

Management of headache: from evidence to practice

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Anecdotal evidence indicates that physiotherapists have been successfully treating headache with manual therapy for many years (Edeling, 1974). Patients seek skilled practitioners for such treatment, confident in the belief that manual therapy is effective in reducing headache symptoms and associated impairment. There is now growing evidence to support the confidence that patients place in physiotherapist in managing headache (Hall, 2011).

One of the difficulties in managing headache is that headache is both a disease and a symptom, which makes diagnosis and management complex. Recent evidence suggests that physiotherapists are capable of differentiating different headache forms, particularly with respect to identifying those people with headache likely to benefit from manual therapy (Hall et al., 2008). This article will present a case study highlighting how evidence can be used in the diagnostic process to identify patients likely to respond to physical intervention.

Peter is a 38-year old male with a 7-year history of gradually worsening, right-side dominant daily occipital headache (Figure 1). He has a history of neck pain which preceded the headache by 2-years. Headache and neck pain both occur together. Symptoms had plateaued in the previous year and were rated at 56/100 on the Headache Disability Index (Andrasik et al., 2005), indicating a substantial burden.

Peter was working full time, with little break away from his desk. He had also changed exercise routine and was no longer physically active due to starting a family 4-years previously. His children had initially slept poorly, leading to his current poor sleeping habit. He found that sitting at a desk provoked his headaches, to the point that he now stood to avoid exacerbation. Sitting for 20 minutes usually aggravated his headache, which would then last till the next morning. Driving was also provocative. If sitting was avoided headache would be present on a daily basis but was less severe than if sitting were attempted. There were no associated features such as aura, nausea, or photophobia.

A neurologist had assessed Peter, who instigated various tests including imaging of the neck and head. Only minor degenerative change was noted at C5/6, an inconsequential finding as previous studies have failed to identify a link between changes seen on imaging and cervicogenic headache (CGH) (Antonaci et al., 2001, Coskun et al., 2003, Knackstedt et al., 2012). The neurologist trialed unsuccessfully various medications (including Triptan's), but Peter best controlled his symptoms by avoiding sitting and taking paracetamol daily. Despite this, the headache remained daily, with an average intensity of 4/10. Peter was otherwise healthy and there were no features indicating red or yellow flags.

Based on the International Study Group (ISG) diagnostic criteria (Sjaastad et al., 1998) Peter's headache are defined as probable CGH (Antonaci, Ghirmai, 2001). That is based on the headache being unilateral without side-shift with associated neck pain preceding headache, with moderate non-throbbing symptoms

aggravated by neck postures, with symptoms of varying duration and intensity, and in the absence of associated features.

Table 1 shows the ISG diagnostic criteria for CGH. These criteria have been investigated for reliability and validity (Vincent and Luna, 1999) and are more comprehensive than the criteria published by the International Headache Society (Headache Classification Committee of the International Headache, 2013). One important aspect of International Headache Society diagnostic criteria for headache is that symptoms are relieved following neck treatment. This perhaps provides some leeway for including headache of potential cervical origin, when the patient responds to neck treatment.

Despite diagnostic features of CGH, these could also be ascribed to migraine. As headache features overlap, up to 50% of patients receive an incorrect diagnosis (Pfaffenrath and Kaube, 1990, Moeller et al., 2008). To explain this, it has been postulated that primary headaches form a spectrum, with shared common patho-physiological mechanisms (Cady et al., 2002). It has also been suggested that CGH forms part of this spectrum, with a shared underlying patho-biological process (Watson and Drummond, 2012).

The underlying patho-physiology of CGH is one of convergence, where afferents from the upper 3 cervical nerve roots converge in the trigeminocervical nucleus (TCN) with trigeminal afferents (Bogduk and Govind, 2009). Hence, cervical afferent input is misinterpreted as headache. Sensitization of the TCN is an important factor in headache generation in patients with migraine (Akerman and Romero-Reyes, 2013) as well as patients with CGH (Niere, 2009, Chua et al., 2011). In a recent study comparing patients with cervical facet joint pain with and without headache, differences were found in sensory processing indicating TCN sensitization as a key feature of CGH (Chua, van Suijlekom, 2011).

It would seem logical that if TCN sensitization is the common cause of headache symptoms, then reducing TCN sensitivity may be key to headache management. Despite the similarity in the mechanisms underlying different headache forms, physical treatment has different effects on each headache form (Biondi, 2005, Bronfort et al., 2010), again underpinning the importance of examination and headache classification.

On physical examination Peter sat with an unusual posture. He had a very flat spine, posterior pelvic tilt, and markedly retracted head on neck (Figure 2). This was not the case when he stood, where he adopted a sway-back posture and forward head position. On questioning, he said that an internet search had led him to adopt the upright posture when sitting. It was his belief that this was good posture, despite the fact that this position provoked headache, and he could not sustain it. Correcting his sitting position, by altering his pelvis, spine posture and head position felt “easier” to him and his headache/neck pain were immediately reduced, but not eliminated.

Previous studies have raised questions regarding the link between cervical posture and headache, with some studies reporting an association (Watson and Trott, 1993, Budelmann et al., 2013), which is not substantiated by others (Treleaven et al., 1994, Dumas et al., 2001, Zito et al., 2006b). One explanation for

this might be the wide variation in cervical posture seen in normal people (Miyazaki et al., 2008). Hence, a common postural abnormality is unlikely to be seen in headache. Peter's case reflects this. His posture does not fit with a stereotypical postural deviation. However, his symptoms are clearly linked to his posture, and avoiding this reduces his symptoms. This highlights one of the problems of blindly applying evidence to all patients with a specific condition. Future research may indicate a range of postural abnormalities associated with different forms of headache.

Due to the postural abnormality, a decision was made to apply a Mulligan Headache SNAG (Mulligan, 2010). The C2 vertebra was fixed posteriorly, while the head was retracted (Figure 3). This caused an immediate increase in symptoms, while reversing the direction of force (Reverse Headache SNAG) immediately reduced symptoms (Figure 4). This indicated that symptoms are likely to arise from at least the joints between Occiput and C2. Head retraction and protraction has been shown to induce maximum range of movement (ROM) at C0/2 (Ordway et al., 1999), without maximally stressing to end range articulations below C2. Hence increased symptoms associated with protraction and retraction in comparison to whole cervical flexion and extension point to the vertebral segments above C2 as probably symptomatic.

Examination of cervical ROM triggered localized neck pain and revealed moderate limitation on cervical flexion. Symptoms were increased and headache provoked on upper cervical retraction, while ROM was limited to half the expected 15° ROM (Panjabi et al., 1988). Other cervical movements were not problematic. Examination of upper cervical rotation with C2 stabilized was normal (Figure 5). A recent study measured 11° right rotation for this test in supine position in healthy people when measured using MRI (Osmotherly et al., 2013). Limitation of ROM on this test is likely to indicate an impairment of C1/2 movement, and should be corroborated with the C1/2 flexion-rotation test (Hall and Robinson, 2004).

The primary movement at C1/2 is rotation (Takasaki et al., 2011), with minimal rotation occurring at C0/1 (Karhu et al., 1999). As rotation was normal, it is unlikely that C1/2 is the source of symptoms in this case, and together with limitation and pain on retraction points towards the C0/1 as the symptomatic source.

One of the major diagnostic criteria for CGH is restricted cervical ROM (Sjaastad, Fredriksen, 1998). However, some (Zito, Jull, 2006b, Jull et al., 2007b) but not all studies (Dumas, Arsenault, 2001, Hall and Robinson, 2004) have identified this in CGH. Furthermore, there are reports of diminished cervical ROM in people with migraine (Bevilaqua-Grossi et al., 2009) and Tension-Type headache (Fernandez-Mayoralas et al., 2010), although this is not always the case (Oksanen et al., 2006, Amiri et al., 2007, Jull et al., 2007a). One explanation for the presence of ROM impairment in primary headache is that sensitization of the TCN influences cervical segmental muscle tone potentially impairing ROM. A good example of this is seen in patients with temporomandibular disorder, where upper cervical spine movement impairment is a commonly associated feature (von Piekartz and Hall, 2013, Grondin and Hall, 2014).

Segmental examination revealed hypomobility at C0/1 & C2/3 vertebral levels. The flexion-rotation test was negative (Figure 6), with an eyeballed estimate of close to the normal range of 44° to each side (Hall and Robinson, 2004). The minimum cut-off value for a positive test is approximately 33° and there is a close relationship between headache severity and a ROM on a positive test (Ogince et al., 2007, Hall et al., 2010a, Hall et al., 2010c, Hall et al., 2010d). This test has also been shown to be helpful in distinguishing people with migraine from those with CGH (Hall, Briffa, 2010c). Impairment found on this test is not universal in people with CGH and neck pain (Hall, Briffa, 2010a). According to one report a positive test is seen in 72% of people suffering CGH (Hall, Briffa, 2010d). While the test has been shown to be a valid marker of C1/2 segmental movement (Takasaki, Hall, 2011), potentially an altered starting position of the C2 vertebral (rotated segment) may influence the test result and give the impression of C1/2 dysfunction. This was not the case, as the C2 spinous process was found to be centrally located.

Critical to the diagnosis of a cervical component in headache is the presence of signs of restriction and pain on cervical palpation (Jull, Amiri, 2007a), in particular headache reproduction (Watson and Drummond, 2012). Palpation also helps identify the likely segment involved (Hall et al., 2010b). In this case headache was reproduced on palpation of the right posterior arch of C1 when the neck was positioned in flexion/right rotation (Edwards, 1992). Local neck pain only was reproduced on palpation testing of the right C2/3 facet joint.

Impairment of muscle function has also been identified as an important feature of CGH (Watson and Trott, 1993, Jull et al., 1999, Zito, Jull, 2006b, Jull, Amiri, 2007a). A reflection of this is shown by the long-term improvement in headache symptoms following retraining the muscle system (Jull et al., 2002). Impairment includes loss of postural alignment and neuromuscular control as well as muscle weakness, endurance and extensibility (Jull and Niere, 2004). Impairments of the deep neck flexors (DNF) are some of the defining features of neck related headache (Watson and Trott, 1993, Jull, Barrett, 1999, Zito et al., 2006a). Similar impairments have not been found in migraine or TTH according to one study (Jull, Amiri, 2007a), but were in TTH in another (Fernandez-de-las-Penas et al., 2007). Other changes to the muscle system in CGH include evidence of muscle tightness (Zito, Jull, 2006b) and trigger points (Roth et al., 2007).

In the current case, there was significant reduction in the ability to perform the craniocervical flexion test for DNF function (Figure 7), with marked substitution of superficial muscles. Muscle length was not an issue, but there were trigger points in muscles associated with CGH. Hence, when taken in conjunction with the whole examination, there was substantial evidence for a cervical component to headache genesis. Jull et al. (2007) have reported that the combination of poor control of the DNF, limitation of cervical ROM, and pain on palpation of the upper cervical spine can distinguish CGH from primary headache forms with high sensitivity and specificity. All 3 signs were present in Peter's case, raising confidence in diagnosis.

On reflection, Peter's symptoms developed following a change from a physically active to sedentary job. A previous neck problem was the precursor, which was

made worse by maladaptive sitting posture, poor muscle control, lack of exercise, and poor sleep all potentially contributing to sensitization of the TCN. Inappropriate medication use on a daily basis is also a potential contributing factor to sensitization of the TCN (Srikiatkachorn et al., 2014). Hence, management for this case is likely to require a multifaceted approach.

Management to address the maladaptive postural control was instigated first, as this was felt to be the main driver. This involved correction of the pelvis, head and spine position, training for progressively longer periods in sitting. Accessory mobilization of the impaired joints was included soon after, in particular progressing towards flexion/right rotation at C0/1 (Figure 8). Once improvement in upper cervical flexion was achieved, exercises to improve the DNF control were commenced (Jull et al., 2008). Specific exercise is more effective at improving activation of the DNF than mobilization (Lluch et al., 2014), but the combination of specific exercise with mobilization is even better (Jull, Trott, 2002). Peter was also given extensive education regarding the association between TCN sensitization and headache, particularly with respect to adequate sleep, exercise, and medication use.

Peter made good progress over 8 treatment sessions. His neck disability score dropped to 8/100, much more than the 30 points change required to reflect a long-term improvement (Jacobson et al., 1994). He was able to sit at work for extended periods, and headache was reduced to once per week at a 3/10 level. He had stopped taking medication, improved his sleep pattern and was walking daily for exercise.

This case illustrates the value of an evidence-based, clinically reasoned approach to examination and management of headache.

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Table 1: International Headache Society diagnostic criteria for cervicogenic headache (Sjaastad, Fredriksen, 1998)

Major criteria	<p>I. Symptoms and signs of neck involvement</p> <p>a) Precipitation of comparable symptoms by:</p> <ol style="list-style-type: none">1) neck movement and/or sustained, awkward head positioning, and/or2) external pressure over the upper cervical or occipital region <p>b) Restriction of range of motion in the neck</p> <p>c) Ipsilateral neck, shoulder or arm pain</p> <p>II. Confirmatory evidence by diagnostic anaesthetic block</p> <p>III. Unilaterality of the head pain, without sideshift</p>
Head pain characteristics	<p>IV. Moderate-severe, non-throbbing pain, usually starting in the neck</p>

	Episodes of varying duration, or fluctuating, continuous pain
Other characteristics of some importance	<p>V. Only marginal or lack of effect of indomethacin</p> <p>Only marginal or lack of effect of ergotamine and sumatriptan</p> <p>Female gender</p> <p>Not infrequent history of head or indirect neck trauma, usually of more than medium severity</p>
Other features of lesser importance	<p>VI. Various attack-related phenomena, only occasionally present, and/or moderately expressed when present:</p> <ul style="list-style-type: none"> a) nausea b) phono- and photophobia c) dizziness, d) ipsilateral “blurred vision” e) difficulties swallowing f) ipsilateral oedema, mostly in the periorcular area