Physical Therapy in the Treatment of Chronic Headache

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Careful evaluation of the chronic headache patient can reveal the presence or absence of musculoskeletal factors to be causative or contributory to the headache condition. This article presents a review of the literature surrounding physical findings in patients with cervicogenic and musculoskeletal sources of pain and specific treatment with physical therapy. Included in the discussion is the identification of muscular and joint involvement in a given headache, the relevance of postural, range of motion, and strength deficits, and appropriate individualized treatment strategies with a review of relevant outcome studies. Possible mechanisms for treatment effects also are considered.

Introduction

Patterns of referral to and use of physical therapy to treat patients with headache vary widely from practitioner to practitioner. The decision that a particular headache patient is appropriate for physical therapy lies in determining to what extent peripheral mechanisms are contributing to a particular headache condition. On one end of the spectrum, a headache may result solely from musculoskeletal and biomechanical dysfunction and may resolve completely with appropriate physical therapy intervention. On the other end, a patient may complain of neck stiffness during classic migraine with aura and the process may be entirely centrally mediated, with poor prognosis for managing symptoms with physical intervention. However, many headache conditions fall somewhere between these two presentations, raising questions about the use of physical therapy. What to do with the chronic daily headache sufferer? What is the prognosis for the patient with tension type headache? How do we determine a cervical component of a mixed headache condition? What, if any, precise physical therapy intervention is appropriate for a particular headache patient?

Diagnosis

Classification of cervicogenic pain

For most patients with cervicogenic headache or cervicogenic component to headache, cervical disease is not significant or causative. These patients usually present with minimal findings on magnetic resonance imaging, but a cervical source of pain is revealed when a history and examination are performed. In these cases, the source of pain likely lies in the musculoskeletal system. Part of the difficulty in accurate identification of causative cervical dysfunction lies in our current classification system for headache and in our less-than-adequate understanding of the pathogenesis of headaches, particularly when it comes to peripheral mechanisms. There continues to be considerable debate regarding the extent to which neuromusculoskeletal structures in the neck are responsible for chronic headache. The International Headache Society recognizes headache "associated with biomechanical lesions of the cervical spine" as a diagnostic category. Diagnostic criteria include cervical/occipital pain with possible projection to the frontal, orbital, temporal, vertex, or ear regions, pain precipitated by neck movements or sustained postures, and physical findings on examination (*eg,* range of motion restrictions or increased muscle tenderness or tone) [1]. The International Association for Study of Pain [2] and Sjaastad *et al.* [3] add unilaterality of headache to the diagnostic criteria. Nonetheless, debate remains active. Sjaastad *et al.* [4] introduced the term cervicogenic headache (and much research has ensued) using the alleviation of pain by diagnostic blockade as the gold standard for diagnosis. The difficulty with diagnosing cervicogenic headache using nerve blockade lies in the fact that successful obliteration of the pain implies that the pain is transmitted by the nerve in question or, in the case of facet blocks, identifies an irritable joint. However, it does not identify the possible biomechanical perpetuators of the pain, which may go untreated in favor of more invasive procedures such as rhizolysis [5]. Although facet blocks can be a useful adjunct to physical therapy by reducing pain so that the patient can tolerate therapy better, conservative measures should not be overlooked in favor of invasive procedures.

Furthering the difficulty in determining whether musculoskeletal factors are significant in a given headache is the presence of mixed headache and the overlap of the diagnostic criteria and common features of various headache syndromes. Fishbain *et al.* [6] examined 1466 consecutive patients referred for treatment of neck or back pain and found 10.5% of them suffering from severe disabling headaches. Using the International Headache Society criteria, 44.2% had more than one headache diagnosis. Patients with cervicogenic headache had the greatest degree of overlap (94.2%); patients with migraine headache experienced the second greatest degree of overlap at 68.3%. The most frequent headache trigger was mental stress followed by neck position and activity/exercise. Vincent and Luna [7] found that 30% of a group of cervicogenic headache patients also met the criteria for migraine. Sjaastad *et al.* [8] have demonstrated the coexistence of cervicogenic headache and migraine without aura. Pfaffenrath *et al.* [9] found one-third of a group of patients with cervicogenic headache presenting with symptoms frequently associated with migrainous headache, including lacrimation, conjunctival injection, lid edema, and visual blurring. Nausea, vomiting, photophobia, and phonophobia also were experienced by some of the patients in the study. Photophobia and phonophobia have been shown to be equally prevalent in patients with tension-type headache and cervicogenic headache [10]. With the frequent presence of symptoms commonly associated with migraine and tension-type headache in patients with cervicogenic headache, specific examination skills adequate to reveal the presence of treatable musculoskeletal dysfunction contributing to a headache become crucial to the practitioner. Only in this way can appropriate and effective management ensue. Cervicogenic headache prevalence is similar to that of migraine. Studies indicate that nearly 18% of headache sufferers in the general population have cervicogenic headache [11], as do approximately 14% of those seeking treatment [12].

The role of the muscles in tension-type headache

It was once thought that tension headache primarily resulted from pericranial and cervical muscle contraction [13]. Although no longer the prevailing paradigm for understanding the relationship between muscle tension and headache, the role of myofascial structures in tension-type headache is still not well understood. It has been suggested that myofascial nociception, defined loosely as pericranial tenderness, is critical in the genesis of episodic tension-type headache, but eventual central sensitization accounts for transformation into chronic tension-type headache [14]. However, local tenderness in pericranial tissues on manual palpation is not a very sensitive diagnostic tool in determining the degree of a peripheral source of pain. Patients with fibromyalgia show significantly increased local tenderness on palpation and the mechanisms are entirely central [15]. The diagnosis of myofascial pain (to be contrasted with tenderness of the myofascial tissues) is made based on a physical examination that includes palpation of tender nodules or trigger points (TrPs) that refer pain to a distant site, reproducing all or some of a patient's primary pain complaint. The presence of a "jump sign" with palpation and relief of pain when the TrP is inactivated are added to this reproduction of pain to complete the minimal criteria for diagnosing myofascial pain [16••]. The occurrence of palpable taut bands in the skeletal muscles and the presence of a local twitch response on snapping palpation or needle insertion further affirm the diagnosis, but may be challenging to achieve if the TrP occurs in deep muscles or when the overlying subcutaneous tissue is thick. However, these criteria generally are not applied in the clinical diagnosis of tension-type headache, nor are they widely used in the literature surrounding tension-type headache. Therefore, it would be safe to say that, in any patient, the presence or absence of myofascial pain as a primary source of head pain needs to be determined by the evaluator. Gerwin *et al.* [17] demonstrated good interrater reliability in the diagnosis of TrPs with precise examiner training and extensive clinical experience only.

Although TrPs in the cervical and masticatory muscles may be primary sources of headache [18,19], they also may contribute to a headache or act as a trigger or precipitant to a headache that is not cervicogenic [20]. Kidd and Nelson [21] demonstrated increased cervical musculoskeletal dysfunction in patients diagnosed with migraine and tension-type headache compared with control subjects and suggested a contributory rather than causative role of the musculoskeletal system in these conditions. Aggressive palpation of TrPs may trigger migraine attacks [22] and pressure over TrPs during a migraine headache can increase the pain [23].

Upper cervical facet joint dysfunction

Biomechanical dysfunction of the upper cervical joints, particularly C0–C1, C1–C2, and C2–C3, is consistently identified in patients with cervicogenic headache when these levels are assessed specifically. In one study, 91% of a group of patients with cervicogenic headache had segmental movement dysfunction of C0–C1 or C1–C2 joints and all of them exhibited three or more TrPs on the symptomatic side [19]. In another study [24], segmental mobility and pain on passive intervertebral motion testing was assessed by blinded examiners in a group of patients with postconcussion headache and healthy control subjects. Symptomatic hypomobile joints differentiated the headache patients from the control subjects. Jull *et al.* [25] investigated the accuracy of a manual diagnosis of cervical zygapophysial joint dysfunction and pain. Twenty patients were divided into two groups. The first group was evaluated by means of diagnostic nerve blocks to establish the presence and location of symptomatic zygapophysial joints. Then they were seen by the therapist for a blinded biomechanical assessment. The second group was examined first by the therapist who diagnosed the presence and level of joint dysfunction and, subsequently, a diagnostic facet block was administered to confirm the musculoskeletal findings. The therapist had 100% accuracy in diagnosing the presence and level of symptomatic joints. A study conducted as a prelude to a large, randomly controlled trial on the effectiveness of physical therapy as a treatment for cervicogenic headache looked at interexaminer reliability in the detection of upper cervical joint dysfunction [26]. Examiners at four different centers, using a variety of clinical techniques, manually assessed each patient's upper cervical spine in a single-blind manner. There was excellent agreement between each pair of examiners regarding whether dysfunction existed and 70% agreement among examiners on the two most dysfunctional joints in symptomatic patients. These are important findings because the routine use of invasive blockade as a screening tool for cervicogenic headache is difficult to justify when more conservative, less costly, reliable diagnostic measures exist. Diagnostic nerve blocks should be used for patients who have failed appropriate conservative management to determine whether more long-term procedures such as rhizolysis should be pursued.

Posture, range of motion, strength, and neuromuscular control

As screening measures for cervicogenic headache, active cervical range of motion (AROM) and the presence of a forward head posture (FHP) have not been shown conclusively to be reliable predictors, although there is some evidence supporting their value. For example, there is evidence that gross range of motion is reduced in patients with cervicogenic headaches compared with those with tension-type headache and common migraine [27]. Cervical AROM also has been reported to accurately discriminate between patients with persistent whiplash-related disorders (including headache) and asymptomatic patients [28]. Restrictions in length of the upper trapezius muscles in patients with cervicogenic headache also have been reported [5]. Dumas *et al.* [29] found significantly reduced flexion/extension and rotation range of motion in patients with post-traumatic cervicogenic headaches, but not in those with idiopathic onset. However, several studies have failed to find significant restriction in AROM in patients with post-concussive headache, even in the presence of upper cervical joint dysfunction and increased muscle tightness [24] or in a group of randomly selected patients with neck pain [30]. The relevance of postural dysfunction is equally unclear. Watson and Trott [31] compared a group of patients with cervicogenic headache with control subjects and demonstrated a statistically significant relationship between FHP and the presence of cervicogenic headache. However, subsequent studies have failed to reproduce this finding [5,24,29]. A study of 88 healthy volunteers revealed that those with more severe postural abnormalities reported significantly increased incidences of pain. Specifically, those with a substantial FHP had an increased incidence of cervical, interscapular, and head pain [32]. An interesting finding in patients with tension-type headache (not cervicogenic pain) was reported in a roentgenographic study of 372 patients with tension-type headache and 225 healthy control subjects. The results indicated that a straightened cervical spine and low-set shoulders were present more frequently in those with tension-type headache. The authors postulate that tensioning of the brachial plexus may be relevant in this finding [33].

There has been great consistency in the finding of weakness in the deep cervical flexor musculature for patients with cervicogenic headaches of traumatic and nontraumatic onset. Jull *et al.* [5] have developed a test for measuring the strength and endurance of the deep cervical flexors, specifically the longus colli and capitus and rectus capitus. The longus colli muscle has been shown to be a significant stabilizer of the cervical spine, with increased activity during talking, coughing, and swallowing [34]. It lies just anterior to the spine, with origins and insertions confined to the vertebrae, thus directly producing intersegmental flexion on contraction [35]. The longus colli has been shown to counteract the cervical lordosis [36]. The more superficial neck flexors, such as the sternocleidomastoid and scalenes, exert their force through a longer lever arm, with direction of force essentially creating lower cervical flexion with a tendency to upper cervical extension. Therefore, to test the deep cervical flexors, one must isolate upper cervical flexion motion (cranio-cervical flexion) and capture strength and endurance measurements. Jull *et al.* [5] used an inflatable pressure sensor (Stabilizer, Chattanooga, South Pacific) placed under the supine patient's cervical spine and inflated to 20 mm Hg. The monitor measures the slight flattening of the cervical lordosis on contraction of the deep cervical flexors. Using this technology in controlled, blinded studies, the group has been able to demonstrate measurable reduction in strength and endurance of the deep flexors, with concurrent tendency toward overuse of the superficial flexors, in patients with cervicogenic headache and those with post-traumatic headache [5,37]. Other examiners also have reported findings of weak flexors in patients with head pain, neck pain, and post-traumatic headache [24,29,31,38].

The concepts of neuromuscular control and re-education are important when discussing the evaluation of patients with pain. The presence of pain produces complex changes in the neuromuscular system, with increased muscular activity in some muscle groups and decreased or inhibited activity in others [39]. Lund *et al.* [40] described reduced agonist activity and increased antagonist activity in the presence of pain, thereby reducing force production and velocity of movement. Nederhand *et al.* [41] demonstrated a difference in muscle activation patterns in patients with chronic whiplash disorder compared with control subjects. Those with pain showed higher activation levels of the upper trapezius muscles during physical exercise and a decreased ability to relax the muscles afterwards. Further studies have demonstrated an imbalance in the recruitment and motor control of the deep segmental stability system (such as in the case of the deep versus superficial cervical flexors) [42], with evidence that correction of improper muscle firing patterns results in decreased pain and improved function [43••]. When these concepts are applied in rehabilitation, the focus moves away from aggressive, repetitive strengthening of large groups of muscles using weight machines and toward re-education of proper muscle firing patterns, with patients practicing exercises designed to enhance motor recruitment and control.

Although active range of motion and postural dysfunction have not been proven definitively to be predictors of cervicogenic headaches, clinical experience supports their merit in the clinical setting. These measures are relatively simple and accessible to examiners without advanced training in palpation and manual assessment. Their appropriate application will identify at least a portion of those with musculoskeletal dysfunction. The more subtle evaluation of intersegmental joint range of motion and deep cervical flexor strength requires more precise training, but also will reveal a greater number of patients who may benefit from physical therapy.

Treatment

Treating myofascial pain

If myofascial TrPs are identified as sources of head pain in an individual patient (whether they appear to be primary or merely contributory to the headache condition), they must be addressed. Treatment of myofascial TrPs has two primary objectives: to increase the resting length of the involved muscle fibers and to reduce or eliminate perpetuating factors that sustain myofascial pain. Stretching the involved muscle after TrP injection has been found to improve efficacy of the injection [44] and stretching in combination with ultrasonography or injections has been found to be more effective than stretching alone [45]. Stretches should be held for a long duration to maximize effectiveness [46] and can incorporate contract/relax techniques using the properties of post-contraction relaxation of a muscle to further progress the stretch [47]. Ischemic compression has been shown to reduce TrP sensitivity after application [48]. A home exercise program using ischemic compression and sustained stretching was shown to be significantly better than range-of-motion exercises in reducing pressure pain thresholds and average pain intensity [49]. Treatment directed at myofascial pain generally is less effective when associated with concurrent widespread pain or fibromyalgia [50,51].

Although stretching the involved musculature is of critical importance, arguably even more crucial is adequate reduction of perpetuators of myofascial pain. Perpetuators can include improper ergonomics, faulty postures, underlying joint or somatic dysfunction, structural asymmetries (*eg,* short leg), poor strength or motor control, psychologic stress, and metabolic/endocrine disorders [16••]. Correction of faulty ergonomics in the workplace has been shown to reduce muscular stress and pain over subsequent months and years [52]. When sitting for prolonged periods of time, frequent microbreaks taken every 20 minutes are effective in reducing discomfort and myoelectric activity in muscles [53]. Graff-Radford *et al.* [18] demonstrated that a comprehensive musculoskeletal rehabilitation program for patients diagnosed with myofascial pain, which systematically addresses all of the identified perpetuating factors (*eg,* postural stresses, poor body mechanics, dysfunctions of the cervical spine, psychologic stress, and depression), is effective in reducing self reports of pain and medication intake, with treatment gains maintained at 1 year. Although most practitioners treating myofascial pain do not have access to such a comprehensive program, lessons can be taken from this study. The identification of perpetuating factors and the provision of suggestions for reducing or eliminating them should be a part of each office visit until all of the factors are addressed adequately.

Patient education cannot be overemphasized for those with myofascial pain. Studies comparing patients with myofascial pain with patients with other chronic pain syndromes indicate that those with myofascial pain are more likely to think there is something more seriously wrong with them than what their diagnosis implies [54]. They also are more likely to subscribe to maladaptive belief systems, such as catastrophizing about their pain and endorsing the belief that their pain represents a signal of harm or damage, that have been shown to predict disability among a chronic pain population [55]. These data support the importance of thorough patient education, including the nature and behavior of myofascial pain and musculoskeletal dysfunction. Patients should be disavowed of any faulty beliefs about the genesis of their pain and should be supported through their treatment with repeated reassurance and encouragement, particularly as it relates to ongoing compliance with necessary exercises and lifestyle changes.

Physical therapy for cervicogenic headache

Once it has been determined that cervical factors are present in a particular headache condition and that physical therapy is indicated, a treatment plan that is highly specific to the individual patient's needs should be developed. Success of therapy depends on its comprehensiveness. One must attend to all of the biomechanical dysfunctions of the musculoskeletal system, including joint hypomobility or hypermobility, short or restricted muscles and connective tissue, and weak or inhibited muscles. Postural dysfunctions, ergonomics, and work and recreational activities must be addressed as they relate to the patient's musculoskeletal dysfunctions. For example, the therapist may work with the patient on increasing suboccipital muscle length, restoring occipitoatlantal range of motion, and strengthening the scapular and deep cervical musculature. However, if the patient then returns to a slumped sitting posture in front of a computer all day, with forward head positioning compressing the upper cervical region and rounded shoulders creating tension on the cervical muscles, any success realized in therapy likely will be temporary.

Unfortunately, the term "physical therapy" is frequently used to mean the application of modalities such as heat, electrical stimulation, generalized massage, or instruction in nonspecific exercise programs. Modalities in the absence of other interventions are useful for temporary relief of symptoms only and should be used by the patient on an as-needed basis. The occasional use of a modality to assist in patient treatment (*eg,* heat before stretching, ice after mobilization) is within reason. However, the use of modalities as the sole or primary content of ongoing physical therapy treatment lacks empirical support.

Studies that focused on specific treatment strategies generally have found a combination of manual therapy, exercise, and education to be most effective. Jull *et al.* [43••] completed a multicenter, randomized, controlled trial with unblinded treatment and blinded outcome assessment of physical therapy for patients with cervicogenic headache. Two hundred patients were included and treatment took place at five treatment centers. The treatment lasted for 6 weeks and follow-up assessment was performed at 3, 6, and 12 months. Study participants were diagnosed with cervicogenic headache according to the criteria suggested by Sjaastad *et al.* [3] and were divided into four groups. Group one received manual therapies to address cervical joint dysfunction. Group two received low-load exercises to improve muscle control in the deep cervical flexors and scapular muscles, postural retraining exercises, and stretching exercises as needed. Group three received both interventions and group four served as a nontreatment control group. All of the treatment groups showed significant improvement in terms of reduced headache frequency and intensity and the effects were maintained at 1 year, with 72% of the patients in the treatment groups having achieved pain reduction of 50% or more and 42% reporting 80% to 100% pain relief. The mixed treatment group had a 10% better chance of achieving good to excellent outcome, although the difference was not statistically significant compared with the other treatment groups. At 12 months, medication intake had decreased by 93% to 100% in the treatment groups and increased by 33% in the control group. Forty-six percent of the control group, but only 19% of the treatment group received some outside intervention for their headaches during the study period, suggesting possible underestimation of the treatment effects.

Another large randomly controlled trial compared active exercises for strength, range of motion, and posture in conjunction with modalities with a program of manual therapies and specific exercise (joint mobilizations/articular techniques, soft tissue mobilization, and stabilization and postural control work) [56]. A third control group received continuing care from a general practitioner, consisting of advice on self-care, prognosis, ergonomics, and encouragement. Treatment success was defined as "much improved" or "completely recovered." The results indicated that 68.3% of the manual therapy group, 50.8% of the exercise group, and 35.9% of the continued care group achieved a successful outcome.

Joint mobilization of occiput through C3 has been shown to be effective for reducing the frequency of cervicogenic headaches [57] and manual therapy is superior to cold packs in the treatment of post-traumatic headache [58]. Mobilization, which is defined as joint oscillations of varying amplitude without thrust, has been shown to be equally effective as high-velocity manipulation for headache reduction [59]. In light of reports of infrequent, but potentially serious risks associated with high-velocity manipulation of the cervical spine [60], mobilization may be preferred.

Patients with cervical pain after whiplash injury and those with pain of nontraumatic onset demonstrate reduced cervicocephalic kinesthesia, which means that patients have an altered proprioception and a reduced ability to reposition the head and neck to a targeted position in space [61,62]. A rehabilitation program based on eye-head coupling can be effective in improving cervicocephalic kinesthesia and can lead to reduced head pain and improved daily functioning [63].

Physical therapy for other headache conditions

When physical therapy is reported to be effective for noncervicogenic headaches, it begs the question of what mechanisms underlie positive treatment outcomes. The treatment effects may be mediated centrally, the headache may have musculoskeletal components (*eg*, the presence of myofascial pain in a patient with tension-type headache or cervicogenic triggers for migraine), or the headache may have been misdiagnosed. For example, in a study by Marcus *et al.* [64], a group of 69 patients diagnosed with migraine with or without aura received physical therapy or relaxation training/biofeedback (RTB). Only 13% reported a 50% or greater headache reduction with physical therapy; 51% had success with RTB. In the second phase of the study, those who did not achieve success with RTB and chose to continue with the study were treated with physical therapy; 47% of those patients reported good outcomes. The improvement seen in those patients treated with RTB and physical therapy may represent an increased responsiveness to physical therapy when preceded by the muscle relaxation inherent in RTB and suggests a possible musculoskeletal contribution to their head pain. Although the authors evaluated the musculoskeletal system of the patients and found positive muscle and joint signs with cervicogenic features in some, the subjects were diagnosed with migraine with aura, migraine without aura, or migraine without aura and tension-type headache.

Hammill *et al.* [65] investigated the effectiveness of physical therapy in a group of patients with tension-type headache. Treatment included posture and ergonomic education, stretching and strengthening exercises, and soft tissue massage. Headache frequency and Sickness Impact Profile scores were significantly improved at post-treatment assessment and remained so at the 12-month follow-up. When the raw data were examined, they showed that individual patients had extremely good reduction in headache or none at all, with only a few patients demonstrating moderate improvement only. This seems suggestive that the responders had headache "associated with biomechanical (or myofascial) dysfunction of the cervical spine" compared with a more centrally mediated condition in the nonresponders. The difficulty in differentiating and classifying headache types may be relevant.

Musculoskeletal factors can be pertinent when they coexist with other headache diagnoses. There are reports of treatment of myofascial and craniomandibular dysfunction reducing symptoms of cluster-like headache [66]. It is not uncommon for patients to catalogue their known migraine triggers such as flickering lights, red wine, stress, and prolonged awkward cervical positioning. Because musculoskeletal factors have been shown to be capable of triggering migraine [20], eliminating the trigger could reduce the frequency of attacks.

Temporomandibular dysfunctions represent another entire realm of musculoskeletal disorders that can produce headache and respond well to physical therapy interventions [67,68]. The principles in treating temporomandibular dysfunctions are the same as in treating cervical dysfunctions (*ie*, address faulty biomechanics, joint hypomobility or hypermobility, myofascial pain, and muscle firing imbalances).

Physical therapy also may be helpful in treating migraine-related nonpain symptoms. Physical therapy in the form of vestibular rehabilitation has been proven effective in treating migraine-related vestibulopathy, reducing severity of dizziness and the frequency of falls [69,70]. Cervical mechanoreceptor dysfunction has been implicated as a cause of dizziness in whiplash-related disorders [71] and treating musculoskeletal dysfunction of the cervical spine, even in the absence of vestibular rehabilitation, can reduce symptoms of dizziness of suspected cervical origin [72,73].

Mechanisms of treatment effects

The mechanism by which physical therapy effects positive therapeutic outcomes for patients with chronic headache has not been determined conclusively and an exhaustive analysis is beyond the scope of this review. There is controversy regarding the degree to which these effects are mediated by afferent input stimulating neural inhibitory pathways at the spinal cord and midbrain or by affecting change in peripheral musculoskeletal tissues. It is likely that both mechanisms contribute to the overall picture. It is clear that central mechanisms are accessed in physical therapy because cervical mobilization and manual therapies have been shown to produce hypoalgesic and sympathoexcitatory effects [74]. There is evidence of disinhibition of weak muscles after mobilization of the appropriate spinal segment, resulting in improved muscular control [75]. Joint oscillations used in spinal mobilization stimulate dynamic mechanoreceptors, which results in reduction in pain and muscle splinting around a painful joint [76]. These mechanisms may account for some of the immediate pain reduction patients sometimes experience after treatment sessions. However, the plastic deformation area under the stress-strain curve also is accessed, producing mechanical effects of tissue lengthening, thereby creating lasting changes in soft tissue length and range of motion. The interested reader is referred to recent discussions of possible physiologic mechanisms for pain reduction produced by physical therapy for musculoskeletal dysfunction [74,75,78].

Conclusions

Physical therapy should be a routine component of treatment for patients with musculoskeletal contribution to their head pain. Treatment needs to be individualized and highly specific, using manual therapies to restore normal joint mechanics, stretching exercises to lengthen shortened muscles and reduce TrP activity, neuromuscular re-education and strengthening to address weakness and poor motor control, and patient education regarding posture, ergonomics, and avoidance of perpetuating factors for pain. Of utmost importance is thorough and consistent education regarding the nature and behavior of musculoskeletal pain and clear identification of factors that will facilitate a favorable prognosis. The efficacy of physical therapy will differ depending on the extent to which the musculoskeletal system is involved in a particular headache; across the variety of headache diagnoses, physical therapy may be expected to remediate the problem, eliminate a trigger, or reduce the frequency or severity of symptoms. The patient, physician, and physical therapist should concur on expectations for therapy so that the patients' understanding regarding their diagnosis is maximized, encouraging positive treatment outcome and high patient satisfaction.

References and Recommended Reading

Papers of particular interest, published recently, have been highlighted as:

- • Of importance
- •• Of major importance

1. International Headache Society: **ICD-10 Guide for Headaches.** *Cephalalgia* 1997, **17(supp19):**29–30.
2. Merskey H, Bogduk N: *Classification of Chronic Pain: Descriptions of Chronic Pain Syndromes and Definition of Pain Terms,* edn 2. Seattle: IASP Press; 1994:94–95.
3. Sjaastad O, Fredriksen TA, Pfaffenrath V: **Cervicogenic headache: diagnostic criteria. The Cervicogenic Headache International Study Group.** *Headache* 1998, **38:**442–445.
4. Sjaastad O, Saunte C, Hovdahl H, *et al.*: **"Cervicogenic" headache: a hypothesis.** *Cephalagia* 1983, **3:**249–256.
5. Jull G, Barrett C, Magee R, *et al.*: **Further clinical clarification of the muscle dysfunction in cervical headache.** *Cephalalgia* 1999, **19:**179–185.
6. Fishbain D, Cutler R, Cole B, *et al.*: **International Headache Society headache diagnostic patterns in pain facility patients.** *Clin J Pain* 2001, **17:**78–93.
7. Vincent MB, Luna RA: **Cervicogenic headache: a comparison with migraine and tension-type headache.** *Cephalalgia* 1999, **19(suppl 25):**11–16.
8. Sjaastad O, Fredriksen T, Pareja J, *et al.*: **Coexistence of cervicogenic headache and migraine without aura (?).** *Funct Neurol* 1999, **14:**209–218.
9. Pfaffenrath V, Dandekar R, Pollmann W: **Cervicogenic headache: the clinical picture, radiological findings, and hypotheses on its pathophysiology.** *Headache* 1987, **27:**495–499.
10. Vanagaite Vingen J, Stovner LJ: **Photophobia and phonophobia in tension-type and cervicogenic headache.** *Cephalalgia* 1998, **18:**313–318.
11. Nilsson N: **The prevalence of cervicogenic headache in a random population sample of 20–59 year olds.** *Spine* 1995, **17:**1884–1888.
12. Pfaffenrath V, Kaube H: **Diagnostics of cervicogenic headache.** *Funct Neurol* 1990, **5:**159–164.

13. Hoyt WH, Shafer F, Bard DA, *et al.*: **Osteopathic manipulation in the treatment of muscle-contraction headache.** *J Am Osteopath Assoc* 1979, **78:**322–325.
14. Jensen R, Bendtsen L, Olesen J: **Muscular factors are of importance in tension-type headache.** *Headache* 1998, **38:**10–17.
15. Crofford LJ, Pillemar SR, Kalogeras KT, *et al.*: **Hypothalamic-pituitary-adrenal axis perturbations in patients with fibromyalgia.** *Arthritis Rheum* 1994, **37:**1583–1592.
16.•• Simons D, Travell J, Simons L: *Myofascial Pain and Dysfunction: The Trigger Point Manual*, vol 1, edn 2. Baltimore: Williams & Wilkins; 1999.

This is the definitive text on myofascial pain. It includes an extensive review of the state of the science of the pathophysiology of myofascial pain and comprehensive clinical information including referral patterns.

17. Gerwin RD, Shannon S, Hong CZ, *et al.*: **Interrater reliability in myofascial trigger point examination.** *Pain* 1997, **69:**65–73.
18. Graff-Radford SB, Reeves JL, Jaeger B: **Management of chronic head and neck pain: effectiveness of altering factors perpetuating myofascial pain.** *Headache* 1987, **27:**186–190.
19. Jaeger B: **Are "cervicogenic" headaches due to myofascial pain and cervical spine dysfunction?** *Cephalalgia* 1989, **9:**157–164.
20. Davidoff RA: **Trigger points and myofascial pain: toward understanding how they affect headaches.** *Cephalalgia* 1998, **18:**436–448.
21. Kidd RF, Nelson R: **Musculoskeletal dysfunction of the neck in migraine and tension headache.** *Headache* 1993, **33:**566–569.
22. Lous I, Olesen J: **Evaluation of pericranial tenderness and oral function in patients with common migraine, muscle contraction headache and "combination headache."** *Pain* 1982, **12:**385–393.
23. Tfelt-Hansen P, Lous I, Olesen J: **Prevalence and significance of muscle tenderness during common migraine attacks.** *Headache* 1981, **21:**49–54.
24. Treleaven J, Jull G, Atkinson L: **Cervical musculoskeletal dysfunction in post-concussional headache.** *Cephalalgia* 1994, **14:**273–279.
25. Jull G, Bogduk N, Marsland A: **The accuracy of manual diagnosis for cervical zygapophyseal joint pain syndromes.** *Med J Aust* 1988, **148:**233–236.
26. Jull G, Zito G, Trott P, *et al.*: **Interexaminer reliability to detect painful upper cervical joint dysfunction.** *Aust J Physiother* 1997, **43:**125–129.
27. Zwart JA: **Neck mobility in different headache disorders.** *Headache* 1997, **37:**6–11.
28. Dall'Alba PT, Sterling MM, Treleaven JM, *et al.*: **Cervical range of motion discriminates between asymptomatic persons and those with whiplash.** *Spine* 2001, **26:**2090–2094.
29. Dumas JP, Arsenault AB, Boudreau G, *et al.*: **Physical impairments in cervicogenic headache: traumatic vs nontraumatic onset.** *Cephalalgia* 2001, **21:**884–893.
30. Sandmark H, Nisell R: **Validity of five common manual neck pain provoking tests.** *Scand J Rehab Med* 1995, **27:**131–136.
31. Watson DH, Trott PH: **Cervical headache: an investigation of natural head posture and upper cervical flexor muscle performance.** *Cephalagia* 1993, **13:**272–284.
32. Griegel-Morris P, Larson K, Mueller-Klaus K, *et al.*: **Incidence of common postural abnormalities in the cervical, shoulder, and thoracic regions and their association with pain in two age groups of healthy subjects.** *Phys Ther* 1992, **72:**425–431.
33. Nagasawa A, Sakakibara T, Takahashi A: **Roentgenographic findings of the cervical spine in tension-type headache.** *Headache* 1993, **33:**90–95.
34. Fountain FP, Minear WL, Allison RD: **Function of longus colli and longissimus cervicis muscles in man.** *Arch Phys Med Rehabil* 1966, **47:**665–669.
35. Vitti M, Fujiwara M, Basmajian JV, *et al.*: **The integrated roles of longus colli and sternocleidomastoid muscles: an electromyographic study.** *Anat Rec* 1973, **177:**471–484.
36. Mayoux-Benhamou MA, Revel M, Vallee C, *et al.*: **Longus colli has a postural function on cervical curvature.** *Surg Radiol Anat* 1994, **16:**367–371.
37. Jull GA: **Deep cervical flexor muscle dysfunction in whiplash.** *J Musculoskel Pain* 2000, **8:**143–154.
38. Silverman JL, Rodriquez AA, Agre JC: **Quantitative cervical flexor strength in healthy subjects and in subjects with mechanical neck pain.** *Arch Phys Med Rehabil* 1991, **72:**679–681.
39. Sterling M, Jull G, Wright A: **The effect of musculoskeletal pain on motor activity and control.** *J Pain* 2001, **2:**135–145.
40. Lund JP, Donga R, Widmer CG, *et al.*: **The pain-adaptation model: a discussion of the relationship between chronic musculoskeletal pain and motor activity.** *Can J Physiol Pharmacol* 1991, **69:**683–694.
41. Nederhand MJ, Jzerman MJ, Hermens HJ, *et al.*: **Cervical muscle dysfunction in the chronic whiplash associated disorder grade II (WAD-II).** *Spine* 2000, **25:**1938–1943.
42. Richardson CA, Jull G: **Muscle control: pain control. What exercises would you prescribe?** *Man Ther* 1995, **1:**1–9.
43.•• Jull G, Trott P, Potter H, *et al.*: **A randomized controlled trial of exercise and manipulative therapy for cervicogenic headache.** *Spine* 2002, **27:**1835–1843.

This is a large, well-designed, and carefully controlled study that demonstrates the efficacy of manual therapies and specific low-load exercises in the long-term management of cervicogenic headaches.

44. Zohn DA, Mennell JM: *Musculoskeletal Pain: Diagnosis and Physical Treatment.* Boston: Little, Brown; 1976:126–129, 190–193.
45. Esenyel M, Caglar N, Aldemir T: **Treatment of myofascial pain.** *Am J Phys Med Rehabil* 2000, **79:**48–52.
46. Roberts JM, Wilson K: **Effect of stretching duration on active and passive range of motion in the lower extremity.** *Br J Sports Med* 1999, **33:**259–263.
47. Lewit K: **Postisometric relaxation in combination with other methods of muscular facilitation and inhibition.** *Man Med* 1986, **2:**101–104.
48. Hou CR, Tsai LC, Chen KF, *et al.*: **Immediate effects of various physical therapeutic modalities on cervical myofascial pain and trigger-point sensitivity.** *Arch Phys Med Rehabil* 2002, **83:**1406–1414.
49. Hanten WP, Olson SL, Butts NL, *et al.*: **Effectiveness of a home program of ischemic pressure followed by sustained stretch for treatment of myofascial trigger points.** *Phys Ther* 2000, **80:**997–1003.
50. Hong CZ, Hsueh TC: **Difference in pain relief after trigger point injections in myofascial pain patients with and without fibromyalgia.** *Arch Phys Med Rehabil* 1996, **77:**1161–1166.
51. Raphael KG, Marbach JJ: **Widespread pain and the effectiveness of oral splints in myofascial face pain.** *J Am Dent Assoc* 2001, **132:**305–316.
52. Aaras A, Horgen G, Bjorset HH, *et al.*: **Musculoskeletal, visual and psychosocial stress in VDU operators before and after multidisciplinary ergonomic interventions.** *Appl Ergon* 1998, **29:**335–354.
53. Mclean L, Tingley M, Scott RN, *et al.*: **Computer terminal work and the benefit of microbreaks.** *Appl Ergon* 2001, **32:**225–237.
54. Roth RS, Horowitz K, Bachman JE: **Chronic myofascial pain: knowledge of diagnosis and satisfaction with treatment.** *Arch Phys Med Rehab* 1998, **79:**966–970.
55. Geisser ME, Roth RS: **Knowledge of and agreement with chronic pain diagnosis: relation to affective distress, pain beliefs and coping, pain intensity and disability.** *J Occup Rehab* 1998, **8:**73–88.
56. Hoving JL, Koes BW, de Vet HCW, *et al.*: **Manual therapy, physical therapy, or continued care by a general practitioner for patients with neck pain: a randomized, controlled trial.** *Ann Intern Med* 2002, **136:**713–722.
57. Schoensee SK, Jensen G, Nicholson G, *et al.*: **The effect of mobilization on cervical headaches.** *J Orthop Sports Phys Ther* 1995, **21:**184–196.

58. Jensen OK, Nielsen FF, Vosmar L: **An open study comparing manual therapy with the use of cold packs in the treatment of post-traumatic headache.** *Cephalalgia* 1990, **10:**241–250.
59. Parker GB, Tupling H, Pryor DS: **A controlled trial of cervical manipulation for migraine.** *Aust N Z J Med* 1978, **8:**589–593.
60. Rothwell DM, Bandy SJ, Williams JC: **Chiropractic manipulation and stroke: a population-based case-control study.** *Stroke* 2001, **32:**1054–1060.
61. Heikkila HV, Wenngren BI: **Cervicocephalic kinesthetic sensibility, active range of cervical motion, and oculomotor function in patients with whiplash injury.** *Arch Phys Med Rehabil* 1998, **79:**1089–1094.
62. Revel M, Andre-Deshays C, Minguet M: **Cervicocephalic kinesthetic sensibility in patients with cervical pain.** *Arch Phys Med Rehabil* 1991, **72:**288–291.
63. Revel M, Minguet M, Gergoy P, *et al.*: **Changes in cervicocephalic kinesthesia after a proprioceptive rehabilitation program in patients with neck pain: a randomized controlled study.** *Arch Phys Med Rehabil* 1994, **75:**895–899.
64. Marcus DA, Scharff L, Mercer S, *et al.*: **Nonpharmacological treatment for migraine: incremental utility of physical therapy with relaxation and thermal biofeedback.** *Cephalalgia* 1998, **18:**266–272.
65. Hammill JM, Cook TM, Rosecrance JC: **Effectiveness of a physical therapy regimen in the treatment of tension-type headache.** *Headache* 1996, **36:**149–153.
66. Vargo CP, Hickman DM: **Cluster-like signs and symptoms respond to myofascial/craniomandibular treatment: a report of two cases.** *Cranio* 1997, **15:**89–93.
67. Nicolakis P, Erdogmus B, Kopf A, *et al.*: **Effectiveness of exercise therapy in patients with myofascial pain dysfunction syndrome.** *J Oral Rehabil* 2002, **29:**362–368.
68. Suvinen TI, Hanes KR, Reade PC: **Outcome of therapy in the conservative management of temporomandibular pain dysfunction disorder.** *J Oral Rehabil* 1997, **24:**718–724.
69. Whitney SL, Wrisley DM, Brown KE, *et al.*: **Physical therapy for migraine-related vestibulopathy and vestibular dysfunction with history of migraine.** *Laryngoscope* 2000, **110:**1528–1534.
70. Wrisley DM, Whitney SL, Furman JM: **Vestibular rehabilitation outcomes in patients with a history of migraine.** *Otol Neurotol* 2002, **23:**483–487.
71. Treleaven J, Jull G, Sterling M: **Dizziness and unsteadiness following whiplash injury: characteristic features and relationship with cervical joint position error.** *J Rehabil Med* 2003, **35:**36–43.
72. Karlberg M, Magnusson M, Malmstrom EM, *et al.*: **Postural and symptomatic improvement after physiotherapy in patients with dizziness of suspected cervical origin.** *Arch Phys Med Rehabil* 1996, **77:**874–882.
73. Heikkila H, Johansson M, Wenngren BI: **Effects of acupuncture, cervical manipulation, and NSAID therapy on dizziness and impaired head repositioning of suspected cervical origin: a pilot study.** *Man Ther* 2000, **5:**151–157.
74. Sterling M, Jull G, Wright A: **Cervical mobilization: concurrent effects on pain, sympathetic nervous system activity, and motor activity.** *Man Ther* 2001, **6:**72–81.
75. Wilson E: **Central facilitation and remote effects: treating both ends of the system.** *Man Ther* 1997, **2:**165–168.
76. Greenman PE: **Manipulation and mobilization.** In *Headache: Diagnosis and Treatment.* Edited by Tollison CD, Kunkel RJ. Philadelphia: Lippincott Williams & Wilkins; 1993:347–355.
77. Paris SV: **Mobilization of the spine.** *Phys Ther* 1979, **59:**988–995.
78. Wright A: **Hypoalgesia post-manipulative therapy: a review of a potential neurophysiological mechanism.** *Man Ther* 1995, **1:**11–16.