Top Ten Things Rheumatologists Should (And Might Not) Know About the Physiatrist's Perspective on Rehabilitation Strategies and Interventions for Neuromusculoskeletal Conditions

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Rehabilitation strategies and interventions encompass a broad range of treatment modalities, from activity modification and exercise prescriptions to medication management and interventional procedures. Physical medicine and rehabilitation is a broad specialty, caring for individuals with a wide range of neurological and musculoskeletal disorders. This article provides insight into the physiatrist's perspective regarding neuromusculoskeletal conditions frequently encountered by rheumatologists.

1. Low back pain, a leading cause of disability, requires determination of potential pain generators to guide interventional treatments. Non-inflammatory back pain is divided as axial, affecting the back itself, or radicular, with pain radiating to the buttocks or legs. Facet-joint-mediated pain contributes to 40% of axial low back pain and can be successfully treated with radiofrequency denervation techniques. For radicular pain, transforaminal epidural steroid injections can provide significant symptomatic relief and expedite recovery. In refractory cases, neurostimulation is an emerging therapy. Surgical management is usually restricted to patients with progressive neurological deficits.[1-3]

2. Greater trochanteric pain syndrome is commonly labelled as bursitis but should instead be considered a tendinopathy affecting the gluteus medius/minimus and iliotibial band. True bursitis is present in only a minority of patients. Gluteal tears can be evaluated using the resisted external derotation test. Ultrasound-guided needle tenotomy, in combination with physiotherapy, can provide reasonable medium- to long-term relief, and represents a better option than corticosteroid injections.[4-6]

3. The sacroiliac joint (SIJ) is an important pain localization in non-inflammatory back pain. Pain generators in the SIJ include the joint capsule, surrounding ligaments, and the intra-articular portion of the joint, all innervated by the lateral branches of the S1-S3 nerve roots. Due to this complex anatomy, physical examination maneuvers may not be as accurate and intra-articular injections may not adequately interrogate all pain generators, resulting in false negative diagnoses. Techniques utilizing imaging-guided blocks to the posterior sacral network may represent a new gold standard in diagnosis and management of SIJ-mediated pain.[7-11]

4. Myofascial pain syndrome needs to be differentiated from fibromyalgia. Clinical features of palpable taut bands and trigger points are usually present, and the area of pain involvement is more focal, compared to the widespread pain typical of fibromyalgia. Treatment includes targeted stretching and active strengthening exercises of the involved muscles, while techniques such as intramuscular stimulation ("dry needling") and trigger point injections with local anesthetic can be helpful for short-term pain reduction to facilitate active rehabilitation.[12,13]

5. Nerve conduction studies (NCS) and electromyography (EMG) testing have technical limitations and knowing when to order them is important. Standard NCS and EMG testing is very useful for identifying abnormalities in the major large-fiber peripheral nerves, such as focal entrapment neuropathies (e.g. carpal tunnel syndrome) or traumatic nerve injuries. EMG studies

are also helpful for distinguishing acute inflammatory myopathies from chronic myopathies. However, pathology involving small-fiber peripheral nerves, a common cause of painful distal polyneuropathies, is more difficult to measure and standard NCS/EMG can be normal in these cases.[14]

6. Small-fiber polyneuropathy (SFPN), involving the myelinated Aδ-fibers and unmyelinated C-fibers, is found in approximately 40-50% of patients with fibromyalgia. Symptoms of dysautonomia and paresthesias may predict underlying SFPN, and abnormalities in sural and medial plantar sensory NCS can aid diagnosis. Identifying this overlap is important to rule-out reversible causes of SFPN and identify patients who may respond better to antiepileptics or antidepressants for pain. Opioids are discouraged, but adjuvant treatments including topical local anesthetics, capsaicin, and acupuncture may be helpful.[15-18]

7. Complex regional pain syndrome (CRPS) is a rehabilitative emergency, and requires urgent treatment with appropriate analgesic medication, possible oral corticosteroids, and aggressive active rehabilitation strategies. When early treatment is not possible or there is a lack of response, CRPS unfortunately develops into a chronic neurological and pain condition. The key feature of CRPS is regional pain out of proportion to any inciting event, with features of neuropathic pain, skin and temperature changes, and significant loss of functional movement. Level 1 evidence exists for use of oral corticosteroids in early or acute cases, and appropriate analgesia is important to promote participation in active rehabilitation exercises and modalities.[19,20]

8. Post stroke joint pain is often complex and may arise from multiple etiologies. Shoulder pain can arise from subluxation due to neuromuscular weakness, rotator cuff tendinopathy or glenohumeral osteoarthritis flare due to altered mechanics, spasticity of the shoulder girdle muscles, or adhesive capsulitis. If hand and shoulder pain is noted, assess for shoulder-hand syndrome, a form of post-stroke CRPS. Post-stroke knee pain is common, due to altered mechanics aggravating underlying knee osteoarthritis or flares of gout from the acute medical event and associated medications. Consider use of functional electrical stimulation (FES), topical NSAIDs, and short courses of oral NSAIDs. Targeted injections of intra-articular corticosteroids are effective in providing medium-term pain relief to promote active rehabilitation for neurological recovery.[21]

9. Inflammatory arthritis may remit on the hemiparetic side after stroke, but the pathophysiology of this phenomenon is unclear. Case reports have suggested that inflammatory arthritis resolves on the hemiparetic side following stroke or other significant central nervous system injury. Proposed mechanisms include altered mechanical factors on the hemiparetic side, changes in the autonomic nervous system affecting inflammation, or changes in limb perfusion. Hemiparetic limbs frequently develop autonomic changes such as edema, altered temperature, and altered skin colour and sweat pattern. Further work to elucidate the role of the central nervous system on inflammation will be helpful to understand this anecdotal phenomenon.[22,23]

10. Plantar fasciitis is a common cause of heel pain and can be related to systemic inflammatory conditions or specific biomechanical issues. Predisposing factors include pes cavus deformity, limited range of ankle dorsiflexion, tightness of the gastrocnemius and soleus, and excessive foot pronation/supination. Correction of biomechanical abnormalities with measures such as targeted stretching, modified shoe wear, use of orthoses (e.g. heel lift), strengthening of the intrinsic foot muscles, and deep friction massage can resolve this condition. In refractory cases, imaging-guided corticosteroid injections provide short-term relief, allowing rehabilitation techniques to be better tolerated and more effective. Other options include extracorporeal shock-wave therapy, botulinum toxin A intramuscular injections, prolotherapy and autologous platelet-rich plasma, but these interventions have conflicting evidence regarding efficacy. Surgical management is reserved for rare cases.[24-28]

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