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ScienceDirect
Manual Therapy xx (2008) 1–10
MANUAL THERAPY
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Masterclass

From acute musculoskeletal pain to chronic widespread pain and fibromyalgia: Application of pain neurophysiology in manual therapy practice

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Received 4 December 2007; accepted 9 March 2008

Abstract

During the past decade, scientific research has provided new insight into the development from an acute, localised musculoskeletal disorder towards chronic widespread pain/fibromyalgia (FM). Chronic widespread pain/FM is characterised by sensitisation of central pain pathways. An in-depth review of basic and clinical research was performed to design a theoretical framework for manual therapy in these patients. It is explained that manual therapy might be able to influence the process of chronicity in three different ways. (I) In order to prevent chronicity in (sub)acute musculoskeletal disorders, it seems crucial to limit the time course of afferent stimulation of peripheral nociceptors. (II) In the case of chronic widespread pain and established sensitisation of central pain pathways, relatively minor injuries/trauma at any locations are likely to sustain the process of central sensitisation and should be treated appropriately with manual therapy accounting for the decreased sensory threshold. Inappropriate pain beliefs should be addressed and exercise interventions should account for the process of central sensitisation. (III) However, manual therapists ignoring the processes involved in the development and maintenance of chronic widespread pain/FM may cause more harm then benefit to the patient by triggering or sustaining central sensitisation.

Keywords: Fibromyalgia; Central sensitisation; Whiplash; Chronic fatigue syndrome

1. Introduction

The 1990 American College of Rheumatology criteria for the classification of fibromyalgia (FM) define chronic widespread pain as a history of at least 3 months of axial skeletal pain, pain in the right and left sides of the body, and pain above and below the waist (Wolfe et al., 1990). In addition to the presence of chronic widespread pain, pain in 11 of 18 tender point sites must be present on digital palpation with an approximate force of 4 kg (Wolfe et al., 1990). FM is classified as a rheumatic illness and is often treated by manual therapists. In the United States, patients with FM are frequently seen in chiropractic practice. Studying the health-care use of 402 patients from a university-based clinic, it was found that nearly 56% of the patients fulfilling the diagnostic criteria for both FM and the related chronic fatigue syndrome visited chiropractors, and 32% of the primary FM subjects consulted chiropractors (Bombardier and Buchwald, 1996). Trigger point

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doi:10.1016/j.math.2008.03.001

Please cite this article in press as: Nijs J, Van Houdenhove B, From acute musculoskeletal pain to chronic widespread pain and fibromyalgia: Application of pain neurophysiology in manual therapy practice, Manual Therapy (2008), doi:10.1016/j.math.2008.03.001

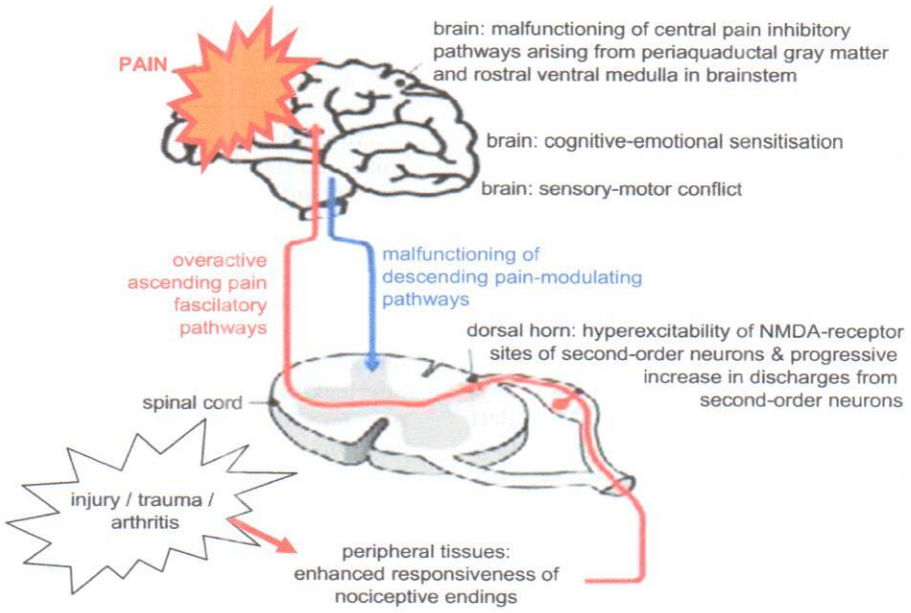

Fig. 1. Anatomical localisations of hyperexcitability of peripheral and central pain pathways.

(Staud et al., 2001; Price et al., 2002; Banic et al., 2004). Malfunctioning of central pain-inhibitory pathways in people with chronic pain becomes particularly apparent to clinicians during exercise interventions: both isometric and aerobic exercises activate endogenous opoid and adrenergic pain-inhibitory mechanisms in healthy subjects, while it increases experimental pain ratings in patients with FM (Staud et al., 2005) and chronic fatigue syndrome (Whiteside et al., 2004).

So how do we account for the process of central sensitisation during manual treatment of those with musculoskeletal pain? This will be discussed in the following sections.

3. Manual therapy to prevent hypersensitivity of pain pathways in (sub)acute musculoskeletal pain

An important and ongoing source of pain is required before the process of peripheral sensitisation can establish central sensitisation. It seems crucial to limit the time course of afferent stimulation of peripheral nociceptors: tissue injury healing and focal pain recovery should occur within a period of approximately 3 months to prevent development of chronic widespread pain/FM (Vierck, 2006). Progression towards chronic widespread pain is associated with injuries to deep tissues which do not heal within several months (Vierck, 2006). Consequently, appropriate and effective manual therapy in those with (sub)acute musculoskeletal disorders is important to prevent evolvement from an acute, localised musculoskeletal pain problem to complex clinical cases, characterised by chronic widespread pain and even symptoms outside the musculoskeletal system such as increased sensitivity to bright lights, auditory loudness, odours, and other sensory stimuli.

Pain due to damage or inflammation of peripheral tissues is clearly capable of causing chronic widespread pain/FM (Clauw, 2007). 15–20% people with whiplash injuries develop chronic pain and disability (Spitzer et al., 1995; Radanov and Sturzenegger, 1996; Côté et al., 2001). Regardless of whether FM is present in chronic whiplash, altered central pain processing and central sensitisation is evident (Curatolo et al., 2001; Sterling et al., 2002, 2003, 2006; Banic et al., 2004). Moreover, altered central pain processing rather than impaired motor control has been identified as one of the prime prognostic factors for developing chronic whiplash (Sterling et al., 2003, 2006).

Another example of a local musculoskeletal disorder associated with FM and frequently seen in manual therapy practice is arthritis (rheumatoid arthritis and osteoarthritis), possibly causing continuous activation of local nociceptors that initiate or sustain central sensitisation (Yunus, 2007). Thus, effective manual therapy in (sub)acute cases of arthritis should be able to limit the (time course of) afferent barrage of noxious input to the central nervous system and thus prevent chronicity.

In addition, manual therapy aimed at improving motor control in symptomatic regions/joints is likely to have its place in the prevention of chronicity. Indeed, a sustained mismatch between motor activity and

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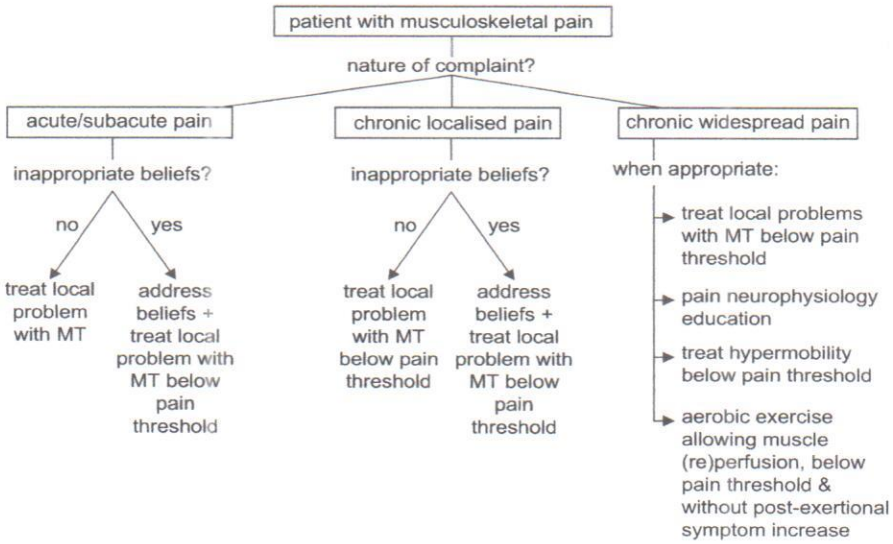

Fig. 2. Proposed treatment options accounting for central hyperexcitability of central pain pathways. MT, manual therapy.

pain exaggeration. However, there is currently no evidence in support of an association between pain complaints and generalised joint hypermobility in those with FM (Nijs, 2005). In the absence of such evidence, it is recommended to search for such an association on an individual basis. If the manual therapist suspects a role for joint hypermobility in a particular FM case, then a treatment consisting of end-range stabilisation exercises, postural advice, movement advice, self-management strategies and the application of protective and supportive devices is recommended (Nijs, 2005).

5. Changing inappropriate beliefs in an attempt to desensitise the central nervous system

Although the dysfunctional descending pain-inhibitory mechanism as seen in those with chronic widespread pain/FM is primary biological, it is influenced by inappropriate cognitions, emotions and behaviour like catastrophizing, hypervigilance, avoidance behaviour, and somatisation. In case of more intense pain levels, pain catastrophizing is associated with decreased activity in brain regions involved in top-down pain suppression like the dorsolateral prefrontal cortex and the medial prefrontal cortex (Seminowicz and Davies, 2006). In addition to catastrophizing, avoidance behaviour and somatisation may result in sensitisation of dorsal horn spinal cord neurons (through inhibition of descending tracts in the central nervous system – Fig. 1), or alternatively, may be the result of central sensitisation (Zusman, 2002). Sustained arousal is likely to maintain sensitisation of the neurobiological loops (Ursin and Eriksen, 2001). It is important for clinicians to recognise that pain cognitions such as fear of movement and catastrophizing are not only of importance to chronic pain patients, but may even be crucial at the stage of acute/subacute musculoskeletal disorders (Swinkels-Meewisse et al., 2006).

So how do you screen for this in daily practice? Screening patients for maladaptive beliefs and subsequently changing them should not be limited to multidisciplinary settings applying cognitive behavioural therapies. Instead, all practitioners can use easy-administered questionnaires (e.g. the pain vigilance and awareness questionnaire, the pain catastrophizing scale) to screen their patients with (sub)acute/chronic musculoskeletal pain for maladaptive beliefs. In the case of hypervigilance, catastrophic beliefs about pain (i.e. helplessness, rumination and magnification) or passive coping strategies like avoidance behaviour, intensive education about the exact nature of chronic widespread pain is likely to clear the path for effective manual physiotherapy (including exercise interventions). Pain neurophysiology education aims at reconceptualising pain, and was found to be effective in reducing pain catastrophising in those with chronic low back pain (Moseley et al., 2004) and chronic widespread pain (Meeus et al., submitted for publication). In

Table 1
Practical guidelines for hands-on manual therapy skills in those with hypersensitive pain pathways

-Educate patient to report adverse reactions during treatment
-Do not elicit identical nociceptive stimuli > once every 3 s
-Adopt techniques to reduced sensory threshold
-Do not increase nociceptive barrage
-Initiate soft-tissue mobilisation with superficial stripping techniques
-Progress soft-tissue mobilisation with deeper cross-fibre techniques
-Careful with ischaemic compression

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the development of chronic widespread pain in a group of psychologically 'at risk' subjects (McBeth et al., 2007). In line with the patients' history, this abnormal functioning of the stress system seems to occur mostly in the aftermath of a long period of overburdening by physical and/or emotional stressors and to be precipitated by an additional trigger in the form of an acute physical or emotional event (McLean et al., 2005). In those with chronic widespread pain/FM, the inability of the central nervous system to activate the descending pain-inhibitory pathways is likely to be related to the stress system (i.e. the initial stress response to the collision during whiplash trauma and via behavioural changes that occur in response to the trauma) (McLean et al., 2005). The stress system is capable of influencing pain processing via dorsal horn glucocorticoid receptors (receptors having pain-inhibitory capacity) (McLean et al., 2005). However, many unsolved questions remain about the precise role of stress and stress system disturbance in FM (Cleare, 2004).

8. Exercise therapy accounting for central sensitisation

There is evidence to support specific exercise therapies as a cornerstone in the comprehensive management of FM (McCain et al., 1988; Isomeri et al., 1993; Burckhardt et al., 1994; Martin et al., 1996; Buckelew et al., 1998; Mannerkorpi et al., 2000; Nijs and Van Parijs, 2004). The evidence from randomised clinical trials is underscored by the conclusions of a systematic literature review (Karjalainen et al., 2000). In relation to the use of exercise therapy, various treatment goals of potential relevance to those with chronic widespread pain and FM can be identified (Table 2) including effort tolerance rather than low effort capacity (Van Houdenhove et al., 2007).

However, clinicians should be cautious not to sustain or even amplify the process of central sensitisation. As outlined above, isometric and aerobic exercises activate endogenous opoid and adrenergic pain-inhibitory mechanisms in healthy subjects, while it increases experimental pain ratings in patients with FM (Staud et al., 2005). Thus, people with FM are increasingly susceptible to activation of nociceptors during exercise. As is the case with hands-on manual therapy skills, exercise interventions that are too vigorous are likely to activate muscle and joint nociceptors and thus cause afferent painful barrage. Altered central pain processing is further augmented by isometric exercise (Staud et al., 2005). Post-exertional complaints should be prevented and closely monitored, if not aversive consequences of exercise therapy can arise and may be a deterrent to compliance with the intervention (Dupree Jones et al., 2006; Vierck, 2006).

In those with FM, post-exertional complaints are more pronounced than the well documented delayed onset muscle soreness experienced by healthy deconditioned persons without FM who engage in unfamiliar muscle activity. Post-exertional complaints are typically seen in exercise programs using higher intensities, higher impact movements and those where subjects cannot self-adjust exercise intensity (Dupree Jones et al., 2006). Exercises that are too vigorous might trigger immune activation with release of pro-inflammatory cytokines provoking a so-called 'sickness response' (Maier and Watkins, 1998), possibly explaining a variety of post-exertional complaints. Therefore, low to moderate intensity exercise (approximately 50% of maximum heart rate) of any types has lower attrition and better symptom improvement than those with the higher intensity (Dupree Jones et al., 2006).

Further support in favour of mild to moderate exercise over vigorous exercise interventions comes from the study showing a blunted increase in muscular vascularity in response to both dynamic and static contractions (Elvin et al., 2006). This can result in diminishing blood flow towards the working muscles both during and following exercises (Elvin et al., 2006). These data are in line with other observations pointing to widespread muscular ischaemia in patients with FM (reviewed in Vierck, 2006). Since muscle nociceptors are highly sensitive to ischaemia, exercise interventions should account for the widespread muscular ischaemia and blunted increase in muscular vascularity in response to muscle contractions. If not, exercise is likely to increase afferent painful barrage and thus sustain or accelerate the process of central sensitisation. Apart from using mild to moderate exercise intensity, aerobic exercises using multiple recovery periods (to allow muscular reperfusion) within training sessions might be beneficial (Fig. 2). If available, hydrotherapy in warm water, known to be beneficial to those with FM (Mannerkorpi et al., 2000), might even be able to account for the decreased muscle perfusion during exercise (Vierck, 2006).

9. Conclusion

Chronic widespread pain/FM is characterised by sensitisation of central pain pathways. An important and ongoing source of pain is required before the process of peripheral sensitisation can establish central sensitisation. Ongoing nociceptive barrage results in adaptation of dorsal horn neurons, and the process of central sensitisation encompasses altered sensory processing in the brain and malfunctioning of pain-inhibitory mechanisms as well. There is evidence that a stress response system dysfunction may play a role in central sensitisation. Moreover, inappropriate cognitions, emotions and behaviour may have a negative impact on the descending pain-inhibitory mechanisms. In order to

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