REVIEW ARTICLE

CERVICOGENIC HEADACHE PART 1: AN ANATOMIC AND CLINICAL OVERVIEW

(AWARDED THE OPTP AWARD FOR EXCELLENCE IN A PUBLISHED REVIEW OF THE LITERATURE**)

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ABSTRACT: *Cervicogenic headache is a headache arising from painful dysfunction of the upper cervical spine. This paper reviews current literature on the anatomy, etiology, clinical presentation and differential diagnosis of cervicogenic headache. Lower cervical spine levels and cervical soft tissue components will be incorporated where they have a direct influence on the upper three segments.*

KEY WORDS: CERVICOGENIC HEADACHE, HEADACHE, NECK PAIN, PATHOLOGY

INTRODUCTION

Cervicogenic headache (CGH) is a headache (HA) arising from painful dysfunction of the cervical spine, particularly the upper three segments. Studies have shown that a connection exists between HA and poor posture, weak cervical flexors, facet joint athropathy, cervical joint trauma, and joint hypomobility and hypermobility[1-7]. The term cervicogenic headache is recognised but not yet officially accepted by the International Headache Society (IHS) Classification Committee[8,9]. For the purposes of this paper, the term cervicogenic headache will be used as it is in current literature. This paper will focus on CGH as it emanates from the upper three cervical segments.

ANATOMY AND ETIOLOGY

Many pain sensitive structures in and around the upper cervical spine may be implicated in CGH. These structures can be broadly categorized into joints (occiput to C3), ligaments, nerve roots and nerves, and muscles and their attachments to bone. Vascular components may also be implicated; these will be dealt with briefly later in this paper.

UPPER CERVICAL SPINE DYNAMICS

The upper cervical spine is the transitional area between the fixed skull and mobile lower cervical spine. Transitional areas are subject to greater static and dynamic strain than other vertebral levels[10]. The cervical area has two normally occurring curves: the larger lordotic curve in the lower cervical area and a reversal of this in the upper cervical spine, observed when the head and neck are in a neutral position. The persistence of the upper cervical curve is responsible for maintaining the horizontal line of vision. This makes possible flexion and extension of the upper cervical spine independent of the lower cervical spine, and vice versa[11].

The cervical spine has divisions occurring both morphologically and histologically at the C2/3 level[11]. The occipito-axial (C0/1. C1/2) articulation is well recognised as a transitional area between the fixed skull and mobile cervical spine[10]. The concept of two transitional areas in the upper cervical region is supported by a study by Jull[10]. Two hundred and twenty-five volunteers (92 males and 133 females) 10 to 65 years of age participated in the study. No prior mention was made of either headaches or neck pain. Passive accessory and physiological inter vertebral movements of all the cervical spine joints were manually examined by two experienced manual therapists who performed a total of 78 separate tests on each patient. Results of each test were graded according to perceived mobility on a scale of 1-5, 1 being classified as hypermobile and 5 as very limited to no movement. There was complete agreement in 88% of cases and only minor discrepancies in the other 12%. Stiffness was the most common finding, with hypermobility being rare. The percentage incidence of involvement was highest at C0/1, followed by C2/3, then C1/2. There was a rapid decrease in comparable joint signs opposite C3/4 (Fig. 1).

RANGE OF MOTION

Marked variability exists in the reported ranges of motion at the cranio-vertebral joints. This reflects various methods of describing movements, different measuring techniques, lack of reliability and validity studies, and errors in measurement. In a literature review Bogduk[12] concludes that it is not worth pursuing intervertebral range of motion as a diagnostic test. Intersegmental range of motion is an unstable parameter, with the uncertainty of any measurement being approximately 5 degrees.

The movement of greatest amplitude at the C0/1 joints is flexion/extension or nodding. Literature varies in measurement from 10 to 35 degrees for this joint[11]. Lateral flexion here is often described as a lateral tilt with a simultaneous contralateral rotation within this joint[11,13]. Penning and Wilmink[13] in a CT study of normal subjects, describe a lateral displacement of 4.4 mm of the atlas relative to the foramen magnum. This lateral displacement occurred contralateral to the direction of rotation. Rotation is described as a slipping movement of one occipital condyle forwards and the other backwards[11,13]. Jull[10] reports an essentially equal division between subjects in relation to symmetry and asymmetry, and left and right asymmetry of the transverse process of the atlas. No correlation was found between those subjects with abnormal accessory movement of C0/1, C1/2, C2/3 and asymmetry at the atlas. Also within the population with asymmetry, no relationship could be determined between the findings of abnormality of the left or right joint tests in the presence of left or right asymmetrical prominence or non-prominence. Jull concludes that this anomaly of the atlas has no apparent effect on upper cervical joint mobility and should be interpreted as an incidental finding.

The movement of greatest amplitude at the C1/2 joints is rotation. Literature varies in measurement here from 9 to 40.5 degrees[11-13]. Rotation is accompanied by a screw-like action, accounting for vertical translation of the atlas in relation to the axis. Rotation to the left is checked by the tension of those fibres of the left alar ligament which are attached to the dens in front of the axis of movement and those of the right alar ligament which are attached to the process behind the axis of

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movement[11]. Flexion and extension are seen by the relative separation and approximation of the spinous process of the axis relative to the posterior arch of the atlas. This movement also involves gliding of the atlantal arch superiorly on the odontoid in extension and inferiorly in flexion. Approximately the same amount of lateral flexion occurs between the axis and atlas (4.07° ± 2.01°), and the occiput and atlas (3.97° ± 1.54°). This lateral flexion is coupled with contralateral axial rotation between the atlas and axis[11].

Saldinger and Dvorak[14] investigated the histology of the transverse and alar ligaments. They conclude that collagen, as the most exclusive constituent together with the fiber orientation determine the mechanical properties of these ligaments. This supports the hypothesis that the ligaments can be irreversibly over-stretched or even ruptured when the head is rotated, and, in addition, flexed by impact trauma as in unexpected rear-end collisions.

Structurally and biomechanically the cervical intervertebral joints, including C2/3, are saddle joints, allowing movements in only two planes at right angles to each other[12]. The uncinate processes form a concave joint surface facing upwards and forwards in the plane of the facet joints. The upper vertebral body has a concave lower surface in the sagittal plane. Movement can only occur in the plane of the facet joints and in the sagittal plane. It is well established that lateral flexion results in a coupled range of ipsilateral rotation at each of the cervical motion segments. This is usually compensated for by a de-rotation of the atlanto-axial level. This explains why it is possible for the face to remain in the frontal plane while side flexion is performed and coupled range of ipsilateral rotation below the atlanto-axial level still occurs[11].

The instantaneous axis of rotation (IAR) is a mathematical concept that has been used to describe motion in contemporary cervical spine biomechanical studies[12-15]. It is the centre about which a moving vertebra appears to rotate if its motion during flexion and extension is perceived as a uniform arcuate movement. The IAR can be located by simple goniometric means if radiographs showing the starting and ending positions of the moving vertebra are superimposed[12]. Mayer *et al*[16] reported that patients with cervical headache exhibited abnormal IARs, particularly at C2/3. There was also a small widening of the C2/3 joint in extension as compared to flexion. Pain was elicited by pressure to the cervical spine in this area; therefore, Mayer *et al* suggested a correlation between clinical symptomatology and radiographic findings at the same location. Amevo *et al*[17] found 72% of 109 symptomatic patients exhibited at least one clearly abnormal IAR at or above the symptomatic segment. The mechanical disturbances were most common at C2/3 and C3/4, implicating symptomatic joints at C1/2 and C2/3. They concluded that abnormal IARs were, therefore, a valid marker of neck pain. Bogduk[12] hypothesizes that the IAR changes are brought about by muscle spasm secondary to the pain. This may well suggest that a lower cervical injury may cause upper cervical segments to become symptomatic. It thus follows that the headache that commonly accompanies C5/6 pain may not be directly related to that segment but may arise as a result of secondary mechanical disturbances at C2/3 or C3/4[13]. Worth[11] writes that abnormal patterns of movement may cause persistent irritability in the joint above. This irritability may be temporarily relieved by treatment but is likely to recur unless the motion segments are restored to as normal a pattern of movement as possible.

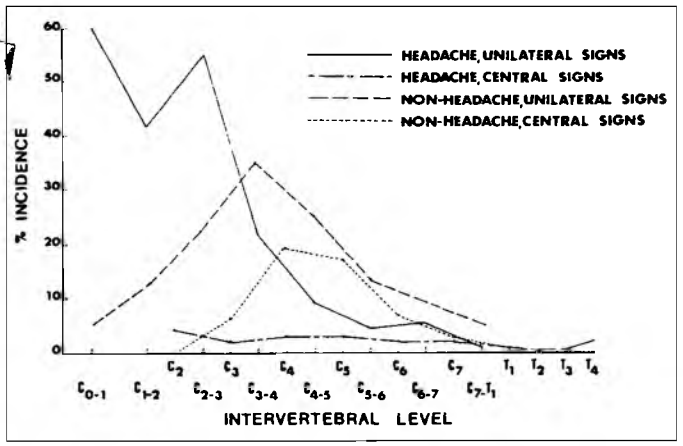

Fig 1: The percentage incidence of positive joint signs found in the left or right apophyseal joints and the central intervertebral joints for the 96 headache and the 107 non headache subjects (from Jull GA. Headaches associated with the cervical spine - a clinical review. In Grieve GP (ed), Modern manual therapy of the vertebral column, Edinburgh, Churchill Livingstone, 1986:323)

FACET JOINTS

Recent and current research has been directed at investigating cervical facet joint pain referral patterns[3,6,18-20]. The presence of mechanoreceptive and nociceptive endings in cervical facet joint capsules proves that these tissues are monitored by the central nervous system[20]. This implies that neural input from the facets is important to proprioception and pain sensation in the cervical spine. In a study published in 1990, Dwyer, Aprill and Bogduk[3] present a composite map of characteristic pain distribution from facets C2/3 to C6/7 (Fig. 2). Normal cervical facets in five volunteers were stimulated by distending the joint capsule with injections of contrast medium. As joints from above downward were stimulated, the evoked areas of pain were centered over progressively more caudal levels, and pain from the lower cervical joints extended laterally into the shoulder girdle. Pain from C2/3 was distinguished from C3/4 by its extension into the head. The authors report that pain from the C2/3 area resembled the distribution of pain reported by patients with headaches stemming from the C2/3 facet joint.

In a follow-up study the same year, Aprill, Dwyer and Bogduk tested the predictive value of the facet joint segmental pain chart[4]. Predictions were made as to the segmental location of the symptomatic segment in ten patients with suspected cervical facet pain. Correct predictions were made in all nine patients shown to have symptomatic joints on the basis of diagnostic joint blocks. The remaining patient was not relieved by blocks of either of the joints predicted. The authors claim that the results vindicate the accuracy of pain charts for predicting the segmental location of symptomatic joints.

Dreyfuss *et al*[6] investigated atlanto-occipital and lateral atlanto-axial joint pain patterns. C0/1 and C1/2 joints of five asymptomatic individuals were isolated and stimulated through mechanical distention of the joint capsules via intra-articular fluoroscopically guided injections. Head and/or neck pain was generated by each injection. All joint referral patterns were ipsilateral. The evoked referral pattern for all C1/2 joints was primarily lateral and slightly posterior at the C1/2 level. Evoked referral patterns

from C0/1 varied considerably; inferiorly from the C5 vertebral level to superiorly approaching the vertex of the skull. The authors were unable to chart a composite pain referral map because of the variability of referred pain produced, and the small sample size. They did not note that the perceived pain/sensation was greater, and that the referral pain patterns were broader, with the C0/1 joint injections.

CERVICAL NERVE ROOTS

The cervical nerve roots exit from each intervertebral foramen and split into a posterior and anterior division (Figs. 3-5). The anterior divisions of the four upper cervical nerves form the cervical plexus[21]. The first and second cervical nerve roots emerge from behind the lateral articular pillars of C1 and C2. The roots are not protected posteriorly by the pedicles and facets present in the rest of the vertebral column[15]. The ganglion of the first two cervical nerve roots are found on the vertebral arches of C1 and C2 and not inside the intervertebral foramen. The nerves travel upward and laterally and split into two primary divisions. The anterior primary ramus of the first cervical root passes over the posterior arch of C1. The anterior primary ramus of the second cervical root passes between the arches of C1 and C2; the posterior primary ramus passes backward between the posterior arches of C1 and C2 and extends to the soft tissues of the neck[15]. The posterior primary ramus of the third cervical root proceeds obliquely downwards and outwards. It gives rise to the third occipital nerve which pierces trapezius and supplies the skin on the lower and back part of the head[15,21].

The second cervical root supplies the C2 dermatome, myotome and the C1/2 joint. The third cervical root supplies the C3 dermatome, myotome and C2/3 joint (Fig. 6). The first cervical root's function is controversial. Classically, it has been described as a pure motor root supplying the C1 myotome, which are the muscles around the C0/1 joint. Gray however, describes a small sensory root consisting of three rootlets[21]. Orbital, frontal and occipital pain may be produced on stimulation of a specific rootlet[15]. This may be explained by the neural anastomosis of the occipital and supraorbital nerves. The greater and lesser occipital nerves originate in the C1 to C3 region. The trigeminal tract extends down to, and synapses between, C1 and C4. The auriculotemporal and zygomaticotemporal nerves, which supply the side of the head, are branches of the trigeminal nerve. Any problem in the suboccipatal spine can, therefore, produce any combination of headache, facial, or cervical pain.

It is generally agreed that neck trauma, such as whiplash, is one of the most common causes of chronic headache[21,15,18]. Nerve root compression as well as disc herniation may occur. Mark[15] reports that the occipital nerve can become entrapped or compressed from a hyperflexion injury by the semispinalis and possibly the splenius capitus muscles, but he does not specify greater occipital nerve, lesser occipital nerve, or both. Tearing of these muscles may occur causing muscle guarding, pulling the cranium down and compressing the suboccipital region.

The posterior primary ramus of the second cervical root, which continues into the scalp as the greater occipital nerve, may be injured by the approximation of the bony arches of C1 and C2, or by excessive mobility at this joint. With a forward head posture, posterior rotation of the cranium on the cervical spine may cause compression of the spinal tract of the trigeminal nerve, which can in turn give rise to facial or head pain separate from trigger points or any lesion occurring on the head or facial areas.

Lord *et al*[5] reports that third occipital nerve headache is a common condition in patients with chronic neck pain and headache after whiplash. One hundred patients with at least a three month history of post-whiplash neck pain participated in this double-blind study. Seventy-

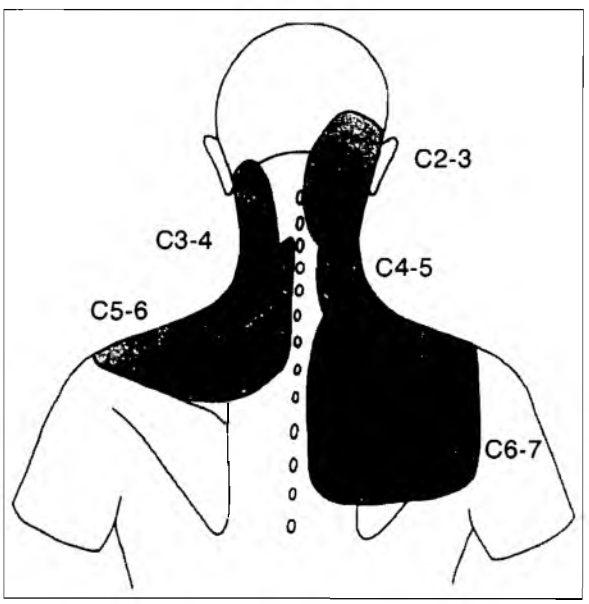

Fig. 2: A composite map of the results in all volunteers depicting the putative characteristic distribution of pain from zygapophoseal joints at segments C2-3 to C6-7. (From Dwyer A, Aprill C, Bogduk N. Cervical zygapophoseal joint pain patterns 1; a study in normal volunteers. Spine 1990; 15(6):453-457)

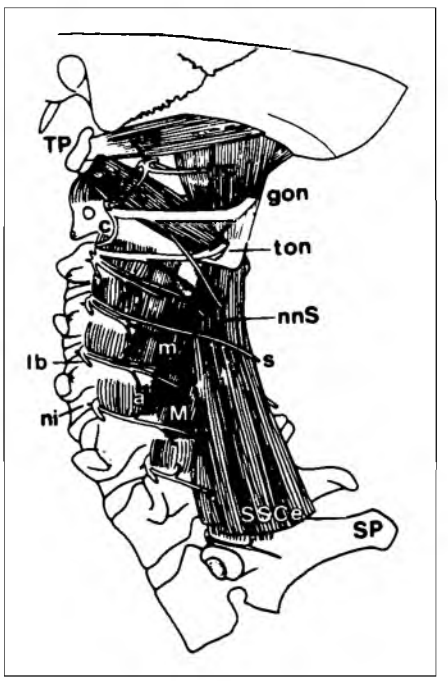

Fig 3: Deep dissection of the left cervical dorsal rami. The superficial posterior neck muscles have been resected. The lateral branches (lb) of the dorsal rami and the nerves to the intertransversarii (ni) have been transected, leaving only the medial branches (m) intact. The C1 dorsal ramus supplies the obliquus superior (os), obliquus inferior (oi) and rectus capitis (rc) muscles. The medial branches of the C2 and C3 dorsal rami, respectively, form the greater occipital (gon) and third occipital (lon) nerves. Communicating loops (c) connect the C1, C2 and C3 dorsal rami. Three medial branches (nnS) of the C2 innervate the semi-spinalis capitis, while the C3 to C8 medial branches send articular branches (a) to the zygapophoseal joints before innervating the multifidus (M) and semispinalis cervicis (SSCe), and those at C4 and C5 from superficial cutaneous branches (s). TP, transverse process of atlas; SP, spinous process of T1. (From Bogduk N. Innervation and pain patterns of the cervical spine, In Grant R. (ed), Physical therapy of the cervical and thoracic spine, 2nd edition, New York, Churchill Livingstone 1994:66)

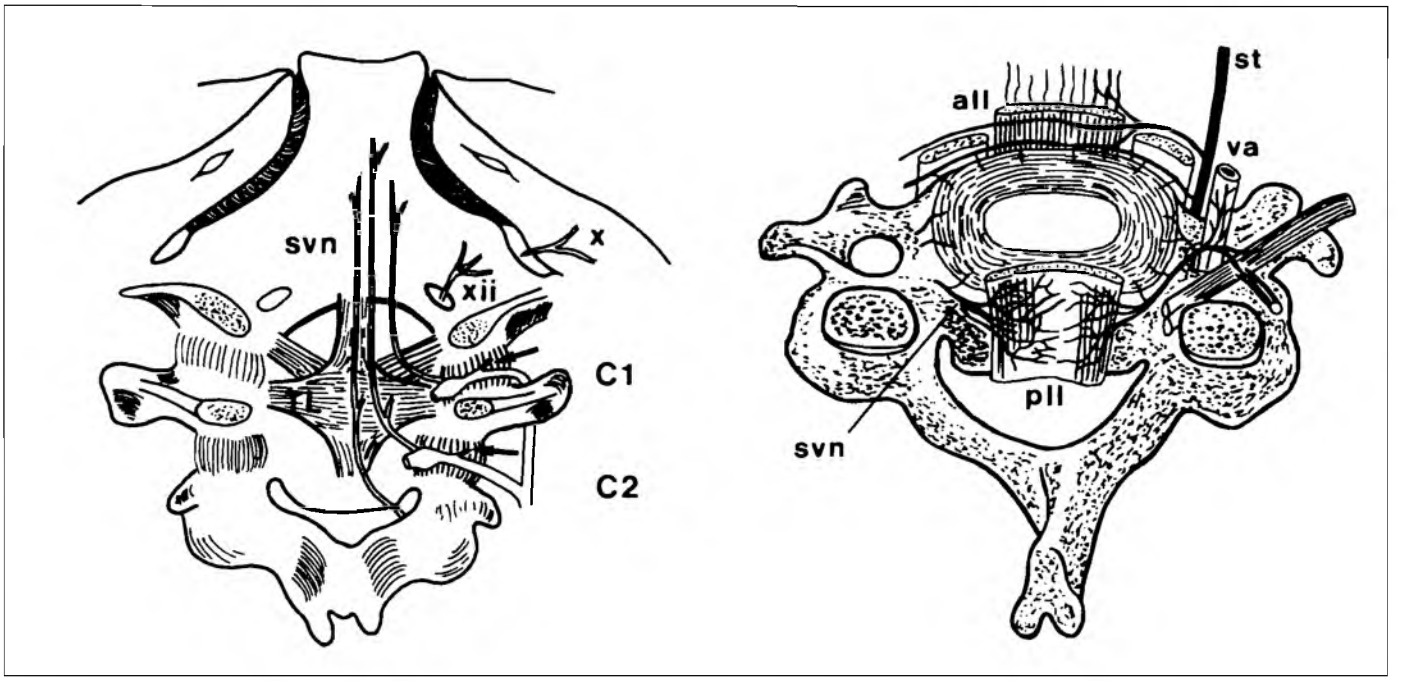

Fig 4: Distribution of the upper three cervical sinuvertebral nerves and the innervation of the atlanto-occipital and atlantoaxial joints. Articular branches (arrowed) to the atlanto-occipital and atlantoaxial joints arise from the C1 and C2 ventral rami, respectively. The C1 to C3 sinuvertebral nerves (svn) pass through the foramen magnum to innervate the duramater over the clivus. En route, they cross and supply the transverse ligament of the atlas (TL). The dura mater of the more lateral parts of the posterior cranial fossa is innervated by meningeal branches of the hypoglossal (xii) and vagus (x) nerves. (From Bogduk N. Innervation and pain patterns of the cervical spine, In Grant R. (ed), Physical therapy of the cervical and thoracic spine, 2nd edition, New York, Churchill Livingstone 1994:67)

Fig 5: The innervation of a cervical intervertebral disc. Nerve fibres enter the anterior and anterolateral aspect of the annulus fibrosus from branches of the cervical sympathetic trunk (st) that form a plexus accompanying the anterior longitudinal ligament (all). Nerve fibers enter the posterolateral aspect of the annulus fibrosus from branches of the vertebral nerve, which accompanies the vertebral artery (va). From this nerve arise the cervical sinuvertebral nerves (svn), which from a plexus accompanying the posterior longitudinal ligament (pll) and from which branches enter the posterior aspect of the annulus fibrosus. (From Bagduk N. Innervation and pain patterns of the cervical spine, In Grant R. (ed), Physical therapy of the cervical and thoracic spine, 2nd edition, New York, Churchill Livingstone 1994:67)

one complained of headache associated with their neck pain; for 40 it was the dominant complaint; for 31 a secondary problem. Diagnostic blocks of lignocaine and bupivacaine were administered in random order to the third suboccipital nerve. The diagnosis of third suboccipital nerve headache was made only if both blocks completely relieved the patient's upper neck pain and headache and the relief lasted longer with bupivacaine. The prevalence of third occipital nerve HA in the 100 patients was 27%. Among those with dominant HA (40 patients), the prevalence was 53%; the cause of HA in the other 47% of these patients was not explained. This study also found that patients with a positive diagnosis were significantly more tender over the C2/3 facet joint in comparison to other cervical levels.

SOFT TISSUE

The associated soft tissues undoubtedly play an integral role in cervicogenic headaches. Janda[22] writes that muscle dysfunction is closely related to joint dysfunction, and that treatment of impaired muscle function is a most effective way to restore normal functioning of a particular joint. He also emphasises that it would be wrong to consider muscle dysfunction independently of the functional status of other structures. It is important as well not to focus only on a symptomatic area but rather to consider it in relation to the functional status of the whole motor system. The "proximal or shoulder crossed syndrome" describes the most important muscle imbalance syndrome in the upper body (Fig.7). This is characterized by tightness in the upper trapezius, levator scapulae, and pectoral muscles, and weakness of the lower stabilizers of the scapula and the deep neck flexors. When the weakened muscles and shortened muscles are linked, they form a cross. In addition, the sternocleidomastoid and suboccipital muscles are tight in this syndrome. A forward head posture would also be observed. From a muscular point of view, a forward head posture is due to weakness of the deep neck flexors and dominance or even tightness of the sternocleidomastoid. A forward head posture particularly stresses the cervicocranial and cervicothoracic junctions and may case painful symptoms in these areas. Watson and Trott[7] investigated natural head posture and upper cervical flexor muscle performance. Sixty female subjects were divided into two equal groups on the basis of the absence or presence of headache. The headache group had confirmed upper cervical articular cause and was found to be significantly different from the non-headache group in respect to forward head posture; they also had less isometric strength and less endurance of the upper cervical flexors.

Myofascial pain patterns should also be considered in soft tissue evaluation. Travell[23] describes several cervical muscles that refer to pain specifically to the cranial area, and may be implicated as a cause of pain the cervicogenic headache. Recent literature indicates that muscles

may directly influence the pain sensitive dura mater[24], thus suggesting an alternative mechanism for headache generation. Anatomical research has shown that a connective tissue band exists between the rectus capitus posterior minor muscle and the posterior atlanto-occipital membrane[24]. This membrane is attached to the underlying spinal dura, thus contraction of the rectus capitus posterior minor can exert tension on the dura, thus producing headache.

CLINICAL PRESENTATION

Cervicogenic headache is referred to as a "syndrome" rather than a single headache entity[27,28]. Clinical manifestations vary in terms of pattern and may present in acute, sub-acute or commonly chronic forms[1].

Sjaastad *et al*[8] describe clinically consistent characteristics for CGH and propose diagnostic criteria. The presentation of CGH with the major signs and symptoms as outlined by Sjaastad *et al* are referred to consistently by several authors in discussion and research on different headache types[15,28-32]. The criterion of unilaterality is discussed by Jull[33] as being significant in terms of onset pain, which is usually in the neck of CGH. Jull[11] refers to the fact that Sjaastad *et al*[8] first characterized CGH as strictly unilateral, but she reports that subsequent studies have shown that the headache can be unilateral, unilateral with spread, or bilateral[34]. Jull[1] confirms unilaterity in that she describes the CGH as not changing sides after onset. Robarth[35] reports that the North American Cervicogenic Headache Society has adopted a definition which addresses cause rather than symptoms for CGH. A shortened version of this understanding of CGH presentation is "Referred pain perceived in any region of the head caused by a primary nociceptive source in the muscule skeletal tissues innervated by cervical nerves".

D'Amico *et al*[29] examined side-locked unilaterality and pain distribution at onset and at peak headache in 74 patients with different forms of long-lasting headache: migraine, tension-type, and cervicogenic headache as described by Sjaastad *et al*[8]. The findings of the D'Amico et al study showed that side-locked unilaterality in migraine was 20,8%, in tension-type HA 12.5%, and in CGH 100% (only 4% of the entire sample). Pain localization described for migraine was initially anterior and then spread over the entire hemicranium. In tension type HA pain was more variable around central sides, tending to localize anteriorly; and in CGH, the pain always began in the occipito-nuchal region, and it could shift to the peri-orbital area or could remain confirmed posteriorly.

Jull[1] states that one established diagnostic criterion for cervical headache is the presence of symptomatic articular dysfunction manifested as a painful abnormality of motion at a relevant segment in the cervical spine. A primary diagnosis of cervical headache is consistent with an abnormality within the upper cervical joints (occiput to C3). The motion abnormality may result from joint trauma, chronic strain, degenerative joint disease, or inflammatory joint disease. The motion abnormality may present as symptomatic joint hypomobility, hypermomobility, or instability.

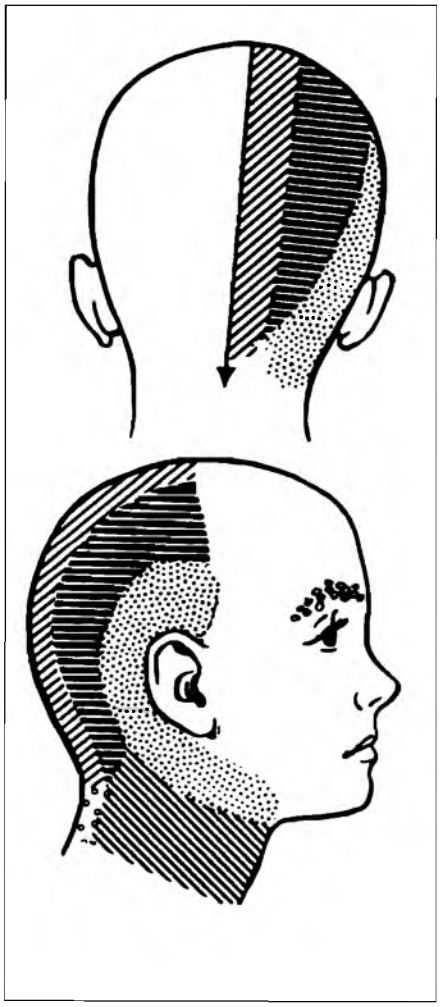

Fig 6: Dermatomic pattern of the anterior and posterior branches of C2 and C3 (according to Maigne) (From the Quebec Headache Study Group: Meloche et al: Painful Intervertebral Dysfunction: Robert Maigne's Original Contribution to Headache of Cervical Origin. Headache 33:328-334, 1993)

Jull[34] developed a cervical headache profile after reviewing 203 patients, 96 of whom had CGH. This study demonstrated that the headaches were either unilateral or bilateral. Pain was described as being in any area of the head or face but was commonly occipital, subocipital, frontal, or retro-orbital. A common history was one of trauma, but the headaches would also be related to prolonged postural and functional strain. Dizziness, nausea, lightheadedness, inability to concentrate and visual disturbances are discussed as being common[34,36,37] as opposed to "minor" by Sjaastad *et al*[8]".

The diagnostic criteria as outlined by Sjaastad *et al*[8] are as follows:

Major Signs and Symptoms

i Unilaterality of the head pain (defined as no change in side from onset)[31].

ii Neck involvement:
   a Provocation of attacks:
      - Pain, seemingly of a similar nature, triggered by neck move-

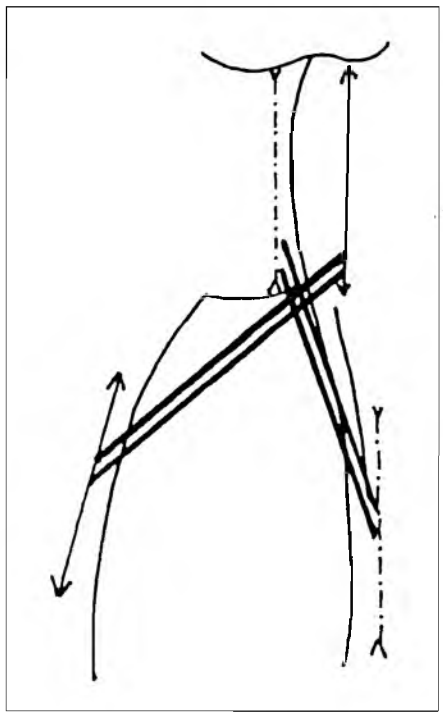

Fig 7: Schematic representation of the proximal crossed syndrome. The thick lines connect shortened and weakened muscles. The short, tight muscles are the levator scapulae, upper trapezius, and pectorals; the weak muscles are the deep neck flexors and lower stabilizers of the scapula. (From JandaV: Muscles and Cervicogenic Pain Syndromes. In Grant (ed), Physical Therapy of the Cervical and Thoracic Spine, Ch 9. Edinburgh: Churchill Livingstone, 1988.)

ment and/or sustained awkward head positioning.
- Pain similar in distribution and character to the spontaneously occurring pain elicited by external pressure over the ipsilateral upper, posterior neck region or occiput.

b) Ipsilateral neck, should and arm pain of a rather vague, non-radicular nature.
c) Reduced ROM in the cervical spine.

Pain Characteristics

iii Non-clustering pain episodes.
iv Pain episodes of varying duration or fluctuating continuous pain.
v Moderate, non-excruciating pain, usually of a non-throbbing nature.
vi Pain starting in the neck, eventually spreading to oculo-frontal-temporal areas, where the maximum pain is often located.

Other important criteria

vii Anesthetic blockades of the major occipital nerve and/or of the C2 root on the symptomatic side abolish the pain transiently, provided complete anesthesia is obtained.
viii Affects more women than men.
ix A history of head and/or neck trauma (whiplash).

Minor, more rarely occurring, non-obligatory symptoms and signs

Various attack related phenomena:

x Autonomic:
a) Nausea
b) Vomiting
c) Ipsilateral edema and less frequently flushing, mostly in the periocular areas.
xi Dizziness.
xii Phono-and photophobia
xii "Blurred vision" in the eye ipsila teral to the pain
xiv Difficulty swallowing

Classic areas of pain referral from spinal segments are described[34,36,38] and represented in figure 8.

C0/1 - Occipital, ear pain.
CI/2 - Suboccipital, posterior head to forehead, into and behind the eye, temporal pain, band around head.
C2/3 - May irritate C2 or C3 nerve root, anterior neck pain, top of head, neck tongue syndrome (transient).

Differential Diagnosis

Headache is one of the most frequently reported symptoms in the general adult population[13]. Grieve[39] describes cranial and facial pain as a frequent complaint in cervical spine disorders but points out that headache may be the presenting symptom of disorders not involving the musculoskeletal tissues. Pain sensitive structures need to be considered in differential diagnosis; these include blood vessels, dura, ligaments, fascia, periosteum, muscles, joints, skin, subcutaneous tissues, glandular tissue, sinus and nasal mucosa, dental pulp, gums in the lining of the oropharynx, endoneurium, epineurium, and perineurium of the cranial and cervical nerves[40]. Pain can also be referred into the cranium from structures located elsewhere, but pain sensitive structures are primarily involved[41].

Migraine, Tension-type, and Cluster headache syndromes

Jull' states that clinical physical therapists who are consulted on a first-contact basis by a patient with headache symptoms must decide whether cervical dysfunction is a primary cause, a partial cause in a headache continuum, is enhancing symptoms of other forms of headache, or has no role in the patient's headache. Headache types overlap within the classification of varying headache syndromes[1,9,15,40,42]. Differentiation of headache type is typically by area and behaviour of symptoms [1,29-31,34,43]. (Table 1). D'Amico *et al*[29] found that unilaterity is characteristic of all forms of long lasting headache (>4 hours) but to differing extents in different headache categories. Localization of pain, particularly at the beginning of an attack, can assist with differentiation as CGH pain is typically felt in the posterior parts of the head or neck (occipitonuchal) region at onset. The pain may remain posteriorly or shift to the peri-orbital area, and will be associated with a cervical spine movement dysfunction pattern[1,29]. Migraine pain is often located in the anterior regions of the head initially (periorbital-frontal) and then spreads over the entire hemicranium[29]. Migraine is differentiated from CGH in that there is marked associated autonomic symptoms of nausea/vomiting, photo and phonophobia[40]. Migraine may be influenced by movement of the craniovertebral region 40 whereas CGH is typically triggered by neck movement[31]. Migraines are episodic, have a reasonably consistent duration for a particular individual and frequently terminate within 24 hours. Periods between headaches are usually pain free[1]. Tension -type headache is differentiated from CGH in that pain tends to localize in the anterior regions. Pain is variable, may be present in the occipical region and is more typically bilateral with a description of a tight band around the head[30,32,34]. Tension headaches are usually long lasting and protracted and may be further differentiated from CGH in that movement dysfunction patterns in the cervical spine are not typically aggravating.

Muscle skeletal dysfunction may be a contributory factor to the etiology of both migraine and tension headaches[30,44]. Limitation of ROM and prevalence of trigger points in the cervical musculature in these types of headaches have been described[44], Vernon et al[30] challenge the "narrow prescriptive definition of cervicogenic headaches". They report findings of occipital and neck pain during headaches, tender points in the upper cervical spine region, greatly reduced or absent cervical curve and X-ray evidence of joint dysfunction in the upper and lower cervical spine in migraine and tension-type headaches. Leone *et al*[31] investigated the identification of CGH amongst patients with migraine analysing 374 headaches. In the migraine group, 44.6% experienced side-locked unilateral headaches; in these cases the pain was not precipitated by trauma or associated with neck movements and, therefore, did not fully meet the criteria of Sjaastad[8]. Leone *et al*[31] suggest that additional clinical and instrumental criteria are required to distinguish migraine from CGH.

Cluster headaches are more easily differentiated from CGH as the typical cluster pattern of pain is not found in CGH[1,15]. Cluster headaches are typically short-lasting and episodic. Pain is predominantly ocular/periocular and may be associated with a Horner's syndrome (ocular sympathetic paresis, ptosis). There is no palpable neck muscle tightness[1,15,32]. (Table 1).

Hemicrania Continua and Chronic Paroxysmal Hemicrania

Hemicrania continua is a unilateral headache syndrome similar in presentation to CGH, however it differs in that it has no mechanical precipitator, it starts in the front-temporal region and it is stopped typically with indomethacin[42]. Chronic paroxysmal hemicrania differs

from CGH as it has a modified cluster pattern of pain. Symptoms can be bilateral, occur in the ocular/periocular area and may be diffuse in the ipsilateral shoulder and arm, and it may have a mechanical precipitator. There may be signs of cervical spine involvement but ROM is normal. Chronic paroxysmal hemicrania also responds well to indomethacin[15].

Serious Pathology

Makofsky[40] outlines the following headache danger signals:

- Sudden onset of new severe headache (subarachnoid hemorrhage, meningoencephalitis, vertebral artery dissection[45], internal carotid artery dissection[46]);
- Progressively worsening headache (advancing intracranial process, brain tumor, subdural haematoma or extracranial process of temporal arthritis);
- Onset after physical exertion, straining, coughing or sexual activity (worsening vascular headaches, increasing intracranial pressure);
- Associated symptoms such as drowsiness, confusion, loss of memory, focal neurological signs, fever, arthralgia, myalgia;
- Onset after 50 years of age (most benign recurrent headaches begin before middle age, patient should be referred to a physician immediately).

Headache can be the presenting feature in the presence of ominous disease, and serious consideration must be given to the history[40]. Early diagnosis of vertebral artery dissection (VAD) or internal carotid artery dissection (ICAD) is essential due to the progressive nature of these disorders and the potential for delayed cerebral lesion[45,46]. Sturzenegger[45] reports that VAD presents in a similar way to CGH with unilateral head and neck pain (always on the side of the dissection). Sudden onset, severe sharp nature of pain with no previous history of HA are differentiating factors. VAD is usually apparent only when focal ischaemia develops. Prior to this stage these headaches can be misdiagnosed as muscle contraction or CGH[46]. ICAD[46] also initially manifests with sharp ipsilateral HA but is differentiated from CGH by site of pain. Head pain is predominantly anterior or on one entire side (frontal, temporal, parieto-occipital), is constant, steady, aching and is associated with a Horner's syndrome of oculosympathetic paresis[46].

Presentation of headache due to brain tumor is possible[39,41]. Symptoms may include a deep, steady, dull, aching pain which is sometimes severe but not as intense as migraine, can be relieved by cold packs or aspirin, and does not usually interfere with sleep. Pain occurs from distortion of pain sensitive structures such as dural membranes, blood vessels, and periosteum. In contrast to CGH it may be provoked by coughing, straining at stool, or postural changes, and the increase in intracranial pressure can cause vomiting but no nausea. Blurred vision, and a loss of lateral gaze with eyes deviated medially (cranial n.VI) may occur depending on location of the tumor. Headache is rarely caused early on by a tumor. Symptoms of neurologic CNS dysfunction are more frequent[41].

Trigeminal Neuralgia (tic dolouroux)

Trigeminal neuralgia[36] affects the cranial nerve which is responsible for sensation to the face and anterior scalp and for motor innervation of the muscles of mastication[41]. Pain occurs in this distribution with sudden, intense, superficial shooting or stabbing pain which immobilizes the face (cheek, chin, lips, and tongue). It is triggered by light touch, may come intermittently with one or two paroxysms, may last a minute only or come with repeated paroxysms giving the feeling of continuous pain. Post-herpetic neuralgia commonly involves the opthalmic branch of the trigeminal nerve and is associated with annoying parasthesias or sharp jabbing pain on a background of steady burning or aching. Post-herpetic neuralgia is easily differentiated from CGH by the behaviour, onset and area of pain. Physical therapy has no described role in the treatment of post-herpetic neuralgia.

Temporomandibular Joint

Temporormandibular disorders (TMD) are frequently the cause of headaches and facial pain[37,47-49] and can be both myogenic or arthrogenic. Arthrogenic pain may radiate to the temple, zygoma, ramus of the mandible or the ear. Myogenic pain may result in tenderness on palpation to masseter, temporalis and pterygoid muscles. TMD patients may have associated craniovertebral and postural dysfunction[37,49]. Grieve[39] comments that patients with or without upper cervical joint problems may have cheek, ear, temporal and postauricular pain. Origin of these symptoms can be either cervical, TMD or a combination. TMD can be differentiated from CGH in that TMD is aggravated by chewing hard foods or anything with excessive overuse of the jaw, and by clenching or grinding of teeth (this may be evident on intraoral examination). It may wake the person at night with jaw pain and may be associated with sleep disorders[49]. Haley *et al*[47] compared patients suffering from TMD and a general HA population; both groups demonstrated pericranial tenderness although the TMD patients had significantly more jaw dysfunction and the HA patients exhibited more neck muscle tenderness.

MYOFASCIAL TRIGGER POINTS

Janet Travell *et al*[23] describes myofascial trigger points that refer pain to the head and neck region. Muscles to consider are the sternocleidomastoid (SCM) trapezius, temporalis, splenius capitus and cervicus, multifidus, scalenes, the suboccipital group, masseter, and medial and lateral pterygoid. These muscles may be implicated in classic CGH as a primary or secondary cause.

OTHER

Grieve[39] describes the list of possible cause of headaches (ocular, aural, nasal, dental structures and sinuses) as "formidable". Other types of headaches which the clinician may be faced with can be vascular in origin in the form of essential hypertension: can be related to pre-menstrual tension, temporal arthritis, dilatation of cranial arteries due to infections, poisons, or foreign protein reactions [37,39,41].

CONCLUSION

The upper cervical spine is a complex region. A good understanding of the local anatomy and biomechanics will enhance the ability of the clinician to identify dysfunction and the tissues responsible for the generation of pain. Recognising and understanding the clinical presentation of headaches emanating from this region will direct the clinician in examine and treatment. The clinician should also be aware of the many other causes of headache and that other headache presentations may have a cervicogenic component.

TABLE 1: Headaches

	CERVICAL	TENSION	MIGRAINE	CLUSTER
Area of symptoms	• frontal, retro-orbital, temporal, occipital, assoc with subocc & neck pain (Sx usually start in neck subocc or occipital) • unilateral spread to other side or bilateral does not change sides	• frontal, retro-orbital, temporal, occipital; may have cervical Sx • bilateral	• frontal, retro-orbital, temporal, occipital; may have cervical Sx (usually start in frontal & temporal then spread to other areas) • unilateral, can change sides	• frontal, retro-orbital, temporal, occipital: may have cervical Sx (neck pain is more of an ache compare to the excruciating head pain) • unilateral, can change sides
Quality of symptoms	• Dull boring pain • can have shooting pain (deep) • Moderate to severe level (20%)	• tight band or heavy weight around the head • moderate to severe level	• throbbing bursting boring • moderate to severe level (if uncontrolled can reach disabling)	• significant ocular Sx & intense pressure behind the eye • typically excruciating
Associated symptoms	• nausea, vomiting & photophobia (ipsilateral to side of pain)	• nausea, vomiting & photophobia	• nausea, vomiting & photophobia (no correlation between side of pain & neurological features)	• autonomic disturbances (forehead sweating, tearing of the eye, ipsilateral ptosis, 7 nasal stuffiness or secretion)
Neurological signs	• not common (tongue neck syndrome occipital pain radiating to ear with ipsilateral tongue numbness: abnormal sublux of lat AA joint)			• surgically confirmed compression of C2 C3 C4 NR
Frequency and duration	• can be episodic (few hrs to few days) or chronic (semi-continuous or 2-3/ week)	• can be episodic (few hrs to few days) or chronic (semi-continuous or 2-3/week)	• few hrs - days but generally less than 24 hrs • 1/yr - several per week (distinct periodicity appropriate assoc features with painfree periods)	• 15 min - 2 hrs • 1/2/24 hr period but can range from 2 week to 8/24 hrs • typically last 1-2 mnths: chronic up to 1 yr, Remission 6 months - 2 yrs
Time and Mode of Onset	• may be present when waking & worsen as day goes on: activity dependent	• less frequently wakes with headache	• wakes with headache • warning or aura of focal neuro Sx prior to headache	• neck movements can trigger headaches
Precipitating Factors & Relieving	• sustained neck postures or movements • may not know particular pattern • stress or tension may increase headache	• stress or tension	• stress induced or stress released situations • food allergies, bright lights, exertion, noise etc.	• neck movements can trigger headaches
General Medical History	• no family history more common from 20-50 age group • F>M	• familial tendency • F>M	• familial tendency • F>M	• no familial history • M>F
History of Onset	• trauma or DJD or UCx joints	• classically not related to cervical trauma or strain	• onset related to puberty	• classically not related to cervical trauma or strain

Adapted from Jull, CA: Cervical Headache a Review. In Grieve GP (ed), *Modern Manual Therapy of the Vertebral Column*, p 333-347, New York, Churchill Livingstone, 1994.

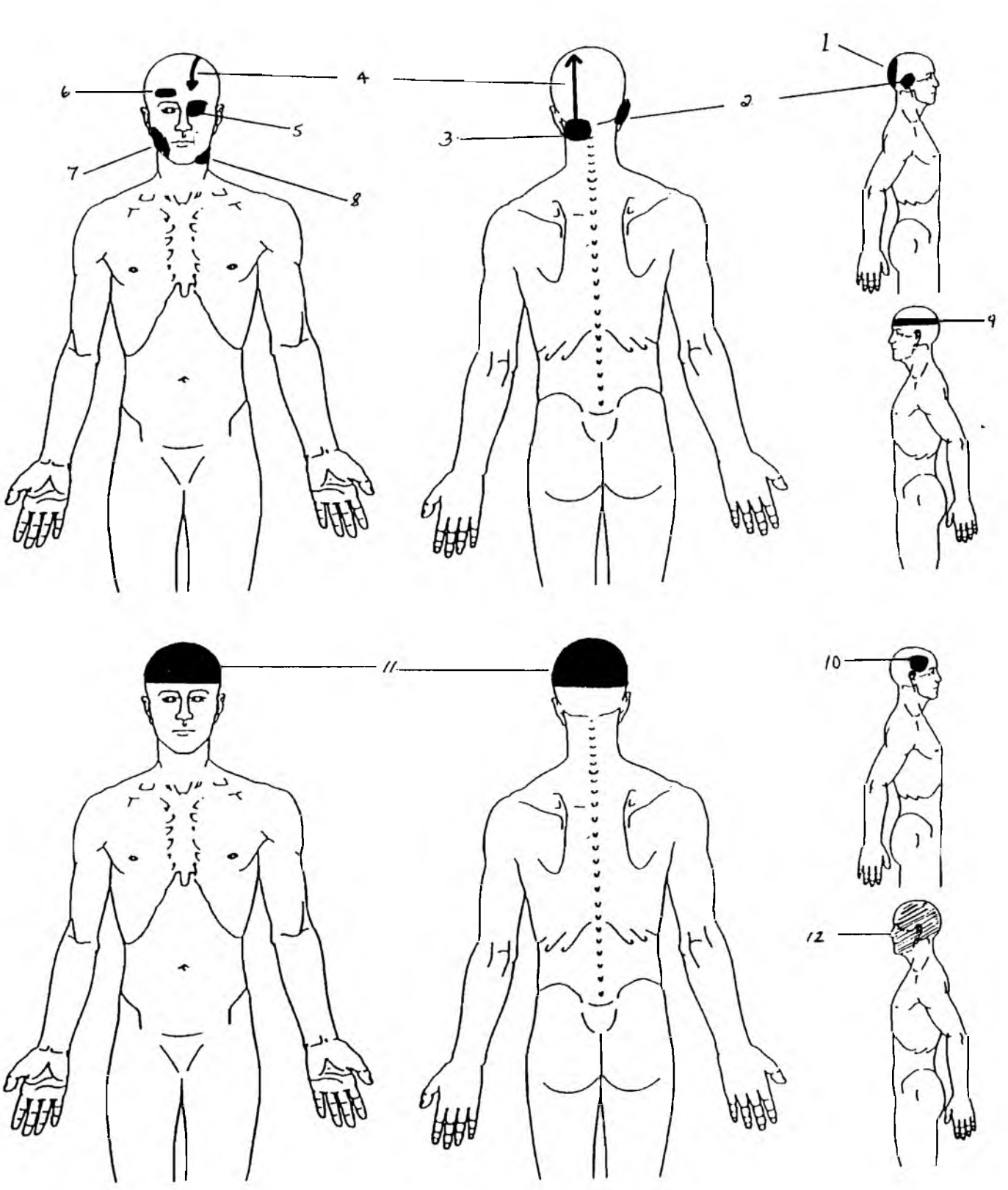

Fig 8: Cervical headache pain patterns SITE 1–C0/1, SITE 2–C0/1, SITE 3–C1/2, C2/3, SITE 4–C1/2, C2/3, SITE 5–C1/2, C2/3, SITE 6–C2/3, SITE 7–C1/2, C2/3, SITE 8–C2/3, SITE 9–C1/2, C2/3, SITE 10–C1/2, C2/3, SITE 11–C5/6 Discogenic, SITE 12–T4, Lumbar (From Bang M. From Kaiser Northern California region. Teleconference: Sink or Swim–streamlining the initial evaluation, 1996)

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