Residual Limb Pain Is Not a Diagnosis
A Proposed Algorithm to Classify Postamputation Pain

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Background: Although postamputation pain (PAP) syndromes have been described since the 16th century, taxonomy of these conditions remains ill-defined. The term "Residual Limb Pain" fails to distinguish between distinct diagnostic entities such as neuroma, complex regional pain syndrome, and somatic pathology. Even phantom limb pain (PLP), although easily distinguished from residual limb pain (RLP), has not been consistently delineated from other PAP syndromes.

Methods: A systematic review of the literature was conducted to identify the degree of delineation of various post amputation pain states and what diagnostic criteria were utilized if any. Furthermore, papers that involved treatment modalities were reviewed to determine efficacy of treatment.

Results: Of the 151 papers reviewed, none further categorized RLP into more specific diagnostic criteria. Furthermore, the literature contains numerous case reports, case series, letters to the editors, and grossly underpowered studies demonstrating significant positive results, yet few high-quality randomized controlled trials.

Conclusions: Describing and defining the distinct clinical entities, intuitively, is a prerequisite to developing optimal treatments. The reported variation in the incidence of PAP phenomena may well represent inconsistency in assessment tools and diagnostic categories rather than variation in prevalence of these conditions. In this paper, we review the historical evolution of the current understanding of these syndromes and propose an algorithm for uniform classification.

Key Words: residual limb pain, stump pain, phantom limb pain, postamputation pain, pain taxonomy

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Although the postamputation syndromes of phantom limb pain (PLP) and residual limb pain (RLP) are not new to the human condition, formal discussions of these afflictions were not noted in the medical literature until the mid-16th century. At that time, the French military surgeon Ambroise Paré noted that months after amputation, soldiers continued to complain of pain in the missing limb. In addition to the original descriptions of PLP, he contributed detailed descriptions of RLP to early medical texts.[1]

RLP is a common problem among amputees and has multiple etiologies, both neuropathic and nociceptive.[2,3] Although diagnoses of neuroma, complex regional pain syndrome (CRPS), and somatic pathology exist in the postamputation pain (PAP) literature, to date, there has not been a concerted effort to delineate these conditions in a formal manner in the context of PAP.

The first published description of RLP subtypes was in 1864. Silas Weir Mitchell labeled one clinical entity as neuromata (neuroma) and another as causalgia, which we now know as complex regional pain syndrome, type II (CRPS II).[4] The literature continued to highlight the neurological origins of RLP in 1948, with Craig[5] discussing neuromas and causalgia (CRPS II) as being mediators of RLP. More recently, Wiffen et al[3] describe characteristics of RLP, and assert that specific pathology needs to be identified, focusing on ruling out somatic causes.

Identification of CRPS II, or sympathetically mediated PAP, as a significant cause of pain after amputation was common during the civil war.[6] However, in the 1940s it was reported that causalgia was rare, occurring only in amputations that were not performed with care.[7] Diagnostic criteria for CRPS II in the postamputation patient may be challenging with the use of currently accepted criteria because the missing limb results in absence of many of the physical findings. Nonetheless, autonomic, sudomotor, trophic, and sensory changes are often found in the residual limb. The patient may not meet criteria for CRPS, but a CRPS-like syndrome appears to exist. Isakov et al[8] demonstrated a case series of Below-Knee-Amputation patients that would meet the Budapest criteria for CRPS.

The neuroma phenomenon has been well addressed in the literature; in the early 1940s much attention was focused on PAP as soldiers were returning from World War II. Histologic examination of neuromas revealed branching masses of Schwann cells with proliferating axons embedded in scar tissue. Neuromas have naked nerve endings that are devoid of myelin and are more likely to repeatedly fire within the local anoxic environment of scar tissue.[9] Neuroma sensitization yields changes in the central nervous system that result in wind-up and central sensitization.[3] Over time, the appreciation of neuroma has grown and it remains frequently listed as a predominant cause of RLP.[10]

The cause of pain in the residual limb is not limited to neuropathic mechanisms.[2] In evaluating a patient with RLP, it is important to discriminate between neuropathic and somatic pain, as this has implications for treatment options.[11] Infection, failure of flap closure, bone spurs, vascular insufficiency, or soft tissue inflammation around the prosthesis are all common causes of somatic pain.[12] A significant number of amputees who use prostheses have symptoms arising from improper prosthetic fit or

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alignment, lack of proper training, development of poor habits, or compensation for a secondary physical limitation. The historical literature definitively supports the notion that RLP represents manifestations of somatic pain CRPS II, sensitized neuromas, or potentially diffuse/mosaic neuralgic pains.

Of the PAP syndromes, phantom pain has been most frequently described and characterized over time. The classical description of a phantom limb is the persistent perception of sensation or pain originating from a body part after it has been removed by amputation or trauma. The majority of patients report phantom sensations immediately after their amputation. Although few patients lacking phantom limb sensations report PLP, the majority of patients with PLP also describe sensations.[13]

Mitchell[6] solidified the concept of PLP within the medical literature in his 1872 text *Injuries of Nerves and Their Consequences*. In 1954, the term "phantom limb" was granted its own heading in Index Medicus, making it a defined medical phenomenon.[14]

It is an unfortunate truth that many patients have PLP without receiving medical treatment as many physicians, until recently, regarded this condition as psychiatric disease that was "in the patient's head."[15] Fortunately, Melzack[16] solidified a central model of PLP with the publishing of his neuromatrix theory, postulating that PLP originates from alterations in the neurosynaptic architecture after amputation. Imaging studies have supported the neuromatrix theory as a cause of phantom pain, establishing a correlation between the severity of PLP and the degree of adjacent sensory invasion into the deafferented area.

There seems to be a wide range of reported prevalence of RLP and PLP. This variability likely results not only from the sensitivity of tests used, but also with potential changes in severity over time. The prevalence of RLP in observational studies has been reported between 21%[17] and 74%.[18] Observational studies of PLP have cited prevalence as low as 35%[19] and as high as 85%.[20] Sherman, who reported PLP prevalence at 85%, postulated that the wide variations reported in PLP may be because of the way in which patient populations were interviewed. Prevalence values remain inconsistent, suggesting that there is still considerable variation in assessment techniques.

There is a need for a standardized assessment tool as well as a classification system for the different pain subtypes that may occur in the residual limb. The lack of assessment and classification systems has led to ambiguity in our understanding of postamputation RLP.[21] Therapeutic algorithms are likely to be easier to follow once we better understand the conditions we are treating.

In this paper, we systematically review and highlight the lack of a uniform classification system for PAP which yields diagnostic limitations. Furthermore, we propose a direction for future classification and investigation.

METHODS

In conjunction with the United States Department of Defense grant on Veteran Integrated Pain Evaluation Research, an oversight committee was formed to address the diagnostic variability that was identified within the PAP clinics at the Durham Veteran's Administration Medical Center (DVAMC) (Durham, NC). The committee consisted of 4 pain practitioners involved in PAP treatment at DVAMC and/or Duke University Medical Center (Durham, NC) and the principle investigator of Veteran Integrated Pain Evaluation Research. A structured search of the literature to investigate previous clinical trials on PAP was conducted. A PubMed database query for "stump pain" or "residual limb pain" or "phantom limb pain" yielded 2710 results. Further limiting this to human clinical trials reported in English decreased this to 151 papers (June 14, 2011). These papers were each reviewed to identify patient categorization or sole descriptions of specific entities of PAP states. The following questions were then posed: (1) Was a subtype of PAP studied? (2) If so what subtypes were described? (3) If the subtypes were identified, was there a diagnostic criteria or algorithm?

RESULTS

The search results, as aforementioned, yielded 151 papers. Sixty-six of these papers contained no description of a PAP state, often containing references of phantom sensations only or were completely unrelated to pain states. A further 9 papers referred only to PAP and did not further delineate actual symptoms or diagnosis from this. Forty papers identified PLP as a patient population within the study, however, did not specifically identify any other PAP demographic. Eight papers specifically categorized patients with RLP phenomenon. Twenty papers differentiated PAP into either PLP or RLP, without further subtype differentiation—this was the furthest of global categorization seen. Eight papers identified neuroma as a cause of RLP but did not speak to the identification or differentiation of this phenomenon from other known entities (Table 1).

Furthermore, when assessing proposed treatments of PAP it became clear that the only delineation that is commonly evident is for that of PLP and RLP. Although ideally every case of PLP could be successfully treated the clinical reality is bleak, with $<10\%$ of PLP and RLP patients stating that they get benefit from their treatments.[174] The unfortunate state of affairs is that the literature contains numerous case reports, case series, letters to the editors, and grossly underpowered studies demonstrating significant positive results, yet few high-quality randomized controlled trials have been conducted.[175] There is also little differentiation in the literature with regard to the treatment of PLP versus RLP.

A PubMed database review for human clinical trials in treatment of PLP yields 72 papers. Fifteen of these papers contained no specific intervention or trial or were not related to PLP, but rather to phantom sensation or other neuropathic phenomena.[34,40,47,64,71,79,80,110,118,124,130,135,157,171,173] A further 16 papers detailed periamputation interventions for the prevention of PLP or the assessment of predictive factors for PL.[22,29,45,52,58,76,95,98,101,120,127,137,143,144,149,160] Seventeen papers contained PLP patients as subjects, but analysis of their outcome data did not distinguish between PLP and RLP.[35,36,42,43,50,56,61,62,84,87,113,123,125,147,164,166,169] The remaining 24 studies either specifically assess PLP reduction as an outcome or report changes in PLP as a component of the study.[25,32,38,41,53–55,65,68,85,90,91,93,94,97,99,104,138,148,152,158,162,168,172] Studies within this group contain small numbers and yield conflicting information.

There is some encouraging data regarding the use of the ketamine in the treatment of PLP when compared with placebo.[38,138] Likewise, dextromethorphan, another

TABLE 1. Degree of Classification of Post-amputation Pain States Within Human Clinical Trails

	Populations Identified					
References	**No PAP**	**PAP Only**	**PLP Only**	**PLP and RLP**	**RLP Only**	**Subtypes of RLP**
Borghi et al[22]				X		
Lindenhovius et al[23]	X					
Sivan et al[24]		X				
de Roos et al[25]		X				
Bosmans et al[26]		X				
Walsh et al[27]	X					
Ang et al[28]	X					
Behr et al[29]				X		
Smyrniotis et al[30]	X					
Balcin et al[31]						X (only neuroma)
Casale et al[32]			X			
Hall et al[33]	X					
Raichle et al[34]				X		
Wu et al[35]		X				
Gruber et al[36]				X		States neuroma as a cause of both
Kang et al[37]	X					
Eichenberger et al[38]			X			
Kalteis et al[39]	X					
Manias and Williams[40]	X					
Chan et al[41]			X			
Owen et al[42]			X			
Lazorthes et al[43]			X			
Nabhan et al[44]			X			
Wilson et al[45]				X		
Heidari et al[46]		X				
Bach and Clement[47]	X					
Lenti et al[48]	X					
Canadian Orthopaedic Trauma Society[49]	X					
Moseley[50]			X			Discusses CRPS, not in PAP
Pessaux et al[51]	X					
Nikolajsen et al[52]				X		
Kern et al[53]			X			
Yamamoto et al[54]			X			
Brodie et al[55]			X			
Blankertz et al[56]	X					
Lin et al[57]					X	
Schley et al[58]			X			
Ertem et al[59]	X					
Inan et al[60]	X					
Smith et al[61]				X		
Saitoh et al[62]	X					
Thome et al[63]	X					
Dhillon and Horch[64]	X					
Harden et al[65]			X			
Cuignet et al[66]	X					
Ephraim et al[67]				X		
Wilder-Smith et al[68]				X		
Schaefer et al[69]	X					
Suputtitada and Suwanwela[70]	X					
Hunter et al[71]				X		
Kane et al[72]	X					
Chiodo and Miller[73]						X (neuroma only)
Wong et al[74]	X					
MacKenzie et al[75]		X				
Hayes et al[76]				X		
Ben Gal et al[77]	X					
Barnett-Cowan and Peters[78]			X			
MacLachlan et al[79]			X			
Moseley[80]	X					CRPS (but not as PAP)
Kornblum et al[81]	X					
Paqueron et al[82]	X					
Beldi et al[83]	X					

(*Continued*)

TABLE 1. (*continued*)

	Populations Identified					
References	**No PAP**	**PAP Only**	**PLP Only**	**PLP and RLP**	**RLP Only**	**Subtypes of RLP**
Robinson et al[84]				X		
Wiech et al[85]			X			
Gimbel et al[86]	X					
Saitoh et al[87]			X			
Schwenkreis et al[88]			X			
Millisdotter et al[89]	X					
Maier et al[90]			X			
Ben Abraham et al[91]			X			
Goh et al[92]	X					
Bone et al[93]			X			
Ben Abraham et al[94]			X			
Techanivate et al[95]	X					
Gentili et al[96]	X					
Wu et al[97]				X		
Lambert et al[98]				X		
Flor et al[99]			X			
Maruno et al[100]	X					
Chu[101]	X					
Karl et al[102]			X			
da Paz et al[103]	X					
Huse et al[104]			X			
Grusser et al[105]				X		
Nikolajsen et al[106]				X		
Belcher and Pandya[107]						X (neuroma only)
Bakheit et al[108]	X					
Devers and Galer[109]						X (neuroma only)
Isaacson et al[110]	X					
Buchner et al[111]	X					
Angrilli and Koster[112]			X			
Carroll et al[113]			X			
Paya et al[114]	X					
Combes et al[115]	X					
Pucher et al[116]			X			
Muhlnickel et al[117]		X				
Ramazanov et al[118]	X					
Sirnes et al[119]	X					
Nikolajsen et al[120]					X	
Ramos-e-Silva et al[121]	X					
Montoya et al[122]			X			
Kumar et al[123]			X			Mentions CRPS but not PAP
Persson et al[124]	X					
Lenz et al[125]		X				
Kosasih and Silver-Thorn[126]					X	
Nikolajsen et al[127]				X		
Chow et al[128]	X					
Montoya et al[129]			X			
Dasgupta et al[130]					X	
Yuksel et al[131]						X (neuroma only)
Postema et al[132]					X	
Nikolajsen et al[133]					X	
Barkun et al[134]	X					
Merimsky et al[135]				X		
Ersland et al[136]	X					
Pinzur et al[137]		X				
Nikolajsen et al[138]				X		
Singh et al[139]	X					
Hill et al[140]			X			
Hunter et al[141]	X					
Gonzalez-Fajardo et al[142]	X					
Jahangiri et al[143]				X		
Elizaga et al[144]			X			
Arena et al[145]	X					
Dorman et al[146]	X					

(*Continued*)

TABLE 1. (*continued*)

	Populations Identified					
References	**No PAP**	**PAP Only**	**PLP Only**	**PLP and RLP**	**RLP Only**	**Subtypes of RLP**
Broggi et al[147]			X			CRPS mentioned but not PAP
Jaeger and Maier[148]			X			
Sicuteri et al[149]	X					
Jonson et al[150]	X					
Bossaert et al[151]	X					
Katz and Melzack[152]			X			
Sane et al[153]	X					
Schreiber et al[154]	X					
Panerai et al[155]			X			
Chaitman et al[156]	X					
Katz et al[157]	X					
Chabal et al[158]						X (neuroma only)
Topol et al[159]	X					
Finsen et al[160]				X		
Crist et al[161]	X					
Lundeberg[162]			X			
Corsini et al[163]	X					
Swerdlow[164]			X			
Steardo et al[165]	X					
Scadding et al[166]						X (neuroma only)
Mueller[167]					X	
Winnem and Amundsen[168]			X			
Langohr et al[169]						X (neuroma only)
Thorpe et al[170]					X	
Nathan[171]	X					
Melzack[172]			X			
Brenning[173]	X					
Totals	66	9	40	20	8	8

CRPS indicates complex regional pain syndrome; PAP, postamputation pain; PLP, phantom limb pain; RLP, residual limb pain.

NMDA antagonist, has demonstrated efficacy in small case series and randomized control trials.[91,94] All NMDA antagonists, however, have not shown equal efficacy, as memantine failed in randomized controlled trials to demonstrate significant pain reductions in the PLP population.[85,90]

Antiepileptic medications are frequently utilized by pain physicians for various pain syndromes thought to be of neurogenic origins. Of this class, gabapentin is the only member that has been studied in a randomized controlled trial specific for PLP. A randomized, double-blind, placebo-controlled, cross-over study with 19 patients demonstrated significant reductions in pain intensity with use of gabapentin relative to placebo.[93] Gabapentin has also been studied in the immediate postoperative period in an attempt to prevent or reduce the incidence of PLP. Unfortunately, a randomized trial did not demonstrate efficacy as compared with placebo.[52] Other trials involving gabapentin have shown equivocal results as compared with placebo.[61] The antiepileptic drug Topiramate has also shown some promise, although experience is limited to a small case series.[65]

Tricyclic antidepressants have long demonstrated efficacy in many neuropathic pain conditions. Amitriptyline was studied in a randomized control study for management of PLP and RLP. It was found to have similar benefits in pain reduction for both conditions with an average daily dose of 56 mg/d and a low side-effect profile.[68]

Opioids are often the mainstay of analgesia in both acute and chronic pain conditions. Several studies have demonstrated that opioids are effective at helping manage PLP and appear to have greater benefit than other drug classes.[68,97] Furthermore, it has been demonstrated that opioids have an effect on central pain mechanisms and may partly reverse some of the cortical shifting seen in patients with phantom pain. One investigator noted the correlation between analgesic response to morphine and reduction in cortical reorganization.[104] However, it remains unclear if the central changes noted in this study persist beyond the initial treatment period.

Studies investigating the use of the hormone calcitonin to treat PLP have been conducted. Although the studies are small, it appears that calcitonin may be beneficial if utilized at the early onset of PLP.[148] Studies conducted farther from the onset of PLP have failed to demonstrate any benefit from its use.[38]

The expanding role of surgical intervention is evident within the medical literature. Case series and reports demonstrate successful management of PLP with the use of deep brain stimulation at a variety of sites including the thalamic sensory relay nucleus and central sulcus.[54,87] Although not specifically captured in the aforementioned review, there exist case series and reports of successful management of PLP with the use of spinal cord stimulation.[176,177] We are careful to suggest that this option is to be considered only when other conservative management has failed.

In addition to medical and interventional management, psychological and rehabilitative strategies play a successful role in the management of PLP. Psychological

training using a variety of strategies, including management in processing emotional and somatosensory memories related to the amputation, have been demonstrated as effective in pain reduction.[25] Furthermore, sensory discrimination training has been shown to not only reduce PLP but also to significantly influence cortical reorganization. Some of the most promising work, which may give support to the neuromatrix theory, is the work done with mirror box therapy.[41,55]

Additional reports and studies have demonstrated varying efficacy with transcutaneous electrical nerve stimulation units applied to various locations; including the periauricular area, contralateral limb, and stump.[152,168,172] A double-blind, randomized, crossover trial in a total of 30 leg amputees demonstrated that an electromagnetically shielded stump stocking significantly reduced the incidence and intensity of PLP.[53] Although no specific etiology can explain these findings, they are interesting nonetheless.

Outside of the limited drug trials noted above, there is a paucity of evidence regarding effective pharmaceutical treatment strategies for PLP. Future trials should focus on therapies for patients who have ongoing PLP despite medical therapy with amitriptyline, ketamine, and opioid medications. In addition, methods to decrease the incidence and severity of phantom pain should be investigated for use at the time of elective amputation.

As a distinct entity, RLP has been underrepresented in the literature. Studies frequently combine PLP and RLP into 1 single category—PAP. However, the etiology and manifestation of these conditions are entirely separate. Existing studies often contain low patient numbers and infrequently speak to whether the cause of the RLP is somatic or neuropathic.

A PubMed database query for human clinical trials in the treatment of postamputation stump pain yields a total of 35 papers. Twelve of these papers do not address interventions related to postamputation RLP management.[41,53,57,71,92,99,104,125,126,138,157,165] A further 14 studies involve interventions for the prevention of RLP or positive predictors for the development of RLP.[45,46,52,75,76,98,107,120,127,131,143,170] Another 3 studies do not specifically delineate in the results the difference in outcomes between PLP and RLP.[35,61,160] This leaves 6 studies that note outcomes specific to RLP.

Wilder-Smith et al[68] demonstrated efficacy of both tramadol and amitriptyline in a 3-armed randomized control trial. Patients received individually titrated doses of tramadol, placebo (double-blind comparison), or amitriptyline (open comparison). Nonresponders were crossed over to the alternative active treatment. Eighty-one percent of patients who failed treatment in one arm saw success in another arm. This highlights the fact that RLP may have different etiologies and trialing a second medication may be very successful. In a randomized, double-blind, active-placebo-controlled, crossover trial with 32 patients, lidocaine and morphine were both found to be effective at ameliorating self-reported pain in the residual limb. Interestingly, in the same study only morphine was found to be effective on PLP.[97] Although there have been a few successful reports of NMDA antagonists for the management of PLP, the use of this drug class for RLP is sparse. This literature search provided a single case report of a single ketamine infusion giving pain relief for 31 hours to a patient with RLP.[133] An observational study of 20 RLP patients by Combes[178] showed efficacy of the dopamine antagonist tiapride in reduction of RLP, demonstrating an increased tolerance and duration of prosthesis use. These results, however, have not been reproduced elsewhere.

As neuroma is believed to be a significant mediator of RLP in many patients, it is logical that interventions performed on this trigger may have potential benefit. Although local anesthetic and steroid injections are frequently performed in the treatment of neuroma-related pain,[179] this treatment is not well supported with controlled trials. Neurolytic therapies using cryoablation and injection of phenol have also been used with success.[36,180] In a prospective manner, Gruber et al[36] demonstrated significant reductions in pain among 82 patients treated with ultrasound-guided neuroma injection with phenol. In addition to steroid injection and neurolytic therapies, immune system modulators such as the anti–tumor necrosis-α drug etanercept have been demonstrated promising analgesia when injected perineurally in patients with traumatic amputation.[181] It is interesting to note that the 6 PAP soldiers in this series reported significant improvements not only in their visual analogue pain score for RLP, but also in their PLP.

There have been additional complementary and alternative medicine reports of analgesia with the use of aroma and music therapy for RLP, particularly during dressing changes.[72] This review of the literature makes it apparent that there is no accepted documented system for differentiating PAP into specific categories; at best these patients are subclassified into RLP or PLP.

DISCUSSION

Identifying the need of a formal diagnostic algorithm for PAP a series of committee meetings were held over approximately 12 months conducted in think tank type sessions with several didactic presentations of solutions eventually culminating in a consensus among committee members, identifying 5 clinically distinct diagnoses. The result was the Durham Pain Investigations Group, Post-Amputation Pain Algorithm (DPIG-PAPA).

The DPIG-PAPA, utilizing simple questions, allows practitioners at almost any level of training, to classify

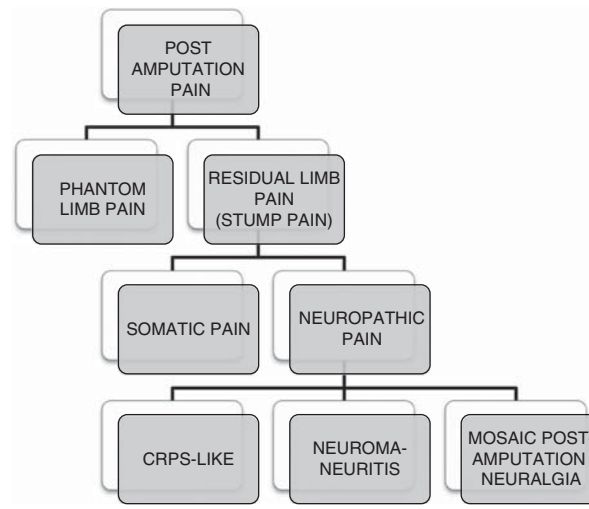

FIGURE 1. Proposed taxonomy of postamputation pain.

patients into one of the following categories: (1) PLP, (2) somatic RLP, (3) neuroma/neuritis RLP, (4) CRPS-like RLP, (5) mosaic postamputation neuralgia (MPAN) (Fig. 1). With the exception of MPAN these classifications are well embedded within the literature and lexicon of pain practice. MPAN is a classification developed in recognition that a small subset of patients present with a mixed neuropathic picture not easily delineated into typical diagnoses. The simple questions in conjunction with the validated Leeds Assessment of Neuropathic Symptoms and Signs (LANSS) Pain Scale (sensitivity 81% to 91%, specificity 80% to 94%)[182] and the Budapest Clinical Criteria for Complex Regional Pain Syndrome (sensitivity 70%, specificity 94%)[183] have been readily used and are now well accepted within the DVAMC pain clinic. One weakness noted of this algorithm is that the LANSS criteria traditionally delineate outcomes into the likeliness or the unlikeliness of neuropathic pain. In order for the DPIG-PAPA to be a useful tool we felt it was important to assert patients into neuropathic or somatic pain pathologies. With the sensitivity and specificity of the LANSS criteria approaching that of other "gold standards," such as the Budapest criteria, we did not foresee any significant detriment to this. Certainly we have not experienced any issues with its application and use in our center.

PAP: Conclusions and Future Directions

Similar to the improvements in cancer therapy that evolve after receptor and biomarker classification of tumors, we foresee improvements in the treatment of amputation pain when the various subtypes are better recognized and treated as discreet clinical conditions. Although descriptions of postamputation sensation and pain syndromes have clearly been recorded in the recent and remote past, we still lack a uniform approach to PAP subtype classification.

Diagnostic clarity is of increasing importance given the recent global conflicts with both military and civilian casualties. Describing and defining the distinct clinical entities, especially in regards to RLP, is a likely prerequisite to developing optimal treatments. An intervention that may be effective for a sensitized neuroma may not be effective for more diffuse symptoms. Treatments for somatic and soft-tissue pathology may not alleviate the pain of nerve injury. Lumping widely disparate pathologic states together in clinical trials is likely to mask the effectiveness of these treatments for any individual PAP subtype. We propose a common classification system for the study of PAP that should allow for the development of disease-specific therapies and allow these to be evaluated in a more systematic way.

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APPENDIX A: MASTER ALGORITHM

Step #1	
Does the patient percieve pain in part of the missing lim?	**YES** **NO**
If **YES** then:	***Phantom Limb Pain***
If **NO** then:	Proceed to Step 2
Step #2	
Complete **LANSS** screening tool to identify neuropathic pain (attached):	
If LANSS < 12 then:	***Somatic Pain***
If LANNS ≥ 12 then:	Proceed to Step 3
Step #3	
Part A: Is the pain localized to a specific nerve distribution?:	**YES** **NO**
Part B: Is there a Tinel's Sign?:	**YES** **NO**
Part C: Complete the budapest criteria (attached), does the patient meet the budapest criteria?:	**YES** **NO**
If Yes to A and or B but not C then:	***Neuroma/Neuritis***
If Yes to A and/or B and C then:	***Mosaic Post-Amputation Neuralgia***
If yes to only C then:	***CRPS-like***

APPENDIX B: LANSS PAGE 1

The Leeds Assessment of Neuropathic Symptoms and Signs (LANSS) Pain Scale

Name..... Date

This pain scale can help to determine whether the nerves that are carrying your pain signals are working normally or not. It is important to find this out in case different treatments are needed to control your pain.

A. PAIN QUESTIONNAIRE

- Think about how your pain has felt over the last week.
- Please say whether any of the descriptions match your pain exactly.

1. Does your pain feel like strange, unpleasant sensations in your skin?
Words like pricking, tingling, pins and needles might describe these sensations.
a) NO – My pain doesn't really feel like this (0)
b) YES – I get these sensations quite a lot (5)

2. Does your pain make the skin in the painful area look different from normal?
Words like mottled or looking more red or pink might describe the appearance.
a) NO – My pain doesn't affect the colour of my skin (0)
b) YES – I've noticed that the pain does make my skin look different from normal (5)

3. Does your pain make the affected skin abnormally sensitive to touch?
Getting unpleasant sensations when lightly stroking the skin, or getting pain when wearing tight clothes might describe the abnormal sensitivity.
a) NO – My pain doesn't make my skin abnormally sensitive in that area (0)
b) YES – My skin seems abnormally sensitive to touch in that area (3)

4. Does your pain come on suddenly and in bursts for no apparent reason when you're still?
Words like electric shocks, jumping and bursting describe these sensations.
a) NO – My pain doesn't really feel like this (0)
b) YES – I get these sensations quite a lot (2)

5. Does your pain feel as if the skin temperature in the painful area has changed abnormally?
Words like hot and burning describe these sensations.
a) NO – I don't really get these sensations (0)
b) YES – I get these sensations quite a lot (1)

APPENDIX C: LANSS PAGE 2

Leeds Assessment of Neuropathic Symptoms and Signs (continued)
B. SENSORY TESTING
Skin sensitivity can be examined by comparing the painful area with a contralateral or adjacent non-painful area for the presence of allodynia and an altered pin-prick threshold (PPT).

1. Allodynia
Examine the response to lightly stroking cotton wool across the non-painful area and then the painful area. If normal sensations are experienced in the non-painful site, but pain or unpleasant sensations (tingling, nausea) are experienced in the painful area when stroking, allodynia is present.
a) NO – Normal sensations in both area (0)
b) YES – Allodynia in painful area only (5)

2. Altered pin-prick threshold
Determine the pin-prick threshold by comparing the response to a 23-gauge (blue) needle mounted inside a 2ml syringe barrel placed gently onto the skin in a non-painful and then painful areas.
If a sharp pin prick is felt in the non-painful area, but a different sensation is experienced in the painful area, eg. none/ blunt only (raised PPT) or a very painful sensation (lowered PPT), an altered PPT is present.
If a pinprick is not felt in either area, mount the syringe onto the needle to increase the weight and repeat.
a) NO – Equal sensation in both areas (0)
b) YES – Altered PPT in painful area (3)

SCORING:
Add values in parentheses for sensory description and examination findings to obtain overall score.

TOTAL SCORE (maximum 24) ***If score < 12, neuropathic mechanisms are unlikely to be contributing to the patient's pain. If score ≥ 12, neuropathic mechanisms are likely to be contributing to the patient's pain.***

APPENDIX D: MODIFIED BUDAPEST CRITERIA OF CRPS

Must report at least one symptom in three of the four categories:

Sensory: reports of hyperesthesia and/or allodynia	
Vasomotor: reports of temperature asymmetry and/or skin color changes and/or asymetry	
Sudomotor/edema: reports of edema and/or sweating changes and/or asymmetry	
Motor/Trophic: reports of decreased range of motion and/or motor dysfunction (weakness, tremor, dystonia) and/or trophic changes (nails, hair, skin)	
SCORE	

Must report at least one sign in 2 of the four categories:

Sensory: evidence of hyperesthesia(to pinprick) and/or allodynia (to light touch and/or deep pressure and/or joint movement)	
Vasomotor: evidence of temperature asymmetry and/or skin color changes and/or asymmetry	
Sudomotor/edema: evidence of edema and/or sweating changes and/or asymmetry	
Motor/Trophic: evidence of decreased range of motion and/or motor dysfunction (weakness, tremor, dystonia) and/or trophic changes (nails, hair, skin)	
SCORE	