Making sense of common headache presentations in Musculoskeletal Physiotherapy: Differentiation, assessment, and treatment.

Dr Neil Langridge DClinP MSc BSc (Hons) FCSP FMACP

Introduction

This article aims to provide the reader with a primary care overview of the most common types of headache seen in clinical practice by Musculoskeletal (MSK) physiotherapists. It will offer some support for introducing clinical reasoning within this highly complex area of practice. The paper will also look at where MSK physiotherapy can aid in the treatment of the neck related headache, and cast an eye over the evidence underpinning the approaches.

The vast majority of individuals will experience headache at least one time in their lives (Gobel et al 1994; Rasmussen et al 1991). The global prevalence of headache has been reported to be 47%, whilst among adults, the individual prevalence of migraine is reported to be 10%, tension type headache (TTH) around 40%, and current Chronic Daily Headache at 3%. Although TTH is generally less burdensome than migraine to the individual sufferer, the total societal burden of this headache type seems to be even larger than that of migraine because of its much higher prevalence (Stovner et al 2007).

Classification

Headache classification can be accessed through *The International classification of headache Disorders* (2018), which delivers a deep exploration into the multitude of classifications identified. It outlines sub-groups of headache and also highlights that many headache disorders can be co-diagnosed in patients, and so singular headache diagnoses are not compartmentalised in this way. It firstly describes two distinct types of classification; Primary Headache, where the headache is the medical disorder and there is no other

underlying pathology causing the headache and associated symptoms, and Secondary Headaches where the headache is a symptom arising from an underlying pathology (i.e. the headache is caused by another medical condition). Examples of primary headache are migraine, TTH and trigeminal autonomic cephalalgias (ICHD 2018). Secondary headache examples may present in patients with intracranial processes, (including tumour, haemorrhage, infection or vascular disorders) and disorders such as using/overusing various drugs, infections, and head injury. Secondary headaches generally are associated with other symptoms reflecting the underlying pathology, and it is important to recognise that the overall incidence of serious headaches due to secondary causes is very low, but many are associated with Red Flags (Bigal and Lipton 2007). A useful mnemonic that can alert the clinician to possible serious pathologies is "*SNOOP*" and this is outlined in the table below:

Signs and symptoms in history	Possible Pathology to consider
Systemic symptoms/signs	Fever, malaise, weight loss
Systemic disease	Malignancy, inflammatory
Neurological presentation	Mass lesion, stroke, encephalitis
Onset (sudden, new)	Subarachnoid haemorrhage,
Onset after 40 years of age	Temporal arteritis
Positional and pattern change	Intracranial hypotension, posterior fossa pathology; overuse of medication.

(Adapted from Dodick 2003)

The three most common headaches seen in primary care are the primary headache disorders, and for the clinician being able to recognise fairly classical signs of symptoms when Red Flags have been excluded, is vital in terms of primary care management.

TTH is the most common primary headache (Robbins and Lipton 2010). People with infrequent episodic TTH are unlikely to seek medical advice and will generally self-manage. As the frequency of TTH increases, so commonly does the severity of the pain and the likelihood that the patient will present for treatment; younger patients are also more likely

to consult a practitioner in these cases (Holroyd et al 2000). Usually, patients report a mild to moderate, bilateral sensation of muscle tightness or pressure lasting hours to days and not associated with constitutional or neurological symptoms. Patients may describe and indicate the location of the pain as a "band-like feeling" around the head. Sufferers may present with bilateral tightening of the cervical spinal musculature, peri-cranial tenderness which can be felt by the patient and also recognised as part of a physical assessment (Loder and Rizzoli 2008).

Migraine is a common disabling primary headache disorder. Many epidemiological studies have documented its high prevalence and socio-economic and personal impacts. In the Global Burden of Disease Study 2010 (GBD2010), it was ranked as the third most prevalent disorder in the world. Migraine has two major types: Migraine without aura is a clinical syndrome characterised by headache with specific features and associated symptoms, whilst Migraine with aura is primarily characterised by the transient focal neurological symptoms that usually precedes or sometimes accompany the headache (Viana et al 2017).

Migraine without aura has characteristics of unilateral presentation, throbbing moderate intensity and worsens with physical exertion. Clear differentials from TTH are the associated symptoms of nausea, photophobia/phonophobia and no preceding aura, whilst migraine with aura, that constitutes approximately 15%-30% of all migraines, is a headache with a transient associated neurological symptom. This is described as the aura and can be visual, motor or sensory. A visual aura is the most common and may include flashing lights, and/or zig zag lines. The sensory aura can be numbness or paraesthesia whilst motor symptoms can be as severe as hemiplegia (Martin 2004). The reason for the symptoms in migraine are not fully explained; but proposals have been made after observations of imaging concerning alterations in cerebral blood flow, cortical spreading depression (CSD), and possible neurogenic inflammation, leading to the possible sequalae to the symptoms experienced by the individual (De-Simone et al 2013; Lauritzen 2001). CSD is a slowly propagated wave of depolarisation followed by suppression of brain activity, and is a remarkably complex event that involves dramatic changes in neural and vascular function (Charles and Baca 2013). The early authors that described this work such as Leao (Dalkaro et al 2017), suggested that vascular change is due to vasodilation, however further work has suggested the vasodilation

is then followed by vasoconstriction of the cerebral blood flow (Borgdorff 2018). (*It is beyond the scope of this paper to detail the complexities of the theories underpinning Migraine.*)

The trigeminal autonomic cephalalgias (TACs) are described as a group of primary headache disorders characterised by unilateral head pain that occurs in association with ipsilateral cranial autonomic features (Goadsby and Edvinnson 2020). The TACs include such presentations as cluster headache (CH) and paroxysmal hemicrania (PH). CH is a unilateral headache associated with autonomic features and is commonly triggered by alcohol (Cohen et al 2007). They are characterised by the attacks that may have three broad forms: single stabs, which are usually short-lived; groups of stabs; or a longer attack comprised of many stabs between which the pain does not resolve to normal, thus giving a ‘‘saw-tooth’’ phenomenon with attacks lasting many minutes, whilst similar aura like features to migraine have been described (Goadsby et al 2020). The autonomic features seen in the ipsilateral cranial dysfunction such as lacrimation (watery eyes), rhinorrhoea (runny nose) and miosis (pupil constriction) (Lambru et al 2019) are also features to be aware of.

The most common presentation that an MSK Physiotherapist encounters is commonly known as the cervico-genic headache disorder (CGH). CGH is defined as a secondary headache disorder, arising from nociceptive structures in the cervical spine or occipital region. CGH arises primarily from musculoskeletal dysfunction in the upper three cervical segments. The pathway by which pain originating in the neck can be referred to the head is proposed to be the trigeminocervical nucleus (TCN), which descends in the spinal cord to the level of C3/4, and is in anatomical and functional continuity with the dorsal gray columns of these spinal segments. The trigeminal nucleus is divided into the main sensory nucleus and spinal tract nuclei which are located caudally in the cervical spinal cord. Marked convergence of the primary afferents of the upper 3 level in the cervical spine with the TCN have been established (Choi and Sang 2016). Hence, input via sensory afferents, principally from any of the upper three cervical nerve roots, may mistakenly be perceived as pain in the head, a concept known as convergence and this convergence forms the neuro-anatomical basis for the CGH.

Provocation of headache by applying experimental nociceptive stimuli to upper cervical structures has been reported in several studies. In a review of the diagnosis and treatment of cervicogenic headache, several experimental studies on humans reporting referred pain patterns to the head, caused by stimulation of nociceptive afferent input from myofascial structures of the upper cervical spine, have been cited (Bogduk and Bartsch 2020). Mechanical nociceptive afferent stimuli elicited by giving a firm pressure to myofascial structures of upper cervical segments (C0-3) also has been shown to provoke the patient's typical headache in patients with CGH, TTH, and migraine, leading to challenges in differentiation (Anarte et al 2019; Cescon et al 2019; Jull and Hall 2018). Therefore, a painful soft tissue structure due to convergence may sensitise cells such as “wide-dynamic range” at the dorsal horn leading to the experience of painful symptoms in the distributions of the TCN. Cervical musculoskeletal dysfunctions of joints and muscles have been observed in patients with migraine, TTH and cervicogenic headache. In the context of the neurophysiological interconnection between the dorsal root of C2 (greater occipital nerve) and the TCN, it may be not surprising that in participants with headache, most cervical musculoskeletal dysfunctions reported are present in the upper cervical spine (Amiri et al 2007; Zito et al 2006). Therefore, common practice in manual therapy assessment may include palpation of the sub-occipital muscles and trapezius, local assessed restricted motion of the cervical segments C0-3, and direct stress on joints in the upper cervical spine (Luedtke et al 2016;2017).

The TCN distribution is highlighted below:

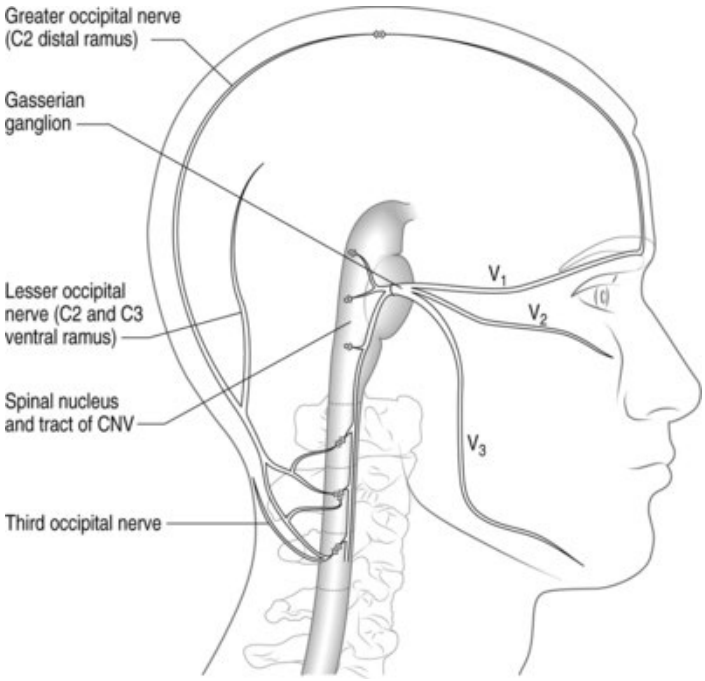


Fig 1:The distribution of the TCN via Convergence theory

Taken from Bhagia et al 2015

The most commonly reported clinical signs and symptom in CGH are commonly defined after excluding Red Flags and secondary headaches, followed by exclusion of TTH and migraine. The pain normally is felt arising from the neck and is commonly unilateral but can be bilateral. Patients will normally describe neck stiffness with no neurological features; autonomic features may be apparent due to the relationship with the TCN. The symptoms of the cranial autonomic system will not be associated with the same pain distribution as seen in TAC, such as cluster headaches and this would also be a clear differential.

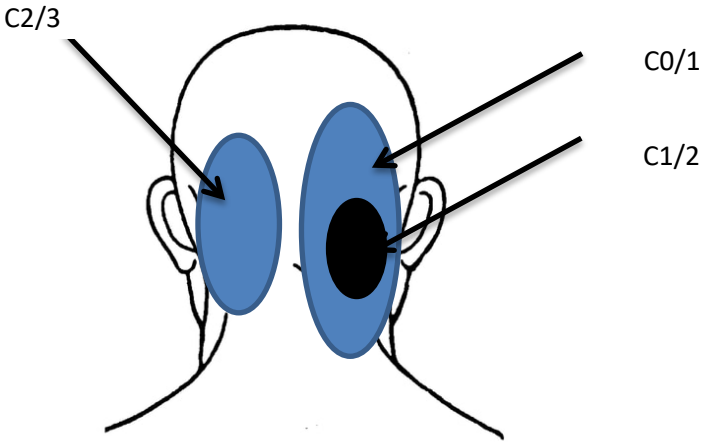


Fig 2:Referred pain patterns after noxious stimulation of upper cervical joints and C2/3 disc (adapted from Bogduk and Govinid 2009)

Clinical Assessment

Manual examination of the cervical spine structures reviewing local tone and pain responses seeking to reproduce the features of the headache are widely advised as part of a multi-modal assessment. Specific tests, such as the cervical flexion rotation test (Ogince et al 2007), have been validated as an examination procedure in the presence of CGH. Further features leading to sensitisation of structures and pain experiences are also vitally important to consider as part of a broad assessment. Emotional, psychological features coupled with general health, well-being and sleep, would be key elements to consider when generating a management plan (Lewis and O'Sullivan 2018). Considerations of muscular strength, general spinal mobility and sensorimotor capacity would also be advised when delivering a comprehensive MSK assessment.

When considering the hypothesis of CGH, the clinical examination process is firstly underpinned through exclusion of Red Flags followed by other forms of primary headache. There are no bio-markers or specific anatomical markers currently attributed to CGH however exclusion of significant underlying pathology via MRI has been recommended in some cases when Red Flags or signs of neurological dysfunction are present (Coskun et al 2003). It is then appropriate to consider CGH as an underlying driver of a neck-related headache which can be supported by further interpretations, assessments and examinations such as below:

	Provocation of the headache radiating from the cervical spine.
1.	Loss of Neck movement
2.	External Pressure on the occipital or higher cervical region on the symptomatic side.
3.	Ipsilateral; neck, shoulder, arm pain that is non-radicular in origin.
4.	Positive response to diagnostic blocks in the upper cervical spine.

Fig 3:Supportive criteria in cervicogenic headache (Adapted from Sjaastad et al 1998 IHCD 2018)

Initial observations of patient posture inclusive of range of motion will then further inform the possible diagnosis. According to Dumas (2001), they were unable to associate a forward head posture with patients reporting CGH, whilst Watson and Trott (1993) found a weak correlation with a reduction in neck angle. In a meta-analysis conducted by Gadotti et al (2008), they were able to associate a reduction in head range of motion to the CGH group when compared to matched controls. The flexion –rotation test, as advocated by Hall, is a test that has reported validity and reliability in the assessment of CGH (Hall et al 2010).

According to the description of Hall and Robinson (2004), the flexion-rotation test is conducted with the cervical spine fully flexed in an attempt to block as much rotational movement as possible above and below C1/2. The head is then rotated to the left and the right. If firm resistance is encountered and range is limited before the expected end range, then this is said to be clinically significant, with a presumptive diagnosis of limited rotation of the atlas on the axis. Manual examination has had high sensitivity and specificity reported to detect the presence or absence of cervical joint dysfunction in neck pain and headache (Jull et al 1988; Sanmark and Nisell 1995). Moreover, Zito et al (2006) determined that the presence of upper cervical joint dysfunction measured by manual examination, in comparison to measures of posture, range of motion, cervical kinesthesia, and cranio-cervical muscle function, most clearly identified CGH sufferers. The term manual examination incorporates tests of passive physiological intervertebral motion, as well as passive accessory intervertebral motion, such as postero-anterior pressures. Motion restriction and symptom responses indicate the most painful dysfunctional cervical motion segment (Jull et al 1997), although the reliability of these tests has been questioned (Jonsson and Rasmussen-Barr 2018).

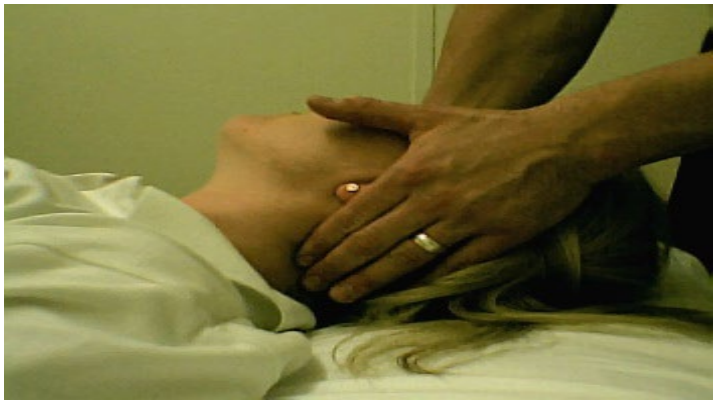


Fig 4: Manual examination of the upper cervical spine.

Oliver et al (2018), in a study that assessed muscular sensitivity in cervicogenic headache versus non CGH found a strong correlation with pain in the upper trapezius and CGH. Muscular sensitivity is often associated with muscle tightness—that is, increased muscle tone (Chen et al 2016). Jull et al (1999) investigated upper cervical muscle tightness in 15 CGH patients and 15 asymptomatic controls. They found that resistance to passive muscle stretch was significantly increased in the upper trapezius muscles in the CGH group, but not in any of the other muscles tested—namely, levator scapulae, scalenes (anterior, middle, and posterior divisions), and the short upper cervical extensors. Lastly, strength of the deep neck flexors, extensors and proprioception had mixed results in that although strongly associated in whiplash, proprioceptive loss was not as prevalent in this meta-analysis in CGH whilst impairments in deep flexors and treatment of these impairments have been shown to help with pain and disability in CGH, the results from the meta-analysis did not strongly support these individual findings.

Efficacy of Treatment

Bogduk and Govind (2009) state clearly that for "*probable cervicogenic headache, exercises with or without manual therapy seems to be the best option among conservative therapies. All other treatment strategies are entirely speculative*".

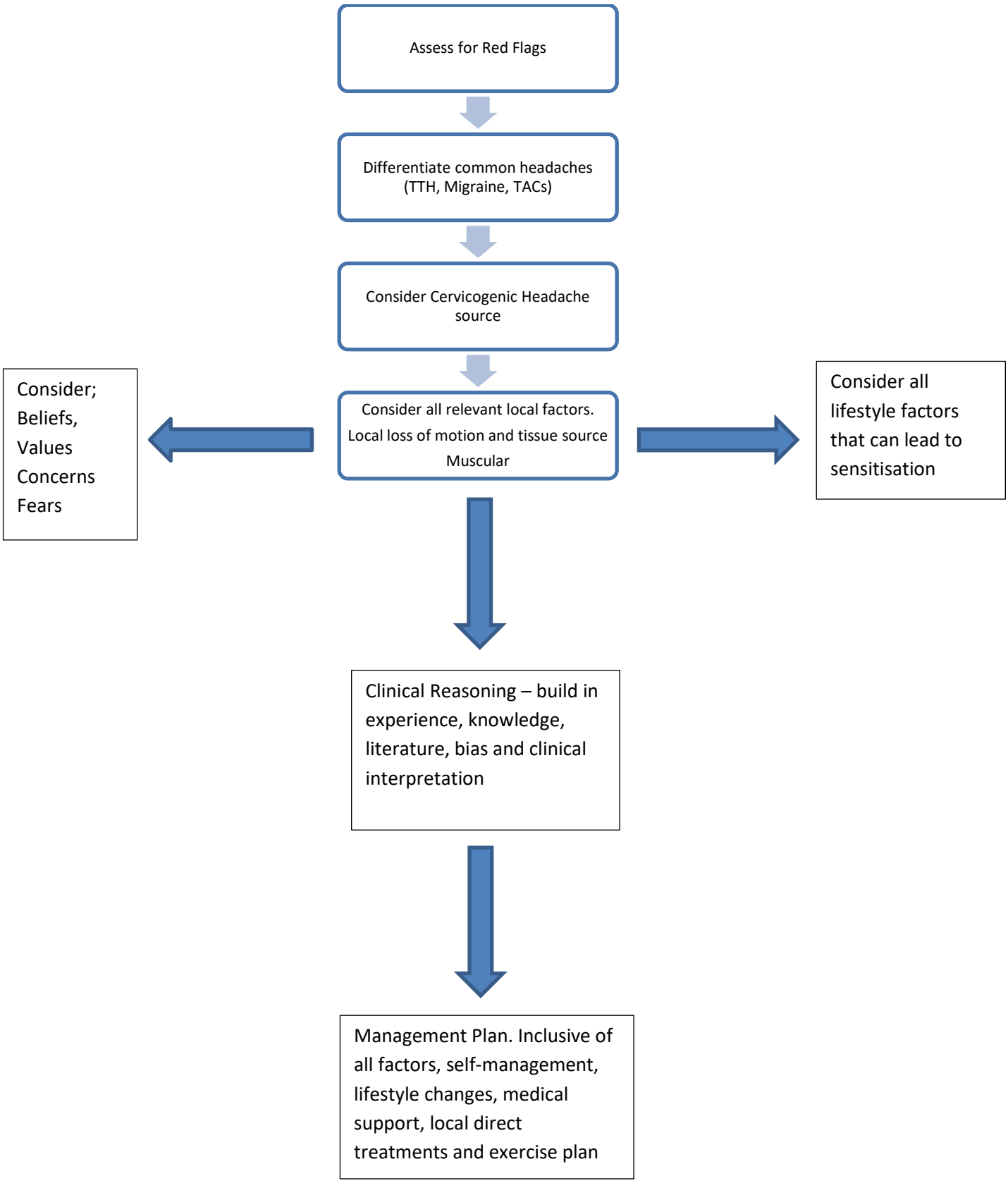
Among the different manual therapies proposed for headaches, cervical manipulation or mobilisation are the most commonly used by physical therapists (Grant and Niere 2000; Nillson et al 1997). A systematic review found six randomised controlled trials suggesting that spinal manipulation is more effective than gentle massage, drug therapy, or no intervention at all in patients with CGH (Posadzki and Ernst 2011), whilst Racicki et al (2013) also concluded that spinal manipulative therapy was effective for reducing the pain in CGH. Garcia et al (2016) concluded after a systematic review of spinal manual therapy, that it is difficult to draw a firm clinical significance, however a review of literature completed by Rani et al (2019), based on five moderate quality systematic reviews, concluded that physiotherapy interventions are effective in CGH treatment and noted that manipulation

and mobilisation (particularly SNAGS) were the most effective treatment options among all available physical therapy interventions. Taken independently, the findings of the studies suggest that manual therapy on the cervical spine is more effective than traditional physical therapy interventions or sham intervention in reducing pain intensity and frequency of headaches in this population. There are differing sources of evidence such as Boursiak et al (2010) who assessed the efficacy of thrust cervical manipulative therapy (CMT) in children and adolescents with recurring CGH, of ages 7–15 years. Outcomes measures utilised were: percentage of days with headache, duration of headache, percentage of missed school days due to headache, percentage of days where medication was needed, and intensity of headache. The authors found no statistically significant differences between the CMT group and the sham CMT group. Non-thrust self-management techniques such as sustained natural apophyseal glides have been shown in a double blinded placebo RCT of 32 subjects that headache intensity and severity were significantly reduced in the SNAG group (Hall et al 2007).

The use of exercise in a multi-modal model is advised inclusive of aerobic conditioning, and this is supported in the literature with the additional application of muscle endurance and strength training exercises targeting the deep cervical flexor muscles that has been shown to be more effective than stretching exercises for reducing pain (Ylinen et al 2010). A Cochrane Review concluded that there is moderate quality evidence supporting cervico-scapulothoracic strengthening and endurance exercises for improving pain and function in patients with CGH, although further studies were concluded as needed (Gross et al 2016). Jull et al (2002) via RCT investigated the effects of only exercise in the treatment of CGH. The exercise-only group displayed statistically significant improvements (P-0.001) at 7 weeks when compared to the control group for headache frequency, headache intensity, and neck pain, but not headache duration. Therefore, it is proposed that therapeutic exercise programs consisting of re-education of the deep neck flexors should be incorporated into the management with modification of lifestyle factors with general exercise management (Fernandez –de-las-Penas and Cuadrado 2016). Making these in-roads into improving outcomes for CGH requires the appropriate diagnosis in the first instance. Differentiation of common headache disorders is a challenge for Physiotherapists and in a study among the 384 respondents, 32.3% classified the tension-type headache case consistent with IHS

criteria. The cervicogenic and migraine headache cases were classified at 54.8% and 41.7% consistent with IHS categories, respectively (Dale et al 2019).

Therefore, when postulating the most effective treatment in neck related headache clinicians should primarily link to the clinical reasoning surrounding differentiation and potential sensitising factors in the patient presentation, and so any treatment guidelines must be linked to a reasoning framework that encapsulates best principles in effective decision-making. Assuming any one tissue-based mechanism is the sole driver to benefits and positive outcomes is naïve, and fails to recognise the complexity in pain presentations seen in standard MSK Physiotherapy practice. Although there are many proposed mechanisms surrounding how migraine, TTH and CGH may occur independently or in concert and therefore how treatments may help, many treatment studies fail to really consider the multi-dimensional elements that are seen particularly in more persistent symptoms. The research as in many areas of MSK practice does not really deliver on the complexity of the person in front of the clinician and the emotional drivers and wider concepts associated with the patient narrative that we see daily in practice. Therefore, when considering the assessment of headache that may be amenable to MSK physiotherapy, it is suggested that a multi-dimensional approach inclusive of medical management is built into the management plan. A simple clinical reasoning model to consider when met with a headache presentation is presented below:


Assess for Red Flags
Differentiate common headaches (TTH, Migraine, TACs)
Consider Cervicogenic Headache source
Consider all relevant local factors.
Local loss of motion and tissue source
Muscular
Consider;
Beliefs,
Values
Concerns
Fears
Consider all lifestyle factors that can lead to sensitisation
Clinical Reasoning – build in experience, knowledge, literature, bias and clinical interpretation
Management Plan. Inclusive of all factors, self-management, lifestyle changes, medical support, local direct treatments and exercise plan

Conclusions

The assessment and clinical reasoning of even the most common headache disorders seen in MSK Physiotherapy are considered highly complex. There remains no clear differential standard that make diagnosis simple, and the clinician is advised to ensure the clear exclusion of Red Flags with consideration of potential underlying pathology in all cases. Building some pattern recognition of common signs and symptoms is advised as helpful, and in terms of clinical examination clear assessment of mechanical and pain provocation tests in the upper cervical spine are needed.

Treatments should be multi-modal, building upon the patient narrative and not limited to mechanical treatments. Exercise coupled with appropriate de-sensitising and life appropriate measures should be considered throughout.

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