MANUAL THERAPY of HEADACHES of SPINAL ORIGIN:
An investigation of etiology, mechanisms, diagnosis and treatment

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I have been privileged to work with many superb researchers outside my own profession. These individuals have eschewed the old conflicts between chiropractors and other biomedical professionals and have welcomed me into their midst. Dr.'s Barry Sessle and James Hu have provided me with a truly unique opportunity to pursue the basic science studies which I always knew were crucial to the elucidation of the importance of the spine in musculoskeletal medicine. Dr. Gwen Jansz has collaborated with me on my current work, a randomized clinical trial of chiropractic and medical treatments for tension-type headache. The generosity, collegiality and friendship of these colleagues have been outstanding.

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Finally, I extend my eternal gratitude to my family, my mother and my sons and, particularly, my late father, whose example I always follow and to whom this thesis is dedicated.
ABSTRACT:

This thesis has proposed the following: "that mechanical disorders of the upper cervical spine are an important cause of headaches".

Four hypotheses were developed to investigate this thesis.

1. Upper cervical spine dysfunction plays an important role in headache etiology.
2. The importance of this role is more or less, depending on the type of headache.
3. Cervical dysfunction can be assessed and characterized by a range of standard clinical methodologies employed in manual therapy.
4. The cervical dysfunction involved in these types of headaches is treatable by spinal manipulative therapy and this results in clinical benefit for patients with these conditions.

Four areas of evidence from my published work have been summarized to support this thesis.

A. An anaesthetized animal model of upper cervical deep somatic inflammation has provided pathophysiologic responses which could be the origins of headaches of cervical origin, particularly the type currently known as "cervicogenic headache".

B. Clinical descriptions have been redefined to reveal common features which could be grouped together and attributed to pathologies or dysfunctions in the regions investigated in the animal model.

C. A new form of questionnaire was developed which produced evidence of an association between headache and neck pathology or dysfunction.
D. An extensive literature review revealed reported associations between headache and neck pathology in diverse works, particularly those involving manual assessment and therapeutic procedures in the cervical spine.

The evidence from such diverse sources strongly supports the hypotheses developed here and my thesis, that mechanical disorders of the upper cervical spine are an important cause of headache.
I. INTRODUCTION:

Painful conditions related to the cervical spine are the second largest group of complaints which present to chiropractors [1-3]. These conditions can be categorized as: 1) neck pain only, 2) neck and arm pain, and, 3) various types of headaches (HA). The mechanisms of pain generation in these categories include local tissue injury and inflammation, neuropathic pain and referred pain from the deep somatic structures in this region.

The etiologies of these conditions span a wide spectrum from relatively benign causes to seriously traumatic, and clearly pathologic, causes. The benign mechanical causes include postural strain, stress-related strain, work-related injury and external traumata. Sources of traumata extend from simple mechanical strains through sport-related injuries to motor vehicle accident-induced whiplash, with or without head concussion. The differential diagnosis of these pain syndromes should always consider organic pathology of the musculoskeletal, cardiovascular and nervous systems.

Any investigation of pain related to the cervical spine and its potential relation to head pain, requires knowledge drawn from many sources, and a holistic approach such as the "biopsychosocial model [4] is useful. Basic sciences provide the necessary anatomical, physiologic and biomechanical foundations. Clinical epidemiology defines population issues such as incidence and prevalence as well as associated societal, occupational and environmental co-factors. Clinical sciences define the various syndromes of interest, their associated symptoms, their methods of clinical assessment and the best methods of treatment. The psychosocial disciplines provide a basis for humanistic care, recognizing that the best practitioners do not merely care for cervical spine-related pains, but for the people suffering with these problems.

The foregoing discussion illustrates the tableau from which the investigator interested in neck-related pain syndromes can draw inspiration for his or her specialized studies. Figure 1 shows the schema which I have adopted for my studies into the relationship
between headache and the cervical spine and its assessment and management by manual therapeutics.

My primary research interest has been headache pain associated with the cervical spine. *My primary thesis is that mechanical disorders (cervical dysfunctions) of the upper cervical spine are an important cause of headache.* The role of mechanical disorders of the cervical spine can be described as primary, secondary or unrelated [5]. Primary disorders are ones in which cervical dysfunction is the causal agent of the headache. In secondary disorders, cervical dysfunction is understood either as one of the perpetuating factors or as one of the somatic manifestations of other primary processes. In the third category (unrelated), cervical dysfunction plays no role. I have attempted to elucidate, from a variety of sources, the precise relationships between upper cervical joint dysfunction and headache in the primary and secondary categories.
**Hypotheses:**

My thesis is supported by the results of testing four hypotheses:

1. Upper cervical spine dysfunction plays an important role in headache etiology.
2. The importance of this role is more or less, depending on the type of headache.
3. Cervical dysfunction can be assessed and characterized by a range of standard clinical methodologies employed in manual therapy.
4. The cervical dysfunction involved in these types of headaches is treatable by spinal manipulative therapy and this results in clinical benefit for patients with these conditions.

1. I first wished to investigate the underlying physiologic and pathophysiologic mechanisms of upper cervical spinal pain and the motor and sensory responses it evokes. In collaboration with Dr's. Barry Sessle and Jim Hu, as a clinical consultant, we developed an animal model of cervicogenic headache [6-9, 10-14]. This work will be described in the section on “Animal Model of Cervicogenic Headache”. I have also proposed, in numerous articles and text chapters, a set of mechanisms of muscular, joint and neural dysfunction in the upper cervical spine and the relation of these to headaches. This work will be discussed under “Mechanisms of Cervicogenic Headache”.

2. Applying the pathophysiologic mechanisms which I have proposed to the clinical situation rationalizes manual therapy. This part of my work involved methodologies of clinical description and clinical assessment. Much of my work has been to develop and refine many of these methodologies. This work appears in [15-19]. I have also contributed to the debate as chief editor of two texts on the upper cervical spine which included contributions from leading experts on the nature and treatment of these types of headache [20,21].
One of my major propositions in these publications is the need for a broad perspective regarding CH, in contrast to the narrower perspective advanced by other groups who use this term [22-26]. This work is summarized in the section of this thesis on “Clinical Mechanisms in Headaches of Cervical Origin”.

3. The next stage of my work investigated methods of clinical assessment of neck pain and CH [27]. This required the development of several new instruments and methods, since cervical dysfunction had typically not been incorporated into the methodologies of medical diagnostics. In this area, I developed the first instrument for assessing self-rated disability due to neck pain: the Neck Disability Index (NDI) [28-31]. Virtually all of the subjects included in these studies have suffered with headaches. In fact, the item “headache” has consistently been the highest scored one, often surpassing “neck pain” itself. This work is summarized in “Manual Therapy Assessment of HCO”.

4. The final component of the evidence supporting my thesis derives from an analysis of the results of studies of therapeutic interventions in the cervical spine for headache. This involved reviewing the literature on all clinical trials employing such methods [16-19, 32], conducting a survey of chiropractic specialists’ best-practices [33] and, most recently, by initiating a randomized, placebo-controlled clinical trial to investigate the effect of chiropractic treatment of tension-type headache. This work is summarized in “The Treatment of HCO by Manual Therapeutics”.

**Approach to a thesis:**

This thesis will be organized according to the areas outlined above, rather than as a chronologic review of the studies included. This approach is preferred because, as with most studies extending over several years, the individual investigations following a specific theme did not follow strict chronologic order.
Prior to a review of the studies, the epidemiology of tension-type and cervicogenic headaches will be reviewed in order to inform the general reader of the extent of these conditions. I will then discuss the pathophysiology of cranio-cervical dysfunction and review basic science studies we conducted which explored the pathophysiology of upper cervical-evoked cranial pain and muscular reactions in an animal model. I will then discuss my work on the clinical features of upper cervical dysfunction in headache patients. Finally, I will discuss my work on the clinical efficacy of spinal manipulative therapy in the treatment of headache.
II. THE EPIDEMIOLOGY OF TENSION-TYPE AND CERVICOGENIC HEADACHES

This section of the thesis derives from work published in [20; and Vernon et al. Clinical Trial Proposal]

II.A TENSION-TYPE HEADACHE (TTH)

Tension-type headache (TTH) is the most prevalent form of benign, primary headache [34-37]. The terminology of this category of headache has evolved from the earliest classification of the NIH Ad Hoc Committee [38]. This type of headache has been called "tension headache" as well as "muscle contraction headache". The most recent definitions derive from the Classification of the International Headache Society (IHS) [39], where the term "tension-type headache" (TTH) was proposed and was described as: "a bilateral headache of mild-to-moderate intensity experienced with an aching, tightening or pressing quality of pain which may last from 30 minutes to 7 days and which is not accompanied by nausea or vomiting and in which only one of photo-or phonophobia may be experienced" [39].

The IHS defined two forms of TTH, "episodic" and "chronic". The distinction between the two forms is based solely on the frequency of headache days. In "episodic" TTH (ETTH), headaches are experienced in no more than 180 days per year, while in "chronic" TTH (CTTH), headaches (HA's) occur more than 180 days per year.

IIA.1 EPIDEMIOLOGY OF TTH

IIA.1.i Prevalence

A small number of population-based studies on TTH exists, the most recent of these reporting on Canadian [40], US [41], Danish [42], German [43] and Finnish [44] populations. These studies have employed various survey methods, including
telephone interviews [40, 41], mail surveys [43, 44] and subject interviews [42]. All but one [42] of the most recent of these studies have employed the IHS criteria for TTH described above. With one exception [42], these studies have involved large, randomly selected samples with good response rates (see Table 1).

<table>
<thead>
<tr>
<th>Authors</th>
<th>Locale</th>
<th>Survey Method</th>
<th>Sample Size</th>
<th>Prevalence Rate</th>
</tr>
</thead>
<tbody>
<tr>
<td>Pryse-Phillips et al., 1992 [40]</td>
<td>Canada</td>
<td>telephone</td>
<td>2,905</td>
<td>Lifetime prev. for HA = 58%*; for TTH = 21%</td>
</tr>
<tr>
<td>Honskaalo et al., 1993 [44]</td>
<td>Finland</td>
<td>mail</td>
<td>22,809</td>
<td>Prev. for once weekly TTH = 7-17%</td>
</tr>
<tr>
<td>Gobel et al., 1994 [43]</td>
<td>Germany</td>
<td>mail</td>
<td>5,000</td>
<td>Lifetime prev. for TTH = 38.3%</td>
</tr>
<tr>
<td>Rasmussen et al. 1995 [42]</td>
<td>Denmark</td>
<td>interview</td>
<td>1,000</td>
<td>Lifetime prev. for all TTH = 66%; for once-weekly TTH = 20-30%</td>
</tr>
<tr>
<td>Schwartz et al., 1997 [41]</td>
<td>America</td>
<td>telephone</td>
<td>13,345</td>
<td>Lifetime prev. for TTH = 38.3%</td>
</tr>
</tbody>
</table>

(* % = percent of population reporting having headache)

Several studies reported a slight predilection in females for TTH [40-42, 44]; but Gobel et al.[43] failed to confirm this. Similarly, some studies reported an increased prevalence in those aged 25-45 [40], while others found no increasing trend with age [41-44]. Schwartz et al. reported higher prevalence rates in whites and those with higher education.
In summary, the reported prevalence rates vary from approximately 10 - 65%, depending on the classification, the description and the severity of headache features. In general, these rates derive from large, randomly selected, well-representative samples. The consistency of findings, particularly among urban populations, leads to the conclusion that slightly more than one third of the adult population suffers from this problem.

IIA.1.ii Frequency of Headaches

The findings of several studies [40, 43, 44] agree that only 3% of TTH sufferers experience headaches more than 180 times yearly (in other words, are classified as CTTH). Rasmussen et al. [42] reported that the percentage who suffer TTH more than once weekly (over 52/yr) is 20-30%. Gobel et al. [43] reported that only 28% suffer TTH more than 36 times yearly. Honskalo et al. [44] reported that yearly frequency increased with age, varying from 7.3 and 13.5 per year for males and females aged below 30 years to 27.7 per year for both genders older than 65 years. This increase appears to be linear from one decade of adulthood to the next.

IIA.1.iii Severity of Headaches

TTH is, by definition, a milder, less severely painful form of headache than migraine or other primary categories (cluster headache, cervicogenic headache). Gobel et al. [43] reported that headache severity varied from mild, moderate to severe in 22, 68 and 10% of TTH sufferers, respectively. Rasmussen et al. [42] reported that 58% of their TTH sample had “mild, infrequent conditions.” Including these subjects in their study produced the highest reported prevalence rate. Schwartz et al. [41] used a pain-rating scale and reported mean (+/- 1SD) headache severities of 4.98 (1.99)/10 for ETTH and 5.55 (2.10)/10 for CTTH. In their sample, 62% were classed as moderately severe, while 25% were mild and 13% severe, closely matching Gobel et al.’s figures.
In summary, it appears that at least 50% of TTH sufferers rate their headaches as moderately or severely painful.

IIA.1.iv Psychosocial Impact

The range of psychosocial impacts of TTH includes disturbances of daily activities, disturbed quality of life, workdays lost or disturbed as well as the costs of these disruptions. In Pryse-Phillips et al.’s study [40], 44% of TTH sufferers reported a significant reduction in their activities of daily living (ADL) due to headache (with a mean duration of 18 hours of disrupted ADL per year) while 8% reported taking at least one day off work from their last headache. In a follow-up study of the Canadian sample, Edmeads et al. [45] reported that a high proportion of TTH sufferers endured adverse effects on their relationships with 89% reporting adverse effects within the family, 71% with friends and colleagues and 80% with their physical activities.

According to Rasmussen et al. [42] 60% of TTH sufferers reported an adverse effect on work capacities, with 12% missing one or more work days per year. They estimated a work-loss rate of 820 days/1000 people per year for a total of 2,300,000 annual work days lost in Danish society.

In Schwartz et al.’s US sample [41], 8.3% of subjects reported lost work days (with a mean of 9 days per person per year) while 43.6% reported “reduced-effectiveness” days (mean: 5 days).

IIA.2 Treatment of TTH

Treatment of TTH varies widely within both medical and non-medical circles. Also, the patterns of health care utilization by TTH sufferers are variable. According to Edmeads et al. [45], 45% of their sample of TTH sufferers had consulted with a physician and, of these, 32% were subsequently referred to a specialist.
In contrast, the Danish results reported by Rasmussen et al. [42] are much lower, with only 14% attending a physician and 4% receiving specialist referrals.

IIA.2.1 Medical Approaches:

Medications remain the mainstay of the medical approach to managing TTH. Two basic approaches exist, namely, symptom relief or abortive therapy (given every time a HA occurs) and prophylactic therapy (given on a regular basis for HA prevention). Edmeads et al. [45] reported that 90% of their TTH sample used over-the-counter (OTC) medications which typically included analgesic or anti-inflammatory drugs. Twenty-four percent (24%) used prescription medications, which typically included stronger doses of the OTC drugs as well as muscle relaxants and combination drugs. Only 3% were on prophylactic medications, which included low-level anti-depressants and serotonin-enhancing agents (SSRI’s).

Wober-Bingol et al. [46] reported on 210 headache sufferers referred to two Austrian specialist centers. Thirty-nine percent (39%) of subjects were on some form of prophylactic regimen; however, in only 9% was this a medication regimen. Of the 19 cases who were receiving anti-depressants, 11 reported them to be effective.

IIA.2.ii Non-medical approaches:

There is considerable variety in the non-medical approaches to the treatment of TTH. Edmeads et al. [45] reported that 34% of their headache sample has used non-medical forms of treatment, although these may have included a broad spectrum from psychological-based to physical therapies to non-pharmacologic remedies.
Rasmussen et al. [42] reported that 5-8% of their headache respondents had sought care from physiotherapists or chiropractors. Wober-Bingol et al.'s [46] study of specialist-level patients reported that 29% had received prophylactic physiotherapy with only a quarter reporting that it was effective.

In an earlier study, Graff-Radford et al. [47] reported that 35% of their specialist-level headache subjects (U.S. sample) had previously received chiropractic treatment.

Several studies from chiropractic college clinics (see [1]) estimate between 5-10% of patients present to chiropractors with a primary complaint of headache. According to Kellner and Wellman [2], 10% of the chiropractic patients in their small survey of alternative health practitioners reported that headache was their primary complaint. Interestingly, this figure was the largest amongst the five complementary/alternative medicine (CAM) practitioners surveyed and was considerably larger than the percentage reported for the family physicians who were also surveyed.

Most recently, Hurwitz et al. [3] reported that 2.3% of US chiropractors' patients present primarily with headache, although 13.5% presented with neck pain which may include headache.

II B. CERVICOGENIC HEADACHE (CH)

II B.1 Description and Epidemiology

Only recently, the role of the cervical spine in headache has become increasingly well acknowledged. Despite the wealth of writing in chiropractic, osteopathic, physiotherapeutic and manual medical circles (see reviews in [5, 20, 21]), as recently as the late 1980’s, cervicogenic headache was poorly recognized in orthodox circles. The diagnostic label "cervicogenic headache" (CH) was coined in 1983 by Sjaastad [22-25],
although numerous other labels existed such as “headache of cervical origin”, “cervical headache”, vertebrogenic headache” and “spondylitic headache”.

As Pollman et al. [25] and Vincent and Luna [26] have recently noted, many older publications dating from 1926, (with the cases of Barre), to those of Bartschi-Rochaix [48] and Hunter and Mayfield [49] in 1949, and Campbell and Parsons [50] in 1944 and 1954 likely involved cases of putative cervicogenic headache. From 1983-1987 Sjaastad and his colleagues [22-24] as well as other European investigators [51, 52] expanded on the topic and provided sufficient basis for the International Headache Society Classification [39] to include “cervicogenic headache” as a distinct entity (category number 11). The criteria for this category of headache, shown in Table 2, were broader and more inclusive than Sjaastad’s original description, as this was challenged by some (Vernon [16-19], Bogduk [53-55]) as being so restrictive as to relegate this headache type to a rare variant. Recognizing this situation, Bogduk, has proposed a new definition of CH, which has been adopted by the North American Cervicogenic Headache Society:

“Referred pain perceived in any region of the head caused by a primary nociceptive source in the musculoskeletal tissues innervated by cervical nerves” (Bogduk, personal communication).

Furthermore, a recent classification from the International Association for the Study of Pain (IASP) [56] adds the additional criterion of successful abolition of the headache by anaesthetic blockade of the appropriate upper cervical nerves as a diagnostic factor. The confusion in applying these various classification or diagnostic schemes is exemplified in two recent studies. Persson and Carlsson [57] reported on 81 subjects with cervical and/or radicular arm pain, 67% of whom also reported experiencing headache. When they applied the IHS criteria, 81% of these cases were classified as having CH. When using Sjaastad’s 1990 criteria [58], only 28% received the same diagnosis. They concluded that “cervical headache has no unique features that differ
from those of tension-type headache, and it would perhaps be appropriate that the diagnosis of CH is incorporated in the diagnosis of TTH" ([57] pg. 223).

Leone et al. [59] applied all of the differing classification schemes to 940 primary headache cases and found very few who manifested any unique features of CH which could not be subsumed in either TTH or migraine diagnoses. In fact, the boundaries between cervicogenic headache, tension-type and migraine headaches were and continue to be quite blurred, particularly on the issue of bilaterality. Another limitation which confuses the diagnostic picture is that many of the features of CH cited in Table 2 are physical signs which the astute clinician must obtain from careful physical and radiologic examinations [58].

If the headache pain pattern is not so distinct as to immediately point to the diagnosis (as in cluster headache or, perhaps, migraine with aura) and if the less astute clinician does not include examination of the neck in his or her assessment, then those features critical to the CH diagnosis could be absent from the clinical assessment. An erroneous diagnosis of tension-type or migraine without aura might then be made, and the opportunity to address the possible cervicogenic etiology of the headache will be lost.

In this way, the prevalence of CH is very likely underestimated.

Table 2: Features of Cervicogenic Headache

1. Unilateral pain without sideshift.
2. Reduced range of neck motion.
3. Provocation of pain by neck movements, awkward neck positions or suboccipital pressure.
4. Associated neck or non-radicular shoulder/arm pain.
5. Pain radiates from neck to anterior head (particularly frontal & ocular).
6. Moderate pain intensity (no throbbing pain).
7. Varying durations of pain including continuous fluctuating pain.
8. Minor associated symptoms and signs (non-obligatory) including:

- nausea, vomiting, dizziness
- photo-and phonophobia
- difficulty swallowing
- "blurred vision" in ipsilateral eye.

(Modified from [39]).

Given the diagnostic confusion cited above, it is understandable that studies of the prevalence of CH (in either the general population or in headache samples) are fraught with difficulties and variations. A wide range of frequencies is reported in the literature. Leone et al. [59] found CH in only 0.7% of their headache sample, while Pfaffenrath and Kaube [60] found CH in 13.8% of their larger headache sample, with 6% suffering exclusively from CH. Using the IHS criteria along with careful questioning about cervical dysfunction, Nilsson [61] found an annual prevalence of CH in a Scandinavian population to be approximately 15%. This is roughly at the lower end of prevalence estimates for TTH [41, 42] and is identical to the prevalence of migraine headaches cited in these reports. This is far from the rare variant headache suggested by the work of the early 1980's.

In conclusion here, TTH and CH are highly prevalent clinical conditions which are associated with significant impact on the daily lives of sufferers and on the health care system. While the IHS Classification provided a needed step forward in clarifying the characteristics of these two types of headaches, investigators have since found that they both share many features which make accurate diagnosis more difficult than was originally thought. One of these areas is the role of the cervical spinal dysfunction, as proposed in the hypotheses of this thesis. In order to further investigate this issue, the pathophysiological mechanisms of referred pain from the upper cervical spine become an important area of interest.
III PATHOPHYSIOLOGY OF HEADACHE OF CERVICAL ORIGIN

IIIA Mechanisms of Cervicogenic Headache

According to Bogduk the cervical source of headache may lie in any of the structures innervated by the first three cervical nerves [53, 54]. As such, a thorough knowledge of upper cervical innervation patterns is useful in understanding the pathophysiology of the condition. Before considering these patterns, it is convenient to categorize these somatic tissues according to location, as follows:

1. **Extrasegmental**

   Long occipito-thoracic muscles lie relatively superficial in the neck and include trapezius, sternocleidomastoid and splenius cervicus. The occipito-frontalis muscle may also be related to cranial pain. Other important structures lying extrasegmentally include the vertebral artery (implicated in the Barre-Lieou syndrome [5, 20, 62] and vertebrobasilar ischemic syndrome) as well as the ascending sympathetic chain and superior cervical ganglion. Older theories implicated compression or irritation of these sympathetic structures in generating cranial pain and cranial vasomotor dysregulation. These theories have fallen out of favour.

2. **Intersegmental**

   These structures include the classic spinal joints and deep spinal muscles, i.e. the semispinalis occiput and cervicalis, multifidus and suboccipital muscles (posterior, lateral and anterior). There is no intervertebral disc between C0-1 and C1-C2. The suboccipital articulations include the bilateral atlanto-occipital joints, the bilateral atlantoaxial joints, the atlanto-dental joint, Joints of Luschka and the C2-C3 intervertebral disc. The suboccipital region contains a large number of specialized ligamentous structures (see: Kapandji [63] for an excellent review).
3. **Intrasegmental**

This category involves the neural and vascular structures contained in the intervertebral environment of C1-C2 and the intervertebral foramina of C2-C3. Specifically, the anterior and posterior rami of C1 and C2, the C2 dorsal root ganglion as well as the C3 posterior nerve root. Bogduk’s reviews of upper cervical anatomy [53, 54] are particularly extensive.

4. **Infrasegmental**

This category includes the spinal cord and lower brainstem. Of particular importance is the spinal tract of the trigeminal nerve which consists of descending afferents from the trigeminal sensory ganglion which terminate as far caudally as C3 in the spinal nucleus of the V nerve. The descending tract contains three components: the pars oralis (upper), pars intermedialis and the pars or subnucleus caudalis (lowest). These afferent fibres terminate on the same second order neurons as do the afferents from the upper three cervical roots. The second order neurons form a continuous column of cells called the “trigemino-cervical nucleus” by Bogduk [64] and the “medullary dorsal horn” by Gobel [65] This “neural anastomosis” of converging afferents is the fundamental neuroanatomical basis by which painful structures in the upper cervical region might generate referred pain to the cranium (see below).

**IIIA.1 Innervation patterns:**

C1 and C2 anterior ramus:

- deep anterior suboccipital muscles
- posterior dura
- posterior cranial vessels
the C2 anterior ramus contains the sensory fibres of the hypoglossal nerve which run in the ansa hypoglossus.

C1 posterior ramus:

The C1 posterior ramus is very small, but its existence was shown by Kerr [70].

The C1 anterior ramus:

superior oblique muscle

C2 posterior ramus:

the C2 posterior ramus has two branches - the medial branch becomes the lesser occipital nerve and innervates Rectus Capitus Posterior Major & Minor and the medial C1-C2 joint and ligaments.

the lateral branch is the largest posterior ramus of the spine and is called the Greater Occipital Nerve (GON). The GON gives off an articular branch to the lateral C1-C2 joint as well as a muscular branch to the inferior oblique. It then courses posteriorly and superior to pierce between the semispinalis capitus and trapezius muscle insertions where it becomes cutaneous and innervates the skin of the posterior skull till the midline.

The C3 posterior ramus has been referred to as the “third occipital nerve” by Bogduk [53, 55] and it innervates the C2-C3 Z-joint and deep muscles as well as providing a recurrent meningeal nerve which innervates the C2-C3 IVD.
IIIB  An Animal Model of Cervicogenic Headache:

In this next section, a series of studies examining brainstem/spinal cord mechanisms of head/face/neck pain will be discussed. In 1988, I began a collaboration with Dr. Barry Sessle of the University of Toronto, Faculty of Dentistry, Oral Physiology Laboratory. Sessle and his colleagues had already conducted investigations into the input of upper cervical afferents, predominantly through C2, into trigeminal brainstem mechanisms as part of their extensive mapping of the sensory organization of the medullary dorsal horn. This term, coined by Gobel [65], was used to refer to the extension of the spinal tract and nucleus of the fifth cranial nerve (V), particularly the subnucleus caudalis, into the upper cervical spinal cord. They were developing the work of Kerr [66] and Bogduk [54, 64], among others, which had established the clinical implications of upper cervical pain being referred to the cranial region and perceived as headache.

While their previous work (see below) had concentrated on oro-facial sources and mechanisms of pain, they welcomed the opportunity to expand their research into the upper cervical region. They also appreciated the importance, to chiropractic science, of developing an animal model of what (as described above) had come to be termed “cervicogenic headache”.

The first group of studies (1981 - 1992) that I will review represent earlier work from the University of Toronto, Faculty of Dentistry, Oral Physiology Laboratory of Dr. Barry Sessle and his co-investigator Dr. James Hu. I was not involved in these studies; however, they form the foundation for the later work in which I was involved. These studies were mainly concerned with mechanisms subserving facial, oral and dental pain, with some emphasis on deep somatic pain from the tongue and temporomandibular joint (TMJ).

The later work (1993-2001) includes studies of deep somatic pain from TMJ and upper cervical spinal tissues. The methodologies of these studies, while briefly and
generically reviewed below, are presented in the published works, and the reader is referred directly to these publications (Appendix 1) for much greater detail. All these studies were conducted with the full approval of the University of Toronto Animal Care Committee and conformed to the guidelines for pain studies in animals from the International Association for the Study of Pain [67] and the Ontario Act for Animal Research.

In 1981, Hu et al. [68] reported on the functional properties of feline trigeminal subnucleus caudalis neurones in response to both noxious and non-noxious stimuli in the oro-facial region in anaesthetized cats. Populations of low threshold mechanoreceptors (LTM), nociceptor-specific (NS) and wide dynamic range (WDR) neurones were located. WDR/NS neurones (as well as some LTM's) responded to stimulation of the hypoglossal and superior lingual nerve (all neurones had oro-facial cutaneous RF's). One third of all caudalis neurones recorded also responded to noxious tooth pulp stimulation. A minority of these caudalis neurons (from 12-15%) were found to project to the thalamus, while 33 - 50% projected to the subnucleus oralis of V, another part of the trigeminal system.

Subsequently, Sessle et al. [69] demonstrated that stimulation of the Nucleus Raphe Magnus (NRM) and the Periaqueductal Grey Matter (PAG), two nuclei known to be involved in antinociceptive mechanisms at the spinal cord level, produced brief periods of inhibition of caudalis neuronal firing. Additionally, peripheral conditioning stimuli in the oro-facial region (and even in the forelimb) induced similar inhibitory effects.

Sessle et al. [70] investigated the wide range of tissue sources providing inputs into the feline subnucleus caudalis itself. For LTM, WDR and NS neurones respectively, the percentage of neurones (numbers tested in parentheses) which responded to electrical stimulation is shown in Table 3:
Table 3: Sources of afferent input to the subnucleus caudalis

<table>
<thead>
<tr>
<th>TISSUE</th>
<th>LTM (%/#)</th>
<th>WDR (%/#)</th>
<th>NS (%/#)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Skin</td>
<td>100 (209)</td>
<td>100 (19)</td>
<td>100 (31)</td>
</tr>
<tr>
<td>Superior Lingual N.</td>
<td>28 (160)</td>
<td>68 (19)</td>
<td>56 (34)</td>
</tr>
<tr>
<td>Tooth pulp</td>
<td>37 (173)</td>
<td>84 (19)</td>
<td>50 (36)</td>
</tr>
<tr>
<td>C2/C3 mixed nerve</td>
<td>17 (152)</td>
<td>61 (18)</td>
<td>46 (33)</td>
</tr>
<tr>
<td>C3 muscle nerve</td>
<td>25 (32)</td>
<td>100 (3)</td>
<td>29 (7)</td>
</tr>
<tr>
<td>Hypoglossal N.</td>
<td>21 (166)</td>
<td>58 (19)</td>
<td>52 (33)</td>
</tr>
<tr>
<td>Temporalis or masseter muscle</td>
<td>7 (123)</td>
<td>25 (12)</td>
<td>21 (28)</td>
</tr>
</tbody>
</table>

% = percent of neurons responding; # = total number of neurons mapped; LTM = low threshold mechanical; WDR = wide dynamic range; NS = nociceptive specific.

These data demonstrate the wide range of inputs into these multi-convergent cells including skin, deep somatic and visceral sources. The high prevalence of C3 input onto WDR cells confirms their importance in potentially generating referred head or neck pain.

Amano et al. [71] then highlighted the contribution of muscle afferents from the jaw and tongue in the feline model. Muscle afferents stimulated by chemical or electrical means activated predominantly WDR and NS caudalis neurones. The majority of these afferents were regarded as nociceptive by virtue of their long latency/high threshold responses and the predominance of small fibers in the muscle nerves tested. Many of these neurones also responded to tooth pulp stimulation. This study provided evidence in the subnucleus caudalis of inputs from both cutaneous and deep (muscular) structures and illustrated central convergence – a form of functional organization - of these afferents which might provide a basis for referred craniofacial pain.

Broton et al. [72] extended this work to include articular afferents from the TMJ in the anaesthetized cat. Again, WDR and NS neurons were especially responsive to algesic and electrical stimulation of the TMJ capsule. These afferents were likely nociceptive.
for the same reasons as described above for Amano et al. (36). A picture of multi-
convergence within the subnucleus caudalis was now emerging involving the
organization of cutaneous oro-facial inputs with those from tooth pulp, facial/jaw
muscles, tongue and the TMJ.

Hu [73] described similar features in the rat subnucleus caudalis. Overlap of cutaneous
oro-facial and deep (hypoglossal nerve) inputs was a feature of the WDR and NS
populations. Hu demonstrated that the evoked responses in the rat subnucleus
caudalis could be attenuated by tail and forepaw noxious stimulation, thereby
demonstrating that these neurones were subject to diffuse noxious inhibitory controls
(DNIC) [74], further evidence of their role in craniofacial nociceptive processing.

This investigation was followed by a study of changes in the response properties of
single caudalis neurones after algesic stimulation (via 5% mustard oil) of the masseter
muscle [75]. Algesic (vs control) stimulation resulted in long-lasting (20-30 min) but
reversible expansion of the cutaneous receptive fields, increase in spontaneous firing
and decrease in the threshold to firing.

I joined this group in 1988 with the intent of developing an animal model of spinal
inflammatory pain. Given their expertise in the cranio-facial area (both theoretically and
technically), a collaboration involving a model of upper cervical deep tissue
inflammatory irritation was a natural and easy extension of their previous work. In
several instances, we were able to repeat previous experiments with the simple
modification of the site of inflammatory irritant injection. In several other instances,
important technical modifications were required and occupied considerable time and
resources of the group. Interestingly, these latter experiments proved to be very fruitful
and served to advance the group’s experimental repertoire.
In our first study, Yu, Hu, Sessle and Vernon (unpublished), sought to identify the input characteristics of the entire medullary dorsal horn, i.e. from caudalis subnucleus to the C2 level in the rat. We found noci-responsive neurones were somatotopically organized in a medial-to-lateral direction as one moved caudally within the nucleus. At 2mm caudal to the obex (caudalis) neurons received only trigeminal (V) input, while at 3mm (lower caudalis), 8% of neurons recorded from received C2 input. Table 4 displays data on the proportions of caudalis/upper cervical neurones from 4mm below the obex receiving convergent input from various levels tissue sources.

<table>
<thead>
<tr>
<th>LEVEL</th>
<th>C2 only</th>
<th>V only</th>
<th>C and V</th>
<th>Cervical</th>
<th>C and L</th>
<th>L only</th>
</tr>
</thead>
<tbody>
<tr>
<td>C1(4mm)</td>
<td>29%</td>
<td>57</td>
<td>29</td>
<td>14</td>
<td>0</td>
<td>0</td>
</tr>
<tr>
<td>C1(5mm)</td>
<td>45</td>
<td>41</td>
<td>35</td>
<td>21</td>
<td>0</td>
<td>0</td>
</tr>
<tr>
<td>C2(6mm)</td>
<td>41</td>
<td>23</td>
<td>41</td>
<td>18</td>
<td>9</td>
<td>9</td>
</tr>
<tr>
<td>C2(8mm)</td>
<td>53</td>
<td>0</td>
<td>12</td>
<td>50</td>
<td>19</td>
<td>19</td>
</tr>
<tr>
<td>C2(9mm)</td>
<td>19</td>
<td>0</td>
<td>0</td>
<td>35</td>
<td>41</td>
<td>24</td>
</tr>
</tbody>
</table>

Table 4. Percentage of neurones receiving somatic input from C2 only, Trigeminal (V) input only, combined C2/V, cervical, cervical and limb and limb only sources.

As such, no cervical inputs were found in the mid-caudalis and a minor amount of C2 input was found in the lower caudalis. C2 and V inputs were found extensively, but exclusively at the C1 level, while, at the C2 level, this pattern continued but also included minor lower cervical and limb inputs. Below C2, C2 inputs were still found, V inputs were decreasing, while cervical and limb inputs predominated. These data identified the C1-C2 spinal cord region as one which is somatotopically organized and with extensive C2 and trigeminal convergence, with a significant proportion of these neurones responding to both C2 and V input. We also found that no cervical units responded to hypoglossal nerve stimulation while many V units did.
Yu et al. [76] explored the differential effects of cutaneous vs deep (tongue muscle) sources of input on neuroplasticity of caudalis neurones. They found that mustard oil injection in the tongue induced expansion of both cutaneous and deep receptive fields, while cutaneous (facial) injection induced expansion of cutaneous RF's only. They concluded that "deep inputs are especially effective in inducing neuroplastic changes" [76] (pg. 1704). This would appear to be due to the finding that deep inputs are effective in inducing expansion of both deep and cutaneous RF's, whereas cutaneous inputs result in RF expansion in cutaneous RF's only. This may explain the greater and longer lasting sensory disturbances in pain conditions involving deep tissues as compared to those involving only the skin.

In 1993, Hu et al. [6] published a study which has significant implications for chiropractors and others interested in spinal pain of mechanical origin. Notwithstanding the obvious interest of Sessle's group in cranio-facial/TMJ pain, almost all of the studies on deep pain mechanisms by other investigators in the field had involved the tissues of the peripheral limbs [77, 78]. Their work was crucial in elucidating many of the neurophysiologic mechanisms described above. Remarkably, however, virtually none of this work involved the deep tissues of the spine; this is in spite of the very high prevalence of mechanical spinal pain syndromes [79]. Gillette and his colleagues [80, 81] published studies on deep pain of lumbar spine origin, but no animal studies of deep pain mechanisms related to the cervical spine had been conducted. This includes the upper cervical region, the afferents which, as described above, are known to connect with the trigemino-cervical nucleus. As such, Hu et al.'s report on the response of neck and jaw muscles in rats in response to mustard oil injection in the deep upper cervical paraspinal muscles represented a significant and unique step forward in our understanding of spinal pain mechanisms. This was of particular relevance to mechanisms of cervico-cranial pain.
In this model, EMG recordings were made of two jaw muscles - digastric and masseter - and three neck/spinal muscles - the trapezius and bilateral rectus capitus posticus major. Injections were made percutaneously into the left deep paraspinal region around the C1-C2 level, typically in the deep, peri-articular fibers of the left rectus capitus posticus major and multifidus muscles. The inflammation induced by this injection was confirmed subsequently, both morphologically and histologically.

After first conducting paired trials of mineral oil (control) injection to confirm that serial injections did not volumetrically provoke any EMG increases, a series of 19 preparations were studied whereby a 20-minute baseline period was followed by a control injection of mineral oil. EMG recordings were made for a further 10 minutes, followed by an injection of either mineral oil or mustard oil (MO), with 30 - 40 minutes of further EMG recordings. The sites of injection and the magnitudes of EMG recordings for 9 rats are shown in Fig. 2, while the aggregate data for all 19 rats are shown in time series in Fig. 3. Several important points emerge:

![Figure 2. Location of left deep upper cervical inflammatory irritation by mustard oil injection. Ipsi.= ipsilateral; contr. = contralateral](image-url)
Figure 3. EMG responses in neck and TMJ muscles to inflammatory irritation of the left deep neck region. * p < .05, ** p < .01

1. All muscles recorded an early increase of EMG activity which was statistically significant compared to controls (black vs open circles in Fig. 3);

2. Irritation of a deep paraspinal source evoked very large increases of EMG activity in a superficial muscle, the trapezius, which did not receive injectate;
3. Activation of the contralateral rectus capitus posticus major (RCPMa) muscle occurred in every case, although to a lesser degree than on the ipsilateral side. While spread of the injectate to the opposite side may have occurred, Fig. 3 shows that this was the exception;

4. Irritation in a paraspinal site provoked activation of ipsilateral jaw muscles, although to a lesser degree. The duration of jaw muscle activation was well beyond the time frame for the clench reflex typically observed in any pain experiment;

5. Activation of the deep spinal muscles followed a biphasic course with an early response lasting for 5 - 8 min followed by a silent period of 15 - 20 min which was then followed by an increase in EMG activity. This biphasic activation is very reminiscent of the response reported to occur in the formalin model in awake rats [82].

These findings supported, for the first time, the notion that injury to the deep spinal tissues provokes both local and contralateral as well as "distal" muscular activation, likely as a protective response. These results also imply that reflex connections exist between upper cervical afferents and both local spinal and trigeminal motoneurones which subserve the muscular activity observed. In terms of chiropractic theory, these findings provide evidence of muscular hypertonicity induced by a form of painful spinal dysfunction ("subluxation inducing local and distal muscular reactions").

In 1995, Yu et al. [7] replicated the EMG study described above while injecting mustard oil into the rat TMJ. Of interest are the following findings, particularly as compared to the neck/EMG study (see Figure 4):

1. Ipsilateral digastric and masseter muscle activity increased significantly over control levels;
2. Only, slight, brief increases in contralateral masseter muscle EMG activity over control was found. This is in contrast to the larger, more prolonged responses in the contralateral RCPMa found in the neck/EMG model;

3. Only one phase of activation was noted in any of the muscles, lasting up to 10 min post-injection. This is in contrast to the biphasic pattern observed in the RCPMa muscles in the neck model;

4. No increase in trapezius or deep neck muscle EMG was found. This is in contrast to the finding of increased (albeit of a smaller magnitude) activity in the jaw muscles evoked by irritation of the deep neck muscles.

A comparison of the neck vs TMJ EMG studies reveals differences in the pattern of evoked muscle activity depending on the site of experimental inflammatory algesia. Responses in rats to TMJ injection appear to be more focal and confined to the jaw muscles on the lesioned side. No neck muscle activity is evoked. Irritation of the spinal (axial) tissues appears to evoke a more diffuse pattern involving contralateral segmental muscles, superficial regional muscles and, to at least some degree, activation of "distal" jaw muscles. One explanation for these differences may be a more diffuse divergence pattern of spinal nociceptive afferents and either their direct or indirect connections to the cervical and trigeminal motor nuclei.
In 1995, Hu et al. [10] reported on a similar EMG study using mustard oil applied to posterior meningeal/dural vascular tissues (see Figure 5). These tissues are exposed during preparation of the experiment because of the required removal of the posterior/vertex region of the skull. Mustard oil (MO)/mineral oil applications were made directly onto the dura and the sagittal sinus. Again, after a 20 minute baseline where no EMG activation occurred, mustard oil, but not mineral oil induced EMG activity.
increases in the left masseter muscle (small increases), left deep neck muscles (large increases, lasting for up to 40 minutes), the right deep neck muscles (moderate increases, lasting up to 35 min) and the left trapezius (large increases lasting up to 30 min).

![EMG responses to inflammatory irritation of the posterior meningeal tissues](image)

Figure 5. EMG responses to inflammatory irritation of the posterior meningeal tissues

These results confirm that posterior cranial structures, which are innervated by the C1 - C2 anterior primary rami, make similar synaptic convergence into the trigemino-cervical nucleus and provoke similar muscular responses as do the posterior deep muscles innervated by the posterior primary rami of C1-C2. These results support the notion
that both referred pain and "referred" reflex muscle spasm can arise from painful disorders of the meninges and dural vessels of the posterior cranium such as meningitis and, more importantly for chiropractors, migraine headache. The "subluxation"-type findings in the upper cervical spine which may be observed by clinicians on careful manual assessment of their patients [27] may, therefore be a manifestation of visceral-somatic reflex disturbances, making diagnosis and treatment of these disorders all the more challenging. To our knowledge, this study represented the first report of spinal somatic dysfunction (as spinal muscular hypertonicity) induced by a "visceral" irritative source, i.e. the classical visceral-somatic reflex disorder.

In related studies Angus-Leppan et al. [83] and Goadsby et al. [84] demonstrated that stimulation of the superior sagittal sinus and the occipital nerve (posterior ramus of C2) activate similar second order neurones in the dorsolateral area of the upper cervical cord and the subnucleus caudalis.

The recent work in Sessle's laboratory has concentrated on two areas: 1] the neurochemical mechanisms involved in deep pain from the TMJ and deep neck tissues, and, 2] single unit recording studies of upper cervical neurones in response to deep neck (as opposed to TMJ) experimental inflammatory pain.

In 1996, Hu et al. [13] replicated the 1993 neck/EMG study with a view to elucidate aspects of neurochemical involvement. The reader will recall that the response pattern of the deep neck muscles (bilaterally) was often biphasic, beginning with an initial elevation lasting about 10 min followed by a return to baseline and, after about 20 min, a return of elevated EMG activity. This second activation phase was proposed to be due to a combination of central sensitization of dorsal horn neurones and sustained tissue inflammation producing summation of enhanced C-fibre firing. We hypothesized that the middle phase might be produced as a result of active suppressive or inhibitory mechanisms evoked in response to the initial nociception. We further hypothesized that
this suppressive phase may be subserved by an opioid mechanism. This mechanism might have features similar to DNIC.

Previously, in 1994, Yu et al. [8] had demonstrated that injection of naloxone, an opioid antagonist, could produce a "rekindling" effect on the EMG output of TMJ-inflammation induced muscular activation. In order to confirm this hypothesis, we "manipulated" the paraspinal-inflammation model by injecting an opioid receptor antagonist, naloxone, within one to two minutes of the commencement of the second phase of EMG silence.

A dose-dependent naloxone-induced "rekindling" effect on the EMG recordings was demonstrated (see Figure 6). At the highest dose, injection of naloxone resulted in an average EMG increase of 83% of the original mustard-oil induced EMG activity. It appears that the normal physiologic response to injury involves mechanisms designed to suppress the initial protective muscular responses (perhaps as part of an overall antinociceptive response). However, in the presence of sufficient and persistent tissue irritation, this inhibitory process is overcome and the nociceptor-driven EMG activity is reactivated.
Yu et al. [11] explored the neurochemical mechanisms involved in mustard oil-induced central sensitization. The TMJ/EMG model was combined with injections of several blocking agents in the periphery, including lidocaine, a local anaesthetic, naloxone, and MK801, an NMDA-receptor blocker. They found that lidocaine significantly attenuated the EMG activation of the masseter and digastric muscles but not the plasma extravasation in the TMJ region. This confirms that the MO-induced EMG increase is reflex-based. Naloxone induced rekindling of the MO-induced EMG activity once suppression had occurred. Various dosages of MK801 (locations: intracerebroventricular, intravenous and directly into the TMJ; dosages: ranging from .01 to .5 mg/kg) reduced EMG activity. Particularly, the application of MK801 peripherally into the TMJ region (at .1 but not at .01 mg/kg) reduced muscular responses. These results
support the conclusion that glutamate mechanisms are involved in the induced muscular responses for both central and peripheral mechanisms, a finding not previously reported.

Finally, Hu, Sun, Zhang and Vernon (in preparation, and [14]) have created a model whereby single dorsal horn neuron (DHN) recordings can be made of responses to deep neck mustard oil injection. This model required considerable innovation in the surgical procedures used for the TMJ study. This is because the standard method of implantation of the recording electrodes requires the use of a stereotaxic apparatus to stabilize the preparation while the recording electrode is implanted. Furthermore, the usual procedure is to perform a laminectomy of atlas and axis posterior structures in order to expose the spinal cord for implantation of the recording electrode from above. Performing these procedures produces three highly undesirable effects when the objective is to study DHN responses to inflammatory irritation of the deep neck muscles:

1] Obviously, the posterior neck tissues are either removed or damaged, rendering them useless for MO injection;

2] a significant amount of bleeding occurs which would further prevent posterior tissue injection;

3] the surgical procedure itself may induce considerable nociception (despite the adequate general anaesthesia) and may produce a degree of central modulation such as DNIC, that might have (and probably has) affected the dorsal horn neuronal responses.
Figure 7. Electrode path for single unit recording in the C1 dorsal horn (DH)

10 Min Before MO

5 Min Before MO

2 Min After MO

10 Min After MO

20 Min

30 Min

40 Min

Brush RF

Pinch RF

Figure 8. Expansion of receptive field of neuron in Figure 7 pre- and post-deep upper cervical inflammatory irritation
To avoid these difficulties, a new surgical technique was devised known as the “minimal trauma technique”. Under fluoroscopic guidance, the recording electrode is threaded down the medulla through a small hole in the superior skull. The termination of this electrode implantation is confirmed, both radiographically and, subsequently, histologically. Figs. 7 and 8 show one preparation with the recording electrode implanted near a wide dynamic range (WDR) neuron in the upper cervical cord. The rat figurines demonstrate the baseline touch and pinch receptive fields (RF’s) which, after percutaneous injection of MO into the C1-C2 paraspinal region, are seen to expand to include, by 20 min, the entire facial region. While this work is still in progress, it represents the first report of the somato-sensory consequences of cervical spinal joint/muscle injury. Gillette and his colleagues have reported similar results in the lumbar region of the rat [85, 86]. These expanded RF’s represent a state of central sensitization whereby the excitability of DHN’s is increased, silent afferent connections may be activated and spontaneous firing can be induced.

The expansion of receptive fields may provide the mechanism of referred pain. In the case of these studies, the original pain generator arises from the upper cervical paraspinal region, including the posterior cranium. Subsequent to the MO injection, pain referral expanding forward to include the facial region may be a manifestation of the receptive field expansion in the same distribution. The extent and pattern of this expansion is very reminiscent of the zones of referred pain elicited from experimental irritation of upper cervical tissues by Feinstein [85] and Travell and Simons [86]. These zones are also identical to those demonstrated to be abolished in studies by Bogduk and his colleagues [87, 88] after anaesthetic injections of the C2-C3 zygapophyseal joints.
The expansion of receptive fields of nociceptive neurons to include extensive skin areas is also a likely mechanism for the hyperaesthesia and allodynia that may be seen after injury to the deep tissues.

**Animal studies: summary**

One of the primary hypotheses of this thesis is that upper cervical spine dysfunction is an important cause of headaches. The development of an animal model was a crucial step to investigate this hypothesis. These studies have shown that experimentally-induced deep paraspinal somatic inflammatory pain replicates the clinical components of upper cervical spinal joint dysfunction. These components include local and distal muscular hyperactivity (as measured by EMG) and expansion of cranio-facial receptive fields, which, itself, is a behavioral indicator of central sensitization. Increased cranio-cervical muscular activity (which, itself, can be a source of pain) and referred cranial pain are the hallmarks of benign headaches.

Of additional interest is the opportunity to compare the findings of the upper cervical inflammatory model in the rat with findings from studies involving experimental inflammation of the tempero-mandibular joint in the same animal model. This combination of data sets allows for a comparison between deep somatic pain of axial origin vs that of proximal joint origin (analogous to the lower cervical spine and the shoulder region). Our studies have shown that deep somatic pain of axial (spinal) origin is particularly effective in producing extensive, multi-joint activation of musculature and multi-dermatomal expansion of receptive fields. As such, our findings have added to a broader understanding of spinal pain mechanisms beyond the field of interest to this thesis.

As with any animal model, extrapolation to humans is to be done cautiously. In this case of our investigations, the rather simple sensory-motor phenomena investigated in the rat bear excellent representation to the same mechanisms in humans.
IV Clinical Mechanisms in Headache of Cervical Origin

Numerous mechanical and arthritic processes affect the region and may give rise to upper cervical pain (i.e. develop into a “pain generator”). This discussion will omit mention of the many pathologic processes which can afflict the region and give rise to pain.

1. Extrasegmental mechanisms

Postural strain and micro or macrotrauma can create myofascial dysfunction. Trigger points in the large regional muscles have been charted by Travell and Simons [86] and create typical referred pain patterns. Stress and occupational repetitive strain can produce static overload of these muscles predisposing to local and referred pain.

2. Intersegmental mechanisms

Painful disorders of the C0-C3 joint structures are currently thought to be the most common disorder in cervical headaches. Pain patterns, both local and referred, have been mapped by provocation and anaesthetic procedures in humans for the C0-C1 joint by Dreyfuss et al. [89], for the C0-C1, C1-C2 and lower cervical joints by Campbell and Parsons [50], for C1-C2 and C2-C3 by Feinstein [85] and Aprill and Bogduk [87, 88, 90]. Barnsley et al. [87, 90] have used double-blind anaesthetic blockades to identify the C2-C3 zygapophyseal (Z-) joint as the primary pain generator in over 50% of a group of whiplash sufferers with headaches.

Trigger points have also been mapped in the deeper intersegmental and suboccipital muscles. Tenderness in the deep suboccipital muscles is the most commonly reported finding in the large number of clinical reports (see review by Vernon et al. [21]). In our 1992 study [27] at least one tender point was identified in at least 84% of a sample of tension-type and migraine sufferers with most having two or more. Sjaastad et al. [22,
23] reported on the high prevalence of paraspinal tenderness at the C2-C3 level. This finding has eventually become a hallmark of CH. Bouquet et al. [91] reported on 24 cervicogenic headache sufferers, 21 of whom had an ipsilateral trigger point at C2-C3. They also commented on a frequently noted finding of what they called an “enlarged C2 spinous process”, which they proposed was due to rotational misalignment at that level. In Jaeger’s report on 11 cervicogenic headache patients [92], tenderness and misalignment around the transverse process of C1 were the most frequently noted findings.

Several authors have reported on standardized methods of measuring tender points in the cranio-cervical region. In 1989, Langemark et al. [93] reported a method of rating manual palpation for muscular tenderness. Tender points are rated on a three-point scale to a standardized manual pressure. It was determined that the pressure sufficient to blanch the examiner’s thumb nail was sufficient to elicit tenderness which is then rated as follows:

0 = no reaction
1 = slight reaction, no vocalization, no movement
2 = moderate reaction with vocalization
3 = severe reaction with vocalization and flinching or other movements

Scores for a variety of muscles are added up bilaterally for a “Total Tenderness Score” (TTS). TTS in fifty tension-type headache subjects were found to be highly reproducible on two examinations separated by three weeks. Comparison of findings in 24 healthy controls indicated significantly higher TTS in headache subjects. Numerous replications of this methodology have verified its reliability and validity [94, 95]. While the manual palpation method has been employed in both neck pain and headache subjects and has been found to very reliable, no study has yet investigated the role of tenderness in cranial, sub-occipital and neck/scapular muscles in TTH sufferers. In an unpublished study, the author used pressure algometry, another method frequently reported to
measure tender points [96-104] in a comparison of 14 headache and 14 control subjects. There was a significant trend toward multiple tender points to be found in headache subjects (four or more). Overall values for each of eight tender points were lower in the headache as compared to control subjects (mean pressure pain threshold values (kg/sq.cm): headaches = 1.9 ± 1.7; controls = 3.8 ± 1.9; p = .02)

3. **Intrasegmental mechanisms**

Entrapment of the greater occipital nerve (GON) and its ganglion has long been purported to cause Greater Occipital Neuralgia [105]. Recent evidence by Bogduk [59, 105] casts doubt on this theory, as anaesthetization of the GON would reduce pain from any of the tissues it innervates, making this procedure too non-specific for anatomical localization. Irritation of the sensory fibres in the anterior ramus of C2 by inflammation or osteophytic outgrowths from the C1-C2 lateral joint has been implicated as a cause of the uncommon “Neck-Tongue Syndrome” [106].

4. **Infrasegmental mechanisms**

Only two direct mechanisms related to mechanical disturbances have been identified for the upper cervical cord. The first concerns a controversial mechanism reported by Hack et al. [107] involving a ligamentous connection between the rectus capitis posticus minor and the dural lining at the foramen magnum. Hack et al. postulated that tightness or strain of this ligament may be responsible for some cranial pain of cervicogenic origin.

The second mechanism involves a herniation of the C2-C3 IVD. This is relatively rare, and, up until recently only Elvidge [108] had reported on it. Recently, the use of C2-C3 discograms and disc fusion surgery have resurrected this idea. The most important role for the spinal cord in CH lies in the phenomenon of afferent convergence of the upper cervical and trigeminal systems, as described above [5, 75].
This mechanism provides an explanation for referred pain to the cranium resulting from upper cervical deep tissue pain. The same convergence phenomenon can explain why posterior intracranial pathologies result in referred upper cervical pain. This may be one of the mechanisms underlying the creation of upper cervical pain and myofascial dysfunction in migraine. Painful and inflamed posterior cranial vessels can refer pain to the sub-occipital region - one more cause of diagnostic confusion!
V Manual Therapy Assessment of TTH and CH

Table 3 lists the procedures and their purported pathophysiologic mechanisms associated with chiropractic assessment in CH. These procedures and their associated dysfunction targets are described in-depth below.

Table 5: Methods of Clinical Assessment in TTH and CH

<table>
<thead>
<tr>
<th>Regional</th>
<th>Mechanism</th>
</tr>
</thead>
<tbody>
<tr>
<td>1. Plumbline/postural observation</td>
<td>1. Anterior head carriage</td>
</tr>
<tr>
<td>2. Range of motion</td>
<td>2. Lateral and/or rotational craniocervical distortion</td>
</tr>
<tr>
<td>3. Radiography - plain film</td>
<td>1. Reduced regional ranges of motion.</td>
</tr>
<tr>
<td>Segmental</td>
<td>1. Dynamometry</td>
</tr>
<tr>
<td>3. Motion palpation</td>
<td>1. Quantification of myofascial tenderness.</td>
</tr>
</tbody>
</table>
<pre><code>                                                             | 1. Detection of joint fixation or hypomobility. |
                                                             | 2. Appreciation of myofascial tissues during movement. |
</code></pre>
V.A Physical Assessment
V.A.1. Regional Considerations

1.1 Postural observations / plumbline testing

Anterior head posture has been correlated with increased incidence of headache and neck pain [109-111]. Many sedentary occupational postures in our modern world predispose to anterior head posture. Watson and Trott [111] provide a theoretical explanation as follows: shortened anterior neck and shoulder muscles induce an anterior shift of the head and neck. In order to compensate for this and maintain optimal horizontal alignment of the cranium, the sub-occipital and long occipital extensors would tighten. This would induce sub-occipital joint compression and pain, as well as create myofascially generated pain in the affected muscles.

In Watson and Trott’s study, 30 cervical headache subjects were found to have significantly greater forward head posture than a similar number of control subjects. Treleaven et al. [112] however, failed to confirm this in a smaller group of post-traumatic headache sufferers.

Grimmer et al. [113] recently reported that CH sufferers have significantly longer anterior neck length measurements than do non-HA sufferers, implying that anterior head shift may be present. Placzek et al. [114] found no difference in head posture between chronic headache sufferers and normal controls.
Anterior head carriage can be assessed using a plumbline and by photographic analysis [113, 114]

1.2 Ranges of motion.

Reduced active ranges of motion (AROM) in the neck have been reported frequently in subjects with neck pain and headaches. Several devices for measuring active and passive ranges of neck motion have been reported, including cap goniometers [115, 116] and magnetic inclinometers [117-120], with findings of good test-retest and inter-examiner reliability.

Several studies have reported on AROM in headache subjects. Stodolny and Chmielewski [121] used a goniometer to measure cervical AROM as an outcome of manipulation therapy in patients with "cervical migraine". After several treatments, AROM values were reported to be increased in these subjects. Kidd and Nelson [122] used a simple visual assessment of cervical AROM in benign headache subjects, reporting that two or more ranges were reduced in headache subjects more frequently than in controls.

Sandmark and Nisell [123] studied the degree to which active rotations and flexion/extension reproduced pain in neck pain subjects as compared to healthy controls. No actual measure of the range of motion was taken. The sensitivity and specificity of rotations and flexion/extension in correctly identifying symptom status were 77% and 92%, and 27% and 90%, respectively. These results imply that there is relatively little error in identifying non-painful subjects when AROM's are not painful, but there is poor correlation between positive findings and pain status.

Recently, Placzek et al. [114] reported significantly lower values for cervical extension in chronic headache sufferers as compared to controls. This implicates shortening of the anterior cervical musculature as part of the postural adaptation associated with chronic
headache. Stolk-Hornsveld et al. [124] reported significant reductions in all cervical ranges of motion with the exception of forward flexion in CH subjects versus those with other types of headaches. This corroborates the validity of "reduced ranges of neck motion" as a criterion of CH diagnosis.

On the other hand, Persson and Carlsson [57] found no differences in cervical ROM's between groups on neck pain patients with or without headache. However, these subjects may all have had reduced ROM's, thereby leaving little room for differences between the sub-groups. Grimmer et al. [113] reported reduced extension in frequent vs infrequent female CH subjects; however, this difference was not significant after age-adjustment. This is important, as AROM's have been shown to reduce progressively after the third decade of life [125].

Several classic prognostic studies [126-128] have reported that active ranges of motion (AROM) are reduced in chronic whiplash-injured patients. Osterbauer et al. [129] recently reported that 10 whiplash-associated disorder (WAD) cases had a combined AROM of 234 deg., considerably less than the normal 360 deg. After six weeks of conservative treatment, total AROM increased to 297 deg. Hagstrom and Carlsson [130] compared 30 WAD cases with 30 normal subjects and found reduced AROM in all ranges.

In a recent study, Vernon [30] objectively measured active ranges of neck motion (AROM) in a sample of 44 chronic WAD patients (all of whom reported headache symptoms) and found moderately high correlations ($r = 0.55-0.70; p = .001$ to .0001) between all range of motion scores and the subjects' self-rated disability scores (using the Neck Disability Index (see below)). This is the first demonstration of a link between validly measured aspects of WAD-related impairments with levels of disability suffered by these patients. Interestingly, AROM scores were not correlated with age and duration of complaint, again, leading to the notion that chronic WAD sufferers reach a stable plateau of self-rated disability and impaired ranges of neck motion. In contrast,
Jordan et al. [131] compared self-rated disability scores (using the Copenhagen Neck Functional Disability Scale (see below)) with active neck extension and found no significant correlation.

### 1.3 Radiography – plain film

Two x-ray studies [Vernon et al. [27], Nagasawa et al. [132]] have demonstrated higher levels of straightened lordotic curves in headache subjects which is likely associated with forward head posture.

### 1.4 Cervical muscular function.

Cervical strength testing has recently been reported for neck pain and headache subjects. Vernon et al. [104] reported significantly lower strength values in all cervical ranges among chronic neck pain/whiplash sufferers (almost all of whom also complained of headaches) as compared to controls. Most importantly, the ratio of flexion : extension strength was much lower (28% vs 69%) in the pain group, implicating the importance of flexor muscle weakness in this condition.

Grimmer et al. [113] reported that reduced cervical extensor and flexor strength predicted increasing headache frequency in women, but not men. Placzek et al. [114] reported reduced extensor and flexor strength as well as reduced endurance of the anterior cervical musculature in women with chronic headaches as compared to controls.

To summarize this section of the thesis, it is hypothesized by many of these authors that the separate factors of chronic pain, postural decompensation, reduced ranges of motion and reduced muscular strength and endurance of the cervical spine as a whole interact together in a self-promoting or vicious cycle of progressive mechanical dysfunction, resulting in greater persistence of neck pain. It is also hypothesized that
the focus of these regional dysfunctions is the intersegmental tissues, whose
dysfunction we now discuss.

V.A.2 Segmental Considerations

2.1 Static palpation for tenderness and misalignment.

Conventional manual palpation can provide the clinician with information about
myofascial and joint dysfunction. Careful manual assessment can identify
misalignments between upper cervical segments (particularly the position of the C1
TVP's, the C2 spinous process and the C0-C3 posterior articulations). Tissue texture
changes include tightened muscles, rotated spinous process of C2 [91] and the taut,
tender bands of trigger points. Tender points can be located by pressure over any of
the soft tissues and bony insertion sites. Tenderness on palpation of the tissues of the
craniovertebral and paraspinal region is the most commonly reported sign of cervical
dysfunction in headache subjects. Many authors have reported on the subject, from
Lewit [133] who reported on "pain over the posterior arch of atlas", to Sachse et al. [134]
who reported similar findings of sub-occipital and scapular tenderness, and Graff-
Radford et al. [47] and Jaeger [92] who reported on the numerous cervical tender points
which they proposed served to perpetuate myofascial head pain.

Sjaastad et al. [22-24] reported on the high prevalence of tenderness at C2-C3. This
finding eventually became a hallmark of "cervicogenic headache". Bouquet et al. [91]
reported on 24 cervicogenic headache sufferers, 21 of whom had an ipsilateral trigger
point at C2-3. The also commented on a frequently noted finding of an enlarged
spinous process of C2, which they proposed was due to static rotational misalignment
at that level. In Jaeger's report on 11 cervicogenic headache patients [92], tenderness
and misalignment around the transverse process of C1 were the most frequently noted
findings.
The findings of more recent reports which have employed the standardized methods of tender point analysis in headache subjects which were described above are listed in Table 6.
# Table 6: Studies of manual tenderness assessment in neck pain and headaches

<table>
<thead>
<tr>
<th>AUTHOR(S)</th>
<th>FINDINGS</th>
<th>LOCATION</th>
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<tbody>
<tr>
<td>Lebbink et al. [135]</td>
<td>Neck muscle soreness and stiffness as well as prior neck injury were more common in 164 headache sufferers than 108 controls.</td>
<td>Neck muscles</td>
</tr>
<tr>
<td>Jensen et al. [136]</td>
<td>Studied 14 muscle sites bilaterally in normals; used Langemark et al.'s method of scoring. Norms reported. Older subjects had lower TTS values while females had higher TTS scores.</td>
<td>Cranial and large neck muscles</td>
</tr>
<tr>
<td>Jensen et al. [137]</td>
<td>TTS scores in TTH and migraine headache sufferers compared. TTH's had lower overall scores. TTH's with headache that day had higher TTS than matched non-HA group</td>
<td>Cranial and large neck muscles</td>
</tr>
<tr>
<td>Jensen et al. [138]</td>
<td>In 19 PTHA's 42% had tenderness at C2-C3, 89% at C3-C4 and 63% at C4-C5.</td>
<td>Neck paraspinal muscles</td>
</tr>
<tr>
<td>Hatch et al. [139]</td>
<td>HA subjects had at least one tender muscle more often than controls; TTS in HA's greater than controls; EMG findings not correlated to tenderness.</td>
<td>4 cranial muscles 2 posterior cervical muscles</td>
</tr>
<tr>
<td>Watson and Trott [111]</td>
<td>PTHA's had more tenderness findings than controls, particularly in upper cervical spine.</td>
<td>Neck paraspinal muscles</td>
</tr>
<tr>
<td>Mercer et al. [140]</td>
<td>HA subjects had higher values of tenderness than controls.</td>
<td>Neck paraspinal muscles</td>
</tr>
<tr>
<td>Levoska et al. [141]</td>
<td>Test-retest correlation of manual palpation of scapular muscles was high; inter-rater reliability only fair.</td>
<td>Scapular muscles</td>
</tr>
<tr>
<td>Levoska et al. [142]</td>
<td>Neck pain sufferers had high number of tender points than controls.</td>
<td>Neck paraspinal muscles</td>
</tr>
<tr>
<td>Hubka and Phelan [143]</td>
<td>Inter-rater reliability of segmental TTS scores were highly correlated (Kappa = 0.68).</td>
<td>Neck paraspinal muscles</td>
</tr>
<tr>
<td>Sandmark and Nisell [123]</td>
<td>Cervical tenderness was most sensitive (62%) and specific (79%) for neck pain patient discrimination.</td>
<td>Neck paraspinal muscles</td>
</tr>
<tr>
<td>Nilsson [144]</td>
<td>TTS scores in neck pain patients high inter-rater reliability,</td>
<td>Neck paraspinal muscles</td>
</tr>
<tr>
<td>Sandrini et al. [145]</td>
<td>Mean TTS scores higher in ETTH and CTTH subjects than controls</td>
<td>Trapezius</td>
</tr>
<tr>
<td>Persson and Carlsson [57]</td>
<td>TTS scores higher in CH vs controls</td>
<td>Sub-occipital, neck paraspinal and scapular muscles</td>
</tr>
<tr>
<td>Stolk-Hornsveld et al. [124]</td>
<td>Segmental tenderness on passive motion at C1 - C4 higher in CH vs other headache types. Good inter-rater reliability</td>
<td>Sub-occipital and neck paraspinal muscles</td>
</tr>
</tbody>
</table>

Legend: TTS = total tenderness score; HA = headache; TTH = tension-type headache; ETTH = episodic tension-type headache; CTTH = chronic tension-type headache; CH = cervicogenic headache; PTHA = post-traumatic headache; EMG = electromyography
2.2 Algometry

While procedures for manual palpation permit the location (i.e., the identification) of tender points and, to some degree, an assessment of the severity of tenderness present at that point, they are limited in the degree to which such quantification of severity can be accomplished. To address this deficiency, numerous instruments have been devised to which would allow for manually controlled and measurable force application, thereby permitting discrete quantification of the degree of tenderness. Since the object of greatest concern in myofascial and joint dysfunction is the deeper somatic tissues, devices which permit pressure stimuli – pressure algometers – have become widely used. Fischer's work, in particular, [98, 99] enabled the standardization of this type of investigation. According to his protocol, side-to-side differences of 1 kg/sq.cm or absolute tender point values less than 2.5 kg/cm² in the cervical region indicate an active tender point. Table 7 lists studies which employed a pressure algometer to assess cranio-cervical tenderness in headache and neck pain subjects.
Table 7: Studies of pressure algometry in neck pain and headache

<table>
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<tr>
<th>AUTHOR(S)</th>
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<th>LOCATION</th>
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<tbody>
<tr>
<td>Reeves et al. [100]</td>
<td>High correlation coefficients for intra- and inter-examiner reliability. Average value for C0-C1 = 3.0 kg/sq cm; for trapezius = 3.5 kg/sq cm</td>
<td>Occipital and suboccipital</td>
</tr>
<tr>
<td>Jensen et al. [136]</td>
<td>Highly consistent values bilaterally and over three week interval in normals.</td>
<td>Temporalsis muscle</td>
</tr>
<tr>
<td>Drummond [146]</td>
<td>High intra-examiner reliability; HA subjects had lower algometer values than normals; no difference between TTH and migraine HA.</td>
<td>Scalp and upper cervical muscles</td>
</tr>
<tr>
<td>List et al. [147]</td>
<td>High reliability coefficients; algometry scores highly correlated to manual palpation findings; TMJ pain subjects had lower values than normals.</td>
<td>Temporalsis and suboccipital muscles</td>
</tr>
<tr>
<td>Langemark et al. [93]</td>
<td>Temporalsis algometry negatively correlated to headache intensity and to TTS on manual palpation. High correlation between temporal and occipital sites.</td>
<td>Cranial muscles</td>
</tr>
<tr>
<td>Takala [148]</td>
<td>High intra- and inter-rater reliability in normal subjects; women had lower algometry values than men; lower values in sub-group with minor neck pain and HA.</td>
<td>Scapular muscles</td>
</tr>
<tr>
<td>Hogeweg et al. [149]</td>
<td>Good reliability in normals; cervical points have lower algometry values than lumbar points.</td>
<td>Spinal muscles</td>
</tr>
<tr>
<td>Bovim [94]</td>
<td>Lower algometry values in cervicogenic HA group vs. migraine, TTH and controls. CH group had lower values in posterior cranial area and on the affected side.</td>
<td>Cranial and suboccipital muscles</td>
</tr>
<tr>
<td>Chung et al. [150]</td>
<td>Electronic pressure algometer showed good reliability and test-retest consistency in normals.</td>
<td>TMJ and neck muscles</td>
</tr>
<tr>
<td>Jensen et al. [151]</td>
<td>Algometry values lower in TTH vs controls.</td>
<td>Cranial muscles</td>
</tr>
<tr>
<td>Kosek et al. [152]</td>
<td>Algometry in normals showed good one-week consistency; lower values in upper part of body.</td>
<td>Whole body</td>
</tr>
<tr>
<td>Levoska et al. [141]</td>
<td>Reliability high in neck pain and normals; pain group had lower values.</td>
<td>Scapular muscles</td>
</tr>
<tr>
<td>Mazzotta et al. [153]</td>
<td>PPT values signif. lower in ETTH vs controls.</td>
<td>Temporalsis</td>
</tr>
<tr>
<td>Sandrini et al. [145]</td>
<td>PPT values signif. lower in ETTH and CTTH vs controls.</td>
<td>Frontal and Trapezius</td>
</tr>
<tr>
<td>Stolk-Horsnved et al. [124]</td>
<td>High levels of inter-rater reliability. Sensitivity and specificity for CH vs controls = 82% and 92%</td>
<td>Sub-occipital and neck paraspinal muscles</td>
</tr>
<tr>
<td>Bendtsen et al. [95]</td>
<td>Reported on an electronic finger pressure pad for palpatind tenderness. High levels of inter-examiner reliability.</td>
<td>Cranial muscles</td>
</tr>
</tbody>
</table>

Legend: as for Table 6. PPT = pressure pain threshold
2.3 **Motion palpation.**

One of the components of cervicogenic dysfunction most commonly cited by practitioners of manual therapy is disturbance of motion at individual spinal motion segments. This phenomenon has been variously termed "subluxation" or "fixation" (in chiropractic circles), "joint" or "somatic dysfunction" (in osteopathic circles) and "joint blockage" in manual medicine circles. The common feature of these terms is that they refer to **hypomobility** of the joints.

The procedures used to assess hypomobility are variations of manual palpation techniques of either the active, passive or accessory motions of the individual spinal motions. These procedures have been devised and described by many experts in the manual therapy disciplines, from Gillet [154], Liekens [155], Faye [156], Grice [157], and Fligg [158] in chiropractic, to Lewit [159] and Mennell [160] in manual medicine. The generic term for these procedures is "segmental motion palpation".

The inter-examiner repeatability of these assessments is unclear. Several authors have reported poor agreement [161, 162], but there are many methodological flaws in these studies. These include the use of asymptomatic students as subjects, the use of inexperienced students as examiners and the use of multiple replications of the procedures, such that the minor intervertebral derangements meant to be found were, temporarily, removed. In Watson and Trott's well-conducted study [111], multiple outcomes of segmental dysfunction, including segmental motion palpation, were employed to assess cervical headache subjects. They reported on the reliability of posterior-to-anterior glide palpation in 12 of their subjects examined on two occasions by the same examiner. Their Kappa reliability values ranged from 0.67 to 1.00, depending on the segment.
More recently, Strender et al. [163] reported poor inter-examiner reliability for segmental motion assessment in 50 subjects, half of whom complained of neck or shoulder pain. On the other hand, Jull et al. [164] reported very high rates of agreement between several pairs of examiners in their ability to detect the presence or absence of any “treatable upper cervical dysfunction”. Agreement levels as to the exact segment of dysfunction were somewhat lower, but still acceptable (70% overall agreement). The C1-2 segment showed the highest frequency of joint dysfunction.

With regard to the validity of the notion of spinal joint hypomobility in cervical headache patients, several studies have used both non-controlled and controlled comparisons. Jull [165 - 167] compared motion palpation findings in headache and non-headache subjects. Hypomobility was found at C0-C1, C1-C2 and C2-C3 in 60%, 40% and 55% of headache subjects and 5%, 12% and 22% of non-HA subjects, respectively. These findings were later confirmed in a study comparing motion palpation findings (with tenderness) compared to local anaesthetic blockades of the zygapophyseal joints in neck pain and headache subjects [167]. The sensitivity of motion palpation was reported as 100%. Jull et al. [164] also reported high levels of agreement between examiners’ motion palpation findings which were obtained without pain cues from their headache subjects and without knowing the subjects’ subsequent report of pain during each procedure at each cervical segmental level. In other words, joint dysfunction can be validly determined without the subject providing pain-related feedback. Much greater levels of significant joint dysfunction were found in the upper cervical segments of cervical headache subjects vs controls in this study.

Jensen et al.'s treatment study [138] of 19 post-traumatic headache patients reported on the findings of hypomobility before and after treatment with a short course of spinal manipulation. Fourteen of the subjects had at least one level of joint blockage in the upper cervical and upper thoracic region, while 4 had blockage only in the upper cervical region, for a total of 18/19 with upper cervical hypomobility. The most frequently blocked segment was C1-C2.
In Vernon et al. [27], 54% of TTH headache sufferers had hypomobility at two upper cervical segments while 30% had all three levels (C0-1, C1-2, C2-3) affected at least unilaterally. In the group of migraine headache sufferers, these figures were 42% and 42%, respectively. In both groups, a total of 84% had at least two upper cervical segments demonstrating hypomobility.

In Watson and Trott's study [111], far more headache subjects than controls demonstrated painful segmental hypomobility, with the most frequent blockage at CO-C1.

Treleaven et al. [112] reported on 12 subjects with post-traumatic headache compared to an age and gender-matched control group. Joint dysfunction (with tenderness) was rated as mild, moderate or marked. Ten of twelve HA subjects had at least one segment demonstrating marked hypomobility in the upper cervical spine. Much more significant joint dysfunction in the headache group was noted between C0 - C3 in the headache group as compared to the control group.

Stodolny and Chmielewski [121] reported that all 31 of their “cervical migraine” cases had significant joint dysfunction at C0-1 on manual palpation. Over 80% of subjects had at least 2 cervical segments demonstrating joint dysfunction, which is remarkably similar to the findings of Vernon et al. [27] in TTH and migraine subjects.

Manual Therapy Assessment: Summary

The third hypothesis of this thesis is that cervical spinal dysfunction can be assessed and characterized by a range of standard clinical methodologies employed in manual therapy. Figure 9 provides a schematic model to interpret studies relative to manual therapy assessment procedures for headache of cervical origin. Regional assessment procedures include the assessment of global head/neck and shoulder posture, by both observation and x-ray, as well as cervical ranges of motion, by observation or
instrumentation. At present, there is insufficient data to conclusively identify a pattern of regional disturbance in headaches of cervical origin, although it is noteworthy that the IHS Classification includes several of these features.

With regard to segmental features of dysfunction, local tenderness has been the most studied. The literature and our studies support the importance of assessing the upper cervical spine for tenderness and any clinical pain referral patterns which emerge in this assessment. The role of spinal joint hypomobility is more controversial; yet, this aspect of dysfunction is at the core of all manual therapy theories, and there is a growing body of evidence that it plays a role in HCO. The 1992 study by Vernon et al. [27] clearly shows how predominant this dysfunction is in tension-type and migraine sufferers and the work of Jensen et al. [138] has done the same for post-traumatic cervicogenic headache. As with regional features, these local or segmental features are key components of the HIS Classification criteria for cervicogenic headache. Their role in other benign headaches will be clarified by further studies.
Figure 9. Schematic for the assessment of cervical dysfunction in HCO
V.B  Self-rated Questionnaires

V.B.1  The Neck Disability Index:

The first instrument designed for assessing self-rated disability specifically due to neck pain, in particular, was the Neck Disability Index (NDI) [28]. Designed by Vernon as a modification of the Oswestry Low Back Pain Disability Questionnaire (OLBPDQ) [168], the NDI is a ten-item questionnaire with well-established psychometric properties such as high test-retest reliability, good internal consistency and good sensitivity to change. Hains et al. [169] recently established a single factor structure to the Index as well as reporting that no response bias could be found amongst the items.

Riddle and Stratford [170] have recently added to the psychometric profile of the NDI by determining three important values for its use in clinical and research settings. These are: “variation around a measured value”, minimal detectable change (MDC) and minimal clinically important difference (MCID). The former of these values addresses the error margin inherent in any single use of the NDI, typically in a practice setting. This value was found to be 5 NDI points, at a 90% confidence interval. To paraphrase Binkley [171] in her recent review article, “if the error associated with a 50 point scale (such as the NDI) is 5 points, with a 90% confidence interval, the interpretation of this is that, given a score of 20/50, a clinician can be 90% sure that the true score lies between 15 and 25” ([171], pg. 10).

According to Riddle and Stratford [170], the MDC and the MCID values are both 5 NDI points. This means that the sampling error of the instrument limits the range of minimal detectable change in a patient’s status to 5 NDI points. However, as a result of its use in a cohort of neck pain patients, they determined that the minimal clinically important difference is also 5 NDI points. Several studies have reported mean change scores well beyond that level [172, 173].
In a recent study, of 44 chronic WAD claimants [30], Vernon reported on additional psychometric features of the NDI. First, this sample’s responses were in almost identical rank order as the initial sample [28]. The items “headache”, “lifting”, “recreation” and “reading” were still among the five most highly rated items, confirming their importance in chronic WAD.

Second, NDI scores were not well-correlated with age and gender, as in the original work; however, in this new sample, duration of complaint was also not well-correlated (r = 0.17, NS). This was explained as follows, “whiplash-injured patients who go on to experience chronic difficulties may reach a plateau of pain, impairment and self-rated disability, the complex of which (may remain) approximately static from that time onwards” ([30], pg. 211).

Third, two subsets of items, namely, “symptoms” (four items) and “activities” (six items) were compared to one another, with a moderate, but significant level of correlation (r = 0.55, p = .05). One interpretation of the lack of strong correlation is that these two subsets may offer unique (i.e., non-redundant) information on the WAD-sufferer’s perception of the effect of their condition on their activities of daily living (ADL’s).

Finally, NDI scores in this sample were correlated with scores on a newer “generic” instrument for self-rating of disability, the Disability Rating Index [174]. The two questionnaires were strongly correlated (r = .89, p = .001). This finding further confirms the construct validity of the NDI as a measure of physical disability.

V.B.2 The Neck Pain Questionnaire (NPQ):

In 1994, Leak et al. [175] reported on their development of the Northwick Park Neck Pain Questionnaire (NPQ). The authors reported that, as with Vernon and Mior [28], they used the OLBPDQ as a basis for their instrument. No report of the methodology for adapting the OLBPDQ was given.
The NPQ contains nine items which are scored from 0 to 4 for a total score out of 36. The items consist of the following: pain intensity, sleeping, numbness, duration, carrying, reading/television, work, social life and driving. These items represent a mix of symptoms as well as activities thought to be important to neck pain patients. Of these "activity items", all but one ("carrying" vs "lifting") had already been incorporated in the NDI, published three years earlier. Both instruments also retained the "pain intensity" item of the original OLBPDQ.

Forty-four subjects completed an NPQ at their original consultation while 31 completed a second NPQ three to five days later. Thirty-five of these subjects also completed NPQ's at 4 and 12 weeks later. No formal treatment was offered in the study, but many subjects did receive some form of treatment.

Short-term repeatability was reported as high, with a Pearson's coefficient of \( r = 0.84 \) and Kappa = 0.62. Inter-item agreements ranged from \( K = 0.53 \) to 0.76. Internal consistency was not formally tested, but was said to be adequate via graphical depiction.

Initial NPQ scores did not correlate well with age, gender, duration or previous history of neck pain. While NPQ scores did not change significantly over the three-month study interval, they did correlate well with a separate question rating the subject's perception of improvement. This was cited as an indicator of "sensitivity to change". Subsets of subjects who either received physiotherapy or performed home exercises had what was described as "significant improvement" in their NPQ scores.

Given the similarities between the NDI and the NPQ, this author considers Leak et al.'s report to be, essentially, a replication study of the NDI. That the same set of psychometric properties was reported, namely, high levels of test-retest reliability, internal consistency and sensitivity to change, as well as poor correlations with age,
gender and duration of complaint is therefore not surprising and confirms the original report [28]. To this author's knowledge, no additional studies on the NPQ have been reported since 1994.

V.B.3 The Copenhagen Neck Functional Disability Scale (CNFDS):

The CNFDS was devised by Jordan et al. [176] as an attempt to improve the existing questionnaires (NDI and NPQ) for assessing disability due to neck pain. Jordan et al. asserted that, since both previous instruments incorporated some items related to "symptoms" (pain, numbness and duration in the NPQ; pain, headache and concentration in the NDI), these questionnaires lacked some precision for measuring solely the disability due to neck pain itself. This assertion was based on the theory that pain, disability and impairment are separate but inter-related constructs.

The CNFDS contains 15 items with a three-point scale (yes = 0, occasionally = 1, no = 2) for a maximum of 30 points. Many of the item constructs are similar to those in the NDI (sleep, personal care, lifting, reading, headaches, concentration and recreation), while three additional items focus on psycho-social issues.

Jordan et al. reported very high test-retest reliability ($r = 0.99$), excellent internal consistency (Cronbach's alpha = 0.89) and no significant correlation between initial scores and age or gender. Additionally, initial CNFDS scores were highly correlated with patients' global assessment of their condition ($r = 0.83$) and moderately correlated with doctors' global assessments ($r = 0.56$). Initial scores also correlated highly with a separate 11-point pain ratings for neck and arm pain, which somewhat questions the authors' original premise of a clinically important distinction between self-ratings of pain and disability.
Finally, the authors reported good sensitivity to change in a larger sample of subjects enrolled in a clinical trial for neck pain [131]. At 6, 24 and 52 weeks of this trial, changes in pain scores correlated with changes in CNFDS scores at $r = 0.49$, $0.48$ and $0.54$ respectively.

Table 8 provides a comparison of the different items in each of these instruments.

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<td>ITEMS</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>1</td>
<td>Pain $^2$</td>
<td>pain $^2$</td>
<td>sleeping $^3$</td>
</tr>
<tr>
<td>2</td>
<td>Personal care $^2$</td>
<td>sleeping $^3$</td>
<td>daily activities $^1$</td>
</tr>
<tr>
<td>3</td>
<td>Lifting $^2$</td>
<td>numbness $^1$</td>
<td>daily activities $^3$</td>
</tr>
<tr>
<td>4</td>
<td>reading $^3$</td>
<td>duration $^1$</td>
<td>dressing $^2$</td>
</tr>
<tr>
<td>5</td>
<td>headache $^2$</td>
<td>carrying $^1$</td>
<td>washing $^2$</td>
</tr>
<tr>
<td>6</td>
<td>concentration $^2$</td>
<td>reading/TV $^3$</td>
<td>at home $^1$</td>
</tr>
<tr>
<td>7</td>
<td>Work $^3$</td>
<td>work $^3$</td>
<td>lifting $^2$</td>
</tr>
<tr>
<td>8</td>
<td>driving $^2$</td>
<td>social $^2$</td>
<td>reading $^3$</td>
</tr>
<tr>
<td>9</td>
<td>sleeping $^3$</td>
<td>driving $^2$</td>
<td>headaches $^2$</td>
</tr>
<tr>
<td>10</td>
<td>recreation $^2$</td>
<td></td>
<td>concentration $^2$</td>
</tr>
<tr>
<td>11</td>
<td></td>
<td></td>
<td>recreation $^2$</td>
</tr>
<tr>
<td>12</td>
<td></td>
<td></td>
<td>resting $^2$</td>
</tr>
<tr>
<td>13</td>
<td></td>
<td></td>
<td>family $^1$</td>
</tr>
<tr>
<td>14</td>
<td></td>
<td></td>
<td>social $^2$</td>
</tr>
<tr>
<td>15</td>
<td></td>
<td></td>
<td>“future” $^1$</td>
</tr>
</tbody>
</table>

$^3$ = item found in all three instruments  
$^2$ = item found in two instruments  
$^1$ = item found in only one instrument
V.B.4 The Headache Disability Inventory

This 25-item scale was developed by Jacobsen et al. [177] to assess the impact of recurrent headaches on daily function. The items are organized into two scales: "emotional" and "functional". Good stability of scores has been reported over one week and eight-week intervals. Unfortunately, Jacobsen et al. [179] reported very poor sensitivity to change in a sample of treated patients. As well, they reported that change in HDI was not well-correlated with change in frequency of headaches.

V.B.5 Activities of Daily Living (ADL)

A series of validated questions to monitor the impact of headaches on activities of daily living has been developed by von Korff et al. [178]. These ADL-related questions employ a relatively long interval of six months' time. For clinical usage, the intervals of time might be shortened.

1. How many days in the last six months have you been kept from your usual activities (work, school or housework) because of headaches?

2. Using the following 0 - 10 scale, where 0 = "no interference" and 10 = "unable to carry out any activities,

   I. In the past 6 months, how much have your headaches interfered with your daily activities?

   II. In the past 6 months, how much have your headaches interfered with your ability to take part in recreational, social or family activities?

   III. In the past 6 months, how much have your headaches interfered with your ability to work (including housework)?
Holroyd et al. [179] have recently reported that these questions loaded most highly on a separate factor labeled “headache disability” in the responses of their headache sample. Scores from the “disability” scale were not highly correlated to “pain intensity” scores”, indicating that self-rated disability is a separate construct in the experience of headache sufferers and should be assessed separately by clinicians and researchers.

V.B.6 The Dizziness Handicap Inventory (DHI)

This instrument was developed in 1990 [180] as a 25-item scale to assess the impact of dizziness and vestibular problems on daily life. The DHI is composed of three subscales: functional, emotional and physical. The original paper reported good test-retest reliability and internal consistency. DHI scores were found to correlate well with increasing frequency of dizziness episodes and with scores from balance tests [180].

V.C Headache Monitoring

V.C.1 The Headache Diary:

The primary instrument for monitoring headache activity is the Headache Diary. Numerous versions of such diaries exist, depending on the particular interest of the research involved [181]. For typical clinical use, the once-daily diary recording format is optimal. This type of diary permits calculations of the following parameters: frequency of headache days per week/month; headache severity for any particular day or averaged over any interval; peak headache severity per any interval (i.e., worst headache in a month); medication usage. Some diaries record the duration of headaches, but, often, the use of analgesic medications will shorten the duration, thereby providing false impressions of the true duration of ongoing headaches.
For clinical purposes, it is often sufficient to monitor the frequency and average severity of headaches. The desirable outcome of clinical management would be a reduction in both of these parameters. This is typically the kind of outcome which has been reported in clinical trials of prophylactic treatments for TTH or CH.

**Self-rated Questionnaires: Summary**

Headache very frequently accompanies mechanical neck injuries. The first instrument for assessing the self-rated disability associated with neck pain (The Neck Disability Index) has been employed in several studies of neck pain patients, and “headache” is the highest ranked item by these patients. While this Index contains only one item for assessing the impact of headache on neck-related disability, several other instruments have been developed to assess headache activity more comprehensively. The standard instrument for research and clinical purposes is the Headache Diary, which provides information on the daily presentation of headaches, their severity and duration as well as the medications used to relieve them. In the short-term, reduction of headache intensity (analgesia) is the primary clinical outcome of interest following a therapeutic intervention (use of medications, chiropractic treatment, massage, etc). Reductions of headache frequency and impact on activities of daily living are the outcomes of interest in long-term or prophylactic treatments such as low-level anti-depressant therapy or a course of chiropractic treatments. Most of these instruments have been employed in studies of the effectiveness of these treatments which will be reviewed in the following section.
VI. THE TREATMENT OF TENSION-TYPE AND CERVICOGENIC HEADACHE BY
MANUAL THERAPY

A number of systematic reviews and meta-analyses have been reported on treatments for headache (182-188). The interventions studied in these reviews have been confined to pharmacological therapies and cognitive/behavioral therapies. The majority of these reviews have been for treatments of migraine-type headache. Bogaards and ter Kuile's recent meta-analytic review of treatments for "recurrent tension headache" [184] confined itself to the following categories of intervention: pharmacological, cognitive therapy, relaxation therapy, EMG biofeedback therapy and combinations, although some complementary/alternative (CAM) therapies (acupuncture and physiotherapy) were regarded as control or "pseudo-placebo" treatments. No primary CAM therapies were included. It appears that no systematic review of CAM therapies for non-migrainous headaches currently exists in the literature.

Since the publication of Eisenberg's important article describing the usage of CAM therapies by Americans [189] interest in the topic within orthodox medical circles has grown and the use of CAM therapies in society has increased considerably. Rates of utilization of CAM therapies by sufferers of TTH are poorly understood. Several studies cite the proportion of patients seeking chiropractic care for headache to be approximately 3 -10% of patients in a typical practice [1, 190].

Our group is pursuing a number of clinical studies in TTH and CH, with a particular interest in non-pharmacologic therapies used for non-migrainous headaches. In the non-pharmacologic group there are two main categories of therapies. The first of these includes psychologically-based treatments involving cognitive or behavioral therapies such as biofeedback and counseling. The second involves CAM therapies, including acupuncture, chiropractic, physiotherapy, massage, homeopathy and others. This discussion will confine itself to the latter category.
My group undertook to analyze all of the randomized clinical trials (RCT’s) of the efficacy or effectiveness of CAM treatments for non-migrainous headache. We defined “non-migrainous headache” as excluding migraine (with or without aura), cluster and any organic types of headache. In this study, we employed standardized methods for literature searching and evaluating the quality of the relevant studies. Quality scores were obtained from an 18-point instrument modified from van Tulder [191] which assessed issues of internal and external validity. Two of my colleagues, a clinician researcher and a statistician applied the instrument to each of the trials. Their scores were averaged for the value shown in Table 9 below.

VI.2. Spinal manipulation trials:

Three RCT’s of spinal manipulation for TTH [192-194], two for cervicogenic headache [195, 196] and one for “post-traumatic headache” [138] were identified. The quality scores ranged from 56 - 80%, with a mean score of 67.5%.

Table 9 reviews these trials. No trial included an exclusively sham or placebo-type control group, so that the “efficacy” of spinal manipulation treatment cannot yet be determined. With respect to determining the effectiveness of spinal manipulation, comparative treatments include soft tissue mobilization [193], resting briefly [193], ice pack [138], amitryptiline [194] and soft tissue therapy [192, 195, 196]. A total of 286 subjects were included in these reports.

There is some inconsistency with regard to the diagnostic classifications used in these studies. The report by Hoyt et al. [193] involved a single manipulative session provided to nine subjects with a concurrent “muscle contraction” headache (versus 13 other control subjects). Jensen et al.’s [138] study was conducted on a small group of subjects with “post-traumatic headache”. Boline et al.’s [194] and Bove and Nilsson’s [192] studies were the only ones to explicitly include “tension-type headache” according to the IHS criteria [39]. The former study included a six-week intervention phase and a
four-week follow-up, while the latter study involved 4 weeks of treatments with no follow-up phase. Nilsson’s study [195, 196] was the only one conducted on subjects with cervicogenic headache.

<table>
<thead>
<tr>
<th>AUTHORS</th>
<th>HEADACHE TYPE</th>
<th>SAMPLE SIZE</th>
<th>NUMBER OF TX’S</th>
<th>TREATMENT GROUPS (n)</th>
<th>RESULTS</th>
<th>SIDE EFFECTS</th>
<th>QUALITY SCORES</th>
</tr>
</thead>
<tbody>
<tr>
<td>Hoyt et al., 1979 [193]</td>
<td>&quot;muscle contraction&quot;</td>
<td>22</td>
<td>1</td>
<td>(1) MANIP =10 (2) MOB = 6 (3) REST = 6</td>
<td>Post-Tx S: (1) -48%*** (2) 0 (3) 0</td>
<td>Not mentioned</td>
<td>56</td>
</tr>
<tr>
<td>Jensen et al., 1981 [138]</td>
<td>post-traumatic</td>
<td>19</td>
<td>2</td>
<td>(1) MANIP = 10 (2) ICE = 9</td>
<td>Post-Tx S: (1) -30.7/100 ** (2) +6.7/100</td>
<td>Not mentioned</td>
<td>60</td>
</tr>
<tr>
<td>Nilsson, 1995 [195]</td>
<td>cervicogenic</td>
<td>39</td>
<td>6</td>
<td>(1) MANIP = 20 (2) STT = 19</td>
<td>Post-Tx E: (1) -3.4 (-59%) (2) -2.1 (-45%) Post-Tx S: (1) -15 (-45%) (2) -10 (-24%)</td>
<td>Not Mentioned</td>
<td>64</td>
</tr>
<tr>
<td>Nilsson, 1997 [196]</td>
<td>cervicogenic</td>
<td>53</td>
<td>6</td>
<td>(1) MANIP = 28 (2) STT = 25</td>
<td>Post-Tx E: (1) -3.2 (-69%) (2) -1.6 (-37%) Post-Tx S: (1) -17 (-36%) (2) -4.2 (-17%)</td>
<td>Not mentioned</td>
<td>72</td>
</tr>
<tr>
<td>Boline et al., 1995 [194]</td>
<td>tension-type headache (IHS)</td>
<td>126</td>
<td>12</td>
<td>(1) MANIP =70 (2) AMIT = 56</td>
<td>Post-Tx E: (1) -3.8/28 (2) -4.0/28 Fol low-up E: (1) -1.0** (2) +5.0 Post-Tx S: (1) -1.3/20 (2) -1.8/20** Fol low-up S: (1) -5 ** (2) +2.0</td>
<td>(1) 4.3% neck stiffness (2) 82.1% dry mouth, drowsy, or weight gain</td>
<td>75</td>
</tr>
<tr>
<td>Bove and Nilsson, 1998 [192]</td>
<td>tension-type headache (IHS)</td>
<td>75</td>
<td>8</td>
<td>(1) MANIP + STT = 38</td>
<td>Post-Tx F: (1) -1.5 hr (2) -1.9 hr</td>
<td>Post-Tx S: (1) No change (2) No change</td>
<td>Not mentioned</td>
</tr>
<tr>
<td>-------</td>
<td>--------------------------</td>
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<td>----</td>
<td>---------------------</td>
<td>-------------------------------</td>
<td>---------------------------------</td>
<td>----------------</td>
</tr>
<tr>
<td>TOTAL OR AVERAGE</td>
<td></td>
<td>286</td>
<td>6</td>
<td>(2) SHAM + STT = 37</td>
<td></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

Legend: (n) = sample size in each treatment group; (IHS) = inclusion based on criteria of the International Headache Society Classification (9);

Treatment types: SHAM = sham placebo treatment, MANIP = chiropractic spinal manipulation, MCB = mobilization, STT = soft tissue therapy, AMIT = amitriptyline.

Outcomes: Tx = treatment; HA = headache; S = severity; F = frequency; mm = millimetres on a visual analogue scale; /wk = per week; wk = week(s); mo = month; (1), (2), = treatment group; for “severity”: mod = moderate; sev = severe; v. sev = very severe
for “frequency”: sev = several; for “global relief”: hi = high, mod = moderate, min = minimal; improv. = improvement; > = statistically significantly better than; NS = not statistically significantly better than.

Statistical significance: NS = not significant, * = .05, ** = .01, *** = .001

As no high quality studies exist which employed an exclusive placebo or sham-control group, the efficacy of SMT for TTH-like or cervicogenic headache cannot be determined. Four high quality studies do exist which compare SMT to other forms of therapy, although two of them have relatively small sample sizes. Three of these studies report a benefit of SMT. In these studies, SMT is more effective than ice pack applications and soft tissue therapy in post-traumatic and cervicogenic headache. SMT appears to be as effective as amitriptyline in producing short-term benefit for TTH; however, there may be a longer-term benefit with SMT once the treatments are withdrawn. In one study, the addition of SMT to a group already receiving therapeutic levels of deep massage did not improve outcomes in TTH sufferers beyond the level obtained by a group receiving the massage and a placebo treatment. This study is the only one to report no additional benefit from SMT.
VI.4. Physiotherapy trials:

Three RCT's were identified involving multi-modality physiotherapy treatment programmes. The quality scores for these trials ranged from 33 - 58% (low - to - moderately high quality). The study with the highest rating (Carlsson et al. [197]) compared physiotherapy treatment to acupuncture. In this trial, “physiotherapy” consisted of a variety of patient-initiated modalities, including relaxation techniques, stretching, TENS and ice therapy, as well as education regarding muscle tension and how to control it “autogenically”. Both treatments produced positive benefit in mood state and overall health function as well as in the intensity and frequency of headaches. Physiotherapy produced greater gains in mood state and in headache intensity.

In both other studies [198, 199], the physiotherapy modalities employed included TENS, heat, massage and ultrasound therapy to the painful areas, trigger point therapy, exercise therapies, biofeedback and education. In Jay et al.’s study [198] all subjects received amitryptiline medication. They reported that subjects receiving the additional physiotherapy treatments fared better than those receiving only the medication. Only the study by Marcus et al. [199] employed a control procedure consisting of education and “skin-cooling” biofeedback. They reported that the combined physiotherapy group “was more likely to experience significant headache relief” than the attention control group (72.7% vs 28.6%, p<.03).
Table 10. Evidence table for physiotherapy studies

<table>
<thead>
<tr>
<th>AUTHORS</th>
<th>HEADACHE TYPE</th>
<th>SAMPLE SIZE</th>
<th>STUDY DURATION</th>
<th>TREATMENT GROUPS (N)</th>
<th>RESULTS</th>
<th>SIDE EFFECTS</th>
<th>QUALITY SCORES</th>
</tr>
</thead>
<tbody>
<tr>
<td>Carlsson et al., 1990 [197]</td>
<td>chronic tension headache</td>
<td>62</td>
<td>60 - 90 days</td>
<td>(1) P/T = 29</td>
<td>$P_g$</td>
<td>Not mentioned</td>
<td>58</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
<td>(2) ACUP = 23</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Marcus et al., 1995 [198]</td>
<td>migraine = 36% tension = 28% combined = 36% (IHS)</td>
<td>25</td>
<td>60 days</td>
<td>(1) P/T + BIOF = 11</td>
<td>$P_g$</td>
<td>Not mentioned</td>
<td>55</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
<td>(2) ATTEN. CONTROL = 14</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Jay et al., 1988 [199]</td>
<td>chronic muscle contraction headache</td>
<td>60</td>
<td>90 days Tx 90 days Follow-up</td>
<td>(1) MEDS+ BIOF (2) + P/T + TENS (3) + P/T ONLY</td>
<td>$P_g$</td>
<td>Not mentioned</td>
<td>33</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>TOTAL OR AVERAGE</td>
<td>147</td>
<td>80 days</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td>47</td>
</tr>
</tbody>
</table>

Legend: As with Table 9. Treatment types: ACUP = acupuncture, P/T = physical therapy or physiotherapy, MEDS = medication, TENS = transcutaneous electrical nerve stimulation, BIOF = biofeedback, ATTEN. = attention control.

In all three studies, (see Table 10) various combinations of these “physiotherapeutic” and “cognitive/behavioral” therapies (as well as medications, in one study) were employed, making the determination of the effect of each of these components impossible. A total of 147 subjects were included in these three studies.
VI.5. Massage trials:

No RCT was found on the effects of manual massage as the primary therapy for non-migrainous headache. The study by Bove and Nilsson [192] employed deep muscular massage to the trapezius and sub-occipital region as a control treatment. Subjects in both groups received this therapy, while they were randomly allocated to additionally receive spinal manipulation or sham treatment. As such, no randomized comparison of massage alone versus another treatment has been reported.

VI.6 Conclusion:

Manual therapies for non-migrainous headache appear to operate by several mechanism. The more general of these involves the relief of pain by endogenous antinociceptive processes [81, 200, 201]. The mechanism by which these therapies may work could be described as “systemic”, and may be activated by massage therapy, as well as some of the relaxation techniques employed within “physiotherapy” (although these were not reviewed in this thesis). The mechanism of these therapies may overlap with cognitive and behavioral therapies which have demonstrated effectiveness [185,187,188].

A second mechanism appears to involve treatments of the cervical spine or cranial muscles as putative sources of headache pain. Manual therapies, including spinal manipulation, mobilization, massage, and other “physiotherapeutic” procedures such as guided exercise and guided postural education appear to target the soft tissues of cervical spine and cranio-cervical junction which may be producing referred head pain.

The other regional mechanism involves therapies directed to the cranial area, including massage techniques and those involved in “alternative medicine” approaches such as cranio-sacral therapy (which, again, has not been reviewed here).
These latter two mechanisms may be described as “local” and appear to involve either
the relief of referred cranial pain from cervical sources or the reduction of local cranial
pain by counter-irritation. These therapies might also exert a relaxant effect on local
musculature.

Vernon et al. [21, 32] conclude that randomized clinical trials of manual therapies for
tension-type and cervicogenic headache have been conducted and reported at a
sufficiently high level of quality to support the existence of the conditions and the
efficacy of this type of treatment. It is claimed that it is not possible to investigate the
benefit of manual therapies with RCT’s, in that, in requiring an appropriate level of
standardization and methodological rigor, compromises may be created to the treatment
context, which may then invalidate the results obtained [32]. While this may be true to
some extent, it would appear that this is not inevitable. Efforts to create placebo-like
interventions were undertaken by Bove and Nilsson [192], by the use of de-tuned short-
wave therapy. The sham procedures used in acupuncture trials (see: full review by
Vernon et al. [32]) could be employed in future manual therapy trials. Finally, the
clinical trial in which the author is currently engaged incorporates sham cervical
manipulation maneuvers comparable to those which have been used in lumbar spine
manipulation trials [202].

It has also been shown that investigators in these areas can develop well-designed,
high quality studies and recruit appropriately large samples of subjects. As this
development evolves, the database of outcomes for at least some of these treatment
approaches should become large enough to conduct meta-analyses so that more robust
evidence-based decisions can be made by practitioners.

Methodological deficiencies certainly exist in these trials and they point to areas where
future clinical trials should be improved. Careful selection of headache subjects
according to explicit inclusion and exclusion criteria following the IHS classification
guidelines [39] should be employed. Provider and subject blinding may be difficult to
achieve in studies of manual therapy treatments, but every effort should be made to blind the treatment allocation from all parties not directly involved in the treatment, particularly the assessors. The issue of long-term follow-up must be dealt with in future trials in order to establish the true value of these treatments to society at large and their impact on the health care system.
VII CONCLUSION

This thesis has proposed the following:
"that mechanical disorders of the upper cervical spine are an important cause of headaches".

Four hypotheses were developed to investigate this thesis.

1. Upper cervical spine dysfunction plays an important role in headache etiology.
2. The importance of this role is more or less, depending on the type of headache.
3. Cervical dysfunction can be assessed and characterized by a range of standard clinical methodologies employed in manual therapy.
4. The cervical dysfunction involved in these types of headaches is treatable by spinal manipulative therapy and this results in clinical benefit for patients with these conditions.

I have presented evidence of four kinds from my published work to support this thesis.

A. An anaesthetized animal model of upper cervical deep somatic inflammation has provided pathophysiologic responses which could be the origins of headaches of cervical origin, particularly the type currently known as "cervicogenic headache".

B. Clinical descriptions have been redefined to reveal common features which could be grouped together and attributed to pathologies or dysfunctions in the regions investigated in the animal model.

C. A new form of questionnaire was developed which produced evidence of an association between headache and neck pathology or dysfunction.
D. An extensive literature review revealed reported associations between headache and neck pathology in diverse works, particularly those involving manual assessment and therapeutic procedures in the cervical spine.

The evidence from such diverse sources strongly supports the hypotheses developed here and my thesis, that mechanical disorders of the upper cervical spine are an important cause of headache.
REFERENCES


140. Mercer S, Marcus D, Nash J. Cervical musculoskeletal disorders in migraine and tension-type headache. Phys Ther 1993;73(105 (abstr)).


Appendix 1

RELATED PUBLICATIONS:

1982 - 2002
Published Papers for this Thesis


**BOOKS AND CHAPTERS**


The Role of Plethysmography in the Chiropractic Management of Costoclavicular Syndromes: Review of Principles and Case Report

HOWARD VERNON, D.C.¹

ABSTRACT
Accurate clinical assessment of the costoclavicular syndrome may be difficult especially since the standard clinical tests may be confusing. The photoelectric plethysmogram greatly improves diagnostic and therapeutic decision-making, and provides an excellent outcome measure of treatment. A description of this syndrome with an illustrative case history is presented. The utility of the plethysmogram is demonstrated as an objective outcome measure of chiropractic manipulative therapy in this case. (J Manip Physiol Ther 1982; 4:17-20)

Key words: costoclavicular syndrome, photoelectric plethysmography, chiropractic manipulation.

INTRODUCTION
The costoclavicular syndrome is probably the least typical and least documented variety of the thoracic outlet syndrome. A wide variety of symptoms may comprise this syndrome (see Table I). The patho-mechanical basis for this syndrome is reduction of volume between the first rib (the “floor” of the space) and the overlying clavicle just distal to the entrance of the thoracic outlet (see Figure 1). Some mechanisms of this reduction are listed below in Table II.

Under such conditions, the brachial neurovascular bundle undergoes biomechanical compromise. Two mechanisms are involved in this process. One involves traction of the neurovascular bundle over the “shelf” of the first rib. In this respect, both neck movements (especially forward flexion) and arm movements (toward the dependent position) will exert traction on the neurovascular structures. The nerve trunks are vulnerable to this stress, and painless paresthesias will result, especially in the ulnar distribution.

The second mechanism involves compression of the neurovascular structures by the clavicle on the first rib. When the arm is pulled downward, this impingement is maximized. This forms the basis for the diagnostic provocation test used to identify this syndrome. Obliteration of the radial pulse and aggravation of brachial symptoms with the arm tractioned downwards is the diagnostic sign (see Figure 2).

However, as with all clinical tests used in assessing the thoracic outlet, the dependence on observer-based assessments of the response of the radial pulse to provocation predisposes to excessive false positive and false negative findings. This is largely because the test is an all-or-none finding, at least with respect to the pulse deficit. A slight reduction of pulse to palpation may either be ignored or deemed insignificant, when in fact this may represent significant reduction of brachial perfusion. More subtle reductions may not be reproducible and cannot be reliably graded for any comparisons because the level at which the reproduction of symptoms in the arm is significant is not well established.

The use of photoelectric plethysmography provides substantial improvement in diagnostic precision in this type of test. Previous investigations have utilized the plethysmogram to focus on cervicobrachial radicular syndromes. These investigations showed that digital pulse anomalies are related to the reflex disturbances in radiculitis.

This instrument provides objective, on-line continuous data in the form of both pulse wave amplitude measurements and a graphic display of the wave form. This provides both quantitative and qualitative assessment of digital pulse response to the costoclavicular provocation test.

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Table 1. Symptoms of the costoclavicular syndrome

- acroparesthesia
- dysesthesia (esp. ulnar)
- radiating pain to the arm and hand
- diffuse aching in the arm
- hypothenar weakness and atrophy
- trophic changes in the hand
- subjective temperature changes (esp. upon provocation)

Table 2. Etiological mechanisms of the costoclavicular syndrome

Structural: Fractures of Clavicle (acute, mal-unioned)
- Anomalous Clavicles
- Anomalous First Ribs
- Hypertrophied Subclavius Muscle
- Abnormalities of the Costocoracoid Ligament

Functional (Biomechanical):
- cephalad subluxation of the first rib
- convexity of cervico-thoracic curvature
- rounded shoulders

The following study describes a subject with the costoclavicular syndrome. It illustrates the usefulness of photoelectric plethysmography in both the selective diagnosis of the syndrome and as an objective outcome measure of treatment effect.

CASE REPORT

Mrs. B, 39, presented with acroparesthesia on the right of three months’ duration. The complaint began after a session of house painting with prolonged elevation of the arms. These sensations presented on the radial side of the arm to the thumb and first finger. There was also a radiation of dull pain over the lateral arm to the elbow as well as neck pain and paresthesias. The condition was aggravated by lying on the right side with the arm elevated. There were no relieving factors. The patient was employed as a secretary.

The examination revealed positive findings on Adson’s maneuver on the right side; painful restriction of neck movements on the right, hypertonicity of the right scalene muscles; cervical spinal fixations and fixation of the first rib. Mild hyperesthesia over the palmar side of the thumb and forefinger was present. All other orthopedic and neurological tests were unremarkable.

The pre-treatment plethysmographic findings are illustrated in Figure 3 revealing the characteristic pattern of the costoclavicular syndrome. Treatment was aimed at correction of the first rib subluxation and scalene muscle hypertonicity. After 12 visits, the patient reported 90% relief from pain and 75% relief of numbness. After
COSTOCLAVICULAR SYNDROMES • VERNON

Figure 4. Clinical improvement in subject No. 1 related to improvement in plethysmographic reading of costoclavicular maneuver.

Figure 5. Plethysmographic recordings of right hand of patient. Note the obliteration on the costoclavicular maneuver.

18 visits this had improved to 95% for both levels. The plethysmogram shows progressive improvement in the costoclavicular test. (see Figure 4).

DISCUSSION

The costoclavicular maneuver differs from the other thoracic outlet tests in that the neurovascular bundle is compressed with the arm in the depressed position, not in the elevated or hyperre elevated position. It follows from this, that individuals who suffer as a result of this syndrome are aggravated with the arm chronically in the dependent position. This is quite contrary to the typical aggravation of paresthesias and pain provoked in the elevated position in the more common scalenus anticus or hyperabduction syndromes.

The clinician must therefore be aware of the following pattern of pulse deficit upon palpation which is a characteristic of this syndrome alone:

- neutral measurement — mildly weakened pulse
- elevation of arm — increased pulse
- maximal depression of — pulse reduced or obliterated arm (costoclavicular maneuver)

The plethysmographic record of this finding is illustrated in Figure 5 where the Adson's and hyperabduction maneuvers are elevated with the costoclavicular maneuver showing reduction.

Manipulative therapy may play a pivotal role in the management of this syndrome. The cephalad, or "bucket handle-up" type (3) subluxation of the first rib is a relatively common finding which responds well to specific manipulative correction. If present, the cervicothoracic curvature can be reduced by exercise and manipulative therapy. Specific exercises to be used are scapular retraction (to strengthen the rhomboids) and scapular elevation (to strengthen the suspensory muscles). Stretching the pectoral group and shoulder rolling exercises help to improve regional flexibility. Thoracic extension exercises may help with the rounded shoulders.

Excellent clinical results were obtained in the case described above. Most importantly, though, the objective evidence of improvements in the thoracic outlet tests is demonstrated by the photoelectric plethysmogram.

CONCLUSION

Unless there is complete obliteration of the radial pulse, the clinical (subjective) interpretation of thoracic outlet testing may be confusing and inaccurate.

Photoelectric plethysmography greatly improves diagnostic and therapeutic decision-making. The costoclavicular syndrome, specifically, is described, and an illustrative case history is presented. The usefulness of photoelectric plethysmography in demonstrating objectively the effect of manipulative therapy on this syndrome is thus described.

ACKNOWLEDGEMENTS

Sincere appreciation is extended to Terry Bernstein for artwork and to Paulette Companion for manuscript preparation.
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Chiropractic Manipulative Therapy in the Treatment of Headaches: A Retrospective and Prospective Study

HOWARD VERNON, D.C.*

ABSTRACT
The treatment of benign, chronic, headache by chiropractic manipulative therapy is investigated in this study. Thirty three subjects were assessed, 15 retrospectively and 18 prospectively. The treatment outcomes used in the study included frequency, duration, severity and treatment satisfaction. These outcomes were assessed by questionnaire. The results indicated statistically significant outcomes in at least nine treatments. The possible mechanisms of action of manipulative therapy are discussed. (J Manipulative Physiol Ther 1982; 5:109-112)

Key indexing terms: manipulation, chiropractic, headaches.

INTRODUCTION
The role of chiropractic manipulative therapy (CMT) in relieving headaches has long been proclaimed by proponents.1-5 Many case reports and review papers have empirically documented such therapeutic successes. The results of these case reports can be generalized to a large patient base that have been successfully treated by chiropractors, without documentation.

Retrospectively, multiple-case studies have documented similar effects of manipulative therapy in larger patient groups than this present study. Wight6 reported 74.7% overall improvement in a sample of 87 migraine subjects. Rose (6) reported 62.5% improvement in 17 patients. Parker,7 in a controlled clinical trial, demonstrated 28% improvement in migraine; however, he failed to demonstrate significant differences between the chiropractic manipulative and control groups. While that study created much controversy, it is by no means the final word on the role of manipulative therapy and headaches. In fact, it is barely an appropriate beginning in this area of research.

To this end, a prospective cohort study is presently underway at the outpatient clinic of the Canadian Memorial Chiropractic College (C.M.C.C.). In order to substantiate a baseline of treatment outcomes and to test both the data-gathering instruments and the framework of statistical analysis for that study, a combined retrospective/prospective study of the effects of CMT was undertaken on intern-treated patients. The results of that preliminary study are presented in this paper.

MATERIALS AND METHODS
Two groups of subjects were assessed. The first group was selected in an unbiased fashion from available files in the C.M.C.C. Clinic during the period of September to December, 1980. A headache questionnaire was devised, with emphasis on quantitative data, which was mailed to the study group. All subjects had completed treatment and so were asked to report on both the pre- and post-treatment status of their complaint. Out of 25 subjects selected, 15 responded sufficiently to be included in the study.

The second group was selected prospectively from concurrent cases in the clinic after being fully informed of the nature of this study. Patients were selected on the basis of complaint alone, not by specific diagnostic category (such as migraine or cervicogenic cephalgia). Pretreatment diagnostic assessment was completed prior to the commencement of therapy. A fixed time-frame of nine visits was chosen in which to evaluate the outcomes of treatment, after which post-treatment assessment was conducted using the same questionnaire as mentioned above. The treatment program consisted of the standard regimen of chiropractic manipulative therapeutics with emphasis on specific spinal adjustment. Of 35 cases so chosen, 18 were complete enough for inclusion in the study. Of the 17 subjects not included, eight reported for less than nine visits before discharge, four produced incomplete or confusing forms, and three did not actually initiate their therapy for non-clinical reasons.

The relevant dependent variables and their indices of measurement are shown in Table 1. The results were
The retrospective study consisted of 18 subjects, 11 females and five males. The mean age was 30.68 ± 8.1 years. The mean duration of the complaint was 9.8 years. The raw scores for each subject are shown in Table 3. The mean scores of the dependent variables were compared before and after nine treatments in Figures 1 through 4. The t-values are shown for each variable indicating a statistically significant difference (p < .05) between each of these scores. The F values for frequency and duration are 10.18 and 13.46 respectively, which is statistically significant (df = 15, p < .01) and indicates greatly improved consistency in the post-treatment scores for these variables. The mean satisfaction score for the group was 12.79, which corresponds with a 90% level of improvement.

Finally, Table 4 demonstrates the incidence of autonomic symptoms which occurred in the sample and their response to treatment. This response is also statistically significant ($x^2 = 15.69, df = 3, p < .01$).

The prospectively studied group consisted of 15 subjects, 14 female and one male. The mean age was 34.2 years. The mean duration of complaint was seven years. The mean number of treatments was 12.08 ± 4.1. The mean values of the dependent variables at pre- and post-treatment assessments are shown in Figure 1. All values decreased significantly to the 99% confidence limit. The satisfaction score was compiled from three subjective questions, as shown in Figure 1: (1) was chiropractic effective in relieving your headache?, (2) what percentage of benefit do you feel you received?, and (3) looking back, would you choose chiropractic if you had it to do over again? The maximum score was 15. The mean score in the sample was 12.5. No significant correlation existed between the length of time (as a function of the number of treatments) and the extent of relief as measured by any of the variables.

### TABLE 1. Headache dependent-variables and their indices

<table>
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<tr>
<th>Frequency</th>
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### TABLE 2. Chiropractic treatment satisfaction scores

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<th>Benefit/Amount</th>
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<td>1 Strongly Disagree</td>
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<tr>
<td>Disagree</td>
<td>2 60%</td>
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<td>Agree</td>
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<tr>
<td>Strongly Agree</td>
<td>5 100%</td>
<td>5 Strongly Agree</td>
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### TABLE 3. Prospective subject data

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<th>Dura­tion (yrs)</th>
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### Figure 1. The retrospective study group comparison of the mean values of the dependent variables before and after treatment. The average subject in this study suffered frequent, long and intense headaches prior to treatment. Significant improvement was demonstrated following chiropractic manipulative therapy.

subjected to the student's t-test for dependent measures with 95% confidence limits.

### RESULTS

The retrospectively assessed group consisted of 15 subjects, 14 female and one male. The mean age was 34.2 years. The mean duration of complaint was seven years. The mean number of treatments was 12.08 ± 4.1. The mean values of the dependent variables at pre- and post-treatment assessments are shown in Figure 1. All values decreased significantly to the 99% confidence limit. The satisfaction score was compiled from three subjective questions, as shown in Figure 1: (1) was chiropractic effective in relieving your headache?, (2) what percentage of benefit do you feel you received?, and (3) looking back, would you choose chiropractic if you had it to do over again? The maximum score was 15. The mean score in the sample was 12.5. No significant correlation existed between the length of time (as a function of the number of treatments) and the extent of relief as measured by any of the variables.
CHIROPRACTIC TREATMENT OF HEADACHES

DISCUSSION

The role of somatic tissues and the mode of action of CMT in adult benign headache is still without precise definition. Early distinctions between muscle contraction and migraine headache types are now being contested. Similarities have now been documented between the two types of headaches with respect to psychogenesis,\(^6\) autonomic dysfunction,\(^5-11\) and most surprisingly, the biochemistry involved in active headache states.\(^12\) As a result, somatic dysfunction may come to play an increasingly important role in headache management.

Manipulative therapists have alluded to numerous components of somatic dysfunction in headache conditions. Among these are:

1. **Vertebral Subluxation (Joint Dysfunction)**
   
   Ligamentous headache is aggravated on anteflexion of the head causing sub-occipital muscle spasm with local, radiating and referred cranial pain.\(^1\)

2. **Regional Myofasciitis**
   
   Hypertonicity and trigger points in the suspensory muscles (trapezius, levator scapula and sternomastoid) and the long occipital extensors frequently refer pain to the cranium and may be associated with secondary symptoms, especially nausea.\(^13\)

3. **Combined Somatic Effects**
   
   Joint pain and muscle spasm may disturb reflex connections via the spinal tract of the trigeminal nerve, causing referred cranio-facial pain and hypertonicity of the temporalis and masseter muscles.\(^14,15\) Trapezius hypertonicity has been implicated in entrapments of the greater occipital nerve, causing referred occipital and temporal pain.\(^16\) The temporomandibular joint syndrome is also well known to involve headache.\(^21\)

4. **Vertebrogenic Autonomic Dysfunction**
   
   This clinical and pathophysiological complex has been described by many authors from many different perspectives. Traditional thought has implicated the role of trauma to the cervical spine resulting in relatively direct mechanical irritation of the posterior cervical sympathetic system and resultant autonomic dysfunction (cervical syndrome of Barré-Lieou). Recent scientific discussion has focused on the interaction between somato-spinal dysfunction, or intervertebral subluxation, and the autonomic nervous system through the somato-autonomic reflexes.\(^17-20\) Facilitation of the lateral horn cells of the spinal cord results in a central excitatory state. The resultant excess sympathetic discharge may result in protean manifestations of distal symptomatology. Most recently, radioisotope studies of monosynaptic connections of neurons in the primate have demonstrated direct connections to the medial vestibular nuclei, hypoglossal nuclei, facial nuclei, reticular formation and lateral cuneatus nucleus. The possible ramifications of direct induction of autonomic dysfunction in the CNS are again protean.\(^22\)

The results of the retrospective study show a statistically significant therapeutic effect on adult benign headaches in an acceptable time-frame of treatment (average \(= 12\) visits). It does not, however, identify when manipulation produces its positive effect, only that it is noticeable after 12 visits. Nor is it possible to relate the treatment causally to the results; since no control group was used, although the results of the study should not have arisen randomly.

In the prospective study, these conclusions are strengthened. Since the data is collected before treatment begins, a reliable baseline is established against which statistical inferences can be made. Also, the fixed time-
frame allows us to assess the treatment effect in a more controlled fashion. The similarly excellent results of this part of the study, therefore, greatly reinforce the conclusion that CMT is efficacious in the management of adult benign headaches. It is well worth mentioning that no side effects or worsening was reported by any of the subjects.

An interesting finding which relates to the previous discussion on vertebrogenic autonomic dysfunction is the extent to which autonomic symptoms prevail in the prospective sample (30.2%) and the extent to which they were relieved by manipulation as shown in Table 4. This lends credence to the hypothesis that CMT has a positive therapeutic impact through modulation in the autonomic nervous system. Also, it indicates that headache patients with demonstrable cervical-spine dysfunction, associated nausea and dizziness, have a good prognosis under CMT. The cohort study, presently underway, will expand on this report by establishing more specific diagnostic categorization and by exploring the effect of treatment in a more controlled setting and with more objective outcome measures.

CONCLUSIONS

Two separate groups of headache sufferers were assessed in an uncontrolled study of the outcome of treatment under chiropractic manipulative therapy. One study utilized a retrospective approach; while the other prospectively assessed subjective improvement. This corroborates previous anecdotal and clinical studies and indicates the need for further controlled studies in this area.

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MANIPULATION AND PAIN TOLERANCE

A CONTROLLED STUDY OF THE EFFECT OF SPINAL MANIPULATION ON PARASPINAL CUTANEOUS PAIN TOLERANCE LEVELS

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INTRODUCTION

The topic of the relief of spinal pain by manipulation has received much attention lately (1, 2, 3). The weight of empirical evidence of the clinical success of chiropractic manipulative therapy has forced a new scientific focus on this issue. Gitelman (4) provided an excellent review of the issue both from an historical and scientific viewpoint. He crystallized the current hypotheses concerning the mode of action of manipulation, emphasizing those mechanisms which have come to be called impulse and non-impulse based. Denslow (5, 6) promulgated the concept of central facilitation as an explanation for pain propagation at the spinal segmental level. Korr (7–9) promoted the importance of sensory feedback from the muscle spindles in the maintenance of central facilitation. Haldeman (10) has reviewed these concepts and emphasizes the role of the interneuronal pools in pain propagation. Will (11) proposed a multifaceted approach towards understanding the mechanisms of spinal manipulation in the relief of pain, integrating most of the known neurological and biochemical mechanisms. Numerous clinical trials have demonstrated relief of spinal pain by manipulation. Manipulation has produced relief in low back pain (12–17), neck pain (18) and headache (19).

Little attention has, however, been paid to the effect of manipulation on experimental pain. Glover (20) studied a phenomenon of hyperaesthesia associated with spinal lesions in low back pain sufferers. He found a zone of increased sensitivity to pin prick lateral to the facet joint of the painful segment. He postulated that this reflected a lowered cutaneous threshold of excitation which he proposed was due to facilitation of cutaneous pain reflexes by nociceptive inputs from the joint receptors. He found this phenomenon disappeared after rotational manipulation to the lesioned segment. However, no quantitation of the lowered pain threshold was attempted, nor was the pain induced experimentally.

With regard to the effect of other conservative therapies, Francini et al. (21) demonstrated a normalization of pain thresholds and H-reflex responses in

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was rejected from further study. Of 53 subjects tested, 50 evidenced at least one zone of maximal pain, so that this became the test site.

The applicator was then set to zero and applied over the skin at the localized segment, 1" lateral to the spinous process. The current was then increased at the rate of 0.2 mA per second. The subject was instructed to indicate the point at which the first painful sensation was perceived (threshold) and then the point of maximal pain which would elicit an aversive response (tolerance) (32). These levels were recorded by an observer who was independent of the measuring and the subsequent procedures.

After determining the threshold and tolerance levels, the investigator placed both thumbs over the paraspinal tissues bilaterally and produced a posterior-anterior joint springing action, starting from T1 down to T10. This is a passive joint play manoeuvre which stresses the motion segment into the zone of "end-feel" at which point a slight and further springing pressure is applied (see fig. 1). This served as the control procedure and as a localizing procedure for the manipulation (as determined by resistance to joint springing). Half of the subjects were randomly chosen for a further manipulative procedure at the test site vertebral level consisting of direct thrust/pisiform contact manipulation (cross bilateral of States (24) (see fig. 2). The manipulation was delivered on the side of the greatest resistance, not necessarily (but mostly) on the side of the zone of increased pain. This was done in order to remain consistent with the clinical imperative of directing a manipulation to the hypomobile aspect of the motion segment. All of the subjects were told that they would

![The bilateral postero-anterior joint springing manoeuvre (see text).](image)
unilateral leg pain syndromes by the application of TENS. Malow (22) showed an increase in pain threshold in the facial area after successful conservative treatment of temporomandibular and facial pain syndromes. Laitinen (23) reported on a case of inhibition of cutaneous nociception by a deep myofascial pain syndrome. While this latter case seems to contradict the phenomenon of lowered pain thresholds associated with somatic lesions, all of the above examples demonstrate that inter-relationships do exist between somatic afferent inputs and cutaneous pain thresholds.

The purpose of this study is to assess the response of paraspinal cutaneous pain tolerance measurements to spinal manipulation. Specifically, the hypothesis investigated is that local paraspinal pain tolerance will increase following manipulation.

**METHODS AND MATERIALS**

In order to proceed with such a study, a model of experimental pain induction had to be constructed. Typical pain study models involve three components: 1) a target tissue; 2) a method of pain induction with an interval scale of measurement; and 3) a treatment modality which acts as an independent variable on every subject. The model chosen for our study was an amplification of that which was used in Glover's study. It involved: 1) the paraspinal cutaneous tissue; 2) electrical induction of pain; and 3) spinal manipulation.

Fifty male Caucasian subjects were studied after informed consent had been obtained. All subjects were undergraduate students at a chiropractic college. The mean age of the subjects was 28.6 years. All subjects were without any spinal pain at the time of the study. The electrical pain induction was achieved with a Siemens Neurotron Stimulator. This instrument delivered a constant current of 110 volts at 60 Hz. The unipolar technique was used with the cathode as the active electrode. The dispersive electrode was placed on the calf.

The current was applied by a large ball-point applicator and was measured in milliamps with an operative range from 0.2 mA to approximately 5.0 mA. Previous assessment had determined that 0.4 mA produced a definite sense of discomfort in most subjects. This level was used as the initial stimulus for the detection of areas of heightened pain sensitivity. The ball-point surface did not provide sufficient surface contact to elicit muscle stimulation, so that the primary component of the stimulus was sensory stimulation of the skin.

After resting prone in a quiet room for 10 minutes, each subject was tested according to the following protocol. The initial 0.4 mA stimulus was applied approximately 1" lateral to the spinous processes proceeding from the first to the tenth dorsal segments on the right. The applicator was drawn slowly over the skin at an approximate rate of 2 segments per second. This was repeated on the left. The subject was asked to indicate whether and where there was a zone of most intense pain. When present, this was localized with a grease pen mark. If no zone of maximal pain emerged after this procedure, the subject...
receive a manipulation. This protocol is summarized in table 1. The threshold/tolerance perception procedure was then repeated at intervals of 30 seconds and 2, 5 and 10 minutes. The maximal tolerance levels were used as the data points for analysis. The mean scores of each group were compared by analysis of variance with 95% confidence limits.

**RESULTS**

The mean age of the control group was 29.2 years. The mean age of the experimental group was 28.1 years. The control group mean baseline value is 1.62 ± (1.13). At 30 seconds, 2, 5 and minutes the values are 1.46 (±.93), 1.46 (±1.02), 1.56 (±1.2) and 1.86 (±1.36). The experimental group values were computed by removing those of two subjects who were inordinately high throughout the study. Their values tended to distort the standard deviations. The mean baseline value was 1.37 (±76); the values at 30 seconds, 2, 5, 10 minutes were 2.05 (±1.1), 2.43 (±2.9), 2.7 (±1.75 and 3.3 (±2.8). A comparison of these mean pain tolerance values is displayed in figure 3, indicating a statistically significant difference (p. < 0.05) between the treated and control groups which reached 140% by ten minutes. Further analysis of the treated group indicated three different kinds of response curves. These are displayed in figure 4.

**DISCUSSION**

Pain tolerance is subject to much intersubject and intrasubject variation (32). We attempted to reduce these sources of variation by selecting a homo-
geneous population, by reducing environmental and psychological stress and by randomly allocating subjects to each of the study groups. All of the subjects were very familiar with manipulation of the dorsal spine and all subjects expected to receive a manipulation so that the potential Hawthorne and Pygmalion effects would be consistent within the whole sample.

The first finding of note is the remarkably high prevalence of at least one area of increased pain sensitivity in the paraspinal area in these asymptomatic subjects (50/53 or 94%). The literature is replete with references to similar findings of paraspinal tender zones, "hyperalgesic zones", areas of decreased galvanic skin response and areas of skin temperature difference, all of which have been associated with empirically demonstrable spinal joint dysfunction. Neumann has described a sub-clinical complex known as "silent somatic
dysfunction" which can be identified by tenderness upon joint pressure and skin rolling techniques (35). Our finding corroborates the presence of such a complex of findings suggesting an underlying sub-clinical facilitation of cutaneous sensory reflex pathways coupled with a biochemical fault in an adjacent spinal motion segment.

With regard to the main hypothesis, the control group mean tolerance levels were unchanged indicating a sustained sensitivity of the cutaneous pain receptors in the paraspinal area. The group receiving a spinal manipulation demonstrated a completely different response with a distinct and progressive elevation in pain tolerance. This effect is noticeable within two minutes and lasts for at least ten minutes.

As discussed previously, there is an emerging model of the relief of spinal pain by manipulation. The role of sensory input of large fiber transmission in modulating the activity of the pain pathways has been well documented (25–28). This is the reflex pathway along which both TENS (29) and acupuncture (30, 31) are believed to operate. Wyke purports that the impact of spinal joint receptor afferentation is a potent component of this pain modulation (33, 34). Korr and Denslow drew attention to the role of the muscle spindle afferents in both sustaining and abolishing segmental central facilitation depending upon output behaviour. They also postulated that spinal manipulation produced short-term bursts of proprioceptive sensory bombardment which could produce secondary effects such as gamma efferent inhibition and inhibition of the pain pathways. This sensory bombardment would occur as a result of instantaneous stretch of the articular and myofascial receptors as their elastic barriers were exceeded in a dynamic fashion.
The results of this study indicate experimental corroboration of this hypothetical model and they establish a behavioural baseline upon which studies seeking to identify the physiological mechanisms of manipulation can be predicated. As well, they indicate a high prevalence of a finding involving a complex of spinal biomechanical and reflex disturbances, the diagnosis of which may be made by an assessment of cutaneous hyperalgesia in the paraspinal area.

CONCLUSION

A model of experimental pain induction in the spinal area has been proposed and used to test the response of paraspinal pain tolerance levels to chiropractic manipulative therapy. A statistically significant elevation of pain tolerance (140%) occurred after manipulation as compared to a control group. This is consistent with previous hypotheses regarding the mode of action of manipulation as a potent therapy in the relief of spinal pain.

SUMMARY

The response of paraspinal cutaneous pain tolerance levels to spinal manipulation has not been studied in an experimental model. This paper proposes
such a model of pain tolerance measurement and describes the results of a controlled study of 50 asymptomatic subjects. The group receiving a spinal manipulation demonstrated a 140% increase in local cutaneous pain tolerance levels which was statistically significant (p < 0.05). This is consistent with previous hypotheses regarding the mode of action of manipulation in the relief of spinal pain.

REFERENCES

In this review article, the scientific biomedical literature related to migraine is examined under the currently proposed etiological mechanisms and classifications of: (1) autonomic/vascular, (2) biochemical/platelets, (3) cellular/immunological/allergy, (4) psychophysiological, (5) neurogenic and (6) somatic. Convention acknowledges a confluence of these components interacting to produce susceptibility to migraine in certain individuals. This paper examines the axis of interaction of the following components: (1) autonomic/vascular, (2) neurogenic and (3) somatic. Each of these models is examined separately, and then the concept of vertebrogenic migraine is discussed.

KEY WORDS: chiropractic, manipulation, migraine, vertebrogenic

Since the writings of Wolff, migraine has been considered to be a disturbance of vascular regulation in the cerebral circulation. Excellent summaries of migraine characteristics and symptomatology have been presented recently by Theissler, Diamond, Graham, and Cohen. As well, the epidemiological characteristics that prevail in our past and present cultures have been described. A number of proposed etiological mechanisms have been previously reported in the literature. The following categories hopefully suffice to organize these etiological classifications: (see references)

1. autonomic/vascular
2. biochemical/platelets
3. cellular/immunological/allergy
4. psychophysiological
5. neurogenic
6. somatic

It is more widely accepted that migraine is really a confluence of a number of these components interacting to produce susceptibility, symptomatology and progressive pathogenesis in specific individuals.

Of considerable interest to this paper is the axis of interaction of the following components: autonomic/vascular—neurogenic—somatic. Each of these etiological models will be examined separately; then a synthesis of these models will be proffered under the rubric of "vertebrogenic migraine" and then vasodilation. Heyck proposed a shunt-theory whereby arterio-venous shunts opened up as a consequence of alterations in arteriovenous oxygen concentrations. O'Brien using Xe inhalation techniques, showed regional variations in cerebral blood flow (CBF) in a migraine attack. Skinjo using intracarotid injections of xenon, demonstrated a decreased CBF in the aura stage and an increased CBF in the headache phase. He also demonstrated a decrease of bicarbonate and an increase of lactate during the headache phase which lead him to postulate that the headache was due to a lactic acidosis and an increase of lactate during the headache phase which led him to postulate that the headache was due to a lactic acidosis from the initial cerebral ischemia (produced by anaerobic metabolism) which lead to a reactive hyperemia.

Feuerstein has recently shown that the temporal artery undergoes dilation upon stress induction, and he has proposed a four-stage model of vascular instability in the genesis of a migraine attack. He postulates a sequence starting with vasodilation—leading to vascular instability—leading to vasoconstriction—leading to the final vasodilation of the headache phase. This sequence has been found to begin four days prior to the headache.

Oleson, on the other hand, questions the vascular model. His studies of intracarotid injections of xenon, showed:

1. a diminution of CBF in the occipitoparietal region which spreads forward and may ultimately effect the entire hemisphere.
2. oligemia was the only blood flow abnormality found during the headache.
3. the oligemia was not sufficient to cause ischemic symptoms which, therefore, could not be due to vasospasm.
4. He suggests that symptoms may arise from the cerebral parenchyma.
Additional evidence for the role of the autonomic nervous system is that migraineurs tend to be "sympathicotonic" and id to maladapt to stress. This tendency may lead to the tial vasoconstrictive activity. Similarities of migraine prodromes to those involved in autonomic dysfunction is that migraineurs tend to be "sympathicotonic" and id to maladapt to stress. This tendency may lead to the tial vasoconstrictive activity.

Lance hypothesizes that "an exhaustion phase" might occur the locus ceruleus output via the descending inhibitory pathway which would then allow the trigeminal system to over vasocontrol and pain transmission systems thus effect the migraine cascade. Lance concludes that "since the loc ceruleus is a central analogue of the sympathetic ganglia, of the accepted migraine triggers could influence locus cerulea activity".

The Somatic model: With respect to the somatic component the migraine complex, attention must be focused on cranio-cervical musculoskeletal structures and their interaction with the spinal segmental and cranial neuromeres. The role the cervical spine in promoting autonomic disturbances well-documented. This interaction is known clinical as the "Cervical Syndrome" or the "syndrome of Ban Lieou". The protean list of symptoms in this syndrome and others that have been documented in relation to the cervical spine bear a remarkable resemblance to those of the migraine prodrome. In these cervical syndromes, afferent stimulation arising from cervical joint or som dysfunction creates a reflex disturbance of autonomic effe activity resulting in imbalanced vasocontrol. Korr and Denslow have established both hypothetically and experimentally the existence of the central facilitated segmental Figar, and Figar and Stary have demonstrated a disturban in peripheral vascular symmetry in the upper limb in associ with cervical radicular syndromes. They have also demonst a normalization of this behavior in response to manipulation the cervical spine. Evidence in other areas of the spine has shown abnormalities of skin temperature and electrical resi and pain tolerance in the response to spinal manipulatio Korr, Haldeman, Vernon, Neuman, and recently Beurger have proposed models of interaction betwe spinal somatic structures and the spinal cord reflexes regard to pain transmission, efferent somatic activity autonomic regulation.

The somatic dysfunctions that would pertain to migraines involve somato-somatic reflex disturbances which create hyperactivity in the distributions of paraspinal muscular hypertonicity in the distributions of paraspinal primary rami, local myalgic tenderness and pain, joint motion disturbance or hypomobility. To this fundamen pattern of somatic dysfunction we may add important reg elements: direct mechanical aggravation of the greater occipital nerves; direct referral of pain in the cranial distributions of the trigeminal nerve and muscular hypertonicity and myalgia in the distributions of the upper primary rami-specifically, the occipito-frontalis muscles.
impressive discussion of these interactions and their potential impact on the migraine complex has been presented by Bouquet et al. Bouquet studied trapezius muscle tone by electromyography and peripheral vegetative tone by measures of blood pressure, heart rate and electrical skin resistance in 12 migraineurs as well as 12 subjects with post-concussional vertigo. He found that sympathetic tonus increases preceded the headache and vertigo crises and were maximal during the headache. As well, he found that trapezius muscle tone increased ipsilaterally to the side of pain and to the side of vegetative disorder. The muscle tonus increases correlated with increases in vegetative tonus. Bouquet proposed the following model to explain the interaction of somatic structures and the migraine cascade: somatic dysfunction in the cranio-cervical area consists of muscular hypertonicity and joint/muscle nociception — this disturbed afference leads to activation of the spino-reticular tract which produces a central facilitation of the autonomic system — from a behavioral point of view this is perceived as a tachycardia, increased aggressivity, hypersudation, peripheral vasoconstriction and increased postural muscle tonus (in short, to the sympathecotonia of Korr) this produces an overall state of vascular dysregulation, an increase in post-synaptic excitation potentials which in the somatic motor pools create excess muscular activity, and, most surprisingly a destabilization of the central aminergic system with increased aminergic discharges. This would lead to a deactivation of the central pain inhibiting pathways and a vicious cycle of escalating dysfunction. Since these behavioral correlates were found to increase in relation to headache behavior it is plausible to view these mechanisms as etiological and not just consequential.

Discussion: This leads to the proposal of a vertebrogenic model of migraine etiology. This model links three phenomena, 1) somatic dysfunction (specifically in the cervico-thoracic spine), 2) disturbance in autonomic tone (specifically a facilitation of the sympathetic aminergic system with increased aminergic discharges), and 3) the migraine cascade of symptomatology as a consequence of vertebrogenic autonomic dysfunction-VAD. This model would operate as follows:

1. Vertebral lesions of the cranio-cervico-thoracic spine create;
2. Pain and altered afference, which creates;
3. Alteration of local segmental reflexes, which creates;
4. Central facilitation and hypersensitivity of autonomic efferent pools (via post-synaptic excitation potentials), which creates the environment for;
5. Vasoconstriction of the posterior cerebral circulation;
6. Increases in circulating levels of catecholamines;
7. Activation of the trigeminal pain transmission pathway;
8. Increases in circulating levels of catecholamines;
9. Vasoconstriction of the posterior cervical sympathetic chain;
10. Indirect cerebral vasoconstriction via stimulation from the locus ceruleus;
11. Indirect cerebral vasoconstriction through the biochemical cascade initiated by increased catecholamine levels which destabilize platelet membranes.

At this point in the discussion, an operational description of this model is needed. There are two such descriptions that can be proffered.

A Somatic dysfunction of the C7-T4 vertebral levels causes joint pain and joint fixation. This leads to local segmental facilitation and consequently, to sustained discharges from the sympathetic chain. This leads to transient cerebral ischemia which under potentiated conditions might reach a threshold level sufficient to create cerebral ischemia which then activates the migraine cascade. Increased catecholamine levels would result from the subthreshold central excitation and would have direct effects on platelet membranes.

B Somatic dysfunction in the upper cervical spine—CO-C2 produces local pain and fixation. The upper cervical neurons become facilitated reducing the inhibitory effect of the descending pain pathways (at least one of which arises from the locus ceruleus) and increasing the facilitation of the second order neurons in the spinal tract of the trigeminus. This leads to transient stimulation of the locus ceruleus, which, upon reaching threshold levels, results in focal and spreading vasoconstriction in the intracerebral circulation which then leads to a secondary vasodilation of the extracranial circulation with cranial pain being mediated by the ipsilateral trigeminal nerve.

Support for these operational descriptions, and thence for the vertebrogenic model of migraine is as follows:

1. Sachse has shown that the most frequent movement restrictions in migraineurs are at CO-C1, at the cervicothoracic junction, and the first rib.
2. Figar, and Figar and Stary have demonstrated the involvement of the cervical spine in the genesis and treatment of vascular abnormalities associated with radicular syndromes and migraines. They have also demonstrated clinical relief of symptomatology in uncontrolled studies.
3. Evidence exists linking the cervical apophyseal joints and vertigo, nystagmus, and tinnitus.
4. Clinical evidence in the form of observational surveys and preliminary controlled clinical trials especially by Parker, Tupling and Wight has demonstrated relief of migraines by spinal manipulation.
5. The entity of "cervical migraine" has been proposed by many authors including Bartschi-Rochaix, Geiger...
graine symptoms by surgical decompression of the lower cervical nerve roots. It was his proposal that compression and irritation of the deep sympathetic fibres was the inciting factor to lead derive primary benefit from spinal manipulative therapy. It is possible, then, to describe three categories of migraine. The first would be those in whom the vertebrogenic pain would be etiological. It is proposed that this group : second group would be those in whom the vertebrogenic component was secondary, but synergistic. It is proposed that : group could benefit from spinal manipulative therapy. The d group would be those in whom the vertebrogenic component was not active, and for whom manipulation would not vide benefit.

knowledgement

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Spinal Manipulation and Beta-Endorphin: A Controlled Study of the Effect of a Spinal Manipulation on Plasma Beta-Endorphin Levels in Normal Males

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ABSTRACT

The role of spinal manipulation in the relief of pain is becoming clearer and more demonstrable as time passes. One approach to this study is the effect of manipulation on the neurochemical mechanisms of antinociception. Chief among these is beta-endorphin, which has been found to produce a wide range of beneficial effects, especially analgesia.

The intent of our study was to demonstrate the effect of spinal manipulation on plasma beta-endorphin levels. Three groups of male subjects were randomly created: the experimental, sham and control groups. All three groups were screened for symptomatology, present use of medications and the present use of innocuous stimulants, such as nicotine and caffeine. A standard protocol involving a 20-min pretest resting period, an intervention and a 40-min test period ensued. The experimental group received a manipulation in the region of the cervical spine; the placebo group received a sham maneuver with no dynamic thrust; the control group received no intervention. Samples were taken by venapuncture at -20, -5, +5, +10 and +30 min. The data were analyzed by repeated measures analysis of variance and by Scheffe’s post-hoc multiple comparison tests. Plasma beta-endorphin levels were assessed by radioimmune assay technique (according to the method described by Harber and Sutton in 1984).

The results of our study demonstrated a small, but statistically significant, increase in serum beta-endorphin levels in the experimental group at the 5-min postintervention point. The levels in the placebo and control groups demonstrated a steady decrease that was distinct from the response in the experimental group. With reservations regarding the sample size, our findings appear to demonstrate a small but unexpected increase of serum beta-endorphin in response to a single cervical manipulation. This finding allows us to hypothesize that the pain-relieving effect of manipulation is, in part, due to a short-term increase in beta-endorphin levels. (J Manipulative Physiol Ther 1986; 9:115-123)

Key Indexing Terms: Manipulation, Endorphins, Cervical Spine.

INTRODUCTION

In the mid-1970s it was shown that endogenous opioid peptides exist that bind to receptor sites in the nervous system and that produce, among other things, profound analgesia (1-4). The first of these identified was beta-endorphin. Since that time it has become clear that many therapeutic modalities exert their pain-relieving effect, in large part, due to their capacity to induce an increase in endorphin levels (5-9). It is also becoming clearer that pain sufferers may, in fact, become endorphin-deficient. Disturbances of endorphin physiology are suspected in the etiology of numerous clinical entities, including migraine (10), postpartum depression (11) and others (12).

During the same time period there has arisen a much more intense scientific interest in spinal manipulation. A firm conceptual and theoretical base had been provided by early investigators in chiropractic (13-16) and...
The studies of the past decade have focused on elucidating the clinical efficacy of spinal manipulation [see the review by Brunarski (20)] and the neurobiological phenomena associated with dysfunction of the spinal motion segment and its treatment by spinal manipulation (21-30).

The currently accepted model of neurobiological effects of spinal manipulation can be described by two major conceptual categories that have been created for this purpose: impulse-based and nonimpulse-based mechanisms (28). The term “impulse-based” applies to phenomena related to reflex behaviors of the spinal neuroregulatory and the central nervous system as they are both sustained and disturbed by motion segment dysfunction. The term “nonimpulse based” applies to phenomena related to the physical or structural status of nerves, especially with regard to compression of neural structures in and around the vertebral motion segment, and to the behavior of materials internal to nerve structures, specifically axonal flow. The model interrelates the structural considerations, especially compression effects, on nerve roots and other neural elements located in the intervertebral foramen and the peripheral nerves, with functional considerations involving pain behavior and sensorimotor reflex patterns governing the behavior of the motion segment and the locomotor system. Manipulation is hypothesized to produce significant short-term bursts of proprioceptive transmission in the large-caliber myelinated alpha-afferent fibers arising from the spinal joint capsules and ligaments (30) and in the muscle spindles of the local paraspinal musculature (28). These large-fiber signals are believed to modulate the interneuronal pool via the dorsal spinal ganglion and the substantia gelatinosa and act to “close the gate” on pain transmission (31). Evidence exists that sensorimotor reflex connections are also influenced by manipulation via stimulation of the segmental motor pools (32, 33). These two sets of behaviors would result in a reduction of pain transmission (via inhibition of the ascending pain pathways) and a reduction of muscle hypertonicity (via inhibition of alpha motorneurons). These are the two most evident clinical effects of manipulation (23-39).

This model has been a useful starting point, but we believe it suffers from being somewhat mechanistic. Less attention has been paid to the role of neurotransmitters in these spinal segmental or central reflex phenomena. Will (34), in his modeling of the effects of manipulation, considered the role of local neurotransmitters, especially serotonin and noradrenaline. Gitelman (21) mentioned the role of substance P in his review of these effects. Haldeman (14) included a summary of neurotransmitters involved in both the segmental and descending inhibitory pathways. A recent review by Pressman and Nickles (35) considered the broad range of potential neurotransmitter effects. By placing spinal manipulation within the generic group of “stimulus-produced analgesic” (SPA) modalities, Pressman and Nickles implicate the same neuroendocrine model as that of transcutaneous electrical nerve stimulation (TENS) and acupuncture (35).

The results of a previous study on pain tolerance levels (29) by one of the authors demonstrated that manipulation can increase paraspinal cutaneous pain tolerance levels. This has lead us to pursue the possibility of the release of a neurotransmitter substance capable of modulating pain transmission as a result of a spinal manipulation.

A review of the pertinent literature reveals only two studies that have examined this possibility experimentally. Luisetto et al. (36) studied endorphin and calcitonin response to chiropractic spinal manipulative therapy in 11 patients with neck and arm pain. Using only pre- and posttherapy measures, they showed no change in basal endorphin levels even though all of the subjects demonstrated significant relief of pain. Richardson et al. (37) studied 31 males and females in a controlled experiment and showed that an unspecified manipulation did not change endorphin levels as compared to a control group. Endorphin levels were measured 1 hr before, immediately after and 1 hr after the manipulation.

Because the neurobiological model postulated for manipulation is very similar to that of TENS and acupuncture, it is useful to review some of the literature in this area. Elkins et al. (38), in a pilot study of the effect of TENS on plasma beta-endorphin levels, propose three mechanisms for the effect of TENS on pain. Two of these are consistent with the model for spinal manipulation: 1) a gating effect of nociceptive afferent stimulation and 2) release of endorphins. Their study showed no consistent changes in plasma beta-endorphin levels after 2 hr of TENS. O'Brien et al. (39), in a study comparing several different frequencies of TENS application, showed no significant differences in pre- and posttreatment or treated vs. control group plasma beta-endorphin levels. Hughes et al. (9) compared low frequency-high intensity to high frequency-low intensity TENS and showed a significant elevation of plasma beta-endorphin levels that correlated with increased evoked potential response. Sjolund and Erikson (40) and Clement-Jones (41) found that TENS increased cerebral spinal fluid (CSF) endorphin levels; however, the latter study showed no increase in plasma beta-
endoorphin levels. Malizia et al. (42) reported that electroacupuncture caused elevations of beta-endorphin in the plasma of healthy volunteers. Cheng and Pomeranz (43) have shown similar elevations with traditional acupuncture. Szczudlik and Lypka (8) demonstrated a decrease in plasma beta-endorphin upon electroacupuncture stimulation. They hypothesized that increased receptor binding might result from the treatment; this would account for a reduction in plasma beta-endorphin. Chapman et al. (44) have shown a lack of reduction of pain tolerance in electroacupuncture subjects upon naloxone blockade, inferring that the analgesia of electroacupuncture is not endorphin-mediated. No doubt experimental variability accounts for a large measure of the conflicting results that appear in the literature.

It is the purpose of the present study to investigate further the hypothesis that spinal manipulation might exert its pain-relieving effect through activation of the endogenous opiate system. In this study we examined the effect of spinal manipulation on plasma beta-endorphin levels in healthy male volunteers.

MATERIALS AND METHODS

Twenty-seven male subjects with an average age of 23 were investigated. The subjects were freshman students at a chiropractic college. Subjects were excluded if they were currently suffering from any kind of disease or ill health, if they were on any prescribed medication or if they had experienced any spinal pain in the 6 months prior to the experiment. After receiving informed consent from the subjects, they were randomly allocated to one of three groups—the control group, the sham group and the experimental group—and their height and weight were recorded. The basic procedure undergone by the control group subjects consisted of resting supine on a treatment table, with the head supported, in a quiet room of relaxed ambiance. After a 20-min rest, the sham group received the following maneuver: The upper cervical spine was assessed in the supine position with joint play maneuvers (45) for intersegmental fixation. The head and neck were then rotated in one direction to the maximal passive range. After another 20-min rest, the sham group received the following maneuver: The upper cervical spine was assessed in the supine position with joint play maneuvers (45) for intersegmental fixation. The head and neck were then rotated in one direction to the maximal passive range. A contact was maintained on the fixed segment, and very slightly oscillatory pressure was exerted on this segment, pressing into the elastic barrier of the structures, but not through it. No audible or palpatory release was elicited. This procedure constitutes a mobilization of the motion segment. The experimental group received the same procedure, except that, after pressure on the segment was introduced, a rotary manipulation was given of a high-velocity, low-amplitude type, taking the joint through the elastic barrier. From this point on, the subject continued to rest supine. Table 1 outlines the measurements that were taken at various times during the 1-hr duration of the experiment. Heart rate and blood pressure measurements were taken manually at 5-min intervals before and after intervention, until the 20-min postintervention mark, after which one more measurement was taken at the end of the experiment. Anxiety levels were assessed by means of a self-rating scale ranging from 1 to 10, with 10 being the highest score. These ratings were done 20 and 5 min before intervention and 5 and 20 min after intervention. Those subjects in the sham and treatment groups were asked whether or not the maneuver was painful.

Blood samples were taken at 15 and 5 min before intervention and at 5, 15 and 30 min after intervention. These blood samples were taken by venipuncture with tourniquet from the antecubital vein. Approximately 8-10 ml was extracted into an EDTA tube. The samples were immediately centrifuged and then frozen. Beta-endorphin levels were determined by an 1125 radioimmunoassay method (47), which involves the extraction of beta-endorphin from the plasma using sepharose anti-beta-endorphin particles; the adsorbed beta-endorphin is then eluted for immediate measurement. The radioimmuno assay method is based on an antibody with high sensitivity to beta-endorphins. A tracer is added, followed by a 16-hr incubation period at 4°C. Phase separation is done in 25 min at 4°C with a precipitated complex of second antibodies carried and with PEG added in a single pipetting step. The antibody to beta-endorphin cross-reacts 100% with human beta-endorphin. The antibody demonstrates less than 5% cross reactivity with beta-lipotropin. The beta-endorphin antibody has no cross-reactivity with other endorphin substrates, dynorphin, leucine, enkephalin, met-enkephalin, ACTH, alpha MSH, prolactin, luteinizing hormone and other pituitary hormones. Normal base-

TABLE 1. Study design

<table>
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<tr>
<th>Time (min)</th>
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<tr>
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<td>Postintervention</td>
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<tr>
<td>10</td>
<td>Postintervention</td>
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<tr>
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<td>Postintervention</td>
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<tr>
<td>20</td>
<td>Postintervention</td>
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Abbreviations: BP, blood pressure; HR, heart rate; ANX, anxiety rating; BL, blood sample.
The serial data from the blood samples, heart rates, blood pressures and anxiety ratings were subjected to repeated measures analyses of variance (ANOVAs) to determine the effects of time and treatment. One-way ANOVAs were used to test for differences among the three treatment groups on the variables height, weight, age and 5-min postintervention beta-endorphin level. Posterior multiple comparisons between pairs of treatment groups were performed using the Scheffe's procedure; the α-level was set at 0.05.

RESULTS

The results from the analyses of these data are summarized in Tables 2–9. The means and standard deviations of the age, height and weight measurements are presented in Table 2 for each group separately and for the whole sample. One-way ANOVAs detected no significant differences among groups on any of these variables.

Table 3 shows that the anxiety levels were relatively low, indicating a general lack of apprehension. Still, there was a significant decrease in anxiety as the experiment progressed (P = 0.001), but there was no differences among groups (P = 0.90). When asked whether the sham or experimental manipulation was painful, no subject responded affirmatively.

The analyses of the blood pressure and heart rates are presented in Tables 4–6. No significant differences among treatment groups were found for any of these three variables (all P > 0.10), but there were highly significant differences among times (all P < 0.0001). All three measures tended to decrease over the first few times and to level off after the intervention.

Table 7 and Figure 1 show the plasma beta-endorphin levels. In order to control for any differences in baseline levels among subjects, each subject’s postintervention levels were expressed as percentages of the mean of the average of the subject’s two baseline levels. These percentage data were analyzed using repeated measures ANOVAs (see Tables 8 and 9) that showed significant differences among groups (P = 0.005) and among times (P = 0.002). Beta-endorphin levels tended to decrease as the time after intervention increased. The experimental group showed higher mean levels than did the control and sham groups. The among-group differences were most noticeable shortly after intervention. A one-way ANOVA showed very highly significant differences among the three groups at this time (P < 0.0001).

Multiple comparisons using the Scheffe’s procedure and a significance level of 0.05 revealed that all three groups were significantly different from one another.
DISCUSSION

Studies such as these possess numerous difficulties and problems in interpretation. The first of these relates to the design of such studies, specifically the use of

<table>
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<tr>
<th>TABLE 6. Heart rate</th>
<th>Treatment groups</th>
<th>Time (min)</th>
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<th>Sham (N = 8)</th>
<th>Control (N = 10)</th>
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ANOVA time effect, P < 0.0001.

<table>
<thead>
<tr>
<th>TABLE 7. Plasma beta-endorphin levels (in pmol/L)</th>
<th>Treatment groups</th>
<th>Time (min)</th>
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<th>Sham (N = 6)</th>
<th>Control (N = 10)</th>
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<td>7.74 ± 1.61</td>
<td>7.36 ± 1.41</td>
<td>7.65 ± 1.48</td>
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</tbody>
</table>

*One observation was missing at the -5 time.

countrol groups and the size of the experimental sample. Luissetto et al. (36) did not employ a control group, nor did they establish a stable baseline before commencement of treatment. The small sample size also limits the reliability of their study. Richardson et al. (37) employed a control group as we did, and the total sample was equal in size to that of this present study. The use of a control group provides a baseline for the total set of experimental conditions, which in this case included the subject's position, the laboratory environment, the subject's expectations and, of course, the blood sampling procedures. Our study employed a further improvement in the form of a sham treatment group. This group not only provides a control for the subject's expectation of the provision of a "treatment" but controls for the effects of treater-subject physical interaction ("laying on of hands") and the effects of passive rotation of the cervical joints and muscular holding elements to the end-range.

The second issue involves the nature of the subjects themselves. Luissetto et al. assessed the clinical progress of a heterogeneous group of middle-aged patients with clinical degenerative joint disease. Richardson et al., in assessing the short-term effect of manipulation on normal subjects, included both males and females. It is possible that intersex and age differences have a confounding influence on the neurohormonal responses under study here. Also, the presence of pain presents a
different and more complex psychosocial context for preliminary studies in this area. As such, our study involved only asymptomatic males in the third decade of life.

A third issue in interpreting these studies is the sequence of endorphin assays. Luisetto et al. provide only pre- and posttreatment mean values, without a control group and without stable baselines. These results are difficult to interpret. Richardson et al. sampled plasma levels at only three intervals: 1 hr before, immediately after and 1 hr after the intervention. The pretreatment measure in this case is solitary and may reflect the state of the subject at entry to the study and not just before the intervention. The other two measurements, we contend, were not taken at the most appropriate times, because a time-dependent sequence of physiological responses must take place in order to induce a release of endorphins, which at the same time, in the framework of Richardson’s design, must therefore be stable enough to induce a large enough response in order to be measurable up to 1 hr later. As we shall discuss below, this situation does not correspond with the physiological response to be expected from manipulation, nor does it take into account the relatively short half-life of plasma beta-endorphin (38). Our study employed two measures within 20 min as a baseline and three postintervention measures to more accurately track the course of the hypothesized response.

Finally, consideration must be given to the natural diurnal variation of beta-endorphin in the human (48). Specifying and limiting the time in the day of the experiment is therefore critical in order to provide a stable range of expected values. Our study was conducted between 0900 and 1200 hr, a period of time in which the expected mean values of plasma beta-endorphin are 80% of the maximum and are in the declining phase of the diurnal cycle. This suggests that endorphin levels should not naturally rise during this time period and that any increases found in an experiment such as ours would be modest due to a “ceiling effect.”

In summary, a very homogeneous study group was employed with two levels of control for our experiment. Measures that reflect the subject’s emotional responses to the experiment all showed either a steady state or even a tendency toward greater response. A slight but statistically significant elevation (8.5% increase) over baseline in plasma beta-endorphin levels in the treated group occurred at a point 5 min after manipulation.

The significant differences between the treated and sham groups are particularly important because they belie the contention that elevations of beta-endorphin levels arise from merely the laying on of hands or from joint mobilization. This decidedly is not the case. This increase, although detectable, is rather low compared to other studies involving exercise, TENS and acupuncture. In a review of the effect of exercise on endorphin levels, Harber and Sutton (7) indicate a range of slight to fivefold increases. The average level reported in their study is in the 100–200% range. Malizia et al. (42) reported an eightfold increase in beta-endorphin levels upon electroacupuncture studies. Hughes et al. (9) report 30–40% increase in plasma beta-endorphin levels using TENS.

We consider, however, that our study approached this phenomenon from a “minimalist” point of view. Only one manipulation was performed, and this constitutes the minimum act sufficient to distinguish sham from active treatment. The amount of somatic stimulation from a single manipulation is therefore the minimum expected experimentally, and as such, our expectations of outcome were correspondingly low. The extent to which this elevation in plasma beta-endorphin clinically manifests itself cannot be determined at this time.

One critical question arising from these results involves the nature of the physiological responses to manipulation, which might mediate an increase in plasma beta-endorphin levels. In other words, what is the neurophysiological rationale behind the original hypothesis of this study? The model of spinal antinociception proposed for TENS and acupuncture would seem an equally fitting one for spinal manipulation. Thus, short-term bursts, presumably of high intensity, of alpha-afferent transmission may activate a “spinal gate” and block pain. The neurons of the substantia

<table>
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<tr>
<th>TABLE 8. Percentage change from baseline level</th>
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<td>Time (min)</td>
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<table>
<thead>
<tr>
<th>TABLE 9. Percentage change from baseline levels</th>
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<tr>
<td>Repeated measures ANOVA</td>
</tr>
<tr>
<td>Source of variation</td>
</tr>
<tr>
<td>Treatment</td>
</tr>
<tr>
<td>Time</td>
</tr>
<tr>
<td>Subject</td>
</tr>
<tr>
<td>Treatment × time</td>
</tr>
</tbody>
</table>
Segmental inhibition of T cells

Activates large fiber transmission

Stimulation of mid brain nuclei

Activation of ascending sensory pathways

Activation of hypothalamic-pituitary axis

Endorphin

Figure 2. Conceptual model of the neurohormonal effects of spinal manipulation.

gelatinosa are enkephalergic and thus, a local spinal or segmental release of enkephalin might be expected with spinal manipulation.

However, ascending tracts are also implicated in antinociception. Spinal manipulation may, indeed be involved in the feedback loop that involves hindbrain nuclei and their descending inhibitory pathways (49). The most cephalad projections purported for acupuncture and TENS involve activation of the hypothalamic-pituitary axis, presumably through somesthetic projections in the thalamus and the sensory cortex (48). Plasma beta-endorphin arises almost exclusively from the pituitary gland (5). Plasma beta-endorphin is purported to act more like a hormone than a neurotransmitter (48) and, indeed, a precise correlation of endorphin increases in the CSF and plasma compartments has been disputed (50). The two compartments may function differently with regard to increases in beta-endorphin produced by various and putative therapeutic agencies; in other words, plasma beta-endorphin is only a selective outcome of a more complete set of responses (as yet poorly understood).

Since our study was confined to plasma beta-endorphin, we conclude that the minimal stimulus generated by our experimental maneuver was sufficient to induce a slight response in the hypothalamic-pituitary axis, resulting in a mild elevation of plasma beta-endorphin. It is possible that this release phenomenon may be dose-dependent. It has been shown that high intensity-low frequency TENS can selectively produce increases in plasma beta-endorphin (39). As such, the quality and quantity of peripheral stimulation may be an important determinant of the pituitary response. A more substantial response than the one found in this study might therefore be induced by a higher dosage of spinal manipulation (either at one time or in series) (Figure 2).

Since plasma beta-endorphin levels do not necessarily reflect spinal met-enkephalin levels (50), then the supposition regarding the segmental effect of spinal manipulation is still tenable. Since the neurotransmitter in the substantia gelatinosa is met-enkephalin (5), procedures to assay this substance in humans should be explored to determine the short-term effect of manipulation on the local spinal-cord reflexes.

CONCLUSION

The hypothesis that chiropractic spinal manipulation activates the endogenous opioid antinociceptive systems has been explored in this study.

Under very controlled and artificial conditions, an experimental group of normal asymptomatic young males demonstrated a small but statistically significant elevation of plasma beta-endorphin levels.

There are numerous contingencies and difficulties with this and other similar studies. The findings of this study should be taken as an encouraging sign to continue investigating this hypothesis linking spinal manipulation to the endogenous opiate system (see Figure 2). Future research into this area is essential. This study should be replicated with a larger sample size. An indwelling catheter should be employed for blood sampling to reduce trauma and pain. Very homogeneous groups of pain sufferers should be similarly studied. Short-term relief of pain should be correlated to increases of beta-endorphins in those patients who respond to manipulation. These studies could also be done employing a pain threshold tolerance instrument (29) that could allow for a study of the effect of naloxone
on pain tolerance levels following manipulation. It might also be possible to develop an animal model in which neurophysiological manipulations (such as spinal tract or dorsal root transections) could be induced in order to investigate the neuroanatomical pathways subserving this phenomenon.

ACKNOWLEDGEMENTS

The authors would like to thank the Canadian Memorial Chiropractic College Research Board, for its generous assistance. The principal author would like to thank Mrs. Sheila Talaga for typing the manuscript, Ms. Margaret Dunn for typing the charts and Dr. Normal Allen for reviewing the text.

REFERENCES

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46. Deleted in proof.
Vertebrogenic Headache

Howard Vernon, D.C., F.C.C.S.

"In my experience, cervical migraine is the type of headache most frequently seen in general practice and also the type most frequently misinterpreted. It is usually erroneously diagnosed as classical migraine, tension headache, vascular headache, hypertensive encephalopathy or post-traumatic encephalopathy. Such patients have usually received an inadequate treatment and have often become neurotic and drug-dependent."

Frykholm (1971)

Headache is one of the most ubiquitous and frequent complaints in our society(1). Headache is the most frequent reason for patients attending their general physician. Medications for headache constitute one of the largest groups of prescribed and self-administered drugs(2). While there are many varieties and categories of headaches, muscular contraction and migraine-type headaches constitute the vast majority of headaches, especially of those seen by primary care practitioners(3).

Chiropractic interest in headaches dates back to the inception of the profession. As early as 1928, Loban(4) gathered anecdotal statistics on the results of chiropractic treatment of migraine. Since that time, a host of chiropractic writers (and other practitioners of manual therapeutics) have addressed the topic. The clinical literature is composed of a variety of studies—from single case studies, multiple case reports, to retrospective, open and controlled clinical trials. While this literature will be reviewed in depth in this chapter, the consensus of these studies, as well as of numerous theoretical treatises on the mechanisms of benign headache, is that the cervical spine plays a very important but largely undervalued role in the etiology of such headaches and that chiropractic treatment can be efficacious in the management of this broad category of headaches.

Despite this consensus of opinion and data, the role of the cervical (especially the upper cervical) spine is poorly understood (and almost wholly unaccepted) by most orthodox practitioners and their patients. According to the data of Kellner, Hall, and Coulter(5), headache constitutes only 9% of the chief complaints seen by Canadian chiropractors. The diagnosis of occipital headache occurred in only 3% of cases in a recently reported survey conducted by a respected Swiss chiropractor in his clinic(6).

In all eight editions of the Swiss Annals from 1960 to 1986, there exist only three articles on headaches(6–8). A review of the typical medical literature on headache reveals very little attention to the role of the cervical spine. In the last three years, the journal HEADACHE has published only six articles on headache and the cervical spine. Three of these dealt with the effects of biofeedback treatment, focusing solely on the large regional musculature(9–11). Three of these do, in fact, deal with the cervical spine as a wholistic entity comprised of bone, joint, muscle, and nerve structures, each component being capable of involvement in headache(12–14).

The current medical paradigms of psychosomatic and vascular etiologies of the vast majority of headaches are, therefore, firmly entrenched (see Figs. 9.1 and 9.2). This is so much so that even when a vertebrogenic origin attributed to the cervical
Table 9.5. Retrospective study group comparison of the mean values of the dependent variables before and after treatment. The average subject in this study suffered frequent, long, and intense headaches prior to treatment. Significant improvement was demonstrated following chiropractic manipulative therapy.

(1) How much improvement did they obtain?
(2) How satisfied were they with chiropractic treatment?
(3) Would they return for similar chiropractic treatments?

These ratings formed a satisfaction index (scored out of 15).

The results of the study in both the retrospective and prospective groups are displayed in Tables 9.5 to 9.9. The levels of statistical significance are very high on all of the indices. The satisfaction index scores range from 12.2 to 12.7 of 15 or 90%. Of seven subjects who reported nausea present prior to treatment, 100% were relieved of that symptom. Similarly, 9 of 9 or 94% of subjects reporting dizziness reported complete relief.

In 1984, Schimek and Mohr(66) conducted a descriptive survey of 78 patients with chronic headache. It is assumed that these subjects were suffering from benign headaches of the muscle contraction, migraine or mixed types. The approach taken in this study was to determine the characteristics of these patients at presentation to a manual therapy clinic as opposed to the outcome of manipulative treatment. The clinical protocol is very similar to that used included a determination of the presence of active and latent trigger points, the presence and degree of "vertebral blockage" patterns of referred pain, and patterns of x-ray findings in the cervical spine. This clinical protocol is very similar to that used by many chiropractors in the examination of chronic headache sufferers.

These authors conclude their study with the following statements: "... pathological nociceptive impulses from a blocked joint increase, reflexly, muscle tension in certain neck muscles. Referred pain develops which may be co-related with a certain
area of the head. . . . Vertebral dysfunction is of primary importance in the development of chronic headache and is considered to be its generator. . . ."

Miller, Maxwell and De Boer, in 1984(67), reported on a single case study of "long-standing, uncomplicated tension headache." According to the authors, 13 adjunctive treatments were rendered, resulting in a reduction in headache frequency from 2.6/day to 1.0/day within one week. Use of analgesic medication similarly decreased.

In 1985, Jirout(68) reported on 200 patients who had previously been involved in a roentgenological study of motion blockage at the C2–C3 segment. Of subjects

with this vertebral dysfunction 90% reported the presence of headaches. Manipulation produced complete relief in approximately 80% of this sample.

Droz and Crot reported in the 1985 Swiss Annals VIII on "Occipital Headaches"(6). A retrospective survey was conducted of clinic files from 1962 to 1963 and from 1973 to 1983. Of approximately 10,000 patient files surveyed, 332 (approximately 3%) were found to suffer from "occipital headaches" (taken here to mean a variant of muscle contraction headache). Average values of number of treatments and case outcome were compiled. The average number of treatments was nine. Treatment outcome was rated as very good (90% relief), good (75%), slightly better (50%), same, or worse. Of the sample, 80% had a very good result, while 10% had a good and 3% had a slightly better outcome. As such, 93% had a positive treatment outcome, while 7% had no improvement. Droz noted that the vast majority of successful cases had no more than 10–15 treatments, so that treatment success can be achieved quickly, but in cases in which the number of treatments exceeds 15, a poorer prognosis is to be expected.

Most recently, Turk and Rotklob (150) have reported on 100 cases of cervicogenic headache treated by manipulation. Nine manipulative treatments produced significant reduction of headaches in 75% of subjects. At six-month follow-up, 65% still had significant improvement while 35% had recurrence of symptoms. A significant reduction of analgesic usage was noted.

The research literature then includes five descriptive surveys (two in the paper by
Lewit) of treatment outcome and three studies describing the characteristics of vertebrogenic dysfunction in chronic benign headache sufferers. Included in these three are one study noting an association between findings of segmental misalignment on static x-ray studies of the upper cervical spine and two studies noting the association of upper cervical vertebral motion dysfunction with chronic headaches.

Migraine Headache

In 1964, Figar and Jarisky(29) studied vascular reflexes of subjects who suffered from "vertebrogenic migraine." This is a diagnostic entity fully accepted by the authors. Vascular reflexes were studied by photoplethysmography of the superficial temporal artery. Both the resting state levels and those evoked as a response to external stressors were studied. Subjects were stimulated by acoustically loud noise, psychological-mental arithmetic, and nociceptive stresses. Nine migraine patients were compared to nine age-matched control subjects. Subjects were studied in between their headaches.

In the normal controls, the vasomotor response of the superficial temporal artery was uniformly and bilaterally vasodilatory. In the patients, 23% showed parallel vasoconstriction, while numerous others showed asymmetric reactions. Nine migraine patients were compared to nine age-matched control subjects. Subjects were studied in between their headaches.

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A landmark descriptive survey of a chiropractor's clinical experience in the treatment of migraine was reported by Wight in 1983(70). In this study, 87 patients (57 female and 30 male) were included, 34 with common migraine and 53 with classical migraine. The results of typical chiropractic treatment regimes were as follows: In the common migraine group, 85% of the females and 50% of the males were greatly improved; in the classical migraine group, 78% of the female and 75% of the male patients were greatly improved. The overall success rate for both migraine categories for both sexes was 74.7%. In those not reporting a greatly improved status after treatment, a reduction of severe attacks was achieved for 81.5% of female and 45.8% of the male patients in the common migraine group, and 43.2% and 34.5%, respectively, of the classical migraine group.

Wight also cites the improvement rates reported in three other chiropractic surveys by Loban(4), International Chiropractic Association, and Rose(72). These are shown in Table 9.10.

The most important study and indeed the only randomized clinical trial on chiropractic manipulation and migraine was conducted by Parker, Tupling, and Pryor in 1978(73). In this study, 85 subjects (33 male, 52 female) with a mean age of 41 years and a mean headache duration of 19 years were randomly allocated to three treatment groups. Of these subjects, 70% suffered common migraine while 30% suffered classical migraine. The subjects received a baseline assessment consisting of psychological tests (especially regarding attitudes toward manipulative therapy), x-ray analysis of the cervical spine, and a migraine diary wherein daily data on headache duration, intensity, and frequency were...
gathered. Medical and neurological tests were conducted to ensure that no organic cause existed for the headache condition. After the baseline examination, subjects were randomly allocated to one of three treatment groups—chiropractic manipulation, medical manipulation, a control/mobilization performed by physiotherapists. Subjects underwent a two-month pretreatment stage, a two-month treatment stage, and a further two-month follow-up period.

This study tested a number of hypotheses which essentially compared the treated and nontreated groups as a whole, compared to each other and separately on the response to headache frequency, severity, and duration. These hypotheses were tested with statistical tests using a probability level of .01.

An average of seven treatments were performed in the treatment phase. The mean improvement level for all three groups was 28%. Only one hypothesis achieved an acceptable level of statistical significance (.01), that being that headache severity (i.e., pain intensity) was reduced in the chiropractic manipulation group. Measures of treatment expectation of the subjects and the treaters indicated a higher degree of expectation of treatment response by the patients and the chiropractors in the chiropractic manipulation group. The authors concluded that such high expectations had a direct effect on pain ratings and, as such, the finding that a single significant hypothesis did attribute treatment success to chiropractors was probably due to a placebo-type response.

This study was the subject of much criticism and independent review. An independent statistical consultant was commissioned by the New Zealand Commission on Chiropractic and asked to revise the statistical tests in order to more properly analyze the available data. Several problems were delineated, which, in the opinion of that consultant, contributed to the negative findings of the clinical trial.

First, the control group did in fact receive a form of treatment, i.e., mobilization, so that a true treatment—no treatment comparison could not be conducted. Second, the alpha level of .01 was very stringent—even higher than that of many medical clinical trials. Third, some of the statistical tests were inappropriate. A revision of the statistical analysis was performed that did not produce an acceptable level of statistical significance in any of the hypotheses, even at a lowered (.05) alpha level. However, the consultant noted that subjects in the chiropractic manipulation group consistently scored higher in every rating than did the other two groups. Given that a relatively low number of treatments was performed on average and that the chiropractic manipulation group was favored in all of the statistical tests, the consultant concluded that the study had a low power. This meant that it was not designed effectively enough to detect a difference between treatment groups when such a difference might, in fact, have been present.

A follow-up study was published in 1980 by Parker and his colleagues that was very much a response to his critics. It was noted that at a 20-month follow-up period, a further 19% of subjects had achieved an improved rating. This constituted a 47% success rate for manual therapy in the treatment of migraine. An interesting aspect of the data that did not receive statistical analysis was that 14 of the subjects in the study achieved a complete recovery. Of these, 8 were in the chiropractic manipulation, 1 in the medical manipulation, and 5 in control/mobilization groups.

The follow-up study attempted to correlate certain psychosocial variables with treatment success. Only one—sex (.01)—achieved the appropriate level of statistical significance to be accepted as a correlate of therapeutic success. In this regard, female patients responded better than male patients to the forms of manual therapy studied by Parker et al.

It should be evident from the preceding discussion that the "Parker clinical trial" raised more questions than it provided answers for. It appears to have been a study with very worthwhile intentions, but which fell short of an important but often overlooked error known as Type B error. This is the statistical error in which a clinical trial fails to demonstrate significant differences when they do in fact exist. In other words, this appeared to have been a false negative trial. Upon reflection, a 47% total success
<table>
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<tr>
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<th>Date</th>
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<td>1. Age</td>
<td>Date</td>
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<td>2. M F</td>
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<tr>
<td>3. Occupation</td>
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<tr>
<td>4. Onset</td>
<td>Total Duration</td>
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<tr>
<td>5. Initial Precipitating Event</td>
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<tr>
<td>1. Trauma</td>
<td></td>
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<tr>
<td>2. Menses</td>
<td></td>
</tr>
<tr>
<td>3. 1st Work stress</td>
<td></td>
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<tr>
<td>4. Childhood</td>
<td></td>
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<tr>
<td>5. Illness</td>
<td></td>
</tr>
<tr>
<td>6. Other</td>
<td></td>
</tr>
<tr>
<td>Typical Headache</td>
<td></td>
</tr>
<tr>
<td>6. Frequency</td>
<td></td>
</tr>
<tr>
<td>Daily</td>
<td>Week</td>
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<tr>
<td></td>
<td>Month</td>
</tr>
<tr>
<td>7. Duration</td>
<td>Hours</td>
</tr>
<tr>
<td>8. Severity</td>
<td></td>
</tr>
<tr>
<td>0</td>
<td>10</td>
</tr>
<tr>
<td>none</td>
<td></td>
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<tr>
<td></td>
<td></td>
</tr>
<tr>
<td>1. Occipital</td>
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<tr>
<td>2. Temporoparietal</td>
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<td>3. Frontal</td>
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<td>4. Facial</td>
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<td>5. Orbital</td>
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<td>6. Vertex</td>
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<td>7. Mandibular</td>
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<td></td>
</tr>
<tr>
<td>1. Neck pain</td>
<td>No</td>
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<tr>
<td>2. Upper back pain</td>
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<td>11.</td>
<td></td>
</tr>
<tr>
<td>1. Unilateral</td>
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<tr>
<td>Right</td>
<td></td>
</tr>
<tr>
<td>Left</td>
<td></td>
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<tr>
<td>2. Bilateral</td>
<td></td>
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<td>12.</td>
<td></td>
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<tr>
<td>1. Focal</td>
<td>No</td>
</tr>
<tr>
<td>2. Diffuse</td>
<td></td>
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<tr>
<td>3. Radiating</td>
<td></td>
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<tr>
<td>13. Character</td>
<td>No</td>
</tr>
<tr>
<td>1. Pressure</td>
<td></td>
</tr>
<tr>
<td>2. Steady ache</td>
<td></td>
</tr>
<tr>
<td>3. Throbbing</td>
<td></td>
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<tr>
<td>4. Stabbing</td>
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<tr>
<td>14. Associated Symptoms</td>
<td>No</td>
</tr>
<tr>
<td>1. Nausea</td>
<td></td>
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<tr>
<td>2. Vomiting</td>
<td></td>
</tr>
<tr>
<td>3. Dizziness</td>
<td></td>
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| 4. Aura | |
| 4.1 Visual | |
| 4.2 Smell | |
| 4.3 Kinesthetic | |
| 4.4 Other | |
| 5. Visual disturbance | |
| 6. Abdominal pain | |
| 7. Photophobia | |
| 8. Phonophobia | |
| 9. Jaw pain | |
| 15. Diurnal | No | Yes* |
| 1. Awakes at night | |
| 2. Morning | |
| 3. Afternoon | |
| 4. Evening | |
| 5. All day | |
| 6. Variable | |
| 16. Precipitation | No | Yes* |
| 1. Hunger | |
| 2. Tension/stress | |
| 3. Fatigue | |
| 4. Weather changes | |
| 5. Movements of head/neck | |
| 6. Certain foods | |
| 17. Aggravation | No | Yes |
| 1. Menstruation | |
| 2. Sneeze/cough | |
| 3. Exertion | |
| 4. Head/neck movements | |
| 5. Motion | |
| 6. Noise | |
| 7. Light | |
| 18. Medications | No | Yes |
| 1. None | |
| Analgesic | |
| 2. Aspirin | |
| 3. Tylenol | |
| 4. Fiorinal | |
| 5. Ibuprofen | |
| 6. Other | |
| 19. Self-Help | |
| 1. Lying down | |
| 2. Massage | |
| 3. Heat | |
| 4. Cold | |
| 5. Food | |
| 6. Other | |

Figure 9.4. Canadian Memorial Chiropractic College Migraine Trial Headache History.
### Past Treatment

20. Previous diagnosis? [ ] No [ ] Yes

21. Past diagnosis?

- Specialist [ ] No [ ] Yes
- 1. CT scan [ ] No [ ] Yes
- 2. EEG [ ] No [ ] Yes
- 3. Other [ ] No [ ] Yes

### Past Treatments

22. Past Treatments

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<th>Treatment</th>
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<tr>
<td>G.P.</td>
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<td></td>
<td></td>
</tr>
<tr>
<td>Biofeedback</td>
<td></td>
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<td></td>
</tr>
<tr>
<td>Acupuncture</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Chiropractor</td>
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<td></td>
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<tr>
<td>Dentist</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Other</td>
<td></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

### General Health

23. Do you have any separate pain in

- Neck [ ] No [ ] Yes
- Upper back [ ] No [ ] Yes
- Lower back [ ] No [ ] Yes

24. Are you taking any other medication?

<table>
<thead>
<tr>
<th>Medication</th>
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<tbody>
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<td>Antidepressant</td>
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<td></td>
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<tr>
<td>Antihistamine</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Other</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

25. Have you had any recent illnesses or operations?

<table>
<thead>
<tr>
<th>Illness/Disease</th>
<th>No</th>
<th>Yes</th>
</tr>
</thead>
<tbody>
<tr>
<td>High blood pressure</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Vascular disease</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Neurological disease</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Other diseases</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

26. Do you have

<table>
<thead>
<tr>
<th>Disease</th>
<th>No</th>
<th>Yes</th>
</tr>
</thead>
<tbody>
<tr>
<td>High blood pressure</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Vascular disease</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Neurological disease</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Other diseases</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

### Lifestyle

27. Are you married? [ ] No [ ] Yes
28. Are you a smoker? [ ] No [ ] Yes

### Diagnosis


### Patient Summary

#### Tension/Muscle Contraction/Cervical

- Migraine
- Tension/Muscle Contraction/Cervical

#### Diagnosis

#### Vertebrogenic Pattern

#### Prognosis for Manipulation/Contraindications

#### Treatment Plan

---

**MECHANISMS OF VERTEBROGENIC HEADACHE**

At this point, I will attempt to synthesize all of the information in the clinical, theoretical, and experimental literature into an integrated and coherent model. This model will be organized along two major lines—anatomical and physiological.

**Anatomical Model**

Table 9.11 displays the components of a generic vertebrogenic model organized along anatomical lines. The major categories of structures include:

1. Extrasegmental—largely involves the large regional musculature and their fascial structures;
Vertebrogenic Headache

**Figure 9.5.** Canadian Memorial Chiropractic College Migraine Trial Headache Diary.

<table>
<thead>
<tr>
<th>Month</th>
<th>Day</th>
<th>Year</th>
<th>Office Use</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

| 1. Did you have a headache today? |   |
| Answer YES or NO |   |

| 2. If YES, how long did it last? |   |
| (in hours) |   |

| 3. How severe was it? (0—10) |   |
| None ................ = 0 |   |
| Mild .................. = 2 |   |
| Moderate ............. = 4 |   |
| Heavy ............... = 6 |   |
| Severe .............. = 8 |   |
| Horrible .......... = 10 |   |

| 4. Rate your activity today. |   |
| Normal ................ = 0 |   |
| Minimal Effect on Activity ............... = 1 |   |
| Interference with work/leisure ........... = 2 |   |
| Absent from work/school ........... = 3 |   |

| 5. Medication taken: |   |
| (a) How much each time? |   |
| (b) How often? |   |

**Table 9.11**

<table>
<thead>
<tr>
<th>An Anatomical Model of Vertebrogenic Mechanisms</th>
</tr>
</thead>
<tbody>
<tr>
<td>Extrasegmental</td>
</tr>
<tr>
<td>----------------</td>
</tr>
<tr>
<td>Regional muscles</td>
</tr>
<tr>
<td>Ligaments</td>
</tr>
<tr>
<td>Deep/short muscles</td>
</tr>
<tr>
<td>&quot;Postural&quot; or configuration causes</td>
</tr>
</tbody>
</table>

\[\text{Diagram of anatomical model}\]
PATIENT NAME ____________________________ AGE ______ M/F ______
Number of Headaches: ______/mo  Headache Duration: ______ hr  Headache Severity: ______
1 2 3 4 5 6 7 8 9 10

STRUCTURAL EXAM:

1) Ranges of Motion (Full = 0, partial = 1, moderate = 2, severe = 3)

<table>
<thead>
<tr>
<th>LATERAL BEND</th>
<th>ROTATION</th>
<th>ANTEROPOSTERIOR BLOCK</th>
</tr>
</thead>
<tbody>
<tr>
<td>L(2)</td>
<td>R(1)</td>
<td>L(2)</td>
</tr>
<tr>
<td>___</td>
<td>___</td>
<td>___</td>
</tr>
<tr>
<td>C0</td>
<td>C0</td>
<td>C0</td>
</tr>
<tr>
<td>C1</td>
<td>C1</td>
<td>C1</td>
</tr>
<tr>
<td>C2</td>
<td>C2</td>
<td>C2</td>
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<td>C3</td>
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<td>C4</td>
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<td>C5</td>
<td>C5</td>
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<td>C6</td>
<td>C6</td>
<td>C6</td>
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<tr>
<td>C7</td>
<td>C7</td>
<td>C7</td>
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<tr>
<td>T1</td>
<td>T1</td>
<td>T1</td>
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<tr>
<td>T2</td>
<td>T2</td>
<td>T2</td>
</tr>
<tr>
<td>T3</td>
<td>T3</td>
<td>T3</td>
</tr>
</tbody>
</table>

3. Fixations (Score 2 for each fix or hyper, X = Not Done. 0 = Normal)

(2) Intersegmental—involves the holding elements of the motion segment: anterior and posterior joint structures as well as the deep, short intersegmental muscles;

(3) Infrasegmental—involves structures in the intervertebral foramen or, in the case of the upper cervical vertebrae that lack true foramina, the bony environs of the nerve roots. Specifically, these structures include the ventral and dorsal nerve roots, the mixed spinal nerve, the dorsal root ganglion, the autonomic fibers, the recurrent meningeal nerve, and the vascular structures of the intervertebral foramina;

(4) Intrasegmental—applies to the spinal cord, in particular, the ascending pain pathways, the anterior horn cells, and those of the sympathetic nervous system. These spinal neuronal pools are involved in reflex connections that govern pain behavior, muscle tone and coordination, and autonomic tone (specifically vasomotor tone), respectively.

An anatomical model of vertebrogenic headache applies these categories to the
Figure 9.7. Data from a single case study of a migraine subject in the manipulated group. Headache frequency.

Figure 9.8. Data from a single case study of a migraine subject in the manipulated group. Headache severity.
craniovertebral anatomy in order to rationalize mechanisms that could be productive of the kind of head pain and dysfunction clinically associated with benign headaches, i.e., muscle contraction, migraine (common and classic), and mixed types. This model will be applied presently to each of these headache categories separately.

**Vertebrogenic Muscle Contraction Headache**

Mechanisms

*Extrasegmental.* The structures involved in this category are the large regional cervicothoracic and cervicocranial muscles, specifically, trapezius, sternocleidomastoid, levator scapula, splenius, and semispinalis capitus, and the occipitofrontalis muscle. In addition, one would include the fascial investments surrounding and interconnecting these large muscles that, as a group (excluding occipitofrontalis), we would define as "regional craniovertebral extensors."

These muscular structures are susceptible to a variety of chronic low-level but accumulative stresses that alter their function and prepare the way for primary or secondary pain states. These stresses arise from postural strain as well as occupational strain and are aggravated when individuals assume slumped postures with cervical hyperlordosis and occipital extension (see Fig. 9.9) as well as seated postures that involve forward tilt of the head and neck (especially with superimposed rotation) and constant upright support of the active arms. These postures are adopted daily by millions of workers in offices around the world (see Fig. 9.10). These muscles may suffer direct trauma as in anteflexion strains or in hyperextension-hyperflexion injuries of the whiplash type. Cervical joint derangement (see below) may also produce muscular dysfunction by inducing secondary arthrogenic contractions.

How do these myofascial dysfunctions produce headache? The most direct mechanism involves irritation of the tendoperiosteal junctions producing low-level chronic foci of tenderness and pain. This produces
Vertebrogenic Headache

local occipital or nuchal and suboccipital pain. Lewit(34) has remarked on "pain over the posterior arch of atlas" and others(38, 84, 85) have noted the presence of tenderness over the insertions of the large regional muscles to the posterior occiput and to the vertebral arches.

The second mechanism involves a chronic myofasciitis of the muscle structure as a whole, which is typically expressed as a low-level hypertonicity with a focal zone of pain and tenderness. This may or may not be accompanied by radiation zones of pain, typically in a cephalad direction into various predictable cranial locations. This phenomenon is referred to by various names but is most commonly known as the "trigger point" phenomenon of Travell(82-84). The trigger point phenomenon may be an actual focal lesion within the muscle structure, typically located in the vicinity of the motor point with local histopathological changes(84), or it may be a zone of referred pain and hypersensitivity resulting from proximal peripheral neuropathy as described by Gunn(81). Regardless of its origin, this myofascial dysfunction produces muscle-specific zones of radiating head pain that may completely mimic the pain of cranial muscular contraction. Some of these typical referral zones, adapted from Travell(147), are displayed in Figures 9.11—9.19.

Muscular hypertonicity or contracture as found in chronic postural dysfunction may also produce a secondary entrapment of accompanying neurovascular structures. There are two such conditions that are associated with headache: entrapment of the greater occipital nerve and the thoracic outlet syndrome. Skillem(23) and Arnold(86) described a syndrome resulting from entrapment of the greater occipital


Figure 9.16. Trigger points and pain referral zone for the upper and middle trapezius muscles. After Travell JG, Simons DG. Myofascial pain and dysfunction. The trigger point manual. Baltimore: Williams & Wilkins, 1983.

Figure 9.15. Trigger points and pain referral zone for the middle and lower trapezius muscles. After Travell JG, Simons DG. Myofascial pain and dysfunction. The trigger point manual. Baltimore: Williams & Wilkins, 1983.

Figure 9.17. Trigger points and pain referral zone for the semispinalis capitus muscle. After Travell JG, Simons DG. Myofascial pain and dysfunction. The trigger point manual. Baltimore: Williams & Wilkins, 1983.
nerve as it pierced the cephalad portion of the trapezius muscle. Bogduk (53) has refined our appreciation of the anatomical basis of this "occipital neuralgia." Entrapment appears to occur where the nerve courses between the insertions of the trapezius and the splenius capitus. Focal tenderness is uniformly present on the occiput unilaterally with sharp, more intense pain radiating forward over the cranium, very often to the frontal region. Conservative therapy (87), myofascial release (85), injection therapy, and occasionally removal of the C2 dorsal root have been proposed in the treatment of this type of headache (86).

According to Raskin et al (12), headache is the leading symptom of thoracic outlet syndrome. They proposed that muscular hypertonicity produced a nonspecific trigger to the existing headache mechanisms, that is, to vascular dysfunction or to cranial muscle tension.

In summary, the large occipital extensors may undergo stresses leading to greater and more persistent degrees of contraction, which then may irritate tendinous in-

**Figure 9.18.** Trigger points and pain referral zone for the semispinalis cervicis muscle. After Travell JG, Simons DG. Myofascial pain and dysfunction. The trigger point manual. Baltimore: Williams & Wilkins, 1983.

**Figure 9.19.** Trigger points and pain referral zone for the multifidus muscle. After Travell JG, Simons DG. Myofascial pain and dysfunction. The trigger point manual. Baltimore: Williams & Wilkins, 1983.
sertions, create focal trigger points with myotomal radiations, and entrap neural structures so that primary and/or referred head pain might arise.

**Intersegmental.** This category refers to the joint structures of the motion segment—posterior ligament complex, apophyseal joints, intervertebral disc, and uncovertebral joints, as well as the deeper short segmental muscles that have been labeled subocciptital muscles in the craniocervical region. Considering first the posterior motion segment, attention focuses on articular and ligamentous nociceptors (Type IV receptors of Wyke)(79) and the intraarticular nociceptors that appear to accompany intra-articular inclusions such as joint meniscioids(88). These nociceptors, when irritated by mechanical or chemical stimuli, transmit local joint pain that may then initiate arthrogenic muscle spasms especially of the segmental musculature(79). In addition, referred pain may arise as a consequence of activation of convergent neurones(89).

This referred pain has been described as a "sclerotogenous" type of referral. Feinstein et al(90) and Kellgren(91) have experimentally irritated facet joints and mapped the corresponding zones of pain. Representative depictions from Feinstein's work of pain referrals from upper cervical joints are shown in Figure 9.20.

Involvement of the cervical and craniocervical joints in headache takes two general forms: first, joint dysfunction and second, osteoarthritis. Joint dysfunction describes a spectrum of mechanical derangements affecting the static and dynamic capacities of a synovial joint. These have been alluded to by manipulative practitioners in all of their writings and have been described as "subluxations" (chiropractic)(146), "fixation" (chiropractic), osteopathic lesion, and joint blockage (medical manipulators). This dysfunction involves a static misalignment, a hypomobility, a paradoxical mobility, or an excess of mobility below the threshold of frank instability. The role of joint dysfunction in headaches was alluded to by numerous authors, some of whom were mentioned in the preceding literature review (see also Ref. 46). Certainly, Schimek and Mohr's(66) description of joint dysfunction as the "generator" of muscle spasm and pain about the head, Boake's(36) description of upper cervical joint dysfunction being responsible for 70% of all headaches, Jirout's(68) finding of headaches in 90% of individuals with C2—C3 dysfunction, and Bogduk's(52) mention of upper cervical hypomobility as the major cause of headaches previously termed "muscle contraction-type" all point to a consensus in the literature on the primary role of joint dysfunction in the production of headache.

Joint derangements of C0—C1, C1—C2, and C2—C3 may initiate direct sclerotogenous referral of cranial pain. The finding of C1 afferents in the facial region(44) gives rise to the possibility of a wide distribution of referred pain from the occiput-atlas segment. Joint derangements cause arthrogenic spasm of the suboccipital muscles, which may then lead to muscular pain syndromes in a manner similar to that described above for the regional musculature.

Pain arising from the upper cervical joints may converge with and facilitate the sec-

second order neurons in the spinal tract of the trigeminal nerve. Pain from upper cervical joint dysfunction may then appear to radiate to the frontotemporal region, an area distinct from the typical "dermatomal" distributions of the C1 and C2 nerve roots.

The second category of joint afflictions involved in headaches is osteoarthrotic changes. Edmeads' review(69) of this aspect of cervicogenic head pain is exhaustive. Suffice it to say that joint pain and stiffness that accompany the degenerative joint process often give rise to headaches.

At least one author(148) has implicated intervertebral disc lesions in disturbances of the spinal tract of the trigeminal nerve. These would likely occur at C2—C3 or C3—C4 as it is doubtful that the spinal tract of V descends any lower in the cervical cord(128). An important feature of osteoarthrosis is the degenerative changes that occur in the uncovertebral joints or the lateral atlantoaxial joints. Osteophytic outgrowths from these joints may affect the C2 nerve root (at C1—C2), the vertebral artery (at C1—C6), and the posterior sympathetic chain that ascends with the vertebral artery. Encroachments on these structures may initiate reflex spasm or occlusion of the vertebral artery or other neurogenic disturbances (see Neck-Tongue Syndrome in Chapter 12).

**Infrasegmental.** This mechanism involves disturbances of the nerve roots, dorsal root ganglia, and preganglionic sympathetic fibers in the environs of the intervertebral foramina or, as in the case of the C1 and C2 nerve roots, in the osseoligamentous environs of the posterior arches of the atlas and axis. Hunter and Mayfield(20) were the first to report on the significance and vulnerability of the C2 nerve root in the genesis of head and facial pains. The earlier chiropractic conception of a "pinched nerve" has given way to the concept of progressive, low-level compressive neuropathy created by a spectrum of pre-degenerate(81) and degenerative changes in the boney environs of the nerve root. Gunn(81) and Luttges and Triano(92, 93) have summarized these progressive alterations in nerve function. Gunn uses the term "denervation hypersensitivity" to describe the functional changes that arise from such a neuropathy. The nerve root is far more vulnerable to lower levels of compression than is the peripheral nerve(93). Luttges and Triano report significant reduction of conduction velocity at 10 times less compression of the nerve root than the peripheral nerve. Gunn's description of these changes is depicted in Table 9.12. The important consideration for our discussion on vertebrogenic headaches is the vulnerability of the C1 and C2 nerve roots, which are not protected by an intervertebral foramen or a nerve root canal. Inflammatory reactions around the synovial

<table>
<thead>
<tr>
<th>Table 9.12</th>
</tr>
</thead>
<tbody>
<tr>
<td>Effects of Denervation Supersensitivity and Chronic Nerve Root Irritation according to Gunn and Luttges and Gerren</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Accord to Gunn*</th>
<th>According to Luttges and Gerren b</th>
</tr>
</thead>
<tbody>
<tr>
<td>(1) Increased sensitivity at motor end plate to circulating neurotransmitters—especially to acetylcholine.</td>
<td>(1) Modification of receptor sensitivity.</td>
</tr>
<tr>
<td>(2) Spontaneous (ectopic) discharge or fibrillation.</td>
<td>(2) Altered fiber projection into cord.</td>
</tr>
<tr>
<td>(3) Increased ability to receive synaptic contacts.</td>
<td>(3) Altered sensory input into cord with subsequent changes in cord cells.</td>
</tr>
<tr>
<td>(4) Disturbed afference.</td>
<td>(4) Altered function of Schwann and other supporting cells.</td>
</tr>
<tr>
<td>(5) Denervated primary afferent fibers may reduce endorphin and enkephalin stores in laminae I, II, and III.</td>
<td>(5) Sprouting.</td>
</tr>
<tr>
<td></td>
<td>(6) Abnormal reinnervation.</td>
</tr>
<tr>
<td></td>
<td>(7) Ventral root afferentation.</td>
</tr>
<tr>
<td></td>
<td>(8) Altered temporal pattern of input.</td>
</tr>
<tr>
<td></td>
<td>(9) Ephaptic transmission (sensory activation by sympathetics).</td>
</tr>
</tbody>
</table>


joints between C0 and C3, hypermobility especially at C1—C2, combined hyperextension/torsional impingement of the C2 dorsal root ganglion (see Chapter 1), and osteophytic encroachments from the posterolateral margins of C1—C2 and C2—C3 joints are all potential sources of nerve root irritation. According to the model of Gunn, these low-level chronic stresses, so much a part of the consequences of the typical pathogenesis of spinal degenerative joint disease, produce referred pains in the cranium, myalgic tender zones, sustained hypertonicity of the craniocervical muscles subserved by these nerve roots, and disturbed afference into the spinal cord resulting in a lowering of the "pain gate"(94) and facilitation of the motor pools. As such, the pain of "muscle contraction headache" may have a decidedly more neurogenic cause than is presently considered.

Included in this infrasegmental set of mechanisms is irritation of the ascending sympathetic chain structures. As Grillo pointed out(7), the autonomic structures are divided into posterior and anterior compartments. The posterior sympathetic structures ascend with the vertebral artery. They are loosely referred to as the vertebral nerve. Compression or rotational irritation can be exerted from the posterior facet joints in the manner described by Kovacs that was presented earlier.

The anterior chain is composed of the preganglionic fibers melding into three cervical ganglia: inferior, middle, and superior. Postganglionic fibers ascend from the superior cervical ganglion into the cranium. A discussion of sympathetic mechanisms in vertebrogenic headache will be presented below in the section on vertebrogenic migraine.

**Infrasegmental.** Normal reflex function and the consequent control of normal physiological responses depend in large part on the nature of sensory input into the spinal cord. Melzack and Wall(94) first proposed that particular patterns and intensities of sensory input might exert a modulating effect on the ultimate transmission of pain in the central nervous system. Korr and his colleagues(95—97, 98) proposed that somatic stimulation could have profound effects on the spinal motor and autonomic pools by creating what was called a state of central facilitation or the "central excitatory state." In this state, more or less constant excitatory postsynaptic potentials combined with a release of inhibitory or suppressive mechanisms serve to increase the "gain" on output behavior of the effector pools or the second-order sensory pathways, thus producing persistent and inappropriate motor and autonomic discharge. This is expressed as sustained muscular contraction, sustained vasoconstriction, and hypersudation, as well as persistent transmission of pain(98).

When these disturbances of central or spinal reflex behavior are considered in the area of the upper cervical spine, attention must focus on the local motor pools and the spinal tract of the trigeminal nerve. The first-order sensory neurons of the trigeminal descend into the spinal cord and terminate on second-order neurons in the C1—C2 and possibly C3 neuromeres (see Figure 9.21). These second-order neurons also receive terminations from the C1 and C2 nerve roots. Boake refers to this situation as an "anastomosis" of nerve endings"(36). The practical considerations, for our purposes, are that disturbed or nociceptive impulses from the C1 and C2 nerve roots (via any of the sources of somatic irritation discussed above) will converge on and facilitate—by spatial and temporal

![Figure 9.21. Synapse of C2 and C3 afferents with the spinal tract of the fifth cranial nerve in the nucleus subcaudalis.](image-url)
summation—the second-order neurons of the spinal tract of the V. In the discussion below on vertebrogenic migraine, further central terminations from the locus ceruleus will be discussed. Thus, referred pain in the trigeminal sensory distribution can arise from upper cervical joint dysfunction. Facilitation of the motor pools would create sustained muscular hypertonicity in the craniocervical region consistent with the model of the arthrogenic reflex of Wyke(79).

In summary, central facilitation of the upper cervical cord by somatic input from joint dysfunction would create pain and muscle contraction in the cranial region—again, pointing to a neurogenic etiology of muscle contraction headache pain contrasted with the psychogenic etiology of primary muscle contractions in the craniocervical muscles as proposed in the current medical model.

Vertebrogenic Migraine

There are several mechanisms that are of particular interest to a vertebrogenic model of migraine.

Mechanisms

**Intersegmental.** Osteophytic or facet joint encroachment on the vertebral artery, vertebral nerve, or sympathetic plexus has been implicated by numerous authors including Bartschi-Rochaix(17), Kovacs(24), Frykholm(37), and Edmeads(69) in the production of migraine-like prodromata and pain.

**Infrasegmental.** Irritation of the postganglionic fibers in the lowest cervical intervertebral foramina has been implicated in the production of cerebral vasoconstriction. Ghavamian(35) reported on relief of migraine symptoms in individuals undergoing surgical decompression of the lower cervical nerve roots. Direct irritation of the superior cervical ganglion(108) would produce vasoconstriction of the internal cerebral vasculature along with a host of other "vegetative symptoms" that have as their basis reflex reduction of blood supply to various cranial structures(71). This direct irritation can occur in posttraumatic states or possibly as a result of mechanical rearrangement (torsional hypermobility) of the atlantoaxial joint(99).
**Intrasegmental.** The vertebrogenic model of migraine proposes that disturbed somatoautonomic reflexes can be responsible for disordered cerebrovascular tone—specifically for the vasoconstrictive phase of the migraine cascade. Clinical and anecdotal observations support the existence of symptoms arising from the cervical spine, which are mediated by excessive discharge from the cervical sympathetic system (see Chapter 10).

**Basic Scientific Evidence**

It has been known for over 25 years that the arterial vessels on the surface and inside the brain are innervated by sympathetic nerve fibers that arise from the superior cervical ganglion (see Figure 9.22). Rosenblum (100) and Krogi (101) provided early experimental evidence for the effect of neuronal influences on cerebral circulation. Several studies in the early 1970s (102–106) convincingly demonstrated the presence of adrenergic receptors on the pial vessels. The functional role of this innervation has been the subject of much study and debate. As reviewed by Wei et al, some of the early studies showed that pial vessels were responsive to chemical or electrical stimulation of the adrenergic fibers that terminated in their vessel walls (107), while other studies could not demonstrate such a response. Kobayashi, Waltz, and Rhoton (108) found that stimulation of the cervical sympathetic trunk produced ipsilateral reduction of cerebral blood flow in cats as well as arteriolar constriction. Inappropriate or dissociated responses of cerebral blood flow and arteriolar caliber also occurred leading them to propose "that vessels smaller than 50 microns are largely responsible for the regulation of cerebral blood flow (CBF), are innervated by sympathetic fibers, and may react to sympathetic stimulation differently than do larger arterioles. Impairment of vascular reactivity by stimulation may be an acute phenomenon induced by repetitive vasoconstriction" (Ref. 108 p 302). Wei et al on the other hand found that only arterioles larger than 100 microns were responsive to stimulation and manifested a narrowing of the vessel caliber (i.e., vasoconstriction). They found that physiological levels of experimental stimulation produced an average of 7% reduction in vessel caliber with a maximum of 18% under potentiated conditions. They concluded that such vasoconstriction could account for a "modest reduction in cerebral blood flow" (Ref. 107 p 654).

As reported by Rubin et al (109), a study

![Figure 9.22. Sympathetic nerve supply in the cervical spine.](image-url)
by Meyer et al [110], demonstrated a 30% decrease in the arterial flow in the internal carotid artery in the monkey upon stimulation of the cervical sympathetic trunk, while Edvinsson et al [102] found that regional cerebral blood flow is reduced in response to electrical stimulation of the cervical sympathetic nerves and that the magnitude of this response was related to the amount of local perivascular innervation. It is agreed that numerous factors determine the arterial response to sympathetic stimulation including vessel size [107] and the number of vasodilatory mechanisms and local circulating metabolites [106] (also see: 123–127, 129).

This brief review of the neurophysiological mechanisms of cerebrovascular regulation prompts us to ask about its clinical relevance or application. It is obvious that if the vascular model of migraine is to be accepted, then these experiments support the contention that there is a role for the autonomic nervous system in the etiology of migraine. Rubin et al have written an important work that both reviews the implications of the “autonomic theory” of migraine and provides evidence in support of this theory. This theory is described as follows: “The neurological symptoms of the prodromal phase of classic migraine result from vasoconstriction due to the neurally released noradrenaline acting on alpha and beta adrenoreceptors. The vessels ultimately dilate possibly assisted by the presence of vasodilator metabolites accumulated during the constrictor phase or perhaps by a reflex inhibition of the constrictor drive” (Ref. 109 P 40). This theory is markedly in contrast to the biochemical model that ascerts the role of serotonin as derived from altered platelet membranes in the vasoconstriction of cerebral arterioles [111]. Rubin et al measured pupillary function and psychological measures of neuroticism, anxiety, and depression. They found a sympathetic dysfunction consisting of a hypofunction of the sympathetic nervous system under stress in common migraineurs. This hypofunctional disturbance of autonomic tone was correlated with higher states of personality dysfunction, i.e., higher neurotic and depressive tendencies. They assert that since the autonomic nervous system is the mediating system of emotional behavior in the physiological milieu of the body, emotional disturbances might manifest themselves as a disturbance of autonomic regulatory tone sufficient to produce the episodic and acute disruptions in cerebrovascular circulation characteristic of migraine.

The vertebrogenic model of migraine accepts the “autonomic theory” but asserts a somatic/neurogenic source of disturbed autonomic regulatory function. Is there experimental support for this contention?

Bouquet et al [57] have studied somatic and vegetative parameters thought to be associated in the etiology of migraine. They studied trapezius muscle tone (by electromyography) and peripheral autonomic tone (by blood pressure, heart rate, and electrical skin resistance) in 12 migraineurs and 12 subjects with postconcussional vertigo. They found that trapezius muscle tone always increased on the side of increases in sympathetic tone that appeared in the period antecedent to a migraine or vertigo crisis. Trapezius muscle hypertonicity was always ipsilateral to the side of pain in the migraineurs. The muscle tone increases were correlated with increases in vegetative tone. Bouquet et al proposed the following model to explain the interaction of somatic structures and the autonomic system in the genesis of the migraine cascade: somatic dysfunction in the craniovertebral region (consisting of muscular hypertonicity and joint/muscle nociception [66] creates disturbed afference (noxious irritation), which leads to activation of the spinoreticular tract that produces central facilitation of the autonomic system. From a behavioral point of view, this is experienced as tachycardia, increased aggressivity, hypersudation, peripheral vasoconstriction, and increase in postural muscle tonus (i.e., the state of “sympathicotonia” of Korr) [95], (also see below). This produces an overall state of vascular dysregulation, an increase in postsynaptic excitation potentials that in the somatic motor pools, creates excess muscular activity and, most surprisingly, a destabilization of the central aminergic system with increased aminergic discharges. This would lead to deactivation of the central pain inhibiting pathways and a vicious
cycle of escalating dysfunction. Under potentiated conditions, this would lead to cerebral vasoconstriction via discharges from facilitated preganglionic cells.

These experimental findings are predicted by the “facilitation theory” of Korr(96), which states that nociceptive somatic input will facilitate the segmental effector pools (motor, autonomic, pain pathways) leading to abnormal input/output relationships. Hinoki and Niki(112) have found that excessive sympathetic discharge is a cause of muscular hypertonicity by causing overexcitation of beta-receptors in injured neck muscles of subjects suffering from postwhiplash cervicogenic vertigo. These findings probably parallel those of Bouquet et al and allow us to conceptualize a vicious cycle of increased sympathetic and motor tone leading to transient cerebral vasoconstriction and cervicogenic vertigo. When this information is integrated with that describing the excitation of the peripheral sympathetic trunks in the cervical region (especially the superior cervical ganglion and the “third neuronal sympathetic pathway,” according to Vijayan(45), then a very plausible mechanism emerges, which causally links somatic dysfunction to the autonomic disturbance that is responsible for the migraine phenomenon.

This somatoautonomic model can also be integrated with the emerging neuronal model arising from the work of Lance et al(113), Bates et al(114) and De U Torre et al(115) among others(135).

This work involves the experimental delineation of a neuronal center—the locus ceruleus—as the source of neural control of the migraine cascade. Their work on cats and monkeys has shown that low-frequency stimulation of the locus ceruleus produces vasoconstriction of the internal cerebral vasculature; and increasing frequencies of stimulation produce vasodilation of the external carotid artery and its branches. These changes are predominantly ipsilateral. The vasoconstriction was presumed to be effected by noradrenaline since “the locus ceruleus is known to project to the cerebral cortex via noradrenergic fibers”(113). The vasodilation was found to be mediated by the facial and greater superficial petrosal nerves “employing a non-cholinergic transmitter agent.” This work also highlights the importance of the trigeminal nerve in the production of vasodilation of the external carotid circulation and in the modulation and transmission of pain in the frontotemporal region, which is so typical a location for the migraine headache. The trigeminal sensory system interacts with the second-order neurons found in the first to third cervical neuromeres via the spinal tract of the trigeminus. These same second-order neurons receive pain afferents from the first to third cervical roots.

The locus ceruleus projects a noradrenergic pathway to the upper cervical spinal cord that inhibits pain transmission. Lance et al hypothesize that “an exhaustion phase” might occur in the locus ceruleus output via the descending inhibitory pathways, which would then allow the trigeminal system to override vascular control and pain transmission systems thus effecting the migraine cascade. Lance et al conclude that “since the locus ceruleus is a central analogue of the sympathetic ganglia(113), any of the accepted migraine triggers could influence locus ceruleus activity.”

In summary, two operational descriptions consistent with the clinical and experimental literature can be proposed:

(1) Somatic dysfunction of the C7–T4 vertebral levels causes joint pain and joint fixation. This leads to local segmental facilitation and consequently, to sustained discharges from the sympathetic chain. This leads to transient cerebral ischemia, which under potentiated conditions, might reach a threshold level sufficient to create cerebral ischemia that then activates the migraine cascade. Increased catecholamine levels would result from the subthreshold central excitation and would have direct effects on platelet membranes.

(2) Somatic dysfunction in the upper cervical spine-C0–C2 produces local pain and fixation. The upper cervical neuromeres become facilitated reducing the inhibitory effect of the descending pain pathways (at least one of which arises from the locus ceruleus) and increasing the facilitation of the second-order neurons in the spinal tract of the
Vertebrogenic Headache

**Figure 9.23.** Somatoautonomic model of migraine etiology. Dotted line, somatosympathetic positive feedback cycle.

trigeminus. This leads to transient stimulation of the locus ceruleus, which, upon reaching threshold levels, results in focal and spreading vasoconstriction in the intracerebral circulation, which then leads to a secondary vasodilation of the extracerebral circulation with cranial pain being mediated by the ipsilateral trigeminal nerve.

Since, as Rubin et al have stated, emotional or psychogenic factors are also extremely important correlates of autonomic dysfunction and migraine(109), it is most appropriate to propose that the somatic and psychogenic components discussed above act in an integrated fashion, one potentiating the other in the manner described in Figure 9.23.

**The Vertebrogenic Model: An Integrated Model?**

While the Ad Hoc Committee on Headache definitions for muscle contraction, common and classical migraine, and mixed headache are undoubtedly distinct and, excepting the latter, nonoverlapping(116 and 136), there is a spectrum of thought on the etiology of benign headache types that ranges from a set of totally distinct etiological theories for each type to an integrated model that proposes a single underlying etiology or etiological complex for these various headache types. Exemplifying the former is the work of Olesen and his colleagues and Rose(117, 118, 119) in which it is proposed that even common and classical migraine are distinct both symptomatically and etiologically. At the other end of the spectrum, Featherstone(120) has asked whether muscle contraction headache and migraine are separate entities. He has proposed a "severity model" for which he supplies supportive data in the finding that various clusters of different headache symptoms do not correlate well with specific diagnoses such as muscle contraction headache or migraine, but that severity of a large pool of common symptoms does distinguish the groups. Muscle contraction headache sufferers experience the same type of symptoms as do migraineurs, however, the symptoms are far less severe, and in some cases, insignificantly so, than those of migraine sufferers. Muscle contraction and common and classical migraine are then linked to one another on a severity spectrum from low to high (see Figure 9.24 and Refs. 130, 131, 137).

Ramirez(121) has recently reviewed the biofeedback literature and draws attention to the tenuous nature of the assumption that muscle contraction headache results from increased tonicity of the craniocervical musculature. He cites differential and contradictory results from the therapeutic literature, as well as evidence of a lack of correspondence between high-tension states and head pain and low-tension states and lack of head pain(122). His conclusion is consistent with that of this author, that "increments in muscle tension are an effect of headache pain rather than the cause" (Ref. 122 p 339). While he does not propose an alternative mechanism for the production of headache pain, and while he ultimately favors psychosocial interventions in the treatment of headache, his articulation of an emerging hypothesis debunking the traditional model of muscle contraction headache is supportive of the etiological mechanisms proposed by the vertebrogenic model, i.e., that headache pain is
derived from noncranial sources of somatic pain, particularly from the upper cervical spine, which are referred to the cranial area.

Can the vertebrogenic model be interfaced with the severity model? And if so, would this increase the validity of the model itself? It is my proposal that a mechanism can be proposed that fulfills the requirements of the severity model, which are that a lower order activation of this mechanism would find, as its expression, muscle contraction headache and a higher order activation of the same mechanism would produce a set of consequences whose increasing severity would express itself as the migraine phenomenon. Simply put, this mechanism would have to produce, at one end of the spectrum, somatic head pain, and at the other end, somatic head pain plus vascular dysregulation. Figures 9.25 and 9.26 depict a model that conceptually links somatic dysfunction with facilitation of the central nervous system, which in a progressive fashion, would produce the necessary spectrum of events.

This model also allows us to propose that the somatic dysfunction-facilitation complex may act as a potentiator of other etiological processes. The effect of psychogenic stressors on the craniocervical muscles would be potentiated by coexisting somatic dysfunction. This would apply equally to the responsiveness of the sympathetic system to a variety of stressors that have been causally linked to migraine.

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**Figure 9.24.** Headache severity model. Adapted from Featherstone HJ. Migraine and muscle contraction headaches: a continuum. Headache 1985;25:194–198.

**Figure 9.25.** Facilitation theory of somatic dysfunction applied to a chiropractic model of benign headaches.
CONCLUSION

It is the conclusion of this paper that the head pains of "muscle contraction headache sufferers" may in fact be the pain referred to the cranium by somatic structures in the craniocervical region, (Fig. 9.27) and somatic dysfunctions in this same region may be responsible for the autonomic dysfunction of migraine. (Fig. 9.28).

This allows us to propose three categories of headache sufferers:

1) Those in whom the vertebrogenic component is etiological. This group ideally will derive primary benefit from spinal manipulative therapy;

2) Those in whom the vertebrogenic component is secondary but synergistic. This group ideally could benefit from...
spinal manipulative therapy in conjunction with other therapeutic measures;

(3) Those in whom there was a negligible vertebrogenic component and for whom manipulation would provide no benefit.

The challenge of the future is to design effective research strategies with the following objectives:

(1) To determine the clinical ramifications of the vertebrogenic model and to demonstrate to what extent chiropractic manipulative therapeutics might be efficacious in the treatment of benign headaches;

(2) To further explain the mechanisms whereby vertebral dysfunction might promote headache and spinal manipulation might relieve it. As this is accomplished, the ultimate objective of integrating chiropractic management with orthodox therapies in the treatment of benign headache will be accomplished.

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Upper Cervical Syndrome


Reviews of the Literature

Spinal Manipulation and Headaches of Cervical Origin

Howard T. Vernon, D.C.

ABSTRACT
The role of the cervical spine in headache remains controversial. Often confused as tension or common migraine headache, headaches arising from the neck pose a diagnostic and therapeutic challenge. Practitioners of spinal manipulation have reported very satisfactory results, although the only published randomized controlled trial did not demonstrate that manipulation was significantly helpful. This article reviews the published clinical studies of manipulation in the treatment of tension and migraine headaches. The topic of cervical headaches in general is reviewed and the current model of cervicogenic headache is critiqued. A representative case history is used to illustrate the thesis that the current model of cervicogenic headache may be too restrictive. The role of spinal manipulation as a trial of therapy in individual patients is also discussed. A retrospective diagnosis of cervical headache can often be confirmed by a successful outcome. (J Manipulative Physiol Ther 1989; 12:455-468).


INTRODUCTION
The role of the cervical spine in the etiology of headache remains controversial and confusing. The standard medical etiological paradigm of migraine is accepted as vascular (1), and that of muscle contraction as psychosomatic (1). It thus can be understood that the contribution of the somatic structures of the cranio-cervical spine has largely been ignored by most, considered to be of minor significance by some, and emphasized by only a very few. Even so, the body of literature supporting a cervicogenic origin of headache is substantial, and the case made for greater recognition of the involvement of the cervical spine is compelling. Recent writers addressing this issue, including Bogduk (2-4), Edmeads (5), Farina et al. (6) and Sjaastad and his colleagues (7-9), have added much to our current understanding. However, even these authors appear to have included only a small portion of the supportive literature in their reports, leaving a diminished sense of the historical attention and the current clinical importance of this category of headaches.

This article has two main objectives. The first is to present a complete review of the data from clinical studies investigating the effect of spinal manipulation and the methods of investigation of its practitioners in the treatment of headaches. The second objective is to present a critical review of the current literature on what has come to be called “cervicogenic headache.” By reviewing the above and by presenting an illustrative case history, an argument will be made that the current description of “cervicogenic headache” attributed to Sjaastad et al. may be too narrow, and its use too restrictive.

DISCUSSION
Muscle Contraction Headache
In 1971 Lewit (10) studied 41 late adolescents (21 females and 20 males) who had sustained cervical spine anteflexion injuries in gymnastic activities and who were now suffering from headache. Seventy-one percent of these subjects were found to have “fixation” (of the cranio-cervical joints) compared to 53% who were found to have hypermobility. Throughout this discussion, fixation will be defined as a reversible restriction of the spinal motion segment (11), while hypermobility will...
refers to an increase in motion short of frank instability. Lewit reported that manipulation was to be the single most effective treatment from a number of physical modalities. In this same study Lewit also reported on a series of 93 adults who had sustained head/neck trauma. Ninety-six percent of these subjects demonstrated movement restriction in the upper cervical spine. Again manipulation was reported to be very successful in treating the headache associated with these injuries.

In 1980 Ng (12) investigated the association between static vertebral misalignment, as viewed on plain film X-rays, and the incidence of occipital headache. Thirty-eight subjects suffering exclusively from occipital headache were studied (10 males, 28 females). These subjects were compared to an age and sex-matched control group. Lateral inclinations of C1, C2 and C3 were measured using a standardized X-ray marking technique. Lateral curvature of the entire cervical spine was also measured. Statistically significant differences were found between headache sufferers and controls in the incidence and degree of lateral tilting at C1 and C3, while a tendency toward a similar distortion existed at C2. No differences were found in the lateral spinal curve in the two groups. Ng concluded that abnormalities of alignment of the upper cervical spine may be associated with occipital headaches.

Mannen (13), reported a single-case study in which nonvascular headache was relieved by a course of chiropractic treatment.

Vernon (14), in a descriptive survey, studied two groups of headache sufferers presenting at a chiropractic college outpatient clinic. One group (n = 15) was studied retrospectively by mail survey, while the other group (n = 18) was studied prospectively within the clinic. Headache sufferers were asked to rate the level of headache frequency, duration and severity, as well as the presence of four symptoms; nausea, dizziness, tinnitus and aura. Subjects were also asked to rate the level of subjective improvement by rating their response to three questions. These ratings were made for the condition before and after treatment:

1. How much improvement did they obtain?
2. How satisfied were they with chiropractic treatment?
3. Would they return for similar chiropractic treatments?

These ratings formed a satisfaction index (scored out of 15).

In the prospective group, a fixed number of treatments (nine) was used. The results of the study in both the retrospective and prospective groups demonstrated statistically significant reductions in all headache-related activity. The satisfaction index scores ranged from 12.2-12.7 out of 15, or 90%. Of the seven subjects who reported nausea present prior to treatment, 100% were relieved of the symptom. Similarly, 90% or 94% of subjects reporting dizziness reported complete relief.

In 1984, Schimek and Mohr (15) conducted a descriptive survey of 78 patients with chronic headache. It is assumed that these subjects were suffering from benign headaches of the muscle contraction, migraine or mixed types. The approach taken in this study was to determine the characteristics of these patients at presentation to a manual therapy clinic as opposed to the outcome of manipulative treatment. The clinic investigation employed in this study included a determination of the presence of active and latent trigger points, the presence and degree of "vertebral fixation", patterns of referred pain and patterns of X-ray findings in the cervical spine. These authors conclude their study with the following statements: "... pathological nociceptive impulses from a blocked joint increase, reflexly, muscle tension in certain neck muscles. Referred pain develops which may be correlated with a certain area of the head ... Vertebral dysfunction is of primary importance in the development of chronic headache and is considered to be its generator ... (15)."

Miller et al. (16), reported on a single case-study of "long-standing, uncomplicated tension headache." According to the authors 13 adjunctive treatments were rendered resulting in a reduction in headache frequency from 2.6/day to 1.0/day within 1 week. Use of analgesic medication similarly decreased.

In 1985, Jirout (17) reported on 200 patients who had previously been involved in a roentgenological study of motion blockage or fixation at the C2-C3 segment. Ninety percent of the subjects with this vertebral dysfunction reported the presence of headaches. Manipulation directed to the areas of fixation resulted in complete relief in approximately 80% of this sample.

Droz and Crot (in 1985) reported on chiropractic care of "occipital headaches (18). They conducted a retrospective survey of clinic files from 1962-1963, and 1973-1983. Of approximately 10,000 patients files surveyed, 332 (approximately 3%) were found to suffer from "occipital headaches." Average numbers of treatments and case outcomes were compiled. The average number of treatments was nine. Treatment outcome was rated as very good (90% relief), good (75%), slightly better (50%), same or worse. Eighty percent of the sample had a very good result, while 10% had a good and 3% had a slightly better outcome. As such, 93%
had a positive treatment outcome, while 7% had no improvement. Droz noted that the vast majority of successful cases had no more than 10–15 treatments, so that treatment success can be achieved quickly, but in cases where the number of treatments exceeds 15, a poorer prognosis is to be expected.

Most recently, Turk and Ratkolb (19) reported on 100 cases of headaches attributable to the cervical spine treated by manipulation. Nine manipulative treatments produced significant reduction of headaches in 75% of subjects. At 6-month followup 65% still had significant improvement while 35% had recurrence of symptoms. A significant reduction in analgesic use was also noted.

In summary, the research literature includes six descriptive surveys (two in the paper by Lewit) of treatment outcome and three studies describing the characteristics of vertebrogenic dysfunction in chronic benign headache sufferers. Included in these are one study notating an association between findings of segmental misalignment on static X-ray studies of the upper cervical spine and two studies noting the association of upper cervical vertebral motion dysfunction (fixation) with chronic headaches (Table 1).

### Migraine Headaches

In 1964, Figar and Jansky (20) studied vascular reflexes of subjects who suffered from what they termed "vertebrogenic migraine." Vascular reflexes were studied by photoplethysmography of the superficial temporal artery. Both the resting state levels and those evoked as a response to external stressors were studied. Subjects were stimulated by loud noise, mental arithmetic, and nociceptive stresses. Nine migraine patients were compared to nine age-matched control subjects. Subjects were studied between headaches. In the normal controls, the vasomotor response of the superficial temporal artery was uniformly and bilaterally vasodilatory. In the patient group, 23% showed parallel vasoconstriction, while numerous others showed asymmetric reactions. The authors concluded that vertebrogenic migraine is associated with abnormal cerebrovascular tone and regulation.

A landmark descriptive survey of a chiropractor’s clinical experience in the treatment of migraine was reported by Wight in 1978 (21). In this study, 87 patients (57 females and 30 males) were included, 34 with common migraine and 53 with classical migraine. The results of chiropractic treatment were as follows. In the common migraine group, 85% of the females and 50% of the males were greatly improved. In the classical migraine group, 78% of the females and 75% of the males were greatly improved. The overall success rate for both migraine categories for both sexes was 74.7%. In those not reporting a greatly improved status after treatment, a reduction of severe attacks was achieved for 81.5% of females and 45.8% of the males in the common migraine group, and 43.2% and 34.5%, respectively, in the classical migraine group.

The most important study, and, indeed the only randomized clinical trial of chiropractic manipulation and migraine, was conducted by Parker et al. in 1978 (22). In this study, 85 subjects (33 males, 52 females) with a mean age of 41 years and mean duration of headache symptoms of 19 years were randomly allocated to three treatment groups. Of these subjects, 70% suffered common migraine while 30% suffered classical migraine. The treatment consisted of chiropractic manipulation, medical manipulation, and a control/mobilization performed by physiotherapists. Subjects underwent a 2-month pretreatment stage, a 2-month treatment stage, and a further 2-month follow-up Stage. Table 1 shows the summary of clinical studies of spinal manipulation and headaches.

### Table 1. Summary of clinical studies of spinal manipulation and headaches

<table>
<thead>
<tr>
<th>Author</th>
<th>Date</th>
<th>Manipulators (MD, DC)</th>
<th># Subjects</th>
<th>HA</th>
<th>Results</th>
</tr>
</thead>
<tbody>
<tr>
<td>Lewit</td>
<td>1971</td>
<td>MD</td>
<td>41</td>
<td>MC</td>
<td>Manipulation was most effective</td>
</tr>
<tr>
<td>Lewit</td>
<td>1971</td>
<td>MD</td>
<td>93</td>
<td>MC</td>
<td>Manipulation most effective</td>
</tr>
<tr>
<td>Vernon</td>
<td>1987</td>
<td>DC</td>
<td>33</td>
<td>MC</td>
<td>85–90% success</td>
</tr>
<tr>
<td>Jirout</td>
<td>1985</td>
<td>MD</td>
<td>200</td>
<td>MC</td>
<td>80% success</td>
</tr>
<tr>
<td>Droz &amp; Crot</td>
<td>1985</td>
<td>DC</td>
<td>332</td>
<td>Occipital</td>
<td>80% very successful</td>
</tr>
<tr>
<td>Turk &amp; Ratkolb</td>
<td>1987</td>
<td>MD</td>
<td>100</td>
<td>MC</td>
<td>75% success</td>
</tr>
<tr>
<td>Wight</td>
<td>1987</td>
<td>DC</td>
<td>67 (57F, 30M)</td>
<td>52F, 33M</td>
<td>75% success</td>
</tr>
<tr>
<td>Parker, Tupling &amp; Pryor</td>
<td>1978</td>
<td>MD &amp; DC</td>
<td>85</td>
<td>NIG</td>
<td>28% success</td>
</tr>
</tbody>
</table>

Total subjects: 971
Average Outcome: 71% (w/o Parker et al. = 80%)

F = female, M = male, MC = muscle contraction, NIG = common and/or classical migraine.
period. An average of seven treatments were performed in the treatment phase. The mean improvement level for all three groups was 28%. Only one hypothesis (that headache severity (i.e., pain intensity) would be reduced in the chiropractic manipulation group) achieved an acceptable level of statistical significance (0.01). Measures of treatment expectation of the subjects and the treaters indicated a higher degree of expectation of treatment response by the patients and the chiropractors in the chiropractic manipulation group. The authors concluded that such high expectations had a direct effect on pain ratings and, as such, the finding that a single significant hypothesis did attribute treatment success to chiropractors was probably due to a placebo-type response.

This study was the subject of much criticism and independent review. An independent statistical consultant was commissioned by the New Zealand Commission on Chiropractic (23) and asked to revise the statistical tests in order to more properly analyze the available data. Several problems were delineated, which in the opinion of the consultant, contributed to the negative findings of the clinical trial. First, the control group did in fact receive a form of treatment, i.e., mobilization, so that a true treatment vs. no treatment comparison could not be conducted. Second, the alpha level of 0.01 was very stringent, even higher than that of many medical clinical trials. Third, some of the statistical tests were inappropriate. A revision of the statistical analysis was performed which did not produce an acceptable level of statistical significance in any of the hypotheses, even at a lowered (0.05) alpha level. However, the consultant noted the subjects in the chiropractic manipulation group consistently scored higher in every rating than did the other two groups. Given that a relatively low number of treatments was performed on average (seven), and that the chiropractic manipulation group was favored in all of the statistical tests, the consultant concluded that the study had a low power. This meant that it was not designed effectively enough to detect a difference between treatment groups when such a difference might, in fact, have been present.

A follow-up study was published in 1980 by Parker and his colleagues as a response to his critics (24). It was noted that at a 20-month follow-up period, a further 19% of subjects had achieved an improved rating. This constituted a 47% success rate for manual therapy in the treatment of migraine. An interesting aspect of the data which did not receive statistical analysis was that 14 of the subjects in the study achieved a complete recovery. Of these, eight received chiropractic manipulation; one received medical manipulation; and five were in the control/mobilization groups (Vernon, H, unpublished data).

This follow-up study attempted to correlate certain psychosocial variables with treatment success. Only one variable (sex) achieved the appropriate level of statistical significance to be accepted as a correlate of therapeutic success (0.01). In this regard, females responded better than males to the forms of manual therapy studied by Parker et al.

It should be evident from the preceding discussion that the “Parker clinical trial” raised more questions than it provided answers for. It appears to have been a study with very worthwhile intentions, which fell heir to an important but often overlooked error known as Type B error (25). Upon reflection, a 47% total success rate, with the chiropractic group producing a significantly larger number of complete recoveries is an impressive set of findings which should encourage further study.

Sachse and Eckhardt (26) reported on 22 migraine patients in which a structural assessment very similar to a chiropractic clinical examination was conducted. They found the most frequent segmental fixations to be C0-C1 and C7-T1, as well as in the first costovertebral joint (first rib fixation). They found the most severe tenderness to palpation present in the shoulder (scapular) region and over the posterior arch of atlas. The most severe muscle hypertonicity was found in the trapezius and levator scapula muscles.

Cervical Headaches: Review of Theoretical and Descriptive Models

Barre (1926) (27) and Lieou (1928) (28) were among the first to implicate the cervical spine in the genesis of a wide array of neurological symptoms, foremost which were headache and vertigo. In their writings, irritation of the cervical sympathetic system was linked to circulatory disturbances, primarily as a disturbance of vasomotor tone, in the distributions of the vertebral artery or the internal auditory artery.

In 1948, Barschi-Rochaix (29) coined the term “migraine cervicale” to describe a type of headache, potentially very severe in character, which was caused by disturbances of the cervical spine. The impact of these mechanical disturbances was thought to be mediated through the autonomic nervous system. At this stage of its development, the vertebrogenic theory focused largely on changes in the vertebral artery and the resultant symptomatology arising from transient ischemia of the posterior cranial structures.

Following Barschi-Rochaix, Beckwith (30,31),
Hunter and Mayfield (32), Geiger (33), Gayral and Neuwirth (34, 35), and Skillern (36) addressed the topic. Kovacs (37) summarized the emergent model in his article entitled “Subluxation and deformation of the cervical apophyseal joints: a contribution to the etiology of headaches.” He bridged the gap between theories focusing largely on the arterial system as it was affected by the neck structures, and those which expanded to include the nervous system more directly as a source of mediation between the spine and pain or disturbances about the head. However, Kovacs went further than these structural considerations by emphasizing the role of functional changes in intervertebral joint behavior. He proposed a sequence of events which lead to joint derangement, namely that restriction and disturbance of joint motion would lead to reflex muscle spasm which would alter the curve of the cervical spine and accelerate degenerative changes which would then compromise the vertebral artery and nerves. This shift to a functional perspective is pivotal in the development of the modern neurogenic model.

The landmark work of Ruth Jackson appeared in 1958 (38). It reiterated that headache and a variety of neuro-otological symptoms were found to be attributable to direct mechanical irritation of the ascending posterior cervical sympathetic system, especially (and, perhaps, too exclusively) in the typical trauma of the cervical spine, namely the whiplash injury. Seletz’s work “Headache of extra-cranial origin” (39) also appeared in 1958. In it, he both summarized the existing model of neurogenic effects on the arterial supply to the cranium and proposed what we might call a neurogenic model arising from vertebral derangements. In this neurogenic model, Seletz emphasized the following.

1. The uncovertebral joints may directly irritate the cervical nerve roots and thus cause pain and muscular dysfunction.
2. Noxious stimulations from the cervical joints and muscles might produce reflex irritation of the spinal accessory nerve (XI) and thus produce spasm of the sternocleidomastoid and trapezius muscles inducing a local occipital headache syndrome.
3. Such spasms of the trapezius muscle may produce entrapment and give rise to unilateral occipital neuralgic headache.

The most important contribution to this neurogenic model arose from Seletz’s discussion of a mechanism which had been discussed 3 years earlier by Skillern (36) involving the connections in the spinal cord between the sensory pathways of C1 and C2 and the second order neurons of the spinal tract of the trigeminal nerve. This convergence of the peripheral sensory pathway from the joints of the upper cervical spine with the sensory projection involving the cranial distributions of the trigeminal nerve creates the most fertile mechanism for head pains to arise from the structures of the neck.

Grillo (40), writing in the Swiss Annals of Chiropractic II on the “Differential diagnosis of therapy and headache”, presented a complete review of the knowledge of the day on what he termed “vertebragenous headaches.” He described these as a syndrome of neck-cranial-facial pains, presenting unilaterally, with tenderness in the suboccipital region and with associated “neurovegetative symptoms.” He proposed that the major cause of this syndrome was mechanical irritation of the nerve roots, particularly in the foraminal environs.

Boake (41) provided a thorough description of the “cervical headache” as being unilateral, of chronic duration, with maximal pain in the fronto-orbital distribution while occipital pain is always present, associated with neck stiffness, tenderness and spasm over the facet joints of C2 and C3 and with ”trigger radiations” possibly present. He proposed that this sort of headache was responsible for 70% of all headaches seen by medical doctors.

He also proposed a number of mechanisms to explain the etiology of cervical headache. It is interesting to note how his proposals amalgamated those of the previous vascular model and extended the neurogenic model. He proposed that the major cause of cervical headache was trauma to the apophyseal joints of the upper cervical spine, leading to joint fixation or restriction of motion. In his view, muscle spasms associated with the craniocervical region, and which were typically associated with headache of the “tension” type, were secondary to these joint disturbances. Boake further emphasized the convergence of sensory input from C1 and C2 with spinal tract of the fifth cranial nerve, as well as the role of the postganglionic sympathetic fibers from the superior cervical ganglion.

Edmeads’s authoritative report (5) reviewed a number of mechanisms proposed to explain the role of cervical spine disorders in headache. Edmeads cites degenerative joint disease, myofascial pain and entrapment of the greater occipital nerve, mechanical irritation of the C1 and C2 nerve roots, the vertebral nerve, and the postganglionic sympathetic chain as potential peripheral sources of cervical spine dysfunction capable of producing headache. The role of the spinal tract of the
trigeminal nerve (V) is given special consideration as a central convergence mechanism whereby referred head pains, particularly in the distribution of the ophthalmic division, might arise as a result of noxious irritation in the upper cervical spine.

Bogduk (2) reviewed the previous literature and responded to the criticism generated by the “Parker” clinical trial in which chiropractic treatment of migraine was investigated. It is this response which forms the basis of his position on the vertebrogenic model of headache. In “Cervical manipulation and headache” (2), Bogduk initially debunked the concept of a “cervical migraine” citing the obvious biochemical and autonomic mechanisms for which, at that time, he failed to find any justified correlation with the cervical spine. This is in spite of the fact that he provided an excellent review of the mechanisms previously proposed to explain the “cervical migraine” phenomenon, i.e., he cited Bartschi-Rochaix’s work, and he noted that mechanical irritation of the vertebral artery, vertebral nerve and the ascending sympathetic chain can initiate an “autonomic barrage” sufficient to create cerebral vasospasm. As well, he accepted Kovac’s contention that subluxation of the cervical apophyseal joints might compromise these structures (37). However, he reserved his most enthusiastic support for the role of the apophyseal joints in nonmigraine headache. He asserted a direct, causative role for mechanical derangements of the cervical apophyseal and most importantly, craniocervical synovial joints in the production of headache. This mechanical derangement is characterized as a “chronic hypomobility”, previously referred to as a fixation. This mechanism allows for an explanation of the role of the cervical spine in headache in a much younger population, contrary to the limitations of the “degenerative disease” model proposed by Edmeads.

Bogduk does, in fact, include osteoarthritic afflictions of the apophyseal joints in the group of mechanisms of headache production attributable to the cervical spine. He includes derangements arising from meniscoid entrapments as well, but this is seen as a secondary mechanism of joint hypomobility.

The contribution of Sjaastad and his colleagues consists of a series of studies from 1983–1987 on what he has termed “cervicogenic headache” (7–9). This is described as a variant of the chronic paroxysmal headache which is almost always unilateral, accompanied by autonomic symptoms and provable by movements of the head and neck: primarily forward flexion. Sjaastad has proposed that this headache may be due to entrapment of the occipital nerve or to a C2-C3 rhizopathy and that this diagnosis may be greatly under-
original article) refer to 16 such studies (2–5, 27, 32, 43–52), chiefly the works of Bartschi-Rochaix, (43). Bogduk, (2–4, 44), Hunter and Mayfield (32) and Barre (27). Four other “cervicogenic” articles are added, by Farina et al.; the work of Maigne (53) and Ehni (54, 55); and Pfaffenrath et al., refers to the works of Pawl (56), Travel (57) and Newill (58). Altogether, this small but coherent body of work on “cervicogenic headache” has cited 21 references prior to 1983. Of these, seven are concerned with surgical approaches to treatment (45–49, 51, 56) and are not material to the issue of conservative treatment herein addressed. Only five authors; Barre, Bartschi-Rochaix, Hunter and Mayfield, and Bogduk, are referenced in all five articles. These studies may then be said to form the core of the supportive literature of the “cervicogenic headache” group. The total number of patients reported on by this group is 47.

Sjaastad et al. have reviewed the cases presented by Hunter and Mayfield, and have concluded that they appear sufficiently similar to the current descriptive profile of cervicogenic headache as to justify tacit acceptance of this case series as predating that of the first of Sjaastad et al.’s articles.

Both Bartschi-Rochaiz and Bogduk are cited as having used a term related to the cervical spine (i.e., “migraine cervical” in the former case, and “cervical headache” in the latter), and of having provided a conceptualization of mechanisms involved in this type of headache (52, 59). Bogduk is also cited as providing evidence of therapeutic success in the use of anaesthetic blocking in the suboccipital region (44), although this work appeared just prior to Sjaastad et al.’s first work.

The present author has also conducted a broad literature and citation search on the topic of headaches and the cervical spine. Such literature was found in computerized data bases, in specialized but readily available textual data bases, or in citations of works which arise from these two sources. Some of this work has been reviewed above. In all, 42 references dealing directly
with the topic of headache arising from the cervical spine, have been accumulated in the period prior to 1983, which have not been cited by the "cervicogenic headache" group (2, 10, 14, 20–22, 26, 30, 31, 33, 34, 36–41, 57, 60–83). Thirty of these articles have been written by medical doctors (only eight by specialists in manual medicine), five by osteopaths, five by chiropractors, and two by physiotherapists. No doubt additional articles exist as well. Chapters or sections in texts and presentations at conferences have not been included in this reference list, although these would undoubtedly increase the total literature base.

The importance of this unmentioned body of literature is two-fold. First, the extent of recognition of the role of the cervical spine is far greater than has been acknowledged by the "cervicogenic headache" group. As described in the discussion above, an understanding of this contribution has evolved since the original work of Barre (27) and Lieou (28) first having been framed as a vascular disorder, subsequently as a neurogenic, then somatic, and finally as a combined neurogenic/somatic disorder of considerable complexity and with variable clinical manifestations. Each of the authors in this unacknowledged literature base has elaborated or emphasized on one or another important factor contributing to the final cohesive picture. A good example is the work of Skillern (36). He described this headache type as the "greater occipital trigeminal syndrome", but most importantly he was the first author to note the importance of the connections between the descending spinal tract of the V cranial nerve and the upper cervical nerve roots. This connection is now accepted as the neuroanatomical basis for referred cranial pain from the neck as described by Pfaffenrath et al. (42). Unfortunately these authors, and the others as well, fail to mention Skillern’s work.

Secondly, if one reviews the breadth of this literature it is apparent that the clinical manifestations of headaches arising from the cervical spine vary considerably and are more extensive than that which is currently described as "cervicogenic". While the most typical clinical picture which emerges is of a unilateral headache, bilaterality is commonly acknowledged. Cervical headaches have been described both with and without an accompanying occipital, suboccipital or cervical component, or in other words, a frontotemporal, orbital, or commonly, as a vertex type of headache. Autonomic symptoms are not a prerequisite although they are acknowledged by many previous authors as a frequent accompaniment. This clinical picture mimics both the conventional "tension" and "common migraine" categories, and the resulting diagnostic confusion has been described by numerous authors, perhaps most eloquently by Frykholm who has written: "In my experience, cervical migraine is the type of headache most frequently seen in general practice and also the type most frequently misinterpreted. It is usually erroneously diagnosed as classical migraine, tension headache, vascular headache, hypertensive encephalopathy or posttraumatic encephalopathy. Such patients have usually received an inadequate treatment and have often become neurotic and drug-dependent" (60).

Cervical headaches have also been described by numerous authors as being far more prevalent than is recognized by the "cervicogenic headache" group. Both Boake (41) and Frykholm (60) state that this type of headache represents 70% of all headaches seen by general practitioners.

The situation described above is in distinct contrast to the restrictive profile of "cervicogenic headache." In their defense, Fredrickson et al. (9), and Sjaastad et al.
(7, 8) cite their desire to apply this diagnostic label only to a precise clinical entity with features as exclusive of other headache types as possible. Unfortunately, as even Sjaastad et al. (8) have recognized that this is an admirable if improbable goal. His 1986 editorial acknowledges the tremendous potential overlap between "cervicogenic", tension and common migraine headaches. Diagnostic precision will likely remain elusive, except in circumstances where a therapeutic trial is included in the diagnosis. As indomethacin response is used to rule out chronic paroxysmal headache so therapeutic measures directed to the cervical spine remain a potent source of retrospective diagnostic confirmation. Bogduk (44) and others have confirmed this for therapeutic nerve and facet blocks. The literature review cited above adds support to the role of spinal manipulation in the diagnosis and treatment of cervical headache.

The following case history is illustrative of both the diagnostic and therapeutic challenges involved in patients with cervical headache.

A 26-yr-old female homemaker reported to our clinic initially complaining of right-sided suboccipital pain of two day's duration which had appeared suddenly upon awakening. The pain was a constant severe ache punctuated by sharp stabs of focal pain upon head/neck movements. The patient denied any past history of neck or suboccipital pain or discomfort but upon further questioning gave a much longer history of intermittent headaches which she described as "migraine", which were in fact bilateral, frontal headaches sometimes accompanied by nausea, but never accompanied by an aura or any other premonitory symptom. These headaches generally started as milder frontal pain with no posterior (i.e., occipital or neck) pain. Most often (3-4/wk) the headache remained at this level of intensity. She described these as "tension headaches."

Less frequently the headache progressed in pain, still bilaterally, to a much more severe level of intensity, accompanied by nausea, which significantly disturbed her activities of daily living and for which analgesic medications were often poorly effective.

Upon examination, exquisite tenderness to palpation

Figure 4. Note suspected spasm of inferior obliquus on the right.
was noted in the suboccipital region on the right. In particular deep palpation of the inferior oblique muscle from the tip of the C2 spinous process superior-laterally to the tip of the transverse process of C1 elicited the most severe replication of her pain. Rotation of the head to the right was restricted by 75%. Forward flexion of the head was restricted by 50%, these movements recreating the focal suboccipital pain. X-rays of the cervical spine were obtained in the neutral and dynamic views (Figure 1–6). The neutral A-P suboccipital view shows marked offset of the C2 spinous process to the right, interpreted to indicate rotation of C2 to the left. The lateral view demonstrated an inferior positioning of the posterior arch of C1, interpreted to indicate a backward nodding of C1 in the neutral position. Combined together these static X-ray findings of vertebral malpositioning are interpreted to reveal involvement of the inferior oblique muscle, in spasm around the C1-C2 articulation on the right (Figure 4). The dynamic views reveal some forward tipping of C1 on C2 in flexion, although less than the same motion at C0-C1.

This clinical picture evokes confusion preventing a precise diagnosis. The chronic headache picture could be described as a “mixed headache” or it could be described as intermittently severe, common migraine. When the unilateral suboccipital pain is superimposed, does this resemble, if only for the short duration of the combined problem, a bilateral cervicogenic headache? If not, and since the headache was not precipitated by the suboccipital pain, are they two separate entities? It would appear so. and as such they could at best be described as coincidental. However, the response to treatment further complicates the picture. This patient was treated with a series of manipulations delivered as high velocity, low amplitude adjustive thrusts to the C1-C2 and C2-C3 segments on the right. As Figures 7 and 8 demonstrate, not only was relief of suboccipital pain obtained very quickly, but there was also a complete remission of headaches of both varieties which
Figure 7. Change in suboccipital pain over the treatment time.

Figure 8. Change in headache frequency over the treatment time. ★ = migraines. □ = muscle contraction headaches.
had lasted for 2 months until the patient was discharged from treatment.

One hypothesis to explain this somewhat confusing clinical picture is that the segmental dysfunction of C1-C2 did not commence suddenly and was in fact present prior to the incidence of acute suboccipital pain, acting as the cause of the headache complaint in this patient and as a referred pain syndrome (84, 85). Since patients very often apply diagnostic labels incorrectly and as a product solely of their own self-perceptions of pain, this patient labelled herself as a "tension/migraine" sufferer and had no sense of the contribution of a suboccipital component. If this is reinforced by health care practitioners it becomes a self-fulfilling prophecy. Any suggestion to the contrary would have been viewed with deep suspicion. Only when an apparently unrelated event occurred did the patient seek alternative treatment. The latent C1-C2 dysfunction was exacerbated acutely in the manner typical of benign, acute torticollis. The therapeutic intervention delivered for this latter complaint then connected the two separate pain components into one meaningful, if retrospective, whole.

This complaint is dissimilar to the "cervicogenic headache" of Sjaastad in the following ways: The headaches were bilateral; a suboccipital component was absent, as was aggravation by forward flexion and nausea was only infrequently present while other autonomic symptoms appeared not to be involved (they were denied in the history). Since the criteria for cervicogenic headache are presently applied quite rigorously (8, 9) this headache would not have been so diagnosed. Yet this case is quite typical of the kind of headache previously described in the clinical studies of manipulation, and, by its excellent response to manipulation, would most decidedly be described as "of cervical origin."

CONCLUSION

Sjaastad and his colleagues have proposed a C2-C3 rhizopathy as one etiology of a currently narrowly defined headache category known as "cervicogenic." Practitioners of manipulative therapy, especially chiropractors, have characterized the rhizopathic condition on functional grounds to include disturbances of segmental alignment, motion and muscle tone, as well as focal joint pain arising from the apophyseal nociceptors (86). Referred pain may radiate from this lesion. In the case of the cranio-cervical joints, these referred pain patterns may project anteriorly, thereby mimicking headache states. In the patient reported herein as in many others described in the literature, the rhizopathy might be located in the C1-C2 joint and might be headache symptomatology more broadly mani. As such, the term "vertebrogenic headache" is proposed as a more all-encompassing term (Figure 9). A manipulative treatment affords the opportunity to firm this diagnosis on a retrospective basis.

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REFERENCES


Pressure pain threshold evaluation of the effect of spinal manipulation on chronic neck pain: a single case study

HT Vernon, DC, FCCS(C)

A single case study is presented in which the use of the pressure pain threshold meter (PPT) allows for the objective evaluation of the treatment effect of spinal manipulation. A 22 year old male with a five year history of right-sided neck, scapular and arm pain demonstrated an average of 45.7% increase in PPT readings in maximal tender points after spinal manipulation. The results are discussed in light of current theories of pain mechanisms related to spinal manipulation. (JCCA 1988; 32(4): 191-194)

KEY WORDS: tender point, pressure pain threshold, manipulation, chiropractic.

Introduction

Reports of the objective evaluation of the effect of spinal manipulation on clinical pain states are scarce. The clinical trials reported since 1974 (see reviews by Brunarski1, Ottenbacher and Difabio2, Deyo3) give evidence of an important benefit obtained in patients who receive spinal manipulation. However, amongst a number of methodological concerns, these studies are generally based on self-reports of relief of pain and are subject to a variety of non-specific influences. The introduction of objective methods of evaluating the kind of clinical pain states suffered by the majority of patients treated by chiropractors4 would be an important improvement.

In this case report, we use the Pressure Pain Threshold meter5 (PPT) to provide objective evidence of pain relief obtained in a single chiropractic treatment. The protocol for the use of the PPT, as well as normative and reliability data have been reported extensively by Fischer5 and Jaeger et al6. The device is intuitively attractive to the chiropractic clinical setting as it measures tenderness to pressure in the deeper myofascial structures. This gives it a distinct advantage over skin-rolling7 and electrical pain stimulators8 in evaluating the structures more typically of clinical interest to chiropractors.

Methods

A single-case study is presented of a 22 year old Caucasian male with a five (5) year history of chronic neck, scapular and arm pain on the right side. The patient is a hairdresser, and so an occupational context to his problems was recognized in that he worked with his arms up and out while standing for hours at a time. He was referred to our Specialty Clinic with a diagnosis of unilateral thoracic outlet syndrome. While this was confirmed with photo-plethysmographic investigation9 the involvement of joint and myofascial dysfunction was broadened to include a diagnosis of concomitant unilateral scapulo-costal pain syndrome. In the chiropractic assessment the following significant findings were noted: The patient pointed to areas of maximal pain over the trapezius muscle and in the mid-cervical area on the right. He localized point tenderness in the right trapezius and levator scapula tender points (TP)10, as well as a tender point in the right mid-cervical paraspinal region. (see Figure 1) Palpable hypertonicity was found in these underlying muscles on the right side, especially the trapezius and levator scapula.

L'étude d'un cas isolé présentée, pour laquelle l'utilisation d'un compteur du seuil de douleur à la pression (SDP) a permis d'évaluer de façon objective les effets d'un traitement par manipulation de la colonne vertébrale. Un patient masculin de 22 ans, ayant à son actif cinq années de douleur au bras et à l'omoplate avec un cou coincé vers la droite, a montré un accroissement moyen en indices de SDP de 45.7% aux points les plus sensibles à la suite d'une manipulation spinale. Ces résultats sont discutés en fonction de théories récentes sur les mécanismes de douleur liés de colonne vertébrale. JCCA 1988; 32(4): 191-194)

MOTS-CLÉ: point sensible, seuil de douleur à la pression, manipulation, chiropraxie.
Motion palpation revealed joint dysfunction (fixations) at C2-C3, T2-T3 and in the right scapula (this latter finding from restricted joint play movements). The patient was asked to score his present pain on a standard visual analogue scale (VAS), scoring a 6 on a scale from 0–10.

Table I displays the PTT findings in seven standard TP’s prior to treatment. The author uses the following pressure pain threshold technique: The patient is oriented to the PPT (see Figure 2) Instructions as to how to indicate the threshold level of tenderness are given. Simply stated, the PPT is placed perpendicularly over the contact area. Even pressure is applied so as to increase the load by one or two kg/cm² per second. The patient is asked to indicate the point at which the sensation of pressure changes to tenderness. They do this by saying “now”. This point is the pressure pain threshold point, and has been used by Fischer and his colleagues as a critical diagnostic parameter. A trial run is performed on the lateral forearm extensor muscles to acquaint the patient with the procedure. The patient then lays prone on the adjusting table with the headpiece tipped forward. The effective technique is to use the maximum tender point where it is located first by digital palpation. Once identified, the PPT is then placed perpendicular to the surface and even pressure is applied for the measurement. (see Figure 3, a and b). Measurements are made unilaterally at all of the relevant TP’s, then the contralateral side is measured so as not to induce comparison/order bias in the patient. Treatment consisted of two adjuvant manipulations – a scapular stretch technique with an audible release on the right, and an anterior thoracic adjustment of T2-T3 on the right. The patient had never received chiropractic treatment. He was instructed to a

Table 1 Pre-treatment PTT measurements

<table>
<thead>
<tr>
<th>Tender Point</th>
<th>PTT value (kg/cm²)</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Left</td>
</tr>
<tr>
<td>Medial occiput</td>
<td>2.3</td>
</tr>
<tr>
<td>Lateral occiput</td>
<td>3.0</td>
</tr>
<tr>
<td>Sub-occipital</td>
<td>3.1</td>
</tr>
<tr>
<td>Mid-cervical</td>
<td>3.3</td>
</tr>
<tr>
<td>Trapezius</td>
<td>4.2</td>
</tr>
<tr>
<td>Levator scapula</td>
<td>3.2</td>
</tr>
<tr>
<td>Rhomboid</td>
<td>4.0</td>
</tr>
<tr>
<td><strong>Average</strong></td>
<td>3.3</td>
</tr>
</tbody>
</table>

Table 2 Post-treatment comparison of right TP’s

<table>
<thead>
<tr>
<th>Tender Point</th>
<th>PTT value (kg/cm²)</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Pre</td>
</tr>
<tr>
<td>Medial occiput</td>
<td>1.4</td>
</tr>
<tr>
<td>Lateral occiput</td>
<td>2.0</td>
</tr>
<tr>
<td>Sub-occipital</td>
<td>1.5</td>
</tr>
<tr>
<td>Mid-cervical</td>
<td>2.1</td>
</tr>
<tr>
<td>Trapezius</td>
<td>2.6</td>
</tr>
<tr>
<td>Levator scapula</td>
<td>1.9</td>
</tr>
<tr>
<td>Rhomboid</td>
<td>2.6</td>
</tr>
<tr>
<td><strong>Average</strong></td>
<td>2.01</td>
</tr>
<tr>
<td><strong>Average increase</strong></td>
<td>45%</td>
</tr>
</tbody>
</table>

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supine for five (5) minutes, whereupon the PTT for the right-sided TP’s was redone. He denied experiencing any pain during the manipulations.

Results
The post-treatment PTT values are shown in Table II. The largest increases in pressure pain threshold occurred in the TP’s of the scapular muscles. The average increase at all seven (7) TP’s was 45.7%. The mobility of the T2-T3 fixation palpated as greatly improved, while the right scapula palpated as moderately more mobile after the treatment. The post-treatment VAS was 1. The patient indicated verbally, that he felt significantly better, even in his general mood state.

Discussion
This single-case study gains some generalizability from the following aspects: the patient was totally naive to adjustive treatment, and so, had no preconceptions about the effect such treatment might have. The complaint was long-standing and no relief had previously been obtained. The complaint was quite typical of those seen by chiropractors and others in “physical” or manual medicine. It bears pointing out that the chiropractic assessment identified findings of both muscle and joint dysfunction and that these are, arguably, concommitant, both in their shared location, and in specific linkages of discrete muscles to discrete joint sites (i.e. C2-C3 to scapula - for levator scapula. T2-T3 fixation subjacent to trapezius TP). This is in distinction to the findings of “myofascial pain specialists” who omit joint dysfunction assessment.

Without the PPT measurements this presentation would be no more than an anecdotal report of a chiropractor’s palpation findings and opinions about the patient’s status. In other words, a mundane report of anecdotal improvement typical of that which we commonly encounter in practice. With the addition of the objective findings, the palpatory findings of fixation and hypertonicity are given a quantitative context which matches the patient’s subjective complaint. The improvement post-adjustment is verified objectively and quantitatively. Variations in treatment effect can be discerned between various areas within the region of pain (i.e. upper cervical vs scapular).

Finally, quantitative evidence is forthcoming which adds to the data which supports certain theoretical explanations regarding the beneficial effects of spinal manipulation. These have been advanced (and reviewed) in recent studies by the author8,13 and most recently by Gillette14 and Zusman15. The controversy exists in accepting the “afferent bombardment model” originally proposed by Korr16 and Wyke and17 and elaborated by many others19,19,20.

In our first study8 we demonstrated an increase in cutaneous pain tolerance to electrical stimulation which was significantly greater after a spinal manipulation as compared to a joint mobilization. The mean increase in pain tolerance at five minutes in that study was 100%, while at 10 minutes it was 140%. The case presented here demonstrates that a similar pattern is obtained when PPT is conducted in the deeper tissues. Since the manipulations were not painful, we continue to maintain that the most tenable explanation for these findings lies in changes in spinal cord reflexes produced by bombardment of Type I and/or Type II afferent input from the myofascial and articular low threshold mechanoreceptors. The phenomenon of manipulation-induced plasticity of spinal reflex mechanisms is essentially the opposite of those which have been demonstrated in the presence of, or as an effect of, painful input of nerve, muscle and to a lesser extent cutaneous experimental lesions. (See Wall and Woolf, 1984).21
Where pain is excitatory, producing long-term potentiation of motoneurons and pain-transmitting spinal cord centers, (i.e. the "central facilitation" of Korr\textsuperscript{2}), manipulation exerts inhibitory influences thereby producing salutory clinical effects.

**Conclusion**

The PPT assessment has been shown to be useful not only as previously reported in the objective evaluation of clinical pain status, but now, in the objective demonstration of relief of pain by spinal manipulation. Larger group studies are necessary to confirm these findings and to substantiate the current theoretical models of manipulation-induced effects.

**References**

Pressure Pain Threshold Evaluation of the Effect of Spinal Manipulation in the Treatment of Chronic Neck Pain: A Pilot Study

HOWARD T. VERNON, D.C.,* PETER AKER,† STEPHEN BURNS,† STEVEN VILJAKAANEN,† AND LARRY SHORT†

ABSTRACT
Nine subjects with chronic mechanical neck pain syndromes were evaluated for pressure pain threshold (PPT) over standardized tender points in the paraspinal area surrounding a manipulable spinal lesion. The subjects were then allocated randomly to an intervention consisting of either an oscillatory mobilization of the cervical spine (n = 4), which was designated as the control procedure, or a rotational manipulation of the cervical spine (n = 5). An assessor-blinded re-evaluation of the pressure pain threshold levels was conducted after 5 min. In the group receiving a manipulation the mean increases in pressure pain threshold ranged from 40–56% with an average of 45%. In the control group no change in any of the pressure pain thresholds was found. These results were analyzed using ANOVA and were found to be statistically significant (p < 0.0001). This study confirms that manipulation can increase local paraspinal pain threshold levels. The use of the pressure pain threshold meter allows for the determination of such a beneficial effect in the deeper tissues.

INTRODUCTION
Objective evidence of clinical improvement following manipulative treatment by chiropractors of patients with spinal pain syndromes is still rather scarce. A number of clinical trials have been published [see recent reviews by Brunarski (1), Ottenbacher and Difabio (2) and Curtis (3)] in the past decade and a half. The consensus of the clinical trials is that manipulation appears to produce faster relief of pain than do control and comparative treatments. This is especially so for low back pain, particularly if it is of the acute, mechanical variety.

The data is less substantive for neck pain syndromes. Howe et al. (4) reported statistically significant results in favor of manipulation in the treatment of neck pain.

The results of Sloop et al. (5) tended toward this result. Mealey et al. (6) compared early mobilization to rest and support in the treatment of whiplash. Their results favored mobilization. A number of open trials, especially in neck pain (7, 8), in muscle contraction headache (9–14), and in migraine headaches (15–17) have reported very favorable success rates with manipulation. Parker et al. (18), in a randomized comparative trial, reported poorer results in the treatment of migraine by manipulation.

Clinical analogue studies have been conducted by the senior author (H.V.) in which the results of a single manipulation were compared to control procedures. In the first study (19), cutaneous pain tolerance levels were measured with an electrical pain stimulator and were found to rise significantly higher in the group receiving a single thoracic manipulation as compared to the sham/mobilization group. In the second study (20), a single manipulation in the cervical spine produced a modest increase (9%) in plasma beta-endorphin levels while control and sham procedures produced a slight drop. These studies offered preliminary (and in the case of beta-endorphin levels, modest) support for the empirical finding of pain relief subsequent to manipula-
tion, and to the theory that this pain relief is a result of reflex mechanisms activated specifically by the dynamic thrust. We characterized these mechanisms as afferent bombardment from the articular (21) and myofascial (22) receptors, which produces presynaptic inhibition of segmental pain pathways and possibly activation of the endogenous opiate system (23-25).

In the present study we have extended this earlier work to symptomatic subjects. As well, we have used the pressure threshold meter (Pain Diagnostics and Treatment, Inc., 17 Wooley Lane, Great Neck, NY) reported upon extensively by Fischer (26), and Reeves and Jaeger (27). This device allows for the objective measurement of pressure pain threshold over tender points in muscles. Data on reliability and on the average values of a variety of muscles in normal subjects have been reported by the authors mentioned above (28, 29). As well, the instrument has been used in small clinical surveys of treatment effect, objectively demonstrating that physiotherapy, trigger point injections (30) and muscle energy stretch techniques (27) result in reduction of tenderness in muscles.

Another advantage of the pressure threshold meter is that it produces mechanical irritation of deeper somatic structures (i.e., myofascial structures). This distinguishes it from electrical pain stimulators which confine their stimulation to the skin only. As such it is a more relevant device for measuring functional changes in the deeper tissues around a joint, especially those produced by a treatment modality directed at the joint (i.e., manipulation). Veccheit et al. (31) have shown that pain threshold levels are quite different in the skin, subcutaneous and muscle tissues of a singular experimental lesion. In their study, chemically-induced muscle hyperalgesia lasted for up to 24 hr after an experimental lesion, while cutaneous hyperalgesia lasted for only 2 hr. This phenomenon may reflect differences in nociceptor innervation between skin and the deeper structures (32) and in spinal cord processing of these inputs (33).

The present study reports on a small series of subjects all of whom were suffering from mechanical neck pain syndromes. Our hypothesis was that a single manipulation would produce a significantly greater rise in pressure pain threshold levels in the paraspinal area surrounding a spinal fixation (34) as compared to a control mobilization procedure.

METHODS

Nine subjects participated; six males (average age 27 yr) and three females (average age 38 yr), all reporting what was diagnosed as chronic mechanical neck pain. The duration of these complaints ranged from 2 wk to 8 yr, with most subjects reporting a duration less than 3 months. The subjects were identified by a research assistant. The study's objectives and general methods were explained to them, and informed consent was obtained. All of the subjects were in the course of receiving chiropractic care at the time of the study.

The research treating physician conducted an assessment of joint dysfunction in the cervical spine (35) and a test segment was identified as a "fixation." This is identified as a spinal motion segment which is characterized by manual palpation as hypomobile (34). This level was marked with a grease pencil.

The examiner left the room while the assessor entered and conducted a pressure pain threshold assessment consisting of four points [tender points (TP)] above and below, and on each side of the test/fixation level in the paraspinal area. These points were consistently measured as: a) ipsilateral to the clinically painful side, slightly below the fixation; b) ipsilateral, above; c) contralateral, above; and d) contralateral, below.

The technique of pressure pain threshold (PPT) measurement has been described by Fischer (26, 28, 29). Careful application of the PPT meter is made over the skin with graded vertical pressure applied 1 kg/sec into the deeper tissues. The patient is instructed to indicate verbally (by saying "yes") the point at which the pressure sensation changes to tenderness. A test run was conducted on the extensor surface of the forearm to acquaint the subject with the sensations associated with the test. Two measurements were taken at each point and the assessor left the room. The treater re-entered and applied the test treatment, allocated randomly as either a rotational mobilization with gentle thrust. We characterized these mechanisms as afferent bombardment from the articular (21) and myofascial (22) receptors, which produces presynaptic inhibition of segmental pain pathways and possibly activation of the endogenous opiate system (23-25).

In the present study we have extended this earlier work to symptomatic subjects. As well, we have used the pressure threshold meter (Pain Diagnostics and Treatment, Inc., 17 Wooley Lane, Great Neck, NY) reported upon extensively by Fischer (26), and Reeves and Jaeger (27). This device allows for the objective measurement of pressure pain threshold over tender points in muscles. Data on reliability and on the average values of a variety of muscles in normal subjects have been reported by the authors mentioned above (28, 29). As well, the instrument has been used in small clinical surveys of treatment effect, objectively demonstrating that physiotherapy, trigger point injections (30) and muscle energy stretch techniques (27) result in reduction of tenderness in muscles.

Another advantage of the pressure threshold meter is that it produces mechanical irritation of deeper somatic structures (i.e., myofascial structures). This distinguishes it from electrical pain stimulators which confine their stimulation to the skin only. As such it is a more relevant device for measuring functional changes in the deeper tissues around a joint, especially those produced by a treatment modality directed at the joint (i.e., manipulation). Veccheit et al. (31) have shown that pain threshold levels are quite different in the skin, subcutaneous and muscle tissues of a singular experimental lesion. In their study, chemically-induced muscle hyperalgesia lasted for up to 24 hr after an experimental lesion, while cutaneous hyperalgesia lasted for only 2 hr. This phenomenon may reflect differences in nociceptor innervation between skin and the deeper structures (32) and in spinal cord processing of these inputs (33).

The present study reports on a small series of subjects all of whom were suffering from mechanical neck pain syndromes. Our hypothesis was that a single manipulation would produce a significantly greater rise in pressure pain threshold levels in the paraspinal area surrounding a spinal fixation (34) as compared to a control mobilization procedure.

METHODS

Nine subjects participated; six males (average age 27 yr) and three females (average age 38 yr), all reporting what was diagnosed as chronic mechanical neck pain.
treatment mean levels of pressure pain threshold in the manipulation group are slightly higher than those of the sham group, however, these values are consistent with the previously reported range of tender point levels in symptomatic subjects (29). In percentage terms, a rise in pressure pain threshold ranging from 40-55% was found in all four points around the fixation level in the group receiving manipulation, while there was virtually no change in the pre- to postintervention levels of the group receiving a mobilization. These differences between the groups, at each point, were highly statistically significant (Table 1); \( F = 26.052 \), and \( p \leq 0.0001 \). Of five subjects receiving a manipulation none reported any pain associated with the maneuver. All five reported the manipulation to be a “real” treatment. Of four mobilized subjects, three reported no pain, and none described the intervention as a “real” treatment.

**DISCUSSION**

Although the sample size is small, there is clear evidence of a difference between the groups. Manipulation produced significantly higher increases in pressure pain threshold of tender points surrounding the fixation. We believe that this is the first time such evidence has been reported. These findings can be applied only to the cervical spine.

Several points require elaboration. The finding of 0% change in the mobilization group should be understood as a reflection of the mean values of the data sets. In some cases a change in pressure pain threshold was recorded; however, the small increases or decreases in one or another point in different patients tended to cancel each other out producing no change in the mean of the group (Table 1).

### TABLE 1. Comparison of changes in PPT in neck tender points: manipulation (M) vs. sham (S)

<table>
<thead>
<tr>
<th>Tender points</th>
<th>Pretreatment</th>
<th>Posttreatment</th>
<th>Change (%)</th>
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</thead>
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<tr>
<td>One M</td>
<td>3.4 ± 1.3</td>
<td>4.9 ± 2.3*</td>
<td>44</td>
</tr>
<tr>
<td>One S</td>
<td>2.8 ± 1.7</td>
<td>2.8 ± 1.7</td>
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</tr>
<tr>
<td>Two M</td>
<td>3.4 ± 1.7</td>
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<td>41</td>
</tr>
<tr>
<td>Two S</td>
<td>2.3 ± 1.9</td>
<td>2.3 ± 1.7</td>
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</tr>
<tr>
<td>Three M</td>
<td>3.3 ± 0.51</td>
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</tr>
<tr>
<td>Three S</td>
<td>2.3 ± 1.4</td>
<td>2.4 ± 1.7</td>
<td>0.4</td>
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<tr>
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<tr>
<td>Four S</td>
<td>2.4 ± 1.5</td>
<td>2.6 ± 1.5</td>
<td>0.8</td>
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</table>

\* \( p \leq 0.0001, F = 26.052 \).

Pain threshold increases were recorded in all four points; in other words, apparently in tender points ipsi- and contralateral to the side identified as painful. This reflects several possibilities. First, some subjects may have identified one side as more painful than the other reflecting their own conscious choice of pain location. Tenderness, on the other hand, can often be evoked where there is no conscious perception of pain (28) or where there is a conscious perception of relatively less pain than at another site. Secondly, the effects of manipulation may manifest themselves contralaterally as well as on the side of consciously perceived pain. Sessle et al. (37) have recently demonstrated that 30% of convergent neurones in the trigeminal nucleus caudalis receive input contralaterally. These structure-function relationships (if applied to the spinal cord) may explain changes in pain threshold levels bilaterally.

These data are consistent with our previous work, although the extent of increase in pain threshold is slightly smaller at the 5-min post-treatment point (100% in cutaneous pain tolerance as compared to 45% with PPT). This may be a reflection of differences in pain sensitivity in skin and the deeper tissues, which corroborates the notion that pressure pain threshold measures a different parameter than does electrical stimulation of the skin.

Although the mobilization maneuver was intended as a sham manipulation it failed to produce the necessary requisites of a “real” treatment. To this end, we feel that the absence of both a thrust and an articular crack, both of which would be expected by the subject to be associated with manipulative treatment by a chiropractor, were responsible for the sham failure. The extent to which this affected the posttest results is, we believe, minimal, because the assessor was blinded, and pain threshold levels are less susceptible to unapparent cognitive influences (38). All four subjects would have to have concluded that the absence of a “crack” was a cue to pretend that there was no change. We believe this is highly unlikely.

These findings are entirely of a behavioral nature related to the subjects perception of pain threshold. The underlying mechanism still remains implied, and certainly the previously reported slight increase of endorphins cannot be definitively associated with our present findings, as endorphin assay was not conducted on these subjects. We feel, however, that our previous hypothesis concerning the effect of manipulation on spinal cord reflexes subserving pain as reflected in pain threshold changes, is lent further support. This is especially so since no subject admitted that the manipulation was painful. Gillette (39) has recently proposed a co-activation of receptors subserving both large and small
diameter fibers during a manipulation. Once again, the finding that there is no pain during a manipulation, which is then demonstrated to increase local pain threshold levels tends to mitigate against small fiber co-activation.

CONCLUSION

The pressure pain threshold meter, applied according to the protocol of Fischer, has proven to be very useful in objectifying the effect of manipulation as compared to a mobilization in the cervical spine of subjects suffering from chronic mechanical neck pain. These findings add further support to the theoretical mechanisms proposed to explain the effects of spinal manipulation on spinal pain.

REFERENCES

Skin rolling technique as an indicator of spinal joint dysfunction*

Perry Taylor
Gerald Tole, DC
Howard Vernon, DC, FCCS

The use of the skin roll technique as an indicator of spinal joint dysfunction (fixations) has not previously been subjected to validity testing. This study was undertaken to determine the degree of correlation between the skin roll technique and spinal joint dysfunction in the thoracic spine.

Twenty-five (25) subjects reported tenderness to a paraspinal skin roll along the first ten thoracic vertebrae. At the tender areas elicited during the skin roll, a pressure algometer was used to determine the subject's pressure pain threshold. Static joint challenging as described by Maigne was utilized by an independent examiner to identify levels of spinal joint dysfunction in the first ten thoracic vertebrae in the same subjects. A moderate, but highly significant correlation (Kappa = 0.48, p < .001) was found relating a tender point on skin roll to a spinal joint dysfunction within one vertebra above or below the level of the tender point. Pressure algometer readings revealed a highly statistically significant (p £ 0.0005) decrease in pain threshold tolerance at the level of a tender skin roll as compared to control (non-tender) points.

These findings suggest a moderate level of support for the validity of the notion that spinal dysfunction is characterized by loss of joint motion and contiguous paraspinal tenderness. (JCCA 1990; 34(2): 82–86)

KEY WORDS: Skin roll technique, spinal joint dysfunction, chiropractic, pressure algometer, vertebral joint challenge, thoracic spine, manipulation.

Introduction

An important component of the chiropractic physical assessment is the determination of areas of spinal joint dysfunction. These areas have been associated with a decreased pain threshold of the paraspinal skin. The skin rolling technique, as described by Maigne is one of the methods that is used to locate areas of paraspinal skin which demonstrate a subjective decrease in pain threshold, amongst other clinical phenomena.

The causal relationship between spinal joint dysfunctions (fixations) and alterations of pain tolerance is supported by many authors. Korr reported that areas of spinal joint dysfunction demonstrated a segmentally sustained level of sympathetic hyperactivity which correlated well with deep and superficial skin tenderness. He concluded that a state of segmental chronic sympathetic facilitation was caused by spinal joint dysfunctions. Other authors also agree that increased sympathetic outflow (facilitation) increases sensory firing and thus enhances pain.

L'emploi de la technique du massage de la peau comme indicateur d'un dysfonctionnement de la jointure spinale (fixations) n'a pas subi de test de validité. Cette étude a été entreprise pour déterminer le degré de corrélation entre cette technique et les dysfonctionnements de la jointure spinale dans la colonne dorsale.

Vingt cinq (25) sujets ont fait part d'une sensibilité à un massage de la peau paraspinale sur les dix premières vertèbres dorsales. Un algésemètre a été utilisé sur les parties sensibilisées pendant le massage de la peau, pour déterminer le seuil nociceptif à la pression. La provocations de la jointure statique, telle que décrite par Maigne, a été par un examinateur indépendant pour identifier le dysfonctionnement de la jointure spinale dans les dix premières vertèbres dorsales, chez les mêmes sujets. Une corrélation modérée, mais très importante (Kappa = 0.48, p < .001) a permis de relier un point sensible du peau roulée à une dysfonction de la jointure spinale sur une vertèbre au-dessus ou en-dessous du niveau du point sensible. Les lectures de l’algésemètre ont révélé une diminution statistiquement très importante (p < 0.0005) de la tolérance au seuil nociceptif au niveau de la peau massée sensible par rapport aux points de contrôle (non sensibles).

Ces constatations suggèrent un niveau modéré de soutien pour la validité de la notion que la dysfonction spinale se caractérise par la perte de mouvement de la jointure et par une sensibilité paraspinale contiguë. (JCCA 1990; 34(2): 82–86)

MOTS CLÉ: Technique de massage de la peau, dysfonction de la jointure spinale, chiropractie, algésemètre, provocation de la jointure vertébrale, colonne dorsale, manipulation.

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© JCCA 1990.
Wyke\(^7\) noted the close morphologic relationship between the mechanoreceptors of joints and nociceptors in the somatic tissues. It was his opinion that any loss of normal sensory input from the mechanoreceptors in the joint capsule may result in various abnormalities, which include decrease in pain threshold. Numerous chiropractic authors suggest that this phenomenon occurs in the state of spinal joint dysfunction which has been termed "subluxation". (For further review see 4 and 18.)

Feinstein\(^4\) stated that skin hyperalgesia to prickling or pinching (essence of skin rolling) has often been observed to accompany pain arising from visceral and somatic structures. The hyperalgesic areas are not random but are dermatomally situated corresponding to the cord level of the affected deep tissues.

The chronicity of spinal joint dysfunction and subsequent dermatomal decreased pain threshold may have a feedback effect on the pain receptors themselves. Slosberg\(^3\) noted that not only are pain receptors of high threshold and non-adapting but they can actually have a lowering of their firing thresholds. Thus, less stimulus becomes necessary to activate the nociceptors.

The pressure algometer (dolorimeter) has been used by many researchers to reliably evaluate subjective tender points in pain patients.\(^{10,13,14,15,16,17,21,22,24,25}\) This device enables the researcher to obtain quantitative measurement of a patient's pain threshold. Tunks \textit{et al.}\(^{11}\) have most recently confirmed that the hand-held pressure algometer is a reliable tool that can be used for clinical research into the tender point phenomena.

Terrett and Vernon\(^8\) studied paraspinal tenderness in response to manipulation. They first determined a high association of tender spots with local spinal joint fixations. Of 54 tender spots, 50 had accompanying fixations. Half of the study received a spinal manipulation, while the other half received a sham mobilization. Cutaneous pain tolerance levels rose 140% in the treated groups as compared to 5% in the controls after 10 minutes. This finding was statistically significant.

Similarly, increases in myofascial pressure pain threshold have recently been reported by Vernon \textit{et al.}\(^{19,20}\) which average approximately 50% of baseline levels, confirming that manipulation of a spinal joint fixation can result in inhibition of pain transmission and reductions of local paraspinal tenderness.

The current literature, as cited above addresses the relationship between 1) spinal joint dysfunction with lowered skin segmental pain thresholds; and 2) the use of the skin rolling technique to locate areas of decreased pain threshold in paraspinal skin. However, no study has reported on the skin roll technique as a reliable method of detecting the locations of a spinal joint dysfunction. It is thus the purpose of this study to investigate the following two hypotheses, herein stated in the null:

\begin{itemize}
  \item \textbf{H1} There exists no correlation between the level of skin roll tenderness and the level of a spinal dysfunction.
  \item \textbf{H2} There is no objective decrease in pain tolerance in the areas of skin roll tenderness relative to areas on non-tender skin roll.
\end{itemize}

Materials and methods

Twenty-five subjects were included in this study. There were 15 males and 10 females. The ages ranged from 20-73 years of age. The mean age was 27.5 years.

The study group consisted of a non-random selection of chiropractic patients already receiving treatment for mechanical pain syndromes in the dorsal spine at the Canadian Memorial Chiropractic College Outpatient Clinic. The only exclusion criterion necessary was that these patients had not received spinal manipulative therapy within the previous 24 hours. All subjects gave informed consent.

The following method was used: the subject was brought into a chiropractic examining room by Examiner A. The subject was placed prone on the examining table. Examiner A placed a small round adhesive sticker on the skin over each of the spinous processes of T1, T4, T7 and T10 as determined by palpation. These were kept in place throughout the entire examination so that each examiner had a common reference to indicate the spinal levels. Examiner A then told the patient: "I'm going to roll your skin between my fingers along your back. Please tell me if it feels that I'm pinching you in one spot more than another."

Examiner A then used both hands to lift the initial roll of skin on the left side of the subject at approximately two centimetres lateral from the midline at the level of the tenth dorsal vertebrae. The skin was then rolled in a continuous manner using both hands upward toward the first dorsal spinous process. The skin was then rolled in a continuous manner using both hands upward toward the first dorsal spinous process. According to Maigne,\(^5\) this first pass serves to introduce the patient to the "feel" of the technique. The roll was then repeated twice more on the left. During the last two passes the subject was asked to report the "pinched" areas. Upon location of a tender area on the third pass, the painful skin was maintained between the fingers of one hand so as to keep the location for the algometer reading as stable as possible.

Using the pressure algometer, (according to previously reported protocols by Fischer\(^21\) and Vernon\(^19\) Examiner A applied steady pressure over the tender spot while securing the skin between the thumb and first finger. The examiner requested the patient to indicate when there was a change from a pressure sensation to a sensation of pain. This point was then recorded in pounds/cm\(^2\) as the pain threshold at the level of the tender skin roll.

Pain threshold levels of each of the tender points were successively measured with the pressure algometer in this manner by Examiner A. Readings were then recorded as to the spinous level at which they were measured. All pressure readings were completed on the left side before repeating skin roll and pressure readings on the right.

Randomly chosen areas of the right and left paraspinal regions from T1-T10 that were not reported as tender during the skin rolling technique were measured with the pressure algometer and recorded to give a control pain threshold value for that patient. These were located at least three segments above or below the tender spot. Examiner A took three to five minutes to...
Skin rolling technique

locate and record this data.

Examiner A then left the room and Examiner B entered. Examiner B then performed a lateral joint challenge from T1–T10. The lateral spinous challenge was performed with thumb pressure on a single spinous process first, with alternating contralateral apposition on the same spinous process (i.e. right to left challenge) and then with contralateral apposition on the segments above or below. The first method located a region of fixation, while the second method confirmed the specific segment. This joint challenge method was performed on each of the spinous processes of T1–T10. At each level where Examiner B determined a relative loss of joint play, it was recorded as a fixed segment (spinal joint dysfunction). The fixations were recorded by spinous levels only and irrespective of direction of the perceived fixation. The subject did not provide feedback as to whether any palpatory manoeuvre was painful. After Examiner B had recorded his findings, the patient was allowed to leave the room. Examiner B took approximately three minutes to locate and record the data. While this form of motion palpation has not yet been studied for inter-rater reliability, it has certainly been adequately described and endorsed by its proponents including Maigne\(^4\) and Gillett\(^22\). Throughout the experiment, each examiner was blinded as to the findings of the other examiner.

The data on levels of skin rolling tenderness and joint play fixation were analyzed for concordance using the Kappa statistic. Kappa (K) values were calculated for exact level agreement and agreement within one level above or below. At each level from T1–T10 the presence of a positive skin roll was assumed to agree with a positive joint challenge if the joint challenge fixation occurred at the same spinal level, the spinal segment above, or the segment below the positive skin roll.

A separate analysis was conducted to compare pressure algometer threshold readings at the point of a tender skin roll to the algometer reading taken at a level with a non-tender skin roll (control threshold point). For each individual patient, all algometer readings at levels of tender skin roll were used to give a mean value. For each patient the mean value of control points was also determined (see Table 1). These values were then used to conduct a paired t-test with \( p < 0.05 \).

Results

When a tender skin roll was used as an indication of spinal fixation at the same spinal level as determined by the joint challenge technique, the level of agreement (true positives and true negatives combined) was 67%. The K value was 0.164 \((n = 250 \text{ (10 spinal levels on each of 25 patients)})\). When the spinal level of fixation as determined by joint challenge was expanded to one level above and level below a tender skin roll level, level of true agreement rose to 77%. (See Appendix 1) The K value increased to 0.48 \((n = 250)\). This was significant at the .001 level.

For each individual patient, the mean algometer reading at the positive skin roll areas was compared to the control paraspinal areas mean algometer reading (Table 1). In all 25 subjects with the exception of one, the control threshold point for pain was higher than the threshold for pain at the level of a positive skin roll. For the one exception, the control point and tender skin point had equal algometer readings.

The values for a tender skin roll for all subjects were combined to give an overall mean value. This was repeated for the non-tender skin roll points. The overall mean value of the algometer readings at the location of a tender skin roll for the patient pool was 11.5 lbs., while the mean of the control points was 15.1 lbs., a 23.9% difference favouring decreased threshold to pain-producing pressure at the level of a tender skin roll when compared to a paraspinal area of a non-tender skin roll. The paired t-test analysis of the algometer results indicated a highly statistically significant difference (\( p < 0.0005 \)).

Discussion

The small Kappa value of the first comparison appears to indicate that when exact level agreement is used, there is poor agreement between the skin roll technique and the joint challenge method. However, the second Kappa value (K = 0.48) relating a tender skin roll to a fixation at, above, or below the spinous level indicates a moderate correlation between skin roll tenderness and fixation. There is, therefore modest support for accepting hypothesis 1. This is especially so, given that the cutaneous branches of a spinal nerve do not exit directly at the surface at the spinal level of origin and there is extensive overlap of adjacent dermatomes in the posterior vertebral region. Maigne\(^4\) postulates that in the upper dorsal spine the posterior branches of the spinal nerves traverse the paravertebral groove by three or four spinal segments before becoming superficial. An example is that the T2 posterior branch emerges superficially at the level of the T5 which can imitate the tenderness of the corresponding articular structure. As well, the acceptance of a match plus/minus one segment (which has been reported previously by DeBoer et al.\(^15\) and Nansel et al.\(^27\)) reproduces the maximum permissible “window” of agreement of segmental levels identified by two independent examiners which still retains the anatomical fidelity required for such an experiment. Since one vertebral level is a component of two contiguous motion segments, then a complex of three levels/two motion segments, is the minimum denotable unit in such a study. The fixation is still contained within that unit, so any of the three vertebral levels serves to designate its presence.

While this will necessarily increase the chance that a match will be found between the levels identified by the two examiners, and while this, alone will increase the Kappa value, we feel that a trade-off is justified. This is analogous to the setting of alpha and beta limits which are set in an experiment in order to contain the element of chance agreement. In this case, exact segment agreement increases (we believe unacceptably) the chance of a false-negative error (type B), while the \( \pm 1 \) segment “window” offers an acceptable (and clinically justifiable) limit on false positives (type A error).
The latter Kappa value ($K = 0.48$) suggests that dorsal spine skin roll tenderness is a moderately good indicator of the presence of contiguous joint fixation, although a poorer indicator of the exact level of fixation. As such, by demonstrating a moderately high correlation between independently measured aspects of spinal dysfunction (i.e., fixation and tenderness) the validity of the "fixation" concept is given further support, even in the absence of data relative to inter-rater reliability of palpation for same. In some sense, this may be even more important, in that, while raters may agree quite highly on the presence of certain findings, if these findings are unrelated to any other clinically important parameters (such as skin tenderness) then the whole question of reliability is moot. We suggest that dorsal skin roll tenderness be used as a screening procedure to indicate that a dorsal joint fixation may be present within a few spinal segments. The high level of agreement between skin rolling findings and the pressure algometer findings, allows us to accept hypothesis 2 and indicates that the skin roll technique provides an accurate indication of local areas of decreased pain threshold along the dorsal spine.

Future studies in this area might distinguish between acute and chronic patients. As well, further exploration as to the sidedness of these two phenomena and the degree of consistency therein would be useful.

### TABLE 1

<table>
<thead>
<tr>
<th>SUBJECT NUMBER</th>
<th>AVERAGE OF ALGOMETER READINGS AT LOCATIONS OF TENDER SKIN ROLL (lbs.)</th>
<th>AVERAGE OF ALGOMETER READING AT CONTROLS (NON-TENDER SKIN ROLL) (lbs.)</th>
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$X_1 = 11.5$  
$X_2 = 15.1$  
$p < .0005$
Conclusion

The investigators found that a correlation between tender skin roll areas and spinal joint dysfunction (fixations) does exist. This study also revealed that subjects who experienced tenderness in areas of their thoracic spine during the skin roll demonstrated a statistically significant difference between these areas of non-tender skin roll.

References


APPENDIX 1: Two by two contingency tables.

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± 1 SEGMENT
Pressure algometry and tissue compliance measures in the treatment of chronic headache by spinal manipulation: a single case/single treatment report

Howard Vernon, DC, FCCS(C)*
Ronald Gitelman, DC, FCCS(C)

A single-case study of chronic bilateral headache is presented with data provided on pressure algometry, visual analogue scale (VAS), and tissue compliance assessment, the former two measured pre-and-post-maniupulation. Pre-treatment recordings demonstrated correlation of findings of joint and muscle dysfunction in the upper cervical spine in this headache sufferer. Post-treatment recordings demonstrated improvements not only in the local spinal tissues but in the distal referred pain pattern (i.e. headache). This case is discussed in regard to both the methods of assessment of soft tissue dysfunction and the theories correlating spinal joint dysfunction with myofascial pain and their relief with manipulation.

(Key Words: headache, upper cervical, pressure algometer, tissue compliance, manipulation, chiropractic.)

Introduction

Our present knowledge of spinal pain syndromes is not yet so adequate that we can precisely distinguish the role of dysfunction of articular structures as opposed to that of the myofascial elements. The view in some circles is a dichotomous one, in which joint and muscle structures are treated separately - thus leading to such concepts as "trigger points" being considered solely a myofascial phenomenon, while joint strains are purely of articular concern, with no impact on muscle function. Others find this dichotomy artificial and, in some sense, irrelevant given that the object of treatment is the anatomical/functional unit as a whole. Myofascial pain specialists, generally subscribers to the former view, have elevated the myofascial tender point to its rightful place in the musculoskeletal pathophysiolog-
multitude of empirical reports exist in the literature of all the disciplines involved in manipulation (see review in Vernon) on the connections between upper cervical dysfunction, headache and relief by manipulation.

However, very few reports provide objective quantification of the myofascial component (tenderness, and increased stiffness (i.e. hypertonicity) and its association with the articular component (motion blockage or fixation). Jaeger has recently reported on a small case series in which disturbances measured by algometry, motion and static palpation and cervical spine gross ranges of motion have been associated with headache. Bogduk has reported on the association between motion palpation and facet injection such that the results of injection therapy improved when specifically targeted to the spinal joint segment level identified as blocked on motion palpation. This work follows closely from that of Jull in which headache and non-headache groups were distinguished by the higher prevalence of fixations in those with headaches.

Methods and results
A 39-year-old married, female occupational health nurse presented with a 25 year history of headaches. The predominant headache mode was a daily, bilateral frontal aching pain which conformed to the classification of chronic muscle contraction headache (MCH). The patient was symptomatic when presented to the clinic and had been so for the previous six hours. Her chronic condition was punctuated by very infrequent episodes of more acute severe headaches, which in the past have been preceded by aura-like phenomena and which had been labelled as migraines. She had not suffered one of these headaches for eight years although she had the occasional severe variation of the MCH. She also complained of chronic neck and upper back pain, but the headache was not described as part of this cervical pain condition. The patient had been under long-term chiropractic care which she acknowledged was the only source of relief she had obtained. Treatment had apparently reduced the overall severity of the headaches and had prevented the severe attacks almost completely. She was referred to our specialty clinic for evaluation of any perpetuating factors which might not previously have been detected.

Postural evaluation revealed a forward head carriage with shrugged shoulders. Inspection and palpation revealed hypertonicity in the upper trapezius muscles bilaterally, worse on the right, as well as in the suboccipital muscles, again worse on the right. Tender points (TP) were detected in each of these muscles, with the right-side points being more tender than those on the left. Motion palpation revealed a major fixation at Cl-C2 on the right, so that a complex of findings - restriction of anterior rotation of atlas, palpable spasm and local tenderness of the ipsilateral suboccipital muscles - appeared to be the major clinical finding.

Two methods of quantifying the myofascial component in this type of disorder are pressure threshold algometry and tissue compliance assessment. Both of these methods have been introduced by Fischer, who has reported extensively on reliability and normative data of these clinical instruments. We have also reviewed the method of pressure algometry and have shown its usefulness in studies of the effects of spinal manipulation. Tissue compliance assessment is a variation of pressure algometry which purports to measure the stiffness of the soft tissues - an objective finding which does not require the patient's participation in the measurement. The tissue compliance meter (TCM) measures the depth of penetration of the pressure plunger into the surface of the soft tissues at various levels of force, so as to detect the relative stiffness of these tissues. Force/penetration graphs are obtained for 2, 3 and 4 kg/cm² force levels (see figures 1 and 2). The normal values reported by Fischer for the upper trapezius muscle are 12, 14 and 17 mm, for 2, 3 and 4 kg/cm² levels, respectively. The critical criteria in pressure algometry (tenderness) are such that values below 3 kg/cm² are clinically significant, as well as any bilateral difference which exceeds 1 kg/cm².

Pressure algometry, TCM and VAS (visual analogue scale) scores were conducted in this case, the results of which are presented in Table 1. The clinically relevant findings were the increased stiffness (or hypertonicity) of both upper trapezius muscles, the right being worse than the left, and the significant reduction of the pressure threshold in the medial occipital point on the right (corresponding to the insertion of rectus capitis anterior muscle), which was greater than 15 mm.

Table 1

<table>
<thead>
<tr>
<th>Table 1: ASSESSMENT PROFILE</th>
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<tbody>
<tr>
<td>1 – Tender Points (kg/cm²)</td>
</tr>
<tr>
<td>Pre-Rx</td>
</tr>
<tr>
<td>Medial-occipital</td>
</tr>
<tr>
<td>Sub-occipital</td>
</tr>
<tr>
<td>Trapezius</td>
</tr>
<tr>
<td>Scalenues</td>
</tr>
<tr>
<td>2 – Muscle Compliance (pre-treatment only)</td>
</tr>
<tr>
<td>Normal</td>
</tr>
<tr>
<td>Trapezius – 2 kg</td>
</tr>
<tr>
<td>– 3 kg</td>
</tr>
<tr>
<td>– 4 kg</td>
</tr>
<tr>
<td>3 – VAS (mm)</td>
</tr>
<tr>
<td>Pre-Rx</td>
</tr>
<tr>
<td>63</td>
</tr>
</tbody>
</table>
Figure 1  Pressure algometer used over occipital tender point.

Figure 2  Tissue compliance meter used on trapezius muscle.
posticus minor from C1–C0). These findings quantitatively corroborate those clinical findings noted previously, namely – increased hypertonicity of the trapezius muscle upon digital palpation and increased tenderness to digital pressure of the suboccipital muscles (trigger point) on the right. During the history taking, the patient was asked to rate her present headache intensity on a standard 10 cm VAS whose left and right anchors were “no headache” and “worst headache imaginable” respectively. Her VAS score was 63 (on the 100mm scale).

A rotational manipulation of atlas-axis (see Szaraz10) was performed and the medial occipital TP was remeasured after five minutes of rest. The post-treatment value was 2.3 kg/cm² which represented an increase of 53%. Just prior to leaving the clinic she was asked to score her headache again on the VAS. An interval of approximately 15 minutes had elapsed. She scored 14, which represented approximately 80% improvement. Tissue compliance data was not obtained post-treatment.

Discussion
In our previous small randomized controlled study of the effects of manipulation on cervical tender points, we reported an average 45% increase in pressure threshold in manipulated subjects, as compared to those receiving a sham mobilization.15 In a single-case study, Vernon reported that manipulation resulted in pressure threshold increases in seven cervico-thoracic muscles, which also averaged 45%.14

The clinical result reported here is consistent with a great number of studies reporting relief of headache and neck pain by spinal manipulation (see reviews in Vernon et al.3 and Vernon11). Although the quantitative measurements in this single-case study were obtained in an unblinded fashion, cautious interpretation is always urged. However, even these data would corroborate the common anecdotal experience of most clinicians, that pain, tenderness and spasm in the soft tissues are often accompanied by focal joint dysfunction (typically related neuromerically12) and that these symptoms and signs are often improved after spinal manipulation.

Conclusion
Quantification of clinical findings reported in a single-case study is important both because it adds precision to the diagnosis and because generalization of the findings in this type of case report can also aid in the verification of important components of the manipulation paradigm. In this paradigm, joint and muscle dysfunctions are correlated, and manipulation results in improvement in the clinical behaviour of the functional unit. In this case, a referred pain – bilateral frontal headache – was shown to decrease in correspondence with reductions in local spinal soft tissue tenderness after a spinal manipulation to the related segment.

References
Spinal manipulation and headaches of cervical origin
A review of literature and presentation of cases

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Summary. Claims of therapeutic efficacy for spinal manipulation in headache have been reported by all of its proponents, although in the mainstream of headache management it is virtually ignored. The validity of these claims and the mechanisms of action of spinal manipulation are largely undetermined. This report presents a critical review of the quantitative clinical research to date. In addition, three case reports are presented to illustrate the empirical experience of spinal manipulation in the treatment of headaches.

Key words: Cervicogenic headache – Spinal manipulation

The case for spinal manipulation in the treatment of headaches of cervical origin contains within it two controversies. The first is the nature and validity of the issue of “headaches of cervical origin.” There is still great debate about the role of the cervical spine in the etiology of a variety of headache types. This is despite a long-standing tradition of writing in medical and heterodox circles supporting the notion of cervicogenic headache. The recent works published by the groups of Bogduk [2-5], Sjaastad [3, 32], Fredrickson [12-14], Pfaffenrath [27, 28] and Jaeger [17] have all supported the validity of “cervicogenic headaches” and have produced important descriptive and explanatory models. Others, notably Edmeads [9, 10] remain unconvinced. The reader is referred to the author’s extensive reviews for a more in depth treatment of this issue [37, 38].

The second controversy, of course, builds on the first. If there is still confusion and doubt regarding the cervical origin of some headache types, there is certainly virtual ignorance concerning the role of spinal manipulation in the treatment of headaches. Nonetheless, a number of professionals groups do practice spinal manipulation, some more exclusively than others, including chiropractors, medical doctors (manual medicine), osteopaths and physical therapists. Members of each of these groups have claimed success from spinal manipulation in the treatment of headache [38]. This report will review all the reported clinical studies in which spinal manipulation was applied as the primary therapeutic agent and will present three case studies exemplifying the role of spinal manipulation in the management of acute headaches.

Literature review: muscle contraction headaches

The role of manipulation in the treatment of muscle contraction or non-migrainous headache has been alluded to by a number of authors who initially wrote on headaches of cervical origin. In particular, Kovacs [21], Seletz [29], Frykholm [15], Grillo [16], Bogduk [2] and others have specifically reported success with manipulation. However, only in the past two decades have quantitative reports emerged. In 1971, Lewit [22] reported on 41 late adolescents (21 females, 20 males) who had sustained cervical spine anteflexion sprain injuries in gymnastic activities and who were presenting with headache. Seventy percent (70%) of these subjects were found to have hypermobility of C1-2. Lewit reported that manipulation was the single most effective treatment from amongst a number of physical modalities.

In the same study, Lewit also reported on a series of 93 adults who had sustained head/neck trauma. Ninety-six percent (96%) of these subjects demonstrated movement restriction in the upper cervical spinal joints. Again, manipulation was reported to be very successful in treating the headache associated with these injuries. Unfortunately, in these two case series presentations, no quantified data on outcomes was presented by Lewit, nor was treatment by manipulation compared with either a control or any other form treatment, so that the degree of natural remission cannot be determined from the reported success rates. Nonetheless, in this early report, manipulation is presented as a treatment of some significant value.

Single-case studies have been reported by several investigators. In 1980, Mannen [23] reported a single-case study in which non-vascular headache was relieved by a course of chiropractic treatment. Data on segmental motion of the cervical spine as analyzed on flexion/extension X-rays were provided, which appeared to indicate...
that improvement in upper cervical mobility accompanied the improvement in symptoms.

Vernon [36] in 1983, reported on a descriptive survey of two groups of non-vascular headache subjects presenting to a chiropractic college out-patient clinic. One group (n = 15) was studied retrospectively by mail survey, while the other group (n = 18) was studied prospectively within the clinic. In each group, statistically significant reductions in headache characteristics, including frequency, duration and severity, were obtained with a mean of nine treatments. In addition, mean satisfaction scores, obtained from an index of three Likert scales (1-5, total = 15) were 12.2 in the retrospective and 12.7 in the prospective groups, respectively, indicating approximately 90% satisfaction with treatment. Finally, subjects were asked to indicate the presence of four (4) "autonomic" symptoms - nausea, dizziness, tinnitus and aura. Of 7 subjects reporting nausea prior to treatment, all reported complete relief, and 8 out of 9 (94%) subjects reported relief of dizziness. No subjects reported experiencing tinnitus or aura. These data were also statistically significant.

Miller et al. [24], in 1984, reported on a single case of "long-standing uncomplicated tension headache". Thirteen (13) manipulative treatments were rendered, resulting in a reduction of headache frequency from 2.6/day to 1.0/day within 1/week. Use of analgesic medication was similarly reduced.

In 1985, Jirout [18] reported on 200 subjects who had previously been involved in a roentgenological study of motion blockage or fixation at the C2-3 segments. Ninety percent (90%) of subjects with this vertebral dysfunction reported the presence of headaches. Manipulation, directed to these areas of fixation, resulted in complete relief in approximately 80% of this sample.

Droz and Crot [8], in 1985, reported on chiropractic care of "occipital headaches." They conducted a retrospective survey of 332 subjects. Treatment results were reported as follows: 80% had complete relief, 10% had good relief, (75% reduction) and 3% were slightly better (50%), giving a 93% success rate. They noted that no subject was worsened.

Turk and Ratkolb [35] in 1987, reported on 100 cases of headaches attributable to the cervical spine that were treated by manipulation. Nine manipulation treatments produced significant reduction of headaches in 75% of subjects. At the 6-month follow-up, 65% still had significant improvement, while 35% had recurrence of symptoms. A significant reduction of analgesic usage was reported.

In summary, the literature includes six descriptive surveys of treatment outcomes (see Table 1). None of these studies was a randomized controlled trial, nor are there any comparative trials. The empirical literature reports success rates ranging from 80% to 90%, but no scientific test of these claims has yet been forthcoming.

### Migraine headaches

Numerous authors cite the advantage of spinal manipulation in the treatment of migraine, without actually reporting any quantitative results. The work of Figar and Jan-sky [11] on cranial-vascular reflexes in subjects with "vertebrogenic migraine" refers to the success obtained with manipulation. A host of chiropractic and osteopathic writers [38] have extolled the virtues of manipulation in the treatment of migraine. Notwithstanding this extensive empirical tradition, only two studies have provided data on treatment outcomes, one of these being the only randomized clinical trial in the literature.

Wight [39] in 1978, reported on 87 non-randomly selected subjects (57 females, 30 males), 34 of whom had common migraine, and 53 with classic migraine. The

<table>
<thead>
<tr>
<th>Authors</th>
<th>Reference</th>
<th>Manipulators</th>
<th>No. of subjects</th>
<th>Headache</th>
<th>Results</th>
</tr>
</thead>
<tbody>
<tr>
<td>Lewit</td>
<td>[22]</td>
<td>A</td>
<td>41</td>
<td>MC</td>
<td>Manipulation was the most effective treatment</td>
</tr>
<tr>
<td>Lewit</td>
<td>[22]</td>
<td>A</td>
<td>93</td>
<td>MC</td>
<td>Manipulation most effective</td>
</tr>
<tr>
<td>Vernon</td>
<td>[36]</td>
<td>B</td>
<td>33</td>
<td>MC</td>
<td>85 - 90% success</td>
</tr>
<tr>
<td>Jirout</td>
<td>[18]</td>
<td>A</td>
<td>200</td>
<td>MC</td>
<td>80% success</td>
</tr>
<tr>
<td>Droz &amp; Crot</td>
<td>[8]</td>
<td>B</td>
<td>332</td>
<td>MC</td>
<td>80% very successful</td>
</tr>
<tr>
<td>Turk &amp; Ratkolb</td>
<td>[35]</td>
<td>A</td>
<td>100</td>
<td>MIG</td>
<td>75% success</td>
</tr>
<tr>
<td>Wight</td>
<td>[39]</td>
<td>B</td>
<td>57 (F)</td>
<td>MIG</td>
<td>75% success</td>
</tr>
<tr>
<td>Parker et al.</td>
<td>[25]</td>
<td>A and B</td>
<td>52 (F)</td>
<td>MIG</td>
<td>71% success (w/o Parker et al. = 80%)</td>
</tr>
<tr>
<td>Total no. of subjects</td>
<td></td>
<td></td>
<td>971</td>
<td></td>
<td></td>
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<tr>
<td>Average outcome</td>
<td></td>
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<td></td>
<td>71% success (w/o Parker et al. = 80%)</td>
</tr>
</tbody>
</table>

A, Doctor of medicine; B, chiropractor; MC, muscle contraction; MIG, migraine (adapted from [18])
results of chiropractic treatment were as follows: 80% improvement levels were classed as “greatly improved,” and 85% of females and 50% of males in the common migraine group obtained this level of improvement, while 78% of females and 75% of males in the classic migraine group were similarly helped. The overall success rate for both categories for both sexes was 74.7%. In those subjects not reporting a greatly improved status after treatment, a reduction of severe attacks was achieved for 81.5% of females and 45.8% of males in the common migraine group and 43.2% and 34.5%, respectively, in the classic migraine group.

Parker et al. [25] also in 1978, conducted a randomized controlled trial in 85 subjects (33 males, 52 females) with “migraine headaches”, comparing chiropractic manipulation, medical manipulation and control/mobilization performed by physiotherapists. The mean age of the subjects was 41 years, while the mean duration of the complaint was 19 years. Seventy percent (70%) suffered from common migraine, while 30% suffered from classic migraine. Subjects underwent a 2-month baseline, from a 2-month treatment stage, and a further 2-month follow-up period. An average of seven (7) treatments were delivered in the treatment stage.

The frequency of migraines was reduced by 40% in the chiropractic group, 34% in the mobilization/control, and 13% in the medical manipulation group. The overall improvement for all three groups was 28%. Only one hypothesis achieved statistical significance (0.01), that being that the severity of headaches was reduced in the chiropractic manipulation group. Pretreatment measures of expectation of outcome response by subjects and treaters indicated a higher response in the chiropractic group. The authors concluded that such high expectations exerted a direct effect on pain ratings, and, as such, the finding that a single parameter of headache activity (severity) was significantly reduced in chiropractic-treated subjects was probably due to a placebo response.

A follow-up study was published in 1980 [26] examining the psychosocial correlates to successful outcomes in the first study. It was noted that at 20-month follow-up, a further 19% of subjects had achieved a good recovery, constituting an overall 47% success rate. An interesting aspect of the data that did not receive statistical attention was the number of subjects who achieved a complete recovery. Of these, 14 subjects (16%), 8 (57%) were in the chiropractic group, 1 (7%) was in the medical group, while 5 (36%) were in the control group. Regarding the psychosocial correlates of successful treatment outcome, only one — sex — achieved statistical significance (0.01) so that, according to Parker et al., female patients respond significantly better than male ones to “physical therapy” treatments for migraine.

Wight’s study was, of course, no more than a retrospective survey of a clinician’s own level of success within his practice, and it cannot therefore be regarded as having high validity. While the Parker et al. study was a randomized controlled trial, it was not without flaws, which combined to create a threat to the external validity of the trial. First, a relatively small number of treatments (7) was employed, which, especially since they extended over a 2-month period (i.e., one treatment per week) may very well have been insufficient to effect much change in what is accepted by all to be a chronic, recurrent condition. More importantly, though, in all of the hypotheses tested, which included reductions in headache frequency, duration and use of medication, and which, as reported in the study, did not achieve statistical significance, the greatest improvements were in the chiropractic-treated subjects. As such, this study suffered from a low power, a common fault in many clinical trials (especially involving manipulation) [7]. With a larger sample size, this clinical trial would have produced a positive result for chiropractic manipulative treatment of migraine.

Table 1 summarizes the extent of clinical studies in which spinal manipulation was used as a primary treatment for both muscle contraction and migraine headache.

Case reports

Case 1: A 35-year-old woman presented with an 11-day history of unilateral upper neck pain and headache. She related the onset of her pain to a stay in hospital during which she underwent a surgical procedure with general anesthesia. At postoperative recovery, she began to experience intense pain localized to her right suboccipital region which radiated forward around the temple to the supraorbital area (see Fig. 1). She had no previous history of neck pain and/or headaches. By the 11th day, the headache was described as a constant sharp pain of high severity (see Fig. 2: baseline.) The headache was not accompanied by nausea, lacrimation, pain in the right eye or dizziness [13]. The patient was quite debilitated by her condition.

Examination showed an analgic head/neck posture with persistent tilt and rotation of the head to the left. Head/neck movements to the right in rotation and lateral flexion as well as in backward extension reproduced the upper cervical pain and mildly provoked the headache. Manual palpation revealed the presence of suboccipital muscle spasm on the right with a trigger point located in the right rectus capitis posterior major muscle, which is easily palpated between the spinous process of C2 and the occipital brim, which were also sites of
tenderness. The spinous process of C2 was felt to be quite prominently rotated to the right, and joint play movement was completely absent when pressure was applied to the spinous process toward the left (i.e., to stress C2–3 in right rotation) indicating hypomobility or fixation of C2–3 on the right [6, 19]. This joint play maneuver was the most tender of all examination procedures. Palpation of the tender points in the joint play maneuver reproduced the radiating head pain.

Plain film X-rays were obtained of the cervical spine. The open-mouth A-P view revealed severe offset of the spinous process of C2 to the right confirming the physical findings of the examination (Figs. 3, 4). The lateral view demonstrated alordosis.

It was concluded that a hyperextension and rotation strain of the upper cervical spine had occurred during surgery (i.e., related to general anesthesia), which had resulted in a traumatic facet syndrome of C2–3 on the right producing local and radiating neck and head pain symptoms, which were easily reproduced during the examination.

This patient was treated with a course of manipulative adjustments directed to the hypomobile C2–3 facet joint. Figure 2 shows the time course of the severity and medication usage as well as the incidence of headache on a day-to-day basis. A single manipulation directed at the hypomobile C2–3 facet joint resulted in over 80% relief within 1 day. The second manipulation resulted in complete remission of the symptoms.

Follow-up at weekly intervals for 3 weeks revealed no recurrence of any neck or head pain and no tenderness, spasm or hypomobility associated with the C2–3 facet joint. The patient was discharged with a complete recovery.

Case 2: A 37-year-old man presented with a 3-day history of neck pain and headaches brought on by a twist of the neck during some horseplay with his children. He located his pain focally in the suboccipital region on the left with a more diffuse radiation into the left scapular area (Fig. 5). In addition, he complained of a bilateral frontal headache which was felt as a constant severe ache. He denied any previous history of neck pain and headaches. There was no nausea or lacrimation.

On examination his head was held stiffly in the mid-line. Painful ranges of head/neck movement included left rotation, left lateral flexion and extension. Spasm was noted on manual palpation in the left suboccipital muscles and the left anterior cervical muscles in the lower cervical area. There were trigger points in these muscles as well, each of
which reproduced a component of the complaint — the suboccipital trigger point referred pain to the left supraorbital area, the scalene trigger point referred pain to the scapular region on the left. Joint dysfunction (hypomobility) was detected at C2–3 and C5–6 on the left associated with the scalene muscle spasm. X-rays were not obtained.

At the time of our consultation the headache was rated as daily, with a severity score of 10/10 on days 1 and 2, and 5/10 on the 3rd day. Aspirins were being used three times daily. The diagnosis was C2–3 facet syndrome on the left with a bilateral cervicogenic headache.

A course of manipulation was initiated which produced immediate results. Figure 6 charts the outcome parameters. The active treatment phase lasted 1 week at which time the patient was virtually painfree. Three-week follow-up showed no recurrence.

Case 3: A 43-year-old woman presented with a 10-day history of right-sided suboccipital pain of no attributable onset. The pain was described as constant, sharp and quite localized and accompanied by a radiation of headache over the right temple to the right supraorbital area. This radiation was described as a sharp pain which had a zone of tenderness and sensitivity located over the right parietal area (Fig. 7). The patient denied any previous history of neck complaints or severe headache.

Examination revealed that her head was held stiffly in the mid-line. There was painful limitation of rotation and lateral bending to the right which reproduced the local suboccipital pain. Forward flexion was painfully limited and reproduced both neck and head pain. There was spasm in the suboccipital muscles on the right, chiefly those around the first cervical joint. There was no significant tenderness over the right C2–3 facet joint. There was exquisite tenderness over the occipital rim at the middle occipital point which was interpreted to be over the exit of the greater occipital nerve. This pressure recreated the head pain precisely. Joint fixations were palpated between occiput-atlas, and C1–2.

The headache was rated as daily in frequency and as a 10/10 in severity. There was no nausea, lacrimation or photophobia. There was no jaw pain and no pain in the ear or in the eye on that side. There were no other ocular symptoms. The diagnosis of occipital neuralgia secondary to upper cervical joint dysfunction was made. Treatment by manipulation was commenced.

By the third treatment (1 week), a 50% improvement was obtained. After another 9 days (or after six treatments), all the patient's symptoms had resolved and her medication use was terminated. This status was maintained for the next 6 weeks until the patient was discharged (Fig. 8).
Discussion

Sjaastad, Fredrickson and their colleagues have insisted that the diagnosis of cervicogenic headache currently be restricted to unilateral headaches aggravated by neck movements and accompanied by autonomic signs, primarily nausea and lacrimation. They have speculated that the cause of this syndrome is most probably a "rhizopathy at C2-3" [32]. Bogduk [2, 5] and others [8, 15, 16, 18, 21, 22, 24, 35, 36] have consistently characterized this rhizopathy as a "hypomobility" of the upper cervical apophyseal joints which is best detected by the skillful manual palpatory techniques practiced typically by chiropractors and other practitioners of spinal manipulation. It is the contention of Bogduk [2] and of numerous other authors [38] that manipulation is an ideal form of treatment for this problem and, hence for cervicogenic headache.

This case series demonstrates the kind of therapeutic efficacy typical of the case series literature on manipulation. In the first two cases improvement was noted very rapidly (each within 1 day), while in the third case improvement was more gradual. Each of these cases deserves comment both for its relationships to the "cervicogenic" diagnostic category and for its response to manipulation.

Case 1 is noteworthy for the mode of onset. Traumatic injury during operative anesthesia is a rarely reported cause of headache and one which in this case was dismissed as "stress-related." The cervicogenic etiology was detected only later, even though the clinical picture was the most similar to that of the cases of Sjaastad et al. [32]. Even so, the autonomic concomitants were absent, and provocation occurred with extension, not flexion. While numerous manual diagnostic procedures and X-ray findings confirmed the cervical origin, the response to corrective treatment directed at the offending spinal joint dysfunction by manipulation certainly clinched the diagnosis.

While case 2 is quite similar in its ultimate presentation and in the obvious involvement of the C2-3 facet joint, it is distinguished by the bilaterality of the headache. Even while Sjaastad et al. [32] have insisted on unilaterality, they have in fact proposed the curious category of double unilateral headaches involving both sides of the cervical spine separately, creating individual but fused head pain patterns. Sessle et al. have found that over 30% of convergent neurons in the nucleus subcaudalis are stimulated by contralateral input from the C3 nerve root [30], providing a neuro-anatomical basis for contralateral as well as ipsilateral pain referral from a unilateral facet dysfunction. As such, the insistence on unilaterality may have some a priori appeal in increasing diagnostic specificity, but it will ultimately fail to include the full range of headaches of cervical origin.

Case 3 is noteworthy in that it represents a fairly typical case of occipital neuralgia but an untypical "cervicogenic" headache, since that the prominent facet involvement was higher in the upper cervical complex, especially at occiput-atlas. Hypomobility of this joint is rarely mentioned; however, Lewit did remark on "pain arising from the posterior arch of atlas" [22]. Pfaffenrath et al. found the most consistent hypomobility in the cervical spine on computer-aided motion X-ray analysis was at C0-1 [28]. This dysfunction is theoretically just as potent a source of noxious input and is also potentially a source of direct irritation of the greater occipital nerve as it passes over the occipital bim. There are no reports of manipulation in the treatment of "occipital neuralgia," but even here, diagnostic confusion and mislabelling have probably clouded the picture.

The mechanism of effect of spinal manipulation is still speculative. Nonetheless, its proponents argue that spinal manipulation results in reduction of joint pain [40], reduction in muscle spasm [20], and reduction of tenderness in myofascial structures [34]. The reduction of nociception from upper cervical joint structures would certainly be reflected in reduced activation of the neural pathways of referred head pain subserved by the spinal tract of the trigeminal nerve [1] and the C1 and C2 nerve roots [33]. Manual therapeutics have traditionally been associated with manual diagnostic procedures such as have been described above. Bogduk [5] and Jull [19] have provided validation for this empirical situation in which joint dysfunctions can be reliably detected and effectively corrected by specific manual methods.

Conclusion

Three cases of acute headache of cervical origin have been described. Manual diagnostic procedures allow for confidence in the diagnosis prior to a treatment trial, while the response to manipulation clinches the diagnosis retrospectively. Headache specialists are urged to consider broadening the criteria for "cervicogenic headaches." In addition, more attention should be paid to spinal manipulation as a potentially effective modality of treatment for this broader spectrum of headache categories.

References

The Neck Disability Index: A Study of Reliability and Validity

HOWARD VERNON, D.C.* AND SILVANO MIOR, D.C.*

ABSTRACT

Injuries to the cervical spine, especially those involving the soft tissues, represent a significant source of chronic disability. Methods of assessment for such disability, especially those targeted at activities of daily living which are most affected by neck pain, are few in number. A modification of the Oswestry Low Back Pain Index was conducted producing a 10-item scaled questionnaire entitled the Neck Disability Index (NDI). Face validity was ensured through peer-review and patient feedback sessions. Test-retest reliability was conducted on an initial sample of 17 consecutive "whiplash"-injured patients in an outpatient clinic, resulting in good statistical significance (Pearson's $r = 0.89$, $p < .05$). The alpha coefficients were calculated from a pool of questionnaires completed by 52 such subjects resulting in a total index alpha of 0.80, with all items having individual alpha scores above 0.75. Concurrent validity was assessed in two ways. First, on a smaller subset of 10 patients who completed a course of conservative care, the percentage of change on NDI scores before and after treatment was compared to visual analogue scale scores of percent of perceived improvement in activity levels. These scores correlated at 0.60. Secondly, in a larger subset of 30 subjects, NDI scores were compared to scores on the McGill Pain Questionnaire, with similar moderately high correlations (0.69–0.70). While the sample size of some of the analyses is somewhat small, this study demonstrated that the NDI achieved a high degree of reliability and internal consistency. As well, it appears to be sensitive to the levels of severity of complaint, and to changes in severity in the course of treatment. While further study is recommended, the NDI is offered as a potentially useful instrument in the assessment of neck pain complaints.

Key Indexing Terms: Cervical Vertebrae, Pain Measurement, Disability.

INTRODUCTION

Neck pain complaints, especially those arising from motor vehicle accidents (MVA), affect an increasing number of persons. Fortunately, the majority of such accidents do not result in death, but unfortunately, bodily injuries do occur. In 1986, available Canadian MVA statistics (excluding Quebec) revealed an increase of 15% in bodily injuries as opposed to a 4% decline in deaths during the previous 3 yr (1) (Table 1). In addition to these annually reported "new" injuries, those previous injuries whose symptoms have not resolved must be taken into account. According to Ameis (2), 15% of MVA victims fail to achieve full functional recovery, while 40–70% have some mild to moderate symptom persistence. Consequently, the annual prevalence rates of persons suffering as a result of motor vehicle accident-related injuries significantly increases.

Such increases are not only reflected in prevalence rates but in cost estimates as well. From the data cited above (1), of approximately 3.75 billion dollars spent on MVA-related claims, 46% (or about 1.75 billion dollars), was paid for bodily injuries. These figures may underestimate the actual costs, since they fail to consider lost work days and litigation-related expenses.

Considering the costs to society of these and other nontraumatic neck pain complaints, it is surprising that there is a paucity of methods of evaluating patients with soft tissue injuries of the cervical spine. The existing evaluations are quite underdeveloped and unsystematic. They are conducted by a variety of practitioners, some of whom are designated as "independent" or "expert." Their evaluations are typically qualitative, subjective, uninstrumented, and often based solely...
TABLE 1. Validity analysis of NDI

<table>
<thead>
<tr>
<th>Validity analysis</th>
<th>Correlation (Pearson’s r)</th>
</tr>
</thead>
<tbody>
<tr>
<td>1. NDI vs. MPQ total score</td>
<td>0.70</td>
</tr>
<tr>
<td>2. NDI vs. MPQ number of words</td>
<td>0.69</td>
</tr>
<tr>
<td>3. NDI % change vs. VAS % change</td>
<td>0.60</td>
</tr>
</tbody>
</table>

upon clinical experience and the reputation of the evaluator.

Since whiplash-type injuries create far more morbidity than mortality, it is sensible to approach the evaluation of such injuries via an assessment of the disability which is eventually experienced by so many of its sufferers. One of the strategies that has been adopted to assess disability in other musculoskeletal pain conditions is to assess the effect of the condition on activities of daily living (ADL) (3–5). In assessing low back pain, for example, Fairbank et al. developed the Oswestry Low Back Pain Index (6), a 10-item ordinal scale questionnaire, with a maximum score of 50 (maximum of 5 on each of the 10 items). This instrument was found to reliably and sensitively measure the impact of low back pain on specific, typical and relevant activities of daily living. Triano and Schultz (7) have recently shown that Oswestry scores above 13 out of 50 correlate highly with the presence of objective measures of lumbar muscle dysfunction and high pain scores; scores below 13 are, conversely, highly correlated with the absence of these findings. The Roland-Morris scale, was developed as an easy-to-use form of the Sickness Impact Profile (SIP), which has been found to correlate well with Oswestry scores (8). The SIP, itself, is a much larger instrument consisting of 136 items. The need for shorter, complaint-specific versions of the SIP has been recognized even by its proponents (9).

Another instrument, recently developed by Pollard (10), is the Pain Disability Index (PDI). The PDI is composed of seven visual analogue scales that rate a subset of activities of daily living. The PDI has been shown to have good reliability, and to validly discriminate levels of complaint severity. It also correlates well with other measures of psychosocial disturbance, notably, abnormal illness behavior (11). The PDI was not designed for any specific pain complaint (in fact, its great usefulness lies in its broad applicability), and it appears not to have been used in the study of neck pain complaints.

After conducting the standard computerized searches, we were unable to find an instrument which assesses ADL in persons with neck pain. An instrument like this would hopefully have an impact on the management of such patients at a number of separate, but interdependent levels, including general clinical practice, medicolegal and insurance proceedings, as well as in research into the characteristics and responses to the treatment of whiplash and chronic neck pain sufferers. Toward that end, an instrument has been developed known as the Neck Disability Index (NDI). This is a revised form of the Oswestry Low Back Pain Index.

The purpose of this paper is to report the reliability and validity of the NDI.

METHODS

After permission from the authors was obtained, the Oswestry Index was reviewed with the intention of identifying those items which appeared most relevant to the needs of assessing patients with neck pain. Six items were initially found to be suitable (pain intensity, personal care, lifting, sleep, driving, and sex life). A list of additional ADL applicable to neck pain complaints was compiled from a review of the descriptive literature on whiplash and chronic neck pain (1, 12–18). This list was submitted to a consulting team of clinical practitioners whose consensus ratings resulted in four additional items: headaches, concentration, reading, and work. Rating scales similar in structure to those of the Oswestry Index were then prepared and submitted to the team for review. Syntactic and scaling errors were corrected and a first draft was prepared. This was submitted to a pilot group of five whiplash sufferers in the Outpatient Clinic of the Canadian Memorial Chiropractic College, who reviewed the form for ease of reading, comprehensibility and personal relevance of the different items.

The only item that provoked unanimous disconcertment, and which several subjects refused to answer, was "sex life." This was removed and replaced by "recreation," which was found to be acceptable to all of the pilot group of patients. One final revision occurred at the suggestion of the review team. Two items in the original Oswestry Index involved scales that contained the "use of tablets" as the dependent measure ("pain intensity" and "sleep"). It was suggested that such phrasing would be, on the one hand, semantically awkward to most North Americans, and, on the other hand, would prove difficult to answer for those patients not taking "tablets" (i.e., medication or "pills") for pain relief or for the management of sleep disturbance. The rating scale for "pain intensity" was thus altered to reflect only the intensity of the disturbance as opposed to the use of a particular form of treatment (such a correction has since occurred for the "pain intensity" item on the original Oswestry Index [J. Fairbank, personal communication]). A similar modification was
made to the "sleep" item, unfortunately only later in the study.

Therefore, the final form of the NDI consisted of five scales from the original Oswestry Index, two of which were revised considerably, and five new scales (Figure 1). This stage of development completed the determination of face validity, resulting in a comprehensible, relevant instrument, which shared the features of ease of use, scoring and the general format of presentation as derived from the original Oswestry Index.

Forty-eight subjects, 17 males and 31 females, suffering from neck pain who visited an outpatient chiropractic clinic (at the Canadian Memorial Chiropractic College) from October 1987 to June 1988 were studied. The average age of the subjects was 35 yr for the females (range 22-48 yr) and 41 yr for the males (range 18-55 yr).

**NECK DISABILITY INDEX**

This questionnaire has been designed to give the doctor information as to how your neck pain has affected your ability to manage in everyday life. Please answer every section and mark in each section only the ONE box which applies to you. We realize you may consider that two of the statements in any one section relate to you, but please just mark the box which most closely describes your problem.

<table>
<thead>
<tr>
<th>Section 1 - Pain Intensity</th>
</tr>
</thead>
<tbody>
<tr>
<td>I have no pain at the moment.</td>
</tr>
<tr>
<td>The pain is very mild at the moment.</td>
</tr>
<tr>
<td>The pain is moderate at the moment.</td>
</tr>
<tr>
<td>The pain is severe at the moment.</td>
</tr>
<tr>
<td>The pain is the worst imaginable at the moment.</td>
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</tbody>
</table>

<table>
<thead>
<tr>
<th>Section 2 - Personal Care (Washing, Dressing etc.)</th>
</tr>
</thead>
<tbody>
<tr>
<td>I can look after myself normally without causing extra pain.</td>
</tr>
<tr>
<td>I can look after myself normally, but it causes extra pain.</td>
</tr>
<tr>
<td>It is painful to look after myself and I am slow and careful.</td>
</tr>
<tr>
<td>I need some help, but manage most of my personal care.</td>
</tr>
<tr>
<td>I need help every day in most aspects of self care.</td>
</tr>
<tr>
<td>I do not get dressed, I wash with difficulty and stay in bed.</td>
</tr>
</tbody>
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<table>
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<tr>
<th>Section 3 - Lifting</th>
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<tbody>
<tr>
<td>I can lift heavy weights without extra pain.</td>
</tr>
<tr>
<td>I can lift heavy weights but it gives extra pain.</td>
</tr>
<tr>
<td>Pain prevents me from lifting heavy weights off the floor, but I can manage if they are conveniently positioned, for example on a table.</td>
</tr>
<tr>
<td>Pain prevents me from lifting heavy weights, but I can manage light to medium weights if they are conveniently positioned.</td>
</tr>
<tr>
<td>I can lift very light weights.</td>
</tr>
<tr>
<td>I cannot lift or carry anything at all.</td>
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<thead>
<tr>
<th>Section 4 - Reading</th>
</tr>
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<tbody>
<tr>
<td>I can read as much as I want to with no pain in my neck.</td>
</tr>
<tr>
<td>I can read as much as I want to with slight pain in my neck.</td>
</tr>
<tr>
<td>I can read as much as I want with moderate pain in my neck.</td>
</tr>
<tr>
<td>I cannot read as much as I want because of moderate pain in my neck.</td>
</tr>
<tr>
<td>I cannot read at all because of severe pain in my neck.</td>
</tr>
<tr>
<td>I cannot read at all.</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Section 5 - Headaches</th>
</tr>
</thead>
<tbody>
<tr>
<td>I have no headaches at all.</td>
</tr>
<tr>
<td>I have slight headaches which come in-frequently.</td>
</tr>
<tr>
<td>I have moderate headaches which come in-frequently.</td>
</tr>
<tr>
<td>I have severe headaches which come frequently.</td>
</tr>
<tr>
<td>I have headaches almost all the time.</td>
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<tr>
<th>Section 6 - Concentration</th>
</tr>
</thead>
<tbody>
<tr>
<td>I can concentrate fully when I want to with no difficulty.</td>
</tr>
<tr>
<td>I can concentrate fully when I want to with slight difficulty.</td>
</tr>
<tr>
<td>I have a fair degree of difficulty in concentrating when I want to.</td>
</tr>
<tr>
<td>I have a lot of difficulty in concentrating when I want to.</td>
</tr>
<tr>
<td>I can concentrate at all.</td>
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</tbody>
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<thead>
<tr>
<th>Section 7 - Work</th>
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<tbody>
<tr>
<td>I can do as much work as I want to.</td>
</tr>
<tr>
<td>I can only do my usual work, but not more.</td>
</tr>
<tr>
<td>I can do most of my usual work, but not more.</td>
</tr>
<tr>
<td>I cannot do my usual work.</td>
</tr>
<tr>
<td>I cannot do any work at all.</td>
</tr>
<tr>
<td>I can do any work at all.</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Section 8 - Driving</th>
</tr>
</thead>
<tbody>
<tr>
<td>I can drive my car without neck pain.</td>
</tr>
<tr>
<td>I can drive my car as long as I want with slight pain in my neck.</td>
</tr>
<tr>
<td>I can drive my car as long as I want with moderate pain in my neck.</td>
</tr>
<tr>
<td>I cannot drive my car as long as I want because of severe pain in my neck.</td>
</tr>
<tr>
<td>I cannot drive my car at all.</td>
</tr>
</tbody>
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<table>
<thead>
<tr>
<th>Section 9 - Sleeping</th>
</tr>
</thead>
<tbody>
<tr>
<td>I have no trouble sleeping.</td>
</tr>
<tr>
<td>My sleep is slightly disturbed (less than 1 hr. sleepless).</td>
</tr>
<tr>
<td>My sleep is moderately disturbed (2-5 hrs. sleepless).</td>
</tr>
<tr>
<td>My sleep is greatly disturbed (6-10 hrs. sleepless).</td>
</tr>
<tr>
<td>My sleep is completely disturbed (11-16 hrs. sleepless).</td>
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</table>

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<thead>
<tr>
<th>Section 10 - Recreation</th>
</tr>
</thead>
<tbody>
<tr>
<td>I am able to engage in all my recreation activities with no neck pain at all.</td>
</tr>
<tr>
<td>I am able to engage in all my recreation activities with some pain in my neck.</td>
</tr>
<tr>
<td>I am able to engage in most, but not all of my usual recreation activities because of pain in my neck.</td>
</tr>
<tr>
<td>I am able to engage in a few of my usual recreation activities because of pain in my neck.</td>
</tr>
<tr>
<td>I cannot do any recreation activities at all.</td>
</tr>
</tbody>
</table>

Figure 1. The Neck Disability Index (modified from [10], with permission).
Thirty-four (70%) of the subjects had sustained a whiplash-type injury within the past 4-6 wk, while 14 (30%) of the subjects presented with more chronic nontraumatic neck complaints. Two of these had suffered a whiplash-type injury within 1.5 yr, while the remainder suffered from mechanical pain syndromes, chiefly related to occupational or lifestyle stresses, with an average duration of 14 wk (range: 2–128 wk).

The first 17 subjects were given the NDI as part of their initial consultation in the clinic. Consent was obtained, instructions on completing the form were given, and it was filled out in the office. The subject’s next visit was appointed 2 days later and no treatment was delivered on the first visit. Upon return to the clinic, the second NDI was filled out immediately. As such, a consistent 2-day no treatment interval was obtained between the completion of the two forms. A Pearson Product Moment correlation coefficient was calculated on the test scores, while age and gender effects were analyzed using regression analysis.

The reliability of each item and for the total index was calculated by using the alpha coefficient of Cronbach (19).

Mean scores of the total index were tabulated on a frequency histogram, the plot of which reveals the distribution of scores from very low to very high. The degree to which this distribution was seen to match that of the clinical population in the study was used as a rough measure of construct validity.

Concurrent validity was assessed in two separate analyses. First, a subset of 10 subjects who completed a course of conservative treatment in the clinic were further studied by having them fill out a subsequent NDI form after 3 wk of treatment. At the same time, subjects completed a visual analogue scale which rated the improvement (i.e., the percent change) in activity levels since the start of treatment (20). The percentage of improvement from the VAS was compared to the percentage change in the pre- and posttreatment NDI scores. Treatment consisted of conservative (chiropractic) therapeutics (14–18). A Pearson Product Moment correlation coefficient was obtained on the change scores, and was taken to indicate the degree of sensitivity of the NDI to treatment effects.

Second, a subset of 30 subjects completed a McGill Pain Questionnaire (21). This instrument has been reported extensively in the assessment of low back pain (22), and was viewed, in the context of the present study, as a “gold standard” with regard to severity of the pain experienced by the subjects. Again, a Pearson Product Moment correlation was conducted on the NDI and MPQ scores. In all analyses, alpha was set at 0.05.

RESULTS

Test-retest Reliability
The correlation coefficient was calculated as 0.89 ($p \leq 0.05$). The effect of age and gender was found not to be statistically significant.

Internal Consistency
The mean score, ranks and alpha values for each item are shown in Table 2. The items with the highest scores included: headaches (2.6), lifting (2.2), recreation (2.2), reading (2.1) and driving (2.0). All items achieved an alpha coefficient above 0.76, while the total index alpha coefficient was calculated as 0.80.

Construct Validity
The frequency histogram of the range of scores is shown in Figure 2. Simple units of 5 or 10 were used to calculate the intervals, which when matched to the data revealed a nearly normal distribution with the largest category being in the mid-range. Eighty-three
percent of the scores were in the mild to moderate categories.

Concurrent Validity

The correlation between changes in NDI scores pre- and posttreatment and those of an improvement VAS for activity levels was 0.60, indicating a moderately high degree of positive correlation. The average change in NDI scores was 33.2%, while for the VAS scores it was 56% (Table 3). The correlation coefficients for NDI/MPQ-total score and NDI/MPQ-number of words were 0.70 and 0.69, respectively.


<table>
<thead>
<tr>
<th>Year</th>
<th>Injuries</th>
<th>Deaths</th>
</tr>
</thead>
<tbody>
<tr>
<td>1980</td>
<td>239,640</td>
<td>5,409</td>
</tr>
<tr>
<td>1983</td>
<td>224,304</td>
<td>4,216</td>
</tr>
<tr>
<td>1986</td>
<td>264,481</td>
<td>4,071</td>
</tr>
</tbody>
</table>

*Source: Insurance Bureau of Canada.

DISCUSSION

Reliability and Item Analysis

This study is largely a modification and replication of the original study design of Fairbank et al., which promulgated the Oswestry Index (6). Remarkably, very little systematic evaluation of that Index has been reported. In one evaluation, Waddell and Main (23, 24) modified the original scales, choosing to use singular cut-off points in each item, thereby creating a simpler dichotomous rating of “less than” or “more than” a criterion level of each activity. Other scales for low back pain have been created, although very little comparative validation has been conducted specifically including the Oswestry Index.

To our knowledge, no revision of the Oswestry Index has been reported for assessment of disabilities related to problems in other regions of the spine. Considering the magnitude of disabling neck pain, such a modification seemed warranted. The Oswestry format was used as it seemed to best serve the identification of separate and distinct components of ADL, while also allowing for a global disability score. Our revision consisted of substituting the four least relevant items from the original low back pain scale (walking, sitting, standing and sex life), with four items of greater relevance for patients suffering from disabling neck pain (reading, headaches, concentration and personal care). Our revision item, indicate a significant disturbance of an important social context, although for many, this may be focused on one particular recreational activity.

Interestingly, scores for the headache item were higher in our subjects than were the pain intensity ratings, illustrating the significance of this symptom in chronic whiplash and nontraumatic neck pain syndrome. This is consistent with the findings of Balla and Kernaghan (27) on the prevalence and severity of headaches in whiplash-injured patients.

Aside from pain intensity, the four lowest scoring items (work, sleeping, concentration and personal care) present an interpretive challenge. Tait et al. (11) recently proposed a two-factor interpretation, the original model being attributed to Fordyce et al. (28) of the ADL which comprise the Pain Disability Index. The two categories were voluntary as opposed to obligatory activities. Most of the lower scoring items on the NDI fall into the “obligatory activity” category. In our subjects, with moderate but not severe levels of complaint (see below), it appears that obligatory activities are not greatly disturbed and may therefore contribute much less to the overall disability experienced. It remains to be seen if this is also true of subjects who suffer with more severe complaints.

Finally, age and gender appeared to have no effect on NDI scores, thus rendering the index suitable for the wide base of subjects such as is typically seen with disabling neck pain conditions. Overall, the high degree of reliability and internal consistency found in the original Oswestry Index has been replicated in the NDI.

Validity

This study was conducted in an outpatient chiropractic clinic. Subjects were necessarily ambulatory and...
self-selected with regard to the severity of their injuries, many of them having undergone lengthy courses of previous care (29, 30). As such, the condition of these subjects were not as severe as, for example, subjects seen in a hospital emergency ward. This is seen in the distribution of the total NDI scores (Figure 2), where the largest single group (48%) was in the moderately severe category, while the vast majority of scores (83%) ranked from mild to moderate. This finding also supports the scaling intervals which were used for interpreting the scores, these being slightly different from those in the Oswestry Index in that the scores of the group rated as “no disability” ranged only from 0–4. We interpret these findings to indicate that the NDI is sensitive to the overall level of complaint severity found in this subject pool.

The concurrent validity is further confirmed by the moderately high positive correlation between NDI and MPQ scores. This is especially important, given that the pain intensity scores, as rated on the NDI, were only in the low to moderate range. The overall disability score, however, appears to be approximately as sensitive as the gold standard in discriminating levels of complaint severity.

The NDI also appears to be sensitive to changes that occur in time, particularly as a result of a therapeutic trial. The correlation with a simpler self-report measure of clinical improvement (the progress VAS) was positive and moderately high \( (r = 0.60) \). The NDI scores were typically lower than those of the VAS (by approximately 40%). We interpret this to indicate that the NDI may be a more accurate indicator of the improvement in activity levels, since VAS scores, unreferenced to pre-treatment baselines, might have been overreported. However, since comparison of different pain rating scales involves significant psychometric complexities (31), further interpretation of the changes in the NDI scores as compared to those of the VAS would be speculative.

CONCLUSION

The Neck Disability Index, a revised form of the Oswestry Low Back Pain Index, has been offered as a self-reporting instrument for the assessment of ADL of sufferers of disabling neck pain, particularly from whiplash-type injuries. Studies of patients with these sorts of conditions would be greatly assisted by a condition-specific disability rating instrument. This has certainly been the case with research in low back pain since the advent of the Oswestry Index and other similar instruments.

The NDI has been shown to demonstrate a high degree of test-retest reliability and internal consistency; to be applicable to a wide age range, unaffected by gender; and to have an acceptable level of validity, being sensitive to severity levels and to changes in severity over time.

Larger group studies, especially on more severe levels of complaint, should be conducted to fortify the psychometric properties and the overall relevance of the NDI. If the findings of this initial study are replicated, then this instrument should find wide application in research on neck-disabled patients, in medicolegal circles and in the general practice assessment of patients with disabling neck pain.

REFERENCES

Cervicogenic Dysfunction in Muscle Contraction Headache and Migraine: A Descriptive Study

HOWARD VERNON, D.C.,* IGOR STEIMAN, D.C.+ AND CAROL HAGINO†

ABSTRACT

Objective: The prevalence and nature of findings of cervicogenic dysfunction is explored in subjects with muscle contraction/tension-type (MCH) headache and common migraine without aura (CM).

Design: Descriptive survey.

Setting: Chiropractic outpatient research clinic.

Patients: Forty-seven (47) subjects, aged 18-55 with two categories of benign headache, were studied: MCH (tension-type), n = 19 (6 males, 13 females) and CM (without aura), n = 28 (3 males, 25 females). Subjects were recruited as part of an intervention trial and, thus, form a consecutive sample of patients. The present findings were elicited as part of the initial assessment.

Intervention: No therapeutic intervention is reported.

Main Outcome Measures: Standardized headache history; plain film and dynamic spinal X rays; motion palpation; and pressure algometry.

Results: For CM, the most prevalent headache locations were frontal (81%) and occipital (78%). Neck pain and upper back pain accompanied headache in 90% and 41% of subjects, respectively. For MCH, the most prevalent headache locations were occipital (87%) and frontal (81%). Neck and upper back pain accompanied headache in 100% and 27%, respectively, of all subjects. For the total group, 77% of all subjects and 89% of females exhibited a marked reduction, absence or reversal of the normal cervical lordosis. Ninety-seven percent of all subjects exhibited, on dynamic X-ray studies, at least one significant abnormality of segmental mobility from C1 to C7, while 43% exhibited abnormalities at four or more segments. Segmental motion at C0-C1 was reduced in 90% of subjects in flexion and 70% of subjects in extension. On motion palpation, 84% of CM and MCH subjects were found to have at least two major fixations from C0 to C2. On pressure algometry, 92% of CM and 85% of MCH had at least one verifiable tender point (TP) in the upper cervical region. The most common locations for TPs were mid-cervical (C2-C3), lateral occipital and suboccipital.

Conclusions: Both MCH and CM subjects demonstrate high occurrences of: a) occipital and neck pain during headaches; b) tender points in the upper cervical region; c) greatly reduced or absent cervical curve; and d) X-ray evidence of joint dysfunction in the upper and lower cervical spine. These findings support the premise that the neck plays an important, but largely ignored role in the manifestation of adult benign headaches. A case-control study should be conducted to confirm the greater prevalence of cervicogenic dysfunction in headache as compared to nonheadache subjects. (J Manipulative Physiol Ther 1992; 15:418-429).

Key Indexing Terms: Muscle Contraction, Headache, Migraine, Cervical Vertebrae.

INTRODUCTION

The involvement of the cervical spine in the etiology of headache has a rich tradition of support in medical and heterodox writings over the past century (1, 2).

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However, even as a modern collection of work from authors such as Edmeads, Bogduk, Sjaastad and his colleagues and others (see below) is emerging, the role of the neck receives scant attention in the orthodox paradigm of cephalalgia.

It is even more the case that the conservative, heterodox treatment of headache directed at treatment of dysfunction of the cervical spine is virtually an ignored approach (3). To deal with this, a controlled clinical trial comparing spinal manipulation with a placebo was undertaken. This present study reports on characteristics of dysfunction of the cervical spine in the study group, which, most importantly, was comprised of sub-
jects diagnosed with muscle contraction (MCH) or common migraine (CM) headaches. This is noteworthy since all of the recent reports describing what has come to be called "cervicogenic headache" have been on subjects who have been designated as deviant, and therefore distinct from, the classical categories of MCH and migraine headaches. As such, our report extends the identification of signs and symptoms of neck dysfunction from the smaller subgroup to the much larger generic categories (4).

The reader is referred to extensive reviews of the cervicogenic headache model by the first author (1, 2).

Barre (5) and Lieou (6) were among the first to implicate the cervical spine in the genesis of a wide array of neurological symptoms, foremost of which were headache and vertigo. In their writings, irritation of the cervical sympathetic system was linked to circulatory disturbances, primarily as a disturbance of vasomotor tone, in the distribution of the vertebral artery or of the internal auditory artery.

In 1948, Bartschi-Rochaix (7) coined the term "migraine cervicale" to describe a type of headache, potentially very severe in character, which was caused by disturbances of the cervical spine. The impact of these mechanical disturbances was thought to be mediated through the autonomic nervous system. Following Bartschi-Rochaix through the next three decades, numerous reports are found in the medical, chiropractic, osteopathic and physiotherapy literature (8–24). For the sake of brevity, the reader is referred to the extensive reviews of these works by Vernon (1, 2).

Regarding the more current literature, Bogduk, in "Cervical Manipulation and Headache" (25), initially debunked the concept of a "cervical migraine," citing the obvious biochemical and autonomic mechanisms for which, at that time, he failed to find any justified correlation with the cervical spine. He noted that mechanical irritation of the vertebral artery, vertebral nerve and ascending sympathetic chain can initiate an "autonomic barrage" sufficient to create cerebral vasospasm. As well, he accepted Kovac's contention (12) that subluxation of the cervical apophyseal joints might compromise these structures. However, he reserved his most enthusiastic support (26-28) for the role of the apophyseal joints in nonmigrainous headache. He asserted a direct, causative role for mechanical derangements of the cervical apophyseal and, most importantly, cranio cervical, synovial joints in the production of headache. This mechanical derangement is characterized as a "chronic hypomobility," hereafter referred to as a fixation. This mechanism allows for an explanation of the role of the cervical spine in headache in a much younger population, contrary to the limitations of the "degenerative disease" model proposed by Edmeads (21).

Sachse et al. (29) found the most frequent segmental fixations in 22 migraineurs to be C0-C1 and C7-T1, as well as in the first costovertebral joint (first rib fixation). They found the most severe tenderness to palpation present in the shoulder (scapular) region and over the posterior arch of atlas. The most severe muscle hypertonicity was found in the trapezius and levator scapulae muscles.

The contribution of Sjaastad and his colleagues consists of a series of studies from 1983 to 1988 on what he has termed "cervicogenic headache" (4, 30–33). This is described as a variant of the chronic paroxysmal headache, which is almost always unilateral, accompanied by autonomic symptoms and provokable by movements of the head and neck, primarily forward flexion. Sjaastad has proposed that this headache may be due to entrapment of the occipital nerve or a C2-C3 rhizopathy, and that this diagnosis may be greatly under-reported (30).

Graff-Radford et al. (34) reported on 25 subjects with resistant headache syndromes who were treated by a multimodal approach to altering "perpetuators" of myofascial pain. Central to their evaluation of the subjects was the investigation of active and latent "trigger points" (TP) that reproduced the headache, noting posture of the head and neck, and the detection of "underlying cervical joint dysfunction," especially in the upper cervical spine. Their multimodal treatment program emphasized TP therapies and joint mobilizations among a variety of interventions and was shown to be quite successful in reducing headache pain and medication usage.

Jaeger (35) subsequently reported on a descriptive study of 11 cervicogenic headache patients. These subjects were found to have a high prevalence of tender (trigger) points, postural abnormalities, misalignment and tenderness of the C1-C2 segment and reduced ranges of neck movement. Most recently, Pfaffenrath et al. (36) reported on the "clinical picture" and X-ray findings in cervicogenic headache patients. Fifteen subjects were selected with clinical features similar to those of Sjaastad et al. (30). Head/neck movements provoked headaches in all subjects. Most subjects had tender points around C2 which reproduced headache. C2 blockades resulted in significant improvement in headache lasting from 2 hr to 42 days. In their original work, X-ray studies did not reveal a significant pattern of abnormality in the cervical spine as compared to a control group. However, a follow-up study (37) using a computer-based technique of analysis of cervical motion X rays did reveal a statistically significant increase.
of upper cervical hypomobility and a reduction of general mobility from C0 to C5 in headache subjects as compared to controls. The most prevalent hypomobility was at C0-C1.

The nature of the disorder(s) in the cervical spine which may typically give rise to headache syndromes is controversial, although two major categories have been identified by Edmeads (3). These are a) structural or organic disorders or diseases; and 2) disorders of function of the cervical spine, which consist primarily of mechanical abnormalities of the articular and myofascial tissues. Since almost all of the studies on cervical or cervicogenic headache presume to deal with the second of these categories, the present study confines itself to these issues as well.

Several components of cervical spine dysfunction can be identified. These are a) hypomobility of the cervical motion segments (fixation, blockage, etc.); b) hypermobility of the cervical motion segments; c) tender points in various cervical and suboccipital muscles; d) spasm or sustained contraction of these muscles; e) reduced and/or painful movements of the neck (especially as they provoke headache); f) X-ray-detectable changes in the lordotic curve of the cervical spine; g) X-ray-detectable changes in the alignment of the neck in the coronal plane; and h) X-ray-detectable alterations in the motion of the cervical segments. Table 1 summarizes these components of dysfunction and gives the findings of various authors as to their nature and validity.

Sjaastad et al. (30) have remarked on the potential overlap of diagnostic categories between cervicogenic, CM and MCH. In regard to the presence of the above-mentioned elements of cervical dysfunction, it appears that they are used to more or less confirm the cervicogenic designation. In other words, in cases where the symptoms overlap and diagnostic confusion exists, the presence of cervical dysfunction determines the cervicogenic diagnosis. When signs of cervical dysfunction are absent (or, perhaps, ignored), then the other diagnoses are selected.

What has not been determined is the extent to which signs of cervical dysfunction are present in headache sufferers already designated as CM or MCH. If the extent is large, then the proposition that diagnostic overlap exists is supported. If the extent is small, then the cervicogenic designation and category ought to be accepted as distinct, exceptional and probably low in prevalence as compared to CM and MCH.

This present study was undertaken to determine the extent of four of the major elements of dysfunction of the cervical spine in subjects suffering from MCH and CM headaches: hypomobility, tender points in muscles, X-ray-detectable changes in the lateral curve of the cervical spine and X-ray-detectable changes in movement of the cervical spine.

**METHODS**

Forty-seven subjects, 38 females (81%) and 9 males (19%), were included in the study. Their average ages are shown in Table 2 and ranged from 17 to 48 yr. These subjects volunteered for a treatment study comparing chiropractic spinal manipulation to a placebo. They consented to a comprehensive diagnostic protocol which included an extensive, standardized case history, physical examination of the cranium and cervical spine and X-ray studies of the neck (see below). As a result of the history and examination, and taking into account the previous classification by medical examiners, the subjects were categorized as MCH or CM according to the Ad Hoc committee standards (38). Twenty-eight (28), or 60%, of the subjects were classified predominantly as CM; 19, or 40%, were classified predominantly as MCH.

The examination protocol for cervical dysfunction included the following procedures.

**Hypomobility of Cervical Motion Segments**

The manual palpatory procedure devised to determine segmental motion is termed “motion palpation.”
This procedure owes its original popularization to Gillet and Liekens (19), who devised procedures specific for each spinal region and for detecting motion in the various anatomical planes. Grice modified and improved on the operational description of cervical motion palpation procedures (39). DeBoer et al. (40) found a suitable degree of reliability in upper cervical motion palpation procedures, while Mior and King (41) were unable to replicate this level of reliability. Fligg (42) has recently reviewed the palpatory procedures for upper cervical motion analysis.

In our study, three tests of motion in the upper cervical spine were used: forward flexion, lateral bending and anterior rotation in the upper three cervical segments (see Figures 1-3). The specialist (IS) who conducted this examination was blinded to the headache category of each subject. Only significant findings of hypomobility were recorded. A positive finding in any of the three tests was taken to indicate fixation of that motion segment. The percentage of subjects in each group with fixations in one, two or three segments were calculated for analysis.

Tender Points in the Muscles of the Cervical Spine

Pressure algometry has been validated for use in cervical spine (43-46) and headache (43, 44) conditions. We employed the Fischer algometer (43) in measuring pressure pain threshold in the following standardized anatomical locations bilaterally: medial occipital brim, lateral occipital brim, suboccipital, midcervical, trapezius and levator scapulae. The technique employed was similar to that described by Fischer (43) and Reeves et al. (46). The examiner was blinded to the headache category. Positive findings (i.e., tender points) were identified by pressure readings lower than 3 kg/cm², or where bilateral asymmetry of greater than 1.5 kg/cm² was noted. Frequency counts were tabulated for each muscle bilaterally.

Cervical Lordosis

Measurements were made on the neutral lateral cervical X ray of the lordotic angle from the bisector of the C1 vertebra, and a line drawn parallel to the inferior endplate of the C7 vertebral body (see Figure 4). Five categories of configuration were created from these measurements and from other visual assessments made by an independent X-ray reviewer: a) normal lordosis (>25°); b) hypolordosis (<25°); c) alordosis (+5 to −5°); d) quasi-alordotic (combinations of b and c, with alordosis usually extending from C3-C6); and e) kyphotic (>5°). Frequency counts were tabulated for the various categories. The assessor was blinded as to the clinical
Segmental Motion Analysis by Radiography

The analysis of segmental motoricity by analysis of flexion-neutral-extension views of the cervical spine has been discussed by many, including Grice (47), Arlen (48), Buetti-Bauml (49) and Henderson and Dormon (50). Dvorak et al. (51) have recently validated the Arlen measurement system and have derived normal ranges of segmental motion in flexion and extension. Pfaffenrath et al. (37) used a similar approach, aug-
prominent are "weather" and "tension," which occupy the first two ranks in each headache group. Notable is the prevalence of "head and neck movements," which were cited as a precipitating factor by 64% of CM subjects (4th rank) and 47% of MCH subjects (3rd rank). The "food" and "menstruation" items were two items more common in the CM subjects. The Spearman rank coefficient was statistically significant ($r = .91, p < .05$).

Table 6 lists the three most prevalent factors which produced aggravation of headache during an episode. Notably, the prevalence of photo- and phonophobia as well as coughing/sneezing are quite similar for both groups.

Table 7 lists 11 associated symptoms, ranging from the most typical ones ("nausea," "visual disturbances" and "dizziness") to the less common ("sensory changes" and "numbness"). The Spearman rank coefficient was statistically significant ($r = .70, p < .001$), indicating that the general ranking of the symptoms was similar in both groups. The Student's $t$ test for dependent means demonstrated a statistically significant difference between the lists of percentage figures, and those of the CM group were significantly higher than those of the MCH group ($t = 15.3, p < .001$). Four symptoms were found exclusively in the migraine group: "speech disturbances" (31%), "sensitivity to smell" (27%), "mood changes" (24%) and "sensory changes" (16%), indicating that 25-30% of migraineurs experience a group of symptoms quite distinct from MCH sufferers.

**Examination Findings**

Table 8 depicts the percentage of subjects with hypomobility (fixation) of the three segments of the upper cervical spine. Eighty-four percent of both migraine and MCH subjects had a hypomobility of at least two out of three segments.

Table 9 depicts the algometry findings. Eighty-nine percent of all subjects had at least one TP (92% of CM and 85% of MCM groups). Half of all subjects had two or three TPs, with the most prevalent category being three TPs (27% of all subjects). Twenty-seven percent (27%) had four or more TPs. Spearman's $r$ was .98, indicating highly similar ranks for both headache groups.

Table 10 shows the distribution of TPs by their location in the subjects with at least one TP. The vast majority (85%) had a TP in the midcervical location, while the second and third most prevalent TPs were the occipital (62.5%) and suboccipital (60%). The rankings for both headache groups were identical, even though there were slightly fewer TPs in the MCH group as compared to the CM group.

Table 11 indicates the distribution of the categories of sagittal curve alignment in the cervical spine on the lateral X ray. Seventy-seven percent (77%) of all subjects had a substantial alteration of the sagittal cervical curve, consisting of near-total or total reduction, or, in fact, reversal of the normal lordotic configuration. A minor reduction in the sagittal lordosis was recorded.
TABLE 8. Prevalence of fixations on manual motion palpation

<table>
<thead>
<tr>
<th>Number of levels</th>
<th>Migraine</th>
<th>MCH</th>
</tr>
</thead>
<tbody>
<tr>
<td>0</td>
<td>0</td>
<td>0</td>
</tr>
<tr>
<td>1</td>
<td>16</td>
<td>16</td>
</tr>
<tr>
<td>2</td>
<td>54</td>
<td>42</td>
</tr>
<tr>
<td>3</td>
<td>30</td>
<td>42</td>
</tr>
<tr>
<td>2 or 3</td>
<td>84</td>
<td>84</td>
</tr>
</tbody>
</table>

TABLE 9. Tender points: Distribution by the number of points

<table>
<thead>
<tr>
<th>No. of TPs</th>
<th>CM</th>
<th>MCH</th>
<th>Total</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>%</td>
<td></td>
<td></td>
</tr>
<tr>
<td>6</td>
<td>8</td>
<td>10</td>
<td>9</td>
</tr>
<tr>
<td>5</td>
<td>16</td>
<td>5</td>
<td>11</td>
</tr>
<tr>
<td>4</td>
<td>4</td>
<td>10</td>
<td>7</td>
</tr>
<tr>
<td>3</td>
<td>28</td>
<td>25</td>
<td>27</td>
</tr>
<tr>
<td>2</td>
<td>20</td>
<td>25</td>
<td>22</td>
</tr>
<tr>
<td>1</td>
<td>16</td>
<td>10</td>
<td>13</td>
</tr>
<tr>
<td>0</td>
<td>8</td>
<td>15</td>
<td>11</td>
</tr>
</tbody>
</table>

Spearman $r = .98, p \leq .05$.

TABLE 10. Tender points: Distribution by the location of points

<table>
<thead>
<tr>
<th>Location of TP</th>
<th>Headache groups</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>CM</td>
</tr>
<tr>
<td></td>
<td>%</td>
</tr>
<tr>
<td>Midcervical</td>
<td>91</td>
</tr>
<tr>
<td>Lateral-occipital</td>
<td>68</td>
</tr>
<tr>
<td>Suboccipital</td>
<td>64</td>
</tr>
<tr>
<td>Trapezius</td>
<td>55</td>
</tr>
<tr>
<td>Levator scapula</td>
<td>32</td>
</tr>
<tr>
<td>Medial-occipital</td>
<td>23</td>
</tr>
</tbody>
</table>

Spearman $r = 1.00, p \leq .05$.

for 11.5%. This distribution was statistically significant ($\chi^2 = 11.79, 1 df, p \leq .001$).

Abnormal segmental motion for combined flexion/extension in the sagittal plane is depicted in Table 12.

In this analysis, computer-generated measurements for flexion and for extension in the segments from C1 to C6 were combined and evaluated against the standard of segmental motion provided by Dvorak et al. (51) (see Figure 5). Values 1 SD above or below the mean for combined flexion/extension are rated as hypermobile or hypomobile, respectively. The total count of abnormalities per segment is expressed as a percentage of the total group of subjects for whom the motion analysis was possible ($n = 37$). One hundred percent of subjects had at least one abnormality between C1 and C6, and 80% of subjects had an abnormality (hypomobility) at C0-C1. Ninety-seven percent of subjects had at least two abnormal segments from C1 to C6. Almost three-fourths had three abnormal segments.

Table 13 shows that 65% of the subjects had at least one abnormality at C1 or C2, while Table 14 shows that an abnormality at C0-C1 existed in 90% of subjects in flexion and 70% in extension.

The character of segmental motion abnormality for C1-C6 in the study group is depicted in Figure 5. It can be seen from this figure and from Tables 12-14 that a pattern of abnormal segmental mobility in the sagittal plane exists consisting of reduction of movement in the lower cervical spine, relatively normal movement in the region from C2-C4, hypermobility of C1-C2 and hypomobility of C0-C1. The most predominant segmental restriction in flexion/extension in the whole cervical spine is at C0-C1.

Finally, Table 15 lists the findings of a set of clinical tests which, taken together, address the potential for vertebrobasilar insufficiency. Of particular interest are the "Wallenberg" tests, which have been proposed to screen for potential contraindication to manipulative treatment (54). It can be seen that no subject from either CM or MCH groups demonstrated a positive response to this test protocol.

TABLE 11. Distribution of categories of sagittal curve of the cervical spine on plain film X-ray

<table>
<thead>
<tr>
<th>Total no. of subjects</th>
<th>Normal</th>
<th>Hyperlordotic</th>
<th>Quasi-alordotic</th>
<th>Alordotic</th>
<th>Reversal</th>
</tr>
</thead>
<tbody>
<tr>
<td>Percent of total no. of subjects</td>
<td>11.5%</td>
<td>11.5%</td>
<td>22.5%</td>
<td>43%</td>
<td>11.5%</td>
</tr>
<tr>
<td>No. of males</td>
<td>5</td>
<td>5</td>
<td>10</td>
<td>19</td>
<td>5</td>
</tr>
<tr>
<td>Percent male</td>
<td>100%</td>
<td>100%</td>
<td>100%</td>
<td>100%</td>
<td>100%</td>
</tr>
<tr>
<td>No. of females</td>
<td>0</td>
<td>0</td>
<td>0</td>
<td>0</td>
<td>0</td>
</tr>
<tr>
<td>Percent female</td>
<td>0%</td>
<td>0%</td>
<td>0%</td>
<td>0%</td>
<td>0%</td>
</tr>
<tr>
<td>$\chi^2$ with continuity correction</td>
<td>11.79*</td>
<td>$p \leq .001$ df = 1</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Fisher exact test (two-tailed probability)</td>
<td>.001</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Phi</td>
<td>.588</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

* To be used with caution, since two quadrants have values less than 5.
TABLE 12. Computer-aided analysis of segmental motion on flexion/extension X rays: C1–C6

<table>
<thead>
<tr>
<th>No. of abnormal segments</th>
<th>Percent of subjects</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>100</td>
</tr>
<tr>
<td>2</td>
<td>97</td>
</tr>
<tr>
<td>3</td>
<td>73</td>
</tr>
<tr>
<td>4</td>
<td>43</td>
</tr>
<tr>
<td>5</td>
<td>16</td>
</tr>
</tbody>
</table>

73% have three or more abnormal segments; 59% had four or more.

TABLE 13. Distribution of subjects with none, one or two abnormal segments at C1 and C2: Computer-aided X-ray analysis

<table>
<thead>
<tr>
<th>No. of abnormalities at C1 and C2</th>
<th>Percent of subjects</th>
</tr>
</thead>
<tbody>
<tr>
<td>0</td>
<td>35</td>
</tr>
<tr>
<td>1</td>
<td>65</td>
</tr>
<tr>
<td>2</td>
<td>16</td>
</tr>
</tbody>
</table>

TABLE 14. Abnormal motion of C0–C1: Overlay method of X-ray analysis

1. C0–C1 Flexion (normal 10°/cutoff = 6°)
   Mean: 3.17°
   SD: 2.24°
   Percent hypomobile (under 6°) = 90%

2. C0–C1 Extension (normal 25°/cutoff = 18°)
   Mean: 10.60°
   SD: 7.70°
   Percent hypomobile (under 18°) = 70%

TABLE 15. Signs related to vertebrobasilar insufficiency

<table>
<thead>
<tr>
<th></th>
<th>Migraine</th>
<th>MCH</th>
</tr>
</thead>
<tbody>
<tr>
<td>Cranial bruits</td>
<td>0</td>
<td>0</td>
</tr>
<tr>
<td>Carotid</td>
<td>0</td>
<td>0</td>
</tr>
<tr>
<td>Vertebral</td>
<td>0</td>
<td>0</td>
</tr>
<tr>
<td>Wallenberg tests (bilateral)</td>
<td>0</td>
<td>0</td>
</tr>
<tr>
<td>Fundoscopy</td>
<td>7% sl. positive</td>
<td>7% sl. positive</td>
</tr>
<tr>
<td>Blood pressure</td>
<td>120/76</td>
<td>106/70</td>
</tr>
<tr>
<td>History of high blood pressure</td>
<td>3%</td>
<td>0%</td>
</tr>
</tbody>
</table>

DISCUSSION

Taken together, our findings describe a pattern of cervical spine dysfunction in MCH and CM headache sufferers which, we believe, has not previously been reported. There is a significant loss of the normal lordosis of the cervical spine in the majority of subjects in both headache groups; there is also a predominant pattern of palpable hypomobility in the upper cervical spine that is confirmed on computer-aided dynamic X-ray analysis in flexion/extension, which appears to most frequently involve C0–C1, and there is also a tendency toward hypomobility in the lower cervical spine. Tender points are highly prevalent with an overwhelming predominance of TP's in both headache groups in the mid- to upper cervical area, with most subjects having two to three TP's in this region.

As far as the association of neck pain with the headaches, subjects in both groups demonstrated a high incidence of both suboccipital and neck pain during headaches, with an additional upper back (cervicothoracic) pain accompanying the headache in 41% of CM and 27% of MCH sufferers. As well, head and neck movements precipitated headaches in 50–60% of subjects in both categories.

These findings are in good agreement with many of those reported for "cervicogenic headaches." Jaeger (35) noted a high prevalence of hypomobility in the upper cervical spine of 11 cervicogenic headache sufferers. Pfaffenrath et al. (38), using computer-aided dynamic X-ray analysis, found the most prevalent hypomobility in flexion/extension to be at C0-C1. Sjaastad et al. (4, 30) require head and neck movements to precipitate head pain in cervicogenic headache sufferers, which we found in 50–60% of CM and MCH sufferers in our sample.

The most universally accepted finding in the cervical spine in cervicogenic headache sufferers is suboccipital tenderness upon palpation (4, 30, 31, 36, 57–59). Our findings in CM and MCH sufferers are in complete agreement with this literature, especially with Jaeger (35), who found a high prevalence of tenderness over the lateral C1 transverse process. That may correspond with our finding of tenderness over the lateral occipital TP in 63% of our subjects. Regardless of the segmental location (i.e., from C0-C4), the overwhelming majority
of our CM and MCH sufferers had at least one to three TPs in the upper cervical area.

These findings of tenderness in muscles in the upper cervical spine of MCH and CM sufferers have been previously reported. Pozniak-Patewicz (60) reported on 183 headache subjects and 51 controls. Electromyogram (EMG) recordings were made of neck and temporal muscles during headaches and in headache-free intervals. Increases in EMG activity, which reflected sustained spasm, were found almost ubiquitously during headaches and quite frequently between headaches. In fact, neck muscles showed significantly higher EMG amplitudes than temporal muscles during headaches in three of four headache categories (85% of the subjects).

Hudzinski (61) found that a significant proportion of MCH sufferers (70%) reported pain and tension in the muscles of the neck as the source of their headaches. Biofeedback training directed at these tissue sites was far more effective in this group than frontalis muscle relaxation. Ahles, Sturgis and their colleagues (62, 63) have found that EMG activity of trapezius and frontalis muscles increases during dynamic activities, including walking upright and tilting the head. These positional variations were proposed as important for both diagnosis and treatment, since the standard protocols involve the supine resting position. In other words, physical activation of the neck musculature by movement and positional changes (i.e., more real life conditions) may contribute to headache genesis and may be important factors to target for treatment interventions.

Drummond (64), using a pressure-threshold algometer, found increased tenderness levels in scalp and neck muscles of both migraine and tension headache sufferers that were (statistically) significantly higher than in nonheadache controls, but which were not significantly different between the headache groups. In fact, neck and/or occipital tenderness was greater than forehead and temple sites in four out of five headache categories.

Drummond speculated that neck pain might excite trigemino-vascular reflexes by way of the convergence of C1 and C2 afferents with the trigeminal nucleus subcaudalis (64, 65). This mechanism has been cited ever since Sklern's report (11) to explain the role of the neck in headache, and more recently, in the "cervicogenic headache" category.

Our findings reveal a pattern of cervicogenic myofascial and joint dysfunction in muscle contraction and common migraine headache sufferers that is similar to that described in the more narrowly defined category of "cervicogenic" headache, and which is found with equal prevalence in CM and MCH groups. This calls into question the narrowly defined attribution of neck involvement in only the "cervicogenic" category, leaving the others supposedly "neck-free."

Our findings also support the severity model (67), which links MCH and CM on the same etiological spectrum. If this is so, then perhaps on the basis of this profile of cervicogenic dysfunction, it could be proposed that the common ground of these two headache categories is a disorder in the cervical spine. The recent therapeutic success reported by Graff-Radford et al. (34) in the multimodal treatment of cervical pain generated in chronic headache cases, the use of cervical facet joint interjections by Hildebrandt and Argyrakis (68) and Bogduk (69) and the reported success with spinal manipulation (1, 2, 70) would seem to bear out this claim.

Nonetheless, until a carefully controlled study of signs of cervicogenic dysfunction in nonheadache subjects is conducted, the findings presented here should be extrapolated with some caution.

CONCLUSION

Two groups of headache sufferers, one diagnosed with MCH and the other with CM, have been investigated to determine the prevalence of signs and symptoms of neck joint and myofascial dysfunction. A high prevalence was found, with similar levels in each group, of neck pain during headache, of cervical joint dysfunction and postural malalignment and of myofascial tenderness. These findings urge us to reconsider the narrow prescriptive definition of "cervicogenic headaches" and to consider that the cervical spine may play a much larger role than has previously been recognized in the etiology of these headache types, giving new meaning to the phrase "these headaches are a pain in the neck."

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64. Drummond FD. Scalp tenderness and sensitivity to pain in migraine and tension headache. Headache 1987; 27:45-50.
Original Articles

Evaluation of Neck Muscle Strength with a Modified Sphygmomanometer Dynamometer: Reliability and Validity

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ABSTRACT

Objective: Determine test-retest reliability, normative data and clinical validity of isometric muscle strength testing in the neck with a modified sphygmomanometer dynamometer (MSD).

Design: Analytic survey. Paired trials of various muscle strength tests were conducted on convenience samples of normal subjects and consecutive samples of symptomatic subjects.

Setting: Outpatient chiropractic research clinic.

Patients/Subjects: For study 2, 40 normal male subjects, average age 25 ± 2 yr, were studied for reliability and normative data. For study 3, 24 symptomatic patients, 12 males and 12 females, average age 39 ± 7 yr, were studied, 8 with “whiplash”-type injuries (average duration 22.5 wk) and 16 with nontraumatic chronic neck pain (average duration 110 wk).

Intervention: No therapeutic intervention is reported.

Main outcome measure: Pressure levels generated by subjects against a modified sphygmomanometer-type dynamometer as measured in kilopascals.

Results: Study 1. Repeated paired trials of a standardized weight column (20 lbs) produced a coefficient of variation of 0.84% and virtually no difference between the means of the first vs. second trials.

Study 2. High test-retest correlation coefficients were found for all ranges of motion (.79-.97). Right-to-left asymmetry in rotation and lateral flexion was within 6-8%. The flexion/extension ratio was .57:1, indicating that in normal subjects, flexion was approximately 40% lower than extension. Lower cutoffs were established as the mean – 1 SD as follows (in kPa): flexion – 3300, extension – 5800, rotation – 3200 and lateral flexion – 6200. Coefficients of variation ranged from 25 to 29%.

Study 3. Differences between paired trials were analyzed by intraclass coefficients, which were very high (.95-.99), and by percentages, which ranged from 4 to 10.45%, with an average of 7%, indicating a high degree of test-retest consistency. The mean values for all symptomatic subjects for flexion, extension, right rotation and right lateral bending were all well below the normal cutoff values as found in study 2. The flexion/extension ratio for whiplash subjects was 0.25:1.00, which is half of that of normal subjects.

Conclusions: The MSD has been found to be a reliable instrument for the evaluation of isometric muscle strength in the neck in normal and symptomatic subjects. Normative values for absolute test levels, bilateral symmetry and flexion/extension ratios have been determined. A symptomatic group demonstrated significant deviations from these norms in the form of reduced strength levels and reduced flexion/extension ratios, while still maintaining very high levels of test-retest consistency and bilateral symmetry. The MSD appears very promising in the evaluation of neck-injured patients. (J Manipulative Physiol Ther 1992; 15:343-349).

Key Indexing Terms: Cervical Vertebrae, Reliability and Validity, Muscles.
trunk and extremity muscles is commonplace (7–12), there exist very few quantitative reports on the assessment of neck muscle strength in the literature.

Manual muscle tests are a standard part of every clinical examination (13–16); however, they suffer from subjectivity and bias, lack of uniformity and lack of quantification (17–22). Isometric instrumentation devices have been studied, including manual dynamometers (17, 23–28), spring-loaded devices (29–32) and electromyogram (EMG)-based instrumentation (32–35). Agre et al. (36) reported on the use of a portable, hand-held dynamometer in the testing of a wide variety of muscle functions. The sole cervical action tested in this study was forward flexion. Intra- and interexaminer correlation coefficients for neck flexion ranged from .63 to .99 for three to four trials by three examiners. The aggregate coefficient of variation (37) was 17.3%.

Schuldt and Harms-Ringdahl and colleagues extensively studied cervical muscle function using an EMG-based experimental model (33, 38–40). Their work has demonstrated that certain cervical muscles predominate in action-specific modes: erector spinae during resisted extension and splenius capitus and levator scapula in resisted lateral flexion. They have also shown that increased force is generated in the cervical extensors during isometric extension when the neck is flexed somewhat forward, and that raising the arms to a horizontal position increases the level of activity of the thoracic erector spinae and rhomboid muscles during resisted cervical extension.

A newly reported device for isometric muscle testing is the modified sphygmomanometer-type dynamometer (MSD). This device offers some advantages in the quantification of muscle strength, namely: a) force is applied to a mechanical frame, not by a tester; resistance can, thus, be made constant. The frame may allow for variable positioning of the test platform (see Figure 1). b) The use of an air bladder provides comfortable resistance, and its physical compliance contributes actively to the mensuration (i.e., the displacement of air). This level of comfort during testing increases subject compliance. c) If interfaced with a computer, data can be digitized by A-D conversion and pretimed trials can be conducted and compared.

Two reports of the reliability of MSD have recently appeared. Wright and Goldsmith (41) studied hip abductor strength testing in children and reported an intertester intraclass coefficient of .79. Busch and Arnold (42) studied four extremity joint muscle tests and found significant correlations for three, ranging from .62 to .79. To our knowledge, no report exists regarding the use of MSD in the assessment of the strength of the cervical musculature.

This investigation was undertaken to determine the test-retest reliability of the MSD as well as to establish normative and clinical data bases from which future studies might proceed.

METHODS AND MATERIALS
The MSD used in this study is known as the Comparative Muscle Tester (CMT) (Magnatec Co. Ltd., Concord, Ontario, Canada). This device consists of a wall-mounted frame on which a platform slides vertically (see Figure 1). On the platform is an air bladder measuring approximately 8 by 25 cm, and 6 cm high when fully inflated. The hoses from the air bladder connect to a potentiometer that is interfaced with an A-D converter, which produces a digital display and is interfaced to a printer. On-board software drives a
timing mechanism capable of inflating the air bladder to 1 mm Hg (a form of calibration) and controlling various preprogrammed intervals of operation. Typical measurement periods range from 5 to 60 sec. The unit of measurement provided by the CMT is millimeters of mercury, which, for this study, is converted to kilopascals.

**Study 1**

Prior to its use in clinical-trials, it was necessary to determine the accuracy of the unit, specifically, the amount and nature of instrument-related variability. A column of four 5-lb weight discs was placed on and off the air bladder for 10 paired trials during a period of approximately 3 min. Means and SD of the first and second trials were compiled and the coefficients of variation (SD/mean) were computed (37). Of interest was whether a consistent pattern of variability existed under these standardized test conditions such that values from the second trial might be less or more than those of the first, or that values from the first three paired trials might be less or more than those of the last three. If required, the Student's t test with p ≤ .05 would be used to determine the level of significant differences.

**Study 2**

Forty young adult males (average age 25 ± 2 yr) were tested in a protocol that involved paired trials of isometric muscle tests of all six ranges of neck movement: forward flexion (FF); extension (EXT); right (RLF) and left lateral flexion; and right (RR) and left rotation. These subjects were without any current neck pain or pain within 1 yr of the study.

The measurement protocol was as follows. The subject remained standing throughout the test procedure. The air bladder platform was moved to a position such that its top edge was level with the subject's eyebrows. The subject was guided into a position with his feet lined up with a floor marker directly under the platform. In each of the test positions, care was taken to position the subject so that the subject's head rested in the neutral position against the surface of the bladder (see Figure 1). As such, a slight preload of the bladder involved no more than 10° of rotation of the head in any plane. Prior testing of this preload showed that it produced, on average, 15–35 mm Hg of pressure, an insignificant amount compared to the range of subsequent test values.

In order to optimize subject performance, verbal instructions and demonstration of each test procedure were provided by the assessor. Trial runs of each test procedure were conducted in order to ensure proper positioning of the equipment, to confirm proper performance by the subject and to reduce any test apprehension (i.e., to give the subject the feel of the equipment and the test performance). These trial runs were not recorded.

In order to reduce recruitment of trunk and lower limb muscle forces to maximize isolation of the neck musculature, two special procedures were incorporated into the test protocol. First, the assessor maintained gentle manual stabilization of the subject's pelvis during the test. This guarded against inward drift, which would increase the leverage of the trunk muscles in any of the test positions. Second, a standardized position of the subject's arms was adopted for all tests such that the arms were raised to the 90°/90° position. This greatly reduced the involvement of the scapular and pectoral muscles in the test actions. This protocol reproduces the National Institute of Occupational Safety and Health recommendations for isolation and stabilization in isometric muscle testing (43–45).

While isolation and stabilization were not construed so that specific muscles or even muscle groups could be tested, this set of procedures was deemed sufficient to test specific regional ranges of resisted motion. Accordingly, the test procedures are named according to the cervical range of motion and not the specific muscles thought to be involved (e.g., cervical flexion, as opposed to scalene muscle group, or anterior cervical flexors).

With regard to the actual test procedure, subjects were instructed to commence, upon visual signal, a gradual sustained pressure on the air bladder. They continued with maximal pressure until a buzzer sounded ending the test.

In this study, the test interval was 5 sec. The instrument is programmed with a 2-sec delay such that data are acquired over the last 3 sec of sustained maximal effort (18). During the resisted test, the assessor gave verbal feedback to the subject instructing them to "keep pushing" or "push as hard as you can" until the buzzer sounded. After a 5-sec rest interval, a second trial of the same resisted action was done.

Analysis of the data consisted of computing means and SD that provided a normative data base. Coefficients of variation for each motion were computed. Test-retest correlations were computed using Pearson Product Moment correlations.

**Study 3**

In this study, 24 symptomatic subjects presented consecutively to the problem case clinic of a chiroprac-
tic college teaching clinic, and repeated the protocol outlined in study 2. This group consisted of 12 males [average age: 39 ± 7 yr (1 SD)] and 12 females (average age: 36 ± 10 yr) who suffered from mechanical neck pain syndrome. Eight (33%) had suffered a recent whiplash-type cervical sprain/strain injury. The average duration of complaint was 22.5 wk (range: 2-102 wk). Sixteen (67%) suffered from nontraumatic mechanical complaints related to postural or occupational strains. The average duration of these complaints was 110 wk. None suffered from overt degenerative disc disease, and none had radiating brachialgic pain. Informed consent was obtained from all symptomatic subjects.

In order to reduce the strain of the paired strength testing protocol, only FF, EXT, RR and RLF were tested. As well, a modification was instituted such that, where constant maximal pressure was required of the asymptomatic subjects in study 2, for the symptomatic subjects, the protocol of acceptable (but then sustained) maximal effort was used (46). Subjects were instructed to produce a level of pressure that reached, but did not exceed, tolerable pain. They were instructed to sustain that level of pressure and not to suddenly and prematurely “give up,” as that occurrence would be deemed a test failure.

Analysis consisted of computing means, SD and coefficients of variations. Percentage differences between paired trials were computed, as well as intraclass coefficients. Descriptive comparisons between the data sets of studies 2 and 3 were conducted to determine where important differences might exist. Student’s t tests were to be used with \( p \leq .05 \) to compare means in studies 2 and 3 (see text).

RESULTS

Study 1

Table 1 depicts the results of 10 paired trials of a standard 20-lb column of weights. The two paired trials were virtually identical. The mean of the first three paired trials was 8845 ± 67.8 kPa, while that of the last three paired trials was 8831 ± 71.8 kPa. The average coefficient of variation for all trials was .84%. No \( t \) tests were conducted, given the obvious similarity of the data sets.

<table>
<thead>
<tr>
<th>Trial</th>
<th>Mean ± 1 SD</th>
<th>Coefficient of variation</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>kPa</td>
<td>%</td>
</tr>
<tr>
<td>1 (n = 10)</td>
<td>8845 ± 67.8</td>
<td>.76</td>
</tr>
<tr>
<td>2 (n = 10)</td>
<td>8831 ± 71.8</td>
<td>.84</td>
</tr>
</tbody>
</table>

Study 2

Table 2 depicts the means, SD and coefficients of variation for all six isometric tests. Table 3 lists lower cutoff values computed by subtracting 1 SD from the mean and rounding off. Table 4 depicts the Pearson’s coefficients rounded off. These are all in the range of moderate to high reliability. Comparisons of mean values of left and right lateral flexion and rotation indicate bilateral symmetry within 6-8%. A ratio was computed between mean flexion and mean EXT values as has been reported for trunk musculature (47). This value was 0.57 (F/E).

Study 3

Table 5 displays the means and SD for the four tests as well as the percentage differences between first and second trials. The intraclass coefficients are displayed in Table 6, with a range of .95-99. Table 7 displays mean values of three data sets for the four tests [A: normals (mean values for comparison); B: symptomatic subjects as a whole; and C: a subgroup from the study...
EVALUATION OF NECK STRENGTH • VERNON ET AL.

TABLE 5. Study 3. Means, SD and percentage differences, trial 1 vs. 2

<table>
<thead>
<tr>
<th>Movement</th>
<th>Mean ± 1 SD</th>
<th>Average % difference (trial 1 vs. 2)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Flexion</td>
<td>1982 ± 1250</td>
<td>10.4</td>
</tr>
<tr>
<td>EXT</td>
<td>4336 ± 1516</td>
<td>7.0</td>
</tr>
<tr>
<td>RLF</td>
<td>3684 ± 2195</td>
<td>6.4</td>
</tr>
<tr>
<td>RR</td>
<td>3857 ± 1583</td>
<td>4.0</td>
</tr>
<tr>
<td>Total</td>
<td></td>
<td>7.0</td>
</tr>
</tbody>
</table>

TABLE 6. Study 3. Intraclass correlation coefficients

- Flexion 1 vs. flexion 2: 0.98
- EXT 1 vs. EXT 2: 0.95
- Rotation 1 vs. Rotation 2: 0.99
- Lateral bend 1 vs. lateral bend 2: 0.98
- All ranges of motion 1 vs. all ranges of motion 2: 0.98

TABLE 7. Study 3. Comparison of values from symptomatic subjects with normal values (as in study 2)

<table>
<thead>
<tr>
<th>Movement</th>
<th>Normal (mean)</th>
<th>Symptomatic Total</th>
<th>Whiplash</th>
</tr>
</thead>
<tbody>
<tr>
<td>Flexion</td>
<td>4615</td>
<td>1982</td>
<td>1184</td>
</tr>
<tr>
<td>EXT</td>
<td>7927</td>
<td>4336</td>
<td>4655</td>
</tr>
<tr>
<td>RLF</td>
<td>7950</td>
<td>3684</td>
<td>4735</td>
</tr>
<tr>
<td>RR</td>
<td>7315</td>
<td>3857</td>
<td>2647</td>
</tr>
</tbody>
</table>

3 sample, all of whom had suffered a whiplash-type injury. The percentage differences between the mean normal values and the mean values in group B are 43% (FF), 55% (EXT), 53% (RR) and 46% (RLF). The differences between the mean normal values and the mean values in group C are 26% (FF), 59% (EXT), 36% (RR), and 60% (RLF), and all values are reductions. Student's t tests on each of these comparisons were significant at at least the p < .05 level.

Table 8 compares the flexion/extension (F/E) ratios in each of the three groups. While the F/E ratio for the total symptomatic group is somewhat less than for normals, the ratio computed from the whiplash group is reduced by more than half.

DISCUSSION

From the results of study 1, it can be stated quite confidently that the MSD is highly accurate, with instrument-related variability or error at less than 1%. This validates the next stages of investigation into performance and clinical-related parameters as in studies 2 and 3.

Regarding performance criteria, the above-described protocol of instructions and subject assistance appears to generate a high level of test-retest reliability in isometric strength evaluations in the cervical spine. The coefficients of variation are all in the low range of clinical testing, indicating that group data can be collected with acceptably low variability.

The normative values from study 2 are self-explanatory; however, the bilateral symmetry in lateral flexion and rotation (within 6-8%) is noteworthy, especially as a baseline for comparison to subjects with unilateral pain syndromes. The relative weakness of cervical flexion as compared to EXT is consistent with findings reported for the trunk (47). This is an expected finding since the cervical spine is, like the lumbar, lordotic in curvature. Since this lordosis is maintained in large measure by sustained postural activity of the extensor musculature, a bias of strength toward these muscles under isometric testing would be expected.

In summary, the chief finding of the normative data base is its consistency with expected and theoretical values, even if these have not all been previously reported. Given the findings of studies 1 and 2, a high level of accuracy (low instrument-related error) and a reasonably high level of performance-related reliability and consistency, the MSD-isometric testing of clinical subjects is logical. The findings of study 3 confirm that cervical pain patients do indeed experience a reduction of isometric strength in all test conditions.

Since there is, overall, a similar level of variability between patients as compared to normal subjects (approximately 10%), the significant reduction of test values must originate from some important feature or features of the clinical status. These could include apprehension, pain or true disuse, or injury-related weakness. Exactly which of these is operational and in what proportions remain to be determined.

This is further confirmed by the progressive reduction in the strength of the cervical flexor muscles as opposed to the extensors in the symptomatic subjects. As such, the consequences of neck pain appear to include not only general reductions of isometric muscle strength, but also a progressive anterior-to-posterior imbalance, apparently related to the severity of the clinical status.
The implications of this finding both in regard to the natural history of mechanical neck pain complaints and their amenability to treatment regimes remain an important set of issues for future studies. At this point, it should be noted that these findings are in general agreement with data from trunk strength tests of patients with low back pain. In these subjects, general levels of weakness are present, as well as a selective weakness of the extensor muscles. Again, this apparently correlates to clinical severity (7, 8, 29–31, 46–48).

CONCLUSION

The modified sphygmomanometer dynamometer has been shown to be a highly accurate and reliable instrument for the clinical assessment of isometric strength of neck musculature. A test protocol and a normative data base have been described which, when applied to a clinical population, reveal significant global weakness in neck pain sufferers. Those with whiplash-type injuries, in particular, appear to suffer even greater reductions of strength values, specifically related to the cervical flexor muscles.

The use of MSD-isometric strength testing offers exciting potential in the study of mechanical neck pain and in validating potentially useful treatments for this all-too-common malady.

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Excitatory effects on neck and jaw muscle activity of inflammatory irritant applied to cervical paraspinal tissues


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Summary A study was carried out in 19 anaesthetized rats to determine if the electromyographic (EMG) activity of jaw and neck muscles could be influenced by injection of the inflammatory irritant mustard oil into deep paraspinal tissues surrounding the C1–3 vertebrae. The EMG activity was recorded ipsilaterally in the digastric, masseter and trapezius muscles and bilaterally in deep neck muscles (rectus capitis posterior). In comparison with control (vehicle) injections, mustard oil (20 μl, 20%) injected into the deep paraspinal tissues induced significant increases in EMG activity in the neck muscles in all the animals and in the jaw muscles in the majority of the animals; the effects of mustard oil were more prominent in the former. The EMG response evoked by mustard oil injection was frequently reflected in two phases of enhanced activity. The early phase of the increase in EMG activity was usually initiated immediately following mustard oil injection (mean latency: 20.4 ± 17.7 sec) and lasted 1.6 ± 1.1 min. The second phase occurred 11.3 ± 7.6 min later and lasted 11.0 ± 8.1 min. Evans Blue extravasation was apparent in the deep paraspinal tissues surrounding the C1–3 vertebrae after mustard oil injection, and histological examination showed that mustard oil injection induced an inflammatory reaction in the rectus capitis posterior muscle. These results document that injection of the inflammatory irritant mustard oil into deep paraspinal tissues results in a sustained and reversible activation of both jaw and neck muscles. Such effects may be related to the reported clinical occurrence of increased muscle activity associated with trauma to deep tissues.

Key words: Electromyography; Inflammatory irritant; Neck muscle; Jaw muscle

Introduction

Noxious stimulation of muscle or articular tissues can reflexly evoke somatic and autonomic changes. In the craniofacial region, electrical or algesic chemical stimulation of high-threshold afferents in the Vth, VIth, and XIIth cranial nerves can elicit reflex responses in the jaw, tongue, and facial muscles that result from activation of nociceptive afferents supplying the temporomandibular joint (TMJ) or craniofacial musculature (e.g., Hanson and Widén 1970; Lindquist and Mårtensson 1970; Willer and Lamour 1975; Sessle 1977, 1990; Dubner et al. 1978; Broton and Sessle 1988; Sessle and Hu 1991). These findings suggest that trauma or noxious stimulation of deep craniofacial tissues may produce a sustained excitation of several masticatory muscles that may serve to protect the masticatory system from potentially damaging movements (Sessle and Hu 1991). Similarly in the spinal system, a limb flexion reflex can also be evoked by noxious mechanical, algesic chemical, or high-threshold electrical stimulation of muscular or articular tissues of the limbs. Deep noxious stimuli generally activate alpha-motoneurones supplying limb flexor muscles and inhibit alpha-motoneurones innervating limb extensors (e.g., Wall and Woolf 1984; Mense 1991). Although of short latency and brief duration, these effects have nonetheless also been viewed as nociceptive reflex responses serving important protective functions, in these cases to protect the limb from further noxious stimulation and to counteract excessive movement so as to prevent further damage to its muscular or articular tissues.

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Studies in the spinal cord have also recently shown that high-intensity electrical stimulation as well as noxious mechanical or chemical stimuli applied to joint and muscle can activate small-diameter afferents and produce a prolonged excitation of spinal dorsal horn and ventral horn neurones in rats and cats (e.g., Woolf and Wall 1986; Guilbaud 1988; Schaible and Schmidt 1988; Hoheisel and Mense 1989; Neugebauer and Schaible 1990; Mense 1991; Woolf and Thompson 1991). One of these chemical stimuli is mustard oil, a small-fibre excitant and inflammatory irritant (Jancso et al. 1967, 1977; Jancso-Gabor et al. 1976; Reeh et al. 1991) which may induce a prolonged facilitation of craniofacial and cervical reflex pathways after experimental injury or inflammation of cervical tissues. Yet such effects have been implicated clinically in myofascial dysfunction in these regions after injury or inflammation of cervical muscles and paraspinal tissues of the neck (e.g., Feinstein 1977; Wyke 1979; Goodman 1988; Simons 1988; Jaeger 1989).

Therefore, effects of deep cervical inputs to the central nervous system were tested on jaw and neck muscle activity in rats. The application to the cervical paraspinal tissues of the inflammatory irritant, mustard oil, was used to activate these deep inputs.

Some of these data have been briefly presented in abstract form (Sessle et al. 1991).

**Methods**

The study was carried out on 19 male Sprague-Dawley rats (250-350 g). Anaesthesia was initially induced by ketamine (130 mg/kg, i.p.) so that a tracheal cannula could be inserted and the right external jugular vein cannulated. Then anaesthesia was maintained by a mixture of 1:2 O₂, 2/3 N₂O, and 1/2 halothane. The animal was placed in a stereotaxic apparatus and two screws were inserted into the exposed dorsal surface of the skull. A bar that was connected to the stereotaxic apparatus was placed in contact with the screws and fixed in place with dental cement so that the stereotaxic ear and incisor bars could be removed. After surgery, the halothane concentration was maintained at 0.3-0.5% so that noxious pressure applied to the hindpaw could induce a slight flexion reflex response of the hindlimb. Heart rate, expired percent CO₂ and body core temperature were continuously monitored and maintained at physiological levels of 52-53°C/min, 3.5-5.5% and 37-37.5°C, respectively. A pair of bipolar EMG electrodes (36-40-ga. single-stranded, Teflon-coated stainless-steel wire: interelectrode distance 0.5 cm; exposed tips 0.5 mm) was inserted through the intact skin into each of the digastic, masseter and trapezius muscles on the side ipsilateral to the injection site (see below) and bilaterally into the deep neck muscles (rectus capitis posterior). Electrode locations were checked by dissection after each experiment, and confirmed in all the target muscles mentioned above, except for the digastic muscle of 1 rat and trapezius muscles of 2 rats: the EMG data recorded from these three muscles were not included in the subsequent analysis. Two to 3 h after the initial ketamine induction, the resting EMG activity in the muscles was monitored before, during and after the injection of either mineral (20 μl) or mustard oil (20% allyl-isothiocyanate, BDH. Pools: in mineral oil, 20 μl) into the deep paraspinal tissues of the left side of the neck. The injection was made from a dorsal approach. 1.5 cm behind the occipital ridge and 0.5 cm lateral to the midline (e.g., Fig. 3a); the injection site was 1 cm below the skin surface, within the paraspinal tissues around the Cl–3 vertebrae.

In order to assess the stability of the EMG recording conditions, the EMG activity of the five muscles was continuously recorded in 3 animals for 2-4 h without any mineral and mustard oil injection. The effect of mustard oil was assessed in another 9 animals in which the EMG activity was first recorded for 20 min before and for 10-20 min after vehicle (mineral oil) injection, and then for another 30-40 min after mustard oil was injected. As control for the effect (see Results) of mustard oil injection into the deep paraspinal tissues, the EMG activity in another 4 animals was recorded according to the same protocol described above for the 9 animals except that a second injection of mineral instead of mustard oil was delivered, and in 3 animals the effect of mustard oil injected into the gastrocnemiussoleus muscle on the EMG activity of the jaw and neck muscles was tested.

The EMG activity of each muscle was amplified (gain: 1000-5000 X; bandwidth: 20-3000 Hz) and displayed on oscilloscopes, and also recorded on a FM tape recorder, for 1 min every 3-5 min before vehicle injection; immediately after vehicle or mustard oil injection, EMG activity was continuously recorded for 3–5 min and thereafter for 1 min every 3–5 min. The EMG data recorded into the computer was processed offline with the program Spike2 (CED, Cambridge). The EMG signals recorded were rectified and integrated every 1 sec to allow the latency of onset of EMG activity to be determined; in addition, since the increase in EMG activity induced by the injection of mustard oil lasted up to 20 min (see Results), the EMG activity was also rectified and integrated every 60 sec to allow the duration of any EMG response to be noted. Because insertion of the injection needle could itself sometimes induce a small transient (5-10 sec) increase in neck EMG activity, injection of mineral or mustard oil did not commence until the EMG activity had returned to pre-insertion levels; the integrated EMG activity associated with any such transient response evoked by needle insertion was not included in the data analysis.

Since in all rats injected with mineral or mustard oil there was no significant variation in EMG activity of each muscle before vehicle injection (Wilcoxon test P > 0.05, e.g., Fig. 3), all pre-injection data points for each muscle were pooled in each of these 16 animals to produce a mean value which represented the pre-injection level of EMG activity for the muscle in each animal. All post-injection data points were normalized relative to this mean value and expressed as a percentage value of the mean pre-injection level (EMG activity). Changes in EMG activity after injection of either mineral or mustard oil were regarded as an increase if one or more EMG data points rose 2 standard deviations (S.D.) above this mean baseline level. The time from the beginning of injection to the increase in EMG activity was designated as the latency of the response, and that from the increase in EMG activity to recovery to the pre-injection level of EMG activity was designated as the duration of the response.
The incidence of these EMG changes for each muscle were compared statistically with Fisher's exact probability test (Fisher test). The Wilcoxon test was used for statistical comparisons between the mean value representing the pre-injection level of EMG activity (see above) and any data point of the postinjection periods. The Mann-Whitney test was used for a population comparison of the EMG activity between the group of 9 animals receiving mineral oil followed by mustard oil and the 4 control animals receiving injections of mineral oil twice. Values were expressed as mean ± S.D., and P values less than 0.05 (2-tailed) were regarded as significant.

Recent studies in our laboratories (Haas et al. 1992) have shown that with mustard oil injected into deep craniofacial tissues, the visual localization of the mustard oil-induced Evans Blue extravasation correlates well with the spectrophotometric detection of its presence and histological parameters of inflammation. Therefore, in the present study, at the end of each experiment 40 min after the injection of mustard oil (n = 9) or the second injection of mineral oil (n = 4), Evans Blue (0.2 ml, 1%) was injected through the catheter in the right external jugular vein and 20 min later the animal was given a lethal injection (T61, Hoechst, Montreal). The mustard oil injection site was visually localized according to the appearance of extravasated dye and was then outlined on a standard drawing of the neck region of the rat; the tissues were also examined histologically for evidence of inflammation.

Results

None of 3 animals examined to assess the stability of the EMG recording conditions showed any change greater than 2 S.D. in the baseline level of EMG activity over the 2-4 h observation period. In the remaining 16 animals, there was also no significant variation in EMG activity in any muscle in the 20 min period before injection of mineral oil into the deep paraspinal tissues of Cl-3 or of mustard oil into the gastrocnemius-soleus muscle. Injection of mineral oil produced a transient increase above the baseline EMG activity level in only a few animals (e.g., Fig. 1). When the data for each of five muscles of all the animals receiving mineral oil followed by mustard oil was statistically analyzed, there was no significant increase (P > 0.05, Wilcoxon test) in EMG activity induced by mineral oil injection for any muscle of the group of animals (Fig. 2). The experiments in which the control animals received injections of mineral oil twice verified that the second injection of mineral oil did not induce a significant change in EMG activity (see Table I and Fig. 2), and the magnitude of the mineral oil-induced increase in EMG activity was significantly smaller (P < 0.05, Mann-Whitney test) than that induced by mustard oil (Fig. 2).

In the 9 animals receiving an injection of mustard oil into the deep paraspinal tissues, a large increase in EMG activity occurred in the majority of animals (e.g., Figs. 1 and 3) and the incidence was significantly higher (P < 0.05, Fisher test) than that associated with the injection of mineral oil (Table I). Furthermore, the magnitude of the mustard oil-induced EMG increase in this group of animals was significantly higher (P <
contrast had a lower frequency of occurrence: 40% for the ipsilateral digastric muscles, 25% for the ipsilateral masseter muscles, 30% for the ipsilateral trapezius muscles and 60% for the ipsilateral as well as the contralateral deep neck muscles. The early EMG increase usually occurred soon after the mustard oil injection (mean latency: 20.4 ± 17.7 sec) and lasted only 1.6 ± 1.1 min before returning to the pre-injection levels (Figs. 1 and 2). The second phase in EMG activity occurred at a mean latency 11.3 ± 7.6 min after the mustard oil injection, and its amplitude was sometimes (30%) even greater than that of the early phase (e.g., Fig. 1). The mean duration of the second phase (11.0 ± 8.1 min) was significantly ($P < 0.01$, Wilcoxon test) longer than that of the early phase; thereafter the EMG activity returned to pre-injection levels and no further increase in EMG activity was noted for the rest of the recording period (up to 1 h). Fig. 2 shows the changes in EMG activity induced by injections of mineral and mustard oil into deep paraspinal tissues surrounding the C1-3 vertebrae. The means ± S.D. data shown are normalized values relative to the mean value of the pre-injection EMG activity (100%) in each rat. The solid line shows the changes in EMG activity of one group of rats receiving mustard oil at 20 min after the vehicle (mineral oil) injection (n: number of rats tested by the injection of mustard oil). The dashed line shows the changes in EMG activity of the control group of rats receiving the vehicle, instead of mustard oil, again 20 min after the first vehicle injection. * and **: $P < 0.05$ and $P < 0.01$ (Wilcoxon test) indicate significant difference between pre-injection and postinjection level of EMG activity. * and **: $P < 0.05$ and $P < 0.01$ not only indicate significant difference between the pre-injection and the postinjection level of EMG activity (Wilcoxon test), but also between the animals receiving mineral then mustard oil and the animals receiving mineral oil injection twice (Mann-Whitney test). Abbreviations as for Fig. 1.
TABLE I
INCIDENCE OF INCREASE IN EMG ACTIVITY AFTER INJECTION OF EITHER MINERAL OR MUSTARD OIL INTO THE DEEP PARASPINAL TISSUES SURROUNDING THE C1-3 VERTEBRAE

Ipsi., ipsilateral; Contr., contralateral; deep neck, rectus capitis posterior muscle. Numbers in parentheses represent number of rats tested (see Methods). Values in the left two columns are results recorded in the group of 4 control animals, and in the right two columns are results in the group of animals receiving mineral oil then mustard oil.

<table>
<thead>
<tr>
<th>Muscles</th>
<th>First injection Mineral oil (%)</th>
<th>Second injection Mineral oil (%)</th>
<th>First injection Mustard oil (%)</th>
<th>Second injection Mustard oil (%)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Ipsi. digastric</td>
<td>0 (4)</td>
<td>25 (4)</td>
<td>75 (8)</td>
<td></td>
</tr>
<tr>
<td>Ipsi. masseter</td>
<td>50 (4)</td>
<td>0 (4)</td>
<td>33 (9)</td>
<td></td>
</tr>
<tr>
<td>Ipsi. trapezius</td>
<td>50 (4)</td>
<td>57 (4)</td>
<td>86 (7)</td>
<td></td>
</tr>
<tr>
<td>Ipsi. deep neck</td>
<td>25 (4)</td>
<td>22 (4)</td>
<td>89 (9)</td>
<td></td>
</tr>
<tr>
<td>Contr. deep neck</td>
<td>25 (4)</td>
<td>22 (4)</td>
<td>100 (9)</td>
<td></td>
</tr>
</tbody>
</table>

* Indicates that incidence of the increase in EMG activity induced by injection of mustard oil was significantly higher than that induced by injection of mineral oil in the group of animals receiving mineral oil then mustard oil (P < 0.05 Fisher test).
** Indicates that the incidence of the increase in EMG activity induced by injection of mustard oil was not only significantly higher than that induced by injection of mineral oil in the group of animals receiving mineral oil then mustard oil, but also higher than that induced by the second injection of mineral oil in the group of control animals (P < 0.05 Fisher test).

phases of the increased EMG activity induced by mustard oil injection. Note that the second phase was especially prominent in the deep neck muscles.

There was no evidence of Evans Blue extravasation or histological evidence of inflammation in the deep paraspinal tissues of C1-3 of any of the 4 animals receiving two injections of mineral oil. Evans Blue extravasation and an inflammatory reaction were, however, apparent in the 9 animals receiving mustard oil. The dye was localized to the deep paraspinal tissues surrounding the C1-3 vertebrae (e.g., Fig. 3). Although the dye was mainly found in the deep paraspinal tissues on the side ipsilateral to the injection, in 5 of the 9 animals the dye also spread to the contralateral side. No relationship was apparent between the magnitude of the increased EMG activity in each muscle produced by the injection of mustard oil and the location of the dye (Fig. 3). Histological examination of these deep paraspinal cervical tissues revealed, in accor-

Fig. 3. Location of extravasated dye, and magnitude of mustard oil-induced EMG activity in 9 rats: a: site of injection shown by the spot with soft tissue and skeletal structures outlined. Shaded regions in b-j show the visualised location of dye. In the lower parts of the figure, symbols (++, +++ and ++) reflect magnitudes of the peak increases in the EMG activity induced by mustard oil injection (regardless of the response phase). The pre-injection EMG activity level in each muscle was represented by the mean value of EMG activity before the vehicle injection (= 100%). +++ EMG activity increased by more than 1000% of the pre-injection level; +++ EMG activity increased by 500-1000% of the pre-injection level; ++ EMG activity increased by 200-500% of the pre-injection level; + EMG activity increased by less than 200% of the pre-injection level; - no increase in EMG activity. Abbreviations as for Fig. 1.

TABLE II
MUSTARD OIL-INDUCED PEAK INCREASE IN EMG ACTIVITY

Abbreviations as for Table I. n, Number of animals in which the mustard oil injection into the deep paraspinal tissues surrounding the C1-3 vertebrae induced an increase in EMG activity. The peak increases in EMG activity are expressed in terms of the percentage of the pre-injection level of the EMG activity in each muscle. The values in the table are given as mean ± S.D.

<table>
<thead>
<tr>
<th>Muscles</th>
<th>Increase in EMG activities</th>
</tr>
</thead>
<tbody>
<tr>
<td>Ipsi. digastric (n = 6)</td>
<td>377 ± 254%</td>
</tr>
<tr>
<td>Ipsi. masseter (n = 8)</td>
<td>204 ± 52% *</td>
</tr>
<tr>
<td>Ipsi. trapezius (n = 6)</td>
<td>968 ± 153%</td>
</tr>
<tr>
<td>Ipsi. deep neck (n = 8)</td>
<td>932 ± 826%</td>
</tr>
<tr>
<td>Contr. deep neck (n = 9)</td>
<td>424 ± 30%</td>
</tr>
</tbody>
</table>

* Indicates that the EMG response amplitude of the masseter muscle was significantly smaller than that of the ipsi. trapezius, ipsi. deep neck and contr. deep neck muscles (P < 0.05 Wilcoxon test).
dance with analogous findings when mustard oil is injected into deep craniofacial tissues (Haas et al. 1992), an inflammatory reaction in the connective tissues between the rectus capitis posterior muscle fibres and clear evidence of congestion, extravasation and invasion of tissues by polymorphonuclear leucocytes. No such inflammatory reaction was apparent in these or other tissues when only mineral oil was injected.

Discussion

The present study has provided the first documentation that stimulation of cervical paraspinal tissues by an inflammatory irritant results in an inflammatory response in the paraspinal tissues and in a sustained and reversible activation of both jaw and neck muscles. This activation is especially prominent in the deep neck muscles, and may be manifested as two phases of increased EMG activity.

In the present study, we have documented that the use of an O₂/N₂/O/halothane mixture is associated with a stable preparation and stable baseline EMG activity. This is consistent with other studies (see Le Bars et al. 1980; Benoist et al. 1984; Dallal et al. 1990). Variability in the EMG response to mustard oil injection was observed but it is presently unclear what factors may have accounted for this. e.g., under our experimental conditions, we did not find any relationship between the incidence or magnitude of the EMG response and the anaesthesia level, or the incidence or magnitude of the EMG response and location or extent of the Evans Blue extravasation induced by mustard oil. Sympathetic efferents may have a role in the peripheral modulation of the response, but their role in mustard oil-induced effects here yet to be examined.

Mustard oil has been shown to excite small-diameter nociceptive afferents, but not the large-diameter non-nociceptive afferents supplying skin (Reeh et al. 1986; Woolf and Wall 1986; Handwerker and Reeh 1991); it also acts as an inflammatory irritant (Jancsó et al. 1967, 1977; Jancsó-Gábor et al. 1976; Woolf and Wall 1986; Handwerker and Reeh 1991). The time course of the increased EMG activity induced by mustard oil that was observed in this study is not inconsistent with that of the activation of C-fibre afferents that may last for up to 30 min (Reeh et al. 1986). High-intensity electrical stimulation of limb muscle afferents at C-fibre strength can indeed produce a prolonged facilitation of the evoked flexion reflex and this effect has been related to pathophysiological mechanisms occurring in response to injury or inflammation (Wall and Woolf 1984; Woolf and Wall 1986).

Mechanisms underlying the mustard oil-induced increased excitability of the jaw and neck muscles also are likely related to the activation of nociceptive afferents supplying in this case the paraspinal tissues of the neck, with the subsequent activation of brain stem and cervical interneurones and then motoneurones supplying the muscles (Dubner et al. 1978; Abrahams 1986; Abrahams and Richmond 1988). As noted above, there is no evidence that large-diameter afferents are involved in the excitatory effects induced by mustard oil. Furthermore, the activity of large-diameter muscle afferents innervating the gastrocnemius-soleus muscle is indeed significantly inhibited following the injection of inflammatory irritants such as carrageenan into the muscle (Yu, Scherotzke and Mense, unpublished data). Unmyelinated and small-diameter myelinated afferents and free nerve endings predominate in the neck muscles (Abrahams and Richmond 1988; Richmond et al. 1988). Unfortunately, there has been no detailed study of the response properties of the nociceptive afferents supplying deep cervical tissues, but it is reasonable to assume that they show properties similar to nociceptive afferents supplying deep tissues of the limbs, trunk and head (e.g., Dubner et al. 1978; Mense 1986, 1991; Handwerker and Reeh 1991; Sessle and Hu 1991). The latency of the EMG response was longer than that reported for mustard oil-induced excitation of primary afferent fibres, and the time course of the EMG response is comparable to the temporal features of the increased excitability of sensory neurones in the trigeminal spinal tract nucleus induced by the injection of mustard oil into the deep masseter muscle (Hu et al. 1992) and tongue (Yu et al. 1991); this nucleus is the site of reflex interneurones serving in many craniofacial and cervical reflex pathways (Dubner et al. 1978; Rossignol et al. 1988; Olsson and Westberg 1989). However, it should be noted that the time courses of both the increased EMG activity and the neuronal hyperexcitability induced by mustard oil (Yu et al. 1991, 1992; Hu et al. 1992) are not comparable to the temporal features of the inflammation process which may last more than 2 h after application of mustard oil (Reeh et al. 1986; Haas et al. 1992).

A small transient increase in EMG activity sometimes occurred after injection of mineral oil. However, in contrast to mustard oil, EMG changes produced by the mineral oil injection occurred only in a few animals and were much smaller in amplitude and shorter in duration. This was verified in the experiments in which the control animals received two injections of mineral oil. The short-lasting increase in EMG activity probably reflected the response to mechanical stimulation produced by the needle or to the volumetric stimulation of the tissues.

Our data indicate that the effects of mustard oil sometimes included two phases of increased EMG activity, especially in the deep neck muscles. There has been no report detailing two phases of activation of nociceptive afferents by mustard oil, or of a 2-
reflex effect of mustard oil, other than the late phase of hyperexcitability noted for some neurones of the trigeminal spinal tract nucleus (Yu et al. 1991; Hu et al. 1992). However, mustard oil applied to the skin of the rat hindpaw first induces a short burst of activity of flexor alpha-motoneurones lasting less than 3 min, and then a facilitation of the pinch-evoked limp flexor reflex that lasts up to about 100 min (Woolf and Wall 1986). The two phases of increased EMG activity are somewhat reminiscent of the temporal features of the effects of formalin injected into subcutaneous tissues (Dubuisson and Dennis 1977; Dickenson and Sullivan 1987; Hunskaar and Hole 1987). Central mechanisms, however, also may contribute since the central neural and reflex changes induced by mustard oil have been viewed, at least in part, as reflecting a 'central sensitization' or 'functional plasticity' of central nociceptive neurones that is initiated, but not necessarily maintained, by nociceptive primary afferent input (Woolf 1989; Wall 1985; Neugebauer and Schäible 1990; Dubner et al. 1992).

The effects that we observed can be attributed to reflex responses, as opposed to direct effects on the muscles by peripheral spread of the mustard oil, for several reasons: (i) as mentioned above, the time course of the increase in EMG activity is consistent with that seen in primary afferents (Reeh et al. 1986) and neurones in the trigeminal spinal tract nucleus (Yu et al. 1991; Hu et al. 1992), (ii) the limited extent of extravasation of Evans Blue suggests that the acute inflammation induced by mustard oil was localized to deep paraspinal tissues surrounding the Cl-3 vertebrae, (iii) the increased EMG activity was seen in muscles remote from the injection site as well as on the side contralateral to the injection site, and (iv) pre-application of local anaesthetic into the mustard oil injection site (Woolf and Wall 1986; Yu et al. 1993) or of the C-fibre neurotoxin capsaicin to afferent nerves (Woolf and Wall 1986) can prevent the excitatory effects of mustard oil.

In contrast to the lack of EMG changes associated with the application of noxious pressure to the hindpaw or the injection of mustard oil into the gastrocnemius-soleus muscle, injection of mustard oil into the deep paraspinal tissues induced an increase in EMG activity in the jaw muscles as well as the neck musculature in most of the animals tested. These effects were more prominent in the neck muscles which is suggestive of a segmental or somatotopic organization to the responses that we have documented, with more limited expression in muscles (e.g., jaw muscles) more remote from the site of the irritant stimulus. This also has clinical implications since trauma or inflammation of deep structures is more likely to manifest myofascial responses adjacent to the injury (Guilbaud 1988; Goodman 1988; Kehlet 1991), although there was no apparent relationship between the EMG response amplitudes and the location and spread of the extravasated Evans Blue in the deep paraspinal tissues surrounding the Cl-3 vertebrae.

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Effects of inflammatory irritant application to the rat temporomandibular joint on jaw and neck muscle activity

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Summary An electromyographic (EMG) study was carried out in 40 anaesthetized rats to determine if the activity of jaw and neck muscles could be influenced by injection of the small-fibre excitant and inflammatory irritant mustard oil into the region of the temporomandibular joint (TMJ). Injection of a vehicle (mineral oil, 20 μl) did not produce any significant change in EMG activity. In contrast, injection of mustard oil (20 μl, 20%) evoked increases in EMG activity in the jaw muscles but not in the neck muscles. The increased EMG activity evoked by mustard oil was reflected in 1 or 2 phases of increased activity. The early EMG increase occurred soon after the mustard oil injection (mean latency ± SD: 3.5 ± 2.3 sec), peaked within 1 min, and then subsided (mean duration: 7.5 ± 5.2 min). The later EMG increase occurred at 14.6 ± 10.0 min after the mustard oil injection and lasted 14.3 ± 12.3 min. These excitatory effects of mustard oil on the EMG activity of jaw muscles appear to have a reflex basis since they could be abolished by pre-administration of local anaesthetic into the TMJ region. These results document that TMJ injection of mustard oil results in a sustained and reversible activation of jaw muscles that may be related to the reported clinical occurrence of increased muscle activity associated with trauma to the TMJ.

Key words: Electromyography; Inflammatory irritant; Temporomandibular joint; Jaw muscle; Neck muscle

Introduction  

It has previously been documented in cats (Broton and Sessle 1988) that electrical stimulation of temporomandibular joint (TMJ) afferents or injection of algesic chemicals (7% NaCl, KCl, histamine) into the TMJ resulted in a sustained (≥ 30 sec) reflex increase in electromyography (EMG) activity of the genioglossus, anterior digastric and middle temporalis muscles. Another algesic chemical is mustard oil, which is a small-fibre excitant and inflammatory irritant (Jancsó et al. 1967, 1977; Woolf and Wall 1986; Handwerker and Reeh 1991), and our recent study in rats (Hu et al. 1993) has demonstrated that mustard oil injected into deep paraspinal cervical tissues can induce a sustained and reversible increase in EMG activity in both neck and jaw muscles. These findings suggest that trauma or noxious stimulation of craniofacial or paraspinal deep tissues can produce a sustained excitation of several masticatory muscles that may serve to protect the masticatory system from potentially damaging movements (Sessle and Hu 1991). Such reflex effects have been implicated clinically in TMJ pain and dysfunction (Dubner et al. 1978; Storey 1979; Zarb and Speck 1979; Laskin et al. 1983; Möller 1985; Bell 1986), and may have as their basis the ‘central sensitization’ of V brain-stem neurones (Hu et al. 1992), many of which serve as reflex interneurones in jaw and neck motor pathways (e.g., Dubner et al. 1978; Sessle and Hu 1991).

While free nerve endings and small-diameter slowly conducting afferents have been implicated in pain and nociceptive reflexes associated with trauma or dysfunction of the TMJ (Klineberg 1971; Dubner et al. 1978; Broton and Sessle 1988; Broton et al. 1988; Sessle and Hu 1991), there are no available data dealing with the effect of the small-fibre excitant and inflammatory irritant mustard oil injected into TMJ on jaw and neck

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muscle activities. In the present study, therefore, we tested whether injection of mustard oil into the TMJ area could reflexly induce an EMG increase in jaw and neck muscles. Some of these data have been briefly presented in abstract form (Yu et al. 1992, 1993b).

**Methods**

The study was carried out on 40 Sprague-Dawley rats of either sex (250–450 g). The animals were initially anaesthetized by a mixture of O₂/N₂O (1:2), and 2–3% halothane. A tracheal cannula was inserted and the right external jugular vein cannulated. The animal was placed in a stereotaxic apparatus and two screws were inserted into the exposed dorsal surface of the skull. A bar that was connected to the stereotaxic apparatus was placed in contact with the screws and fixed in place with dental cement so that the stereotaxic bar and incisor bars could be removed. After surgery, a cannula consisting of 3 needles (30-ga) connected to 2 Hamilton syringes (20 μl) with polyethylene tubes for injection was passed through the TMJ capsule into the TMJ on the left side and fixed in place. During the EMG recordings (see below), the halothane concentration was maintained at 0.5–0.8% so that noxious pressure applied to the hind paw could induce only a slight flexion reflex response of the hind limb; Heart rate, percent expired CO₂, and body core temperatures were continuously monitored and maintained at physiological levels of 33.0–34.0°C/min, 4–5%, and 37.0–37.5°C, respectively.

Pairs of bipolar EMG electrodes (36–40-ga, single-stranded, Teflon-coated stainless-steel wire; interpolar distance: 0.3–0.5 cm; exposed tips: 0.5–1 mm) were inserted into the digastic muscle on the side ipsilateral to the injection site and bilaterally in the masseter muscles in all the 40 animals and also in the ipsilateral trapezius and bilateral deep neck muscles (rectus capitis posterior) in 7 of these animals. The EMG electrode locations were confirmed by postmortem dissection immediately after each experiment.

The EMG activity of each muscle was amplified (gain: 10,000–50,000×; bandwidth: 30–3000 Hz) and displayed on oscilloscopes, and also recorded online with a data acquisition system (consisting of an IBM AT 386 computer, CED 1401 Plus hardware and software 'Spike2'; CED, Cambridge). Signal sampling rate was 2000 Hz. The EMG activity was recorded for 1 min every 3–5 min for the first 20 min; immediately after either the mineral or mustard oil injection, EMG activity was continuously recorded for 3–5 min and thereafter for 1 min every 3–5 min, except that in the 15 animals tested only with the mustard oil injection (see below), EMG activity was continuously recorded for 20 min before the mustard oil injection and for 30–240 min after the mustard oil injection.

In all animals, EMG activity was first observed for 20 min prior to any injection. In 15 animals, mustard oil (30% allylthiocyana- nat, BDH, Poole; in mineral oil, 20 μl) was injected into the TMJ region and EMG activity was continuously recorded thereafter for 30–240 min. As a control for the effect of the mustard oil injection, mineral oil (vehicle, 20 μl) was injected into the TMJ region of another 15 animals. In 7 of these 13 animals, mustard oil was also injected into the same site at 30 min after the mineral oil injection; and EMG observations continued for another 30–40 min. In another 12 animals the local anaesthetic lidocaine (1.4 mg/kg, 2%, Abbott, Chicago, IL; n = 6) or saline (vehicle, 20 μl; n = 6) was injected into the TMJ region, followed by mustard oil into the same site 5 min later.

At the end of each experiment, Evans Blue dye (5 mg/kg) was injected through the catheter in the right external jugular vein, and 10 min later a euthanasic agent (Th6, Hoechst) was administered. The mustard oil injection site was visually localized according to the appearance of extravasated dye and the tissues were also examined histologically for evidence of inflammation (Haus et al. 1992; Hu et al. 1993).

The recorded EMG data was processed offline with the CED 1401 Plus software 'Spike2': at first, EMG signals were rectified, and then EMG area integrated separately for each 1 and 60 sec. EMG activity of each muscle was expressed with the data points representing the integrated area for each 60-sec segment.

Data points of EMG activity for each muscle recorded in the initial 20 min in each animal were pooled to produce a mean rate which represented the baseline level of EMG activity for the muscle. Since EMG recording is a multi-unit recording, variability may be produced by the EMG recording technique itself. Therefore, relative changes in EMG activity with respect to the baseline EMG level were used to assess the effects of chemical injections into the TMJ. All data points were normalized relative to the mean value and expressed as a percentage value of the mean baseline level of the EMG activity; ‘ceiling’ or ‘floor’ effects can distort normalized data but were not a confound in the study. Changes in EMG activity after the injection of either mineral oil or mustard oil were regarded as an increase if one or more EMG data points rose 2 standard deviations above the mean baseline level. The time period from the beginning of the injection to the increase in EMG activity was designated as the latency of the response, and the time period from the increase in EMG activity to recovery to the baseline level of EMG activity was designated as the duration of the response.

Fisher’s Exact Probability test (Fisher test) was used to compute the incidence of any EMG increase after the mustard oil injection.

**Table 1**

**EMG Increases Evoked by Mineral or Mustard Oil Injections Into the TMJ Region**

<table>
<thead>
<tr>
<th>Mineral oil (13)</th>
<th>Mustard oil (15)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Jaw muscles</td>
<td>Characteristic</td>
</tr>
<tr>
<td>Incidence</td>
<td>Latency (sec)</td>
</tr>
<tr>
<td><strong>Ip. digastric</strong></td>
<td><strong>33%</strong></td>
</tr>
<tr>
<td><strong>Ip. masseter</strong></td>
<td><strong>38%</strong></td>
</tr>
<tr>
<td><strong>Ct. masseter</strong></td>
<td><strong>31%</strong></td>
</tr>
</tbody>
</table>

**P < 0.01 and *** P < 0.001** (Fisher test) indicates that the incidence of the EMG increase evoked by mustard oil injection was significantly higher than that evoked by mineral oil injection.
the 13 animals with the incidence of any EMG increase after the mineral oil injection in the 13 animals tested. The Wilcoxon test was used for the comparison between the mean value representing the baseline level of the resting EMG activity (see above) and any data point of the postinjection periods. The Mann-Whitney test was used for statistical comparison of EMG activity between the animals tested only by mustard oil injection and the animals tested by mineral oil injection, and also used for statistical comparison of EMG activity between the animals receiving mustard oil injection following lidocaine administration and the animals receiving mineral oil injection following saline administration. A probability level of less than 0.05 (2-tailed) was regarded as significant. In this paper values are given as mean ± SD.

Results

Effects of mineral and mustard oil injections into the TMJ region on EMG activity of jaw muscles

For all 40 animals there was no significant variation in EMG activities in any jaw muscle during the initial 20-min period before any injections into the TMJ region. Injection of mineral oil produced a transient increase above the baseline level of the EMG activity in the ipsilateral digastric muscle in 3 animals, in the contralateral masseter muscle in 5 animals and in the contralateral masseter muscle in 4 of the 13 animals tested (see Table I). Compared with the baseline level, the mean peak amplitude of the mineral oil-evoked EMG activity of the ipsilateral digastric and masseter and contralateral masseter were 125 ± 72%, 131% ± 64%, and 121 ± 49%, respectively. No significant increase (P > 0.05, Wilcoxon test) above the baseline level of EMG activity evoked by the mineral oil injection could be demonstrated (Fig. 1).

Mustard oil injection alone (n = 15) evoked an increase of EMG activity in the ipsilateral digastric muscle of all the animals, in the ipsilateral masseter muscle of 13 animals (87%) and in the contralateral masseter muscle of 8 animals (53%) (e.g., Fig. 2 and Table I). The incidence of the evoked ipsilateral digastric EMG increase as well as the incidence of the ipsilateral masseter EMG increase were significantly (P < 0.001 and P < 0.01, Fisher test) higher than those evoked by the mineral oil injection (Table I). The mean peak amplitude of the mustard oil-evoked EMG activity of the ipsilateral digastric and masseter and contralateral masseter were 357 ± 253%, 345 ± 200%, and 113 ± 38%, respectively. The amplitude of EMG activity evoked by the mustard oil injection was significantly greater than the baseline EMG level for the ipsilateral digastric and masseter muscles (P < 0.01, Wilcoxon test) but not for the contralateral masseter muscle (Fig. 1) in these 15 animals, and also significantly (P < 0.01, Mann-Whitney test) greater than that evoked by the mineral oil injection in another 13 animals (Fig. 1). In the 7 animals receiving the mustard oil injection following the mineral oil injection into the same TMJ region, a significant (P < 0.05, Wilcoxon test) EMG increase above the baseline level was not only noted in the ipsilateral digastric and masseter muscles but also in the contralateral masseter muscle (Fig. 3). In these 7 animals, in contrast, mineral oil injected into the same TMJ region did not produce such effects.

The increased EMG activity evoked by mustard oil was reflected in 1 or 2 phases of enhanced activity (e.g., Fig. 2). The early increase of the EMG activity occurred soon after the mustard oil injection (mean latency: 3.5 ± 2.3 sec), peaked within 1 min, and then the increase subsided. The early increase in EMG activity lasted 7.5 ± 5.2 min. In each of the jaw muscles in which the mustard oil injection evoked an increase in EMG activity the early phase of the increased EMG activity was documented in all animals (Table I). The later phase, in contrast, had a lower frequency of occurrence: 47% for the ipsilateral digastric muscles,
53% for ipsilateral masseter and 47% for contralateral masseter muscles (Table I). The later increase of the EMG activity occurred at 14.6 ± 10.0 min after the mustard oil injection and lasted 14.3 ± 12.3 min (Table I).

Effects of mineral and mustard oil injections into the TMJ region on EMG activity of neck muscles

Mustard oil injection evoked an EMG increase in the ipsilateral trapezius and deep neck muscles of 3 animals and in the contralateral deep muscles of 5 animals. However, neither mineral nor mustard oil evoked any statistically significant increase in EMG of the neck muscles (P > 0.05, Wilcoxon test, see Fig. 3). Compared with the baseline level, the mean peak amplitude of the mineral and mustard oil-evoked EMG activity of the ipsilateral trapezius and deep neck muscles and contralateral deep neck muscles were 115 ± 68%, 178 ± 99%, 145 ± 79%, and 105 ± 58%, 134 ± 177%, 155 ± 88%, respectively.

Effect of pre-administration of lidocaine into the TMJ region

A transient EMG increase (Fig. 4) was evoked in some animals by the injection of either saline (4 of 6) or lidocaine (2 of 6). This transient EMG increase was not statistically significant and disappeared within 1 to 5 min after injection.

Mustard oil following the lidocaine injection in the same TMJ site did not produce any significant (P > 0.05, Wilcoxon test) increase of EMG activity. The mean peak amplitudes of the mustard oil-evoked EMG activity of the ipsilateral digastic and masseter and contralateral masseter were 108 ± 5.6%, 104 ± 4.5%, and 109% ± 61%, respectively. EMG activity associated with mustard oil injection in the animals receiving lidocaine pre-injection was significantly (P < 0.01 and 0.05, Mann-Whitney test) smaller than that in the animals receiving saline pre-injection (Fig. 4).

Inflammation in the TMJ region induced by mustard oil injection

Plasma extravasation shown by Evans Blue dye after mustard oil injection into the TMJ region could be seen in the TMJ capsule of all the animals and in the articular disc and synovial membranes of one-third of animals. The dotted region shown in Fig. 2B indicates the location of the extravasated dye. Postmortem examination.
Discussion

This study has provided documentation that application of a small-fibre excitant and inflammatory irritant to TMJ tissues results in an inflammatory response in the tissues and a sustained and reversible activation of jaw muscles. This activation may be manifested as 2 phases of increased EMG activity.

Our finding that the use of an O₂/N₂O/halothane mixture is associated with a stable preparation and stable resting EMG activity is consistent with our previous findings (Hu et al. 1993). Variability in the EMG responses to mustard oil injection were observed. However, it is presently unclear what factors may contribute to these EMG response variations. Under our experimental conditions, we did not find any relationship between the incidence or magnitude of the EMG response and the anaesthesia level, or the incidence or magnitude of the EMG response and location or extent of the Evans Blue extravasation induced by mustard oil.

A small transient increase in EMG activity sometimes occurred after injection of either mineral oil, saline or lidocaine. However, compared to the changes in EMG activity after mustard oil injection, EMG changes produced by injection of mineral oil, saline or lidocaine occurred only in a few animals and were much smaller in amplitude and shorter in duration. The short-lasting increase in EMG activity probably reflected the response to the volumetric stimulation of the tissue (Hu et al. 1993).

The time course of the increased EMG activity evoked by mustard oil injected into the TMJ region is consistent with that of the neck and jaw EMG increase evoked by mustard oil injected into deep paraspinal tissues (Hu et al. 1993), except that the latency of the EMG increase in the jaw muscles evoked by mustard oil injected into the TMJ region was shorter.

No significant EMG increase above the baseline EMG level after mustard oil injection into the TMJ region was found in the neck muscles. In contrast, injection of mustard oil into the deep paraspinal tissues induces a significant increase in EMG activity in neck as well as jaw muscles, and the increase of the EMG activity evoked by mustard oil injected into the paraspinal tissues is more prominent in the neck muscles (Hu et al. 1993). Such findings, together with the lack of neck and jaw EMG changes associated with the application of noxious pressure to the hind paw or the injection of mustard oil into the gastrocnemius-soleus muscle (Hu et al. 1993), suggest a segmental or somatotopic organization of EMG responses, i.e., the response is smaller or absent in muscles (e.g., neck muscles) more remote from the site of the irritant stimulus. This is also consistent with the clinical finding that trauma or inflammation of deep structures is more likely to manifest myofascial responses adjacent to the injury (Goodman 1988; Guilbaud 1988; Kehlet 1991).

Mustard oil-evoked EMG activity in the animals receiving pre-administration of lidocaine to the TMJ region was significantly blocked. This finding indicates that the EMG increase evoked by mustard oil injection into the TMJ region may be attributed to a centrally mediated reflex response. This view is further supported by findings that (1) the increased EMG activity was seen in muscles remote from or contralateral to the injection site, (2) the time course of the EMG increase is consistent with that of the mustard oil-evoked response in primary afferents (Reeh et al. 1986).
and nociceptive neurones in the trigeminal spinal tract nucleus (Hu et al. 1992; Yu et al. 1993a), and (3) application of the C-fibre neurotoxin capsaicin to afferent nerves can prevent the central neuronal excitatory effects of mustard oil (Woolf and Wall 1986).

Our previous study (Hu et al. 1993) has indicated that the effects of mustard oil sometimes included 2 phases of increased EMG activity. This feature is somewhat reminiscent to that of the effects of formalin injected into subcutaneous tissues (Dubuisson and Dennis 1977; Dickenson and Sullivan 1987; Hunskaar and Hole 1987). The early phase of the EMG increase is very likely a reflex response associated with an activation of the nociceptive afferents, as we have mentioned above; however, the mechanisms underlying the later phase of the response to the mustard oil injection remain unclear. Central neural mechanisms might contribute to the later phase, because the time course of mustard oil-evoked EMG increase does not parallel that of plasma extravasation in the TMJ tissues evoked by mustard oil: the increase in jaw EMG activity disappears within 30 min, while plasma extravasation peaks at 2 h and lasts more than 4 h (Yu et al. 1993b). It is also noteworthy that application of the opiate antagonist naloxone after the disappearance of the mustard oil-evoked EMG increase is associated with a significant increase in jaw EMG activity (Yu et al. 1993b) which suggests that deep injury and inflammation may also provoke and recruit an opiate-related suppressive mechanism. Furthermore, administration of the NMDA antagonist MK-801 can significantly reduce the jaw EMG response (Yu, Hu and Sessle, unpublished data) as well as the facilitation of spinal cord α-motoneurones and the flexion reflex (Woolf and Thompson 1991) induced by mustard oil. A 'central sensitization' or 'functional plasticity' of central nociceptive neurones that is initiated, but not necessarily maintained, by nociceptive primary afferent input (Wall 1985; Woolf 1989; Neugebauer and Schaible 1990; Dubner 1992; Hu et al. 1992) may be induced by mustard oil. Our present data suggest that activation of TMJ nociceptive afferents and changes in central neural plasticity induced by inputs from the TMJ nociceptive afferents may be related to clinically based concepts of muscle dysfunction such as muscle splitting, myospastic activity or trigger point in the case of TMJ disorders (Dubner et al. 1978; Storey 1979; Laskin et al. 1983; Bell 1986; Simons 1988).

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Administration of Opiate Antagonist Naloxone Induces Recurrence of Increased Jaw Muscle Activities Related to Inflammatory Irritant Application to Rat Temporomandibular Joint Region

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SUMMARY AND CONCLUSIONS

1. Our recent studies in rats have demonstrated that the small-fiber excitant and inflammatory irritant mustard oil injected into the temporomandibular joint (TMJ) region can evoke a sustained and reversible increase of electromyographic (EMG) activity in jaw muscles and an acute inflammatory response. The aim of the present study was to test if opioid mechanisms are involved in modulating the EMG increase evoked by mustard oil.

2. Mustard oil injected into the rat TMJ region evoked significant increases of jaw muscle EMG activity; the vehicle mineral oil had no such effect. The increased EMG activity lasted up to 20 min, and by 30 min after the mustard oil injection had returned to control (preinjection) levels, at which time administration of the opiate antagonist naloxone (1.3 mg/kg iv) induced a significant recurrence of the increase in EMG activity. This "rekindling" of EMG activity appeared at 3 to 5 min after the naloxone administration and lasted for 10 to 20 min. In contrast, naloxone administration in the animals receiving mineral oil injection into the TMJ region did not "rekindle" the EMG activity, nor did the administration of the peripherally acting opiate antagonist methylnaloxone or the vehicle of naloxone.

3. These findings reveal that the application of the opiate antagonist naloxone produces a recurrence of increased jaw muscle activity reflexively evoked by mustard oil injection into the rat TMJ region. They suggest that central opioid depressive mechanisms activated by the mustard oil-induced adherent barrage limit the duration of the evoked EMG changes.

INTRODUCTION

Our recent studies in rats have demonstrated that the small-fiber excitant and inflammatory irritant mustard oil (Handwerker and Reeh 1991; Jancso et al., 1977; Woolf and Wall 1986) injected into either deep paraspinal cervical tissues (Hu et al. 1993) or the temporomandibular joint (TMJ) region (Yu et al. 1994) can evoke a sustained and reversible increase in electromyographic (EMG) activity in neck add/or jaw muscles and an acute inflammatory characteristic by plasma extravasation lasting >4 hours (Haas et al. 1992; Hu et al. 1993; Yu et al. 1993, 1994). The present study was initiated to determine what mechanisms may be involved in limiting the increased EMG activity despite the presence of an ongoing inflammatory response. Because endogenous opioids have been implicated in the modulation of central and peripheral nociceptive transmission associated with injury or inflammation of peripheral tissues (Besouc and Chaouch 1987; Dubas and Ruda, 1992; Hargreaves and Joris 1993; Ramakrishnan and Banasri 1990; Stein 1993), opioid antagonists were used to test for the possible involvement of endogenous opioids in modulating the EMG response evoked by the injection of mustard oil into the TMJ region. Some of these data have been briefly presented in abstract form (Yu et al. 1993).

METHODS

The study was carried out in 24 male Sprague-Dawley rats (250-380 g) and involved methods that were in general similar to those previously described in detail (Hu et al. 1993; Yu et al. 1994); thus only a brief description follows. Under general anesthesia (1.4-1.5% O2, 3-3.5% N2O and 2-3% halothane), a cannula consisting of a needle (30 gauge) connected to a Hamilton-syringe (20 ml) with a polyethylene tube for injection was passed through the TMJ capsule into the TMJ on the left side and fixed in place. During EMG recordings (see below), the halothane concentration was maintained at 0.5-0.8% so that noxious pressure applied to the hindpaw could induce only a slight reflex flexion of the hindlimb. Heart rate, expired percent CO2, and body core temperature were continuously monitored and maintained at 330-340/111, 4-5/, and 37-37.5°C, respectively.

Bipolar recordings were made of the EMG activities of the left digastric and left and right masseter muscles, and EMG electrodes locations were confirmed by postmortem dissection. The EMG activity of each muscle was amplified (gain: ×10,000-30,000; bandwidth: 30-3,000 Hz) and displayed on oscilloscopes, and also was recorded online with a data acquisition and processing system (CED 1401, Plus hardware and software "Spike2"; CED, Cambridge; signal sampling rate was 2,000 Hz).

The 24 animals were divided into four groups of six animals each in which EMG activities were first recorded for 20 min before any injection to establish a baseline level of EMG activity. Then, in the animals of group 1, mustard oil (20% allylisothiocyanate [in 20 μl mineral oil; BDH, Poole]) was injected into the TMJ region, followed 30 min later by the administration of the opiate antagonist naloxone (1.3 mg/kg iv; Narcan, Du Pont, Scarborough); after the naloxone administration, the EMG activity was continuously observed for another 30 min. The animals of groups 2, 3, and 4 were tested with a protocol similar to that described above except that mineral oil (20 μl) was used instead of mustard oil for group 2, and instead of naloxone the peripherally acting (CHuyomi solution of 0.86% sodium chloride and 0.2% mixture of methyl- and 4 were tested with a protocol similar to that described above previously for another 30 min. The animals of groups 2, 3, and 4 were tested with a protocol similar to that described above except that mineral oil (20 μl) was used instead of mustard oil for group 2, and instead of naloxone the peripherally acting (CHuyomi solution of 0.86% sodium chloride and 0.2% mixture of methyl- and
Effects of mineral oil and mustard oil injections on EMG activity of jaw muscles

For all animals in the four groups there was no significant variation in EMG activities of any jaw muscle during the initial 20 min-period before any injections into the TMJ region (e.g., Fig. 1). The injection of mineral oil did not evoke any significant increases in EMG activity ($P > 0.05$, Wilcoxon test) although a transient increase above the baseline level of EMG activity occurred in the ipsilateral digastric muscle of one animal and the masseter muscles of three animals. This transient increase in the EMG activity disappeared within 5 min. The mean peak amplitudes of the mineral oil-evoked EMG activity of the ipsilateral digastric and masseter and contralateral masseter were 95 ± 16, 114 ± 18, and 117 ± 27%, respectively.

Compared with baseline EMG activity, the mustard oil injection evoked a significant increase of EMG activity in all the jaw muscles of the animals of groups 1, 3, and 4 ($P < 0.05$, Wilcoxon test). The mustard oil-evoked EMG changes in these muscles were also significantly greater than the mineral oil-evoked EMG changes in the same muscles of the animals of group 2 ($P < 0.05$, Mann-Whitney test, Fig. 1). The mean peak amplitudes of the mustard oil-evoked EMG activity of the ipsilateral digastric and masseter and contralateral masseter in the animals of group 1 were 273 ± 56, 340 ± 49, and 177 ± 23%, respectively. These values were 339 ± 94, 280 ± 116, and 138 ± 25%, respectively, in the animals of group 3, and 252 ± 153, 290 ± 46, and 133 ± 12%, respectively, in the animals of group 4. There was no significant difference in the mustard oil-evoked EMG changes among groups 1, 3, and 4 ($P > 0.05$, Mann-Whitney test, cf. Fig. 1). Consistent with our previous findings (Yu et al. 1994), the mustard oil-evoked increased EMG activity, which lasted up to 20 min, was reflected in two phases of enhanced activity in most (15/18) of the animals. The early increase of EMG activity occurred soon (4 ± 0.7 s) after the mustard oil injection and peaked within 1–2 min and then subsided. The later increase of the EMG activity occurred at 12.4 ± 1.2 min after the mustard oil injection and lasted 11.5 ± 1.4 min.

Effects of naloxone on EMG activity of jaw muscles

At 30 min after the mustard oil injection into the TMJ region, the EMG activity in each jaw muscle of animals in groups 1, 3, and 4 had returned to the baseline level, and there was no significant difference in the EMG activity of each jaw muscle between the animals receiving the mustard oil injection (groups 1, 3, and 4) and the animals receiving the mineral oil injection (group 2). The administration of naloxone in the latter group did not produce any significant EMG increase above baseline ($P > 0.05$, Wilcoxon test) although the EMG increase was noted in the ipsilateral digastric of three animals and masseter muscles of four animals. The administration of either methylnaloxone (group 3) or the vehicle for naloxone (group 4) also produced no significant EMG increase ($P > 0.05$, Wilcoxon test, Fig. 1), although an EMG increase was noted in the ipsilateral digastric of one animal and ipsilateral masseter muscles of two animals of group 3, and in the ipsilateral digastric of three animals and ipsilateral masseter muscles of two animals of group 4. In contrast, the naloxone administration in the animals receiving mustard oil injected into the TMJ region (group 1) induced a significant recurrence of the EMG activity ($P < 0.05$, Wilcoxon test) that lasted 10–20 min at 5 to 10 min after the naloxone administration, an EMG increase occurred in the ipsilateral digastric and masseter of all six animals and in the contralateral
Central inhibition may be enhanced during the development of inflammation (Schaible and Grubb 1993) and that central inhibitory pathways acting on neurons receiving nociceptive inputs from deep tissues are controlled by inhibitory pathways in the spinal cord (Besson and Banisnath 1990; Seale et al. 1981; Yu et al. 1991).

Administration of naloxone alone has been shown to enhance nociceptive motor responses induced by electrical stimulation (e.g., Catley et al. 1983; Goldfarb and Hu 1976), but our present data that naloxone administration to animals receiving the TMJ injection of mineral oil did not induce a recurrence of the increase in EMG activity indicate that the EMG increase is dependent on the previous occurrence of mustard oil-induced effects. The findings suggest that an opioid mechanism is triggered by the mustard oil and limits the EMG activity which can be "rekindled" by naloxone.

Both centrally and peripherally acting mechanisms of endogenous opioids have been implicated in the modulation of nociceptive transmission associated with injury or inflammation of peripheral tissues (see Besson and Chauoc 1987; Hu 1993; Ramahbadir and Banisnath 1990; Seale 1987). The time course of the "rekindling" effect that we documented in this study with a single administration of naloxone is consistent with that of the effects of naloxone in antagonizing these modulatory effects and neural and reflex responses to noxious stimuli (Ramahbadir and Banisnath 1990; Seale et al. 1981; Yu et al. 1991).

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OPIATE-RELATED EMG CHANGES


Electromyographic and Trigeminal Brainstem Neuronal Changes Associated with Inflammatory Irritation of Superficial and Deep Craniofacial Tissues in Rats

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INTRODUCTION

Sensitization of peripheral nociceptors is important in the increased pain sensitivity that develops in the skin immediately surrounding an injury site (so-called primary hyperalgesia, Meyer et al. 1985). However, recent studies also suggest that central as well as peripheral mechanisms are responsible for the hyperalgesia and allodynia that extends beyond the site of cutaneous injury, termed secondary hyperalgesia (LaMotte et al. 1991; Willis 1992). It has been proposed that second-order nociceptive neurons in the spinal dorsal horn are the major sites for this central sensitization in the spinal somatosensory system (Neugebauer and Schaible 1990; Woolf 1992; Cervero et al. 1993; Willis 1993). This central process can be produced by injury or inflammation of deep or visceral tissues and is related to secondary hyperalgesia and manifestations of pain spread and referral associated with deep injury or inflammation (Woolf and Wall 1986; Cook et al. 1987; Hoheisel and Mense
Inflammatory pain may be associated with central opioid changes (Lombard and Besson 1989; Dubner and Ruda 1992), and the initiation and maintenance of the central sensitization process appears to involve N-methyl-D-aspartate (NMDA) receptor mechanisms (Dubner and Ruda 1992; Woolf and Thompson 1992). Recently, nitric oxide (NO) has been suggested as a second messenger for these NMDA receptor mechanisms (Meller and Gebhart 1993).

Our recent studies have sought to determine whether a similar process of central sensitization exists in the trigeminal (V) brainstem sensory nuclear complex and what neurochemical mechanisms may be involved in this process. Our single-unit recording studies have focused on the V subnucleus caudalis because it has been particularly implicated in craniofacial deep pain mechanisms (Sessle and Hu 1991; Hu et al. 1992; Hu 1993; Sessle et al. 1993a,b; Yu et al. 1993b). These studies have been supplemented by studies (Hu et al. 1993; Yu et al. 1993a,c) of possible neuromuscular changes that may accompany neuroplastic changes in the V brainstem complex. We have used mustard oil as a means to produce irritation and inflammation of deep tissues to study the central sensitization process in the V brainstem complex. Mustard oil is an excitant of small-fiber afferents (Reeh et al. 1986; Woolf and Wall 1986; Handwerker et al. 1991) and also acts as an inflammatory irritant (Jancso et al. 1977; Handwerker and Reeh 1991).

METHODS

Experimentation on all animals conformed to the regulations of the Canadian Council on Animal Care and the Ontario Animals for Research Act and was approved by the University of Toronto Animal Care Committee. Single brainstem units were recorded in the V subnuclei caudalis (1-3 mm behind the obex) or oralis of anesthetized rats paralyzed with a muscle relaxant. Methods and procedures are outlined in detail in our published papers (Hu 1990; Hu et al. 1992; Yu et al. 1993b). A wide range of mechanical, electrical, and thermal stimuli was used to classify each neuron and delineate its mechanoreceptive field (RF) properties. Low-threshold mechanical stimuli included a camel hair brush, and high-threshold mechanical stimuli were applied by watchmaker forceps (pinch). Noxious radiant heat (51-53°C) and bipolar electrical stimulation (2 ms, < 5 mA) were also used for assessment of cutaneous nociceptive properties. A deep RF was documented if no response could be evoked by this wide range of cutaneous stimuli and if a clear response occurred from deep pressure applied by a blunt probe to muscle, bone, tendon, or joint. Furthermore, each neuron functionally classified as a nociceptive skin/mucosa and deep convergent neuron also was classified as either wide...
dynamic range (WDR) or nociceptive-specific (NS) on the basis of its cutaneous RF properties. In addition, we also tested the excitability of the neuron’s responsiveness to electrical stimulation of A-fiber and C-fiber cutaneous afferent inputs. Our aim was to determine whether there are differential neuroplastic effects of cutaneous and deep stimuli on the RF properties of these nociceptive brainstem neurons. Either mineral oil (vehicle for mustard oil) or mustard oil (allyl-isothiocyanate, BDH, Poole) was injected into the deep masseter or tongue (5 mm behind the tongue tip, 3 mm lateral to the tongue midline, and 2 mm below the tongue surface) muscles. Alternatively, either mineral oil or mustard oil also was applied with a cotton swab to the skin 10 mm distal to the cutaneous RF border but overlapping the deep RF of the neuron.

We also conducted a series of electromyographic (EMG) studies in anesthetized, unparalyzed rats to determine if the EMG activity of the jaw and neck muscles can be influenced by mustard oil application to craniofacial tissues. Rats were lightly anesthetized with a mixture of one-third oxygen, two-thirds nitrous oxide, and 0.5–0.8% halothane to achieve a stable plane of anesthesia; noxious pressure applied to the hindpaw could induce a slight flexion reflex response of the hindlimb. EMG activity was recorded by bipolar EMG electrodes (36–40 gauge, single-stranded, Teflon-coated stainless-steel wire; interpolar distance 0.5 cm; exposed tips 0.5 mm) inserted into the ipsilateral digastric and bilateral masseter muscles, and for some experiments, also into the ipsilateral trapezius and bilateral deep neck muscles. Mustard oil (or vehicle) was injected into neck, temporomandibular (TM) joint, or dental tissues. For the neck injection, mustard oil (20%, 20 μl in mineral oil) or mineral oil (20 μl) was injected by a needle into deep paraspinal muscles (Hu et al. 1993). For the TM injection, a preimplanted two-needle assembly with either mustard oil (20%, 20 μl in mineral oil) or mineral oil was placed into the TM region before the EMG recording and injected according to a predetermined schedule (Yu et al. 1992). For the dental injection, mustard oil (98%, 0.25 μl) or mineral oil was applied to a cavity preparation made in the maxillary first molar tooth (Sunakawa et al. 1993a,b).

Inflammation was assessed by the plasma extravasation of Evans’ Blue (EB) dye bound to plasma protein (Haas et al. 1992); the EB dye concentration was assessed spectrophotometrically. We also conducted histologic examination and morphometric assessment of the injected tissues to reveal other indicators of inflammation (e.g., congestion and invasion of polymorphonuclear leukocytes; Haas et al. 1992).

The EMG activity of each muscle was amplified (gain: 1000–5000X; bandwidth 30–3000 Hz) and displayed on oscilloscopes, and also recorded on a FM tape recorder, for one minute every 3–5 minutes before the vehicle
injection. Immediately after either the vehicle or mustard oil injection, the EMG activity was continuously recorded for 3–5 minutes and thereafter for one minute every 3–5 minutes. The EMG data recorded into the computer was processed off-line with the program Spike2 (CED, Cambridge). The recorded EMG signals were rectified and integrated every second and every 60 seconds to measure the duration of any EMG response induced by mustard oil or vehicle. The integrated EMG area for each 60-second period was used to express EMG activity. The insertion of the injection needle into the deep tissues could itself sometimes induce a small and transient (5–10 second) increase in EMG activity, so we did not begin the injection of the mineral oil or mustard oil until the EMG activity had returned to preinsertion levels. The data analysis did not include the integrated EMG activity associated with any such transient response evoked by insertion of the injection needle.

Before vehicle injection, none of the muscles revealed any variation in EMG activity greater than two standard deviations above the mean EMG amplitude level, so we pooled all preinjection data points for each muscle in each animal to produce a mean value that represented the preinjection level of EMG activity for the muscle in each animal. All postinjection data points were normalized relative to this mean value and expressed as a percentage value of the mean preinjection level of EMG activity. Changes in EMG activity after the injection of either mineral oil or mustard oil were regarded as an increase if one or more EMG data points rose two standard deviations above this mean baseline level.

We also initiated preliminary experiments to test the possible involvement of excitatory amino acid receptor processes in these mustard oil-induced EMG changes: an NMDA noncompetitive inhibitor (MK-801, at a dose of 0.5 mg/kg) and a NO synthase inhibitor (L-NAME, at doses of 10, 30, and 100 mg/kg) were systemically injected 5–10 minutes before mustard oil application. In addition, to test if endogenous opioid receptor mechanisms also might be involved, we administered the opioid antagonist naloxone (1.2–1.3 mg/kg, iv.) 30 minutes after mustard oil application.

RESULTS AND DISCUSSION

CHANGES IN NOCICEPTIVE NEURONS

The injection of mustard oil into the deep masseter muscle enhanced the C-fiber evoked responses of 17 of 39 (44%) nociceptive neurons tested in the rat's subnuclei caudalis and oralis to electrical stimuli applied to their cutane-
C-fiber strength electrical stimulation of hypoglossal muscle afferents for about one minute also markedly enhanced the excitability of WDR and NS neurons tested in the subnucleus caudalis (Sessle et al. 1993a). These changes were reversible and were associated with an expansion of the cutaneous RF of the nociceptive neurons, a reduction in their mechanical threshold for activation, and an increase in spontaneous firing. The neuronal changes appeared within 2–5 minutes of the mustard oil injection and lasted for 20–30 minutes; sometimes two phases of mustard oil–induced increased excitability were noted. As we have discussed (Hu et al. 1992; Sessle and Hu 1991; Sessle et al. 1993a), these effects of deep afferent inputs on the cutaneous RF properties of the V nociceptive neurons may relate to neural processes underlying the spread and referral of pain, particularly to cutaneous facial sites. These mechanisms may also contribute to the tenderness and hyperalgesia of superficial tissues that are characteristic of many cases involving injury to deep craniofacial tissues.

The deep application of mustard oil could produce even greater enhancement of the deep RF of caudalis nociceptive neurons. A total of 31 caudalis neurons were functionally identified as WDR or NS neurons receiving convergent cutaneous and deep afferent inputs. We tested the effects of mustard oil applied to either tongue muscle or facial skin on their cutaneous and deep RFs (Yu et al. 1993b) and found that the expression of neuroplasticity depended on the site of application of this irritant. Mustard oil injected into the tongue muscle produced a statistically significant and reversible expansion of both the cutaneous and deep RFs (17 of 23 neurons tested). In contrast, application of mustard oil to facial skin resulted in a significant and reversible expansion of the cutaneous RF (7 out of 8 neurons tested), but no deep RF expansion occurred in the 8 neurons tested.

These differential effects of deep and cutaneous afferent inputs may relate to differences in the central sensitization effects between the two types of inputs. While there are differences in neuropeptide content between cutaneous and muscle C-fiber afferents (McMahon et al. 1984), the consequence of these differences on the excitability of central neurons is still unclear. Woolf and Wall (1986), however, have reported that deep (muscle) noxious stimulation at C-fiber strength in spinalized rats produces greater facilitatory effects on the flexion reflex than does cutaneous noxious stimulation at C-fiber strength. Thus, the central effects of activating C-fibers from deep tissue appear to be different from cutaneous C-fiber activation. The time course of the central neuronal changes is much longer than the 3–4 minutes of mustard oil–induced excitation reported with cutaneous primary afferents (Handwerker et al. 1991), thus supporting the view that a central sensitization process contributes to the neuronal changes.
We first conducted a neck EMG study (Hu et al. 1993) to determine if the EMG activity of the jaw and neck muscles could be influenced by the injection of mustard oil into deep paraspinal tissues surrounding the C1 to C3 vertebrae. Vehicle injection (mineral oil, n = 4 rats) did not produce any significant change in EMG activity. In contrast, mustard oil injection (n = 13 rats) induced a statistically significant and reversible increase in EMG activity of the bilateral neck and ipsilateral jaw muscles that was most prominent in the ipsilateral deep neck muscles (Fig. 1A). The increased EMG response evoked by mustard oil was frequently reflected in two phases of enhanced activity. The early phase was initiated immediately (< 20 seconds) following the mustard oil injection and lasted 3 minutes; the second phase occurred 10 to 15 minutes later and lasted 10–15 minutes. The time course of these EMG changes is thus comparable to that (see above) induced by mustard oil in caudalis or oralis neurons (Hu et al. 1992; Yu et al. 1993b), many of which serve as reflex interneurons in jaw and neck motor pathways (Dubner et al. 1978; Sessle and Hu 1991). These findings support the view that the EMG changes are associated with a central sensitization process.

The injection of mustard oil (but not the vehicle, mineral oil) induced an inflammatory reaction in the tissues between the fibers of the rectus capitis posterior muscle that was characterized by congestion and invasion of polymorphonuclear leukocytes (Hu et al. 1993). Plasma extravasation as reflected in increased EB dye content of the tissues also occurred.

We also studied a TM inflammatory model by injecting mustard oil into the periarticular TM tissues of anesthetized rats (Haas et al. 1992; Yu et al. 1992, 1993a). Inflammation was assessed by the plasma extravasation of EB dye, and a spectrophotometric analysis revealed a significantly greater ($P < 0.001$) dye concentration in the injected tissues compared to the contralateral untreated side. A morphometric analysis also revealed a significant increase ($P < 0.001$) in polymorphonuclear leucocyte infiltration compared to the contralateral side (Haas et al. 1992; Yu et al. 1993a). A time-course study of the effect of mustard oil on EB dye extravasation revealed a gradually increasing extravasation of EB dye that was maximal at two hours after administration of mustard oil. In eight anesthetized rats studied to date (Yu et al. 1992), EMG activity was significantly increased above baseline levels in both ipsilateral digastric and masseter muscles after mustard oil injection into the TM region but not after vehicle injection (Fig. 1B). Mustard oil injection into the TM region provoked a strong early phase of EMG increase that gradually subsided. These effects lasted for several minutes, in contrast to the more
Fig. 1. Changes in EMG activity induced by application of mineral oil or mustard oil to: A: the deep paraspinal tissues surrounding the C1-3 vertebrae (mineral oil, n = 4 rats; mustard oil, n = 13 rats); B: the temporomandibular (TM) region (mineral oil, n = 6 rats; mustard oil, n = 8 rats); and C: the maxillary first molar (mineral oil, n = 6 rats; mustard oil, n = 8 rats). The mean (± SD) data shown are normalized values relative to the mean value of the preinjection level of EMG activity (= 100%) in each rat. Changes in EMG activity of rats receiving mustard oil or mineral oil are indicated by filled or unfilled circles, respectively. * P < 0.05 and ** P < 0.01 (Wilcoxon test) indicate significant difference between preinjection and postinjection levels of EMG activity. Only one prominent EMG increase induced by mustard oil injected into each tissue has been illustrated. Mineral oil applied to deep paraspinal tissues of the neck, the TM joint region, or the tooth did not induce any significant EMG increase. In contrast, mustard oil induced significant increases in EMG activity of the neck muscles (bilateral deep neck muscles, ipsilateral trapezius) or jaw muscles (ipsilateral digastric and masseter muscles). The opiate antagonist naloxone (1.2-1.3 mg/kg, iv.) was injected in animals receiving mustard oil or mineral oil application to TM region or tooth. When it was administered after the disappearance of the mustard oil-induced EMG response, another significant increase in EMG activity occurred and lasted for 10 minutes. In contrast, administration of naloxone in rats receiving mineral oil did not produce any significant increase in EMG activity (B and C).
transient EMG effects observed in cats when other algesic compounds such as bradykinin and histamine are applied to TM tissues (Broton and Sessle 1988). The findings suggest that increased EMG activity of jaw muscles may be associated with TM inflammatory conditions and involve a central sensitization process. Moreover, local anesthetic (lidocaine, 1.4 mg/kg) injected into the TM region before the mustard oil injection blocked the EMG activity (but not plasma extravasation) (Yu et al. 1993a). Mustard oil is a small-fiber excitant (see above), so this finding points to the necessity of small-fiber afferent activation in reflexively inducing the central sensitization process after deep injury.

Additional preliminary data also showed that the enhanced EMG activity induced by mustard oil could be blocked by the systemic administration of either the noncompetitive NMDA receptor antagonist, MK-801 (0.5 mg/kg, iv., n = 16 rats), or the NO synthase inhibitor, (L-NAME, 30 and 100 mg/kg but not 10 mg/kg and vehicle injection, iv., n = 24 rats). We have also recently found that MK-801 (0.1 mg/kg, n=5; 0.01 mg/kg, n=5) injected into the third ventricle can also block the evoked EMG activity. These findings suggest that NO and NMDA receptor mechanisms may be involved in the central V neuroplastic changes that we have documented and are consistent with other studies (Dubner and Ruda 1992; Woolf and Thompson 1992; Meller and Gebhart 1993) pointing to their involvement in central sensitization.

The involvement of opioids in the modulation of pain is well known (Basbaum and Fields 1984; Hammond 1986; Sessle 1987; Dubner and Ruda 1992). Lombard and Besson (1989) tested opioid involvement in inflammatory-related pain and showed that the opioid antagonist naloxone (1 mg/kg, iv.) could induce a significant increase in the spontaneous firing as well as enhancement of C-fiber-related responses of dorsal horn nociceptive neurons in polyarthritic rats. We tested the possible involvement of endogenous opioid mechanisms in the increased EMG activities induced by the TM injection of mustard oil by administering naloxone (1.3 mg/kg, iv.) 30 minutes after the mustard oil injection to see if it “rekindled” the EMG activity that had returned to baseline levels by this time (Fig. 1B). The resulting increase of EMG activity suggests that naloxone may have counteracted an opioid suppressive mechanism triggered by the mustard oil–induced inflammatory process or deep afferent input activation. However, naloxone (1.3 mg/kg, iv.), injected 5 minutes before the application of mustard oil did cause significant enhancement of EMG activity induced by the mustard oil injection but had no effect on the extravasation, and did not cause any EMG changes when it was applied either before or after mineral oil injection into the TM region (Yu et al. 1993a).
Another model of the central sensitization process has recently been tested by applying mustard oil directly into the tooth (Sunakawa et al. 1993a,b). The rationale for these experiments is based on the notion that some types of toothache may be associated with activation of pulpal C-fiber afferents, and the possibility that this pain also may reflect a central sensitization process in tooth pulp-related V brainstem sensory and motor neuronal pathways. Mustard oil (n = 8 rats) applied to the first maxillary molar (Sunakawa et al. 1993a,b) evoked a significant increase in EMG activity, often with early and late phases, in the ipsilateral masseter muscle (Fig. 1C). No significant EMG changes occurred following mineral oil application to the tooth (n = 6), or in pulpectomized rats following mustard oil application to the tooth (n = 4). In accordance with our findings outlined above, naloxone (1.2 mg/kg, iv., 30 minutes after mustard oil application to the tooth) also could induce a late enhancement of EMG activity (Fig. 1C).

CONCLUSION

This chapter provides documentation that stimulation of different craniofacial sites as well as cervical paraspinal tissues by an inflammatory irritant results in an inflammatory response in these tissues and in a sustained and reversible activation of jaw or neck muscles. The enhanced EMG activity evoked from the various tissues injected with mustard oil and its abolition by local anesthesia of the injected tissues demonstrate that these enhanced EMG effects can be attributed to reflex responses involving segmental as well as suprasegmental pathways, as opposed to direct effects on muscles by the peripheral spread of mustard oil. In neck injection experiments, activation of neck muscles was more prominent following the mustard oil injection, whereas activation of jaw muscles was more prominent following the mustard oil injection into TM or dental tissues. These results suggest a segmental or somatotopic organization of EMG responses in the head and neck region. The latency and time course of the mustard oil–induced increases in EMG activity are also comparable to the temporal features of the mustard oil–induced increased excitability of sensory neurons in the V spinal tract nucleus, which are the sites of reflex interneurons involved in craniofacial and cervical reflex pathways as well as projection neurons contributing to ascending somatosensory pathways (Dubner et al. 1978; Rossignol et al. 1988; Olsson and Westberg 1989). However, the time courses of both the increased EMG activity and the neuronal hyperexcitability induced by mustard oil (Hu et al. 1992; Yu et al. 1992, 1993a,c) are different from the temporal features of the inflammation process, which may last more than two hours after the application of mustard oil.
oil (Reeh et al. 1986; Haas et al. 1992). One factor limiting the duration of the V neuronal and EMG changes may be opioid depressive mechanisms induced by the mustard oil-evoked afferent input. Our data of "rekindling" of EMG activity by the opiate antagonist naloxone demonstrate the involvement of opioid mechanisms in the neuroplastic changes associated with deep tissue injury. Our preliminary EMG data also have raised the possibility that NMDA receptor and NO mechanisms may be involved in the mustard oil-induced changes. These EMG findings are supported by preliminary data obtained from caudalis nociceptive neurons where three of five skin and deep convergent nociceptive caudalis neurons demonstrated a second (i.e., "rekindling") RF expansion following naloxone administration after the initial mustard oil-induced RF expansion had subsided.

In addition to their significance in revealing V central sensitization and possible neurochemical mechanisms involved in the neuroplastic changes, these findings also have clinical significance. They provide insights into how increased cutaneous sensitivity, spread and referral of pain, and associated neuromuscular changes may occur after craniofacial injury or inflammation. The findings also point out the need for therapeutic approaches to reduce nociceptive inputs to the CNS (e.g., with local anesthesia) or their effects (e.g., with opiate drugs) that can potentially prolong posttraumatic craniofacial pain by the central neuroplastic changes that they may induce (e.g., Dubner 1991; Woolf 1992; Mense 1993).

ACKNOWLEDGMENTS

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Spinal manipulation and headaches: An update

The role of the cervical spine and of spinal manipulation in headache remain unresolved. This article will review the existing literature in three important areas which relate to these controversies, namely: (1) studies of spinal manipulation for headache; (2) mechanisms of cervicogenic head pain; and (3) components of cervicogenic dysfunction. The main thesis of this article is that the cervical spine plays an important role in benign headache and that evidence exists supporting chiropractic spinal manipulation as a beneficial therapy. Key words: headache; chiropractic; tension headache; neck; manipulation, orthopedic; algorithm

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Algorithms appear in Appendix C.

The role of the cervical spine in headache continues to be an unresolved controversy. In 1988, a spectrum of headache subtypes that might have some kind of cervicogenic involvement was defined1 (Fig 1). The spectrum ranged from "tension headache with neck muscle pain" through "cervicogenic headache" (defined in chiropractic terms as symptomatic head pain and cephalic dysfunction caused by spinal joint dysfunction), to a proposed "vertebrogenic migraine." Coincidentally, in 1988 the International Headache Society (IHS)2 published its classification of headaches and for the first time recognized a headache subtype known as "cervicogenic headache" (CH). The definition of CH according to the IHS classification is shown in Table 1. This headache owes much of its definition to the work of Sjaastad and colleagues,3-5 which first appeared in print in 1983.

In a subsequent publication,7 this narrow definition of CH was challenged and contrasted with the characteristic headache subtypes that chiropractic, manual medicine, osteopathic, and physiotherapeutic experts had addressed in the literature spanning the greater part of this century. This also contrasted the IHS version of CH with the headache subtypes that had been included in the clinical studies of the outcome of spinal manipulation—again, by the same practitioners. In these studies of both tension and migraine headaches (definitely different from the narrow IHS features of the CH category), the results of manipulation for (presumably) some cervical spine dysfunction ranged from fair to excellent.
This leaves a quandary, necessitating answers to the following questions:

- Is there a putative cervical component in tension and migraine headaches?
- Is it similar to or different from the dysfunction in CH?
- Do the results of manipulation studies automatically imply the existence of an etiologic cervical component to these forms of benign headaches?

Alternately, is there one kind of cervical component that might contribute to, or manifest as, different forms of headache experiences and thus be diagnostically labeled as different forms of headache categories? This article will explore this quandary, with a clear bias toward accepting this alternate hypothesis. This will be done by first briefly reviewing the results of clinical studies of spinal manipulation for headaches. Then the current research on pain mechanisms in CH will be reviewed. Finally, the literature on findings of cervicogenic dysfunction in headache groups that are clearly not IHS-CH will be reviewed, and the argument for the alternative hypothesis will be summarized—that is, that subluxation/dysfunction of the spine makes a significant contribution to the etiology of a number of benign forms of headache.

**HEADACHE AND SPINAL MANIPULATION**

In 1991 a review article of all studies of manipulation for the treatment of headaches summarized work up to 1988. This work is outlined in Tables 2 and 3. Since 1988, five treatment studies employing manipulation have appeared in the scientific literature. In 1989 Stodolny and Chmielewski reported on 31 subjects (24 females, 7 males; average age, 48 years) with "cervical migraine." They employed tests for intersegmental blockage and found that 100% of subjects demonstrated spinal motion segment blockage at C0–1, 75% at C7-T1, and 25% between C1–2 and C-3 (see "Cervicogenic Dysfunction"). The treatments consisted of two to three manual therapy sessions, largely employing manipulative techniques. Reported results at the end of 7 days included:

- complete relief of headache in 75% of subjects,
- an average increase of cervical range of motion of 9 degrees (statistically significant).
Table 1. Diagnostic criteria for CH

1. Pain localized to neck and suboccipital region. May project to forehead, orbital region, temples, vertex, or ears. 
2. Pain is precipitated or aggravated by special neck movements or sustained neck postures. 
3. At least one of the following is present: 
   a. Resistance or limitation of passive neck movements. 
   b. Changes in neck muscle contour, texture, tone, or response to passive stretching or contraction. 
   c. Abnormal tenderness of neck muscles. 
4. Radiologic examination reveals at least one of the following: 
   a. Movement abnormalities in flexion-extension. 
   b. Abnormal posture. 
   c. Fractures, congenital abnormalities, bone tumors, rheumatoid arthritis, or other distinct pathology (not spondylosis or osteochondrosis).


- reduced fixations in 28 of 31 subjects, and 
- greatly reduced reports of dizziness in subjects.

There was no further follow-up reported. No control comparison or blinding of the assessors was included in the protocol, so these results must be interpreted cautiously, all the more so because of the short treatment period.

In 1990 Jensen et al reported on 19 subjects with posttraumatic headache. Seven males and 12 females with an average age of 31 years and an average duration of complaint of 12 months were randomly allocated to either manual therapy (n = 10) or ice packs (n = 9). In the manual therapy group, two treatments were rendered within 1 week; these consisted of mobilization and manipulation to the upper and lower cervical spine and the upper thoracic spine. In the control group, two sessions of ice treatment to the neck were rendered, lasting 15 to 20 minutes each.

The outcomes measured were pain score, regional range of motion, segmental range of motion, and the presence of accompanying symptoms (dizziness, visual disturbances, ear pain).

In 1990 Jensen et al reported on 19 subjects with posttraumatic headache. Seven males and 12 females with an average age of 31 years and an average duration of complaint of 12 months were randomly allocated to either manual therapy (n = 10) or ice packs (n = 9). In the manual therapy group, two treatments were rendered within 1 week; these consisted of mobilization and manipulation to the upper and lower cervical spine and the upper thoracic spine. In the control group, two sessions of ice treatment to the neck were rendered, lasting 15 to 20 minutes each.

The first outcome interval was 2 weeks after treatment. At this point there was a 57% reduction in pain intensity measurements and a 64% reduction in analgesic use in the manual therapy group as compared with controls. This was statistically significant. At 5 weeks there was also a difference favoring the manual therapy group, but this difference did not reach statistical significance.

There was a significant positive correlation between pain index and (1) the frequency of visual disturbances (r = .65; P

Table 2. Studies of manipulation for tension headaches

<table>
<thead>
<tr>
<th>Study</th>
<th>Treaters</th>
<th>Design</th>
<th>No. studied</th>
<th>Headache type</th>
<th>Results</th>
</tr>
</thead>
<tbody>
<tr>
<td>Lewit (1971)</td>
<td>A</td>
<td>CS</td>
<td>41</td>
<td>TT</td>
<td>Manipulation most effective</td>
</tr>
<tr>
<td>Lewit (1971)</td>
<td>A</td>
<td>CS</td>
<td>93</td>
<td>TT</td>
<td>Manipulation most effective</td>
</tr>
<tr>
<td>Vernon (1982)</td>
<td>B</td>
<td>CS</td>
<td>33</td>
<td>TT</td>
<td>85%-90% success; pre-post differences statistically significant</td>
</tr>
<tr>
<td>Jirout (1985)</td>
<td>A</td>
<td>CS</td>
<td>200</td>
<td>TT</td>
<td>80% success</td>
</tr>
<tr>
<td>Droz and Crot (1985)</td>
<td>B</td>
<td>CS</td>
<td>332</td>
<td>Occ</td>
<td>75% success</td>
</tr>
<tr>
<td>Turk and Ratkolb (1987)</td>
<td>A</td>
<td>COH</td>
<td>100</td>
<td>TT</td>
<td>Significant difference, manipulation vs ice</td>
</tr>
<tr>
<td>Whittingham et al (1994)</td>
<td>B</td>
<td>COH</td>
<td>26</td>
<td>TT</td>
<td>60%-77% headache reduction; pre-post differences statistically significant</td>
</tr>
<tr>
<td>Mootz et al (1994)</td>
<td>B</td>
<td>COH</td>
<td>11</td>
<td>TT</td>
<td>Statistically significant reduction of headache frequency and duration after chiropractic care</td>
</tr>
</tbody>
</table>

Table 3. Studies of manipulation for migraine headaches

<table>
<thead>
<tr>
<th>Study</th>
<th>Treaters</th>
<th>Design</th>
<th>No. studied</th>
<th>Headache type</th>
<th>Results</th>
</tr>
</thead>
<tbody>
<tr>
<td>Wight (1987)</td>
<td>B</td>
<td>CS</td>
<td>57</td>
<td>Mig</td>
<td>75% success</td>
</tr>
<tr>
<td>Stodolny and Chmielewski (1989)</td>
<td>A</td>
<td>COH</td>
<td>31</td>
<td>Mig</td>
<td>75% success</td>
</tr>
</tbody>
</table>

A, Medical doctor; B, chiropractor; CS, case series; COH, cohort; RCT, randomized controlled trial; Mig, migraine; Source: Vernon HT. Spinal manipulation and headaches of cervical origin: a review of literature and presentation of cases. J Manipulative Med. 1991:6:73-79.

< .02) and (2) all three associated symptoms (including dizziness) together (r = .70; P < .01).

Although this study employed a small sample size in an unblinded trial, there was a clear difference favoring manual therapy in reducing headache pain and associated symptoms. This is even more remarkable given that only a short-course and short "dosage" of manual therapy were provided.

In 1995 Boline et al reported on their randomized comparative trial of chiropractic manipulation and the antidepressant drug amitriptyline in the treatment of "muscle contraction headache." This trial included 126 subjects with an average headache duration of 6 years. After the 6-week treatment phase, subjects in both treatment groups demonstrated clinically and statistically significant reductions in headache activity. Neither group had statistically greater reductions than the other. However, at the end of a 6-week no-treatment follow-up phase, subjects treated with chiropractic manipulation had significantly less headache activity than those receiving drug therapy, possibly indicating that the benefit obtained by chiropractic manipulation was more durable and may have corrected some of the underlying causative (but still poorly understood) mechanisms for the kind of headaches suffered by these subjects.

In 1994 Whittingham et al reported on 26 subjects (16 females, 10 males) with chronic headache and upper cervical dysfunction. A self-controlled time series design was employed to study the effect of four manipulative sessions of a toggle recoil technique during a 2-week treatment period. After 2 weeks, they reported a 62% reduction in frequency, a 77% reduction in duration, and a 60% reduction in severity of headaches. These results are consistent with those of other noncontrolled trials.

This study is of particular interest to chiropractors because of the use of a specific manipulative technique directed toward the upper cervical spine. That this extent of improvement —50% to 77% reduction in headache activity — was achieved in only four treatments may indicate a particular effectiveness for this manipulative approach. Of course, controlled trials are needed to confirm this speculation.

In 1994 Mootz et al reported on 11 males ranging in age from 18 to 40 years. These subjects suffered from "mild to severe head and/or neck pain," with at least four episodes per month (ie, chronic episodic tension headache). The study design was a prospective case series with a 2-week nontreatment baseline and an 8-week treatment period of 16 interventions. The treatment protocol consisted of spinal manipulation to the cervical spine supplemented by myofascial trigger point therapy and moist heat packs.

Mootz et al reported statistically significant reductions in headache frequency (from 6.4 per 2 weeks to 3.1 per 2 weeks) and duration (from 6.7 h to 3.9 h). A strong trend toward a reduction in headache pain severity (5.05 to 3.37 out of 10) was also noted.

When these data are combined with those of the previous studies, it can be concluded that spinal manipulation appears to provide clinically significant levels of relief for benign types of headache in patients who receive the treatment. Unfortunately, the dearth of controlled studies prohibits any firm conclusions regarding the efficacy of treatment. In addition, it is virtually impossible to assert the conclusion that relief of headache by manipulation proves the hypothesis that spinal dysfunction (presumably the target of the manipulative treatment) causes or is associated with these forms of headache. Although correction of the causative agent is one acceptable hypothesis to explain these results, other equally plausible explanations exist, including the strong placebo effect that is typically generated in headache patients, the natural history effect, and selection bias. Once again, controlled studies are required for theoretical and pathophysiologic conclusions to be drawn.

MECHANISMS OF PAIN IN CERVICOGENIC HEADACHE

In 1988 Vernon presented a vertebrogenic model of headache that contained four categories:
1. **Extrasegmental:** Referring to the long regional myofascial structures such as the trapezius and long extensor muscles, the ligamentum nuchae, and the interface between the occipitofrontalis muscle and these regional cervicothoracic structures.

2. **Intersegmental:** Referring to the three joint complexes of C2-3-4 and the articulations of C0-1-2, with their ligaments and deep intersegmental muscles.

3. **Infrasegmental:** Referring to the nerve structures in and around the intervertebral foraminae and in the cervical spine near the lateral portions of the vertebrae (i.e., the sympathetic trunk; the vertebral nerve; the C-2 dorsal root ganglion; the greater, lesser, and third occipital nerves; and the sensory roots of C-1).

4. **Intrasegmental:** Referring to the spinal cord and medullary dorsal horn with the nucleus subcaudalis of the trigeminal nerve.

Much has been learned about these mechanisms since 1988, centering on the pain pathways of the upper cervical cord and their convergence on cells in the nucleus subcaudalis, a mechanism Bogduk\(^1\) calls the “trigemino-cervical nucleus” (Fig 2).

Sessle and colleagues\(^{14-21}\) have extensively investigated these cephalic and craniofacial pain pathways, particularly with regard to the effect of deep tissue (joint, muscle, and visceral) nociception on trigeminal sensorimotor mechanisms. Their work extends previous investigations of spinal nociceptive mechanisms\(^{22,23}\) and provides a replication of many similar mechanisms underlying pain transmission in the spinal cord itself. Specifically, they have identified pain-transmitting neurons in laminae I, II, V, and VI of the dorsal medullary horn and have classified these cells according to their response properties as (1) low-threshold mechanoreceptors (LTM) (non-noxious); (2) nociceptor-specific (NS) receptors; and (3) wide dynamic range (WDR) receptors, which respond to both noxious and non-noxious stimuli. Furthermore, they have identified the receptive fields of these central neurons as skin only (S), (68%); deep inputs only (S-D) (14%); and combined skin and deep (S/D) (18%). The range of tissues providing afferent input into neurons in the last two categories is remarkably extensive and includes orofacial skin, tooth pulp, temporomandibular joint (TMJ) tissues, tongue, masticatory muscles, hypoglossal nerve fibers, and, finally and most importantly for this discussion, deep cervical paraspinal tissues via the C-2 somatic nerve.

Under normal, nonpainful conditions, these deep afferent inputs are ineffective in stimulating transmission in these second-order neurons. As such, although multidimensional convergence of many of these neurons (superficial vs deep, skin/somatic/viscera) exists, the normal “bias” in the system is toward interpreting input as arising from the predominant cutaneous sources (which in this case are largely in the craniofronto-orbital and facial locations). Put in more sophisticated terms, the central nervous system (CNS) interprets the receptive fields of these neurons in terms of their cutaneous inputs.

Under pathophysiologic conditions (both experimental and clinical), this system changes dramatically. Sessle and colleagues\(^{14-21}\) have investigated alterations in trigemino-cervical pain mechanisms under conditions of experimentally produced pain and inflammation. The sites of these experimental inflammatory lesions have predominantly been in deep craniofacial tissues and include the following: tongue muscle, masticatory muscles, TMJ ligaments, and, most important for this discussion, deep upper cervical paraspinal tissues. Two experimental models have been employed. The first involves electrophysiologic recordings of single dorsal horn or nucleus subcaudalis neurons in response to peripheral analgesic/inflammatory agents. In this model investigators study the altered response characteristics of these neurons, which include such phenomena as (1) changes in the cutaneous receptive field of the neuron, (2) changes in the deep receptive field, (3) persistent discharges from the cell, and (4) lowered threshold of excitation of the cell (including the phenomenon known as “wind-up”).\(^{24}\)

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The second model involves electromyographic (EMG) recordings of the muscles related to the cranio cervical region. In this model investigators study the motor response to inflammatory nociception, thus elucidating the reflex sensorimotor responses to deep pain inputs.

Sessle and his colleagues have explored these nociceptive effects using electrical stimulation of the hypoglossal and C-2 nerves, as well as chemical/inflammatory stimulation, particularly with the C fiber irritant, mustard oil. Put simply, an inflammatory focus is created in, for example, the tongue, the TMJ, or the deep cervical paraspinal muscles, and the response of the spinal cord/V nucleus subcaudalis neurons is explored by using one of the two models just described.

The findings of these studies are partially summarized as follows:

- When mustard oil is injected into both the tongue and the TMJ, there is a statistically significant expansion of both the deep and cutaneous receptive fields (RFs) of the neurons receiving both skin and deep inputs. The expansion of the deep RFs is significantly greater than that of the cutaneous RFs.

- The threshold of excitation of many of these neurons becomes lowered during the phase of RF expansion. EMG activity of neck and jaw muscles becomes significantly increased, not only immediately after the induction of the inflammatory lesion (in both the deep paraspinal muscles and the TMJ), but also in a second phase of excitation that begins some 10 minutes afterward and can last for up to 20 minutes afterward (Fig 3).

- A differential pattern of EMG activation appears to occur with mustard oil injection into the TMJ, as compared with the deep paraspinal muscles around Cl–3. In the former case, only jaw muscles, and not neck muscles, demonstrated significantly early and/or late EMG activation. In the case of injection into the deep upper cervical muscles, EMG activation occurred in both superficial (ie, trapezius) and deep muscles in the region, as well as in jaw muscles (albeit of considerably less magnitude and duration) (Fig 4).

- The EMG increase in jaw muscles induced by injections into the TMJ can be blocked by both peripheral injection of lidocaine (a peripheral anesthetic agent) and systemic injection of MK-801 (an NMDA [N = methyl D = aspartic acid] receptor antagonist). Also, systemic injection of naltrexone after the early phase of EMG activation can enhance (or “rekindle”) the second, later phase of EMG activation, actually increasing its magnitude as compared with mustard oil injection alone.

These findings can be interpreted as follows:

- Upper cervical cord and trigeminal nucleus subcaudalis neurons are particularly responsive to deep nociceptive inputs, which as Hu et al have said, “unmask or strengthen... central somatosensory neuronal relays of convergent afferent inputs that normally are relatively ‘ineffective’ (‘silent’) in exciting (these) neurons.” As such, deep nociceptive inputs are particularly effective in creating the most significant increase in cutaneous hypersensitivity and an increase in the receptive fields of dorsal medullary horn neurons. In clinical terms, this explains several important features of deep tissue pain, including its poor localization (explained by multiconvergence on numerous central neurons), hyperalgesia (so-called “secondary hyperalgesia”), and spread of hyperalgesia and its referral to distant cutaneous regions. These phenomena are all well-known attributes of myofascial and, in particular, spinal pain syndromes. They are also important components of myofascial dysfunction and pain referral likely to be operative in headache of cervical origin.

- Deep pain inputs activate local and, in some cases, distant muscles, presumably in some kind of early protective response. However, it can be presumed that this muscular reactivity contributes to the pain and dysfunction of clinical syndromes involving the neck and jaw articulations.

- There is a complex neurochemical control of these mechanisms that balances inhibitory and excitatory influences within the entire sensorimotor system involved with cephalic and facial pain.

- All of these mechanisms are consistent with the phenomenon of “central sensitization” demonstrated previously in spinal systems. This phenomenon is consistent with the model of neuroplasticity proposed by Dubner and Ruda, in that changes in central processing of nociceptive transmission contribute to the development and prolonged maintenance of the pathophysiology associated with pain arising from deep somatic tissues.

IMPLICATIONS FOR HEADACHE OF CERVICAL ORIGIN

Central sensitization provides a mechanism to explain the clinical phenomena seen so regularly in headache sufferers of (1) persistent somatic pain; (2) pain referred from the cervical spine or posterior occipital region into the fronto-orbital regions, perceived as “headache” but in fact being referred neck pain; (3) tender hyperalgesic muscle zones (“‘trigger points’”) that often expand as the headache pain increases; and (4) muscular tension in the deep suboccipital, superficial occipital, and craniofacial muscles that for many years has been thought to be the sole basis of muscle contraction or tension headache.

On the other hand, the mechanism of “convergence-sensitization-projection” also serves to explain how pain arising from
inflammatory pain arising from the posterior suboccipital muscles and joints, is explained by the phenomenon of “central sensitization” and the neuroplastic changes that these second-order neurons undergo in response to prolonged peripheral deep somatic pain. These are the mechanisms that have been proposed by chiropractors to arise from the subluxation or dysfunction state of the vertebral motion segment. Certainly this is consistent with the older model of “central facilitation” proposed by Korst\(^9\) and adhered to by several generations of chiropractors.

CERVICOGENIC DYSFUNCTION IN HEADACHE

The final section of this article explores evidence for the spinal subluxation phenomenon in groups of headache sufferers.

In Vernon et al.'s 1992 report on cervicogenic dysfunction in muscle contraction (i.e., tension-type headache [TTH]) and common migraine (i.e., migraine without aura [MWA]), the components of cervicogenic dysfunction were defined, and the literature up to 1988 was reviewed in defense of the notion of a broad, highly prevalent basis for cervicogenic dysfunction in headache. In a 1991 report, Vernon had also addressed how this high prevalence of cervicogenic dysfunction in these types of headaches argued against the position adopted by the IHS (based principally on the work of Sjaastad and colleagues) that CH was a narrowly defined, infrequently encountered form of headache.

In their 1992 report, Vernon et al. outlined the components of cervicogenic dysfunction, as follows:

- hypomobility—variously termed "subluxation," "joint blockage," "segmental dysfunction," and "fixation;"
- tender points in the soft tissues—variously called "tender muscle points," "triggerpoints," and "hyperalgesic zones;"
- reduced regional ranges of cervical motion; and
- radiographic findings of static misalignment and dynamic intersegmental abnormality.

To this list, two other features should now be added:
- static segmental misalignment on palpation (most often C-1 or C-2) and
- static malposition of the head and neck (specifically, anterior carriage of the head and low, rounded shoulders).

The reader is directed to Vernon et al.'s report for literature supportive of each of these components prior to 1998. The more recent literature (with a few notable exceptions) is reviewed here.

Hypomobility

In 1985 and 1986 Jull reported on both the reliability of upper cervical joint motion palpation and its use in headache subjects to determine the lesioned segment. A comparison between headache and nonheadache subjects revealed dysfunction at C0-1, C1-2, and C2-3 in 60%, 40%, and 55%, respectively, of headache subjects, compared with 5%, 12%, and 22%, respectively, in controls. These palpatory findings were confirmed by Jull and colleagues and Dwyer et al. using diagnostic anesthetic blocks as the gold standard.

Jensen et al.'s treatment study of posttraumatic headache sufferers reported on findings of hypomobility in the 19 subjects entered into the trial and on the presence of "joint block" in one or more of the upper three segments of the cervical and thoracic spinal regions. Fourteen of 19 subjects had at least one block in both regions, while 4 of 19 had a blockage only in the upper thoracic spine. Thus, 18 of 19 subjects had a joint blockage in at least one of the three upper cervical segments. The segment demonstrating joint blockage most frequently was C1-2.

In Vernon et al.'s 1992 report, three motion palpation procedures (as described by Fligg) were utilized: anteroposterior (AP) glide, rotation, and lateral flexion. A major blockage in any of these three procedures on either side at C0-3 was indicative of segmental fixation. Not a single subject in either the muscle contraction or the migraine group had no fixations at any of the upper cervical segments, and only 16% in each group had a fixation at only one level. In the muscle contraction group, 54% had fixations at two levels and 30% at all three, while for migraine subjects, these figures were 42% and 42% respectively. Thus, in both groups, 84% had a fixation at least two of three upper cervical segments.

In 1993 Watson and Trotter used multiple outcomes (others are discussed below) to assess cervical headache subjects.
They reported on the reliability of posterior-to-anterior glide palpation in 12 of their subjects examined on two occasions by the same examiner, with \( r \) values ranging from 0.67 to 1.0, depending on the segment. They also included, as positive signs of joint dysfunction, the presence of tenderness and muscle stiffness (see below). When all three signs—fixation, tenderness, and palpatory stiffness—were included, far more positive findings were found in headache subjects than in controls. The most prevalent level was C0-1.

In 1994 Trelleaven et al.\(^4\) reported on 12 subjects with posttraumatic headache compared with an age- and sex-matched control group. Signs of cervicogenic dysfunction included in this report were joint blockage, range of cervical motion, forward head posture, and neck flexor muscle endurance; the last three will be discussed separately below. The protocol employed for palpation of joint blockage included the following three features, assessed on a segmental basis:

1. presence of abnormal motion (hypermobility),
2. abnormal tissue quality (hypertonicity), and
3. provocation of pain (tenderness).

Joint dysfunction was rated as mild, moderate, or marked. Ten of 12 headache subjects had at least one segment showing marked joint dysfunction in the upper cervical spine. A qualitative comparison between headache and control subjects showed much more hypomobility between C-0 and C-3 in headache sufferers. The dysfunction detected in controls tended to be milder and spread evenly throughout the cervical spine.

**Craniovertebral tender points**

Tenderness on palpation of the skin\(^a\) and deep tissues of the craniovertebral and paraspinal region is the most commonly reported sign of headache of cervical origin. Virtually every relevant author has reported on the subject, from Lewit,\(^40\) who reported on "pain over the posterior arch of atlas," to Sachse et al.\(^41\) who reported similar suboccipital and scapular tenderness, and Graff-Radford et al.\(^42\) and Jaeger,\(^43\) who reported on the numerous cervical tender points that serve to perpetuate myofascial head pain. Sjaastad et al.\(^44\) reported on the high prevalence of tenderness at C2-3, and Bouquet et al.\(^45\) reported on 24 cervicogenic headache sufferers, 21 of whom had an ipsilateral trigger point at C2-3. Bouquet et al also commented on a frequently rotated and enlarged C-2 spinous process as evidence of some static misalignment that typically accompanies the spinal subluxation. In Jaeger's\(^46\) report on 11 cervicogenic headache sufferers, tenderness and misalignment around the transverse process (TVP) of the atlas were the most frequent palpatory findings.

In 1991 Lebbink et al.\(^47\) compared 164 headache sufferers with 108 controls. Subjects completed a survey form that included questions on the presence and severity of neck muscle tightness and soreness, as well as the presence of head/neck injury in the past. Statistically significant increases in most of these variables occurred in headache subjects as compared with controls and also in headache subjects during a headache as compared with when headache free, although 49% of headache subjects had neck muscle soreness even when headache free.

In Jensen et al.'s\(^48\) clinical trial report,\(^9\) tenderness was reported in 42% of subjects at C2-3, in 89% at C3-4, and in 63% at C4-5.

Vernon et al.\(^49\) used the pressure algometer\(^46\) to verify true tenderness in cervical tender points in TTH and MWA sufferers. This type of assessment has been used with great success by fibromyalgia\(^50\) and headache\(^6\) researchers. In fact, the IHS classification\(^5\) includes the presence (or absence) of pericranial tender points as part of the subclassification of TTH. Recently, Bovim\(^51\) has verified the importance of pericranial (but not cervical) tender points in CH sufferers, and although this study was able to distinguish CH from TTH and migraine with regard to the severity and location of tender points, other studies have found greater similarities among types of headache in this regard.

Vernon et al.'s\(^52\) study reported on the prevalence of six standard craniocervical tender points in these headache groups:

1. medial occipital brim,
2. lateral occipital brim (near the TVP of the atlas),
3. suboccipital (C1-2),
4. midcervical (C2-3),
5. trapezius, and
6. levator scapulae.

Table 4 shows the distribution of numbers of tender points (bilateral points counted as 1; therefore, maximal count was 6) in the two headache groups. Eighty-five percent of TTH patients had at least one; 75%, at least two; and 50%, four or more. In the migraine group, these figures were 92%, 76%, and 44%, respectively, indicating a high prevalence of tender points in both groups. Table 5 lists the most prevalent locations, showing a strong (predictable) prevalence for the upper cervical region.

In an unpublished study by Vernon (H.V., unpublished data, 1992), these findings were replicated in a case–control comparison of TTH sufferers (14 subjects in each group). In the headache group there was a stronger predilection for multiple tender points (four or more). All tender points were measured by pressure algometry. The mean score in the headache group (2.94 kg/cm\(^2\)) was significantly lower than that of the controls (3.44 kg/cm\(^2\); \( t = 1.94; P < .05 \)).

The findings of Watson and Trott,\(^53\) in which detection of joint dysfunction in headache subjects and nonheadache subjects included pressure palpation for tenderness, have already been described. Again, this procedure was found to have good
Within 5 minutes after a spinal manipulation to the lesioned patients (some of whom also complained of headaches). Vernon et al.52 who employed pressure algometry in neck pain found clinical differences among the groups.


Table 4. Distribution of tender points (TPs) by the number of points

<table>
<thead>
<tr>
<th>Headache groups</th>
<th>No of TPs</th>
<th>CM</th>
<th>MCH</th>
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Intraexaminer reliability and to distinguish headache from nonheadache subjects, with a higher prevalence of findings in the headache subjects.

In 1993 Mercer et al.50 reported on 42 posttraumatic headache subjects, 39 with migraine, 11 with TTH, and 9 with combined headaches according to IHS criteria. A high prevalence of myofascial tenderness and joint dysfunction was found in each of these three headache groups (n=59), with particular emphasis on neck-related "myofascial features."

In 1994 Duckro et al.51 reported on 42 posttraumatic headache subjects who were divided according to IHS categories (TTH, 70%; combined, 24%; migraine, 19%; cervicogenic, 7%; others, 24%). A high prevalence of tender points in head, neck, and upper back muscles was found, with no significant differences among the groups.

Vecchiet et al.51 studied tender points in the semispinalis and trapezius muscle of 10 TTH sufferers. They employed an electrical pain threshold test applied to the skin, subcutaneous, and deep tissues separately. In addition, resting EMG levels were recorded in these muscles. All tender points that were measured in these cervical muscles had been found to create referred frontal headache. The pain threshold measurements were made before and again 2 and 10 days after injection of a local anesthetic agent. At 10 days after injection, pain threshold values were significantly higher in all three tissues at all injected tender point sites. Also, EMG activity was significantly lower, and headache activity was significantly reduced.

These findings are somewhat similar to the findings of Vernon et al.52 who employed pressure algometry in neck pain patients (some of whom also complained of headaches). Within 5 minutes after a spinal manipulation to the lesioned segment, bilateral pressure pain thresholds increased an average of 45%; in control subjects, no increase was recorded.

Finally, tenderness to palpation has been used as one of the cardinal signs by Vernon, Jull, and colleagues53-54 to locate the level of zygapophyseal joint dysfunction potentially responsible for neck pain and headache. These tenderness findings correlate very well with the signs of joint hypomobility discussed above. This combination of tenderness and hypomobility (as in the study by Watson and Trott55) correlate very highly with anesthetic joint blockades, considered the gold standard for diagnosis and used with greater frequency as a successful therapy for headaches.

Increased muscle stiffness is a finding associated with muscle tenderness. In 1992 Vernon and Gitelman56 reported on a single case of bilateral TTH with cervical dysfunction. Pressure algometry revealed clinically significant tenderness bilaterally in the suboccipital region. Muscle stiffness of the midcervical paraspinal and trapezius muscles was measured using Fischer's tissue compliance meter. Higher than expected values were found in both muscle sites.

Sakai et al.57 used a computerized compliance meter in a comparison of 37 TTH subjects and 63 controls. In 65% of TTH subjects, there was significantly increased trapezius stiffness, while the overall mean values (756+121 vs 538+89) significantly distinguished headache from control subjects. An orally administered muscle relaxant greatly reduced this increased stiffness in headache subjects, implying that active tension contributed to the stiffness.

It is evident that the more recent studies of myofascial dysfunction (pain/tenderness and stiffness) employ more sophisticated methodologies and instrumentation and are being...
conducted in case–control design formats. The findings of these studies even more strongly substantiate the clinical and empirical experience of a high prevalence of craniocervical tenderness in headache sufferers, which is strongly associated with other signs (including misalignment and joint hypomobility) of spinal segmental subluxation. Studies that also employ manipulation or anesthetic joint blockade have, as indicated by the relief of symptoms, further implicated cervicogenic joint dysfunction in the etiology of headache.

**Radiologic findings of cervicogenic dysfunction**

Pfaffenrath et al. used a computer-aided method of analyzing segmental cervical motion on flexion-extension radiographs. They found a statistically higher incidence of restrictions at C0–1 in cervicogenic headache subjects as compared with controls.

In 1991 Jensen et al. published a second report, a descriptive study of the sample of posttraumatic headache sufferers studied in the clinical trial. In this study, flexion-extension radiographs were obtained in 19 headache sufferers and in a group of age- and sex-matched controls. Total C1–7 motion and motion at each segment were measured with a computer-aided protocol. Findings correlated with age, pain severity, and the presence of three associated symptoms: dizziness, visual symptoms, and ear symptoms.

Jensen et al. reported that total flexion-extension was reduced in headache subjects compared with controls (88° vs 100°; \(P<0.05\)). There was a tendency toward a reduced cervical curve in the headache subjects. Three segments—C2–3, C5–6, and C6–7—demonstrated reductions in motion in the headache subjects. Age-corrected C1–7 motion was negatively correlated with the log (pain) scores, indicating that an increase in pain was strongly associated with a reduction in motion.

Vernon et al. report on TTH and MWA subjects used a similar method that rated segmental movement against the normative data from Dvorak et al. They also used Penning's method for C0–1. The mean + 1 SD for occipital flexion in both headache groups was 3.17° + 2.24°, while for extension it was 10.6° + 7.7°. The percentage of subjects showing reductions in motion below a lower cut-off for normal (−1 SD) was 90% for flexion and 70% for extension. These results agree with those of Pfaffenrath et al. For the rest of the cervical spine, a pattern emerged of greater hypermobility at C1–2 and hypomobility at C4–6. Finally, with regard to the neutral cervical curve, 77% of all subjects had a substantial reduction in (quasi- or fully alordotic) or abnormal reversal of the cervical lordosis.

Nagawasa et al.'s recent report adds further to our knowledge of changes in cervical function in headache sufferers as determined by radiographic analysis. They compared 372 TTH subjects with 225 controls and found a statistically significant reduction in the neutral curve as measured by a "cervical spine curvature index" (14.6±11.9% vs 19.4±11.1%; \(P<0.001\)). They also found that segmental instability was less frequent in TTH patients than in controls, but there was a higher frequency of low-set (ie, rounded) shoulders in TTH patients (57.5%) than in controls (41.8%; \(P<0.01\); \(\chi^2=16.6\)). These findings confirm that TTH sufferers have significant cervical postural and segmental motion abnormalities, typical of subjects whose headaches would be labeled as "cervicogenic."

**Cervical posture, muscular weakness, and range of motion**

Watson and Trott studied 60 subjects. With recurrent cervical headache (a combination of TTH, MWA, and CH) and 30 controls. They studied (1) the degree of anterior head carriage, which was measured photographically; (2) the isometric strength and endurance of the upper cervical flexors, measured by strain gauge dynamometry; and (3) the presence of "joint dysfunction," measured by combining manual palpation findings of restricted joint play, tenderness, and stiffness (described above).

They found a smaller mean angle of forward head position (FHP) in headache sufferers (ie, a straightened cervical spine). Headache sufferers also had smaller strength values and smaller endurance values. These findings correlated well with the degree of FHP. Watson and Trott concluded that FHP increases the load on the posterior muscles that rotate the head (occiput) backward to maintain the orthostatic horizontal position of the eyes. This in turn weakens the antagonist muscles (upper cervical flexors) and contributes to upper cervical joint and myofascial dysfunction, ultimately leading to upper cervical pain. This nociception reinforces the local muscle spasm (found experimentally by Hu et al.) and creates a vicious cycle of pain–spasm–altered mechanics–pain, all leading to the potential for referred cranial pain.

Further evidence for the development of regional muscular stiffness and reduced range of cervical motion comes from Kidd and Nelson's report using a very simplistic observer's evaluation of neck range of motion in 64 subjects, 37 with and 37 without benign headache. The headache sufferers more frequently had a reduction of two or more ranges of motion.

In Treleaven et al.'s study, several other parameters were measured in posttraumatic headache suffers and controls, including neutral head posture, range of cervical motion, "muscle tightness," and endurance of the upper cervical flexor muscles. These tests were similar to the protocol used by Watson and Trott. However, contrary to Watson and Trott's report, the degree of forward head posture was not significantly greater in headache subjects. Also, gross ranges of motion were not greatly reduced, although more muscles...
tested positive for "tightness" on manual examination. The endurance of the upper cervical flexor muscles was significantly reduced in headache vs control subjects (1+1.4 s vs 2.9±2.2 s; P<.02).

All of these findings combine to create a composite of cervicogenic dysfunction, much of which has been observed in noncervicogenic headache sufferers (ie. TTH and MWA). This profile includes regional alterations in anterior head posture; straightened cervical curve and low-set shoulders; regional muscular stiffness and reduced range of motion; and upper cervical subluxogenic signs, including misalignment, joint hypomobility, and frequent segmental myofascial tenderness. The high potential for upper cervical pain to occur, not only unilaterally but also bilaterally, creates potent opportunities for cranial pain referral.

SUMMARY

The literature on headaches of cervical origin has been reviewed, focusing on three areas: the results of studies employing manipulation and facet or neural anesthetic blockade; recent advances in the understanding of craniovertebral pain mechanisms; and finally, recent studies of cervicogenic dysfunction in several categories of headache. Including TTH, MWA, and CH. The current IHS approach to CH is too narrow and will create many false misattributions, typically tending toward underdiagnosis of the cervicogenic component of many benign headache conditions. Vernon's1988 model of vertebrogenic headache should be modified slightly. There are three likely categories of benign headache:

1. Those in whom the cervicogenic component is etiologic. This group ideally will derive primary benefit from spinal manipulative therapy (SMT) or other treatments aimed at cervical dysfunction.

2. Those in whom the cervicogenic component is secondary but synergistic. This group ideally could derive significant benefit from SMT in conjunction with other therapeutic measures.

3. Those in whom the cervicogenic component is negligible, reactive, or fully absent. This group would derive little or no benefit from SMT.

As in 1988, it is still the case that only careful but comprehensive research, taking full account of cervicogenic dysfunction, will ultimately determine the validity of this model.

REFERENCES


Chiropractic rehabilitation of spinal pain patients: principles, practices and outcome data

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Deborah Kopansky-Giles, DC, FCCS(C)
Carol Hagino, MBA
Shirley Fuligni

Objective: To review basic principles and practices of chiropractic rehabilitation for spine pain patients and to present data on outcomes of an active care program.

Design: Pre-post statistical comparisons of patient outcomes in a 6-week program of active care.

Setting: Rehabilitation clinic.

Participants: A convenience sample of seventy-three work-injured spine-pain patients from January 1993 to September 1994 who completed a 6-week intervention program. Forty-eight (48) males with an average age of 41 years, and 25 females with an average age of 39 years were included.

Outcome measures: VAS for pain seventy; Oswestry and Neck Disability Indices; self-ratings for improvement; an outcomes satisfaction index.

Results: The average duration of complaint was 48 days. Mean pre-post changes in pain scores (6.7 to 3.4) and disability scores (27.3 to 17.1) were highly significant (p < .0001). Eighty-one percent (81%) of subjects were discharged as fit to return to at least modified work. The average level of self-rated improvement was 68%. Eighty-one percent were discharged as fit to return to at least modified work. The average level of satisfaction with outcome was 39/50. The highest correlations were found between disability status, self-rated improvement and outcomes satisfaction (.57—.81).

Conclusion: An active care program has been shown to produce high levels of clinical improvement and patient satisfaction in a sample of moderate-to-severely disabled spine-pain patients. While this study has limitations, investigations such as this are essential to improve the quality of care provided to work-injured spine-pain patients.


KEY WORDS: chiropractic, manipulation, rehabilitation.

Introduction
Within recent years, the incorporation of active-care, exercise-based rehabilitation programs into the chiropractic private practice setting has expanded considerably. Several developments have likely contributed to this.

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behaviour of the spine within the context of the total locomotor system. In other words, chiropractors were receptive to and ready for this paradigm shift.

2 It is now recognized that chiropractors are particularly effective in providing quality education to their patients as to the nature of their pain problems. Thus, chiropractors were ideally positioned to blend the newer "back school" components of spine rehabilitation with their practice model.

3 "Muscular re-education models" have been developed and incorporated into the mainstream of chiropractic thinking, particularly with the work of Lewit and Janda.

4 During this same time period, parallel developments occurred in medicine toward a "sports medicine" approach to the care of back pain, largely motivated by studies which debunked the value of bed rest. The new motto appears to be "keep it moving."

5 The infusion of Waddell's insights into the behavioral dimension of low back pain, particularly in focussing on the distinction between disease/impairment and illness/disability, and particularly as it relates to the occupational setting, has contributed to this trend.

Chiropractors appear to be participating in these new "active care" models in ever-increasing fashion. This is reflected in several trends in the profession, including the development, in the USA, of the Chiropractic Rehabilitation Association, and in Canada, of the emerging Fellowship in Chiropractic Rehabilitation. These bodies and others have initiated considerable postgraduate course work in musculoskeletal rehabilitation.

In Ontario, chiropractic rehabilitation clinics were created in 1989 in response to initiatives taken by the Workers' Compensation Board. The Ontario WCB developed the Medical Rehabilitation Plan which created a "Community Clinic Program" of outpatient clinics for the treatment of work-related soft tissue injuries. These clinics have also been active in the area of motor vehicle accident-related personal injury rehabilitati. Other provinces are heading in similar directions.

In this paper, we outline some of the principles and practices of chiropractic spinal rehabilitation, particularly those which we have incorporated in a program of early, active intervention for work-related injuries. We report on a convenience sample of patients from 1993–1994, providing data on baseline characteristics at referral, and on the outcomes of this structured rehabilitation program. We conclude with some opinions and recommendations for the future of this challenging trend in chiropractic.

Principles and practices of chiropractic spinal rehabilitation

Burns and Liebenson have recently published excellent reviews of the history and scientific basis for prescribing therapeutic exercise and conducting spinal rehabilitation in the private-practice setting. Lahad et al. have recently conducted a review of the effectiveness of exercise therapy in the pain and secondary prevention of low back pain, concluding that sufficient evidence exists supporting its use for this purpose. The foundation of the rehabilitation paradigm rests on shifting the perspective of mechanical spinal pain disorders from a biomedical/pathology model to a biopsychosocial/function model. In this model, a systems approach is used to deal with the various biomedical dimensions of physiology, pathology, and social sciences into a holistic perspective, particular the emphasis is less on disordered physiology and more on disordered function.

As such, while the narrow goal of chiropractic treatment centers on the correction of discrete mechanical lesions and their accompanying consequences, and while the expected result of treatment is the reduction of symptoms and improving function of this discrete lesion, there is a broader, more holistic context which includes the overall flexibility, strength, and integration of movement of the spine and the locomotor system. Beyond this, consideration must be given to the demands each individual's environments, both in the workplace, activities, and at play - on the spine and locomotor system. Maximal recovery depends upon appropriately re-integrating the patient - physically and psychosocially - with these environments. From this biopsychosocial model, one can derive the need for interventions which extend beyond the manipulation treatment of the spinal subluxation, to therapeutic exercise to help the goal of which is to improve regional and locomotor function, strength, endurance and coordination, as well as improve overall aerobic fitness levels.

In fact, the more recent exercise movement in the treatment of low back pain (LBP) - the "sports medicine" model — centered on this last issue, particularly the development of what came to be known as the "deconditioning syndrome". Following the work of Mayer and others, acute and, certainly, chronic low back pain patients have been found to be exhibiting progressive weakness of trunk muscle function, reduction of aerobic fitness and an increase in a constellation of abnormal illness behaviors centering around the "fear-avoidance beliefs". Patterns of pain and restriction are seen to be progressively reinforced in this patient by avoidance of activity which is seen as a proximate threat to provoke more pain. Additionally, such patients have been seen to rely more heavily on external agents (therapists, drug therapy) to effect therapeutic change in their lives and become increasingly unable or unwilling to participate actively in their own recovery. Taken together, these and other factors form a picture of the deconditioning syndrome - which is an ideal target for supervised, guided and progressive exercise programs. Therapeutic program employed in this study has a multiple characteristics and components. Referall for the program should be within 70 days post-injury, thus emphasizing that intervention is as early as possible and appropriate. The underlying assumption is that primary care practitioners will provide level treatment for the acute phase of a work-related
injury10 and will then refer for secondary-level active care to complete the recovery path of their patient.

The program begins with a comprehensive intake assessment which includes attention to injury history, course of condition, subjective symptom rating, physical, neurologic and functional testing. The key objectives of the intake assessment are to:

1. Establish or confirm the diagnosis
2. Identify key pain generators
3. Identify key functional impairments
4. Identify critical psychosocial barriers to recovery
5. Identify an individualized profile of functional deficiencies for which exercise and education might be beneficial interventions.

The outcome of the intake assessment process is an individualized, injury-specific intervention plan which has a high likelihood of success at discharge. Additionally, the intake process should develop a reliable, quantitative baseline of clinical information against which clinical progress and outcome can be measured. The variables included in this individualized patient data base may be related to symptom-rating, functional impairment, psychosocial status etc. (See: Methods, below)

The intervention program must have three components:
1. Treatment
2. Exercises
3. Education.

1. The Treatment program offered to patients in this study largely consisted of chiropractic therapeutic measures, primarily spinal manipulation, provided by the primary referring practitioners.10
2. The Exercise program is characterized by the following features:
   (a) The program must be individualized. This applies to the kind of exercises employed, the target tissues or structures, the target functions and the intensity levels.
   (b) The program must be injury-specific, however it should also be comprehensive and holistic, i.e. total-body conditioning, coordination, and functional capacities, particularly those which are relevant to work demands.
   (c) The exercises must be progressively increased based on the patient’s tolerance, capacities and needs.
   (d) The program is performed on a daily basis.
   (e) The exercise program reported in this study also increases progressively with regard to time in the program. The initial period of exercise activity may start at 30–45 minutes per day within the first week and then progress to 60–90 minutes in the second week. From week 3–6 the program may involve 120–150 minutes of exercise per day.

Table 1

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<tr>
<th>Goal</th>
<th>Methods</th>
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<td>Flexibility</td>
<td>Stretching exercises: passive, assisted active neuromuscular facilitation</td>
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<td>Regional strength recovery</td>
<td>Isometric, then isotonic strength exercises</td>
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<td>Recovery of coordination of motor patterns</td>
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<td>Recovery of aerobic fitness</td>
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The exercise program is modified on an ongoing basis particularly by feedback from the patient. This is provided verbally, by daily log entries, and on the basis of regular re-assessments. The progressive incremental expansion of the program, in terms of the numbers, kinds and intensity level of exercises and in terms of their goals and applications (see below) is consistent with the concept of "continuous quality improvement" in clinical management. In fact, these types of exercise programs are ideal models for integrating concepts of CQP into the health care arena.

The education program evolves with respect to the goals and applications for the patient. The typical schema is outlined in Table 1.

The Education Program is delivered in several models: i - individual and/or group models; ii - formal and/or informal. The focus of the education program must be injury-specific, but must also include attention to general health issues, particularly those related to the injury site. An example would be as follows:

For a lumbar sprain injury—
- Information on the anatomy and function of the lumbar spine;
- Biomechanical and ergonomic information on the daily use of the spine;
- Information on weight control and smoking cessation as they relate to back pain;
- Discussion on benefits of proper sleep, and techniques to achieve relaxation.

The education program is delivered in a multi-media context, including: 1 - written documents, pamphlets, fact-sheets etc., 2 - the use of charts, slides, videos, 3 - interactive learning sessions.

The principle aims of the education program are: 1 - reduction of distress and fear-avoidance behaviours; 2 - enhancement of motivation and capacity to participate effectively in the exercise program — this goal being consistent with the notion of "client empowerment" contained in the CQI model; 3 - prevention of future recurrences through management of pain and through ergonomic and biomechanical modifications in the patient's lifestyle, and, 4 - having the patient accept an increased level of responsibility for his/her recovery.

The intervention program ends with a comprehensive discharge evaluation which includes:

1. Debriefing of the patient and providing a home exercise program;
2. Final clinical evaluation with symptom, disability and psychosocial rating;
3. Rating of program and outcomes satisfaction;
4. Determination of work readiness and discharge status for the Workers' Compensation Board (see: Methods).

In 1994, Gill et al. reported on a series of 50 patients enrolled in a similar "community-clinic" program in Hamilton, Ontario. They reported statistically significant reductions in pain, disability, and handicap level as well as reductions in Sickness Impact Profile scores. As well, a high rate of return to work (74%) was reported.

Methods

Patients are referred to the rehabilitation facility by their treating practitioner. Ninety-five percent of patients in this group were referred from chiropractors, with only 5% from medical doctors. The intake assessment served as the baseline for obtaining baseline data. Demographic data included: age, duration of current complaint, occupation, primary language.

Clinical information was obtained by using standardized history protocols for neck or low back pain, as well as use of pain and disability rating instruments. Pain was rated using the visual analogue scale and scored out of 10, while received disability was rated on the Oswestry Low Back Index or the Neck Disability Index (scored out of 30). First pain score was designated as VAS1, while the disability rating was designated as DIS1. Comprehensive clinical examinations were conducted but the results of these assessments will not be reported here.

At discharge, pain (VAS2) and disability (DIS2) ratings were obtained. Additional outcomes included:

1. A self-rated improvement scale consisting of a ten cm visual analogue scale. The patient was asked to consider much improvement in your activity level? Has occurred since the beginning of the program. This VAS was anchored "none" and "complete";
2. Doctor's discharge rating as one of the following: a) return to work, b) return to modified work, or c) no return to work, and,
3. A questionnaire consisting of ten items which rated level of satisfaction with the goals of the program. Goals ranged from "reducing my pain" to "helping me learn more about my pain in order to prevent future recurrences". This instrument was named the "Outcomes Satisfaction Index". A more comprehensive report of the properties of this instrument is forthcoming.

Analyses of the data consisted of descriptive statistics, t-tests for pre-post comparisons, and Pearson's Product-Moment correlations and Chi Square tests for inter-instrument correlations. As this was an exploratory study of the possible correlations between various outcome variables, the level of significance was set at .05.

Results

This report includes all patients who completed the rehabilitation program from January 1993 to September 1994. Seventy-three (73) subjects (48 males and 25 females) were eligible. The mean age of the males was 40.5 ± 11.2 years, while for the females it was 38.5 ± 11.3 years. All patients sustained a work-related injury within 70 days of the initial visit was 48 days (range: 8-69). All patients received at least one week of chiropractic treatment.
With regard to the sites of injury, 73% had sustained injuries in the lumbopelvic region (the vast majority being of the sprain/strain variety), 22% in the cervical region and 5% in the dorsal region.

The mean intake pain score (VAS1) was 6.7/10 ± 2.2, while the mean intake disability score (DIS1) was 27.3/50 ± 8.3. At discharge, the mean VAS2 was 3.4 ± 2.4 and the mean disability score was 17.1 ± 9.9. Figure 1 displays these data with the corresponding statistical values. The distribution of discharge status ratings was: Full return to work (RTW) = 34%, Modified-RTW = 47%, and no return to work = 19%. The average outcomes satisfaction score was 39.4 ± 6.8/50. The average level of self-reported improvement in functional ability was 68.36 ± 24.9%.

Table 2 lists the correlations found between these variables.
Discussion

When the average time to referral from the initial injury of 48 days (almost 7 weeks) is combined with the high intake pain and disability scores, this group of work-injured spinal pain patients can be characterized as sub-acute to chronic with serious unresolved complaints, and who are at a high risk for converting to a chronic pain state.\(^3\)\(^5\)\(^6\) As such, the outcomes of this program are of considerable clinical and social magnitude, particularly regarding the potential for effective secondary prevention of chronicity. Taken together, the outcomes of this program are characterized by an average 50% reduction in measured pain level and an average 42% reduction in measured disability level. Eighty-one (81%) percent of patients were discharged as fit for at least modified work. There was also a high level of patient satisfaction for the achievement of a variety of clinical and psychosocial recovery goals (average 76% rating). Patients’ self-rated levels of improvement in function (68%) were slightly higher than the average reduction in disability scores (see below).

A high level of congruence was noted among many of the outcome variables. The high negative correlations between the “pain” and “disability ratings” at discharge, separately, with the “self-rated level of functional improvement”, and the high positive correlation between “change-in-disability scores” (i.e., reduction of disability) and the “self-rated improvement score” indicate that patient’s self-perceptions of improvement generally good reflections of actual clinical status, especially regarding perceived state of disability.

When these findings are added to the above-mentioned that “self-rated functional improvement levels” were, on average, 20% higher than the “change-in-disability scores”, considerable support is lent to this simple measure as a potential outcome indicator.

The high positive correlations between “improvement function level” and “change-in-disability scores”, together with “outcomes satisfaction” scores provides good evidence for construct and concurrent validity of the Outcomes Satisfaction Index, thus warranting further investigation with this instrument.

The poor correlations between “change-in-pain score” to the other variables is puzzling, as is the reduction in correlation between “pain scores” and “disability scores” as compared to intake. These items need further study in subsequent studies.

The findings of our study compare very favourably to those of Gill et al.\(^2\)\(^9\) who reported on a similar physiotherapy program. They reported an average 36% reduction in pain scores, 39% improvement in disability status and a 71% return-to-work rate. In our study group, pain reduction averaged 50%, disability score improvement averaged 38%, while return-to-work rate (full and modified) was 81%.

This study has limitations, particularly with regard to sampling issues and non-randomization. There is a strong selection bias created when using a convenience sample of patients who are referred for rehabilitation, as patients are deemed to be recovering poorly. However, our patient base is more representative of the population of pain patients in the current system of rehabilitation programs, and, as such, our findings are probably highly relevant to such programs. Additionally, the sample size in this study was somewhat small, limiting the statistical power.

Another limitation is the use of the multiple correlation matrix, especially with the p-value at .05. It is possible that many of the correlations can arise in these circumstances. In our findings, many of the correlations had p-values lower than .05. As well, the correlations reported here all have consistency, and so, taken together, they represent a diverse profile of findings.

Further studies ought to include larger sample sizes of objective data from clinical and physical assessments, more in-depth reporting on exactly which areas of therapy generate the highest levels of patient satisfaction.

Conclusion

A convenience sample of work-related spine-pain enrolled in an active program of chiropractic rehabilitation has been described. Descriptive data at intake revealed serious, unresolved complaints. A battery of clinical psychosocial instruments has been used to assess the...
of a six-week intervention program consisting of treatment, exercise therapy and education.

Given the study design limitations and sampling bias, this program is seen to produce high levels of clinical improvement with a high level of discharge success rate. Good correlations have been found between many of the outcomes variables indicating good construct and concurrent validity of these measures. Patient satisfaction with outcomes is rated as quite good and may be based upon the combination of clinical/physical improvement and improvements in attitudes regarding their pain and disability.

Studies such as this are essential in the ongoing effort to improve the quality of care provided to work-injured spine pain patients.

References
The Effectiveness of Chiropractic Manipulation in the Treatment of Headache: An Exploration in the Literature

Howard T. Vernon, D.C.*

Abstract

Objective: To review the literature on outcome studies of chiropractic/manipulation for tension-type and migraine headaches.

Methods: Of nine studies of manipulation for tension-type headaches that reported quantitative outcomes, four were randomized clinical trials and were case series designs. These studies reported 729 subjects, 613 of whom received manipulation. Outcomes ranged from good to excellent. Manipulation seems to be better than no treatment, some types of mobilization and ice, it seems to be equivalent to amitriptyline but with greater ability of effect than this medication. Of three studies on migraine, only one was a randomized clinical trial. These studies reported on 202 subjects, 156 of whom received manipulation. The outcomes ranged from fair to very good.

Conclusion: A modest body of clinical studies exists dealing with the effect of manipulation and headache. The overall results are encouraging, even if not quite supportive in the case of tension-type headache. Further studies in this area are definitely warranted, particularly well-controlled studies in migraine.

Key Indexing Terms: Chiropractic, Manipulation, Cervical Vertebrae, Headache, Migraine

Introduction

Chiropractors of spinal manipulation have long been proposing its effectiveness in the treatment of headaches. Reports of effect exist in the literature of each of the main branches of manual therapy—chiropractic, osteopathy, manual medicine, and physiotherapy. These reports range from anecdotal reports, theoretical treatises, pathophysiologic and clinicopathologic studies to case series, cohort and randomized controlled studies (1, 2). There is a remarkable longevity and consistency of literature, contributing to a coherent body of opinion and supporting the role of the cervical spine in the etiology of headache, significant proportion of what we might call “benign headache.” There is growing evidence for the efficacious role of manual therapies in the treatment of these kinds of headache.

However, this coherent “manual therapy” paradigm is still disregarded in orthodox circles, despite the 1988 introduction of a “cervicogenic headache” category in the Classification of Headaches of the International Headache Society (3). Neglect of the “manual therapy model” also continues despite the large body of data that clearly delineates the role of interconnection of neural pathways in what Bogduk has called the “trigemino-cervical nucleus” in the genesis of headache pain and, possibly, face pain (4). It is now indisputable that the afferent connections from the upper cervical joints have an enormous capacity to create referred head and facial pain (5–7) as well as muscle dysfunction in the cranio-vertebral region (8). Manual therapy researchers have consistently reported on signs of cervicogenic dysfunction, including local pain, referred pain and myofascial dysfunction in the upper cervical region of many sufferers of what has now come to be called “tension-type” and “migraine without aura” headache types (9, 10).

This presentation will focus on the evidence that does exist in the literature mentioned above as to the “effectiveness” of spinal manipulation in the treatment of these types of headaches. The reader is referred to other sources on the issues of the cervicogenic basis of headache and the nature of the kinds of dysfunction that chiropractors and others target in their treatment approaches.

Methods

This paper consists of a qualitative/critical review of all empirical reports on the treatment of a headache type by spinal manipulation. Only studies that report outcomes data for a clearly defined sample of headache sufferers are included. Studies such as those by Lewit (11), Jirout (12) and Boake (13), which report primarily on signs of cervicogenic headache and only mention secondarily that manipulation is, for example, “the most effective form of treatment,” are not included. Single-case studies have also been excluded from this discus-
sion. As such, three types of studies have been included: case series, cohort designs, and randomized trials, whether they be controlled or comparative in nature. These studies are divided into the two main headache subgroups: tension-type and migraine-type. One study concerns subjects with "post-traumatic" headache, although the tension and migraine subtypes predominated in this study sample. This presentation draws heavily from the author's previous works, which essentially presented studies from the period up to 1988 in one group of publications (1, 2) and the work from 1988 to 1994 in a more recent publication (14). The search strategy in these reports included on-line searches in the medical databases (Medline, in particular), manual searches in the chiropractic data bases (CRAC, CHIROLARS), citation searches and peer inquiries.

RESULTS

Tension-type Headache

Although mention of the effectiveness of spinal adjustments for the treatment of headaches was being made in the writings of the earliest founders of the chiropractic profession (15), the first formal report on a clearly delineated sample of patients came in 1928, in the text on chiropractic by Loy Loban (16). Wight summarized Loban's data: in 89 subjects, 58% reported complete relief, 32% marked relief and 10% no relief after a series of chiropractic treatments (17). No subjects were reported to have had their headaches worsened. Naturally, this early report suffers from all the faults of the "prestatistical" era, including the bias that comes from a retrospective report of a practitioner's own patients and the lack of clearly delineated methods. Nonetheless, this report establishes a pattern of outcome, which will be consistent down through the years, of a large percentage of patients (75-90%, as will be shown) who report considerable relief of headache conditions with chiropractic spinal manipulation.

Would that more practitioners had followed Loban’s example and reported in the chiropractic literature on discrete samples of patients or subjects, because in the period from 1930 to 1970, there are no studies that qualify for inclusion in this present review. Wight mentions two other "surveys,” one by the ICA, which was part of a larger survey on public and patient usage of chiropractors, and the other by Rose, in which a small sample (n = 15) was described, 72% of whom had relief of their headaches by chiropractic treatment (18).

In 1979, Hoyt et al. reported on 22 subjects with tension-type headache who were randomly allocated to three groups: cervical palpation and manipulation, cervical palpation only and no intervention (19). Subjects rated the intensities of their headache before the intervention. In addition, EMG recordings of the frontalis muscle were obtained throughout the study. The intervention consisted of one 10-min session, after which pain intensity and EMG readings were obtained.

Hoyt et al. report that the two outcome variables were similar for subjects in all three groups at baseline. After the intervention, only the group receiving a manipulation to the cervical spine demonstrated a reduction of headache severity of about 50%, on average, which was statistically significant.

EMG values were unchanged in all three groups. The first experimental report showing that a single manipulation could have measurable and statistically greater benefit was a procedure or one that involved only low-level manual therapy. Unfortunately, the duration of this effect was not more than any follow-up. As such, this treatment protocol does not extrapolate to typical practice settings, and this study can be regarded as a focused analytical experiment.

In 1982, Vernon reported on 33 subjects, 18 of whom in a prospective study of chiropractic treatment for tension-type headache (20). Statistically significant reductions in headache activity, as measured by frequency, duration and intensity, were obtained in an average of nine treatment sessions of chiropractic manipulation directed principally to the cervical spine. The average frequency was reduced from 13 headaches per month. The average duration was reduced from 2 hr, and the average severity was reduced from 3.5 to 1.5. In addition, the mean satisfaction-with-care score was obtained from an index of three Likert-scaled questions: (a) Were you satisfied with the treatment? Finally, subjects were asked to self-report the presence of four "autonomic" symptoms: nausea, tinnitus and aura. Of seven subjects reporting nausea as the treatment, 100% reported complete relief; of nine reporting dizziness, 94% reported complete relief. These data were also statistically significant.

Although only an uncontrolled study, this report was worthy on several accounts. First, it represented a prospective cohort of tension headache sufferers with standardized outcome variables using a standardized instrument, the headache diary. The outcome analyzed separately and with appropriate statistical approach, the actual magnitude of improvement was determined for the first time, as opposed to the determination of outcomes of subjects who achieved certain percentages as was the case for the few previous case series reported. The average dosage of treatment was determined in future studies with this figure as a benchmark. Finally, quantitative data was reported on two associated autonomic symptoms, providing evidence that manipulation might exert an effect by inhibiting excess sympathetic activity headache sufferers.

On the other hand, the inclusion criteria employed in this study could have been more precise: at present, it is difficult to compare this cohort with other subject samples. Non-randomized uncontrolled design could not provide a basis for any hypotheses of efficacy or effectiveness of manipulation.

In 1985, Droz and Crot reported on a retrospective study of 332 patients with "occipital headache" (taken here as a variant of tension-type headache) (21). The average number of treatments rendered to these patients was nine. Eighty percent of this sample had a clinical result rated as "very good" (less than 90% relief), whereas 10% had a good outcome.
remaining 10% had little or no relief. They noted that the vast majority of successful cases had no more than 10–15 treatments, establishing an upper limit on the effective treatment dosage. They also reported no case of worsening or any adverse effect of spinal manipulation.

Obviously, this report suffers from the range of biases that affect any retrospective analysis, including strong selection biases and the fact that the investigators themselves provided the evaluations of their own care. Nonetheless, their results provide some useful parameters for future investigations, particularly on the issue of effective treatment dosage.

In 1987, Turk and Ratkolb reported on 100 cases of chronic nonvascular headache (22). Once again, nine treatments of spinal manipulation to the cervical spine produced clinically significant reduction of headache activity in 75% of subjects. This was reflected in reduced analgesic usage. Of great interest is the fact that these authors conducted a 6-month follow-up, something very rarely done in the literature under review here. At this point, 65% of subjects still had significant improvement. This is the first finding that hints that manipulation might have its effect by correcting some underlying pain-producing disorder (itself most likely originating in the neck) and that this effect has an important durability.

In 1990, Jensen et al. reported on 19 subjects with post-traumatic headache (23). Seven men and twelve women, whose ages averaged 31 yr and whose average duration of complaint was 12 months, were randomly allocated to either manual therapy (n = 10) or ice packs (n = 9). In the manual therapy group, two treatments were rendered within 1 wk, consisting of mobilization and manipulation to the upper and lower cervical spine and to the upper thoracic spine. In the control group, two sessions of ice treatment were rendered to the neck, each lasting 15–20 min.

The outcomes measured were: pain score, regional ranges of motion, segmental ranges of motion and the presence of accompanying symptoms (dizziness, visual disturbances and ear pain).

The first outcome interval was 2 wk after treatment. At this time, there was a 57% reduction of pain intensity measurements and a 64% reduction in analgesic use in the manual therapy group as compared with the control group. This was statistically significant. At 5 wk, there was also a difference in these variables favoring the manual therapy group; however, it did not reach statistical significance. There was a positive correlation between pain index and the frequency of visual disturbances (r = .65, p < .02) and all three accompanying symptoms (r = .70, p > .01).

This study is one of the few randomized controlled trials in the literature. Investigators were blind to the allocation of the subjects, even though subjects were aware of their allocation. Although this study employed a small sample size, there was a clear clinically and statistically significant difference favoring the manual therapy treatment in reducing headache pain and accompanying symptoms. The finding of improvement in the accompanying symptoms (which seem to have a basis in autonomic dysfunction) is similar to Vernon’s findings (20).

These favorable results are all the more remarkable given the relatively low dosage of treatment (i.e., 2 treatments over 1 wk).

In 1994, Whittingham et al. reported on 26 subjects (16 women, 10 men) with chronic headache and upper cervical dysfunction (24). A self-control time-series design was employed to study the effect of four manipulations with the “toggle recoil technique.” The treatments were delivered over a fixed period of 2 wk. After treatment, these mean findings were reported: 62% reduction in frequency, 77% reduction in duration and 60% reduction in severity of headaches. These variables were collected from a headache diary. This study is of particular interest to chiropractors because of the use, for the first time, of one of the unique manipulative techniques devised by chiropractors. The profession has certainly lacked empirical investigations of these different techniques. That the improvements reported in this study (ranging from 60–77% reductions) were achieved in only four treatments may indicate a particular effectiveness of this manipulative approach. Of course, controlled and comparative trials are necessary to confirm this speculation.

In 1994, Mootz et al. reported on 11 men aged 18–40 yr who were suffering from chronic, episodic, tension-type headache (25). Subjects were required to have at least four episodes per month. The study design was a prospective case series with a 2-wk, no-treatment baseline period and an 8-wk treatment period of 16 interventions. Treatments consisted of spinal manipulation to the cervical spine supplemented by myofascial trigger point therapy and moist, hot packs. This treatment protocol has excellent generalizability to typical practice approaches.

Mootz et al. reported statistically significant pre-post treatment reductions in headache frequency (6.4 per 2 wk to 3.1 per 2 wk), and duration (6.7 hr to 3.9 hr of headache). A strong trend toward a reduction of severity of headache (5.05/10 to 3.37/10) just failed to obtain statistical significance. These findings are very similar to those of Vernon (20) and Whittingham et al. (24).

In 1995, Boline et al. reported on a randomized comparative trial of chiropractic manipulation vs. amitriptyline in the treatment of tension-type headache (26). This trial included 126 subjects with an average headache duration of 6 yr. After the 6-wk/12-treatment intervention stage, subjects in both groups demonstrated clinically and statistically significant reductions of headache activity as measured by the Blanchard Headache Diary (27), although neither group had statistically greater results than the other. However, at the end of a further 6-wk/no-treatment follow-up phase, subjects treated with chiropractic manipulation had significantly less headache activity than those in the drug therapy group, whose headache activity began to approach the baseline level. This finding, like that of Turk and Ratkolb, may indicate that the benefit obtained by manipulation was more durable, possibly by correcting some underlying disorder potentially responsible for the kind of headache suffered by a majority of these subjects.

This study is the most robust of all the trials of manipulation.
The authors reported on an ill-defined variable that they termed “aches” was reduced to a greater level in the chiropractic group. Statistical significance (at the .01 level): the severity of headache in the chiropractic group. 34% in the mobilization control and 13% in the medical group. The overall improvement for all three groups was less than one per wk.

For classical migraine sufferers, 78% of women and 75% of men with a history of more than one per wk. of severe attacks was noted in 81.5% of women and 45.8% of men in the common migraine group and 43.2% and 34.5% of women and men, respectively, in the classical migraine group.

This study suffers from all of the biases of a clinician’s retrospective analysis of his own patients; however, it served a useful function as the first attempt to quantify the levels of improvement possible with this challenging condition in a typical clinical setting.

In 1978, Wight reported on a convenience sample of 87 patients (57 women, 30 men), 34 of whom were found to have what was then called “common migraine” and 53 had “classical migraine” (17). Wight presented the results of his own retrospective analysis of chiropractic treatment in this clinical sample. Eighty-five percent of women and 50% of men with common migraine obtained a “greatly improved” level, which corresponded to 80% or greater reduction in headache activity. For classical migraine sufferers, 78% of women and 75% of men achieved similar levels of improvement. The overall success rate for both categories in both sexes was 74.7%. In those subjects not reporting a greatly improved status after treatment, a reduction of severe attacks was noted in 81.5% of women and 45.8% of men in the common migraine group and 43.2% and 34.5% of women and men, respectively, in the classical migraine group.

This study suffers from all of the biases of a clinician’s retrospective analysis of his own patients; however, it served a useful function as the first attempt to quantify the levels of improvement possible with this challenging condition in a typical clinical setting.

In 1978, Parker et al. reported on a randomized clinical trial of manipulation for migraine (29). Eighty-five subjects (52 women, 33 men), with an average age of 41 yr and an average duration of complaint of 19 yr, were randomly allocated to one of three treatment groups: (a) chiropractic manipulation, (b) medical manipulation, and (c) control/mobilization performed by physiotherapists. Seventy percent suffered from common migraine and the rest with classical migraine. Subjects were not stratified according to the type of migraine headache. Subjects underwent a 2-month baseline period, a 2-month treatment stage and a 2-month follow-up. An average of seven treatments were delivered in the treatment stage, or slightly less than one per wk.

The frequency of migraines was reduced by 40% in the chiropractic group, 34% in the mobilization control and 13% in the medical group. The overall improvement for all three groups was 25%. Only one of the study’s hypotheses achieved statistical significance (at the .01 level): the severity of headaches was reduced to a greater level in the chiropractic group. The authors reported on an ill-defined variable that they termed the “level of enthusiasm” of the patients and practitioners in each group. This seems to be akin to the level of one’s expectation that might have been held by each subject at the outset of the trial. A higher response to a placebo was noted (but not quantified) in the chiropractic group (both subjects and practitioners). Because of this authors concluded that the reduction of headache levels in the chiropractic-treated subjects could be attributed to a placebo response.

A follow-up study was published in 1980, examining psychosocial correlates to successful outcome in the chiropractic manipulation for migraine (30). It was reported that, at 20-month follow-up, further 19% of subjects had achieved a good recovery, overall 47% success rate for all subjects. When the number of subjects who had achieved a complete recovery was analyzed, 57% had received chiropractic manipulation, mobilization and only 7% medical manipulation. A simple test of the hypothesis revealed a statistically significant difference in favor of the chiropractic treatment.

Although the original study was indeed a randomized trial, it was not without its flaws. An understanding of this is tant, not so much for those who wish to use this study as a sole measure of the benefit (or lack of it) of the treatments, but for those who continue to seek hard objective investigations in future, and who wish to improve the quality of future clinical trials. This study employed relatively small number of treatments (seven) delivered over an extended period of time. This may very well have been insufficient dosage of treatment to produce a clinically significant (never mind statistical significance) difference for one group or another. When one examines the data on the effects of “chiropractic treatments” the participating chiropractors found acceptable to offer these subjects (including treatments such as “liver deficiencies,” etc.), one cannot escape the conclusion that the optimum therapy might not have been offered.

Clearly this study did not employ an effective control in the classic sense of receiving a comparative treatment known low or no benefit. The control group actually offered a manual treatment (i.e., mobilization), which seemed to have some value. The differences between the groups on variables tested (regardless of whether the p-value was .01) consistently favored chiropractic manipulation. Along with the relatively small sample size for a randomized clinical trial, indicates that this study sufficed to what we now know as low power to detect a clinically relevant difference favoring the chiropractic-treated group. Therefore, the difference between chiropractic manipulation and mobilization for migraine subjects had a relatively high capacity for on-treatment response, especially in the short-term, the results of which should be viewed as encouraging for the use of manual therapies in general and spinal manipulation in particular in the treatment of migraine headache.

In 1989, Stodolny and Chmielewski reported on a randomized trial of manipulation for migraine (31). Eighty-five percent of women and 61% of men with a history of more than one per wk. of severe attacks was noted in 81.5% of women and 45.8% of men in the common migraine group and 43.2% and 34.5% of women and men, respectively, in the classical migraine group. This study suffered from all of the biases of a clinician’s retrospective analysis of his own patients; however, it served a useful function as the first attempt to quantify the levels of improvement possible with this challenging condition in a typical clinical setting.

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with what they termed “cervical migraine” (31). It seems that their subjects conformed to the criteria for “migraine without aura” according to the International Headache Society Classification (3). Included were 24 women and 7 men, with an average age of 48 yr. The treatment protocol consisted of 2–3 manual therapy sessions emphasizing manipulation to the cervical spine. After 1 wk, they reported complete relief of headache in 75% of subjects. Other findings included a statistically significant increase in range of cervical motion; greatly reduced levels of intersegmental dysfunctions, which had been found in 100% of subjects at C0–C1 and in 75% of subjects at C7–T1 and reductions of dizziness levels in most subjects.

Unfortunately, these authors did not employ pre/post comparison statistical analysis of headache variables such as frequency, severity and duration. The categorical measurement of the outcome as “complete relief” probably reflects substantial reductions in all three variables. Naturally, because no comparison or control group was employed, these results cannot confirm the causal correlation between the treatment and the outcome. On the other hand, these results are consistent with other uncontrolled reports that, when taken together, indicate a high rate (75–90%) of success in the reduction of headache activity after spinal manipulation. These three studies on manipulation in the treatment of migraine are summarized in Table 2.

### Table 2. Studies of the effect of manipulation on tension-type headaches

<table>
<thead>
<tr>
<th>Authors</th>
<th>Treaters</th>
<th>Design</th>
<th>n</th>
<th>HA</th>
<th>Results</th>
</tr>
</thead>
<tbody>
<tr>
<td>Lewit (1971)</td>
<td>MD</td>
<td>CS</td>
<td>41</td>
<td>T-t</td>
<td>Manip. most effective</td>
</tr>
<tr>
<td>Lewit (1971)</td>
<td>MD</td>
<td>CS</td>
<td>93</td>
<td>T-t</td>
<td>Manip. most effective</td>
</tr>
<tr>
<td>Vernon (1982)</td>
<td>DC</td>
<td>CS</td>
<td>33</td>
<td>T-t</td>
<td>85–90% success: pre/post diffs. statis. signif. 80% success</td>
</tr>
<tr>
<td>Jirout (1985)</td>
<td>MD</td>
<td>CS</td>
<td>200</td>
<td>T-t</td>
<td>75% success</td>
</tr>
<tr>
<td>Droz and Crot (1985)</td>
<td>DC</td>
<td>CS</td>
<td>332</td>
<td>Occ</td>
<td>80% success</td>
</tr>
<tr>
<td>Turk and Ratkolb (1987)</td>
<td>MD</td>
<td>COH</td>
<td>100</td>
<td>T-t</td>
<td>75% success</td>
</tr>
<tr>
<td>Jensen et al. (1991)</td>
<td>MD</td>
<td>RCT</td>
<td>19</td>
<td>PTH</td>
<td>Signif. diff. manip. vs. tce</td>
</tr>
<tr>
<td>Whittingham et al. (1994)</td>
<td>DC</td>
<td>COH</td>
<td>26</td>
<td>T-t</td>
<td>60–77% HA reducit. pre/post diffs. statis. signif.</td>
</tr>
<tr>
<td>Mootz et al. (1994)</td>
<td>DC</td>
<td>COH</td>
<td>11</td>
<td>T-t</td>
<td>Statis. signif. reduction of headache frequency and duration after chiropractic care</td>
</tr>
<tr>
<td>Boline et al. (1995)</td>
<td>DC, MD</td>
<td>RCT</td>
<td>126</td>
<td>T-t</td>
<td>Manip $&gt;$ drug at follow-up</td>
</tr>
</tbody>
</table>

CS = case series; COH = cohort; RCT = randomized controlled trial; HA = headache; T-t = tension-type; PTH = post-traumatic headache.

Inasmuch as the strengths and weaknesses of this body of literature have been summarized above in the text and in the accompanying tables, and because this review was not intended to quantitatively synthesize the evidence for manipulation in the treatment of headache, the discussion will be confined to the consideration of issues that will qualitatively improve future research in this area. First, much stronger emphasis will need to be placed on precision in categorizing the headache types to be investigated. The inclusion and exclusion criteria of future studies should conform to the IHS Classification of tension-type, migraine-type, cervicogenic and other headaches (cluster headache would certainly be an interesting condition to investigate). Studies should be directed to one headache type only and should not lump vascular and nonvascular types together in the same study sample. This will strengthen the internal validity of studies by creating more homogeneous samples. It will also strengthen external validity by improving the generalizability of the results, thereby allowing other health care providers to accept the findings based upon standardized headache groupings.

Researchers will need to pay much greater attention to design issues which strengthen the internal validity of their studies. Appropriate baseline phases should always be incorporated to establish stable pretreatment values. The treatment dosage should be sufficient to achieve a therapeutic effect, if one is to be found at all. The consensus in the previous studies is 9–12 treatments over a 2-month interval, at which point optimum results would be expected. Although some studies have employed an appropriate baseline period, none have employed a fairly standard procedure used in other literature (drug and biofeedback studies), i.e., a waiting-list control group. This is a well-accepted method of creating a true no-treatment control group with which to test the efficacy of chiropractic manipulation while not unnecessarily withholding treatment.

Studies should employ standard outcome measures and instruments such as the Headache Diary. Results should be reported in terms of means and standard errors, and appropriate robust statistical testing should be done. This is in contrast to the categorical results such as “very good” or “complete relief” that have appeared in some of the studies reviewed above. Sample sizes should be determined before the study, using variance results from those studies having reported these data. Sample sizes should be large enough so that the power of the study is large enough to detect clinically important differences regardless of the number of treatment groups.

Investigators must refine the diagnostic and assessment procedures used to characterize cervicogenic dysfunction in various headache groups. Improved reliability and validity of man-
Table 2. Studies of manipulation in the treatment of migraine headaches

<table>
<thead>
<tr>
<th>Authors</th>
<th>Treaters</th>
<th>Design</th>
<th>n</th>
<th>HA</th>
<th>Results</th>
</tr>
</thead>
<tbody>
<tr>
<td>Wight (1978)</td>
<td>DC</td>
<td>CS</td>
<td>57</td>
<td>MIG</td>
<td>75% success</td>
</tr>
<tr>
<td>Parker et al. (1978, 1982)</td>
<td>MD, DC</td>
<td>RCT</td>
<td>85</td>
<td>MIG</td>
<td>Manip. = 28% success. At 2-yr follow-up = 47% success</td>
</tr>
<tr>
<td>Stodolny and Chemielewski (1989)</td>
<td>MD</td>
<td>COH</td>
<td>31</td>
<td>MIG</td>
<td>75% success</td>
</tr>
</tbody>
</table>

CS = case series; COH = cohort; RCT = randomized controlled trial; HA = headache; MIG = migraine.

Conclusions

Only by evolving to this level of scientific quality will chiropractors really be able to determine if their therapeutic approaches truly offer anything of value to the vast number of headache sufferers awaiting our efforts. This is certainly a worthy goal.

References


Cervicogenic Headache

Howard Vernon

Key Words
Cervicogenic headache, tension headache, vertebrogenic migraine, convergence projection, central sensitization, central facilitation, aura

After reading this chapter you should be able to answer the following questions:

Question #1 What is the most common level of motion segment blockage (subluxation) found in subjects suffering “cervical headache”?

Question #2 What is the proposed neuroanatomic basis of headache referred from the neck?

Question #3 What cervical subluxogenic signs have been noted in headache sufferers?
The role of the cervical spine in headache continues to be an unresolved controversy. In 1988, a spectrum of headache subtypes that might have some kind of cervicogenic involvement was defined (1), (Figure 16-1). At that time the spectrum ranged from “tension headache with neck muscle pain” through “cervicogenic headache,” defined in chiropractic terms as symptomatic head pain and cephalic dysfunction caused by subluxation of a spinal joint, to a proposed “vertebrogenic migraine.” Coincidentally, in 1988, the International Headache Society (IHS) classification of headaches (2) published its report and, for the first time, recognized a headache subtype known as cervicogenic headache (CH). The definition of CH according to the IHS classification is shown in the box on p. 305. This headache owes much of its definition to the work of Ottar Sjaastad and his colleagues (3-6), which first appeared in print in 1983.

In a subsequent publication (7), this very narrow definition of CH was challenged, and contrasted with the characteristic headache subtypes that chiropractic, manual medicine, osteopathic, and physiotherapeutic experts had addressed in the literature spanning the greater part of this century. The IHS version of CH was also contrasted with the headache subtypes that had been included in the clinical studies of the outcome of spinal manipulation, again, by the same array of practitioners. In these studies of both tension-type and migraine-type headaches (definitely different from the narrow IHS features of the CH category), the results of manipulation to (presumably) some cervical spine dysfunction range from fair to excellent.

This leaves us with a quandary, asking the following questions: is there a putative cervical component in tension-type and migraine-type headaches?; is it similar to or different from the dysfunction in CH?; and, do the results of manipulation studies automatically imply the existence of a causative cervical component to these forms of benign headaches?

Alternatively, is there one kind of cervical component that might contribute to, or manifest as, different forms of headache experiences, and thus may be diagnostically labeled as different headache categories? This chapter explores this quandary with a clear bias toward accepting this alternate hypothesis. This is done by first briefly reviewing the results of clinical studies of spinal manipulation for headaches. Then, current research on pain mechanisms in CH is reviewed. Also, literature on findings of cervicogenic dysfunction in headache groups that are clearly not IHS-CH is reviewed. Finally, the argument is made for the alternative hypothesis: that subluxation/dysfunction of the spine makes a significant contribution to the cause of a number of benign forms of headache.

**Headache and Spinal Manipulation**

In 1991, a review article of all studies of manipulation for the treatment of headaches (7) summarized the work extent to 1988.

This work is summarized in Table 16-1. Since that time, only two treatment studies employing manipulation have appeared in the scientific literature; only one of them in a peer-reviewed publication. In 1989, Stodolny and Chmielewski (8) reported on 31 subjects (24 women, 7 men; average age, 48 years) with “cervical migraine.” They employed tests for intersegmental blockage and found that 100% of subjects demonstrated spinal motion segment blockage at C0-C1; 75% at C7-T1, and 25% between C1-C2 and C3 (see Cervicogenic Dysfunction). The treatments consisted of two to three manual therapy sessions, largely employing manipulative techniques. They reported results at the end of 7 days that included:

- Complete relief of headache in 75% of subjects
- Average increases of cervical range of motion of 9° (statistically significant)
- Fixations reduced in 28 of 31 subjects
- Reports of dizziness in subjects greatly reduced

There was no further follow-up reported. Obviously, no control comparison or blinding of
The assessors was included in their protocol, so their results must be interpreted cautiously, all the more so because of the short treatment period.

In 1992, Boline and Nelson (9) reported at a scientific meeting on the progress of their randomized comparative trial of chiropractic manipulation and the antidepressant drug amitryptyline in the treatment of “muscle contraction headache.” This trial included 150 subjects (87 women, 63 men) with an average headache duration of 6 years. After the 6-week treatment phase, subjects in both treatment groups demonstrated clinically and statistically significant reductions of headache activity. Neither group had statistically greater reductions than the other. However, at the end of a further 6-week no-treatment follow-up phase, subjects treated with chiropractic manipulation had significantly less headache activity than those receiving drug therapy, possibly indicating that the benefit obtained by chiropractic manipulation was more durable and may have corrected some of the underlying causative (but still poorly
Mechanisms of Pain in Cervicogenic Headache

In 1988, a vertebrogenic model of headache was presented, (1) which contained four categories: (1) extrasegmental, referring to the long regional myofascial structures such as the trapezius and long extensor muscles, the ligamentum nuchae, and the interface between the occipitofrontalis muscle and these regional cervicothoracic structures; (2) intersegmental, referring to the three-joint complexes of C2-C3-C4 and the articulations of C0-C1-C2, with their ligaments and deep intersegmental muscles; (3) infrasegmental, referring to the nerve structures in and around the intervertebral foramina, and, in the cervical spine, lying near the lateral portions of the vertebral (in other words, the sympathetic trunk, the vertebral nerve, the C2 dorsal root ganglion, the greater, lesser, and third occipital nerves, and the sensory roots of C1); and, finally, (4) intrasegmental, referring to the spinal cord and medullary dorsal horn with the nucleus subcaudalis of the trigeminal nerve.

Much has been learned about these mechanisms since 1988, centering on the pain pathways of the upper cervical cord and their convergence on cells in the nucleus subcaudalis, a mechanism called by Bogduk (10) the trigemino-cervical nucleus (Figure 16-2).

Upper cervical cord and trigeminal nucleus subcaudalis neurons are particularly responsive to deep nociceptive inputs that, as Hu has said, "unmask or strengthen . . . central somatosensory neuronal relays of convergent afferent inputs that..."
### Summary of Clinical Studies of Spinal Manipulation for Tension Headache and Migraine

<table>
<thead>
<tr>
<th>Authors</th>
<th>Treators</th>
<th>No. of Subjects</th>
<th>Headache</th>
<th>Results</th>
</tr>
</thead>
<tbody>
<tr>
<td>Lewit (1971)</td>
<td>A</td>
<td>41</td>
<td>T-t</td>
<td>Manipulation most effective</td>
</tr>
<tr>
<td>Lewit (1971)</td>
<td>A</td>
<td>93</td>
<td>T-t</td>
<td>Manipulation most effective</td>
</tr>
<tr>
<td>Vernon (1982)</td>
<td>B</td>
<td>33</td>
<td>T-t</td>
<td>85%-90% success</td>
</tr>
<tr>
<td>Jirout (1985)</td>
<td>A</td>
<td>200</td>
<td>T-t</td>
<td>80% success</td>
</tr>
<tr>
<td>Droz and Crot (1985)</td>
<td>B</td>
<td>332</td>
<td>Occipital</td>
<td>80% success</td>
</tr>
<tr>
<td>Turk and Ratkolb (1987)</td>
<td>A</td>
<td>100</td>
<td>T-t</td>
<td>75% success</td>
</tr>
<tr>
<td>Boline et al. (1993)</td>
<td>B</td>
<td>150</td>
<td>T-t</td>
<td>Manipulation &gt; drug at follow-up</td>
</tr>
<tr>
<td>Wight (1978)</td>
<td>B</td>
<td>57</td>
<td>Mig</td>
<td>75% success</td>
</tr>
<tr>
<td>Parker et al. (1978, 1982)</td>
<td>A and B</td>
<td>85</td>
<td>Mig</td>
<td>Manipulation = 28% success initially</td>
</tr>
<tr>
<td>Stodolny and Chmielewski (1989)</td>
<td>A</td>
<td>31</td>
<td>Mig</td>
<td>47% at 2-yr follow-up</td>
</tr>
</tbody>
</table>

A, Medical Doctor; B, Doctor of Chiropractic; T-t, tension-type; Mig, migraine

Normally are relatively "ineffective" ("silent" [11]) in exciting (these) neurons" ([12] p. 72).

As such, deep nociceptive inputs are particularly effective in creating the most significant increase of cutaneous hypersensitivity and an increase in the receptive fields of dorsal medullary horn neurons. In clinical terms, this underlies several important features of deep tissue pain, including its poor localization (explained by multiconvergence on numerous central neurons), hyperalgesia (so-called secondary hyperalgesia), spread of hyperalgesia, and its referral to distant cutaneous regions. All of these phenomena are well-known attributes of myofascial and, in particular, spinal pain syndromes. These are also all important components of myofascial dysfunction and pain referral likely to be operative in headache of cervical origin.

Deep pain inputs activate local and, in some cases, distant muscles, presumably in some kind of early protective response. However, it can be presumed that this muscular reactivity contributes to the pain and dysfunction of clinical syndromes involving the neck and jaw articulations.

There is a complex neurochemical control of these mechanisms that balances inhibitory and excitatory influences within the entire sensorimotor system involved with cephalic and facial pain.

All of these mechanisms are consistent with the phenomenon of central sensitization, which has been demonstrated previously in spinal sys-
Figure 18-2 The trigeminocervical nucleus (From Hooshmand H. Chronic pain. London: CRC Press, 1993:52.)
This phenomenon is consistent with the model of neuroplasticity proposed by Dubner and Ruda (14), in that changes in central processing of nociceptive transmission contribute to the development and prolonged maintenance of the pathophysiology associated with pain arising from deep somatic tissues.

Implications for Headache of Cervical Origin

Central sensitization provides a mechanism to explain the clinical phenomena seen so regularly in headache sufferers of (1) persistent somatic pain; (2) pain referred from the cervical spine or posterior occipital region into the frontoorbital regions being perceived as "headache," but, in fact, being referred neck pain; (3) tender hyperalgesic muscle zones ("trigger points"), which often expand as the headache pain increases; (4) muscular tension in the deep suboccipital, superficial occipital, and craniofacial muscles, which has, for many years, been thought to be the sole basis of muscle contraction or tension-type headache (15).

Conversely, the mechanism of "convergence-sensitization-projection" also serves to explain how pain arising from intracranial structures such as posterior cerebellar tumors or the intracerebral blood vessels (as proposed by Moskowitz [16] in the case of migraine) may be referred to the posterior occipital and suboccipital regions, masquerading as cervical pain.

In summary, the trigeminocervical nucleus and the extensive afferent convergence from numerous cranio-cervical peripheral tissues onto these second-order neurons (a phenomenon called convergence-projection [17]) serves to explain the neuroanatomic basis of headache referred from the neck. The neurophysiologic basis of such pain referral, particularly from inflammatory pain arising from the posterior suboccipital muscles and joints, is explained by the phenomenon of "central sensitization" and the neuroplastic changes that these second-order neurons undergo in response to prolonged peripheral deep somatic pain. These are the mechanisms thought for many years by chiropractors to arise from the subluxation or dysfunction state of the vertebral motion segment. Certainly this is consistent with the older model of "central facilitation" proposed by Korr and his colleagues (18) and adhered to by several generations of chiropractors.

Cervicogenic Dysfunction in Headache

In the final section of this chapter, evidence for the spinal subluxation phenomenon in groups of headache sufferers is explored.

A 1992 report (19) on cervicogenic dysfunction in muscle-contraction (that is, "tension-type" [TTH]) and common migraine (that is, "migraine without aura" [MWA]) defined the components of cervicogenic dysfunction, and the literature up to 1988 was reviewed in defence of the notion of a broad, highly prevalent basis of cervicogenic dysfunction in headache. A 1991 report (7) also addressed how this high prevalence of cervicogenic dysfunction in these types of headaches argued against the position adopted by the IHS (based principally on the work of Sjaastad and his colleagues (3-6) that "cervicogenic headache" was a narrowly defined, infrequently encountered form of headache.

In the 1992 report, the components of cervicogenic dysfunction were outlined as follows:

1. Hypomobility—variously termed subluxation, joint blockage, segmental dysfunction, fixation
2. Tender points in the soft tissues—variously called tender muscle points, trigger points, hyperalgesic zones
3. Reduced regional ranges of cervical motion
4. Radiographic findings of:
   a. Static misalignment
   b. Dynamic intersegmental abnormality

Two other features should be added to this list now:

5. Static segmental misalignment on palpation (most often, C1 or C2 is cited)
6. Static malposition of the head and neck (specifically, anterior carriage of the head and low rounded shoulders)

The reader is directed to the 1992 report for a review of the literature supportive of each of these components before 1988. The recent literature (with a few notable exceptions) is now reviewed.

**Hypomobility**

In 1985 and 1986, Jull (20,21) had reported on both the reliability of upper cervical joint motion palpation and its use in headache subjects to determine the lesioned segment. The comparison between headache and nonheadache subjects (21) showed dysfunction at C0–C1, C1–2, and C2–3 in 60%, 40%, and 55% of headache subjects, as compared with 5%, 12%, and 22%, respectively, in controls. These palpatory findings were confirmed by Jull, Bogduk, and Marsland (22) and Dwyer et al. (23) using diagnostic anesthetic blocks as the gold standard.

In the 1992 report by Vernon et al., three motion palpation procedures (as described by Fligg [24]) were used: A-P glide, rotation, and lateral flexion. A major blockage in either of these three procedures on either side at C0–C3 was indicative of segmental fixation.

It was reported that no subject in either the tension-type or migraine group had fixations at none of the upper cervical segments, whereas only 16% in each group had a fixation at only one level. In the tension-type group, 54% had fixations at two levels and 30% at all three, whereas for migraine subjects these figures were 42% and 42%, respectively. In both groups 84% had a fixation at least two of the three upper cervical segments.

In 1993, Watson and Trott (25) used multiple outcomes (others are discussed later) to assess cervical headache subjects. They reported on the reliability of posterior-to-anterior glide palpation in 12 of their subjects examined on two occasions by the same examiner, with Kappa values ranging from 0.67 to 1.0, depending on the segment. They also included as positive signs of joint dysfunction the presence of tenderness and muscle stiffness (see later discussion). When all three signs—fixation, tenderness, and palpatory stiffness—were included, far more positive findings were found in headache subjects (N = 30) than in controls (N = 30). The most prevalent level was C0–C1.

**Craniovertebral Tender Points**

Tenderness to palpation of the skin (26) and deep tissues of the craniovertebral and paraspinal region is the most commonly reported sign of headache of cervical origin. Virtually every relevant author has reported on the subject, from Lewit (27), who reported on “pain over the posterior arch of atlas,” to Sachse et al. (28), who reported similar suboccipital and scapular tenderness, to Graff-Radford et al. (29) and Jaeger (30), who have reported on the numerous cervical tender points that serve to perpetuate myofascial head pain, to Sjaastad et al. (3,4), who report on the high prevalence of tenderness at C2–3. Bouquet et al. (31) reported on 24 cervicogenic headache sufferers, 21 of whom had an ipsilateral trigger point at C2–3. They also commented on a frequently rotated and enlarged C2 spinous process, evidence of some static misalignment that typically accompanies the spinal subluxation. In Jaeger's report on 11 cervicogenic headache sufferers, tenderness and misalignment around the transverse process (TVP) of atlas were the most frequent palpatory findings.

In the 1992 report, the pressure algometer (32) was used to verify true tenderness in cervical tender points in tension-type and migraine-without-aura sufferers. This type of assessment has been used with great success by fibromyalgia (33) and headache (34) researchers. In fact, the IHS classification (2) includes the presence (or absence) of pericranial tender points as part of the subclassification of tension-type headaches.

The 1992 study reported on the prevalence of six standard craniocervical tender points in these headache groups, namely:

- Medial occipital brim
- Lateral occipital brim (near the TVP of atlas)
Tender Points: Prevalence of Cervicoscapular Tender Points in Tension-type and Migraine Patients: Distribution by the Number of Points

<table>
<thead>
<tr>
<th>Headache groups</th>
<th>No. of TPs</th>
<th>CM %</th>
<th>MCH %</th>
<th>Total</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>6</td>
<td>8</td>
<td>10</td>
<td>9</td>
</tr>
<tr>
<td></td>
<td>5</td>
<td>16</td>
<td>5</td>
<td>21</td>
</tr>
<tr>
<td></td>
<td>4</td>
<td>4</td>
<td>10</td>
<td>7</td>
</tr>
<tr>
<td></td>
<td>3</td>
<td>28</td>
<td>25</td>
<td>53</td>
</tr>
<tr>
<td></td>
<td>2</td>
<td>20</td>
<td>25</td>
<td>45</td>
</tr>
<tr>
<td></td>
<td>1</td>
<td>16</td>
<td>10</td>
<td>26</td>
</tr>
<tr>
<td></td>
<td>0</td>
<td>8</td>
<td>15</td>
<td>23</td>
</tr>
</tbody>
</table>

Spearman $r = .98$, $P < .05$.

- Suboccipital (C1–2)
- Midcervical (C2–3)
- Trapezius
- Levator scapulae

Table 16-2 shows the distribution of numbers of tender points (bilateral points counted as 1; therefore, the maximal count was 6) in the two headache groups. Eighty-five percent (85%) of TTH had at least one; 75%, at least two; and 50% had four or more. In the migraine group, these figures were 92%, 76%, and 44%, respectively, indicating a high prevalence of tender points in both groups. Table 16-3 shows the most prevalent locations, showing a strong (predictable) prevalence for the upper cervical region.

The findings of Watson and Trott (25), in which detection of joint dysfunction in headache (HA) subjects and non-HA subjects included pressure palpation for tenderness, were described above. Again, this procedure was found to have good intraexaminer reliability, and was found to distinguish HA subjects from non-HA subjects with higher prevalence of findings in the HA subjects.

Finally, tenderness to palpation has been used as one of the cardinal signs by Bogduk and his colleagues (20-23) to locate the level of zygapophyseal joint dysfunction potentially responsible for neck pain and headache. These tenderness findings correlate very well with the signs of joint hypomobility previously discussed. This combination of tenderness and hypomobility (as with Watson and Trott) correlate very highly with joint blockades used as a gold standard for diagnosis, and are used with greater frequency (35,36) as a successful therapy in headaches.

A finding associated with muscle tenderness is increased muscle stiffness. In 1992, Vernon and Gitelman (37) reported on a single case of bilateral TTH with cervical dysfunction. Pressure algometry showed clinically significant tenderness bilaterally in the suboccipital region. Muscle stiffness of the midcervical paraspinal and trapezius muscles was measured using Fischer’s tissue compliance meter (38). Higher than expected values were found in both muscle sites.

Tender Points: Distribution of Cervicoscapular Tender Points in Tension-type and Migraine Patients: Distribution by the Location of Points

<table>
<thead>
<tr>
<th>Location of TP</th>
<th>Headache groups</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>CM</td>
</tr>
<tr>
<td>Midcervical</td>
<td>91</td>
</tr>
<tr>
<td>Lateral-occipital</td>
<td>68</td>
</tr>
<tr>
<td>Suboccipital</td>
<td>64</td>
</tr>
<tr>
<td>Trapezius</td>
<td>55</td>
</tr>
<tr>
<td>Levator scapula</td>
<td>32</td>
</tr>
<tr>
<td>Medial-occipital</td>
<td>23</td>
</tr>
</tbody>
</table>

Spearman $r = 1.00$, $P < .05$.
Sakai et al (39) used a computerized compliance meter in a comparison of 37 TTH subjects and 63 normals. In 65% of HA subjects there was significantly increased trapezius stiffness, whereas the overall mean values (756 ± 121 versus 538 ± 89) significantly distinguished headache from control subjects. An orally administered muscle relaxant greatly reduced this increased stiffness in headache subjects, implying that active tension contributed to the stiffness.

It is evident that the more recent studies of myofascial dysfunction (pain/tenderness and stiffness) employ more sophisticated methodologies and instrumentation and are being conducted in case-control design formats. The findings of these studies even more strongly substantiate the clinical and empirical experience of a high prevalence of craniocervical tenderness in headache sufferers, which is strongly associated with other signs (including misalignment and joint hypomobility) of spinal segmental subluxation. Studies that also employ manipulation or joint blockade have, by the relief of symptoms obtained, further implicated cervicogenic joint dysfunction in the cause of headache.

**Radiologic Findings of Cervicogenic Dysfunction**

Pfaffenrath et al. (40) used a computer-aided method of analyzing segmental cervical motion on flexion-extension radiographs. They found a statistically higher incidence of restrictions at C0–C1 in cervicogenic headache subjects as compared with normal controls.

In a 1992 report on TTH and MWA subjects, a similar method that rated segmental movement against the normative data from Dvorak et al. was used (41). In addition, Penning’s method (42) was used for C0–C1 (with Fielding’s norms for flexion and extension at C0–C1). The mean ± 1 SD (standard deviation) for occipital flexion in both headache groups was 3.17° ± 2.24°; whereas for extension it was 10.6 ± 7.7°. The percentage of subjects showing reductions in motion below a lower cutoff for normal (-1 SD) was 90% for flexion and 70% for extension. These results agree with those of Pfaffenrath et al.

For the rest of the cervical spine, a pattern emerged of greater hypermobility at C1–C2, and hypomobility at C4–5–6.

Finally, regarding the neutral cervical curve, it was found that 77% of all subjects had a substantial reduction (quaialordotic or fully, even, or postural) or abnormal reversal of the cervical lordosis.

Nagasawa et al.’s (43) recent report adds further to our knowledge of changes in cervical function in headache sufferers as determined by x-ray analysis. They compared 372 TTH subjects with 225 normal controls. They found a statistically significant reduction of the neutral curve as measured by a “cervical spine curvature index” (14.6 ± 11.9% versus 19.4 ± 11.1%, P < .001) They also found that segmental instability was less frequent in TTH than in controls. Finally, they found a higher frequency of low-set (in other words, rounded) shoulders in TTH (57.5%) versus controls (41.8%, P < .01, χ² = 16.6). These findings confirm that TTH sufferers have significant cervical postural and segmental motion abnormalities, typical of subjects whose headaches would be labeled as “cervicogenic.”

**Cervical Posture, Muscular Weakness, and Range of Motion**

Watson and Trott (25) studied 60 subjects, 33 with recurrent cervical headache (a combination of TTH, MWA, and “cervicogenic” HA) and 33 controls. They studied: (1) the degree of anterior head carriage, which they measured photographically, (2) the isometric strength and endurance of the upper cervical flexor muscles, measured by strain gauge dynamometry, and (3) the presence of “joint dysfunction,” by combining manual palpatorv findings of restricted joint play, tenderness and stiffness (as described above).

They found a smaller mean angle of forward head position (FHP) in headache sufferers (that is, a straightened cervical spine). As well, headache sufferers had smaller strength values.
and smaller endurance values of upper cervical flexors. These findings correlated well with the degree of FHP. They concluded that FHP increases the load on the posterior muscles that rotate the head (occiput) backward to maintain the orthostatic horizontal position of the eyes. This, in turn, weakens the antagonist muscles (upper cervical flexors) and contributes to upper cervical joint and myofascial dysfunction, ultimately leading to upper cervical pain. This nociception reinforces the local muscle spasm (as found experimentally by Hu et al [12]) and creates a vicious cycle of pain, spasm, altered mechanics, pain, etc., all leading to the potential for referred cranial pain.

Further evidence for the development of regional muscular stiffness and reduced range of cervical motion comes from Kidd and Nelson's report (44), using a very simplistic observer's evaluation of neck ROM in 64 subjects, 37 with and 37 without benign headache. The headache sufferers more frequently had a reduction of two or more ranges of motion.

All of these findings combine to create a composite of cervicogenic dysfunction, much of which has been observed in noncervicogenic-type headache sufferers (for example, TTH and MWA). This profile includes regional alterations in anterior head posture, straightened cervical curve and low-set shoulders, regional muscular stiffness and reduced ROM, upper cervical subluxogenic signs including misalignment, joint hypomobility, and frequent segmental myofascial tenderness. The high potential for upper cervical pain to occur, not only unilaterally, but bilaterally, creates potent opportunities for cranial pain referral.

Summary
The literature on headaches of cervical origin has been reviewed, focusing on three areas: (1) the results of studies employing manipulation and facet or neural anesthetic blockade, (2) recent advances in the understanding of craniocephalic pain mechanisms, and finally (3) recent studies of cervicogenic dysfunction in several categories of headache, including TTH, MWA, and "cervicogenic." This chapter author believes the current IHS approach to "cervicogenic headache" is too narrow and will create many false misattributions, typically in the direction of underdiagnosing the cervicogenic component of a great many more benign headache conditions.

In conclusion, this chapter author would reiterate the 1988 (1) model of vertebrogenic headache, modifying it slightly, as follows:

... there are likely three categories of benign headache:

1. Those in whom the cervicogenic component is etiological. This group, ideally, will derive primary benefit from spinal manipulative therapy (SMT) or other treatments aimed at cervical dysfunction;
2. Those in whom the cervicogenic component is secondary but synergistic. This group ideally could derive significant benefit from SMT in conjunction with other therapeutic measures; and,
3. Those in whom the cervicogenic component is negligible, reactive or fully absent. This group would derive little if any benefit from SMT.

As in 1988, it is still the case today that only careful yet comprehensive research, taking full account of cervicogenic dysfunction, will ultimately determine the validity of this model.

References
39. Sakai F, Ebihara S, Horikava M, Akiyama M. Quantitative measurement of muscle stiffness in tension-type


Changes in Neck Electromyography Associated with Meningeal Noxious Stimulation

James W. Hu, Ph.D.*, Howard Vernon, D.C.,†, and Iskosi Tatourian, Ph.D.‡

ABSTRACT

Objective: To determine if the activity of jaw and neck muscles in a rat model is influenced by the application of small-fiber irritant mustard oil to meningeal/dural vascular tissues.

Design: Controlled animal experiment.

Setting: University neurophysiology laboratory.

Interventions: Applications of mineral oil (vehicle control) and mustard oil to exposed meningeal/dural vascular tissues.

Main Outcome Measure: Electromyographic (EMG) recordings from deep suboccipital muscles, bilaterally, and the left trapezius and left masseter muscles.

Results: Mineral oil evoked no EMG responses in any muscles. The incidences of mustard oil-evoked EMG increases were 100%, 100%, 89% and 78% for left deep neck, right deep neck, left trapezius and left masseter muscles, respectively. The durations of EMG responses were (mean ± SD) 19.2 ± 6.6 min, 17.3 ± 7.5 min, 14.5 ± 6.8 min and 12.7 ± 8.5 min, respectively.

Conclusions: These results document that meningeal/dural vascular irritation leads to sustained and reversible activation of neck and jaw muscles that may be related to the clinical occurrence of muscular tension and pain associated with certain types of headaches, particularly migraine. (J Manipulative Physiol Ther 1995; 18:577-81).

Key Indexing Terms: Electromyography (EMG), Muscles, Neck, Pain, Meningeal Tissue. Animal Study.

INTRODUCTION

The etiology of headache is unclear (1). Tension headache and cervicogenic injury-induced headache are common forms of headache and have been treated routinely by spinal manipulation (2). Involvement of meningeal/vascular tissue in tension headache or other forms of headache is uncertain (2), but the inclusion of neck muscle tension is a common clinical occurrence in many forms of headache. Anatomically, the peptide-rich, unmyelinated, C-afferent fibers innervate meningeal blood vessels (3); the central terminals of these trigeminovascular fibers are located within the trigeminal subnucleus caudalis (4). Activation of trigeminovascular fibers triggers neuronal responses within brain regions previously associated with the transmission of trigeminal (V) nociceptive information (4) and could potentially provoke abnormal muscle activities or tensions. Currently, there is no evidence to support a physiological or anatomical connection between neck muscle activity and meningeal/vascular abnormality. This connection could be one of the primary manifestations of the headaches, especially in the case of migraine. Moskowitz pointed out that (sterile/neurogenic) inflammation of meningeal tissue may be a cause of headache (4). Sumatriptan is especially effective against migraine headache (and somewhat effective on other types) because of its inhibitory action on the serotonin system; it blocks the neurogenic inflammation and nociceptive information transmission that is involved in the V pathway, including trigeminal subnucleus caudalis. Thus, hyperactivity in neck muscles may be an important clinical symptom associated with many forms of headache.

Our recent studies have provided evidence suggesting neuroplasticity of the V brainstem complex and of the brainstem mechanisms underlying muscle, temporomandibular joint (TMJ) and cutaneous (e.g., facial) pain (5–9). These single-unit recording studies have focused especially on the V subnucleus caudalis because it has been implicated in craniofacial deep pain mechanisms. These studies also suggest that deep (muscle and TMJ) nociceptive inputs are especially effective in inducing deep and cutaneous mechanoreceptive field (RF) expansions of nociceptive neurons, whereas the cutaneous nociceptive inputs induce only cutaneous RF expansion (10). These RF changes, which may be accompanied by increased spontaneous activity and excitability to evoke afferent inputs, seem to reflect a "central sensitization" (6, 7, 11, 12).

We have also demonstrated the presence of prolonged increased muscle activity [measured by electromyography (EMG)] associated with deep tissue injury and/or inflammation. These EMG studies have been carried out on anesthetized
rats to determine if the activity of the jaw and neck muscles can be influenced by the injection of the inflammatory irritant and C-fiber excitant mustard oil into deep paraspinal tissues surrounding the C1–C3 vertebrae (17). The most prominent EMG responses occurred in the neck muscles, as compared with those in the jaw muscles. The increased EMG response evoked by mustard oil was frequently reflected in two phases of enhanced activity. The early phase of the increase in EMG activity was initiated immediately (< 20 sec) after the mustard oil injection and lasted 1–3 min. The second phase occurred 5–10 min later and lasted 10–15 min. Histological examination showed that in the tissues between fibers of the rectus capitis posterior muscle, the mustard oil (but not the vehicle, mineral oil) injection induced an inflammatory reaction characterized by extravasation, congestion, and invasion of polymorphonuclear leukocytes. These results provide documentation that inflammatory irritation by mustard oil in the deep paraspinal tissues results in a sustained and reversible activation of both jaw and neck muscles. Similar findings have also demonstrated the increase of muscle activity after the injection of mustard oil into the TMJ region and involvement of opioid suppressive mechanisms to limit the duration of this mustard oil–induced EMG activity increase (13).

The aim of this study is to examine the relationship between meningeal noxious stimulation (by meningeal application of mustard oil) and neck and jaw muscle activity. Thus, the viscerosomatic reflex may provoke muscle tension and, in association with neck joint and muscle injury, may be amenable to treatment (15). Some of these data have been briefly presented in abstract form (16).

**METHODS**

The description of the various methods to be used can be found in previously related papers (13, 14, 17). Only a brief description relevant to this study follows and any details not found in those papers will be emphasized below. Thirteen Sprague-Dawley rats were initially anesthesia with halothane. Tracheotomy was performed to allow the use of a gas anaesthetic machine. A mixture of 1/3 O<sub>2</sub>, 2/3 N<sub>2</sub>O and 2% halothane was used during the surgery. A venous cannula was inserted. A wide range of body parameters were monitored and kept within an acceptable physiological range, including heart rate, expired CO<sub>2</sub>, pupil size, body temperature and skin color. Next, the rat was put onto a stereotaxic frame and two screws were inserted into the exposed dorsal surface of the skull. A cranial cap that was connected to the stereotaxic apparatus was put in place to allow contact with the screws and then fixed in place with dental cement so that the stereotaxic ear and incisor bars could be removed. A hole (anterior to Bregma, 2 x 3 mm in the midline) was opened on the top of the skull to apply mustard oil. This hole was covered immediately with mineral oil to keep the meningeal tissue viable. After the surgery was completed, the cranial cap provided support for the head, neck, and body in a posture natural for the rat and also allowed EMG recording. Bipolar EMG recording electrodes (32-gauge wire coated with teflon insulation except the 1-mm exposed tip) were inserted into deep neck muscle (suboccipital) bilateral left trapezius and left masseter muscle. Upon finishing surgery, the halothane concentration was reduced to 0.6–0.9% with 1 O<sub>2</sub> and 2/3 N<sub>2</sub>O. At this anesthetic plane, the rat is quiet and unresponsive to innocuous external stimuli, but retains a motor (reflexive) response to noxious stimuli (i.e., pinch hindpaw) separate control experiments, we have already shown that arousal or extraneous stimuli did not produce the prolong EMG increase after mustard oil was injected into temporalis diurnal joint tissues, and the EMG pattern demonstrated stable state over 4 hr without any spontaneous discharge pattern (15). Baseline recordings of 20 min from each muscle group were obtained after a waiting period of at least 60 min without any surgical interventions. The standard intervention consists of a bolus of mustard oil (98% allyl-isothiocyanate 0.5 μl; BDH, Poole, UK) applied into the skull hole after mineral oil is temporarily removed. Mineral oil application similar meningeal tissue served as a control. At the end of each experiment, Evans Blue dye (5 mg/kg) was injected through the cannula in the vein; 10 min later, a euthanasis agent (10 Hoehchst) was injected. The mustard oil application site was visually localized.

The mustard oil was administered after 20 min baseline data had been taken. EMG responses were obtained for an additional 30–60 min after the mustard oil injection. EMG recordings of multiple muscles were recorded continuously on computer-interface system (CED 1401Plus/Spike 2 system). This system can digitize, rectify, integrate and store multiple channel EMG signals on a real time basis. These multiple channel EMG data were displayed on the computer screen ensure that any short-term effects of mustard oil injection were missed. Overall results were analyzed offline. Each 1-min record was processed as a data point for statistical comparisons in accordance with our recent publications (13, 14, 17). Occasionally, the EMG electrodes were not working properly, therefore, only those rats that demonstrated at least three more proper EMG recordings were used for data analysis. Thus, the number of each muscle tested under different experimental conditions would be different. The mean value (± SE) of those baseline data in each of the muscles would be calculated as 100% and the increase of EMG activity will be expressed as the percent of this mean value. Both area, latency and duration data counted for those EMG electrodes that were producing positive effects, i.e., 2 SD above the mean EM level values from 20 1-min data points.

**RESULTS**

The EMG activity in each of the muscles recorded showed clearly the nature of the multiple motor unit activities (example, Figure 1). For all rats tested, there was no significant variation in EMG activities in any neck and masseter muscle during the initial 20 minute period before any application of mustard oil into the meningeal tissues (Figure 1). Neither initial application nor repeated replacement of mineral oil produced any EMG activities. Mustard oil application alone (n = 9) evoked an increase of EMG activity in at least two of
Fig. 1 An example of EMG changes evoked by application of mustard oil to the meningeal/vascular tissue in one rat. Note the multiple motor unit activities in each of the muscles tested here. Initial large response amplitude is made from the sum of these multiple unit activities whereas the gradual decreased amplitude is caused by the cease of individual motor unit activity. The solid line indicates the application time of mustard oil to meningeal tissue.

three neck muscles (ipsilateral or contralateral deep neck or trapezius muscles) as well as the masseter muscle (Figure 1). The incidences of mustard oil-induced EMG responses was similar in all muscles (χ² test, p > .05); 100% (9 of 9), 100% (9 of 9), 89% (8 of 9) and 78% (7 of 9) for left deep neck, right deep neck, left trapezius and left masseter muscles, respectively (Table 1). The magnitude of EMG activity increases (i.e., the area under the curve) was larger in the neck muscles (both deep neck and trapezius M.) than jaw muscle (masseter M.) (Figure 2). The durations of EMG responses were (mean ± SD) 19.2 ± 6.6 min, 17.3 ± 7.5 min, 14.5 ± 6.8 min and 12.7 ± 8.5 min, respectively. The latencies of EMG responses were 1.2 ± 2.2 min, 2.8 ± 3.7 min, 2.1 ± 3.1 and 1.3 ± 1.8 respectively. No difference was found between different muscles for both latency and duration data (ANOVA, p > .05). The latency data yielded very different results from those observed in TMJ-injected, mustard oil-induced EMG responses, which usually had short (< 10 sec) and synchronized activation from different jaw and neck muscles (13). In 22% (2 of 9) of rats, mustard oil evoked only one deep neck muscle increase of EMG activity without the activities from contralateral deep neck muscle within a 5-min period. Eighty-nine percent (8 of 9) of rats evoked a bilateral neck muscle increase of EMG activities. In pooled data from 31 of 36 possible EMG recording sites (i.e., these sites showed EMG increases over 2 SD of control-period values), 19% of them showed a brief early-phase EMG response, (i.e., EMG activities lasted less than 10 min), 65% showed a prolonged early phase (up to 30 min continuous response), 13% showed clearly discerned early- and late-phase responses, and 3% showed only late-phase responses. Compared with the baseline level, the mean peak amplitude of the mustard oil-evoked EMG activity of the left neck, right neck, left trapezius and left masseter muscles were 838 ± 851%, 641 ± 482%, 726 ± 476% and 271 ± 189% respectively. Please note the remarkable large trapezius EMG responses. Figure 2 shows the averaged EMG response patterns in all four muscles from all nine rats.

Discussion

This study has provided documentation that application of a small-fiber excitant and inflammatory irritant to meningeal tissues results in increased EMG activity in the neck and jaw muscles. No increase in EMG activity was found when the mineral oil was applied. Especially large EMG responses were found in the trapezius muscle. The results establish physiological connections between abnormality of the meningeal tissue and the neck muscular activities. These connections may contribute to the clinical manifestation of headaches. As well, the general implication of these results is to support the long-held theory that visceral nociception induces spinal somatic reactions in a segmentally organized fashion (19–21). These reactions have been termed “somatic dysfunction” and consist, primarily, of increased paravertebral muscular activity along with local tenderness. To chiropractors and osteopaths, these reactions in the spinal paravertebral tissues provide clues not only to local segmental dysfunction, but also to distal visceral disorders as well (22).

Our present study demonstrates similar effects of other jaw and neck EMG activity increases induced by mustard oil-injection/application into TMJ (14), tooth pulp (8) and paraspinal neck region (17). The time course of meningeal/vascular mustard oil-induced effects is similar to those previous results; however, there are two important differences: incidence and latency. In some cases, a muscle-recording electrode pair did not generate any EMG activity when mustard oil was first applied to the meningeal/vascular tissues but showed either a late-phase response (8%) or a clear EMG response when the opiate antagonist naloxone was injected intravenously to test the opioid receptor involvement in many of the rats of this study (these results will be reported in a separate communication). Unlike previous synchronized mustard oil-induced EMG activity changes in different muscles (14, 17), present results show longer latencies and variable response patterns (Figure 1). For example, Yu et al. showed the mustard oil-induced EMG activities had latencies less than 4 sec (mean) in all EMG electrodes, whereas only late-phase EMG responses can be demonstrated in some cases (8%) (14). There are two possible explanations: either the meningeal afferent terminals within dural or vascular tissue are extremely sparse, which may require temporal and spatial summation to reach the critical central exciting state that initiates the EMG activity, or the neural pathway involved in this meningeal/vascular nociceptive transmission may require higher threshold activation.

In this study, mustard oil quickly (latency < 3 min) induces neck muscle activities; thus, it is unlikely that meningeal/vascular inflammation plays an important role. Rather, the
Table 1. EMG increases evoked by mustard oil application to meningeal/vascular tissue

<table>
<thead>
<tr>
<th>Muscle</th>
<th>Incidence</th>
<th>Area (mV·sec)*</th>
<th>Duration (min)</th>
<th>Latency (min)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Left deep neck</td>
<td>100%</td>
<td>21.4 ± 21.1</td>
<td>19.2 ± 6.6</td>
<td>1.2 ± 2.2</td>
</tr>
<tr>
<td>Right deep neck</td>
<td>100%</td>
<td>13.6 ± 13.3</td>
<td>17.3 ± 7.5</td>
<td>2.8 ± 7.7</td>
</tr>
<tr>
<td>Left trapezius</td>
<td>89%</td>
<td>8.5 ± 8.0</td>
<td>14.5 ± 6.8</td>
<td>2.1 ± 1.1</td>
</tr>
<tr>
<td>Left masseter</td>
<td>78%</td>
<td>4.2 ± 5.5</td>
<td>12.7 ± 8.5</td>
<td>1.3 ± 1.8</td>
</tr>
</tbody>
</table>

* Values under area, duration and latency are represented as mean ± SD.

inflammation activates small-diameter afferent fibers distributing in either meningeal or vascular tissues. Moskowitz proposed that the sterile/neurogenic inflammation of meningeal tissue may be a cause of headache, especially in cases of migraine (4). The prolonged inflammatory process that was triggered by mustard oil may cause the release of inflammatory mediators, such as substance-P (bradykinin), to a certain critical concentration that activates the nociceptive afferents. Thus, inflammation may have a role in the activation of the nociceptive afferent; however, this effect may take much longer (> 30 min) than the time span employed in this study.

Because the pattern and time course of the meningeal/vascular activation by noxious stimulation is similar to those seen in TMJ (14), tooth pulp (8) and the paraspinal neck region (11), the most likely explanation for this EMG activity is the result of the "central sensitization" (6, 7, 11, 12) of trigeminal second order nociceptive neurons (7, 8). After excitation, the EMG activity may be subjected to an endogenous modulatory process, such as opioid suppressive mechanisms (13), to reduce its activity back to the baseline.

Furthermore, meningitis caused by bacterial infection produces inflammation in the meningeal/vascular tissue, and the physician depends upon the subtle signs of meningitis, such as a feeling of stiffness in the neck (23). Our present results also show the implication of the stiff neck meningitis symptom. It is interesting to find that the trapezius muscle peaks at a higher peak (normalized) of mustard oil-induced effects on other muscles. The reason for this finding is still unclear; a possible reason is that these deep neck muscles are engaged in posture maintenance; thus, these muscles show a higher tonic activity, whereas the trapezius remains present. This difference in muscle activity between different muscles may explain the present results. Meningeal/vascular inflammation induced by mustard oil may result in activation of small-diameter nerves in associated dural/vascular tissues. In turn, trigeminal, spinal-subnucleus, caudalis is activated and triggers a neural pathway to activate the C1-C3 neuronal pool. The existence of this latter pathway is speculative; however, this study provides solid experimental evidence to demonstrate this reflex pathway (14). We still have to apply local anesthetics to the dural/vascular tissue before application of mustard oil to prove that this EMG activity is reflex-based.

ACKNOWLEDGMENTS

We are grateful to K. MacLeod for electronic data assistance and to R. Attaran who participated in some experiments.

REFERENCES


Involvement of NMDA receptor mechanisms in jaw electromyographic activity and plasma extravasation induced by inflammatory irritant application to temporomandibular joint region of rats


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Summary The aim of this study was to examine the possible role of N-methyl-D-aspartate (NMDA) receptor mechanisms in responses induced by the small-fibre excitant and inflammatory irritant mustard oil injected into the temporomandibular joint (TMJ) region of rats. The effects of the non-competitive NMDA antagonist MK-801 were tested on the mustard oil-evoked increases in electromyographic (EMG) activity of the masseter and digastric muscles and Evans Blue plasma extravasation. Five minutes before the mustard oil injection, MK-801 or its vehicle was administered systemically (i.v.), into the third ventricle (i.c.v.), or locally into the TMJ region. Compared with control animals receiving vehicle, the rats receiving MK-801 at an i.v. dose of 0.5 mg/kg (n = 5) showed a significant reduction in the incidence and magnitude of EMG responses as well as in the plasma extravasation evoked by mustard oil; MK-801 at an i.v. dose of 0.1 mg/kg (n = 5) had no significant effect on plasma extravasation or on the incidence and magnitude of EMG responses but did significantly increase the latency of EMG responses. An i.c.v. dose of 0.1 mg/kg (n = 5) or 0.01 mg/kg (n = 5) had no significant effect on plasma extravasation or incidence of EMG responses but did significantly reduce the magnitudes of the masseter EMG response; the 0.01 mg/kg dose also significantly increased the latency of the digastric EMG response. The magnitudes of both the masseter and digastric EMG responses were also significantly reduced by MK-801 administered into the TMJ region at a dose of 0.1 mg/kg (n = 5) but not by 0.01 mg/kg (n = 5); neither dose significantly affected the incidence of EMG responses or the plasma extravasation. These data suggest that both central and peripheral NMDA receptor mechanisms may play an important role in EMG responses evoked by the small-fibre excitant and inflammatory irritant mustard oil, but that different neurochemical mechanisms may be involved in the plasma extravasation induced by mustard oil.

Key words: EMG; Temporomandibular joint; Jaw muscles; NMDA; Inflammation

Introduction

Excitatory amino acids (EAAs) are major excitatory neurotransmitters in the mammalian central nervous system (CNS). Receptors for EAAs are broadly classified into metabotropic and ionotropic types (for review, see Collingridge and Lester 1989; Monaghan et al. 1989; Nakanishi 1992). One ionotropic EAA receptor subtype is the N-methyl-D-aspartate (NMDA) receptor, and there are two non-NMDA receptor subtypes, kainate and α-amino-3-hydroxy-5-methyl-4-isoxazolepropionate (AMPA). Both NMDA and non-NMDA receptors have been found to exist not only in the CNS but also in the peripheral nervous system (Shigemoto et al. 1992; Berger et al. 1995; for review, see Collingridge and Lester 1989; Monaghan et al. 1989; Erdö 1991; Nakanishi 1992). Functionally, non-NMDA receptor mechanisms are implicated in generating fast excitatory post-synaptic potentials in the CNS, whereas NMDA receptor mechanisms mediate more prolonged excitatory post-synaptic potentials (for review, see Collingridge and Lester 1989; Monaghan et al. 1989; Nakanishi 1992) and are considered of primary importance in nociceptive transmission in ascending somatosensory pathways, particularly in 'central sensitization' states that can be evoked by inflammation and other conditions associated with increased small-fibre nociceptive afferent inputs into the CNS (Aanonsen and Wilcox 1987; Dickenson and Sullivan...
MK-801 administration

MK-801 (NMSD, Harlow) was administered into the right external jugular vein (n = 10), or the third ventricle (i.c.v., n = 10) or the TMJ region (n = 10) 5 min before the injection of mustard oil. For the i.v. administration, MK-801 was given in a dose of 0.1 mg/kg (n = 5) or 0.5 mg/kg (n = 5) in 0.3 ml saline. The vehicle saline was administered in an equal volume (0.3 ml) in another six (control) animals. For the i.c.v. administration, a 30-gauge cannula was connected to a 20 μl Hamilton syringe with a polyethylene tube, mounted in a stereotaxic holder, and introduced stereotactically into the third ventricle before the animal’s cranium was fixed to the stereotaxic apparatus (see above). The tip of the cannula was positioned at the coordinates AP ~0.08, L 0, and V ~3 (Paxinos and Watson 1986) and the cannula was fixed in place with dental cement. After the experiment, the correct placement of the cannula in the third ventricle was visually assessed by determining the location of the cannula and the distribution of Evans Blue solution (10 μl) injected through the cannula. In each case, the dye was found to extend over the entire cerebroventricular system and the cerebral isthmuses. The MK-801 was given in a dose of 0.01 mg/kg (n = 5) or 0.1 mg/kg (n = 5) in 10 μl artificial cerebrospinal fluid (ACSF). The vehicle ACSF alone was also administered in an equal volume (10 μl) in another six (control) rats. The ACSF consisted of 140 mM NaCl, 3 mM KCl, 2.5 mM CaCl2, 1 mM MgCl2, 1.2 mM NaH2PO4, 0.27 mM NaH2PO4, and 3 mM glucose, at a pH of 7.2–7.3 (Philippe 1984).

For the local TMJ administration of MK-801, two 30-gauge cannula were first cemented side-by-side and connected to two Hamilton-syringes with polyethylene tubes. One cannula was filled with either MK-801 (n = 10) or vehicle saline (n = 6) and the other one with mustard oil. They were then implanted into TMJ on the left side. The MK-801 was given in a dose of 0.01 mg/kg (n = 5) or 0.1 mg/kg (n = 5) in 20 μl saline. For control animals (n = 6), the vehicle was administered in an equal volume (20 μl).

EMG recording

Bipolar recordings were made of the EMG activities of the left e […

EMG data processing

The recorded EMG data was processed offline and analyzed as previously described (Hu et al. 1993; Yu et al. 1994a, 1995). In brief, the on under the rectified EMG curve for every second as well as for each 60 sec period was measured. The mean value of EMG activity for each muscle recorded in the initial 20 min in each animal was regarded as the baseline level of resting EMG activity for the muscle. Increases in EMG activity were determined by a change above the baseline level if one or more EMG data points rose two standard deviations above this baseline value. All data points were normalized relative to this mean value and expressed as a percentage of the mean baseline level of EMG activity and values reported as mean ± S.D. The time period from the beginning of the injection to the increase in EMG activity was designated as the latency of the response, and the time period from the increase in EMG activity to recovery to the baseline level of EMG activity was designated as the duration of the response. The Wilcoxon test, Mann-Whitney U-test, and Fisher test were used for statistical analysis. A probability level less than 0.05 (two-tailed) was regarded as significant.
Effects of MK-801 on mustard oil-evoked EMG responses

Consistent with our previous finding (Yu et al. 1994a, 1995), none of the animals revealed any variation in baseline EMG activities greater than two standard deviations (e.g. Figs. 1, 2 and 3) during the initial 20 min period prior to mustard oil injection into the TMJ region.

Plasma extravasation measurement

The plasma extravasation was measured as previously described (Haas et al. 1992). In brief, Evans Blue dye (5 mg/kg, i.v.) was injected in each animal at 20 min following the mustard oil injection. At 10 min after the injection of Evans Blue solution, the animals were decapitated and tissues (1.5 x 1.5 cm) of the left and right TMJ regions were excised. The tissues were analyzed by spectrophotometric measurement of absorbance at 620 nm for the amount of Evans Blue dye present. In the present study, the amount of Evans Blue dye in the TMJ region receiving mustard oil was expressed as the dye value obtained by subtracting the dye amount of the contralateral control side from that of the ipsilateral side.

Results

Fig. 1. EMG responses evoked by mustard oil injection into the TMJ region in animals with i.v. pre-administration of saline (0.3 ml) or MK-801 at a dose 0.1 mg/kg or 0.5 mg/kg. The EMG activity is shown at 5 min intervals (except for the EMG activity immediately after mustard oil injection, where it is shown every minute for 5 min). Each data point represents the mean (±SD) of the normalized values relative to the baseline EMG activity (=100%) in each rat (in some cases, symbols cover the SD bars). Compared with the baseline EMG activity, the mustard oil injection evoked a significant \((P < 0.05, \text{Wilcoxon test})\) increase of EMG activity in ipsilateral digastric and masseter muscles of the animals with saline pre-administration. \(*P < 0.05\) and \(**P < 0.01\) (Mann-Whitney test) indicate statistically significant differences in the EMG response between the animals with the pre-administration of saline and MK-801 at the dose 0.5 mg/kg. \(\text{Ip.} = \text{ipsilateral}; \text{Cl.} = \text{contralateral}; \text{M.O.} = \text{mustard oil.}\)

Fig. 2. EMG responses evoked by mustard oil injection into the TMJ region in animals with pre-administration of ACSF (10 μl) or MK-801 at a dose 0.01 mg/kg or 0.1 mg/kg into the third ventricle. The EMG activity is shown at 5 min intervals (except for the EMG activity immediately after mustard oil injection, where it is shown every minute for 5 min). Each data point represents the mean (±SD) of the normalized values relative to the baseline EMG activity (=100%) in each rat (in some cases, symbols cover the SD bars). Compared with the baseline EMG activity, the mustard oil injection evoked a significant \((P < 0.05, \text{Wilcoxon test})\) increase of EMG activity in ipsilateral digastric and masseter muscles of the animals with ACSF pre-administration. \(*P < 0.05\) and \(**P < 0.01\) (Mann-Whitney test) indicates statistically significant differences in the EMG response between the animals with the pre-administration of ACSF and MK-801 at the dose 0.01 mg/kg into the third ventricle, and \(*P < 0.05\) and \(**P < 0.01\) (Mann-Whitney test) between the animals with the pre-administration of ACSF and MK-801 at the dose of 0.1 mg/kg.

Abbreviations as in Fig. 1.
TABLE I
THE MUSTARD OIL-EVOKED EMG RESPONSES AFTER I.V. PRE-ADMINISTRATION OF SALINE OR MK-801

<table>
<thead>
<tr>
<th>Incidence of the EMG response (%)</th>
<th>Peak magnitude of the EMG response (%)</th>
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<tbody>
<tr>
<td></td>
<td>Saline (n = 6)</td>
</tr>
<tr>
<td>I.p. digastric</td>
<td>100 (6)</td>
</tr>
<tr>
<td>I.p. masseter</td>
<td>100 (6)</td>
</tr>
<tr>
<td>C.t. masseter</td>
<td>50 (3)</td>
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<tr>
<td></td>
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Saline (n = 6) 0.1 mg/kg (n = 5) 0.5 mg/kg (n = 5)

<table>
<thead>
<tr>
<th>Incidence of the EMG response (%)</th>
<th>Peak magnitude of the EMG response (%)</th>
</tr>
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<tbody>
<tr>
<td></td>
<td>ACSF (n = 6)</td>
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<tr>
<td>I.p. digastric</td>
<td>100 (6)</td>
</tr>
<tr>
<td>I.p. masseter</td>
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<tr>
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</table>

ACSF (n = 6) 0.01 mg/kg (n = 5) 0.1 mg/kg (n = 5)

n = number of animals tested. Numbers in brackets refer to the number of animals showing an increase in EMG activity after mustard oil injection. Mavitudes of the EMG responses to mustard oil injection are expressed as percentages of the EMG baseline and given as mean ± SD. I.p. = ipsilateral. C.t. = contralateral.

†P < 0.05 (Fisher test) indicates that the EMG magnitude is significantly smaller than that in the animals pre-administered with MK-801, and
‡P < 0.05 (Mann-Whitney test) indicates that the EMG magnitude is significantly smaller than that in the animals pre-administered with vehicle (saline).

**I.v. administration**

Neither vehicle nor MK-801 (i.v.) alone induced any significant changes in EMG activity when compared with baseline. However, an increase in EMG activity was noted in one animal after the vehicle administration, and in one and three animals after 0.1 and 0.5 mg/kg MK-801, respectively; these insignificant EMG activity increases lasted only 2-5 min.

The mustard oil injection following the i.v. pre-administration of the vehicle saline (n = 6) evoked a significant (P < 0.05, Wilcoxon test) increase in EMG activity in the ipsilateral digastric and masseter muscles of each animal (Table I and Fig. 1). The mean latencies of the mustard oil-evoked EMG activity in the ipsilateral digastric, ipsilateral masseter, and contralateral masseter were 3.5 ± 0.8, 3.8 ± 1.6, and 5.3 ± 2.3 sec, respectively, and the mean peak amplitudes (Table I) were 434 ± 157%, 331 ± 262%, and 144 ± 90%.

Compared with the EMG response in the animals receiving pre-administration of vehicle (saline), the pre-administration of 0.1 mg/kg MK-801 (i.v.) did not significantly affect the incidence and magnitude of the mustard oil-evoked EMG response, although the latencies of the EMG response in the ipsilateral digastric and masseter muscles were significantly (P < 0.01 and P < 0.05, respectively, Mann-Whitney test) increased to 10 ± 5.0 and 32 ± 49.4 sec, respectively. Pre-administration of 0.5 mg/kg MK-801 did, however, significantly (P < 0.05, Fisher) reduce the incidence of the mustard oil-evoked EMG response in the ipsilateral digastric and the magnitude of EMG response in the ipsilateral masseter as well as in a contralateral masseter (Fig. 1 and Table I).

**I.c.v. administration**

Consistent with the finding for the i.v. administration MK-801, neither vehicle nor MK-801 i.c.v. alone induced any significant changes in EMG activity when compared with baseline. However, an increase in EMG activity was noted in one animal after the vehicle administration, and one and two animals after 0.01 and 0.1 mg/kg MK-801, respectively; these insignificant EMG increases lasted 1-2 min.

Following the i.c.v. pre-administration of ACSF for mustard oil injected into the TMJ region evoked a significant increase in EMG activity (P < 0.05, Wilcoxon test; Fig. 2) in all the jaw muscles of each animal when compared with baseline. The mean latencies of mustard oil-evoked EMG activity in the ipsilateral digastric, ipsilateral masseter, and contralateral masseter muscles were 3.8 ± 0.8, 4.5 ± 1.5, and 16.8 ± 21.2 sec, respectively, and the mean peak amplitudes (Table II) were 413 ± 280%, 431 ± 167%, and 179 ± 70%.

TABLE II
THE MUSTARD OIL-EVOKED EMG RESPONSES AFTER I.C.V. PRE-ADMINISTRATION OF ACSF OR MK-801

<table>
<thead>
<tr>
<th>Incidence of the EMG response (%)</th>
<th>Peak magnitude of the EMG response (%)</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>ACSF (n = 6)</td>
</tr>
<tr>
<td>I.p. digastric</td>
<td>100 (6)</td>
</tr>
<tr>
<td>I.p. masseter</td>
<td>100 (6)</td>
</tr>
<tr>
<td>C.t. masseter</td>
<td>100 (6)</td>
</tr>
<tr>
<td></td>
<td></td>
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<td></td>
<td></td>
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</tbody>
</table>

ACSF (n = 6) 0.01 mg/kg (n = 5) 0.1 mg/kg (n = 5)

n = number of animals tested. Numbers in brackets refer to the number of animals showing an increase in EMG activity after mustard oil injection. Mavitudes of the EMG responses to mustard oil injection are expressed as percentages of the EMG baseline and given as mean ± SD. Abbreviations in Table I.

*P < 0.05 and †P < 0.01 (Mann-Whitney test) indicate that the EMG magnitude is significantly smaller than that in the animals pre-administered with vehicle (saline).
TABLE III
THE MUSTARD OIL-EVOKED EMG RESPONSES AFTER TMJ PRE-ADMINISTRATION OF SALINE OR MK-801

<table>
<thead>
<tr>
<th>Incidence of the EMG response (%)</th>
<th>Peak magnitude of the EMG response (%)</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Saline (n = 6)</td>
</tr>
<tr>
<td>p. digastric</td>
<td>100 (6)</td>
</tr>
<tr>
<td>Ip. masseter</td>
<td>100 (6)</td>
</tr>
<tr>
<td>Ct. masseter</td>
<td>100 (6)</td>
</tr>
</tbody>
</table>

n = number of animals tested. Numbers in brackets refer to the number of animals showing an increase in EMG activity after mustard oil injection. Magnitudes of the EMG responses to mustard oil injection are expressed as percentages of the EMG baseline and given as mean ± SD. Abbreviations as in Table I.

*P < 0.05 and **P < 0.01 (Mann-Whitney test) indicate that the EMG magnitude is significantly smaller than that in the animals pre-administered with vehicle (saline).

Compared with the EMG response in the animals receiving pre-administration of vehicle (ACSF), pre-administration of 0.01 mg/kg MK-801 (i.c.v.) did not significantly reduce the incidence and magnitude of mustard oil-evoked EMG response in the ipsilateral digastric muscle, although the latency of the EMG response in this muscle significantly (P < 0.05, Mann-Whitney test) increased to 7.8 ± 3.1 sec. However, this dose did significantly reduce the magnitude of the EMG response in the ipsilateral masseter muscle. With the increase of the MK-801 dose to 0.1 mg/kg, a significant (P < 0.05, Mann-Whitney test) reduction was again noted in the magnitude of the mustard oil-evoked EMG response in the ipsilateral masseter muscle (Fig. 2 and Table II).

TMJ administration
Compared with baseline, administration of either the vehicle saline (n = 6) or MK-801 into the TMJ region (n = 10) did not evoke any significant change in EMG activity (P > 0.05, Wilcoxon test). However, an increase above the baseline level of EMG activity was noted in four animals after the vehicle administration, and in two and three animals after 0.01 and 0.1 mg/kg MK-801, respectively; these insignificant EMG increases lasted 2–5 min.

Compared with baseline, the mustard oil injection following the pre-administration of saline into the TMJ region evoked a significant increase of EMG activity (P < 0.05, Wilcoxon test; Fig. 3) in all three jaw muscles of each animal (Table III). The mean latencies of the mustard oil-evoked EMG activity in the ipsilateral digastric, ipsilateral masseter, and contralateral masseter were 5.3 ± 3.9, 5.4 ± 4.0 and 7.2 ± 3.9 sec, respectively, and the mean peak amplitudes (Table III) were 392 ± 164%, 485 ± 176%, and 306 ± 70%.

Compared with the EMG response in the animals receiving pre-administration of vehicle (saline), the pre-administration of MK-801 at a dose 0.01 mg/kg into the TMJ region did not significantly reduce the incidence and magnitude of the mustard oil-evoked EMG response in any of the three muscles, although a reduction in the mean amplitudes of the EMG response could be noted (Fig. 3). The pre-administration of 0.1 mg/kg MK-801 did significa-

Fig. 3. EMG responses evoked by mustard oil injection into the TMJ region in animals with pre-administration of saline (20 μl) or MK-801 at a dose 0.01 mg/kg or 0.1 mg/kg into the TMJ region. The EMG activity is shown at 5 min intervals (except for the EMG activity immediately after mustard oil injection, where it is shown every minute for 5 min). Each data point represents the mean (±SD) of the normalized values relative to the baseline EMG activity (×100%) in each rat (in some cases, symbols cover the SD bars). Compared with the baseline EMG activity, the mustard oil injection evoked a significant (P < 0.05, Wilcoxon test) increase of EMG activity in all jaw muscles of the animals with saline pre-administration. *P < 0.05 and **P < 0.01 (Mann-Whitney test) indicate statistically significant differences in the EMG response between the animals with the pre-administration of saline and MK-801 at the dose of 0.1 mg/kg into the TMJ region. Abbreviations as in Fig. 1.
I.V.

**Effects of MK-801 on the mustard oil-evoked plasma extravasation**

Consistent with our previous findings (Haas et al. 1992; Hu et al. 1993; Yu et al. 1995), mustard oil induced significant plasma extravasation (**P** < 0.05, Wilcoxon test) when compared with the contralateral TMJ region receiving no mustard oil injection. In comparison to the animals receiving i.v. administration of saline, animals receiving the systemic administration of 0.5 mg/kg MK-801 showed a significant reduction in the mustard oil-evoked plasma extravasation (**P** < 0.05, Mann–Whitney test; Fig. 4A) but 0.1 mg/kg had no such effect. Neither i.c.v. (Fig. 4B) nor TMJ (Fig. 4C) administration of MK-801 at the doses used significantly reduced plasma extravasation.

**Discussion**

It is well known that MK-801 is a potent non-competitive NMDA receptor antagonist which binds to the cationic channel and prevents ion fluxes, and that only a very low dose of MK-801 is required to produce an almost complete block of NMDA-induced responses (for review, see Collingridge and Lester 1989; Wong and Kemp 1991). The present study has demonstrated that MK-801 may significantly reduce digastric and masseter EMG responses reflexly evoked by the small-fibre excitant and inflammatory irritant mustard oil injected into the TMJ region. The effective doses of MK-801 administered into the TMJ region and the third ventricle were considerably lower than the effective dose when MK-801 was administered systematically and point to both a peripheral and a central involvement of NMDA receptor mechanisms in the EMG responses evoked by mustard oil injected into deep tissues.

Our finding that systemic administration of MK-801 at a dose of 0.5 mg/kg, but not 0.1 mg/kg, significantly reduces mustard oil-evoked EMG responses is consistent with the effects of the drug on the reflexly evoked activity of motoneurons in the spinal cord (Woolf and Thompson 1991). However, behavioral studies have indicated that a high dose of NMDA antagonists (e.g. >0.3 mg/kg MK-801) may produce circling behaviour, head weaving and paralysis of the hindlimb characterized by an increase in extensor tone (Cahusac et al. 1984; Ren et al. 1992). Since a muscle with increased tone may show an increase in EMG activity (Douglas et al. 1989; Mense 1993), and we noted that MK-801 itself did not produce a significant change in EMG activity (Douglas et al. 1989; Mense 1993), and we noted that MK-801 itself did not produce a significant change in EMG activity of either group of muscles that are analogous to limb extensor muscles (i.e. masseter) or limb flexor muscles (i.e. digastric) (Dubner et al. 1978; Hannam and Sessle 1994), it would appear that the MK-801 effect on the mustard oil-evoked EMG activity could not be explained by change in muscle tone induced by MK-801 per se.

Administration of 0.01 mg/kg MK-801 into the third ventricle significantly reduced the magnitude of the mustard oil-evoked EMG response in the ipsilateral masseter and significantly increased the latency of the EMG response in the digastric muscle. A similar dose administered intrathecally is required for blocking both the first and second phases of appearance of flinching behaviour induced by formalin injected into subcutaneous tissues (Yamamoto and Yaksh 1992). However, the magnitude of the mustard oil-evoked EMG response in the digastric muscle was not significantly reduced by i.c.v. administration of MK-801 even at the dose of 0.1 mg/kg. There have been described differential facilitatory or inhibitory effects of descending modulatory influences on masseter and digastric motoneurons (for review, see Dubner et al. 1978) and so it is possible that the i.c.v. application of MK-801 differentially affected one of these descending inputs. If the site of NMDA action on digastric activity is in the brainstem (e.g. trigeminal subnucleus caudalis or trigeminal motor nucleus...)
while its action on masseter activity involves other inputs (e.g. from higher brain centres), this could explain why i.v. administration of MK-801 reduced the EMG response in both the digastric and masseter muscles while i.c.v. application of MK-801 only significantly reduced the magnitude of the EMG response in the masseter muscle. NMDA receptor mechanisms do appear to be involved in the control of motoneurons in the spinal cord (Bohmer et al. 1991; Durand 1993; Pinco and Lev-Tov 1993, 1994) and trigeminal motor nucleus (Katakura and Chandler 1991; Boucher et al. 1993). For example, it has been reported that NMDA receptor antagonists applied to the trigeminal brainstem sensory complex significantly reduce the amplitude of the jaw-opening reflex evoked by electrical stimulation of the rat tooth pulp (Boucher et al. 1993), and Katakura and Chandler (1991) have reported that intraoral afferent-evoked activity of motoneurons innervating the guinea pig digastric muscle is regulated by NMDA receptor mechanisms, but the activity of masseter motoneurons evoked by stimulation of the trigeminal mesencephalic nucleus does not appear to involve such mechanisms (Chandler 1989). The difference between our findings indicating NMDA receptor involvement and this latter observation with respect to the masseter might be explained by the different experimental models used. In the earlier experiments (Chandler 1989; Katakura and Chandler 1991) the use of trigeminal mesencephalic nucleus stimulation tested the involvement of NMDA receptor mechanisms only in the pathway from low-threshold (i.e. muscle spindle or periodontal ligament) trigeminal inputs to masseter motoneurons. In contrast, our experimental model, where the masseter response was evoked by the small-fibre excitant and inflammatory irritant mustard oil (Jancso et al. 1977; Woolf and Wall 1986; Handwerker and Reeh 1991), specifically tests for high-threshold afferent inputs to masseter motoneurons. Data from other experimental models suggest that NMDA receptor antagonists affect the central facilitatory state induced by noxious stimuli and are particularly effective on the protracted nociceptive processes associated with peripheral inflammation (Aanonsen and Wilcox 1987; Dickenson and Sullivan 1987; Yaksh 1989; Schaible et al. 1991; Woolf and Thompson 1991; Ren et al. 1992; Yamamoto and Yaksh 1992; for review, see Wilcox 1991; Dubner and Ruda 1992; Codera 1993; Meller and Gebhart 1993; Urban et al. 1994). Recent studies have also documented that noxious stimuli applied to the TMJ region (Broton and Sessle 1988; Yu et al. 1994a) or tooth pulp (Hu et al. 1994) can produce a sustained excitation of several masticatory muscles including the jaw-closing (e.g. masseter) as well as the jaw-opening (e.g. digastric) muscles. The present study suggests that NMDA receptor mechanisms may be differentially involved centrally in these responses of the jaw-opening and jaw-closing muscles to noxious stimuli.

MK-801 administered locally into the TMJ region may reduce the mustard oil-evoked EMG responses. It is unlikely that the effect of 0.1 mg/kg MK-801 administered into the TMJ region is produced by a systemic action, because this dose administered i.v. did not significantly reduce the mustard oil-evoked EMG responses. These findings appear to be the first to document that the NMDA antagonist MK-801 administered locally may reduce a nociceptive reflex, although the detailed mechanisms underlying the peripheral action of MK-801 remain to be clarified. Nonetheless, immunohistochemical studies have confirmed that NMDA receptors exist in primary afferent neurons (Shigemoto et al. 1992) and neuromuscular junctions (Berger et al. 1995), and ligand binding studies and other physiological and pharmacological investigations have also suggested that NMDA receptors may be located in peripheral tissues (for review, see Erdö 1991). Consistent with our previous findings (Haas et al. 1992; Yu et al. 1994a, 1995), we noted that mustard oil also induces an acute inflammatory response in the TMJ region. The plasma extravasation induced by mustard oil may be significantly reduced by depletion of C fibres (Jancso et al. 1977; Woolf and Wall 1986; Kwan et al. 1995) or by the application of local anaesthetic (Yu et al. 1995) and enhanced by the application of the peripherally acting opioid antagonist methylnaloxone (Haas, unpublished data; Yu et al. 1994a). These data suggest that a neurogenic component is involved in the mustard oil-induced inflammation (Jancso et al. 1977; Woolf and Wall 1986). We found that the systemic administration of 0.5 mg/kg MK-801 significantly reduced the plasma extravasation induced by mustard oil. The mechanisms underlying this effect, however, are not clear. One possibility is that the drug at a high systemic dose might influence the function of the adrenal gland and autonomic nerves involved in the control of vascular permeability (Codexe et al. 1989; Catania et al. 1991; Lam and Ferrell 1993; for review, see McCall 1988; Erdö 1991). Several other central mechanisms which may be involved in regulating the development of neurogenic inflammation have also been proposed, including descending inhibitory systems which can be activated by non-steroidal anti-inflammatory drugs applied to the CNS (Catania et al. 1991). However, the present study documented that neither i.c.v. administration nor local TMJ administration of MK-801 at the dose effective for reducing the mustard oil-evoked EMG responses reduces the plasma extravasation, and Sluka and Westlund (1993) found that the spinal administration of MK-801 significantly reduces carrageenan-induced hyperalgesia but not inflammation. These findings raise the possibility that separate neurochemical mechanisms may be involved in nociceptive responses and the inflammatory responses. This viewpoint is supported by several other lines of evidence: (i) the time courses of the EMG responses and the plasma extravasation induced by mustard oil are not parallel (Yu et al. 1994a, 1995); (ii) the doses of the opioid antagonists naloxone and methylnaloxone required for enhancing the EMG responses and the plasma extravasation are different (Yu et al. 1994a);
and (iii) the doses of the local anaesthetic lidocaine required for blocking the EMG responses and the plasma extravasation also are different (Yu et al. 1995).

The increased excitability of α-motoneurons induced by noxious stimulation and the expansion of the mechanoreceptive fields of nociceptive trigeminal brainstem neurons in subnucleus caudalis (medullary dorsal horn) induced by mustard oil injected into deep tissues may reflect a 'central sensitization' state (Woolf and Wall 1986; Hu et al. 1992, 1994; Woolf 1992; Sessle et al. 1993; Yu et al. 1993). The time course of the mustard oil-evoked EMG responses is similar to that of the mechanoreceptive field expansion of the nociceptive trigeminal caudalis neurons (Yu et al. 1993). Moreover, transection of the trigeminal subnucleus caudalis and trigeminal spinal tract below the obex level and ibotenic acid lesions of caudalis significantly reduces the mustard oil-evoked EMG responses in the masseter and digastric muscles (Tsai et al. 1994), and micro-stimulation of caudalis evokes EMG activity in these muscles (Tsai et al. 1995). These findings suggest that 'central sensitization' processes at the level of trigeminal subnucleus caudalis may be involved in the mustard oil-evoked EMG responses (Sessle et al. 1993; Hu et al. 1994).

These 'central sensitization' processes in subnucleus caudalis may involve NMDA receptor mechanisms since the mustard oil-evoked expansion of the mechanoreceptive field of nociceptive trigeminal caudalis neurons can be reduced by i.v. application of MK-801 (Yu, Hu and Sessle, unpublished data), and NMDA receptor mechanisms are considered of primary importance in 'central sensitization' states evoked by inflammation and other conditions associated with increased small-fibre nociceptive afferent inputs onto spinal dorsal horn neurons (Aanonsen and Wilcox 1987; Dickenson and Sullivan 1987; Yaksh 1989; Woolf and Thompson 1991; Dougherty et al. 1992; for review, see Dubner and Ruda 1992; Coderre 1993; Meller and Gebhart 1993; Urban et al. 1994). Activation of the NMDA receptor may increase the release of presynaptic excitatory neurotransmitters (Montague et al. 1994) and produce prolonged excitatory post-synaptic potentials in the CNS (for review, see Collingridge and Lester 1989; Monaghan et al. 1989; Nakaniishi 1992). Antagonism of the mustard oil-evoked 'central sensitization' by MK-801 in the present study may have then resulted, at least in part, from the blockade of the central release of excitatory neurotransmitters and/or the reduction of the excitability of central neurons (Aanonsen and Wilcox 1987; Dickenson and Sullivan 1987; Dougherty et al. 1992; Yu, Hu and Sessle, unpublished data; for review, see Dubner and Ruda 1992; Coderre 1993; Meller and Gebhart 1993; Urban et al. 1994), and in view of the potential sites involving NMDA receptor mechanisms noted above, this antagonism could involve actions within the trigeminal brainstem sensory or motor nuclei or at suprabulbar sites influencing the activity of neurons in these nuclei. In addition, however, the present study suggests that peripheral as well as central NMDA receptor mechanisms must also be considered in the reflex changes evoked in muscles by small-fiber nociceptive afferent inputs.

Acknowledgements

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The Neck Disability Index: Patient Assessment and Outcome Monitoring in Whiplash

Howard Vernon

SUMMARY. Whiplash associated disorders have pain and disability affecting multiple systems. A key issue is to determine, however possible, those problems that lead to disability, since physical dysfunction has a major effect upon adjustments to life. The current study has taken the Oswestry Low Back Disability Index, and with adaptations that consider the neck and whiplash, and using current language and social issues, have developed a Neck Disability Index [NDI]. This self-report index has been tested reliable and valid as a measure of neck disability. The NDI has been shown to be a simple tool that could improve clinical assessments of whiplash; it is duplicated here, and its duplication and use is encouraged. [Article copies available from The Haworth Document Delivery Service: 1-800-342-9678. E-mail address: getinfo@haworth.com]

KEYWORDS. Whiplash, disability, questionnaire, index

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INTRODUCTION

Injuries to the neck, and, in particular, those originating from a motor vehicle accident, constitute a significant burden to the health care system. Lifetime prevalence estimates for neck pain in general range from 45-71% (1,2,3), while the point prevalence has been reported at 9-12% (4).

Estimates of the incidence of whiplash-related injuries as reported in Barnsley et al. (5) range from 0.44 per 1000 (6) to 1.06 per 1000 (7). The recent Quebec Task Force Group [QTFG] (8) reported a one year incidence rate of whiplash claims in Quebec of 0.7 per 1000. Large differences between Canadian provinces were noted and thought to be due to differing tort law and compensation systems.

The degree to which whiplash-related symptoms persist in time appears variable in the reported literature. Norris and Watt (9) reported that 66% and 43% of their subjects continued to experience neck pain and headache respectively beyond six months post-accident. These figures are smaller in the report of Hildingsson and Toolanen (10), being 29% and 25%, respectively. The QTFG re-analyzed the data from Norris and Watt's study and found a trend toward persistence of neck pain and headache as the severity of initial presentation increased [i.e., from category 1 through 3]. Barnsley, Lord and Bogduk summarize the literature on chronicity by concluding that “between 14 and 42% of patients with whiplash injuries develop chronic neck pain and that approximately 10% will have constant severe pain indefinitely” (5).

Methods of determining both the clinical course of whiplash-related complaints and their impact upon the individual and society are rather crude. Most studies report symptom lists (8) but few have developed systematic symptom check lists. Outcomes such as claims data on length of disability, return to work and costs of claims are often used, and, although these data are useful to health policy analysts and insurers, they have less relevance to practicing clinicians. It was to address this need, i.e., for an instrument to assess what the QTFG was later to call “Whiplash-associated disorder (WAD)” that Vernon and Mior first developed the Neck Disability Index [NDI] (11).

THE NECK DISABILITY INDEX

The Neck Disability Index was modified [with permission] from the Oswestry Low Back Pain Disability Index [OLBPDI] of Fairbank et al. (12). The NDI, thus, has 10 items selected from the literature to have relevance
to whiplash-associated disorder. Four items relate to subjective symptomatology, namely: "pain intensity," "headache," "concentration" and "sleeping." Four items relate to obligatory activities of daily living, namely: "lifting," "work," "driving," and "recreation." Two items, "personal care" and "reading" are discretionary activities of daily living. Items are scored on an ordinal scale from 0-5, with a maximum score of 50. The seminal study reported on 52 subjects, 70% of whom had suffered a whiplash-type injury within the past 4-6 weeks. Tables 1 and 2 display the mean item scores, ranks and alpha coefficients, and the range of total index scores, respectively.

The Index has good internal consistency [alpha = 0.80], while the total scores display a normal distribution, peaking in the moderately severe interval [15-24 out of 50]. In 17 subjects, test-retest reliability was determined using a two-day/no treatment interval as R = 0.90.

The NDI scores were found to correlate reasonably strongly with McGill Pain Questionnaire (13) scores [R = 0.72]. Finally, in order to determine the NDI’s responsivity to change, change scores were compared to a visual analogue scale for improvement. The correlation coefficient was 0.60. The degree to which any of the subjects with a whiplash-type injury were receiving any disability benefits was not determined.

TABLE 1. Total Score and Item Reliability Analysis.

<table>
<thead>
<tr>
<th>Item</th>
<th>Mean Score</th>
<th>Rank</th>
<th>Alpha</th>
</tr>
</thead>
<tbody>
<tr>
<td>1. Pain intensity</td>
<td>1.70</td>
<td>6</td>
<td>0.79</td>
</tr>
<tr>
<td>2. Personal care</td>
<td>0.75</td>
<td>10</td>
<td>0.78</td>
</tr>
<tr>
<td>3. Lifting</td>
<td>2.20</td>
<td>2</td>
<td>0.78</td>
</tr>
<tr>
<td>4. Reading</td>
<td>2.10</td>
<td>4</td>
<td>0.78</td>
</tr>
<tr>
<td>5. Headache</td>
<td>2.60</td>
<td>1</td>
<td>0.84</td>
</tr>
<tr>
<td>6. Concentration</td>
<td>1.10</td>
<td>9</td>
<td>0.77</td>
</tr>
<tr>
<td>7. Work</td>
<td>1.50</td>
<td>7</td>
<td>0.76</td>
</tr>
<tr>
<td>8. Driving</td>
<td>2.00</td>
<td>5</td>
<td>0.76</td>
</tr>
<tr>
<td>9. Sleep</td>
<td>1.40</td>
<td>8</td>
<td>0.81</td>
</tr>
<tr>
<td>10. Recreation</td>
<td>2.20</td>
<td>3</td>
<td>0.77</td>
</tr>
<tr>
<td>Total Index =</td>
<td>0.80</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>
In 1994, Hains et al. (14) reported on response set bias and internal factor structure of the NDI in 219 subjects with neck pain. After multivariate analysis of variance was conducted, no order or sequencing effect was detected. The internal consistency was determined by Cronbach’s alpha at 0.90. The inter-item correlations ranged from $R = 0.31$ to 0.77, suggesting non-redundancy of items.

On factor analysis, one factor was extracted accounting for 59% of the variance, demonstrating that the NDI is, by and large, unidimensional. This factor was labelled “physical functioning.” Factor weights were calculated and revealed no significant differences amongst the items. As such, it would seem that no inter-item weighting is required. Finally, a strong correlation between pain score [on a visual analog scale [VAS]] and NDI scores was found [$R = 0.70$].

Knaap et al. (15) reported on 46 subjects with neck pain. Test-retest reliability was high [ICC = 0.91, $P < 0.001$] as was internal consistency [Cronbach’s alpha = 0.81]. As with the original report, no effect of age or gender was found.

Wallace et al. (16) used the NDI as an outcome measure in an uncontrolled study of 38 subjects with neck pain treated with spinal manipulation. The NDI scores were reduced from a mean of $19.3 \pm 11.1$ to $7.95 \pm 5.76$ [$P < 0.0001$]. The overall percentage change in NDI scores [over a 12 week period] was 58.8% which was nearly identical to the reduction in VAS pain scores [56%].

In recent unpublished work in our clinic (Vernon et al. unpublished) a high correlation has been observed between NDI scores in whiplash patients and scores on a new instrument—the Disability Rating Index (17). This finding further strengthens the concurrent validity of the NDI.
Vernon et al. (18) have recently reported on the NDI and the Oswestry Index in a sample of patients undergoing physical rehabilitation for work-related or motor-vehicle accident-related neck or back pain. A strong correlation $R = 0.67$, $P = 0.018$ was observed between change in disability index scores [NDI = 20] and a measure of patient satisfaction with the outcome of care [the Outcomes Satisfaction Index] (19).

CONCLUSION

Psychometric studies of the NDI have confirmed that it possesses high test-retest reliability, high internal consistency, a single-factor structure with only moderately high inter-item correlations making each item clinically and statistically useful. Clinical studies have confirmed good concurrent validity and good responsiveness. The NDI is a useful instrument for clinicians and researchers alike to employ in studies of whiplash-injured patients.

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Deep Craniofacial Pain: Involvement of Trigeminal Subnucleus Caudalis and Its Modulation

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While damage or inflammation of deep craniofacial tissues such as the temporomandibular joint (TMJ) and masticatory muscles, as well as abnormal muscular activity, are often considered important in the pathophysiology of temporomandibular disorders (TMDs) and related craniofacial pain conditions, the pathogenesis, diagnosis, and treatment of these deep pain conditions are still controversial. Furthermore, the central neural mechanisms underlying deep craniofacial pain are poorly understood. This chapter outlines our recent findings pointing to a crucial role played by subnucleus caudalis of the brain stem trigeminal (V) spinal tract nucleus (also known as the medullary dorsal horn) in the relay and modulation of nociceptive input from deep craniofacial tissues.

METHODS

All experimental work involving animals was approved by the Animal Care Committee (University of Toronto) and conformed to the regulations of the Canadian Council on Animal Care and the Ontario Animals for Research Act. The studies involved a series of electromyographic (EMG) or single-neuron recording experiments in anesthetized rats (Yu et al. 1995; also see...
Briefly, rats were prepared under surgical anesthesia and then anesthetized with a mixture of \( \frac{1}{3} \) \( \text{O}_2 \), \( \frac{2}{3} \) \( \text{N}_2\text{O} \), and \( 0.5-0.8\% \) halothane to achieve a stable plane of light anesthesia; noxious pressure applied to the hindpaw induced a slight flexion reflex response of the hindlimb. In the EMG experiments, we recorded EMG activity with bipolar electrodes inserted into the ipsilateral and contralateral anterior digastric and masseter muscles. The EMG activity of each muscle was amplified (gain: 1000-5000x; bandwidth 30-3000 Hz) and displayed on oscilloscopes, and also recorded directly into a computer system (CED 1401/Spike2 system, Cambridge). In one series of EMG experiments, the small-fiber excitant and inflammatory irritant mustard oil (MO, 20%, 20 \( \mu \text{l} \) in mineral oil) or its vehicle mineral oil was injected into the ipsilateral TMJ region via a preimplanted needle according to a predetermined schedule (Yu et al. 1995). Lesions of subnucleus caudalis were made surgically or with ibotenic acid injections prior to MO injection to determine if MO evoked a nociceptive reflex that could be interrupted by lesioning of certain regions of the subnucleus caudalis. In another series, either electrical microstimulation (1-ms duration train of three 50-ms cathodal pulses; < 1 mA; train frequency 0.3 Hz) via a microelectrode or microinjection of glutamate (0.5 M in saline, 40 nl) introduced into various loci within the subnucleus caudalis and upper cervical dorsal horn was used to see if stimulation of a specific region within caudalis could also evoke EMG activity. In a third series, we injected neurokinin-receptor antagonists (NK-1 antagonist, CP-99,994, Pfizer; and NK-2 antagonist, MEN-10,376, RBI) intraventricularly (i.c.v.) five to 10 minutes prior to MO injection into the TMJ to see if neurokinin-related neuropeptides are involved in modulating the MO-evoked EMG activity. In the fourth series, we applied opioid receptor antagonists (naloxone hydrochloride or CTOP, D-Phe-Cys-Tyr-D-Tyr-Orn-Thr-Pen-Thr-NH\(_2\), RBI) either systemically or intrathecally (via a cannula placed directly over the subnucleus caudalis) 30 minutes after the initial EMG activity evoked by MO had subsided, to see if the MO-evoked EMG activity was modulated by an opioid mechanism. Ten minutes before the conclusion of each of these physiological experiments, Evans blue dye (5 mg/kg) was injected systemically to confirm the TMJ injection site at postmortem by the visual examination of MO-induced plasma extravasation of Evans blue dye bound to plasma protein (Haas et al. 1992).

As the baseline EMG data revealed no significant variation in EMG activity of each muscle before the treatment (see Yu et al. 1995), we pooled all prestimulation/injection data points for each muscle in each animal to produce a mean value that represented the prestimulation/injection level of EMG activity for each muscle in each animal. All poststimulation/injection data points were normalized relative to this mean value and expressed as a
percentage value of the mean prestimulation/injection level of EMG activity. Changes in EMG activity after the stimulation/injection of either mineral oil or MO were regarded as an increase if one or more EMG data points rose two standard deviations above this mean baseline level.

In some anesthetized and paralyzed rats, single-neuron recordings were made in subnucleus caudalis to determine if MO application to craniofacial tissues influenced caudalis neuronal properties and whether naloxone or the NMDA antagonist MK-801 modulated this influence; the methods for neuronal recording and classification have been previously described in detail (see Hu 1990; Yu et al. 1993). In another group of anesthetized rats, we examined the expression of the immediate early gene, c-fos, within subnucleus caudalis following TMJ stimulation (see Hathaway et al. 1995).

RESULTS AND DISCUSSION

NEURAL PATHWAY FOR TRIGEMINAL NOCICEPTIVE REFLEX ACTIVITY

Subnucleus caudalis contains nociceptive neurons (e.g., Price et al. 1976; Hu 1990), some of which can be activated by electrical and algesic chemical stimulation of the TMJ and other deep tissues; many of these neurons can be antidromically activated from posterior thalamus, which indicates their involvement in ascending nociceptive pathways (Amano et al. 1986; Broton et al. 1988; Sessle et al. 1993). The long latency and high threshold of the neuronal responses evoked by peripheral stimulation indicate inputs from small-diameter afferents. Indeed, an abundance of small-diameter afferents exists in nerves supplying the TMJ and masticatory muscles, and many of these afferents project to subnucleus caudalis (see Shigenaga et al. 1988; Sessle and Hu 1991). Most of the neurons activated by electrical stimulation of TMJ or masticatory muscle afferents can also be excited by noxious mechanical or chemical stimulation of masticatory muscle, TMJ, and skin. Caudalis neuronal activity (Hu et al. 1994; Tsai, Sessle and Hu, unpublished data) and c-fos expression (Hathaway et al. 1995) in lamina I and V caudalis neurons can be produced by MO injection into the TMJ region, both in the posterior part of subnucleus caudalis and CI dorsal horn, and more rostrally at the caudalis/posterior subnucleus interpolaris transition zone. Furthermore, Shigenaga et al. (1988) have indicated that the central terminals of major craniofacial muscle afferents, such as those in masseter and anterior digastric nerves, are mainly distributed in the posterior two-thirds of subnucleus caudalis and upper cervical dorsal horn.

The injection of MO into deep craniofacial tissues such as the TMJ and
masticatory musculature can also lower the threshold for neuronal activation by peripheral afferent inputs and produce an expansion of the deep and the cutaneous components of the mechanoreceptive field (RF) of caudalis nociceptive neurons receiving both superficial and deep craniofacial nociceptive inputs (Hu et al. 1992; Yu et al. 1993; Hu et al. 1994). In contrast, the application of MO to facial skin may be effective in inducing analogous changes in the cutaneous but not the deep RF properties of these neurons. These neuroplastic changes appear to reflect a "central sensitization" process akin to that observed in spinal nociceptive pathways (see Woolf 1992;Coderre et al. 1993; Willis 1993; Dubner 1995). Moreover, we have provided the first demonstration that these MO-induced neuroplastic changes in V nociceptive neurons are associated with an increase in EMG activity in the rat digastric and masseter muscles (Hu et al. 1994; Yu et al. 1995). The increase in jaw muscle EMG activity can be evoked, for example, by MO application to the TMJ region and has a latency of a few seconds and a duration of three to 20 minutes or more; it can be blocked by prior local anesthesia of the TMJ region, which indicates the reflex basis for the EMG activity. Also in adult rats in which over 80% of C-fiber afferents were depleted by neonatal treatment with capsaicin (50 mg/kg), MO injection into the TMJ tissues failed to evoke EMG activities in any of the jaw muscles (unpublished results, Tsai, Sessle, and Hu), which demonstrates the importance of C fibers in mediating the MO-evoked EMG activities.

Although previous studies have indicated that critical relays in many craniofacial reflexes are located in rostral elements of the V brain stem complex such as subnucleus oralis and the surrounding reticular formation (see Sumino 1971; Dubner et al. 1978; Donga and Lund 1991), the above findings indicate that caudalis is most likely involved as a critical relay in jaw nociceptive reflexes and in ascending pathways that transmit nociceptive information from deep craniofacial tissues. To further study the apparent involvement of subnucleus caudalis in these reflex effects induced by MO, we surgically transected caudalis and V spinal tract at the obex (n = 8) or made large ibotenic acid injections (0.5%, 2 injections, 2.5 mg in 0.5 ml, n = 8) into the left caudalis to destroy selectively caudalis neurons. Both manipulations were designed to see if the surgical or chemical lesion could block the MO/TMJ-induced EMG activity (Tsai et al. 1994). After a four- to six-hour recovery period and demonstration of normal physiological conditions, both manipulations were found to be associated with significantly reduced MO-evoked ipsilateral digastric and masseter muscle activities (but normal contralateral EMG activities when MO was injected into contralateral TMJ region) compared to the evoked responses seen in control rats. Four additional control groups in which lesions were made elsewhere in the medulla/spinal cord (to rule out that
the surgical preparation itself could not account for the lesion effects) had normal MO-induced EMG activity. We also have used a more localized ibotenic acid injection (20 nl, 0.5%, n = 12) to produce a lesion (diameter about 600 μm) and this also could block the MO-induced EMG increase (Tsai et al. 1996b). The anterior/posterior location of the effective lesions within caudalis corresponded to the posterior peak distribution of MO/TMJ evoked fos-like immunoreactive cells (see above) and to the most effective micro-stimulation loci (see below).

We also tested whether microstimulation within caudalis can produce excitatory effects on digastric and masseter muscles (Tsai et al. 1995). In 16 rats anesthetized with halothane, electrical microstimulation introduced into the posterior brain stem at six rostrocaudal planes (0.5 mm rostral, and 1.0, 2.5, 4.0, 5.5 and 7.0 mm caudal to obex, i.e., from posterior pole of subnucleus interpolaris to C2 dorsal horn) was delivered at different depths within medial and lateral caudalis and adjacent reticular formation. EMG activity in both ipsilateral digastric and masseter muscles (but not contralateral muscles) could be readily evoked in all animals, with the lowest threshold sites (mean ± SD, 153 ± 63 μA for digastric and 157 ± 59 μA for masseter) being in superficial loci within the posterior caudalis (i.e., 4 mm caudal to obex). The threshold for evoking EMG activity in both muscles gradually decreased from rostral to caudal planes and was significantly lower in the 4-mm caudal plane of caudalis than in the rostral planes; further caudal advancement to the C2 level (7 mm caudal to obex) also caused the threshold to increase dramatically. The shortest EMG latency (at three times threshold stimulation) was also significantly lower with microstimulation at the 4-mm caudal plane (4.4 ± 0.8 ms for digastric and 4.8 ± 0.9 ms for masseter) than at the most rostral planes (7.2 ± 2.7 ms for digastric and 8.3 ± 3.3 ms for masseter). Similar excitatory effects were found after microinjection of the cell-excitant glutamate into the posterior caudalis region, which indicates that neurons and not fibers of passage were the critical elements. The evoked EMG activity was limited to ipsilateral digastric and masseter muscles with a latency of 4.2 ± 2.8 s and duration of 8.5 ± 5.9 min (Tsai et al. 1996a). These findings indicate that neurons in the posterior caudalis may produce excitatory effects on both jaw-opening and jaw-closing muscles, at latencies suggesting multisynaptic or slow conducting paths from caudalis to digastric and masseter motoneurons. Together with the lesioning data and earlier documentation of inputs to caudalis from deep craniofacial tissues (see above) and for direct or indirect projections from caudalis to the V motor nucleus (see Dubner et al. 1978; Mizuno et al. 1983; Jacquin et al. 1990; Yoshida et al. 1994), the findings support the view that subnucleus caudalis is a critical relay in craniofacial nociceptive reflexes evoked by deep noxious stimulation.
At the spinal level, stimulation of nociceptive afferents, particularly C fibers, induces the central sensitization process mentioned above; excitatory amino acids (EAA), including NMDA-receptor mechanisms as well as tachykinins appear to contribute significantly to this central neuroplasticity (Woolf and Thompson 1991; Dubner and Ruda 1992; Urban et al. 1994). It has been proposed that long-term changes in spinal neurons involve a combination of increased peripheral drive, increased central neuron excitability, and altered intersegmental or descending controls from higher brain regions such as the rostral ventromedial medulla (including nucleus raphe magnus) provoked by the initial injury (Coderre et al. 1993; Ren and Dubner 1995). We have recently examined the neurochemical mechanisms underlying central sensitization by testing for the involvement of EAA and neurokinin mechanisms in the central excitatory effects of MO. In related experiments, we also tested for the involvement of a central opioid-related modulatory process induced by the afferent barrage evoked by MO injection into the TMJ region.

We documented the involvement of NMDA receptor mechanisms in our MO/TMJ/EMG model: the NMDA-receptor antagonist MK-801 injected systemically (0.5 mg/kg, i.v., n = 5) in the third ventricle (i.c.v., 0.1 mg/kg, n = 5) or locally into the TMJ region (0.1 mg/kg, n = 5) five minutes before the MO injection into the TMJ region can significantly block EMG activity (Yu et al. 1996). Interestingly, the i.c.v. injection blocked the masseter but not the digastric muscle activity, whereas injection of MK-801 systemically and locally blocked MO-induced EMG activity increases in both muscles. This finding suggests that NMDA-receptor mechanisms may be differentially involved in the responses of the jaw-opening and jaw-closing muscles to noxious stimuli such as MO. In a single-neuron recording pilot study (Yu, Sessle and Hu, unpublished data), we tested five nociceptive neurons receiving cutaneous and deep convergent afferent inputs with MK-801 (0.5 mg/kg, i.v.) delivered five to 10 minutes prior to MO injection into the tongue muscle; MO-801 blocked the cutaneous RF expansion that commonly occurred after MO injection in three of five cases and deep RF expansion in five of five cases. These EMG findings and neuronal data point to the involvement of both peripheral and central NMDA-receptor mechanisms in the relay of craniofacial deep nociceptive inputs through subnucleus caudalis.

Recently, we have examined the involvement of neurokinin receptor mechanisms in the MO-evoked EMG activity (Bakke et al. 1996). The initial EMG activity increase was not affected by prior administration (i.c.v., 50 nM, 20 µl, n = 7) of the NK-1 (substance-P) receptor antagonist CP-99,994. However, the NK-1 antagonist significantly reduced the EMG response to a second MO injection into the contralateral TMJ region. With prior adminis-
tration of the NK-2 receptor (neurokinin A) antagonist MEN-10,376 (i.c.v. 1 nM, 20 µl, n = 7), the EMG responses to the first and second MO injections did not differ significantly but the duration and magnitude of EMG activity was significantly reduced in ipsilateral and contralateral muscles compared with the vehicle control group (Bakke et al. 1996). Together with our MK-801 data (see above), these results suggest that MO may evoke two types of excitatory inputs, an initial one involving EAA but not substance P release and a later one involving in particular the central release of EAA and neurokinin A.

We have previously documented the involvement of central opioid mechanisms in the MO-induced neuroplastic changes in caudalis nociceptive neurons and associated EMG activity: when systemic (i.v.) or intrathecal (i.t.) naloxone was administered 30 minutes after the MO injection, it induced a second EMG activity increase ("rekindling") of the MO-evoked EMG activity that had returned to baseline level by this time (Yu et al. 1994; Seo et al. 1995). This rekindling was dependent on the prior occurrence of the MO-induced EMG activity because naloxone administration 30 minutes after vehicle (mineral oil) injection into TMJ tissue (which itself evoked no EMG activity) resulted in no rekindling effect. Naloxone administration five to 10 minutes prior to the MO injection also significantly enhanced the MO-evoked EMG activity. Thus, as we have suggested (Hu et al. 1994; Yu et al. 1994), a central opioid modulatory influence on EMG activity appears to be recruited by the MO application to the TMJ region and serves to limit the activity evoked by the inflammatory irritant. Our data also support the suggestion that this opioid suppressive mechanism may be a centrally based process and not involve sites in the periphery because the quaternary opioid antagonist methylnaloxone (i.v.), which is reported (Schroeder et al. 1991) not to cross the blood-brain barrier, was not effective in inducing rekindling.

We have recently demonstrated (Seo et al. 1995) a dose-response dependent process in the naloxone-induced rekindling effect for both i.v. (0.65–2.6 mg/kg, n = 5 each group) or i.t. applications (1–30 µg, n = 5 each group) and rekindling of both ipsilateral and contralateral muscle activities. Furthermore, this naloxone-induced rekindling effect may involve the activation of the mu-opiate receptor subtype as we have recently demonstrated similar rekindling effects with CTOP (i.t., 0.06–0.18 µg, n = 7 each group), a peptide and potent specific mu-receptor subtype antagonist (Tambeli et al. 1997). Given that CTOP (i.t.) likely remains locally in the caudalis region because of its hydrophilic nature (Xu et al. 1992), the target site of the MO-induced release of endogenous opioids may be within subnucleus caudalis. Moreover, using single-neuron recordings, we have also found that naloxone administered (i.v., 1.3 mg/kg) 30 minutes after MO was injected into the tongue muscle and after the initial RF expansion had subsided, could induce a second RF expansion in
three of five V caudalis nociceptive neurons tested; 30 minutes after the naloxone injection, RF size had returned to its original dimensions (Hu et al. 1994). These EMG findings and neuronal data clearly point to the likely involvement of central opioid mechanisms in a modulatory process related to central sensitization.

CONCLUSIONS

Our findings point to the critical involvement of subnucleus caudalis not only in the relay of deep nociception to higher brain centers but also to its crucial interneuronal role in jaw reflex responses to deep noxious stimuli. TMJ and muscle inputs to subnucleus caudalis evoke excitatory responses in nociceptive neurons and associated reflex EMG activity in both jaw-opening and jaw-closing muscles. Disruption of caudalis, especially its posterior part, by surgical or chemical lesions can block the evoked EMG activity whereas chemical or electrical microstimulation in the posterior caudalis can evoke EMG response patterns, consistent with a role for caudalis as a critical relay in jaw nociceptive reflexes. Thus, it appears that physiological processes involving deep nociceptive inputs may activate neurons in subnucleus caudalis, which, through their connections with brainstem reflex centers, can result in a cocontraction of agonist and antagonist muscles that can serve to limit movement in pathophysiological conditions affecting the jaw musculature. Furthermore, the neuroplastic changes in caudalis nociceptive neurons by injection of MO into deep craniofacial tissues point also to the occurrence of a central sensitization process that accompanies the MO-evoked EMG activity. Neurochemical mechanisms involving NMDA, neurokinins, and opioids appear to modulate these neuroplastic effects in caudalis and the associated neuromuscular changes. These data support the view that the posterior subnucleus caudalis is a crucial relay of nociceptive information from deep craniofacial tissues and is critically involved in the expression of nociceptive reflexes and their modulation.

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Opioid Involvement in Electromyographic (EMG) Responses Induced by Injection of Inflammatory Irritant into Deep Neck Tissues

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Abstract Previously, we have demonstrated (Hu et al., 1993) that injection of the small-fiber excitant and inflammatory irritant mustard oil (MO) into deep paraspinal tissues surrounding C1-C3 vertebrae can evoke a sustained and reversible increase of electromyographic (EMG) activity of neck and jaw muscles, and can also produce an acute inflammatory response. This increased EMG activity lasts up to 20 min; within 30 min following MO injection, the activity returns to preinjection levels. The aim of our present study was to determine whether an opioid suppressive mechanism may be involved in limiting the increased EMG activity, despite the presence of an ongoing inflammatory response. Three doses (0.6 mg/kg, 1.2 mg/kg, and 2.5 mg/kg) of the opioid antagonist naloxone, along with vehicle (saline), were administered intravenously to determine whether naloxone is capable of inducing a significant recurrence ("rekindling" effect) of EMG activity. A dose-dependent process in the naloxone-induced rekindling effect was demonstrated for the area under the curve of rectified and integrated EMG activity. At the highest dose (2.5 mg/kg), the relative area of naloxone-evoked EMG activity increases reached 83% of the original MO-induced EMG activity level. These results suggest that a central opioid suppressive mechanism is activated by the MO-induced small-afferent barrage, and that this may limit the duration and magnitude of the evoked EMG changes.

Key words endogenous opioids, naloxone, neck, electromyography, inflammatory irritant

Our recent studies in rats have demonstrated that injection of the small-fiber excitant and inflammatory irritant mustard oil (MO; Jancso et al., 1967; Wooff and Wall, 1986; Handwerker and Reeh, 1991) into deep paraspinal cervical tissues evokes a sustained and reversible increase in electromyographic (EMG) activity in neck and/or jaw muscles, and also produces an acute inflammatory response characterized by plasma extravasation (Hu et al., 1993). Similar studies in which MO was injected into the temporomandibular joint (TMJ) region have also documented a sustained and reversible increase in EMG activity in jaw muscles (Yu et al., 1994, 1995). The increased EMG activity usually lasts up to 20 min, although the plasma extravasation lasts longer than 4 hr (Haas et al., 1992; Hu et al., 1993; Yu et al., 1994, 1995). These EMG activity increases may have a reflex basis, since prior injection of a local anesthetic into the TMJ blocks increased EMG activity (Yu et al., 1995). These findings suggest that trauma or noxious stimulation of deep tissues may produce a sustained excitation of several neck/masticatory muscles, which may serve to protect these regions from potentially damaging movements (Sessle and Hu, 1991). In addition, activation of C fibers by electrical stimulation or by application of irritants such as MO or formalin is particularly effective in inducing these trigeminal (V) brainstem neuronal changes (Hu et al., 1992, 1994, Yu et al., 1993; Raboisson et al., 1995). Such reflex effects have also been implicated in neck pain conditions and may have their basis in the "central sensitization" of central nociceptive neurons (Yu et al., 1995; see also Sessle and Hu, 1991; Hu et al., 1994). These nociceptive neurons may serve as reflex interneurons in jaw and neck motor pathways (Abrahams and Richmond, 1988; Hu et al., 1992; Yu et al., 1993; Tsai et al., 1994).

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Subsequent work has shown that once the EMG activity returns to baseline level, and naloxone is administered (i.v.) 30 min after the MO injection into the TMJ, a recurrence or "rekindling" of the MO-evoked EMG activity is induced (Yu et al., 1994). This rekindling is dependent on the prior occurrence of MO-induced EMG activity, since naloxone administration 30 min after injection of a vehicle (mineral oil) into TMJ or muscle tissue (which itself evokes no EMG activity) does not demonstrate this rekindling effect. This opioid suppressive mechanism associated with EMG increases induced by MO injected into the rat TMJ region (Yu et al., 1994) may be a centrally based process and may not involve sites in the periphery, since the quaternary opioid antagonist methylhaloxone (i.v.), which is reported (Schroeder et al., 1991) not to cross the blood–brain barrier, is ineffective in inducing rekindling. The present study was initiated to determine whether similar opioid mechanisms may also be involved in limiting the increased EMG activity found in the neck region following injection of MO into the paraspinal tissues. Because endogenous opioids have been implicated in the modulation (limiting) of central and peripheral nociceptive transmission associated with injury or inflammation of peripheral tissues (Besson and Chaouch, 1987; Ramabadran and Bansinath, 1990; Dubner and Rudy, 1992; Hargreaves and Joris, 1993; Stein, 1993), the opiate antagonist naloxone was used to test for the possible involvement of endogenous opioids in modulating the EMG response evoked by the injection of MO into the paraspinal (neck) region.

Some of these data have been briefly presented in abstract form (Hu et al., 1995).

MATERIALS AND METHODS

All procedures carried out in this study were approved by the University of Toronto Animal Care Committee in accordance with the regulations of the Ontario Animal Research Act. The study was conducted on 27 male Sprague-Dawley rats (250–380 g) and involved methods similar to those previously described in detail (Hu et al., 1993; Yu et al., 1994). Thus, only a brief description follows. Under general anesthesia (1/3 O2, 2/3 N2O, and 2–3% halothane), a cannula consisting of a 30-gauge needle connected to a 20-ml Hamilton syringe with polyethylene tubing for injection was placed in the paraspinal tissue near the C1–C2 vertebrae, passed through the left trapezius and deep neck muscle, and then fixed in place. A special head connector (see Hu et al., 1993) was cemented onto the skull with two small jewel screws and dental cement. This head connector apparatus functioned to stabilize the head without the use of stereotaxic instruments, thereby eliminating any possible noxious input induced by the tooth or ear bars of the stereotaxic equipment. The head and neck positions were adjusted especially carefully, to insure that minimal EMG activity would be elicited from any of the neck muscles because of postural variation. During EMG recordings (see below), the halothane concentration was maintained at 0.6–0.8% so that when noxious pressure was applied to the hindpaw, it would induce only a slight flexion reflex of the hindlimb. Heart rate, percentage of expired CO2, and body core temperature were continuously monitored and maintained at 330–430/min, 4–5%, and 37–37.5°C, respectively. These are the same physiological parameters maintained in a similar procedure in which a C-fiber-related reflex was studied in the hindpaws of similarly anesthetized rats (Fallinower et al., 1994; Guirmand et al., 1994).

The animals were divided into four groups. In each group, EMG activity levels were first recorded for 20 min before MO injection to establish a baseline. MO (20% allylisothiocyanate [in 20 ml mineral oil] BDH, Poole) was then injected into the cervical paraspinal region of the C1–C2 vertebrae region via the cannula–needle system described above, followed 30 min later by the administration of either saline (vehicle for naloxone, i.v., group 1, n = 6) or the opiate antagonist naloxone (groups 2–4). After the saline/naloxone administration, the EMG activity was continuously monitored for another 30 min. The animals in groups 2, 3, and 4 (n = 7 for each group) were tested using three different doses of naloxone (0.6, 1.2, and 2.5 mg/kg, i.v., respectively). Pairs of bipolar EMG electrodes (36- to 40-gauge, single-stranded, Teflon-coated stainless steel wire; interpolar distance, 0.5 cm; exposed tips, 0.5 mm) were then inserted through the intact skin into the masseter and trapezius muscles on the side ipsilateral (left) to the injection site (see below) and bilaterally into the deep neck muscles (rectus capitis posterior). Electrode locations were checked by dissection after each experiment, and confirmed in all the target muscles mentioned above. From 2 to 3 hr after the initial anesthesia induction, the resting EMG activity in these muscles was monitored before, during, and after the injection of MO into the deep paraspinal tissues of the left side of the neck, followed by intravenous injection of saline or naloxone. The MO was injected via a dorsal approach, 1.5 cm behind the occipital ridge and 5 cm lateral to the midline (Hu et al., 1993). The injection site was 1 cm below the skin surface, within the paraspinal tissues around the C1–C2 vertebrae.

Before the MO injection, the EMG activity of each muscle was amplified for 20 min. EMG activity was continuously recorded 30 min after MO injection (gain, ×1000–5000; bandwidth, 30–3000 Hz) and displayed on oscilloscopes, and also recorded on a computer system (see below). Naloxone or saline was injected when the EMG activity passed through the left trapezius and deep neck muscle, and then returned to baseline level, and then an additional 30 min of EMG recording was carried out. The EMG data were recorded directly into the computer via an analog-to-digital converting interface (1401plus, CED, Cambridge) and were processed off-line with the Spike2 program (CED, Cam-
bridge). The EMG signals were rectified and integrated. Since the increase in EMG activity induced by the injection of MO lasted up to 20–30 min (see "Results"), the EMG activity was rectified and integrated after every 1-min block to allow the area under the curve of any EMG response to be calculated. During the pre-MO-injection period of 20 min, the area under the curve of EMG activity in each minute was converted into twenty 1-min block data points, and the mean and standard deviation (SD) were expressed. Increases in EMG activity were determined by a change above the baseline level if one or more EMG data points rose 2 SD above this mean value. All data points were normalized relative to this mean value and expressed as a percentage of the mean baseline level of EMG activity; the values are reported here as mean ± SD. In order to avoid the intrinsic property differences existing between muscles, we also normalized the rekindling effect according to each muscle’s own baseline (i.e., its own original MO-induced EMG activity increase) as 100%. The time period from the beginning of the injection to the increase in EMG activity was designated as the latency of the response, and the time period from the increase in EMG activity to recovery to the baseline level of EMG activity was designated as the duration of the response.

There was no significant variation in the EMG activity of each muscle before vehicle injection in all rats injected with MO (Wilcoxon test, p > 0.05). As such, all preinjection data points for each muscle were pooled. In order to assess the effect of naloxone, an a priori Fisher’s exact probability test between the saline group and one of the three naloxone groups was used. The Wilcoxon test was used for statistical comparisons between the mean value representing the preinjection level of EMG activity (see above) and any data point of the postinjection periods. In order to determine the naloxone effect on other measurements (i.e., duration, latency, and area under the curve), an a priori Dunnett’s test between the saline group and one of the three naloxone groups was used after the analysis of variance (ANOVA; F test). If the homogeneity test failed (p < 0.01; Winer, 1962), a Kruskal–Wallis H test was substituted for further analysis. If the F or H value was significant, the likelihood of a slope for the dose–response curve’s being zero would be low; thus the dose–response relationship was established. Dunnett’s test followed the F or H test between the saline (vehicle control) group and one of the three naloxone groups. Values were expressed as mean ± SD and p < 0.05 (two-tailed) was regarded as significant.

Previous studies in our laboratories (Haas et al., 1992) showed that when MO was injected into deep craniofacial tissues, visual localization of Evans blue dye conjugated with extravasated plasma protein correlated well with the spectrophotometric detection of Evans blue dye, as well as with the presence of other histological parameters of inflammation (such as the presence of polymorphonuclear leucocytes). Therefore, at the end of each experiment in the present study, Evans blue (0.2 ml, 1%) was injected through a catheter inserted into the right external jugular vein 70 min after injection of MO. The animal was given a lethal injection (T61, Hoechst, Montreal) 20 min later. The MO injection site was visually localized according to the appearance of extravasated dye, and was then outlined on a standardized drawing of the neck region of the rat.

RESULTS

The Effects of MO Injections on EMG Activity

As in our previously published results (Hu et al., 1993), MO injected into the paraspinal region produced an increase in EMG activity in all the muscles examined in all four groups (the saline group plus the three different naloxone dose groups). The latency of this MO-induced EMG response was very short (<1 min; see Fig. 1 and Hu et al., 1993), and thus the data were not analyzed further and are not presented in Table 1. In order to simplify the graphic presentation, only the saline group and the 2.5-mg/kg naloxone group are presented in Figure 1 (the 0.6- and 1.2-mg/kg groups are not shown). Table 1 shows that MO-induced EMG responses were similar (p > 0.05) for the incidences (Fisher’s exact probability test), areas (ANOVA), and durations (ANOVA) in all four muscles examined. Thus, we have replicated our previously published results and have provided an equal basis for naloxone treatment (see below).

The MO injection site was visually localized according to the appearance of extravasated Evans blue dye and was found within the region of the C1–C2 paraspinal tissues in all rats (Hu et al., 1993). Also note that the masseter EMG response pattern was generally smaller than those observed in the neck (left and right deep neck and trapezius) muscles.

The Effects of Naloxone Injection on EMG Activity

The saline injection group did not produce significant EMG increases (p > 0.05, Wilcoxon test) 30 min after the MO injection into the paraspinal region. In contrast, the opiate antagonist naloxone at various doses produced significant increases in the incidence of the recurrence of EMG activity (Table 2; Fisher exact probability test between the saline group and all three naloxone groups within the same muscle). Similarly, latency data showed that all naloxone groups were significantly different from the saline group in all four muscles (Kruskal–Wallis H tests). With respect to area under the curve, naloxone (0.6 mg/kg, 1.2 mg/kg, 2.5 mg/kg) administered (i.v.) 30 min after MO injection produced dose-dependent EMG activity increases in all four muscles (the data are presented in Table 2 and Fig. 2). For area measurement, the 1.2-mg/kg group (only masseter muscle)
Figure 1. The effects of MO injection into the left deep neck region, and the effects of naloxone, on EMG activity. For clarity, the data from the 0.6-mg/kg and 1.2-mg/kg naloxone groups are not shown; the data from the saline and 2.5-mg/kg groups are shown in A and B, respectively. Vertical bars represent SDs. Asterisks represent the significant differences (Wilcoxon test, *p < 0.05). Note that only significant data points are shown in the 2.5-mg/kg naloxone group. MO-induced EMG activity was higher in amplitude but shorter in duration, whereas naloxone-induced EMG activity was lower in amplitude and more prolonged in duration. Filled triangles represent the data from the saline (vehicle) group; filled circles represent the data from the 2.5-mg/kg naloxone group.

and the 2.5-mg/kg group (except the right deep neck muscle) were significantly different from the saline group. Even more interesting, the relative ratio of the rekindling effect related to the original MO-induced EMG activity (as 100% of the area) was a dose-dependent function. When all four muscle observations were averaged into a single mean, these mean relative ratios were 3.3% (saline group), 18% (0.6-mg/kg naloxone group), 37% (1.2-mg/kg naloxone group), and 83% (2.5-mg/kg naloxone group), and the pattern of these four data points fit a monotonic function (see Fig. 2). Like the area measure, the duration measure showed that both the 1.2- and the 2.5-mg/kg groups were significantly different from the saline group. For these measurements of incidence, latency, and duration, no clear demonstration of dose-response function was found. As noted earlier, the 0.6- and 1.2-mg/kg groups are not shown in Figure 1 to simplify the presentation, but the detailed properties are described in Table 2. However, Figure 1 clearly demonstrates the time course and magnitude of this naloxone rekindling effect. As stated above, the averaged magnitude of this effect reached a maximum of 83% of the original MO-induced muscular electrical activity level. However, the naloxone-induced effect was rather a slow process (6–15 min to reach 2 SD), had a prolonged time course, and had lower peak electrical activity levels (see example in Fig. 1) than the original MO-induced EMG activity.

DISCUSSION

In addition to replicating our previously published results concerning MO-induced EMG activity increase in the neck region (Hu et al., 1993), our present study demonstrates a dose-dependent process in the naloxone-induced neck "rekindling" EMG effects, suggesting that an opioid receptor mechanism may be involved. Both ipsilateral and contralateral neck muscle activities were rekindled by intravenous naloxone. At the highest naloxone dose of 2.5 mg/kg, the relative area under the curve of naloxone-produced rekar-
## TABLE 1. The Effects of MO Injections on EMG Activity

<table>
<thead>
<tr>
<th></th>
<th>L. masseter</th>
<th>L. deep neck</th>
<th>R. deep neck</th>
<th>L. trapezius</th>
</tr>
</thead>
<tbody>
<tr>
<td>Saline (n = 6)</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Incidence (%)</td>
<td>83%</td>
<td>100%</td>
<td>100%</td>
<td>100%</td>
</tr>
<tr>
<td>Area (mV/sec)</td>
<td>13.8 ± 8.4</td>
<td>14.7 ± 8.7</td>
<td>14.8 ± 6.8</td>
<td>16.7 ± 14.7</td>
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<tr>
<td>Duration (min)</td>
<td>12.2 ± 11.3</td>
<td>15.7 ± 12.9</td>
<td>12.2 ± 8.7</td>
<td>16.7 ± 11.9</td>
</tr>
<tr>
<td>0.6 mg/kg naloxone (n = 7)</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Incidence (%)</td>
<td>57%</td>
<td>100%</td>
<td>100%</td>
<td>100%</td>
</tr>
<tr>
<td>Area (mV/sec)</td>
<td>2.1 ± 2.3</td>
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<td>16.8 ± 19.1</td>
<td>13.1 ± 13.6</td>
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<td>Duration (min)</td>
<td>13.3 ± 13.0</td>
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<td>17.1 ± 6.6</td>
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<td></td>
<td></td>
<td></td>
</tr>
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<td>Incidence (%)</td>
<td>100%</td>
<td>100%</td>
<td>86%</td>
<td>100%</td>
</tr>
<tr>
<td>Area (mV/sec)</td>
<td>9.8 ± 7.6</td>
<td>16.4 ± 8.2</td>
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<td>23.7 ± 7.5</td>
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<tr>
<td>Duration (min)</td>
<td>20.4 ± 9.3</td>
<td>20.4 ± 9.5</td>
<td>18.7 ± 11.4</td>
<td>21.7 ± 5.9</td>
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<td>2.5 mg/kg naloxone (n = 7)</td>
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<td></td>
<td></td>
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<tr>
<td>Incidence (%)</td>
<td>100%</td>
<td>100%</td>
<td>86%</td>
<td>86%</td>
</tr>
<tr>
<td>Area (mV/sec)</td>
<td>3.4 ± 3.9</td>
<td>31.9 ± 19.7</td>
<td>13.8 ± 17.8</td>
<td>24.1 ± 20.0</td>
</tr>
<tr>
<td>Duration (min)</td>
<td>16.6 ± 10.0</td>
<td>20.3 ± 8.4</td>
<td>12.7 ± 10.56</td>
<td>20.0 ± 10.0</td>
</tr>
</tbody>
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Note. No comparison between measurements in any muscle was significantly different. All incidence measurements were tested with Fisher's exact probability test. The area and duration measurements were tested with Dunnett's test after the ANOVA test, except the area of the left masseter, for which the Kruskal–Wallis H test was used instead of the ANOVA test, and p = 0.045 (not significant, two-tailed).

## TABLE 2. The Effects of Naloxone Injections on EMG Activity

<table>
<thead>
<tr>
<th></th>
<th>L. masseter</th>
<th>L. deep neck</th>
<th>R. deep neck</th>
<th>L. trapezius</th>
</tr>
</thead>
<tbody>
<tr>
<td>Saline (n = 6)</td>
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<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Incidence (%)</td>
<td>0%</td>
<td>0%</td>
<td>0%</td>
<td>0%</td>
</tr>
<tr>
<td>Area (mV/sec)</td>
<td>0.24 ± 0.4</td>
<td>0.48 ± 0.67</td>
<td>0.42 ± 0.37</td>
<td>0.6 ± 0.52</td>
</tr>
<tr>
<td>Relative ratio</td>
<td>1.7%</td>
<td>3.3%</td>
<td>2.8%</td>
<td>3.6%</td>
</tr>
<tr>
<td>Latency (min)*</td>
<td>---</td>
<td>---</td>
<td>---</td>
<td>---</td>
</tr>
<tr>
<td>Duration (min)</td>
<td>0</td>
<td>0</td>
<td>0</td>
<td>0</td>
</tr>
<tr>
<td>0.6 mg/kg naloxone (n = 7)</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Incidence (%)</td>
<td>86%*</td>
<td>86%*</td>
<td>86%*</td>
<td>71%*</td>
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<tr>
<td>Area (mV/sec)</td>
<td>0.68 ± 0.61</td>
<td>4.8 ± 5.0</td>
<td>5.0 ± 7.9</td>
<td>0.8 ± 0.7</td>
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<td>Relative ratio</td>
<td>32%</td>
<td>5.2%</td>
<td>29%</td>
<td>6%</td>
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<tr>
<td>Latency (min)</td>
<td>16.7 ± 5.5</td>
<td>8.0 ± 4.4</td>
<td>7.4 ± 4.3</td>
<td>12.6 ± 1.5</td>
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<tr>
<td>Duration (min)</td>
<td>6.1 ± 3.2</td>
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<td>8.2 ± 6.8</td>
<td>13.0 ± 5.5</td>
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<tr>
<td>1.2 mg/kg naloxone (n = 7)</td>
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<td></td>
<td></td>
<td></td>
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<td>Incidence (%)</td>
<td>71%*</td>
<td>86%*</td>
<td>86%*</td>
<td>100%*</td>
</tr>
<tr>
<td>Area (mV/sec)</td>
<td>2.1 ± 2.0</td>
<td>5.6 ± 6.4</td>
<td>7.6 ± 17.8</td>
<td>12.6 ± 1.5</td>
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<tr>
<td>Relative ratio</td>
<td>21%</td>
<td>34%</td>
<td>39%</td>
<td>53%</td>
</tr>
<tr>
<td>Latency (min)</td>
<td>13.3 ± 2.4</td>
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<td>Duration (min)</td>
<td>10.4 ± 8.5</td>
<td>18.4 ± 8.2</td>
<td>15.1 ± 5.9</td>
<td>14.4 ± 6.7</td>
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<tr>
<td>2.5 mg/kg naloxone (n = 7)</td>
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<td></td>
<td></td>
</tr>
<tr>
<td>Incidence (%)</td>
<td>86%*</td>
<td>100%*</td>
<td>86%*</td>
<td>100%*</td>
</tr>
<tr>
<td>Area (mV/sec)</td>
<td>4.4 ± 6.4*</td>
<td>14.8 ± 13.3*</td>
<td>10.7 ± 13.7*</td>
<td>18.8 ± 24.4*</td>
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<tr>
<td>Relative ratio</td>
<td>129%</td>
<td>46%</td>
<td>78%</td>
<td>78%</td>
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<tr>
<td>Latency (min)</td>
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<td>10.3 ± 4.4</td>
<td>12.3 ± 5.4</td>
<td>9.7 ± 3.9</td>
</tr>
<tr>
<td>Duration (min)</td>
<td>9.7 ± 6.3*</td>
<td>16.7 ± 7.2*</td>
<td>14.6 ± 8.8*</td>
<td>14.7 ± 7.9*</td>
</tr>
</tbody>
</table>

Note. All significance levels (p < 0.05, two-tailed) were calculated by a priori test between the saline group and one of the three naloxone groups. Incidences were tested with Fisher's test. Latency data were not tested for significance, because the saline group did not have a latency value. Duration data were tested with F test. The areas of the masseter, right deep neck, and trapezius muscles were tested with H test, whereas the area of the left deep neck muscle was tested with F test.

* Latency for the saline group could not be estimated, and a cutoff value (30 min) was used for consequent calculation because no data point was found to be significant.

**Relative ratio** is the area value of the naloxone-induced EMG activity versus the original MO-induced EMG activity for each of the individual muscles. See "Methods" for details.
resolve which of these opioid receptor subtypes is involved in this rekindling effect. Moreover, Guirimand et al. (1994) have demonstrated that opioid antagonists, including naloxone and mu, delta, and kappa subtypes, could antagonize the suppressive effects caused by the intrathecal injection of morphine to the C-fiber evoked by electrical stimulation. Our model and Guirimand et al.'s model relate to C-fiber-evoked reflexes, but there are two important differences: (1) In their model rats received an exogenous opioid agonist (morphine) by intrathecal application, whereas in our model an endogenous opioid (or opioids) was triggered and released by MO application. (2) Our EMG recordings were provoked by a single MO application, whereas the C-fiber reflex was evoked by repeated electrical stimuli at C-fiber strength. Nonetheless, their results suggest the possibility that all three opioid receptor subtypes may be involved in our neck-MO-EMG model.

The time course (latency and duration) of the rekindling effect is consistent with the effects of naloxone in antagonizing these modulatory effects (including periaqueductal grey [PAG] or nucleus raphe magnus [NRM] stimulation-induced analgesia) and to neuronal and reflex responses to noxious stimuli in the V system (Sessle et al., 1981; Yu et al., 1991). Administration of naloxone alone has been shown to enhance spinal motor reflexes induced by electrical stimulation (e.g., Goldfarb and Hu, 1976; Catley et al., 1983). However, we have preliminary data to indicate that the action site for this naloxone-induced rekindling effect is most likely involved with sensory second-order nociceptive neurons in subnucleus caudalis or cervical dorsal horn; therefore, naloxone is unlikely to act on the motor neurons. In a pilot study using single V subnucleus caudalis neuron recordings (Hu et al., 1994), we found that naloxone administered (i.v.) 30 min after MO was injected into the tongue muscle after the initial receptive field (RF) expansion had subsided could induce a second RF expansion in three of five caudalis nociceptive neurons tested; 30 min after naloxone injection, RF size had recovered to its original dimensions. These EMG findings plus our pilot neuronal data point clearly to the probable involvement of central opioid mechanisms in the central sensitization process.

A previous study (Yu et al., 1994) found that naloxone did not produce any rekindling effects after MO injection into the TMJ region; thus, the EMG increase was dependent on the previous occurrence of MO-induced effects. These findings suggest that an opioid mechanism is triggered by the MO injection and attenuates the EMG activity, which can then be rekindled by naloxone. Our present study used a dose-response approach, as opposed to the use of a single dose (1.3 mg/kg) of naloxone in the previous study (Yu et al., 1994). However, our present study did not use the peripherally acting opioid antagonist methylnaloxone (Yu et al., 1994) to rule out the peripheral opioid activity. Nevertheless, in conjunction with our previous TMJ study (Yu et al., 1994), the evidence suggests that the rekindling of the...
EMG activity induced by naloxone may be produced by an antagonism of an inhibitory effect of central origin. Both the central sensitization process and the opioid modulatory mechanism may play important roles in the clinical situation relating to pain and dysfunction in the neck region. Previously, we suggested (Yu et al., 1994; see also Hu et al., 1994) that the central opioid modulatory influence on EMG activity appears to be recruited by the MO application to the TMJ region, and that it serves to limit the activity evoked by the inflammatory irritant. Local and/or descending modulation could underlie the opioid-related rekindling effects that the present study has discovered; however, the exact origin and the loci of this endogenous opioid remain to be elucidated. The intricate organization and the variation of inputs from neck tissues and interconnections of cervical dorsal horn (Smith et al., 1991; Abrams et al., 1993) as well as V subnucleus caudalis (Sessle et al., 1986; Sessle and Hu, 1991) provide the basis for numerous central interactions between the various inputs derived from peripheral tissues (e.g., segmental inhibition) or from intrinsic brain regions (e.g., descending inhibition from the PAG, NRM, and other brain centers). These modulatory processes involve a variety of endogenous neurochemical substances, including an endogenous opioid, that may involve presynaptic or postsynaptic regulatory processes (Fields and Basbaum, 1994; Yaksh and Malmberg, 1994).

Extensive sensory and reflex studies have been performed in the V system, but few studies have been conducted in the neck region (see Abrams et al., 1993). The present study, in addition to our previous studies of MO-induced EMG activity increase in the neck muscles, further our understanding of nociceptive processes and reflex pathways in the neck region. In conclusion, the neck system may operate with neural processes similar to those found in the V (and the limb) system, including central sensitization and opioid modulatory mechanisms.

ACKNOWLEDGMENTS

We are grateful to K. MacLeod for his electronic and computer technical expertise. This research was supported by a grant from the Foundation for Chiropractic Education and Research (No. 94-03-08) to James W. Hu.

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Muscle strength testing of the neck with a manual modified sphygmomanometer dynamometer

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Muscle strength testing of the neck with a manual modified sphygmomanometer dynamometer

The purpose of this study was to assess intra- and inter-examiner reliability of the manual modified sphygmomanometer dynamometer (MSD) for use in manual muscle testing of neck muscles. A secondary objective was to provide a preliminary determination for both genders of the normative range of values of neck muscle strength with this device. A convenience sample of 40 asymptomatic volunteer subjects, consisting of 20 males and 20 females, selected from the Canadian Memorial Chiropractic College, Toronto, Ontario, student and staff population, was obtained, ranging in age from 26–30 years. Each examiner conducted two trials of isometric, subject-initiated neck muscle strength testing in four cervical ranges of motion (flexion, extension, and right and left lateral bending) with a 35-s rest interval between trials. Data were analysed descriptively for each test for both genders. Coefficients of variation were calculated. The unpaired t-test was used to determine any differences between male and female neck muscle strengths. The Intra-class Correlation Coefficient (ICC) was used to determine intra- and inter-examiner reliability. The ICCs greater than 0.70 were considered to be clinically significant for reliability. A normative database was calculated for all four tests for each gender showing generally acceptable coefficients of variation. Side-to-side (right-to-left) differences and flexion/extension ratios were consistent with previously reported values in normal subjects. Values for males were substantially greater than for females on all tests. The ICCs for intra-examiner reliability ranged from 0.81 to 0.98, while these ranged from 0.65 to 0.86 for inter-examiner reliability. A first approximation of a normative database is presented which conforms to expected findings such as lower values for flexion as compared to extension and limited side-to-side differences. Strength values for males appear to be significantly greater than for females. With minor exceptions for some male subjects, the manual MSD appears to be an instrument which can be employed consistently between and among examiners in testing the strength of neck muscles in normal subjects. The manual MSD is offered as a potentially useful tool in the quantitative assessment of neck function.

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Introduction

Manual muscle testing has proved useful in the clinical setting for determining the strengths of various muscles, the limitations and level of sincerity of patients, and the prognosis and progress of treatment programmes (Iddings et al. 1961; Kendall & McGcreary 1982; Marino et al. 1982; Wadsworth et al. 1987; Hsieh & Phillips 1990). This type of testing is not entirely objective and relies on such factors as the skill of the tester, strength of the tester and the testing procedure itself (Beasley 1956; Nicholas et al. 1978; Wikholm & Bohannon 1991). For these reasons, obtaining an accurate quantitative measure of muscle strength is challenging to the clinician and researcher alike.

When considering the cervical spine in particular, any weakness in neck muscle strength may be important in revealing clinically significant information on neck, shoulder and arm disorders (Defreitas & Vitti 1980; Bull et al. 1984; Harms-Ringdahl & Schuldt 1988; Schulte & Harms-Ringdahl 1988a,b). It has been shown that specific cervical muscles predominate in specific actions of neck motions, thereby making it possible to test fairly specific muscles in the neck (Defreitas & Vitti 1980; Bull et al. 1984; Harms-Ringdahl & Schuldt 1988; Schulte & Harms-Ringdahl 1988a,b). It has also been shown that certain occupational conditions can result in fatigue and weakness of various muscles around the neck and shoulders (Hagberg 1984; Harms-Ringdahl & Schuldt 1990; Viikara-Juntura et al. 1994), thereby leading to pain complaints in these areas.

Several instruments, including strain gauges (Kenney 1965; Bohannon 1987; Triano & Schultz 1987; Schulte & Harms-Ringdahl 1988a,b), hand-held dynamometers (Edwards & McDonnel 1974; Hyde et al. 1983; Bohannon 1986; Bohannon & Andrews 1987) and modified sphygmomanometer dynamometers (MSDs) (Helewa et al. 1981; Isherwood et al. 1983; Helewa et al. 1986; Bohannon & Lusardi 1991; Vernon et al. 1992; Perossa et al. 1996) have been studied, with high levels of reliability being reported. Vernon et al. (1992) studied the use of a wall-mounted MSD in neck strength testing and reported high reliability for all ranges. A preliminary normative data base was also reported. Vernon et al. (1992) found that flexion strength was typically 60% of extension strength and side-to-side differences were less than 10%. Symptomatic subjects showed some clinically important differences from the normal subjects; namely, greatly reduced strength values and a greatly reduced flexion/extension ratio. Vernon et al. (1992) concluded that chronic neck pain may be associated with reduced neck flexor muscle strength. Bussieres (1994) reviewed a variety of assessment procedures for the cervical spine.

The data on strength testing in this review is essentially in agreement with the findings of Vernon et al. (1992).

Several studies have been published on various kinds of MSDs have shown these instruments to be reliable when testing the strength of neck (Vernon et al. 1992), hip (Perossa et al. 1996), shoulder (Helewa et al. 1981; 1986) and elbow (Bohannon & Lusardi 1991) muscles. The device used in this study is a manual MSD. This device may provide several advantages over other instruments used for quantitative muscle strength testing. First, it is a practical device that neither requires any mounting apparatus nor complicated set-up. Secondly, it is inexpensive and portable, and thus, can be used very effectively in a clinical setting. Thirdly, it is comfortable to the patient and the examiner, thereby maximizing subject compliance.

Since no studies have measured neck muscle strength using the manual MSD, the present authors initiated this study in asymptomatic male and female subjects between the ages of 25 and 30 years. The objectives of the study were: (1) to establish a preliminary normative data base for neck muscle strengths in normal subjects; 2) to test the hypothesis that males would provide higher strength values than female subjects in any tested motion; and (2) to determine the level of intra- and inter-examiner reliability of manual muscle testing using the manual MSD, with clinically significant reliability indicated by an Intra-class Correlation Coefficient (ICC) greater than 0.70.

Materials and methods

Subjects

A total of 40 subjects between the ages of 25 and 30 years participated in the study. Out of this group, 22 were male and 20 were female. The subjects were verbally screened for any cervical spine pathology, such as any known congenital anomaly, history of fracture or history of surgery. Other inclusion criteria consisted of no active profile of headaches (greater than one per week), no neck pain within the past 6 months and no known history of hypertension. Each volunteer was given a written informed consent form to sign prior to testing.

Examiners

Two graduating chiropractors served as the examiners. The examiners practised the testing procedure for several
hours prior to the study in order to standardize the method of testing.

**Instrument**

The test instrument used in this study was the manual MSD devised by Dr A. Schulte (see Fig. 1). The device was created by altering the position of the air tube and meter on a standard sphygmomanometer to a fixed position on the front of the cuff. The length of the cuff was also shortened so as to produce a ring of material suitable for the size of a single hand. To begin testing, the cuff was pumped to 20 mmHg as a starting point so that the bladder became inflated. The inflated cuff was then held in the tester’s right hand while the left hand stabilized the subject being tested.

**Test protocol**

The subject was seated comfortably in a chair with a high seat-back in order to stabilize their trunk. The hands were held comfortably in the lap in order to reduce any attempted addition of resistance from the arms and trunk. No other stabilization or isolation was provided. The subject was instructed to gradually push to maximum contraction against the inflated bladder, a method known as the patient-initiated method (Hsieh & Phillips 1990). The pressure exerted against the cuff, which is a measure of the peak force output of the muscle groups being tested, was measured on the cuff dial. A one-way valve at the air tube ensured that the needle position was maintained so that an accurate measurement could be taken.

Manual muscle testing was performed on the neck muscles in flexion, extension, and right and left lateral bending. Each subject was instructed on how to perform the test and was given one practice trial in neck extension to familiarize him or herself with the test procedure. Examiner 1 measured all four neck strengths with a 35-s rest period between duplicate measurements. Examiner 2 repeated the above procedure on the same subject immediately after Examiner 1. The order of the motions tested was randomly altered between examiners. An assistant read and recorded the values obtained from the meter.
The patient-initiated method was utilized which involves the patient initiating maximal exertion against the device gradually while the tester resists any motion in the opposite direction of the force.

The procedure for neck flexion involved seating the subject with the head in slight extension so that the head was held in a neutral position once contraction began (see Fig. 2). The tester placed the partially inflated bladder on her right hand and ensured a starting pressure of 20 mmHg. The tester stood at the subject’s right shoulder and stabilized it with her left hand while the cuffed right hand was place on the subject’s forehead. To start the test, the subject was given a verbal command to gradually exert maximum flexion of the neck until told to stop. The tester resisted forward flexion of the neck for approximately 5 s, at which time the subject was told to relax and the measurement was recorded.

Similarly, procedures for neck extension, and right and left lateral bending were performed by the first examiner (see Figs 3 & 4). A minimum 35-s rest was given before the second examiner measured the randomly ordered neck ranges of resisted motion. Measurements were recorded on the data sheet for each trial for each examiner.

Data analysis

The data were first analysed descriptively with means and standard deviations for each combination of strength test and gender. Coefficients of variations were calculated to determine the distribution of each data set. The unpaired t-test was used to determine any significant difference in male versus female neck muscle strength levels with the level of significance being set at 0.05. The ICC (Bartko 1966) was used to determine intra- and inter-examiner reliability with clinical significance set at ICC greater than 0.70.

Results

Table 1 displays the means and standard deviations for each combination of movement for both genders (in = 80 measurements per gender). This table also displays the t-test values for male versus female measures, all of which are highly significantly different. The coefficients of
Figure 3. The procedure for testing neck extension.

variation for this data set are as follows: flexion, 21% (males) and 21% (females); extension, 18% (males) and 20% (females); right lateral bending, 23% (males) and 16% (females); and left lateral bending, 24% (males) and 19% (females). The ratio between the mean flexion and extension values was 80% for males and 74% for females.

The ICCs for intra-examiner reliability are shown in Table 2 and range from 0.808 to 0.975. Table 3 displays the ICCs for inter-examiner reliability, which range from 0.654 to 0.742 for males and 0.738 to 0.858 in females (n=40 measurements for each ICC per gender).

Discussion

A variety of devices have been designed in order to assess muscle strength by manual testing (Bussieres 1994). This study of the manual MSD was conducted in order to provide preliminary determination of the normal ranges of neck strengths in both male and female subjects, and to determine the intra- and inter-examiner reliability of the procedures. The relative values obtained for individual strength measures in this study compare favourably to those published previously with a wall-mounted MSD (Vernon et al. 1992). Values for all tests were found to be higher for males than for females (Foust et al. 1973; Vernon et al. 1992; Perossa et al. 1996). The differences between side-to-side tests in lateral bending were well below the 10% level in both males (5%) and females (4%). This is in agreement with Vernon et al.’s (1992) finding with the wall-mounted MSD, and with the findings of Levoska et al. (1992) and Franko & Herzog (1987).

The issue regarding the comparison of flexion versus extension strengths is somewhat controversial. Several authors (Foust et al. 1973, as reported in Bussieres 1994; Franko & Herzog 1987; Levoska et al. 1992; Vernon et al. 1992) found extension strength to be higher than that of flexion, at least in normal subjects. In the first three of these reports, a flexion/extension ratio of 0.6/1.0 was obtained on average. However, Petrofsky & Phillips (1982) found the opposite. In the present study, flexion strengths in both males and females were found to be lower than those of extension by 80% and 74%, respectively. It appears that manual as opposed to wall-mounted
For clinical purposes, Table 4 displays a set of values (in mmHg) which represent lower cut-offs of normal measures derived in this young healthy sample by subtracting one standard deviation from the mean of each test value for male and females. Because of the consistency within the overall data set (i.e., relatively similar coefficients of variation and low side-to-side differences), the present authors suggest that this represents a workable approximation of values below which some clinically important weakness may be present. It remains for a study in symptomatic subjects with the manual MSD to be done for this to be confirmed.

The ICCs for the intra-examiner reliability were found to be clinically and statistically significant for both male and female subjects. In addition, the ICCs for the inter-examiner reliability also show clinical and statistical significance for female subjects while three out of the four ICC values for the male subjects fall below the critical value of 0.70, although only marginally so. These values are in agreement with previous reports of high test-retest and inter-rater reliability of MSDs in general usage (Helewa et al. 1981, 1986; Isherwood et al. 1983).

---

Table 1. Summary of means and standard deviations in (SD) in mmHg for males and females, and t-test values for males versus females

<table>
<thead>
<tr>
<th>Movement</th>
<th>Male</th>
<th>Female</th>
<th>t-test*</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Mean</td>
<td>SD</td>
<td>Mean</td>
</tr>
<tr>
<td>Right lateral bend</td>
<td>141.3</td>
<td>32.3</td>
<td>100.4</td>
</tr>
<tr>
<td>Left lateral bend</td>
<td>133.7</td>
<td>31.7</td>
<td>104.1</td>
</tr>
<tr>
<td>Extension</td>
<td>169.4</td>
<td>30.7</td>
<td>127.8</td>
</tr>
<tr>
<td>Flexion</td>
<td>134.7</td>
<td>27.8</td>
<td>95.1</td>
</tr>
</tbody>
</table>

*Significance set at $P<0.05$.

---

Figure 4. The procedure for testing right and left lateral bending.
Table 2. Intra-examiner reliability of tested movements for males and females

<table>
<thead>
<tr>
<th>Movement</th>
<th>Examiner 1</th>
<th>Examiner 2</th>
<th>Examiner 1</th>
<th>Examiner 2</th>
</tr>
</thead>
<tbody>
<tr>
<td>Right lateral bend</td>
<td>0.964</td>
<td>0.884</td>
<td>0.831</td>
<td>0.823</td>
</tr>
<tr>
<td>Left lateral bend</td>
<td>0.937</td>
<td>0.868</td>
<td>0.927</td>
<td>0.927</td>
</tr>
<tr>
<td>Extension</td>
<td>0.907</td>
<td>0.808</td>
<td>0.945</td>
<td>0.995</td>
</tr>
<tr>
<td>Flexion</td>
<td>0.914</td>
<td>0.691</td>
<td>0.918</td>
<td>0.945</td>
</tr>
</tbody>
</table>

*Significance: ICC>0.70.

Table 3. Inter-examiner reliability of tested movements for males and females

<table>
<thead>
<tr>
<th>Movement</th>
<th>Male</th>
<th>Female</th>
</tr>
</thead>
<tbody>
<tr>
<td>Right lateral bend</td>
<td>0.654</td>
<td>0.77</td>
</tr>
<tr>
<td>Left lateral bend</td>
<td>0.693</td>
<td>0.858</td>
</tr>
<tr>
<td>Extension</td>
<td>0.657</td>
<td>0.738</td>
</tr>
<tr>
<td>Flexion</td>
<td>0.742</td>
<td>0.768</td>
</tr>
</tbody>
</table>

*Significance: ICC>0.70.

Table 4. Values representing lower cut-offs of normal measures in mmHg

<table>
<thead>
<tr>
<th>Range</th>
<th>Males (mm)</th>
<th>Female (mm)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Flexion</td>
<td>100</td>
<td>75</td>
</tr>
<tr>
<td>Extension</td>
<td>140</td>
<td>100</td>
</tr>
<tr>
<td>Flexion/extension ratio</td>
<td>80%</td>
<td>75%</td>
</tr>
<tr>
<td>Right bend</td>
<td>100</td>
<td>80</td>
</tr>
<tr>
<td>Left bend</td>
<td>100</td>
<td>80</td>
</tr>
</tbody>
</table>

Bohannon & Lusardi (1991); Perossa et al. (1996) and specifically in testing neck strengths (Vernon et al. 1992; Bussieres 1994).

There are several limiting factors which may have contributed to the lack of statistical and clinical significance of some of the results of the study. The first factor is the variable strength levels of the subjects and the testers. Wikholm & Bohannon (1991) have confirmed that tester strength does influence the magnitude of tested levels as well as the consistency of the measures obtained. In their work, stronger testers obtained higher and less consistent values than testers of average and more equal strength. In the present study, this problem mainly concerned the male volunteers who may have produced variable results by attempting to overpower the examiners. This problem was considerably minimized by instructing both male and female subjects to gradually start exerting pressure against the hand-held sphygmomanometer, thus allowing the tester to gain more control.

The second factor is the potential recruitment of additional trunk musculature during resistance of the neck movements (Bussieres 1994). This factor was minimized by utilizing a chair with a back rest, giving the volunteers a practice trial during which instructions were given and ensuring that the subject’s hands remained in their lap. Since it was the present authors’ aim to develop a test protocol which could be utilized simply and easily in clinical practice, the protocol deliberately omitted some of the measures necessary for the ‘high-tech’ strength testing devices, particularly those involving the need for a special apparatus, and stabilization of the trunk and shoulder in order to isolate specific muscles.

Furthermore, we have not designated the test results as indicative of any specific muscle group (e.g. cervical extensors or the scalene muscles), but rather, of a resisted range of motion (i.e. extension, flexion, and right and left lateral bending).

A third factor involves any potential errors in the process of obtaining and recording the actual test values. The MSD used in this study was a mechanical device with no electronic display or capture of data. As with any circumstances of repetitive testing, the device may have undergone some deterioration of precision, and the examiners may have made errors in reading the test values from the dial. However, the similarity of coefficients of variation for each tested motion argues against a major loss of precision in this study.

Conclusion

The average values obtained in this sample of normal subjects represent a first approximation in determining a normative data base for the manual MSD and neck strength testing. Several features of this data base conform to expected findings, such as the low side-to-side difference in lateral flexions, the predicted ratio of flexion to extension, and the limited range of motion in right and left lateral bending.
to extension and the relatively low coefficients of variation, indicating generally consistent results. The null hypothesis was rejected by demonstrating a significant difference between male and female neck strengths on all tests.

This study has demonstrated excellent levels of intra-examiner reliability for both males and females in neck strength testing. Acceptably high levels of reliability were not shown for inter-examiner measurements in all movements in male subjects, but were shown for female subjects in all tested movements. From this, it can be concluded that the manual MSD may prove useful in neck strength testing, especially when performed by the same examiner. Further studies involving asymptomatic subjects will help determine the instrument’s usefulness in monitoring patient progress in a clinical setting.

Acknowledgements

The authors wish to acknowledge the efforts of Dr. A. Schulte, Saskatchewan, Canada, in devising the manual modified sphygmomanometer dynamometer used in this study, and the assistance of Dr. Cam McDiarmid in providing helpful reviews of initial drafts.

References


Correlations among ratings of pain, disability and impairment in chronic whiplash-associated disorder

Howard Vernon DC FCCS

OBJECTIVE: To investigate the level of correlation among pain, disability and physical impairment scores in chronic whiplash-associated disorder patients.

SUBJECTS: Adults with chronic whiplash-associated disorder referred for secondary independent assessment.

METHODS: Forty-four subjects (16 males, 28 females) were included in the sample. Self-rated pain was measured on a five-point verbal rating scale. Self-rated disability was measured using the Neck Disability Index (NDI) and the Disability Rating Index (DRI). Physical impairment was measured as active cervical ranges of motion obtained with a cap goniometer. Descriptive statistics were reported and Pearson product moment correlations were obtained with the P value at 0.01.

RESULTS: Mean ± SD age of the subjects was 36.4±8.7 years. Mean duration of complaint was 15.2±12.3 months. Mean pain score out of 5 was 2.5±1.2. Mean NDI and DRI scores were 23.2±9.3 out of 50 and 21.6±9.1 out of 48, respectively. These scores correlated very highly (r=0.89, P=0.0001). The average reduction of ranges of motion compared with published norms was approximately 25%. The correlations among ranges of motion, NDI, DRI and pain scores ranged from -0.32 to -0.66 (P<0.05 to P=0.0001). Age and duration of complaint correlated poorly with ranges of motion.

CONCLUSIONS: The self-ratings of pain and disability obtained from these chronic whiplash-associated disorder sufferers appear to be consistent with, and correlate reasonably well with, levels of physical impairment. Physical impairment ratings do not appear to correlate well with duration, which suggests that factors related to pain and physical impairment may play an important role in the development of chronicity in whiplash-associated disorder.

Key Words: Disability evaluation, Impairment, Neck pain, Questionnaires, Whiplash injuries

Corrélations parmi les cotations de la douleur, de l’incapacité et de la déficience dans les troubles associés à l’entorse cervicale chronique

OBJECTIF: Investiger le niveau de corrélation parmi les scores de la douleur, de l’incapacité et de la déficience physique chez les patients accusant des troubles associés à l’entorse cervicale chronique.

Sujets: Les adultes accusant des troubles associés à l’entorse cervicale chronique adressés pour une deuxième évaluation indépendante.

Méthodes: Quarante-quatre sujets (16 hommes, 28 femmes) ont été inclus dans l’échantillon. L’auto-évaluation de la douleur a été mesurée sur une échelle de cotation verbale de 5 points. L’auto-évaluation de l’incapacité a été mesurée à l’aide du Neck Disability Index (NDI) et du Disability Rating Index (DRI). La déficience physique a été mesurée comme amplitude des mouvements cervicaux actifs obtenus à l’aide d’un goniomètre. Les statistiques descriptives ont été rapportées et le coefficient de corrélation produit-moment de Pearson a été obtenu avec la valeur P à 0.01.

Résultats: L’âge moyen ± ET des sujets était de 36,4±8,7 ans. Les patients se plaignaient en moyenne depuis 15,2±12,3 mois. Le score moyen de la douleur sur une échelle à 5 degrés était de 2,5±1,2. Les scores moyens du NDI et du DRI étaient respectivement de 23,2±9,3 et de 21,6±9,1. Ces scores correspondaient fortement (r=0,89, P=0,0001). La réduction moyenne de l’amplitude des mouvements comparée aux normes publiées était d’environ 25%. Les corrélations parmi les amplitudes des mouvements, le NDI, le DRI et les scores de la douleur allaient de -0,32 à -0,66 (P<0,05 à P=0,0001). L’âge et la durée de la plainte des malades correspondaient peu avec les amplitudes des mouvements.

Conclusions: Les auto-évaluations de la douleur et de l’incapacité obtenues chez ces patients souffrant de troubles associés à l’entorse cervicale chronique semblaient en accord avec les niveaux de déficience physique et corrétaient raisonnablement avec ces derniers. Par contre, les cotations de la déficience physique ne semblaient pas bien corrélées avec la durée, ce qui permet de croire que les facteurs liés à la douleur et la déficience physique joueraient un rôle dans le développement de la chronicité dans les troubles associés à l’entorse cervicale.
Morbidity due to 'whiplash-type' injuries resulting from motor vehicle accidents continues to plague Western society (1-12). While the reported incidence of whiplash-related insurance claims appears to vary widely (12), whiplash-associated injuries continue to account for huge health care and social costs. Recent benchmark reports by Spitzer et al (12) and Barnsley et al (1) have crystallized the current state of the art and have concluded that research into the whiplash phenomenon is sadly deficient.

The report by Spitzer and colleagues (12) particularly emphasizes the need for better means to assess whiplash-induced injuries. One of the challenges facing clinicians and researchers is the wide array of symptoms reported by whiplash-injured patients, which complicates diagnosis, classification and prognosis for this complaint. This situation prompted Spitzer et al to propose the term 'whiplash-associated disorder' to encompass the combination of somatic, psychological and social dysfunction associated with whiplash-type injuries.

In addition to their wide range of types and severity, these dysfunction symptoms have been reported to be very persistent (2,3,5,7,11,13-21), leading Barnsley et al (1) to conclude that between 20% and 40% of whiplash-associated disorder sufferers will continue to report symptoms one year after injury, while at least 10% will have virtually permanent problems. On the other hand, Bovim et al (22) and Schrader et al (23) recently reported that the prevalence of 'late whiplash' symptoms was nearly identical to the level of neck pain reported in the general population.

While the scope of symptoms experienced by sufferers of whiplash-associated disorder is broad, relatively few attempts have been made to assess the impact of these symptoms on one's ability to engage in important activities of daily living. In reports on whiplash-associated disorder, symptom checklists are usually reported (2,6,7,11,15,17,21,24), and data on occupational status have been used as a surrogate measure of recovery (12).

In 1991, Vernon and Mior (25) and Vernon (26) developed an instrument designed to assess the disability associated with neck pain, particularly arising from whiplash-induced injuries: the Neck Disability Index (NDI). This instrument was designed as a substantial modification of an existing, well-accepted measure for self-rating of disability due to low back pain (27). The original report, which included data on 48 subjects, 70% of whom had suffered a recent whiplash-induced neck injury, provided evidence of high test-retest reliability (over a two day/no treatment interval), high internal consistency of the index's items, good concurrent validity with the McGill Pain Questionnaire (28) (r=0.70) and good responsivity in a treatment setting.

Since 1991, several reports (reviewed in 26) and one replication study (29) have appeared, all supporting the NDI's psychometric properties. Hains et al (personal communication) found, first, that no order effect existed in either the items or the detractors; second, that one factor -- physical disability -- accounted for 69% of variance; third, that no item weighting was necessary; and fourth, that the correlations between pain scores and disability scores was 0.70.

To the author's knowledge, no study exists in which a condition-specific instrument for measuring neck pain-related disability has been studied in relation to findings of pain and impairment in whiplash injured patients, particularly those whose conditions persist to become chronic. This kind of investigation is particularly important regarding whiplash-injured patients because the usual approach to investigating their clinical status and course has been to employ symptom checklists and measures of psychosocial and/or cognitive dysfunction. Persistence of a variety of symptoms in various proportions of whiplash-associated disorder patients for 12 or more months has been reported (2,6,7,11,15,17,21,24).

What has not yet been investigated in these patients is the degree to which pain and self-perceived disability ratings are associated with levels of physical impairment. This was the aim of the present study, which employed one measure of current self-rated pain intensity, two disability measures and the measurement of active cervical ranges of motion (AROM) in chronic whiplash sufferers referred for secondary assessment. In this study, the questions explored are: is a sample of chronic whiplash-associated disorder sufferers, is there a correlation between levels of self-reported pain and levels of self-reported disability; and, is there a correlation between either or both of self-rated pain and disability levels and levels of objectively measured physical impairment? It was hypothesized that high correlations would be obtained between the two different disability measures (one condition-specific, the other generic [30]) and between these measures and the pain intensity rating. As well, it was hypothesized that there would be low correlations among range of motion measures (impairment), self-rated pain scores and self-rated disability measures, and moderate correlations between age and duration of complaint.

Finally, the possibility that two distinct subsets of items might exist in the NDI -- symptoms and activities -- prompted an exploratory analysis, with moderate correlations between these subsets predicted.

PATIENTS AND METHODS

This study involved an analysis of selected cases of chronic whiplash-injured patients living in a large metropolitan area. The case material was derived from examinations of these subjects conducted for the purpose of third-party payer assessments and by referral from these sources. All cases involved claimants who had suffered motor vehicle accident-related injuries and were continuing to receive ongoing therapy (mainly chiropractic treatment).

Subjects were examined by using a standardized protocol that included the following measures: demographic, clinical and accident-related data; a measure of self-rated current pain intensity (five-point verbal rating scale [28]); the NDI, a condition-specific instrument for self-rating of disability (25); the Disability Rating Index (DRI), a generic instrument for self-rating of disability that is a recently validated 12-item measure of generic self-rated disability (30); and active ranges of cervical motion (see below).

Inclusion of cases in this study was based upon the following criteria.

1. Primary complaint of neck pain. Other symptoms such as headache and low back pain might have been present but were not the primary complaint.

2. Completion of at least one of the disability questionnaires. The primary reason for incompleteness of this criterion was language difficulties.

Of 82 cases presenting from December 1994 to March 1996, 44 satisfied these inclusion criteria and form the case material of this study.
Pain, impairment and disability in whiplash-associated disorder

report. Of those rejected for analysis, 21 were non-English speaking claimants who were unable to complete the disability questionnaires, 11 had a primary complaint of low back pain and six had incomplete files.

Data were extracted by a research assistant. The demographic, clinical and accident-related variables obtained from all files comprised sex, age, ethnic category, English as first or second language, duration of complaint, occupant status in the vehicle (driver, passenger or pedestrian) and mechanism of collision (rear, front or side).

All 44 claimants completed a single NDI (which included a six-point pain rating scale from 0 to 5), while 25 completed the DRI. All cases contained AROM measurements obtained from one trial in all six standard ranges for the neck using a cap goniometer (31,32). AROM measurements were made in a consistent fashion, involving gentle manual guidance at the end range to stabilize the movement so that the maximum voluntary excursion could be measured accurately. No end range pressure was applied, so the subject's pain was solely that experienced from the active movement. AROM assessment has been used in several classic prognostic studies (5,7,11) and in one recent prognostic study (15). Good intra- and interexaminer reliability has been reported for a magnetic goniometer (33-36) as well as for the cap-goniometer used in this study (31). In previous work within our group (unpublished data) interexaminer reliability coefficients obtained with this instrument ranged from 0.64 to 0.86.

The data were analyzed descriptively and with Pearson product moment correlation coefficients with P<0.01. This alpha level was selected to avoid accepting spurious significance given the large number of correlations analyzed. Correlations were conducted between the two disability instruments, and between the condition-specific disability instrument and each of age and duration of complaint. Correlations were also conducted between the range of motion data and both disability instruments, pain scores, age and duration of complaint.

Finally, an exploratory analysis was conducted by separating the NDI items into two categories: symptoms (items 1, 5, 6 and 9) and activities (items 2, 3, 4, 7, 8 and 10). Scores of these items were added to form one category score out of 20 for symptoms and out of 30 for activities. Each of these scores was then correlated with the range of motion data.

RESULTS

Demographic characteristics of the sample, presented in Table 1, show a predominance of Caucasian females in the third to fifth decades of life, with relatively lengthy durations of complaint postaccident. The majority of cases were drivers who were involved in rear-end collisions, although in about one-third of cases, a frontal collision precipitated the pain. The mean ± SD pain intensity score was 2.48±1.09 out of 5. The mean ± SD of scores on the NDI and DRI are shown in Table 2. The correlation between the two instruments was 0.89, P=0.0001. Table 2 also displays correlation coefficients between NDI scores and age and duration of complaint, neither of which was significant.

Table 3 displays data on the AROM, including the mean ± SD for each range as well the average reduction in AROM from published norms (37-39). AROM results from this study were compared with those obtained by Hagstrom and Carlsson (15) and with American Medical Association guides; results are outlined in Table 4. Correlations of the AROMs with the NDI, DRI and pain scores are displayed in Table 5. All correlations were moderately strong and were highly significant, although the correlations with NDI scores were slightly higher.

Table 6 displays the correlations between age and duration of...
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TABLE 4
Comparison of active cervical ranges of motion values (mean ± SD) (in degrees)

<table>
<thead>
<tr>
<th>Range</th>
<th>Present study</th>
<th>Patients</th>
<th>Controls</th>
<th>AMA guides (reference 37)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Extension</td>
<td>48±7</td>
<td>49±2</td>
<td>74±12</td>
<td>60</td>
</tr>
<tr>
<td>Flexion</td>
<td>47±13</td>
<td>38±17</td>
<td>60±9</td>
<td>50</td>
</tr>
<tr>
<td>RLB</td>
<td>37±12</td>
<td>31±13</td>
<td>45±6</td>
<td>45</td>
</tr>
<tr>
<td>LRB</td>
<td>38±12</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>RRot</td>
<td>48±18</td>
<td>51±15</td>
<td>79±7</td>
<td>80</td>
</tr>
<tr>
<td>LRot</td>
<td>51±18</td>
<td></td>
<td></td>
<td></td>
</tr>
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</table>

AMA American Medical Association; LRL Left lateral bend; LRot Left rotation; RLB Right lateral bend; RRot Right rotation.

TABLE 5
Correlations of active cervical ranges of motion with the NDI, DRI and pain scores

<table>
<thead>
<tr>
<th>Range</th>
<th>NDI</th>
<th>DRI</th>
<th>Pain score</th>
</tr>
</thead>
<tbody>
<tr>
<td>Extension</td>
<td>-0.58</td>
<td>-0.54</td>
<td>-0.51</td>
</tr>
<tr>
<td>Flexion</td>
<td>-0.58</td>
<td>-0.52</td>
<td>-0.59</td>
</tr>
<tr>
<td>RLB</td>
<td>-0.66</td>
<td>-0.59</td>
<td>-0.35*</td>
</tr>
<tr>
<td>LRB</td>
<td>-0.49**</td>
<td>-0.32*</td>
<td>-0.34*</td>
</tr>
<tr>
<td>RRot</td>
<td>-0.63</td>
<td>-0.54</td>
<td>-0.53</td>
</tr>
<tr>
<td>LRot</td>
<td>-0.52</td>
<td>-0.45**</td>
<td>-0.53</td>
</tr>
</tbody>
</table>

All results are significant at *P<0.001 except **P=0.05 and ***P=0.001. DRI Disability Rating Index; LRL Left lateral bend; LRot Left rotation; NDI Neck Disability Index; RLB Right lateral bend; RRot Right rotation.

TABLE 6
Correlation of active cervical ranges of motion values with age and duration

<table>
<thead>
<tr>
<th>Range</th>
<th>Age</th>
<th>Duration of complaint</th>
</tr>
</thead>
<tbody>
<tr>
<td>Extension</td>
<td>-0.16</td>
<td>0.14</td>
</tr>
<tr>
<td>Flexion</td>
<td>-0.19</td>
<td>0.15*</td>
</tr>
<tr>
<td>RLB</td>
<td>-0.32</td>
<td>0.03</td>
</tr>
<tr>
<td>LRB</td>
<td>-0.29</td>
<td>0.14</td>
</tr>
<tr>
<td>RRot</td>
<td>-0.12</td>
<td>0.17</td>
</tr>
<tr>
<td>LRot</td>
<td>-0.14</td>
<td>0.01</td>
</tr>
</tbody>
</table>

All values were not significant except *P<0.05. LRL Left lateral bend; LRot Left rotation; RLB Right lateral bend; RRot Right rotation.

TABLE 7
Comparison of Neck Disability Index (NDI) item analysis with the author's original report (25)

<table>
<thead>
<tr>
<th>NDI Items</th>
<th>Mean ± SD</th>
<th>Rank in original 1991 study</th>
</tr>
</thead>
<tbody>
<tr>
<td>Headache</td>
<td>2.91±1.44</td>
<td>1</td>
</tr>
<tr>
<td>Lifting</td>
<td>2.67±1.42</td>
<td>2</td>
</tr>
<tr>
<td>Recreation</td>
<td>2.63±1.29</td>
<td>3</td>
</tr>
<tr>
<td>Reading</td>
<td>2.59±1.05</td>
<td>4</td>
</tr>
<tr>
<td>Pain intensity</td>
<td>2.48±1.09</td>
<td>6</td>
</tr>
<tr>
<td>Work</td>
<td>2.41±1.27</td>
<td>7</td>
</tr>
<tr>
<td>Sleeping</td>
<td>2.37±1.42</td>
<td>8</td>
</tr>
<tr>
<td>Driving</td>
<td>2.34±1.27</td>
<td>5</td>
</tr>
<tr>
<td>Concentration</td>
<td>1.98±1.34</td>
<td>9</td>
</tr>
<tr>
<td>Personal care</td>
<td>1.00±0.98</td>
<td>10</td>
</tr>
</tbody>
</table>

DISCUSSION

The link among pain, impairment and disability is likely not symmetrical (40-50). This phenomenon has been studied much more extensively in low back pain patients than in those with neck pain. In the 1980s, several instruments were developed to assess functional disability relative to activities of daily living in low back pain sufferers including the Oswestry Low Back Pain Disability Questionnaire (OLBPDQ) (27), and the Roland-Morris Questionnaire (51). Others followed, including generic (as opposed to condition-specific) instruments for measuring pain-related disability (50,52-55).

Investigators have used these instruments to assess the relationship among pain, impairment and disability in low back pain. Triano and Schultz (56) found that low back pain sufferers with complaint and the ranges of motion. These were all generally low, with none being found significant at P=0.01. Item analyses for the NDI, showing the ranking of the mean ± SD for all 10 items, are compared with those from the author's original report (25) in Table 7. For the exploratory analyses, the average correlation coefficient for AROM/category 1 (symptoms) was 0.45, while it was 0.61 for AROM/category 2 (activities). The two categories were moderately but significantly correlated (r=0.55, P<0.05).

OLBPDQ scores of 13 or higher out of 50 were much more likely to demonstrate paraspinal muscular dysfunction, as measured by electromyogram, and altered trunk active ranges of motion, particularly in forward flexion.

Waddell and Main (47) and Waddell et al (48,57) reported that disability levels in low back pain sufferers correlated more highly with psychosocial measures of distress than with measures of pain severity.

Gronblad et al (58-62) reported relatively good correlations in chronic low back pain patients between disability scores, as measured by a condition-specific instrument, the OLBPDQ, and a generic instrument, the Pain Disability Index (63) (r=0.83), and measures of impairment, particularly the performance of single physical performance tests in the trunk (r=0.30 to 0.41) (62). Their most recent work, however, failed to demonstrate strong correlations between disability ratings and trunk AROM scores.

The investigation of this relationship is in its infancy regarding the assessment of disability in neck pain sufferers. In Morris and Watt's classic report (11), it is indicated that AROM measurements were obtained in their sample of 61 whiplash-associated disorder patients, but these values were not reported. Hildingsson and Tolman (6,7) reported on the persistence of reduced AROM at 25 months' follow-up in their sample of 93 motor vehicle accident...
subsets of items, which might be regarded as subfactors related to the primary factor of physical disability, namely symptoms and activities. This analysis suggests that these two subsets may offer unique information and that some subjects may endorse higher levels of symptom expression compared with activity reduction, and vice versa.

The average reduction in active range of motion compared with published norms (2,4,39) was approximately 25%, with extension and right rotation the ranges with the highest reduction (31%). The reduction in extension is consistent with previous reports of the long term stiffness of the anterior cervical musculature (2,8,10). The bilateral implication of this finding would be manifested in reduced extension (i.e., reduced movement in the sagittal plane), while unilateral anterior myofascial stiffness would result in reduced rotation (ipsilateral) and lateral bending (contralateral).

Neither of the bilateral measures (rotation or side bending) demonstrated any significant side-to-side differences. This is consistent with data on neck range of motion testing in controls and sincere pain subjects (31-36). Taken together, however, the range of motion deficits observed in these subjects constitute only a moderately severe impairment level that is somewhat less than would be expected given the chronicity of these cases.

The degree to which current pain severity levels in chronic whiplash-associated disorder sufferers is associated with self-rated disability and objectively measured impairment is important because, as Radanov and Sturzenegger (17) and Radanov et al (18-20) have shown, there is a strong association among initial pain, injury severity ratings and the development of persistent symptoms (i.e., chronicity). The correlations of pain and disability measures with impairment measures (AROM) found in this present sample are surprisingly high, with the highest correlations generally observed between AROM and the NDI scores. All correlations were in the expected negative direction given the nature of the data used for the analysis; i.e., lower range of motion measures were reasonably well-correlated with higher self-rated pain and disability scores. Because NDI, DRI and pain scores are all reasonably well-correlated, the fact that the correlations between these scores and AROM were all in generally the same range (0.30 to 0.60) is not surprising.

The lack of correlation between age and AROM is interesting given that AROM is known to reduce with advancing age (38,39). However, the age range of our subjects was relatively small and confined to the third to fifth decades, which is before the age range when progressive degenerative disc disease becomes significant. As well, the progressive, age-related reductions in AROM are generally small and continuous, making it unlikely that age accounts for the reductions observed in this sample.

The lack of correlation between AROM and duration is somewhat surprising, in that chronicity may be associated with progressive increase in impairment. One possible explanation for this unexpected finding is that those whiplash-injured claimants who go on to experience chronic difficulties may reach a plateau of pain, impairment and self-rated disability, the complex of which remains approximately static from that time onwards. Because this was not a prospective study, this explanation remains to be confirmed in a longitudinal study. It is, however, consistent with the follow-up data reported in the literature (2,6,7,11,15,17,21,24) on persistent symptomatology and cognitive difficulties in the minority of whiplash-injured cases that go on to become chronic.
This study has several important limitations. While the self-report measures were provided by the subjects themselves, and while all case data were extracted by a research assistant, the AROM values were obtained by the author in an unblinded fashion. Bias may have been introduced here. Future studies should employ an independent examiner for this or any other physical impairment measures. A case-control design that blinds the examiner as to subject status would be ideal.

Second, the study sample may have been skewed by factors related to the referral for assessment. Not all chronic whiplash-associated disorder cases are necessarily chosen for assessment referral. Factors acting to facilitate referral, such as case management issues, litigation status and dispute with the insurer, may have created a selection bias that then influenced the measures obtained in the study. However, study sample characteristics were similar to those of previously reported whiplash-associated disorder groups regarding sex, age, occupant status and collision type (2,7,9,11,12,14). As well, the results of the analysis of NDI scores in this sample were quite similar to those presented in the first report (25) whose sample comprised 75% whiplash-associated disorder sufferers. Nonetheless, an ideal study would involve a prospective cohort assessed with these instruments on a serial basis in order to investigate the evolution of the condition and the extent to which similar correlations between the variables reported here might exist.

REFERENCES

CONCLUSIONS
A sample of chronic sufferers of whiplash-associated disorder has been investigated to explore the relationships among self-reported pain, disability scores and impairments as measured by reductions in AROM. Two instruments for measuring self-rated disability, one condition-specific and the other generic, correlated very well with each other and with pain scores. The correlations between these two measures and pain scores with AROM scores were moderately strong and highly statistically significant. AROM scores did not correlate well with age and duration of complaint, which suggests that chronicity in whiplash-associated disorder may be more directly linked to loss of physical function, and that whiplash-associated disorder sufferers may reach a plateau of reduced function and increased self-ratings of pain and disability, which then remain stationary. These findings also suggest that treatment interventions that target this link may be fruitful, either by directly increasing neck range of motion or by reducing self-ratings of pain and disability, particularly by focusing on attitudes and behaviours related to the avoidance of the very movements (57) that are actively restricted.

ACKNOWLEDGEMENTS: The author thanks Dr Cam McDermaid for thoughtful reviews and Bev Fuller for typing.
Pain, impairment and disability in whiplash-associated disorder


Chiropractic management of episodic tension-type headache: a survey of clinical specialists

Howard Vernon, DC. FCCS(C)*
Cameron McDermaid, DC*

Tension-type headache (TTH) is a highly prevalent condition experienced annually by 30–70% of the population. As a chief complaint, it occupies 5–8% of chiropractors’ caseloads, but is probably more prevalent in multiple complaint cases. While numerous clinical descriptions exist in the literature of the management of TTH by chiropractors, and while there is a small body of clinical trials of the treatment of non-migrainous headache by spinal manipulation, there is no systematic survey of the approaches to its treatment by chiropractors.

The goals of this study were to determine the test-retest reliability of a questionnaire designed to identify the most commonly used treatments for TTH and to report on any consistent findings as a potential profile of typical practice approach. The respondents consisted of a group of Canadian chiropractic clinical specialists. Respondents were asked to complete a survey which consisted of a comprehensive list of chiropractic treatment procedures including standard manual manipulations and mobilisations, soft tissue therapies, modalities, exercises, behavioral therapies, acupuncture, nutrition and four “systems” techniques. The respondents were asked to rate their frequency of use of these procedures on a 4-point scale ranging from “always” to “never”. The surveys were completed twice within a two day interval.

The response rate was 18/25 (72%). Eighty-seven percent (87%) of the items were rated identically on both surveys. All but one of the items achieved a statistically significant reliability coefficient. The highest rated items were “upper cervical manipulations”, “upper cervical soft tissue therapy” and “neck stretching exercises”. The items which received the lowest endorsement were:

Les céphalées de tension (CT) sont très répandues et touchent chaque année 30 pour cent de la population. Les CT motivent à elles seules de 5 à 8 pour cent des consultations auprès des chiropraticiens. mais elles sont sans doute plus fréquentes si on les considère comme l’un des symptômes d’une affection. Bien que la documentation scientifique fasse état de nombreuses descriptions cliniques de traitement des CT par des chiropraticiens et d’un petit nombre d’essais cliniques de traitement des céphalées non migraineuses par des manipulations vertébrales, aucune enquête systématique n’a été menée sur les méthodes de traitement des CT par les chiropraticiens.

La présente étude avait pour objet de vérifier la fiabilité d’un questionnaire visant à dégager les traitements les plus courants des CT et de rendre compte des réponses les plus fréquentes pouvant aider à dresser le profil d’une démarche typique. Les personnes interrogées avaient été recrutées parmi un groupe de spécialistes cliniques canadiens en chiropratique. On leur a demandé de remplir un questionnaire présentant la liste exhaustive de traitements en chiropratique, à savoir les manipulations et les mobilisations manuelles courantes, les traitements des tissus mous, les modes de traitement, les exercices, les thérapies comportementales, l’acupuncture, l’alimentation et les techniques des quatre “systèmes”. Il s’agissait d’évaluer, de un à quatre, ou de « Toujours » à « Jamais », la fréquence d’utilisation des techniques mentionnées ci-dessus. Le questionnaire devait être rempli deux fois, à deux jours d’intervalle.

Le taux de réponse a été de 18 sur 25, soit de 72 %. Quatre-vingtième pour cent des réponses ont été identiques les deux fois. Toutes les questions, à

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chiropractic procedures to the dorso-lumbo-pelvic spine, most therapy modalities and the "systems" techniques which were included in the survey. Years in practice appeared to have very little effect on the use of the various procedures.

With very minor exceptions, this group of respondents provided reliable and consistent responses which were also consistent with the clinical trial literature on the treatment of headache by spinal manipulation. We interpret this to indicate that the survey instrument has an acceptable level of reliability and validity for use in any larger study of field practitioners' approaches to the treatment of TTH. The set of procedures endorsed by this group of specialists is presented as a possible set of "best-evidence practices" in the chiropractic management of tension-type headache.


KEY WORDS: chiropractic, manipulation, headache.

Introduction

Tension-type headache (TTH) is a highly prevalent condition. Annual prevalence rates reported in a Canadian population-based survey were 36% of respondents or 30% of the population for moderately severe headaches. A recent Danish survey reported a 66% annual prevalence rate for any level of tension-type headache (56% for men; 71% for women).

In 1988, the International Headache Society (IHS) reclassified tension or muscle contraction headache as "tension-type" (Category 2) and created two major subcategories: Episodic TTH (ETTH) in which headache frequency is less than 15 per month, and "Chronic" TTH for sufferers with more than 15 headaches per month. The former category is by far the most prevalent, with less than 5% of TTH sufferers in the "chronic" category. In headache groups, adult sufferers typically report long durations of their headache conditions. The other IHS criteria for TTH are listed in Table 1.

Estimates of the proportion of headache sufferers treated by chiropractors range from 3-9% of patients general practice. In one study, 35% of headache patients in a chronic pain programme had received prior chiropractic treatment.

There is a small body of clinical trials of spinal manipulative therapy for non-migrainous headaches, although only one study applied the criteria for TTH. Spinal manipulative therapy (SMT) appears to be more efficacious than ice treatment or soft tissue therapy and is equal in effectiveness to a sham treatment.
Table 1
Criteria for TTH from the IHS Classification\(^3\)

**Description:** "Recurrent episodes of headache lasting minutes to days. The pain is typically pressing/tightening in quality, of mild or moderate intensity, bilateral in location and does not worsen with physical activity. Nausea is absent, but photophobia or phonophobia may be present".

**Diagnostic Criteria:**
A. At least 10 previous headache episodes fulfilling criteria B–D below.
B. Headache lasting from 30 min to 7 days.
C. At least 2 of the following pain characteristics:
   1. Pressing/tightening (non-pulsating) quality.
   2. Mild or moderate intensity (may inhibit, but does not prohibit activities).
   3. Bilateral location.
   4. No aggravation by walking stairs or similar physical routine.
D. Both of the following:
   1. No nausea or vomiting (anorexia may occur)
   2. Photophobia and phonophobia are absent, or one but not the other is present.
E. At least one of the following:
   1. History, physical and neurological examinations do not suggest any other headache diagnosis.
   2. History and/or physical and/or neurological examination do suggest such disorder, but it is ruled out by appropriate investigations.
   3. Such disorder is present, but tension-type headache does not occur for the first time in close temporal relation to the disorder.

course of low-dose amitriptyline therapy,\(^8\) although the authors of this latter study reported longer-lasting relief post-treatment in the group receiving chiropractic SMT. While many articles and book chapters exist which outline a variety of approaches to the conservative, non-pharmacologic treatment of TTH,\(^12,13\) no systematic survey of practitioners’ approaches has been reported. In other words, there is no reliable data on which treatment approaches chiropractors actually use in daily practice in the treatment and management of TTH.

This study presents data from a relatively simple questionnaire completed by a group of chiropractic clinical specialists. By having practitioners complete the survey twice within a two-day interval, we determined the test-retest reliability of the instrument. This is a necessary requisite for employing this survey in a larger, random sample of field practitioners to determine their common practices. This report also presents some preliminary data on the set of common procedures used by this specialist group which, taken together, may represent a collection of “best-evidence” practices in the chiropractic management of TTH.

**Methods**
The questionnaire contains a list of manual and conservative therapy procedures which were gleaned from reviewing the published clinical trials and other clinical reports (as reviewed above). The list and its format were reviewed by a group of content and methodology experts at our institution for face and construct validity. After incorporating their suggested revisions, the final questionnaire was prepared.

Respondents were asked to rate their use of each procedure using a four-point scale: 3 = always, 2 = sometimes, 1 = infrequently and 0 = never. No attempt was made to solicit ratings of the respondents’ perceived effectiveness of these procedures. A small set of demographic items was included as well as an item asking respondents to indicate...
whether their approach to scoring the questionnaire (and therefore, their management approach) was "typical of most chiropractors".

The survey was mailed to a convenience sample of 25 Fellows of the College of Chiropractic Clinical Sciences (CCS) (approximately half the members of this organization). It was felt that such practitioners would be more inclined to respond, and would therefore better suit the needs of this reliability study.

The practitioners were mailed two copies of the questionnaire and asked to complete one immediately and then the other two days later. Aside from instructions on the scoring scale, respondents were instructed to consider their answers with respect to the treatment of "their last five tension-type headache patients".

Data were analyzed using SPSS for Windows. Mean (sd) scores of each item were produced. The paired questionnaires were analyzed for the percentage of items which were scored identically both times as well as for scores of a one-rank or a two-rank difference. These differences were analyzed by McNemar's non-parametric Chi Square test as compared to chance findings. Spearman Rank Order Correlation Coefficients were also calculated for each item which was scored at least 85% of the time, then paired answers would be pooled and cross-tabulations of career length (< 10 years) would be computed in order to determine if the responses were truly homogeneous across practice experience.

**Results**

The survey response rate was 18/25 or 72%. Respondent mean age was 42 years (range: 30 to 60). All but one of the respondents was male. The mean (sd) number of years in practice was 16.4 (8.5), with 31% under 10 years and 69% ten years or over. All respondents were Fellows of CCS.

The percentage of identical answers was 87%, while Table 2:

### Results for Manual and Physiotherapeutic Procedures

<table>
<thead>
<tr>
<th>Procedure</th>
<th>Mean (sd)</th>
<th>Spearman's r</th>
</tr>
</thead>
<tbody>
<tr>
<td>Upper cervical SMT</td>
<td>2.5 (.63)</td>
<td>.74 p &lt; .0005</td>
</tr>
<tr>
<td>Soft tissue therapy</td>
<td>2.2 (.77)</td>
<td>.85 p &lt; .0005</td>
</tr>
<tr>
<td>Trigger point work</td>
<td>1.93 (.80)</td>
<td>.94 p &lt; .0005</td>
</tr>
<tr>
<td>Upper cerv. mobilizations</td>
<td>1.75 (.77)</td>
<td>.84 p &lt; .0005</td>
</tr>
<tr>
<td>Massage</td>
<td>1.73 (.88)</td>
<td>.86 p &lt; .0005</td>
</tr>
<tr>
<td>Mid-cerv. SMT</td>
<td>1.56 (.51)</td>
<td>.88 p &lt; .0005</td>
</tr>
<tr>
<td>Upper thoracic / rib SMT</td>
<td>1.27 (.46)</td>
<td>.44 p = .12</td>
</tr>
<tr>
<td>Lower cervical mobilization</td>
<td>1.25 (.77)</td>
<td>.95 p &lt; .0005</td>
</tr>
<tr>
<td>Modalities (any)</td>
<td>.93 (.70)</td>
<td>.87 p &lt; .0005</td>
</tr>
<tr>
<td>IFC</td>
<td>.82 (.98)</td>
<td>.87 p &lt; .0005</td>
</tr>
<tr>
<td>Ultrasound</td>
<td>.75 (.75)</td>
<td>.91 p = .0005</td>
</tr>
<tr>
<td>Thoracic mobilizations</td>
<td>.75 (.77)</td>
<td>.72 p = .0005</td>
</tr>
<tr>
<td>Low Volt</td>
<td>.67 (.89)</td>
<td>.84 p = .0005</td>
</tr>
<tr>
<td>TMJ STT</td>
<td>.67 (.72)</td>
<td>.82 p = .0005</td>
</tr>
<tr>
<td>Shoulder mobilizations</td>
<td>.50 (.63)</td>
<td>.78 p = .0005</td>
</tr>
<tr>
<td>TMJ SMT</td>
<td>.47 (.52)</td>
<td>1.0 p = .0005</td>
</tr>
<tr>
<td>Lower dorsal SMT</td>
<td>.44 (.63)</td>
<td>.77 p = .0005</td>
</tr>
<tr>
<td>Cranial STT</td>
<td>.40 (.74)</td>
<td>.64 p = .21</td>
</tr>
<tr>
<td>Sacroiliac SMT</td>
<td>.38 (.62)</td>
<td>.88 p &lt; .0005</td>
</tr>
<tr>
<td>Lumbar SMT</td>
<td>.31 (.48)</td>
<td>.86 p &lt; .0005</td>
</tr>
<tr>
<td>Scapular SMT</td>
<td>.25 (.45)</td>
<td>.83 p &lt; .0005</td>
</tr>
</tbody>
</table>
percentage of one-rank and two-rank differences were 12% and 1%, respectively. In other words, respondents agreed with themselves within one ranking 97% of the time. None of the McNemar's tests achieved a p value greater than .03, indicating that all paired comparisons achieved agreement levels which were significantly different from chance.

The mean(sd) scores (ranked from highest to lowest) and the Spearman Correlation coefficients for each item are shown in Tables 2 and 3, for manual and non-manual procedures, respectively. The only item which achieved a test-retest agreement above 85% and which demonstrated differences related to career length on cross-tab analysis was “postural exercises”, with 76% percent of more experienced practitioners endorsing this as compared to 19% of those with less than ten years’ experience.

Table 4 lists additional procedures volunteered by the respondents which were not directly included in the survey. Forty-seven percent (47%) of respondents indicated that their approach to treating TTH was “typical of most chiropractors”, while 88% indicated that there were no other treatment approaches which they used which were not included in the questionnaire.

### Discussion
The 72% response rate was deemed adequate for our purposes. The sample of respondents certainly differs from the field practitioner profile in being Clinical Specialists, virtually all of whom were males. Aside from the gender bias, this sample was preferred because the additional training and experience of these specialists permits the results of the survey to represent the “best-evidence practice” approach to the chiropractic management of TTH. As well, CCS members were thought to be more likely to have a consistent approach to treating TTH, thereby increasing the likelihood of highly consistent responses both within and between respondents. Our data bears this supposition out.

A review of Tables 2 and 3 reveals that most items achieved a very high level of test-retest reliability. No item with a correlation below 0.70 achieved a mean score above 2/3, indicating that these less reliably endorsed procedures were employed much less frequently than those achieving higher levels of reliability. The items whose reliability coefficients were below 0.70 but which achieved an average endorsement between “sometimes” and “usually” (i.e., between 1–2/3) were: upper thoracic and costovertebral

<table>
<thead>
<tr>
<th>Procedure</th>
<th>Mean(sd)</th>
<th>Spearman’s r</th>
</tr>
</thead>
<tbody>
<tr>
<td>Neck stretching</td>
<td>2.13 (.81)</td>
<td>.88 p = .0005</td>
</tr>
<tr>
<td>Shoulder stretching</td>
<td>1.5 (.82)</td>
<td>.70 p = .003</td>
</tr>
<tr>
<td>Postural exercises</td>
<td>1.5 (.73)</td>
<td>.85 p = .0005</td>
</tr>
<tr>
<td>Neck strengthening</td>
<td>1.3 (.58)</td>
<td>.68 p = .003</td>
</tr>
<tr>
<td>Self - STT</td>
<td>1.1 (.62)</td>
<td>.53 p = .03</td>
</tr>
<tr>
<td>Relaxation advice</td>
<td>1.1 (.72)</td>
<td>.66 p = .005</td>
</tr>
<tr>
<td>Aerobic exercise</td>
<td>1.1 (.72)</td>
<td>.83 p = .0005</td>
</tr>
<tr>
<td>Stress management</td>
<td>1.0 (.57)</td>
<td>.54 p = .03</td>
</tr>
<tr>
<td>Psychological counsel.</td>
<td>.75 (.68)</td>
<td>.96 p = .0005</td>
</tr>
<tr>
<td>Shoulder strengthening</td>
<td>.56 (.63)</td>
<td>.90 p = .0005</td>
</tr>
<tr>
<td>Activator</td>
<td>.38 (.62)</td>
<td>.99 p = .0005</td>
</tr>
<tr>
<td>Somatics</td>
<td>.38 (.50)</td>
<td>.62 p = .01</td>
</tr>
<tr>
<td>AK</td>
<td>.19 (.54)</td>
<td>.99 p = .0005</td>
</tr>
<tr>
<td>SOT</td>
<td>.13 (.34)</td>
<td>.68 p = .004</td>
</tr>
<tr>
<td>Heel lifts</td>
<td>.06 (.25)</td>
<td>.68 p = .004</td>
</tr>
<tr>
<td>Craniosacral Therapy</td>
<td>0</td>
<td></td>
</tr>
<tr>
<td>Homeopathy</td>
<td>0</td>
<td></td>
</tr>
</tbody>
</table>

Table 3
Results for Non-manual and “systems” therapies
manipulation, stress management, advice on relaxation therapy, self-stretching advice and the prescription of neck strengthening exercises. With these few minor exceptions, we consider that this survey has adequate test-retest reliability for use in a larger sample of field practitioners.

The procedures which received the highest level of endorsement (i.e., used at least half the time (using a cut-off value of 1.5/3)) were, in descending order:

- upper cervical manipulation;
- soft tissue therapy, in general;
- neck stretching exercises;
- trigger point therapy;
- upper cervical mobilization;
- massage;
- mid-cervical manipulation;
- shoulder stretches, and
- postural exercises.

It is interesting that these procedures reflect what might be described as a "holistic structural" approach, in that they combine segmental and regional manipulation, mobilization and exercises to both (putatively) correct underlying spinal dysfunction and to provide supportive or rehabilitative benefit. These procedures may be considered, and are thus offered, as a set of "best-evidence" practices endorsed by this group of Clinical Specialists. There were virtually no differences in the treatment approaches endorsed by those practitioners with less or more than ten years of practice experience. The one exception, i.e., the greater endorsement of "postural exercises" by the more experienced group, may reflect an even more holistic model in which these practitioners were trained.

These results, of course, do not imply that these procedures actually have proven effectiveness, particularly in every case of TTH, and particularly in the case of the non-manipulative procedures, but that they are a set of procedures deemed by specialist-level practitioners to have value in the management of ETTH. These findings are also consistent with the clinical trial literature (as reviewed above) and with the recommendations of a recent RAND consensus panel on the appropriateness of manipulation for cervical spine disorders, and headache in particular.14 This leads to our conclusion that the survey form has good construct validity, a conclusion which is further strengthened by the finding that so few respondents felt that there were any important additional procedures.

The set of procedures which received low endorsement (mean values below 1/3) include, with one exception ("psychological counseling"), additional manual therapy to areas which are apparently viewed by these respondents as of much less importance in the management of TTH. These include: manipulation of the lower dorsal, lumbar, and sacroiliac joints as well as the temporomandibular joint (TMJ); mobilizations to the shoulders and thoracic spine; soft tissue therapy to the cranial and TMJ area; specific physiotherapeutic modalities (although the use of any physiotherapeutic modality was endorsed at just below the "sometimes" level); strengthening exercises to the shoulder area; generalized approaches to somatic therapy and nutrition, and four "systems" approaches to chiropractic treatment (Applied Kinesiology, Sacro-Occipital Technique, Activator Methods and Craniosacral Therapy).

The lack of endorsement of treatment procedures for the TMJ area is interesting, particularly given the well-established sensory-motor connections between the TMJ and upper cervical regions. One could surmise that these specialists hold the opinion that sufficient benefit can be obtained by directing their treatments at the upper cervical region alone.

Two procedures, homeopathy and craniosacral therapy, were endorsed by none of the respondents. These results might be interpreted as representative of a narrower, "non-holistic" perspective which some may attribute to clinical specialists. We prefer to interpret these results as representing the strong evidence-based approach to practice adopted by most such specialists, although it should be noted that even some of these respondents did endorse some of these less commonly used practices. The proce
dures which were not included in the questionnaire and which were offered by the respondents are shown in Table 4. Perhaps it was felt that these procedures could be of some benefit to a subset of their TTH patients. The most commonly cited reason for the respondent to have the view that their approach to treating TTH might not be fully representative of the typical chiropractor’s approach was that field practitioners might “adjust more of the full spine”. To date, such a “full spine” approach to chiropractic spinal manipulation has not been investigated in any of the clinical studies, so its use is strictly empirical.

Conclusion
The questionnaire used in this survey has been found to have an acceptable level of test-retest reliability. The procedures most highly endorsed by the respondents, themselves a somewhat selected group of practitioners, are consistent with the evidence in the literature as to which procedures have demonstrated effectiveness in the treatment of TTH or non-vascular headache. In this respect, this questionnaire has been shown to have good content and construct validity. The questionnaire contains a sufficiently wide variety of items so that its ability to capture the breadth of practice approaches amongst general practitioners is likely very high. This would apply to those practitioners who employ both standard and unconventional procedures such as nutritional counseling, somatic therapy, full-spine manipulation and “systems” techniques.

At present, we offer this profile of specialist-endorsed procedures, particularly upper cervical manipulation and soft tissue therapy accompanied by a stretching exercise prescription, as a set of “best-evidence practices” for use in chiropractic practice for the treatment of tension-type headache.

Acknowledgments
The authors wish to thank the respondents of the survey for their participation and Dr. Judy Waalen for her assistance with data analysis and for her thoughtful review of the manuscript. We are grateful for the funding support provided for this study by the Ontario Chiropractic Association and the National Chiropractic Mutual Insurance Company.

References
SUMMARY. Objectives: To conduct a systematic review of the randomized controlled clinical trials (RCTs) of complementary/alternative (CAM) therapies in the treatment of non-migrainous headache (i.e. excluding migraine, cluster and organic headaches).

Design: Systematic review with quality scoring and evidence tables. Main outcome measures: Number of RCTs per therapy, quality scores, evidence tables. Results: Twenty-four RCTs were identified in the categories of acupuncture, spinal manipulation, electrotherapy, physiotherapy, homeopathy and other therapies. Headache categories included tension-type (under various names pre-1988), cervicogenic and post-traumatic. Quality scores for the RCT reports ranged from approximately 30 to 80 on a 100 point scale. Conclusion: RCTs for CAM therapies of the treatment of non-migrainous headache exist in the literature and demonstrate that clinical experimental studies of these forms of headache can be conducted. Evidence from a sub-set of high quality studies indicates that some CAM therapies may be useful in the treatment of these common forms of headache.

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INTRODUCTION

Tension-type headache (TTH) is the most prevalent form of adult benign headache. Recent population-based studies have estimated its prevalence as 35–40% of the adult population in Western societies.1–3 TTH contributes to a large burden of disability, resulting in lost work-days, diminished quality of life, and considerable health care costs to both governments and institutional payers.4,5 Individual sufferers share in these costs, because the predominant approach to treatment of TTH is the use of over-the-counter analgesic medications5 for symptomatic relief.

The etiology of TTH is unclear at present. Older models of painful cranial muscular contraction, possibly induced by psychological tension, have been rejected.3–8 In 1988, the International Headache Society (IHS) promulgated a classification of headaches9 which did not endorse any particular etiological mechanism for TTH, and, as such, recommended changing the name of this condition from ‘tension headache’ to the current form of ‘tension-type headache’. The IHS classification includes two categories: headaches less frequently than 15 per month are known as episodic TTH, while the term chronic TTH is reserved for the small minority (about 3%)14 who suffer headache more frequently than every other day. The IHS-based definition of TTH is given in Figure 1.

Cervicogenic headache (CH) is a recently validated type of headache,10–11 although its existence
Review of RCTs of CAM therapies

1) Tension-Type Headache:
A. At least 10 previous headache episodes fulfilling criteria B to D.
B. Headache lasting from 30 minutes to 7 days.
C. At least two of the following criteria:
   i. Pressing /tightening (non-pulsatile) quality
   ii. Mild or moderate intensity (may inhibit, but does not prohibit activity)
   iii. Bilateral location
   iv. No aggravation by walking, stairs or similar routine physical activity.
D. Both of the following:
   i. No nausea or vomiting (anorexia may occur)
   ii. One of photo-or phonophobia may be present, but not both.

2) Cervicogenic Headache:
A. Pain localized to the neck and occipital region. May project to forehead, orbital region, temples, vertex or ears.
B. Pain is precipitated or aggravated by special neck movements or sustained postures.
C. At least one of the following:
   i. Resistance to or limitation of passive neck movements
   ii. Changes in neck muscle contour, texture, tone or response to active and passive stretching and contraction
   iii. Abnormal tenderness of neck muscles.
D. Radiological examination reveals at least one of the following:
   i. Movement abnormalities in flexion/extension
   ii. Abnormal posture
   iii. Fractures, congenital abnormalities, bone tumours, rheumatoid arthritis or other distinct pathology (not spondylosis or osteochondrosis).

Fig. 1 Criteria for tension-type headache and cervicogenic headache from the IHS classification.

had been proposed by investigators in the medical field for many decades. Nilsson, using the criteria established by the IHS, reported the prevalence of cervicogenic headache in a Scandinavian population to be approximately 16%. There is still some confusion in the clinical profiles of TTH and CH. The IHS-based definition of CH is given in Figure 1.

A lack of clarity also exists for the etiology of CH; however, by definition, it must involve pain referred to the head originating from structures in the cervical spine. The upper cervical spine has been particularly implicated, principally as the upper cervical spinal cord and lower brain stem share a common input of pain afferent fibres from the trigeminal and upper cervical sensory systems.

A number of systematic reviews and meta-analyses have been reported on treatments for headache. The interventions studied in these reviews have been confined to pharmacological therapies and cognitive/behavioural therapies. The majority of these reviews have been for treatments of migraine-type headache. Bogaards and ter Kuile's recent meta-analytic review of treatments for 'recurrent tension headache' confined itself to the following categories of intervention: pharmacological, cognitive therapy, relaxation therapy, EMG biofeedback therapy and combinations, although some CAM therapies (acupuncture and physiotherapy) were regarded as control or 'pseudo-placebo' treatments. No primary complementary/alternative (CAM) therapies were included. It appears that systematic review of CAM therapies non-migrainous headaches currently exists in the literature.

Since the publication of Eisenberg's impulsive Health article describing the usage of CAM therapies among Americans interest in the topic within orthopaedic and medical circles has grown and the use of CAM therapies in society has increased considerably. Therapy utilization rates in suffers of TTH are poorly understood. Several studies cite the proportion of patients seeking chiropractic care for headache to be approximately 3–10% of patients presenting.

Our group is pursuing a number of clinical studies in TTH and CH, with a particular interest in non-pharmacological therapies which have been used for migraineous headaches. In the non-pharmacological group there are two main categories of therapy first of these includes psychologically-based treatments involving cognitive or behavioural therapy such as biofeedback and counselling. The second category involves CAM therapies, including acupuncture, chiropractic, physiotherapy, homoeopathy and others. This report will refer to this latter category.
The goal of this report was to present an analysis of the randomized clinical trials of the efficacy of CAM treatments for non-migrainous headache. We have defined 'non-migrainous headache' as excluding migraine (with or without aura), cluster and any organic types of headache. In this study, we employed standardized methods for literature searching and evaluating the quality of the relevant studies.

METHODS

Literature review

A literature search was conducted of MEDLINE (English-language, 1966 to mid-1998), PsychInfo and CINHAL databases. The MEDLINE search strategy is given in Figure 2. Once these searches were obtained, supplementary searches of citations and reference lists in other systematic literature reviews, as well as author queries, were undertaken.

Inclusion criteria

From the total initial citation lists, a screening process was undertaken by the senior author to identify the study design as one of the following: clinical trial, case series, case report or letter to the editor. Only randomized controlled trials (RCTs) were retained for analysis in this report. Relevant RCTs were defined as prospective studies with a sample of adult headache sufferers in which at least two groups were randomly allocated to receive one or more interventions. Studies involving exclusively migraine, cluster or organic types of headache were excluded. A small number of studies included both tension (-type) and migraine groups, and were included in the review. In some reports, older terminology such as 'muscle contraction' or 'tension' headache was used and these studies were included.

Studies were included in the review if they reported clinical outcomes related to headache activity (i.e. headache index, severity, frequency, medication usage). Studies which reported only physiological outcomes (EMG measurements only, blood chemistry, eye function), without clinical measures were not included. Papers had to be published in the English language.

Quality scoring

Data abstraction and quality reviews were independently conducted by two reviewers (CM and CH), using a standardized abstraction form and a quality review protocol modified from van Tulder et al.26 (further details are available from the authors on request). This quality review protocol was deemed most appropriate for our purposes as it was devised for reviewing clinical trials of non-medical treatments for spinal pain. As such, items pertaining to medications (dosages, side effects, monitoring via blood samples, etc.), which would be relevant to drug trials and which appear in other quality review schemes,17 were excluded to prevent quality decrements from unfairly being applied to the CAM studies.

The reviewers were not blinded to the source of the citations. While there is evidence that a difference may exist between blinded and unblinded reviews, the differences demonstrate little consistency in direction of bias or its magnitude.24

The reviewers included one clinician (CM) as well as a non-clinician methodologist (CH). The reviewers’ scores were assessed for consistency with the Intraclass Correlation Coefficient. The standard error of the mean of the difference scores was calculated to determine the absolute level of difference between the reviewers’ scores.

Evidence tables were constructed to include author(s), year of publication, study duration, sample size, headache type (as well as use of IHS classification) and a review of the outcome of the trial, specifically whether a positive or negative result was obtained when comparing the experimental to the comparative or control treatment(s). No statistical pooling was attempted.

The quality review protocol contains eighteen items answered by ‘yes, no, don’t know’ scores. The latter two response categories were collapsed, making the scoring dichotomous. No weighting factor was used. Scores, therefore, range from 0–18, and were converted to percentages for ease of interpretation and reporting. A rating of 0–40% was deemed to indicate ‘poor’ quality; ratings of 40–60% were deemed ‘moderately high’ and ratings above 60% were deemed to indicate ‘high’ quality.

RESULTS

The MEDLINE search resulted in 444 citations. Three hundred and forty-nine of these were excluded immediately because they were irrelevant to our study, involved migraine headaches or involved studies of behavioural or cognitive-type treatments. Of the remaining 95 citations related to CAM, 73 were not RCTs, giving a total of 22 RCTs. The PsychInfo and CINHAL searches revealed no additional RCTs. Citation searches revealed one additional RCT.29 One RCT30 was identified in the recent literature. Five additional reports did not deal directly with clinical outcomes but were investigations of physiological measures,31–34 or, in one case, did not involve symptomatic subjects.35 They were excluded from the review.

The 24 studies included in this review were organized into groups according to the primary modality of treatment. This was straightforward for studies involving acupuncture, spinal manipulation and homeopathy. The category of ‘physiotherapy’ was less straightforward. Studies investigating electrical
therapies alone and as the primary modality were placed in the 'electrotherapy' category. Studies included in the 'physiotherapy' category, involved multiple modalities, including electrotherapy in some. Table 1 gives the breakdown of number of RCTs by treatment category. The largest group involved studies of acupuncture, with no other group having more than five distinct trials. In the manipulation group, one of the trials was reported twice, with different sample sizes. We conducted
The quality scores for each of these reports separately.

Twenty-two of the trials involved tension headache subjects. Only two reports (one trial) involved CH. Only one study involved post-traumatic headache (PTTH) subjects. Subjects in these three trials received spinal manipulation as compared to soft tissue therapy (for the CH trials) or ice therapy (in the PTTH trial).

The quality raters' scores achieved a reliability coefficient of 0.72 (P = 0.0015). Scores were not statistically significantly different from one another (\( t = -1.5, P = 0.14 \)) and the 95% CI of the mean difference between scores was 1.9–0.3. Given this level of consistency, we averaged the two raters' scores for a final trial quality score.

**Quality reviews**

**Acupuncture trials**

The quality scores for the eight acupuncture trials ranged from 44 to 69, with an average score of 58 and a median of 61. The quality scores for the four high quality trials ranged from 61 to 69%. These trials were published during 1979–1996. The total number of subjects reported in these trials is 99, with an average of about 25 subjects per trial. Three of these trials were sham-controlled, with an average treatment duration of 52.5 days. The other trial, compared acupuncture to physiotherapy. Tavola et al. reported a 'negative' outcome, in that the acupuncture treatment was no better than the placebo, while Ahonen et al. reported significant improvement in the acupuncture and physiotherapy groups, with no difference between the two groups. Two trials reported a significant difference favouring acupuncture over sham placebo with regard to the frequency of headaches, but these two trials have a combined total of 39 subjects, thus precluding any definitive conclusions.

The quality scores for the other four trials ranged from 44 to 50 (moderately high quality). They were published from 1984 to 1991. The total number of subjects in these reports was 173, with an average of 43 per trial. Only one of these studies was sham-controlled, while one used a no-treatment control. The average duration of treatment was 99 days. Three of these studies reported a positive benefit in that acupuncture was shown to be better than sham-control for frequency, better than no-treatment control and better than medication. On the other hand, Johansson et al. did not demonstrate differences between acupuncture and an occlusal splint for TMJ-related tension headache and Carlsson et al. reported that subjects receiving physiotherapy obtained greater benefit than acupuncture.

In summary, (see Tables 2 and 3) the total number of TTH subjects reported in the literature receiving acupuncture is 264. The treatment durations of these studies range from 6 to 12 weeks. Five (63%) of these studies were controlled (4/5 employed sham controls). Two of four higher quality studies reported negative results, although, with the small sample sizes in all of these trials, the likelihood of a type II error is quite high. Acupuncture has been shown in at least one study (low quality) to be more beneficial than medication over a 3 month period, and equivalent to an occlusal splint in the treatment of TMJ-related tension-type headache. Acupuncture does not appear to be more effective than a course of physiotherapy.

**Spinal manipulation trials**

Three RCTs of spinal manipulation for TTH, two for cervicogenic headache and one for 'post-traumatic headache' were identified. The quality scores ranged from 56–80%, with a mean score of 67.5%.

Table 4 reviews these trials. No trial included an exclusively sham or placebo-type control group, so that the 'efficacy' of spinal manipulation treatment cannot yet be determined. With respect to determining the effectiveness of spinal manipulation, comparative treatments include soft-tissue mobilization, resting briefly, ice pack, amitriptyline and soft tissue therapy. A total of 286 subjects were included in these reports.

There is some inconsistency with regard to the diagnostic classifications used in these studies. The report by Hoyt et al. involved a single manipulative session provided to nine subjects with a concurrent 'muscle contraction' headache (versus 13 other control subjects). Jensen et al.'s study was conducted on a small group of subjects with 'post-traumatic headache'. Boline et al.'s study included the only ones to explicitly include 'tension-type headache' according to the IHS criteria. The former study included a 6 week intervention phase and a 4 week follow-up, while the latter study involved 4 weeks of treatments with no follow-up phase. Nilsson's study was conducted on subjects with cervicogenic headache.

As no high quality studies exist which employed an exclusive placebo or sham-control group, the efficacy of SMT for TTH or CH cannot be determined. Four high quality studies do exist which compare SMT to other forms of therapy, although two of them have relatively small sample sizes. Three of these studies report a benefit of SMT.
In these studies SMT is more effective than ice pack applications and soft tissue therapy in post-traumatic and CH. SMT appears to be as effective as amitriptyline in producing short-term benefit for TTH; however, there may be a longer term benefit with SMT once the treatments are withdrawn. In one study, the addition of SMT to a group already receiving therapeutic levels of deep massage did not improve outcomes in TTH sufferers beyond the level obtained by a group receiving the massage and a placebo treatment. This study is the only one to report no additional benefit from SMT.

**Electrotherapy studies**

Four RCTs were obtained which investigated electrotherapy as the sole modality. Three studied transcutaneous electrical nerve stimulation (TENS) and one used a form of 'cranial electrotherapy'. The latter study and two of the TENS studies were placebo-controlled, while the other study compared TENS to relaxation therapy, biofeedback and a combination of all three treatments. The quality scores for these studies ranged from 39 to 61%, with an average score of 50%. Only one study achieved a rating which would qualify it as of 'high' quality.*

The studies by Reich and Solomon et al. included both tension-type and migraine sufferers, while the studies by Airaksinen and Pontinen and Solomon and Guglielmo involved only tension-type headache. Airaksinen and Pontinen investigated the short-term changes in pressure pain threshold at 'trigger points' in TTH sufferers.
<table>
<thead>
<tr>
<th>Authors</th>
<th>Headache type</th>
<th>Sample size</th>
<th>Study duration</th>
<th>Treatment groups (n)</th>
<th>Results</th>
<th>Side effects</th>
<th>Quality scores</th>
</tr>
</thead>
<tbody>
<tr>
<td>Hansen and Hansen</td>
<td>chronic tension</td>
<td>18</td>
<td>105 days (crossover)</td>
<td>(1) ACUP = 9</td>
<td>Pre-Tx F: (1) = 42.2, (2) = 40.7</td>
<td>1 subject had aggravation of pain</td>
<td>50</td>
</tr>
<tr>
<td>1985</td>
<td>headaches</td>
<td></td>
<td></td>
<td>(2) SHAM = 9</td>
<td>Post-Tx F: (6 weeks) (1) = 26,4* (3) = 35.2</td>
<td>from needing</td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td>Post-Tx F: (12 weeks) (1) = 30.1 (3) = 30.9*</td>
<td>1 = primary measure is period index</td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td>2 = groups crossed-over</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Johansson et al.,</td>
<td>muscle tension</td>
<td>45</td>
<td>120 days</td>
<td>(1) ACUP = 15</td>
<td>Not mentioned</td>
<td>50</td>
<td></td>
</tr>
<tr>
<td>1991</td>
<td>headache</td>
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<td></td>
<td>(2) SPLINT = 15</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
<td>(3) CONTR = 15</td>
<td></td>
<td></td>
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</tr>
<tr>
<td>Carlson et al.,</td>
<td>chronic tension</td>
<td>62</td>
<td>60-90 days</td>
<td>(1) ACUP = 23</td>
<td>None</td>
<td>50</td>
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<tr>
<td>1990</td>
<td>type HA</td>
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<td></td>
<td>(2) P/T = 29</td>
<td>(1)</td>
<td>(2)</td>
<td></td>
</tr>
<tr>
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<td></td>
<td></td>
<td></td>
<td>none = 3</td>
<td>(1) = 3</td>
<td>0</td>
<td>None</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
<td>mild = 3</td>
<td>3</td>
<td>3</td>
<td></td>
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<tr>
<td></td>
<td></td>
<td></td>
<td></td>
<td>mod. = 17</td>
<td>17</td>
<td>29</td>
<td></td>
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<td></td>
<td></td>
<td></td>
<td></td>
<td>sev. = 59</td>
<td>59</td>
<td>58</td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
<td>v. sev. = 17</td>
<td>17</td>
<td>10</td>
<td></td>
</tr>
<tr>
<td>Loh et al., 1984</td>
<td>'muscle tension'</td>
<td>48</td>
<td>120 days</td>
<td>(1) ACUP = 41</td>
<td>Not mentioned</td>
<td>44</td>
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<tr>
<td></td>
<td>migraine</td>
<td></td>
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<td>(2) MEDS = 36</td>
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<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>mixed</td>
<td></td>
<td></td>
<td>(1) = 7</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
<td>mild = 31</td>
<td></td>
<td></td>
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<td></td>
<td>mixed = 10</td>
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<tr>
<td>Total or average</td>
<td></td>
<td>173</td>
<td>105 days</td>
<td></td>
<td></td>
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<td></td>
</tr>
</tbody>
</table>

(n) = sample size in each treatment group; (IHS) = inclusion based on criteria of the International Headache Society Classification.

Treatment types: ACUP = acupuncture; SHAM = sham placebo treatment; SPLINT = occlusal splint; CONTR = no-treatment control; P/T = physical therapy or physiotherapy; MEDS = medication; MANIP = chiropractic spinal manipulation; STT = soft tissue therapy; AMIT = amitriptyline; RELAX = relaxation therapy; TENS = transcutaneous electrical nerve stimulation; ELEC. STIM. = electrical stimulation; LO- = low level; BIOF = biofeedback; ATTN = attention control. Outcomes T = treatment; HA = headache; S = severity; F = frequency; mm = millimetres on a visual analogue scale; severity. Global relief: mod = moderate; sev = severe; very sev = very severe frequency; sev = several, hi = high; mod = moderate; min = minimal; impv = improvement; ≥ = statistically significantly better than; = means not statistically significantly better than. NS = not significant; * = 0.05; ** = 0.01; *** = 0.001.
### Table 5: Evidence of CAM therapies for headache

<table>
<thead>
<tr>
<th>Authors</th>
<th>Headache type</th>
<th>Sample size</th>
<th>Number of TXs</th>
<th>Treatment groups (n)</th>
<th>Results</th>
<th>Side effects</th>
<th>Quality scores</th>
</tr>
</thead>
<tbody>
<tr>
<td>Hoyt et al., 1979</td>
<td>‘muscle contraction’</td>
<td>22</td>
<td>1</td>
<td>(1) MANIP = 10</td>
<td>Post-TX S: (1) -48%*** (2) 0 (3) 0</td>
<td>Not mentioned</td>
<td>56</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
<td>(2) MOB = 6</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
<td>(3) REST = 6</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Jensen et al., 1981</td>
<td>post-traumatic</td>
<td>19</td>
<td>2</td>
<td>(1) MANIP = 10</td>
<td>Post-TX S: (1) -30.7/100** (2) + 6.7/100</td>
<td>Not mentioned</td>
<td>60</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
<td>(2) ICE = 9</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Nilsson 1995</td>
<td>cervicogenic</td>
<td>39</td>
<td>6</td>
<td>(1) MANIP = 20</td>
<td>Post-TX F: (1) -3.4 (-59%) (2) -2.1 (-45%)</td>
<td>Not mentioned</td>
<td>64</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
<td>(2) STT = 19</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Nilsson 1997</td>
<td>cervicogenic</td>
<td>53</td>
<td>6</td>
<td>(1) MANIP = 28</td>
<td>Post-TX S: (1) -3.2* (-69%) (2) -1.6 (-37%)</td>
<td>Not mentioned</td>
<td>72</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
<td>(2) STT = 25</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Boline et al., 1995</td>
<td>tension-type headache (IHS)</td>
<td>126</td>
<td>12</td>
<td>(1) MANIP = 70</td>
<td>Post-TX F: (1) -3.8/28 (2) -4.0/28</td>
<td>(1) 4.3% neck stiffness</td>
<td>75</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
<td>(2) AMIT = 56</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
<td>(2) + 5.0</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Bove and Nilsson, 1998</td>
<td>tension-type headache (IHS)</td>
<td>75</td>
<td>8</td>
<td>(1) MANIP + STT = 38</td>
<td>Post-TX S: (1) -1.5 hours (2) -1.9 hours</td>
<td>Not mentioned</td>
<td>80</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
<td>(2) SHAM + STT = 37</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Total or average</td>
<td></td>
<td>286</td>
<td>6</td>
<td></td>
<td></td>
<td></td>
<td>68</td>
</tr>
</tbody>
</table>

*(n)* = sample size in each treatment group; (IHS) = inclusion based on criteria of the International Headache Society Classification. Treatment types: ACUP = acupuncture; SHAM = sham placebo treatment; SPLINT = occlusal splint; CONTR = no-treatment control; P/T = physical therapy or physiotherapy; MEDS = medication; MANIP = chiropractic spinal manipulation; STT = soft tissue therapy; AMIT = amitriptyline; RELAX = relaxation therapy; TENS = transcutaneous electrical nerve stimulation; ELEC. STIM. = electrical stimulation; LO- = Low level; BIOF = biofeedback; ATTEN. = attention control. Outcomes: Tx = treatment; HA = headache; S = severity; F = frequency; mm = millimetres on a visual analogue scale; severity: Global relief: mod = moderate; sev = severe; v. sev. = very severe Frequency: hi = high; mod = moderate; m/n = minimal; improv. = improvement; ≥ = statistically significantly better than; = means: not statistically significantly better than. NS = not significant; * = 0.05; ** = 0.01; *** = 0.001.

At least one high quality RCT and two others of moderately high quality demonstrate that electrotherapy is more efficacious than placebo in the treatment of TTH. One moderately high quality study demonstrated that TENS is at least as effective as other cognitive/behavioural therapies in (presumably as a measure of pain relief for concurrent headache), while the other three studies investigated the prophylactic benefit of a series or programme of treatments. A total of 507 tension headache subjects were included in these four studies (see Table 5).
reducing headache activity, although patient variables such as the duration of headache complaint and the number of treatments rendered have an impact on individual patient response.

Physiotherapy trials
Three RCTs were identified involving multimodality physiotherapy treatment programmes. The quality scores for these trials ranged from 33 to 58% (low-to-moderately-high quality). The study with the highest rating (Carlsson et al.52 compared physiotherapy treatment to acupuncture. In this trial, ‘physiotherapy’ consisted of a variety of patient-initiated modalities, including relaxation techniques, stretching, TENS and ice therapy, as well as education regarding muscle tension and how to control it

<table>
<thead>
<tr>
<th>Authors</th>
<th>Headache type</th>
<th>Sample size</th>
<th>Study duration</th>
<th>Treatment groups (n)</th>
<th>Results</th>
<th>Side effects</th>
<th>Quality scores</th>
</tr>
</thead>
<tbody>
<tr>
<td>Reich, 1989</td>
<td>muscle contraction headache</td>
<td>331</td>
<td>at least 15 weeks TX; 36 months follow-up</td>
<td>(1) RELAX; (2) TENS; (3) BIOF; (4) COMB</td>
<td>Post-Tx S: (At discharge) (1) -1.5/5 (2) -2.1/5 (3) -2.4/5*** (4) -2.1/5</td>
<td>Not mentioned</td>
<td>44</td>
</tr>
<tr>
<td>Solomon et al., 1989</td>
<td>tension headache</td>
<td>100</td>
<td>6-10 weeks</td>
<td>(1) CRANIAL ELEC. STIM = 50       (2) SHAM = 50</td>
<td>Post-Tx S: (At discharge) (1) -2.1 (35%)* (2) -1.2 (18%) Global relief (%) (1) (2) hi 124 mod 2412 min 2620 none 3863 **</td>
<td>61</td>
<td></td>
</tr>
<tr>
<td>Airaksinen and Pontinen, 1992</td>
<td>chronic tension headache 14</td>
<td>1 week, 2 sessions</td>
<td>(self-control)</td>
<td>(1) ELEC. STIM. = 14 (2) SHAM = 14</td>
<td>Pre-Post Pressure Thresholds: (hp) (1) pre = 2.83 (0.16) post = 3.46 (0.21) (2) pre = 3.34 (0.2) post = 3.48 (2.1) Percent showing clinically significant improvement (Muscle contraction headache) (1) = 55% (2) &amp; (3) = 10%</td>
<td>39</td>
<td></td>
</tr>
<tr>
<td>Solomon and Guglielmo, 1985</td>
<td>migraine</td>
<td>62</td>
<td>one treatment</td>
<td>(1) ACTIVE TENS = 18 (2) LO- TENS = 18 (3) SHAM = 22</td>
<td>Not mentioned</td>
<td>56</td>
<td></td>
</tr>
</tbody>
</table>

Total or Average 507 50

(n) = sample size in each treatment group; (IHS) = inclusion based on criteria of the International Headache Society Classification; (S) Treatment types: ACUP = acupuncture; SHAM = sham placebo treatment; SPLINT = occlusal splint; CONTR = no-treatment control; P/T = physical therapy or physiotherapy; MEDS = medication; MANIP = chiropractic spinal manipulation; STT = soft tissue therapy; AMIT = amitriptyline; RELAX = relaxation therapy; TENS = transcutaneous electrical nerve stimulation; ELEC. STIM. = electrical stimulation; LO- = low level; BIOF = biofeedback; ATTEN. = attention control. Outcomes Tx = treatment; HA = headache; S = severity; F = frequency; mm = millimetres on a visual analogue scale; severity: Global relief: mod = moderate; sev = severe. v. sev. = very severe Frequency: low, sev = several; hi = high; mod = moderate; min = minimal; improv. = improvement; ≥ = statistically significantly better than; Φ = means: not statistically significantly better than. NS = not significant; * = 0.05; ** = 0.01; *** = 0.001.
'autogenically'. Both treatments produced positive benefit in mood state and overall health function as well as in the intensity and frequency of headaches. Physiotherapy produced greater gains in mood state and in reduced headache intensity.

In both other studies, the physiotherapy modalities employed included TENS, heat, massage and ultrasound therapy to the painful areas, trigger point therapy, exercise therapies, biofeedback and education. In Jay et al.'s study, all subjects received amitriptyline medication. They reported that subjects receiving the additional physiotherapy treatments fared better than those receiving only the medication. Only the study by Marcus et al. employed a control procedure consisting of education and ‘skin-cooling’ biofeedback. They reported that the combined physiotherapy group ‘was more likely to experience significant headache relief’ than the attention control group (72.7 vs 28.6%, P<0.03).

In all three studies (see Table 6) various combinations of these ‘physiotherapeutic’ and ‘cognitive/behavioural’ therapies (as well as medications, in one study) were employed, making the determination of the effect of each of these components impossible. A total of 147 subjects were included in these three studies (two additional reports by Carlsson et al. on the same group of subjects and were excluded from this review).

The evidence from the three studies on TENS adds to the evidence of the studies reviewed above under ‘electrotherapy’. There are no high quality studies to support the efficacy of any other form of ‘physiotherapy’ in the treatment of TTH.
some lower quality evidence supporting the effectiveness of combined physiotherapy regimens in treating TTH.

Massage trials

No RCT was found on the effects of manual massage as the primary therapy for non-migrainous headache. The study by Bove and Nilsson\(^5\) employed deep muscular massage to the trapezius and sub-occipital region as a control treatment. Subjects in both groups received this therapy, while they were randomly allocated to additionally receive spinal manipulation or sham treatment. As such, no randomized comparison of massage alone versus another treatment has been reported.

Homeopathy trials

Only one RCT was identified for homeopathic treatments of TTH. Walach et al.\(^5\) reported on 98 subjects, about half of whom had chronic tension-type headaches and were randomly allocated to receive either an individualized homeopathic remedy or an inert, indistinguishable placebo for 12 weeks. This trial achieved a quality score of 86%, which was the highest in our series, chiefly as a result of the high methodologic rigour which included an appropriate sample size and double-blinded, placebo controls. This trial reported no difference between the two groups on any important clinical variables related to headache activity.

Other remedies

One clinical trial was retrieved which investigated the use of an analgesic/counter-irritant ointment (‘Tiger Balm’) in the treatment of tension headache.\(^5\) This study achieved a high quality rating of 72%. Fifty-seven tension headache subjects were randomly allocated to receive Tiger Balm, topical placebo or paracetamol (1000 mg dose) as a treatment for a concurrent headache. Both Tiger Balm and paracetamol produced greater pain relief than placebo in a single headache episode (\(P < 0.05\)) for up to 3 hours, with no difference between these two.

One study was found on the effects of ‘therapeutic touch’ on TTH\(^5\) which achieved a quality score of 47%. The therapeutic benefit is purported to derive from the ‘therapeutic intent’ of the therapist. No manual contact is applied in this therapy. This trial involved the application of either true or ‘placebo’ therapeutic touch to 60 randomly allocated tension headache subjects who were experiencing a headache concurrently. Subjects in the ‘experimental group’ obtained twice as much pain relief as those in the control group immediately and 4 hours after the 5 minute intervention.

Methodological aspects of the reviewed studies

Table 7 presents the results of the quality ratings per item of the rating checklist, based upon agreement between raters for ‘no’ or ‘don’t know’. Those items scoring higher than 30% represent critical deficiencies in this body of studies, most of which relate to internal validity.

DISCUSSION

CAM therapies for non-migrainous headache appear to operate within several intersecting theoretical models. The more general of these involves the amelioration of pain states by activation of putative endogenous anti-nociceptive processes.\(^5\)-\(^8\) The mechanism by which these therapies may work could be described as ‘systemic’, and could include acupuncture and homeopathy, as well as some of the relaxation techniques employed within ‘physiotherapy’. These latter therapies are consistent with cognitive and behavioural therapies which have demonstrated effectiveness.\(^8\)-\(^12\)

A second mechanism appears to involve treatments targeted at the cervical spine or cranial muscles as putative sources of headache pain. The notion that headache pain may arise from the cervical spine is generally well accepted today, based upon the work of Kerr,\(^13\) Sjaastad et al.\(^10\)-\(^12\), and Bogduk et al.\(^6\)-\(^5\) This work has contributed to the acceptance of a category of headache known as ‘cervicogenic’.\(^9\) The degree to which problems in the cervical spine may contribute to tension-type headache is still unresolved, from both theoretical and nosological perspectives. Cervical musculo-ligamentous dysfunction has been demonstrated in tension-type headache sufferers.\(^6\) Despite the controversy, spinal manipulation, mobilization, massage, electrotherapy and other ‘physiotherapeutic’
procedures such as exercise and postural education, target the soft tissues of cervical spine and cranio-cervical junction which may be producing referred head pain.

The other regional mechanism involves therapies directed to the cranial area, including electrotherapy to cranial skin and muscles as well as topical creams applied to the cranial skin, the purpose of which is to reduce local pain and muscle spasm.

These latter two mechanisms may be described as 'local' and appear to involve either the amelioration of possible referred cranial pain from cervical sources or the reduction of local cranial pain by counterirritation. In addition, these therapies might theoretically exert a relaxant effect on local musculature.

The findings of our review demonstrate that RCTs of CAM therapies for non-migrainous headache do exist, and that some of them have been conducted and reported at a sufficiently high level of rigour. There are some who claim that it is not possible to investigate the benefit of CAM therapies with RCTs, in that, in requiring an appropriate level of standardization and methodological rigour, compromises the treatment context which may invalidate the results obtained are created.20 While this may be true to some extent, it would appear that this is not an absolute circumstance. In fact, several of the trials have successfully incorporated sham/placebo treatments in order to investigate the efficacy of the primary treatment.

It has also been shown that investigators in these areas can develop well-designed, high-quality studies and recruit appropriately large samples of subjects interested in participating. As this development evolves, the database of outcomes for at least some of these treatment approaches should become large enough to conduct meta-analyses so that more robust evidence-based decisions can be made by practitioners.

It is noteworthy that one therapy, electrotherapy to cranial muscles, would appear to have sufficient strength of evidence to support its use in treating TTH. Additionally, for another therapy, homeopathy, there is at least one high-quality trial whose results might recommend against its use in TTH. For the other therapeutic modalities, the evidence base either contains too few trials or contains trials resulting in contradictory findings which preclude any definitive summary.

The methodological deficiencies cited in Table 7 indicate the areas where future clinical trials should be improved. Careful selection of headache subjects according to explicit inclusion and exclusion criteria following the IHS classification guidelines9 should be employed. Provider and subject blinding may be difficult to achieve in studies of some CAM treatments, but every effort should be made to blind the treatment allocation from all parties not directly involved in the treatment, particularly the assessors.

The issue of long-term follow-up must be dealt with in future trials in order to establish the true value of these treatments to society at large and their impact on the health-care system.

CONCLUSION

We have reported on 24 published RCTs of acupuncture, spinal manipulative therapy, electrotherapy, physiotherapy, massage, homeopathy and other therapies for non-migrainous headache.

Pooling of trial data would be the most desirable representation of the evidence; however, the small number of trials in each category, as well as the variability in outcome measures in the trials, precluded this type of analysis at present.

Quality issues that require attention in further trials include: similarly of groups at baseline, description of co-interventions, compliance monitoring, subject blinding (where possible), monitoring of adverse effects, describing drop-outs, long-term follow-up and intention-to-treat analysis.

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REFERENCES


FACIAL, HEAD AND NECK PAIN are very common complaints in Western society and are highly prevalent in chiropractic practice. The physiologic basis of the association between neck pain and headaches (if not also some forms of facial and/or jaw pain) lies in the fact that the nociceptive afferents from upper cervical and posterior cranial structures terminate in the same second-order neuron pool, termed the "medullary dorsal horn," by Gobel et al. and the "trigemino-cervical nucleus" by Bogduk. A series of experiments beginning in the mid-1980s by Sessle and his colleagues has investigated these interactions in an exploration of craniofacial pain mechanisms. This article first presents a review of current pain physiology as a background to a serial review of the relevant experiments by Sessle and his colleagues. These studies are reviewed qualitatively, in order to inform the reader about their breadth and scope. Our work has confirmed that complex patterns of sensorimotor processing underly the phenomena associated with craniofacial pain, and that various named clinical syndromes such as "upper cervical dysfunction," "greater occipital neuralgia," "cervicogenic headache," "neck-tongue syndrome," and some forms of temporomandibular joint pain may all share at least some aspects of this common mechanism. The clinical manifestations of disturbances in this mechanism are likely to include referred pain and muscular hypertonicity which are so common in myofascial pain syndromes in the craniofacial region. The astute clinician is then presented with much greater diagnostic complexity, but is also given expanded opportunities for therapeutic intervention in the challenging area of craniofacial pain. (JNMS: Journal of the Neuromusculoskeletal System 7:51-64, 1999)

Key words: Chiropractic, Neurophysiology, Pain mechanisms, Upper cervical

Facial, head, and neck pain are very common complaints in Western society and are highly prevalent in chiropractic practice (1). Estimates for the annual prevalence rate of neck pain average approximately 30% (2,3), while headache prevalence rates are 45%-70% for tension type (4-6) and 15%-20% for migraine (5,6). There is considerable overlap between neck pain and headache complaints, with 70%-80% of whiplash sufferers also experiencing headache (7,8). In two studies of a disability rating index for neck pain complaints (the Neck Disability Index), headache was the highest rated item (9,10).

The physiologic basis of this association between neck pain and headaches (if not also with some forms of facial and/or jaw pain) lies in the fact that the nociceptive afferents from upper cervical and posterior cranial structures (both intra and extra-cranial) terminate in the same second-order neuron pool. This consists of an uninterrupted column of cells in the brainstem and upper cervical cord, including the substantia gelatinosa and marginal laminae. First mention of this connection appears to have been made by Skillel in 1949 (11) in his description of the "greater occipital nerve syndrome." However, it was the work of F. Kerr a few years later (12) which clearly identified the phenomenon of referred head pain from stimulation of the upper cervical sensory roots during posterior cranial surgery. In fact, in some circles, this convergence of trigeminal and upper cervical afferents is referred to as the "Kerr" phenomenon. Gobel (13) has termed this nucleus the "medullary dorsal horn," while Bogduk et al. referred to this as the "trigemino-cervical nucleus" (14).

Put in colloquial terms, the pain center of the head (spinal nucleus of the trigeminal nerve) is continuous with the pain center of the upper cervical spine, and there is considerable convergence between inputs from the head and neck within this system (see below). Figure 1 shows the spinal tract of the trigeminal nerve (V) descending in the brainstem, containing three parts: pars oralis, pars interpolaris, and pars caudalis. The pars or subnucleus caudalis has a laminar structure and has been identified as descending to the level of C3 (14). These trigeminal afferent fibers synapse on the same second-order neurons as do the afferents from levels C1 to C3, a process known as afferent conver-
FIGURE 1. Trigemino-cervical nucleus: Afferents from subnucleus caudalis of V terminate on the same second-order neurons as do the afferents from C1–C3.

The clinical implications of this convergence phenomenon are discussed later in this review.

For a number of years, work has been pursued in the laboratory of Dr. Barry Sessle (Faculty of Dentistry, Oral Physiology Laboratory, University of Toronto) on the brainstem mechanisms of craniofacial/cervical pain. This article reviews this work which extends from the early 1980s to the present. While the experimental data have already been reported, speculation on the clinical implications of these findings has, necessarily, been limited. This review expands upon these potential implications. First, it is necessary to review some principles of basic pain neuroscience, especially for the novice reader.

CURRENT CONCEPTS IN PAIN NEUROSCIENCE

The reader is referred to specific references from which much of the following is taken (15–19). Pain is mediated by small myelinated (A-delta) and unmyelinated (C) fibers which, collectively, comprise nociceptive nerve fibers. Spe-
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cialized craniofacial structures such as teeth, cornea, and meninges are innervated solely by these small-diameter fibers. While skin is also innervated by both the large- (A-beta) and small diameter fibers, nociceptive sensation from the deep structures, such as muscles and joints, is mediated by group III (A-delta) and group IV (C-fiber) afferents. These small-diameter fibers terminate mainly in laminae I and II of the spinal cord and in the subnucleus caudalis of the trigeminal nucleus.

Three types of dorsal horn neurons (DHN) have been identified, two of which are normally involved in pain transmission, classified as nociceptive neurons. Non-nociceptive inputs activate low-threshold mechanoreceptive cells (LTMs) which are responsive to low-intensity afferent signaling arising from mechanoreceptors in the peripheral tissues. These signals are conveyed along large A-beta fibers. LTM cells signal non-noxious modalities such as touch, pressure, kinesthesia, and vibration. They are primarily located in laminae III and IV of the dorsal horn (DH) and lower medulla (see Fig. 2).

Two types of DHNs are involved in signaling pain, one exclusively—the nociceptor-specific cell (NS)—and one in a mixed spectrum of painful and nonpainful stimuli—the wide dynamic range cell (WDR). Under normal conditions, NS cells are activated only in response to high-intensity noxious stimuli (nociception). They are mainly located in the “marginal zone” (lamina I) of the dorsal horn. WDR cells are concentrated in lamina V and have several unique characteristics, namely: 1) they respond to both noxious and non-noxious stimuli; 2) they have the ability to code stimulus intensity, i.e., they manifest a low rate of response to light stimuli (nonpainful), whereas a high rate of response is manifested to intense (painful) stimuli; and 3) they receive inputs from a variety of tissue sources, including skin, viscera, muscles, and joints (hence the term “convergent neuron”) (see Fig. 2).

It is now known that small-diameter nociceptive afferents contain both glutamate/aspartate as well as neuropeptides such as substance P and CGRP (calcitonin gene-related peptide). Glutamate appears to be the major transmitter involved in the small-diameter pain fibers. An initial painful stimulus may provoke only glutamate release. Glutamate activates both NMDA (N-methyl-D-aspartic acid) and non-NMDA receptors in the dorsal horn and caudalis. The NMDA receptor is a specialized feature of the nociceptive neurons (NS and WDR) in the DH. Slow wave “afferent depolarization” along with intracellular second messengers act on the NMDA receptor causing a magnesium ion to be dislodged, thus allowing an influx of calcium. This increase in intracellular Ca^{2+} may activate several signal transduction systems (so-called “second messengers”), including phosphokinase C (PKC), as well as the facilitation of nitric oxide (NO) production. These events may alter DH neuronal properties profoundly, including a feed-forward facilitation by NO on the presynaptic ending, resulting in the release of additional glutamate as well as substance P, SP, which is known to act on the neurokinin 1 receptor, may produce prolonged postsynaptic membrane depolarization. Increased intracellular Ca^{2+} also produces more postsynaptic ionic gate opening, thus increasing the depolarization. However, noxious stimulation produces these changes only under certain physiological conditions. One of these conditions may be inflammation, particularly the peripheral effect of inflammatory mediators such as PGE2, bradykinin, and substance P in inducing nociceptive afferent sensitiza-
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Peripheral sensitization. This peripheral sensitization is further augmented by a process called neurogenic inflammation. The inflammatory mediators activate nociceptive afferents and initiate neural spikes which, while traveling centrally, activate axonal collateral branches which activate more terminals to release additional inflammatory mediators. In this way, inflammation can provide the central nervous system (CNS) with a constant "afferent barrage," producing intense pain sensations and inducing the cascade of events which leads to central sensitization.

An injury or a potentially injurious stimulus may provoke a withdrawal response which is a normal defensive response. However, inflammation and repeated strong stimuli may trigger the "central sensitization" process. Central sensitization involves neuroplastic changes in the second-order neurons (in dorsal horn or subnucleus caudalis) as well as potential neural reorganization in supraspinal structures, the latter being poorly understood at present. This longer term neuroplastic change may also result in "central modulatory" changes, which are seen in some neuropathic pain conditions and which can become refractory to opiate treatment.

A second important central phenomenon associated with inflammation results from a high rate of repeated noxious stimulation, and is known as "wind-up." Strong stimuli occurring at a rate of at least once every 2 seconds may cause the DHN membrane to gradually shift toward depolarization, leading it to become hyperactive. On the other hand, if the rate of electrical impulses is slower than 1/3 second, no such temporal summation will occur. Wind-up and inflammation-induced stimuli are associated with the central neuronal neuroplastic changes described above. These include increased intracellular Ca²⁺ concentration, membrane hyperdepolarization, second-messenger activation, and release of neuropeptides. Central sensitization induces transient or "long-term" changes in neuronal properties (neuroplasticity) and eventually results in a hyperalgesic state (see Fig. 4).

The changes in response characteristics manifested by central (spinal DH and trigeminal subnucleus caudalis) neurons include: 1) enlargement of the receptive fields (RF) of neurons (i.e., they respond to stimuli that are normally outside their range); 2) reduction of nociceptive thresholds; 3) increase in response magnitude, and 4) increase in spontaneous activity. The first three changes are related to hyperalgesia. In extreme cases, some NS neurons may possess a novel receptive field responsive even to touch or other low-threshold stimuli. These stimuli may then activate the NS neuron, rendering them "WDR-like." This touch-evoked pain, or "allodynia," is a feature in such chronic pain conditions as trigeminal neuralgia or causalgia (reflex sympathetic dystrophy) (see below). This phenomenon may occur when inflammation activates these so-called "silent nociceptors." Once their activation threshold has been reduced and spontaneous activity is more likely, these silent nociceptors become active nociceptors, and normal, nonpainful movements may trigger pain sensations. This becomes a further source of noxious barrage to the central neurons, further increasing their excitability as well as neuroplasticity.

When nerve injury is also involved, these neuroplastic changes may produce a long-term pain state. The critical element in preventing the development of these changes is early treatment. In addition to their role in sensory functions, the NMDA receptor mechanism is also known to be involved in memory and learning function in the central nervous system. If these nociceptor-induced pathophysiological changes are permitted to develop fully, then neuroplastic changes may become permanent and refractory to treatment. The short-term pathophysiology converts to a longer term pathophysiology with "memory" and permanent structural change.

Numerous experimental models have been developed to investigate one or more of these "neuroplastic" elements. These models include the assessing of behavioral responses in awake animals, assessments of more focal physiologic responses in experimental preparations, and highly refined studies of the molecular responses within the DHNs.

Before the mid-1980s, most pain researchers employed cutaneous nociceptive models because of the ease of delivery of the noxious stimulus to the skin. However, seminal work by British (20,21) and German (22,23) researchers opened the door to the study of deep pain mechanisms through the use of chemical noxious stimuli. For example, Schmidt and his colleagues (23), in studying the discharge from single group III and IV afferents from the cat knee joint, developed the concept of the "silent nociceptor" described above.
In the mid-1980s, models were developed which extended nociceptive studies to the deeper tissues and for longer periods of time (tonic pain). The formalin model (24) involved injection of formalin subcutaneously into the paw pad of an awake, unrestrained animal. Withdrawals of the paw for licking or biting were measured. Longer lasting and more complex pain behavioral mechanisms could be studied in this model. It was determined that a standard formalin exposure provoked a triphasic response consisting of an early reactive phase, a phase of diminished activity, followed by a phase of reactivation of withdrawal responses. This third phase was found to involve C-fiber firing, to be attenuated by nonsteroidal anti-inflammatory drugs (NSAIDs), and to represent manifestations of “central sensitization.”

Another model for studying deep tissue pain is the Freund’s adjuvant model which produces a full-blown inflammatory reaction in the hindquarters and limbs of the animal, thereby providing a model for rheumatoid arthritis. Carrageenan, kaolin, and bradykinin (see refs. 15 and 18 for reviews).

The chronic sciatic constrictive model of neuropathic pain was promulgated by Bennett and Xie (25) in 1988, although Triano and Luftges (26) had reported this model in 1980 and Christiansen and Meyer (27) had elaborated upon it in 1987. Bennett and Xie’s work revolutionized the study of neuropathic pain and opened up a whole new area of investigation, which has since expanded to include multiple neuropathic models, particularly involving nerve root and dorsal root ganglion irritation models. These models distinguished neuropathic from peripheral mechanisms of pain, the latter including inflammation and other experimental irritations of the peripheral tissues.

In the early 1980s, Woolf and Wall (20) began to connect the neuroplasticity observed in deafferentation processes with mechanisms related to pain. First, they observed that the nociceptive neuronal receptive field (RF) could be expanded to incorporate a nearby fresh “wound” created experimentally (28). Furthermore, they created a rat decorrelated flexion reflex model with which they could test their hypotheses concerning neuroplastic changes associated with deep versus cutaneous stimulation (20). They found that the facilitation of the flexion reflex (a nociceptive reflex) produced by stimulation of a deep somatic nerve (gastrocnemius nerve) was more prolonged (>2 hours) than for stimulation of a cutaneous nerve in the same body segment (sural nerve). They attributed these reflex changes to spinal DH neuronal property changes (i.e., to central sensitization).

Subsequently, they employed mustard oil (MO), a C-fiber irritant, to induce deep tissue pain. They found that such application in the hind limb could facilitate the flexion reflex. When lidocaine (a peripheral anesthetic) was applied 5 minutes before MO application, the facilitation of the flexion reflex was blocked, whereas lidocaine application 5 minutes after MO application did not block the MO-induced facilitation of the flexion reflex. These studies reinforced the importance of central neurons in the facilitatory process, leading to the development of the concept of “central sensitization” and, furthermore, to the notion that deep tissue sources of painful input may have a larger “sensitizing” effect than cutaneous sources.

Sophisticated electrophysiologic recording procedures have made it possible to study single nerve fibers in the periphery and single cells (units) in the dorsal horn. Single unit recordings permit the identification of a number of important properties of the sensory neurons in the dorsal horn. Since virtually all cells in laminae I–VI receive cutaneous inputs, the cutaneous receptive field (RF) of the neuron can be mapped using both non-noxious and noxious stimuli to classify them as LTM, NS, or WDR types. Stimulation of relevant deeper structures such as subjacent muscles, joints, and blood vessels as well as stimulation of the nerves which supply these structures permits determination of whether a WDR neuron has superficial and/or deep RFs. Evaluating the baseline size and location of the RF (including where RFs do not exist under normal, resting circumstances) is the first step in such single unit recording experiments.

The next phase of these types of experiments involves some intervention such as: 1) the injection of an algogenic substance into some target tissue (substances such as mustard oil, carrageenan, capsaicin, hypertonic saline, bradykinin, etc.); 2) a control injection (mineral oil or vehicle); or 3) electrical stimulation, particularly of nerve structures. The findings of numerous studies such as these (reviewed in refs. 15–19), which are consistent with the model of central sensitization, typically involve manifestations of increased excitability or, to use Dubner and Ruda’s phrase (29), “activity-dependent changes” in DHNs.

Recently, investigators have taken advantage of another manifestation of central neuroplasticity, namely the mechanism of transduction of proto-oncogenes such as c-Fos. These proto-oncogenes are activated early in the process of activation of other genes within a cell and may serve as a marker for long-term changes in neuronal function. Various studies have shown that c-Fos is activated by experimental noxious stimulation and is then localized in the CNS by immunohistochemical methods. These studies have provided opportunities to localize segments and specific spinal cord regions where nociceptive transmission likely takes place. The localization of c-Fos-like immunoreactivity (FLI) in laminae I and V/VI in many of these studies provides further evidence that these areas are involved in pain transmission, for example, after MO is injected into the...
temporomandibular joint (30). A number of studies have reported bilateral distributions of FLI, most often in supraspinal structures, indicating that the neural connectivity associated with nociception is quite complex and likely involves multiple pain processing areas (31).

**REVIEW OF EXPERIMENTAL EVIDENCE**

With the above general review as a background, a series of studies examining brainstem/spinal cord mechanisms of head/face/neck pain can now be presented. The first group of studies (1981-1992) represent earlier work from the University of Toronto, Faculty of Dentistry, Oral Physiology Laboratory of Dr. Barry Sessle and his co-investigator Dr. James Hu. These studies were mainly concerned with mechanisms subserving facial, oral, and dental pain, with some emphasis on deep somatic pain from the tongue and temporomandibular joint (TMJ). The later work includes studies of deep somatic pain from TMJ and upper cervical spinal tissues. The methodologies of these studies, while briefly and generically reviewed above, are presented in the published works, and the reader is referred directly to these publications for much greater detail. Also, all of these studies were conducted with the full approval of the University of Toronto Animal Care Committee and conformed to the guidelines for pain studies in animals from the International Association for the Study of Pain (32) and the Ontario Act for Animal Research.

In 1981, Hu et al. (33) reported on the functional properties of feline trigeminal subnucleus caudalis neurones in response to both noxious and non-noxious stimuli in the orofacial region. Populations of LTM, NS, and WDR neurones were located. WDR/NS neurones (as well as some LTM) responded to stimulation of the hypoglossal and superior lingual nerve (all neurons had orofacial-cutaneous RFs). One third of all caudalis neurones recorded also responded to noxious tooth pulp stimulation. A minority of these caudalis neurones (from 12% to 15%) were found to project to the thalamus, while 33%-50% projected to the aqueductal grey matter (PAG), two nuclei known to be involved in antinociceptive mechanisms at the spinal cord level, produced brief periods of inhibition of caudalis neuronal firing. Additionally, peripheral conditioning stimuli in the orofacial region (and even in the forelimb) induced similar inhibitory effects.

Subsequently, Sessle et al. (34) demonstrated that stimulation of the nucleus raphe magnus (NRM) and the periaqueductal grey matter (PAG), two nuclei known to be involved in antinociceptive mechanisms at the spinal cord level, produced brief periods of inhibition of caudalis neuronal firing. Additionally, peripheral conditioning stimuli in the orofacial region (and even in the forelimb) induced similar inhibitory effects.

Sessle et al. (35) investigated the wide range of tissue sources providing inputs into the feline subnucleus caudalis itself. For LTM, WDR, and NS neurones respectively, the percentage of neurones which responded to electrical stimulation is given in Table 1. These data reinforce the wide range of inputs into these multiconvergent cells including skin, deep somatic, and visceral sources. The high prevalence of C3 input onto WDR cells confirms its importance in potentially generating referred head or neck pain. Amano et al. (36) then highlighted the contribution of muscle afferents from the jaw and tongue in the feline model. Muscle afferents stimulated by chemical or electrical means activated predominantly WDR and NS caudalis neurones. The majority of these afferents were found to be nociceptive by virtue of their long latency/high threshold responses and the predominance of small fibers in the muscle nerves tested. Many of these neurones also respond to tooth pulp stimulation. This study provided evidence of functional organization in the subnucleus caudalis of inputs from both cutaneous and deep (muscular) structures and illustrated central convergence of these afferents which might provide a basis for referred craniofacial pain.

Broton et al. (37) extended this work to include articular afferents from the TMJ in the cat. Again, WDR and NS neurones were especially responsive to algesic and electrical stimulation of the TMJ capsule. These afferents were likely nociceptive for the same reasons as described above for Amano et al. (36). A picture of multiconvergence within the subnucleus caudalis was now emerging involving the organization of cutaneous oro-facial inputs with those from tooth pulp, facial/jaw muscles, tongue, and the TMJ.

Hu (38) described similar features in the rat subnucleus caudalis. Overlap of cutaneous oro-facial and deep (hypoglossal nerve) input was a feature of the WDR and NS populations. Hu demonstrated that the evoked responses in the rat subnucleus caudalis could be attenuated by tail axon forepaw noxious stimulation, thereby demonstrating that these neurones were subject to diffuse noxious inhibitory controls (DNIC) (39), further evidence of their role in craniofacial nociceptive processing.

**TABLE 1. Percentage of Neurons Responding to Stimulation**

<table>
<thead>
<tr>
<th>Tissue</th>
<th>LTM</th>
<th>WDR</th>
<th>NS</th>
</tr>
</thead>
<tbody>
<tr>
<td>Skin</td>
<td>100% (209)</td>
<td>100% (19)</td>
<td>100% (31)</td>
</tr>
<tr>
<td>Superior lingual nerve</td>
<td>28 (160)</td>
<td>68 (19)</td>
<td>56 (34)</td>
</tr>
<tr>
<td>Tooth pulp</td>
<td>37 (173)</td>
<td>84 (19)</td>
<td>50 (36)</td>
</tr>
<tr>
<td>C2/C3 mixed nerve</td>
<td>17 (152)</td>
<td>61 (18)</td>
<td>46 (33)</td>
</tr>
<tr>
<td>C3 muscle nerve</td>
<td>25 (32)</td>
<td>100 (3)</td>
<td>29 (7)</td>
</tr>
<tr>
<td>Hypoglossal nerve</td>
<td>21 (166)</td>
<td>58 (19)</td>
<td>52 (33)</td>
</tr>
<tr>
<td>Temporalis or masseter muscle</td>
<td>7 (123)</td>
<td>25 (12)</td>
<td>21 (28)</td>
</tr>
</tbody>
</table>

Numbers tested are parentheses. LTM, low-threshold mechanoreceptive cell; WDR, wide dynamic range cell; NS nociceptor-specific cell.
This investigation was followed by a study of changes in the response properties of single caudalis neurons after algesic stimulation (via 5% mustard oil) of the masseter muscle (40). Algesic (vs. control) stimulation resulted in long-lasting (20–30 minutes) but reversible expansion of the cutaneous receptive fields, increase in spontaneous firing, and decrease in the threshold to firing.

In unpublished work following these studies, Yu, Hu, Sessle, and Vernon identified input characteristics of the entire medullary dorsal horn (i.e., from caudalis subnucleus to the C2 level in the rat). Our results included the finding that noxious-responsive neurons were organized in a medial to lateral direction as one moved caudally within the nucleus. At 2 mm caudal to the obex (caudalis), neurons received only V input, while at 3 mm (lower caudalis), 8% of neurons recorded from received C2 input. Table 2 displays data on the proportions of caudalis/upper cervical neurons from 4 mm below the obex receiving convergent input from various levels of tissue sources.

As such, no cervical inputs were found in the midcaudalis and a minor amount of C2 input was found in the lower caudalis. C2 and V inputs were found liberally, but exclusively at the C1 level, while, at the C2 level, this continued but also included minor lower cervical and limb inputs. Below C2, C2 inputs were still found. V inputs were decreasing, while cervical and limb inputs predominated. These data verify the C1–C2 spinal cord region as one which is somatotopically organized and with extensive C2 and trigeminal convergence, with a significant proportion of these neurons responding to both C2 and V input. We also found that no cervical units responded to hypoglossal nerve stimulation, while many V units did.

Yu et al. (41) explored the differential effects of cutaneous versus deep (tongue muscle) sources of input on neuroplasticity of caudalis neurons. They found that mustard oil injection in the tongue induced expansion of both cutaneous and deep receptive fields, while cutaneous (facial) injection induced expansion of cutaneous RFs only. They concluded that "deep inputs are especially effective in inducing neuroplastic changes" (41) (p. 1704). This would appear to be due to the finding that deep inputs are effective in inducing expansion of both deep and cutaneous RFs, whereas cutaneous inputs result in RF expansion in cutaneous RFs only. This may explain the greater sensory disturbances in pain conditions involving deep tissues as compared to those involving only the skin.

In 1993, Hu et al. (42) published a study which has significant implications for chiropractors and others interested in mechanical spinal pain. Notwithstanding the obvious interest of Sessle's group in craniofacial/TMJ pain, almost all of the studies on deep pain mechanisms by other investigators in the field had involved the tissues of the peripheral limbs (22,23). Their work was crucial in elucidating many of the neurophysiologic mechanisms described above. Remarkably, however, virtually none of this work involved the deep tissues of the spine; this is in spite of the very high prevalence of mechanical spinal pain syndromes (43). Gillette and his colleagues (44,45) had begun to publish studies on deep pain of lumbar spine origin, but no animal studies of deep pain mechanisms related to the cervical spine had been conducted. This includes the upper cervical region, the afferents from which, as described above, are known to connect with the trigemino-cervical nucleus. As such, Hu et al.'s report on the response of neck and jaw muscles in rats to mustard oil injection in the deep upper cervical paraspinal muscles represented a significant and unique step forward in our understanding of spinal pain mechanisms. This was of particular relevance to mechanisms of cervicocranial pain.

In this model, EMG recordings were made of two jaw muscles (digastric and masseter) and three neck/spinal muscles (the trapezius and bilateral rectus capitus posticus major, RCPMs). Injections were made percutaneously into the left deep paraspinal region around the C1–C2 level, typically in the deep, periarticular fibers of the left rectus capitus posticus major and multifidus muscles. The inflation-induced by this injection was confirmed subsequently, both morphologically and histologically.

After first conducting paired trials of mineral oil (control) injection to confirm that serial injections did not volumetrically provoke any EMG increases, a series of 19 preparations were studied whereby a 20 minute baseline period was followed by a control injection of mineral oil. EMG recordings were made for a further 10 minutes, followed by an injection of either mineral oil or mustard oil, with 30–40 minutes of further EMG recordings. The sites of injection and the magnitudes of EMG recordings for nine rats are shown in Figure 5, while the aggregate data for all 19 rats are shown in time series in Figure 6. Several important points bear mentioning with regard to these results:

1. All muscles recorded an early increase of EMG activity which was statistically significant compared to controls (black vs. open circles in Fig. 6).
2. Irritation of a deep paraspinal source evoked very large increases of EMG activity in a superficial

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**TABLE 2. Percentage of Neurons Receiving Somatic Input from C2 Only, Trigeminal (V) Input Only, Combined C2/V, Cervical, Cervical and Limb, and Limb Only Sources**

<table>
<thead>
<tr>
<th>Level</th>
<th>C2 only</th>
<th>V only</th>
<th>C and V</th>
<th>Cervical</th>
<th>C and L</th>
<th>L only</th>
</tr>
</thead>
<tbody>
<tr>
<td>C1 (4 mm)</td>
<td>29%</td>
<td>57%</td>
<td>29%</td>
<td>14%</td>
<td>0%</td>
<td>0%</td>
</tr>
<tr>
<td>C1 (5 mm)</td>
<td>45</td>
<td>41</td>
<td>35</td>
<td>21</td>
<td>0</td>
<td>0</td>
</tr>
<tr>
<td>C2 (6 mm)</td>
<td>41</td>
<td>23</td>
<td>41</td>
<td>18</td>
<td>9</td>
<td>9</td>
</tr>
<tr>
<td>C2 (8 mm)</td>
<td>53</td>
<td>0</td>
<td>12</td>
<td>50</td>
<td>19</td>
<td>19</td>
</tr>
<tr>
<td>C2 (9 mm)</td>
<td>19</td>
<td>0</td>
<td>0</td>
<td>35</td>
<td>41</td>
<td>24</td>
</tr>
</tbody>
</table>

*JNMS: Journal of the Neuromusculoskeletal System, Vol. 7, No. 2, Summer 1999*
muscle, the trapezius, which never received any injectate.

3. Activation of the contralateral RCPMa occurred in every case, although to a lesser degree than on the ipsilateral side. While spread of the injectate to the opposite side may have occurred, Figure 5 shows that this was the exception.

4. Irritation in a paraspinal site provoked activation of ipsilateral jaw muscles, although to a lesser degree. The duration of jaw muscle activation was well beyond the time frame for the clench reflex typically observed in any pain experiment.

5. Activation of the deep spinal muscles followed a biphasic course with an early response lasting for 5–8 minutes followed by a silent period of 15–20 minutes which was then followed by an increase in EMG activity. This biphasic activation is very reminiscent of the response reported to occur in the formalin model in awake rats (24).

These findings supported, for the first time, the notion that injury to the deep spinal tissues provokes both local and contralateral as well as “distal” muscular activation, likely as a protective response. These results also imply that reflex connections exist between upper cervical afferents and both local spinal and trigeminal motoneurons which subserve the muscular activity observed. In terms of chiropractic theory, these findings provide evidence of muscular hypertonicity induced by a form of painful spinal dysfunction (“subluxation inducing local and distal muscular reactions”).

In 1995, Yu et al. (46) replicated the EMG study described above while injecting mustard oil into the rat TMJ. Of interest are the following findings, particularly as compared to the neck/EMG study:

1. Ipsilateral digastric and masseter muscle activity increased significantly over control levels.
2. Only slight, brief increases in contralateral masseter muscle EMG activity over control was found. This is in contrast to the larger, more prolonged responses in the contralateral RCPMa found in the neck/EMG model.
3. Only one phase of activation was noted in any of the muscles, lasting up to 10 minutes postinjection. This is in contrast to the biphasic pattern observed in the RCPMa muscles in the neck model.
4. No increase in trapezius or deep neck muscle EMG was found. This is in contrast to the finding of increased (albeit of a smaller magnitude) activity in the jaw muscles evoked by irritation of the deep neck muscles.

Therefore, a comparison of the neck versus TMJ EMG studies reveals differences in the pattern of evoked muscle activity depending on the site of experimental inflammatory algesia. Responses in rats to TMJ injection appear to be more focal and confined to the jaw muscles on the lesioned
Neuroplasticity of Neck/Craniofacial Pain Mechanisms

FIGURE 6. Average changes in EMG activity induced by injections of mineral and mustard oil into deep paraspinal tissues surrounding the C1–C3 vertebrae. The means (±S.D.) data shown are normalized values relative to the mean value of the preinjection EMG activity (= 100%) in each rat. The solid line shows the changes in EMG activity of one group of rats receiving mustard oil at 20 minutes after the vehicle (mineral oil) injection (n, number of rats tested by the injection of mustard oil). The dashed line shows the changes in EMG activity of the control group of four rats receiving the vehicle, instead of mustard oil, again 20 minutes after the first vehicle injection. # and ##, p < .05 and p < .01 (Wilcoxon test) indicate significant difference between preinjection and postinjection level of EMG activity.* and **, p < .05 and p < .01 not only indicate significant difference between the preinjection and the postinjection level of EMG activity (Wilcoxon test), but also between the animals receiving mineral then mustard oil and the animals receiving mineral oil injection twice (Mann-Whitney test). (Reproduced, with permission, from Hu J, et al. Pain 1993; 55:243–250. Copyright © 1993 by Elsevier Science.)
side. No neck muscle activity is evoked. Irritation of the spinal (axial) tissues appears to evoke a more diffuse pattern involving contralateral segmental muscles, superficial regional muscles, and, to at least some degree, activation of "distal" jaw muscles. One explanation for these differences may involve a more diffuse divergence pattern of spinal nociceptive afferents and their either direct or indirect (through interneurons) connection to the cervical and trigeminal motor nuclei.

In 1995, Hu et al. (47) reported on a similar EMG study using mustard oil applied to posterior meningeal/dural vascular tissues. These tissues are exposed during preparation of the experiment because of the required removal of the posterior/vertex region of the skull. Mustard oil/mineral oil applications were made directly onto the dura and the sagittal sinus. Again, after a 20-minute baseline where no EMG activation occurred, mustard oil, but not mineral oil induced EMG activity increases in the left masseter muscle (small increases), left deep neck muscles (large increases, lasting for up to 40 minutes), the right deep neck muscles (moderate increases, lasting up to 35 minutes), and the left trapezius (large increases lasting up to 30 minutes). These results confirm that posterior cranial structures, which are innervated by the C1–C2 anterior primary rami, make similar synaptic convergence into the trigemino-cervical nucleus and provoke similar muscular responses as do the posterior deep muscles innervated by the posterior primary rami of C1–C2. These results support the notion of both referred pain and "referred" reflex muscle spasm arising from painful disorders of the meninges and dural vessels of the posterior cranium such as meningitis and, more importantly for chiropractors, migraine headache. The "subluxation"-type findings in the upper cervical spine which may be observed by clinicians on careful manual assessment of their patients (48) may, therefore, be a manifestation of viscerosomatic reflex disturbances, making diagnosis and treatment of these disorders all the more challenging. To our knowledge, this study represents the first report of spinal somatic dysfunction (as spinal muscular hypertonicity) induced by a "visceral" irritative source (i.e., the classical viscerosomatic reflex disorder).

In related studies, Angus-Leppan et al. (49) and Goadsby et al. (50) have demonstrated that stimulation of the superior sagittal sinus and the occipital nerve (posterior ramus of C2) activate similar second-order neurons in the dorsolateral area of the upper cervical cord and the subnucleus caudalis.

The recent work in Seskle's laboratory has concentrated on two areas: 1) the neurochemical mechanisms involved in deep pain from the TMJ and deep neck tissues, and 2) single unit recording studies of upper cervical neurons in response to deep neck (as opposed to TMJ) experimental inflammatory pain.

In 1996, Hu et al. (51) replicated the 1993 neck/EMG study with a view toward elucidating aspects of neurochemical involvement. Recall that the response pattern of the deep neck muscles (bilaterally) was often biphasic, beginning with an initial elevation lasting about 10 minutes followed by a return to baseline and, after about 20 minutes, a return of elevated EMG activity. This second activation phase was proposed to be due to a combination of central sensitization of dorsal horn neurons and sustained tissue inflammation producing summation of enhanced C-fiber firing. We hypothesized that the middle phase might be produced as a result of active suppressive or inhibitory mechanisms evoked in response to the initial nociception. We further hypothesized that this suppressive phase may be subserved by an opioid mechanism.

In 1994, Yu et al. (52) had demonstrated that injection of naloxone, an opioid antagonist, could produce a "rekindling" effect on the EMG output of TMJ inflammation-induced muscular activation. In order to confirm this hypothesis, we "manipulated" the paraspinal-inflammation model by injecting an opioid receptor antagonist, naloxone, once the second phase of EMG silence had begun.

A dose-dependent naloxone-induced "rekindling" effect on the EMG recordings was demonstrated. At the highest dose, injection of naloxone resulted in an average EMG increase of 83% of the original mustard oil-induced EMG activity. It appears that the normal physiologic response to injury involves mechanisms designed to suppress the initial protective muscular responses (perhaps as part of an overall anti-nociceptive response). However, in the presence of sufficient and persistent tissue irritation, this inhibitory process is overcome and the nociceptor-driven EMG activity is reactivated.

Yu et al. (53) explored the neurochemical mechanisms involved in mustard oil-induced central sensitization. Using the TMJ/EMG model combined with injection of several blocking agents including lidocaine, a peripheral anesthetic, naloxone, and MK801, an NMDA-receptor blocker, they found that lidocaine significantly attenuated the EMG activation of the masseter and digastric muscles but not the plasma extravasation in the TMJ region. This confirms that the MO-induced EMG increase is reflex-based. Naloxone induced rekindling of the MO-induced EMG activity once suppression had occurred. Various dosages of MK801 (locations: intracerebroventricular, intravenous, and directly into the TMJ; dosages: ranging from 0.01 to 0.5 mg/kg) reduced EMG activity. Particularly, the application of MK801 peripherally into the TMJ region (at 0.1 but not at 0.01 mg/kg) reduced muscular responses. These results support the conclusion that glutamate mechanisms are involved in the induced muscular responses for both central and peripheral mechanisms, a finding not previously reported.
Finally, Hu, Sun, Zhang, and Vernon (unpublished data) have created a model whereby single dorsal horn unit recordings can be made of responses to deep neck mustard oil injection. This model required considerable innovation in the surgical procedures used for the TMJ study. This is because the standard method of implantation of the recording electrodes requires the use of a stereotaxic apparatus to stabilize the preparation while the recording electrode is implanted. Furthermore, the usual procedure is to perform a laminectomy of atlas and axis posterior structures in order to expose the spinal cord for implantation of the recording electrode from above. Performing these procedures produces three highly undesirable effects when the objective is to study DHN responses to inflammatory irritation of the deep neck muscles:

1. Obviously, the posterior neck tissues are either removed or damaged, rendering them useless for MO injection.
2. A significant amount of bleeding occurs which would further prevent posterior tissue injection.
3. The surgical procedure itself may induce considerable nociception (despite the adequate central anesthesia) and may produce a degree of central modulation such as DNIC, that might have (and probably has) affected the dorsal horn neuronal responses.

A new surgical technique was devised, known as the "minimal trauma technique." Under fluoroscopic guidance, the recording electrode is threaded down the medulla through a small hole in the superior skull. The termination of this electrode implantation is confirmed both radiographically and, subsequently, histologically. Figures 7 and 8 show one preparation with the recording electrode implanted near a WDR neuron in the upper cervical cord. The rat figureines demonstrate the baseline touch and pinch RFs which, after percutaneous injection of mustard oil into the C1–C2 paraspinal region, are seen to expand to include, by 20 minutes, the entire facial region. While this work is still in progress, it represents the first report of the somatosensory consequences of cervical spinal joint/muscle injury. Gillette and his colleagues have reported similar results in the lumbar region of the rat (45,46). These expanded RFs represent a state of central sensitization whereby the excitability of DHNs is increased, silent afferent connections are activated, and spontaneous firing is induced.

The expanded receptive fields themselves are probably at least demonstrative of, if not the basis of, referred pain which, in the case of these studies, arises in the upper cervical paraspinal region and initially includes the posterior cranium, but then, subsequent to the MO injection, expands forward to include the facial region. The extent and pattern of this expansion are very reminiscent of the zones of referred pain elicited from upper cervical tissues by Feinstein (54) and Travell and Simons (55) and demonstrated to be abolished by studies from the laboratory of Bogduk and his colleagues (56,57).

The expansion of receptive fields of nociceptive neurons to include extensive skin areas is also a likely mechanism for the hyperesthesia and allodynia that may be seen after injury to the deep tissues.

CONCLUSION

Nociceptive afferents from tissues innervated by the upper two or three cervical segments and the first division of the trigeminal nerve converge on the same second-order neurons residing in the "trigemino-cervical nucleus." That this occurs should not surprise anyone, as rostro-caudal connectivity exists at all levels of the cord, and the first
FIGURE 8. Time course of mechanoreceptive field (RF) change in a C, nociceptive neuron after injection of mustard oil into the paraspinal tissue.

trigeminal division is, in reality, the next rostral neuromere to the upper cord. Our work has confirmed that complex patterns of sensorimotor processing underly the phenomena associated with craniofacial pain, and that various named "syndromes," such as upper cervical dysfunction, "greater occipital neuralgia," cervicogenic headache, neck-tongue syndrome, and temporomandibular joint pain, may all share at least some aspects of this common mechanism.

Notwithstanding this overall commonality, there appear to be some important differences between the spinal and extraspinal components of this "system," in that, pain arising from spinal sources appears, from our data, to evoke more widespread manifestations such as the expansion of receptive fields and more extensive muscular activity. The clinical manifestations of these mechanisms are likely to include referred pain and muscular hypertonicity which are so common in the myofascial pain syndromes of this region. The astute clinician is, then, presented with much greater diagnostic complexity, but is also given expanded opportunities for therapeutic intervention in the challenging area of craniofacial pain syndromes.

REFERENCES
Neuroplasticity of Neck/Craniofacial Pain Mechanisms


Assessment of Self-Rated Disability, Impairment, and Sincerity of Effort in Whiplash-Associated Disorder

Howard Vernon

ABSTRACT. Objectives: To review the reliability and comparability of different pain and disability self-report questionnaires as they relate to patients with whiplash-associated disorders [WAD] and to conduct analytic cohort studies of two samples of subjects with respect to impairment and sincerity of effort.

Methods: The Neck Disability Index, Neck Pain Questionnaire, and the Copenhagen Neck Functional Disability Index were reviewed for comparability of pain, impairment, and disability items. Two small clinical samples were assessed with disability and strength tests.

Results: The three instruments for self-rated disability in WAD have numerous similarities in content and format, and have equally good basic psychometric efficacy. The Neck Disability Index has been used in a larger number of reports, and more is known about its psychometric properties, particularly as they apply to the assessments made in research studies.

The two small studies demonstrate that sincerity of effort is an important variable to consider in interpreting both the subjective reports of pain and self-rated disability as well as the objectively obtained measurements of movements and strength in WAD patients.

Conclusions: High quality instruments now exist for the assessment of self-rated disability and, in two cases, self-rated pain in WAD patients. These instruments are suitable for both clinical and research settings in the clinical management of WAD. The Neck Disability Index...
has been studied more extensively than the other two and is probably
the instrument of choice for research settings. The original investiga­
tions in this report suggest that the link between subjective reports and
objective test performances by WAD patients should be calibrated by an
assessment of the patient's sincerity of effort. [Article copies available for a
fee from The Haworth Document Delivery Service: 1-800-342-9678. E-mail
address: getinfo@haworthpressinc.com <Website: http://www.haworthpressinc.
com>]

KEYWORDS. Whiplash, pain, assessment, sincerity, impairment

INTRODUCTION

The term "whiplash-associated disorder" [WAD], coined by the
Quebec Task Force Report (1), was designed to denote that injuries to
the cervical spine in automobile collisions can create a complex array
of symptoms, impairments, and disabilities. Understanding this com­
plex has become a challenge to clinicians and researchers alike.

The grading system adopted by the Task Force was designed to
assist in the sorting of the major categories of injury status, and their
likely sequelae. Musculoskeletal soft tissue injuries [Grades 1 and 2]
were distinguished from those which also involve peripheral neural
disorders [Grade 3] and serious bony or joint lesions such as fractures
and dislocations [Grade 4]. Extrapolating from statistics from the On­
tario Ministry of Transport database (2), it is likely that over 65% of
post-motor vehicle accident [MVA] injuries are in the first grade,
while no more than 15% are in the third and fourth grades, leaving
about 20% of MVA claims in the second grade of severity.

However, once this level of categorization has been applied, clini­
cians of all health care groups are challenged to conduct accurate
clinical diagnoses as to the specific tissues injured and the specific
impairments incurred by each of their patients. As well, they and their
clinical research counterparts are challenged to conduct accurate eval­
uations of the health status of their injured patients. Furthermore, they
are challenged to accurately assess changes in health status, particular­
ly improvements, as these changes are often evaluated by way of
guideline-derived recovery patterns. Deviations in the expected pat­
tern of health recovery following WAD may lead to inappropriate care
and prolonged disability.
has been studied more extensively than the other two and is probably the instrument of choice for research settings. The original investigations in this report suggest that the link between subjective reports and objective test performances by WAD patients should be calibrated by an assessment of the patient's sincerity of effort.

KEYWORDS. Whiplash, pain, assessment, sincerity, impairment

INTRODUCTION

The term "whiplash-associated disorder" [WAD], coined by the Quebec Task Force Report (1), was designed to denote that injuries to the cervical spine in automobile collisions can create a complex array of symptoms, impairments, and disabilities. Understanding this complexity has become a challenge to clinicians and researchers alike. The grading system adopted by the Task Force was designed to assist in the sorting of the major categories of injury status, and their likely sequelae. Musculoskeletal soft tissue injuries [Grades 1 and 2] were distinguished from those which also involve peripheral neural disorders [Grade 3] and serious bony or joint lesions such as fractures and dislocations [Grade 4]. Extrapolating from statistics from the Ontario Ministry of Transport database (2), it is likely that over 65% of post-motor vehicle accident [MVA] injuries are in the first grade, while no more than 15% are in the third and fourth grades, leaving about 20% of MVA claims in the second grade of severity.

However, once this level of categorization has been applied, clinicians of all health care groups are challenged to conduct accurate clinical diagnoses as to the specific tissues injured and the specific impairments incurred by each of their patients. As well, they and their clinical research counterparts are challenged to conduct accurate evaluations of the health status of their injured patients. Furthermore, they are challenged to accurately assess changes in health status, particularly improvements, as these changes are often evaluated by way of guideline-derived recovery patterns. Deviations in the expected pattern of health recovery following WAD may lead to inappropriate care and prolonged disability.

One way of considering the complexity of WAD is to apply multi-dimensional pain theories to the problem. According to Melzack and Torgerson's (3) theoretical model, pain can be considered to have several dimensions: sensory, affective, and cognitive. When applied to WAD, it can be seen that sensory dimensions include local and/or radiating neck-arm pains, upper back pains, headache, and paraesthesia or other sensory abnormalities. Common manifestations of the affective dimension include distress and anxiety, particularly in the early post-accident period, activity-avoidance behaviors based upon fear of pain and, later, in the more chronic phase, depression. Aspects of cognitive dysfunction include changes in memory and concentration function as well as sleep disturbances.

According to Waddell (4), a biopsychosocial perspective is necessary for a full understanding of patients with musculoskeletal injury. A somatic pain complaint is at the core of this model, but just as important are the means by which the injured patient copes with his/her condition, its impact on their daily activities and roles, and the abnormal influence on their health status. This model also conforms to the World Health model which links disorders to impairments which lead to disability, and possibly to handicap. These models provide a framework within which a comprehensive clinical evaluation can be conducted.

This paper will review a set of instruments which can be employed by clinicians and researchers alike in the evaluation of self-rated disability. Then, a set of studies in which measures of self-rated disability have been correlated to measures of impairment and sincerity of effort will be reviewed. In order to complete this section of the paper, a review of studies in which measures of self-rated disability have been correlated to measures of impairment and sincerity of effort will be provided.

MEASURES OF SELF-RATED DISABILITY

A number of instruments are available which measure the patient's ability to perform important activities of daily living [ADL] and/or functional capacity measures. There is debate, however, as to the theoretical construct underlying these instruments, as there should be a distinction between true versus self-rated functional capacities. When viewed from the latter perspective, scores on "functional" instruments may depict the patient's self-perception of functional capability, which may be more accurate than true functional capacity.
Neck Disability Index

The first instrument designed for assessing self-rated disability due to neck pain was the Neck Disability Index (NDI) (7). Designed as a modification of the Oswestry Low Back Pain Disability Index (OLBPDI) (8), the NDI is a ten-item questionnaire with well-established psychometric properties such as high test-retest reliability, good internal consistency, and good sensitivity to change. Mains et al. (9) established a single factor structure to the NDI as well as reporting that no response bias could be found amongst the items.

Riddle and Stratford (10) have recently added to the psychometric profile of the NDI by determining three important values for its use in clinical and research settings. These are: variation around a measured value, minimal detectable change, and minimal clinically important difference (MCID). Variation around a measured value addresses the error margin inherent in any single use of the NDI, typically in a practice setting. This value was found to be 5 NDI points, at a 90% confidence interval. For example, given a 20/50 score (NDI is a 50 point scale) with a 5 point error at 90% confidence interval, then a clinician can be 90% sure that the true score lies between 15 and 25 (11).

Minimal detectable change and the MCID values have been estimated at 5 NDI points each (10). This means that the sampling error of the instrument limits the range of minimal detectable change in a patient's status to 5 NDI points. As a result of its use in a cohort of neck pain patients, it was determined that the minimal clinically important difference is also 5 NDI points. However, several studies have reported mean change scores well beyond that level (12, Bronfort-personal communication).

In a study of 44 chronic WAD claimants (13), additional psychometric features of the NDI were reported. First, this sample's responses were in almost identical rank order as the initial sample (7). The items "headache," "lifting," "recreation," and "reading" were still among the five most highly rated items, confirming their importance in chronic WAD. Second, NDI scores were not well-correlated with age and gender, as would be expected in chronic WAD. Second, NDI scores were not well-correlated with age and gender, as would be expected in chronic WAD.

Neck Pain Questionnaire

The Neck Pain Questionnaire (NPQ) was developed in 1994 (15), again as an outgrowth of the OLBPDIQ. No report of the methodology for adapting the OLBPDI was given. The NPQ contains nine items scored 0 to 4, total = 36. The items consist of the following: pain intensity, sleeping, numbness, duration, carrying, reading/television, work, social life, and driving. These items represent a mix of symptoms and activities thought to be important to neck pain patients. Of these activity items, all but one, carrying vs. lifting, had already been incorporated in the NDI. Both instruments featured the "pain component," which was incorporated in both questionnaires. The items "headache," "lifting," "recreation," and "reading" were still among the five most highly rated items, confirming their importance in chronic WAD. Second, NDI scores were not well-correlated with age and gender, as would be expected in chronic WAD.

Forty-four subjects completed an NPQ at their original consultation while 31 completed a second NPQ three to five days later. Thirty-five of these subjects also completed the NPQ four and 12 weeks later. No formal treatment was offered in the study, but many subjects did receive some form of treatment. Short-term reliability was high (Pearson's coefficient R = 0.84, Kappa = 0.62). Inter-item agreement ranged from K = 0.53 to 0.76. Internal consistency was not formally tested, but was graphically depicted as adequate (15).

Initial NPQ scores did not correlate well with age, gender, duration of complaint, or any of the other clinical variables. As a result, the NDI may be a more appropriate instrument for assessing self-rated disability in chronic WAD.
NPQ scores did not change significantly over the three-month study interval, they did correlate well with a separate question rating the subject's perception of improvement. This was cited as an indicator of "sensitivity to change." Subsets of subjects who either received physiotherapy or performed home exercises had what was described as "significant improvement" in their NPQ scores.

Given the similarities between the NDI and the NPQ, this author considers Leak et al.'s report (15) to be essentially a replication study of the NDI. That the same set of psychometric properties was reported, namely, high levels of test-retest reliability, internal consistency, and sensitivity to change, as well as poor correlation with age, gender, and duration of complaint, is therefore not surprising and confirms the findings of the original NDI study (7). To this author's knowledge, no additional studies on the NPQ have been reported since 1994.

### Copenhagen Neck Functional Disability Scale

The Copenhagen Neck Functional Disability Scale (CNFDS) by Jordan et al. (16) was an attempt to improve the existing questionnaires (NDI, NPQ). The authors asserted that, since both previous instruments incorporated some items related to subjective symptoms (pain, numbness, and duration in the NPQ; pain, headache, and concentration in the NDI), these questionnaires lacked precision for measuring solely the disability due to neck pain itself. This assertion was based on the theory that pain, disability, and impairment are separate but inter-related constructs.

The CNFDS contains 15 items with a three-point scale [yes = 0, occasionally = 1, no = 2] with a maximum of 30 points. Many of the item constructs are similar to those in the NDI [sleep, personal care, lifting, reading, headaches, concentration, and recreation], while three additional items focus on psycho-social issues.

They reported very high test-retest reliability [R = 0.99], excellent internal consistency [Cronbach's alpha = 0.89], and no significant correlation between initial scores and age or gender. Initial CNFDS scores were highly correlated with patients' global assessment of their pain and disability [R = 0.80] and moderately correlated with doctors' global assessment of their pain and disability [R = 0.59]. Initial scores also highly correlated with a global assessment of physical condition [R = 0.59] and moderately correlated with doctors' global assessment of their physical condition [R = 0.49].

The CNFDS correlates with a larger sample of subjects enrolled in a clinical trial for neck pain (17). At 6, 24, and 52 weeks of this trial, changes in pain scores correlated with changes in CNFDS scores at R = 0.49, 0.48, and 0.54, respectively. Over the three-month study interval, the correlation was similar in the NDI [sleep, personal care, lifting, reading, headaches, concentration, and recreation], while Item 2 "pain" was well correlated with the CNFDS [sleeping, social, future].

### Table 1: Item Comparison of Neck Disability Scales

<table>
<thead>
<tr>
<th>INSTRUMENT</th>
<th>(Publication date)</th>
<th>ITEMS</th>
</tr>
</thead>
<tbody>
<tr>
<td>NDI</td>
<td>[1991]</td>
<td>pain, personal care, lifting, reading, headaches, concentration, recreation, work, driving, sleeping, recreation</td>
</tr>
<tr>
<td>NPQ</td>
<td>[1994]</td>
<td>pain, sleeping, numbness, duration, carrying, reading/TV, work, social, driving, work, social</td>
</tr>
<tr>
<td>CNFDS</td>
<td>[1998]</td>
<td>sleeping, daily activities, dressing, washing, at home, lifting, reading, headaches, concentration, recreation, resting, family, social, future</td>
</tr>
</tbody>
</table>

**Additional Notes:**
- NDI = Neck Disability Index
- NPQ = Neck Pain Questionnaire
- CNFDS = Copenhagen Neck Functional Disability Scale
- Active Ranges of Motion (AROM): Reduced in chronic whiplash-injured patients (18,19). Os-Pherson studies have reported that active range of motion is reduced in chronic whiplash-injured patients (18,19).
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terbauer et al. (20) recently reported that 10 WAD cases had a combined AROM of 234°, less than a normal 360°. After six weeks of conservative treatment, total AROM increased to 297°. Hagstrom and Carlsson (21) compared 30 WAD cases with 30 normal subjects and found reduced AROM in all ranges tested.

Vernon (13) measured AROM in a sample of 44 chronic WAD patients and found moderately high correlation \( P = 0.001 \) to 0.0001 between all ranges and the subjects' NDI scores. This was the first demonstrated link between physical neck measures and self-reported disability in WAD patients. Interestingly, AROM scores were not correlated with age and duration of complaint, again, leading to the notion that chronic WAD sufferers reach a stable plateau of self-rated disability and impaired ranges of neck motion. In contrast, comparisons of CNFDS scores with active neck extension found no significant correlation.

Strength Testing in the Cervical Spine

A study of isometric strength differences between paired trials of both groups (mean inter-trial difference = 7%) (22). Variance of bilateral range of rotation and lateral bending was typically no more than 10%. The flexion/extension (F/E) strength values was confirmed in normal subjects was 0.60. In WAD patients, this ratio decreased to 0.24, suggesting that flexor muscle strength loss is greater in these cases.

Study 1

Methods:

Ten normal, healthy male subjects were asked to perform maximum voluntary effort (MAX) and also to simulate malingerers (INSIN). The subjects included 17 cases with pairs of their contemporaries were included. The sample included 17 WAD cases with persistent complaint of 11.3 months [SD 1.7]. Four subjects had been involved in motorcycle accidents with a mean age of 33.5 years [SD 9.8].

Results:

The differences between paired trials of both groups were significant \( P < 0.05 \) [mean inter-trial difference = 15.5%]. All differences between modes with the exception of extension were statistically significant. The mean percentage differences between MAX-INSIN values was 36%, with all differences being statistically significant. The mean resistance to INSIN was 0.77, with all differences being statistically significant. The mean resistance to MAX was 0.60.

Conclusions: This study was able to provide a model for deliberate reduction in effort in cervical muscle strength testing. Factors identified in the modeled behavior included:

1. A consistent reduction in the level of effort,
2. Consistent inconsistency between paired trials of three of four ranges in the insincere mode,
3. A significant increase in the flexion/extension ratio, indicating that modeled "malingerers" are not aware that greater reduction in flexion strength is the expected norm of effort.

Study 2

Methods:

In this preliminary study, 17 WAD cases with persistent complaints were recruited. The sample included 12 females and five males with a mean age of 33.5 years [SD 9.8], and a mean duration of 11.3 months [SD 1.7]. Two-thirds had been involved in motorcycle accidents with a mean age of 33.5 years [SD 9.8].

Results: Table 2 shows the percent differences between the paired trials. The average difference in the INSIN mode was 15.5%, all differences between modes with the exception of extension were statistically significant. The mean percentage differences between MAX-INSIN values was 36%, with all differences being statistically significant [Chi-square \( P < 0.05 \)].

Conclusions: This study attempted to provide a model for insincere effort in cervical muscle strength testing. Factors identified in the modeled behavior included:

1. A consistent reduction in the level of effort,
2. Consistent inconsistency between paired trials of three of four ranges in the insincere mode,
3. A significant increase in the flexion/extension ratio, indicating that modeled "malingerers" are not aware that greater reduction in flexion strength is the expected norm of effort.

| Table 2: Shows Results of Study 1 Isometric Strength Efforts of the Cervical Spine. MAX = Maximum Voluntary Effort. INSIN = Insincere or Sub-Maximum Effort. The Differences Between MAX vs. INSIN Were Significant \( P = 0.05 \). |
| --- | --- | --- | --- | --- |
| Range of Motion | Paired MAX | Paired INSIN | Difference of means | Difference of \% |
| Flexion | 42 | 10 | 32 | 155 |
| Extension | 46 | 7 | 39 | 36 |
| Right lateral flexion | 46 | 7 | 39 | 36 |
| Right rotation | 46 | 7 | 39 | 36 |
| Mean values | 36 | 115 | 7 | 7 |

STRENGTH STUDIES, AND SINCERITY OF EFFORT

Strength Testing in the Cervical Spine

A study of isometric strength differences between paired trials of both groups (mean inter-trial difference = 7%). Variance of bilateral range of rotation and lateral bending was typically no more than 10%. The flexion/extension (F/E) strength values was confirmed in normal subjects was 0.60. In WAD patients, this ratio decreased to 0.24, suggesting that flexor muscle strength loss is greater in these cases.

Formulas: Ten normal, healthy male subjects were asked to perform a maximum voluntary effort (MAX) and also to simulate malingerers (INSIN). The mean resistance to INSIN was 0.77, with all differences being statistically significant. The mean resistance to MAX was 0.60.
rear-end collisions, 24% were in front-end, and 11% in side collisions. All but one of the subjects complained of pain in the neck and lower back regions.

Isometric cervical flexion and extension values were obtained, with one trial using the manual MSD described above. The F/E ratio was compared to previous norms noted above (22,23). Next, a set of seven “non-organic signs” [NOS] modified from Waddell et al. (24) were used (Table 3). Based upon the results of the measurements, the subjects were grouped into those with four or more NOS [high NOS] and those with three or less NOS [low NOS]. The differences between the NOS groups comparing NDI and F/E ratios are shown in Table 4.

### Table 3. “Non-organic signs” [NOS] Used in This Study [Modified from Waddell et al. (25). [* = Standard Item in “Waddell’s non-organic signs”]]

<table>
<thead>
<tr>
<th>Subject</th>
<th>Cervical active ROM less than ROM’s observed informally [in at least 2 ranges].</th>
<th>Cervical active ROM greater than passive ROM’s [in at least 2 ranges].</th>
<th>Sitting Straight Leg Raise [SLR] greater than supine SLR by at least 30° in at least one leg.</th>
<th>Supine SLR does not increase by at least 20° with opposite knee bent.</th>
<th>Plantar-flexion of foot in the SLR provokes back pain [either leg].</th>
<th>Waddell Rotation Test positive. [*]</th>
<th>Excessive symptom amplification during examination [frustration].</th>
</tr>
</thead>
<tbody>
<tr>
<td>Low-NOS</td>
<td>No</td>
<td>No</td>
<td>No</td>
<td>No</td>
<td>No</td>
<td>No</td>
<td>No</td>
</tr>
<tr>
<td>High-NOS</td>
<td>Yes</td>
<td>Yes</td>
<td>Yes</td>
<td>Yes</td>
<td>Yes</td>
<td>Yes</td>
<td>Yes</td>
</tr>
</tbody>
</table>

### Table 4. Shows Results of Study 2, Where Two Groups Were Identified by High or Low Non-Organic Signs [NOS], Which Correlated to the Mean Neck Disability Index [NDI] and F/E Ratios for Each Group

<table>
<thead>
<tr>
<th>Variable</th>
<th>Low-NOS Group</th>
<th>High-NOS Group</th>
</tr>
</thead>
<tbody>
<tr>
<td>Mean NDI score/50</td>
<td>17.6 [9.1]</td>
<td>36 [3.7]</td>
</tr>
<tr>
<td>Flexion/Extension ratio [F/E]</td>
<td>0.81 [0.21]</td>
<td>1.07 [0.29]</td>
</tr>
</tbody>
</table>

**Results:**

The high NOS F/E values [1.07] obtained with the manual MSD, were elevated in approximately the same proportion as seen with the stationary MSD, when comparing normal [0.60] to “modeled insincere” [0.77].

**Discussion:** Chronic whiplash subjects who demonstrated a high level of non-organic pain amplification behavior had self-rated disability scores which were more than twice that of the low NOS group. These scores were more than twice that of the low NOS group.

**Conclusion:** This paper has reviewed a small set of instruments for self-rating of perceived disability suitable for use in evaluating WAD. It is not yet clear that the condition-specific instruments for self-rating of disability are more valid than generic instruments such as the DRI, but they all have the same conceptual appeal of face validity. The NDI has been shown to correlate moderately with actual disability.

Mean NDI score of the whole sample was 24.6 [SD 11.8, range 1 to 39/50].

**Note:** Table 4 shows results of Study 2, where two groups were identified by high or low non-organic signs [NOS] which correlated to the mean neck disability index [NDI].
should result in improved evaluation of WAD. Furthermore, new information on the importance of many of the individual item characteristics in these instruments will help in clarifying the typical patterns of WAD expression and development. For instance, does a high score on any of the items in the NDI, NPQ or CNFCD [headache, concentration, etc.] correlate with poorer prognosis? Can clusters of high-scoring items be identified and correlated with other measures of physical or cognitive functioning? Do certain items appear to respond better to certain kinds of treatment approaches? These and many other questions are the challenges for future investigations into the WAD phenomenon.

REFERENCES


Spinal Manipulation in the Management of Tension-type Migraine and Cervicogenic Headaches: The State of the Evidence

**Purpose:** A cervicogenic origin for various types of headache has been postulated and debated for decades. By 1988, the International Headache Society published its *Classification and Diagnostic Criteria for Headache Disorders, Cranial Neuralgias and Facial Pain*, including a category for "cervicogenic headache." Studies that have explored the effectiveness of spinal manipulation for various headache complaints are reviewed. **Method:** Systematic literature review. **Summary:** A strong case can be made for the pathophysiology of headaches of cervical origin. Such a cervical component may be present in tension-type and migraine headaches as well as the currently accepted category of "cervicogenic headache." Key words: cervical vertebrae, chiropractic, headache, headache disorders, orthopedic manipulation

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Center for the Study of the Cervical Spine
Canadian Memorial Chiropractic College
Toronto, Ontario, Canada

In the decade of the 1990s, a number of important advances took place in the understanding of the role of the cervical spine in headaches. In 1988, the International Headache Society (IHS) published its *Classification and Diagnostic Criteria for Headache Disorders, Cranial Neuralgias and Facial Pain*. For the first time, a category known as "cervicogenic headache" (CH) was included. The IHS description of CH was an expanded version of the definition that had originated with the work of Sjaastad and his colleagues in the earlier part of the 1980s. By that point in time, both the IHS and Sjaastad and his colleagues were acknowledging the work of authors extending back as far as the 1940s in developing the role of the cervical spine in headaches (see reviews in references 6–10).

In 1991, Jensen et al undertook a study of the role of the cervical spine in posttraumatic headache. In addition to reporting the beneficial effect of spinal manipulation compared with ice treatment in the short-term management of this type of headache, they reported on the high prevalence of cervical spine dysfunction or joint hypomobility in their sample.

The phenomenon of cervical joint and myofascial dysfunction in headache was further advanced by the work of Vernon and colleagues who, in 1992, reported on a high prevalence of cervical joint and myofascial dysfunction findings in subjects with tension-type and migraine headache. Additionally,
through the decade, Jull and her colleagues published a series of studies\textsuperscript{19-21} that also supported the role of this type of dysfunction in posttraumatic cervical headaches. They, and other Australian investigators, also reported relatively high levels of reliability and validity of the manual palpatory procedures designed to identify clinically important joint dysfunction in the cervical spine in headache sufferers.\textsuperscript{16-18}

In 1993, Hu et al\textsuperscript{19} published an important study that demonstrated the response patterns of craniovascular muscles in a rat model of upper cervical deep paraspinal inflammatory irritation. Their study constituted the first animal model of painful cervical joint dysfunction and showed a wide-ranging pattern of evoked muscular responses, similar to those predicted to occur in humans who suffer CH. This muscular response pattern was more wide-ranging than the one that had been previously published involving the same inflammatory irritation of the temporomandibular joint. In other words, the response pattern evoked by irritation of spinal deep tissues was shown to be different from and more extensive than the one evoked by proximally-located joint tissues; a pattern that was also demonstrated in the work of Gillette and his colleagues with respect to the lumbar spinal tissues.\textsuperscript{20}

Hu et al\textsuperscript{21} went on to demonstrate that the same craniovascular muscular response pattern was evoked by inflammatory irritation of the posterior cerebral dura and blood vessels, pointing to the kind of “viscero”-somatic reflexes that might be present in migraine headache. Finally, Hu et al have investigated the response of single dorsal horn neurons in the upper cervical cord to deep paraspinal irritation. They have showed the kind of expanded craniofacial cutaneous receptive fields that are indicative of central sensitization (personal communication, 2001). All of these basic science studies have expanded knowledge of the role of deep upper cervical paraspinal inflammatory pain in the maintenance of the sensory-motor reflex dysfunctions that may lie at the heart of cervicogenic headache.\textsuperscript{22-24}

A crucial contribution to these developments came from the Australian group headed by Nikolai Bogduk.\textsuperscript{25-27} He and his colleagues developed a methodology for identifying painful cervical zygapophyseal joints in patients who suffered chronic whiplash by using double-blind trials of injectable anesthetic blocking agents. The prevalence of cervical “z-joint” dysfunction in patients suffering from whiplash, particularly those with headaches as a primary symptom, was reported to be in the order of 50%. Procedures to produce long-lasting analgesia of these joints were validated by several important clinical trials.\textsuperscript{26}

In 1995, Hack et al\textsuperscript{28} reported a new human anatomic finding consisting of a musculofibrous band connecting the deep rectus capitis posticus minor muscle to the cervico-occipital dura mater. This connection was proposed as another potential mechanism of cervicogenic headache, in that traction from the suboccipital muscle on the dura would likely be painful and could create referred pain into the cranial area. Humphreys et al\textsuperscript{29} have since identified another such connection between the ligamentum nuchae and the cervical dura mater, thus strengthening the role of this mechanism.

In 1994, the RAND panel on the Appropriateness of Manipulation and Mobilization of the Cervical Spine was convened. Its report\textsuperscript{29} was published in 1996 and outlined the kind of clinical scenarios, many of them involving headache, for which these manual therapies were deemed appropriate by these clinical experts. The literature review for this study was published by Hurwitz et al in 1996.\textsuperscript{30} This article was followed by an important meta-analysis by Aker and colleagues\textsuperscript{31} that also reported on clinical trials that demonstrated the relief of headaches with cervical spinal manipulation.

The status of the evidence regarding the treatment of benign adult headaches, including tension-types, migraine types, and CH, has also improved greatly in the past decade. Eleven randomized clinical trials,\textsuperscript{11,22-24} eight published in the 1990s,\textsuperscript{11,22-24,26,39-41} and one presented as an abstract,\textsuperscript{41} have been reported. Two of these have been for tension-type headaches,\textsuperscript{35-37} two for migraine,\textsuperscript{39-40} and four for CH.\textsuperscript{11,33,35,41} As well, six systematic reviews have been published since 1990, four of these specifically the treatment of headache,\textsuperscript{42-44} whereas two have addressed neck pain and headaches.\textsuperscript{30,31}

These studies will be reviewed in this article to give the reader an overview of the current state of the evidence regarding the effectiveness of spinal manipulation in the treatment of these most common headache types.

METHODS

The majority of information contained in this review was obtained from the work of Vernon, McDermaid, and Hagino.\textsuperscript{45} The search strategy employed in that review covers all reports of original studies on spinal manipulation for tension-type and CH up to 1999. Additional retrieval strategies included citation reviews of the systematic reviews done before 1999\textsuperscript{30} as well as materials from the author’s files. These last strategies were particularly relevant to the clinical trials on migraine headaches.

RESULTS

Before beginning the review of clinical evidence, one ancillary study should be discussed. To describe more fully the mode of treatment of headache by the majority of chiropractors, Vernon and McDermaid\textsuperscript{46} conducted a survey of Canadian chiropractic specialists. The overwhelming majority of these specialists endorsed the use of spinal manipulation, along with soft tissue therapies and postural exercises, as the most commonly used and most valuable therapies.

Table 1 provides the relevant data from the body of randomized clinical trials published since the late 1970s on the
Table 1. Evidence for studies of spinal manipulation for headaches

<table>
<thead>
<tr>
<th>Authors</th>
<th>Headache type</th>
<th>Sample size</th>
<th>Number of treatments</th>
<th>Treatment groups (n)</th>
<th>Results</th>
<th>Side effects</th>
<th>Quality scores</th>
</tr>
</thead>
<tbody>
<tr>
<td>Hoyt et al12</td>
<td>“muscle contraction”</td>
<td>22</td>
<td>1</td>
<td>(1) manipulation = 10 (2) MOB = 6 (3) rest = 6</td>
<td>Posttreatment S: (1) -48%* (2) 0 (3) 0</td>
<td>Not mentioned</td>
<td>56</td>
</tr>
<tr>
<td>Jensen et al11</td>
<td>Posttraumatic</td>
<td>19</td>
<td>2</td>
<td>(1) manipulation = 10 (2) ice = 9</td>
<td>Posttreatment S: (1) -3.2*(-69%) (2) +6.7/100</td>
<td>Not mentioned</td>
<td>60</td>
</tr>
<tr>
<td>Nilsson33</td>
<td>Cervicogenic</td>
<td>39</td>
<td>6</td>
<td>(1) manipulation = 20 (2) STT = 19</td>
<td>Posttreatment F: (1) -3.4 (-59%) (2) -2.1 (-45%) Posttreatment S: (1) -15 (-45%) (2) -10 (-24%)</td>
<td>Not mentioned</td>
<td>64</td>
</tr>
<tr>
<td>Nilsson et al34</td>
<td>Cervicogenic</td>
<td>53</td>
<td>6</td>
<td>(1) manipulation = 28 (2) STT = 25</td>
<td>Posttreatment F: (1) -3.2*(-69%) (2) -1.6 (-37%) Posttreatment S: (1) -17*(-36%) (2) -4.2 (-17%)</td>
<td>Not mentioned</td>
<td>72</td>
</tr>
<tr>
<td>Boline et al35</td>
<td>Tension-type headache</td>
<td>126</td>
<td>12</td>
<td>(1) manipulation = 70 (2) AMIT = 56</td>
<td>Posttreatment F: (1) -3.8/28 (2) -4.0/28 Follow-up F: (1) -1.0 (2) +5.0 Posttreatment S: (1) -1.3/20 (2) -1.8/20* Follow-up S: (1) -0.5 (2) +2.0</td>
<td>(1) 4.3% neck stiffness (2) 82.1% dry mouth, drowsy, or weight gain</td>
<td>75</td>
</tr>
<tr>
<td>Bove and Nilson36</td>
<td>Tension-type headache</td>
<td>75</td>
<td>8</td>
<td>(1) manipulation + STT = 38 (2) sham + STT = 37</td>
<td>Posttreatment F: (1) -1.5 h (2) -1.9 h Posttreatment S: (1) No change (2) No change</td>
<td>Not mentioned</td>
<td>80</td>
</tr>
<tr>
<td>Parker, Tupling, and Pryor37</td>
<td>Migraine</td>
<td>85</td>
<td>(1) CMT = 30 (2) MT = 27 (3) MOB = 28</td>
<td>Pre-Post F: (1) 8.5-5.1 (2) 11.4-9.9 (3) 8.7-5.7</td>
<td>Not mentioned</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

(Continues)
Table 1. Continued

<table>
<thead>
<tr>
<th>Authors</th>
<th>Headache type</th>
<th>Sample size</th>
<th>Number of treatments</th>
<th>Treatment groups (n)</th>
<th>Results</th>
<th>Side effects</th>
<th>Quality scores</th>
</tr>
</thead>
<tbody>
<tr>
<td>Parker, Pryor, and Tupling⁹⁸</td>
<td>Migraine</td>
<td>218</td>
<td>14</td>
<td>(1) CMT = 77 (2) AMI = 70 (3) CMT +AMI = 71</td>
<td>Pre-Post duration (hours): (1) 30.5-19.4 (2) 12.2-11.2 (3) 14.9-11.9 Pre-Post DIS(0-5): (1) 2.8-1.8 (2) 2.7-2.6 (3) 2.8-2.2 Pre-Post S (0-10): (1) 4.9-2.8* (2) 5.0-4.4 (3) 5.3-4.4</td>
<td>Not mentioned</td>
<td>continues</td>
</tr>
<tr>
<td>Nelson et al³⁹</td>
<td>Migraine</td>
<td>218</td>
<td>14</td>
<td>(1) CMT = 77 (2) AMI = 70 (3) CMT +AMI = 71</td>
<td>Headache Index(0-70): (1) Pre: 18.6 (9.6) Post: 11.1 (8.6) Fl/up: 10.8 (9.6) (2) Pre: 16.5 (8.7) Post: 8.4 (9.0) Fl/up: 12.5 (8.3) (3) Pre: 15.1 (6.5) Post: 8.9 (7.5) Fl/up: 11.3 (7.5) Pre-Post S (0-10): (1) Pre: 5.3 (1.3) Post: 4.3 (1.5) Fl/up: 4.4 (1.7) (2) Pre: 4.6 (1.1) Post: 4.3 (1.6) Fl/up: 4.5 (1.3) (3) Pre: 4.4 (1.1) Post: 4.1 (1.4) Fl/up: 4.3 (1.4)</td>
<td>Posttreatment Fl/up F (20 months): (1) 5.1-3.8 (2) 9.9-8.0 (3) 5.7-4.9</td>
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<tr>
<td>Tuchin et al⁴⁰</td>
<td>Migraine</td>
<td>123</td>
<td>16</td>
<td>(1) CMT = 83 (2) PLA = 40</td>
<td>Pre-Post F (number per month): (1) 7.1 (7.0) →4.1 (6.6)* (2) 7.3 (6.5) →6.9 (6.6) Pre-Post S (0-10): (1) 7.9 (1.4) 6.9 (1.8) (2) 7.9 (1.2) 6.2 (1.7) Pre-Post duration (hours): (1) 22.3 (28.3) 14.8 (19.8)* (2) 22.6 (27.4) 19.8 (17.7)</td>
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Table 1. Continued

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<th>Authors</th>
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<td>(1) 19.8 (21.2) 13.0 (18.2)</td>
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<td>(2) 18.9 (21.2) 15.6 (18.2)</td>
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<td>(1) 21.3 (28.4) 9.8 (12.4)*</td>
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<td>(2) 20.1 (28.4) 16.2 (12.4)</td>
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Total or average 712 7 68

*p = .05.
*p = .01.
*p = .001.

MOB indicates mobilization; STT, soft tissue injury; AMI, amitriptyline; CMT, chiropractic manipulative therapy; MT, manual therapy; PLA, placebo; F, frequency; S, severity; DIS, disability; Fl/up F, follow-up frequency

Treatment of headache by spinal manipulation. It should also be noted that several studies of neck pain treated by spinal manipulation have also reported relief of headaches in large percentages of the subjects.47-48 These studies are not included in this review, although they appear in Aker et al.31

In 2001, Whittingham et al41 reported on the results of a clinical trial of a specific chiropractic manipulative technique (upper cervical recoil adjustment) in patients with chronic CH. Subjects participated in a crossover design, alternating between active adjustments and inactive or "placebo/sham" adjustments. Statistically significant intergroup differences in the first phase for headache frequency, duration, severity, and medication usage were reported in abstract form.41 The researchers also reported on significant improvements in Neck Disability Index49 scores, pressure algometry measurements in the head and neck region, and cervical ranges of motion.

In addition to the systematic reviews of Harwitz et al and Aker et al, which were cited above, four other important systematic reviews on the benefit of spinal manipulation for headache were published toward the end of the decade and into 2001. Pryse-Phillips et al42 published a systematic review of clinical trials of nonpharmacologic management of migraine headaches. They cited Parker et al's two studies37-38 and concluded that "chiropractic manipulations reduced migraine frequency and severity."51 They rated this conclusion as Level I evidence (at least one strong clinical trial) with a class B recommendation (promising).

In 1999, Vernon et al43 published a systematic review of clinical trials of all complementary and alternative therapies for tension-type headaches and CH. The evidence table in this article (with the exception of the studies on migraine headaches) is taken from that study. A total of 286 subjects were included in these studies. The quality scores ranged from 56%-80%, with a mean score of 67.5%. No trial included an exclusively placebo or sham-controlled group, so that the efficacy of spinal manipulation (compared with the "effectiveness") could not be determined.

In patients with tension-type headaches, chiropractic spin manipulation (CSM) was shown to be more effective than no treatment (in a single-treatment session32), as effective as amitriptyline after a 4-week follow-up period,33 and no better than sham therapy plus soft tissue therapy when CSM was combined with soft tissue therapy (in a short course of treatment34).

In patients with CHs, Nilsson's trials33-34 as well as the trial of Jensen et al,1 showed a superior benefit for CSM compared with soft tissue therapy and ice therapy, respectively.

In 2001, McCrory et al44 published a systematic review that was comparable to that of Vernon et al but also included trial for behavioral treatments. With regard to spinal manipulation, they concluded that it was effective in patients with CH. Its effectiveness in patients with tension-type headache was rated as unproven. They found no convincing evidence to support the use of any other physical treatments for patients with these two headache types.

In 2001, Bronfort et al45 published a systematic review an meta-analysis of all trials for the three types of headache discussed in this article. They included two additional trial: The study by Howe et al47 primarily involved subjects with neck pain, but these investigators also reported on their result pertaining to headache symptoms that showed improvement with spinal manipulation. The study by Bitterli et al48 was published in the German literature in 1977 and was not included in Vernon et al's review for that reason.

Bronfort et al reported effect sizes for all these trials, the majority of which were in the range of 0.5 to 1 (fair
moderately strong) in favor of spinal manipulation. The primary exception to this trend was the trial by Bove and Nilsson. Bronfort et al suggested a revised explanation for the negative results from this trial, stating that "SMT [spinal manipulation therapy], when combined with soft tissue therapy, is no better than soft tissue therapy alone for episodic tension-type headache. This conclusion neither supports nor refutes the efficacy of SMT as a separate therapy."45(P463)

CONCLUSION

The data reviewed in this article present a rather strong case for the pathophysiology of headaches of cervical origin and shows that such a cervical component may be present in tension-type and migraine headaches as well as the currently accepted category of CH. This author has argued for this case since the decade began. Now that the specific entity of CH has been accepted by most in the "headache world," the larger premise may come into greater acceptance as well. Although it is hoped that this acceptance will lead to a larger number and higher quality of clinical trials for CSM and headaches, it is also hoped that this will lead to greater use of spinal manipulation in the management of these highly prevalent chronic conditions. As well, we might hope to see such research and clinical use in other headache conditions such as cluster headaches, menstrually related headaches, analgesic rebound headaches, and, especially, pediatric and adolescent headaches.

REFERENCES

33. Nilsson N. A randomized controlled trial of the effect of spinal


