* 2004;36:2017-23.
43. Rees K, Taylor RS, Singh S, Coats AJ, Ebrahim S. Exercise based rehabilitation for heart failure. *Cochrane Database Syst Rev* 2004;3:CD003331.
44. van Tol BA, Huijsmans RJ, Kroon DW, Schothorst M, Kwakkel G. Effects of exercise training on cardiac performance, exercise capacity and quality of life in patients with heart failure. A meta-analysis. *Eur J Heart Fail* 2006;8:841-50.
45. Conn EH, Williams RS, Wallace AG. Exercise responses before and after physical conditioning in patients with severely depressed left ventricular function. *Am J Cardiol* 1982;49:296-300.
46. Dubach P, Myers J, Dziekan G, Goebbels U, Reinhart W, Vogt P, et al. Effect of exercise training on myocardial remodeling in patients with reduced left ventricular function after myocardial infarction. Application of magnetic resonance imaging. *Circulation* 1997;95:2060-7.
47. Piepoli MF, Davos C, Francis DP, Coats AJ. Exercise training meta-analysis of trials in patients with chronic heart failure (ExTraMATCH). *BMJ* 2004;328:189.
48. Boule NG, Haddad E, Kenny GP, Wells GA, Sigal RJ. Effects of exercise on glycemic control and body mass in type 2 diabetes mellitus. A meta-analysis of controlled clinical trials. *JAMA* 2001;286:1218-27.

49. Pescatello LS, Franklin BA, Fagard R, Farquhar WB, Kelley GA, Ray CA. American College of Sports Medicine Position Stand. Exercise and hypertension. *Med Sci Sports Exerc* 2004;36:533-53.
50. Delagardelle C, Feiereisen P. Strength training for patients with chronic heart failure. *Eura Medicophys* 2005;41:57-65.
51. Laoutaris I, Dritsas A, Brown MD, Manginas A, Alivizatos PA, Cokkinos DV. Inspiratory muscle training using an incremental endurance test alleviates dyspnea and improves functional status in patients with chronic heart failure. *Eur J Cardiovasc Prev Rehabil* 2004;11:489-96.
52. Mancini DM, Henson D, La Manca J, Donchez L, Levine S. Benefit of selective respiratory muscle training on exercise capacity in patients with chronic congestive heart failure. *Circulation* 1995;91:320-9.
53. McKelvie R. Exercise training in heart failure. How? *Heart Failure Reviews* 1999;3:263-73.
54. Pina IL, Apstein CS, Balady GJ, Belardinelli R, Chaitman BR, Duscha BD, et al. Exercise and heart failure. A statement from the American Heart Association Committee on exercise, rehabilitation, and prevention. *Circulation* 2003;107:1210-25.
55. Corra U, Giannuzzi P, Adamopoulos S, Bjornstad H, Bjarnason-Wehrens B, Cohen-Solal A, et al. Executive summary of the position paper of the Working Group on Cardiac Rehabilitation and Exercise Physiology of the European Society of Cardiology (ESC). Core components of cardiac rehabilitation in chronic heart failure. *Eur J Cardiovasc Prev Rehabil* 2005;12:321-5.
56. Sabelis LW, Senden PJ, Fijnheer R, de Groot PG, Huisveld IA, Mosterd WL, et al. Endothelial markers in chronic heart failure. Training normalizes exercise-induced vWF release. *Eur J Clin Invest* 2004;34:583-9.
57. European Heart Failure Training Group. Experience from controlled trials of physical training in chronic heart failure. Protocol and patient factors in effectiveness in the improvement in exercise tolerance. *Eur Heart J* 1998;19:466-75.
58. Gielen S, Adams V, Linke A, Erbs S, Mobius-Winkler S, Schubert A, et al. Exercise training in chronic heart failure. Correlation between reduced local inflammation and improved oxidative capacity in the skeletal muscle. *Eur J Cardiovasc Prev Rehabil* 2005;12:393-400.
59. Myers J, Hadley D, Oswald U, Bruner K, Kottman W, Hsu L, et al. Effects of exercise training on heart rate recovery in patients with chronic heart failure. *Am Heart J* 2007;153:1056-63.
60. Hagerman I, Tyni-Lenne R, Gordon A. Outcome of exercise training on the long-term burden of hospitalisation in patients with chronic heart failure. A retrospective study. *Int J Cardiol* 2005;98:487-91.
61. Taylor A. Physiological response to a short period of exercise training in patients with chronic heart failure. *Physiother Res Int* 1999;4:237-49.
62. Rees K, Bennett P, West R, Davey SG, Ebrahim S. Psychological interventions for coronary heart disease. *Cochrane Database Syst Rev* 2004;2:CD002902.

63. Thompson PD, Buchner D, Pina IL, Balady GJ, Williams MA, Marcus BH, et al. Exercise and physical activity in the prevention and treatment of atherosclerotic cardiovascular disease. A statement from the Council on Clinical Cardiology (Subcommittee on Exercise, Rehabilitation, and Prevention) and the Council on Nutrition, Physical Activity, and Metabolism (Subcommittee on Physical Activity). *Circulation* 2003;107:3109-16.
64. Guyatt G. Use of the six-minute walk test as an outcome measure in clinical trials in chronic heart failure. *Heart Failure* 1987;21:211-7.
65. Lipkin DP, Scrivin AJ, Crake T, Poole-Wilson PA. Six minute walk test for assessing exercise capacity in chronic heart failure patients. *BMJ* 1986;292:653-5.
66. Borg G. Perceived exertion as an indicator of somatic stress. *Scand J Rehab Med* 1970;2:92-8.
67. Cider A, Carlsson S, Arvidsson C, Andersson B, Stibrant Sunnerhagen K. Reliability of clinical muscular endurance tests in patients with chronic heart failure. *Eur J Cardiovasc Nurs* 2006;5:122-6.
68. Ware JE Jr, Sherbourne CD. The MOS 36-item short-form health survey (SF-36). I. Conceptual framework and item selection. *Med Care* 1992;30:473-83.
69. Rector TS, Cohn JN. Assessment of patient outcome with the Minnesota Living with Heart Failure Questionnaire. Reliability and validity during a randomized, double-blind, placebo-controlled trial of pimobendan. Pimobendan Multicenter Research Group. *Am Heart J* 1992;124:1017-25.
70. Carlsson AM. Assessment of chronic pain. I. Aspects of the reliability and validity of the visual analogue scale. *Pain* 1983;16:87-101.
71. Schaufelberger M, Andersson G, Eriksson BO, Grimby G, Held P, Swedberg K. Skeletal muscle changes in patients with chronic heart failure before and after treatment with enalapril. *Eur Heart J* 1996;17:1678-85.
72. Schaufelberger M, Eriksson BO, Grimby G, Held P, Swedberg K. Skeletal muscle alterations in patients with chronic heart failure. *Eur Heart J* 1997;18:971-80.
73. Swedberg K. Exercise testing in heart failure. A critical review. *Drugs* 1994;47:14-24.
74. Slatton ML, Irani WN, Hall SA, Marcoux LG, Page RL, Grayburn PA, et al. Does digoxin provide additional hemodynamic and autonomic benefit at higher doses in patients with mild to moderate heart failure and normal sinus rhythm? *J Am Coll Cardiol* 1997;29:1206-13.
75. Vescovo G, Dalla Libera L, Serafini F, Leprotti C, Facchin L, Volterrani M, et al. Improved exercise tolerance after losartan and enalapril in heart failure. Correlation with changes in skeletal muscle myosin heavy chain composition. *Circulation* 1998;98:1742-9.
76. Mattioli AV, Castellani ET, Casali E, Mattioli G. Symptomatic achievements with diuretics in congestive heart failure. *Cardiology* 1994;84:131-4.

30. Heart rhythm disturbances

Authors

Agneta Ståhle, PT, PhD, Associate Professor, Department of Neurobiology, Care Sciences and Society, Division of Physiotherapy, Karolinska Institutet, Stockholm, Sweden

Lennart Bergfeldt, MD, PhD, Professor, Sahlgrenska Academy, Gothenburg University and Sahlgrenska University Hospital, Gothenburg, Sweden

Anders Englund, MD, PhD, Associate Professor, Arrhythmia Center and Karolinska Institutet, South Hospital, Stockholm, Sweden

Knut Gjesdal, MD, PhD, Professor, Department of Cardiology, Ullevål University Hospital, Oslo, Norway

Summary

Heart rhythm disturbances is a collective description of deviations in the heart's electrical impulse generation and/or conduction. Included in this concept at one end of the spectrum are extra beats, which do not usually produce symptoms and are therefore perceived as a normal variant in the absence of other heart disease, and at the other end directly life-threatening ventricular fibrillation or interruptions in the impulse generation. Many patients with arrhythmia problems have an underlying cardiovascular disease such as hypertension, coronary heart disease and/or heart failure, which is why care must be taken when prescribing physical activity. The principles for exercise training that apply to other heart patients are also applicable to patients with arrhythmias. The prescription must always include definition of the frequency, duration and intensity.

Training method	Intensity	Frequency (times/week)	Duration (min./session)
Aerobic training	50–80% of VO ₂ max* RPE** 9–15/20	2–3	45–60
Strength training	40–60% of 1 RM*** 1–3 sets of 8–10 exercises with 12–15 reps/set RPE 11–13/20	2–3	30–40
Muscle endurance training	40–80% of 1 RM*** > 15 reps/set RPE 9–15/20	2–3	45–60

* VO₂ max = Maximal Oxygen Uptake.

** Rating of Perceived Exertion, according to the Borg scale.

*** RM = Repetition Maximum, which corresponds to the maximum weight that can be lifted throughout the entire exercise movement one time.

Suitable activities include brisk walking, jogging, cycling, swimming, exercise or aquafit classes, skiing, skating, dance and ball sports, depending on the individual’s interests. For patients with an implantable cardioverter defibrillator (ICD), activities like jogging in proximity to heavily trafficked roadways, swimming and cycling involve a certain risk of injury to themselves or others as there is a 10 to 20 second delay between detection of the arrhythmia and generation of the ICD shock.

Definition

The heart is a muscular pump through which blood flow is controlled by valves (one-way valves). In order for the pumping motion to occur, an electrical activation (electromechanical coupling) is required. This originates in the heart’s own electrical system which is made up of a generator (the sinoatrial node), a reserve generator and filter between the atrium and ventricle (the AV node), and a main cable (the His bundle), which branches into three main divisions – one to the right– and two to the left ventricle. The heart is also a secretory organ with hormones that affect the heart itself and other organs such as the kidneys.

Heart rhythm disturbances is a collective description of deviations in the heart’s electrical impulse generation and/or conduction. At one end of the spectrum are extra beats, which do not usually produce symptoms and are therefore perceived as a normal variant in the absence of other heart disease, and at the other end are directly life-threatening ventricular fibrillation or interruptions in the impulse generation. Deviations in the heart’s electrical activity can give rise to both too slow a rhythm (bradycardia) and a racing heart (tachycardia). An estimated 1–1.5 per cent of the population has some form of heart rhythm disorder that will at some point become the object of assessment and/or treatment. Commonly occurring disorders are atrial fibrillation (approx. 1 per cent of the entire population, but approx. 10 per cent of the population over 80 years), bradycardia that requires artificial pacing (0.3 per cent) and attacks of regular atrial tachycardia

(0.5–1.0 per cent of the adult population). The extent of life-threatening ventricular tachycardia is more difficult to estimate. In Sweden, about 30,000 people per year are diagnosed with acute myocardial infarction. Against this background, it is important to know that approximately 30 per cent of those who die a sudden death in acute myocardial infarction had no earlier symptoms of coronary heart disease.

Cause

Cardiac arrhythmias, another general description, can be primary or secondary. Primary electrical problems can be congenital, caused by extra pathways outside the regular conduction system (WPW syndrome), or acquired, extra impulse pathway within the heart's own electrical system (AV nodal reentry tachycardia). Functional and structural changes in the pores (ionic channels) that control the flow of electrically charged particles (above all sodium, potassium and calcium ions) across the cell membrane are also among the primary arrhythmias. An example of such a disease condition is congenital long QT syndrome, which involves an increased risk for serious arrhythmias and fainting. By secondary problems are meant disorders in the heart muscle and/or valve function that have consequences for the heart's electrical function, as well as other diseases, such as toxic goitre, which can lead to arrhythmia.

Risk factors

There is a genetic origin to certain types of arrhythmia, for example, long QT syndrome, certain types of atrial fibrillation, and some types of ventricular arrhythmias. In most patients with atrial fibrillation and ventricular arrhythmias, however, there is an underlying cardiovascular disease, such as hypertension and coronary heart disease. Because there are a number of predisposing factors, at least with respect to the last disease condition noted, such as diabetes, smoking, overweight, blood lipid disorders, etc., these must also be seen as risk factors for arrhythmias. It is suspected that strenuous physical exercise can predispose to atrial fibrillation.

Pathophysiological mechanisms

The cause of too slow a heart rhythm (bradycardia) requiring pacemaker treatment is just as often disorders in sinoatrial node function (sinus bradycardia, sinus pause), blocking conduction of the impulse between the atrium and ventricle (AV block). When it comes to a rapid heart rhythm (tachycardia), the predominant mechanism is an electrical circuit (a reconnection or reentry mechanism) This electrical circuit can be relatively stable, such as in AV nodal reentry tachycardia and WPW syndrome, as well as some ventricular tachycardias associated with scarring after myocardial infarction, but can also vary as with atrial fibrillation and ventricular arrhythmias in relation to congenital or acquired long QT syndrome. The latter includes effects of drugs and thickening of the heart muscle as a result of hypertension, heart failure and cardiomyopathy (disease of the heart muscle).

Abnormal impulse generation is less common as a cause of atrial arrhythmias, but is relevant in the initiation of ventricular arrhythmia with respect to acquired long QT syndrome.

Common symptoms

Heart palpitations involve the patient's perception of his or her heart rhythm, but are not necessarily a symptom of arrhythmia, as palpitations also occur in sinus tachycardia of a purely physiological nature. Sudden onset palpitations are the most predominant symptom in patients with tachycardias but who are otherwise in good cardiac health. Shortness of breath, pressure across the chest or chest pain and disturbances in consciousness (dizziness and/or fainting) are more common during rapid heart rhythms in individuals with other concurrent heart disease. In the case of atrial fibrillation, impaired performance capacity is a common symptom. When it comes to bradycardia-related symptoms, sudden onset dizziness or fainting is most common, but shortness of breath, fatigue and impaired performance capacity upon exertion are also common symptoms that lead the patient to seek medical attention.

The heart's pumping capacity, that is its ability to meet the varying demands of the body is seen in the cardiac output (heart minute volume), which at rest is 4–5 litres per minute depending on body size, and can increase to 25–30 litres per minute during maximal exertion. This ability to adjust is primarily dependent on variations in heart rate, which at rest is most often between 50 and 70 beats per minute and up to 170–200 per minute at maximal exertion. The heart's stroke volume (volume per beat) can increase by approximately 50 per cent. Another important factor to consider in this context is that 85 per cent of the blood supply to the heart itself takes place in the heart's electrical resting phase (diastole), and when heart rate increases, regardless of the cause, this mainly reduces the heart's resting phase. In concrete terms, this means that while a heart rate that is too slow yields good filling of the heart's chambers, it yields poor adjustment to increased demands, whereas a high heart rate reduces the filling and places high demands on the heart muscle's energy supply since every heart beat expends energy, while the time for supplying this energy and oxygen is reduced, relatively speaking. Both ends of this heart rate spectrum can be life-threatening.

In summary, however, the patient's symptoms are due firstly to the heart rate, secondly to cardiac function in general, and thirdly to the patient's general fitness, which can vary from one instance to another.

Diagnosis

Manual pulse-taking, electrocardiography (ECG) conducted as resting ECG or long-term ECG monitoring and electrophysiological recording and stimulation methods, in part from outside the heart via the oesophagus and in part from inside the heart via blood vessels, for example, in the groin, are important diagnostic tools. Stress tests have a poor capacity (sensitivity) to provoke (diagnose) both tachycardia and bradycardia, but are a valuable method for assessing the patient's overall aerobic fitness and possible presence of coronary heart disease.

Treatment

A correct diagnosis is decisive in the care of these patients. Once a link between a rhythm disturbance and symptoms has been established and an assessment of the level of danger and prognosis has been made, it is often possible to refrain from other treatment. When it comes to bradycardias, the decision is usually a pacemaker in a secondary prophylactic aim (when arrhythmia is already producing symptoms) and less often in a primary prophylactic aim (before symptoms appear). For tachycardias, it is usually a question of no treatment at all, drug therapy, ablation therapy (an invasive catheter technique in which the electrical disorder or pathway is treated with heat), surgery (e.g. maze surgery for atrial fibrillation), or combinations of treatments such as a pacemaker plus drug therapy, drug therapy plus ablation, etc. Implantable cardiac defibrillators (ICDs) are used both to treat ventricular arrhythmia relapses and to prevent sudden arrhythmia death in high-risk patients.

Effects of physical activity

Long-term effects

Exercise training has effects on the autonomic nervous system's parasympathetic system, which can affect heart rhythm disturbances in different directions. An increase in vagal activity, particularly at night, can give rise to both sinus pauses and AV blocks, mainly in the form of AV block II (Wenckebach type). In a physically fit person, this rhythm disturbance is of little prognostic importance as long as conditions during physical exertion are completely normal (1).

A less common subgroup of atrial fibrillation presents primarily at night and is considered to be linked to vagal dominance. In the case of this condition, exercise training can possibly predispose, even if scientific evidence here is lacking. In animal trials, vagal activity during concurrent sympathetic stimulation and arrhythmia provocation has been shown to have a beneficial effect in that the fibrillation threshold in the ventricles is raised and it becomes harder to induce fibrillation. This has nevertheless not been convincingly documented in humans. Because good aerobic fitness can improve a person's tolerance to disturbances in cardiac function, good overall fitness is worth striving for. After many years of training at the competitive level, arrhythmias are relatively common (2), but the mechanism behind this has not been established. This applies in particular to atrial fibrillation (3, 4).

Effects of arrhythmia

A tendency to bradycardia is often linked to an inability to increase the heart rate sufficiently in connection with exertion (chronotropic incompetence). This results in reduced maximal performance capacity. Certain tachycardias, and tachycardias in certain people, present especially in connection with physical and/or mental exertion, which the patient has usually observed. As a rule, one's performance capacity decreases in connection with the tachycardia.

Exercise training for different arrhythmia

Pronounced sinus arrhythmia

Young, physically fit persons often have a slow and uneven resting heart rate. This is due mainly to breathing-related vagal reflexes and disappears when the heart rate increases during activity.

Permanent atrial fibrillation

In a randomised clinical study of 30 patients with chronic atrial fibrillation, Hegbom and colleagues showed that 2 months of aerobic fitness and strength training yielded an increase in performance capacity on the exercise bike (41% at 17/20 on the Borg scale) and better heart rate control (5). Heart symptoms as well as quality of life were also significantly improved (6).

Atrial flutter

In the case of atrial flutter, the atrium beats regularly at approximately 250 beats per minute. The AV node normally blocks the impulses so that every second, third or fourth impulse is conducted. In physical exertion, there is always a risk that conduction of the impulses will increase to 1:1 (7). This gives worse circulation and many people experience a drop in blood pressure and shortness of breath. These patients may therefore require drugs to slow the AV conduction while they are exercising (beta blockers, calcium antagonists). These patients can be cured with ablation treatment and possible drugs against the flutter can be withdrawn.

Patients with pacemaker-ICD

An artificial pacemaker sends impulses to the heart at the rate set at the hospital and most have an activity sensor that increases the stimulation during physical exertion. The most common sensors respond to vibrations or movement and resting heart rate, maximal heart rate, and how quickly the heart rate should increase and decrease can be programmed. Different types of exercise training produce different responses in heart rate: running increases the heart rate a lot, cycling a bit less, and swimming yields a weaker stimulus and can in fact lead to a drop in blood pressure during exertion. An active person with a pacemaker should therefore adjust the programming to his or her activities. Occasionally other sensor systems are needed, for example, respiratory-controlled or impedance-controlled systems (the resistance in the system decreases when sympathetic tone increases).

Problems can also arise with exertion in ICD patients. The system can sometimes have trouble distinguishing between when the heart is beating rapidly due to exertion and serious arrhythmias that must be treated with pacing or shock. This can lead to the patient receiving shock unnecessarily. With proper programming, however, it generally works well, and according to a large controlled study ICD patients have good results from exercising (8).

Patients with tetraplegia

Spinal injuries in vertebrae Th 1–4 have an impact on the sympathetic innervation of the heart. This results in a poorer pulse increase and sometimes a drop in blood pressure during exercise. Arm training can improve the metabolism (9), however, and training with electrical stimulation of paralysed muscles yields better oxygen uptake, increased muscle and less fatty tissue (10). In most of these patients, however, an increase in heart rate cannot be used as a measure of exercise intensity (11).

Arrhythmias that occur in connection with exercise

Arrhythmias sometimes present during exercise load, and a rapid start and high load is sometimes used as a provocation test. Supraventricular arrhythmias induced by intensive exercise do not affect future risk of cardiac death (12). When a patient experiences arrhythmia during training or competition, the current activity should be stopped so that the stimulation from the sympathetic nervous system decreases, which reduces and normalises the heart rate. In atrial fibrillation and flutter in particular, continued activity can lead to a very strong and potentially dangerous heart rhythm.

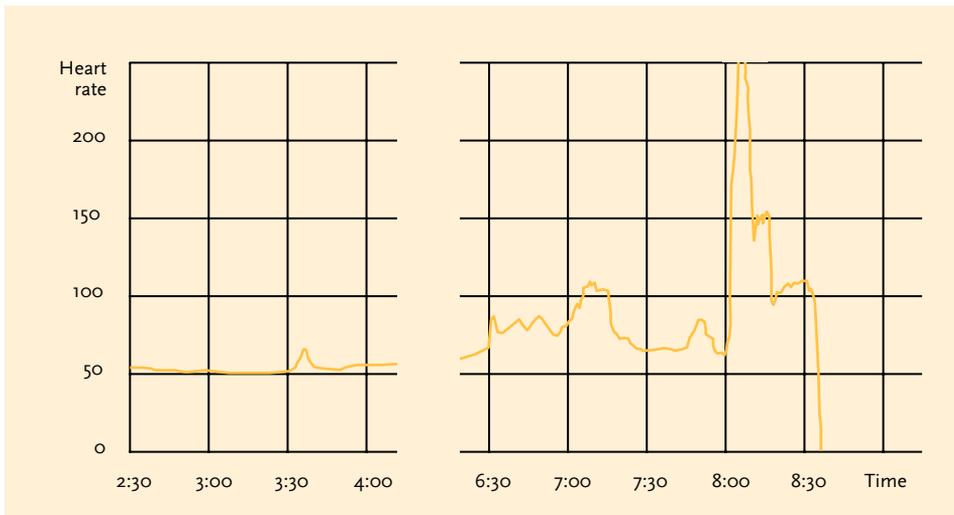


Figure 1. Trend curve from 24-hour ECG in a 50-year-old man with atrial fibrillation. At 8:10 he has an attack, and at 8:20 he attempts to “run it off”. Maximal heart rate is 275 beats per minute.

Indications

Because good aerobic fitness can improve one's tolerance for disturbances in cardiac function, regular physical activity, preferably with the goal of endurance training, is of particular importance for this group of patients. If it has been present for a long time, a tendency to arrhythmia often has a negative impact on the level of physical activity, that is, the patient is afraid to exert him/herself for fear of provoking an arrhythmia or other symptoms during exertion, such as dizziness or fainting, resulting in a deterioration in aerobic fitness. In such cases, supervised training is especially important in the beginning and preferably with a physiotherapist specialised in heart diseases.

Prescription

The principles for exercise training applicable to other heart patients are also applicable for patients with arrhythmias. The prescription must always include definition of the frequency, duration and intensity. Many patients with arrhythmia problems consequently have other underlying heart disease, such as heart failure and/or coronary heart disease, which is why consideration must be taken to this when prescribing physical activity and training (13). For more on this, see the chapters on Heart Failure and Coronary Heart Disease.

The general goal of exercise training in heart disease is to improve aerobic fitness through loading the central circulatory system. When it comes to the central circulatory system, training is effective and less strenuous if as large muscle groups as possible are engaged in the training. One effective and non-injurious way to conduct this training is in intervals, alternating between harder and easier intervals of 3–5 minutes (14). In order to improve aerobic fitness in healthy, previously physically inactive individuals, a training intensity of about 50 per cent of the individual's maximal oxygen uptake (corresponding to light to moderate breathlessness) for 30 minutes, 3 times per week, appears to be completely sufficient to achieve an improvement of between 5 and 10 per cent (15). Each training session should begin with a warm-up phase and end with a relatively long cool-down phase, regardless of the activity being done. The cool-down phase is of particular importance for patients with arrhythmia problems as arrhythmias generally appear in this phase of training (13, 16). The principle of interval training should be applied both in group exercise classes and cycling, aquafit and other forms of training.

All training should begin with a successive warm-up of 6–10 minutes at an intensity of up to 50 per cent of maximal capacity and an exertion level of “very light to light”, a perceived level of 9–11 on the Borg RPE scale (17). After warm-up, come 3 loading exercise sessions of 4–5 minutes each at an intensity of up to 50–80 per cent of maximal capacity and with an exertion level of “somewhat hard to hard (heavy)”, 13–15 on the RPE scale. Between the loading intervals, follow lighter 4–5-minute intervals at an intensity of up to 50 per cent of maximal capacity and an exertion level of 9–11 on the RPE scale. All training should finish off with a successive cool-down and stretching of at least 6, and optimally 10, minutes.

Supervised training includes individual adaptation of both the load and the time for this during the training period itself. For patients with arrhythmia problems, it can be beneficial to begin by lengthening the loading interval by 2–3 minutes before the load level is increased, that is, a somewhat longer training session than those noted above.

Strength training, which used to be considered contraindicated for patients with coronary heart disease and heart failure, has been shown in recent studies to be a safe and effective way to train (18, 19). The requirement is that the load not exceed 60 per cent of 1 RM (Repetition Maximum = the weight that can be lifted through the entire exercise movement once) and that the number of repetitions per set be somewhat higher (12–15) than in traditional strength training. Sometimes strength training must be preceded by other training in order to enable fitness training, such as a brisk walk. One example of this is exercise training for patients with heart failure, whose muscles may be so weakened that lighter strength training, or peripheral muscle training (with a focus on endurance training), is the only type of exercise initially tolerated by the patient. The load level in muscular endurance training can be determined with the help of Borg's RPE scale or by establishing an RM. Here, the number of repetitions should be higher than 15 in each set, see also the chapter on Heart Failure.

Using a relatively high load enables one to achieve the beneficial effects of physical training more rapidly, but not all elderly persons or patients with concurrent heart failure can handle the heavier load. For these people, a parallel assessment of the central- and peripheral exertion levels should be made. Here, one chooses a lower central load (intensity up to 50–60 per cent of maximal capacity, exertion level 10–11 on the RPE scale), but can have a higher intensity in the peripheral exercise (exertion level 13–15 on the RPE scale).

Table 1. Description of training methods for patients with heart rhythm disturbances.

Training method	Intensity	Frequency (times/week)	Duration (min./session)
Aerobic fitness training	50–80% of VO_2 max* RPE** 9–15/20	2–3	45–60
Strength training	40–60% of 1 RM*** 1–3 sets of 8–10 exercises with 12–15 reps/set RPE 11–13/20	2–3	30–40
Muscle endurance training	40–80% of 1 RM*** > 15 reps/set RPE 9–15/20	2–3	45–60

* VO_2 max = Maximal Oxygen Uptake.

** Rating of Perceived Exertion, according to the Borg scale (17).

*** RM = Repetition Maximum. 1 RM corresponds to the maximum weight load that can be lifted through the entire exercise movement one time.

The choice of activity should always be preceded by a history of the patient's physical activity where consideration is given to fitness level, interests and requirements. Muscle training oriented to activities of daily life can be of particular benefit for the elderly, as declining muscle endurance and strength can prevent them from remaining socially self-reliant and living an independent life. Aerobic fitness training, which can be carried out in the form of brisk walks, jogging, cycling, swimming, exercise or aquafit classes, skiing, skating, dance or ball sports, depending on the individual's interests, should include 45–60 minutes of exercise, 2–3 times per week. For patients with implantable cardioverter defibrillators (ICDs), activities like jogging in proximity to heavily trafficked roadways, swimming and cycling can involve a certain risk of injury to themselves or others as there is a 10–20 second delay between detection of the arrhythmia and activation of the ICD shock (13). Other patients may also experience dizziness or disturbances to consciousness, and exercise training must be adapted with this in mind.

All training should be complemented with at least 30 minutes of physical activity daily, which need not be strenuous nor performed all at once and can include everything from regular moving about to walks and climbing stairs (20, 21). The goal is to achieve a daily energy expenditure of at least 660 kJ (approx. 150 kcal), a level for which there are documented health effects (10).

Functional mechanisms

Decreased heart rate variability is a risk factor for arrhythmia-related death in patients following a myocardial infarction (22). Aerobic fitness training in patients with coronary heart disease, or with heart failure, leads to increased heart rate variability as an expression of a relative increase in parasympathetic activity (23, 24). The latter study suggests a reduced arrhythmia risk, but larger studies than those currently available are needed to confirm this.

Functional tests

A patient history and physical examination complemented by an electrocardiogram (ECG) constitute minimum requirements before arrhythmia patients start training. If cardiovascular disturbance is suspected, or before training at a competitive level, echocardiography (ultrasound examination) of the heart is recommended to show possible structural heart disease and assess ventricular function. Echocardiography should be complemented with a stress ECG, which gives an overall assessment of function and can also give an idea of the tendency to arrhythmia in connection with maximal exertion.

All training at the physiotherapist should be preceded by some form of test in which aerobic and muscular fitness are evaluated before choosing the level of training. A stress ECG is recommended and should be conducted with current medication.

Interactions with drug therapy

Beta blockers and certain calcium antagonists (verapamil, diltiazem) are negative chronotropes, that is, they lead to a lowering of resting heart rate and reduced maximal heart rate. This generally limits maximal performance capacity, though the drugs themselves do not constitute a particular risk in connection with exertion. The underlying treatment indication (disease in question) is decisive for whether individual consultation is needed.

Contraindications

In general, the content and level/intensity of training should be adapted to the individual's requirements and conditions.

Absolute contraindications

Absolute contraindications are exercise-induced ventricular arrhythmias and atrial arrhythmias of high heart rate (>180–200 beats/minute), as well as recent onset and uninvestigated arrhythmia.

Relative contraindications

Tolerance for arrhythmias is normally reduced if the patient is hypoglycemic (low blood sugar level) and/or dehydrated. These factors are therefore important to consider in all types of exercise training, and especially in patients with heart disease, including those with isolated electrical disturbances.

Risks

Hypotension with impaired consciousness and in the worst case cardiac arrest.

References

1. Bergfeldt L. Atrioventricular conduction disturbances. *Cardiac Electrophysiology Review* 1997;1:15-21.
2. Jensen-Urstad K, Bouvier F, Saltin B, Jensen-Urstad M. High prevalence of arrhythmias in elderly male athletes with a lifelong history of regular strenuous exercise. *Heart* 1998;79:161-4.
3. Mont L, Sambola A, Brugada J, Vacca M, Marrugat J, Elosua R, et al. Long-lasting sport practice and lone atrial fibrillation. *Eur Heart J* 2002;23:477-82.
4. Karjalainen J, Kujala UM, Kaprio J, Sarna S, Viitasalo M. Lone atrial fibrillation in vigorously exercising middle aged men. Case-control study. *BMJ* 1998;316:1784-5.
5. Hegbom F, Sire S, Heldal M, Orning OM, Stavem K, Gjesdal K. Short-term exercise training in patients with chronic atrial fibrillation. Effects on exercise capacity, AV conduction, and quality of life. *J Cardiopulm Rehabil* 2006;26:24-9.
6. Hegbom F, Stavem K, Sire S, Heldal M, Orning OM, Gjesdal K. Effects of short-term exercise training on symptoms and quality of life in patients with chronic atrial fibrillation. *Int J Cardiol* 2007;116:86-92.
7. van den Berg MP, Crijns HJ, Szabo BM, Brouwer J, Lie KI. Effect of exercise on cycle length in atrial flutter. *Br Heart J* 1995;73:263-4.
8. Vanhees L, Kornaat M, Defoor J, Aufdemkampe G, Schepers D, Stevens A, et al. Effect of exercise training in patients with an implantable cardioverter defibrillator. *Eur Heart J* 2004;25:1120-6.
9. de Groot PC, Hjeltnes N, Heijboer AC, Stal W, Birkeland K. Effect of training intensity on physical capacity, lipid profile and insulin sensitivity in early rehabilitation of spinal cord injured individuals. *Spinal Cord* 2003;41:673-9.
10. Hjeltnes N, Aksnes AK, Birkeland KI, Johansen J, Lannem A, Wallberg-Henriksson H. Improved body composition after 8 wk of electrically stimulated leg cycling in tetraplegic patients. *Am J Physiol* 1997;273:R1072-9.
11. Valent LJ, Dallmeijer AJ, Houdijk H, Slootman J, Janssen TW, Hollander AP, et al. The individual relationship between heart rate and oxygen uptake in people with a tetraplegia during exercise. *Spinal Cord* 2007;45:104-11.
12. Bunch TJ, Chandrasekaran K, Gersh BJ, Hammill SC, Hodge DO, Khan AH, et al. The prognostic significance of exercise-induced atrial arrhythmias. *J Am Coll Cardiol* 2004;43:1236-40.
13. Pashkow FJ, Schweikert RA, Wilkoff BL. Exercise testing and training in patients with malignant arrhythmias. *Exerc Sport Sci Rev* 1997;25:235-69.
14. Åstrand P-O, Rodahl K. *Textbook of work physiology*. 3. edn. Singapore: McGraw-Hill Co; 1986.
15. Pollock ML. The quantification of endurance training program. In: Wilmore JH, Ed. *Exercise and sport sciences review*. New York: Academic Press Inc; 1973. p. 155.
16. Dimsdale JE, Hartley LH, Guiney T, Ruskin JN, Greenblatt D. Postexercise peril. Plasma catecholamines and exercise. *JAMA* 1984;251:630-2.

17. Borg G. Perceived exertion as an indicator of somatic stress. *Scand J Rehab Med* 1970; 2:92-8.
18. McCartney N. Role of resistance training in heart disease. *Med Sci Sports Exerc* 1998;30:S396-402.
19. McCartney N. Acute responses to resistance training and safety. *Med Sci Sports Exerc* 1999;31:31-7.
20. US Department of Health and Human Services. Physical activity and health. A report of the Surgeon General. Atlanta: US Department of Health and Human Services, Centers for Disease Control and Prevention, National Center for Chronic Disease Prevention and Health Promotion; 1996.
21. Lee IM, Sesso HD, Paffenbarger RSJ. Physical activity and coronary heart disease risk in men. Does the duration of exercise episodes predict risk? *Circulation* 2000;102:981-6.
22. Kleiger RE, Miller JP, Bigger JTJ, Moss AJ. The multicenter post-infarction research group. Decreased heart rate variability and its association with increased mortality after acute myocardial infarction. *Am J Cardiol* 1987;59:256-62.
23. Ståhle A, Nordlander R, Bergfeldt L. Aerobic group training improves exercise capacity and heart rate variability in elderly patients with a recent coronary event. *Eur Heart J* 1999;20:1638-46.
24. Larsen AI, Gjesdal K, Hall C, Aukrust P, Aarsland T, Dickstein K. Effect of exercise training in patients with heart failure. A pilot study on autonomic balance assessed by heart rate variability. *Eur J Cardiovasc Prev Rehabil* 2004;11:162-7.

31. Hypertension

Authors

Mats Börjesson, MD, PhD, Associate Professor, Department of Medicine, Sahlgrenska University Hospital, Gothenburg, Sweden

Sverre Kjeldsen, MD, PhD, Professor, Department of Cardiology, Oslo University Hospital, Ullevål, Norway

Björn Dahlöf, MD, PhD, Associate Professor, Department of Medicine, Sahlgrenska University Hospital Östra, Gothenburg, Sweden

Summary

Hypertension, that is, above-normal blood pressure, is the most important, modifiable risk factor for cardiovascular disease and mortality. The incidence is increasing in most countries and lifestyle factors are considered to play a decisive role in this development. Obesity, physical inactivity and increased salt intake, in particular, have varying degrees of significance in different populations. As a single risk factor, physical inactivity is believed to be responsible for 5–13 per cent of hypertension today.

Most cases of hypertension are currently still undetected, untreated and/or have not reached therapeutic target value for treatment. This leaves much room for improved treatment, both via an increase in non-pharmacological treatment and via different pharmacological options.

Available data from a recent meta-analysis shows that aerobic fitness training lowers blood pressure approximately 7/5 mmHg in people with mild to moderate hypertension. A single episode of physical activity yields an acute lowering of the blood pressure, so-called post-exercise hypotension. Repeated sessions of physical activity are therefore a strategy for lowering blood pressure, but to attain a lasting reduction of pressure regular exercise is required.

Type of exercise training	Prescription
Aerobic fitness training	40–70 per cent of individual maximal oxygen uptake capacity 5–7 days/week, a minimum of 30 minutes per training session
Strength training	many repetitions and low resistance

Dividing the activity, for example, into 4 sets of 10-minute sessions, can be just as effective as a single 40-minute dose of training per day.

Introduction

Hypertension, or more correctly blood pressure that is too high, is defined as a systolic pressure of ≥ 140 mm Hg and/or a diastolic pressure of ≥ 90 mm Hg, on repeated measurements under standardised conditions (1). In certain high-risk individuals, such as patients with diabetes or kidney disease, the target level for blood pressure treatment is even lower (1).

Hypertension is a significant independent risk factor for cardiovascular disease and the most important modifiable cause of mortality (1). More than 7 million deaths per year across the world are due to hypertension (2). Different national and international organisations have defined the concept of “hypertension” and also divided the disease into degrees of severity based on a number of large epidemiological and treatment studies. The most recently published European guidelines give the following classifications of hypertension (1):

Table 1. Classification of hypertension.

	Systolic	Diastolic
Optimal blood pressure	< 120	< 80
Normal blood pressure	< 130	< 85
High normal blood pressure	130–139	85–89
Grade 1 hypertension (mild)	140–159	90–99
Grade 2 hypertension (moderate)	160–179	100–109
Grade 3 hypertension (severe)	≥ 180	≥ 110
Isolated systolic HT	≥ 140	< 90

It is estimated that at least 25 per cent of the Swedish population (increases with age) suffer from hypertension or take antihypertensive drugs. Globally, the number of people with hypertension is growing dramatically and is estimated to reach close to 1.6 billion people, or 30 per cent of the world’s population by 2025 (3, 4).

Ninety-five per cent of all hypertension is called “essential”, that is, where no single identifiable cause can be shown (1, 5). Essential hypertension is probably the product of many factors that interact in a complex manner in the development of high blood pressure, including genetic factors, environmental factors such as diet, physical inactivity and stress, and psychosocial factors (1, 5). Over the past decades, changes in lifestyle for the worse have been decisive for the increased incidence of hypertension and cardiovascular disease, which is especially pronounced in the developing countries (2). Obesity, physical inactivity and increased salt intake are specific factors of varying importance in different populations, where physical inactivity alone is estimated to represent 5–13 per cent of the risk of developing hypertension (6).

The remaining 5 per cent of people with hypertension suffer from secondary hypertension resulting from, for example, renovascular disease (e.g. renal arterial stenosis), Cushing’s syndrome, pheochromocytoma or coarctation of the aorta. These types of secondary hypertension are best addressed through possible correction of the underlying cause.

Simplified, blood pressure is the result of cardiac output multiplied by the peripheral resistance ($BP = CO \times PR$). In the development of hypertension, there is usually an initial increase in cardiac output, which is a product of heart rate and the heart's stroke volume. An increased resistance gradually develops peripherally in the vascular bed, which in turn leads to secondary, more manifest vascular changes (a vicious circle). At this point, the elevation in blood pressure becomes more established and more resistant to treatment.

Risks of high blood pressure

Untreated, high blood pressure leads to secondary vascular changes consisting of a thickening of the blood vessel walls, impaired endothelium function, and atherosclerosis of the large and medium-sized arteries. In addition, left ventricular hypertrophy of the heart and/or albumin leakage in the kidneys, proteinuria, are seen, and are in themselves also associated with increased mortality and morbidity (1, 7, 8). In order for atherosclerosis to develop, high blood pressure must be present: in the venous low pressure system, for example, atherosclerosis never occurs, while if veins are moved to the arterial system (e.g. via bypass grafting in coronary artery surgery) atherosclerosis develops in a matter of months.

The risk for fatal and non-fatal cardiovascular complications (especially coronary artery disease and stroke), kidney disease and other heart and vascular complications increases parallel to increases in both systolic and diastolic blood pressure levels (8).

Advantages of treating high blood pressure

Pharmacological treatment of hypertension has a long, well-established history. Successful lowering of blood pressure yields fewer cardiovascular events parallel to the degree of blood pressure-lowering (1). According to a meta-analysis of 61 studies covering a million individuals, we can expect a 7 per cent reduction in coronary artery disease and 10 per cent reduction in stroke for every 2 mmHg reduction in blood pressure (8). This is especially important in light of the fact that most cases of hypertension today are still undetected, untreated and/or have not yet reached the appropriate treatment target value (1, 9, 10). There is thus much room for improved treatment via both pharmacological and non-pharmacological methods.

Although there is a lack of formal mortality and morbidity studies with respect to effects of physical activity on high blood pressure, it is well-documented that better fitness/higher levels of physical activity are associated with lower mortality in hypertensives (11), as in non-hypertensive people (12). Recent findings from the LIFE study (Losartan versus Atenolol-based therapy for patients with hypertension and left ventricular hypertrophy) report increased physical activity to be associated with fewer complications, independent of treatment (13).

Effects of physical activity on blood pressure

A single episode of physical activity yields an acute lowering of the blood pressure, so-called post-exercise hypotension. Repeated bouts of physical activity are therefore a strategy for lowering blood pressure (14). This acute effect of increased activity is, however, not the whole truth, as physical activity also has a more lasting effect. These different effects of physical activity are mediated by different mechanisms.

Acute effects

Aerobic fitness training

During dynamic physical activity in healthy persons, such as running, systolic blood pressure normally rises during the exercise itself. In people with hypertension, the elevation in blood pressure can be more pronounced (15). The diastolic pressure remains the same or increases slightly during exertion, mainly due to reduced vasodilating capacity (15).

After dynamic physical activity, the blood pressure falls by about 10–20 mmHg for the next few hours, compared to the person's normal resting blood pressure. This effect is called *post-exercise hypotension*. How long this lowered blood pressure lasts (up to almost 24 hours) after the exercise seems to depend on factors such as the duration and intensity of the physical activity, and whether the activity was continuous or not (16, 17). The same total physical activity, divided into smaller sessions, had a greater pressure-lowering effect than one long session (16, 17). It is believed that the blood pressure-lowering in the 24 hours following physical activity is mediated mainly via a transient reduction in stroke volume (15) and/or modulation of the sympathetic nervous tone.

Strength training/Heavy static training

When it comes to heavy static training (strength training with heavy weights), there is a stronger increase in systolic and diastolic blood pressure during the training itself, than in dynamic exercise. One typically sees a moderate increase in heart rate and cardiac output, combined with an increased in peripheral resistance (18).

Circuit training

According to a new meta-analysis of 9 short-term studies covering 341 individuals, strength training can reduce both systolic and diastolic blood pressure by approximately 3–4 mmHg (19). These studies concluded that moderate intensity strength training is not contraindicated in hypertension, and can even lead to a lowering of blood pressure, although more studies are needed with respect to the long-term effects (19). Circuit training in particular, that is, strength training of large muscle groups with lighter load and more repetitions, appears to be able to lower blood pressure (18) in addition to the positive effect on other risk factors such as insulin resistance.

Qi gong

One small study found an equivalent blood pressure-lowering effect from qi gong as compared to traditional aerobic exercise training (20). Qi gong may therefore constitute an alternative form of exercise for people with hypertension, though possible long-term effects are at present unknown and further studies are needed to confirm these initial findings.

Long-term effects

A recent meta-analysis looking at 72 studies shows that dynamic endurance training lowers blood pressure by approximately 7/5 mmHg (21) in people with mild to moderate essential hypertension. The blood pressure-lowering effect of physical activity is, not surprisingly, highest in people with established hypertension compared to those with normal blood pressure (2/2 mmHg (21)). The effects appear to be the same in people taking antihypertensive drugs (12). The blood pressure-lowering effect of dynamic training is not lasting and requires continually maintained regular activity in order to remain.

Regular physical activity not only lowers the resting blood pressure, but also decreases the blood pressure response during both physical exertion (22) and mental stress (23).

The effects usually apply regardless of age, gender and ethnicity (24). The blood pressure-lowering effect may even be stronger in African-Americans (24).

It has been suggested that the reduction in blood pressure was lower in older individuals, who have often had suffered from hypertension for a longer time and can thus be expected to have more established vascular changes. One study seems to confirm this, by showing that 6 months of exercise training produced lower diastolic- but not systolic blood pressure in people 55–75 years, while no measurable improvement in aorta stiffness was seen (25). On the other hand, a Taiwanese study showed that, in hypertensives 60 years and older, systolic blood pressure was reduced significantly more than in a control group in a 6-month walking intervention (26). Also, Fagard and Cornelissen's meta-analysis did not show any reduced efficacy of physical activity in older hypertensive patients (21).

Indications

Physical activity as primary prevention

A number of longitudinal and cross-sectional studies support a relation between low physical activity and an increased risk for developing hypertension (27). It was recently shown that being physically active during leisure time is associated with a lower risk for developing hypertension, independent of the level of physical activity at work or during transit/commuting to and from work (28).

A primary preventive study observed a 50 per cent reduction in development of hypertension development in patients at risk for hypertension aged 30–44 years: 8.8 per cent (compared to 19.2%) of those who increased physical activity and modified their diet developed hypertension in a 5-year period (29). One group with an increased risk for

coronary disease reduced their coronary risk profile to the same level as those with a low genetic risk through regular physical activity (30). To sum up, regular physical activity appears to protect against development of hypertension and reduces the elevation of pressure in predisposed individuals (31).

Predicting hypertension

Older studies have suggested that individuals with a genetic susceptibility to hypertension, but with normal blood pressure, have an exaggerated blood pressure response to dynamic and isometric exercise compared to controls without an inherited susceptibility. Longitudinal studies indicate a 2–3 times higher risk for later development of hypertension in patients with normal blood pressure who show increased elevation of blood pressure in an exercise test (34). The blood pressure after a stress test also predicts future risk (35). It is likely that the individual clinical prediction of risk for cardiovascular disease could improve if one also took into account these results from a stress test (36).

If one knows that a healthy person has an abnormal blood pressure response in a stress test, the person should be informed of an increased risk for later development of hypertension and be given appropriate advice concerning lifestyle modifications and regular blood pressure check-ups.

Treatment of hypertension/Secondary prevention of cardiovascular disease

The latest European recommendations emphasise that the increase in risk with respect to mortality is greater in people with hypertension with signs of organ damage secondary to hypertension and in those with multiple cardiovascular risk factors (1). Different risk factor models (e.g. SCORE) that take into consideration the importance of combined risk factors are used to individualise risk assessment and treatment (1). Unfortunately, neither fitness nor the physical activity level are included even in the newer risk factor models.

All of the written guidelines for treatment of hypertension give lifestyle factors, including physical activity, a prominent role, especially in milder and early forms of the disease. Often several months of initial lifestyle modification are recommended (only) in people with BP < 160/90 who show no signs of secondary organ damage or have no other risk factors and are thereby judged to be at low risk for cardiovascular events in the short term.

Increased physical activity together with other lifestyle factors are thus the first treatment in mild hypertension with low cardiovascular risk (1). At blood pressures of 160–179/100–109 mmHg, lifestyle changes, including increased physical activity are initiated for a few weeks before pharmacological treatment is added if needed (1). For all individuals with even higher blood pressure levels, regular physical activity is recommended in addition to medication, as long as the blood pressure is controlled at < 180/105 mmHg, with drug therapy (1). The benefits of combining lifestyle measures and pharmacological antihypertensive treatment should always be stressed.

When there are signs of secondary organ damage, competitive- or elite level sports may be contraindicated (see below).

Prescription

Type of activity

Despite recent studies and an increased interest in the importance of strength training for treating risk factors and hypertension, the main recommendation for primary and secondary prevention in hypertension is still dynamic endurance training, for example, aerobic fitness training, walking, swimming and cycling (1, 21). As a supplement, strength training with low resistance, perhaps 40–50 per cent of maximal capacity (RM = repetition maximum), expressed as “moderate intensity” strength training, and many repetitions could be recommended (19, 34).

Intensity

Most studies show that exercising at 40–70 per cent of VO_2 max (= maximal oxygen uptake; corresponding to 50–85% of maximal individual heart rate) lowers the blood pressure at least as well as more intensive exercise (34). This has also been shown in rat trials, where only animals that exercised at this intensity reached a lower blood pressure than the controls (37). Researchers have even claimed that high intensity exercise training (dynamic > 90% of VO_2 max) such as marathons or competitive running may increase blood pressure. More recent studies appear to confirm the impression that low to medium intensity activity, at a level of only 40 per cent of VO_2 max, is enough to achieve acute post-exercise hypotension (38) and to maintain a more lasting lowering of pressure (14, 39).

What this means in terms of specific activities for a particular individual depends on the individual's fitness level (VO_2 max) prior to the start of exercise training. In the USA, an initial exercise test is often recommended, enabling prescription of a suitable activity according to the outcome of the performance test. This type of testing cannot be recommended as a general practice in Sweden and Norway today for several reasons, among them resources and the fact that it would theoretically result in many unnecessary coronary investigations due to abnormal and false-positive stress tests. In practice, it has been shown that an activity that makes one breathless, but still able to carry on a conversation relatively easily, corresponds to a medium intensity activity (11–13/20 on the Borg RPE scale). For many (untrained) people with hypertension, relatively simple activities, such as walking, are therefore sufficient to achieve a reduction in blood pressure.

Duration

Episodes of physical activity of short duration, such as 3–20 minutes, can reduce blood pressure, but longer durations appear to be connected with greater and more lasting lowering of BP (14, 40). 30- to 45-minute sessions are therefore recommended as suitable for achieving a good pressure-lowering effect (1, 14, 15, 21, 40).

In recent years, there has also been increasing discussion of how physical activity divided into shorter durations may have an equally positive effect as activity of longer duration. A brisk 10-minute walk 4 times per day may be as effective at lowering blood pressure as a 40-minute walk once a day (17).

Accumulated physical activity (walking at 50% of VO_2 max), four 10-minute sessions, even yielded significantly longer lasting post-exercise blood pressure-reduction (11 hours versus 7 hours) than one 40-minute walk, in individuals who had not yet developed hypertension (16).

The total weekly dose of activity also seems to be important. Ishikawa-Takata and colleagues were able to show that 60–90 minutes of activity per week was more effective at lowering pressure than 30–60 minutes per week, over 8 weeks in previously sedentary people with hypertension (41).

Frequency

To take maximum advantage of the acute blood pressure-lowering effect of physical activity, which lasts up to 24 hours, it is usually recommended that exercising be performed on most days of the week. However, five or six days can be just as good as seven due to a lower risk of overuse injuries (34).

The increased physical activity will need to be kept up on a continual basis in order for the blood pressure-reducing effect to be maintained. After 4–6 months, the maximal effect on blood pressure, with respect to pressure-lowering, seems to be attained (15). If the individual stops exercising, the blood pressure returns to the same level as before exercising began (34). This can occur as quickly as within 10 days (17), possibly depending on how long the individual exercised regularly.

Table 2. Recommended exercise training.

Type of training	Prescription
Aerobic fitness training	40–70 per cent of maximal oxygen uptake 5–7 days/week, a minimum of 30 minutes per training session
Strength training	many repetitions and low resistance

Dividing up the activity into 4 sets of 10-minute sessions, for example, can be just as effective as a single 40-minute dose of training per day. Examples of suitable activities (according to ESC classification of sports [42]) may include running, table tennis, cross-country skiing, brisk walking, badminton, orienteering, football, tennis (moderate to high intensity dynamic activity but low static activity). As discussed above, however, the activity best suited for an individual is dependent on that fitness level of that individual.

Mechanisms of blood pressure lowering

The pressure-lowering effect of physical activity is mediated by several mechanisms, either interacting or of different importance in different individuals. However, the blood pressure-lowering effect is independent of a reduction in weight or body fat (34).

Endurance training is associated with decreased vascular resistance involving the sympathetic nervous system and the renin-angiotensin system. Simultaneous cardiovascular risk factors are also positively impacted (21).

Reduced sympathetic activity

Increased sympathetic system activity is believed to play a role in the development of essential hypertension. People with increased noradrenaline levels show a reduction in blood pressure from physical activity parallel to the decrease in noradrenaline levels (21, 43).

Especially, early in the course of hypertension, physical activity plays an important role in reducing blood pressure (34), as the blood pressure increase at this stage is mediated by an increase in cardiac output and secondary, more manifest, vascular changes with increased peripheral resistance has not yet developed. According to Fagard and Cornelissen's meta-analysis (21), physical activity reduces noradrenaline levels by approximately 30 per cent.

Increase in vasodilating agents

It has been proposed that an increase in endorphins as a result of physical activity has a blood pressure-lowering effect post-exercise (44).

Reduced insulin resistance

The link between hypertension and the metabolic syndrome, including obesity, dyslipidemia and increased insulin resistance, has been shown, and has been suggested to play a role in the development of hypertension (5). Physical activity reduces insulin resistance and thereby secondary hyperinsulinemia (45), which is a potential pressure-lowering mediator. Regular physical activity also reduces the risk of developing diabetes (46).

Changes in kidney function

Because the kidneys play a big role in maintaining abnormally high blood pressure, through regulation of sodium and water balance, and thereby cardiac output, some of exercise's positive effects may be mediated by the kidneys (47). For example, plasma-renin is reduced by 20 per cent by aerobic training in hypertensives (21).

Regression of left ventricular hypertrophy

Physical activity leads to a reduction of the left ventricular hypertrophy associated with hypertension to the same degree as pharmacological treatment with diuretics (45). Left ventricular hypertrophy in itself is an independent risk factor for cardiovascular disease and regression produces independent prognostic value (13).

Effects on other risk factors

It is impossible to analyse the effects of physical activity on blood pressure, and thereby reduced mortality/morbidity, in isolation, without taking into consideration the positive effects on other risk factors for cardiovascular disease, such as obesity, hyperinsulinemia and hypertriglyceridemia (21). In addition to the effect on blood pressure alone, the added effects of physical activity reduce overall risk for the patient (1).

Other effects

Known effects of physical activity like improved physical performance (45) lead to a better prognosis in people with hypertension. One controlled study showed that well-trained individuals with hypertension had a lower mortality than untrained individuals with normal blood pressure (48). Physical activity also reduces inflammatory parameters such as hs-CRP, which is a risk marker for cardiovascular disease. Also hs-CRP is inversely related to level of fitness in people with hypertension (49). Physical activity is associated with improved quality of life in addition to blood pressure reduction in these patients (50). A new study that compares different treatment regimens shows physical activity to be most strongly associated with improved quality of life in several dimensions, most markedly in older women (51).

Interactions

Beta blockers

Treatment with beta blockers leads to a lowering of the maximal heart rate by about 30 beats per minute (15). In addition to lowering resting blood pressure, beta blockers reduce the activity-induced elevation of systolic blood pressure. In comparison with other antihypertensive drugs, the rate pressure product (heart rate multiplied by blood pressure) increases less at a given level of physical intensity with beta blocker therapy (52). This yields a reduction of the patient's maximal performance capacity (15), but is also a positive effect, especially in people with abnormally large increases in blood pressure during dynamic exercise. For normal, medium intensity activity, this likely plays a minor role. But a person with higher demands on performance, for example, a more fit, active runner, may find it hard to accept beta blocker therapy.

Whether or not beta blockade reduces resting energy expenditure and/or reduce weight loss from physical activity (53) has been discussed. However, it is possible that beta blockade mitigates the increase in the energy expenditure observed in physical activity (54).

Diuretics

Has potentially negative effects through increasing the risk for dehydration in warm weather, as well as increasing the risk for hypokalaemia (15).

ACE inhibitors

ACE inhibitors are a first choice for physically active people with hypertension. They can help to lower blood pressure after activity, which can create problems in dehydrated patients, for example, patients with diarrhoea, especially in warmer climates.

Angiotensin receptor blockers

Another possible first choice. At present, there is limited experience and the same precaution must be taken as with ACE inhibitors. They have been shown to reduce blood pressure response in connection with physical exercise (55).

Calcium channel blockers

A possible drug of first choice. There is a risk for a drop in blood pressure immediately after exercise due to vasodilatation (widening of the blood vessels).

Alpha blockers

Alpha blockers are a good secondary alternative, with minor impact on maximal performance capacity (56, 57).

Contraindications

A systolic blood pressure over 200 mmHg or a diastolic pressure over 115 mmHg should contraindicate physical activity until the pressure has stabilised below these levels with blood pressure-lowering medication (15). The American College of Sports Medicines (ACSM) guidelines recommend that individuals with blood pressure > 180/105 first begin pharmacological treatment before any regular physical activity is resumed or started (relative contraindication) (34).

ACSM also recommends, however, that caution be taken with very intensive dynamic training (90–100% of VO_2 max) such as extreme elite sports in people with hypertension, as well as heavier strength training (weight lifting etc.) (34). In the case of strenuous

strength training, extremely high blood pressure can be measured in the heart's left ventricle (> 300 mmHg), which is potentially dangerous. ACSM recommends some caution in the case of established hypertrophy of the left ventricle, which means that these individuals should possibly exercise in the lower region of the proposed interval (40–70% of VO_2 max) (34).

Side-effects

Increase in blood pressure

When the intensity of dynamic or static training is too high, the result can instead be elevated blood pressure (see above).

Stroke

Side-effects can also arise due to the high blood pressure that can develop in people with hypertension exercising at too high an intensity. Research has, however, not been able to show an increased incidence of stroke in these people, and not from strength training either.

General side-effects

Joint injuries from overloading (often concurrent obesity and hypertension) may develop.

Dehydration, electrolyte disorders, hypotension

These side-effects can arise to varying degrees, due in part to the physical activity and in part to possible medications.

Sudden death

A very small increased risk for sudden cardiac death *during* physical activity is offset by a significantly greater decrease in the long-term morbidity/mortality risk.

References

1. ESC. 2007 guidelines for the management of arterial hypertension. *J Hypertension* 2007;25:1105-87.
2. WHO. The World Health Report. Reducing risks, promoting healthy life. Geneva: World Health Organization; 2002.
3. Hajjar I, Kotchen T. Trends in prevalence, awareness, treatment and control of hypertension in the United States, 1998–2000. *JAMA* 2003;290:199-206.
4. Kearney PM, Whelton P, Reynolds K, Muntner P, Whelton PK, He J. Global burden of hypertension. Analysis of worldwide data. *Lancet* 2005;365:217-23.
5. Dahlöf B. Hypertonihandboken [The Hypertension Handbook]. Sollentuna: Merck, Sharp & Dohme (Sweden) AB; 2000.
6. Geleijnse JM, Kok FJ, Grobbee DE. Impact of dietary and lifestyle factors on the prevalence of hypertension in Western populations. *Eur J Public Health* 2004;14:235-9.
7. Vasan RS, Larson MG, Leip EP, Evans JC, O'Donnell CJ, Kannel WB, et al. Impact of high-normal blood pressure on the risk of cardiovascular disease. *N Engl J Med* 2001;345:1291-7.
8. Lewington S, Clarke R, Qizilbach N, Peto R, Collins R, Prospective Studies Collaboration. Age-specific relevance of usual blood pressure to vascular mortality. A meta-analysis of individual data from one million adults in 61 prospective studies. *Lancet* 2002;360:1903-13.
9. Börjesson M, Dahlöf B. Fysisk aktivitet har en nyckelroll i hypertonibehandlingen [Physical activity has a key role in hypertension therapy]. *Läkartidningen* 2005;102:123-9.
10. Wolf-Meier K, Cooper RS, Kramer H, Banegas JR, Giampaoli S, Joffres MR. Hypertension treatment and control in five European countries, Canada, and the United States. *Hypertension* 2004;43:10-7.
11. Evenson KR, Stevens J, Thomas R, Cai J. Effect of cardiorespiratory fitness on mortality among hypertensive and normotensive women and men. *Epidemiology* 2004;15:565-72.
12. Sandvik L, Erikssen J, Thaulow E, Erikssen G, Mundal R, Rodahl K. Physical fitness as a predictor of mortality in healthy middle-aged men. *N Engl J Med* 1993;328:533-7.
13. Devereaux RB, Wachtell K, Gerds E, Boman K, Nieminen MS, Papademitrou V. Prognostic significance of left ventricular mass change during treatment of hypertension. *JAMA* 2004;292:2350-6.
14. Guidry MA, Blanchard BE, Thompson PD, Maresh CM, Seip RL, Taylor AL, et al. The influence of short and long duration on the blood pressure response to an acute bout of dynamic exercise. *Am Heart J* 2006;151:1322.e5-12.
15. Gordon NF. Hypertension. In: Durstine JL, Ed. ACSM's exercise management for persons with chronic diseases and disabilities. Champaign (IL): Human Kinetics; 1997.
16. Park S, Rink LD, Wallace JP. Accumulation of physical activity leads to greater blood pressure reduction than a single continuous session, in prehypertension. *J Hypertens* 2006;24:1761-70.

17. Elley R, Bagrie E, Arroll B. Do snacks of exercise lower blood pressure? A randomised crossover trial. *NZMJ* 2006;119:1-9.
18. Tipton CM. Exercise and hypertension. In: Shephard RJ, Miller HSJ, Eds. *Exercise and the heart in health and disease*. 2. edn. New York: Marcel Dekker, Inc.; 1999, pp. 463-88.
19. Cornelissen VA, Fagard RH. Effect of resistance training on resting blood pressure. A meta-analysis of randomized controlled trials. *J Hypertens* 2005;23:251-9.
20. Cheung BMY, Lo JLF, Fong DYJ, Chan MY, Wong SHT, Wong VCW, et al. Randomised controlled trial of qigong in the treatment of mild essential hypertension. *J Hum Hypertension* 2005;19:697-704.
21. Fagard RH, Cornelissen VA. Effect of exercise on blood pressure control in hypertensive patients. *Eur J Cardiovasc Prev Rehabil* 2007;14:12-7.
22. Ketelhut RG, Franz IW, Scholze J. Regular exercise as an effective approach in anti-hypertensive therapy. *Med Sci Sports Exerc* 2004;36:4-8.
23. Santaella DF, Ara jo EA, Ortega KC, Tinucci T, Mion DJ, Negrao CE, et al. Aftereffects of exercise and relaxation on blood pressure. *Clin J Sport Med* 2006;16:341-7.
24. Svetkey LP, Erlinger TP, Vollmer WM, Feldstein A, Cooper LS, Appel LJ, et al. Effect of lifestyle modifications on blood pressure by race, sex, hypertension status, and age. *J Hum Hypertension* 2005;19:21-31.
25. Stewart KJ, Bacher AC, Turner KL, Fleg JL, Hees PS, Shapiro EP, et al. Effect of exercise on blood pressure in older patients. *Arch Intern Med* 2005;165:756-62.
26. Lee L-L, Arthur A, Avis M. Evaluating a community-based walking intervention for hypertensive older people in Taiwan. A randomized controlled trial. *Prev Med* 2006;44:160-6.
27. Paffenbarger RS Jr, Jung DL, Leung RW, Hyde RT. Physical activity and hypertension. An epidemiological view. *Ann Med* 1991;23:319-27.
28. Barengo NC, Hu G, Kastarinen M, Lakka TA, Pekkarinen H, Nissanen A, et al. Low physical activity as a predictor for antihypertensive drug treatment in 25–64 year-old population in Eastern and South-Western Finland. *J Hypertens* 2005;23:293-9.
29. Stamler R, Stamler J, Gosch FC, Civinelli J, Fishman J, McKeever P, et al. Primary prevention of hypertension by nutritional-hygienic means. *JAMA* 1989;262:1801-7.
30. Greenfield JR, Samaras K, Campbell LV, Jenkins AB, Kelly PJ, Spector TD, et al. Physical activity reduces genetic susceptibility to increased central systolic pressure augmentation. A study of female twins. *J Am Coll Cardiol* 2003;42:264-70.
31. Pescatello LS, Franklin BA, Fagard R, Farquhar WB, Kelley GA, Ray CA. American College of Sports Medicine. Position stand. Exercise and hypertension. *Med Sci Sports Exerc* 2004;36:533-53.
32. Wilson MF, Sung BH, Pincomb GA, Lovallo WR. Exaggerated pressure response to exercise in men at risk for systemic hypertension. *Am J Cardiol* 1990;66:731-6.
33. Benbassat J, Fromm PF. Blood pressure response to exercise as a predictor of hypertension. *Arch Intern Med* 1986;146:2053-5.

34. American College of Sports Medicine. Position stand. Physical activity, physical fitness, and hypertension. *Med Sci Sports Exerc* 1993;25:i-x.
35. Laukkanen JA, Kurl S, Salonen R, Lakka TA, Rauramaa R, Salonen JT. Systolic blood pressure during recovery from exercise and the risk of acute myocardial infarction in middle-aged men. *Hypertension* 2004;44:820-5.
36. Erikssen G, Bodegard J, Bjornholt JV, Liestol K, Thelle DS, Erikssen J. Exercise testing of healthy men in a new perspective. From diagnosis to prognosis. *Eur Heart J* 2004;25:978-86.
37. Tipton CM, MacMahon S, Leininger JR, Pauli EL, Lauber C. Exercise training and incidence of cerebrovascular lesions in stroke-prone spontaneously hypertensive rats. *J Appl Physiol* 1990;68:1080-5.
38. Smelker CL, Foster C, Maher MA, Martinez R, Porcari JP. Effect of exercise intensity on postexercise hypotension. *J Cardiopulm Rehabil* 2004;24:269-73.
39. Pescatello LS, Guiridy MA, Blanchard BE, Kerr A, Taylor AR, Johnson AN, et al. Exercise intensity alters postexercise hypotension. *J Hypertens* 2004;22:1881-8.
40. Mach C, Foster C, Brice G, Mikat RP, Porcari JP. Effect of exercise duration on post-exercise hypotension. *J Cardiopulm Rehabil* 2005;25:366-9.
41. Ishikawa-Takata K, Ohta T, Tanaka H. How much exercise is required to reduce blood pressure in essential hypertensives. A dose-response study. *Am J Hypertens* 2003;16:629-33.
42. Pelliccia A, Fagard R, Bjornstad HH, Anastassakis A, Arbustini E, Assanelli D, et al. Recommendations for competitive sports participation in athletes with cardiovascular disease. ESC Report. *Eur Heart J* 2005;26:1422-45.
43. Duncan JJ, Farr JE, Upton J, Hagan RD, Oglesby ME, Blair SN. The effects of aerobic exercise on plasma catecholamines and blood pressure in patients with mild hypertension. *JAMA* 1985;254:2609-13.
44. Thorén P, Floras JS, Hoffman P, Seals DR. Endorphins and exercise. Physiological mechanisms approach. *Med Sci Sports Exerc* 1990;22:17-28.
45. Rinder MR, Spina RJ, Peterson LR, Koenig CJ, Florence CR, Ehsani AA. Comparison of effects of exercise and diuretic on left ventricular geometry, mass, and insulin resistance in older hypertensive adults. *Am J Physiol Regul Integr Comp Physiol* 2004;287:R360-8.
46. Fossum E, Gleim GW, Kjeldsen SE, Kizer JR, Julius S, Devereaux RB, et al. The effect of baseline physical activity on cardiovascular outcomes and new-onset diabetes in patients treated for hypertension and left ventricular hypertrophy. The LIFE study. *J Intern Med* 2007;262:439-48.
47. Kenney WL, Zambraski EJ. Physical activity in human hypertension. A mechanisms approach. *Sports Med* 1984;1:459-73.
48. Blair SN, Kohl HWI, Paffenbarger Jr RS, Clark DG, Cooper KH, Gibbons LW. Physical fitness and all-cause mortality. A prospective study of healthy men and women. *JAMA* 1989;262:2395-401.

49. Hjelstuen A, Anderssen SA, Holme I, Seljeflot I, Klemsdal TO. Markers of inflammation are inversely related to physical activity and fitness in sedentary men with treated hypertension. *Am J Hypertens* 2006;19:669-75.
50. Tsai J-C, Yang H-Y, Wang W-H, Hsieh M-H, Chen P-T, Kao C-C, et al. The beneficial effect of regular endurance exercise training on blood pressure and quality of life in patients with hypertension. *Clin Exp Hypert* 2004;26:255-65.
51. Fernandez FJC, Garcia MTM, Alvarez CR, Giron MJI, Aguirre-Jaime A. Is there an association between physical exercise and the quality of life of hypertensive patients? *Scand J Med Sci Sports* 2007;17:348-55.
52. Kokkinos P, Chrysohoou C, Panagiotakos D, Narayan P, Greenberg M, Singh S. Beta-blockade mitigates exercise blood pressure in hypertensive male patients. *J Am Coll Cardiol* 2006;47:794-8.
53. Gondoni LA, Tagliaferri MA, Titon AM, Nibbio F, Liuzzi A, Leonetti G. Effect of chronic treatment with beta-blockers on resting energy expenditure in obese hypertensive patients during a low-calorie and physical training program. *Nutr Metab Cardiovasc Dis* 2003;13:232-7.
54. Bélanger M, Boulay P. Effect of an aerobic exercise training program on resting metabolic rate in chronically beta-adrenergic blocked hypertensive patients. *J Cardiopulm Rehabil* 2005;25:354-60.
55. Nashar K, Nguyen JP, Jesri A, Morrow JD, Egan BM. Angiotensin receptor blockade improves arterial distensibility and reduces exercise-induced pressor responses in obese hypertensive patients with the metabolic syndrome. *Am J Hypertens* 2004;17:477-82.
56. Fahrenbach MC, Yurgalevitch SM, Zmuda JM, Thompson PD. Effect of doxazosin or atenolol on exercise performance in physically active, hypertensive men. *Am J Cardiol* 1995;75:258-63.
57. Tomten SE, Kjeldsen SE, Nilsson S, Westheim AS. Effect of alpha-1 adrenoceptor blockade on maximal VO_2 and endurance capacity in well trained athletic hypertensive men. *Am J Hypertens* 1994;7:603-8.

32. Kidney disease (chronic) and kidney transplant

Author

Susanne Heiwe, PT, PhD, the Physiotherapy Clinic, Karolinska University Hospital and Department of Medicine and Department of Clinical Sciences, Karolinska Institutet, Stockholm, Sweden

Summary

Chronic kidney failure is a toxic syndrome caused by a deteriorating kidney function to excrete waste products, regulate the salt-water and acid-base balance and secrete endocrine hormones. Chronic kidney failure leads to progressive fatigue and a gradually decreased physical work capacity concurrently with the deterioration of kidney function. Without physical exercise, the maximum physical work capacity and muscle strength are reduced by up to 50 per cent compared with the normal expected value once the patient commences regular dialysis treatment. However, this reduction in capacity can be prevented and the patient can maintain normal muscle function plus physical and functional ability if a regular physical exercise programme is started pre-uraemia. Patients with chronic kidney failure have the same ability as healthy people of the same age to improve their muscle function and increase their physical and functional ability regardless of how far the disease has progressed. Physical exercise is an important part of the treatment of chronic kidney failure. Recommended physical activities include walking, cycling and strength training.

Training method	Example	Intensity	Frequency (times/week)	Duration
Aerobic fitness training	Walking Interval training on ergometer bike	70% of $\text{VO}_2 \text{ max}^*$ RPE** 14/20	3	60 min.
Strength training	Sequence training Individual training with wrist weights for resistance.	80% of 1 RM***	3	1–2 sets of 8–10 repetitions (reps)
Muscular endurance training	Sequence training Individual training with wrist weights for resistance.	50% of 1 RM	3	Max. number of reps 13–15 acc. to Borg's RPE scale.
Functional training (i.e. walking, balance and coordination training)	Walking for example on a treadmill or balance mat Knee bends. Walking up and down stairs. Standing up from sitting.		3	Max. walking duration and max. reps of other exercises. 13–15 acc. to Borg's RPE scale

* $\text{VO}_2 \text{ max}$ = maximum oxygen uptake capacity.

** RPE = Borg's Ratings of Perceived Exertion, RPE scale (41).

*** RM = Repetition Maximum. 1 RM corresponds to the maximum weight that can be lifted through the entire exercise movement one time.

Definition

Chronic kidney failure is a global health problem. Consequences of chronic kidney failure include the loss of kidney function and cardiovascular disease. Chronic kidney disease can either be defined as kidney damage or a glomerular filtration rate (GFR) of less than 60 ml/min/1.73 m² for a minimum period of three months and diagnosed without knowledge of its cause. There are five stages of chronic kidney disease:

1. GFR \geq 90 Kidney damage with normal or increased kidney function
2. GFR 60–89 Kidney damage with a slight decrease in kidney function
3. GFR 30–59 Average kidney function
4. GFR 15–29 Serious decrease in kidney function
5. GFR < 15 Kidney failure

Prevalence/Incidence

At the end of year 1999, the prevalence or the number of patients in active uraemia treatment in Sweden was 693 for every million people with an annual incidence of 120 new patients for every million people. The average age of patients who start treatment is 66 years, i.e. most patients being treated for uraemia today are pensioners (1).

Cause

The most common causes of chronic kidney disease is chronic glomerulonephritis (30%) followed by diabetes mellitus (18%), hereditary polycystic kidney disease (13%), chronic pyelonephritis (10%) and nephrosclerosis (9%). As for the remaining 20 per cent, the cause cannot be determined (1).

Risk factors

- Hypertension with unsatisfactory blood pressure control.
- Diabetes Types 1 and 2 with proteinuria and unsatisfactorily controlled blood pressure and blood glucose levels.

Pathophysiology

Chronic kidney disease leads to an irreversible loss of the glomerular filtration rate (GFR) and toxic syndrome caused by the kidney's failing ability to excrete waste products, regulate the salt-water and acid-base balance and secrete endocrine hormones. The majority of patients with chronic kidney disease have cardiovascular complications. Around 70 per cent of all patients have left ventricular hypertrophy at the start of dialysis and the risk of a dialysis patient dying due to cardiovascular complications, independent of age, is the same as for an average 80-year old (2, 3).

Symptoms

Patients with chronic kidney disease experience a change in taste sensations accompanied by a dry mouth, thirst and a metallic taste in the mouth. These symptoms together with nausea, abdominal bloating and diarrhoea often lead to a loss of appetite and weight loss (4, 5). Uraemia is in itself a catabolic condition with a reduced ability to excrete nitrogenous waste products, i.e. breakdown products of proteins. This in turn leads to a lower intake of proteins and energy which together with metabolic acidosis leads to muscle degradation and muscle atrophy (6, 7). As a result, patients develop anaemia with further fatigue and weakness. Salt and water retention and the impact it has on the renin-angiotensin system leads to hypertension and cardiovascular complications. Displacements in the calcium phosphate vitamin D parathyroid hormone axis cause decalcification of the skeleton with the added risk of calcification of soft tissue. Untreated uraemia generally leads to fatigue, increased fragility, sleep disorders, itchiness, leg cramps, weight loss, muscle hypotrophy and a reduced physical ability (8, 9).

Diagnosics

The initial diagnosis involves assessing the electrolyte status with serum creatinine and serum urea followed by a GFR determination using iohexol or Chrom EDTA clearance.

Current treatment principles

Pre-uremic stage – GFR < 25 ml/min

At this stage of the disease, the prime objective is to slow down the uraemia progression and alleviate uremic symptoms in addition to preparing the patient for dialysis and/or kidney transplant. The basis of slowing down the uraemia progression is a blood pressure control which should give a systolic blood pressure of < 140 mm Hg and a diastolic blood pressure of < 90 mm Hg. A good metabolic control is also important for persons with diabetes. A reduced dietary protein intake has an alleviating effect on uremic symptoms and is also likely to affect the uraemia progression although there is a lack of documented, clear-cut scientific evidence to support this. Renal anaemia is treated with erythropoietin and iron supplements while hypocalcaemia and vitamin D deficiency are treated with lime substitution and active vitamin D. Hyperphosphatemia is treated with phosphate binding medication. Patients with metabolic acidosis are prescribed sodium bicarbonate tablets. During the pre-uraemia stage, the patient is prepared for the necessity of regular dialysis treatments and informed about the two types of dialysis available, i.e. haemodialysis and peritoneal dialysis and if required, the possibility of a kidney transplant. Patients are also required to undergo a minor operation to gain entry/give access to the bloodstream so that dialysis can be performed.

Haemodialysis – GRF about 5–10 ml/min

Access to the patient's bloodstream is attained by way of an arteriovenous fistula which involves stitching together an artery and a vein in the arm to create a large quick flowing and thick walled artery that can cope with the insertion of two needles three times a week. Using a dialysis machine, the blood is then pumped into a dialysis filter that cleans it. The treatment can either take place at a specialist dialysis ward or at home once the patient has been trained to use a self-test haemodialysis machine.

Peritoneal dialysis – GRF about 5–10 ml/min

Access to the patient's bloodstream is attained in the form of a small catheter inserted into the abdominal cavity below the navel. Here, the richly vascular peritoneum is used as dialysis membrane. Four times a day and night, the cavity of the abdomen is filled with around two litres of a glucose solution with a balanced saline content. The solution remains in the cavity of the abdomen for approximately six hours, absorbing waste products and excess fluids before being drained through the catheter. This method is called Continuous Ambulatory Peritoneal Dialysis (CAPD). Alternatively, a machine can be used to pump smaller volumes of fluids in and out of the abdomen cavity of the patient in short cycles during the night when the patient is sleeping for a maximum period of 9–10 hours. This method is called Continuous Cyclic Peritoneal Dialysis (CCPD). Peritoneal dialysis can be managed by the patient at home.

Kidney transplant

A kidney transplant can be performed using a kidney from a close relative or, if this option is not available to the patient, from a recently deceased person. In the latter case, the patient is put on a waiting list for a so-called necro kidney.

*Effects of physical activity**Exercise response*

A number of studies have shown that persons with chronic kidney disease who are either in the pre-uraemia stage (10–13) or undergoing dialysis treatment (14–19) have the same relative ability to improve their aerobic fitness, muscle strength and endurance as healthy individuals of the same age. However, chronic disease patients need to exercise regularly to counter the decline in their aerobic fitness (20), muscle strength and endurance (21), which will otherwise occur because of the strong catabolic effects of kidney failure.

Natural progression with no exercise

The natural progression of chronic kidney failure is a gradual decline in aerobic capacity during the pre-uraemia stage to around 50 to 60 per cent of the capacity normally seen in a person of the same age and gender undergoing dialysis treatment (22, 23). The most significant etiological factors of this decline is renal anaemia and muscle weakness (23). Today, renal anaemia is treated with erythropoietin and muscle fatigue with physical exercise.

A lack of physical exercise can lead to the individual not being able to lead an active and social life. This may result in deteriorating health-related quality of life and an increasing need for the society's involvement. Adults suffering with chronic kidney disease are also at greater risk for cardiovascular diseases, a risk that increases with inactivity. Excessively degraded physical strength could also result in delayed medical acceptance for a kidney transplant, as the individual in question may not be deemed able to cope with the side-effects of medical treatment.

The level of physical activity spontaneously increases following a successful kidney transplant, but will not return to normal without physical exercise (24, 25).

Acute effects

Patients with chronic kidney disease may suffer from high blood pressure, fluid retention and hyperkalaemia. All of these conditions may worsen due to a lack of physical exercise. Exercise in itself may increase the level of serum potassium due, for example, to exercise induced acidosis (26).

Long-term effects

Firstly, physical exercise increases the maximum working capacity (10–19), muscle strength and muscle endurance (10, 13, 16, 19). Patients in the pre-uraemia stage usually manage to normalise their working capacity, muscle strength and endurance after three months of regular exercise, while patients undergoing dialysis treatment usually see a significant improvement after three to six months of regular exercise. Patients in the pre-uraemia stage or undergoing dialysis treatment can increase their functional ability through exercise (13, 18, 27). Physical exercise will also lead to a decline in depressive symptoms and an enhanced self-esteem and quality of life (28, 29). Moreover, physical exercise has a favourable influence on a number of cardiovascular risk factors in patients with chronic kidney disease, resulting in improved blood pressure and lipid control (30) and increased insulin sensitivity (31) as well as an increased heart-rate variability, vagal activity and a lower frequency of cardiac arrhythmia (32, 33).

Following a successful kidney transplant, physical exercise may result in nearly normal physical capacity (34, 35). Muscle strength training has proven to result in improved muscular strength in adult kidney transplant recipients (36, 37).

So as to reduce the risk of cardiovascular disease, physical exercise should be combined with lifestyle changes, such as an improved diet (38).

Indications

Primary prevention

No human studies indicate that physical exercise has a direct, primary preventive influence on the onset of chronic kidney failure.

Secondary prevention

No human studies indicate that physical exercise prolongs or combats uraemia progression. Nevertheless, an indirect secondary preventive effect on uraemia progression should not be ruled out, bearing in mind the favourable influence that physical exercise has on the blood pressure control of patients with high blood pressure and on the blood glucose control of patients with diabetes.

Prescription

Muscle fatigue is the most restrictive factor for the majority of patients. Hence, exercise should initially emphasize muscle strength and endurance training plus balance and coordination training to be complemented with fitness training at a later stage. See Table 1 for a description of various forms of exercise, intensity, duration and frequency.

Table 1. Description of various forms of exercise.

Training method	Example	Intensity	Frequency (times/week)	Duration
Aerobic fitness training	Walking Interval training on ergometer bike	70% of VO ₂ max* RPE** 14/20	3	60 min.
Strength training	Sequence training Individual training with wrist weights for resistance.	80% of 1 RM***	3	1–2 sets of 8–10 repetitions (reps)
Muscular endurance training	Sequence training Individual training with wrist weights for resistance.	50% of 1 RM	3	Max. number of reps 13–15 acc. to Borg’s RPE scale.
Functional training (i.e. walking, balance and coordination training)	Walking for example on a treadmill or balance mat. Knee bends. Walking up and down stairs. Standing up from sitting.		3	Max. walking duration and max. reps of other exercises, 13–15 acc. to Borg’s RPE scale.

* VO₂ max = maximum oxygen uptake capacity.

** RPE = Borg’s Ratings of Perceived Exertion, RPE scale (41).

*** RM = Repetition Maximum. 1 RM corresponds to the maximum weight that can be lifted through the entire exercise movement one time.

Special considerations in connection with chronic kidney failure

For all patients

- In view of the high prevalence of cardiovascular diseases, the patient must have a stable heart status, well-controlled blood pressured and not suffer from excessive fluid retention.
- Patients with chronic kidney failure can easily develop tendinitis (inflammation of tendon/tendon bone attachment). Subsequently, it is important to incorporate a long warming up and cooling down period as well as flexibility and stretching exercises in the exercise programme. In addition, the intensity and duration of the exercise programme should increase in stages.
- Patients with a polycystic kidney disease should not perform exercises that involve jumping about or lead to added abdominal pressure due to the risk of mechanical damage to the kidney.

Patients undergoing haemodialysis treatment

- Blood pressure must not be taken using the arm with the arteriovenous fistula or graft as the blood vessels could be damaged if the blood flow is blocked.
- Patients with a central dialysis catheter, most often positioned on the neck or chest, should avoid lifting their arms above their head and avoid neck movements as the catheter may be detached and damaged.

Patients undergoing peritoneal dialysis treatment

- The dialysis fluid must be drained from the abdominal cavity before exercise. If dialysis fluid remains in the abdominal cavity, the patient is at risk of having a hernia and/or damage his/her pelvic floor muscles. Furthermore, it renders it more difficult to exercise with the correct intensity and duration.

Functional tests/need for health checks

- All patients with chronic kidney failure should be referred to a specialist physiotherapist by the attending practitioner for functional tests and physical training.
- The attending practitioner should determine whether or not referring a patient for an exercise test is necessary from a medical viewpoint before the patient starts a training programme. An exercise test is sometimes suggested prior to the start of a training programme to optimise exercise dose and intensity.
- The attending physiotherapist will provide information and encourage the patient to exercise and will also put together an individual exercise programme or try to get the patient involved in a group exercise. Regular functional tests are also carried out.
- The attending practitioner monitors the patient's clinical status and regular laboratory test results.

Table 2 illustrates clinical test methods for the assessment of physical and functional ability and evaluation of exercise response.

Table 2. Test methods for the assessment of physical and functional ability and evaluation of exercise response.

Physical capacity	A standardised symptom-limited exercise test on an ergometer bicycle (39) for the purpose of assessing patient complaints of tired legs, breathlessness and possible chest pains in accordance with the Borg CR10 scale (40). The patient's level of exertion is also measured in accordance with the Borg RPE scale (41).
Muscle strength	One repetition maximum (1RM) (42).
Dynamic muscular endurance	Maximum number of muscle contractions with a load equivalent to 50% of 1 RM and a fixed frequency (13). "Standing heel-rise test" (43).
Static muscular endurance	The total time in seconds that the patient manages to maintain an isometric muscle contraction such as a fully extended knee with a load equivalent to 50% of 1 RM (13).
Functional capacity	A 6-minute walking test (44, 45) for the purpose of assessing patient complaints of tired legs, breathlessness and possible chest pains in accordance with the Borg CR10 scale (40). The patient's level of exertion before and after the test is also measured in accordance with the Borg RPE scale (41). Timed Up & Go (46).

Interactions with drug therapy

Beta blockers

To-date, studies have not shown that beta blockers have a negative effect on the ability to improve fitness. Patients with chronic kidney failure often show symptoms of autonomic neuropathy with a lower maximum pulse during maximum exertion than other healthy individuals of the same age and gender not treated with beta blockers.

ACE inhibitors/All antagonists

An increasing number of people with chronic kidney disease are treated with these preparations partly as an anti-hypertensive therapy and partly to delay the uraemia progression in patients with diabetes and chronic glomerulonephritis. Patients in the pre-uraemia stage of chronic kidney disease who are treated with these preparations are often sensitive to dehydration with a risk of hypertension and must be extra cautious and drink plenty of fluids during exercise to compensate for the loss of fluid from sweating.

Erythropoietin

Patients with chronic kidney failure receive a substitute treatment of erythropoietin, i.e. they are treated with erythropoietin to compensate for their own inadequate production of erythropoietin. A lower target haemoglobin concentration is a condition if the patient should have the strength to accomplish adequate training and obtain the desired exercise response.

Essential amino acids (Aminess N)

Patients in the pre-uraemia stage are treated with a protein-reduced diet, which could result in a deficiency of essential amino acids. In order to maximise the response of the skeletal muscles to exercise, patients should be prescribed supplements of essential amino acids.

Sodium bicarbonate

Untreated metabolic acidosis leads to an increased frequency of leg cramps and muscle catabolism that are likely to have a negative effect on the response of the skeletal muscles to exercise. Therefore, the patients should be fully compensated for the metabolic acidosis and prescribed a supplement of sodium bicarbonate tablets.

Calcium tablets and active vitamin D

Hypocalcaemia and hyperparathyroidism lead to a higher frequency of muscle skeletal symptoms. Calcium tablets and active vitamin D should then be used to obtain calcium homeostasis.

Contraindications

Absolute

- Acute infection
- Uncontrolled hypertension (systolic blood pressure > 180 mm Hg and/or diastolic blood pressure > 105 mm Hg).
- Unstable angina.
- Severe cardiac arrhythmia.
- Uncontrolled diabetes.
- Hyperkalaemia.

Relative

- Weight gain of more than 5 per cent of the estimated 'dry weight' between haemodialysis treatments.
- Expressed anaemia and arteriosclerotic cardiovascular disease.

Risks

To-date, no patient has had a serious incident during or after exercise. However, all exercise should be carried out in accordance with the guidelines above under the supervision of a specialist physiotherapist and on the recommendations of a doctor.

References

1. Schön S. Svenskt register för aktiv uremivård 2001 [The Swedish Registry for Active Treatment of Uremia 2001]. The Nephrology Clinic, KSS, SE 541 85 Skövde (srau.kss@vgregion.se).
2. Foley RN, Parfrey PS, Kent GM, Harnett JD, Murray DC, Barre PE. Long-term evolution of cardiomyopathy in dialysis patients. *Kidney Int* 1998 Nov;54:1720-5.
3. Foley RN, Parfrey PS, Sarnak MJ. Epidemiology of cardiovascular disease in chronic renal disease. *J Am Soc Nephrol* 1998 Dec;9:S16-23.
4. Fernström A, Hylander B, Rössner S. Taste acuity in patients with chronic renal failure. *Clin Nephrol* 1966;45:169-74.
5. Klang B, Björvell H, Clyne N. Quality of life in predialytic uremic patients. *Quality of Life Research* 1996;5:109-16.
6. Guarnieri G, Toigo G, Situlin R, et al. Muscle biopsy studies in chronically uremic patients. Evidence for malnutrition. *Kidney Int* 1983;24:187S-93S.
7. Williams B, Hattersley J, Hayward F, Walls J. Metabolic acidosis and skeletal muscle adaptation to low protein diets in chronic renal failure. *Kidn Int* 1991;40:779-86.
8. Klang B, Clyne N. Well-being and functional ability in uremic patients before and after having started dialysis treatment. *Scand J of Caring Sciences* 1997;11:159-66.
9. Moreno F, Lopez Gomez JM, Sanz-Guajardo D, Jofre R, Valderrabano F. Quality of life in dialysis patients. A Spanish multicentre study. *Nephrol Dial Transpl* 1996;11:125-9.
10. Clyne N, Ekholm J, Jogestrand T, Lins LE, Pehrsson SK. Effects of exercise training in predialytic uremic patients. *Nephron* 1991;59:84-9.
11. Eidemak I, Haaber AB, Feldt-Rasmussen B, Kanstrup IL, Strandgaard S. Exercise training and the progression of chronic renal failure. *Nephron* 1997;75:36-40.
12. Boyce M, Robergs R, Avasthi P, Roldan A, Foster A, Montner A, et al. Exercise training by individuals with predialysis renal failure. Cardiorespiratory endurance, hypertension and renal function. *Am J Kidn Dis* 1997;30:180-92.
13. Heiwe S, Tollbäck A, Clyne N. Twelve weeks of exercise training increases muscle function and walking capacity in elderly predialysis patients and healthy subjects. *Nephron* 2001;1:48-56.
14. Goldberg AP, Geltman EM, Hagberg JM, Gavin JR 3rd, Delmez JA, Carney RM, et al. Therapeutic benefits of exercise training for hemodialysis patients. *Kidney International* 1983;24:303S-9.
15. Painter PL, Nelson-Worel JN, Hill MM, Thornbery DR, Shelp WR, Harrington AR, et al. Effects of exercise training during hemodialysis. *Nephron* 1986;43:87-92.
16. Kouidi E, Albani M, Natsis K, Megalopoulos A, Gigis P, Guiba-Tziampiri O, et al. The effects of exercise training on muscle atrophy in hemodialysis patients. *Nephrol Dial Transpl* 1998;13:685-99.

17. Krause R, Abel HH, Bennhold I, Koepchen HP. Long-term cardiovascular and metabolic adaptation to bedside ergometer training in hemodialysis patients. In Doll-Tepper G, Dahms C, Doll B, v Sezam H, red. *Adapted physical activity*. Heidelberg: Springer-Verlag; 1990. pp. 299-304.
18. Koufaki P, Mercer TH, Naish PF. Effects of exercise training on aerobic and functional capacity of patients with end-stage renal disease. *Clinical Physiology and Functional Imaging* 2002;22:115-24.
19. Ota S, Takahashi K, Suzuki H, Nishimura S, Makino H, Ota Z, et al. Exercise rehabilitation for elderly patients on chronic hemodialysis. *Geriatric Nephrology and Urology* 1996;5:157-65.
20. Clyne N, Jogestrand T, Lins L-E, Pehrsson SK. Progressive decline in renal function induces a gradual decrease in total hemoglobin and exercise capacity. *Nephron* 1994;67:322-6.
21. Kettner-Melsheimer A, Weiss M, Huber W. Physical work capacity in chronic renal disease. *Int J Artif Organs* 1987;10:23-30.
22. Painter P, Messer-Rehak D, Hanson P, Zimmerman SW, Glass NR. Exercise capacity in hemodialysis, CAPD and renal transplant patients. *Nephron* 1986;42:47-51.
23. Clyne N, Jogestrand T, Lins LE, Pehrsson SK, Ekelund LG. Factors limiting physical working capacity in predialytic uraemic patients. *Acta Med Scand* 1987;222:183-90.
24. Nielens H, Lejeune TM, Lalaoui A, Squifflet JP, Pirson Y, Goffin E. Increase of physical activity level after successful renal transplantation. A 5-year follow-up study. *Nephrol Dial Transplant* 2001 Jan;16:134-40.
25. Painter P. Exercise for patients with chronic disease. Physician responsibility. *Curr Sports Med Rep* 2003 Jun;2:173-80.
26. Daul AE, Völker K, Hering D, Schäfers RF, Philipp T. Exercise-induced changes in serum potassium in patients with chronic renal failure. *J Amer Soc Nephrol* 1996;7:1348.
27. Mercer TH, Naish PF, Gleeson NP, Crawford C. Low volume exercise rehabilitation improves functional capacity and self-reported functional status of dialysis patients. *American Journal of Physical Medicine and Rehabilitation* 2002;81:162-7.
28. Carney RM, McKevitt PM, Goldberg AP, Hagberg J, Delmez JA, Harter HR. Psychological effects of exercise training in hemodialysis patients. *Nephron* 1983;33:179-81.
29. Kouidi E, Iacovides A, Iordanidis P, Vassiliou S, Deligiannis A, Ierodiakonou C, et al. Exercise renal rehabilitation program (ERRP). Psychosocial effects. *Nephron* 1997;77:2:152-8.
30. Goldberg AP, Geltman EM, Gavin Jr 3d, Carney RM, Hagberg JM, Delmez JA, et al. Exercise training reduces coronary risk and effectively rehabilitates hemodialysis patients. *Nephron* 1986;42:311-6.
31. Eidemak I, Feldt-Rasmussen B, Kanstrup IL, Nielsen SL, Schmitz O, Strandgaard S. Insulin resistance and hyperinsulinaemia in mild to moderate progressive chronic renal failure and its association with aerobic work capacity. *Diabetologia* 1995;38:565-72.

32. Deligiannis A, Kouidi E, Tourkantonis A. The effects of physical training on heart rate variability in hemodialysis patients. *Am J Cardiol* 1999;84:197-202.
33. Deligiannis A, Kouidi E, Tassoulas E, Gigis P, Tourkantonis A, Coats A. Cardiac response to physical training in hemodialysis patients. An echocardiographic study at rest and during exercise. *Int J Cardiol* 1999;70:253-66.
34. Painter C, Monestier M, Bonin B, Bona CA. Functional and molecular studies of V genes expressed in autoantibodies. *Immunol Rev* 1986 Dec;94:75-98.
35. Warburton DE, Sheel AW, Hodges AN, Stewart IB, Yoshida EM, Levy RD, et al. Effects of upper extremity exercise training on peak aerobic and anaerobic fitness in patients after transplantation. *Am J Cardiol* 2004 Apr 1;93:939-43.
36. Horber FF, Hoopeler H, Scheidegger JR, Grünig BE, Howald H, Frey FJ. Impact of physical training on the ultrastructure of midthigh muscle in normal subjects and in patients treated with glucocorticoids. *J Clin Invest* 1987 Apr;79:1181-90.
37. LaPier TK. Glucocorticoid-induced muscle atrophy. The role of exercise in treatment and prevention. Review. *J Cardiopulm Rehabil* 1997 Mar-Apr;17:76-84.
38. Painter P. Exercise for patients with chronic disease. Physician responsibility. *Curr Sports Med Rep* 2003 Jun;2:173-80.
39. Åström H, Jonsson B. Design of exercise tests with special reference to heart patients. *Br Heart J* 1976;38:289-96.
40. Borg G. Perceived exertion as an indicator of somatic stress. *Scand J Rehab Med* 1970;2-3:92-8.
41. Borg G. A category scale with ratio properties for intermodal and interindividual comparisons. In Geissler HG, Petzolds P, red. *Psychophysical judgement and the process of perception*. Berlin: VEB Deutscher Verlag der Wissenschaften; 1982.
42. McDonough M, Davies C. Adaptive response of mammalian skeletal muscle to exercise with high loads. *Eur J Appl Physiol* 1984;52:139-55.
43. Lunsford BR, Perry J. The standing heel-rise test for the ankle plantar flexion. Criterion for normal. *Phys Ther* 1995;75:694-8.
44. Guyatt G, Sullivan M, Thompson P, et al. The 6-minute walk: a new measure of exercise capacity in patients with chronic heart failure. *Can Med Assoc J* 1985;132:919-23.
45. Guyatt G, Thompson P, Berman L, Sullivan M, Townsend M, Jones N, et al. How should we measure function in patients with chronic heart and lung disease? *J Chronic Dis* 1985;38:517-24.
46. Podsiadlo D, Richardson S. The "Timed Up and Go". A test of basic functional mobility for frail elderly persons. *J Am Geriatr Soc* 1991;39:142-8, 517-2.

33. Lipids

Authors

Lena Björck, RN, MSc, Research Laboratory, Sahlgrenska University Hospital, Gothenburg, Sweden

Dag S Thelle, MD, PhD, Professor, University of Oslo, IMB, Department of Biostatistics, Oslo, Norway

Summary

The studies conducted thus far on physical activity and blood fats (lipids) have studied the effect of aerobic fitness training and strength training. There is no evidence today to suggest that strength training has a positive effect on blood lipids, whereas fitness training has an impact on triglycerides, HDL cholesterol (High Density Lipoprotein or alpha-lipoprotein) and to some degree also LDL cholesterol (Low Density Lipoproteins). The activity, which must be regular, 30–45 minutes most days of the week, should be of an intensity equivalent to 40–70 per cent of maximal capacity (after a gradual warm-up). The duration of each training session is dependent on the individual's possibilities and capacity, but should include a minimum of 30 minutes per session. To achieve maximal blood lipid-lowering effect, the training volume should be between 24–32 km per week, for example, a brisk walk or jogging, corresponding to 1200–2000 kcal per week. This calorie expenditure per week is associated with a 5–8 per cent increase in HDL cholesterol and a decrease in triglycerides of approximately 10 per cent.

Suitable activities include jogging, running, skiing, fitness classes, brisk walks, cycling, swimming, and racquet and ball sports.

Background

“The alpha-lipoprotein fraction is smaller and is more constant in amount, practically independent of the diet, and is considered to be non-atherogenic or even protective because its concentration in the serum is higher in premenopausal women than in men and tends to be reduced in CHD patients” (1).

The above was stated by Ancel Keys and Henry Blackburn almost 50 years ago, and they were the first to suggest that alpha-lipoprotein, or what we now call HDL cholesterol, had a protective effect with respect to cardiovascular disease. The interest in HDL cholesterol was weak for many years, overshadowed by studies that concentrated on total cholesterol, triglycerides and lipoproteins of a lower density. The reason for this is that researchers had not observed an ecological connection between alpha-lipoprotein levels and cardiovascular disease in Hawaii, Japan and Finland, three countries with large variations in the risk for heart diseases (2, 3). It was not until the brothers George and Norman Miller published their hypothesis that HDL had an anti-atherogenic effect, and the results of two prospective studies, from Tromsø and Framingham, became known in 1977, that HDL cholesterol received attention as an interesting intermediary risk factor (4–6).

Functional mechanisms

What kind of a causal relationship is there?

What lies behind the reasoning of the relation between physical activity and blood lipids (HDL cholesterol fraction) is that it is HDL that has been shown to have the strongest relation to physical activity.

Figure 1 shows a simplified model of the relationship between physical inactivity and the risk for cardiovascular disease.

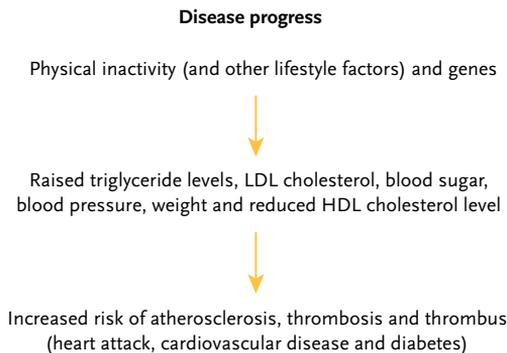


Figure 1. Model of the relation between physical inactivity and the risk for cardiovascular disease.

An individual's behaviour with regard to physical activity (such as frequency, intensity and duration) is assumed to affect different biological factors that play a part in one or more pathogenic mechanisms, for example, hardening of the arteries, risk of blood clots, or development of high blood pressure. After a certain time, the cells alter their function and thereby also the organ's function, which later yields clinical symptoms as a result of this. The above model gives a general picture of the disease mechanism that forms the basis for discussion about the relation between physical activity and blood lipids.

Lipids and their relation to cardiovascular disease

Lipid is a collective term that includes a "diverse range of compounds, like fatty acids and their derivatives, carotenoids, terpenes, steroids and bile acids, which are all soluble in organic solvents such as diethyl ether, benzene, chloroform or methanol" (7). There are also a number of shorter water-soluble fatty acids, but for the sake of discussion it suffices to know that fat molecules ingested with food or produced by the body are included in the term "lipid". The terms *lipid* and *fat* will be used synonymously in this chapter. Fat is a necessary part of our diet, containing the fat-soluble vitamins A, D, E and K, as well as being an energy source. In order to be transported in the blood, fat molecules must bind to proteins and form water-soluble complexes such as lipoproteins.

Lipoproteins transport triglycerides and cholesterol in the blood. Triglycerides are made up of three fatty acids and one glycerol molecule. They make up the body's energy stores and are also an integral part of the cell membrane. 95 per cent of the body's energy is stored as triglycerides. Cholesterol is a complex substance that is produced in the liver or supplied via the animal products in our diet and absorbed in the intestine. Cholesterol is also a crucial part of the cell membrane and is a precursor to sex hormones such as testosterone and progesterone.

Lipoproteins are distinguished according to size and density into different classes (see Table 1): chylomicrons, very low density lipoprotein (VLDL), low density lipoprotein (LDL) and high density lipoprotein (HDL). These can be broken down according to particle size and intermediary groups such as intermediate-density lipoprotein (IDL) and lipoprotein (a) or Lp(a). Lp(a) is a variant of LDL with the addition of a glycoprotein, Apo(a), attached to the LDL core, which is related to LDL and strongly associated with an increased risk of cardiovascular disease.

Table 1. Some physical characteristics and mean composition of lipoprotein fractions from normal triglycerides.

	Chylomicrons ¹	VLDL ²	IDL ³	LDL ⁴	HDL ⁵
Density (g/ml)					
Lower limit	–	0.96	1.006	1.019	1.063
Upper limit	0.96	1.006	1.019	1.063	1.21
Size (nm)	75–1200	30–80	25–35	19–25	5–12

1. Chylomicrons = belong to the lipoprotein group. After meals, cholesterol and triglycerides are transported via chylomicrons from the intestine.

2. VLDL = Very Low Density Lipoprotein.

3. IDL = Intermediate Density Lipoprotein.

4. LDL = Low Density Lipoprotein.

5. HDL = High Density Lipoprotein.

All of these lipoprotein complexes have been shown to be associated with cardiovascular disease at increased levels of LDL cholesterol. VLDL cholesterol, IDL cholesterol and Lp(a) are associated with a higher risk, while increased HDL cholesterol shows a lower risk. The sum of LDL-, VLDL-, IDL- and HDL cholesterol is what is measured as *total cholesterol*.

HDL cholesterol levels are higher in women than in men, and lower among diabetic patients, smokers, and in people who are overweight or physically inactive (8).

Alcohol intake also increases the level of HDL cholesterol (9).

Effects of physical activity

The studies that are of interest are either observation studies, in which physical activity is recorded and serum lipid levels in otherwise healthy individuals are measured, or investigations where the individual exercises at different intensities and the effect of blood fats is studied.

The relation between blood lipids and physical activity is known from observation studies from the 1970s, where individuals who reported low physical activity had 8 per cent higher total cholesterol than the very physically active subjects (10). Correspondingly, physically active men had 7 per cent- and physically active women 6 per cent higher HDL cholesterol than the inactive subjects (8). Findings from observation studies are, however, not the same as proof of a relation. Smaller trials have, however, been able to confirm the relation from epidemiological studies. Table 2 shows the effects of fitness training on blood lipids in women and men. After a programme lasting 12 weeks, HDL cholesterol increased by up to 16 per cent and was directly associated with the amount of exercise training (11–13). Shorter periods of training do not yield the same effect as longer ones. The effect on HDL cholesterol is more pronounced if the initial level is low. The increase in HDL cholesterol is related to the reduction in triglyceride levels, where HDL cholesterol and triglycerides have an inverse relationship (14), that is, the higher the triglycerides, the lower the HDL cholesterol. Physical activity has a direct effect on triglyceride

levels, with close to 30 per cent reduction if the initial value is high. At lower levels, the reduction will be approximately 10 per cent. The effect on LDL cholesterol is less impressive than the effect on triglycerides and HDL cholesterol.

Studies of physical activity in the population

A number of studies have demonstrated the relation between physical activity and lower blood fat levels, and individuals who are physically active have lower total cholesterol and higher HDL cholesterol. Data from a number of studies has shown that there is a break-point regarding the amount of physical activity needed to achieve a beneficial effect on blood lipids, corresponding to 24 to 32 km of brisk walking or jogging per week, that is, a calorie expenditure of 1200 to 2200 kcal is required to gain the desired effect. This weekly calorie expenditure is associated with an increase in HDL cholesterol of 0.5–0.8 mmol per litre and a decrease of triglycerides of 0.1–0.2 mmol per litre (15). Physical activity has a certain lowering effect on total cholesterol and LDL cholesterol.

After menopause, women have higher total cholesterol and higher LDL cholesterol. Physical activity has a beneficial effect in both older men and women, possibly with a greater reduction in LDL compared to younger individuals, as well as a reduction in total cholesterol (16, 17). Even individuals with a physically strenuous job or who are very active in daily life have higher HDL and total cholesterol (18).

Prescription

The studies conducted to date have looked at the effect of fitness training and strength training (resistance training). At present, there is no evidence to suggest that strength training has any particular positive effect on lipids, whereas fitness training has a positive effect on triglycerides, HDL cholesterol, and to a certain extent also LDL cholesterol (see Table 2).

Table 2. Expected protective effect of blood lipids and lipoproteins after a fitness training programme for men, women and seniors.

Lipid/Lipoprotein	Relation to CHD*	Effects of physical activity
Chylomicrons	Positive	↔
VLDL ¹	Somewhat positive	↔
IDL ²	Somewhat positive	↔
LDL ³	Positive	↔
Lp(a) ⁴	Clearly positive	↔
HDL ⁵	Clearly reverse	↑
Total cholesterol	Clearly positive	↔
Triglycerides	Somewhat positive	↓

*CHD = Coronary Heart Disease.

1. VLDL = Very Low Density Lipoprotein.

2. IDL = Intermediate Density Lipoprotein.

3. LDL = Low Density Lipoprotein.

4. Lp(a) is a variant of LDL with the addition of a glycoprotein, Apo(a), attached to the LDL core.

5. HDL = High Density Lipoprotein.

↑ = Increased levels in blood, ↓ = Reduced levels in blood, ↔ = Little or no change in blood levels.

Increased physical activity is a supplement to other interventions, such as changes in diet and/or drugs, aimed at improving the blood lipid profile. The activity, which must be regular and carried out most days of the week, should be of an intensity equivalent to 40–70 per cent of maximal capacity (after a gradual warm-up). The duration of each exercise session is dependent on the individual’s possibilities and capacity, but should include a minimum of 30–45 minutes per day, and can likely be broken up into several shorter sessions throughout the day, though no shorter than 10 minutes per session.

There is currently an ongoing discussion as to the true meaning of HDL cholesterol as a causal factor or simply as a risk marker for coronary artery disease. Thus far, one clinical trial has been conducted with HDL-raisers, but without the expected positive result (19, 20). The only known method of actively raising HDL cholesterol without side-effects is physical activity.

References

1. Keys A, Blackburn H. Background of the patient with coronary heart disease. *Progr in Cardio Dis* 1963;6:14-44.
2. Keys A, Kimura N, Kusunokawa A, Bronte-Stewart B, Larsen N, Keys MH. Lessons from serum cholesterol studies in Japan, Hawaii and Los Angeles. *Ann Intern Med* 1958;48:83-94.
3. Karvonen M, Orma E, Keys A, Fidanza F, Brozek J. Cigarette smoking, serum-cholesterol, blood-pressure, and body fatness. Observations in Finland. *Lancet* 1959;1:492-4.
4. Miller GJ, Miller NE. Plasma-high-density-lipoprotein concentration and development of ischaemic heart disease. *Lancet* 1975;1:16-9.
5. Miller NE, Thelle DS, Førde OH, Mjos OD. The Tromso Heart Study. High-density lipoprotein and coronary heart disease. A prospective case-control study. *Lancet* 1977;1:965-8.
6. Gordon T, Castelli WP, Hjortland MC, Kannel WB, Dawber TR. High density lipoprotein as a protective factor against coronary heart disease. The Framingham Study. *Am J Med* 1977;62:707-14.
7. Christie WW. Lipid analysis. New York: Pergamon Press; 1982.
8. Førde OH, Thelle DS, Arnesen E, Mjos OD. Distribution of high density lipoprotein cholesterol according to relative body weight, cigarette smoking and leisure time physical activity. The Cardiovascular Disease Study in Finnmark 1977. *Acta Med Scand* 1986;219:167-71.
9. Schaefer EJ, Lamon-Fava S, Ordovas JM, Cohn SD, Schaefer MM, Castelli WP, et al. Factors associated with low and elevated plasma high density lipoprotein cholesterol and apolipoprotein A-I levels in the Framingham Offspring Study. *J Lipid Res* 1994; 35:871-82.
10. Thelle DS, Førde OH, Try K, Lehmann EH. The Tromso Heart Study. Methods and main results of the cross-sectional study. *Acta Med Scand* 1976;200:107-18.
11. Wood PD, Haskell WL, Blair SN, Williams PT, Krauss RM, Lindgren FT, et al. Increased exercise level and plasma lipoprotein concentrations. A one-year, randomized, controlled study in sedentary, middle-aged men. *Metabolism* 1983;32:31-9.
12. Dengel DR, Hagberg JM, Pratley RE, Rogus EM, Goldberg AP. Improvements in blood pressure, glucose metabolism, and lipoprotein lipids after aerobic exercise plus weight loss in obese, hypertensive middle-aged men. *Metabolism* 1998;47:1075-82.
13. Hagberg JM, Ferrell RE, Dengel DR, Wilund KR. Exercise training-induced blood pressure and plasma lipid improvements in hypertensives may be genotype dependent. *Hypertension* 1999;34:18-23.
14. Thelle DS, Cramp DG, Patel I, Walker M, Marr JW, Shaper AG. Total cholesterol, high density lipoprotein-cholesterol and triglycerides after a standardized high-fat meal. *Hum Nutr Clin Nutr* 1982;36:469-74.

15. Durstine JL, Grandjean PW, Davis PG, Ferguson MA, Alderson NL, DuBose KD. Blood lipid and lipoprotein adaptations to exercise. A quantitative analysis. *Sports Med* 2001;31:1033-62.
16. Boardley D, Fahlman M, Topp R, Morgan AL, McNevin N. The impact of exercise training on blood lipids in older adults. *Am J Geriatr Cardiol* 2007;16:30-5.
17. Pescatello LS, Murphy D, Costanzo D. Low-intensity physical activity benefits blood lipids and lipoproteins in older adults living at home. *Age Ageing* 2000;29:433-9.
18. Barengo NC, Kastarinen M, Lakka T, Nissinen A, Tuomilehto J. Different forms of physical activity and cardiovascular risk factors among 24–64-year-old men and women in Finland. *Eur J Cardiovasc Prev Rehabil* 2006;13:51-9.
19. Nicola P. Pfizer halts clinical trials of torcetrapib due to patients safety concerns. In: National Electronic Library of Medicines /<http://nelm.nhs.uk>); 2006.
20. Duriez P, Bordet R, Berthelot P. The strange case of Dr HDL and Mr HDL. Does a NO's story illuminate the mystery of HDL's dark side uncovered by Dr HDL's drug targeting CETP? *Med Hypotheses* 2007;69:752-7.

34. *Metabolic syndrome*

Author

Mai-Lis Hellénus, MD, PhD, Professor, Department of Medicine, Karolinska Institutet and Karolinska University Hospital, Stockholm, Sweden

Summary

Metabolic syndrome consists of a cluster of factors such as abdominal obesity, insulin resistance, dyslipidemia and hypertension. In addition, factors such as impaired fibrinolysis, inflammatory status, high levels of uric acid and a fatty liver are not uncommon. Metabolic syndrome increases the risk of cardiovascular disease, type 2 diabetes, dementia, Alzheimer's disease and some common forms of cancer. An increase in the prevalence of metabolic syndrome is evident in the general population, primarily owing to a lack of physical activity in combination with a high energy intake, a poor diet, stress and psychosocial factors.

A high level of physical activity and fitness reduce the risk of metabolic syndrome while physical activity affects all integral components. In order to prevent and treat metabolic syndrome, it is recommended that sedentary time is reduced and moderately strenuous physical activity carried out daily for a minimum of 30, but preferably 60 minutes. Further positive effects are achieved by exercising moderately and regularly 2–3 times a week for a minimum of 30 minutes. Aerobic fitness training may well be combined with adapted strength training.

Individuals with metabolic syndrome are often at risk of developing secondary conditions and it is consequently important that individual risk analyses are performed and, if required, examinations and recommendations for physical activities are adapted to the individual in question. Equally important is the follow-up of the physical activity recommendations.

Definition

Prevalence/Incidence

Many international reports show an alarmingly high prevalence of metabolic syndrome among men, women and children. The prevalence of metabolic syndrome varies depending

on the definition used (1). According to a European Study (DECODE) of 6,156 men and 5,356 women without diabetes and between the ages of 30–89 from Finland, Sweden, Poland, the Netherlands, the United Kingdom and Italy, the age-standardised prevalence was 16 per cent among men and 14 per cent among women (2). The study also showed that the prevalence increases with age. American studies show a high prevalence among both men and women (3–5).

Today, metabolic syndrome is also common in populations with a traditionally low rate of cardiovascular diseases and mortality. In Greece, the overall prevalence of metabolic syndrome among healthy, middle-aged men ($n = 1,128$) and women ($n = 1,154$) was 20 per cent (6). The prevalence of metabolic syndrome was higher among the men (25%) than the women (15%) and increased with age. The prevalence of metabolic syndrome is considerably higher in high-risk populations. Approximately 53 per cent (7) of obese patients in Italy were found to suffer from metabolic syndrome compared with 45 per cent (8) of patients in the Netherlands with cardiovascular disease. Among 3,770 English women aged 60–79, just under 30 per cent were found to suffer from metabolic syndrome (9). The result of a Swedish population-based study of 4,232 individuals (aged 60, 78% participation rate) showed that 26 per cent of the men and 19 per cent of the women had metabolic syndrome according to NCEP/ATP III criteria (10, 11). Extremely alarming are reports on the high prevalence of metabolic syndrome among children and young people (12).

Risk factors for metabolic syndrome

Metabolic syndrome is the result of complex interactions between environment and genes. A change in lifestyle with less physical activity, unhealthy food and drinking habits and an imbalance between energy intake and energy expenditure in addition to chronic stress and psychosocial factors are some fundamentally important reasons for the increase in metabolic syndrome (13–19).

Despite the difficulties in measuring physical activity and the variety of methods used, a large number of international and national reports agree that the majority of adults and children are physically inactive today. Only about 20 per cent of the population is sufficiently physically active (13).

A majority of more recent studies indicate a strong link between the level of physical activity or fitness and the prevalence of metabolic syndrome. A Swedish study of men and women aged 60 showed a strong dose-response relationship between reported physical activity in leisure time and metabolic syndrome (11). Individuals exercising moderately at least twice a week for a minimum of 30 minutes had a 70 per cent lower risk of developing metabolic syndrome compared with individuals reporting low levels of leisure time physical activity (less than 2 hours light physical activity per week). The relationship was not affected by factors such as gender, education, civil status, smoking or intake of fruit, vegetables and alcohol (see Figure 1).

Similar findings were made in other cross-sectional studies and prospective studies where an inactive lifestyle and/or poor fitness were closely linked to the existence of metabolic syndrome (20–25).

Many national and international reports indicate an increase in overweight and obesity among both children and adults (26–28). The waist circumference of children and adults has increased comparatively more than their weight. Today, nearly half of Sweden's adult population is overweight (BMI \geq 25) and approximately 10 per cent suffers from obesity (BMI \geq 30). The prevalence has doubled since the 1980s (29). Abdominal obesity is closely linked to metabolic syndrome (30).

The pathogenesis of metabolic syndrome

The pathogenesis of metabolic syndrome is complex with interactions between genetic and lifestyle factors (19, 20, 31). Overweight and abdominal obesity are principal and recurrent clinical characteristics which, together with insulin resistance in skeletal muscles, adipose tissue and liver, play a central role in the development of metabolic syndrome. A typical dyslipidemia with high levels of triglycerides, low HDL and high ApoB plus small, dense, oxidation prone and very atherogenic LDL particles is a common and important subcomponent of metabolic syndrome. Post-prandial (following food intake) hyperlipidemia and high levels of serum-free fatty acids have also been found. Hypertension is another recurrent condition. Other subcomponents include a reduced fibrinolytic capacity, inflammatory activity, high levels of uric acid, a reduced endothelial function and fatty liver (29, 30). See Fact Box 1.

Most common symptoms – what are the consequences of metabolic syndrome?

Metabolic syndrome is often a symptom-free condition detected in connection with a health check or other contact with the healthcare services. The various subcomponents of metabolic syndrome are common in an adult population (11), but are often symptom-free. High blood pressure, obesity, incipient diabetes or a silent coronary artery disease may naturally result in symptoms such as excessive fatigue or exertion-induced discomfort or chest pains. Abdominal obesity may lead to snoring, insomnia, daily fatigue and a lower quality of life (31, 32).

Metabolic syndrome increases the risk of major public illnesses, type 2 diabetes, dementia, Alzheimer's disease plus other more common forms of cancer. Many cross-sectional studies and prospective studies indicate that individuals with metabolic syndrome have an elevated risk of cardiovascular disease (1, 2, 34–37). This elevated risk applies to all cardiovascular diseases (1, 2) plus cognitive function, dementia and mortality in general (36–38). It also applies to both men and women (39).

The risk of developing type 2 diabetes is considerably higher for individuals with metabolic syndrome while diabetics with metabolic syndrome have a worse prognosis than those without (34, 35).

A number of epidemiological studies over the past few years have also linked metabolic syndrome to prostate cancer (40) and other more common forms of cancer such as colon cancer and breast cancer (41–44). Hyperinsulinemia may be one mechanistic link (45).

Diagnosics

Metabolic syndrome has many definitions. However, all definitions include abdominal obesity/overweight, insulin resistance and disturbed glucose/insulin homeostasis, typical dyslipidemia and hypertension. The four most commonly used definitions are those proposed by WHO (46), the European Group for the Study of Insulin Resistance (EGIR) (47), the National Cholesterol Education Program (NCEP/ATP III) (10) and the International Diabetes Federation (IDF) (48). More recently, a separate definition relating to children was also proposed (12). The definition proposed by American NCEP/ATP II is most commonly used and appropriate for clinical practice (see the Fact Box below).

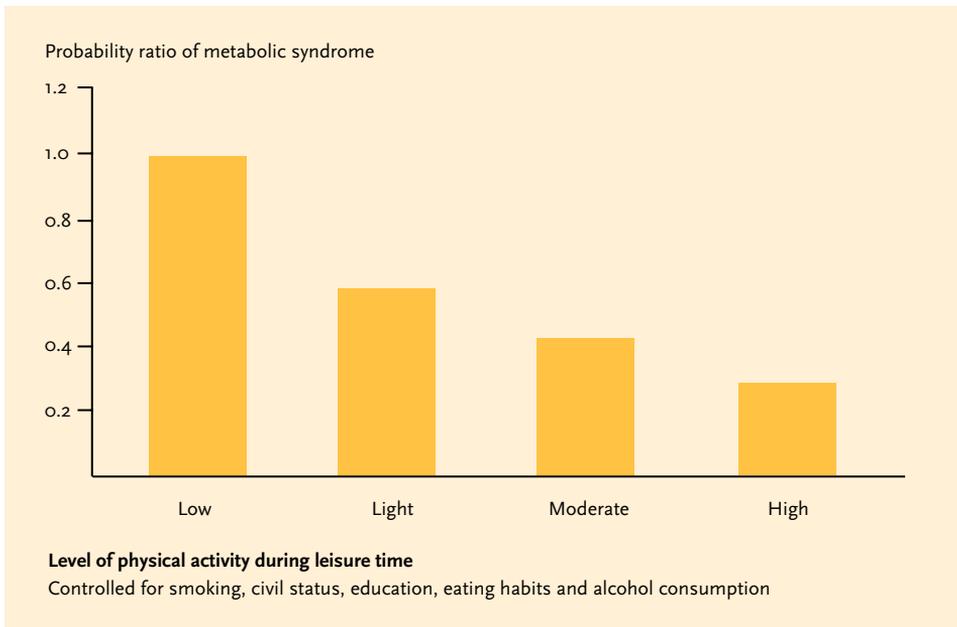


Figure 1. A dose-response relationship between the level of physical activity during leisure time and the occurrence of metabolic syndrome in 60-year-old Swedish men and women.

Criteria for clinical diagnosis of metabolic syndrome, NCEP/ATP III:

Any of five constitute diagnosis of metabolic syndrome

- Waist circumference > 102 cm for men and > 88 cm for women.
- S-triglycerides \geq 1.7 mmol/l (or drug treatment)
- HDL cholesterol < 1.03 mmol/l for men and < 1.3 for women (or drug treatment)
- Blood pressure \geq 130/ 85 mm Hg (or drug treatment)
- Fasting glucose \geq 5.6 mmol/l (or drug treatment)

Treatment

Prevention and treatment of metabolic syndrome is based on changing lifestyle (1, 19, 49–53). Increased physical activity is the cornerstone in the treatment of metabolic syndrome. The treatment should always be individualised and focus on weight loss and reduced abdominal obesity through increased physical activity and improved food patterns. Recommendations on the consumption of food and alcohol follow general dietary guidelines, but have to be adapted to the individual (53). Advice on nicotine replacement therapy and stress management may also be relevant.

Pharmacological treatment of different subcomponents is also a possibility (51, 52) as lifestyle modifications will not counteract the effects of any treatment. However, today's advanced and professional prevention and treatments are always based on changing lifestyle.

Treatment is aimed at lessening the risk of future diseases through the reduction of various risk factors.

Effects of physical activity

Close association between physical activity and metabolic syndrome

A growing number of epidemiological studies indicate that there is a strong dose-response relationship between the level of physical activity or fitness and metabolic syndrome (see "Most common symptoms") (25). For example, the prevalence of the metabolic syndrome is 70 per cent lower in 60-year-old men and women who exercise with a moderate intensity at least twice a week, even when taking into account other relevant factors such as food and alcohol intake, education and smoking (11).

Physical activity reduces the health risk associated with metabolic syndrome

Overweight or abdominally obese men and women who exercise regularly are at much lower risk of cardiovascular disease than those who are inactive (55–57). In an American study, more than 21,000 men aged 30–83 were monitored during an average period of eight years for the purpose of studying cardiovascular diseases and mortality. A fit but

overweight or abdominally obese man was at a lower risk than an unfit man of normal weight (54). When monitoring 88,000 healthy middle-aged women for a period of 20 years as part of the so-called Nurses Health Study, it became evident that physical activity could reduce the risk of coronary disease associated with abdominal obesity (55).

A systematic review of the literature and 10 prospective studies regarding the relationship between physical activity and the risk of type 2 diabetes showed that regular daily physical activity of a moderate intensity for a minimum of 30 minutes would considerably reduce the risk of coronary heart disease (56). A Finnish study monitored 2,017 healthy men and 2,352 healthy women aged 45–64 for an average period of 9.4 years. The risk of type 2 diabetes was reduced by 60–70 per cent among subjects reporting a high level of physical activity compared with subjects reporting a low level of physical activity. These findings apply equally to overweight people and people of a normal weight (57). A follow-up of 1,263 American men with type 2 diabetes showed that the mortality was 50 per cent lower among those reporting to be physically active during a prospective 15-year study (58). Similar findings were made in a study of 3,708 Finnish men and women with type 2 diabetes over a period of 19 years. A moderate or high level of physical activity was associated with a significantly improved prognosis regardless of weight, blood pressure, smoking or blood lipids (59). Moderate physical activity during work, leisure time or as a means of transportation is generally associated with an improved prognosis for type 2 diabetics (60).

A large number of case-control studies and prospective studies have likewise shown a link between the level of physical activity and the cancer forms associated with metabolic syndrome, e.g. prostate cancer, colon cancer and breast cancer (61–63).

Multiple effects of physical activity on metabolic disorders

The effects of physical activity on the metabolic disorders included in metabolic syndrome are evident from a number of clinical studies and have also been summarised in several review articles (19, 64–70). There are many mechanisms behind the preventive effects of physical activity and they are not yet fully known, but include a positive effect on the lipoprotein metabolism. Physical activity increases the blood flow in muscles and adipose tissue and increases lipoprotein lipase activity, reduces triglycerides and increases HDL levels. The particle size and susceptibility to oxidation of LDL particles are also positively affected by increased physical activity. The antihypertensive effects of physical activity are well documented. Peripheral insulin sensitivity and glucose tolerance are improved (66, 67). Physical activity reduces abdominal obesity and bodyweight (68, 69). Thrombogenesis and haemostasis are also positively affected (70). The IGFBP-1, endothelial function and inflammatory markers have also been shown to be affected (19, 68). Because of the multiple effects, increased physical activity is a beneficial way in which to prevent and treat metabolic syndrome.

There are currently no randomised primary prevention studies of the effects of increased physical activity among individuals with metabolic syndrome regarding future incidence or mortality from cardiovascular disease or cancer.

However, randomised primary preventive intervention studies on overweight men and women with reduced glucose tolerance and metabolic syndrome have shown that a combined dietary intervention and increased physical activity regimen can substantially (58%) reduce the risk of developing type 2 diabetes (71–74). The independent effects of increased physical activity are still partially unknown even though a Chinese four-armed study (diet, exercise, diet and exercise or control) indicated that the recommendations given on food intake and exercise were equally effective, each resulting in a risk reduction of 40 per cent (72). Post hoc analyses of the Finnish Diabetes Prevention study indicate that there is also a strong link between the reduction of risk and increased physical activity when taking into account other relevant factors such as eating habits (74). According to the Norwegian ODES Study, increased physical activity in combination with a change in diet may considerably reduce the prevalence of metabolic syndrome as compared with a control group during a 12-month follow-up period (65).

Newly developed molecular biology techniques and molecular genetics based on animal and human research models have over the past few years provided us with a greater understanding of the cellular mechanisms of metabolic syndrome as well as the molecular biology and molecular genetics behind the positive effects of physical activity.

Indications

Increased physical activity is of extensive importance to both primary and secondary prevention of metabolic syndrome. Today, different components (overweight, abdominal obesity, insulin resistance, high blood pressure, lipid disorder, etc.) in addition to metabolic syndrome are so common among the general public that prevention aimed at the individual is no longer enough. Population-based measures undertaken to increase physical activity among children and adults are also needed to reduce the risk of chronic diseases and premature deaths in the future.

Prescription

Reduce sedentary time

Many prospective studies have shown that the number of hours spent in front of the TV is related to the future risk of obesity and diabetes in both men and women (75, 76). There is also a link between the number of hours spent in front of the TV or the computer and the prevalence of metabolic syndrome among men, women and children (77, 78). A dose-response relationship is reported between sedentary time and mortality from all causes and cardiovascular disease (79, 80). Energy expenditure when walking (4.8 km/hour) is approximately 400 per cent more than when resting, e.g. lying in the sofa or sitting on a chair (20 kilojoule/minute compared with 5 kilojoule/minute) (81). In view of this, limiting inactivity is just as important as promoting physical activity.

Advice on physical activity for the prevention and treatment of metabolic syndrome

Individuals with metabolic syndrome should be encouraged to engage in daily physical activity of moderate intensity for a minimum of 30 minutes or 60 minutes if overweight, e.g. a brisk walk (82, 83). Additional health benefits are obtained if, in addition to daily physical activity of 30–60 minutes, some form of exercise is performed 2–3 times a week.

The activities recommended for the prevention or treatment of metabolic syndrome incorporate some form of aerobic fitness training such as walking, Nordic walking, jogging, swimming, cycling, etc. These activities can also be combined with a certain amount of strength training. Muscle mass decreases with age as a result of inactivity. Studies have shown that the lack of muscle strength affects the development of metabolic syndrome while strength training can have an effect on insulin sensitivity, for example (84).

Exercise should be done regularly for a minimum duration of 30 minutes. The recommended daily amount of physical activity can be accumulated through several separate episodes (for example 10 plus 10 plus 10 minutes) throughout the day (85). The exercise should be of a moderate intensity, approximately 60–70 per cent of maximum capacity, i.e. to the point when you begin to perspire and quicken your breathing. The same recommendations are essentially given for the prevention and treatment of cardiovascular diseases, type 2 diabetes and obesity or for maintaining generally good health (82).

The fact that a small amount of physical activity is better than no physical activity at all is common sense and was recently verified by a randomised controlled study of overweight and inactive postmenopausal women (82). The effect of various doses of exercise on general fitness was tested and a clear dose-response relationship was found. As little as 50 per cent of the recommended dose (according to the general guidelines) had a clear beneficial effect on aerobic fitness.

Constructive advice on exercise

A good knowledge of physical activity and health and recent recommendations and guidelines is not always enough. The approach taken by the caregiver as to the importance of lifestyle and lifestyle intervention in connection with metabolic syndrome is essential and requires pedagogical skills in addition to good scientific knowledge. All caregivers, i.e. staff categories, should be offered the opportunity of learning about the effects of physical activity and how to recommend exercise/give advice on physical activities so that all such recommendations and advice are given in unison. This would reinforce the credibility of the recommendations and advice given.

Giving advice on physical activity requires perceptiveness and a patient-centred approach. The patient is often embarrassed about his or her lifestyle, inactive life and obesity, etc. and it is therefore important not to add to this feeling of guilt. The general advice given on physical activity must always be adapted to the individual and transformed into concrete recommendations on exercise. It is important to form a picture of the patient's life circumstances and motivation to change. The information given must be neutral and not convey the caregiver's own opinion of physical activity. The patient should be informed about what type of physical

activity is suitable and the intensity, frequency and duration recommended in order to achieve the optimal effect. In addition, the concept of 50–70 per cent of maximum capacity should be explained to the patient, i.e. all forms of activities of an easy to moderate intensity up to the point when you begin to perspire and your breathing quickens.

The patient should also be given advice on suitable local activities and physical activity on prescription (FaR[®]) for the purpose of individual training or, if appropriate, modified exercise. Exercise referrals or physical activity on prescription (FaR[®]) has been in practice for decades in Sweden and New Zealand, for example (87, 88). According to national studies, approximately one third of the healthcare centres in Sweden follow this practice (89). Patients and the general public can also obtain information on physical activity and health in booklets available for purchase from dispensing pharmacies, for example. The use of a pedometer is a simple way in which to stimulate increased physical activity and monitor the effects of the prescriptions issued. When carrying a pedometer for a couple of weeks, the patient becomes aware of the degree of activity performed in different situations. A joint discussion on the subject of reasonable targets or sub-targets can also be advantageous.

Follow up on the advice given and give feedback

It is important that the advice on physical activity is followed up to ensure compliance and accomplishment. The time that such a follow-up is to take place must be chosen on an individual basis, although six weeks is generally considered an appropriate interval. By then, most people will have had time to make certain changes as verified by their pedometer or diary with positive effects on their waist circumference or metabolic variables. The waist circumference, which can easily be measured in clinical practice and by the patient, is strongly linked to the prevalence of metabolic syndrome in general and to several of the metabolic variables encompassed by metabolic syndrome (90–93). Prospective studies have also shown that waist circumference is linked to the future risk of coronary heart disease, intima-media thickness of the carotid arteries and death (94–98). Elevated blood pressure, lipids, blood glucose, etc. should also be monitored.

Risks and the need for health checks

Excess exertion can increase the risk of a stroke, myocardial infarction or sudden death in high-risk individuals. Such events are dramatic, but unusual. Much more common are injuries due to overloading or overtraining such as tendon inflammation and large joint disorders.

There are generally very few contraindications of increased physical activity. However, some men and women with metabolic syndrome are considered to be high-risk individuals because of the presence of multiple risk factors. Consequently, all advice or recommendations relating to exercise must be preceded by an appropriate assessment and individual risk analysis. An emergency examination and treatment should always be performed

in case of untreated, very high blood pressure or blood glucose levels as well as acute symptoms from the heart and circulation (e.g. TIA, unstable angina, peripheral circulation disorders).

However, professionally recommended physical activity in connection with metabolic syndrome rarely constitutes a risk. Following a standard analysis of cardiovascular symptoms and a family history in addition to a thorough physical examination of the heart and arteries, measurement of height, weight, waist circumference and blood pressure plus the taking of samples for an evaluation of the metabolic condition, it is important to determine whether further analysis such as an exercise test or ultrasonography is required. The patient is informed about warning signals and how to recognize them, as well as the importance of starting slowly and gradually increasing the amount and intensity of any activity. In this way, many types of injuries due to overloading can be prevented and avoided. The benefits of a good pair of shock absorbing shoes cannot be stressed enough, especially for an overweight person.

Interactions with drug therapy

There are a large number of pharmacological agents available for individuals with metabolic syndrome and information about interactions can be obtained from other sources. With the exception of insulin treatment and other pharmacological treatments of diabetes where there is a risk of hypoglycaemia, the risk of any adverse interaction, as a result of increased physical activity on the recommendation of a competent professional, is relatively atypical. Successful changes in lifestyle and increased physical activity may however necessitate a dose reduction. Hence, a regular follow-up is particularly important when lifestyle changes are combined with pharmacological treatment.

Contraindications

There are very few contraindications of physical activity in connection with metabolic syndrome, but they must be taken into consideration (also refer to “Risks”). Absolute contraindications include acute symptoms from the heart and circulation or a pending cardiovascular event (e.g. TIA, stroke, unstable angina, heart attack, acute peripheral circulation disorders), acute bleeding, hypoglycaemia or hyperglycaemia, significantly elevated blood pressure, infection accompanied by fever and poor health in general. As regards relative contraindications such as elevated cardiovascular risk, please refer to the section on “Risks”. Healthcare personnel who give advice on physical activity should always carry out a risk analysis even though the personal responsibility of the patient should not be overlooked. Physical activity and exercise is a natural part of our existence that brings enjoyment and contributes to an enhanced quality of life.

References

1. Galassi A, Reynolds K, He J. Metabolic syndrome and risk of cardiovascular disease. A meta-analysis. *American Journal of Medicine* 2006;119:812-9.
2. Hu G, Qiao Q, Tuomilehto J, Balkau B, Borch-Johnsen K, Pyorala K, et al. Prevalence of the metabolic syndrome and its relation to all-cause and cardiovascular mortality in non-diabetic European men and women. *Archives of Internal Medicine* 2004;164:1066-76.
3. Ford ES. Prevalence of the metabolic syndrome in US populations. *Endocrinology & Metabolism Clinics of North America* 2004;33:333-50.
4. McKeown NM, Meigs JB, Liu S, Saltzman E, Wilson PW, Jacques PF. Carbohydrate nutrition, insulin resistance and the prevalence of the metabolic syndrome in the Framingham Offspring Cohort. *Diabetes Care* 2004;27:538-46.
5. Ridker PM, Buring JE, Cook NR, Rifai N. C-reactive protein, the metabolic syndrome and risk of incident cardiovascular events. An 8-year follow-up of 14,719 initially healthy American women. *Circulation* 2003;107:391-7.
6. Panagiotakos DB, Pitsavos C, Chrysohooou C, Skoumas J, Tousoulis D, Toutouza M, et al. Impact of lifestyle habits on the prevalence of the metabolic syndrome among Greek adults from the ATTICA study. *American Heart Journal* 2004;147:106-12.
7. Marchesini G, Melchionda N, Apolone G, Cuzzolaro M, Mannucci E, Corica F, et al. The metabolic syndrome in treatment-seeking obese persons. *Metabolism: Clinical & Experimental* 2004;53:435-40.
8. Gorter PM, Olijhoek JK, van der Graaf Y, Algra A, Rabelink TJ, Visseren FL, et al. Prevalence of the metabolic syndrome in patients with coronary heart disease, cerebrovascular disease, peripheral arterial disease or abdominal aortic aneurysm. *Atherosclerosis* 2004;173:363-9.
9. Lawlor DA, Ebrahim S, Davey Smith G. The metabolic syndrome and coronary heart disease in older women. Findings from the British Women's Heart and Health Study. *Diabetic Medicine* 2004;21:906-13.
10. Grundy SM, Cleeman JI, Daniels SR, Donato KA, Eckel RH, Franklin BA, et al. Diagnosis and management of the metabolic syndrome. An American Heart Association/National Heart, Lung and Blood Institute Scientific Statement. *Circulation* 2005;112:2735-52.
11. Halldin M, Rosell M, De Faire U, Hellenius ML. The metabolic syndrome. Prevalence and association to leisure-time and work-related physical activity in 60-year-old men and women. *Nutr Metabol Cardiovasc Dis* 2007;17:349-57.
12. Zimmet P, Alberti G, Kaufman F, Tajima N, Silink M, Arslanian S, et al. International Diabetes Federation Task Force on Epidemiology and Prevention of Diabetes. The metabolic syndrome in children and adolescents. *Lancet* 2007;369:2059-61.
13. Swedish National Board of Health and Welfare. *Folkhälsorapport 2005* [Public Health Report 2005]. Stockholm: Swedish National Board of Health and Welfare; 2005.
14. Becker W. Vi äter nyttigare men har blivit tyngre [We eat better but have become heavier]. *Vår föda* [Our food] 1999;2:3-7.

15. Bjorntorp P. Do stress reactions cause abdominal obesity and comorbidities? *Obesity Reviews* 2001;2:73-86.
16. Hellenius ML. Metabola syndromet [Metabolic syndrome]. Betydelsen av fysisk aktivitet [Effects of physical activity]. *Scand J Nutr* 2002;46:91-3.
17. Muldoon MF, Mackey RH, Williams KV, Korytkowski MT, Flory JD, Manuck SB. Low central nervous system serotonergic responsivity is associated with the metabolic syndrome and physical inactivity. *Journal of Clinical Endocrinology & Metabolism* 2004;89:266-71.
18. Zimmet P, Shaw J, Alberti KG. Preventing Type 2 diabetes and the dysmetabolic syndrome in the real world. A realistic view. *Diabetic Medicine* 2003;20:693-702.
19. Lakka T, Laaksonen DE. Physical activity in the prevention and treatment of the metabolic syndrome. *Appl Physiol Nutr Metab* 2007;32:76-88.
20. Lakka TA, Laaksonen DE, Lakka HM, Mannikko N, Niskanen LK, Rauramaa R, et al. Sedentary lifestyle, poor cardiorespiratory fitness and the metabolic syndrome. *Med Sci Sports Exerc* 2003;35:1279-86.
21. Finley CE, LaMonte MJ, Waslien CI, Barlow CE, Blair SN, Nichaman MZ. Cardiorespiratory fitness, macronutrient intake and the metabolic syndrome. The Aerobics Center Longitudinal Study. *Journal of the American Dietetic Association* 2006;106:673-9.
22. Ekelund U, Brage S, Franks PW, Hennings S, Emms S, Wareham NJ. Physical activity energy expenditure predicts progression toward the metabolic syndrome independently of aerobic fitness in middle-aged healthy Caucasians. The Medical Research Council Ely Study. *Diabetes Care* 2005;28:1195-200.
23. Ekelund U, Franks P, Sharp S, Brage S, Nicholas J, Wareham NJ. Increase in physical activity energy expenditure is associated with reduced metabolic risk independent of changes in fatness and fitness. *Diabetes Care* 2007;in press.
24. Laaksonen DE, Lakka HM, Salonen JT, Niskanen LK, Rauramaa R, Lakka TA. Low levels of leisure-time physical activity and cardiorespiratory fitness predict development of the metabolic syndrome. *Diabetes Care* 2002;25:1612-8.
25. Physical Activity Guidelines Advisory Committee. Physical Activity Guidelines Advisory Committee Report, 2008. Part G. Washington, DC:U.S. Department of Health and Human Services, 2008.
26. Rosengren A, Eriksson H, Larsson B, Svärdsudd K, Tibblin G, Welin L, et al. Secular changes in cardiovascular risk factors over 30 years in Swedish men aged 50. The study of men born in 1913, 1923, 1933 and 1943. *J Intern Med* 2000;247:111-8.
27. Berg C, Rosengren A, Aires N, Lappas G, Toren K, Thelle D, et al. Trends in overweight and obesity from 1985 to 2002 in Goteborg, West Sweden. *International Journal of Obesity* 2005;29:916-24.
28. McCarthy HD, Jarrett KV, Emmett PM, Rogers I. Trends in waist circumferences in young British children. A comparative study. *International Journal of Obesity* 2005;29:157-62.
29. SCB, http://www.scb.se/templates/tableOrChart____48681.asp. 2006.

30. Despres JP, Lemieux I. Abdominal obesity and metabolic syndrome. *Nature* 2006;444:881-7.
31. Eckel RH, Grundy SM, Zimmet PZ. The metabolic syndrome. *Lancet* 2005;365:1415-28.
32. Coughlin SR, Mawdsley L, Mugarza JA, et al. Obstructive sleep apnoea is independently associated with an increased prevalence of metabolic syndrome. *European Heart Journal* 2004;25:735-41.
33. Leineweber C, Kecklund G, Akerstedt T, et al. Snoring and the metabolic syndrome in women. *Sleep Medicine* 2003;4:531-6.
34. Isomaa B, Almgren P, Tuomi T, Forsen B, Lahti K, Nissen M, et al. Cardiovascular morbidity and mortality associated with the metabolic syndrome. *Diabetes Care* 2001;24:683-9.
35. Eschwege E. The dysmetabolic syndrome, insulin resistance and increased cardiovascular (CV) morbidity and mortality in Type 2 diabetes. Aetiological factors in the development of CV complications. *Diabetes & Metabolism* 2003;29:6S19-27.
36. Nakanishi N, Takatorige T, Fukuda H, Shirai K, Li W, Okamoto M, et al. Components of the metabolic syndrome as predictors of cardiovascular disease and Type 2 diabetes in middle-aged Japanese men. *Diabetes Research & Clinical Practice* 2004;64:59-70.
37. Ford ES. The metabolic syndrome and mortality from cardiovascular disease and all-causes. Findings from the National Health and Nutrition Examination Survey II Mortality Study. *Atherosclerosis* 2004;173:309-14.
38. Kalmijn S, Foley D, White L, Burchfiel CM, Curb JD, Petrovitch H, et al. Metabolic cardiovascular syndrome and risk of dementia in Japanese-American elderly men. The Honolulu-Asia aging study. *Arteriosclerosis, Thrombosis & Vascular Biology* 2000;20:2255-60.
39. Komulainen P, Lakka TA, Kivipelto M, Hassinen M, Helkala EL, Haapala I, et al. Metabolic syndrome and cognitive function. A population-based follow-up study in elderly women. *Dementia & Geriatric Cognitive Disorders* 2007;23:29-34.
40. Steinbaum SR. The metabolic syndrome. An emerging health epidemic in women. *Progress in Cardiovascular Diseases* 2004;46:321-36.
41. Barnard RJ, Aronson WJ, Tymchuk CN, Ngo TH. Prostate cancer. Another aspect of the insulin-resistance syndrome? *Obesity Reviews* 2002;3:303-8.
42. Hammarsten J, Hogstedt B. Clinical, haemodynamic, anthropometric, metabolic and insulin profile of men with high-stage and high-grade clinical prostate cancer. *Blood Pressure* 2004;13:47-55.
43. Colangelo LA, Gapstur SM, Gann PH, Dyer AR, Liu K. Colorectal cancer mortality and factors related to the insulin resistance syndrome. *Cancer Epidemiology, Biomarkers & Prevention* 2002;11:385-91.
44. Furberg AS, Veierod MB, Wilsgaard T, Bernstein L, Thune I. Serum high-density lipoprotein cholesterol, metabolic profile and breast cancer risk. *Journal of the National Cancer Institute* 2004;96:1152-60.

45. Sinagra D, Amato C, Scarpilta AM, Brigandi M, Amato M, Saura G, et al. Metabolic syndrome and breast cancer risk. *European Review for Medical & Pharmacological Sciences* 2002;6:55-9.
46. Boyd DB. Insulin and cancer. *Integrative Cancer Therapies* 2003;2:315-29.
47. Alberti KG, Zimmet PZ. Definition, diagnosis and classification of diabetes mellitus and its complications. Part 1. Diagnosis and classification of diabetes mellitus provisional report of a WHO consultation. *Diabetic Medicine* 1998;15:539-53.
48. Balkau B, Charles MA. Comment on the provisional report from the WHO consultation. European Group for the Study of Insulin Resistance (EGIR). *Diabetic Medicine* 1999;16:442-3.
49. Alberti KG, Zimmet P, Shaw J, Group, IDFETFC. The metabolic syndrome. A new worldwide definition. *Lancet* 2005;366:1059-62.
50. Swedish Medical Products Agency. Förebyggande av aterosklerotisk hjärtsjukdom [Prevention of atherosclerotic heart disease]. *Behandlingsrekommendationer [Treatment recommendations]*. Volume 15. Uppsala: Swedish Medical Products Agency; 2006.
51. Laaksonen DE, Niskanen L, Lakka H-M, Lakka TA, Uusitupa M. Epidemiology and treatment of the metabolic syndrome. *Ann Med* 2004;36:332-46.
52. Tuomilehto J. Cardiovascular risk. Prevention and treatment of the metabolic syndrome. *Diab Res Clin Pract* 2005;68:S28-35.
53. Eyre H, Kahn R, Robertson RM, ACS/ADA/AHA Collaborative Writing Committee. Preventing cancer, cardiovascular disease and diabetes. A common agenda for the American Cancer Society, the American Diabetes Association and the American Heart Association. *Circulation* 2004;109:3244-55.
54. Nordic Council of Ministers, Nordic Nutrition Recommendations 2004. Integrating nutrition and physical activity. 4. edn. Vol 13. Copenhagen: Nord; 2004.
55. Lee CD, Blair SN, Jackson AS. Cardiorespiratory fitness, body composition and all-cause and cardiovascular disease mortality in men. *Am J Clin Nutr* 1999;69:373-80.
56. Li TY, Rana JS, Manson JE, Willett WC, Stampfer MJ, Colditz GA, et al. Obesity as compared with physical activity in predicting risk of coronary heart disease in women. *Circulation* 2006;113:499-506.
57. Ekblom-Bak E, Hellenius ML, Ekblom O, Engström LM, Ekblom B. Fitness and abdominal obesity are independently associated with cardiovascular risk. *J Intern Med* 2009; DOI: 10.1111/j.1365-2796.2009.02131.x.
58. Jeon CY, Lokken RP, Hu FB, van Dam RM. Physical activity of moderate intensity and risk of type 2 diabetes. A systematic review. *Diabetes Care* 2007;30:744-52.
59. Hu G, Lindstrom J, Valle TT, Eriksson JG, Jousilahti P, Silventoinen K, et al. Physical activity, body mass index and risk of type 2 diabetes in patients with normal or impaired glucose regulation. *Arch Intern Med* 2004;164:892-6.
60. Wei M, Gibbons LW, Kampert JB, Nichaman MZ, Blair SN. Low cardiorespiratory fitness and physical inactivity as predictors of mortality in men with type 2 diabetes. *Ann Intern Med* 2000;132:605-11.

61. Hu G, Jousilahti P, Barengo NC, Qiao Q, Lakka TA, Tuomilehto J. Physical activity, cardiovascular risk factors and mortality among Finnish adults with diabetes. *Diabetes Care* 2005;28:799-805.
62. Hu G, Eriksson J, Barengo NC, Lakka TA, Valle TT, Nissinen A, et al. Occupational, commuting and leisure-time physical activity in relation to total and cardiovascular mortality among Finnish subjects with type 2 diabetes. *Circulation* 2004;110:666-73.
63. Lagerros YT, Hsieh SF, Hsieh CC. Physical activity in adolescence and young adulthood and breast cancer risk. A quantitative review. *European Journal of Cancer Prevention* 2004;13:5-12.
64. Slattery ML. Physical activity and colorectal cancer. *Sports Medicine* 2004;34:239-52.
65. Hu G, Tuomilehto J, Silventoinen K, Barengo NC, Peltonen M, Jousilahti P. The effect of physical activity and body mass index on cardiovascular, cancer and all-cause mortality among 47 212 middle-aged Finnish men and women. *Int J Obes* 2005;29:894-902.
66. Carroll S, Dudfield M. What is the relationship between exercise and metabolic abnormalities? A review of the metabolic syndrome. *Sports Med* 2004;34:371-418.
67. Anderssen SA, Carroll S, Urdal P, Holme I. Combined diet and exercise intervention reverses the metabolic syndrome in middle-aged males. Results from the Oslo Diet and Exercise Study. *Scand J Med Sci Sports* 2007 ;17:687-95.
68. Hellenius ML, Brismar KE, Berglund BH, de Faire U. Effects on glucose tolerance, insulin secretion, insulin-like growth factor 1 and its binding protein, IGFBP-1, in a randomized controlled diet and exercise study in healthy, middle-aged men. *J Intern Med* 1995;238:121-30.
69. Anderssen SA, Hjermmann I, Urdal P, Torjesen PA, Holme I. Improved carbohydrate metabolism after physical training and dietary intervention in individuals with the "atherothrombogenic syndrome". Oslo Diet and Exercise Study (ODES). A randomized trial. *J Intern Med* 1996;Oct;240:203-9.
70. Hellénius ML, de Faire U, Berglund B, Hamsten A, Krakau I. Diet and exercise are equally effective in reducing risk for cardiovascular disease. Results of a randomized controlled study in men with slightly to moderately raised cardiovascular risk factors. *Atherosclerosis* 1993;103:81-91.
71. Irwin ML, Yasui Y, Ulrich CM, Bowen D, Rudolph RE, Schwartz RS, et al. Effect of exercise on total and intra-abdominal body fat in postmenopausal women. A randomized controlled trial. *JAMA* 2003;289:323-30.
72. Väisänen B, Hellenius ML, Penttilä I, Rauramaa R. Physical activity and hemostasis. *Klinisk Kjem i Norden* 2002;4:8-11 (Biological Chemistry in Scandinavia).
73. Knowler WC, Barrett-Connor E, Fowler SE, Hamman RF, Lachin JM, Walker EA, et al. Reduction in the incidence of type 2 diabetes with lifestyle intervention or metformin. *N Engl J Med* 2002;346:393-403.
74. Pan XR, Li GW, Hu YH, Wang JX, Yang WY, An ZX, et al. Effects of diet and exercise in preventing NIDDM in people with impaired glucose tolerance. The Da Qing IGT and Diabetes Study. *Diabetes Care* 1997;20:537-44.

75. Tuomilehto J, Lindstrom J, Eriksson JG, Valle TT, Hämäläinen H, Ilanne-Parikka P, et al. Prevention of type 2 diabetes mellitus by changes in lifestyle among subjects with impaired glucose tolerance. *N Engl J Med* 2001;344:1343-50.
76. Laaksonen DE, Lindstrom J, Lakka TA, Eriksson JG, Niskanen L, Wikstrom K, et al. Physical activity in the prevention of Type 2 diabetes. The Finnish diabetes prevention study. *Diabetes* 2005;54:158-65.
77. Hu FB, Leitzmann MF, Stampfer MJ, Colditz GA, Willett WC, Rimm EB. Physical activity and television watching in relation to risk for Type 2 diabetes mellitus in men. *Archives of Internal Medicine* 2001;161:1542-8.
78. Hu FB, Li TY, Colditz GA, Willett WC, Manson JE. Television watching and other sedentary behaviors in relation to risk of obesity and Type 2 diabetes mellitus in women. *JAMA* 2003;289:1785-91.
79. Ford ES, Kohl HW, 3rd, Mokdad AH, Ajani UA. Sedentary behavior, physical activity and the metabolic syndrome among U.S. adults. *Obesity Research* 2005;13:608-14.
80. Ekelund U, Brage S, Froberg K, Harro M, Anderssen SA, Sardinha LB, et al. TV viewing and physical activity are independently associated with metabolic risk in children. The European Youth Heart Study. *PLoS Med* 2006 Dec;3:e488.
81. Katzmarzyk PT, Church T S, Craig C L, Bouchard C. Sitting time and mortality from all causes, cardiovascular disease and cancer. *Med Sci Sports Exerc* 2009;41:998-1005.
82. Levine JA, Schleusner SJ, Jensen MD. Energy expenditure of nonexercise activity. *Am J Clin Nutr* 2000;72:1451-4.
83. Saris WH, Blair SN, van Baak MA, Eaton SB, Davies PS, Di Pietro L, et al. How much physical activity is enough to prevent unhealthy weight gain? Outcome of the IASO 1st Stock Conference and consensus statement. *Obes Rev* 2003;4:101-14.
84. Jurca R, Lamonte MJ, Barlow CE, Kampert JB, Church TS, Blair SN. Association of muscular strength with incidence of metabolic syndrome in men. *Medicine & Science in Sports & Exercise* 2005;37:1849-55.
85. Murphy MH, Blair SN, Murtagh EM. Accumulated versus continuous exercise for health benefit. A review of empirical studies. *Sports Med* 2009;39:29-43.
86. Church TS, Earnest CP, Skinner JS, Blair SN. Effects of different doses of physical activity on cardiorespiratory fitness among sedentary, overweight or obese postmenopausal women with elevated blood pressure. A randomized controlled trial. *JAMA* 2007;297:2081-91.
87. Hellenius ML, Arborelius E. Prescribed exercise can help patients change their habits. *Läkartidningen* [Swedish Medical Journal] 1999;96:3343-6.
88. Kallings L, Leijon M. Erfarenheter av Fysisk Aktivitet på Recept, FaR. [Experiences of Prescribed Physical Activity, FaR.] Report 2003;53. Stockholm: Swedish National Institute of Public Health; 2003.
89. Kallings LV, Leijon M, Hellénus ML, Ståhle A. Physical activity on prescription in primary health care. A follow-up of physical activity level and quality of life. *Scand J Med Sci Sports* 2007; 2008;18:154-61.

90. Kallings LV, Leijon ME, Kowalski J, Hellénus ML, Ståhle A. Self-reported adherence: A method for evaluating prescribed physical activity in primary health care patients. *J Phys Act Health* 2009;6:483-92.
91. Kallings LV, Sierra Johnson J, Fisher RM, Faire U, Ståhle A, Hemmingsson E, Hellénus ML. Beneficial effects of individualized physical activity on prescription on body composition and cardiometabolic risk factors: results from a randomized controlled trial. *Eur J Cardiovasc Prev Rehabil* 2009;16:80-4.
92. Swedish Council on Technology Assessment in Health Care (SBU). Metoder för att främja fysisk aktivitet. [Methods of promoting physical activity.] En systematisk litteraturöversikt [A systematic literature survey]. English Summary. Swedish Council on Technology Assessment in Health Care Stockholm; 2007.
93. Henriksson J. FYSS för all. En bok om att röra på sig för att må bättre samt att förebygga och behandla sjukdomar. [A book about exercising to feel better and prevent and treat diseases.] Stockholm: Yrkesföreningar för fysisk aktivitet; 2004 [Professional organisations for physical activity].
94. Janssen I, Katzmarzyk PT, Ross R. Waist circumference and not body mass index explains obesity-related health risk. *American Journal of Clinical Nutrition* 2004;79:379-84.
95. Kahn HS, Valdez R. Metabolic risks identified by the combination of enlarged waist and elevated triacylglycerol concentration. *American Journal of Clinical Nutrition* 2003;78:928-34.
96. Palaniappan L, Carnethon MR, Wang Y, Hanley AJ, Fortmann SP, Haffner SM, et al. Predictors of the incident metabolic syndrome in adults. The Insulin Resistance Atherosclerosis Study. *Diabetes Care* 2004;27:788-93.
97. Riserus U, Arnlov J, Brisman K, Zethelius B, Berglund L, Vessby B. Sagittal abdominal diameter is a strong anthropometric marker of insulin resistance and hyperproinsulinemia in obese men. *Diabetes Care* 2004;27:2041-6.
98. Bigaard J, Frederiksen K, Tjonneland A, Thomsen BL, Overvad K, Heitmann BL, et al. Waist and hip circumferences and all-cause mortality. Usefulness of the waist-to-hip ratio? *International Journal of Obesity & Related Metabolic Disorders* 2004;28:741-7.
99. Bigaard J, Thomsen BL, Tjonneland A, Sorensen TI. Does waist circumference alone explain obesity-related health risk? *American Journal of Clinical Nutrition* 2004;80:790-1.
100. Hassinen M, Lakka TA, Komulainen P, Haapala I, Nissinen A, Rauramaa R. Association of waist and hip circumference with 12-year progression of carotid intima-media thickness in elderly women. *Int J Obes* 31:1406-11.
101. Kuk JL, Katzmarzyk PT, Nichaman MZ, Church TS, Blair SN, Ross R. Visceral fat is an independent predictor of all-cause mortality in men. *Obesity* 2006;14:336-41.
102. Zhang X, Shu XO, Gao YT, Yang G, Matthews CE, Li Q, et al. Anthropometric predictors of coronary heart disease in Chinese women. *International Journal of Obesity & Related Metabolic Disorders* 2004;28:734-40.

35. Multiple sclerosis

Authors

Ulrika Einarsson, PT, PhD, Physiotherapy Clinic, Karolinska University Hospital and Institute of Neurobiology, Healthcare Science and Society, Karolinska Institutet, Stockholm, Sweden

Jan Hillert, MD, PhD, Professor, Karolinska University Hospital and Institute of Clinical Neurosciences, Karolinska Institutet, Stockholm, Sweden

Summary

Maintaining muscle activity, strength and aerobic fitness is just as important for people with multiple sclerosis (MS) as for healthy people. Those with MS are currently recommended physical activity as there is strong evidence that it improves muscle function, aerobic fitness and mobility. There is also evidence that physical activity improves the overall quality of life. However, people with MS often have a reduced physical capacity. There are many reasons for this. It is not uncommon for those with MS to limit their physical activity so as to minimise the feeling of tiredness and avoid an elevated body temperature. More often than not, this complies with the advice of healthcare and medical professionals who feel that the patient should save his/her energy for daily chores. However, limited physical activity will lead to further weakness, fatigue and other health risks plus fewer chances of enjoying a leisure time hobby.

Daily activities, walks and water exercises combined with periods of rest/recovery are recommended. Participation in physical activities should be encouraged at home, at work or at a fitness centre. Those with MS should be well informed about potential symptoms that may arise from or increase as a result of physical activity such as fatigue, onsets due to heat intolerance and spasticity, and how such symptoms should be handled. Appropriate activities to be carried out at home as recommended by a physiotherapist or at a physiotherapy clinic should incorporate aerobic fitness and strength training, walking and water exercises. Training should take place at intervals and in a cool environment. There is strong evidence today that periods of intensive rehabilitation at a rehabilitation centre improve the level of activity and participation for those with MS.

In case of warm outdoor or indoor temperatures (for example in the gym), those with MS who suffer from heat intolerance may take a cool shower before and after training or use a cooling suit. Exercise done in connection with a pseudofit should be carefully monitored until all symptoms are stabilised. The same applies to infections such as urinary tract infections as well as cortisone medication.

Definition

Prevalence/Incidence

One in 800 people are diagnosed with MS. That is 11,000–12,000 people in Sweden and around half as many in Norway. Each year, 5 out of 100,000 people in Sweden get MS, i.e. around 450 people. Most of these are diagnosed with MS between the ages of 20 and 40 years old. MS is a chronic, lifelong illness with a variable development that often leads to significant disability.

Causes and risk factors

What causes MS is not known. Hereditary factors play a part in some cases, i.e. children of MS patients have a 2–4 per cent risk of developing the same illness. However, environmental factors are also likely to be of some significance although this has not yet been proven.

The prevailing theory is that MS is caused by an autoimmune reaction, in other words that the immune system incorrectly attacks the myelin of the central nervous system, i.e. the fatty insulating sheath that facilitates neural impulse transmissions. Consequently, the myelin producing cells or oligodendrocytes are damaged and reduced in number. The nervous system has a certain ability to heal, but the damage to the myelin and nerve fibres in the early stages of the illness is permanent.

Symptoms

The symptoms of MS vary greatly from person to person and over time. The early stages of MS are characterised by symptoms of onsets plus periodic symptoms caused by the effects on the central nervous system, such as sensory disorders, transient visual problems (optical nerve inflammation), balance disturbances, weakness (pareses) or problems with urination. In the later stages of the illness, the symptoms are usually more gradual and permanent, such as weakness in the legs leading to problems walking or with balance.

A small number of those with MS never experience any significant problems, but the majority will sooner or later develop various degrees of disability. From a random selection of individuals with MS, one out of ten had a normal walking speed. One in three people is self-reliant and has a normal social life. All areas of health-related quality of life are subjected to a strong negative impact, especially when it comes to walking, domestic chores, recreational and leisure activities, and one in five suffers from depression (1–4). After 15 years, one out of two people with MS will require a walking aid to walk 100 meters and after 25 years, one out of two will be confined to a wheelchair. After a period of 30 years, at least one in three people with MS needs help getting out of bed. MS shortens average life expectancy by 5–10 years.

Diagnostics

A diagnosis is always based on at least two separate symptoms, depending on which parts of the central nervous system that are affected. A brain scan using Magnetic Resonance Imaging (MRI) and a cerebrospinal fluid analysis are of major assistance when determining the diagnosis, especially in the early stages before the symptoms are completely clear.

Treatment

A so-called modification treatment has been available since the middle of the 1990s, initially using interferon-beta and in recent years glatiramer acetate as well, with the addition of natalizumab in 2006. These treatments reduce the frequency and severity of onsets and also slow down the development of other symptoms. However, the treatments are only partially effective and the long-term effect on the development of symptoms is not fully known. In addition, the possibility of alleviating different symptoms such as stiffness, pain, depression, fatigue and urination problems continues to improve. Recurrent rehabilitation periods incorporating physical exercise and identifying the needs for assistance also mean that disability can be reduced.

Effects of physical activity

MS is often associated with increased tiredness and heat intolerance, i.e. an exacerbation of symptoms as a result of an elevated body temperature. Even persons with a slight to moderate neurological deterioration may be severely hindered in life due to acute fatigue or pain.

Maintaining muscle activity, strength and aerobic fitness is just as important for people with MS as it is for people without MS (5). In recent years, scientific publications of rehabilitation (6–14) and exercise studies (15–25) have shown the benefits of regular physical activity. Those with MS are recommended physical activity as there is strong evidence that this improves muscle function, aerobic fitness and mobility (26). Moreover, physical activity has been shown to improve the quality of life for people with MS (27). There is evidence that exercise, regardless of whether it is similar to exercise for healthy individuals or is modified to maintain function, is of great benefit to those with MS and is therefore widely recommended. It is also recommended that exercise and rehabilitation be adapted to the individual needs and requirements of the person with MS, as no specific form or method of exercise has shown to be more effective than any other (26, 28). Rampello and colleagues (29) compared neurological rehabilitation with aerobic exercise in connection with distance and walking speed, and subsequently concluded that aerobic exercise generated the greatest improvements.

There is still limited evidence as to the level of exercise required in order to improve muscle strength and aerobic fitness (15, 16, 28). With regard to aerobic training, White and Dressendorfer (30) make the following recommendations; 20–30 minutes or 2 x 10–15 minutes 2–3 times a week at an intensity of 65–75 per cent of the maximal heart

rate and 50–70 per cent of VO_2 max. The training should be of a moderate intensity (Borg scale 11–14). The time it takes to recover after a training session indicates the level of training required.

Compared with people without MS or with another illness (31–33) and even compared with healthy, but inactive people (34), individuals with MS have been shown to have a lower level of physical activity. This can lead to reduced muscle mass, weakness and reduced endurance not directly caused by MS, but rather as a secondary effect of inactivity. Reduced physical activity may in turn lead to less social interaction, restricted leisure activities and depression, generally affecting the quality of life. Conversely, persons with MS can enhance their quality of life by becoming more physically active.

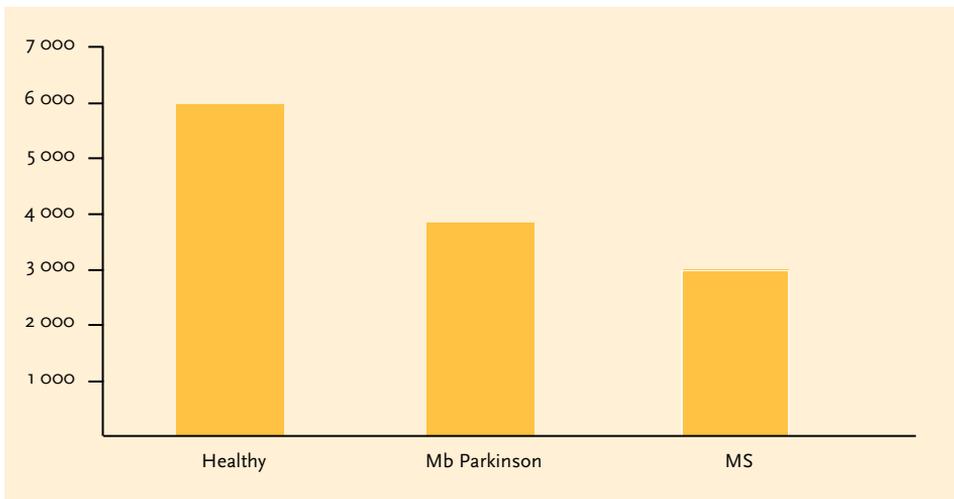


Figure 1. The average number of steps taken in a day for a period of seven days measured with a pedometer by people with Mb Parkinson, multiple sclerosis or in good health (33).

Physical activity and MS – specific aspects

The degree of physical activity should be adapted to the relevant circumstances. Special consideration must be given to people suffering from fatigue or heat intolerance. Physical exercise during a period of recurrent onsets can easily lead to increased symptoms in which case the exercise should be limited or avoided.

Of those with MS, 87 per cent experience severe fatigue. This is typical for the illness (35). The majority of people with a mild form of MS also experience severe fatigue (36). Patients often describe how they suffer from pronounced fatigue, muscular fatigue and the need for long periods of recovery following moderate muscular exertion. The increased level of muscular fatigue is not correlated with muscular weakness (37), although muscular strength is also affected following a short period of exertion (nerve fibre fatigue). This tiredness or fatigue affects all daily activities and the quality of life. Consequently, persons with MS need advice on how to manage the fatigue as it can easily lead to inactivity and further tiredness. Initially, persons with MS may need help to understand the concept of “MS fatigue” and how to best deal with this condition (38, 39). When informing a person with MS about fatigue, it helps to separate the condition into seven different forms of fatigue:

1. **Normal muscular fatigue** is to be expected after any physical/muscular exertion, but arises more rapidly for people with MS. A person with MS consumes more energy during a normal activity such as walking compared with a healthy person (40, 41). Therefore, it is important to find a balance between physical activity, recuperation/rest and daily events.
2. **Compensatory fatigue.** With MS, the stronger muscles compensate for the weaker muscles by taking more of the physical strain. This extra muscular work can lead to overexertion and tiredness. For example, the arm muscles of a person with MS have to cope with the added strain of pushing a wheelchair or supporting the body when walking with the help of crutches or a rollator.
3. **Fatigue due to depression** A loss of energy, desire and motivation is a manifest problem of depression. These feelings add to general physical tiredness, usually leading to reduced physical activity and increased fatigue (4, 26, 36, 42, 43).
4. **Cognitive fatigue.** Half of those with MS are cognitively affected (1). Tiredness may affect the cognitive function for persons with MS. This is often referred to as cognitive fatigue (44, 45). A short rest usually alleviates the problem.
5. **Cardiovascular fatigue.** Reduced fitness owing to inactivity leads to tiredness/reduced endurance. Swimming, water exercises, cycling, exercise bike training and walks will boost endurance and reduce tiredness (15, 18, 21, 22).
6. **MS related muscular fatigue** (46, 47). Individuals with MS require a longer period of recovery following exertion (47). Consequently, it is important to know one’s personal limits. A physical activity should not exhaust the person in question. Sub-maximum effort is recommended. Persons with MS must allow themselves short breaks to gain new strength. A nap in the middle of the day may sometimes be necessary. For a person with MS to be able to continue working, it may even be necessary to reduce the number of working hours to allow time for physical activity and exercise.
7. **General fatigue with or without drowsiness.** Individuals with MS often have an increased need for sleep and may feel extremely tired despite having had a good night’s sleep. In addition, those with MS often describe a feeling of mental and physical fatigue that is quite different from normal drowsiness.

Naturally, these different forms of fatigue require different types of treatment. However, graded exercise is generally thought to lessen the symptoms of MS fatigue (45). Recently, it has also been shown that rehabilitation at a rehabilitation centre can reduce the fatigue experienced by individuals with MS, a finding based on the improved disposition of the people monitored (48).

Table 1. Model for treatment of MS fatigue (49).

Physiology	Medication
Education	Better understand of symptoms
Adaptation	Planned activity/rest
Compensation	Use of aids
Physical activity	Individual/group

Many persons with MS are sensitive to heat. An increase in body temperature of just 0.1° C reduces the speed of nerve transmission, particularly where myelinated nerve fibres are damaged. As a result, previous or existing symptoms are augmented until the body temperature returns to normal.

An inability to endure physical activity and increased functional failure secondary to the increase in body temperature often occur (50–52), which is why a cool down in the form of a cool shower or a cooling suit may be necessary for any physical activity to be carried out (53–60). The cooling suit has been shown to be of benefit to persons with MS who are heat sensitive and is useful to facilitate an active life (60). Air conditioning, cool premises and a peaked cap (in sunshine) may also help.

Until now, the information available on physical exercise and strength training for patients with mild to moderate MS symptoms has been very limited (15, 16). However, recent findings confirm that physical exercise should be recommended to and is safe for persons with mild to moderate MS symptoms (25). Clinical experiences have shown that many people with mild MS symptoms are able to exercise to the same extent as healthy people and will benefit from using their muscles and increasing their level of fitness .

However, a large number of people with MS are worried that physical exercise may aggravate their illness and claims of such an association have been published in literature aimed at those with MS. Such claims have been put forward by alternative medicine practitioners despite the absence of any scientific evidence to support this hypothesis. Consequently, it is very important that people with MS learn how to handle the symptoms that may materialize in connection with physical activity so as not to be afraid of exercising or leading an active life (61, 62).

Physical activity/physiotherapy should be avoided while the patient is undergoing cortisone treatment, but can usually continue despite onsets as it minimises any loss of aerobic fitness and muscle strength. During the initial stages of an onset until a plateau is reached, flexibility exercise is recommended. Sometimes an exercise programme needs to be modified as a result of an onset and if necessary, incorporate additional aids. It is important to encourage, support and motivate a patient to resume physical activity following

an onset although at a lower level of intensity. A new onset also often leads to a certain amount of depression and support is therefore vital.

Acute effects

Four weeks of cycling increased aerobic fitness by 13 per cent, overall work capacity by 11 per cent and the level of physical activity for the people monitored (22). Ten weeks of endurance training including cycling with an ergometer resulted in improved aerobic fitness and strength, reduced fatigue and enhanced quality of life (15). Another study showed that endurance training for a period of 4–6 weeks resulted in reduced fatigue and an enhanced feeling of well-being for people with MS (16). Water exercises increase muscle strength and endurance (18). Swimming has been shown to improve aerobic fitness and muscle strength. White and Dressendorfer (30) recommend training in water at a temperature of 27–29° C, but clinical experiences have shown that MS patients can cope with considerably warmer water (up to 34° C). The same authors (30) claim that progressive resistance training increases muscle strength while improving the chances of an active daily life (ADL) as well as psychosocial well-being. Regular aerobic training has an anti-depressive effect on the mild to moderate clinical depression found among many MS patients (30).

Two months of walking reduced limitations to activity and disability (20). Today, persons with MS are recommended to undergo more intensive rehabilitation at rehabilitation centres as there is strong evidence to suggest that this will quickly improve the level of activity and participation. There is also evidence to suggest that physical activity improves muscle function, aerobic fitness and mobility (26, 28).

Long-term effects

Water exercises increased the quality of life and made it easier for persons with MS to cope with daily household chores (increased functional capacity). The reduction in activity limitations and disability achieved after 6 weeks of rehabilitation remained for a period of 6 months while the health-related quality of life was enhanced for nearly 12 months (12). A different study indicated similar effects lasting for a period of 4 months (17). However, there is now strong new evidence suggesting that a less intensive rehabilitation over a longer period of time also improves the quality of life (28).

When comparing a group of active women with MS with a group of inactive women with MS, the active women had a lower risk of myocardial infarction (63). The increased risk of osteoporosis among women with MS could potentially be reduced by an increased level of activity (64).

Physical activity cannot reduce the risk of an onset or stop the progress of the disease. However, physical activity that takes into account fatigue problems and heat sensitivity will help strengthen bodily functions and parts of the body that are either unaffected or only partly affected by MS. Avoiding physical activity only leads to worse aerobic fitness, less energy, lower motivation and flexibility, which in turn leads to a reduction in

muscle strength. Weight gain caused by inactivity may have an adverse effect on mobility and lead to increased dependency.

Indications

Prevention

There is currently little information on prevention for persons with MS. As in the healthy population, physical activity is likely to:

1. Prevent problems, such as weakness and reduced endurance owing to inactivity.
2. Help improve general well-being and aerobic fitness (heart/lungs).
3. Help maintain good health and provide increased resistance to disease.
4. Stimulate motivation.

Increased physical activity for persons with MS who are not currently as active as the population at large (33, 34) is also likely to reduce the risk of cardiovascular disease and depression. Women with MS have a higher risk of getting osteoporosis, which in turn increases the risk of fractures. Physical activity may contribute to lowering this risk.

Prescription

Type of activity

There is currently no evidence available to determine the training dose (intensity, duration and frequency) for persons with MS. No training method has proven to be more effective than the other. However, there is strong evidence that physical exercise does affect persons with MS, regardless of whether it is similar to that recommended for healthy individuals or comprises exercises adapted to maintain function. Consequently, it is recommended that the training/rehabilitation be adapted to the individual, based on the needs of the person with MS (26, 28). Petjan and White have developed a Physical Activity and Exercise Pyramid for persons with MS (65).

Table 2. Exercise model for the improvement of muscular strength, endurance and physical activity for persons in different stages of MS (65).

	Muscular strength and endurance	Physical activity
No dysfunction, no problems with fatigue and/or heat sensitivity	<p><i>Modified strength training programme</i></p> <ul style="list-style-type: none"> • Large muscle groups can be subjected to 3 sets of 10–12 repetitive tasks. • The person training should not feel completely exhausted at the end of the third set of exercises. • The aim is for the extra strength to result in an improved balance and added motion that would otherwise have been avoided. 	<p><i>Structured fitness training programme</i></p> <p>Persons with no dysfunctions may exercise in the same way as a healthy person, but should perhaps cool down before training.</p>
No dysfunction, but problems with fatigue and/or heat sensitivity	<p><i>Specific muscle strength training</i></p> <ul style="list-style-type: none"> • Strength training programme that takes into account strength, fatigue, motivation and degree of dysfunction. • Programme designed for balance and coordination training in supplement to the above. • Could the programme be adapted to home environment? • Water exercise. 	<p><i>Active recreation</i></p> <ul style="list-style-type: none"> • Regular low-intensity training of less than 30 min./day, e.g. walking, cycling or gardening. • Aerobic training 3 times/week, 65% of VO₂ max for 20–30 min. • Body weight unloading training, e.g. cycling or water exercise.
Minor to moderate dysfunction	<p><i>Active and unloaded active motion</i></p> <ul style="list-style-type: none"> • A weak muscular system may be subjected to actively unloaded muscle training. • No studies with a clear guideline on frequency, degree of loading, etc. have been published to date. 	<p><i>“Built-in inefficiencies”</i></p> <ul style="list-style-type: none"> • Individuals at this level are active, but balance all activities owing to the energy cost, sometimes unconsciously. • Training may involve making persons aware of this compensation mechanism.
Severe dysfunction	<p><i>Passive range of motion</i></p> <ul style="list-style-type: none"> • Passive motion to prevent contractures and maintain mobility. • Passive motion is primarily for hip extension, knee flexion, hip abduction and dorsal extension of the ankle joint. 	<p><i>Activities in daily life (ADL)</i></p> <ul style="list-style-type: none"> • For persons with severe dysfunctions, activities in daily life (ADL) provide sufficient training. • These persons usually need home caregivers or family members to help them with shopping, doing the laundry, etc.

Persons with MS often feel that they lack sufficient knowledge to carry out a physical activity in a correct and safe manner (32) and hence, a general exercise prescription is not recommended. Consequently, a personal exercise plan taking into account the symptoms and effects of physical activity would be preferred in conjunction with a prescription for a physical activity/training programme to underline the importance of exercise (45).

Persons with MS often have a reduced physical capacity and it is important that some form of physical and mental exercise takes place. The physical training should consist of general exercises including aerobic training (fitness), strength training (endurance) and

mobility/flexibility training. Training should start with a warm up and finish with a cool-down plus stretching exercises.

Daily activities, walks and water exercises combined with periods of rest/recovery are recommended. Participation in physical activities should be encouraged and take place either at home, at work or at a fitness centre. Training carried out in the home environment (66) and adapted to the status of the patient is preferred to avoid subjecting the person with MS to tiring travel. It is important that the training be followed up, especially if carried out in the home environment. A person with MS should be well aware of the MS related symptoms that may arise from physical activity such as fatigue, pseudo onset owing to heat intolerance and spasticity and how to handle these in connection with physical activity. Appropriate activities to be carried out at home as recommended by a physiotherapist or at a physiotherapy clinic should incorporate aerobic fitness and strength training, walking and water exercises (25, 26, 66). Training should take place at intervals and in a cool environment. There is strong evidence today that periods of intensive rehabilitation at a rehabilitation centre improve the level of activity and participation for individuals with MS.

Special considerations

Training should be carried out with caution in connection with onset, significant heat intolerance or cortisone treatment. The ability to exercise may be restricted by the fatigue so typical to MS despite good retained strength (see above).

Functional tests/need for health check-ups

A functional test should always be carried out prior to physical training to determine the appropriate individual level of intensity. A functional test should also be carried out at the end of each training session for the purpose of assessing the effects of the training programme and planned prescriptions.

The following is a selection of assessment instruments and areas of use:

Motor assessment

Motor assessments may either be carried out using strength tests on a scale of 0–5 or the Amended Motor Club Assessment (AMCA) (61), the Rivermead Mobility Index (67–68) or Birgitta Lindmark's motor assessment of active range of motions of the upper and lower extremities, limb movement velocity, motion capacity and balance (1, 69).

Walk/balance/endurance

Assessed using for example the 25 Feet Timed Walk (25, 70), 10 Meters Walk (1, 71), Timed Up and Go Test (72, 73), Physiological Cost Index, PCI (74, 75), 6-minute Walk Test (76, 77), Berg Balance Scale (78, 79) or the 12-item MS Walking Scale (MSWS-12) (80–82).

Health-related quality of life

Assessed using for example the Sickness Impact Scale (SIP) (3, 83), 36-item Short Form Health Survey Questionnaire (SF-36) (84, 85), Multiple Sclerosis Impact Scale-29 (MSIS-29) (86) or the Multiple Sclerosis Quality of Life-54 (MSQO-54) (87).

Fatigue

Assessed using for example the Fatigue Severity Scale (88), Fatigue Impact Scale (89) or the Fatigue Descriptive Scale (90).

Activity and participation

Assessed using for example the Guy's Neurological Disability Scale (GNDS) (91), Frenchay Social Activity Index (FAI) (2, 92) or the Barthel Index (BI) (93).

Interactions with drug therapy

Cortisone treatment is sometimes prescribed temporarily to inhibit onset. However, cortisone can lead to an increased risk of damage to bones, muscles and tendons.

A potential side effect of treatment with *interferon beta* is a slightly elevated body temperature. As a result, symptoms of heat intolerance may be aggravated and the ability to exercise restricted. However, this is normally a transient side-effect of treatment with interferon beta.

Contraindications

Exhaustive training should be avoided while submaximal training with a period of rest is recommended. Exercise done in connection with an onset should be carefully monitored until all symptoms have stabilised (26). This also applies to infections such as urinary infection and cortisone treatments.

Risks

Symptoms arising from exercise due to heat intolerance can in rare cases become permanent or only pass after a long period of time. Consequently, patients with pronounced heat intolerance should exercise with caution.

References

1. Einarsson U, Gottberg K, von Koch L, Fredrikson S, Ytterberg C, Jin YP, et al. Cognitive and motor function in people with multiple sclerosis in Stockholm county. *Mult Scler* 2006;12:340-53.
2. Einarsson U, Gottberg K, Fredrikson S, von Koch L, Holmqvist LW. Activities of daily living and social activities in people with multiple sclerosis in Stockholm county. *Clin Rehabil* 2006;20:543-51.
3. Gottberg K, Einarsson U, Ytterberg C, de Pedro Cuesta J, Fredrikson S, von Koch L, et al. Health-related quality of life in a population-based sample of people with multiple sclerosis in Stockholm county. *Mult Scler* 2006;12:605-12.
4. Gottberg K, Einarsson U, Fredrikson S, von Koch L, Holmqvist LW. A population-based study of depressive symptoms in multiple sclerosis in Stockholm county. Association with functioning and sense of coherence. *J Neurol, Neurosurg Psychiatry* 2007;78:60-5.
5. Senior K. Inpatient rehabilitation helps patients with multiple sclerosis. *Lancet* 1999;353:301.
6. Wickström A. Rusta-rapporten. [The Equip Report] Tidig rehabilitering för personer med multipel skleros inom rehabilitering neurologi. [Early rehabilitation for persons with multiple sclerosis in rehabilitation neurology.] Umeå; March 1997.
7. Schapiro RT. The rehabilitation of multiple sclerosis. *J Neurol Rehabil* 1990;4:215-7.
8. La Rocca NG, Kalb RC. Efficacy of rehabilitation in multiple sclerosis. *J Neurol Rehabil* 1992;6:147-55.
9. Kidd D, Howard RS, Losseff NJ, Thompsen AJ. The benefit of inpatient neurorehabilitation in multiple sclerosis. *Clin Rehabil* 1995;9:198-203.
10. Aisen ML, Sevilla D, Fox N. Inpatient rehabilitation for multiple sclerosis. *J Neurol Rehabil* 1996;10:43-6.
11. Freeman JA, Langdon DW, Hobart JC, Thompsen AJ. The impact of inpatient rehabilitation on progressive multiple sclerosis. *Ann Neurol* 1997;42:236-44.
12. Freeman JA, Langdon DW, Hobart JC, Thompsen AJ. Inpatient rehabilitation in multiple sclerosis. Do the benefits carry over into the community? *Neurology* 1999;52:50-6.
13. Kraft GH. Rehabilitation still the only way to improve function in multiple sclerosis. *Lancet* 1999;354:2016.
14. Di Fabio RP, Choi T, Soderberg J, Hansen CR. Health-related quality of life for patients with progressive multiple sclerosis. Influence of rehabilitation. *Phys Ther* 1997;77:1704-16.
15. Petajan JH, Gappmaier E, White AT, Spencer MK, Mino L, Hicks RW. Impact of aerobic training on fitness and quality of life in multiple sclerosis. *Ann Neurol* 1996;39:432-41.
16. Svensson B, Gerdle B, Elert J. Endurance training in patients with multiple sclerosis. Five case studies. *Phys Ther* 1994;74:1017-26.
17. Solari A, Fillippini G, Gasco P, Colla L, Salmaggi A, La Mantia L, et al. Physical rehabilitation has a positive effect on disability in multiple sclerosis patients. *Neurology* 1999;52:57-62.

18. Gehlsen GM, Gisby SA, Winant D. Effects of an aquatic fitness program on the muscular strength and endurance of patients with multiple sclerosis. *Phys Ther* 1984;64:653-7.
19. Wiles CM, Newcombe RG, Fuller KJ, Shaw J, Furnival-Doran J, Pickersgill TP, et al. Controlled randomised crossover trial of the effects of physiotherapy on mobility in chronic multiple sclerosis. *J Neurol Neurosurg Psychiatry* 2001;70:174-9.
20. Lord SE, Wade DT, Halligan PW. A comparison of two physiotherapy treatment approaches to improve walking in multiple sclerosis. A pilot randomized controlled study. *Clin Rehabil* 1998;12:477-86.
21. Ponichtera-Mulcare JA. Exercise and multiple sclerosis. *Med Sci Sports Exer* 1993;25:451-65.
22. Mostert S, Kesselring J. Effects of a short-term exercise training program on aerobic fitness, fatigue, health perception and activity level of subjects with multiple sclerosis. *Multiple Sclerosis* 2002;8:161-8.
23. Di Fabio RP, Soderberg J, Choi T, Hansen CR, Schapiro RT. Extended outpatient rehabilitation. Its influence on symptom frequency, fatigue and functional status for persons with progressive multiple sclerosis. *Arch Phys Med Rehabil* 1998;79:141-6.
24. Ponichtera-Mulcare JA, Mathews T, Glaser RM, Gupta SC. Maximal aerobic exercise of individuals with multiple sclerosis using three modes of ergometry. *Clin Kinesiol* 1995;49:4-13.
25. Romberg A, Virtanen A, Ruutinen J, Aunola S, Karppi SL, Vaara M, et al. Effects of a 6-month exercise program on patients with multiple sclerosis. A randomized study. *Neurology* 2004;63:2034-8.
26. Rietberg MB, Brooks D, Uitdehaag BM, Kwakkel G. Exercise therapy for multiple sclerosis. *Cochrane Database Syst Rev*. 2005 Jan 25;(1):CD003980.
27. Motl RW, Gosney JL. Effect of exercise training on quality of life in multiple sclerosis. A meta-analysis. *Mult Scler* 2007;Sep 19. (Epub ahead of print)
28. Khan F, Turner-Stokes L, Ng L, Kilpatrick T. Multidisciplinary rehabilitation for adults with multiple sclerosis. *Cochrane Database of Systematic Reviews* 2007;2. Art. No. CD006036. DOI: 10.1002/14651858.CD006036.pub2.
29. Rampello A, Franceschini M, Piepoli M, Antenucci R, Lenti G, Olivieri D et al. Effect of aerobic training on walking capacity and maximal exercise tolerance in patients with multiple sclerosis. A randomized crossover controlled study. *Physical Therapy* 2007;87:545-55.
30. White LJ, Dressendorfer RH. Exercise and multiple sclerosis. *Sports med* 2004;34:1077-100.
31. Stuijbergen AK. Physical activity and perceived health status in persons with multiple sclerosis. *J Neurosci Nurs* 1997;29:238-43.
32. Stuijbergen AK, Roberts GJ. Health promotion practices of women with multiple sclerosis. *Arch Phys Med Rehabil* 1997;78:S3-9.
33. Busse ME, Pearson OR, Van Deursen R, Wiles CM. Quantified measurement of activity provides insight into motor function and recovery in neurological disease. *J Neurol Neurosurg Psychiatry* 2004;75:884-8.

34. Ng AV, Kent-Braun J. Quantification of lower physical activity in persons with multiple sclerosis. *Med Sci Sports Exer* 1997;29:517-23.
35. Krupp LB, Alvarez LA, LaRocca NG, Sceinberg LC. Fatigue in multiple sclerosis. *Arch Neurol* 1988;45:435-7.
36. Johansson S, Ytterberg C, Claesson IM, Lindberg J, Hillert J, Andersson M, et al. High concurrent presence of disability in multiple sclerosis. Associations with perceived health. *J Neurol* 2007;254:767-73.
37. Schwid SR, Thornton CA, Pandya S, Manzur KL, Sanjak M, Petrie MD, et al. Quantitative assessment of motor fatigue and strength in MS. *Neurology* 1999;53:743-50.
38. Mathiowetz V, Matuska KM, Murphy ME. Efficacy of an energy conservation course for persons with multiple sclerosis. *Arch Phys Med Rehabil* 2001;82:449-56.
39. Multiple Sclerosis Clinical Practice Guideline. Fatigue and multiple sclerosis. Evidence-based management strategies for fatigue in multiple sclerosis. Washington (DC): Paralyzed Veterans Association; 1999. Downloaded 25.03.2003 via <http://www.pva.org/NEWPVASITE/publications/pubs/mscpg.htm>.
40. Olgati R, Burgunder JM, Mumenthaler M. Increased energy cost of walking in multiple sclerosis. Effect of spasticity, ataxia and weakness. *Arch Phys Med Rehabil* 1988;69:846-9.
41. Zetterberg L, Lindmark B. Energikostnad vid gång. [Energy cost of walking] En jämförande studie av personer med och utan multipel skleros. [A comparative study of persons with and without multiple sclerosis] *Nordisk Fysioterapi* 2000;4:21-8.
42. Whitlock FA, Suskind MM. Depression as a major symptom of multiple sclerosis. *J Neurol Neurosurg Psychiatry* 1980;43:861-5.
43. Bakshi R, Shaikh ZA, Miltich RS, Czarnecki D, Dmochowski J, Henschel K, et al. Fatigue in multiple sclerosis and its relationship to depression and neurologic disability. *Multiple Sclerosis* 2000;6:181-5.
44. Krupp LB, Elkins LE. Fatigue and declines in cognitive functioning in multiple sclerosis. *Neurology* 2000;55:934-9.
45. Krupp LB. Fatigue in multiple sclerosis. A guide to diagnosis and management. New York: Demos Medical Publishing Inc; 2004. pp. 27, 64.
46. Iriate J. Correlation between symptom fatigue and muscular fatigue in multiple sclerosis. *Eur J Neurol* 1998;5:579-85.
47. Sharma KR, Kent-Braun J, Mynhier MA, Weiner MW, Miller RG. Evidence of an abnormal intramuscular component of fatigue in multiple sclerosis. *Muscle Nerve* 1995;18:1403-11.
48. Romberg A, Ruutiainen J, Puukka P, Poikkeus L. Fatigue in multiple sclerosis patients during inpatient rehabilitation. *Disabil Rehabil* 2007;12:1-6.
49. Chan A. Review of common management strategies for fatigue in multiple sclerosis. *Int J Ms Care* 1999;dec:12-9. Downloaded via www.ms-care.org.
50. Bajada S, Mastaglia FL, Black JL, Collins DWK. Effects of induced hyperthermia on visual evoked and saccade parameters in normal subjects and multiple sclerosis patients. *J Neurol Neurosurg Psychiatry* 1980;43:849-52.

51. Namerow NS. Temperature effect on critical flicker fusion in multiple sclerosis. *Arch Neurol* 1971;25:269-75.
52. Davies FA, Michael JA, Tomaszewski JS. Fluctuation of motor functions in multiple sclerosis. *Dis Nerv Syst* 1973;34:33-6.
53. van Diemen HA, van Dongen MM, Dammers JW, Polman CH. Increased visual impairments after exercise (Uhthoff's phenomenon) in multiple sclerosis. Therapeutic possibilities. *Eur Neurol* 1992;32:231-4.
54. Capello E, Gardella M, Leandri M, Abbruzzese G, Minatel C, Tartaglione A, et al. Lowering body temperature with a cooling suit as symptomatic treatment for thermo-sensitive multiple sclerosis patients. *Ital J Neurol Sci* 1995;16:533-9.
55. Kinnman J, Andersson U, Kinnman Y, Wetterqvist L. Temporary improvement of motor function with multiple sclerosis after treatment with a cooling suit. *J Neuro Rehab* 1997;11:109-14.
56. Kinnman J, Andersson T, Andersson G. Effect of cooling suit treatment in patients with multiple sclerosis evaluated by evoked potentials. *Scand J Rehab Med* 2000;32:16-9.
57. Kinnman J, Andersson U, Wetterqvist L, Kinnman Y, Andersson U. Cooling suit for multiple sclerosis. Functional improvement in daily living? *Scand J Rehab Med* 2000;32:20-4.
58. Flensner G, Lindencrona C. The cooling-suit. A study of ten multiple sclerosis patients experiences in daily life. *J Adv Nurs* 1999;30:775.
59. Flensner G, Lindencrona C. The cooling-suit. Case studies of its influence on fatigue among eight individuals with multiple sclerosis. *J Adv Nurs* 2002;37:541-50.
60. Nilsagard Y, Denison E, Gunnarsson L. Evaluation of a single session with cooling garment for persons with multiple sclerosis. A randomized trial. *Disability and Rehabilitation: Assistive Technology* 2006;1:225-33.
61. de Souza LH, Ashburn A. Assessment of motor function in people with multiple sclerosis. *Physiother Res Int* 1996;1:98-111.
62. Thompson AJ. Symptomatic management and rehabilitation in multiple sclerosis. *J Neurol Neurosurg Psychiatry* 2001;71:ii22-i7.
63. Slawta JN, McCubbin JA, Wilcox AR, Fox SD, Nalle DJ, Andersson G. Coronary heart disease risk between active and inactive women with multiple sclerosis. *Med Sci Sports Exerc* 2002;34:905-12.
64. Herndon RM, Mohandas N. Osteoporosis in multiple sclerosis. A frequent, serious and under-recognized problem. *Int Journal of MS Care* 2000;2:5-12.
65. Petjan JH, White AT. Recommendations for physical activity in patients with multiple sclerosis. *Sports Med* 1999;27:179-91.
66. Pozzilli C, Brunetti M, Amicosante AMW, Gasperini C, Ristori G, Palmisano L, et al. Home based management in multiple sclerosis. Results of a randomised controlled trial. *J Neurol Neurosurg Psych* 2002;73:250-5.
67. Collen FM, Wade DT, Robb GF, Bradshaw CM. The Rivermead mobility index. A further development of the Rivermead Motor Assessment. *Int Disabil Studies* 1991;13:50-4.
68. Vaney C, Blaurock H, Gattlen PT, Meisels C. Assessing mobility in multiple sclerosis using the Rivermead mobility index and gait speed. *Clin Rehabil* 1996;10:216-26.

69. Lindmark B, Hamrin E. Evaluation of functional capacity after stroke as a basis for active intervention. Presentation of a modified chart for motor capacity assessment and its reliability. *Scand J Rehabil Med* 1988;20:103-9.
70. Rudick R, Antel J, Confavreux C, Cutter G, Ellison G, Fischer J, et al. Recommendations from the National Multiple Sclerosis Society clinical outcomes assessment task force. *Ann Neurol* 1997;42:379-82.
71. Wade DT, Wood VA, Heller A, Maggs J, Langton Hewer R. Walking after stroke. Measurement and recovery over the first 3 months. *Scand J Rehabil Med* 1987;19:25-30.
72. Podsiadlo D, Richardson S. The 'Timed Up and Go'. A test of basic functional mobility for frail elderly persons. *J Am Geriatr Soc* 1991;39:142-8.
73. Nilsagard Y, Lundholm C, Gunnarsson LG, Denison E. Clinical relevance using timed walk tests and 'timed up and go' testing in persons with multiple sclerosis. *Physiother Res Int* 2007;12:105-14.
74. McGregor J. The objective measurement of physical performance with long term ambulatory physiological surveillance equipment. Proceedings of 3rd International Symposium on Ambulatory Monitoring, Harrow; 1979.
75. Bailey MJ, Ratcliff CM. Reliability of physiological cost index. Measurements in walking normal subjects using steady-state, non steady-state and post exercise heart rate recording. *Physiotherapy* 1995;81:618-23.
76. Guyatt G, Sullivan M, Thompson P, Fallen E, Pugsley S, Taylor D, et al. The 6-minute walk. A new measure of exercise capacity in patients with chronic heart failure. *Can Med Assoc J* 1985;32:919-23.
77. Guyatt G. Use of the six-minute walk test as an outcome measure in clinical trials in chronic heart failure. *Heart Failure* 1987;21:211-7.
78. Berg KO, Wood-Dauphinee SL, Williams JI, Maki B. Measuring balance in the elderly. Validation of an instrument. *Can J Public Health* 1992;83 Suppl 2:S7-11.
79. Berg K, Wood-Dauphinee S, Williams JI. The balance scale. Reliability assessment with elderly residents and patients with an acute stroke. *Scand J Rehabil Med* 1995;27:27-36.
80. Hobart JC, Riazi A, Lamping DL, Fitzpatrick R, Thompson AJ. Measuring the impact of MS on walking ability. The 12-item MS walking scale (MSWS-12). *Neurology* 2003;60:31-6.
81. McGuigan C, Hutchinson M. Confirming the validity and responsiveness of the Multiple Sclerosis Walking Scale-12 (MSWS-12). *Neurology* 2004;62:2103-5.
82. Nilsagard Y, Gunnarsson L-G, Denison E. Self-perceived limitations of gait in persons with multiple sclerosis. *Advances in Physiotherapy* 2007;9:136-43.
83. Bergner M, Bobbit RA, Carter WB, Gilson BS. The Sickness Impact Profile. Development and final revision of a health status measure. *Medical Care* 1981;19:787-805.
84. Ware JE, Sherbourne CD. The MOS 36-Item short form healthy survey (SF-36) I. Conceptual framework and item selection. *Med Care* 1992;30:473-83.
85. McHorney CA, Ware JE Jr, Raczek AE. The MOS 36-Item short form healthy survey (SF-36) II. Psychometric and clinical tests of validity in measuring physical and mental health constructs. *Med Care* 1993;31:247-63.

86. Hobart J, Lamping D, Fitzpatrick R, Riazi A, Thompson A. The Multiple Sclerosis Impact Scale (MSIS-29). A new patient-based outcome measure. *Brain* 2001;124:962-73.
87. Vickery BG, Hays RD, Harooni R, Myers LW, Ellison GW. A health-related quality of life measure for multiple sclerosis. *Quality of Life Research* 1995;4:187-206.
88. Krupp LB, LaRocca NG, Muir-Nash J, Steinberg AD. The Fatigue Severity Scale. *Arch Neurol* 1989;46:1121-3.
89. Fick JD, Pontefract A, Ritvo PG, Archibald CJ, Murray TJ. The impact of fatigue on patients with multiple sclerosis. *Can J Neurol Sci* 1994;21:9-14.
90. Iriarte J, Katsamakos G, de Castro P. The Fatigue Descriptive Scale (FDS). A useful tool to evaluate fatigue in multiple sclerosis. *Mult Scler* 1999;5:10-6.
91. Rossier P, Wade DT. The Guy's Neurological Disability Scale in patients with multiple sclerosis. A clinical evaluation of its reliability and validity. *Clin Rehabil* 2002 Feb;16:75-95.
92. Wade D, Legh-Smith L, Langton Hewer R. Social activities after stroke. Measurement and natural history using Frenchay Activities Index. *Int Rehabil Med* 1985;7:176-81.
93. Katz S, Ford AB, Moskowitz RW, Jackson BA, Jaffe MW. Studies of illness in the aged. The index of ADL. A standardized measure of biological and psychosocial function. *JAMA* 1963;185:914-9.

36. Obesity

Author

Stephan Rössner, MD, PhD, Professor, Obesity Unit, Karolinska University Hospital, Stockholm, Sweden

Summary

Body weight is the outcome of an energy balance, i.e. energy intake minus energy expenditure. While it is relatively easy to limit the daily energy intake by a few hundred kilocalories (kcal), it is significantly more difficult to increase the level of energy expenditure. In addition, activity advice given to overweight or obese individuals must be realistic as their mechanical ability may be impaired as a consequence of being overweight and obese. A small step forward such as an increased daily expenditure of 50–100 kcal is in fact an achievement and improvement as long as it is maintained. The long-term energy balance is most significant, largely involving a change in lifestyle. Choosing activity (walking, cycling, taking the stairs) instead of inactivity (watching TV, driving, taking the escalator). “Everyday physical activity” (also referred to as NEAT – Non-Exercise Activity Thermogenesis) is evidently effective and should be encouraged. Also, the extra muscle mass gained through physical activity improves the basal metabolism, making weight control easier. Successful weight loss through physical activity is just as much about eliminating mental obstacles as about actually performing physical activities.

Definition

Obesity is today the leading nutritional disorder in the Western World. There are more people in the world today that eat themselves to death than starve to death and international comparisons have made it possible to follow this explosive growth in obesity problems (1). The change in lifestyle that has occurred in the Western World over the past three to four decades has, for example, only been evident in Southeast Asia in the last couple of years. In the Pacific Islands, the most obese part of the world, more than 50 per cent of the population is obese. This dramatic trend of increasing overweight and obesity is now evident all over the world with a variety of illnesses that follow in its wake, further highlighting the consequences of obesity.

In Sweden, the number of obese people has increased by around 50 per cent in the past 25 years. This development is illustrated in Figure 1. Today, approximately 10 per cent of

the male population and 12 per cent of the female population are considered obese, defined as a body mass index (BMI) above 30 kg/m^2 . BMI is calculated as the body weight in kg divided by the square of the height in meters (kg/m^2). Whereas there is plenty of data about the prevalence of obesity among the adult population, there is significantly less data about children and adolescents. However, with regard to Swedish military servicemen, i.e. young men enrolling for compulsory military service at the age of 18, the data available is particularly reliable and indicates a dramatic increase in obesity. The proportion of obese 18-year-old men increased from 0.9 to 3.8 per cent from 1970 to 1994 (2). No comprehensive representative data exists in Sweden concerning children, although around 20 per cent are estimated to be obese. This figure is rising and the problem with obesity in Sweden is growing throughout the country regardless of age or social group. Most affected at the moment are middle-aged individuals in the inland parts of Norrland.

Obesity develops gradually. Our metabolism peaks at the age of 20, whereupon the basal metabolism decreases by about 1 per cent per annum. This means that young individuals of a normal weight will gain an average of 3–4 kg every decade. However, the risk of gaining even more weight than this increases for individuals that are overweight in childhood. It is estimated that many young people with childhood obesity gain around 1 kg in weight every year.

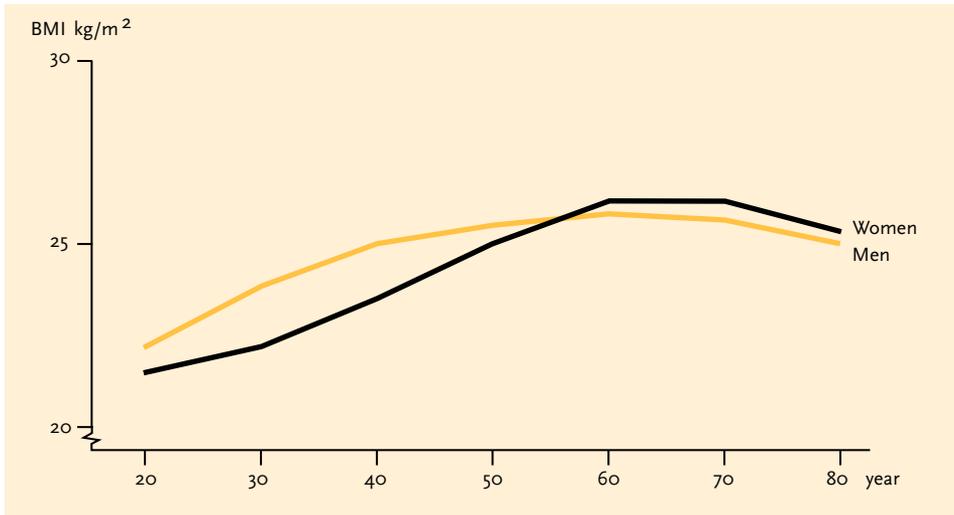


Figure 1. Weight development in Sweden over the past 25 years.

Most of the data available is cross-sectional, i.e. relating to individuals of different ages. No adequate longitudinal study has yet monitored the weight of individuals throughout their entire life. However, the cross-sectional data available indicates that the average weight of the population increases continuously until around 65 years of age when it starts to decrease (see Figure 2). There are many reasons for this break in the trend. It seems reasonable to assume that the older age group would include individuals that have passed away due to

complications caused by obesity and are thereby excluded from the statistics, which leaves behind less weighty and healthier individuals of a similar age. It is also possible that the muscle tissue deterioration or sarcopenia seen in older people leads to a weight loss except this is usually only a shift from less muscle tissue to more or an unchanged amount of fatty tissue. It is merely a shift of tissue proportions. In addition, the degree of tissue hydration decreases with age, affecting the body weight. Longitudinal data is still sparse. No study has monitored the weight of individuals throughout life. However, the preliminary data available shows that older people are also subject to a continuous weight increase.

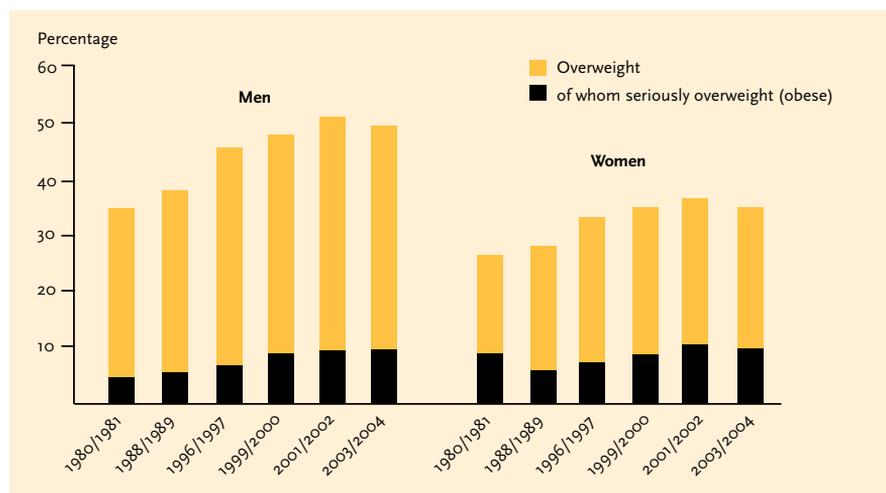


Figure 2. Cross-sectional data relating to weight developments in different age groups in Sweden.

As mentioned above, the obesity is defined using BMI kg/m^2 , a measurement that was first used in the middle of the 19th century to calculate relative body weight and compare individuals. The World Health Organization, WHO, uses BMI to define obesity as illustrated in Table 1. However, it is worth noting that the BMI value is neither age nor gender standardised and consequently provides an approximate estimate of the degree of obesity. A recurring objection to the use of BMI, particularly among athletes, is that BMI is an insufficient index of body fat as many well-trained individuals (with extra muscle mass) can have a BMI of over 30 without actually being fat. This is not untrue in itself, but irrelevant from a population perspective as only a negligible proportion of the population is classified in the incorrect BMI category.

In recent years, the significant risk factor of abdominal obesity has been recognised. The abdominal fat mass surrounds the organs in the gut and even a fairly modest volume has a major impact on metabolism. Abdominal fat mass can easily be determined by measuring the waist circumference, which is an increasingly common way to determine obesity (3). In a couple of years, BMI will probably only be used to classify individuals into categories of defined populations, while waist circumference will provide a more sensitive indication of fat deposits that may lead to health risks and the metabolic syndrome described

below. Table 1 shows the risk factor levels of men and women with a growing waistline. According to recent findings, measuring epicardial fat (the fat surrounding the heart) may also serve as an additional tool to assess the source of the risk, although the discovery of this potentially high-risk area of fat infiltration requires further verification.

Table 1. BMI and waist circumference used as risk classification according to WHO (1).

Classification	BMI	Health risk
Underweight	< 18.5	Low (but elevated risk of other clinical problems) < 18.5
Normal weight	18.5–24.99	Normal risk
Overweight	25–29.99	Slight increase in risk
Obesity Category I	30–34.99	Moderate increase in risk
Obesity Category II	35.00–39.99	High, serious increase in risk
Obesity Category III	≥ 40	Very high, extreme increase in risk

Gender specific waist circumference and risk of obesity related metabolic complications.

Risk of metabolic complications	Waist circumference (cm)	
	Men	Women
Increased risk	≥ 94	≥ 80
Severe increase in risk	≥ 102	≥ 88

Cause

The existence of obesity is very easily explained: Energy intake exceeds energy expenditure (or energy output). However, it is much more difficult to explain the acceleration of obesity recently seen around the world. The most plausible explanation is the fact that our genetic composition is not changing while the environment in which we live is undergoing a dramatic change (4). Our energy intake is influenced by the availability of large volumes of tasty high energy food with a high fat and sugar content. Our energy expenditure is reduced as a result of having created a society that requires minimal physical activity. Technical aids have limited the need for physical work which for many people means that their basal metabolism, i.e. the energy expenditure needed to keep a body ‘alive’, makes up more than 70 per cent of their total energy expenditure. High energy food combined with insignificant energy expenditure is therefore the likely cause of an impending obesity epidemic. Our genes tell us to eat when there is food as we are biologically programmed to cope with recurrent periods of starvation. However, the fact is that we have constant access to food and recurrent periods of starvation do not arise in our prosperity. As a result of learning how to refine sugar and purify fat – nutrients that did not exist in the Stone Age, the food we eat today is extremely high in energy. In addition, the flavours of sugar and fat complement each other very well, sending us irresistible signals that we should eat more.

There is a myth that overweight individuals constantly consume large amounts of fatty food. Binging does of course exist, but the majority of obese individuals have a small, but continuous positive energy balance. By adding as little as three lumps of sugar (approx. 42 kcal) every day to the food needed to maintain equilibrium, you will gain half a kilogramme of fatty tissue every year. The bad news is that even a small amount of excess energy will in the long run lead to a significant risk. However, the good news is that moderate, but regular exercise will keep the weight under control.

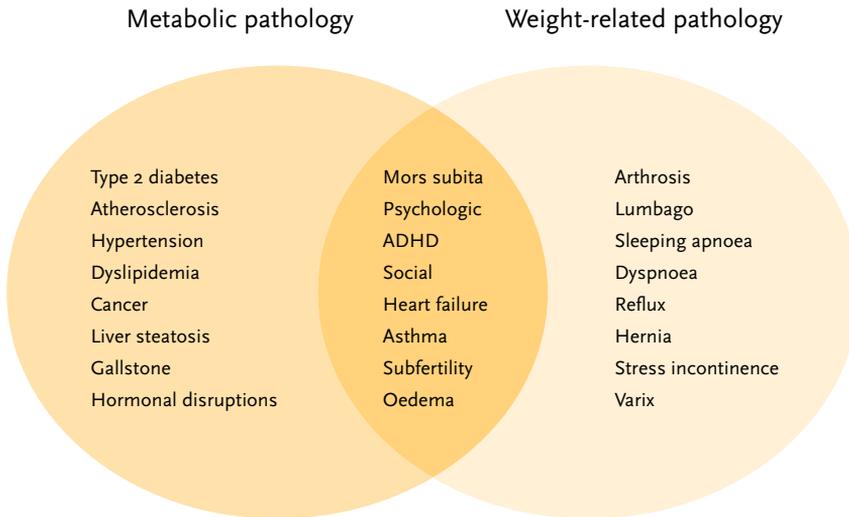


Figure 3. Complications of obesity.

Obesity gives rise to a number of risks (5, 6). Figure 3 shows metabolic and mechanical complications due to obesity as well as an area where these complications overlap. Most people know that high blood pressure (hypertension), lipid disorders (dyslipidemia) and diabetes are the most common companions of obesity, which together make up the so-called metabolic syndrome (7–10). It has been discussed whether metabolic syndrome in itself is an illness or merely coexisting complications of obesity.

It is not difficult to visualize the mechanical complications of obesity. The weight-bearing joints are clearly subject to added stress as a result of the increased weight load, but there are also other mechanical problems that are easily overlooked. Sleep apnoea is an underestimated public health problem that is particularly prevalent among overweight and obese individuals as the accumulation of fat around their air passages and abdomen leads to a feeling of pressure in the chest and difficulties breathing. Losing weight can have a dramatic effect on the health of these people. Incontinence is a problem that affects women in particular and intensifies with age. Obesity adds pressure on the urinary bladder and the short urethra, meaning weight loss would have a beneficial effect.

However, most sufferers find the psychological consequences of incontinence the hardest to deal with. Numerous studies have shown that obese people are subject to discrimination from an early age and that this discrimination continues into adult life (11). Consequently, obese and overweight people are more inclined to suffer from depression and despondency than people of a normal weight, and depression is even more manifest among those seeking help for their obesity.

Diagnosis

It is easy to diagnose someone as overweight or obese. However, in order to determine the type of obesity, it is important to measure the waist circumference as part of routine clinical practice. In case of serious obesity, it is often easier to measure the waist circumference with the patient reclined on a bed. The waist circumference is measured half-way between the twelfth rib and the iliac crest with the feet slightly parted and after normal exhalation.

Bioimpedance (tissue water conductivity converted to proportion of body fat) is a more detailed estimation of fatty tissue volume which becomes less precise as the severity of obesity increases. However, computed tomography and magnetic camera imaging can be used for research purposes, but is not suitable for routine clinical practice. All patients seeking medical care for one reason or another should have their height, weight and waist circumference measured. The baseline values obtained are used as a starting point for any future weight changes. Self-reported data is less useful as it is well-documented that obese individuals tend to give a lower weight than the actual weight as indicated by the scales.

Methodological problems

One of the greatest challenges of assessing the energy balance in overweight individuals is the reliability of the methods used. It is almost impossible to obtain an adequate dietary history of an obese person due to the large number of sources of error. Most people underestimate their true energy intake (12). The same applies to their energy expenditure. Even light exertion is a considerable effort for people with obesity who have a tendency to describe the same exertion as more of an effort than it actually was in terms of energy expenditure. This tendency to amplify the effort involved in physical exertion is also a major methodological problem. Expensive and advanced technology is required to make an objective assessment of an individual's energy intake and expenditure. Pedometers and pulse rate monitors only provide an approximation of the energy expenditure and double labelled water with stable isotopes or metabolic chambers are extremely expensive and resource-intensive technologies that can only be used in individual cases and not on a population level. Everyday movements or NEAT (see below) that are not detected by conventional measurement instruments may still have an effect the total daily metabolic rate.

Physical activity and obesity

The so-called non-exercise activity thermogenesis or NEAT refers to daily physical activities that are not perceived as exercise or fitness training. Standing instead of sitting, moving around and even laughing affects our basal metabolism. Such activities can amount to 150–250 kcal a day. This is a significant part of our energy expenditure that ultimately could improve our chances of maintaining a good energy balance.

There are two main objectives for physical activity in obesity (13). Physical activity obviously boosts the energy expenditure. However, the human body is extremely energy efficient. Losing weight by merely increasing physical activity is very difficult in practice. Many overweight and obese individuals also find it difficult to move around because of the mechanical load, risk of injury, etc. One important positive effect of physical activity is an increase in muscle mass and subsequent improvement in the basal metabolic rate. A large muscle mass improves the chances of maintaining a high level of energy expenditure. The muscle mass can only be increased through physical activity while the volume of fatty tissue can grow without limit through constant overconsumption. Table 2 is a summary of the effects of physical activity on metabolic functions.

Sarcopenia is defined as the age-related loss of muscle mass. This loss of muscle mass cannot be prevented, but can be mitigated with an active lifestyle. Strength training, for example, is an excellent form of exercise in this respect.

The assessment is further complicated by the fact that obesity is the result of a long-term positive energy balance. Not even the most advanced technology can measure disequilibria of less than 100 kcal a day, but a comparable energy imbalance is equivalent to one kilogramme of adipose tissue per year.

Table 2. Effects of high intensity activity on metabolic factors related to weight loss.

Reduction in volume of subcutaneous adipose tissue.
Increased mobilisation of adipose tissue.
Increased muscle lipolysis.
Less tendency to overeat following activity.
Increased metabolic rate and fat oxidation.

Treatment principles

Prevention is obviously a fundamental principle. Because overweight children often become obese adults, focus is currently aimed at the identification of effective and preventative strategies. This falls far outside of the normal realms of national healthcare services: Good food in preschools and primary schools, possibility of physical activities, physical education and athletics not just for the most competent school children, but available to all school children and a lifestyle that promotes a healthy diet and physical activity. The Swedish National Institute of Public Health and the National Food Agency recently presented the results of a study including 79 proposals on how to improve public health (14).

Other treatment principles

Diet, exercise and behavioural change are the cornerstones of obesity treatment. Most people are familiar with these measures. Regular meals, compliance with the Balanced Diet Chart and Eatwell Plate Model based on breakfast, lunch and dinner at regular times with two snacks between meals. The basic advice is a low-fat, fibrous diet and moderation in alcohol consumption. Physical activity is another important ingredient, i.e. everything from general mobility to organised exercise activities as described below (15).

Discount prescription drugs are currently available to obese people with a BMI of over 35 or a BMI of over 28 with an added obesity-related complication like diabetes. There are currently three such drugs available.

Xenical is not absorbed by the body, but passes through the gastrointestinal tract while at the same time reducing lipase activity in the intestine whereupon around 30 per cent of fat is expelled in faeces. The drug is often said to have an “antabuse effect”. A continued diet of fatty food will give unpleasant diarrhoea as a result of which the patient soon learns to eat healthier food. This means low-fat food, which in itself leads to weight loss in addition to the fact that Xenical will also extract and expel fat from a healthier low fat diet. The medication is taken three times a day together with food and there are no real side-effects apart from the discomfort of diarrhoea as mentioned above. However, this is more a pharmacological consequence of the intrinsic properties of the drug rather than a genuine pharmacological side-effect.

Reductil works on the central nervous system as an appetite suppressant, actively lessening the desire for food and with an added thermogenic effect. Reductil is taken once a day and leads to weight loss, but it also has other desirable effects such as an increased level of the HDL cholesterol (“the good cholesterol”). Because Reductil is slightly adrenergic, i.e. it stimulates the sympathetic nervous system, the reduction of blood pressure is not quite as noticeable as with other medications. Consequently, caution should be observed and blood pressure taken before prescribing the drug. It has also been noted that obese people with high blood pressure (a common combination) actually see their blood pressure fall when they start to lose weight. Known side-effects of Reductil include constipation, a dry mouth and initial sleeping problems. However, these side-effects are generally transient.

Acomplia is a so-called cannabinoid receptor blocker that works both on the central nervous system and in the peripheral gastrointestinal tract, as well as other places such as the liver and muscular system. Acomplia produces weight loss by regulating food intake while also possibly affecting the metabolic system. The side-effects associated with this drug are generally psychological in nature and Acomplia should therefore not be prescribed to patients with a risk of depression. Other patients should be monitored to prevent the development of depression, which although unusual, must be considered a serious side-effect.

Many preparations claimed to reduce weight are available for purchase over the Internet or in shops. Some of the products available from health food shops are either hazardous to health or at best completely ineffective. Mechanical aids such as “slimming underwear”, “slimming sauna belts”, etc. have no effect whatsoever. Serious side-effects and even fatalities have been reported as a result of using these types of slimming methods.

Realistic expectations

A normal weight is seldom achieved with treatment. However, a permanent weight loss of 5–10 per cent will have a sufficient beneficial metabolic effect and it is very important for the patient to have a realistic expectation on what can be achieved with a treatment programme. Many patients give up on the programme when they do not achieve the unrealistic weight loss they had hoped for. The greatest weight loss usually takes place within the first six months. Most people are able to lose weight. However, the difficulty lies in maintaining the new lower weight for the foreseeable future as relapses are unfortunately not uncommon. Consequently, most weight-loss strategies focus on a permanent and long-term change in behaviour.

Surgery is the most effective treatment of obesity. A number of operations resulting in weight loss can be performed today. However, these operations always entail a lifelong change of circumstances. However, the Swedish Obese Subjects Study (SOS) showed that obesity surgery gave an average weight loss of around 16 kg after a period of 10 years and a 30 per cent reduction in mortality and morbidity. Also, surgery-induced weight loss eliminates diabetes almost completely. New onset diabetes is reduced by one thirtieth. A transient normalisation of changes in lipids and high blood pressure also takes place.

Effects of physical activity in obesity

The effect of physical activity in obesity is described in Figure 4. The dosage-time product is a health benefit. The more often, intensive and longer we exercise, the greater the amount of added physical activity. The individual ability to foster this new found legacy determines whether the change will result in a permanently improved state of health. Some people find it more difficult than others to change their behaviour and are more likely to relapse. Naturally, it is difficult to maintain a changed approach to physical activity in an environment that resists new and better behaviours. Work, family and financial pressures can put a stop to even the most ambitious individual exercise campaign. Those who manage to change their pattern of behaviour will of course reap the benefits over time. Weight loss not only entails a better reading on the scales, but also has effects that the individual cannot see with the naked eye, such as improved metabolic control, disease prevention and other psychological benefits. Surveys have shown that even a moderate weight loss through increased physical activity improves the quality of life.

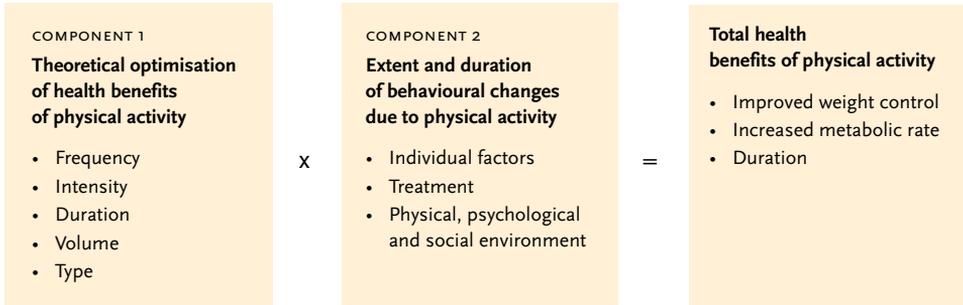


Figure 4. The effect of physical activity on health benefits according to Faskunger (15).

Everyday physical activity

Everyday physical activities are part of NEAT where every moment in life is used to improve the metabolic rate. For example, standing instead of sitting, not using machinery to make work easier and parking the car in the furthest corner of the car park are all examples of actions that promote everyday physical activities and can help shift the energy balance in the right direction.

A standard argument against the use of exercise in obesity is that it is too much of an effort. This incorrect reasoning is based on the old concept that the desired effect is only achieved through great intensity and endurance. This old data relate to an era when the demands and conditions of people in favour of everyday exercise were not taken into consideration. Today, we know that any activity, no matter how small, is better than sitting still and the good news is that accumulated daily physical activities count towards our overall energy expenditure.

Reasonable activity advice

It is easy to make demands on overweight and obese individuals in the belief that they will perform miracles despite their body mass and muscle composition. In fact, quite the opposite occurs. As part of a study conducted by Karolinska University Hospital, women of a normal weight were asked to walk at their own speed across a level floor for a few minutes. To do this, the women that were untrained required approximately 30 per cent of their maximum capacity. However, obese women required more than double this capacity to perform the same task. When asking obese people to ‘take a walk’, we are in fact asking them to perform a laborious task which is naturally met with resistance. Consequently, it is important to find realistic forms of treatment that are enjoyable and reasonable. Water gymnastics, cycling and swimming are exercises that have been shown to be especially effective against obesity as they put less strain on weight-bearing joints.

“Fat, but fit”

As almost 50 per cent of obesity is linked to genetic susceptibility, the best approach to advising obese people on physical activity has come under scrutiny. As a result, researchers in the United States have been able to show that it is possible to be “fat, but fit”. In other words, people with a strong susceptibility to obesity should continue exercising since, although they will never achieve a normal body weight, their fitness will improve and lead to health benefits.

Previous discussions have focused on the need for physical activity and *increased mobility* in obesity including proposals for various activity programmes. However, researchers have recently discovered the opposite, i.e. that the length of time *sitting still every day* is a risk factor. Watching TV, computer games, etc. can in fact be seen as missed opportunities of carrying out a physical activity. The results of many surveys show that the more time we spend idle every day, the higher the risk of being overweight. It is actually possible to lose weight by just cutting down on the time spent in front of the TV or games console, which is very much a focal point in the development of a healthy lifestyle programme for children and adolescents. Disallowing or minimising the time spent in front of the TV/computer will hopefully add appeal to other more energy demanding activities.

Objective registration

There is plenty of evidence to suggest that keeping a record of one’s activities leads to a positive change in behaviour when treating obesity. Physical activity is particularly suitable for this as nearly all daily movement can be monitored with a pedometer. Pedometers come in various qualities and with somewhat different measurement functions, but a simple pedometer counting the steps taken every day is usually sufficient without having to use complicated conversion tables according to age and gender. Recording the amount of steps taken every day is what leads to a change in attitude. Many experts fail to agree on the level of activity, but the general notion is that everyone should be as active as possible. However, people suffering from obesity may find it difficult to start an activity purely due to mechanical problems such as joint arthrosis, breathlessness, sweating and inner thigh abrasions (women in particular).

The American study National Weight Control Registry is a summary of the most important factors for successfully maintained weight loss subsequent to the completion of an initial programme (16–19). It is evident from the self-reported material of this obviously selected and successful group that its achievement is attributable to the following four simple factors:

1. Eating breakfast as a manifestation of a structured existence.
2. A fibrous low-fat diet.
3. Log books to record energy intake and expenditure.
4. More than 150 minutes of activity every week.

These patients had lost weight in quite different ways and hence, it was difficult to find common patterns for success. Yet, identifying the four factors listed above proved to be a lot easier, increasing the odds of maintaining the lower weight.

Physical activity and appetite

From a physiological viewpoint, long-term weight control should lead to a greater appetite and an increased energy intake because of the higher level of physical activity. However, the situation is somewhat more complicated in reality. It appears that obese individuals do not immediately compensate for exercise by eating more afterwards. In fact, it just goes to prove that having dinner after a training session is the rational thing to do. However, activity programmes do not seem to have a long-term effect on food intake or appetite, which is positive, as it allows for activities to be used as weight control instruments without the individual having an uphill struggle with a compensatory appetite increase. Nor do activity programmes seem to manoeuvre the food intake in a specific direction of preference for fat, carbohydrates or protein.

It is widely recognised that physical activity increases the feeling of well-being for obese individuals. Exercise alleviates depression, reduces anxiety, raises the mood and relieves stress. Exercise also appears to have a positive effect on the body perception of obese individuals who as a result of the increased activity adapt a more positive attitude to their personal appearance. Self-confidence and the ability to cope with challenges in life also improve as a result of weight loss due to physical exercise.

Table 2 is a summary of the metabolic effects of intensive physical activity.

Obstacles to change

The psychopathology of obesity is often characterised by procrastination, i.e. the deferment of actions or tasks to a later time. Obese patients often find a reason not to carry out a physical activity, some of which are listed in Table 3.

Table 3. Obstacles to behaviour change and counter-strategies.

1.	I do not have time.	A common reason for not wanting to perform a physical activity. However, not much extra time is needed if the physical activity is planned for and integrated with other everyday activities and, with a little planning, the majority of people can usually manage to squeeze in 30 minutes of physical activity into their daily schedules.
2.	I cannot afford it.	There is no need for expensive gym memberships or training equipment, many excellent forms of exercise are completely free of charge.
3.	I do not like exercising.	Bad experiences at school or during compulsory military service and tedious training can lead to a loss of motivation. Everyone can find an enjoyable form of activity such as ball games, gardening and other activities together with children, friends and colleagues.
4.	I am too old.	Researchers agree that physical activity is generally beneficial for people well into their 80's. One's personal performance can be improved well into the later years of life provided that the skeleton and joints are not put under too much strain.
5.	I might injure myself.	Outdated and tough exercise programmes often lead to injuries, sprains and pains. There is little risk of a problem if one starts carefully and gradually builds up mobility. People with a cardiovascular or serious chronic disease may need a medical assessment before starting a physical activity. However, there is an activity programme to suit everyone.
6.	I am too fat.	This is circular reasoning – if you never start any physical activity, then you will always be obese. The circle must be broken and the simplest way to do so is with moderate and modified activities that do not subject the body to undue strain.
7.	No one that I know exercises.	Someone has to be first and you often get an amazing response from everyone else when taking the initiative. The rewards are improved health, increased mobility and less risk of complications owing to obesity, which must after all be considered worth the effort.
8.	I do not like sweating.	It is not necessary to sweat copious amounts to get enjoyment out of a training session. However, a good rule to follow is that you should need to take a shower and change your clothes after exercise at least 2–3 times a week. The exercise in question may take the form of walking, cycling, picking mushrooms in the forest, a long stroll around a museum or whatever else takes your fancy. A dog provides the perfect motivation to exercise. “Take your dog for a walk even if you do not have one” is sometimes the advice given to obese people.

Social strategies

Primary prevention must permeate our entire society to stop the accelerating obesity epidemic. This is achievable in many ways: Providing the possibility of activity at preschools and primary schools with a school yard that encourages physical activity. So-called walking school buses through residential areas where children are walked to school by parents with the first and last parent in the line of walking children holding a flag. Walking school buses are part of a pilot scheme encouraging children to get to school

safely without having to use means of transport. An outdoor activity at the nursery school or a fun excursion instead of ice-cream and cake for a birthday celebration is a new way of thinking. Physical education at school not just for the elite, but structured so the heavier children also feel a sense of accomplishment, such as activities like swimming, giving the children who need exercise the most a chance. Stairs that encourage activity instead of being cold, dirty and frightening, and alternatives to escalators and lifts make it possible to choose more physical activity. Well-lighted walkways and jogging paths make it safer for older people to exercise outdoors. Side-walks, rather than just roads for cars, make it possible to go for a walk. Cycling paths make cycling safer. Good bicycle stands for locking one's bicycle and preventing theft increases the likelihood of people cycling as a means of transport. A good public transport system makes walking in conjunction with travel possible. TV free evenings involving doing something together rather than sitting still increase the level of activity. Discounts provided through work on health-promoting measures, lunchtime Nordic walking clubs, and competitions between departments at work to see which one is the most active usually raise employee interest in physical activities. Only a lack of imagination limits the list of possible activities.

Functional tests

Individuals with an uncomplicated obesity do not have to undergo complex examinations before commencing a general activity programme that involves, for example, walking and swimming. However, for individuals with a history of cardiovascular disease who need to start a systematic training programme, it is recommended that a medical examination and fitness assessment test are carried out. However, the patients themselves usually have a clear picture of the factors limiting their mobility.

Certain conditions make it difficult to put together a training programme that for example includes swimming. Some people have a fear of water and have never learnt how to swim or they are allergic to chlorine. Unfortunately, this eliminates that specific form of physical activity.

Type 2 diabetes and obesity are closely linked. There is enough of evidence to show that people with diabetes are able to lose weight although not generally as much as people without diabetes (20). However, it is well documented that obese diabetics gain just as much from losing weight as anyone else. There are specific reasons why diabetics cannot lose as much weight as other people. When diagnosed with obesity, diabetics are often slightly older than other sections of the population, and their higher age entails a lower basal metabolic rate and reduced potential for further weight loss. In addition, many diabetics are prescribed beta blockers, making it even more difficult to carry out a physical activity. Vascular spasm (angina pectoris) or diabetic foot conditions also make certain forms of exercise difficult or impossible to perform. In addition, diabetic patients are more inclined to suffer from depression and as a result, more likely to resist the thought of exercise.

Drug interaction

Patients with obesity and metabolic syndrome are often treated with a number of drugs such as beta blockers which make it difficult and burdensome to carry out physical activity. A medication-induced blood pressure reduction may give rise to nausea, making it more difficult to exercise, and diabetes treated with medication may lead to the blood sugar level falling, particularly during physical activity. However, situations like these do not give rise to any diagnostic problems and are usually recognised by both patients and healthcare professionals.

Contraindications

Obese patients often suffer from pain in their weight-bearing joints, back, hips, knees and ankle joints. Consequently, the physical activity must be modified accordingly. Water gymnastics are an excellent form of exercise for patients with joint pain. However, it may prove impossible for the patient to participate in water gymnastics if he or she is allergic to chlorine, a non-swimmer or afraid of water. Another recommended form of low-impact exercise is Nordic walking.

Risks

The paradox of patients losing weight while taking part in an activity programme is that they sometimes experience a higher degree of pain in, for example, the back despite having lost 15 kg. This is usually due to a load displacement from one section of the spine to another and other joints having to carry the load redistribution. The problem is usually transient, but it is important to inform the patient that he or she may still continue with physical activity. The risks involved are generally regarded as insignificant and a little extra care at the gym, in the swimming pool or when walking in the forest or along a foot-path is usually all that is needed.

References

1. WHO. Obesity. Preventing and managing the global epidemic. Report of a WHO consultation. WHO Technical Report Series, No. 894. Geneva: World Health Organization; 2000.
2. Rasmussen F, Johansson M, Hansen HO. Trends in overweight and obesity among 18-year-old males in Sweden between 1971 and 1995. *Acta Paediatr* 1999;88:431-7.
3. Janssen I, Katzmarzyk PT, Ross R. Waist circumference and not body mass index explains obesity-related health risk. *Am J Clin Nutr* 2004;79:379-84.
4. Perusse L, Bouchard C. Genotype-environment interaction in human obesity. *Nutr Rev* 1999;57:31-7.
5. Björntorp P. Overweight is risking fate. *Ballière's Clin Endocr Metab* 1999;13:47-69.
6. Francischetti EA, Genelhu VA. Obesity-hypertension. An ongoing pandemic. *Int J Clin Pract* 2007;61:269-80.
7. Despres JP, Lemieux I. Abdominal obesity and metabolic syndrome. *Nature* 2006;444:881-7.
8. Carey VJ, Walters EE, Colditz GA, et al. Body fat distribution and risk of non-insulin-dependent diabetes mellitus in women. The Nurses' Health Study. *Am J Epidemiol* 1997;145:614-9.
9. Defay R, Delcourt C, Ranvier M, Lacroux A, Papoz L. Relationships between physical activity, obesity and diabetes mellitus in a French elderly population. The POLA study. Pathologies Oculaires liées à l'Age. [Population-based study on age-related eye diseases] *Int J Obes Relat Metab Disord* 2001;25:512-8.
10. Hu G, Lindstrom J, Valle TT, et al. Physical activity, body mass index and risk of Type 2 diabetes in patients with normal or impaired glucose regulation. *Arch Intern Med* 2004;164:892-6.
11. Puhl R, Brownell KD. Bias, discrimination and obesity. *Obes Res* 2001;9:788-805.
12. Lichtman SW, Pisarska K, Berman ER, et al. Discrepancy between self-reported and actual caloric intake and exercise in obese subjects. *N Engl J Med* 1992;327:1893-8.
13. Ainsworth BE. Compendium of physical activities. Classification of energy costs of human physical activities. *Med Sci Sports Exerc* 1993;25:71-80.
14. Swedish National Institute of Public Health Handlingsplan för goda matvanor och ökad fysisk aktivitet i befolkningen. [Action plan on healthy dietary habits and increased physical activity] S2005/1274/FH. Stockholm: Swedish National Institute of Public Health.
15. Hemmingsson E. Behandling av vuxenfetma och dess följsjukdomar. [Treatment of adult obesity and secondary diseases] I: Faskunger J. Motivation för motion [Motivation for exercise] Farsta: SISU Idrottsböcker [Sports books]; 2001.
16. Shick SM, Wing RR, Klem ML, McGuire MT, Hill JO, Seagle H. Persons successful at long-term weight loss and maintenance continue to consume a low calorie, low fat diet. *Journal of the American Dietetic Association* 1998;98:408-13.

17. Klem ML, Wing RR, McGuire MT, Seagle HM, Hill JO. A descriptive study of individuals successful at long-term maintenance of substantial weight loss. *American Journal of Clinical Nutrition* 1997;66:239-46.
18. McGuire MT, Wing RR, Klem ML, Seagle HM, Hill JO. Long-term maintenance of weight loss. Do people who lose weight through various weight loss methods use different behaviors to maintain their weight? *International Journal of Obesity* 1998;22:572-7.
19. Wyatt HR, Grunwald OK, Mosca CL, Klem ML, Wing RR, Hill JO. Long-term weight loss and breakfast in subjects in the National Weight Control Registry. *Obesity Research* 2002;10:78-82.
20. Campbell L, Rössner S. Management of obesity in patients with Type 2 diabetes. *Diabet Med* 2001;18:345-54.

37. Osteoarthritis

Author

Ewa Roos, PT, PhD, Professor, Institute of Sports Science and Biomechanics, University of Southern Denmark, Odense, Denmark

Summary

The purpose of osteoarthritis treatment is to teach the patient about the disease, alleviate pain, optimise and retain physical function, and to prevent or reverse the progress of harmful structural changes in cartilage, bone, ligaments and muscles. A physically active lifestyle, with demands comparable to those placed on good overall health, has been shown to reduce pain and increase function in older patients with osteoarthritis. Both fitness training and dynamic strength training lead to reduced pain and improved function. Initially, however, exercising is more painful, and pain relief is attained after 6–8 weeks. Physiotherapists are able to offer non-pharmacological pain relief and information on pain management.

Osteoarthritis is a chronic disease. In order to achieve lasting results, it is important that the individual chooses a form of exercise that is suitable and that he or she likes and can integrate into daily life. Suitable types of fitness training to start with are cycling and “Nordic walking” (i.e. with poles).

Type of training	Intensity	Frequency (times/week)	Duration (min./session)
Aerobic fitness training	Moderate (13 on the Borg RPE scale)	≥ 3	30 in total (e.g. 3 x 10 min.)
Strength training	1–3 sets of 8–10 exercises with 8–12 repetitions/set, successively increasing load	3	20–60

Definition

Osteoarthritis is not a single, well-defined disease condition but is best described as a final stage failure of the joints that presents similar symptoms and radiographic findings regardless of the original cause. How osteoarthritis is defined depends on the diagnostic criteria applied and varies between different areas of specialty such as orthopaedics, rheumatology, radiology, pathology and epidemiology.

Diagnosis

The main radiological criterion for osteoarthritis is narrowed joint space, which in turn is a result of deterioration of the articular cartilage. A consequence of this is that, when using routine radiographic examination, the diagnosis of osteoarthritis can only be made in a very advanced stage, once the articular cartilage is already eroded. In the early stages, radiographic images often appear essentially normal, even if cartilage changes can be seen with arthroscopy. This can be a contributing factor to the poor link between radiological signs of osteoarthritis and pain. It is not until the radiological signs of osteoarthritis are judged as serious that the link to pain becomes stronger. It is pain that brings the patient to health care, and to begin with the pain appears in connection with movements and loading, and later also at rest and during the night.

Underlying pathophysiological mechanisms

The pathogenesis of osteoarthritis is unclear. Osteoarthritis can be defined as a common final stage in failure of articular cartilage function. If we accept the definition “joint failure”, it follows that osteoarthritis can arise in a normal joint subjected to excessive stress, or in a weakened joint subjected to normal loading. That the same factors that initiate the disease also lead to its advancement is given.

The surfaces of a joint are coated with a layer of articular cartilage several millimetres thick. The cartilage absorbs and distributes weight across the joint and reduces the friction of movements. The articular surfaces are lubricated with a thin layer of viscous synovial fluid with a high concentration of hyaluronic acid. The cartilage matrix can be likened to a fibre-reinforced, water-absorbent gel that seeks to absorb water. When the joint is loaded, the water is pressed out of the tissue and absorbed again when the load decreases. Articular cartilage is a highly specialised tissue in which a constant process of resorption and regeneration of the cartilage matrix occurs. Normally, the cartilage maintains a state of equilibrium, which is a prerequisite for its function. In the case of osteoarthritis, as in excessively high or excessively low joint load, the metabolic equilibrium changes and an imbalance between resorption and repair arises. The cell tries to repair itself but is not able to regenerate a functional matrix and the shock-absorbing function is successively lost.

What does the disease lead to?

Osteoarthritis develops slowly. In cases where the precipitating cause can be determined, it can take 10–30 years before the diagnosis of osteoarthritis can be made with the aid of radiology. Recent studies show that the course of the disease, as measured by radiology measures, is not always progressive but that in approximately 50% of cases slight changes can remain stationary for many years. UK studies show that only a fraction of patients with the diagnosis of osteoarthritis ever become candidates for surgical intervention.

The final stages of osteoarthritis are treated successfully today with arthroplasty (where articular surfaces are replaced with metal or plastic ones), which as a rule yields a stable, pain-free joint and a satisfied patient for many years. Problems that remain with this method are primarily loosening and wear on the new joint surfaces. These problems are most pronounced in younger patients. Arthroplasty surgery is best suited to older patients with lower expectations on their level of physical activity, or with a greater desire to adapt to the new activity level. Patients who receive a new joint are recommended to refrain from activities that involve significant loading, but are encouraged to do things such as cycling and Nordic walking.

Prevalence/Incidence

Osteoarthritis is common. It is a chronic disease and the strain on both the individual and society is considerable. Joint disease is the most common chronic disease in older people and is more common than high blood pressure, heart disease and diabetes. However, osteoarthritis can occur as early as in patients in their 30s. Osteoarthritis that appears early in life is often secondary to a joint injury. These patients make up a sub-group of the osteoarthritic population. They are younger and have a higher need for physical function and are thus less willing to adapt their activities to the problems.

In the US, osteoarthritis costs society an estimated 215 billion USD per year. In Sweden, health economists have estimated the cost of musculoskeletal diseases to be higher than the cost of diseases of the brain and nervous system put together (www.boneandjointdecade.org).

Risk factors

In addition to age, the risk factors for osteoarthritis include heredity, gender and joint overload. Examples of overloading include repetitive bending of the knees in one's work, certain elite sports, high body weight and joint injuries. Another risk factor is muscle weakness. Patients with poor muscle function develop knee osteoarthritis to a larger degree than patients with better muscle function (1–3).

Most common symptoms

The most common symptoms of osteoarthritis are pain and loss of physical ability. Other symptoms include stiffness and joints that click or make other detectable noises.

Current treatment principles

The purpose of osteoarthritis treatment is to:

- Teach the patient about the disease
- Relieve pain
- Optimise and retain physical function
- Prevent or reverse the progression of harmful structural changes to cartilage, bone, ligaments and muscles (4).

When asked about the benefits of different treatments for osteoarthritis, European experts put exercise at the top of the list, ahead of arthroplasty surgery, acetaminophen, NSAIDs (non-steroidal anti-inflammatory drugs) and educating the patient (4). All patients with osteoarthritis should be offered information and education. Since osteoarthritis is a chronic disease, it is of utmost importance that the patient be informed about the disease and understand the mechanisms behind the different treatment options offered. Many communities offer an osteoarthritis school, often led by a physiotherapist. It is essential that patients realise that the basis for osteoarthritis treatment is their own physical activity, which may be complemented, initially or as required, with some form of pain relief. A physically active lifestyle, with demands comparable to those placed on good overall health, has been shown not only to reduce pain and increase function in older patients with osteoarthritis, but also to yield improved overall well-being (5).

Effects of physical activity

The European guidelines, which are based on evidence gathered and the consensus of experts in the field, recommend exercise as treatment for osteoarthritis of the knee (6), hip (7) and hand (8).

Exercise as treatment for osteoarthritis – moderation is best!

Similarly to other biological tissues, cartilage thrives best when subjected to moderate loading. Too little loading, where total decompression is the end point, leads to erosion of the cartilage with reduced ability to absorb shock. Too much loading, as in the case of professional football, leads to an increased risk of osteoarthritis. This applies even when no serious injuries have been noted. Fitness activities (moderate loading) appear, however, rather to protect against the development of osteoarthritis (9, 10).

Positive effects of physical activity

A systematic review of 17 studies, in which a total of approximately 2,500 patients were randomly assigned exercise as treatment and compared to others with other or no treatment, concluded that there is sound evidence that exercise has a positive effect on both pain and physical function in knee osteoarthritis (11). At present, the number of studies

is insufficient to confirm the same conclusions for hip and hand osteoarthritis, but those that have been conducted point in the same direction. Compared to NSAID treatment, a greater reduction in pain is seen after 6–8 weeks of exercise training (4). It is also important to note in this context that the risk of side-effects with NSAID therapy is substantial, while the side-effects seen in exercising osteoarthritic patients are limited to minor muscle injuries (12).

It appears that there is a dose-response relation in exercise training as osteoarthritis treatment, whereby the more strength and fitness improves – the higher the gains. This is based on outcomes from exercise training in older people with osteoarthritis, and even better outcomes can perhaps be achieved in patients who may generally be assumed to want to train harder. It must be noted, however, that exercise training should not be confused with all physical activity. Training entails a well-planned, successive increase in the load on the joint under optimal loading conditions, which is not necessarily characteristic of physical activity as such. For example, a person with knee osteoarthritis benefits from cycling but is made worse by playing football. Data from younger patient groups is, however, lacking.

The scientific basis that exists today with respect to osteoarthritis and exercise training, deals primarily with older people with knee osteoarthritis. On the basis of this data, no one specific form of training can be recommended, as similar pain reduction is seen, for example, in strength training and fitness training. For older people with relatively low training levels, it appears that just doing something is more important than what it is they do. For younger or more physically active people, however, it can be expected that the type of activity plays a larger role since “you become what you train to be.” The main goal is to achieve optimal loading of the knee, by emphasising how one can best load the leg, and through strength and endurance training.

Exercise is a perishable good. In order to be effective, exercise training and physical activity must be ongoing activities. It has been shown that, despite having less pain after training with a physiotherapist, it is difficult for patients to continue on their own. The perception is that, despite the positive effects, exercise wears out the joints (13, 14) and support is needed to get past this. A study done in the UK showed that only 28 per cent of patients on the waiting list for hip arthroplasty exercised or had been referred to a physiotherapist (15).

Indications

Primary prevention

Of the known risk factors, joint loading is the only one that can be modified. Optimal loading of the joints can be achieved through:

- Aerobic fitness activities
- Increased muscle strength
- Weight loss.

Secondary prevention

Osteoarthritis is the most common cause of physical inactivity in seniors. Patients with osteoarthritis have a higher body mass index (BMI) than people without osteoarthritis. Physical inactivity and overweight, both of which osteoarthritic patients suffer from, are known risk factors for increased ill-health and premature death. Thus, treating osteoarthritis early is critical, not only to reduce articular diseases, but also to counteract overall ill-health and the burden on society.

Prescription

Osteoarthritis is a chronic disease. Training must be integrated in daily life and, most importantly, finding a form of exercise that the individual likes is essential. To facilitate the exercise, an initial assessment and training period with a specially adapted programme are required, aimed at optimising the load on the affected joint. This can be achieved through strength training that stresses neuromuscular factors, usually in cooperation with a physiotherapist. 6–8 weeks of exercise training are needed to see definite improvement. In the beginning, it hurts for osteoarthritic patients to exercise. This is permitted, however, as long as the pain diminishes after training and does not increase from day to day. At the physiotherapist, the patient is able to get help with the dosage of training and temporary pain relief, for example, acupuncture, which provides effective pain relief for knee osteoarthritis. As the patient's strength increases, the pain decreases. The reduction of pain has been observed to remain up to 12–18 months after training. Constant maintenance is, however, necessary. Descriptions of various possible activities for patients with osteoarthritis are given below. An activity whose efficacy and tolerance is well-documented is cycling. Clinical experience tells us that patients with osteoarthritis often return to the physiotherapist once a year for "service", that is, for a short period to increase strength under optimal loading. Often this is done in the winter, a time when many find it more difficult to perform regular fitness activities.

A big risk factor for osteoarthritis is injury. Because joint injuries often occur in connection with physical activity, it is wise to consider which physical activities are suitable. Injuries occur more often in football and other contact sports, which should thus be avoided by patients with osteoarthritis.

Training of strength, flexibility, balance and coordination

Training should be individualised with respect to each patient's requirements and, initially, is best carried out with the aid of a physiotherapist. In general, dynamic strength training is recommended, to begin with using the body as the load, and successively increasing load thereafter. The exercises should be carried out in a loaded position. In knee osteoarthritis, training of the front thigh muscles should be emphasised. In hip osteoarthritis, maintaining good flexibility is of particular importance.

Intensity, frequency and duration of aerobic fitness training

The intensity of aerobic fitness training should be such that holding a conversation is possible, corresponding to a stress level of 13/20 on the Borg RPE scale. The activity should be carried out for 30 minutes per day *in total*, meaning that the person could walk for 10 minutes, 3 times a day. The activity should be carried out most days of the week.

Aerobic fitness-promoting activities

Walking

Benefits: Safe for the majority, something everyone can already do, easy to do, inexpensive. Improves aerobic fitness, reduces osteoarthritic pain and depression.

Limitations: Not suitable for severe osteoarthritis of the hips, knees and ankles.

Recommendations: Wear light shoes with good support and shock absorption. Walk on even, flat ground. If possible, avoid pavement. Choose a softer surface. Walk slowly rather than quickly.

Nordic walking

Benefits: The same as for walking without poles. Less loading of the hip, knee and ankle joints. More rapid improvement in fitness compared to walking without poles. Effective also for neck and back problems.

Limitations: Good clinical experience, but scientific data on patients with osteoarthritis is lacking.

Recommendations: Wear light shoes with good support and shock absorption. Walk on even, flat ground. If possible, avoid pavement. Choose a softer surface. Use the poles rhythmically. Walk as if skiing (right foot-left arm, left foot-right arm). Choose a pole length that gives a good grip and comfortable pendulum movement that does not hurt the shoulders. One recommendation states that the poles should reach one decimetre above the elbow when standing with arms at side of body. Another recommendation states that proper pole length can be calculated by multiplying body height by 0.7. Choose poles with adjustable length.

Running

Benefits: Data for patients with osteoarthritis is lacking.

Limitations: Overload injuries are common in the general public, changes in mechanical conditions as in osteoarthritis would suggest an increased risk of injury, and high loading of the hip, knee and ankle joints.

Recommendations: Do training exercises aimed at increasing strength and mobility of the legs before attempting to run. Run on even, flat surface. If possible, avoid pavement. Choose a softer surface. Use shoes with good support and shock absorption. Do not increase the distance or intensity by more than 5 per cent per week.

Treadmill

Benefits: Easy to use, soft even surface. Avoid downhill grades, the uphill grade can often be adjusted.

Limitations: Requires good balance as the surface is moving. On some models the slowest speed is too fast.

Recommendations: Choose a treadmill with a soft surface, sufficient length and breadth, and with hand rails along the sides.

Running in water

Benefits: The same movement pattern as when running on land but without the load on the hips, knees and feet.

Limitations: Requires access to a pool that is deep enough. Heart rate and oxygen intake are 15–20 per cent lower than on a treadmill. Data for patients with osteoarthritis is lacking.

Recommendations: Use the proper technique, that is, more upright than lying position. Use a life jacket designed for the purpose. When prescribing, specify the intensity (steps per minute).

Swimming/aquafit

Benefits: It is easy to perform the movements in the water. Very little stress on the joints.

Limitations: Requires access to a heated pool or suitable aquafit programme. Data on swimming is lacking.

Recommendations: Choose a fitness programme especially for patients with osteoarthritis. Work in water that is deep enough.

Dancing

Benefits: Studies have shown that dancing as treatment increases fitness and facilitates an increased level of activity as well as leading to reduced ill-health, pain and depression.

Limitations: Data is lacking with regard to loading of the joints. Relatively high risk of injury.

Recommendations: Use shoes with good support and shock absorption. Dance on wood floor or other surface with some give in it. Have a chair handy for resting or relieving the load.

Cycling outdoors or on a stationary bike

Benefits: Effective fitness training, activates the larger muscle groups of the legs.

Low joint load (1.2 times the body weight in the knees). Data shows that fitness, exercise tolerance and muscle strength increase at the same time as illness is reduced.

Limitations: Requires 90-degree mobility of the knee. Correct positioning of seat and handlebars is of utmost importance. Cycling outdoors requires good balance, which is not the case when using a stationary bike.

Recommendations: Correct positioning of seat and handlebars is of utmost importance. Seat height should be such that the knee's angle is 10–15° when most extended. Seek the assistance of a bike dealer accustomed to helping racing cyclists. Choose a bike with a comfortable seat and whose seat and handlebars can be easily adjusted.

Stair/step machine

Benefits: Functional activity, similar to walking up stairs. Data shows that younger patients with other knee injuries tolerate step machines well.

Limitations: Can entail considerable load on the joints. Temporary numbness of the fore-foot is common. Data for patients with osteoarthritis is lacking.

Recommendations: Use a model with large pedals/stairs and hand rails. Change foot position often.

Functional mechanisms

Exercise training can be assumed to be an effective treatment for osteoarthritis via a number of mechanisms. The muscle activity relieves pain via the same mechanisms as with acupuncture. Aerobic fitness training increases endorphin levels in the brain, which reduces the sensation of pain. Increased muscle strength and improved neuromuscular function yield increased stability around the joints, factors that help to reduce the load on the joints (16, 17). One study shows that 4 months of training with a physiotherapist not only improves muscle strength, but also the quality of the cartilage of the knee joint (18), confirming earlier animal studies. In a group of older people with knee osteoarthritis, in comparison to mobility training, strength training was shown to lead to slower progression of osteoarthritis (19). Exercise training is often associated with weight loss, which helps to reduce the total load on the joint.

Evaluation

How do we know that exercising training is effective?

In treatment studies of osteoarthritis, efficacy is normally measured with the aid of well-documented *questionnaire surveys* that evaluate the patient's perceived pain, stiffness and other symptoms as well as impairment of function. In some cases, the effect of pain and disability on the patient's quality of life is also evaluated. For younger patients, or patients with early onset knee osteoarthritis, greater improvement is generally seen in the quality of life and physical function in addition to daily demands (such as squatting, kneeling, jumping and running), than in conventional measures such as pain and functions of daily life. Questionnaires designed for this purpose can be downloaded from the internet (www.koos.nu).

Testing of muscle strength and aerobic fitness can be used in part to motivate the patient to exercise, and in part to objectively document the outcomes of training. Simple functional tests of muscle strength in the quadriceps have proven to be able to be carried out by GPs. One example of a practical test that can be carried out is the so-called "step test", where the patient is asked to step up, one leg at a time, as high as he or she can. The step box has a removable shelf that can be set at seven different levels (20). Chairs and stools of

different heights can be used in place of the step box. Ensure, however, that the chair/stool is secured to avoid the risk of falling! Because the stepping height is dependent not only on the patient's height and weight, but also on the mobility of the hip, knee and foot joints, both comparison between patients and between a patient's two sides can be difficult. It is recommended that each patient serve as his or her own control. Good step ability can be measured by whether the patient is able to step up to the height where the initial angle of the knee joint is 90 degrees. This can be done up to 80–85 years of age.

For younger patients, more complex tests can be used. Hopping on one leg is easy to perform and is often used to assess the functional ability of patients with knee problems. The distance of the hop is dependent on several factors, including the patient's strength, joint stability, balance and confidence in the knee. Hopping on one leg has been found to have satisfactory test-retest reliability. Comparison with published normal measures and sample groups requires that the test be conducted in a standardised manner, as factors such as arm position and requirements for safe landing entail high variability in the distance hopped by a particular individual (20). A common requirement for adequate rehabilitation as measured by functional tests is a difference between the two sides of at most 10–15 per cent.

Interactions with drug therapy

As an initial increase in pain may occur during the training period, pain relief in the form of acetaminophen or an NSAID is often used to reduce pain. These drugs have no known interactions with physical activity.

Contraindications

Patients with generalised osteoarthritis or fibromyalgia usually have a very strong response to exercise and should be prescribed very low doses over a prolonged period. The positive effects of exercise are not as great as for osteoarthritis in individual joints either. The prescriptions for patients with osteoarthritis in this chapter are not suitable for these groups!

Risks

A big risk factor for osteoarthritis is injury. Because joint injuries often occur in connection with physical activity, it is wise to consider which physical activities are suitable for patients with osteoarthritis. Injuries occur more often in football/soccer and other contact sports, which should thus be avoided by these patients.

Sports that involve high loading in the form of both axial compression force and twisting can increase the risk for osteoarthritis. Basketball, handball, professional running, football, American football, rugby and waterskiing are examples of sports with high axial compression force and a risk for twisting. Patients with osteoarthritis should avoid these sports.

References

1. Hootman JM, FitzGerald S, Macera CA, Blair SN. Lower extremity muscle strength and risk of self-reported hip or knee osteoarthritis. *J Phys Act Health* 2004;1:321-30.
2. Slemenda C, Heilman DK, Brandt KD, Katz BP, Mazucca SA, Braunstein EM, Byrd D, et al. Reduced quadriceps strength relative to body weight. A risk factor for knee osteoarthritis in women? *Arthritis Rheum* 1998;41:1951-9.
3. Thorstenson CA, Petersson IF, Jacobsson LTH, Boegård TL, Roos EM. Reduced functional performance in the lower extremity predicted radiographic knee osteoarthritis five years later. *Ann Rheum Dis* 2004;63:402-47.
4. Pendleton A, Arden N, Dougados M, Doherty M, Bannwarth B, Bijlsma JW, et al. EULAR recommendations for the management of knee osteoarthritis. Report of a task force of the Standing Committee for International Clinical Studies Including Therapeutic Trials (ESCISIT). *Ann Rheum Dis* 2000;59:936-44.
5. Devos-Comby L, Cronan T, Roesch SC. Do exercise and self-management interventions benefit patients with osteoarthritis of the knee? A metaanalytic review. *J Rheumatol* 2006;33:744-56.
6. Jordan KM, Arden NK, Doherty M, Bannwarth B, Bijlsma JW, Dieppe P, et al. EULAR Recommendations 2003. An evidence based approach to the management of knee osteoarthritis. Report of a Task Force of the Standing Committee for International Clinical Studies Including Therapeutic Trials (ESCISIT). *Ann Rheum Dis* 2003;62:1145-55.
7. Zhang W, Doherty M, Arden N, Bannwarth B, Bijlsma J, Gunther KP, et al. EULAR evidence based recommendations for the management of hip osteoarthritis. Report of a task force of the EULAR Standing Committee for International Clinical Studies Including Therapeutics (ESCISIT). *Ann Rheum Dis* 2005;64:669-81.
8. Zhang W, Doherty M, Leeb BF, Alekseeva L, Arden NK, Bijlsma JW, et al. EULAR evidence based recommendations for the management of hand osteoarthritis. Report of a Task Force of the EULAR Standing Committee for International Clinical Studies Including Therapeutics (ESCISIT). *Ann Rheum Dis* 2007;66:377-88.
9. Manninen P, Riihimäki H, Heliovaara M, Suomalainen O. Physical exercise and risk of severe knee osteoarthritis requiring arthroplasty. *Rheumatology (Oxford)* 2001;40:432-7.
10. Sutton AJ, Muir KR, Mockett S, Fentem P. A case-control study to investigate the relation between low and moderate levels of physical activity and osteoarthritis of the knee using data collected as part of the Allied Dunbar National Fitness Survey. *Ann Rheum Dis* 2001;60:756-64.
11. Fransen M, McConnell S, Bell M. Exercise for osteoarthritis of the hip or knee. *Cochrane Database Syst Rev* 2003;CD004286.
12. Ettinger WH Jr, Burns R, Messier SP, Applegate W, Rejeski WJ, Morgan T, et al. A randomized trial comparing aerobic exercise and resistance exercise with a health education program in older adults with knee osteoarthritis. The Fitness Arthritis and Seniors Trial (FAST). *JAMA* 1997;277:25-31.

13. Hendry M, Williams NH, Markland D, Wilkinson C, Maddison P. Why should we exercise when our knees hurt? A qualitative study of primary care patients with osteoarthritis of the knee. *Fam Pract* 2006;23:558-67.
14. Thorstenson CA, Roos EM, Petersson IF, Arvidsson B. How do patients conceive exercise as treatment of knee osteoarthritis? *Disabil Rehabil* 2006;28:51-9.
15. S Shrier I, Feldman DE, Gaudet MC, Rossignol M, Zukor D, Tanzer M, et al. Conservative non-pharmacological treatment options are not frequently used in the management of hip osteoarthritis. *J Sci Med Sport* 2006;9:81-6.
16. Mikesky AE, Meyer A, Thompson KL. Relationship between quadriceps strength and rate of loading during gait in women. *J Orthop Res* 2000;18:171-5.
17. Thorstenson CA, Henriksson M, von Porat A, Sjö Dahl C, Roos EM. The effect of eight weeks of exercise on knee adduction moment in early knee osteoarthritis. A pilot study. *Osteoarthritis Cartilage* 2007;15(10):1 163-70.
18. Roos EM, Dahlberg L. Positive effects of moderate exercise on knee cartilage glycosaminoglycan content. A four-month randomized controlled trial in patients at risk of osteoarthritis. *Arthritis Rheum* 2005;52:3507-14.
19. Mikesky AE, Mazzuca SA, Brandt KD, Perkins SM, Damush T, Lane KA. Effects of strength training on the incidence and progression of knee osteoarthritis. *Arthritis Rheum* 2006;55:690-9.
20. Roos EM. Hur utvärdera behandlingsresultat vid knäsjukdom? [How do we evaluate treatment outcomes in knee diseases?] In: Karlsson J, Ed. *Knäledens sjukdomar och skador [Diseases and Injuries of the Knee Joint]*. Södertälje: Astra Läkemedel; 2000. p. 120-30.

38. Osteoporosis

Authors

Eva Ljunggren Ribom, PT, PhD, Institute of Surgical Sciences, Orthopedic Unit, Uppsala University, Uppsala, Sweden

Karin Piehl-Aulin, MD, PhD, Professor, Department of Health Science, Örebro University, Örebro, Danderyd University Hospital, Stockholm, Sweden

Summary

The incidence of osteoporosis related fractures has increased in the past decades (1). Accordingly, it is important to try to prevent osteoporosis and current preventative endeavours are focusing on a number of avoidable risk factors. One example is physical activity, a vital ingredient for reinforcing and maintaining bone tissue. The effects of physical activity on the bone tissue are most noticeable when the activity is of a weight-bearing nature, intensive and frequent, around 2–3 times a week. In addition to the effects on the skeleton, weight-bearing exercises will have a positive effect on fitness, muscle strength and coordination and this in turn leads to a reduced risk of fractures and an improved quality of life. Suitable activities include dancing, gymnastics, jogging, strength training, ball and racket sports, brisk walks and walking up and down stairs.

The purpose of the physical activity carried out by individuals with osteoporosis is not just to affect the bone tissue, but also to prevent falls and subsequent fractures. Balance, strength and coordination exercises are therefore a good complement to walking, for example. In the event of a vertebral compression with continued pain, the physical activity should also emphasize pain relief.

Disease definition

Osteoporosis is defined as a systematic skeletal disease characterised by low bone mass (BMD, bone mineral density) and microarchitectural deterioration of bone leading to greater bone fragility and a consequent increase in fracture risk (2).

Prevalence/Incidence

Osteoporosis is common in Sweden. Approximately one out of three women aged 70–79 is diagnosed with osteoporosis in the hip (3).

Cause

Depending on its cause, osteoporosis can be divided into two types:

- *Primary osteoporosis* is either postmenopausal or related to age or lifestyle factors such as a lack of physical activity, smoking, alcohol consumption and inadequate nutrition.
- *Secondary osteoporosis* is caused by certain diseases or as a result of medical treatments.

Risk factors

The risk factors of osteoporosis can be divided into unavoidable (family history, old age, female gender, early onset of menopause, personal history of fractures and height) and avoidable (smoking, physical inactivity, inadequate nutrition, low weight, poor health, secondary osteoporosis and medical treatment).

Pathophysiology

Bone tissue composition and renewal (remodelling)

The remodelling or renewal of bone tissue constitutes 25 per cent of trabecular and 2–3 per cent of cortical bone renewal each year. Bone is a very dynamic tissue that remodels constantly throughout life depending on external demands such as physical activity. This remodelling means that the osteoclasts, i.e. bone resorption cells, resorb bone from the bone surface while the osteoblasts, i.e. bone-forming cells, replace the loss of bone with new bone tissue (Figure 1).

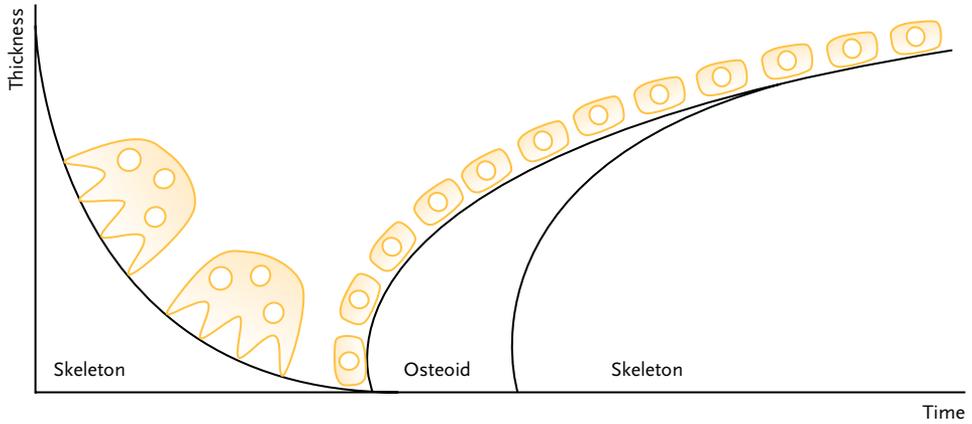


Figure 1. Schematic illustration of bone renewal (remodelling) with osteoclasts breaking down (resorbing) the bone (left in the picture) and osteoblasts producing new bone tissue (right in the picture). Modified according to Ericson (4).

During remodelling, some of the osteoblasts remain encased in bone matrix and are then called osteocytes. The osteocytes communicate with each other and the cells on the bone surface via long cell outgrowths that form a network of small channels. The osteocytes most likely play an important role as they detect and respond to the mechanical load put on the skeleton, thereby initiating the remodelling of relevant bone surfaces. Moreover, the mechanical load may be an important link between bone resorption and bone formation and lead to bone formation without preceding bone resorption (modelling). Hormones (systemic influence) and local growth factors are also likely to be involved in different levels in the remodelling process (Figure 2).

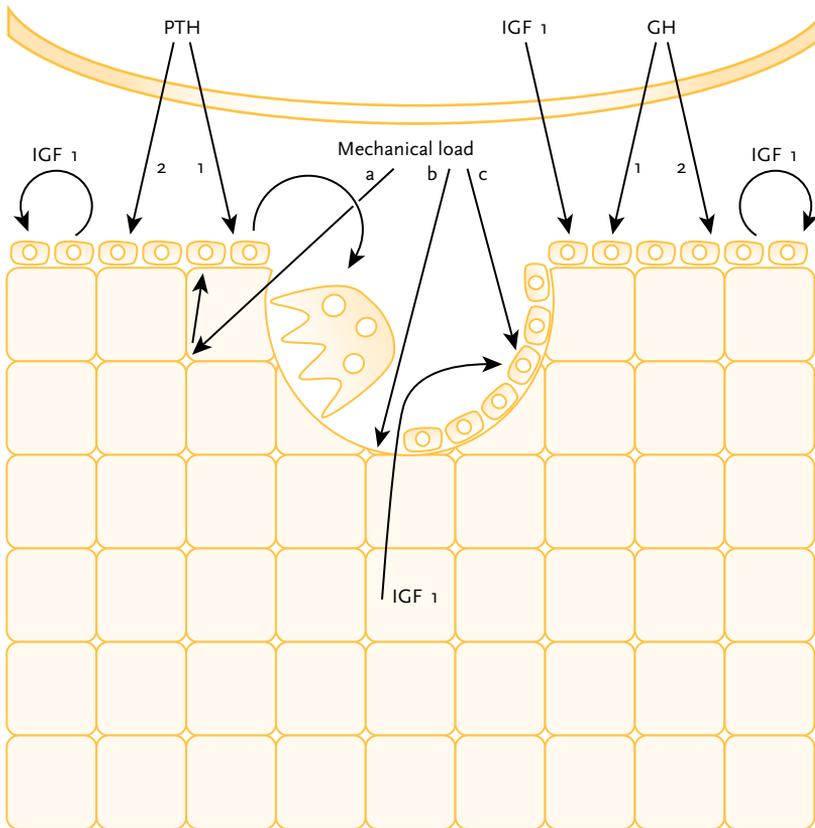


Figure 2. Schematic illustration of local and systemic influence on the bone cells. The mechanical load can intervene and affect various stages in the remodelling cycle (a–c) while endocrine, autocrine and paracrine regulators of the parathyroid hormone (PTH), growth hormone (GH) and insulin-like growth factor 1 (IGF-1) have the potential of modulating or supplementing the mechanical load response. Arrows 1 and 2 show direct and indirect effects of PTH and GH on bone cells. Modified according to Brahm (5).

It is vital for the resorbed bone to be replaced with the same amount of new bone or the bone renewal will be incomplete, resulting in a loss of bone. The loss of bone becomes particularly pronounced when the remodelling rate is high, such as during menopause.

Most common symptoms – what are the consequences of the disease?

Osteoporosis is a symptom-free disease, which only causes suffering in the event of a fracture. Approximately 50 per cent of women and 25 per cent of men aged 50 will at some point during the rest of their lives suffer a fracture because of osteoporosis (6).

Diagnosis

The diagnosis of osteoporosis is based on bone mineral density measurements (dual energy x-ray absorptiometry, DXA) and osteoporosis is defined by the World Health Organization (WHO) as a bone mineral density that is 2.5 standard deviations below the average bone mass of young adults of the same population (7).

Treatment principles

1. Lifestyle changes; physical activity, dietary habits and smoking.
2. Calcium and Vitamin D supplements.
3. Antiresorptive treatment; bisphosphonates, SERM.
4. Anabolic treatment; intermittent PTH.
5. Orthopaedic intervention; kyphoplasty/vertebroplasty (for vertebral compressions)

The effects of physical activity on bone resorption and renewal

A combination of factors are known to affect the dynamic bone tissue (see Figure 3).

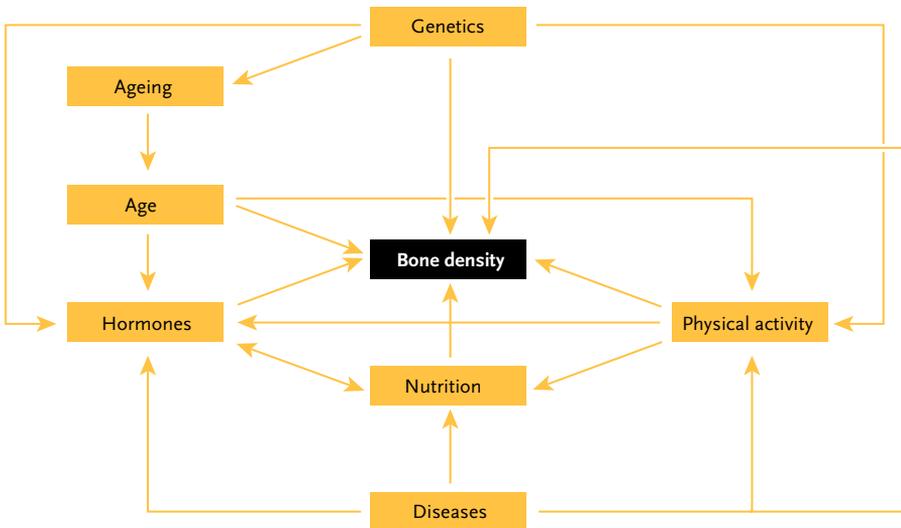


Figure 3. A summary of factors affecting the production and maintenance of bone tissue and the relationship between these factors. Modified according to Ziegler and colleagues (8).

An optimisation of these factors will aid the production, protection and maintenance of a healthy skeleton (9). It is not possible to influence heredity and age, but an improved nutritional intake (incorporating calcium and vitamin D), adequate levels of sex hormones (e.g. oestrogen substitutes during menopause) and increased physical activity can provide considerable benefits in the form of increased bone formation, for example.

In addition, physical activity affects the muscle mass and muscle strength whereupon the latter is closely connected to bone density. Hence, it is not unreasonable to see a strong link between these two factors, i.e. reduced muscle strength followed by reduced bone mass as a result of physical inactivity for example, even if the response from the bone tissue is somewhat delayed. Increased muscle strength would similarly precede an increase in bone mass, even though the load to which the bone is subjected is mainly muscle load and not weight load. Age-related bone loss is essentially linked to age-dependent changes in the muscle strength. However, the claims of numerous researchers that bone loss begins earlier in life than the loss of muscle strength contradict such a strong correlation. A number of studies also indicate that muscle strength is not just linked to the bone density of the “underlying” bone, but also to the bone density of the remainder of the skeleton without any relation to the muscle studied. Consequently, it is important to note that the effect of mechanical stimulus (physical activity) on bone tissue may also be dependent on the hormonal and metabolic environment, i.e. non-mechanical environment, with a potentially changing effect on the sensitivity of the bone cells.

Acute effects

Effects on bone density have been observed after 7–12 months of training (10–13).

Long-term effects

Like muscle strength, bone density is perishable and a change from a physically active life to a physically inactive life will result in a reduced bone density. However, there is evidence that athletes maintain a somewhat higher bone density than others when checked several years after their sports careers have finished (14).

Indications

Physical activity in order to affect the maximum bone mass – primary prevention

A maximum bone mass, known as peak bone mass, is achieved at 20–30 years of age following skeletal maturation throughout the adolescent years. The most important factors for an optimised maximum bone mass during this time in life are diet, hormonal status and weight-loading activity. Consequently, multifaceted physical education at school is likely to be of benefit to the skeleton, although it may be even more important that physical

education fosters continued physical activity in the generation growing up, whereby bone mass is maintained. The choice of lifestyle is therefore of utmost importance.

Prospective training studies of children between the ages of 6–10 with interventions such as jumping, aerobics, weight lifting and school gymnastics indicate an increase in bone density. The length of the training period varied from 7–24 months (10–13, 15). Prospective studies also show that self-chosen physical activities in the adolescent years have a positive effect on bone density (16–19). In addition, a large number of cross-sectional studies have compared the bone density of athletes with that of untrained individuals. These studies highlight the importance of weight-loading physical activity. Sports that involve rapid movements in different directions and/or a weight-bearing load have the greatest effect on bone density while an unloaded activity such as swimming has little or no effect. One study of the effect of loading activity compared the bone density in the dominant arm of female tennis players with the bone density in the non-dominant arm. The result of the study indicates that those who had started playing tennis before reaching puberty had a bone density side-difference of around 20 per cent while those who had started playing tennis after puberty had a difference of just under 10 per cent when comparing the dominant arm with the non-dominant arm (20). This may be seen as further evidence that regular physical activity in the primary school years is of greater benefit to the skeleton than the physical activity carried out in later years.

Physical activity in women between the ages of 20–50 (premenopausal)

A gradual loss of bone density takes place from around the age of 20, when the maximum bone mass is fully developed, until menopause. However, the results of activity studies carried out on women aged 30–50 are somewhat uncertain as to the effect of physical activity on aged-related bone loss. Still, most of the results indicate that weight-bearing activities (such as aerobics) are of the greatest significance – if not to increasing bone mass then at least to maintaining existing bone mass (21–24). A review article on the subject concluded that the quality of the relevant studies was relatively low. There is a large drop-out rate in the training studies, making it difficult to generalize the small positive effects that physical activity has on BMD (25).

Physical activity to reduce bone loss after menopause

Trabecular bone loss accelerates during and after menopause, but cortical bone is also affected. Cross-sectional and longitudinal studies show that the loss of bone taking place during this time in life cannot be prevented by physical activity, only slowed down.

The significance of walking, jogging, aerobics and strength training to the prevention of bone loss is not yet clear, although a number of randomised controlled studies indicate that such activities have a beneficial effect on bone tissue, either in the form of a slight increase or reduced resorption (26–28). Cross-sectional studies also indicate a lower incidence of fractures in postmenopausal women reported to be physically active (29, 30).

Physical activity in osteoporosis – secondary prevention

The effect of physical activity on the bone density of patients with osteoporosis is poorly documented. One study on women with osteopenia showed that 60 minutes of weight-bearing exercise three times a week for 12 months stabilised the BMD in the lumbar region, but did not have an effect on the hips (31). Home exercise programmes can result in an increased quality of life in patients with or without vertebral compression (32, 33). In addition, 10 weeks of physiotherapy incorporating balance exercises, strength training and lumbar stabilisation exercises lead to an improved daily functional capacity and quality of life with better balance and less pain (34). This indicates that the aim of physical activity need not be to increase bone density, but can be to improve balance in order to reduce the tendency to fall, resulting in a lower risk of fractures, less pain following a vertebral fractures and a better quality of life.

Prescription

General exercise advice based on what is known today

To obtain maximal effect on the skeleton, weight-bearing activities must be performed. The dynamic load put on the skeleton should consist of movements that compress, elongate, rotate and flex the skeleton. At a younger age, this would include activities such as jumping and running with quick starts and stops and changes in direction. This could be anything from normal play to an organised sports activity. Later in life, activities such as brisk walks, jogging, fitness training, aerobics, strength training or, if a more competitive form of exercises is preferred, ball and racket sports are recommended.

Even if walking in itself is not the activity that has the greatest positive effect on bone tissue, together with other daily activities (such as taking the stairs instead of the lift) it may nonetheless be of significance. Svenska Osteoporossällskapet [The Swedish Osteoporosis Society] recommends a brisk 30-minute walk five times a week as a general exercise to prevent osteoporosis, which should be feasible and practical for the population at large. However, this activity must be done on a regular basis as bone is a replenishable tissue.

Because osteoporosis is a risk factor for fractures, the objective of the physical activity is also to prevent falls and consequent fractures. Leg training exercises combined with balance and coordination training are recommended, such as in the form of dancing or Thai Chi.

The pain associated with a vertebral compression is often alleviated with lumbar stabilisation and mobility training which reduces muscle tension.

Functional mechanisms

The effect of physical activity on the skeleton

When bone tissue is subjected to a dynamic load, a temporary deformation and subsequent fluid flow induction takes place in the canalicular network surrounding the osteocytes. This is likely to have an effect on intracellular calcium levels and also on local osteoblasts and osteocytes. This effect increases with the size and speed of the load and whether the load has an unusual or variable direction.

Bone tissue responds immediately with a cellular reaction characterised by an acute local release of prostaglandins, which has been shown *in vivo* in humans (35). This leads to a local production of growth factors and subsequent bone renewal in response to the original load. However, a general ‘hormonal’ tissue reaction might also be expected as a consequence of the physical activity. Many of the hormones and growth factors known to affect the skeleton are influenced by physical activity such as growth hormone (GH), parathormone (PTH) and insulin-like growth factor 1 (IGF-1) among others (36). Perhaps the most crucial evidence of the skeleton health benefits gained through loading is the rapid bone resorption that takes place in the absence of weight-bearing load as seen during space missions and immobilisation (37–40).

The Mechanostat, *i.e.* the theoretic model describing the effect of mechanical load on bone tissue can be compared with a thermostat that limits load-related bone tissue deformations. A skeleton deformation is an indication that there is a risk of fracture whereupon the production of bone tissue starts and continues until the risk has been removed. Accordingly, the load put on the skeleton determines how much bone tissue is renewed and resorbed, and the individual in question only gets as much new bone tissue as he or she ‘deserves’.

Reduced muscle strength, balance and coordination constitute risk factors for falls. By training these functions, falls and, consequently, the risk of fractures can be prevented.

Functional tests/need for health check-ups

What tests should be carried out prior to training?

- Assess the DXA measurement to gain an understanding of how fragile the patient is before recommending a load.
- Test balance and muscle strength to assess the risk of falling and evaluate the effects of physical activity.
- Ask if the patient has a tendency to fall.
- Carry out a pain assessment.
- Test lumbar mobility and make an assessment of kyphosis.

How and when should the effects gained be assessed?

Increased bone density should be possible to measure with a DXA bone densitometry scan after 7–12 months.

Assess muscle strength, balance, pain, posture and the number of falls after a period of 2–6 months.

Interactions with drug therapy

Antiresorptive treatment with bisphosphonates have shown to be less effective on patients that are immobilised due to a spinal cord injury (41). Physical activity in combination with medication is recommended to achieve maximum effect.

Contraindications

Acute illness with pain and general deterioration.

Risks

General side-effects such as:

- Load injuries caused to the locomotor system if the intensity and frequency of physical activity is not gradually increased and incorrect footwear used.
- Risk of falling and increased risk of fracture.
- Weight loss due to insufficient nutritional intake.
- Anorexia nervosa with amenorrhea.

It is important that the exercise programme put together for an untrained individual or osteoporosis sufferer with a fragile skeleton incorporates activities that are enjoyable and can be performed with caution so as to avoid injury owing to an unusual load.

References

1. Obrant KJ, Bengnér U, Johnell O, Nilsson BE, Sernbo I. Increasing age-adjusted risk for fragility fractures: A sign of increasing osteoporosis in successive generations? *Calcif Tissue Int* 1989;44:157-67.
2. Consensus Development Conference: Diagnosis, Prophylaxis and Treatment of Osteoporosis. *Am J Med* 1993;94:646-50.
3. SBU – Statens beredning för medicinsk utvärdering [Swedish Council on Technology Assessment in Health Care] Osteoporos – prevention, diagnostik och behandling – En systematisk litteraturöversikt. [Osteoporosis – prevention, diagnosis and treatment – A systematic literature survey] Vol 1. 2003. ISBN 91-87890-86-0. Elanders Graphic Systems, Gothenburg.
4. Ericsen EF. Normal and pathological remodeling of human trabecular bone: Three dimensional reconstruction of the remodeling sequence in normals and in metabolic bone disease. *Endocr Rev*. 1986;7:379-408.
5. Brahm H, Ström H, Piehl-Aulin K, Mallmin H, Ljunghall S, Bone metabolism in endurance trained athletes: A comparison to population-based controls based on DXA, SXA, quantitative ultrasound, and biochemical markers. *Calcif Tissue Int* 1997;61:448-54.
6. Kanis JA, Jonell O, Oden A, Sembo I, Redlund-Johnell I, Dawson A, et al. Longterm risk of osteoporotic fracture in Malmo. *Osteoporos Int* 2000;11:669-74.
7. WHO [World Health Organization] Assessment of osteoporotic fracture risk and its role in screening for postmenopausal osteoporosis. WHO technical report series, Geneva; 1994.
8. Ziegler R, Scheidt-Nave C, Scharla S. Pathophysiology of osteoporosis: Unresolved problems and new insights. *J Nutr*. 1995;125(7 Suppl):2033S-2037S.
9. Frost HM. Perspectives: Bone's mechanical usage windows. *Bone Miner* 1992;19:257-71.
10. Fuchs RK, Bauer JJ, Snow CM. Jumping improves hip and lumbar spine bone mass in prepubescent children: A randomized controlled trial. *J Bone Miner Res* 2001;16:148-56.
11. McKay HA, Petit MA, Schutz RW, Prior JX, Barr SI, Khan KM. Augmented trochanteric bone mineral density after modified physical education classes: A randomized school-based exercise intervention study in prepubescent and early pubescent children. *J Pediatr* 2000;136:156-62.
12. Bradney M, Pearce G, Naughton G, Sullivan C, Bass S, Beck T, et al. Moderate exercise during growth in prepubertal boys: Changes in bone mass, size, volumetric density, and bone strength: A controlled prospective study. *J Bone Miner Res* 1998;13:1814-21.
13. Morris FL, Naughton GA, Gibbs JL, Carlson JS, Wark JD. Prospective ten-month exercise intervention in premenarcheal girls: Positive effects on bone and lean mass. *J Bone Miner Res* 1997;12:1453-62.
14. Karlsson MK, Johnell O, Obrant KJ. Is bone mineral density advantage maintained long-term in previous weight lifters? *Calcif Tissue Int*. 1995;57:325-8.

15. Linden C, Ahlborg HG, Besjakov J, Gardsell P, Karlsson MK. A school curriculum-based exercise program increases bone mineral accrual and bone size in prepubertal girls: Two-year data from the pediatric osteoporosis prevention (POP) study. *J Bone Miner Res* 2006;21:829-35.
16. Kemper HC, Twisk JW, van Mechelen W, Post GB, Roos JC, Lips P. A fifteen-year longitudinal study in young adults on the relation of physical activity and fitness with the development of the bone mass: The Amsterdam Growth and Health Longitudinal Study. *Bone* 2000;27:847-53.
17. Lehtonen-Veromaa M, Mottonen T, Irjala K, Nuotio I, Leino A, Viikari J. A 1-year prospective study on the relationship between physical activity, markers of bone metabolism and bone acquisition in peripubertal girls. *J Clin Endocrin Metab* 2000;85:3726-32.
18. Baily DA, McKay HA, Mirwald RL, Crocker PR, Faulkner RA. A six-year longitudinal study of the relationship of physical activity on bone mineral accrual in growing children: The University of Saskatchewan bone mineral accrual study. *J Bone Miner Res* 1999;14:1672-9.
19. Bass S, Pearce G, Bradney M, Hendrich E, Harding A, Seeman E. Exercise before puberty may confer residual benefits in bone density in adulthood: Studies in active prepubertal and retired female gymnasts. *J Bone Miner Res* 1998;13:500-7.
20. Kannus P, Haaposalu H, Sankelo M, Sievänen H, Pasanen M, Heinonen A, et al. Effect of starting age of physical activity on bone mass in the dominant arm of tennis and squash players. *Ann Intern Med* 1995;123:27-31.
21. Vainionpää A, Korpelainen R, Leppaluoto J, Jamsa T. Effects of high-impact exercise on bone mineral density: A randomized controlled trial in premenopausal women. *Osteoporos Int.* 2005;16:191-7.
22. Heinonen A, Kannus P, Sievänen H, Pasanen M, Oja P, Vuori I. Good maintenance of high-impact activity-induced bone gain by voluntary, unsupervised exercises: An 8-month follow-up of a randomized controlled trial. *J Bone Miner Res* 1999;14:125-8.
23. Heinonen A, Kannus P, Sievänen H, Oja P, Pasanen M, Rinne M, Uusi-Rasi K, Vuori I. Randomised controlled trial of effect of high-impact exercise on selected risk factors for osteoporotic fractures. *Lancet* 1996;348:1343-7.
24. Lohman T, Going S, Pamentier R, Hall M, Boyden T, Houtkooper L, Ritenbaugh C, Bare L, Hill A, Aickin M. Effects of resistance training on regional and total bone mineral density in premenopausal women: A randomized prospective study. *J Bone Miner Res.* 1995;10:1015-24.
25. Martyn-St James M, Carroll S. Progressive high-intensity resistance training and bone mineral density changes among premenopausal women: Evidence of discordant site-specific skeletal effects. *Sports Med.* 2006;36:683-704.
26. Engelke K, Kemmler W, Lauber D, Beeskow C, Pintag R, Kalender WA. Exercise maintains bone density at spine and hip EFOPS: A 3-year longitudinal study in early postmenopausal women. *Osteoporos Int.* 2006;17:133-42.

27. Adami S, Gatti D, Braga V, Bianchini D, Rossini M. Site-specific effects of strength training on bone structure and geometry of ultradistal radius in postmenopausal women. *J Bone Miner Res.* 1999;14:120-4.
28. Brooke-Wavell K, Jones PR, Hardman AE. Brisk walking reduces calcaneal bone loss in post-menopausal women. *Clin Sci.* 1997;92:75-80.
29. Farahmand BY, Persson PG, Michaelsson K, Baron JA, Alberts A, Moradi T, et al. Physical activity and hip fracture: A population-based case-control study. Swedish hip fracture study group. *Int J Epidemiol.* 2000;29:308-14.
30. Gregg EW, Cauley JA, Seeley DG, Ensrud KE, Bauer DC. Physical activity and osteoporotic fracture risk in older women. Study of Osteoporotic fractures research group. *Ann of Intern Med.* 1998;129:81-8.
31. Bravo G, Gauthier P, Roy PM, Payette H, Gaulin P, Harvey M et al. Impact of a 12-month exercise program on the physical and psychological health of osteopenic women. *J Am Geriatr Soc.* 1996;44:756-62.
32. Chien MY, Yang RS, Tsauo JY. Home-based trunk-strengthening exercise for osteoporotic and osteopenic postmenopausal women without fracture – a pilot study. *Clin Rehabil.* 2005;19:579.
33. Papaioannou A, Adachi JD, Winegard K, Ferko N, Parkinson W, Cook RJ, Webber C, McCartney N. Efficacy of home-based exercise for improving quality of life among elderly women with symptomatic osteoporosis-related vertebral fractures. *Osteoporosis Int.* 2003;14:677-82.
34. Malmros B, Mortensen L, Jensen MB, Charles P. Positive effects of physiotherapy on chronic pain and performance in osteoporosis. *Osteoporosis Int.* 1998;8:215-21.
35. Thorsen K, Kristoffersson A, Lerner U, Lorentzon R. In situ microdialysis in bone tissue: Stimulation of prostaglandin E2 release by weight-bearing mechanical loading. *J Clin Invest* 1996;98:2446-9.
36. Gregg EW, Cauley JA, Seeley DG, Ensrud KE, Bauer DC. Physical activity and osteoporotic fracture risk in older women. *Ann Intern Med* 1998;129:81.
37. Burr DB. Muscle strength, bone mass and age-related bone loss. *J Bone Miner Res* 1997;12:1547-51.
38. Lorentzon R. Fysisk aktivitet och benmassa [Physical activity and bone mass] I: Osteoporos 1996- kunskapsunderlag och rekommendationer för Sverige. [Osteoporosis 1996 - knowledge base and recommendations for Sweden] Swedish Osteoporosis Society 1996;18-26.
39. Rodan GA. Bone mass homeostasis and bisphosphonate action. *Bone* 1997;20:1-4.
40. Heaney RP, Barger-Lux MJ, Davies KM, Ryan RA, Johnson ML, Gong G. Bone dimensional change with age: Interactions of genetic, hormonal, and body size variables. *Osteoporosis Int* 1997;7:426-31.
41. Bauman WA, Wecht JM, Kirshblum S, Spungen AM, Morrison N, Ciriigliaro C, Schwartz E. Effect of pamidronate administration on bone in patients with acute spinal cord injury. *J Rehabil Res Dev.* 2005;42:305-13.

39. Pain

Authors

Mats Börjesson, MD, PhD, Associate Professor, Department of Medicine, Sahlgrenska University Hospital, Gothenburg, Sweden

Kaisa Mannerkorpi, PT, Department of Physiotherapy, Sahlgrenska University Hospital, Gothenburg, Sweden

Stein Knardahl, MD, PhD, Professor, National Institute for Working Life and Psychology, Oslo University, Oslo, Norway

Jon Karlsson, MD, PhD, Professor, Department of Orthopaedics, Sahlgrenska University Hospital/Mölndal, Gothenburg, Sweden

Clas Mannheimer, MD, PhD, Professor, Pain Centre, Department of Medicine, Sahlgrenska University Hospital/Östra, Gothenburg, Sweden

Summary

Physical activity is of great significance for the treatment and rehabilitation of patients with long-term pain. There are three distinct effects of physical activity:

1) The “direct” pain-relieving effects of physical activity; 2) other “non-direct” effects on fitness and mood, reduced stress sensitivity and improved sleep, with potentially even greater effects on the pain situation of the patient; and 3) the positive effects of physical activity on lifestyle-related diseases in patients who tend to be inactive.

The physical activity must be regular and consistent to give direct or indirect pain relief. The physical activity should be carried out for at least 10 minutes, preferably for a much longer duration, and be of at least moderate intensity (> 60% of VO_2 max). Fitness and endurance training in the form of walking, jogging, cycling and swimming are often suitable activities. However, the type of physical activity the patient benefits from depends on his/her pain status and initial physical fitness. The physical fitness of patients with long-term pain is often very low and, consequently, the intensity of their chosen activity should ideally be gradually increased, starting at a low intensity level.

Introduction

Pain is a significant clinical problem. Studies show that up to 50 per cent of the population in Sweden and England suffer from chronic pain (9, 10). 13 per cent of the individuals questioned reported that their pain had led to reduced functional capacity (11). Only 40 per cent had been given a definite diagnosis (10). The most common clinical pain condition was chronic lumbago, i.e. chronic back pain (9). It has been described in the literature how individuals manage to continue with a particular sport without feeling any pain despite having suffered a stress fracture (1) or an acute heart attack (2). The importance of coping with pain in order to achieve success in endurance sports has also been the focus of much discussion (3). Elite swimmers have been shown to tolerate pain better than people who swim for the sake of exercise (4), and athletes who play contact sports appear to cope even better with pain than non-contact sport athletes (5). It has also been discussed whether sensitivity to pain in itself could be a predisposition for future physical inactivity (6).

The exact association between physical inactivity and long-term pain is still not fully known. Clinically, physical activity is considered to be of a great indirect and clinical importance in various chronic pain conditions. Aside from pain relief, physical activity may also contribute to increased functional capacity (by increasing fitness) in these patients. Expectancies may affect pain (12) as may improvement in the state of mood (13), which further reduces the pain experienced.

Definition

The International Association for the Study of Pain (IASP) defines pain as “an unpleasant experience that we primarily associated with tissue damage or describe in terms of tissue damage or both” (7). Accordingly, pain is a subjective experience that is not always related to the extent or even existence of tissue damage. Pain is a psychological phenomenon that must be described based on the behaviour and experience of the individual. Nonetheless, it is possible to assess pain using several methods.

Pain is either *acute* or long-term (*chronic*). Acute pain seldom gives rise to serious therapeutic problems and generally responds well to analgesics or cause-related treatment. Acute musculoskeletal pain usually responds well to analgesics or physiotherapy aimed at restoring function. Traditionally, the person administering the treatment (health professional) is normally active and the patient passive.

Chronic non-malignant pain (defined as pain that persists 3 months or more) is more complex, however, and often very difficult to treat. In such cases, ideally the patient should play a more active role while the person administering the treatment should act as an adviser, including encouraging the patient to be more active.

Pain analysis is used to assess whether the pain is *nociceptive*, i.e. stems from the skin, muscles or similar; *visceral*, i.e. stems from the inner organs; or *neuropathic*, i.e. caused by a nerve damage or dysfunction. Complex regional pain syndrome is often characterised by allodynia, which is painful hypersensitivity to stimuli that should normally not cause pain.

The definition of *pain threshold* is “the least stimulus intensity at which the patient perceives pain” (8). *Pain tolerance* is defined as the greatest level of pain that a patient is prepared to “tolerate”. Two individuals can have the same pain threshold but a different tolerance for the same painful event. These two definitions are critical in the pain analysis and for the treatment required.

Pain physiology

Pain, as experienced in the central nervous system (CNS), is the result of a complex processing of pain signals. Pain impulses usually transpire through the activation of peripheral pain receptors (nociceptors, from the Latin *nocere* = to injure). This activates primarily myelinated (A-delta nerve fibres) and thinner unmyelinated (C nerve fibres) neurons. The pain fibres reach the dorsal horn of the spinal cord via several segments. As a result, secondary neurons are activated and the pain signal is transmitted via the *spinothalamic fasciculus* up through the nervous system. Via the thalamo-cortical pathway, the signals reach the somatosensory cortex. Following a cortical processing, the activation typically gives rise to a sharp, well-localised feeling of pain (“it hurts”). Other fasciculi reach deeper and more diffuse subcortical areas (e.g. gyrus cinguli, prefrontal areas), also via the thalamus, for emotional processing. This gives rise to the emotional components of pain (“discomfort”), which are very important from a clinical standpoint.

In addition, there are a number of systems that process pain impulses before they give rise to the perception of pain. The spinal cord includes mechanisms that can both enhance and inhibit pain. Constant stimulation of the nociceptors may lead to central sensitisation, i.e. a heightened sensitivity to stimulation, which is of significant importance to long-term pain.

Descending pain-inhibiting systems from the *periaqueductal grey area* (PAG) and *nucleus raphe magnus* affect the sensory afferents of the spinal dorsal horn. Opioids play a dual role in these systems by activating the descending pain-inhibiting systems and inhibiting the ascending pain impulses of the spinal cord. Opioids can also lead to peripheral modulation of pain at the receptor level in connection with inflammation.

Psychological factors such as expectancies and experiences seem to influence the sensitisation of neurons (11) and, as a result, research into the mechanisms that underlie the placebo effect has intensified over the past few years.

The difficulties with experimental studies on physical activity and pain are the various types of pain-inducing stimuli used and the individual differences in pain sensitivity. In addition, there are methodological issues regarding the type of physical activity, duration and intensity. Experimentally induced pain is not comparable to clinical pain. For example, study subjects are usually less apprehensive/anxious since they know that the pain stimulation can be stopped at any time, which is not the case in real life situations.

The effects of physical activity on experiences of pain

Acute effects

Studies have shown that physical activity has a modulating effect on pain, i.e. it affects the way in which we experience induced pain, both during and after exercise. There are many indications that pain relief is an integral part of physical activity. An integration of behavioural and cardiovascular reactions as well as pain inhibition take place via the PAG (14). In addition, the pressure receptors of the cardiovascular system appear to have an effect on the pain system (15).

During exercise. Experimental studies indicate that the pain threshold for different forms of pain stimulation increases *during* physical activity. For example, this applies to dental pain (16), electrically induced finger pain during a cycle test (17), and pressure-induced pain in the quadriceps muscle during static load (18).

After exercise. Evidence suggests that different forms of physical activity lead to pain relief *using a broad range methods* of assessment, as well as different pain stimuli (e.g. electrical stimulation, heat, pressure and ischaemia) (6, 19–21). Both experimental and “natural” physical activities have been tried (22). A high-intensity activity seems to increase the pain threshold, which then gradually decreases once the activity is finished (see “Prescription” below). Running, for example, has a pronounced effect on one’s pain threshold. In a trial using thermal provocation, the analgesic effect of 45 minutes of high-intensity running corresponded to approximately 10 mg of intravenous morphine (19).

Paradoxal effects of physical activity on pain threshold. Experimental studies on fibromyalgia have shown physical activity to have the opposite effect. Unlike in healthy individuals (in whom the pain threshold decreases), the sensitivity to pain *was found to increase* during and after physical activity (23). This may render it more difficult for patients with fibromyalgia to be physically active, as part of their everyday clinical treatment.

Long-term effects

Do physically active persons *have a higher pain tolerance* than inactive persons? People who are physically active on a regular basis appear to have a higher *pain tolerance*. However, it does not necessarily mean that they have a higher pain threshold (24). Whether this potential difference in pain tolerance is due to the training itself, or simply individual/genetic, is currently subject to debate. However, the effect of physical activity on pain seems to be similar for both active sportsmen and untrained individuals (16, 20). A possible explanation for why certain runners are able to continue running despite an injury (1) could be that they have a high pain tolerance to begin with, which is then boosted by the extra physical load. Pain inhibition is an integrated part of the active pain control system (14). Also, by focusing on other signals from the body, attention is diverted away from the pain.

It is therefore likely that individuals with a high pain tolerance turn out to be the most successful in many athletic areas, such as endurance sports, where athletes often have to tolerate severe muscle pain (25). The training itself may have a positive effect on their sensitivity to pain (3). For example, it has been noted that the pain sensitivity of competitive swimmers varies depending on the intensity of training during a season (4).

When treating patients with *chronic pain*, increased physical activity is a key factor for improving the prognosis and alleviating the patient's suffering. Aerobic fitness and quality of life is gradually reduced in patients with chronic pain, with the risk of social isolation. Activity-induced pain combined with the anxiety and uncertainty that comes with the possibly unknown cause of the pain often leads to a reduced level of activity. Disaster thinking, i.e. expecting that the pain will increase and the prognosis is bad, may worsen the prognosis. Patients with chronic pain are often downhearted and sometimes depressed. This could make the pain situation even worse. In a small number of cases, negative attributes such as pain communication, inadequate and increased pain distribution, as part of the so-called "somatoform pain disorder", have developed.

Increased physical activity has a significant effect on patients with chronic pain. Not only does it reduce pain, but it also positively influences the patient's mood (13, 26), alleviates social isolation, and increases functional capacity (27). Physical activity may also lead to an improved perception of body image (28) and the person's self image of being a healthy individual (29). These effects also increase the possibility of the patient being able to handle and cope with pain. Secondary muscle tension caused by pain can be reduced through physical activity and mobility training.

Mechanisms

Physical activity as pain relief

Endogenous opioids

The most favoured theory behind the effects of physical activity on pain relief is based on endogenous opioids (beta-endorphins; the body's own opiates). These can act as pain inhibitors at different levels, as described above (24). The concentration of beta-endorphin in the blood increases with physical activity (30), although this is probably only partly responsible for the pain relief. According to a theoretical model, the activation of ergoreceptors in major muscle groups during physical activity can lead to increased central opioid activity through the activation of A-delta fibres (31).

However, it seems that physical activity of a high intensity is needed to release significant amounts of endorphins (corresponding to 75–80% of maximum oxygen uptake capacity; VO_2 max) (32, 33), i.e. an almost anaerobic workload (32). However, for physical activity at lower intensities, such as aerobic endurance training with stable lactate levels, a long duration is required (> 1 hour) in order to obtain an increased release of beta-endorphins (32). Some studies support the endorphin theory by showing that pain relief is reduced when patients are given naloxone (an antagonist to morphine and other opiates

and opioids that obstruct the analgesic effect of opiates by blocking their receptors) (19, 21), whereas other studies have been unable to confirm these findings (17, 21). This may be explained by an undefined selectivity to naloxone. One study has also been able to show that low-intensity physical activity equivalent to 63 per cent of VO_2 max may indeed increase endorphin levels and tolerance to pain (27). Thus, there seems to be more than one explanation for the secondary effects that physical activity has on pain. According to another study, aerobic activity at 50 per cent of VO_2 max did not give any relief from pressure induced pain (34), which may be an indication that there is a lower limit of intensity to achieve pain-relief.

Other descending pain-inhibiting systems that use different neurotransmitters (e.g. serotonin and noradrenaline) may potentially also be involved in pain relief secondary to physical activity.

Increased activity in non-pain transmitting sensory fibres

Activation of large afferents (sensory fibres) could, in theory, lead to reduced pain via the activation of pain-inhibiting interneurons (Gate Control Theory) (35). This effect is not mediated through increased opioid activity, but possibly through the transmitter gamma-aminobutyric acid (GABA).

Distraction

A distraction or diversion has been proven to change the experience of pain (36) and can contribute to alleviating pain during and after an activity (37). A sports activity may distract an individual from pain, as also illustrated historically by injured and fleeing soldiers. Consequently, the analgesic effects of physical activity demonstrated in laboratory tests may be underestimated due to insufficient exterior influences (38).

Expectations

It can be difficult to differentiate between the effect that the stress experienced before a competition/activity has on pain and the effect the physical activity itself has on the same pain. *Expectations* prior to a physical activity, in addition to *trepidation*, could in themselves act as pain inhibitors or enhancers/triggers (39). One study showed that more than 50 per cent of study subjects who were told that they might get a headache from electrical stimulation did in fact get such a headache, despite not being exposed to any such electrical stimulation (expected or nocebo pain) (40).

Adaptive reactions – stress

Regulation of pain sensitivity is an integrated part of our adaptive reactions to stress (14). *Acute stress* is usually associated with pain relief (41, 42), while *chronic stress* is usually associated with an increased sensitivity to pain (43).

Regular physical activity may *reduce sympathetic activity*, which in turn can lead to pain relief by way of reduced ischaemia in conditions such as angina pectoris, peripheral vascular disease and dysmenorrhoea (see below).

Sensitisation and previous experiences with pain

Persons with previous pain experiences may feel less pain than persons without such experiences (44). However, other studies seem to suggest the opposite, possibly due to various circumstances. Patients with fibromyalgia experience *hyperalgesia*, i.e. increased pain sensitivity during a similar form of stimulation and activity. This could be explained by sensitisation (23) as part of the development of chronic pain. Direct pain-relieving effects are noted in patients with fibromyalgia who are able to do the same type of moderate to high-intensity exercise as healthy individuals (45), whereas other patients with fibromyalgia doing only lower intensity training see bigger improvements in their general health status and mood state (29, 46).

Indirect effects

Effect on depression and anxiety

According to one study, 8 weeks of physical activity (walking or jogging) led to reduced symptoms of depression and anxiety (13). This has also been shown in other studies (47, 48). Depression may lead to increased pain and a reduced capacity to cope with the pain situation. Consequently, increased physical activity may have a positive effect on the pain situation by boosting the patient's self-esteem and mood state. This positive effect of physical activity appears to be mediated partly through its effect on the central serotonergic system (49). Patients with fibromyalgia report being less depressed and anxious following a period of physical activity. These effects may be achieved with both low-intensity and high-intensity exercise (50, 51).

Effect on sleeping

Regular physical activity of a moderate intensity has been shown to improve the quality of sleep (52). This should, in theory, also contribute to a better pain situation, as the patient feels more rested and content.

Prescription

Duration. Available studies suggest that it is possible to achieve pain relief through short sessions of physical activity, though a minimum duration of 10 minutes is needed (34, 38). However, a further increase in the pain threshold can be expected if the physical activity continues for longer than 10 minutes. Thus, 50 minutes of running increases the ischaemic pain threshold more than 15 minutes of running on a treadmill (38). The effect on pressure-induced pain lasts for at least 5 minutes after exercise (18). The pain thresholds revert to normal within approximately one hour after the physical activity has finished (17).

Type of activity. Almost all data on physical activity and pain relates to aerobic fitness training. A small-scale study showed that 45 minutes of strength training at 75 per cent of an individual's 1 RM (1 RM = Repetition Maximum, i.e. the maximum amount of weight

one can lift in a single repetition for a given exercise) resulted in a significantly heightened pain thresholds and reduced pain intensity compared with control groups (53). The alleviation of pain lasted for 10 minutes, i.e. a considerably shorter effect than after fitness training (53). However, according to another study, pain tolerance did not increase after 12 weeks of strength training (3). Hence, the effect of strength training on the release of beta-endorphins is still unclear (33). More studies are needed to determine whether strength training does have an alleviating effect on pain.

Intensity. High-intensity fitness training, such as cycling at a minimum of 75–80 per cent of VO_2 max (which increases beta-endorphin levels), has shown to alleviate pain (19, 54). However, even low-intensity fitness training (63% of VO_2 max) increases pain tolerance (27). According to a study using pressure induced pain, fitness training at 50 per cent of VO_2 max did not alleviate pain (34), which appears to indicate that there is a low-intensity limit. Yet, the fact that submaximal exertion seems to have an alleviating effect on pain does have important therapeutical implications, as this means that more patients may be able to alleviate their pain through physical activity. Persons with chronic pain often have low functional capacity (55) and, as a result, may find it difficult to engage in relatively high-intensity activities. A simple activity such as walking could therefore be of sufficient relative intensity for an unfit person in this context.

Continuity. The prescribed physical activity has to be regular for continuous effect.

Summary of prescription

A physical activity must be regular and consistent to give direct or indirect pain relief. The physical activity should be carried out for at least 10 minutes, preferably much longer, and be of at least a moderate level of intensity ($> 60\%$ of VO_2 max). Aerobic fitness and endurance training in the form of walking, jogging, cycling and swimming are often suitable, but the type of physical activity the patient benefits from most depends on his/her pain status and initial fitness. The physical capacity of patients with chronic pain is often very low and, consequently, the intensity of their chosen activity should be gradually increased, starting from a relative (individual) low intensity.

Clinical indications

Physical activity as pain relief in patients with various diseases

Unspecified Chronic Pain (UCP) and Chronic Pain Syndrome (CPS)

Whatever the initial cause of the pain, physical activity plays a very important role and is perhaps the most important part of the patient's treatment programme. The patients regularly show a low functional capacity (reduced fitness) and are often passive. Breaking this vicious circle is vital, as is to gradually and carefully increase the patients' level of physical

activity. At the same time, the patients are often in a primary or secondary state of dejection or even depression. If this is the case, then physical activity could act as a positive complement to traditional medical treatments (13).

Before choosing a passive treatment method such as weak analgesics for a non-depressed patient, the patient should reach a basic level of physical activity (walking). More specific training programmes are also of significant importance, not least to show the patient that it is possible to remain active, i.e. to eliminate unconstructive thoughts relating to the pain.

Chronic lumbago

Indication and prescription

Individualised functional training combined with information about the underlying disease, with the aim of increasing the person's level of physical activity, is considered essential for patients with chronic lumbago. Women with lumbago have a lower aerobic fitness level compared to male subjects of the same age (56).

A training programme may incorporate stability training, graded endurance and strength training, as well as stretching of shortened torso and leg muscles. Studies have shown that strength and an increase in training gives improved back function and reduced pain when comparing a group of training patients to an untreated control group (57, 58). Also fitness training has been shown to have positive effects on pain intensity and pain frequency (59). This patient group may even benefit from everyday physical activities (60). Cycling was shown to have an effect on the quality of life and mental capacity of older patients with lumbago (61). In addition, a Pilates-based approach to training seemed to have a better effect than standard treatments (62).

It is recommended that the training begin under the supervision of a physiotherapist who will adapt the various exercises to the patient's functional limitations and pain, and gradually increase the physical load. The long-term effects of supervised training surpass the effects attained from self-training at home (58).

In the case of acute back pain without a known pathologic cause, everyday physical activities seems to be most beneficial (63). The risk of prescribing physiotherapy is that it may enhance the patient's perception of disease rather than improve the situation.

Fibromyalgia

Indication

Many patients with fibromyalgia are physically inactive due to an overall feeling of tiredness and diffuse pain. Inactivity is considered one of the most important reasons for these patients having reduced functional capacity, as well as reduced muscle strength and aerobic fitness (64). However, the ability of the patients to train their muscles does not appear to be affected (65).

Prescription

Numerous studies have shown that patients with fibromyalgia benefit from physical training in that their physical functioning improves and the severity of their symptoms is reduced, which boosts their frame of mind. The feeling of general well-being is further enhanced if the training takes into account the patient's current level of functioning and pain tolerance (29). It is worth noting that the intensity of a physical activity is always relative. In other words, it must be prescribed in relation to the individual capacity of the patient.

Low-intensity training

Patients with fibromyalgia whose level of activity have been low for a longer period of time are often despondent or frightened of experiencing increased pain as a result of physical activity. The initial training should therefore be of a low intensity and gradually increase at a rate that the patient feels comfortable with. If the training is beneficial or manageable, the patient is more likely to feel motivated to continue training. For these patients, regularity is more important than the intensity of the training. Studies have shown that walking (46, 66, 67) and low to moderate-intensity training in a heated pool (50, 68–71) improves physical functioning and lessens the severity of symptoms and feelings of despondency. Patients who are unable or do not have time to exercise for an uninterrupted duration of 30 minutes may divide the training into two 15-minute training sessions daily (72).

Body image therapy and self-care training may be vital for those patients who need to improve their knowledge of their bodies and physical limitations (29, 73).

Moderate to high-intensity training

Patients who are able to do fitness training for at least 20 minutes 2–3 times a week, at an intensity of 55–90 per cent of their maximal heart rate have been shown to have improved fitness and pain situation (45). The types of exercise carried out include cycling (74), combined with fitness training, strength training and stretching (75). Because of the problematic pain situation, not all patients manage to achieve a (relatively) moderate to high-intensity level of training (76). However, most untrained patients with a low aerobic fitness level are able to achieve moderate-intensity training by simply walking on flat ground!

Strength training

Patients who begin strength training at 40–60 per cent of their maximum capacity and gradually increase the resistance to 60–80 per cent have been shown to have improved muscle strength.

In short, fitness training, walking, exercising in water and strength training are generally considered to have a positive influence on the physical functions, symptoms and despondency of patients with fibromyalgia. Since pain often leads to muscle tension and possibly shortening of the muscles, stretching after a training session is considered beneficial. Training that is carried out at an adequate level seems to give patients added self-esteem and a more positive view of their bodies. Many patients prefer training in a group as it offers them the social support needed to continue training regularly.

Neck pain/whiplash

For patients with chronic neck pain, a structured training programme that focuses on the neck area may alleviate pain and increase physical functioning and muscle strength (79). A combination of strength training and stretching is recommended (80). Neck training exercises may also lead to reduced muscle fatigue (81).

In a randomised controlled trial on patients with a “whiplash-associated disorder” (WAD), physical activity carried out in addition to standard treatment had the greatest effect on pain and on functionality in patients with the most pronounced symptoms (82). There are considerable geographical differences in the prevalence of WAD, and it is much more common in Scandinavia/Canada than, for example, Lithuania/Greece (83). This indicates that other factors, such as the expectation of chronic pain, are of importance. Physical activity plays an important role in showing the patient that it is possible to be physically active despite the pain.

Urogenital pain

A 3-month fitness training programme reduced symptoms of primary dysmenorrhea (84). Whether this is due to a reduced sympathetic tone or endorphins has been discussed (85).

A double-blind randomised study examined the effects of fitness training in men with refractory chronic prostatitis/chronic pelvic pain compared with placebo/stretching. The results showed that 18 weeks of aerobic fitness training reduced the pain as well as improving depression and anxiety (86).

Rheumatoid arthritis/osteoarthritis

Reduced functional capacity, restricted movements and pain are common problems among patients with rheumatoid arthritis (RA), the characteristics of which are inflammation of the joints, tendon sheaths and bursae, with subsequent destruction of cartilage and bone. The pain experienced in connection with RA is usually nociceptive, due to inflammation or destruction, or neurogenic, due to compression of peripheral nerves or nerve roots. When putting together an exercise programme, the joint and muscle function of the patient must be taken into account, and the exercise programme be individualised accordingly.

Flexibility training for the prevention of restricted flexibility should be carried out daily, especially during periods of aggravated joint inflammation. If the patient experiences localised pain, with restricted flexibility, muscle fatigue and pain, the flexibility may have to be addressed separately before the patient can commence strength and aerobic fitness training. Patients with RA are able to improve their muscle strength (87) and fitness (88) through strength and fitness training. A number of scientific studies have used endurance/strength training at 50–80 per cent of Maximal Voluntary Contraction, and fitness training at 60–85 per cent of Maximal Heart Rate (88).

Some of the studies reported a reduction in pain subsequent to group strength and fitness training (88). No increased inflammation activity or symptoms (87, 88) were reported following an adequately planned training programme.

Ischaemic pain – vascular disease

An association between physical activity and pain has been noted in various forms of *ischaemic pain situations*, such as angina pectoris and claudication (claudicatio intermittens). In the case of claudication, physical activity has been shown to alleviate pain and lengthen the walking distance (89, 90). The improvement can be attributed to both the improved physical health and increased functional capacity (91).

In the case of a coronary artery disease, the appropriate dose of physical activity has been shown to increase the functional capacity for a given exercise (92). A lower heart rate reduces the oxygen consumption in the heart muscle, which in turn reduces ischaemia (93) and delays the onset of angina pectoris (92). Patients participating in a cardiac rehabilitation programme usually have a low level of fitness at the start of the programme (94), which further reinforces the need for participating in such programmes. These programmes can potentially affect the patients' physical activity levels and quality of life for a number of years (95).

Interactions

Analgesics or other pain-modulating substances, such as non-steroidal anti-inflammatory drugs (NSAID), are normally used to alleviate pain. Products such as NSAIDs are able to modify the pain experience by influencing both the spinal and peripheral receptors, for example, through reduced sensitisation. NSAIDs should not be prescribed to individuals with risk factors for or established cardiovascular disease. However, low-dose NSAIDs are non-prescription drugs. Analgesics are sometimes indicated for minor traumas that do not affect the general activity level, e.g. a toe fracture in a football player. However, by using analgesics, the protective aspects of the sensation of pain itself are partly eliminated, entailing a risk of making the injury worse.

Contraindications

Pain is not altogether a bad thing. Humans are equipped with a pain system that, among other functions, acts as a defense mechanism against trauma and other damaging impacts on the body. It is important to remember that high-intensity training in connection with *fibromyalgia* is usually contraindicated at first (see above), while acute pain may be a sign of injury, whereupon physical activity should be avoided.

Always listen to the body. The treatment of acute pain due to distortion of the knee ligaments or a collision-induced fracture rarely poses a problem. However, the gradual onset of pain, often the result of an overuse injury, can be a bigger problem, while long-standing pain remains the greatest treatment challenge.

References

1. Colt EWD, Spyropoulos E. Running and stress fractures. *Br Med J* 1979;2:706.
2. Colt EWD. Letter to the Editor. *N Engl J Med* 1980;302:57.
3. Anshel MH, Russell KG. Effect of aerobic and strength training on pain tolerance, pain appraisal and mood of unfit males as a function of pain location. *J Sports Sci* 1994; 12:535-47.
4. Scott V, Gijsbers K. Pain perception in competitive swimmers. *Br Med J* 1981;283:91-3.
5. Ryan ED, Kovacic CR. Pain tolerance and athletic participation. *Perc and Motor Skill* 1966;22:383-90.
6. Janal MN, Glusman M, Kuhl JP, Clark WC. Are runners stoical? An examination of pain sensitivity in habitual runners and normally active controls. *Pain* 1994;58:109-16.
7. IASP. IASP Subcommittee on Taxonomy. Pain terms. A list with definitions and notes on usage. *Pain* 1979;6:249-52.
8. Guyton AC. Textbook of medical physiology. 7th edn. Philadelphia (PA): W.B. Saunders; 1986.
9. Elliott AM, Smith BH, Penny KI, Smith WC, Chambers WA. The epidemiology of chronic pain in the community. *Lancet* 1999;354:1248-52.
10. Andersson HI. The epidemiology of chronic pain in a Swedish rural area. *Qual Life Res* 1994;suppl 1:19-26.
11. Matre D, Casey KL, Knardahl S. Placebo-induced changes in spinal cord pain processing. *J Neuroscience* 2006;26:559-63.
12. Finnis DG, Benedetti F. Placebo analgesia, nocebo hyperalgesia. *Pain Clin Updates* 2007;XV(1).
13. Sexton H, Maere Å, Dahl NH. Exercise intensity and reduction in neurotic symptoms. *Acta Psychiatr Scand* 1989;80:231-5.
14. Bandler R, Shipley MT. Columnar organization in the midbrain periaqueductal grey. Modules for emotional expression? *Trends Neurosci* 1994;17:379-89.
15. Rau H, Brody S, Larbig W, Pauli P, Vöhringer M, Harsch B, et al. Effects of PRES baroreceptor stimulation on thermal and mechanical pain threshold in borderline hypertensives and normotensives. *Psychophysiol* 1994;31:480-5.
16. Pertovaara A, Huopaniemi T, Virtanen A, Johansson G. The influence of exercise on dental pain thresholds and the release of stress hormones. *Physiol Behav* 1984;33:923-6.
17. Droste C, Greenlee MW, Schreck M, Roskamm H. Experimental pain thresholds and plasma beta-endorphin levels during exercise. *Med Sci Sports Exerc* 1991;23:334-42.
18. Kosek E, Ekholm J. Modulation of pressure pain thresholds during and following isometric contraction. *Pain* 1995;61:481-6.
19. Janal MN, Colt EWD, Clark WC, Glusman M. Pain sensitivity, mood and plasma endocrine levels in man following long-distance running. Effects of naloxone. *Pain* 1984;19:13-25.

20. Kempainen P, Pertovaara A, Huopaniemi T, Johansson G, Karonen SL. Modification of dental pain and cutaneous thermal sensitivity by physical exercise in man. *Brain Res* 1985;360:33-40.
21. Haier RJ, Quaid K, Mills JSC. Naloxone alters pain perception after jogging. *Psychiatr Res* 1981;5:231-2.
22. Bartholomew JB, Lewis BP, Linder DE, Cook DB. Post-exercise analgesia. Replication and extension. *J Sports Sci* 1996;14:329-34.
23. Kosek E, Ekholm J, Hansson P. Modulation of pressure pain thresholds during and following isometric contraction in patients with fibromyalgia and in healthy controls. *Pain* 1996;64:415-23.
24. O'Connor PJ, Cook DB. Exercise and pain. The neurobiology, measurement, and laboratory study of pain in relation to exercise in humans. *Exerc Sports Sci Rev* 1999;27:119-66.
25. Cook DB, O'Connor PJ, Eubanks SA, Smith JC, Lee M. Naturally occurring muscle pain during exercise. Assessment and experimental evidence. *Med Sci Sports Exerc* 1997;29:999-1012.
26. Morgan WP. Affective beneficence of vigorous physical activity. *Med Sci Sports Exerc* 1985;17:94-100.
27. Gurevich M, Kohn PM, Davis C. Exercise-induced analgesia and the role of reactivity in pain sensitivity. *J Sports Sci* 1994;12:549-59.
28. Tucker L. Effect of weight training and self concept. A profile of those influenced most. *Res Quart Exerc Sports* 1983;54:389-97.
29. Mannerkorpi K, Gard G. Physiotherapy group treatment for patients with fibromyalgia. An embodied learning process. *Disabil Rehabil* 2003;25:1372-80.
30. Colt WD, Wardlaw SL, Frantz AG. The effect of running on plasma beta-endorphin. *Life Sciences* 1981;28:1637-40.
31. Thoren P, Floras JS, Hoffman P, Seals DR. Endorphins and exercise. Physiological mechanisms and clinical implications. *Med Sci Sports Exerc* 1990;22:417-28.
32. Schwarz L, Kindermann W. Review. Changes in beta-endorphin levels in response to aerobic and anaerobic exercise. *Sports Medicine* 1992;13:25-36.
33. Goldfarb AH, Jamurtas AZ. Beta-endorphin response to exercise. *Sports Med* 1997; 24:8-16.
34. Hoffman MD, Shepanski MA, Ruble SB, Valic Z, Buckwalter JB, Clifford PS. Intensity and duration threshold for aerobic exercise-induced analgesia to pressure pain. *Arch Phys Med Rehabil* 2004;85:1183-7.
35. Melzack, Wall D. Pain mechanisms. A new theory. *Science* 1965;150:971-9.
36. Miron D, Duncan GH, Bushnell MC. Effects of attention on the intensity and unpleasantness of thermal pain. *Pain* 1989;39:345-52.
37. Debreuil DL, Endler NS, Spanos NS. Distraction and redefinition in the reduction of low and high intensity experimentally induced pain. *Imag, Cogn and Pers* 1987;7:155-64.
38. Janal MN. Pain sensitivity, exercise and stoicism. *J R Soc Med* 1996;89:376-81.
39. Sternberg WF, Bailin D, Grant M, Gracely RH. Competition alters the perception of noxious stimuli in male and female athletes. *Pain* 1998;76:231-8.

40. Bayer TL, Baer PE, Early C. Situational and psychophysiological factors in psychologically induced pain. *Pain* 1991;44:45-50.
41. Millan MJ, Przewlocki R, Herz A. A non-beta andorphinergic adenohipophyseal mechanism is essential for an analgesic response to stress. *Pain* 1980;33:343-53.
42. Pitman RK, van der Kolk BA, Orr SP, Greenberg MS. Naloxone-reversible analgesic response to combat-related stimuli in posttraumatic stress disorder. *Arch Gen Psychiatry* 1990;47:541-4.
43. van Houdenhove B. Psychosocial stress and chronic pain. *Eur J of Pain* 2000;4:225-8.
44. Kempainen P, Hämäläinen O, Könönen M. Different effects of physical exercise on cold pain sensitivity in fighter pilots with and without the history of acute in-flight neck pain attacks. *Med Sci Sports Exerc* 1998;30:577-82.
45. Busch A, Schacter C, Peloso P, Bombardier C. Exercise for treating fibromyalgia syndrome. Oxford: The Cochrane Library; 2002.
46. Valim V, Oliveira L, Suda A, Silva L, de Assis M, Barros Neto T, et al. Aerobic fitness effects in fibromyalgia. *J Rheumatol* 2003;30:1060-9.
47. Byrne A, Byrne DG. The effect of exercise on depression, anxiety and other mood states. A review. *J Psychosom Res* 1993;37:565-74.
48. LaFontaine TP, DiLorenzo TM, Frensch PA, Stucky-Ropp RC, Bargman EP, McDonald DG. Aerobic exercise and mood. A brief review 1985-90. *Sports Med* 1992;13:160-70.
49. Chaouloff F. Effects of acute physical exercise on central serotonergic systems. *Med Sci Sports Exerc* 1997;29:58-62.
50. Gowans S, deHueck A, Voss S, Silaj A, Abbey S, Reynolds. Effect of a randomized controlled trial of exercise on mood and physical function in individuals with fibromyalgia. *Arthritis Rheum* 2001;45:519-29.
51. Mannerkorpi K, Iversen M. Physical exercise in fibromyalgia and related syndromes. *Best Pract & Res Clin Rheumatol* 2003;17:629-47.
52. King AC, Oman RF, Brassington GS, Bliwise DL, Haskell WL. Moderate-intensity exercise and self-rated quality of sleep in older adults. *JAMA* 1997;277:32-7.
53. Koltyn KF, Arbogast RW. Perception of pain after resistance exercise. *Br J Sports Med* 1998;32:20-4.
54. Koltyn KF, Garvin AW, Gardiner RL, Nelson TF. Perception of pain following aerobic exercise. *Med Sci Sports Exercise* 1996;28:1418-21.
55. Bennett RM, Clark SR, Goldberg L, Nelson D, Bonafede RP, Porter J, et al. Aerobic fitness in patients with fibrositis. *Arthritis and Rheum* 1989;32:454-60.
56. Hoch AZ, Young J, Press J. Aerobic fitness in women with chronic discogenic non-radicular low back pain. *Am J Phys Med Rehabil* 2006;85:607-13.
57. Hayden J, Tulder M, Malmivaara A, Koes B. Meta-analysis. Exercise therapy for non-specific low back pain. *Ann Intern Med* 2005;142:765-75.

58. Liddle S, Baxter G, Gracey J. Exercise and chronic low back pain. What works? *Pain* 2004;107:176-90.
59. Mannion A, Muntener M, Taimela S, Dvorak J. Comparison of three active therapies for chronic low back pain. Results of a randomized clinical trial with one-year follow-up. *Rheumatology* 2001;40:772-8.
60. Chatzitheodorou D, Kabitsis C, Malliou P, Mougios V. A pilot study of the effects of high-intensity aerobic exercise versus passive interventions on pain, disability, psychological strain and serum-cortisol concentrations in people with chronic low back pain. *Phys Ther* 2007;87:304-12.
61. Iversen MD, Fossel AH, Katz JN. Enhancing function in older adults with chronic low back pain. A pilot study of endurance training. *Arch Phys Med Rehabil* 2003;84:1324-31.
62. Rydeard R, Leger A, Smith D. Pilates-based therapeutic exercise. Effect on subjects with nonspecific chronic low back pain and functional disability. A randomized controlled trial. *J Orthop Sports Phys Ther* 2006;36:472-84.
63. Malmivaara A, Häkkinen U, Aro T. The treatment of acute low back pain. Bed rest, exercises, or ordinary activity? *N Engl J Med* 1995;332:351-5.
64. Bennett R, Clark S, Goldenberg L, Nelson D, Bonafede R, Porter J, et al. Aerobic fitness in patients with fibrositis. *Arthritis Rheum* 1989;32:454-60.
65. Valkeinen H, Häkkinen A, Hannonen P, Häkkinen K, Alen M. Acute heavy-resistance exercise-induced pain and neuromuscular fatigue in elderly women with fibromyalgia and in healthy controls. Effects of strength training. *Arthritis Rheum* 2006;54:1334-9.
66. Buckelew S, Conway R, Parker J, Deuser W, Read J, Witty T, et al. Biofeedback/relaxation training and exercise interventions for fibromyalgia. A prospective trial. *Arthritis Care Res* 1995;11:196-209.
67. Richards SCM, Scott DL. Prescribed exercise in people with fibromyalgia. Parallel group randomised controlled trial. *Br Med J* 2002;325:185-7.
68. Cedraschi C, Desmeules J, Rapiti E. Fibromyalgia. A randomised, controlled trial of a treatment programme based on self-management. *Ann Rheum Dis* 2004;63:290-6.
69. Gowans S, deHueck A, Voss S, Richardson M. A randomised controlled trial of exercise and education for individuals with fibromyalgia. *Arthritis Care Res* 1999;12:120-8.
70. Mannerkorpi K, Nyberg B, Ahlmén M, Ekdahl C. Pool exercise combined with an education program for patients with fibromyalgia syndrome. *J Rheumatol* 2000;27:2473-81.
71. Gusi N, Tomas-Carus P, Häkkinen A, Häkkinen K, Ortega-Alonso A. Exercise in waist-high warm water decreases pain and improves health-related quality of life and strength in the lower extremities in women with fibromyalgia. *Arthritis Rheum* 2006;55:66-73.
72. Schachter C, Busch A, Peloso P, Sheppard M. The effects of short versus long bouts of aerobic exercise in sedentary women with fibromyalgia. A randomized controlled trial. *Phys Ther* 2003;83:340-58.
73. Gustafsson M, Ekholm J, Broman L. Effects of a multiprofessional rehabilitation programme for patients with fibromyalgia syndrome. *J Rehabil Med* 2002;34:119-27.

74. McGain G, Bell D, Mai F, Halliday P. A controlled study of the effects of a supervised cardiovascular fitness program on the manifestations of primary fibromyalgia. *Arthritis Rheum* 1988;31:1135-41.
75. Wigers S, Stiles T, Vogel P. Effects of aerobic exercise versus stress management treatment in fibromyalgia. *Scand J Rheumatol* 1996;25:77-86.
76. van Santen M, Bolwijn P, Verstappen F. A randomized clinical trial comparing fitness and biofeedback training versus basic treatment in patients with fibromyalgia. *J Rheumatol* 2002;29:575-81.
77. Häkkinen A, Häkkinen K, Hannonen P, Alen M. Strength training induced adaptations in neuromuscular function of premenopausal women with fibromyalgia. A comparison with healthy women. *Ann Rheum Dis* 2001;60:21-6.
78. Jones K, Burckhardt C, Clark S, Bennet R, Potempa K. A randomized controlled trial of muscle strengthening versus flexibility training in fibromyalgia. *J Rheumatol* 2002;29:1041-8.
79. Chiu TT, Lam TH, Hedley AJ. A randomised controlled trial on the efficacy of exercise for patients with chronic neck pain. *Spine* 2005;30:1-7.
80. Ylinen JJ, Takala EP, Nykänen MJ, Kautianen HJ, Häkkinen AH, Airaksinen OV. Effects of twelve-month strength training subsequent to twelve-month stretching exercise in treatment of chronic neck pain. *J Strength Con Res* 2006;20:304-8.
81. Falla D, Jull G, Hodges P, Vicenzino B. An endurance-strength training regime is effective in reducing myoelectric manifestations of cervical flexor muscle fatigue in females with chronic neck pain. *Clin Neurophysiol* 2006;117:828-37.
82. Stewart MJ, Maher CG, Refshauge KM, Herbert RD, Bogduk N, Nicholas M. Randomized controlled trial of exercise for chronic whiplash-associated disorders. *Pain* 2007;128:59-68.
83. Schrader H, Stovner LJ, Obelieniene D, Surkiene D, Mickeviciene D, Bovim G, et al. Examination of the diagnostic validity of headache attributed to whiplash injury. A controlled, prospective study. *Eur J Neurol* 2006;13:1226-32.
84. Israel RG, Sutton M, O'Brien KF. Effects of aerobic training on primary dysmenorrhea symptomatology in college females. *J Am Coll Health* 1985;33:241-4.
85. Golomb LM, Solidum AA, Warren MP. Primary dysmenorrhea and physical activity. *Med Sci Sports Exerc* 1998;30:906-9.
86. Giubilei G, Mondaini N, Minervini A, Saieva C, Lapini A, Serni S, et al. Physical activity of men with chronic prostatitis/chronic pelvic pain syndrome not satisfied with conventional treatments. Could it represent a valid option? The physical activity and male pelvic pain trial. A double-blind, randomized study. *J Urol* 2007;177:159-65.
87. Häkkinen A. Effectiveness and safety of strength training in rheumatoid arthritis. *Curr Opin Rheumatol* 2004;16:132-7.
88. Stenström C, Minor M. Evidence for the benefit of aerobic and strengthening exercise in rheumatoid arthritis. *Arthritis Rheum* 2003;49:428-34.
89. Hiatt WR, Regensteiner JG, Hargarten ME, Wolfel EE, Brass EP. Benefit of exercise conditioning for patients with peripheral vascular disease. *Circulation* 1990;81:602-9.

90. Gardner AW, Poehlman ET. Exercise rehabilitation programs for the treatment of claudication pain. A meta-analysis. *JAMA* 1995;274:975-80.
91. Tsai JC, Chan P, Wang CH, Jeng C, Hsieh MH, Kao PF, et al. The effects of exercise training on walking function and perception of health status in elderly patients with peripheral arterial occlusive disease. *J Intern Med* 2002;252:448-55.
92. Thompson PD. Exercise for patients with coronary artery and/or coronary heart disease. In: Thompson PD, Ed. *Exercise and Sports Cardiology*. New York: McGraw-Hill; 2001. p. 354-70.
93. Todd IC, Ballantyne D. Antianginal efficacy of exercise training. A comparison with beta blockade. *Br Heart J* 1990;64:14-9.
94. Ades PA, Savage PD, Brawner CA, Lyon CE, Ehrman JK, Bunn JY, et al. Aerobic capacity in patients entering cardiac rehabilitation. *Circulation* 2006;113:2706-12.
95. Hage C, Mattsson E, Ståhle A. Long-term effects of exercise training on physical activity level and quality of life in elderly coronary patients. A three- to six-year follow-up. *Physiother Res Int* 2003;8:13-22.

40. Parkinson's disease

Authors

Kristian Borg, MD, PhD, Professor, Rehabilitation Medicine, Karolinska Institutet, Danderyd University Hospital, Stockholm, Sweden

Svein Ivar Bekkelund, MD, PhD, Professor, Department of Neurology, University Hospital of North Norway, Tromsø, Norway

Marketta Henriksson, PT, PhD, Department of Neurobiology, Care Sciences and Society and Department of Physiology and Pharmacology, Karolinska Institutet, Stockholm, Sweden

Summary

Parkinson's disease is characterised by a general impairment of motor skills. Physical activity is of utmost importance and ought to be introduced in the early stages of the disease. Patients are recommended general physical activities such as hiking, walking, etc. combined with physiotherapy and home exercise programmes. Scientific studies indicate that fitness training and specific endurance training may have beneficial effects and can be recommended in certain cases. Strength training also appears to have beneficial effects, but there is currently not enough scientific evidence to recommend such training. In addition, physical activity is likely to prevent inactivity and a fear of exercise and reduces the risk of fall related injuries. Training on a stepper machine or walking on a treadmill with supports is a preferred form of exercise to cycling for patients with Parkinson's disease as spinal extension is needed to avoid kyphoscoliosis (stooped spine).

Definition

Symptoms

Parkinson's disease is characterised by increased rigidity (stiffness), hypokinesia (diminished muscle movement) and tremors (shaking) (1–3). These characteristics usually develop gradually and slowly. A common initial symptom of the disease is tremors, usually starting in one part of the body until eventually all body extremities are affected. Hypokinesia affects the entire locomotion of the patient. Patients also find it difficult to

start walking, change directions and turn. The stride is short. Parkinson's disease also leads to a change in posture including kyphoscoliosis and flexion of the hips, moving the centre of gravity forward. Patients develop a specific way of walking with the arms hanging motionless at the sides. The head is pushed forward, the spine curves and there is a lateral (sideways) movement in the shoulder blades with the shoulders being pushed forward and the upper arms turned inwards. This tendency for propulsion, i.e. forward movement, increases the risk of falling. The effect on the scalene, sternocleido mastoideus and pectoralis muscles may also lead to a deterioration in the oxygen exchange and respiration function with an increased feeling of tiredness.

In the later stages of the disease, it is not unusual for an on-off phenomenon to take place, especially during treatment with L-dopa, when the patient loses all forms of motor ability for a short period of time. The tremor is characteristic and often referred to as pill-rolling tremor. Mental health problems such as depression are not uncommon among persons suffering from Parkinson's disease nor is the onset of dementia.

Prevalence/Incidence

The overall prevalence estimate of Parkinson's disease is 15 per 10,000 residents (1, 2). The average age at onset is 55–60 years.

Diagnosis/pathophysiology

The diagnosis is an evidence-based clinical diagnosis. A number of possible pathophysiological causes of Parkinson's disease have been identified. The principal cause is a lack of dopamine in the basal ganglia. However, the background to the disease is unclear although genetic factors incorporating various mutations have been reported in families with Parkinson's disease (4). Epidemiological studies have also shown that environmental factors, such as exposure to pesticides (chemical products against fungi, insects and worms) constitute a risk factor (5, 6). A trend towards a lower risk of developing Parkinson's disease has been reported with a high level of physical activity (7, 8).

Current treatment principles

Parkinson's disease is usually treated with a combination of pharmacology and physiotherapy (1–3). In exceptional cases, neurological surgery with deep brain stimulation is carried out for the purpose of alleviating tremor and rigidity. The pharmacological treatment focuses on replacing low levels of dopamine in the basal ganglia. L-dopa is administered orally. Current preparations also contain a peripheral decarboxylase inhibitor that prevents the breakdown of dopamine. Hypokinesia is a dose-related side-effect of L-dopa. In addition, orthostatic hypertension (fall in blood pressure when standing up) and psychological side-effects such as confusion (perplexity) are relatively common, especially among elderly patients. Patients may also experience delusions and hallucinations. Sleeping difficulties and nightmares are not uncommon. The therapeutic arsenal

also includes other preparations which enhance dopaminergic activity. Dopamine agonists such as Bromocriptine and Apomorphine as well as COMT and MAO-B inhibitors that reduce the breakdown of levodopa and prolong the treatment effects of L-dopa are also used. Possible side-effects of these preparations are related to the enhanced dopaminergic activity with hypokinesia, postural hypotension and psychological side-effects.

Effects of physical activity

Due to motor impairment with hypokinesia, patients with Parkinson's disease often demonstrate muscular inactivity, which leads to a reduced working capacity. Increased physical activity leads to improved muscle function and other beneficial effects on the general state of health. In particular, there are no negative effects as a consequence of physical inactivity.

The *objectives of the physiotherapy* are to maintain and improve mobility in the torso and extremities, counteract sluggishness, improve respiration and coordination and reduce rigidity and speech impediments. In the more advanced stages of the disease, it is of utmost importance to counteract contractures (2). Fitness walking and initiation of walking are important parts of the training. A number of studies have shown that patients' ability to walk improves with physiotherapy as does their stride and walking velocity (9, 10).

Only a handful of studies have looked into the physical capacity of patients with Parkinson's disease. Patients with mild to moderate Parkinson's disease usually have a normal maximal oxygen uptake, sub-maximal heart rate and working capacity (11, 12). One of the studies carried out shows that it is possible to maintain the aerobic metabolism for a longer period of time with L-dopa treatment with a constant workload and without the formation of lactic acid. It is also an indication that treatment with Levodopa results in an increased energy efficiency during muscular work (13). Patients with Parkinson's disease also display changes in the frequency modulation of the motor units in connection with the initiation of muscle contraction (14). Motor units are normally recruited when the contraction strength increases. L-dopa treatment has been shown to facilitate the recruitment of motor units and improve frequency modulation.

In 2006, Crizzle and Newhouse (15) summarised the results of earlier studies of Parkinson's disease. They came to the conclusion that physical capacity and ADL functions (activities of daily living) benefited from physical exercise. In recent years, a growing number of studies have shown that various forms of exercise have a positive effect on the symptoms of Parkinson's disease as well as muscle function (16–24).

According to Sunvisson and colleagues (24), the motor efficiency and simultaneous capacity of patients with Parkinson's disease improved after a week of walking 4 kilometres every day in a mountainous region. A comparison between physical training and 'normal' physiotherapy showed that patients performing lower extremity exercises appeared to benefit from improved walking and ADL functions (19). Individuals with mild to moderate Parkinson's disease were also found to benefit from intensive physical training with improved motor ability and increased muscle strength, flexibility and

coordination (21). In addition, the exercise lifted the spirits, gave enhanced feelings of well-being and somewhat unexpectedly improved the ability to manage dyskinesia (21). Another study compared the effects of aerobic fitness training and Qigong with the result that aerobic fitness training gives a greater improvement in motor function (25). However, Qigong has been found to stabilize motor symptoms as well as symptoms of autonomic dysfunction (26).

According to a retrospective interview survey making comparisons between a control group and Parkinson's disease sufferers, the level of participation in sports activities is very similar until the first onset of symptoms. A striking reduction in physical exercise was noted thereafter, although it never stopped completely. The most popular activities were swimming, aerobic fitness training and nature walks, but it seemed impossible for patients with Parkinson's disease to learn new sports (27).

Indications

Physical activity is always recommended in connection with Parkinson's disease. Physical activity combined with physiotherapy should be begun in the early stages of the disease. Physical activities do not affect the progress of the disease, but improve motor skills and ADL functions and lead to a better general state of health. It is evident from a number of scientific studies that work capacity as well as other functions benefit from fitness training and certain endurance training.

Prescription

1. Daily physical activity such as trekking, walking and the like.
2. Physiotherapy once a week with a specialist physiotherapist to improve the ability to walk among other things. Self-training/home exercise programmes should be drawn up and implemented 2–3 times a week.
3. Fitness and endurance training is recommended in some cases.

Functional tests/need for health checks

Because patients with Parkinson's disease are usually of a mature age, cardiac and pulmonary functions should be assessed before any physical activity takes place in addition to general activity and the patient starts physiotherapy.

Interactions with medical treatment

Physical activity is reported to both increase and decrease the absorption of levodopa (28). Yet, the result of another study showed no change in the plasma level, nor any variation in the effects of increased work intensity (29). Consequently, there is no reason to assume that there is any interaction between physical activity and medical treatment.

Contraindications

There are no contraindications for physical activity in general or physiotherapy treatment. There may be cardiac and pulmonary contraindications for strength training. A recently published study showed that half of the Parkinson's sufferers participating in the study had a pathologic cardiovascular reaction when subjected to a maximal load (30). If reproduced, this discovery would perhaps allow for an identification of patients without an increased risk of a cardiovascular side-effect as a consequence of physical activity.

Risks

There is an increased risk of falling, especially among patients in the later stages of the disease who may also suffer with orthostatic hypertension and physical impairment. Special consideration should be given to patients suffering from hallucinations and other similar disorders.

References

1. Aquilonius S-M. Rörelsestörningar. I: [Movement disorders] Aquilonius S-M, Fagius J, red. Neurologi. 4 uppl. [Neurology 4 issue] Stockholm: Liber AB; 2006. p. 258-76.
2. Lexell J. Multipel skleros och Parkinsons sjukdom. [Multiple sclerosis and Parkinson's disease] I: Borg J, Gerdle B, Grimby G, Stibrant-Sunnerhagen K, red. Rehabiliteringsmedicin. Teori och Praktik. [Rehabilitation medicine. Theory and Practice] Lund: Studentlitteratur; 2006. p. 288-95.
3. Midlöv P, Eriksson T, Petersson J. Parkinsons sjukdom. [Parkinson's disease] I: Läkemedelsboken 2007/2008. Stockholm: Apoteket AB; 2007. p. 786-95.
4. Valente EM, Bentivoglio AR, Dixon PH, Ferraris A, Ialongo T, Frontali M, et al. Localization of a novel locus for autosomal recessive early-onset parkinsonism, PARK6, on human chromosome 1p35-p36. *Am J Hum Genet* 2001;68:895-900.
5. Jenner P. Parkinson's disease, pesticides and mitochondrial dysfunction. *Trends Neurosci* 2001;24:245-7.
6. Kirkey KL, Johnson CC, Rybicki BA, Peterson EL, Kortsha GX, Gorell JM. Occupational categories at risk for Parkinson's disease. *Am J Ind Med* 2001;39:564-71.
7. Chen H, Zhang SM, Schwarzschild MA, Hernan MA, Ascherio A. Physical activity and the risk of Parkinson disease. *Neurology* 2005;64:664-9.
8. Sasco AJ, Paffenbarger Jr RS, Gendre I, Wing AL. The role of physical exercise in the occurrence of Parkinson's disease. *Arch Neurol* 1992;49:360-5.
9. Baatile J, Langbein WE, Weaver F, Maloney C, Jost MB. Effect of exercise on perceived quality of life of individuals with Parkinson's disease. *J Rehabil Res Dev* 2000;37:529-34.
10. de Goede CJ, Keus SH, Kwakkel G, Wagenaar RC. The effects of physical therapy in Parkinson's disease. A research synthesis. *Arch Phys Med Rehabil* 2001;82:509-15.
11. Bergen JL, Toole T, Elliott III RG, Wallace B, Robinson K, Maitland CG. Aerobic exercise intervention improves aerobic capacity and movement initiation in Parkinson's disease patients. *NeuroRehabilitation* 2002;17:161-8.
12. Canning CG, Alison JA, Allen NE, Groeller H. Parkinson's disease. An investigation of exercise capacity, respiratory function, and gait. *Arch Phys Med Rehabil* 1997;78:199-207.
13. LeWitt PA, Bharucha A, Chitrit I, Takis C, Patil S, Schork MA, et al. Perceived exertion and muscle efficiency in Parkinson's disease. L-DOPA effects. *Clin Neuropharmacol* 1994;17:454-9.
14. Petajan JH, Jarcho LW. Motor unit control in Parkinson's disease and the influence of levodopa. *Neurology* 1975;25:866-9.
15. Crizzle AM, Newhouse IJ. Is physical exercise beneficial for persons with Parkinson's disease? *Clin J Sport Med* 2006;16:422-5.
16. Dibble LE, Hale T, Marcus RL, Gerber JP, Lastayo PC. The safety and feasibility of high-force eccentric resistance exercise in persons with Parkinson's disease. *Arch Phys Med Rehabil* 2006;87:1280-2.

17. Dibble LE, Hale TF, Marcus RL, Droge J, Gerber JP, LaStayo PC. High-intensity resistance training amplifies muscle hypertrophy and functional gains in persons with Parkinson's disease. *Mov Disord* 2006;21:1444-52.
18. Hirsch MA, Toole T, Maitland CG, Rider RA. The effects of balance training and high-intensity resistance training on persons with idiopathic Parkinson's disease. *Arch Phys Med Rehabil* 2003;84:1109-17.
19. Miyai I, Fujimoto Y, Ueda Y, Yamamoto H, Nozaki S, Saito T, et al. Treadmill training with body weight support. Its effect on Parkinson's disease. *Arch Phys Med Rehabil* 2000;81:849-52.
20. Protas EJ, Mitchell K, Williams A, Qureshy H, Caroline K, Lai EC. Gait and step training to reduce falls in Parkinson's disease. *NeuroRehabilitation* 2005;20:183-90.
21. Reuter I, Engelhardt M, Stecker K, Baas H. Therapeutic value of exercise training in Parkinson's disease. *Med Sci Sports Exerc* 1999;31:1544-9.
22. Scandalis TA, Bosak A, Berliner JC, Helman LL, Wells MR. Resistance training and gait function in patients with Parkinson's disease. *Am J Phys Med Rehabil* 2001;80:38-43, quiz 4-6.
23. Schenkman M, Cutson TM, Kuchibhatla M, Chandler J, Pieper CF, Ray L, et al. Exercise to improve spinal flexibility and function for people with Parkinson's disease. A randomized controlled trial. *J Am Geriatr Soc* 1998;46:1207-16.
24. Sunvisson H, Lökk J, Ericson K, Winblad B, Ekman SL. Changes in motor performance in persons with Parkinson's disease after exercise in a mountain area. *J Neurosci Nurs* 1997;29:255-60.
25. Burini D, Farabollini B, Iacucci S, Rimatori C, Riccardi G, Capecci M, et al. A randomised controlled cross-over trial of aerobic training versus Qigong in advanced Parkinson's disease. *Eura Medicophys* 2006;42:231-8.
26. Schmitz-Hubsch T, Pyfer D, Kielwein K, Fimmers R, Klockgether T, Wullner U. Qigong exercise for the symptoms of Parkinson's disease. A randomized, controlled pilot study. *Mov Disord* 2006;21:543-8.
27. Fertl E, Doppelbauer A, Auff E. Physical activity and sports in patients suffering from Parkinson's disease in comparison with healthy seniors. *J Neural Transm Park Dis Dement Sect* 1993;5:157-61.
28. Carter JH, Nutt JG, Woodward WR. The effect of exercise on levodopa absorption. *Neurology* 1992;42:2042-5.
29. Mouradian MM, Juncos JL, Serrati C, Fabbrini G, Palmeri S, Chase TN. Exercise and the antiparkinsonian response to levodopa. *Clin Neuropharmacol* 1987;10:351-5.
30. Werner WG, DiFrancisco-Donoghue J, Lamberg EM. Cardiovascular response to treadmill testing in Parkinson's disease. *J Neurol Phys Ther* 2006;30:68-73.

41. *Peripheral arterial disease*

Authors

David Bergqvist, MD, PhD, Professor, Department of Surgery, Uppsala University Hospital, Uppsala, Sweden

Agneta Ståhle, PT, PhD, Associate Professor, Department of Neurobiology, Care Sciences and Society, Division of Physiotherapy, Karolinska Institutet, Stockholm, Sweden

Summary

Peripheral arterial disease (intermittent claudication) is typically age-dependent and is not uncommon after the age of about 65 years. Exercise training in peripheral arterial disease leads to an increased walking distance and reduced pain, and likely also a slowed progression of the disease process itself. For optimal effect, the exercise should be carried out as walk training, preferably intermittently, a minimum of 3 times per week, 30 minutes per session, and over a period of at least 6 months. The biggest effect is achieved through supervised training programmes. Suitable activities include brisk walks and Nordic walking.

Definition

Peripheral arterial disease (intermittent claudication) is typically age-dependent and is not uncommon after the age of about 65 years. Claudication refers to pain in the muscles of the legs upon exertion, such as walking, where symptoms disappear after a short rest, usually a few minutes. The prevalence, that is, the frequency in the population, can be estimated at 1.5 per cent before the age of 50 years, increasing to over 10 per cent in people over 65 years. If the symptoms become worse, that is, the patient experiences pain at rest, persistent sores and/or gangrene, one generally speaks of critical ischaemia (insufficient blood supply to the tissues). Progression to critical ischaemia is not particularly common in intermittent claudication, however, but the risk increases in patients who smoke or have diabetes. The problem is due to narrowing or complete blockage of the arteries that supply the muscles, resulting in impaired perfusion (blood supply).

Various studies have determined the risk factors for intermittent claudication in patients with no prior disease as well as progression to more severe ischaemia of the extremities in

patients with claudication. These risk factors include age, male gender, smoking, diabetes, hypertension, high blood lipid levels and high fibrinogen. A patient with claudication usually seeks medical attention for impaired quality of life due to shorter walking distance, and one problem with treatment is that reduction of the noted risk factors, some of which can be influenced, does not have an immediate effect on the patient's symptoms. An effect may instead be seen in the longer perspective, and then also in the form of improved survival. In that the number of elderly persons in the population is increasing and smoking habits have not changed radically, we can anticipate an increased prevalence of the disease in coming decades. The treatment for claudication is usually conservative, whereas critical ischaemia often requires surgery, which can be performed using different methods.

Effects of physical activity

Physical exercise to improve walking distance is a treatment method where experience suggests a beneficial effect on the symptoms. "Stop smoking and keep walking" is the five-word phrase coined by Scottish internal medicine specialist Housley (1) to describe the treatment of claudication. The aim of this chapter is to discuss in more detail the potential effects of exercise and the scientific bases that suggest that an effect actually does exist.

As early as 1898, Erb (2) from Heidelberg gave a detailed description of intermittent claudication, also pointing out the importance of exercise as a treatment, but it was not until the 1950s that the idea was raised again more systematically (3). The effect of exercise training in connection with peripheral vascular disease is well-documented in meta-analyses and review studies (4–7).

An important factor to consider when evaluating treatment therapies for ischaemia of the extremities is whether there is a correlation between walking distance and the patient's subjective well-being, measured as quality of life. Several studies show that this is the case (8, 9). Walking distance must be measured with objective methods because both the patient's and physician's perception of walking distance can be surprisingly inaccurate (10).

There are several possible explanations as to why exercise may have an effect on intermittent claudication:

- **Increase in blood flow.** The discussion of this possible explanation has been lively, and the general opinion today is that good effects can be achieved from exercise without increased blood flow. The increased flow may be a small part of the explanation, but most studies have not shown any effect (11, 12).
- **Increased collateral development** has been considered but is increasingly questioned due to the absence of effect on blood flow and pressure on the ankle (see above). In any case, there is a poor correlation between possible increase in flow and change in walking distance (13).
- **Effects on muscle metabolism.** Exercise gives rise to various structural and functional changes in the muscles, such as a slower metabolism of glycogen, increased oxidation of fatty acids, high levels of oxidative enzymes, and an increase in the number of

mitochondria per volume unit. The increase in walking distance after exercise training correlates to a reduction in acyl-carnitine plasma levels, which reflect metabolic dysfunction.

- **Improved cardiopulmonary function** is due, among other things, to increased oxygen utilisation after exercise and a reduced heart rate.
- **Psychological effect**. Exercise increases overall well-being. According to Rosfors and colleagues (14), the most important factor in predicting a positive effect from a training programme is the patient's expectation that it will have an effect. An effect via the endorphin system has been suggested, though this has not been proven.
- **Increased muscle strength.**
- **Change in gait pattern.**
- **Change in perception of pain.**

There has been discussion of whether exercise can have an adverse effect due to an inflammatory response during the walking or possibly during the resting phase as a partial phenomenon in a reperfusion syndrome (15, 16). A more general harmful effect of exercise has been suggested to be reflected in the form of microalbuminuria (17). However, it has not been proven that exercise leads to any harmful clinical effects, but rather the inflammatory response is reduced with increased exercise (18). Treadmill training does not increase plasma markers that indicate endothelial injury (19).

Prescription

The recommendation of the Trans-Atlantic Inter-Society for Management of Peripheral Arterial Disease (TASC) for exercise therapy for intermittent claudication states that:

- Supervised exercise should be made available as part of the initial treatment for all patients with peripheral arterial disease.
- The most effective programmes employ intermittent walk therapy that is of sufficient intensity to bring on claudication, followed by rest, for 30–60 minutes, 3 times per week for at least 3 months (20)

A good summary of the effect of exercise training can be found in a meta-analysis by Gardener and Poehlman (4). In a final analysis of 21 studies, which met the required inclusion criteria, six components of the exercise programme were registered for evaluation:

1. Exercise frequency (per week).
2. Duration of exercise (minutes per session).
3. Mode of exercise (walking or combined exercise).
4. Total duration of programme (weeks).
5. Pain used as the endpoint (initial pain or maximal walking distance).
6. Level of supervision (supervised systematic exercise or exercising on one's own in the home).

Overall, the walking range increased significantly from the exercise programmes. The distance to onset of pain increased by 179 per cent or 225 metres, and the maximal walking distance increased by 122 per cent or 397 metres. These can be considered clinically significant improvements. Factors of the exercise programme that were of significant value in the increase of walking distance indicated an exercise frequency of 3 or more times per week, a duration of more than 30 minutes, a total programme length of more than 6 months, and only walking as opposed to combined exercise. The level of supervision seemed to be of less importance, but for many patients exercising in a group motivates them more to continue than exercising alone. Randomised studies suggest the importance of proper supervision in the exercise training (9). The clinical effect of exercise is impacted positively if the patient also quits smoking (19). The optimal exercise programme remains, however, to be defined (5).

Exercise programmes also have a generally beneficial effect on cardiovascular risk factors (20–22) and cardiorespiratory function (11). Exercise also improves the quality of life (23).

In conclusion, exercise training for intermittent claudication leads to an increase in walking distance, with higher quality of life and reduced pain, and probably also slowed progression of the arteriosclerotic disease process. It would appear that the biggest effect is achieved with supervised exercise programmes of 3–6 months. A recently published report from the Swedish Council on Technology Assessment in Health Care (SBU) concludes that, in patients with peripheral arterial disease, supervised exercise therapy leads to increased physical activity, measured as walking distance and/or walking time. The best results are achieved if the exercise is supervised to begin with, is conducted in 30–60-minute sessions 3 or more times per week, at an intensity close to the pain threshold, and lasts for at least 6 months (level of evidence: 3) (7).

References

1. Housley E. Treating claudication in five words. *Br Med J (Clin Res Ed)* 1988;296:1483-4.
2. Erb W. Über das “intermittierende Hinken” und andere nervöse Störungen in Folge von Gefässerkrankungen [On “intermittent claudication” and other nervous system disorders resulting from vascular diseases]. *Deutsch Zschr Nervenheilk* 1898;13:1-76.
3. Foley W. Treatment of gangrene of the feet and legs by walking. *Circulation* 1957;15:689-700.
4. Gardner AW, Poehlman ET. Exercise rehabilitation programs for the treatment of claudication pain. A meta-analysis. *JAMA* 1995;274:975-80.
5. Robeer GG, Brandsma JW, van den Heuvel SP, Smit B, Oostendorp RA, Wittens CH. Exercise therapy for intermittent claudication. A review of the quality of randomised clinical trials and evaluation of predictive factors. *Eur J Vasc Endovasc Surg* 1998;15:36-43.
6. Wannamethee SG, Shaper AG. Physical activity in the prevention of cardiovascular disease. An epidemiological perspective. *Sports Med* 2001;31:101-14.
7. SBU (Swedish Council on Technology Assessment in Health Care). Metoder för att främja fysisk aktivitet. En systematisk litteraturoversikt [Methods for promoting physical activity. A systematic literature review]. SBU Report no. 181/2007. Stockholm: SBU; 2007.
8. Barletta G, Perna S, Sabba C, Catalano A, O’Boyle C, Brevetti G. Quality of life in patients with intermittent claudication. Relationship with laboratory exercise performance. *Vasc Med* 1996;1:3-7.
9. Regensteiner JG, Steiner JF, Hiatt WR. Exercise training improves functional status in patients with peripheral arterial disease. *J Vasc Surg* 1996;23:104-15.
10. Watson CJ, Collin J. Estimates of distance by claudicants and vascular surgeons are inherently unreliable [see comments]. *Eur J Vasc Endovasc Surg* 1998;16:429-30.
11. Tan KH, Cotterrell D, Sykes K, Sissons GR, de Cossart L, Edwards PR. Exercise training for claudicants. Changes in blood flow, cardiorespiratory status, metabolic functions, blood rheology and lipid profile. *Eur J Vasc Endovasc Surg* 2000;20:72-8.
12. Tan KH, De Cossart L, Edwards PR. Exercise training and peripheral vascular disease. *Br J Surg* 2000;87:553-62.
13. Hiatt WR, Regensteiner JG, Hargarten ME, Wolfel EE, Brass EP. Benefit of exercise conditioning for patients with peripheral arterial disease. *Circulation* 1990;81:602-9.
14. Rosfors S, Arnetz BB, Bygdeman S, Skoldo L, Lahnborg G, Eneroth P. Important predictors of the outcome of physical training in patients with intermittent claudication. *Scand J Rehabil Med* 1990;22:135-7.
15. Walker PM. Ischemia/reperfusion injury in skeletal muscle. *Ann Vasc Surg* 1991;5:399-402.
16. Nawaz S, Walker RD, Wilkinson CH, Saxton JM, Pockley AG, Wood RF. The inflammatory response to upper and lower limb exercise and the effects of exercise training in patients with claudication. *J Vasc Surg* 2001;33:392-9.

17. Hickey NC, Shearman CP, Gosling P, Simms MH. Assessment of intermittent claudication by quantitation of exercise-induced microalbuminuria. *Eur J Vasc Surg* 1990;4:603-6.
18. Turton EP, Coughlin PA, Kester RC, Scott DJ. Exercise training reduces the acute inflammatory response associated with claudication. *Eur J Vasc Endovasc Surg* 2002;23:309-16.
19. Woodburn KR, Rumley A, Murtagh A, Lowe GD. Acute exercise and markers of endothelial injury in peripheral arterial disease [see comments]. *Eur J Vasc Endovasc Surg* 1997;14:140-2.
20. Norgren L, Hiatt WR, Dormandy JA, Nehler MR, Harris KA, Fowkes FG, Rutherford RB; TASC II Working Group. Inter-society consensus for the management of peripheral arterial disease. *Eur J Vasc Endovasc Surg* 2007;33:S1-75.
21. Naslund GK, Fredrikson M, Hellenius ML, de Faire U. Effect of diet and physical exercise intervention programmes on coronary heart disease risk in smoking and non-smoking men in Sweden. *J Epidemiol Community Health* 1996;50:131-6.
22. Hellenius ML, de Faire U, Berglund B, Hamsten A, Krakau I. Diet and exercise are equally effective in reducing risk for cardiovascular disease. Results of a randomized controlled study in men with slightly to moderately raised cardiovascular risk factors. *Atherosclerosis* 1993;103:81-91.
23. Gartenmann Ch, Kirchberger I, Herzig M, Baumgartner I, Saner H, Mahler F, Meyer K. Effects of exercise training program on functional capacity and quality of life in patients with peripheral arterial occlusive disease. Evaluation of a pilot project. *Vasa* 2002;31:29-34.

42. Post-polio

Authors

Carin Willén, PT, PhD, Department of Occupational Therapy and Physiotherapy, Department of Neuroscience and Physiology, Sahlgrenska Academy, Gothenburg University, Gothenburg, Sweden

Gunnar Grimby, MD, PhD, Professor Emeritus, Department of Clinical Neuroscience and Rehabilitation, Institute of Neuroscience and Physiology, Sahlgrenska Academy, Gothenburg University, Gothenburg, Sweden

Summary

The most common post-polio symptoms include reduced muscle strength, a longer period of recovery for previously affected muscles, general fatigue and painful joints and muscles. This has brought about a new concept called the post-polio syndrome where new or aggravated muscle weakness is present in addition to other symptoms. Possible consequences of these symptoms are limited daily activities at work or in leisure time with the added risk of inactivity and general health-related problems. There is also a risk of overloading because of too much activity. Consequently, a suitable individual level of activity and a carefully monitored exercise programme are recommended.

Examples of suitable forms of exercise are water gymnastics and Nordic pole walking. Traditional fitness and strength training could be recommended for some individuals. Exercise will provide added strength, improved aerobic fitness and reduced pain.

Polio and its residual effects

Causes and prevalence

Polio or poliomyelitis is a viral disease marked by a gastroenteric inflammation which, in a small number of people, leads to an inflammation in the spinal cord and sometimes also in the motor neurons of the brain stem. As a consequence, paralysis occurs to a lesser or greater extent. Whether immunologic changes occur in the later stages of the disease is currently the subject of numerous studies and discussions. Actively administered vaccination programmes have led to polio being practically eradicated throughout the world,

although a limited number of outbreaks have occurred in recent decades, including Europe. However, polio is still present in some parts of Africa and Asia.

It has been known for more than 100 years that individuals who have recovered from acute polio may experience aggravated or additional symptoms following a relatively stable period of time, sometimes several decades after the initial onset.

Yet, the concept of developing strategies for clinical assessment and treatment only gathered serious momentum around 20 years ago (1). The industrialised countries have a significant number of people with post-polio residual paralysis. In Scandinavia, the last epidemic occurred in the first half of the 1950s. Asia, Africa and South America have seen more recent cases of polio. As a consequence, younger people with post-polio syndrome have arrived in the Nordic countries. Even if not all polio patients suffer from additional or aggravated problems – around 80 percent as indicated by clinical evidence and around 50 percent as indicated by population-based evidence – most of them seek medical care. In Sweden, around 10,000–15,000 people are estimated to have been diagnosed with post-polio syndrome. The prevalence of post-polio syndrome is probably even higher than these figures suggest.

Symptoms and underlying mechanisms

The additional problems are of a varying nature (2). Many are associated with an increase in muscular weakness, which is one of the most common post-polio symptoms. The added weakness may affect muscles previously affected by the disease, as well as those muscles in which no earlier weakness has been perceived. Increased muscular fatigue and difficulty in regaining muscular strength following muscular exertion are not unusual. In the event of muscular over-exertion, the individual may experience muscle pain during or after the exertion.

If the respiratory muscles have been affected by polio, causing breathing problems, the respiratory problems may be further accentuated by additional ventilatory problems. A state of underventilation may arise or be aggravated, mainly at night. Although this is a fairly unusual problem, it does lead to anxiety before a correct diagnosis can be made and will have an effect on the individual's general functioning.

Another recurrent problem is the general fatigue that many individuals experience in addition to muscular fatigue. This is usually due to a lower level of fitness, i.e. a reduced circulatory capacity because the patient is out of shape, but also because of an inappropriate degree of exertion, not enough rest periods or the training being of too long duration.

Another type of problem is associated with overloading and consequential instability in joints surrounded by weakened muscles. This problem is not necessarily directly linked to an additional or increased weakness of the muscles. Symptoms due to overloading can also occur without the muscular structure being significantly weakened, but where the load on the “better” side of the body is greater. Other problems that are perhaps a little harder to explain include reduced sensitivity and enhanced intolerance to cold. Psychological symptoms such as apprehension, anxiety, depression, irritability and concentration difficulties are also frequently reported.

The different organ symptoms and general fatigue, the onset of which may occur very quickly, may also restrict daily life activities as well as both work and leisure time. However, a relatively high proportion of post-polio patients in an active age continue to work (3). The problems in managing work are, however, accentuated with the onset of additional symptoms at which point more than half of those in working life report difficulties at work. Many with post-polio symptoms find it difficult to carry on with their normal leisure-time activities and the majority find alternative activities (3). In spite of this, many of these individuals are satisfied with their leisure time activities and have consequently adapted well to a reduced function.

The term post-polio syndrome (PPS) was created at the beginning of the 1980s when a more systematic assessment and recording of additional symptoms began (1). The criteria for diagnosing PPS have since changed somewhat according to the literature and additional or aggravated muscle weakness is no longer a common symptom, but a mandatory criterion. According to Gawne and Halstead (2), the criteria for the post-polio syndrome (PPS) are:

1. A confirmed history of paralytic polio including neurogenic changes as indicated by electromyography scan (EMG).
2. A stable period of at least 20 years following the initial recovery.
3. Additional or aggravated muscle weakness in muscles affected by polio, often with additional symptoms.
4. No other medical reasons for additional symptoms.

Specific muscular changes have specific names such as post-polio muscle atrophy (PPMA) and post-polio muscular dysfunction (4). Not all persons with residual polio symptoms are diagnosed with the post-polio syndrome since the diagnosis requires additional symptoms with increased muscular weakness. Nor is the post-polio syndrome included in the ICD-10 diagnostic criteria. However, the diagnosis code B91 “Sequelae of poliomyelitis” is included.

Muscular function

In case of an anterior horn nerve cell loss as seen in poliomyelitis, compensatory mechanisms in the form of collateral innervation (sprouting) are activated. A reinnervation of denervated muscle fibres occurs through the regrowth of sinuvertebral nerve endings from surviving motor axons. As a result, the remaining motor units will contain a significantly increased amount of muscle fibres. Collateral innervation is an important mechanism for the improvement of muscle function in the early stages of polio. However, denervation and subsequent reinnervation has been noted in some patients several decades after the onset of polio (5). This is interpreted as a loss of anterior horn cells or certain motor units losing a part of the collaterally innervated muscle fibres. These are then taken over by other expanding motor units. There are many different theories as to the cause of the anterior horn nerve cell loss such as aging or a shorter life span owing to over-utilisation or partial nerve damage as a consequence of polio.

The motor unit of the muscles affected by polio is enlarged up to 11 times in size which together with a double transverse surface of individual muscle fibres amount to more than five times the muscle fibres in a normal motor unit. Very large motor units constitute a risk factor for additional weakness (6). Some retrospective studies indicate that patients with an initially severe paralysis that is followed by a stable phase of considerable improvement, reasonably good function and level of activity are at a greater risk of a late onset of aggravated or additional muscle weakness (7).

Another essential compensation for loss of motor units is the growth (hypertrophy) of the remaining muscle fibres. As mentioned above, an approximate doubling is measurable. However, the degree of muscle fibre hypertrophy varies significantly and is probably dependent on the relative load experienced by the muscle in question. An increase in both type 1 and 2 muscle fibre areas may occur. Conversely, no significant compensatory increase in muscle fibres is noticeable in patients with near to normal muscle strength. A long period of “muscle over-utilisation”, as seen in the tibialis anterior muscle when walking, can lead to an increase in the proportion of type 1 muscle fibres (8) as a consequence of fibre type transformation.

The two compensatory mechanisms, i.e. reinnervation and muscle fibre hypertrophy, allows for muscles with a considerable loss of motor units to sustain normal or near to normal muscle strength. Neuromuscular transmission disorders are thought to be one of the reasons for muscle weakness and increased muscular fatigue (9), but are unlikely to be the only explanation for muscle weakness.

A number of mechanisms could cause the muscular fatigue and lack of muscle endurance experienced by patients. Another likely reason is that patients with post-polio syndrome and an increased muscle weakness continue with the same absolute muscle activation despite a lower maximal strength. As a result, the relative load increases, resulting in intensified and sudden tiredness.

Another possible reason for muscular fatigue is an inefficient restitution following muscle activity (10, 11).

Joint structure and pain

Orthopaedic problems in general are fairly common in individuals with post-polio symptoms. Those that walk often report problems with their lower extremities while those using a wheelchair or crutches often report problems with their upper extremities due to the load placed on their shoulders and/or wrists, respectively. The patients that seem to be suffering most from muscle and joint pain have a moderately reduced function and comparatively high level of activity (12). There is a substantial need for orthoses and other types of orthopaedic aids, particularly orthopaedic shoes and inserts.

Principles of assessment and treatment

It is important to distinguish between late effects of polio or other more nonspecific symptoms of weakness or pain. Other simultaneous diseases with similar symptoms occur fairly frequently in post-polio individuals (13). A clinical examination should be carried out by a medical consultant experienced in treating post-polio patients, preferably at a specialist clinic.

Apart from identifying symptoms and reduced functions, the examination should incorporate an EMG scan to verify the extent of the polio disease. The individual's vital capacity is also measured to exclude or confirm the presence of polio effects on respiratory muscles.

No specific pharmacological treatment for muscular weakness and fatigue was previously available. However, in the past few years, clinical studies have been able to identify an inflammatory reaction in the central nervous system in post-polio patients – not a renewed progression of the polio infection. Therefore, trial treatments with intravenous immune globulin have been carried out resulting in improved muscle strength, an enhanced feeling of well-being, reduced pain and increased physical activity (14). However, this type of treatment is still experimental and only available under the supervision of a specialist clinic. It is imperative that pain caused by a relatively high level of activity, specific loading on unstable joints or biomechanical conditions are alleviated. The patient should be given guidance on the appropriate level of activity and the use of mobility aids, and orthotic devices should be prescribed and adjusted. Pain-relieving measures may be necessary in the form of antiflogistic and analgetic preparations combined with heat treatment and/or transcutaneous stimulation of the nerves or acupuncture.

Effects of physical activity

As is evident from the above, muscle fibre hypertrophy is the consequence of a daily and relatively heavy load on specific muscles. Hence, there is a significant “spontaneous” adaptation to the demands placed on an individual's physical activity. Priority should be given to the development of strength rather than endurance (17). The anticipated effect of strength training is an improved neural activation. However, initially large muscle fibres are unlikely to expand any further. Because the muscles in an extremity may be subject to a varying degree of strength reduction, the weakest muscles could be restricting the activity while the “stronger” muscles become relatively inactive. The effects of physical exercise on such muscles will then be similar to those on inactive muscles unaffected by polio. There have been numerous discussions concerning the damaging effects of too much physical activity. It is not unlikely that an inappropriate activity intensity and duration may lead to increased weakness and fatigue that will last for several days. However, if discovered early, such possible “overactivity” should be reversible and an adjustment of the physical activity and exercise should be made.

The level of aerobic fitness is often reduced and worsened by reduced muscle strength, pain and inactivity. If possible, find a mode of physical activity where the muscle weakness is less restrictive than the circulatory capacity. Aerobic fitness will most likely improve

together with the endurance capacity of the muscles. The initial effect of physical activity is almost certainly peripheral with an improved muscle adaptation to aerobic exercise and enhanced efficiency. However, it is important to modify the exercise and physical activity programme to suit the individual in question.

Indications

Physical activity and exercise are only used for the purpose of secondary prevention. However, it is still not known whether a modified physical training of post-polio individuals prevents the development of new symptoms.

The symptoms of post-polio confers an increased risk of inactivity with the development of health problems. It is crucial to inform patients that inactivity can lead to aggravated symptoms such as increased weakness, pain and fatigue in addition to other conditions such diabetes, cardiovascular disease, osteoporosis and obesity. Hence, great importance should be given to promote physical exercise in the prevention of such conditions. It is also recommended to adapt the physical activity with the aim of sustaining and improving biomechanical conditions as well as maintaining the optimal level of aerobic fitness.

Prescription

Strength and muscular endurance training

Because residual effects of polio have a great symptomatic variation, particularly regarding the degree of reduced muscle function, it is important that physical exercise programmes are adapted to the individual patient. Clinical and functional targets should be clearly specified. The exercise programme should not just include the weakest muscles or muscles groups. Try different methods of optimal muscle training.

According to the documentation available, it is possible to exercise and strengthen moderately weakened and polio-affected muscles measuring > 3 on a scale of 0–5 (15). A number of studies indicate that weight training increases the strength of individual muscles. The load of the body itself, together with low-intensity training, has proven to benefit muscular function (16).

Exercise programmes should also include intervals of endurance training as spontaneous adaptations appear to prioritise strength before endurance (17).

Table 1. Recommendations for physical activity considering polio status and reduced strength (18).

Polio status	Muscle strength	Training
Stable	Normal	Without restrictions
Stable	Reduced	Short period of strength training (4–6 weeks)
Unstable	Reduced	Sub-maximal training
Unstable	Significantly reduced	Low-intensity training
Serious atrophy	Serious atrophy	No training

Stable polio does not refer to a subjective perception of progressive muscle weakness. In contrast, unstable polio does refer to a progressive muscle weakness. In the case of unstable polio, it is essential to determine whether there is over-utilisation or inactivity.

It is possible to exercise respiratory muscles affected by polio. Improved endurance is obtained by exercising the respiratory muscles once a day for a period of 10 weeks using an apparatus that provides different levels of inhalation resistance. Before and after each exercise, the patient uses his/her own ventilator for a minimum period of 30 minutes (19).

General rules: The patient's response acts as a training guideline. Initially, the training should be carefully monitored with shorter than normal training sessions. Several short sessions of training are better than one session of a long duration. A long duration of pain or tiredness (24 hours or more) after training is an indication that the muscle load needs to be reduced. A perceived exertion scale (20) can be used to stop the patient from exceeding a certain level during training.

People affected by polio have a longer post-exercise muscle recovery period than people with a normal muscular system. Consequently, a training frequency of more than twice a week is not recommended.

Eccentric strength training is not recommended as it involves a greater load compared to concentric strength training, which confers an increased risk of overloading.

Aerobic fitness training

Fitness training is possible for patients with sufficient strength in larger muscle groups. The basic principle of applied training involves a load of 60–80 percent of the maximal oxygen uptake/heart rate for a minimum duration of 30 minutes, usually twice a week.

If the larger muscle groups lack sufficient strength, a similar effect can be obtained through peripheral muscle strength training, i.e. low-intensity training with many repetitions.

Recommended forms of exercise

A number of examples of suitable forms of training are provided below with references to available scientific evidence. Table 2 lists documented scientific studies.

Table 2. Forms of exercise with documented benefits.

Form of training	Activity	Intensity	Frequency (times/week)	Length of training period	References
Aerobic fitness training	Ergometer bicycle training/legs	70% of max HR*	3	16 weeks	22
Aerobic fitness training	Ergometer cycling/arms	70–75% of max HR for 20 min.	3	16 weeks	23
Strength training	Dynamometer	Max. isokinetic 12 × 8 sec. Max. isometric 12 × 4 sec.	3	6 weeks	15
Strength training	Weights	10 RM** × 3	Every other day	2 years	24
Strength training	Weights	Dynamically controlled level of exertion gradually increased to extreme exertion	4	12 weeks	25
Combination programme (pneumatic resistance/light aerobic fitness training)	75% of 3 RM × 3	Sub-max.	3 2	10 weeks	26, 16
Water exercises		Sub-max.	2	5 months	21

* Max HR = maximal heart rate.

** RM = Repetition maximum. 1 RM is the maximum amount of weight one can lift in a single repetition for a given exercise.

- **Dynamic water exercise:** Training should take place in a heated swimming pool, preferably in a group session. The unloading of body weight in water reduces the load on muscles and joints. The water gently resists movement with many opportunities to vary and modify the training intensity. Various muscle groups are exercised and the water facilitates individual exercise programmes. Research indicates that sub-maximum exertion alleviates pain and reduces heart rate (21).
- **Using a bicycle ergometer:** Best suited for patients with good muscle strength in the lower extremities. The fitness benefits have been established (22). Outdoor cycling is only recommended for patients with very good muscle function. A good balance is vital and there is an increased risk of falling when mounting and dismounting the bicycle due to a reduced muscle function. Cycling uphill is not recommended.

- **Light aerobic fitness training:** Group exercises incorporating modified movement techniques in sitting or lying positions. According to one study, the result is an improved aerobic capacity and increased strength in specific muscle groups (16).
- **Swimming:** Exercising in a normally heated swimming pool is suitable for patients with good muscle function as well as competent swimmers. There are no documented studies on this, however.
- **Nordic pole walking:** Walking with poles unloads the lower extremities which is advantageous. Nordic pole walking is a safer way of walking, but requires good function of the upper extremities to avoid injuries due to overloading. There are no documented studies on this.
- **Weight lifting:** Provided that the exercises have been adapted and are carefully monitored, there is no reason not to do weight training. This type of training can work well as optional home training. Studies indicate improved strength without any muscle damage (15, 24, 25).

Advice and recommendations on physical activity

Experience has shown that those with post-polio symptoms have a fairly high level of physical activity (12). Consequently, the patient's physical activity routines should be reviewed and discussed when constructing a physical exercise programme. It is not usually necessary to change the daily physical activities carried out, although the degree of intensity may need to be reduced and the activities spread all throughout the day.

Appropriate physical activities should be recommended, also based on the patient's personal objectives. It is a question of the patient saving his/her strength for what is important in life and finding a balance between physical activity and rest. It is also important to recommend mobility aids and energy-saving measures for both work and leisure-time.

Functional mechanisms

Because compensatory mechanisms lead to an enlargement of the muscle fibres in muscles affected by polio, the increased strength gained through physical exercise is believed to be the effect of increased neural activity.

The improvement of aerobic fitness follows the same principles that apply to well-trained healthy individuals. In individuals with a significantly reduced muscle function, the peripheral adaptation of muscles with an increased oxygenation is most important.

Participation in a dynamic water exercise group means regular physical activity of a 40-minute duration which partly explains the pain relief experienced (21). It is also evident that taking part in a physical activity in itself has an alleviating effect on pain during and after the activity.

Functional tests

If deemed necessary, a standard health examination should be carried out in order to exclude cardiovascular disease or any other relevant diseases.

- **Functional tests** for the assessment of muscle function, e.g. various movements and standing up motions from different heights of sitting.
- **Muscle strength test.** A rough assessment of muscle function is possible using a scale from 0 to 5 when measuring muscle weakness (3 or below). A myometer can be used in the clinical settings if a more objective measurement is required. If a dynamometer is available for the measurement of isometric and isokinetic muscle strength, this should be used at the start of exercise and at some point later.
- **30 m walking test** at a self-chosen maximum speed. The duration of the session and the number of steps are recorded. If lower extremity muscle function is significantly reduced and the walking velocity slow, i.e. < 1.5 m per second at maximum speed, there is a risk of the walking velocity quickly slowing down if muscle function is further reduced.
- **Provided that the patient has sufficient muscle function, a standardised maximal or submaximal exercise test** should be carried out together with an electrocardiogram (ECG) if a cardiovascular disease exists.
- **Spirometry** for measuring respiratory function. Respiratory muscles may be affected by polio without the patient being aware of it.
- **Pain diagram and VAS** (Visual Analogue Scale) to identify the type of pain and its extent and intensity. The pain diagram provides a useful indicator of potentially overloaded muscles.
- **Different questionnaires** for the assessment of perceived quality of life and health. No disease-related questionnaire exists, but the Life Satisfaction Scale (27) and Nottingham Health Profile (NHP) (28) are both used. The latter produces a clear physical profile and is therefore suitable for this patient category.

Contraindications and risks

There are no known contraindications to correctly prescribed physical activity. However, it is important to have sufficient knowledge of the propagation and gravity of polio and to be able to recognize clinical indications of muscle overload. Physical activity should never lead to increased or additional pain. There is an insignificant and undocumented risk of a permanently reduced function because of muscle overload. The onset of osteoporosis is possible in which case the risk of falling must be taken into consideration when recommending physical activity. In case of a substantially reduced respiratory capacity, respiratory muscle fatigue may set in following exertion. As a number of patients with residual effects of polio have reached a mature age with the added risk of cardiovascular disease and high blood pressure, the physical exercise programme should be planned taking this into consideration.

References

1. Wiechers DO, Halstead LS. Late effects of poliomyelitis. Part I: Report of five cases. *South Med J* 1985;78:1277-80.
2. Gawne AC, Halstead LS. Post-polio syndrome. Pathophysiology and clinical management. *Crit Rev Phys Rehabil Med* 1995;7:147-88.
3. Thorén-Jönsson A-L, Hedberg M, Grimby G. Distress in everyday life in people with poliomyelitis sequelae. *J Rehab Med* 2001;33:119-27.
4. Borg K. Workshop report. Post-polio muscle dysfunction. 29th ENMC workshop 14–16 October 1994, Narden, Netherlands. *Neuromuscul Disord* 1996;6:75-80.
5. Stålberg E, Grimby G. Dynamic electromyography and biopsy changes in a 4 year follow up. Study of patients with a history of polio. *Muscle Nerve* 1995;18:699-707.
6. Grimby G, Stålberg E, Sandberg A, Sunnerhagen K. An eight year longitudinal study of muscle strength, muscle fiber size and dynamic electromyogram in individuals with late polio. *Muscle Nerve* 1998;21:1428-37.
7. Klingman J, Chui H, Corgiat M, Perry J. Functional recovery. A major risk factor for the development of postpoliomyelitis muscular atrophy. *Arch Neurol* 1988;45:645-7.
8. Borg K, Borg J, Edström L, Grimby L. Effects of excessive use of remaining muscle fibers in prior polio and LV lesion. *Muscle Nerve* 1988;11:1219-30.
9. Trojan D, Gendron D, Cashman N. Anticholinesterase-responsive neuromuscular junction transmission defects in postpoliomyelitis fatigue. *J Nerol Science* 1993;114:170-7.
10. Agre JC, Rodriquez AA. Neuromuscular function. A comparison of symptomatic and asymptomatic polio subjects to control subjects. *Arch Phys Med Rehabil* 1990;71:545-51.
11. Agre JC, Rodriquez AA, Franke TM. Subjective recovery time after exhausting muscular activity in postpolio and control subjects. *Am J Phys Med Rehabil* 1998;77:140-4.
12. Willen C, Grimby G. Pain, physical activity, and disability in individuals with late effects of polio. *Arch Phys Med Rehabil* 1998;79:915-9.
13. Schanke A-K, Stanghelle JK. Fatigue in polio survivors. *Spinal Cord* 2001;39:243-51.
14. Gonzales H, Stibrant Sunnerhagen K, Sjöberg I, Kaponides G, Olson R, Borg K. Intravenous immunoglobulin for post polio syndrome. A randomized controlled trial. *Lancet Neurol* 2006;5:493-500.
15. Einarsson G. Muscle conditioning in late poliomyelitis. *Arch Phys Med Rehabil* 1991;72:11-4.
16. Ernstoff B, Wetterqvist H, Kvist H, Grimby G. The effects of endurance training on individuals with post-poliomyelitis. *Arch Phys Med Rehabil* 1996;77:843-8.
17. Tollbäck A. Neuromuscular compensation and adaptation to loss of lower motoneurons. Dissertation. Stockholm: Karolinska Institute, Department of Clinical Neurosciences; 1995.
18. Grimby G, Stålberg E. Dynamic changes in muscle structure and electrophysiology in late effects of polio with aspects on muscular trainability. *Scand J Rehab Med* 1994;Suppl 30:33-44.

19. Klefbeck B, Lagerstrand L, Mattsson E. Inspiratory muscle training in patients with prio polio who used part time assisted ventilation. *Arch Phys Med Rehabil* 2000;81: 1065-71.
20. Borg GA. Psychosocial bases of perceived exertion. *Med Sci Sports Exerc* 1982;14: 377-81.
21. Willén C, Sunnerhagen KS, Grimby G. Dynamic water exercise in individuals with late poliomyelitis. *Arch Phys Med Rehabil* 2001;82:66-72.
22. Jones DR, Speir J, Canine K, Owen R, Stull A. Cardiorespiratory responses to aerobic training by patients with postpoliomyelitis sequelae. *JAMA* 1989;261:3255-8.
23. Kriz JL, Jones DR, Speir JL, Canine JK, Owen RR, Serfass RC. Cardiorespiratory responses to upper extremity aerobic training by postpolio subjects. *Arch Phys Med Rehabil* 1992;73:49-54.
24. Fillyaw MJ, Badger GJ, Goodwin GD, Bradley WG, Fries TJ, Shukla A. The effects of long-term non-fatiguing resistance exercise in subjects with post-polio syndrome. *Orthopedics* 1991;1:1253-56.
25. Agre JC, Rodriquez AA, Todd FM. Strength, endurance and work capacity after muscle strengthening exercise in postpolio subjects. *Arch Phys Med Rehabil* 1997;78:681-6.
26. Spector SA, Gordon PL, Feuerstein IM, Sivakumar K, Hurley B, Dalakas M. Strength gains without muscle injury after strength training in patients with postpolio muscular atrophy. *Muscle & Nerve* 1996;10:1282-90.
27. Fugl-Meyer AR, Brenholm J-B, Fugl-Meyer K. Om livstillfredsställelse, lycka, rehabilitering. [About life satisfaction, happiness and rehabilitation] *Socialmedicinsk Tidsskrift* 1992;1:33-41. [Journal of Social Medicine]
28. Wiklund I. The Nottingham Health Profile. A measure of health-related quality of life. Review. *Scand J Prim Health Care* 1990;1:15-8.

43. Rheumatoid arthritis

Authors

Christina H. Opava, PT, PhD, Professor, Department of Neurobiology, Care Sciences and Society, Division of Physiotherapy, Karolinska Institutet, Department of Rheumatology, Karolinska University Hospital, Stockholm, Sweden

Ralph Nisell, MD, PhD, Associate Professor, Department of Rheumatology, Karolinska University Hospital, Stockholm, Sweden

Summary

The pathophysiology of rheumatoid arthritis (RA), characterised by reduced joint flexibility, muscle function and fitness together with an increased risk of cardiovascular disease and premature death, provides an indication for physical activity and exercise. There is evidence to suggest that moderate intensity exercise will improve the strength and fitness in patients with RA without increasing the level of pain, disease activity or joint destruction. The joint flexibility should be checked on a regular basis and if needed, the range of motion exercised. The strength and aerobic fitness training recommended to patients with RA is similar to the training recommended to people without RA except that it should be a gradual process where the exercise intensity is adapted to the disease development and specific attention is given to initial indications of pain. Regular physical activity with a moderate intensity improves the quality of life and muscle function in patients with RA. Suitable forms of exercise include cycling, cross-country skiing, walking, Nordic walking, water gymnastics, dancing, light fitness and strength training. Extra caution should be observed in connection with inner organ engagement, cortisone treatment, large joint destruction or joint replacement surgery.

Definition

Prevalence/Incidence

Rheumatoid arthritis affects 0.5–1 percent of the population. Twice as many women as men get rheumatoid arthritis and disease onset can occur at any age, but is most common between the ages of 45–65. The incidence rate is approximately 25–50 new cases per 100,000 residents each year (1).

Cause

The fundamental cause of RA is largely unknown. It is characterised by the defence cells and immune system, which are normally activated to fight off infections and other exterior “assaults”, being activated and remaining activated despite the absence of exterior invaders, such as bacteria, viruses or micro-organisms. This inability to suppress or down-regulate the immune system leads to the body’s own organs and structures being attacked and damaged.

Pathophysiology

Rheumatoid arthritis is a chronic, systemic and inflammatory disease. Common symptoms include periodic onsets of symmetric polyarthritis characterised by inflammation of the synovial joint-lining membranes (synovitis), tendon sheaths (tendovaginitis) and bursae sacks (bursitis). Bone and cartilage destruction is a common occurrence as is a gradual weakening of the bones (osteoporosis) and an increased risk of osteoporosis-related fractures (2, 3). Apart from joint problems, general symptoms of inflammation also exist in, for example, the pericardium and pleura and the blood vessels in skin and inner organs. Secondary amyloidosis is caused by collagen deposits accumulating in one or more organ systems in the body, especially the kidneys, and is a serious adverse complication that may set in after a long period of inflammation. In relation to the general population, persons with rheumatoid arthritis are at an increased risk of cardiovascular disease and premature death (4).

The pain experienced by patients with RA is predominantly of a nociceptive character, i.e. in the skin, muscles, etc. and associated with the inflammatory tissue process. Neurogenic pain is also experienced by patients with RA. This is central or peripheral nerve damage caused by, for example, cervical spine instability, median nerve entrapment or vasculitis (inflammation of blood vessels). Fibromyalgia is another relatively common pain condition in patients with RA causing widespread pain and tenderness throughout the body. *Fatigue* is often due to general symptoms of inflammation, but may also be associated with physical inactivity. The stress experienced by many patients is thought to be caused by the unpredictable nature of the disease and consequential feelings of anxiety, depression and uncertainty (4).

Cartilage and bone destruction is also a consequence of the inflammation having a tendency to attack and destroy adjacent tissues, initially noticeable where the synovial membrane attaches to the bone. *Osteoporosis* is thought to be a combined effect of the actual disease process, physical inactivity and, if applicable, cortisone treatment. *A reduced range of joint* motion accompanies increased joint fluid, thickened joint capsules and changes in load conditions as a consequence of cartilage and bone destruction. The *reduced muscle function* is partly explained by muscle inflammation and partly by changes in the joints. This may lead to extended tendons, ligaments and joint capsules and as a result, joint instability, a reduced muscle mass and strength. The joint swelling will also have a direct impact on the ability of surrounding muscles to contract. The unfavourable position of the joints restrains optimum muscle contraction and the impaired

biomechanical conditions give rise to pain, changes in load conditions and a movement pattern that requires extra energy. As a result, physical activity is further reduced in patients with RA which combined with fatigue and, at times, a direct engagement of the heart and lungs can lead to *reduced aerobic fitness*.

Symptoms

Pain is the cardinal symptom of RA which, although it varies in intensity, localisation and quality, is considered to be chronic. Fatigue and reactions to stress are also common.

According to a Swedish study, a large number of patients with RA exhibit reduced joint flexibility, muscle function and aerobic fitness early in the course of the disease despite adequate medical treatment. A normative comparison of age-gender-matched data showed that 72 per cent had a reduced muscle function while 92 per cent had a reduced grip strength. Around 80 per cent of the women and 50 per cent of the men had a lower than average oxygen uptake capacity. Only around half (53 %) reported to have a sufficient level of physical activity to maintain good health (6).

Diagnostics

The diagnostic criteria for RA have been defined by the American College of Rheumatology (7). At least four of the following seven criteria have to be fulfilled: Arthritis in the small joints of the hand and in at least three joints/joint areas, symmetry, morning stiffness, rheumatic nodules, decalcification of bone on joints or erosions (bone cavities) as shown on X-ray and finally, positive rheumatoid factor.

Because of the large number of individual variations in disease manifestations and consequences, a functional classification system has long been used for rheumatoid arthritis (8). Four classification criteria apply where Class I represents an independent life without any major symptoms, Class II is an independent life despite symptoms of pain, stiffness and reduced physical capacity, Class III is a partly dependent life and finally, Class IV represents complete dependency. Nearly 90 per cent of patients with RA belong to Class I or II.

Prognosis

The varied disease progression makes it difficult to establish individual prognoses. Even if the symptoms of rheumatoid arthritis may subside and fully disappear after a short period of time, the majority develop into chronic symptoms. However, alternating acute flares of the disease and calmer periods of remission usually lead to a gradual deterioration. Occasionally, the clinical picture presents severe, rapidly progressive joint destruction with system engagement. However, modern medical treatments have contributed to substantially improving the prognosis and disease progression in patients with RA (9).

Current treatment principles

Rheumatoid arthritis has traditionally been treated with medication, surgery and various forms of rehabilitation. The treatment is usually administered by doctors, nurses, occupational therapists, social workers and physiotherapists and aimed at alleviating inflammatory activity and other symptoms of the disease, preventing joint damage and potential disability as well as maintaining a good quality of life. However, treatment perceptions and objectives have developed significantly over the past few years with remissions or near remissions now being a realistic target (9).

Pharmaceuticals used for treating RA include cortisone, non-steroid anti-inflammatory drugs (NSAID), slow acting anti-rheumatic drugs (SAARD) or disease-modifying anti-rheumatic drugs (DMARD) in addition to biological treatments such as TNF-alpha blockers and inhibitors, interleukin 1, CD 20+ B-cells and T-cells co-stimulation. Treatment with inflammatory inhibitors should be administered effectively and as early as possible in the course of the disease so as to reduce future joint damage and functional disorders. Cortisone is an anti-inflammatory and effective medicinal product that is administered orally in tablet form or as an injection directly into the inflamed joint or tendon. NSAID is a generic term for a group of anti-inflammatory medicinal drugs that are quick and effective to alleviate pain (within a couple of hours) and used in patients with RA to ease pain and morning stiffness. SAARDs (slow acting anti-rheumatic drugs) such as gold salt, chloroquine phosphate, sulfasalazine and methotrexate affect the actual rheumatic disease progression and have been used for many years. Biological treatments, preferably in combination with methotrexate, have revolutionised the treatment of RA in recent years. Near total remission is obtained in many cases or at least a successful inhibition of the inflammation for the duration of the treatment. This means that the disease and arthritis are no longer clinically perceptible. Modern biological treatment also seems to stop the bone and joint destruction identifiable on x-ray (10).

The most common surgical treatment of RA is arthroplasty, arthrodesis, synovectomy of joints and tendons, nerve decompression and reconstructive tendon surgery. However, the use of surgery in patients with RA has decreased over the past few years as new and successful biological treatments have emerged. This gradual decrease in surgery is expected to continue.

All forms of rehabilitation should be implemented on the basis of team work in close collaboration with the patient, focusing on the goals of the patient. Patient education is a very important aspect of rehabilitation and includes informing the patient about the benefits and prospects of physical activity. Pain relief, trying out various mobility aids, psychological and social support plus various forms of physiotherapy and physical exercise are also essential aspects of rehabilitation.

Effects of physical activity

Acute effects

Patients with rheumatoid arthritis often experience increased levels of pain when commencing physical activity or exercise. This temporary pain increase is seen as a harmless soreness caused by joints and muscles being subjected to an unusual load. The pain is usually transient and is not a hinder to continued activity.

Long-term effects

There are indications that regular and moderately intensive physical activity on a daily basis results in improved muscle function and a better quality of life in patients with early rheumatoid arthritis (11).

There are few studies on joint flexibility exercise for patients with RA, but a general level of physical activity appears to be beneficial for maintaining joint flexibility (12–14). Just like healthy patients, patients with rheumatoid arthritis are able to improve their aerobic capacity, muscle function, bone density, daily activity performance and quality of life with the implementation of physical activity (15–19). Recent studies of moderate to high-intensity exercise indicate that, regardless of whether the patient's disease status is stable or active, previous concerns over increased disease activity were unwarranted (12, 16, 20). The few studies that have examined the effects of moderate-intensity exercise on the progression of joint destruction showed no negative effects (15, 21). However, long periods of high-intensity exercise appeared to accelerate joint destruction in individuals with an initial destruction of large joints, particularly the shoulder joints and subtalar joints of the foot (22).

Indications

Physical activity and exercise are only used as secondary prevention in patients with RA as primary prevention is not possible.

For reasons of stress and fatigue and an increased risk of osteoporosis and premature death, mainly caused by cardiovascular disease, the positive health-related effects gained with physical activity are of specific relevance to patients with RA. Also, as mentioned above under "Pathophysiology", it is recommended that joint flexibility, strength and aerobic training programmes are adapted to the individual patient with the aim of maintaining optimal biomechanical properties and preventing a gradual decline in aerobic capacity. Physical activity can also help reduce symptoms of stress, anxiety and depression and stop social isolation.

Prescription

All patients with RA should be encouraged to be physically active so as to improve and maintain their physical and mental health and reduce the risk of comorbidity. Also, it is important to discuss how physical activities can be incorporated into the daily life of the patient. Because of the inconsistent progress of the disease, every organised physical exercise should be adapted to the individual patient for the purpose of improving bodily functions.

It is recommended that the joint range of motion is monitored and exercised, especially in case of an active disease status and threatening contractions. For patients with a very active disease status or significant disability (Functional Class IV), the emphasis should be on flexibility exercise together with innervation training of major muscle groups in the abdomen, seat and front thighs. Low-intensity exercise, either on land or in water, can be safely recommended for all patients with RA regardless of their disease status. In order to increase oxygen uptake capacity, muscle function and ability to carry out daily chores, the patient needs to perform moderate to high-intensity exercise for 30 minutes at least 3 times a week. Moderate to high-intensity exercise is also required to increase bone density.

The prescription specified in Table 1 is based on a systematic review of randomized controlled studies on physical exercise (15), but is not much different from general exercise recommendations. However, the following should be noted in relation to RA:

1. To reduce the risk of aggravated symptoms in connection with increased physical activity in patients with RA, physical activity should be slowly introduced, i.e. initial loads should be smaller than generally recommended and gradually increased in periods of 2–3 weeks.
2. Due to the intermittent nature of rheumatoid arthritis, it is not possible, as with other diseases, to consistently upgrade the exercise, but rather it must be adapted to the variations of the disease.
3. Specific attention paid to a possible increase in the initial pain will facilitate continued exercise.

There are indications that focusing on the goals of the exercise rather than on the symptoms that the exercise may potentially give rise to is beneficial to patients classified as belonging to either functional Class I or II with a stable disease status (23). However, it is still common for the so-called 24-hour rule to be applied and the load temporarily reduced if the increase in pain continues for more than 24 hours after exercise.

Table 1. Prescription of physical activity and exercise for patients with RA.

Objective	Frequency (times/week)	Duration (min./session)	Intensity % of APM*	Intensity acc. to RPE**	Load % of 1 RM***
Promote health	4–7	30	50–70	10–14	–
Improve aerobic fitness	3	30–60	60–80	11–15	–
Increase strength	2–3				50–80
Improve muscular endurance	2–3				30–40

* APM = Age-Predicted Maximum Heart Rate (220 – age).

** RPE = Rating of Perceived Exertion according to the Borg RPE scale.

*** RM = Repetition Maximum. 1 RM is the maximum weight that can be lifted through the entire exercise movement one time.

Suitable forms of exercise for patients with RA are cycling, cross-country skiing, Nordic walking, walking, light fitness training, dancing and strength training using either apparatus or rubber expanders. Exercising in a heated swimming pool can be very beneficial as the water offloads the body weight at the same time as it provides a gentle and even resistance to movement.

Because RA is a lifelong illness, the onset of which usually occurs at middle-age, it is important that the exercise can be as independent as possible. A number of studies indicate that patients with RA who initially exercise under the supervision of a physiotherapist have managed to continue their exercise independently with telephone support once a month and regular physiotherapy treatments 2–4 times a year for assessment, feedback and adjustment of intensity and load (20, 24, 25). Focusing on the benefits of exercise, e.g. an enhanced feeling of being in control, better general fitness, ease of movements and social support, can be motivating and help the individual to find an activity that he/she enjoys and will continue with for a long period of time (26, 27).

One vital aspect of physiotherapy check-up appointments is discussing the correct types of shoes and soles since engagement of the small joints in the foot is often a significant obstacle to physical activity and exercise in patients with RA.

Functional mechanisms

As far as one knows, there is hardly any difference between the functional mechanisms of physical activity in medically well-controlled patients with RA and the functional mechanisms of physical activity in the general public. Positive effects of exercise seen in patients with RA have also been proposed to be due to auto-synovectomy (28), a change in the neuropeptide concentration (29), decreased cachexia with an increased percentage of fat-free body mass (30) and molecular mechanisms such as a raised concentration of serum IGF-1 (31).

Functional tests/need for health checks

Standard health checks should be carried out, i.e. cardiovascular disease screening. Submaximal cycling or treadmill tests are used to assess the level of aerobic fitness while strength levels are assessed using various types of weight-lifting equipment. An evaluation of the effects of physical exercise can also be made using the different function tests and questionnaires found in the so-called REFORM folder (32) and on NRRK's (the National Research Centre for Rehabilitation in Rheumatology) website (www.nrrk.no).

Interactions with medical treatment

Cortisone is a catabolic steroid with alleviating effects that reduces the connective tissue strength in for example ligaments and tendons with an increased risk of rupture. Consequently, extreme physical activity is not recommended in connection with a cortisone treatment. The anti-inflammatory agents of cortisone also benefit from rest. It is recommended that patients undergoing a course of intra-articular cortisone injections should be prescribed rest, preferably bed rest, for the first 24 hours followed by at least one week of refraining from pronounced physical activity or exercise. When injections are given directly into muscular attachments or around tendons, the risk of rupture will remain for far longer, sometimes several months, and as a result, large physical loads should be avoided for a relatively long time. In the event of a long-term low-dose peroral cortisone treatment, the benefits of physical activity and exercising may surpass potential risk factors although this requires an individual assessment.

Subjecting a tender or painful joint to exercise and physical load is facilitated by administering an NSAID prior to exertion. This will not negatively affect the movement organs, at least not in the short term. However, the long-term effects of NSAIDs are unclear, although the general consensus is that it is better to uphold a certain level of mobility and strength, especially in the case of rheumatic sufferers. There are no indications that physical activity or exercise should be restrained during a course of SAARD (slow acting anti-rheumatic drug) treatments.

Contraindications

There are no absolute contraindications for physical activity as a consequence of RA. The conditions described below are all perceived as relative contraindications requiring specialist care.

Inflammation of the membrane surrounding the heart (pericarditis), congestive heart failure, inflammation of the lining around the lungs (pleuritis), pulmonary fibrosis, inflammation of blood vessels (vasculitis) and engagement of the kidney may occur in patients with RA. Patients with these types of complications require special training and guidance by healthcare professionals.

In the case of osteoporosis, a relatively common condition in patients with RA, physical activity constitutes an important part of the treatment, stimulating and strengthening the skeleton. However, it is important not to forget the increased risk of fractures and consequently, the necessity of being extra aware and cautious to prevent falls or similar accidents from happening. Caution should also be observed in connection with cortisone treatment. See above under “Interactions with medical treatment”.

Patients suffering with large joint destruction should be advised to weigh up the benefits of high-intensity training over the risk of an accelerated joint destruction. Exercise programmes designed for patients with large joint destruction should to the greatest extent possible protect shoulder joints and subtalar joints.

Caution should also be observed in the event of joint replacement surgery, especially in association with heavy-load strength training. However, physical activity and exercise is normally beneficial to maintain maximum muscle function and mobility before and after joint replacement surgery.

Acknowledgement

The authors would like to thank Camilla Fongen, Anne Christie and Kåre Birger Hagen at the National Research Centre for Rehabilitation in Rheumatology (NRRK), Diakonhjemmet Sykehus, Oslo, for their constructive observations and advice.

References

1. Uhlig T, Kvien TK. Is rheumatoid arthritis disappearing? *Ann Rheum Dis* 2005;64:7-10.
2. Haugeberg G, Ørstavik RE, Uhlig T, Falch JA, Halse JI, Kvien TK. Bone loss in patients with rheumatoid arthritis. Results from a population-based cohort of 366 patients followed up to two years. *Arthritis Rheum* 2002;46:1720-8.
3. Huusko TM, Korpela M, Karppi P, Avikainen V, Kautiainen H, Sulvaka R. Threefold increased risk of hip fractures with rheumatoid arthritis in central Finland. *Ann Rheum Dis* 2001;60:521-2.
4. Bacon PA, Towend JN. Nails in the coffin. Increasing evidence for the role of rheumatic disease in the cardiovascular mortality of rheumatoid arthritis. *Arthritis Rheum* 2001;44:2707-10.
5. Pincus T, Griffith J, Pearse S, Isenberg D. Prevalence of self-reported depression in patients with rheumatoid arthritis. *Br J Rheumatol* 1996;35:879-83.
6. Eurenus E, Stenström CH, PARA study group. Physical activity, physical fitness and general health perception among individuals with rheumatoid arthritis. *Arthritis Rheum* 2005;53:48-55.
7. Arnett FC, Edworthy SM, Bloch DA, McShane DJ, Fries JF, Cooper NS, et al. The American Rheumatism Association 1987 revised criteria for the classification of rheumatoid arthritis. *Arthritis Rheum* 1988;31:315-24.
8. Steinbröcker O, Traeger CH, Batterman RC. Therapeutic criteria in rheumatoid arthritis. *JAMA* 1949;140:659-62.
9. Ikeda K, Cox S, Emery P. Aspects of early arthritis. Biological therapy in early arthritis – overtreatment or the way to go? *Arthritis Res Ther* 2007;9:211.
10. van der Heijde D, Klareskog L, Rodriguez-Valverde V, Codreanu C, Bolosiu H, Melo-Gomes J, et al. Comparison of etanercept and methotrexate, alone and combined, in the treatment of rheumatoid arthritis. Two-year clinical and radiographic results from the TEMPO study, a double-blind, randomized trial. *Arthritis Rheum* 2006;54:1063-74.
11. Brodin N, Eurenus E, Jensen I, Nisell R, Opava CH, PARA study group. Coaching patients with early rheumatoid arthritis to healthy physical activity. A multicenter randomized, controlled study. *Arthritis Rheum* 2008;59:325-31.
12. van den Ende CH, Breedveld FC, le Cessie L, Dijkmans DA, de Mug AW, Hazes JM. Effect of intensive exercise in patients with active rheumatoid arthritis. A randomized clinical trial. *Ann Rheum Dis* 2000;59:615-21.
13. Van DJ, Harlowe D. The efficacy of the ROM Dance Program for adults with rheumatoid arthritis. *Am J Occup Ther* 1987;4:90-5.
14. Han A, Robinson V, Judd M, Taixiang W, Wells G, Tugwell P. Tai chi for treating rheumatoid arthritis. *Cochrane Database Syst Rev* 2004(3):CD004849.
15. Stenström CH, Minor MA. Evidence for the benefit of aerobic and strengthening exercise in rheumatoid arthritis. *Arthritis Rheum* 2003;49:428-34.
16. de Jong Z, Munneke M, Zwinderman AH, Kroon HM, Jansen A, Ronda KH, et al. Is a long-term high-intensity exercise program effective and safe in patients with rheumatoid arthritis? Results of a randomized controlled trial. *Arthritis Rheum* 2003;48:2415-24.

17. de Jong Z, Munneke M, Lems WF, Zwiderman AH, Kroon HM, Pauwels EAK, et al. Slowing of bone loss in patients with rheumatoid arthritis by long-term high-intensity exercise. *Arthritis Rheum* 2004;50:1066-76.
18. Bilberg A, Ahlmén M, Mannerkorpi K. Moderately intense exercise in a temperate pool for patients with rheumatoid arthritis. A randomized controlled study. *Rheumatol* 2005;44:502-8.
19. Eversden L, Maggs F, Nightingale P, Jobanputra P. A pragmatic randomised controlled trial of hydrotherapy and land exercises on overall being and quality of life in rheumatoid arthritis. *BMC Musculoskeletal Disorders* 2007;8:23.
20. Häkkinen A, Sokka T, Kotaniemi A, Hannonen P. A randomized two-year study of the effects of dynamic strength training on muscle strength, disease activity, functional capacity and bone mineral density in early rheumatoid arthritis. *Arthritis Rheum* 2001;44:515-22.
21. de Jong Z, Vliet Vlieland TPM. Safety of exercise in patients with rheumatoid arthritis. *Curr Opin Rheumatol* 2005;17:177-82.
22. Munneke M, deJong Z, Zwiderman AH, Runday HK, van Schaardenburg D, Dijkmans BAC, et al. The effect of a high-intensity weight-bearing exercise program on radiologic damage progression of the large joints in subgroups of patients with rheumatoid arthritis. *Arthritis Rheum* 2005;53:410-7.
23. Stenström CH. Home exercise in rheumatoid arthritis functional class II. Goal setting versus pain attention. *J Rheumatol* 1994;21:627-34.
24. Ekdahl C, Andersson SI, Moritz U, Svensson B. Dynamic versus static training in patients with rheumatoid arthritis. *Scand J Rheumatol* 1990;19:17-26.
25. Stenström CH, Arge B, Sundbom A. Dynamic training versus relaxation training as home exercise for patients with inflammatory rheumatic diseases. A randomized controlled study. *Scand J Rheum* 1996;25:28-33.
26. Hootman JM, Macera CA, Ham SA, Helmick CG, Sniezek JE. Physical activity levels among the general US adult population and in adults with and without arthritis. *Arthritis Rheum* 2003;49:129-35.
27. Eyler AA. Correlates of physical activity. Who's active and who's not? *Arthritis Rheum* 2003;49:136-40.
28. Bodamyali T, Stevens CR, Billingham MEJ, Ohta S, Blake DR. Influence of hypoxia in inflammatory synovitis. *Ann Rheum Dis* 1998;57:703-10.
29. Stenström CH, Alexanderson H, Lundberg I, Lundeberg T, Theodorsson E, Nisell R. Exercise and variations in neuropeptide levels in rheumatoid arthritis. A pilot study. *Neuropeptides* 1999;33:260-4.
30. Marcora SM, Lemmey AB, Maddison PJ. Can progressive resistance training reverse cachexia in patients with rheumatoid arthritis? Results of a pilot study. *J Rheumatol* 2005;32:1031-9.
31. Melikoglu MA, Karatay S, Senel K, Akcay F. Association between dynamic exercise therapy and IGF-1 and IGFBP-3 concentrations in the patients with rheumatoid arthritis. *Rheumatol Int* 2006;26:309-13.

32. REFORM-gruppen [The REFORM Group] Reumatologisk fysioterapi och riktlinjer för mätmetoder. [Rheumatological physiotherapy and guidelines for assessment] Stockholm: Department of Rheumatology, the Swedish Association of Registered Physiotherapists; 1999.

For training tips and advice

www.reumatikerforbundet.org

www.gigtforeningen.dk

www.idrett.no

www.bhss.no

www.vhss.no

44. Schizophrenia

Authors

Egil W. Martinsen, MD, PhD, Professor, Aker University Hospital, Oslo University, Oslo, Norway

Jill Taube, MD, Center for Family and Community Medicine, Karolinska Institutet, Stockholm, Sweden

Summary

Physical activity cannot replace traditional treatments of serious psychiatric illnesses but plays an important part of the treatment process. However, there is limited documentation and no randomised controlled trials of high scientific quality have studied the effects of physical activity on schizophrenia. Physical activity appears to alleviate negative symptoms and can also be a useful method for keeping positive symptoms under control. Physical activity may contribute to reduced anxiety and depression, and improve the quality of life as well as reduce the risk of relapse. The physical health benefits gained from physical activity are also important. Physical inactivity, smoking and obesity are common among people with schizophrenia, and likely reasons for the increased rate of morbidity and mortality found in this group.

Regular physical activity is an essential factor for maintaining good health and preventing disease, not least in relation to diseases that carry a risk of reduced longevity and greater overall morbidity. For many people, sports and outdoor activities are an important source of happiness in their lives, and there is no reason for people with a serious psychiatric disease not to take pleasure in the same type of activities.

Definition

Schizophrenia is one of the serious psychiatric diseases. The clinical picture of schizophrenia varies from individual to individual. In order to make a diagnosis, the patient must have been psychotic for a certain period of time. There are several types of psychotic symptoms, but the most common are delusions and hallucinations. The patient being convinced that he/she is being followed is one of the more common delusions. Hallucinations are defined as apparent perceptions of external objects when no such objects are present.

Hearing voices or seeing people that do not exist is not uncommon. Incoherent speech or behaviour is less common. These symptoms are called positive symptoms (1).

Negative symptoms are less dramatic but often of a greater significance to the patient's level of function over the long-term. The most common negative symptoms are apathy, passivity, and a lack of initiative and endurance. These symptoms are often difficult to accept for close relatives and caregivers. Schizophrenia is often associated with anxiety and depression, making it difficult to motivate these patients to be physically active

Prevalence/Incidence

Schizophrenia is a serious disease that affects approximately 0.5–1 per cent of the population and is hence as prevalent as insulin-dependent diabetes. Disease onset usually occurs in adolescence and adults aged 15 to 35, and affects both men and women. The prevalence of schizophrenia is noticeably similar irrespective of country or culture.

Causes and risk factors

The causes of schizophrenia are still not fully known but genetic predisposition is a significant factor. Close relatives of a person with schizophrenia have a 10 times greater risk of developing the disease compared to the general population. However, studies indicate that identical twins have a much greater risk of developing the disease than fraternal twins. Psychosocial and environmental factors may therefore also be of significance (1).

Prognosis

Even if there are treatment methods with documented beneficial effects, very few patients fully recover from schizophrenia. Most patients display a reduced functional capacity at school, work and in their social life, with only a small number able to hold regular employment. In the past, many people with schizophrenia spent most of their life in institutions, but recent closures have led to fewer of these institutions, which has led to most schizophrenic patients returning to their local areas (3). A lot of these schizophrenic patients are alone and need some form of help (4). A number of local governments have set up day centres and these have proven to be indispensable for this patient group.

Despite symptoms and suffering, many people with schizophrenia consider themselves to have a good quality of life.

Physical health

There has been a growing interest recently in the physical health of schizophrenic patients. The relative risk of premature death is four times greater in patients with schizophrenia than in the general population, while their life expectancy is at least 10 years shorter. Two thirds of the increased mortality rate may be explained by cardiovascular and respiratory diseases as well as diabetes. In all these diseases, lifestyle factors play an important role (5).

Compared to people without mental illness, a schizophrenic person is twice as likely to smoke, with a 50 per cent greater risk of being overweight or obese owing to an unhealthy diet and physical inactivity (6). In a comparative study of patients with schizophrenia or a serious emotional disorder and the general population, Dickerson and colleagues found that only 1 per cent of schizophrenic people, compared to 10 per cent of the general population, fulfilled the following five health criteria: non-smoker, takes regular physical exercise, good teeth, not overweight/obese and no major physical symptoms (7).

Current treatment principles

It is generally agreed that it is often necessary to use antipsychotic medication in the treatment of schizophrenic patients. The effects are well-documented, both in individual cases of the disease and for the purpose of preventing relapses. When schizophrenia is accompanied by depressive symptoms, the use of antidepressants and mood stabilisers may bring added benefits.

However, medications do not always have the desired effect, and adverse reactions are not uncommon. With the “old” antipsychotics, the most painful side effects were those affecting the neuromuscular functions, i.e. tremors, stiffness and slowness of movement. These are also referred to as drug and chemically induced parkinsonism as the symptoms are very similar to those of Parkinson’s disease. However, these side effects are less pronounced with the latest antipsychotics. Yet, more recent studies indicate that these medications may cause other side effects, such as weight gain, which, if serious, will give rise to additional problems for the individual in question. There is also a risk of the patient developing glucose intolerance, or increased blood fat levels, the ultimate consequence of which is metabolic syndrome and type 2 diabetes (5).

Medication treatment on its own is seldom enough and there are a number of scientifically documented psychosocial treatments. Family therapy sessions help the immediate family of the schizophrenic patient to be less critical or overprotective and instead be more supportive of the patient, which appears to reduce the risk of relapse. The purpose of the social training is to help the patient cope with sociable situations, e.g. how to start a conversation, what to do when he/she feels a relapse coming on and, finally, how to handle medication-induced side effects (8). Cognitive behavioural therapy has successfully been used to treat delusions, helping the patient to find an alternative explanation for what he/she is experiencing. Cognitive behavioural therapy has also been shown to benefit patients suffering from hallucinations (9). The majority of patients benefit from talk therapy.

Despite the treatments mentioned above, there is still a need for alternative treatments. Trial studies using physical activity as part of the treatment programme have been carried out, and the following is a summary of the current expertise in this area.

Effects of physical activity

When established psychiatric institutions began operating back in the 19th century, most treatment activities took place at farms in the countryside since moral treatment was the therapeutic approach recommended at that time. The mentally ill were thought to need a structured day and to improve their functional capacity by participating in the work on the farm. The physical work and contact with animals were considered important therapeutic elements.

Later, however, one began to question the therapeutic benefits of this form of treatment. The farm-located treatment programmes were gradually discontinued and for many patients, this meant increased physical inactivity and passivity. It is, however, interesting to note the renewed interest in manual farm work as a form of therapeutic treatment. These days, working with animals has become a therapeutic treatment and research area in its own right (10).

Primary prevention

Cross-sectional studies continuously indicate that the prevalence of psychiatric illnesses is higher in physically inactive than in physically active people, although the studies do not specifically relate to schizophrenia. No prospective studies have yet been carried out but, based on today's knowledge, there are no data indicating that physical activity can prevent schizophrenia.

Treatment

Schizophrenic individuals are generally in poor physical condition (11). Many intervention studies have been published on the effects of physical activity on positive and negative symptoms, depression and quality of life. However, these studies all have methodical limitations and no randomised study using satisfactory study methods has yet been published (12). Physical activity on its own is unlikely to be sufficient treatment. However, there are many sides to a serious disease such as schizophrenia. The following section describes some of the studies carried out on the benefits gained from physical activity.

Positive symptoms

The results of studies using well-documented measurement tools indicate that physical activity can be linked to a reduction in positive symptoms, above all auditory hallucinations (13, 14). One such example was a study carried out by Chamove (13) on 40 schizophrenic patients at a psychiatric nursing home in Scotland, where the staff were trained to assess the patients' symptoms and behaviours using specific scales. On those days when the patients participated in physical activities, a reduction in the extent and seriousness of their symptoms was observed. The patients least affected by the disease demonstrated the greatest change. In another study, Beebe and colleagues put 10 schizophrenic patients

on a 16-week walking schedule in addition to their regular treatment programme (15). Compared with the control group, the training group displayed fewer psychiatric symptoms after the intervention.

Psychotic symptoms rarely disappear completely. Consequently, a realistic treatment objective is to help patients live with their disease in the best possible way. One example of a psychotic symptom is auditory hallucinations, which are common in patients with schizophrenia. Patients with auditory hallucinations are treated with antipsychotics, though 25–30 per cent do not respond to this type of treatment. Falloon and Talbot (16) asked a group of patients with such symptoms which treatment methods they believed to be most beneficial. One of the treatment strategies mentioned most was physical activity, during which the voices or noises became less distressing. Shergill, Murray and McGuire (17) also asked a group of patients which strategy they believed most useful in controlling auditory hallucinations. As a result, they were able to determine that a distractive activity, or when patients were made to concentrate on a single task, had the best effect. The hallucinations did not disappear, but the distractive activities made them feel less difficult. Holmes and colleagues (18) asked a group of patients to describe the strategies they use to control difficult symptoms in general. The most common strategies included physical activity, stimuli reduction and the use of alcohol, narcotics and tobacco.

Negative symptoms

Physical activity can help to reduce negative symptoms. Chamove (13) describes how patients were less irritated, depressed, introverted and tense, as well as more sociable with better social skills on days with increased activity. Higher self-esteem was also reported. Anxiety and depression are common symptoms, and physical activity can be useful for controlling these symptoms in patients (12).

Body image perception

Sell (19) was interested in how schizophrenic patients perceive their own body and, in order to find this out, he asked them to draw a picture of a human being. Many schizophrenic people have a distorted body image and their drawings often feature the following characteristics: the figures are lifeless, often grotesque, stylised and static. Sell asked the schizophrenic patients to draw human figures before and after a 12-week fitness programme with exercise 3 times a week. Before the programme started, many of the drawings produced were distorted. For example, the proportion of various body parts was wrong and details such as mouths, eyes, fingers and toes were missing. At the end of the fitness programme, the drawings had become more normal, despite the patients not having any drawing lessons. This could be an indication that, by being more physically active, the patients got to know their body better and, as a result, their body image perception improved.

Quality of life

Borge and colleagues (4) studied long-term patients at a psychiatric hospital in Sogn og Fjordane one year after the number of patient spaces had been reduced significantly, with many of the patients returning to their home communities. They were interested in the patients' quality of life and the factors that could explain the variations in this. The factors that emerged from the study related to the patients' degree of loneliness, relationship with their neighbours and local surroundings and whether they participated in meaningful activities during the day. For many of these patients, holding regular employment is unrealistic. Physical activity during the day is thus a constructive alternative that may contribute to an enhanced quality of life.

Relapse prevention

In a Spanish study (20), researchers followed 40 patients with schizophrenia over a period of 10 years. The patients were divided into an exercise group and a control group, the latter of which received treatment as usual. Both groups received standard medication. During this 10-year period, significantly fewer relapses occurred among the patients in the exercise group.

Functional mechanisms

It is unlikely that a single factor can explain all of the psychological effects of physical activity, but there are a number of hypotheses that endeavour to explain the health benefits gained by physical activity in people with schizophrenia (12). The following physiological, neurobiological and psychological hypotheses have been put forward:

- People in a good physical condition generally have better health and greater resistance to disease and other loads. Well-trained individuals cope better with the challenges of everyday life by using a lower percentage of their maximal heart rate, and the heart rate normalises more rapidly after physical load.
- The basis for treating schizophrenia with medication is to influence neurotransmitters in the brain, in particular dopamine. Results from animal studies indicate that physical activity has an effect on these systems.
- Increased secretion of beta-endorphins appears to have a calming effect.
- Reduced interaction among the hypothalamus, pituitary gland and adrenal glands (the "HPA" axis) – which is important for how we respond to and tolerate stress.

Distraction and mastery are the most prominent psychological explanations. A number of studies indicate that patients suffering from auditory hallucinations have fewer and less difficult symptoms during physical activity, which could be explained by diversion or distraction. Physical activity does not make the hallucinations go away, but they become less dominant when the patient is concentrating on something else and, as a result, the patient feels more in control of the problem.

Many mentally ill people feel isolated and lonely, and have few interests or hobbies. In addition, they often lack social competence, which leads to a low self-esteem when in the company of other people. Physical activity can add meaning to their lives and contribute to an enhanced quality of life.

Indications

Primary prevention

People who are physically active tend to be less prone to developing mental disorders than inactive people, but there is no clear evidence to suggest that this relates specifically to schizophrenia.

Secondary prevention

Physical activity cannot be recommended as the only treatment for schizophrenia, but there are good reasons for integrating physical activity into the treatment process. Physical activity can reduce negative symptoms, improve the control of positive symptoms, lead to a better quality of life and a normal body image perception, and reduce the risk of relapse. Regular physical activity is also important for the prevention of somatic diseases, such as obesity, type 2 diabetes, hypertension, cardiovascular disease and metabolic syndrome.

Prescription

Special circumstances

A group of Swedish researchers was the first to evaluate the physical condition in schizophrenic patients (21). They found that patients were in poor physical condition but that they responded normally to fitness training. One exception was people on high doses of antipsychotics (chlorpromazine), who did not respond as well to physical activity. Individual differences in physical condition vary considerably from patient to patient, and it is therefore essential that the intensity and duration of physical activity be adapted to the capacity of the individual. It is easy to aim too high at the start, and it is often surprising how little activity is needed to have an effect on untrained individuals.

Dosage

Normal guidelines for health-promoting physical activity can also be used for schizophrenic people, and most of the health benefits can be achieved with a half hour of activity most days of the week. However, individuals who wish to lose weight should be physically active for one hour a day. It is important to remember that, even in small doses, regular physical activity is beneficial, and it is the overall time spent being physically active that counts.

Functional tests/Need for health check-ups

It is not necessary for schizophrenic patients to undergo any specific examinations before starting regular physical activity or exercise, unless the patient in question has other conditions, e.g. heart disease.

How and when should the effects gained be assessed?

Regular fitness assessments and controls are essential, as they serve to encourage the patient and are also an indication of whether the increased physical activity has the desired effect. In addition, there are simple, readily available tests for measuring mental health changes, including anxiety and depression, and quality of life.

Interaction with drug therapy

Large doses of antipsychotics may reduce the effects of a training programme (21), but there are no risks associated with training while on therapeutic doses of psychotropic drugs (22).

Contraindications

There are no contraindications to physical activity for individuals with schizophrenia unless other physical disease is present.

Risks

For patients without co-existing physical disease for which physical activity is contraindicated, there are no specific risks involved with physical activity.

References

1. American Psychiatric Association. Diagnostic and Statistical Manual of Mental Disorders. 4. edition, Washington (DC): American Psychiatric Association; 1994.
2. Malt UF, Retterstøl N, Dahl AA. Lærebok i psykiatri. [Handbook of Psychiatry] Oslo: Gyldendal; 2003.
3. Martinsen EW, Ruud T, Borge L, Watne Ø, Friis S. The fate of chronic in-patients after closure of psychiatric nursing homes in Norway. A personal follow-up 6 years after. *Acta Psychiatrica Scandinavica* 1998;98:360-5.
4. Borge L, Martinsen EW, Ruud T, Watne Ø, Friis S. Quality of life, loneliness and social contacts among long-term psychiatric patients. *Psychiatric Services* 1999;50:81-4.
5. Connolly M, Kelly C. Lifestyle and physical health in schizophrenia. *Advances in Psychiatric Treatment* 2005;11:125-32.
6. Compton MT, Daumit GL, Druss BG. Cigarette smoking and overweight/obesity among individuals with serious mental illnesses. A preventive perspective. *Harvard Review of Psychiatry* 2006;14:212-22.
7. Dickerson FB, Brown CH, Daumit G, Lijuan F, Goldberg RW, Wohlheiter K, Dixon LB. Health status of individuals with serious mental illness. *Schizophrenia Bulletin* 2006;32:584-9.
8. Nathan PE, Gorman JM. A guide to treatments that work. Oxford: Oxford University Press; 2007.
9. Trower P, Birchwood M, Meaden A, Byrne S, Nelson A, Ross, K. Cognitive therapy for command hallucinations. Randomised controlled trial. *British Journal of Psychiatry* 2004;84:312-20.
10. Berget B. Animal-assisted therapy. Effects on persons with psychiatric disorders working with farm animals. *Philosophia Doctor Thesis*. Oslo: Norwegian University of Life Sciences; 2006.
11. Carlsson C, Dencker S, Grimby G, Heggendal J. Circulatory studies during physical exercise in mentally disordered patients. I. Effects of large doses of chlorpromazine. *Acta Medica Scandinavica* 1968;184:499-509.
12. Faulkner GEJ. Exercise as an adjunct treatment of schizophrenia. In: Faulkner GEJ, Taylor AH, Eds. *Exercise, Health and Mental Health. Emerging relationships*. London: Routledge; 2005.
13. Chamove AS. Positive short-term effect of activity on behaviour in chronic schizophrenic patients. *British Journal of Clinical Psychology* 1986;25:125-33.
14. Lukoff D, Wallace CJ, Lieberman RP, Burke K. A holistic program for schizophrenic patients. *Schizophrenia Bulletin* 1986;12:274-82.
15. Beebe LH, Tian L, Morris N, Goodwin A, Allen SS, Kuldau J. Effects of exercise on mental and physical health parameters of persons with schizophrenia. *Issues in Mental Health Nursing* 2005;26:661-76.
16. Falloon IRH, Talbot RE. Persistent auditory hallucinations. Coping mechanisms and implications for management. *Psychological Medicine* 1981;11:329-39.

17. Shergill SS, Murray RM, McGuire PK. Auditory hallucinations. A review of psychological treatments. *Schizophrenia Research* 1998;32:137-50.
18. Holmes H, Ziemba, J, Evans T, Williams CA. Nursing model for psychoeducation of the seriously mentally ill patient. *Issues on Mental Health Nursing* 1994;5:85-104.
19. Sell H. *The Effect of Physical Training on Psychiatric Patients*. Odense: [Published by the author]; 1994.
20. Torres-Carbajo A, Olivares JM, Merino H, Vazquez H, Diaz A, Cruz E. Efficacy and effectiveness of an exercise program as community support for schizophrenic patients. *American Journal of Recreation Therapy* 2005;4:41-7.
21. Carlsson C, Dencker S, Grimby G, Heggendal J. Circulatory studies during physical exercise in mentally disordered patients. I. Effects of training in patients with and without administration of chlorpromazine. *Acta Medica Scandinavica* 1968;184:511-6.
22. Martinsen EW, Stanghelle JK. Drug therapy and physical activity. In: Morgan WP, Ed. *Physical Activity and Mental Health*. Washington (DC): Taylor & Francis; 1997 p. 81-90.

45. *Spinal cord injury*

Author

Nils Hjeltnes, MD, PhD, Spinal Cord Injury Rehabilitation Department, Sunnaas Hospital, Nesoddtangen, Norway

Summary

Physical activity and exercise following a spinal cord injury is a central part of the rehabilitation programme for everyone with a spinal cord injury. As in individuals with normal function, the best form of rehabilitation/physical activity depends on such factors as age, gender and fitness, etc. However, the level and extent of the injury as well as how much time has passed since the injury are of even greater importance. Patients should be mobilised as soon as medically possible after a spinal cord injury. The initial treatment is passive, e.g. stretching muscles, mobilising joints and removing mucus. This is followed by an active training programme that incorporates individual training for the purpose of improving aerobic fitness, muscle strength, coordination and balance. Much of the training is similar to that recommended for individuals with normal function except that it is of a different intensity and modified to the individual patient. Both the spinal cord injury patient and the physiotherapists managing his or her rehabilitation must know how to avoid and prevent complications during training. Physiotherapists must also be fully aware of the possibilities available to spinal cord injury patients so as to be able to help the patient improve his or her health and physical fitness. Recent epidemiologic studies indicate that there is a relatively large number of patients with incomplete spinal cord injuries. Consequently, it is vital to acquire new knowledge on how these individuals can best train, not just for the purpose of improving aerobic fitness and muscle strength, but also to learn how to do “old activities with new muscles”.

Introduction

A spinal cord injury destroys the neural connections between the cranial part of the central nervous system and the spinal cord caudally of the damaged area. Depending on the localisation and extent of the injury, this usually leads to a varying degree of muscle paralysis and lack of sensation in addition to a reduced control of bodily functions (e.g. urination, defecation and sexual function), blood pressure and body temperature. A spinal cord

injury is either traumatic because of a traffic accident, fall, work-related injury, sports injury or violence, or atraumatic as a consequence of an infection, circulation disorder or tumour (benign or malignant). The injury may also be congenital (inherited or caused at birth) or caused by medical or surgical intervention.

The following section focuses on physical activity and training for people with a spinal cord injury from around 6 months after the occurrence of the injury.

Level and extent of injury

While the spine has 7 cervical, 12 thoracic, 5 lumbar and 5 sacral vertebrae, the spinal cord has 8 cervical, 12 thoracic, 5 lumbar and 5 sacral *spinal cord segments*. Each segment forms a pair of *spinal nerve roots* that innervate the myotomal muscles and consist of sensory fibres from a *dermatome on the skin*. On this anatomical basis, it is not difficult to localise the spinal cord injury level with a clinical neurologic examination.

The level of the neurologic injury is defined on the basis of the degree of normal function found in the caudal spinal cord segment (1, 2). A spinal cord injury that causes paralysis in both arms and legs is called *tetraplegia*. A spinal cord injury that causes paralysis in the legs and torso (truncus) is called *paraplegia*. There is reason to classify patients with the lowest level of spinal cord injury, i.e. a conus medullaris injury, and patients with a cauda equina injury into the same category, i.e. the conus-cauda syndrome. Common for patients with conus-cauda injuries is paralysis of the legs, a dysfunctional urinary bladder and rectal muscles plus pronounced sexual dysfunction.

The cross-sectional extent of a spinal cord injury is just as important as the level of the injury as regards the ability to function properly after a spinal cord injury as classified by the American Spinal Injury Association's (ASIA)* Impairment Scale, Class A to E. Patients classified as:

AIS-A have no motor or sensory function below the spinal cord injury level.

AIS-B have impaired sensory function below the injury level.

AIS-C have impaired motor and sensory function below the injury level.

AIS-D have a certain motor function preserved (muscle grade 3 or more on a scale from 0 to 5) in 50 per cent of the muscles below the injury level.

AIS-E have insignificant neurologic impairment as a consequence of the spinal cord injury (1).

* Original specification of www.asia-spinalinjury.org/contact/ reads:

- A** = Complete: No motor or sensory function is preserved in the sacral segments S4–S5.
- B** = Incomplete: Sensory but not motor function is preserved below the neurological level and includes the sacral segments S4–S5.
- B** = Incomplete: Motor function is preserved below the neurological level, and more than half of key muscles below the neurological level have a muscle grade less than 3.
- D** = Incomplete: Motor function is preserved below the neurological level, and at least half of key muscles below the neurological level have a muscle grade of 3 or more.
- E** = Normal: motor and sensory functions are normal.

AIS-A is considered a complete injury, i.e. complete loss of motor and sensory function below the defined injury level. AIS-B to AIS-D represents an incomplete injury where AIS-B means that the injured person still has a complete motor dysfunction, but an intact sensory function. In order for an injury to be considered incomplete, the injured person must have sensory and/or motor function in the lowest sacral spinal cord segment, i.e. sensation and/or controlled rectal motor function.

Epidemiology

Epidemiologic studies show that even if the annual incidence of spinal cord injuries varies, it is relatively stable over a longer period of time. In Norway, the number of new traumatic spinal cord injuries needing specialised rehabilitation was 16.5 in 1 million in 1974 (3). From 2001 to 2004, this number was between 12 and 18 per 1 million and year (data obtained from NordiskRyggmargskadeRegister). A Nordic Spinal Cord Injury Council has been established and is updated on an annual basis (www.nscic.se).

The most common cause of traumatic spinal cord injuries is road accidents, although this has lately been matched by injuries from falls. Injuries from free-diving have become less common, while injuries as a result of violence are on the rise. Between 20 and 25 per cent of those afflicted are women. No Norwegian prevalence studies exist, but studies carried out outside of Norway show an incidence of between 200 and 1,000 per 1 million inhabitants. This means that there are between 1,000 and 5,000 people in Norway with a permanent functional impairment as a consequence of a spinal cord injury. Recent studies indicate a somewhat lower prevalence in Sweden than Norway. There are no comparative international studies on the prevalence of spinal cord injuries.

In Norway, an estimated annual figure of 60 patients with atraumatic spinal cord injuries require the same rehabilitation and training programme as patients with traumatic spinal cord injuries. However, this figure does not include patients with spinal cord injuries caused by cancer (and metastasis). It is assumed that the same prevalence exists in other Nordic countries.

Treatment of traumatic spinal cord injuries

The treatments and primary rehabilitation methods employed nationally and internationally after a traumatic spinal cord injury of a cross-sectional nature are usually centralised at so-called comprehensive spinal units. The comprehensive spinal units are either made up of emergency and rehabilitation departments at the same hospital or separate institutions. Most importantly, these units have specialist expertise, which is a guarantee that the treatment and rehabilitation programmes offered to patients are on a par with high-quality international standards. Highly specialised and competent spinal cord injury departments cooperate with other specialists (urology, plastic surgery) throughout the lifelong treatment of spinal cord injuries. Other organisations and institutions are also involved in the follow-up process such as primary healthcare and physiotherapy clinics. These offer training camps and physical skills programmes for patients with new or old injuries.

A more thorough description of emergency treatment and primary rehabilitation of spinal cord injuries falls outside the scope of this chapter. The “father” of today’s treatment and rehabilitation of spinal cord injuries, Sir Ludwig Guttmann, said the following back in 1945: “Rehabilitation after spinal cord injuries seeks the fullest possible physical and psychological readjustment of the injured person to his permanent disability with a view to restoring his will to live and working capacity” (4). The cutting-edge expertise that is offered by spinal cord injury treatment units contributes to achieving this objective (5).

Effects of spinal cord injuries on level of activity

A spinal cord injury usually involves a dramatic change in the injured person’s ability and possibility to be physically active. Naturally, the level and extent of the injury is of utmost importance in this context. A person with complete tetraplegia with a damaged respiratory centre (nucleus phrenicus) may be lifetime dependent on a ventilator and thereby restricted to passive physical training. However, a person with an incomplete low spinal cord injury (conus injury) could have an intact skeletal muscle function, but a restricting body dysfunction with a better chance of physical activity regardless of the level of injury than a person with a similar, but complete injury.

Neuromuscular function/spasticity

After a spinal cord injury, spasticity imitates spinal reflexes to replace normal muscle activity. Spasticity is a syndrome that increases resistance to rapid passive movements, involuntary clonic and tonic muscle spasms, conductive time delay, synergist and antagonist coactivation and reduced strength (6). At the same time, these spasms are a manifestation of what “the spinal cord can manage on its own” and hence, should only be treated if functionally restrictive for the injured person (7).

Lung function

Lung function and lung capacity are strongly linked to the level of the spinal cord injury (8–10). Persons with an upper cervical spine injury may need the lifelong support of a ventilator. Some patients need help breathing for certain periods of time and therefore need access to a ventilator or similar equipment in their home (CPAP/Bi-PAP). Tetraplegic patients generally suffer from sleep apnoea, even during the chronic stage of their injury (11). However, the lung capacity of patients needing respiratory assistance is seldom a restrictive factor for physical activity during the chronic stage of their injury, regardless of the level of injury. This is evident from tests using an arm-pedalled bicycle where the respiratory minute volume during maximal exertion continued to increase at the same time that a decrease in the oxygen uptake capacity was noted.

Autonomic regulation weakness

A complete cervical spinal cord injury also destroys the communication between the superior autonomic centre of the brain and the sympathetic intermediolateral nucleus membrane of the spinal cord and corresponding parasympathetic nucleus membrane in the sacral portion of the medulla. This not only of consequence to controlled body functions such as urination, sexual function and defecation, but also to the regulation of heart rate, blood pressure and body temperature.

By using industrial physiological techniques and subjecting a person with a spinal cord injury to maximal load on an arm ergometer while measuring the person's heart rate, blood pressure, maximal oxygen uptake and lactic acid production, it has been possible to show that the heart rate in patients with complete tetraplegia hardly ever exceeds 125 heart beats per minute at maximal load (12, 13). Instead, physical exertion results in a drop in blood pressure and a disproportionate increase in body temperature relative to the low, but maximal load that tetraplegic patients can cope with (14). Some tetraplegic patients will always have a problem with their blood pressure, even when sitting in a wheelchair.

Patients with high paraplegia respond in very much the same way, but can usually increase their heart rate to a normal maximal rate (13) and therefore do not suffer to the same extent from drops in blood pressure due to exertion. However, this group of patients does not suffer from the same rise in blood pressure caused by exertion as non-disabled individuals. The blood-pressure response during physical load is fairly normal in those with spinal injuries where the level of injury is below the tenth (Th10) thoracic vertebrae. The cardiac muscle generally has an overcapacity compared with the load to which a disabled person is subjected in the chronic stage of a spinal cord injury. The load placed on the cardiac muscle of a tetraplegic person is so insignificant that the cardiac muscle decreases in size (hypotrophies) due to the insufficient level of physical activity, low venous back-flow and low blood pressure (15). A varying degree of impaired autonomic regulation may also exist in patients with incomplete cervical injuries. There are numerous examples of persons with mild spasticity and incomplete tetraplegia (ASIA-D) who become pale and feel sick as a result of physical exertion long after the injury occurred.

Autonomic dysreflexia

Autonomic dysreflexia is an autonomic sympathetic “overactivity syndrome” (16). This syndrome is primarily seen in patients with a complete injury above the Th6 level, but is also found in individual patients down to an injury level of Th10. The overactivity syndrome is characterised by high blood pressure with headaches, bradycardia, goose bumps, perspiration above and below the injury level and facial redness which may occur in conjunction with physical activity. The vasoconstriction, i.e. narrowing of blood vessels, especially the visceral vessels, results in a clear rise in blood pressure that can be life-threatening or used by tetraplegic persons to prevent an unwanted fall in blood pressure during exercise (boosting). The triggering factor, whether an overfull bladder, urinary tract infection or anything else that activates the spinal cord below the injury level, should nonetheless be removed without delay.

Metabolic function

Body composition changes following a spinal cord injury at both the macro and micro level. These changes (reduced bone density and muscle mass plus increased body fat) are a consequence of the metabolic changes caused by the spinal cord injury and the physical inactivity created by the paralysis (17). Muscles are metabolically active organs and a reduced muscle mass means that nutrients and other waste products from the metabolic system remain in the blood for a longer period of time than in healthy people.

Studies have shown that persons with a spinal cord injury have a lower glucose tolerance and that insulin resistance is predominantly caused by a reduced muscle mass (18). However, the muscle cells below the injury level have a normal glucose uptake capacity when stimulated by insulin or functional electrical stimulation although most of the muscle fibres are very small, i.e. type 2b fibres (19). In addition, persons with protracted spinal cord injuries appear to have an unfavourable cholesterol profile with low HDL and high LDL levels (20). These verified metabolic changes are linked to the results of descriptive epidemiological studies which signify a predominance of obesity, insulin resistance, Type 2 diabetes and coronary artery disease among people with a protracted spinal cord injury (21).

Other conditions

Like people in good functional health, the level of physical activity in people with impaired functional health is dependent on age, gender, psychosocial and environmental conditions. There is currently no cure for spinal cord injuries and since there are only limited resources to support treatment, rehabilitation and lifetime monitoring, it is vital that expert professionals, researchers, politicians as well as the spinal cord injury patients themselves prioritise those areas that need further development. In an American interview study, more than 96 per cent of the spinal injury patients said that physical activity was important to achieve functional improvement (22). Around 57 per cent of the interviewees had the possibility of exercising, while only around 12 per cent were able to exercise with the assistance of a personal trainer or physiotherapist. The same study showed that tetraplegic patients

missed their finger and hand function, while paraplegic patients would like to have their sexual function restored to normal. Both tetraplegic and paraplegic patients would prefer to have urinary and defecation functions restored more than the ability to walk (22).

Exercise, effects and special conditions

Normal physical activity and exercise after a complete spinal cord injury entail using muscles not affected by the injury, which in the case of tetraplegia means arm and shoulder muscles with the addition of trunk muscles for paraplegic patients and leg muscles for conus injuries and other incomplete injuries. It is well documented in a number of controlled studies that various methods of arm training (arm cycling using an arm ergometer, wheelchair ergometer, manual wheelchair training on a treadmill or rowing) and various training programmes (interval training) designed to achieve maximal oxygen uptake (VO_2 max/peak VO_2) or a specific percentage increase in the heart rate increase the maximal load and the body's ability to cope with lactic acid. A higher aerobic fitness level lessens the load placed on the heart during daily activities (23).

General physical training also involves training the respiratory muscles. In tetraplegic patients, the diaphragm is more than a respiratory muscle. It is also an equilibrium muscle when the patient is sitting and can sometimes become fatigued by having to carry out two functions at the same time (24). A person with high tetraplegia (level C6) has a significantly reduced vital capacity and the daily sitting positions of a person with such a high level injury should not engage the diaphragm to act as a postural muscle. During physical training, such as the use of an arm ergometer, the trunk of the patient should be fixated with a trunk belt to prevent the diaphragm from having to carry out two functions. Hence, it is possible to exercise although the fixation of the trunk is vitally important as it could otherwise lead to injury with negative consequences for other daily functions.

Skin

The patient's skin must be carefully monitored, which is a task that can gradually be shifted over to the patient, who can do it on his/her own using a mirror. At first sign of red pressure marks or abrasions on the skin, an expert adviser should immediately be contacted, bedding and seat cushioning checked and, if required, the pressure unloaded. Unloading pressure is very important during treatment and to prevent pressure sores (25). Some spinal units have the ability to register the pressure that individual patients are subjected to when sitting or lying and as a result, the bedding and seat cushioning can be made to order which is ideal. There are also ready-made, high-quality mattresses and seat cushions to buy which only require minor adjustments.

In addition, a change of position stimulates the vascular reflexes that regulate the blood flow through the skin. Using electrical muscle stimulation, the muscle mass can be increased in areas that are subjected to pressure (gluteal muscles) and so, give added protection, for example around the hips where the skin is close to the bone.

Urinary tract and urination

Some individuals with spinal cord injuries have problems with incontinence when carrying out a physical activity (26). “Training is not fun when you always end up peeing in your pants”, announced a spinal patient whose problem was later solved and who went on to become an Olympic Weightlifting Champion. Irrespective of how a patient’s bladder is emptied, many spinal cord patients end up with urinary tract infections and a limited ability to carry out physical activity or training for certain periods.

Gastrointestinal tractus – alimentary canal

Problems defecating after a spinal cord injury are aggravated by physical inactivity, not least due to the reduced activity and toning of the abdomen muscles. The severity of the problem varies from individual to individual, but is generally dependent on the level and extent of the spinal cord injury. Of spinal cord patients, 30 per cent feel that problems defecating are more troublesome than those associated with urination or sexual function (27). The nutritional regimen, food patterns and physical activity are considered to be important to the functioning of the intestine.

Contracture prophylaxis

Joint mobility should be maintained in the early stages of an injury to avoid joint stiffness and contracture in joints that are not used, irrespective of muscle tonus (muscle tension). In paralysis with reduced muscle tonus, a daily programme of activities is usually sufficient, but joints subjected to a local trauma or oedema need to be moved 2–3 times a day. In special cases (complete injury), taping the patient’s hands or using modified fixation plates to fixate the patient’s finger joints in a better (more functional) handgrip position is an initial possibility. Also, the use of plates preventing the development of club foot can be beneficial.

Reduced bone mineral density (osteopenia)

Bones that are neither subjected to gravity nor muscular strength lose strength and mineral density (28, 29). The rapid decalcification that occurs in the first three months after a spinal cord injury increases the risk of severe *hypercalcemia*. Hypercalcemia usually affects boys or young men with tetraplegia or high tetraplegia (above Th5) who were very physically active (athletes) prior to the accident, although not patients with incomplete injuries (30).

Injuries of the central nervous system with pronounced paralysis increase the risk of decalcification around the joints, *periarticular ossifications* (PAO). This is an inflammatory reaction that primarily occurs adjacent to large joints (laterally around the hips and medially around the knees, ankles and sometimes the elbows). This complication is predominant in patients with a complete spinal cord injury, but seldom in children. The patient’s general health is affected by fever, elevated inflammation test levels (CRP and SR) and an increase in the amount of alkaline phosphates (ALP).

No matter what type of treatment is offered, a massive decalcification of the long femur bones below injury level is unavoidable. There are currently no long-term studies indicating that standing training exercises increase the bone density after a spinal cord injury. However, it is documented that electrically stimulated leg cycling carried out by tetraplegic patients resulted in an increased bone density, particularly around the knee joint (31). *Consequently, spontaneous fractures* in the lower extremities are a common long-term complication in spinal patients and ought to be treated along the same principles as spontaneous fractures in persons with no functional impairments, but extra care should be taken when casting a fracture so as not to subject the skin or nerves near the surface to pressure.

Defective spinal position is also common in persons with spinal cord injuries. However, new surgical procedures to treat the acute phase of an injury reduce the risk of developing severe gibbus (spinal hump) or scoliosis. Because of changes in the paravertebral muscle tonus, weakened abdomen muscles, a lot of sitting combined with a poor sitting position, the spine will have to be checked on a regular basis for the rest of the spinal patient's life. Surgery for the correction of secondary scoliosis is required when the scoliosis is a hindrance to function or exceeds an angle of 35–40 degrees.

Diet and nutrition

The diet of those with spinal cord injuries must be adapted to a changed metabolism. The daily calorie intake is lower than in healthy persons with no functional impairment, usually due to physical inactivity. However, this does not necessarily mean that the individual's appetite is reduced and obesity is consequently a real problem for many patients. There are currently no studies recommending a special diet for this group of patients, which is why the diet recommended for healthy persons with no functional impairment applies, i.e. a nutritional low-calorie diet. To-date, no documentation can verify that spinal patients would benefit from nutritional supplements even though a lack of nutrients appears to prolong the healing of pressure sores (32).

Pain

Chronic pain is a serious problem after a spinal cord injury and a frequent obstacle to physical activity and training. The prevalence of pain varies from study to study, but an average of 65 per cent of spinal injury patients experience chronic pain, one-third of whom were found to have pronounced pain (33). The main difference seems to be whether the pain is nociceptive or neurogenic or localised above, in the centre of or below the spinal cord injury level. Psychological and social factors undoubtedly play an important role too. It is difficult to treat chronic pain (33). When medicinal and/or surgical treatment is not enough, pain management strategies, cognitive therapy and support from other spinal patients is of vital importance.

Syringomyelia

Syringomyelia is a very problematic condition in spinal cord injury patients (34). Post-traumatic syrinxes (spinal cord cysts) form at spinal cord injury level in up to 30 per cent of all spinal patients. However, there is no reason for a surgical procedure before the patient experiences severe pain or loses muscle strength and sensitivity of the skin. Diagnosis is usually clear-cut using magnetic resonance imaging, which shows whether a cyst has formed or spread. Patients with syringomyelia are sometimes recommended to abstain from physical training such as weight lifting.

Hand surgery

Reconstructive hand surgery can lead to an improved finger and hand function in tetraplegic patients (35). Hand surgery is most beneficial for patients with level C6 injuries where initial surgery is aimed at developing the primary stretch function of the elbow followed by key grip surgery between the thumb and index finger. Hand surgery can also result in a patient with level C5 injuries regaining his/her wrist function. Patients with level C7 injuries may also regain finger flexibility. This means added opportunities with respect to physical activity and training.

Orthopaedic aids

A range of plates and orthoses have been developed for the improvement of walking in paraplegic patients. Standard plastic or metal orthoses for knees, ankles or feet plus knee locks and foot/foot joint plates are generally available. A so-called Parawalker and Reciprocating Gait Orthosis can be combined with an electrical function simulation of a hybrid orthosis to improve walking in paraplegic patients (36–39). Despite the use of a mobility aid, it is very energy consuming for a paraplegic patient fitted with plates to walk upright. The walk pattern is slow and many follow-up studies have shown that patients with this type of orthoses would rather use a wheelchair on a daily basis. At any rate, it is recommended that the plates are used in training in an upright position as this could have long-term benefits for the entire body.

Conclusion

It is hoped that future technical developments will lessen the extent of a spinal cord injury in the acute stage and provide better opportunities for supporting weakened body parts in the rehabilitation phase. Around the world, key data is accumulated systematically in so-called spinal cord injury registries such as the Nordic Spinal Cord Injury Council for the purpose of improving treatments offered to those with spinal cord injuries. However, it is still not possible to fully cure a spinal cord injury, and preventive measures, rehabilitation and access to daily physical activity and training are extremely important.

References

1. Ditunno JF, Young W, Donovan WH, Creasy G. The international standards booklet of neurological and functional classification of spinal cord injury. *Paraplegia* 1994;32:70-80.
2. Maynard jr FM, Bracken MB, Creasey G, Ditunno jr JF, Donovan WH, Ducker TB, et al. International standards for neurological and functional classification of spinal cord injury. *Spinal Cord* 1997;35:266-74.
3. Gjone RN, Nordlie L. Incidence of traumatic paraplegia and tetraplegia in Norway. A statistical survey of the years 1974 and 1975. *Paraplegia* 1978-79;16:88-93.
4. Guttmann L. New hope for spinal sufferers. *Ludwig Guttmann*. Reproduced from *Medical Times*, November 1945. *Paraplegia*. 1979;17:6-15.
5. Jones L, Bagnall A. Spinal injuries centres (SICs) for acute traumatic spinal cord injury. *Cochrane Database Syst Rev* 2004;(4):CD004442.
6. Young RR. Spasticity. A review. *Neurology* 1994;44:S12-20.
7. Kirshblum S. Treatment for spinal cord-injury related spasticity. *J Spinal Cord Med* 1999;22:199-217.
8. Borel CO, Guy J. Ventilatory management in critical neurologic illness. *Neurol Clin* 1995 Aug;13:627-44.
9. Carter RE. Respiratory aspects of spinal cord injury management. *Paraplegia* 1987;25:262-6.
10. Anke A, Aksnes AK, Stanghelle JK, Hjeltnes N. Lung volumes in tetraplegic patients according to cervical cord injury level. *Scand J Rehab Med* 1993;25:73-7.
11. Biering Sørensen F, Biering Sørensen M. Sleep disturbances in the spinal cord injured. An epidemiological questionnaire investigation, including a normal population. *Spinal Cord* 2001;39:505-13.
12. Dallmeijer AJ, Hopman MT, van As HH, van der Woude LH. Physical capacity and physical strain in persons with tetraplegia. The role of sport activity. *Spinal Cord* 1996;34:729-35.
13. Hjeltnes N. Cardiorespiratory capacity in tetra- and paraplegia shortly after injury. *Scand J Rehab Med* 1986;18:65-70.
14. Sawka MN. Physiology of upper body exercise. *Exerc Sport Sci Rev* 1986;14:175-211.
15. Nash MS, Bilsker MS, Kearney HM, Ramirez JN, Applegate B, Green BA. Effects of electrically-stimulated exercise and passive motion on echocardiographically-derived wall motion and cardiodynamic function in tetraplegic persons. *Paraplegia* 1995;33:80-9.
16. Stjernberg L, Blumberg H, Wallin BG. Sympathetic activity in man after spinal cord injury. *Brain* 1984;107:183-98.
17. Hjeltnes N. Physical exercise and electrical stimulation in the management of metabolic, cardiovascular and skeletal-muscle alterations in people with tetraplegia. Stockholm: Department of Surgical Science, Department of Clinical Physiology, Karolinska Hospital; 1998.

18. Aksnes AK, Hjeltnes N, Wahlström EO, Katz A, Zierath JR, Wallberg-Henriksson H. Intact glucose transport in morphologically altered denervated skeletal muscle from quadriplegic patients. *Am J Physiol* 1996;271:593-600.
19. Hjeltnes N, Galuska D, Björnholm M, Aksnes AK, Lannem A, Zierath JR, et al. Exercise-induced overexpression of key regulatory proteins involved in glucose uptake and metabolism in tetraplegic persons. Molecular mechanism for improved glucose homeostasis. *FASEB J* 1998;12:1701-12.
20. Bauman WA, Spungen AM, Zhong YG, Rothstein JL, Petry C, Gordon SK. Depressed serum high density lipoprotein levels in veterans with spinal cord injury. *Paraplegia* 1992;30:697-703.
21. Whiteneck GG, Charlifue SW, Frankel HL, Fraser MH, Gardner BP, Gerhart KA, et al. Mortality, morbidity and psychosocial outcomes of persons spinal cord injured more than years ago. *Paraplegia* 1992;30:617-30.
22. Anderson KD. Targeting recovery. Priorities of the spinal cord-injured population. *Journal of Neurotrauma* 2004;21:1371-83.
23. Janssen TW, van Oers CA, Veeger HE, Hollander AP, van der Woude LH, Rozendal RH. Relationship between physical strain during standardised ADL tasks and physical capacity in men with spinal cord injuries. *Paraplegia* 1994;32:844-59.
24. Sinderby C, Weinberg J, Sullivan L, Lindström L, Grassino A. Electromyographical evidence for exercise-induced diaphragm fatigue in patients with chronic cervical cord injury or prior poliomyelitis infection. *Spinal Cord* 1996;34:594-601.
25. Bell GB. Spinal cord injury, pressure ulcers and support surfaces. *Ostomy Wound Manage* 1999;45:48-50, 52-3.
26. Wyndaele JJ, Madersbacher H, Kovindha A. Conservative treatment of the neuro-pathic bladder in spinal cord injured patients. *Spinal Cord* 2001;39:294-300.
27. Lynch AC, Antony A, Dobbs BR, Frizelle FA. Bowel dysfunction following spinal cord injury. *Spinal Cord* 2001;39:193-203.
28. Biering-Sørensen F, Bohr HH, Schaadt OP. Longitudinal study of bone mineral content in the lumbar spine, the forearm and the lower extremities after spinal cord injury. *Eur J Clin Invest* 1990;20:330-5.
29. Garland DE, Stewart CA, Adkins RH, Hu SS, Rosen C, Liotta FJ, et al. Osteoporosis after spinal cord injury. *J Orthop Res* 1992;10:371-8.
30. Massagli TL, Cardenas DD. Immobilization hypercalcemia treatment with pamidronate disodium after spinal cord injury. *Arch Phys Med Rehabil* 1999;80:998-1000.
31. Mohr T, Andersen JL, Biering-Sørensen F, Galbo H, Bangsbo J, Wagner A, et al. Long term adaptation to electrically induced cycle training in severe spinal cord injured individuals. *Spinal Cord* 1997;35:1-16.
32. Cruse JM, Lewis RE, Dilioglou S, Roe DL, Wallace WF, Chen RS. Review of immune function, healing of pressure ulcers and nutritional status in patients with spinal cord injury. *J Spinal Cord Med* 2000;23:129-35.
33. Ragnarsson KT. Management of pain in persons with spinal cord injury. *J Spinal Cord Med* 1997;20:186-99.

34. Schurch B, Wichmann W, Rossier AB. Post-traumatic syringomyelia (cystic myelopathy). A prospective study of 449 patients with spinal cord injury. *J Neurol Neurosurg Psychiatry* 1996;60:61-7.
35. Friden J. Reconstructive hand surgery after cervical spinal injury. A new method increases the possibilities to reconstruct important functions. *Läkartidningen [Swedish Medical Journal]* 1998;95: 3072-4.
36. Andrews BJ, Baxendale RH, Barnett R. Hybrid FES orthosis incorporating closed loop control and sensory feed-back. *J Biomed Eng* 1988;10:189-95.
37. Bajd T, Andrews BJ, Kralj A, Katakis J. Restoration of walking in patients with incomplete spinal cord injuries by use of surface electrical stimulation-preliminary results. *Prosthet Orthot Int* 1985;9:109-11.
38. Peckham PH, Creasey GH. Neural prostheses. Clinical applications of functional electrical stimulation in spinal cord injury. *Paraplegia* 1992;30:96-101.
39. Yang L, Granat MH, Paul JP, Condie DN, Rowley DI. Further development of hybrid functional electrical stimulation orthoses. *Spinal Cord* 1996;34:611-4.

46. Stress

Authors

Ingibjörg H. Jonsdottir, PhD, Associate Professor, Institute of Stress Medicine, Gothenburg, Sweden

Holger Ursin, MD, PhD, Professor Emeritus, Bergen University, Bergen, Norway

Summary

Stress can be defined as a state of increased psychological, physiological and behavioural preparedness. A stress reaction is a normal reaction that is considered necessary rather than harmful. Physical activity is generally deemed a stress factor that activates our physiological stress systems in the same way as exposure to psychological stress. The following chapter describes acute stress reactions and the effect that physical activity has on our well-being, control and stress management. It is impossible to describe stress in relation to an individual diagnosis. Accordingly, only the general effects of physical activity are covered here. For a more in-depth discussion on the prescription of physical activity, please refer to the related chapters on pain and depression.

Definition

Stress is a commonly used concept in physics, but a much more complicated concept in medicine with a number of alternative definitions. In 1936, one of the pioneers of stress research, Hans Selye, described an unspecific and general syndrome (1), which he later referred to as stress. Selye referred to a reaction or physical strain as stress, but, according to the rules of physics, the correct definition of stress is the actual exposure. Subsequently, a more appropriate term for Selye's syndrome would have been "strain". Today, the term "stress" is used to describe exposure (stressors) and an individual's reaction to this exposure.

Stress can be defined as a state of increased psychological, physiological and behavioural preparedness, i.e. the body's own alarm reaction. During stress, sensory impressions are processed by the brain, whereupon the brain's interpretation and coping with the situation influence the characteristics of a subsequent physiological stress reaction (2). The physiological stress reaction that is triggered is the body's way of responding and can be described as a survival reaction. Stress hormones are released to mobilise the energy needed

for coping with the stress, whereupon other systems that restore the balance are activated. A physiological stress reaction is therefore completely normal, helping the body to act and react appropriately to different threats and challenges. Our physiological stress system is adapted for a short period of activity. The risk of stress exposure leading to an illness only becomes relevant during long periods of exposure and when the stress level is so great that the physiological stress system is not given enough time, which all systems of the body need, to recover and function normally. It has been discussed whether this lack of recovery, often in the form of sleeplessness, is an important explanation to why some people suffer from fatigue/exhaustion, which regularly leads to reduced capacity and sick leave (3).

The physiological stress reaction and consequent adaptation are highly complex and influenced by a number of different factors that determine the degree of physiological activation and its consequences. Examples of such consequences include the ability to cope, the level of physical training, and sleep. Genetic factors and personality may also play an important role (2–7).

Prevalence/Incidence

Stress cannot be described as a specific diagnosis, making it impossible to discuss its prevalence. With regard to the increase in long-term sick leave seen in Sweden during the second half of the 1990s and beginning of the 2000s, most of the increase relates to diagnoses associated with mental illness. According to Swedish Social Insurance Agency statistics from 2006, the majority of sick leave taken in Sweden is due to pain diagnoses and mental illness. In 2005, close to 36 per cent of the sick leave taken was due to mental illness, a rise from approximately 28 per cent in 2002 (8). For December 2006, the two largest groups of mental illness were F32 (depressive episode) and F43 (adjustment disorders and reaction to severe stress), constituting approximately 22 per cent of the total number of diagnosed illnesses lasting 14 days or more (Source: Swedish Social Insurance Agency). A survey of 9000 county council employees from 1998 to 2004 showed that a total of 1483 people reported long-term sick leave (in excess of 28 days) during this 5-year period. The most commonly reported diagnoses were “musculoskeletal problems” (46%) and “psychiatric and stress-related complaints” (33%) (9).

Cause

According to Swedish Social Insurance Agency statistics, between 30–40 per cent of people on sick leave have been diagnosed with some form of mental illness. However, it is impossible at present to specify the percentage of mental illness and sick leave associated with stress.

Factors such as changes in working conditions including job cuts, reorganisations and added demands, are mentioned as contributors to the current ill-health in Sweden. An aging workforce, women working “double duty”, fewer people expected to perform more complex tasks, increasing demands in private life and, not least, the absence of recovery are other factors that may explain the escalated numbers of people on long-term sick leave

(10, 11). Studies from other countries also suggest that psychosocial factors may be a contributing cause of the increase in long-term sick leave (12–15). A recent survey among county council employees reported an escalating situation, with one in five participants reporting an increase in stress levels. At the same time, it is important to note that a large majority (over 80 per cent) of the participants felt that their general state of health was good or very good (16).

Diagnosis

Stress is not defined as a separate diagnosis, but is often mentioned as a contributing causal factor in diagnoses such as depression and different pain conditions. How big a role stress plays as a contributing cause is not scientifically documented. The Swedish National Board of Health and Welfare's report on Exhaustion Disorder (ED, F43.8) suggests that the disorder may serve as a model for diagnosing mental exhaustion caused by stress (17). Clinical experience indicates that patients who fulfil the criteria for UMS often display a high burden of disease, sleep disturbances and other conditions in addition to depression and anxiety (18).

An important part of the diagnostic criteria is that the patient must have had physical and psychological symptoms of exhaustion for a minimum of 2 weeks, with the symptoms developing as a result of one or more identified stress factors that must have existed for a minimum of 6 months. Accordingly, the criteria incorporate at least one identified stress exposure (17). Exhaustion Disorder is a relatively new diagnosis and research studies relating to this diagnosis are therefore lacking. Thus, the knowledge in this area is based solely on clinical experience.

Effects of physical activity

Since stress is not defined as a separate diagnosis, only the general effects of physical activity on our mental well-being, ability to cope and physiological stress systems are discussed here. Regular exercise has been shown to an effect on a variety of conditions where stress is considered as one of many contributing causal factors. Examples of such conditions are cardiovascular diseases, diabetes, depression and pain.

Effects of physical activity on psychological well-being

A number of studies have shown that psychological well-being can be influenced by physical activity/training, and it is relatively well-documented that individuals who exercise regularly have better mental health than those who do not (19–23). Psychological well-being is a multifaceted phenomenon that can be described and validated in many ways, and the effects can therefore vary depending on the outcome measures. Many of these studies are also cross-sectional studies of a relatively healthy population. Subsequently, there is a need for long-term follow-up studies and randomised intervention studies of

patients suffering from fatigue and mental illness where stress is a possible contributing causal factor (23).

A recently published study by Heiden and colleagues showed no difference between physical activity or cognitive behavioural therapy and so-called “standard” treatments in patients with long-term diagnoses such as exhaustion disorder or depression where stress was considered a possible contributing causal factor. The outcome measures were autonomic activity, sensitivity to pain, perceived stress and mental health. The physical activity consisted of two training sessions per week, one with low-intensity water exercises and one of the patient’s choice (strength training, aerobics, walking or swimming) (24). More treatment studies are needed in this area. However, looking at the research to date, a number of studies on patients with depression have shown that physical training is of benefit to these patients, and that the intensity and duration of the training may also be of relevance (see chapter on Depression).

Acute effects of physical activity on physiological stress systems

Physical activation is very much a stressor that affects the body’s physiological stress systems. Physiological stress reactions primarily involve the hypothalamic-pituitary-adrenal (HPA) axis and the autonomic nervous system (ANS), the body’s two fundamental physiological stress systems, and interact closely with one another. Corticotropin-releasing hormone (CRH) controls the release of adrenocorticotrophic hormone (ACTH), which in turn stimulates the release of cortisol from the adrenal cortex. The release of ACTH and cortisol may also be controlled by other factors and arginine-vasopressin (AVP) has been shown to be even more important than CRH in the release of ACTH during acute physical exercise (25).

Activation of the HPA axis in connection with physical training is a complicated process. It is influenced by a number of factors, such as the intensity of the training, what time of day the training takes place, and the intake and composition of any meals consumed before the training (25). An acute physiological stress activation is also influenced by psychological factors such as motivation and competition. The release of cortisol varies substantially throughout the day and night, meaning that two identical training sessions may result in a varying increase of cortisol levels depending on what time of day the training takes place. A higher training intensity usually leads to a greater activation of the HPA axis. The sympathetic part of the ANS that releases noradrenaline and adrenaline is activated mainly in connection with acute physical and psychological stress, thus leading to, among other things, increased blood pressure and heart rate. The release of adrenaline, noradrenaline and cortisol that occurs during physical training is similar to the release that occurs in connection with an acute psychological stress reaction. Psychosocial stress often gives rise to an increased heart rate and blood pressure, and, contrary to fitness training, it also leads to increased vascular resistance (25, 26).

Long-term effects of physical activity on physiological stress systems

It has long been known that regular exercise lowers resting heart rate and blood pressure (25). The long-term effects of fitness training mean that a given workload is less physiologically demanding for a well-trained person. Thus, a less pronounced increase in the blood pressure, heart rate, vascular resistance and stress hormone levels is observed during physical activity. Also, compared to an untrained individual, the levels of catecholamines and cortisol released in a well-trained individual are lower when performing a physical activity of exactly the same intensity. In addition, well-trained individuals tend to display a less pronounced physiological stress activation in connection with a psychosocial stress load (4, 27–29). Regular activation of physiological stress systems by way of physical activity should benefit the systems even during psychosocial stress (30). The notion that regular training has an effect on individual stress reactions is based in part on the physiological mechanisms activated in connection with physical activity, such as sensitivity to hormones and influences on autonomic function (31, 32). However, physiological stress reactions are also influenced by an individual's psychological well-being and ability to cope.

Effects of physical activity on coping

Humans (and animals) may react differently to identical loads. The reason for this is that we assess a situation differently – what it means and what to do. This is in turn influenced by our personal experiences, expectations and actions. Physical activity has a positive effect on our expectations and actions in a specific situation. This positive expectation is defined as “coping”, i.e. how we decide to solve a problem in a certain situation. A stress reaction is very much affected by the expected result. An individual who expects a positive outcome from a difficult situation tends to have a much less pronounced physiological stress reaction. Similarly, an individual who expects the “worst” outcome from a situation tends to display a higher level of physiological stress activation. This expectation is a learned behaviour and is often generalised to similar future situations (2).

The psychological well-being achieved through physical activity, providing the activity is perceived as positive, can in other words “spread” to other situations and thereby affect the actual stress reaction. Physical activity that is perceived as negative can likewise have a negative effect on other situations. An example of this are the expectations of parents and coaches on a child's training progress. If the objective of the training is to create a champion, then the child may perceive the situation (i.e. the training) as negative, with negative consequences for the training.

Controlled studies have shown that physical activity with realistic expectations and objectives may have an effect on the well-being and subjective health of an individual (33), regardless of the type of training carried out to achieve these expectations and objectives, at least in patients with muscle and back pain (www.backpaineurope.org).

Effects of physical activity on the brain

Long-term stress is thought to affect the functioning of the brain, including that of the hippocampus, which can lead to impaired regulation of the HPA axis and cognitive disturbances associated with memory loss and impaired learning (34–36). Some clinical observations and recently published studies indicate that long-term stress may have an effect on cognitive functions including the memory function (37, 38), but more scientific studies are needed to confirm this. A less pronounced reaction of the HPA axis resulting from physical training should therefore be good for the brain and any cognitive problems associated with long-term stress. However, randomised treatment studies are needed to confirm this. An interesting aspect of hippocampus function is the ability of nerve cells to regenerate in the adult brain (39). It is now well-documented, in animal experiments, that physical activity has a positive effect on cell proliferation in the hippocampus (40, 41). However, the clinical significance of the regenerated cells remains unclear.

Prescription

As this chapter does not address a particular diagnosis and there are no studies on the treatment of exhaustion disorder, the reader is referred to the prescriptions recommended for depression and pain diagnoses where stress may be one of many contributing causal factors.

References

1. Selye H. A syndrome produced by diverse nocuous agents. *Nature* 1936;138:32.
2. Ursin H, Eriksen HR. The cognitive activation theory of stress. *Psychoneuroendocrinology* 2004;29:567-92.
3. Åkerstedt T. Sömn som återhämtning efter stress. [Sleep as recovery after stress.] *Läkartidningen* 2004;101:1501-5.
4. Traustadottir T, Bosch PR, Matt KS. The HPA axis response to stress in women. Effects of aging and fitness. *Psychoneuroendocrinology* 2005;30:392-402.
5. Wirtz PH, Elsenbruch S, Emini L, Rudisuli K, Groessbauer S, Ehlert U. Perfectionism and the cortisol response to psychosocial stress in men. *Psychosom Med* 2007;69:249-55.
6. Oswald LM, Zandi P, Nestadt G, Potash JB, Kalaydjian AE, Wand GS. Relationship between cortisol responses to stress and personality. *Neuropsychopharmacology* 2006;31:1583-91.
7. Wust S, Van Rossum EF, Federenko IS, Koper JW, Kumsta R, Hellhammer DH. Common polymorphisms in the glucocorticoid receptor gene are associated with adrenocortical responses to psychosocial stress. *J Clin Endocrinol Metab* 2004;89:565-73.
8. Nybeviljade sjukersättningar/aktivitetsersättningar. [Newly granted statutory sickness pay/activity compensation.] Fördelning på län och diagnos, 2003-2005. [Distributed by county and diagnosis.] Report 2006:3. Stockholm: Swedish Social Insurance Agency; 2006.
9. Hållbar arbetshälsa i kommuner och landsting, oktober 2004. Stockholm: [Sustainable occupational health in municipalities and county councils] Department of Personal Injury Prevention, Karolinska Institutet; 2004.
10. Den höga sjukfrånvaron [The high level of sickness absence]. Sanning och konsekvens [Truth and consequence] Stockholm: Swedish National Institute of Public Health; 2004.
11. Theorell T. I spåren av 90-talet. [In the aftermath of the 1990s] Stockholm: Karolinska Institutet University Press; 2006.
12. Mental Health. Facing the challenges, building solutions. Copenhagen: WHO; 2005.
13. Bultmann U, Huibers MJ, van Amelsvoort LP, Kant I, Kasl SV, Swaen GM. Psychological distress, fatigue and long-term sickness absence. Prospective results from the Maastricht Cohort Study. *J Occup Environ Med* 2005;47:941-7.
14. Henderson M, Glozier N, Holland Elliott K. Long term sickness absence. *BMJ* 2005;330:802-3.
15. Melchior M, Niedhammer I, Berkman LF, Goldberg M. Do psychosocial work factors and social relations exert independent effects on sickness absence? A six year prospective study of the GAZEL cohort. *J Epidemiol Community Health* 2003;57:285-93.
16. Ahlborg GJ, Ljung T, Swan G, Glise K, Jonsdottir IH, Hadzibajramovic E, et al. Stressrelaterad ohälsa bland anställda vid västra Götalandsregionen och Försäkringskassan i Västra Götalands län. [Stress-related illness in employees in the West Götaland Region and at the Swedish Social Insurance Agency in West Götaland]. Delrapport 1. Enkätundersökning i maj-juni 2004. Göteborg: [Interim Report 1. Questionnaire survey, May-June 2004] Institute of Stress Medicine; 2006.

17. Utmattningsyndrom. [Exhaustion Disorder] Stressrelaterad psykisk ohälsa. [Stress related psychological illnesses] Underlag från experter. [Expert evidence] Report 2003-123-18. Stockholm: Swedish National Board of Health and Welfare; 2003.
18. Glise K, Björkman A. Utmattningsyndromet. Klinisk bild och terapi. [Exhaustion Disorder. Clinical picture and therapy.] *Läkartidningen* 2004;101:1202-6.
19. Galper DI, Trivedi MH, Barlow CE, Dunn AL, Kampert JB. Inverse association between physical inactivity and mental health in men and women. *Med Sci Sports Exerc* 2006;38:173-8.
20. Hassmén P, Hassmén N. Hälsosam motion. [Healthy exercise.] Stockholm: SISU Idrottsböcker; 2005.
21. Hassmen P, Koivula N, Uutela A. Physical exercise and psychological well-being. A population study in Finland. *Prev Med* 2000;30:17-25.
22. Netz Y, Wu MJ, Becker BJ, Tenenbaum G. Physical activity and psychological well-being in advanced age. A meta-analysis of intervention studies. *Psychol Aging* 2005;20:272-84.
23. Penedo FJ, Dahn JR. Exercise and well-being. A review of mental and physical health benefits associated with physical activity. *Curr Opin Psychiatry* 2005;18:189-93.
24. Heiden M, Lyskov E, Nakata M, Sahlin K, Sahlin T, Barnekow-Bergkvist M. Evaluation of cognitive behavioural training and physical activity for patients with stress-related illnesses. A randomized controlled study. *J Rehabil Med* 2007;39:366-73.
25. Borer KT. Exercise endocrinology. Champaign (IL): Human Kinetics; 2003.
26. Goldberg AD, Becker LC, Bonsall R, Cohen JD, Ketterer MW, Kaufman PG, et al. Ischemic, hemodynamic and neurohormonal responses to mental and exercise stress. Experience from the Psychophysiological Investigations of Myocardial Ischemia Study (PIMI). *Circulation* 1996;94:2402-9.
27. Peronnet F, Cleroux J, Perrault H, Cousineau D, de Champlain J, Nadeau R. Plasma norepinephrine response to exercise before and after training in humans. *J Appl Physiol* 1981;51:812-5.
28. Georgiades A, Sherwood A, Gullette EC, Babyak MA, Hinderliter A, Waugh R, et al. Effects of exercise and weight loss on mental stress-induced cardiovascular responses in individuals with high blood pressure. *Hypertension* 2000;36:171-6.
29. Rimmel U, Zellweger BC, Marti B, Seiler R, Mohiyeddini C, Ehlert U, et al. Trained men show lower cortisol, heart rate and psychological responses to psychosocial stress compared with untrained men. *Psychoneuroendocrinology* 2007;32:627-35.
30. Tsatsoulis A, Fountoulakis S. The protective role of exercise on stress system dysregulation and comorbidities. *Ann N Y Acad Sci* 2006;1083:196-213.
31. Charlton GA, Crawford MH. Physiologic consequences of training. *Cardiol Clin* 1997;15:345-54.
32. Duclos M, Gouarne C, Bonnemaïson D. Acute and chronic effects of exercise on tissue sensitivity to glucocorticoids. *J Appl Physiol* 2003;94:869-75.

33. Eriksen HR, Ihlebaek C, Mikkelsen A, Gronningsaeter H, Sandal GM, Ursin H. Improving subjective health at the worksite. A randomized controlled trial of stress management training, physical exercise and an integrated health programme. *Occup Med (Lond)* 2002;52:383-91.
34. McEwen BS, Sapolsky RM. Stress and cognitive function. *Curr Opin Neurobiol* 1995;5:205-16.
35. Sapolsky RM. Stress hormones. Good and bad. *Neurobiol Dis* 2000;7:540-2.
36. Sapolsky RM. Stress, glucocorticoids and damage to the nervous system. The current state of confusion. *Stress* 1996;1:1-19.
37. Sandstrom A, Rhodin IN, Lundberg M, Olsson T, Nyberg L. Impaired cognitive performance in patients with chronic burnout syndrome. *Biol Psychol* 2005;69:271-9.
38. Rydmark I, Wahlberg K, Ghatan PH, Modell S, Nygren A, Ingvar M, et al. Neuroendocrine, cognitive and structural imaging characteristics of women on longterm sickleave with job stress-induced depression. *Biol Psychiatry* 2006;60:867-73.
39. Eriksson PS, Perfilieva E, Bjork-Eriksson T, Alborn AM, Nordborg C, Peterson DA, et al. Neurogenesis in the adult human hippocampus. *Nat Med* 1998;4:1313-7.
40. van Praag H, Kempermann G, Gage FH. Running increases cell proliferation and neurogenesis in the adult mouse dentate gyrus. *Nat Neurosci* 1999;2:266-70.
41. Bjornebekk A, Mathe AA, Brene S. The antidepressant effect of running is associated with increased hippocampal cell proliferation. *Int J Neuropsychopharmacol* 2005;8:357-68.

47. Stroke

Authors

Gunnar Grimby, MD, PhD, Professor Emeritus, Department of Clinical Neuroscience and Rehabilitation, Institute of Neuroscience and Physiology, Sahlgrenska Academy, Gothenburg University, Gothenburg, Sweden

Carin Willén, PT, PhD, Department of Occupational Therapy and Physiotherapy, Institute of Neuroscience and Physiology, Sahlgrenska Academy, Gothenburg University, Gothenburg, Sweden

Margareta Engardt, PT, PhD, Stockholm, Sweden

Katharina Stibrant Sunnerhagen, MD, PhD, Professor, Sahlgrenska Academy, Gothenburg University, Gothenburg, Sweden, Sunnaas Rehabilitation Hospital, Faculty of Medicine, Oslo University, Oslo, Norway

Summary

People with residual symptoms after a stroke are deconditioned and have reduced physical capacity. Many suffer varying degrees of disability after a stroke. However, many stroke patients are able to adapt and remain physically active. The benefits of strength and aerobic fitness training for stroke patients have previously been the subject of much debate. Exertion was considered contraindicated due to the risk of triggering spasticity. However, none of the studies published over recent years have been able to confirm that this is the case. Strength training of the lower extremities has been shown to yield significantly improved functions. Aerobic fitness training improves the tolerance for daily activities as it enables people who have had a stroke to carry out day-to-day activities with less exertion.

Definition

The World Health Organization (WHO) defines stroke as a condition characterised by rapidly developing symptoms that last for more than 24 hours or lead to death, with no apparent cause other than that of vascular origin. There are three main causes of stroke: 1) brain infarction, representing approximately 85 per cent of all strokes in Scandinavia and usually due to cardiac embolism (approx. 30%), macrovascular disease (approx. 25%)

and microvascular injury (approx. 20%). Other causes represent approximately 5 per cent of all strokes, and the remainder of unidentified causes. The other two most common causes are: 2) cerebral haemorrhage (10%), and 3) subarachnoid haemorrhage (5%), resulting from a ruptured arterial aneurysm. The third cause often presents completely differently than the first two, as the damage to the brain can be diffuse.

Prevalence/Incidence

Each year, approximately 30,000 people in Sweden suffer a stroke, of whom 20,000 for the first time (1). The average age of stroke victims is 75 years (men 73 years, women 77 years). However, stroke also affects younger age groups, with 20 per cent of strokes occurring in people under the age of 65. The incidence is also higher in men than women. This particular somatic disease group represents the highest number of clinical care days at Swedish hospitals and is the most common cause of neurological disabilities. The reported prevalence totals approximately 100,000, of whom 20,000 require considerable levels of assistance (1). Thus a large number of stroke sufferers are left with varying degrees of post-stroke disability. However, with some adaptation, many of these people can continue to be physically active.

Symptomatology

Different functions in the brain are affected depending on where the injury is located. It is common that the motor centre of the brain is affected, resulting in some degree of hemiparesis, along with reduced sensibility, impaired balance and coordination, as well as speech and vision disturbances. Stroke patients may also display reduced cognitive capacity, denial of the affected side of the body, depression and emotional disturbances, as well as different types of pain.

Treatment principles

Space does not permit a detailed description of the treatment principles associated with stroke here in this chapter. For this, the reader is referred instead to clinical guidelines on the topic (1–4). Recent research has clearly shown that the best initial treatment and early rehabilitation measures are those provided by special stroke units that use a multidisciplinary approach (5). The work of the stroke units should then be followed by an efficient chain of care that provides continued rehabilitation and medical follow-up at special rehabilitation centres, as well as in primary care, municipal care, and through home-care services. Physical activities should also be made easier for stroke patients, for example, by providing them access to primary care centres, gyms and health clubs where they can train to improve their strength, aerobic fitness, balance, coordination and relaxation, in enjoyable, modified exercise programmes.

Frequent physiotherapy may also be necessary. Despite paralysis/muscular weakness and loss of fine motor ability being typical residual symptoms after a stroke, there has been much uncertainty as to the benefits of strength and aerobic fitness training for stroke patients. Exertion was considered contraindicated due to the risk of triggering spasticity. However, no recently published studies have been able to confirm that this is the case (6–10). Strength training of the lower extremities has been shown to significantly improve functioning in stroke patients without leading to increased spasticity. The participation of different occupational groups is required in order to maintain the ability to take part in different home activities. Special rehabilitation programmes often enable younger stroke patients to return to work and even resume previous leisure activities.

Effects of physical activity

Once stroke patients have gone through the initial period of rehabilitation, an aerobic fitness training programme can improve their endurance and functional capacity. This can also result in increased self-confidence to activate themselves and take part in physical activities (10, 11).

Training on a treadmill has been used successfully to increase maximal oxygen uptake in stroke patients. Twenty-five people (12) who had suffered a stroke at least 6 months earlier and who still had a hemiparetic gait took part in 40-minute sessions on the treadmill, 3 times a week for 6 months. The results showed an increase in maximal oxygen uptake (peak VO_2) and a decrease in energy expenditure at the same exertion level compared to a control group of 20 people with only low-intensity training. Readers are also referred to a Cochrane Review showing the need for additional studies in order to determine which type of training yields the greatest fitness results. However, improved aerobic fitness does appear to lead to improved walking ability (13).

Physical endurance was improved and heart rate lower at constant load, after 12 weeks of cycle training (14). In addition, the study subjects reported an improved self-perception and general well-being. The increased self-confidence and endurance also seemed to give individuals more confidence and energy to improve other activities as well.

A combined strength and aerobic fitness training programme involving 35 study subjects, all of whom had suffered a stroke at least 6 months earlier and had multiple comorbidities (concurrent illnesses) led to significant improvement in maximal oxygen uptake, increased muscle strength, and weight loss (15).

The studies described below exemplify the effects of physical activity in stroke patients. However, the number of subjects in several of these studies was relatively small. More studies are therefore needed. For further information about the background and effects of fitness training, please refer to subject review articles (17, 18).

Table 1. The effect of fitness and strength training in stroke patients.

Form of training	Intensity	Frequency (times/week)	Duration	Measured pulse (n)	Result	Design	Ref.
Fitness							
Bicycle training (ergometer) Control group: relaxation	60–80% of max HR*	3	30 min. 12 weeks	142	↑ Work load → Functionally independent	RCT**	
Bicycle training	Progressing to → 70% max HR	3	30 min. 12 weeks	42	↑ 13% of VO ₂ max ↓ Blood pressure ↑ Muscular strength → Spasticity	RCT	11
Walking on treadmill	60–70% of max HR	3	40 min. 12 weeks	21	↑ Peak VO ₂ ↓ Energy consumption (20%)	***	12
Circuit training	Functional strength and mobility	3	60 min. 4 weeks	12	↑ Walking endurance ↑ Walking velocity ↑ No. of step tests	RCT	16
Strength							
Weight lifting machines	Reciprocal knee extension/flexion	3	40 min. 6 weeks	15	↑ Muscle strength ↑ Walking capacity ↑ Physical activity → Spasticity	***	7
Isokinetic training Control group: Concentric training	Eccentric training	2	40 min. 6 weeks	20	↑ Except muscle strength ↑ Paretic leg load ↑ Walking velocity → Spasticity	RCT	
Combined fitness and strength							
Circuit training	Aerobics Strength training, lower extremities	3	60–80 min. 12 weeks	13	↑ Muscle strength ↑ Walking velocity ↑ Physical activity ↑ Life quality → Spasticity	RCT	9
Circuit training	30 min. fitness 30 min. strength 20 min. flexing	3	60–80 min. 12 weeks	35	↑ Peak VO ₂ ↑ Muscle strength ↑ Hamstring/ low back flexibility	RCT	15

* Max HR = Maximal Heart Rate (220 – age ± 12).

** RCT = Randomised Controlled Trial, i.e. a trial with a randomly selected study and control group.

*** = Not controlled study.

**** = Peak VO₂ (maximal oxygen uptake).

Since many stroke patients have comorbidities, i.e. concurrent illnesses, such as diabetes, hypertension or cardiovascular disease, the possibility of physical activity and the effects of such activity are also dependent on these other conditions. The effect of muscular training depends ultimately on how well the patient regains motor control. The degree of paralysis, sensory disturbances, balance, etc., in addition to comorbidity, determine the type of activity possible. For further information about the background and effects of strength training, please refer to subject review articles (19, 20).

Indications

Physical activity has a documented *primary preventive* effect against cardiovascular diseases. This primary prevention of stroke is described in a study on 11,000 American men with an average age of 58. Those who walked 20 km a week were at significantly lower risk of suffering a stroke at follow-up 11 years later (21). Four independent cohort studies show a reverse and dose-dependent relationship between physical activity and the risk of stroke, i.e. a little is better than none at all, and a lot is better than a little. Two other studies revealed a reverse but not dose-dependent relationship between physical activity and the risk of stroke (22).

Although the degree of paralysis and sensory disturbances may vary from near normal function to severe disability and significantly reduced mobility, the same principal indications apply to stroke patients as to healthy individuals, i.e. that any physical activity not restricted by disability will improve muscle function and general fitness. Since vascular disease is common in stroke patients, the indications for secondary prevention in these people are generally consistent with the indications for these diseases, as well as for diabetes and hypertension. However, scientific evidence showing that physical activity itself has a *secondary preventive* effect on stroke recurrence is lacking.

Prescription

At present, there are limited possibilities for continued training once stroke patients have been discharged from a hospital or rehabilitation clinic. Many stroke patients have residual symptoms and find it difficult to keep up with a normal exercise class or other desired physical activity. The risk of feeling dejected and experiencing a lower quality of life due to reduced fitness and strength can easily be avoided by creating opportunities for stroke patients with residual symptoms to continue training.

Normal activities that the individual enjoys are recommended, such as walking, climbing stairs, dancing, circuit training, gardening, arm and leg pedaling, training on the exercise bike, walking on the treadmill, wheelchair exercising, group exercise classes and water aerobics. Exercise that can be done with others is both socially and psychologically stimulating. However, the intensity of the training should be adapted to the individual and relevant symptoms. It is important to remember that, along with more organised physical activity/training,

everyday physical activities are also very valuable. Such activities may consist of gardening, household chores, going for a walk or playing with one’s grandchildren. If the intensity of the activity is such that the patient is slightly out of breath but can still carry on a conversation, it is quite sufficient for attaining the desired effect and maintain endurance.

The relative heart rate (percentage of max HR), level of exertion, or degree of breathlessness experienced by the patient can be used as a guide when deciding on an appropriate training intensity. Using relative heart rate in patients treated with beta blockers may be difficult, however, since beta blockers reduce both maximal heart rate and heart rate during submaximal exercise.

According to Åstrand, calculations of maximal oxygen uptake (“aerobic fitness value”) based on submaximal exercise testing are also misleading. The same applies to people with atrial flutter. Consequently, the best guide is the subjective perceived level of exertion (Rating of Perceived Exertion, RPE). (Please refer to the chapter on Assessing and controlling physical activity). Measuring the maximal oxygen uptake is usually not possible as motor functions and cardiac limitations, if present, do not allow for maximal exercise testing.

Table 2. Guidelines for prescribing physical activity to stroke patients.

Form of training	Activity	Intensity	Frequency (times/week)	Duration
Fitness training	Walking	60–80% max	2–5	10–60 min./session 4–6 months – throughout life
	Nordic pole walking:	HR*		
	Circuit training	12–15 RPE**		
	Ergometer bicycle training	Slightly to moderately out of breath		
	Arm/leg cycling			
	Walking on treadmill			
	Step training			
	Water exercises:			
	Dancing			
	Wheelchair driving			
Strength training	Weight lifting machines, e.g. leg press	Start with 50%, increase to 70–80% of RM***	1–3 Increase: increase load, not number of repetitions	1–3 sets of 7–10 reps 10–12 weeks
	Eccentric/concentric training			
	Isokinetic training	12–13 RPE**		
	Functional training			
Muscular endurance training	Circuit training	30–50% of 1 RM***	1–5	3 sets of 25–50 reps (dose-response)
	Sequence training			
	Walking/moving	9–11 RPE**		
Functional training	Balance and coordination training	Increase level of intensity	1–3	
Flexibility	Warm up		With every type of training	
	Cool down			
	Stretch Flex			

* Max HR = Maximal Heart Rate (220 – age ± 12).

** RPE = Rating of Perceived Exertion = subjective perceived level of exertion according to the Borg scale 6–20.

*** RM = Repetition Maximum. 1 RM is the maximum amount of weight one can lift in a single repetition for a given exercise.

Functional mechanisms

Oxygen uptake and cardiac function

The functional mechanisms may vary and be dependent on the possible presence of other disease. A concurrent cardiovascular disease may dominate the functional mechanisms of physical activity and training. If no other diseases are present, as in the case of residual lesion following a subarachnoid hemorrhage in younger stroke patients, the functional mechanisms should be identical to those found in untrained healthy individuals of the same age.

Skeletal muscle function

Strength training facilitates motor unit recruitment and increases discharge rate (23). In order to achieve power, timing and coordination of the muscles stroke patients must be given the opportunity to train at an adequate intensity, frequency and duration. To begin with (6–8 weeks), the increased muscle strength obtained through physical training is the result of neural adaptation (increased recruitment of motor units, less inhibition, improved coordination, reduced coactivation, etc.). Muscle hypertrophy, whereupon muscles cells increase in size, occurs at a later stage. The muscle strength of paralysed stroke patients can be improved by combining eccentric and concentric training methods (6). Using the Stretch-Shortening Cycle (SSC, eccentric/concentric muscle contractions) (24) in a closed muscle chain when training the weight-bearing muscles of the lower extremities (e.g. sit-to-stand position, walking up and down stairs) may have a positive effect on motor functions.

Peripheral muscle endurance

Muscular endurance training leads to increased levels of mitochondria, oxidative enzymes, myoglobin and capillarisation (25).

Aerobic fitness

With an improved aerobic fitness level comes an increased ability to manage everyday activities at a lower percentage of maximal oxygen uptake (lower relative load). Physical activities can then be carried out at a lower heart rate and lower systolic blood pressure. An ineffective movement pattern resulting in increased energy expenditure during activity may be improved by training, also reducing the level of exertion. Aerobic fitness training has a positive effect on the risk factors for cardio- and cerebrovascular diseases.

Functional tests and health controls

An assessment of motor functions should be carried out before any advice is given on physical activity. This is best performed by a physiotherapist using one or more of the assessment tools currently available such as those by Fugl-Meyer and Lindmark, the Rivermead Mobility Index, or the 10-meter Walk Test. If prescribed a certain strength training, the muscle strength of the patient should be measured with a dynamometer. The degree of muscle tone and balance should also be assessed. Mobility and presence of contractures should also be noted. If a patient with concurrent heart disease (angina pectoris, status post heart attack, heart failure and arrhythmia tendencies) is following a specific fitness training programme, the patient's cardiovascular function should be assessed by the physician in charge and include ECG at rest and during exercise.

Table 3 shows clinical test methods used for the assessment of physical ability.

Table 3. Clinical tests for the assessment of physical ability in stroke patients.

Fitness	Standardised ergometer cycling test during which the load (watt), time (minutes), velocity (revolutions/minute), pulse and blood pressure are registered by the test monitor. Perceived exertion (Borg RPE scale), leg tiredness and pain, if any (Borg CR10 scale), are evaluated by the test subject. Pulse and blood pressure are measured while resting before the test and then again 15 minutes after the test. NB! Åstrand's sub-maximal test cannot be carried out on persons taking beta blockers or suffering with atrial flutter.
Muscle strength	1 RM for different muscle groups. Hand held dynamometer. Isometric or isokinetic measuring.
Muscular endurance	Functional endurance test (for example, the number of symmetrical rising/sitting down, heel raising, step tests).
Functional walking capacity	6-minute walking test. The walking distance is measured and perceived leg tiredness and breathlessness assessed according to the Borg CR10 scale. Perceived exertion is also assessed according to the Borg RPE scale. Pulse (HF) and blood pressure should be taken before and after the walking test.

Interaction with drug therapy

Most stroke survivors are prescribed a number of medications. Prophylactic treatments with blood thinners (anticoagulants) to prevent new thromboses or embolisms are commonly used and do not affect one's ability to exercise or recommendations regarding physical activity. In the case of hypertension and heart disease, different medications, such as beta blockers, which reduce the submaximal and maximal heart rate, may have an effect on the person's physiological reaction to physical activity. See also recommendations for physical activity in patients with hypertension and heart disease. Some stroke sufferers

also have diabetes which must naturally be given specific consideration. Antidepressant treatment, such as SSRIs, need not be limiting, but rather may have a synergistic effect on physical activity.

Contraindications and risks

Contraindications to physical activity and exercise are primarily associated with other diseases. (See above.)

With the exception of inappropriate dose-intensity in patients with a cardiovascular disease and the potential triggering of severe angina or arrhythmia, the risks involved with physical activity relate mainly to the increased risk of falling due to impaired motor function and balance. Stroke patients are 2–4 times more likely to fall or suffer hip fractures (26). They are also at greater risk of getting fractures due to osteoporosis as a result of immobilisation and other osteoporosis risk factors. Closer supervision of at-risk patients during physical activity is therefore important, as well as considering the use of hip protectors.

References

1. Swedish National Board of Health and Welfare. Nationella riktlinjer för stroke-sjukvård [National Clinical Guidelines for Stroke] Stockholm: Swedish National Board of Health and Welfare; 2005.
2. Barnes MP, Dobkin BH, Bogousslavsky J. Recovery after stroke. Cambridge (US): Cambridge University Press; 2005. (ISBN 052182236X).
3. Scottish Intercollegiate Guidelines Network. Management of patients with stroke. Rehabilitation, preventions and management of complications, and discharge planning. <http://www.sign.ac.uk/guidelines/fulltext/64/index.html>.
4. National Clinical Guidelines for Stroke. 2nd edn. Prepared by the Intercollegiate Stroke Working Party. <http://www.rcplondon.ac.uk/pubs/contents/78ba394c-c09a-4fce-bdf4-bcd49b33e650.pdf>.
5. Stroke Unit trialists' Collaboration. Organised inpatient (stroke unit) care for stroke. *Cochrane Database Syst Rev* 2000;(2):CD000197.
6. Engardt M, Knutsson E, Jonsson M, Strenhag M. Dynamic muscle strength training in stroke patients. Effects on knee extension torque, electromyographic activity and motor function. *Arch Phys Med Rehabil* 1995;76:419-25.
7. Sharp SA, Brouwer BJ. Isokinetic strength training of the knee. Effects on function and spasticity. *Arch Phys Med Rehabil* 1997;78:1231-6.
8. Brown DA, Kautz SA. Increased workload enhances force output during pedaling exercise in persons with poststroke hemiplegia. *Stroke* 1998;29:598-606.
9. Teixeira-Salmela LF, Olney SJ, Nadeau S, Brouwer B. Muscle strengthening and physical conditioning to reduce impairment and disability in chronic stroke survivors. *Arch Phys Med Rehabil* 1999;80:1211-8.
10. Bateman A, Culpan FJ, Pickering AD, Powell JH, Scott OM, Greenwood RJ. The effect of aerobic training on rehabilitation outcomes after recent severe brain injury. A randomized controlled evaluation. *Arch Phys Med Rehabil* 2001;82:174-82.
11. Potempa K, Lopez M, Braun LT, Szidon JP, Fogg L, Tincknell MS. Physiological outcomes of aerobic exercise training in hemiparetic stroke patients. *Stroke* 1995;26:101-5.
12. Macko RF, Smith GV, Dobrovolny CL, Sorkin JD, Goldberg AP, Silver KH. Treadmill training improves fitness in chronic stroke patients. *Arch Phys Med Rehabil* 2001;82: 879-84.
13. Saunders DH, Greig CA, Young A, Mead GE. Physical fitness training for stroke patients. *Cochrane Database of Systematic Reviews* 2004;1. Art. No. CD003316. DOI: 10.1002/14651858.CD003316.pub2.k.
14. Brinkmann J, Hoskins T. Physical conditioning and altered self-concept in rehabilitated hemiplegic patients. *Phys Ther* 1979;59:859-65.
15. Rimmer JH, Riley B, Creviston T, Nicola T. Exercise training in a predominantly African-American group of stroke survivors. *Med Sci Sports Exerc* 2000;32:1990-6.
16. Dean CM, Richards CL, Malouin F. Task-related circuit training improves performance of locomotor tasks in chronic stroke. A randomized controlled pilot trial. *Arch Phys Med Rehabil* 2000;81:409-17.

17. Pang MY, Eng JJ, Dawson AS, Gylfadóttir S. The use of aerobic exercise training in improving aerobic capacity in individuals with stroke. A meta-analysis. *Clinical Rehabilitation* 2006;20:97-111.
18. Gordon N, et al. Physical activity and exercise recommendations for stroke survivors. An American heart association scientific statement from the council of clinical cardiology. *Stroke* 2004;18:27-39.
19. Patten C, Lexell J, Brown H. Weakness and strength training in persons with post-stroke hemiplegia. Methods and efficacy. *J Rehabil Res Dev* 2004;41:293-312.
20. Bohannon R. Muscle strength and muscle training after stroke. *J Rehabil Med* 2007; 39:14-20.
21. Lee I-M, Pfaffenberger RS. Physical activity and stroke incidence. *Stroke* 1998;29: 2049-54.
22. US Department of Health and Human Services. Physical Activity and Health. A Report of the Surgeon General. Atlanta (GA): US Department of Health and Human Services, Centers for Disease Control and Prevention, National Center for Chronic Disease, Prevention and Health Promotion; 1996.
23. Sale DG. Neural adaptation to resistance training. *Med Sci Sports Exerc* 1988;20: S135-45.
24. Svantesson U, Sunnerhagen KS. Stretch-shortening cycle in patients with upper motor neuron lesion due to stroke. *Eur J Appl Physiol Occup Physiol* 1997;75:312-8.
25. Wilmore JH, Costill DL. Physiology of sport and exercise. Champaign (IL): Human Kinetics; 1994.
26. Ramnemark A, Nyberg L, Lorentzon R, Olsson T, Gustafson Y. Hemiosteoporosis after severe stroke, independent of changes in body composition and weight. *Stroke* 1999; 30:755-60.

Physical activity in the prevention and treatment of disease summarizes the up-to-date scientific knowledge on how to prevent and treat various diseases and conditions using physical activity. The book covers most areas of disease where physical activity has a documented effect. By combining recommendations on suitable exercise activities with a description of the potential risks of physical activity for various patient groups, this handbook can comprehensively be used by anyone working with physical activity and health.

Content:

Thirty-three chapters address the effects of and recommendations of physical activity in diseases and conditions within cardiovascular and metabolic medicine, psychiatry, orthopaedics, neurology, gastro-intestinal medicine, nephrology, rheumatology, pulmonary medicine and more.

Also, fourteen general chapters deal with:

- General effects of physical activity
- General recommendations regarding physical activity
- Promoting physical activity
- Becoming physically active
- Motivational interviewing about physical activity
- Assessing and controlling physical activity
- Various types of physical activity and exercise
- Health aspects of strength training
- Infections and sports
- Sports and sudden death
- Children and young people
- Pregnancy
- Menopause
- Elderly

The handbook is especially tailored to be a tool for licensed healthcare staff when prescribing physical activity and the method is currently used by all county councils in Sweden as well as Norway. The book is also useful for physical activity organisers working with physical activity on prescription and for educational institutions, such as colleges and universities that focus on health sciences and public health.

This book was prepared by Professional Associations for Physical Activity in cooperation with the Swedish National Institute of Public Health (SNIPH). Professional Associations for Physical Activity (see www.fyss.se) constitute an independent sub-association of the Swedish Society of Sports Medicine (Svensk Idrottsmedicinsk Förening – SIMF) which in turn is a section within the Swedish Society of Medicine.



Swedish National Institute of **Public Health**

Swedish National Institute
of Public Health
Distribution
SE-120 88 Stockholm

Fax 08-449 88 11
fhi@strd.se
www.fhi.se

R 2010:14
ISSN 1651-8624
ISBN 978-91-7257-715-2