# Cervical afferents in migraine: orchestrating central sensitisation or innocent bystanders?

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This article aims to augment the comprehension of migraine through an integrative approach, combining insights into upper cervical musculoskeletal dysfunction, contemporary principles of neuroscience, and my own extensive clinical experience. This multidisciplinary perspective is designed to provide a more holistic understanding of the complexities underlying migraine, bridging the gap between musculoskeletal pathology, neuroscientific advancements, and practical clinical observations. I will present my perspective on migraine, which entails a critical examination and potential challenge to some of the established paradigms based on the medical model of headache, as delineated in the International Classification of Headache Disorders -3 (ICHD-3) (International Headache Society 2018).

**LEARNING OUTCOMES**
TO SUPPORT PHYSIO FIRST QAP

1 Macroscopic, discernible lesions are not necessary as evidence for cervicogenic headache or cervical afferent involvement in migraine.
2 Manual cervical reproduction of typical head pain does not confirm cervical relevancy in cervicogenic headache and migraine.
3 Manual cervical reproduction and lessening of typical head pain confirms peripherally (cervical) mediated central sensitisation in migraine.

## Introduction

My extensive clinical background informs my perspective. I am an Australian physiotherapist who has focused exclusively on treating and managing headache and migraine conditions since 1991. My clinical experience encompasses more than 40,000 hours, during which I have treated more than 13,000 patients with a variety of medically diagnosed headache and migraine diagnoses. This extensive, focused clinical experience underpins my insights and contributes to understanding migraine from a manual, cervical perspective.

Consequently, my perspective has undergone a paradigm shift regarding the etiological role of upper cervical afferents in migraine pathogenesis. Initially, I was taught and subscribed to the view that these afferents were non-contributory in migraine causation. However, through extensive clinical observation and research, my position has now become opposed, recognising a significant causative role for upper cervical afferents in the migraine process.

The potential role of cervical afferents in migraine is contingent upon the presence of relevant upper cervical musculoskeletal issues in head pain. Therefore, this discussion will involve critiquing some diagnostic criteria for cervicogenic headache (CGH), the requirement for evidence of macroscopic lesions, the absolute reliance on an appropriate response to anaesthetic blocks, and the reproduction of accustomed head pain.

This discussion also elucidates the role of noxious upper cervical afferents, exploring their potential sensitising influence on the trigemino cervical complex (TCC), thereby shedding light on a crucial aspect of migraine pathophysiology.

## Is demonstrable upper cervical pathology necessary for cervical relevancy?

According to the ICHD-3, a significant requirement for CGH is demonstrable pathology, a macroscopic, discernible lesion recognised as a source of head pain (International Headache Society 2018).

Cervicogenic headache is classified as a "secondary headache" (International Headache Society 2018), i.e. secondary to a cervical lesion or pathology. Consequently, an ICHD-3 diagnosis of CGH is contingent on the identification of demonstrable cervical pathology. Therefore, in the absence of such, discussion on the question of whether it is necessary for cervical relevance is futile. Furthermore, the lack of direct evidence of cervical pathology has

been the subject of significant debate (Edmeads 1988); whilst, in theory, the pathophysiology of CGH is recognised, there are no bona fide, accepted lesions initiating the physiological process.

The role of cervical pathology in migraine suffers the same fate. The absence of demonstrable cervical lesions in migraine (Goadsby & Bartsch 2008; Bartsch & Goadsby 2003; Lambert 2010) has led to the prevailing assumption that cervical dysfunction is not involved in migraine pathophysiology (Goadsby & Bartsch 2008).

However, the lack of identifiable cervical tissue damage does not rule out the presence of relevant pathology in head pain (Curatolo *et al* 2011). This is in contradistinction to the ICHD-3 perspective (International Headache Society 2018) and requires consideration.

As discussed, the previously described clinical pattern hypothesised stresses the top three intervertebral segments, including the facet and zygapophyseal joints (z-joints). As in humans, histological studies utilising animal models have identified nociceptive nerve fibres in the capsular ligament of z-joints, indicating that mechanical hyperalgesia or allodynia, common indicators of pain in animal studies, can result from non-injurious loading of these joints (Inami *et al* 2001; Ohtori *et al* 2001; Yamashita *et al* 1990). This is supported by evidence showing that mechanical hyperalgesia develops from controlled z-joint capsule loading (Lee *et al* 2004a, 2004b; Lee & Winkelstein 2009; Quinn & Winkelstein 2007; Lee *et al* 2008; Winkelstein & Santos 2008; Dong & Winkelstein 2010; Quinn *et al* 2010; Dong *et al* 2013; Kras *et al* 2013, 2014; Crosby *et al* 2014) and its absence when the capsule is removed (Winkelstein & Santos 2008). Additionally, intra-articular ketorolac injections reduce z-joint pain, likely by decreasing protease-activated receptor -1, involved in pain maintenance (Dong *et al* 2013).

Substance P is a nociceptive mediator that enhances joint pain signalling with increased expression after capsular loading and persisting inflammation markers (Lee *et al* 2004a; Yamashita & Cavanaugh 1993; Ebersberger *et al* 1999; Ferreira *et al* 1988; Fiorentino *et al* 2008; O'Byrne *et al* 1990; Quinn & Bazan 1990). Spinal dorsal horn neuronal activity increase after capsular loading indicates central sensitisation (CS) and chronic pain development (Quinn *et al* 2010). Similarly, intra-articular injections of saline trigger nociceptor activation, with effects mitigated by bupivacaine within four hours. This suggests that early spinal sensitisation is driven by glutamatergic signalling (Crosby *et al* 2014).

Notably, mechanical hyperalgesia occurs with relatively small joint loads (Lee *et al* 2004a, 2004b; Lee & Winkelstein 2009; Kras *et al* 2013, 2014; Crosby *et al* 2014), suggesting pain initiation before reaching tensile strain thresholds. This is supported by evidence of capsular fibre realignment and ligament yield at low distraction levels, implying a relationship between these factors and pain (Lee *et al* 2004b; Quinn *et al* 2010; Quinn 2009). These findings in animal models highlight the importance of z-joints in human pain processes, particularly in contributing to CS in headache. It suggests that z-joint compromise can occur without macroscopic lesions, with significant implications for understanding neck pain and headache etiology (Mogil 2009; Graven-Nielsen & Mense 2001; Arendt-Nielsen & Graven-Nielsen 2011).

Furthermore, and similarly, animal model studies have shown that stimulating deep cervical paraspinal muscles with algesic chemicals leads to trigeminal field effects, such as prolonged ipsilateral jaw muscle activity (Hu *et al* 1993, 1996) and changes in the jaw opening reflex (Makowska *et al* 2005); an essential critical trigeminal brainstem sensorimotor processing model (Makowska *et al* 2006). Brainstem neuronal activity recordings during cervical interventions support these findings. One study observed long-term brainstem neuronal excitability increases following a single intramuscular low-dose adenosine 5'-triphosphate (ATP) injection into neck muscles (Makowska *et al* 2006), a common experimental muscle nociception inducer (Ellrich & Makowska 2007; Reitz *et al* 2009). Similar results were seen with mustard oil injections into deep paraspinal structures near the C1-C2 spinal segment, causing 70% of neurons to show heightened excitability (Vernon *et al* 2009). This research also noted post-injection orofacial and cervical neuronal receptive field expansion, underlining the role of (imperceptible) noxious afferents from cervical musculature in TCC sensitisation.

These observations provide essential scientific evidence that noxious afferents from non-injurious cervical z-joint and cervical musculature can result in headache-related neurophysiological changes in the dorsal horn. Moreover, the results challenge the biomechanical assumption that symptom severity should correlate with the extent of soft tissue load. The absence of macroscopic tissue damage does not preclude the existence of significant, relevant cervical pathology (Elliott *et al* 2015; Bogduk 2011).

## Manual cervical reproduction of typical head pain: a key diagnostic criterion of cervical relevancy

Temporary reproduction of usual head pain when examining structures of the cervical spine is considered to be one of the key diagnostic criteria for CGH (International Headache Society 2018; Sjaastad *et al* 1998; Antonaci & Inan 2021), but this might also be important in other forms of headache. For example, reproduction of usual head pain occurred, when examining passive accessory intervertebral movements (PAIVMs) of the atlanto occipital (C0 – C1), and C2 – C3 spinal segments, in 95% of participants (Watson & Drummond 2012) who fulfilled the diagnostic criteria of migraine (International Headache Society 2018). All had alternating unilateral head pain, which excludes a diagnosis of CGH (International Headache Society 2018; Sjaastad *et al* 1998; Antonaci & Inan 2021).

The extremely high incidence of reproduction of headache in migraineurs could suggest an underlying cervicogenic basis for CS of nociceptive second-order neurons in the TCC, with subsequent hyperexcitability to afferent stimulation (McMahon *et al* 2000). The notion of CS considers an increased barrage of afferent noxious information, from C-fibers on to second-order neurons, as crucial in the development of this hyperexcitability (Woolf & Salter 2000; Woolf 2011).

This finding poses a challenging question: does this validate the involvement of cervical afferents in migraine or suggest that this phenomenon is not exclusive to CGH, thereby questioning the current diagnostic criteria used to establish cervical relevance? This quandary arises amidst prevailing uncertainties ab out the mechanisms of CS in migraine and the existing assumption that cervical afferents do not play a role in its pathophysiology (Goadsby & Bartsch 2008; Fernandes-de-las-Penas *et al* 2023).

Based on the authors' clinical observations, it is suggested that whilst the temporary reproduction of familiar head pain may imply cervical relevancy, it does not conclusively establish it (Fernandez-de-las-Penas *et al* 2003). For example, sensitivity to cervical afferents could arise from pre-existing sensitisation of the TCC, potentially due to trigeminal nociceptive activity (Burstein *et al* 1998; Goadsby & Hoskin 1999; Goadsby & Knight 1997; Kaube *et al* 1993; Bartsch & Goadsby 2003; Spekker *et al* 2021), or reduced supraspinal inhibition (Woolf & Salter 2000; Ossipov *et al* 2010; Arendt-Nielsen *et al* 2018), leading to head pain. Therefore, relying solely on symptom reproduction for diagnosis is unreliable and should be reconsidered as a key diagnostic criterion for cervical involvement.

## Central sensitisation in migraine

Earlier discussion in this article not only highlights that discernible lesions are not necessary to substantiate the presence and relevancy of noxious cervical afferents, but also accentuated their role in sensitisation of the TCC.

Research has consistently shown that the TCC is sensitised in migraine, leading to increased responsiveness, i.e. CS, of nociceptive neurons in the central nervous system (Sandrini *et al* 2002; Akerman & Romero-Reyes 2013; Suzuki *et al* 2022). Peripheral nociceptor inputs are a common trigger of CS capable of activating prolonged, but reversible, increase in the excitability and synaptic efficacy of neurons in central nociceptive pathways (Woolf 2011), profoundly increasing or amplifying the sensitivity of the somatosensory system (Woolf 1983).

Whilst increased peripheral somatic and vascular nociceptive activity can lead to hyperexcitability in second-order neurons within the TCC (Woolf 2011; Roch *et al* 2007; Goadsby 2005; Olesen *et al* 2009), CS may also arise from a reduction in supraspinal, i.e. conditioned pain modulation (CPM) inhibition (Woolf 2011; Ossipov *et al* 2010).

Central sensitisation in migraine can, therefore, be driven "peripherally", i.e. due to noxious trigeminal or cervical afferents (Woolf 2011; Arendt-Nielsen *et al* 2018; Sandrini *et al* 2006), or "centrally", i.e. due to changes in synaptic efficacy within the central nervous system (Woolf 2011; Arendt-Nielsen *et al* 2018).

> "RELYING SOLELY ON SYMPTOM REPRODUCTION FOR DIAGNOSIS IS UNRELIABLE AND SHOULD BE RECONSIDERED AS A CRITERION FOR CERVICAL INVOLVEMENT"

Studies examining centrally driven CS in migraine present mixed findings. Some research suggests that endogenous descending inhibitory and facilitatory mechanisms are dysregulated in migraine patients (Noseda & Burstein 2013; Sandrini *et al* 2006), whilst other studies indicate these mechanisms function normally (Coppola *et al* 2007; Serrao *et al* 2005; Teepker *et al* 2014). This dichotomy in research findings underscores the complexity of understanding CS in the context of migraine.

However, research focusing on peripheral nociceptive afferents is unambiguous. Activation of trigeminal nerve-innervated structures leads to the stimulation of neurons in the TCC (Burstein *et al* 1998; Goadsby & Hoskin 1999; Goadsby & Knight 1997; Kaube *et al* 1993; Bartsch & Goadsby 2003; Spekker *et al* 2021) indicative of peripherally driven CS. Likewise, discussing the issue of discernible cervical lesions reveals compelling evidence that the stimulation of cervical afferents also activates TCC neurons. Furthermore, stimulation of the greater occipital nerve (GON) also activates TCC neurons (Goadsby *et al* 1997; Le Doare *et al* 2006), reinforcing the hypothesis that CS in migraine may be primarily driven by cervical afferents (Olesen *et al* 2009).

Additional evidence for cervical afferent activation in migraine comes from the modulation of the R2 component of the nociceptive blink reflex (R2 nBR) following blockade of the GON (Busch *et al* 2006, 2007). The nBR, a trigeminofacial brainstem reflex, is a validated method for assessing central trigeminal transmission (Kaube *et al* 2000; Katsarava *et al* 2002a), and specifically the processing of trigeminal nociception in migraine (Katsarava *et al* 2002b; Kaube *et al* 2002). These findings provide empirical support for the functional influence of cervical afferents on trigeminal nociceptive inputs. Modulation of these inputs could potentially be beneficial in migraine treatment (Busch *et al* 2006). Conceivably, occipital activation of

the TCC represents the cervicogenic equivalent to application of an "inflammatory soup" on to the dura that has been shown to induce central sensitisation (Spekker *et al* 2021) and ensuing increased sensitivity to trigeminal inputs (Goadsby *et al* 1997; Le Doare *et al* 2006).

The precise mechanisms that initiate and maintain CS in migraine are still not fully understood (Olesen *et al* 2009; Akerman *et al* 2011). Resolving this question and determining to what extent it is characterised primarily by dysregulation / disinhibition as opposed to noxious peripheral afferents is crucial for a deeper comprehension of migraine.

## Manual cervical reproduction and lessening of typical head pain: the science substantiating the inference

Clinicians utilising manual therapy (MT) identify spinal dysfunction based on various features; among these are the ability to reproduce local and referred pain and restrictions in spinal joint motion (Westerhuis 2010). It is recognised that local symptoms only, in the presence of restriction in movement, constitute "comparable signs", i.e. inviting an assumption that they represent the source of the symptoms (Maitland *et al* 2005). However, comparable signs are much less reliable than those incorporating head pain referral (Hanten *et al* 2002; Humphreys *et al* 2004; Jull *et al* 1988, 1997). Furthermore, PAIVM testing is more likely to provoke head pain than passive physiological movement testing (Watson & Drummond 2012, 2014; Westerhuis 2010; Watson & Trott 1993). Therefore, PAIVMs are the most important tests to incriminate cervical afferents in head pain.

However, since 1991, my own modus operandi has been to *sustain* the PAIVM examination techniques reproducing familiar head pain. These are applied with sustained, gradually increasing thumb pressure, examining the movements of the top three intervertebral segments in their recognised planes, and within their accepted ranges of movement, specifically to detect early, pathological, soft tissue resistance. In the event of a typical head pain referral, the thumb pressure is maintained, simultaneously assessing the status of referred head pain.

Forty thousand hours of clinical experience and research (Watson & Drummond 2012, 2014) demonstrates that at least 80% of patients fulfilling ICHD-3 criteria for migraine, report lessening to a variable degree but often with complete resolution, of their referred, usual head pain within 90 seconds. Moreover, sustaining the technique repeatedly, for example, five repetitions, results not only in decreasing the intensity of head pain referral but also in more expeditious resolution. Notably, *it is extremely rare for reproduced head pain to worsen as the technique is sustained*.

Furthermore, patients presenting with allodynia frequently report that after lessening of their referred pain, allodynia had decreased or resolved (Mansilla-Ferragut *et al* 2009; La Touche *et al* 2009; Coranado *et al* 2012), perhaps indicating that a pre-existing CS state had diminished. Cutaneous allodynia is a clinical manifestation of central nervous system sensitisation present in 79% of migraineurs (Burstein *et al* 2000a) and is caused by sensitisation of trigeminovascular neurons in the TCC (Burstein *et al* 2000b).

Although the efficacy of MT is documented, the precise mechanisms of its therapeutic impact remain speculative (Bialosky *et al* 2009; Bishop *et al* 2015). One of the principal rationales for MT intervention is that an ongoing barrage of noxious afferent input from neuromusculoskeletal spinal dysfunction may result in maladaptive neuroplastic changes in circuits of the spinal cord (Haavik-Taylor & Murphy 2007; Byl *et al* 1993).

If we assume that CS is driven centrally (Fernandez-de-las-Penas *et al* 2010), a key clinical characteristic is temporal summation, or "wind-up" of pain which serves as an index for augmented central pain facilitation. Facilitated temporal summation results from a gradual escalation in neuronal output in response to a series of consistent nociceptive stimuli. This potentiation of temporal summation signals an amplification in central pain processing pathways. Clinically, this is evident through the exacerbation and intensification of pain (Woolf 2011; Arendt-Nielsen *et al* 2018; de Tommaso *et al* 2014).

This response is in stark contrast to clinical experience with patients fulfilling diagnostic criteria of migraine and migraine with aura, i.e. head pain referral lessens as the technique is sustained. This phenomenon suggests that sensitisation of the TCC in migraineurs is driven peripherally by noxious cervical afferents and also that CS, a neuroplastic phenomenon, can change quickly (Arendt-Nielsen *et al* 2018).

This information piqued my curiosity because it was in contradistinction to what has been and continues to be taught in the global physiotherapy / physical therapy curricula, i.e. that cervical afferents do not play a causal role in sensitisation of the TCC in migraine.

In 2009, a pivotal case involved a migraineur with a 20-year history who presented during an interictal phase. For the past decade, he had experienced consistent, severe tenderness to touch (allodynia) on the left side near the hairline. Following the reproduction of typical head pain and subsequent applications of the previously described technique, which resulted in decreasing intensity of head pain referral and more expeditious resolution to the point where minimal referral occurred, the patient was asked to assess his tenderness and was surprised to find it absent.

This seminal experience inspired my PhD programme, in which the principal study involved assessing the effect of manual cervical referral of typical head pain on the R2 nBR in migraineurs (Watson & Drummond 2014).

The nBR is elicited in the orbicularis oculi muscles through stimulation of the supraorbital nerve using a concentric high-density electrode, primarily activating Aδ afferents. This reflex involves interneurons in the spinal trigeminal nucleus. In migraine, there is a notable interictal lack of R2 nBR habituation to both short (Katsarava *et al* 2003) and long (Di Clemente *et al* 2005) stimulus series, with normalisation of nBR habituation observed during migraine attacks (Katsarava *et al* 2003). Additionally, patients with migraine exhibit temporal summation of the nBR (Griffin *et al* 2004).

The text in the box below is a summary of the seminal of several studies for my PhD under the expert tutelage of Professor Peter Drummond, Murdoch University, Perth, Western Australia (Watson & Drummond 2014).*
To my knowledge, this is the first time a manual cervical examination technique has been shown to influence trigeminal nociceptive neurotransmission, modulating the nBR. The findings of decreased area under the curve (AUC) and increased latency of R2 during the cervical intervention provide a clinical correlation with anaesthetisation of the greater occipital nerve (Busch *et al* 2006) and are supported by a functional magnetic resonance imaging (fMRI) study in which manual therapy was administered to the ankle joints of rats following capsaicin injection. Subsequent to mobilisation, there was decreased activation of the dorsal horn (Malisza *et al* 2003). By analogy, the results of this study support noxious upper cervical afferents as the peripheral source or driver of sensitisation of the TCC that can be ameliorated by manual cervical reproduction and lessening of usual head pain.

Further support for cervical afferents as the peripheral driver of CS comes from the influence of MT on spinal excitability. Manual therapy has been associated with immediate reductions in nociceptive flexion reflexes (Courtney *et al* 2010) and decreased temporal sensory summation (Bialosky *et al* 2014; Bishop *et al* 2011), which could reflect both diminished facilitation and augmented inhibition of nociceptive input within the central nervous system. This is supported by the recognised hypoalgesic effects of MT (Vicenzino *et al* 2001; Mohammadian *et al* 2004; George *et al* 2006) and systematic reviews which have found that MT lowers pressure pain thresholds, indicating its effect on the dorsal horn (Coronado *et al* 2012; Gay *et al* 2013).

Another consideration, however, is that the hyperexcitability of the second-order neurons in the TCC results from a decrease in supraspinal inhibition (Goadsby & Bartsch 2008; Villaneuva *et al* 1984), i.e. a central rather than a peripheral driver.

In animal models, the phenomenon of "wind-up" observed in dorsal horn wide-dynamic range neurons is characterised by a gradual escalation in neuronal output in response to a series of consistent nociceptive stimuli.

*In the study of 15 participants fulfilling diagnostic criteria of migraine[1], intervertebral movements of the atlanto-occipital and C2-3 spinal segments were evaluated interictally. Each participant underwent two sessions. In one session, either the atlanto-occipital or C2-3 segment was examined. In the other, pressure was applied to the common extensor origin of the ipsilateral arm. Both interventions (constant thumb pressure) were sustained for four trials of 90 seconds. The R2 nBR was measured in response to a supraorbital electrical stimulus both before and during each intervention. Main outcome measures included blink count, R2 component area under the curve (AUC), and R2 latencies in the R2 nBR, alongside participant-rated pain intensities for head pain and applied pressure (for cervical and arm) and the supraorbital stimulus (stimulating electrode).*

*During the cervical session, each participant reported typical head pain referral. As the examination technique was sustained, head pain lessened in all participants, decreasing significantly from the beginning to the end of each trial (P = .000) and from the beginning of the first trial to the end of the last (P = .000). Also notable is that referred head pain at the end of each trial decreased progressively across the four trials when compared with ratings at the beginning of each trial (P = .047). The referred head pain eased immediately on cessation of the technique at the end of each trial in all participants. No participant experienced a migraine episode for at least 48 hours following the study.*

*When averaged across the four trials, mean ratings of tenderness to thumb pressure were identical across the four trials for both arm and cervical interventions (P = 1.0). However, participants reported a significant reduction in tenderness across trials during the cervical but not the arm intervention (P = .005). Mean ratings of the supraorbital stimulus were similar across the trials (P = .635) and were comparable for cervical and arm interventions (P = .072).*

*To establish a baseline for R2, blinks were elicited in the absence of either the cervical or arm intervention during the first trial i.e. R2 was recorded over five trials. Cervical and arm interventions were then applied in the ensuing four trials. The number of blinks decreased significantly across the five trials (P = .000) and was comparable for the cervical and arm interventions (P = .624).*

*While the R2 AUC decreased irrespective of intervention, this reduction was significantly greater for the cervical than arm intervention (P = .037).*

*Analysis of the R2 latencies revealed a notable increase across the five trials (P = .037). However, this increase was significantly greater for the cervical than arm intervention (P =.012).*

*** Editor's note: The paragraphs in italics above are taken directly from Dean's PhD dissertation.**

This intensified and repetitive afferent input can extend the increased neuronal output beyond the stimulus duration, leading to CS (Arendt-Nielsen *et al* 2018). In centrally mediated CS, painful stimuli applied between one to three times per second for a duration of five to 10 seconds result in intensification, and increased severity of pain by the end of the stimulus sequence (Arendt-Nielsen *et al* 1994). As an example, in our study (Watson & Drummond 2014), nociceptive input was sustained for four trials of 90 seconds duration. This enhancement of temporal summation is interpreted as an indicator of heightened central pain processing. Beyond temporal summation, nociceptive stimuli also undergo spatial summation (Quevedo & Coghill 2007), where pain intensity escalates with the expansion of the stimulated area. This phenomenon is attributed to central networks and centrally driven CS (Bouhassira *et al* 1995). Enhanced spatial summation is similarly regarded as indicative of elevated central pain facilitation (Arendt-Nielsen *et al* 2018).

Clinically, the phenotype of centrally driven CS is an aggravation of pain, whether through intensity and / or spatially, reflecting neurophysiologically, temporal summation of the nBR (Giffin *et al* 2004); neither happened. Conversely, referred head pain decreased with concomitant habituation of the R2 component of the nBR. This suggests CS of the TCC in study participants (Watson & Drummond 2014) was mediated peripherally rather than centrally (Olesen *et al* 2009).

It is possible, however, that the cervical reproduction of head pain in this study (Watson & Drummond 2014) activated the supraspinal CPM system, modulating nociception at the TCC (Villanueva *et al* 1984; Sessle 2000). Nevertheless, if CPM were operational, we would have expected identical effects on the R2 nBR during the arm and cervical interventions, as mean ratings of local tenderness were the same. This is supported by inconsistent pain ratings across the different sites, i.e. decreased head pain referral and cervical tenderness, whilst arm tenderness and supraorbital stimulus remained unchanged.

Other possible mechanisms for the inhibitory effect on pain demonstrated in the study are placebo, expectations, and emotional or psychological influences (Bialosky *et al* 2009).

In our study, (Watson & Drummond 2014) participants were included only if usual head pain could be produced when stressing either the AO or C2 - C3 segments during the "inclusion / exclusion" session. If head pain was referred, both segments were evaluated to determine which of them more accurately reproduced the usual head pain prior to experimental sessions. This process ensured that the participants' usual head pain was reproducible and would stop immediately on cessation of the technique, effectively "cueing" them to perceive the procedures as non-threatening. Literature indicates that anticipated pain relief can diminish the pain experienced in response to harmful stimuli (Voudouris *et al* 1990; Price *et al* 1999; Colloca & Benedetti 2005; Hoffman *et al* 2005; Koyama *et al* 2005). It was likely that the inclusion / exclusion session set an expectation that head pain would intensify during interventions and subside immediately afterwards. However, participants had no prior knowledge about the trajectory of referred head pain as the technique progressed. Accordingly, it was considered that any placebo effect was minimal.

While the underlying processes of emotional pain modulation remain unclear, evidence suggests that increased anxiety may heighten pain sensitivity (Rhudy & Meagher 2000; Bishop *et al* 2001; McCracken & Turk 2002), and moderate fear could suppress pain (Rhudy & Meagher 2000, 2001, 2003; Martenson *et al* 2009; Flor *et al* 2002; Willer *et al* 1981; Rhudy *et al* 2004; Lewis *et al* 1980; Watkins & Mayer 1982). This indicates that unpredictable threats may intensify pain experiences, whereas predictable threats may induce hypoalgesia (Rhudy & Meagher 2000). The psychological state of participants was not monitored in our study (Watson & Drummond 2014), hence its influence over the course of the experiment is undetermined. Nevertheless, the dissociation between pain perception and R2 nBR response supports the possibility that the reductions in referred head pain, cervical tenderness, and inhibition of R2 nBR were due to a specific cervical neurophysiological effect rather than psychological influences.

## Summary

Previous research has demonstrated that the presence of macroscopic cervical lesions is not a prerequisite for establishing the significance of cervical afferents in the etiology of head pain. Moreover, the limited efficacy, inherent ambiguity in the partial relief outcomes, and the impractical nature of anaesthetic blocks significantly diminish their utility in effectively probing cervical origins of head pain.

Emerging evidence posits that a meticulously conducted cervical PAIVM examination, particularly incorporating the C0-C1 segment, is at least as effective, if not superior, to anaesthetic blocks in assessing cervical afferents to head pain referral. However, the diagnostic reliability of temporarily reproducing typical head pain during such examinations is questionable. This criterion, initially considered pivotal for establishing cervical relevance, should be re-evaluated. A more robust diagnostic indicator would be the "reproduction and resolution" of head pain that resolves as the PAIVM technique is maintained.

This perspective is corroborated by the observed attenuation, or desensitisation, of the TCC in migraineurs, which occurs consequent to the PAIVM-induced reproduction and resolution of typical head pain. Such an outcome not only substantiates the cervical afferents' role in migraine but also confirms them as a peripheral driver of CS in this condition.

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## About the author

Dean Watson graduated in 1976 with Dip Tech Physio, he subsequently gained Grad Dip Adv Manip Ther (Hons), Master Manip Ther (University of South Australia), and in 2017 achieved a PhD from Murdoch University, Perth, Western Australia.

Dean's PhD investigated the role of cervical afferents in migraine, which for the first time demonstrated a manual cervical intervention (the Watson Headache® Approach) ameliorating the underlying disorder in migraine.

Since 1990, Dean has treated headache and migraine conditions exclusively, accumulating more than 40,000 hours of clinical experience with more than 13,000 patients. From this experience, the Watson Headache® Approach has evolved. Dean has taught and presented internationally on numerous occasions since 1997, the Approach having been taught to more than 5,000 practitioners in 25 countries.

Dean has published in *Cephalalgia* and *Headache*, the premier, peer reviewed headache publications. He remains an active clinician, researcher, educationalist, and author. Dean is currently an Adjunct Senior Lecturer at the University of South Australia.

**The Watson Headache® Approach** is

1. a series of specific original and other (non-high velocity thrust techniques) sustained manual techniques/ exercise
2. embedded in an innovative clinical reasoning process to
3. correct a previously unrecognised pattern of relevant musculoskeletal misbehaviour.

## References

Full details of the references in this article can be found by accessing our *In Touch* spring edition online at www.physiofirst.org.uk/resources

### REVIEW SUPPORTING QAP

**How might this article improve patient outcomes?**
This article gives us a greater understanding of the sensory mechanisms behind migraine headache, and by improving our awareness of the trigemino-cervical complex and the effect it has on both cervicogenic and migraine headache, we can adjust our treatment techniques to help to resolve these symptoms in our patients.

There is a clear explanation on why we must think differently about how we treat primary headache patients, and it is highlighted how manual therapy influences these tissues, and the importance of the zygapophyseal joints in the cervical spine as a source of central sensitisation and potential chronic pain.

The author delivers a lot of detail, but it is well worth reading more than once. As a physiotherapist who specialises in treating headache, and a follower of Dean's work, I found this article to be a really useful reminder of the mechanisms behind the treatment.

**How can this article help to achieve or maintain QA status?**
When patients come to see us with either primary or secondary headache symptoms, we can apply the knowledge and treatment principles highlighted in this article to improve our outcomes.

Additionally, in helping us to understand the mechanisms of headache, this article can help us to grow our confidence in treating these problems and deliver the best outcomes for our patients.

Reviewer
**Elizabeth Palmer**