

PASIG MONTHLY CITATION BLAST: No.83

June 2013

Dear Performing Arts SIG members:

Happy June! This month's citation blast is on neuropathic pain in performing artists. I hope you benefit from the monthly citations, and that you facilitate sharing the most recent research on performing artist patient issues in your setting.

Reminder to those interested in participating in the production of a wellness screen for the young, pre-professional dancer: contact Brooke Winder, PT, DPT, OCS, <u>brookeRwinder@gmail.com</u>

Consider compiling Performing Arts-related abstracts for a citation blast this year. It's easy to do, and a great way to become involved with PASIG! Just take a look at our Performing Arts Citations and Endnotes, look for what's missing, and email me your contribution or ideas on future citation blasts.

http://www.orthopt.org/content/special_interest_groups/performing_arts/citations_endnotes

You do not need to analyze each article. All I need is your abstracts pasted on one Word Document, and a short introduction on how you became interested in your topic. If you need help on how to search for abstracts, let me know. We have a great team to help you get started.

Suggested sites to start with:

APTA Hooked on Evidence, Cochrane Library, PEDro, Pubmed

Best regards,

Annette

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PERFORMING ARTS CONTINUING EDUCATION, CONFERENCES, AND RESOURCES

Orthopaedic Section Independent Study Course. 20.3 Physical Therapy for the Performing Artist.

Monographs are available for:

- Figure Skating (J. Flug, J. Schneider, E. Greenberg),

- Artistic Gymnastics (A. Hunter-Giordano, Pongetti-Angeletti, S. Voelker, TJ Manal), and

- Instrumentalist Musicians (J. Dommerholt, B. Collier). Contact: Orthopaedic Section at: <u>www.orthopt.org</u>

Orthopaedic Section Independent Study Course. *Dance Medicine: Strategies for the Prevention and Care of Injuries to Dancers*.

This is a 6-monograph course and includes many PASIG members as authors.

- Epidemiology of Dance Injuries: Biopsychosocial Considerations in the Management of Dancer Health (MJ Liederbach),

- Nutrition, Hydration, Metabolism, and Thinness (B Glace),

- The Dancer's Hip: Anatomic, Biomechanical, and Rehabilitation Considerations (G. Grossman),

- Common Knee Injuries in Dance (MJ Liederbach),

- Foot and Ankle Injuries in the Dancer: Examination and Treatment Strategies (M. Molnar, R. Bernstein, M. Hartog, L. Henry, M. Rodriguez, J. Smith, A. Zujko),

- Developing Expert Physical Therapy Practice in Dance Medicine (J. Gamboa, S. Bronner, TJ Manal). Contact: Orthopaedic Section at: www.orthopt.org

Orthopaedic Section-American Physical Therapy Association, Performing Arts SIG <u>http://www.orthopt.org/content/special_interest_groups/performing_arts</u> Performing Arts Citations and Endnotes <u>http://www.orthopt.org/content/special_interest_groups/performing_arts/citations_endnotes</u>

ADAM Center <u>http://www.adamcenter.net/</u> Publications: <u>http://www.adamcenter.net/#!vstc0=publications</u> Conference abstracts: <u>http://www.adamcenter.net/#!vstc0=conferences</u>

Dance USA Annual conference: Philadelphia, PA, June 12-15, 2013 <u>http://www.danceusa.org/</u> Research resources: <u>http://www.danceusa.org/researchresources</u> Professional Dancer Annual Post-Hire Health Screen: <u>http://www.danceusa.org/dancerhealth</u>

Dancer Wellness Project <u>http://www.dancerwellnessproject.com/</u> Becoming an affiliate: <u>http://www.dancerwellnessproject.com/Information/BecomeAffiliate.aspx</u>

Harkness Center for Dance Injuries, Hospital for Joint Diseases <u>http://hjd.med.nyu.edu/harkness/</u> Continuing education: <u>http://hjd.med.nyu.edu/harkness/education/healthcare-professionals/continuing-education-courses-cme-and-ceu</u> Resource papers: <u>http://hjd.med.nyu.edu/harkness/dance-medicine-resources/resource-papers-and-forms</u> Links: <u>http://hjd.med.nyu.edu/harkness/dance-medicine-resources/links</u> Informative list of common dance injuries: <u>http://hjd.med.nyu.edu/harkness/patients/common-dance-injuries</u> Research publications: <u>http://hjd.med.nyu.edu/harkness/research/research-publications</u> International Association for Dance Medicine and Science (IADMS) http://www.iadms.org/

The 23rd Annual Meeting of the International Association for Dance Medicine & Science (IADMS) will be held in <u>Seattle, Washington, USA</u> from October 17 - 19, 2013. Meeting activities and sessions will be held at the <u>Renaissance Seattle</u> <u>Hotel</u>. On Sunday, October 20, 2013, Special Interest Groups (SIG) Day will be held, with special programs available.

Resource papers:

http://www.iadms.org/displaycommon.cfm?an=1&subarticlenbr=186 Links:

http://www.iadms.org/displaycommon.cfm?an=5

Medicine, arts medicine, and arts education organization links: <u>http://www.iadms.org/displaycommon.cfm?an=1&subarticlenbr=5</u> Publications: <u>http://www.iadms.org/displaycommon.cfm?an=3</u>

Performing Arts Medicine Association (PAMA)

http://www.artsmed.org/

Annual symposium: July 20-23, 2013 Medical Problems of Performing Artists: "Maximizing Performance, Artistry, Implementation, and Empowerment"

http://www.artsmed.org/symposium.html

Interactive bibliography site:

http://www.artsmed.org/bibliography.html

Related links:

http://www.artsmed.org/relatedlinks.html

Member publications:

http://artsmed.org/publications.html

(Educators, researchers, and clinicians, please continue to email me your conference and continuing education information and I will include it in the upcoming blasts.)

Neuropathic pain

I became interested in this topic after having many dancers with lower extremity pain and teaching interns how to practice ruling out to rule in. Some had true lower extremity issues, others had spinal referral patterns, and most recently we had a dancer patient with neuropathic pain questionably related to complex regional pain syndrome. In all cases, PICO questions came up in our clinic of how to retrain the brain to receive movement and even rest as non pain generating, how to educate our patients, and how to collaborate with other practitioners in unusual patient cases. Do we spend our time breathing, moving, talking, listening, addressing ribs, core, gluts, movement patterns, or patient perception? What else needs consideration...cardiorespiratory conditioning, nutrition, psychological or neurological consult, medication, imagery? What are the best self-report outcome measures or standardized physical performance tools? Happy reading!

Annette

Backonja, M., N. Attal, et al. (2013). "Value of quantitative sensory testing in neurological and pain disorders: NEUPSIG consensus." <u>Pain</u>.

Quantitative sensory testing (QST) is a psychophysical method used to quantify somatosensory function in response to controlled stimuli in healthy subjects and patients. Although QST shares similarities with the quantitative assessment of hearing or vision, which is extensively used in clinical practice and research, it has not gained a large acceptance among clinicians, for many reasons and in significant part due to lack of information about standards for performing QST, its potential utility and interpretations of results. A consensus meeting was convened by the Neuropathic Pain Special Interest Group of the International Association for the Study of Pain (NEUPSIG) to formulate recommendations for conducting QST in clinical practice and research. Research studies have confirmed the utility of QST for: a/ the assessment and monitoring of somatosensory deficits, particularly in diabetic and small fiber neuropathies; b/the assessment of evoked pains (mechanical and thermal allodynia or hyperalgesia); c/the diagnosis of sensory neuropathies. Promising applications include the assessment of evoked pains in largescale clinical trials and the study of conditioned pain modulation. In clinical practice, we recommend the use QST for: a/ the screening for small and large fiber neuropathies; b/ monitoring of somatosensory deficits; and c/ monitoring of evoked pains, allodynia and hyperalgesia. QST is not recommended as a standalone test for the diagnosis of neuropathic pain. For the conduct of QST in healthy subjects and in patients, we recommend use of predefined standardized stimuli and instructions, validated algorithms of testing and reference values corrected for anatomical sites, age and gender. Interpretation of results should always take into account the clinical context and patients with language and cognitive difficulties, anxiety or litigation should not be considered eligible for QST. When appropriate standards as discussed here are applied QST can provide important and unique information about the functional status of somatosensory system, which would be complementary to already existing clinical methods.

Baron, R. (2006). "Mechanisms of disease: neuropathic pain--a clinical perspective." <u>Nat Clin Pract Neurol</u> **2**(2): 95-106.

Neuropathic pain syndromes-pain after a lesion or disease of the

peripheral or central nervous system-are clinically characterized by spontaneous and evoked types of pain, which are underpinned by various distinct pathophysiological mechanisms in the peripheral and central nervous systems. In some patients, the nerve lesion triggers molecular changes in nociceptive neurons, which become abnormally sensitive and develop pathological spontaneous activity. Inflammatory reactions of the damaged nerve trunk can induce ectopic nociceptor activity, causing spontaneous pain. The hyperactivity in nociceptors induces secondary changes in processing neurons in the spinal cord and brain, so that input from mechanoreceptive A-fibers is perceived as pain. Neuroplastic changes in the central pain modulatory systems can lead to further hyperexcitability. The treatment of neuropathic pain is still unsatisfactory, and a new hypothetical concept has been proposed, in which pain is analyzed on the basis of underlying mechanisms. The increased knowledge of pain-generating mechanisms and their translation into symptoms and signs might eventually allow a dissection of the mechanisms that operate in each patient. If a precise clinical phenotypic characterization of the neuropathic pain is combined with a selection of drugs that act on those mechanisms, it should ultimately be possible to design optimal treatments for individuals. This review discusses the conceptual framework of the novel mechanism-based classification, encouraging the reader to see neuropathic pain as a clinical entity rather than a compilation of single disease states.

Baron, R. (2009). "Neuropathic pain: a clinical perspective." <u>Handb Exp</u> <u>Pharmacol(194)</u>: 3-30.

Neuropathic pain syndromes, i.e., pain after a lesion or disease of the peripheral or central nervous system, are clinically characterized by spontaneous pain (ongoing, paroxysms) and evoked types of pain (hyperalgesia, allodynia). A variety of distinct pathophysiological mechanisms in the peripheral and central nervous system operate in concert: In some patients the nerve lesion triggers molecular changes in nociceptive neurons that become abnormally sensitive and develop pathological spontaneous activity (upregulation of sodium channels and receptors, e.g., vanilloid TRPV1 receptors, menthol-sensitive TRPM8 receptors, or alpha-receptors). These phenomena may lead to spontaneous pain, shooting pain sensations, as well as heat hyperalgesia, cold hyperalgesia, and sympathetically maintained pain. Spontaneous activity in damaged large nonnociceptive A-fibers may lead to paresthesias. All these changes may also occur in uninjured neurons driven by substances released by adjacent dying cells and should receive more attention in the future. The hyperactivity in nociceptors in turn induces secondary changes (hyperexcitability) in processing neurons in the spinal cord and brain. This central sensitization causes input from

mechanoreceptive A-fibers to be perceived as pain (mechanical allodynia). Neuroplastic changes in the central descending pain modulatory systems (inhibitory or facilitatory) may lead to further hyperexcitability. Neuropathic pain represents a major neurological problem and treatment of patients with such pain has been largely neglected by neurologists in the past. The medical management of neuropathic pain consists of five main classes of oral medication (antidepressants with reuptake blocking effect, anticonvulsants with sodium-blocking action, anticonvulsants with calciummodulating actions, tramadol, and opioids) and several categories of topical medications for patients with cutaneous allodynia and hyperalgesia (capsaicin and local anesthetics). In many cases an early combination of compounds effecting different mechanisms is useful. At present existing trials only provide general pain relief values for specific causes, which in part may explain the failure to obtain complete pain relief in neuropathic pain conditions. In general, the treatment of neuropathic pain is still unsatisfactorily. Therefore, a new hypothetical concept was proposed in which pain is analyzed on the basis of underlying mechanisms. The increased knowledge of pain-generating mechanisms and their translation into symptoms and signs may in the future allow a dissection of the mechanisms that operate in each patient. If a systematic clinical examination of the neuropathic pain patient and a precise phenotypic characterization is combined with a selection of drugs acting against those particular mechanisms, it should ultimately be possible to design optimal treatments for the individual patient.

Bowering, K. J., N. E. O'Connell, et al. (2013). "The effects of graded motor imagery and its components on chronic pain: a systematic review and meta-analysis." <u>J Pain</u> 14(1): 3-13.

Graded motor imagery (GMI) is becoming increasingly used in the treatment of chronic pain conditions. The objective of this systematic review was to synthesize all evidence concerning the effects of GMI and its constituent components on chronic pain. Systematic searches were conducted in 10 electronic databases. All randomized controlled trials (RCTs) of GMI, left/right judgment training, motor imagery, and mirror therapy used as a treatment for chronic pain were included. Methodological guality was assessed using the Cochrane risk of bias tool. Six RCTs met our inclusion criteria, and the methodological quality was generally low. No effect was seen for left/right judgment training, and conflicting results were found for motor imagery used as stand-alone techniques, but positive effects were observed for both mirror therapy and GMI. A meta-analysis of GMI versus usual physiotherapy care favored GMI in reducing pain (2 studies, n = 63; effect size, 1.06 [95%] confidence interval, .41, 1.71]; heterogeneity, I(2) = 15%). Our results suggest that GMI and mirror therapy alone may be effective, although this conclusion is based on limited evidence. Further rigorous studies are needed to investigate the effects of GMI and its components on a wider chronic pain population. PERSPECTIVE: This systematic review synthesizes the evidence for GMI and its constituent components on chronic pain. This review may assist clinicians in making evidence-based decisions on managing patients with chronic pain conditions.

Budrys, V. (2006). "Neurological deficits in the life and works of Frida Kahlo." <u>Eur</u> <u>Neurol</u> **55**(1): 4-10.

World-famous Mexican painter Frida Kahlo is an impressive example of an artist whose entire life and creativity were extremely influenced by chronic, severe illness. Many of her best-known works depict her physical and mental suffering. She was one of those very uncommon artists who dared to show their nude, sick body. This article describes biographical events and works of Frida Kahlo that are closely related to neurology: congenital anomaly (spina bifida), poliomyelitis, spine injury, neuropathic pain.

Clifford, D. B., D. M. Simpson, et al. (2012). "A randomized, double-blind, controlled study of NGX-4010, a capsaicin 8% dermal patch, for the treatment of painful HIV-associated distal sensory polyneuropathy." <u>J Acquir Immune Defic</u> <u>Syndr</u> **59**(2): 126-133.

INTRODUCTION: Effective treatment of HIV-associated distal sensory polyneuropathy remains a significant unmet therapeutic need. METHODS: In this randomized, double-blind, controlled study, patients with pain due to HIV-associated distal sensory polyneuropathy received a single 30minute or 60-minute application of NGX-4010--a capsaicin 8% patch (n = 332)--or a low-dose capsaicin (0.04%) control patch (n = 162). The primary endpoint was the mean percent change from baseline in Numeric Pain Rating Scale score to weeks 2-12. Secondary endpoints included patient global impression of change at week 12. RESULTS: Pain reduction was not significantly different between the total NGX-4010 group (-29.5%) and the total control group (-24.5%; P = 0.097). Greater pain reduction in the 60-minute (-30.0%) versus the 30-minute control group (-19.1%) prevented intended pooling of the control groups to test individual NGX-4010 treatment groups. No significant pain reduction was observed for the 30-minute NGX-4010 group compared with 30-minute control (-26.2% vs.-19.1%, respectively, P = 0.103). Pain reductions in the 60-minute NGX-4010 and control groups were comparable (-32.8% vs. -30.0%, respectively; P = 0.488). Posthoc nonparametric testing demonstrated significant differences favoring the total (P = 0.044) and 30-minute NGX-4010 groups (P = 0.035). Significantly, more patients in the total and 30minute NGX-4010 group felt improved on the patient global impression of change versus control (67% vs. 55%, P = 0.011 and 65% vs. 45%, P = 0.006, respectively). Mild to moderate transient application site pain and ervthema were the most common adverse events. CONCLUSIONS:

Although the primary endpoint analyses were not significant, trends toward pain improvement were observed after a single 30-minute NGX-4010 treatment.

Damian, M. and C. Zalpour (2011). "Trigger point treatment with radial shock waves in musicians with nonspecific shoulder-neck pain: data from a special physio outpatient clinic for musicians." <u>Med Probl Perform Art</u> **26**(4): 211-217. Musicians often suffer from disorders of the musculoskeletal system that are related to their instrument playing. Among the most frequent symptoms are complaints in the shoulder-neck area. Radial shock wave therapy is increasingly used in trigger point treatment, but only few high-level studies have examined of shock wave therapy used together with physical therapy in the treatment of musicians. METHODS: This randomized blinded study in musicians (n = 26) with nonspecific shoulder-

neck problems was done to examine the effect of shock wave therapy in addition to current physical therapy on the symptoms and quality of life of the musicians as well as their habits of playing musical instruments (intervention group shock wave vs reference group placebo). The effects were documented by a pain VAS and other instruments. A guestionnaire designed specifically for musicians (with initial and final guestions) recorded intensity and manifestation of pain and handicaps in daily life, especially when practicing and playing. The Shoulder Pain and Disability Index (SPADI) and the Neck Pain Disability Index Questionnaire (NPDIQ) were also used. RESULTS: Both groups reported subjective improvement in pain, but significance was found only for the intervention group for the SPADI and NPDIQ. CONCLUSIONS: Trigger point treatment with radial shock wave used in combination with physical therapy makes the subjects feel temporarily relieved of neck and shoulder pains. The effects of radial shock wave without physical therapy will need to be examined in further studies.

Eccleston, C., T. M. Palermo, et al. (2012). "Psychological therapies for the management of chronic and recurrent pain in children and adolescents." <u>Cochrane Database Syst Rev</u> **12**: CD003968.

BACKGROUND: Chronic pain affects many children, who report severe pain, distressed mood, and disability. Psychological therapies are emerging as effective interventions to treat children with chronic or recurrent pain. This update adds recently published randomised controlled trials (RCTs) to the review published in 2009. OBJECTIVES: To assess the effectiveness of psychological therapies, principally cognitive behavioural therapy and behavioural therapy, for reducing pain, disability, and improving mood in children and adolescents with recurrent, episodic, or persistent pain. We also assessed the risk of bias and methodological quality of the included studies. SEARCH METHODS: Searches were

undertaken of MEDLINE, EMBASE, and PsycLIT. We searched for RCTs in references of all identified studies, meta-analyses and reviews. Date of most recent search: March 2012. SELECTION CRITERIA: RCTs with at least 10 participants in each arm post-treatment comparing psychological therapies with active treatment were eligible for inclusion (waiting list or standard medical care) for children or adolescents with episodic, recurrent or persistent pain. DATA COLLECTION AND ANALYSIS: All included studies were analysed and the quality of the studies recorded. All treatments were combined into one class: psychological treatments; headache and non-headache outcomes were separately analysed on three outcomes: pain, disability, and mood. Data were extracted at two time points; post-treatment (immediately or the earliest data available following end of treatment) and at follow-up (at least three months after the post-treatment assessment point, but not more than 12 months). MAIN RESULTS: Eight studies were added in this update of the review, giving a total of 37 studies. The total number of participants completing treatments was 1938. Twenty-one studies addressed treatments for headache (including migraine); seven for abdominal pain; four included mixed pain conditions including headache pain, two for fibromyalgia, two for pain associated with sickle cell disease, and one for juvenile idiopathic arthritis. Analyses revealed five significant effects. Pain was found to improve for headache and non-headache groups at post-treatment, and for the headache group at follow-up. Mood significantly improved for the headache group at follow-up, although, this should be interpreted with caution as there were only two small studies entered into the analysis. Finally, disability significantly improved in the non-headache group at posttreatment. There were no other significant effects. AUTHORS' CONCLUSIONS: Psychological treatments are effective in reducing pain intensity for children and adolescents (<18 years) with headache and benefits from therapy appear to be maintained. Psychological treatments also improve pain and disability for children with non-headache pain. There is limited evidence available to estimate the effects of psychological therapies on mood for children and adolescents with headache and nonheadache pain. There is also limited evidence to estimate the effects on disability in children with headache. These conclusions replicate and add to those of the previous review which found psychological therapies were effective in reducing pain intensity for children with headache and nonheadache pain conditions, and these effects were maintained at follow-up.

Fernandez-Perez, A. M., C. Villaverde-Gutierrez, et al. (2012). "Muscle trigger points, pressure pain threshold, and cervical range of motion in patients with high level of disability related to acute whiplash injury." <u>J Orthop Sports Phys Ther</u> **42**(7): 634-641.

STUDY DESIGN: Cross sectional cohort study. OBJECTIVE: To analyze

the differences in the prevalence of trigger points (TrPs) between patients with acute whiplash-associated disorders (WADs) and healthy controls, and to determine if widespread pressure hypersensitivity and reduced cervical range of motion are related to the presence of TrPs in patients with acute WADs. BACKGROUND: The relationship between active TrPs and central sensitization is not well understood in patients with acute WADs. METHODS: Twenty individuals with a high level of disability related to acute WAD and 20 age- and sex-matched controls participated in the study. TrPs in the temporalis, masseter, upper trapezius, levator scapulae, sternocleidomastoid, suboccipital, and scalene muscles were examined. TrPs are defined as hypersensitive spots in a palpable taut band, producing a local twitch response and referred pain when palpated. Pressure pain threshold (PPT) was assessed bilaterally over the C5-6 zygapophyseal joints, second metacarpal, and tibialis anterior muscle. Active cervical range of motion, neck pain, and self-rated disability using the Neck Disability Index were also assessed. RESULTS: The mean +/-SD number of TrPs for the patients with acute WAD was 7.3 +/- 2.8 (3.4 +/- 2.7 were latent TrPs; 3.9 +/- 2.5 were active TrPs). In comparison, healthy controls had 1.7 +/- 2.2 latent and no active TrPs (P<0.01). In patients with acute WAD, the most prevalent sites for active TrPs were the levator scapulae and upper trapezius muscles. The number of active TrPs increased with higher neck pain intensity (P<0.001) and a higher number of days since the accident (P=.003). Patients had significantly lower PPTs in all tested locations and less active cervical range of motion than controls (P<.001). In the patient group, there were significant negative correlations between the number of active TrPs and PPT over the C5-C6 joints and cervical range of motion in flexion, extension, and rotation in both directions: the greater the number of active TrPs, the lower the bilateral PPT over the neck and the greater the cervical range of motion limitation. CONCLUSIONS: The local and referred pain elicited from active TrPs reproduced neck and shoulder pain patterns in individuals with acute WADs with higher levels of disability. Patients with acute WADs exhibited widespread pressure hypersensitivity and reduced cervical mobility. The number of active TrPs was related to higher neck pain intensity, the number of days since the accident, higher pressure pain hypersensitivity over the cervical spine, and reduced active cervical range of motion.

Hassett, A. L. and D. A. Williams (2011). "Non-pharmacological treatment of chronic widespread musculoskeletal pain." <u>Best Pract Res Clin Rheumatol</u> **25**(2): 299-309.

Individuals with chronic widespread pain, including those with fibromyalgia, pose a particular challenge to treatment, given the modest effectiveness of pharmacological agents for this condition. The growing consensus indicates that the best approach to treatment involves the combination of pharmacological and non-pharmacological interventions. Several non-pharmacological interventions, particularly exercise and cognitive-behavioural therapy (CBT), have garnered good evidence of effectiveness as stand-alone, adjunctive treatments for patients with chronic pain. In this article, evidenced-based, non-pharmacological management techniques for chronic widespread pain are described by using two broad categories, exercise and CBT. The evidence for decreasing pain, improving functioning and changing secondary symptoms is highlighted. Lastly, the methods by which exercise and CBT can be combined for a multi-component approach, which is consistent with the current evidence-based guidelines of several American and European medical societies, are addressed.

Jensen, K. B., C. Berna, et al. (2012). "The use of functional neuroimaging to evaluate psychological and other non-pharmacological treatments for clinical pain." <u>Neurosci Lett</u> **520**(2): 156-164.

A large number of studies have provided evidence for the efficacy of psychological and other non-pharmacological interventions in the treatment of chronic pain. While these methods are increasingly used to treat pain, remarkably few studies focused on the exploration of their neural correlates. The aim of this article was to review the findings from neuroimaging studies that evaluated the neural response to distraction-based techniques, cognitive behavioral therapy (CBT), clinical hypnosis, mental imagery, physical therapy/exercise, biofeedback, and mirror therapy. To date, the results from studies that used neuroimaging to evaluate these methods have not been conclusive and the experimental methods have been suboptimal for assessing clinical pain. Still, several different psychological and non-pharmacological treatment modalities were associated with increased pain-related activations of executive cognitive brain regions, such as the ventral- and dorsolateral prefrontal cortex. There was also evidence for decreased pain-related activations in afferent pain regions and limbic structures. If future studies will address the technical and methodological challenges of today's experiments, neuroimaging might have the potential of segregating the neural mechanisms of different treatment interventions and elucidate predictive and mediating factors for successful treatment outcomes. Evaluations of treatment-related brain changes (functional and structural) might also allow for sub-grouping of patients and help to develop individualized treatments.

Kerstman, E., S. Ahn, et al. (2013). "Neuropathic pain." <u>Handb Clin Neurol</u> **110**: 175-187.

Neuropathic pain is a clinical entity that presents unique diagnostic and therapeutic challenges. This chapter addresses the classification, epidemiology, pathophysiology, diagnosis, and treatment of neuropathic pain syndrome. Neuropathic pain can be distinguished from nociceptive

pain based on clinical signs and symptoms. Although neuropathic pain presents a significant burden to individuals and society, a more accurate assessment of resource utilization, costs, and impairments associated with neuropathic pain would facilitate appropriate planning of healthcare policies. The underlying pathophysiology of neuropathic pain is not well defined. Several theories regarding the mechanism of neuropathic pain have been proposed, including central and peripheral nervous system sensitization, deafferentation, neurogenic inflammation, and the wind up theory. Neuropathic pain is a clinical diagnosis and requires a systematic approach to assessment, including a detailed history, physical examination, and appropriate diagnostic testing. The mainstay of treatment for neuropathic pain is pharmacological, including the use of antidepressants, antiepileptics, topical anesthetics, and opioids. Nonpharmacological treatments include psychological approaches, physical therapy, interventional therapy, spinal cord stimulation, and surgical procedures. Neuropathic pain is difficult to treat, but a combination of therapies may be more effective than monotherapy. Clinical practice guidelines provide an evidence-based approach to the treatment of neuropathic pain.

Lane, R., T. Nguyen, et al. (2012). "Functional popliteal entrapment syndrome in the sportsperson." <u>Eur J Vasc Endovasc Surg</u> **43**(1): 81-87.

OBJECTIVE: To define the clinical syndrome of functional popliteal entrapment comparing pre and post surgical clinical outcomes with pre and post-operative provocative ultrasonic investigations. Further, to suggest a management pathway to differentiate chronic exertional compartment syndromes and concomitant venous popliteal compression. METHODS: In 32 claudicant sportspersons, 55 limbs were characterised pre-surgery clinically, with provocative testing including hopping, and following a series of non-invasive tests. The clinical findings, ankle brachial indices (ABI) and duplex outcomes were compared preoperatively, at 3 months post-operatively (n = 52) and in the long term i.e. 16 months (n = 17). RESULTS: At 3 months, all 55 limbs had clinical follow up. 52 of the 55 limbs had follow up with ultrasound with provocative manoeuvres. The ABIs normalised in 46 (88%). There were 40 of 52 (76%) that became asymptomatic post surgery with a normal scan. There were 4 of 52 (8%) who were clinically asymptomatic but with residual obstruction on duplex and who were able to resume their usual lifestyle. There were 4 (8%) that had abnormal findings both on post-operative scan and clinically. Re-operation on 2 limbs corrected the duplex findings and the symptoms. There were 4 (8%) limbs that had normal duplexes but continued with symptoms albeit varied from the presenting symptoms. In the longer term, a further 2 became symptomatic at 2.8 years requiring a further successful intervention. (Concomitant popliteal venous obstruction

was present in 5 limbs (10%) on standing.) CONCLUSIONS: In the claudicating sportsperson, where there are no well characterised specific anatomical abnormalities, the syndrome can be characterised by provocative clinical (particularly hopping) and non-invasive tests. A positive clinical outcome with surgery can be predicted by abnormal presurgical ultrasonic investigations and confirmed later by a similar normal post surgical study. Concomitant venous compression may occur while standing with both syndromes related to muscle hypertrophy.

Linari-Melfi, M., I. Cantarero-Villanueva, et al. (2011). "Analysis of deep tissue hypersensitivity to pressure pain in professional pianists with insidious mechanical neck pain." <u>BMC Musculoskelet Disord</u> **12**: 268.

BACKGROUND: The aim of this study was to investigate whether pressure pain hyperalgesia is a feature of professional pianists suffering from neck pain as their main playing-related musculoskeletal disorder. METHODS: Twenty-three active expert pianists, 6 males and 17 females (age: 36 +/- 12 years) with insidious neck pain and 23 pianists, 9 males and 14 females (age: 38 +/- 10 years) without neck pain the previous year were recruited. A numerical pain rate scale, Neck Disability Index, hand size and pressure pain thresholds (PPT) were assessed bilaterally over the C5-C6 zygapophyseal joint, deltoid muscle, the second metacarpal and the tibialis anterior muscle in a blinded design. RESULTS: The results showed that PPT levels were significantly decreased bilaterally over the second metacarpal and tibialis anterior muscles (P < 0.05), but not over C5-C6 zygapophyseal joint and deltoid muscle (P > 0.10), in pianists with neck pain as compared to healthy pianists. Pianists with neck pain had a smaller (P < 0.05) hand size (mean: 181.8 +/- 11.8) as compared to pianists without neck pain (mean: 188. 6 +/- 13.1). PPT over the tibialis anterior muscles was negatively correlated with the intensity of neck pain. CONCLUSIONS: Our findings revealed pressure pain hypersensitivity over distant non-symptomatic distant points but not over the symptomatic areas in pianists suffering from neck pain. In addition, pianists with neck pain also had smaller hand size than those without neck pain. Future studies are needed to further determine the relevance of these findings in the clinical course of neck pain as playing-related musculoskeletal disorder in professional pianists.

Malouin, F. and C. L. Richards (2010). "Mental practice for relearning locomotor skills." <u>Phys Ther</u> **90**(2): 240-251.

Over the past 2 decades, much work has been carried out on the use of mental practice through motor imagery for optimizing the retraining of motor function in people with physical disabilities. Although much of the clinical work with mental practice has focused on the retraining of upperextremity tasks, this article reviews the evidence supporting the potential of motor imagery for retraining gait and tasks involving coordinated lowerlimb and body movements. First, motor imagery and mental practice are defined, and evidence from physiological and behavioral studies in healthy individuals supporting the capacity to imagine walking activities through motor imagery is examined. Then the effects of stroke, spinal cord injury, lower-limb amputation, and immobilization on motor imagery ability are discussed. Evidence of brain reorganization in healthy individuals following motor imagery training of dancing and of a foot movement sequence is reviewed, and the effects of mental practice on gait and other tasks involving coordinated lower-limb and body movements in people with stroke and in people with Parkinson disease are examined. Lastly, questions pertaining to clinical assessment of motor imagery ability and training strategies are discussed.

Mohamed, M. and C. K. Wong (2011). "More than meets the eye: clinical reflection and evidence-based practice in an unusual case of adolescent chronic ankle sprain." <u>Phys Ther</u> **91**(9): 1395-1402.

BACKGROUND AND PURPOSE: Adolescents who have chronic pain after common orthopedic injuries such as ankle sprains may present a multidimensional clinical problem stemming from both physical and psychological issues. A traumatic incident such as a motor vehicle accident can produce clinical issues ranging from a specific tissue injury to multisystem complications such as complex regional pain syndrome (CRPS) or posttraumatic stress disorder (PTSD). The purpose of this retrospective case report on an adolescent with chronic ankle pain stemming from a motor vehicle accident is to demonstrate how reflection and the evidence base influenced the modification of the plan of care. Description of the screening methods, clinical findings, interventions, and outcomes of the case may help physical therapists identify and improve the guality of care in cases of suspected CRPS and PTSD. CASE DESCRIPTION: The patient was a 12-year-old girl with a medical diagnosis of recurrent right ankle sprain and with signs of potential CRPS and PTSD. Poor initial response to ankle sprain management led to reflective reconsideration of the diagnosis and plan of care. The revised plan of care supported by the evidence base emphasized empathetic consideration of the traumatic motor vehicle accident and focused on CRPS prevention and management of potential non-physical pain via mirror therapy and motor imagery therapy. OUTCOMES: Pain was relieved, behavior improved, and functional movement began to normalize after 3 sessions of mirror therapy and motor imagery therapy. DISCUSSION: Patient symptoms were inconsistent with the medical diagnosis, and the clinical outcome of the original plan of care was unsuccessful. Reflection inspired a more-detailed history and systems review, which led to greater understanding and more-effective care.

Naleschinski, D. and R. Baron (2010). "Complex regional pain syndrome type I: neuropathic or not?" <u>Curr Pain Headache Rep</u> **14**(3): 196-202.

Complex regional pain syndrome (CRPS) is clinically characterized by pain, abnormal regulation of blood flow and sweating, edema of skin and subcutaneous tissues, active and passive movement disorders, and trophic changes. It is classified as type I (reflex sympathetic dystrophy) and type II (causalgia). CRPS cannot be reduced to one system or to one mechanism only. In the past decades, there has been absolutely no doubt that complex regional pain syndromes have to be classified as neuropathic pain disorders. This situation changed when a proposal to redefine neuropathic pain states was recently published, which resulted in an exclusion of CRPS from neuropathic pain disorders. We analyzed the strength of the scientific evidence that supports the neuropathic nature of complex regional pain syndromes.

O'Connell, N. E., B. M. Wand, et al. (2013). "Interventions for treating pain and disability in adults with complex regional pain syndrome." <u>Cochrane Database</u> <u>Syst Rev</u> **4**: CD009416.

BACKGROUND: There is currently no strong consensus regarding the optimal management of complex regional pain syndrome although a multitude of interventions have been described and are commonly used. OBJECTIVES: To summarise the evidence from Cochrane and non-Cochrane systematic reviews of the effectiveness of any therapeutic intervention used to reduce pain, disability or both in adults with complex regional pain syndrome (CRPS). METHODS: We identified Cochrane reviews and non-Cochrane reviews through a systematic search of the following databases: Cochrane Database of Systematic Reviews, Database of Abstracts of Reviews of Effects (DARE), Ovid MEDLINE, Ovid EMBASE, CINAHL, LILACS and PEDro. We included non-Cochrane systematic reviews where they contained evidence not covered by identified Cochrane reviews. The methodological quality of reviews was assessed using the AMSTAR tool. We extracted data for the primary outcomes pain, disability and adverse events, and the secondary outcomes of quality of life, emotional well being and participants' ratings of satisfaction or improvement. Only evidence arising from randomised controlled trials was considered. We used the GRADE system to assess the quality of evidence. MAIN RESULTS: We included six Cochrane reviews and 13 non-Cochrane systematic reviews. Cochrane reviews demonstrated better methodological quality than non-Cochrane reviews. Trials were typically small and the guality variable. There is moderate quality evidence that intravenous regional blockade with guanethidine is not effective in CRPS and that the procedure appears to be associated with the risk of significant adverse events. There is low quality evidence

that bisphosphonates, calcitonin or a daily course of intravenous ketamine may be effective for pain when compared with placebo; graded motor imagery may be effective for pain and function when compared with usual care; and that mirror therapy may be effective for pain in post-stroke CRPS compared with a 'covered mirror' control. This evidence should be interpreted with caution. There is low quality evidence that local anaesthetic sympathetic blockade is not effective. Low quality evidence suggests that physiotherapy or occupational therapy are associated with small positive effects that are unlikely to be clinically important at one year follow up when compared with a social work passive attention control.For a wide range of other interventions, there is either no evidence or very low guality evidence available from which no conclusions should be drawn. AUTHORS' CONCLUSIONS: There is a critical lack of high quality evidence for the effectiveness of most therapies for CRPS. Until further larger trials are undertaken, formulating an evidence-based approach to managing CRPS will remain difficult.

Pasnoor, M., M. M. Dimachkie, et al. (2013). "Cryptogenic sensory polyneuropathy." <u>Neurol Clin</u> **31**(2): 463-476.

Chronic sensory or sensorimotor polyneuropathy is a common cause for referral to neurologists. Despite extensive diagnostic testing, up to one-third of these patients remain without a known cause, and are referred to as having cryptogenic sensory peripheral neuropathy. Symptoms progress slowly. On examination, there may be additional mild toe flexion and extension weakness. Electrophysiologic testing and histology reveals axonal neuropathy. Prognosis is usually favorable, as most patients maintain independent ambulation. Besides patient education and reassurance, management is focused on pharmacotherapy for neuropathic pain and physical therapy for balance training, and, occasionally, assistive devices.

Posadzki, P. and E. Ernst (2011). "Guided imagery for musculoskeletal pain: a systematic review." <u>Clin J Pain</u> **27**(7): 648-653.

AIMS: The objective of this systematic review was to assess the effectiveness of guided imagery (GI) as a treatment option for musculoskeletal pain (MSP). METHOD: Six databases were searched from their inception to May 2010. All controlled clinical trials were considered, if they investigated GI in patients with any MSP in any anatomic location and if they assessed pain as an outcome measure. Trials of motor imagery were excluded. The selection of studies, data extraction, and validation were performed independently by 2 reviewers. RESULTS: Nine randomized clinical trials (RCTs) met the inclusion criteria. Their methodologic quality ranged between 1 and 3 on the Jadad scale. Eight RCTs suggested that GI leads to a significant reduction of MSP. One RCT indicated no change in MSP in comparison with usual

care. CONCLUSIONS: It is concluded that there are too few rigorous RCTs testing the effectiveness of GI in the management of MSP. Therefore, the evidence that GI alleviates MSP is encouraging but inconclusive.

Posadzki, P., W. Lewandowski, et al. (2012). "Guided imagery for nonmusculoskeletal pain: a systematic review of randomized clinical trials." <u>J Pain</u> <u>Symptom Manage</u> **44**(1): 95-104.

CONTEXT: Our previous review of the literature concluded that there is encouraging evidence that guided imagery alleviates musculoskeletal pain, but the value of guided imagery in the management of nonmusculoskeletal pain remains uncertain. OBJECTIVES: The objective of this systematic review was to assess the effectiveness of guided imagery as a treatment option for non-musculoskeletal pain. METHODS: Six databases were searched from their inception to February 2011. Randomized clinical trials were considered if they investigated guided imagery in human patients with any type of non-musculoskeletal pain in any anatomical location and assessed pain as a primary outcome measure. Trials of motor imagery and hypnosis were excluded. The selection of studies, data extraction, and validation were performed independently by two reviewers. RESULTS: Fifteen randomized clinical trials met the inclusion criteria. Their methodological guality was generally poor. Eleven trials found that guided imagery led to a significant reduction of non-musculoskeletal pain. Four studies found no change in nonmusculoskeletal pain with guided imagery in comparison with progressive relaxation, standard care, or no treatment. CONCLUSION: The evidence that guided imagery alleviates non-musculoskeletal pain is encouraging but remains inconclusive.

Shoup, D. (2006). "An osteopathic approach to performing arts medicine." <u>Phys</u> <u>Med Rehabil Clin N Am</u> **17**(4): 853-864, viii.

An osteopathic approach to the performing artist is a complete approach. It involves spending adequate time with the performing artist to obtain a complete history and evaluation. It requires attention to the performer's lifestyle, practice habits, exercise routine, nutrition, stress level, and coexisting medical problems. Because an injury to a performing artist can be physically, emotionally, and financially devastating, these patients deserve a comprehensive treatment plan to allow for the best opportunity for recovery.

Smith, B. K., T. Engelbert, et al. (2013). "Foot claudication with plantar flexion as a result of dorsalis pedis artery impingement in an Irish dancer." <u>J Vasc Surg</u>. Dorsalis pedis artery impingement is an extremely rare cause of foot claudication, with a single case reported in the literature. In this report, we describe the case of a 17-year-old female Irish dancer who presented with intermittent bilateral foot pain and discoloration during active plantar flexion.

Votrubec, M. and I. Thong (2013). "Neuropathic pain--a management update." Aust Fam Physician **42**(3): 92-97.

BACKGROUND: Neuropathic pain is described as burning, painful, cold or electric shocks and may be associated with tingling, pins and needles, numbness or itching. OBJECTIVE: This article summaries the diagnosis and management of four common neuropathic pain presentations. DISCUSSION: A validated diagnostic screening tool can help identify patients with neuropathic pain. A systematic approach to clinical assessment and investigation will clarify the diagnosis. Good glycaemic control is important in the prevention and management of diabetic polyneuropathy; management options include antidepressants. gabapentinoids and controlled release opioids. Pain that lasts for more than 3 months after the onset of a herpes zoster infection is called 'postherpetic neuralgia'; management options include prevention with vaccination, early antiviral treatment and gabapentinoids, tricyclic antidepressants, controlled release opioids, capsaicin cream and lignocaine patches. In trigeminal neuralgia, patients complain of severe brief episodes of pain in the distribution of one or more branches of the fifth cranial nerve; first line management is with carbamazepine. Complex regional pain syndrome is diagnosed using the Budapest Diagnostic Criteria. Few clinical trials are available to guide the treatment of complex regional pain syndrome, which includes pharmacological and surgical options.

Vranken, J. H. (2009). "Mechanisms and treatment of neuropathic pain." <u>Cent</u> <u>Nerv Syst Agents Med Chem</u> **9**(1): 71-78.

Neuropathic pain (pain associated with lesions or dysfunction of nervous system) is relatively common, occurring in about 1% of the population. Studies in animal models describe a number of peripheral and central pathophysiological processes after nerve injury that would be the basis of underlying neuropathic pain mechanism. A change in function, chemistry, and structures of neurons (neural plasticity) underlie the production of the altered sensitivity characteristics of neuropathic pain. Peripheral sensitization acts on the nociceptors, and central sensitization takes place at various levels ranging from the dorsal horn to the brain. In addition, abnormal interactions between the sympathetic and sensory pathways contribute to mechanisms mediating neuropathic pain. Despite recent advances in identification of peripheral and central sensitization mechanisms related to nervous system injury, the effective treatment of patients suffering from neuropathic pain remains a clinical challenge.

Although numerous treatment options are available for relieving neuropathic pain, there is no consensus on the most appropriate treatment. However, recommendations can be proposed for first-line, second-line, and third-line pharmacological treatments based on the level of evidence for the different treatment strategies. Beside opioids, the available therapies shown to be effective in managing neuropathic pain include anticonvulsants, antidepressants, topical treatments (lidocaine patch, capsaicin), and ketamine. Tricyclic antidepressants are often the first drugs selected to alleviate neuropathic pain (first-line pharmacological treatment). Although they are very effective in reducing pain in several neuropathic pain disorders, treatment may be compromised (and outweighed) by their side effects. In patients with a history of cardiovascular disorders, glaucoma, and urine retention, pregabalin and gabapentine are emerging as first-line treatment for neuropathic pain. In addition these anti-epileptic drugs have a favourable safety profile with minimal concerns regarding drug interactions and showing no interference with hepatic enzymes. Despite the numerous treatment options available for relieving neuropathic pain, the most appropriate treatment strategy is only able to reduce pain in 70% of these patients. In the remaining patients, combination therapies using two or more analgesics with different mechanisms of action may also offer adequate pain relief. Although combination treatment is clinical practice and may result in greater pain relief, trials regarding different combinations of analgesics are lacking (which combination to use, occurrence of additive or supra-additive effects, sequential or concurrent treatment, adverse-event profiles of these analgesics, alone and in combination) are lacking. Additionally, 10% of patients still experience intractable pain and are refractory to all forms of pharmacotherapy. If medical treatments fail, invasive therapies such as intrathecal drug administration and neurosurgical interventions may be considered.

Weichman, K., T. Berland, et al. (2010). "Intermittent foot claudication with active dorsiflexion: the seminal case of dorsalis pedis artery entrapment." <u>Ann Vasc</u> <u>Surg</u> **24**(1): 113 e111-115.

BACKGROUND: Atypical claudication is a relatively uncommon problem within the general population. However, suspicion for the diagnosis is raised when young and athletic patients present with symptoms of claudication during exercise. The most common causes of atypical claudication are anatomical variants, including popliteal artery entrapment syndrome and tarsal tunnel syndrome. These variants result in impaired arterial flow and nerve compression, respectively. In this report, we present a seminal case of dorsalis pedis artery entrapment by the extensor hallucis brevis tendon during active dorsiflexion of the foot. METHODS: The patient was a 42-year-old male without significant past

medical history, who presented with claudication in both feet upon active dorsiflexion. He underwent dynamic arterial duplex studies that first revealed normal flow in the neutral position and then revealed complete cessation of flow in both duplex and Doppler modes on dorsiflexion of the foot. He also underwent dynamic magnetic resonance angiography of bilateral lower extremities that revealed an incomplete pedal arch with early termination of the posterior tibial artery on static images and termination of the dorsalis pedis artery at notching on the dorsum of the foot during dorsiflexion. The patient was taken to the operating room for bilateral dorsalis pedis artery exploration. During exploration, the patient was found to have entrapment of the dorsalis pedis artery by the extensor hallucis brevis (EHB) tendon. This was documented by both direct visualization and intraoperative cessation of Doppler signal on dorsiflexion. Since the EHB tendon provides only secondary function to the extensor hallucis longus (EHL) tendon, the EHB was transected near its insertion and transposed directly to the EHL tendon. This allowed for normal extensor function of the great toe and restored triphasic Doppler signals during dorsiflexion. CONCLUSION: Dorsalis pedis arterial entrapment is a novel cause of atypical claudication. It is extremely uncommon as patients must have both abnormal anatomy and an incomplete pedal arch to display symptoms. Similar to other entrapment syndromes, if identified before permanent arterial scarring, the treatment does not require a bypass procedure. Removal of the tendon along with transposition will allow cessation of symptoms without impaired dorsiflexion of the great toe.

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