Clinics in Physical Therapy

Temporomandibular Disorders

Second Edition

Edited by

Steven L. Kraus

Temporomandibular Disorders

Second Edition

Edited by

Steven L. Kraus, P.T., O.C.S.

Instructor
Division of Physical Therapy
Department of Rehabilitation Medicine
Emory University School of Medicine
Private Practice
Physiotherapy Associates
Atlanta, Georgia

CHURCHILL LIVINGSTONE
New York, Edinburgh, London, Madrid, Melbourne, Tokyo

11 Cervical Spine Influences on the Management of TMD

Steven L. Kraus

Management of the symptomatic and dysfunctional TMD patient requires an understanding of the cervical spine that is not commonly realized by many patients, health professionals, and third party payers. The clinician who can differentiate signs and symptoms of cervical spine involvement from the signs and symptoms of TMD and/or masticatory muscle hyperactivity (MMH) will be less likely to make an error in determining the primary source(s) of dysfunction. Chapter objectives include a discussion on:

1. Prevalence of symptoms associated with cervical spine involvement.
2. Local, distal, and cephalic symptoms originating from cervical spine involvement.
3. Mechanisms by which the cervical spine may influence the mandible. Effects of the cervical spine upon mandibular position and mobility may influence the attending dentist to alter the sequence of treatment to include physical therapy treatment of the cervical spine prior to, during, and/or following surgical or nonsurgical intervention for TMD and or MMH.
4. The dilemma of diagnosing. Patients, clinicians, and third party payers often place a great deal of importance on the diagnosis. Unfortunately, they assume that the diagnosis is correct and is the source of the symptoms, which may lead patients to say; "I now know what is wrong with me," clinicians to say; "I now know what to treat," and third party payers to say; "I now know how much it will cost and how long it will take." If the diagnosis is important, then hopefully it is the correct diagnosis.

5. Overall management of cervical spine involvement from a physical therapist perspective.

This chapter does not intend to question the expertise of the medical, dental, and psychological professions in diagnosing and managing the symptomatic and dysfunctional patient. However, in an attempt to discuss reliability and validity issues surrounding diagnosis and management of the cervical spine, the reader will become aware of inconsistencies. This chapter attempts to place in better perspective the essential role of the physical therapist, as well as the medical and dental professions, in the team approach to patient care.

PREVALENCE OF CERVICAL SPINE INVOLVEMENT

Reliable epidemiologic data on the incidence and prevalence of neck pain is lacking, as most studies have been on low back pain. Throughout this chapter, various studies on low back pain or spinal pain are cited. Based upon these studies, inferences to the cervical spine will be postulated.

It has been estimated that some 40 to 50 percent of the general population will experience neck pain with limitation of mobility at some time during their lives.[1] Although most patients improve with time, as many as one-third of them may continue to have moderate or severe pain that interferes with the patient's life-style 15 years later.[2] The etiology of neck pain, unlike other musculoskeletal conditions, is multifactorial. This spectrum emphasizes the frequency of symptoms often originating from the cervical spine, which can occur from but not be limited to the following events and syndromes.

Motor Vehicle Accidents

Of the 3,800,000 rear-end collisions that occur annually in the United States, relatively few result in death or quadriplegias.[3] The majority of motor vehicle accidents (MVA) result in neck and shoulder pain secondary to soft tissue injury without objective neural or osseous spinal involvement.[4] At speeds of less than 15 mph a forewarned driver may escape significant injury caused by being hit from behind by bracing himself against the steering wheel.[4] In rear-end collisions that exceed 20 mph, however, the unrestrained driver may experience facet joint loading and excessive stretching to the anterior cervical tissues.[4] In either case, rebound cervical flexion may occur with reflex contraction of the neck muscles.[4]

Statistics on the prevalence of cervical spine related symptoms as a result of MVA reveal an ever increasing patient population. Canadian MVA statistics in 1986 revealed an increase of 15 percent in bodily injuries as opposed to a 4 percent decline in deaths over the previous 3 years.[5] In addition to these "new" injuries, one must take into account previous MVA in which the symptoms have not resolved. According to Ameis,[6] 15 percent of MVA victims fail to

recover full function while 40 to 70 percent have some mild to moderate symptoms persist. It is reported that 50 percent or more of patients after injury may experience symptoms 5 to 10 years after settlement of litigation.[7,8]

Repetitive Motion Injury

Repetitive motion injuries, also known as cumulative trauma disorders, repetitive strain injuries, or overuse injuries, occur in various professions as well as during sporting and recreational activities. Repetitive motions place individuals at a higher risk of developing musculoskeletal problems due to the duration and position in which they are performed.[9,10] Hagberg and Wegman,[11] following their review of 21 articles, state that there is an association between occupation and diseases of the shoulder and neck that suggest that "highly repetitive shoulder contractions, static contractions, and work at shoulder level are hazardous exposure factors." Several authors report correlation between psychosocial factors and musculoskeletal disorders.[12,13] Anxiety, nervousness, and mental strain are suggested to increase static muscle activity and provoke pain.[12,13]

Repetitive motion injury affects as many as 70 million Americans, according to a study by the National Institute for Occupational Safety and Health.[14] Examples include the use of the hand and/or wrist, elbow, shoulder, and neck while using a computer terminal,[14] working on an assembly line,[14] long distance driving to and from work, or professional and amateur musical instrument playing.[9]

Musculoskeletal pain and discomfort among dentists was investigated based on questionnaires distributed in 1987 and again 2.5 years later in 1990.[15] The results of this prospective study showed the prevalence of musculoskeletal pain and discomfort had increased between 1987 and 1990. From the answers in 1990 it was shown that frequency of headaches and pain and discomfort in the neck, shoulders, and/or lower back was relatively high. Such symptoms were believed to be associated with work environment and work tasks (repetitive motion injury).

Fibromyalgia

Fibromyalgia, a controversial diagnostic syndrome, has been estimated by Goldenberg to afflict between 3 to 6 million patients.[16] Fibromyalgia has been referred to by different names over the years, such as rheumatic pain modulation syndrome,[17] nonarticular rheumatism,[17] and interstitial myofibrositis,[18] to mention a few. Twenty percent of outpatients who see a rheumatologist are diagnosed with fibromyalgia.[19] Myofascial trigger points in a population of patients with spinal pain represent a primary source for clinical symptoms, according to Travell and Simons.[20–22] Differentiation between fibromyalgia and myofascial pain syndrome will be discussed below.

Cervicogenic Headache and Other Cephalic Symptoms

The prevalence of cervical spine symptoms would be greater if past epidemiologic studies included symptoms not commonly associated with the cervical spine. This list of ignored symptoms related to cervical spine involvement includes cervicogenic headaches and dizziness. In the field of headache research, the cervical spine has been a largely unexplored frontier until recent years. Bogduk states,[23]

> Although disorders of the neck are acknowledged as possible causes of headache, there are no established guidelines whereby, on clinical grounds, cervical headache can be reliably differentiated from other forms of headache. . . . It is possible that 'cervical headache' has been avoided as a diagnosis by neurologists dealing with headache problems, and its actual incidence underestimated.

Summary

If the "numbers" are close to being accurate, the prevalence of symptoms associated with cervical spine involvement is indeed high. A patient who is seeking help for their TMD and/or MMH-related symptoms will more than likely have a complaint associated with a cervical spine involvement that may require further investigation.

CERVICAL SPINE AS A SOURCE OF LOCAL AND DISTAL SYMPTOMS

Cervical spine tissues can be the source of local and distal (upper extremity) symptoms. Empirical evidence suggests that if certain tissues are found to be involved, they suggest or indicate the cervical spine as the source of symptoms and limitation in function. Some clinicians may be biased toward one tissue based upon their educational background and what they are capable of evaluating and treating. Such a bias will prevent the clinician from recognizing not only other tissues that might be involved, but also the complexity of the patient's clinical presentation.

Muscle

It is well documented that muscles of the cervical spine and shoulder girdle areas can be a major source of local and referred symptoms into the upper extremities.[24–29] Palpation examination is a procedure used to determine the presence of muscular involvement.[30] Patients may have full pain-free range of motion, normal strength, and normal neurologic reactions but still have mus-

cular involvement.[30] A patient may complain of referred pain in the absence of pain at the primary site of origin. Silent primary pain is discussed in the section on History.

Muscular involvement is commonly identified as either fibromyalgia or myofascial pain syndrome. The criteria to diagnose fibromyalgia have varied as much as the name. Today's general criteria for diagnosing fibromyalgia include multiple tender points (four or more), diffuse musculoskeletal aches, pain, stiffness at many sites, and easy fatigability.[21,31,32] Fibromyalgia is often confused with myofascial pain syndrome.[22] The hallmark of myofascial pain syndrome is a trigger point that is located in one or two muscle(s) as a taut band of exquisite tenderness in response to manual pressure, and a characteristic pattern of referred pain.[22,24] Tender points associated with fibromyalgia are areas of tenderness that may or may not be in muscle tissue, do not have palpable taut bands, and do not refer pain to adjacent areas.[21]

Both fibromyalgia and myofascial pain syndrome cause pain and tenderness and exhibit similar histologic changes, and both conditions are noted to exhibit increase pain with activity.[33] Simons and others suggest that multiple or generalized myofascial trigger points or tender points are in the spectrum of fibromyalgia.[25–28,32] No attempt to differentiate between fibromyalgia or myofascial pain syndrome will be made in this chapter. Managing either fibromyalgia or myofascial trigger points by suggested procedures and/or techniques covered in the section on management should yield satisfactory results (see also Ch. 13). For the rest of this chapter, the term "muscular involvement" will be used to indicate symptoms associated with either fibromyalgia or myofascial pain syndrome.

Facet Joint

A significant source of pain associated with cervical spine involvement originates from facet joints, also known as zygapophyseal joints. Each vertebral segment of the cervical spine has a pair of facet joints starting from occiput and C1 (atlantooccipital) through C6/C7. With the exception of occiput/C1 and C1/C2 (atlantoaxial) facet joints, the remaining paired facets from C2/C3 through C6/C7 are located laterally and posteriorly to each side of the vertebral body (Fig. 11-1). Another set of joints located only in the cervical spine are the joints of von Luschka, also known as the lateral interbody joints (Fig. 11-1). These joints extend upward from the lateral margins of the upper surfaces of the vertebral bodies of the lower five cervical vertebrae.[34] Only the facet joints will be discussed as a primary source for joint pain, mainly because of available documentation. For further information about the joints of von Luschka the reader is referred to the references.[35]

Research into lumbar spinal pain syndromes has shown that the lumbar facet joints can be a major source of low back pain, referred pain into the lower extremity, and even neurologic signs.[36–39] Even though Wyke and co-workers describe in detail the articular neurology of the facet joints in the cervical

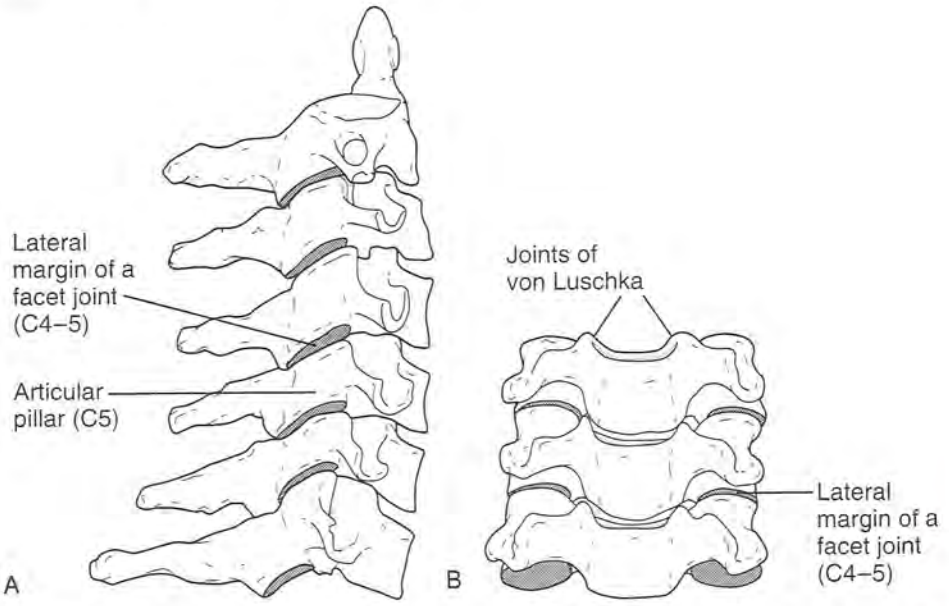


Fig. 11-1. Typical cervical vertebrae. **(A)** The 2nd to 7th cervical vertebrae viewed from the right side. **(B)** The 3rd, 4th and 5th vertebrae viewed from in front.

spine,[40,41] little attention has been given to the cervical spine facet joints as a source of neck and upper arm pain until recently (Fig. 11–2).[42] Dwyer and co-workers[43] mapped out pain patterns evoked by stimulation of normal cervical facet joints by injecting about 1 ml of contrast medium into the facet joint, distending the joint capsule (see Ch. 12, Fig. 12-2). As a follow-up to this study, Aprill and co-workers[44] tested the predictive value of segmental pain charts in patients with suspected cervical facet joint pain. This study concluded that pain charts used for locating the segmental location of a symptomatic joint were accurate in each patient on the basis of the alleviation of pain with diagnostic joint blocks.[44] Inflammation and/or edema is a likely cause of facet joint pain and limited movement since the facet joints are true synovial joints. To identify inflammation and edema occurring at a cervical facet joint, a manual procedure of palpating the lateral margin of the facet joint (Fig. 11-1) will be mentioned in the physical examination. Jull and co-workers[45] evaluated one manual therapist's ability to identify cervical facet joint syndrome in 20 patients, all of whom had complained of chronic neck pain or headaches for at least a year. The authors concluded that manual examination by a trained manual therapist is as accurate as radiologically controlled blocks in the diagnosis of symptomatic cervical facet joints.[45]

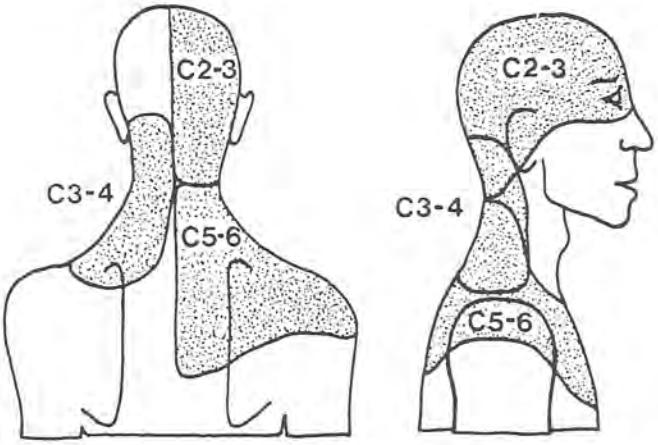


Fig. 11-2. Facet joint pain distribution. Distribution of presenting symptoms in the patients with positive responses correlated with the offending level revealed by the diagnostic blocks to the facet joints.

Neck pain and limited neck movement due to facet joint involvement have also been related to a torn or trapped menisci of the facet joint.[46–49] The form, incidence, and disposition of menisci in facet joints have been studied by several authors.[50,51] The menisci is a peripheral ring of tissue covering from a small fraction up to 50 percent of the articular cartilage of the facet joint, depending upon the vertebral level and age of the subject.[52] The inner margin of the meniscus contains dense collagenous tissue whereas the outer margin contains collagen and fatty tissue attaching firmly to the facet joint capsule.[50] The primary role of the menisci in the cervical facet joints is said to be providing uniform distribution of pressure.[53] Other secondary functions, including compensation for joint incongruency, preservation of articular facet edges, and occupation of cavities or spaces left open during motion, have been suggested.[47] The presence of menisci invites speculation about their causal role in pain and limitation of neck movement if torn or trapped.[54,55] Clinically, certain neck and distal symptoms can be resolved if proper manual techniques are applied to the restricted facet joint(s) (see Fig. 11-22H).

Peripheral Nerve

Peripheral entrapment neuropathy is another source of neck an distal symptoms related to cervical spine involvement. Kopell and Thompson define the term *entrapment neuropathy* as "a region of localized injury and inflammation in a peripheral nerve that is caused by mechanical irritation from some impinging anatomical neighbor."[56] A peripheral entrapment neuropathy occurs lateral to the intervertebral foramen. An entrapment neuropathy occurring in-

side the intervertebral foramen and medially will be considered a central entrapment neuropathy involving the nerve root. A peripheral or central entrapment occurs because the nerve is unable to move in relation to neighboring structures. There are a number of potential sites where a peripheral nerve can become entrapped.

Brachial Plexus

A common peripheral entrapment neuropathy associated with the cervical spine occurs at the interscalene triangle involving the brachial plexus. The interscalene triangle boundaries are the first rib inferiorly, the scalene medius muscle posterolaterally, and the scalene anticus muscle anteromedially.[57] The dimensions of the interscalene are not static. Movement or positioning of the cervical spine can affect the dimensions, especially the anteroposterior dimensions. Scalene muscle hypertrophy linked to physical demands of certain occupations and recreational activities may also alter interscalene dimensions.[57] Contents of the interscalene triangle that are potentially at risk of impingement include the brachial plexus and the subclavian artery. The majority of symptoms are related to neurogenic involvement but the most serious, yet rare, are vascular.[58] To some clinicians an entrapment occurring at the interscalene triangle may be considered a part of the thoracic outlet syndrome (TOS), which would not be an inaccurate assumption. However, no attempt will be made by this author to discuss TOS because of the confusion and contradiction in the literature that underscores our poor understanding of this syndrome.[59]

At the point of nerve entrapment either compression or friction can cause a reaction in a nerve.[60] The degree of such a reaction is proportional to the intensity and duration of the entrapment pressure.[61] Since the connective tissue components of nerves are innervated by pain fibers, inflammation may result in pain reactivity.[61] Based upon the amount of compression, nerve injury can range from the mildest degree of injury involving interruption of conductivity (known as demyelination),[61,62] to severe injury involving destruction of the nerve components (known as axonal degeneration).[61,63] Clinically, most nerve injuries at the interscalene triangle are related to inflammation and mild nerve injury giving rise to the patient's neck and upper extremity symptoms. Once inflammation and compression is removed from the nerve, symptoms and function are restored.[62]

Greater Occipital Nerve

Although the greater occipital nerve (GON) contributes to cephalic symptoms it will be discussed in this section, since it is a peripheral nerve in the cervical spine. The greater occipital nerve is the main sensory nerve in the

occipital area, deriving most of its fibers from the C2 nerve root.[64] Involvement of the C2 nerve root or GON has been collectively referred to as "occipital neuralgia."[65] C2 or GON compression or irritation has been attributed to various causes, ranging from post-traumatic lesions or cervical degenerative arthrosis, to muscle spasm in the upper cervical spine.[66,67] Diagnosis often depends on pain location and the exclusion of other causes. Jansen and coworkers state; "There are no radiological or electrophysiological maneuvers for demonstrating compression or irritation of the C2 root."[68] Unlike the C2 nerve root, the GON is superficial, so compression or irritation may be more verifiable. For over 40 years, occipital nerve zone tenderness has been known to be a feature of GON entrapment.[67,68] Bovim and coworkers examined 20 unselected adult autopsy cases without a history of headache according to the hospital files.[70] They found variations in the anatomic relation of the GON to muscles and tendons. Such variations may contribute to predisposition to entrapment in certain individuals. On the basis of this study, it can be inferred that the semispinalis muscle of the neck most often is penetrated by the GON.[70] Nerve penetration to the trapezius was also a frequent finding, although a greater variation in the course of the nerve was present.[70] Bovin and co-workers indicate that these findings contrast with some classic anatomy books that describe the GON as circumscribing these muscles to reach the occipital insertion of the trapezius.[70] It is speculated that entrapment of the nerve may occur from an increase in muscle tone (guarding, spasm, etc.) of any one or two of the muscles names above.[70] Symptoms associated with GON entrapment may be relieved by physical therapy management.

Location of symptoms associated with GON entrapment would be in the area innervated by cutaneous branches of the GON. Cutaneous branches and their innervation of the GON include:[64]

Medial branch. Innervates the occipital skin.

Lateral branch. Innervates the region above the mastoid process and behind the pinna (the projecting part of the ear lying outside of the head).

Intermediate branches. Run rostrally and ventrally across the top of the skull as far as the coronal suture. At the coronal suture, the GON is traditionally regarded as communicating with terminal branches of the supraorbital nerve.

Based upon the cutaneous innervation, complaints may be located in the occipital area, the top of their skull and/or around or in the ear or TMJ. A burning dysesthesia, described by the patient as "my hair is on fire," may also be a clinical presentation of GON entrapment.[4]

During manual palpation over the GON, certain symptoms may occur that cannot be explained by the GON's cutaneous innervation. Unilateral hemicranial, fronto-ocular or dysaesthesias located in areas innervated by the trigeminal nerve may still be attributed to GON and or C2 involvement.[68,71–73] Symptoms located in these areas are best explained by the convergence of

cervical and trigeminal afferents on common neurons in the trigemino-cervical nucleus. This neuroanatomic connection will be described in more detail in the section on Cervical Spine as a Source of Cephalic Symptoms.

Nerve Root

Nerve root involvement will be referred to as *radiculopathy*. Radiculopathy is a central nerve root entrapment of any portion of a nerve root from the intervertebral foramen and medially. As with peripheral entrapment neuropathy, a central entrapment neuropathy can occur by mechanical irritation or compression from some impinging anatomic neighbor. *Stenosis* is a frequent term meaning a narrowing or stricture. Stenosis can involve any anatomic opening, one of which can be the intervertebral foramen. Various causes have been attributed to stenosis of the intervertebral foramen possibly resulting in radiculopathy.[74] Stenosis of the intervertebral foramen has often been attributed to pathologic change of the intervertebral disc.[75] Symptoms of radiculopathy may range from localized neck symptoms to referral of symptoms into the shoulder and upper extremity. Neurologic assessment may show varying degrees of neurologic deficits.

Intervertebral Disc

Symptoms associated with discogenic involvement resulted from the discovery by Mixter and Barr that the nucleus pulposus in the lumbar discs can "herniate" through the annular fibers to occlude the nerve root within the intervertebral foramen.[76] In the cervical spine, a herniated nucleus pulposus as a *direct* cause of radiculopathy is not as likely as it is in the lumbar spine.[77] The nucleus pulposus of cervical discs are initially very small in the child and, in adults, have only a relatively brief existence as a soft central gel.[78–81] Horizontal fissuring of the annulus begins toward the end of the first decade, usually accompanied by an associated loss in disc height.[81] Horizontal fissuring is universal in the adult cervical disc, completely dividing the posterior two-thirds of the disc in late adult life.[78,82] Progressive and slow loss of nuclear material continues with the central gel converting to firm fibrocartilage.[83] In the adult cervical spine the nucleus pulposus is usually absent at most levels. In many adult discs, the anterior annulus is the only intact part of the disc.[81] The progression of cervical disc degeneration is a relatively linear function of age in that 90 percent of the population is afflicted by age 60 and 50 percent severely so.[82] Thus *nuclear* protrusion and herniation, which has long been a feature of descriptions of lumbar pathology, is not a major problem in the cervical spine.[81,83]

The end product of this nuclear pulposus degeneration is referred to as *cervical spondylosis*. Cervical spondylosis as defined by Parke "is not a single temporal or pathologic entity, but the result of a concatenation of degenerative events of which the initial lesion is the deterioration of the intervertebral

disc."[83] The end product of cervical spondylosis can be radiculopathy or myelopathy. The evaluation for cervical spondylosis can be through x-rays, identifying disc space narrowing, osteophytosis of both the facet joints and joints of von Luschka, and hypertrophy of the ligamentum falvum.[83] The highest incidence of cervical spondylosis is at the level of C5–6.[84] A more in-depth discussion of cervical spondylosis is provided in Chapter 12.

Radiculopathy secondary to cervical spondylosis may require a different physical therapy approach to management. Depending on other clinical and paraclinical findings (Ch. 12), additional medical involvement may be in order. Fortunately, degeneration of the cervical disc is insidious and most often asymptomatic unless suddenly exacerbated by a traumatic stress[83] or dramatic changes in occupational or recreational activities. Studies on neck pain and disc degeneration seen in imaging studies have shown that there is only a weak correlation between symptoms and degeneration.[85,86] A host of medical diagnoses such as disc degeneration, bone spurs, degenerative arthritis, arthrosis, etc., fall under the spectrum of cervical spondylosis. This host of cervical spondylitic diagnoses should not misdirect the clinician's attention away from addressing the other tissues previously covered (muscle, facet joint, peripheral nerve) that could instead be the primary source of the patient's neck and distal symptoms.

In conclusion, cervical radiculopathy appears to be related to degenerative changes secondary to loss of nuclear material rather than from a *direct* herniation of a soft nucleus pulposus. A major study has demonstrated that spinal pain is rarely derived from structural abnormalities such as a herniated disc or stenosis; such conditions exist in only 1 percent of the target population.[87] It remains to be seen whether or not the disc itself can be a *significant* source of symptoms. In a recent study, Mendel and co-workers[88] confirmed previous reports that no nerves are to be found in the nucleus pulposus. Nerves were found throughout the annulus, however, and provide further evidence that human cervical discs are supplied with both nerve fibers and mechanoreceptors.[88,89] It has been suggested that in severe traumatic cases that did not result in death, pain originating from the nerve endings located in the annulus of the disc is possible.[89] Hence, the disc may be implicated as a source of a patient's symptoms. Further discussion on the disc as a source of pain will occur in the section on Management by the Use of Cervical Collars.

Summary

This section addressed key tissues that can be the cause of the patient's neck, shoulder, and upper extremity symptoms. All tissue components can be managed through physical therapy intervention. Depending upon the mechanism of injury, chronicity, patient's irritability, and degree of degeneration, medical management may be necessary (see Ch. 12, 13). Clinicians will recognize that a pure entity of tissue involvement is not common; instead, the patient's symptoms are often of diverse etiologies.

CERVICAL SPINE AS A SOURCE OF CEPHALIC SYMPTOMS

It is evident that cephalic symptoms can be due to serious disease or pathology and require a thorough neurology and often otolaryngology examination. It is just as evident that the cervical spine has not been sufficiently recognized as being a source for nondiseased or pathologic cephalic symptoms.

Neuroanatomy

The following is the documented neuroanatomic pathway explaining how nociceptive activity originating in the cervical spine tissues is perceived by the patient as symptoms in the head, face, and jaw areas (Fig. 11-3).[90]

The spinal nucleus of the trigeminal nerve consists of three parts: pars oralis, par interpolaris, and pars caudalis. The pars caudalis extends cau-

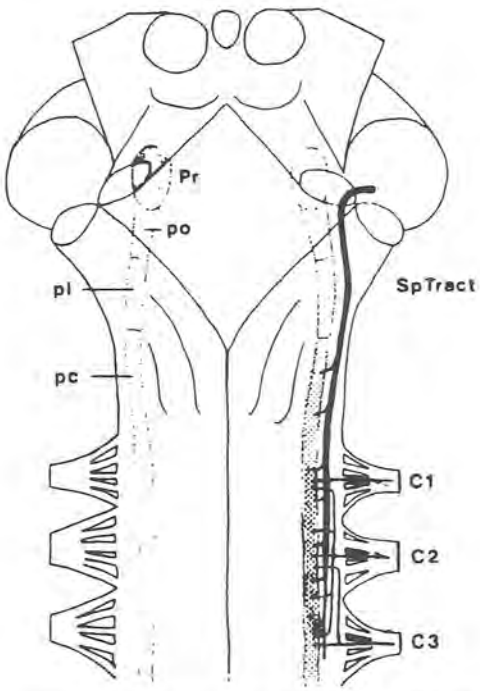


Fig. 11-3. A posterior view of the brainstem illustrating the disposition of nuclei of the trigeminal nerve. The principle nucleus of the trigeminal nerve (Pr) lies at the level of the pons. Below it extends the spinal nucleus of the trigeminal nerve which is subdivided into pars oralis (po), pars interpolaris (pi), and pars caudalis (pc). On the right, the continuity between the pars caudalis and the spinal grey matter is indicated by the shading. This column of grey matter receives afferents from the spinal tract of the trigeminal nerve (Sp Tract) and from the C1, C2, and C3 nerve roots.

dally to merge with the grey matter of the spinal cord. The spinal tract of the trigeminal nerve descends to the level of at least C3 level and possible as far as the C4 level. Fibers from the spinal tract terminate in the pars caudalis and in the upper three segments of the spinal cord. In the spinal cord, termination of the spinal tract of the trigeminal nerve overlaps those of the upper cervical nerves.

From the above description, Bogduk summarizes[90]: ". . . terminals of the trigeminal nerve and the upper three cervical nerves ramify in a continuous column of grey matter formed by the pars caudalis of the spinal nucleus of the trigeminal nerve and the dorsal horns of the upper three cervical segments." Bogduk states that this region of grey matter can legitimately be viewed as a single or combined nucleus, for which he prefers to use the term *trigeminocervical nucleus*.[90] Thus, the anatomic basis for cervical headache appears to be the convergence of trigeminal and cervical afferents in the trigeminocervical nucleus.[90–95] Trigeminocervical nucleus incorporates the essential central nervous structures responsible for the transmission of pain. The trigeminocervical nucleus receiving afferents from the trigeminal and upper cervical nerves is viewed by Bogduk as the nociceptive nucleus for the entire head and upper neck.[90] Essentially, nociceptive information from cervical spine tissues is transmitted to the trigeminocervical nucleus, which in turn gives the patient the perception of symptoms in the head, face, and jaw areas.[90–95] (Figs. 11-4, 11-

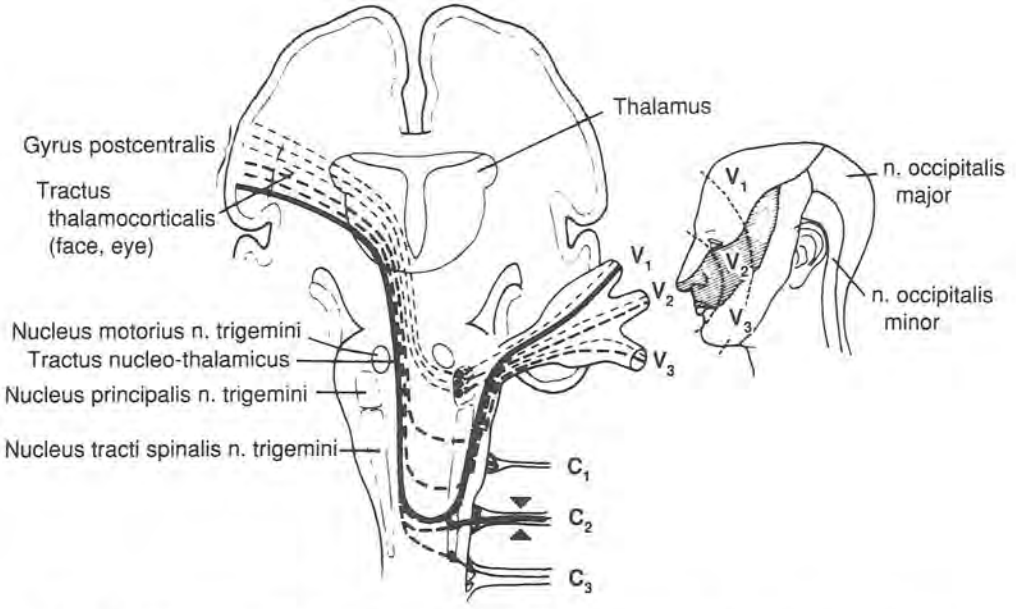


Fig. 11-4. Transference of pain from C2 innervated tissues or greator occipital nerve or C2 nerve root compression into the fronto-ocular region—a hypothesis. (From Jansen et al.,[68] as modified from Jansen and Spoerri,[380] with permission.)

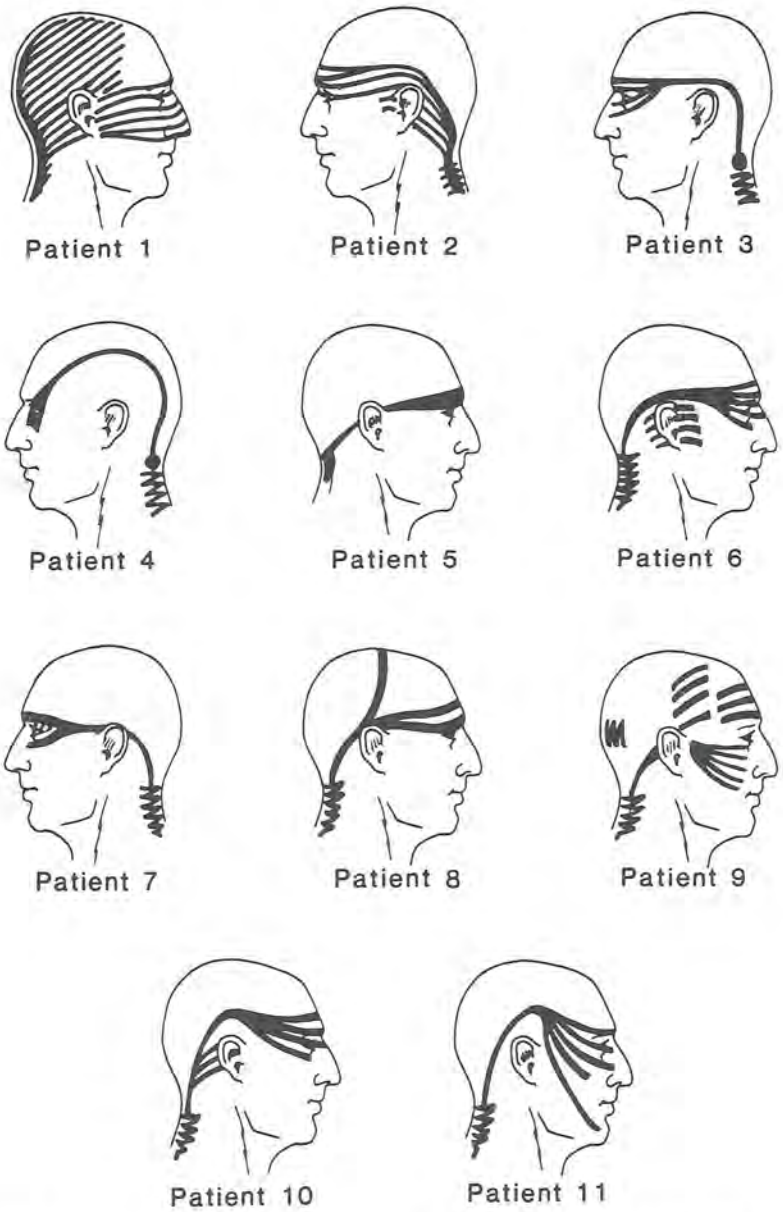


Fig. 11-5. Cervicogenic headache pattern in 11 female patients. See Table 11-1 for explanation. (From Fredriksen et al.,[109] with permission.)

5, Table 11-1). Those tissues with sensory innervation from the upper three cervical nerves are:[90]

C1 Sensory Distribution
- Suboccipital tissues and muscles
- Atlantoccipital and atlantoaxial facets joints
- Paramedian dura of the posterior cranial fossa and dura adjacent to the condylar canal
- Upper prevertebral muscles (longus capitis and cervicis and the rectus capitis anterior and lateralis)

C2 Sensory Innervation
- Skin of the occiput
- Upper posterior neck muscles; semispinalis capitis, longissimus capitis and splenius capitis, the sternocleidomastoid, trapezius, and prevertebral muscles
- Atlantoaxial facet joint

Table 11-1. Cervicogenic Headache Pattern in 11 Female Patients[a,b]

Description	No. of Cases
Accompanying symptoms and signs	
Nausea	7
Vomiting	6
Piloerection	7
Photophobia	5
Phonophobia	10
Reduced hearing, subjectively	2
Dizziness	9
Tinnitus	2
Irritability	10
Discomfort in the throat	6
Rhinorrhea, symptomatic side	4
Tearing, symptomatic side	4
Blurred vision, symptomatic side	9
Redness of eye, symptomatic side	4
Edema of eyelids, symptomatic side	8
Head movement leading to precipitated attacks[c]	
Turning of the head	8
Bending forwards	5
Bending backwards	5
Location of "pressure" points[d]	
Midway between external occipital process and mastoid process	8
C_2 area—that is, behind and just below the mastoid process	8
Transverse processes of the C_3/C_4	8
Muscle insertion of the external occipital protuberance	1

[a] See Figure 11-5 for diagrams.

[b] Mean age of 43 years. Durations of headaches were: 5 patients, <5 years; 2 patients, 5–10 years; 1 patient, 10–15 years; 2 patients, 15–20 years; 1 patient, >20 years. The pain was very constant in duration when considering the single patient.

[c] A total of ten patients could precipitate attacks by one head movement or another.

[d] In a total of 10 patients attacks could be provoked from the pressure points. (Modified from Fredriksen et al.,[109] with permission.)

Paramedian dura of the posterior cranial fossa
Lateral walls of the posterior cranial fossa

C3 Sensory Distribution
Multifidus, semispinalis capitis, sternocleidomastoid, trapezius, and prevertebral muscles
Suboccipital skin
C2/3 facet joint
Cervical portion and intracranial branches of the vertebral artery

Numerous studies over the years have confirmed the trigeminocervical connection. Kerr, through electrical stimulation of the rootlets of the C1 dorsal nerve, produced pain in the orbit, frontal region, and vertex.[94] Cyriax injected 4 percent saline into various posterior neck muscles, which produced local and referred pains to the forehead and temporal regions.[96] Campbell and Parsons injected 6 percent saline in the basal occipital, occipitocervical, and C1/2 areas, which produced frontal pain.[97] Feinstein et al.[98] induced forehead pain by stimulating the midline soft tissues between occiput and C1, and occipital pain by stimulating the upper cervical interspinous spaces. Bogduk and Marsland, through electrical stimulation of the C3 dorsal ramus, evoked referred pain to the occiput, mastoid region, and forehead.[99]

Clinical experiments clearly demonstrate the capacity of experimental painful stimuli in the upper neck to produce referred pain in the head.[90] It is therefore proposed that cervical spine involvement of any of the tissues and/or structures innervated by the upper cervical nerves is equally capable of producing referred pain into the head.[90,100]

Cervicogenic Headache

The dental explanation of the etiology of headache is often TMD.[101] The medical explanation of the etiology of migraine headache is often vascular, and of a tension-type headache is often psychosomatic.[102] In 1988 the Ad Hoc Committee on the Classification and Diagnostic Criteria for Headache Disorders recognized the cervical spine as an etiology for headache.[103]

John Edmeads[104] points out that in order for the cervical spine to be a source of headaches, the following three conditions must be obtained:

1. There should be pain-sensitive structures within the neck
2. There should be identifiable pathologic processes or physiologic dysfunctions within the neck capable of serving as an adequate stimulus to the pain receptors in the cervical structures
3. There should be identifiable neurologic pathways and mechanisms through which pain originating in the cervical structures may be referred to the head.

It appears that all of Edmeads' criteria are present for the cervical spine to be a source of headaches. Referring to #2 above, in the absence of disease processes, the role is less clear when understanding dysfunction. Controversy over this point is inevitable because of the difficulty and, at times impossibility, of demonstrating in a reliable and valid way certain types of dysfunction, other than to say "it is clinical opinion." The reader needs to keep this in mind during the upcoming discussion on diagnosis and management. I believe that the ensuing management for cervical spine involvement can be extremely effective in alleviating local, distal, and cephalic symptoms. The reason for such therapeutic responses may not always be apparent.

Several names have been used to incriminate the cervical spine as the source of headache such as "syndrome cervical sympathique posterieur"[105] and "cervical migraine."[106] The term *cervicogenic* was first used by Sjaastad et al. in 1983.[107] The previous two terms used to describe a headache of cervical origin have been replaced with the term *"cervicogenic headache."*[107] "Cervicogenic" only indicates the region, it does not indicate the structure primarily affected.[107] Since 1983, numerous articles have appeared in the literature providing documentation on the existence of a cervicogenic headache.[71,108–117]

The following is a description of the clinical features of a cervicogenic headache:[109]

> Predominately unilateral symptoms (at times bilateral) always on the same side of a non-excruciating, non-continuous character, without cluster pattern. Unilateral symptoms are combined with signs of neck involvement in the form of ipsilateral pain/stiffness and a decrease range of motion of the neck. Variable presence of ipsilateral shoulder or arm pain are also part of the clinical picture. Precipitation of the headache occurs either by neck movement or pressure on certain tender spots on the neck. Tender spots on the neck [as originally described by Sjaastad[107]] are over the C2 nerve root, greater occipital nerve and over the transverse process of C4/5. The attack typically lasts from 1 to 3 days, and the pain free interval (or at least a period with clearly reduced pain) typically varies from 1 to 3 weeks.

The Ad Hoc Committee on the Classification and Diagnostic Criteria for Headache Disorders makes the following comment on cervicogenic headache[103]:

> Cervical headaches are associated with movement abnormalities in cervical intervertebral segments. The disorder may be located in the joints or ligaments. The abnormal movement may occur in any component of intervertebral movement, and is manifest during either active or passive examination of movement.

The radiologic investigative methods (cervical myelography, cerebral angiography, cerebral computed tomography (CT), cervical CT, plain x-rays of the cervical spine, x-rays of the cranium, and x-rays of the paranasal sinuses)

utilized by Fredriksen and co-workers[114] did not offer any clear evidence regarding the etiology of cervicogenic headache. The etiology for cervicogenic headache, as suggested in this chapter, will be any one or combination of muscle, facet joint, and neural tissue (peripheral and or central) involvement.

Cervical spine involvement needs to be considered even when other forms of headache have been implicated. There is no doubt an overlap of diagnostic categories since most headaches are diagnosed by location of symptoms.[117] The cervicogenic headache is not suggested to replace a more popular headache label (i.e., common migraine or tension-type headache) but at the very least, cervical spine involvement should be considered as a primary or secondary feature or epiphenomena. The following are several classifications of the more frequently seen headaches that may be mistaken for headaches originating from the cervical spine. The reader is referred to the references for further information on this vast topic of headache.[118]

Migraine

Migraine has been considered a disorder of cerebral or extracranial blood vessels, blood constituents, or possibly a primary disorder of the brain itself.[119] Semantically, the term *migraine* derives from the Latin *hemicrania* which ipso facto should designate a unilateral headache.[113]

Classic Migraine. *Premonitory* symptoms apply to changes in mood (i.e., elation, hyperactivity, depression, and/or irritability) which occur up to 24 hours before the attack.[113] *Prodromal* symptoms such as the visual aura, especially the scintillating scotoma, are part of the migraine attack and usually last less than 30 minutes, followed by headache. Nausea and vomiting may also occur with the aura.[113,120]

The certainty of diagnosing a classic migraine usually exceeds that with which one of common migraine can be made.[113] Walters[121] indicated that the symptoms most frequently diagnostic of classic migraine (prodrome, unilateral onset, and nausea) occur together only a little more frequently than by chance, thereby making the diagnosis of classic migraine questionable. Bakal and Kaganov[122] demonstrated that muscle contraction and migraine headache patients were equally familiar with head pain locations thought to be diagnostic of each group. Migraine patients also experienced muscle contraction symptoms more frequently than migraine symptoms according to Bakal and Kaganov.[122] Olesen[123] raised the question, "Is the muscle tenderness which develops in association with a migraine attack caused by the migraine pain (reflex spasm) or is it a primary event causing the migraine?" Kaganov et al.[124] concluded that "There was a strong trend for increasingly problematic/severe headache to be accompanied by an increasing presence of both muscle contraction and migraine symptoms." The question still remains unanswered as to whether or not muscle contractions precipitate a migraine like attack.

Common Migraine. This headache is differentiated from classic migraine by the absence of a visual aura.[113] Common migraine can be easily confused

with cervicogenic headache because of the lack of typical visual disturbances and the fairly long-lasting nature of attacks.[71] There are no scientifically acceptable diagnostic tests for common migraine, so an admixture of cervicogenic headaches could be overlooked and treated as though they were common migraine.[113]

Tension-Type Headache

The clinical features describing cervicogenic headache are close to those of tension-type headache. A tension-type headache is based on the criteria that the headache appears almost daily as a constant tight pressing or bandlike sensation in the occipital, temporal, and/or frontal areas.[118] Tension headache is bilateral but not necessarily symmetrical. The patient cannot provoke a tension headache by head movements and there is no painful limitation of neck mobility.[102,125] The cause is believed to be emotional stress. Cervicogenic headaches that involve the muscles of the cervical spine could also be triggered by emotional stress. The possibility that a patient could experience a cervicogenic headache bilaterally, ("unilaterality on both sides") has been suggested.[126] As was the case with common migraine, an admixture of symptoms originating from the cervical spine could be overlooked and treated as though the symptoms were all caused by a tension-type headache.

Post-Traumatic Headache

The organic source of post-traumatic headache has been thought to be localized in the brain, in the scalp, or in the soft tissue of the neck.[127] Head trauma invariably implies some traumatic impact to the neck. As discussed in the section that follows on dizziness, various visual disturbances and ear symptoms following head trauma could be part of a cervical spine-related headache.[127,128] It is suggested that therapy directed to the cervical spine can influence headaches that are of post-traumatic origin.[127,128]

Summary

In the following quote by Frykholm (1983),[129] if one substitutes "cervicogenic headache" for "cervical migraine" many of the preceding chapter concepts about this type of headache classification are well summarized:

> 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 inadequate treatment and have often become neurotic and drug dependent.

Dizziness

Dizziness "is a general term, implying only the sense of a disturbed relationship to the space outside oneself."[130] Vertigo is the illusion of motion or position, either of the patient or the patient's environment, and has been used more specifically to connote rotation.[131] Both dizziness and vertigo refer to a false sensation of motion of the body and will be considered synonomously in this chapter.[132] Dizziness often is described by the patient as unsteadiness, imbalance, floating, light headedness, spinning and as those features of ataxia noted subjectively.[130–132] Dizziness results from discrepancy or conflict in positional information from the cerebellum, vestibular nuclei, ears, eyes, proprioceptors, and other peripheral receptors.[90,132] Uemura and co-workers[133] state that "if a patient can stand on either leg with eyes closed, he may be regarded as essentially normal, and other vestibular tests are not necessary." Troost[134,135] lists the cause of vertigo as either a peripheral or central disorder. A brief discussion of peripheral disorder will aide the therapist to appreciate and respond accordingly to patients who may get dizzy when manual therapy to the cervical spine is administered. Pertinent to this discussion are central disorders contributing to dizziness in which one of the various etiologies can be cervical spine involvement.

Peripheral Disorders

Benign paroxysmal positional vertigo (BPPV) is the most common diagnosis of the peripheral disorders.[136] BPPV bears discussion since it may often be confused with cervical vertigo. Common clinical findings of BPPV are vertigo and nystagmus when the patient is moved from a sitting to a supine position with the head over the edge of the table and the neck in 30 to 45° of extension and 30 to 45° of rotation, referred to as the Hallpike-Dix maneuver or Nylen-Barany test.[137] Vertigo usually appears after a 1- to 5-second latency period once the patient's head has been placed in the provoking position previously described. In order to observe the nystagmus, the patient must be lowered quickly. Vertigo along with nystagmus increase in intensity and disappear in 30 to 60 seconds while the provoking position is maintained.[138] The cause is usually dysfunction of the vestibular end organ—semicircular canals, utricle, and saccule.[138] Treatment is based on two theories—"cupuloithiasis" and "canalithiasis."[137] Cupulolithiasis theory proposes that degenerative debris from the utricle fall onto the cupula of the posterior canal, making the ampulla gravity-sensitive.[139] Canalithiasis theory proposes that the degenerative debris is not adherent to the cupula of the posterior canal but instead is free-floating in the endolymph.[140] A specific physical therapy exercise program has become a common treatment for BPPV.[136,141–143] The exercise treatment approach purports to dislodge embedded debris in the canals or to habituate the central nervous system (CNS) response to movement-provoked vertigo.[136]

Central Disorder

The most common central disorder involves the vestibular nuclei.[90] The vestibular nuclei are often involved through ischemic processes,[144] or through disturbances of the tonic neck reflexes often referred to as cervical vertigo or reflex vertigo.[134,145]

Ischemic Vertigo. Ischemic vertigo involves the basilar artery or the vertebral artery. Vertebrobasilar artery occlusion can be the first sign of vertebrobasilar insufficiency.[144] Many additional signs and symptoms (nausea, vomiting, faintness, ataxia, and blurred vision) so often accompany dizziness of any origin that they cannot be used as reliable indicators of ischemic vertigo.[144] However, the presence of diplopia, drop-attacks, dysarthria and/or dysphagia in association with dizziness is *highly indicative* of vertebrobasilar occlusion.[146] The serious consequences of ischemic vertigo are obvious and cannot be overlooked. If in doubt, the clinician must have the patient consult with a neurologist or otolaryngologist. Ischemic vertigo can be caused by intrinsic or extrinsic factors that can decrease blood flow to the vestibular nuclei.[90] The clinician who performs manual procedures to the cervical spine must be alert to both intrinsic and extrinsic factors. Emphasis will be on extrinsic factors since the involvement of extrinsic factors can be more readily tested by the physical therapist and, if positive, may become a contraindication for manual therapy to the cervical spine.

Intrinsic factors may involve such conditions as atherosclerosis of the vertebrobasilar system. If this is suspected, secondary to the *highly indicative* symptoms associated with vertebrobasilar occlusion as mentioned above, a referral to medical personnel is forthright.

Extrinsic factors involve the vertebral artery. The close relationship between the cervical spine and the vertebral artery can predispose the vertebral artery to injury.[147] Congenital anomolies altering the spine and route of the vertebral artery as well as spondylitic cervical changes may adversely influence the integrity of the vertebral artery. It is recommended that prior to active/passive cervical spine exercises, the cervical spine be moved or positioned so as to test for vertebral artery symptoms, primarily dizziness.[148,149] A general screening maneuver for vertebral artery symptoms (i.e., dizziness, as related to extrinsic factors) will be discussed in the section on Diagnosis.

Reflex Vertigo. Reflex cervical vertigo can also effect the vestibular nuclei activity and cause dizziness.[90,145] Often the diagnosis of BPPV is confused with reflex vertigo.[145] Both cervical vertigo and BPPV may have a history of trauma and both can induce dizziness by head movement.[145]

Bogduk states that "along with the eyes and labyrinths, the cervical vertebral column is an important source of proprioceptive information that influences the sense of balance, and it is well known, on clinical grounds, that cervical disease or injury can be accompanied by vertigo, but of a nature that does not imply vertebrobasilar insufficiency."[90]

Neck proprioception consists of the muscle spindle reflexes and tonic neck reflexes (TNR).[150,151] Recognition of the role that muscle spindle receptors play in providing abundant proprioceptive feedback cannot be overlooked.[152,153] The proprioceptive outflow from the muscle spindle is a result of gamma motor neuron activity, which is influenced greatly by the TNR.[40,154] The TNR, with its influence over muscle spindle activity, can be considered a main source for neck proprioception. The TNR originates in the facet joints of the upper cervical spine and will be discussed further in the section on Cervical Spine Influences on Mandibular Position and Movement.

Although orientation of the head in space is the special role of the vestibular apparatus, there is, as Cohen states,[155] "no conceivable way by which the semicircular canal or the otoliths can, by themselves, inform the brain of the angle formed by the head and the body." Orientation of the head to the body can only be achieved by neck proprioception. To have information from the vestibular system indicating a position of the head in space without information from the neck proprioceptors indicating relationship of head on neck, would prove greatly insufficient to function.

Vestibular catastrophes such as vertigo and nystagmus can be caused solely by abnormalities related to neck proprioceptors.[155] Various studies show that damage to deep cervical tissues, including neck muscles, produce a generalized ataxia, with symptoms of imbalance, disorientation, and motor incoordination.[153,157–159] Vertigo, ataxia, and nystagmus were induced in animals and humans by injecting local anesthetic into the neck.[160] The injections presumably interrupted the flow of afferent information from neck muscles and joint receptors. Ataxia in humans was associated with a broad-based staggering gait, hypotonia of the ipsilateral arm and leg, and a strong sensation of ipsilateral falling or tilting.[160] Hinoki,[161] through his experiments and clinical studies, suggests that vertigo following a whiplash injury may be due to overexcitation of the cervical soft tissues, such as muscles, ligaments, facet joints, and sensory nerves. Other seemingly unrelated symptoms such as vomiting, tinnitis, and diminished hearing could be related to cervical spine involvement.[145] Several studies have also shown that proprioceptive impulses from various neck receptors influence eye movements.[162–164]

In conclusion, cephalic symptoms such as headaches and dizziness can have a variety of origins and may require input from a variety of health professionals. In all instances, however, the attentive clinician must address cervical spine involvement as one of the primary sources of cephalic symptoms.

CERVICAL SPINE INFLUENCES ON MANDIBULAR POSITION AND MOVEMENT

This section analyzes and outlines the effects of cervical spine involvement on mandibular position and movement. The rest position of the mandible is primary to this analysis. Mandibular rest position and trajectory of mandibular closure as effected by cervical spine dynamics are reviewed. Ways in which

the vertical dimension of an intraoral orthotic appliance potentially alters cervical spine dynamics are highlighted. Clinical observations pertaining to cervical spine influences upon mandibular dynamics complete this section of the chapter.

The Rest Position of the Mandible

The rest position of the mandible is the position at which all functional mandibular movements of the mandible start and end.[165,166] When the mandible is in its rest position, a freeway space exists between the upper and lower arch of teeth.[167] The average freeway space is 3 mm and is measured between the tips of the central upper incisors and central lower incisors.[168] The freeway space has also been referred to as interocclusal distance, interocclusal clearance, interocclusal gap, or the interocclusal rest space.[169] The rest position of the mandible has also been referred to as the clinical rest position, tonic rest position, and rest relation.[170] A more appropriate term to describe the position of the mandible at rest is the *upright postural position of the mandible* (UPPM).[171] The term itself implies the essential interrelationship of the jaw, head, and neck in the upright posture.

In previous terminology the postural position of the mandible at rest implies a state of quiet or repose. However, in the UPPM, there is tonic contraction of the muscles attaching to the mandible.[172] Kawamura and Fujimoto[173] found spontaneous motor activity in masticatory muscles in the clinical rest position. Thus two "rest" positions have been stated to exist with the mandible: the clinical UPPM and the EMG rest position.[174] The mandibular EMG rest position has an average 11-mm freeway space and is not the same as or near the UPPM, which has an average 3-mm freeway space.[172,175,176] In the clinical setting, the diagnosis of an EMG rest position is difficult to arrive at (see Ch. 4) and does not influence treatment options. Clinically, it is therapeutic to decrease the amount of time a patient stays in occlusion and increase the amount of time the patient spends in the UPPM.

Dentistry often uses the UPPM as a guide to determine vertical dimension of occlusion (VDO) for diagnostic and therapeutic purposes in both edentulous as well as dentulous patients.[177,178] Dentistry has and continues to improve scientific evaluation of the UPPM other than by clinical judgment.[177] Conventional methods using a physiologic approach to arrive at an UPPM have involved swallowing, phonetics, and esthetics and are still used today.[179] Electromyography (EMG) with or without biofeedback, the myomonitor/kinesiograph,[180] and the use of cephalometric analysis[181] have been use to evaluate the UPPM. In the final analysis, judgement and clinical trial are still the common choices of the dentist when determining the UPPM.[178]

Numerous subtle factors can influence the UPPM from person to person as well as interpersonally from day to day and moment to moment.[182] Anatomic factors (weight and number of teeth) physiologic factors (tongue positioning and breathing), and pathologic factors (ridge resorption under dentures) may

all influence the UPPM.[169,170,183,184] Other influences such as age, drugs, and emotions have been suggested as influencing the UPPM.[185] Identifiable yet imprecise, the UPPM is still a position to which reference is made as a basic datum in many procedures in clinical dentistry.[176–178]

Cervical Spine Dynamics Influencing Mandibular Position

A variable influencing the UPPM that has not received much consideration is the cervical spine. Effects of cervical spine dynamics on mandibular tissue elasticity and mandibular muscle tone will be considered as factors that influence mandibular dynamics. Before pursuing this discussion, an overview of cervical spine dynamics is offered.

Cervical Spine Dynamics

The term *cervical spine dynamics* will be interchanged with the term *head-neck posture*. The term *altered cervical spine dynamics* will be interchanged with the terms *forward head posture* and *cervical spine involvement*. Cervical spine dynamics infer regional (flexion, extension, rotation, and sidebending) and segmental (movement between vertebrae) movements required to achieve and maintain a given head-neck posture. Terms such as "forward head posture" and "upright posture" can be misleading. Clinicians may mistakenly think a "forward head" posture necessitates a problem. On the other hand "good" head-neck posture may mistakenly suggest an absence of cervical spine dysfunction. Studies that investigate static cervical spine relationships without considering cervical spine dynamics will most likely arrive at invalid conclusions.[186] In the presence of good cervical spine dynamics, a forward head posture may be normal. Focus should be on the patient's posture and on the presence or absence of normal regional and/or segmental cervical spine dynamics. The status of cervical spine dynamics can only be determined by doing a physical examination that considers active and passive mobility assessment.

Achievement and maintanence of head posture in humans is an interaction of numerous factors; factors that are not totally understood and have not yet been recognized. Evolution, heredity, congenital growth and development, and pathologic factors influence the head-neck posture. Once musculoskeletal maturity is reached, other factors such as the aging process[187] and a decline in health[188] will create changes in function and performance capabilities and contribute to a deterioration of head-neck posture. Whereas these factors are recognized as exerting a long-term effect on head-neck posture, the achievement and maintanence of head-neck posture on a moment-to-moment basis will be accomplished through the peripheral and central control system.

Peripheral System

The peripheral system includes the vestibular, occular, and proprioceptive systems. Pertinent to the cervical spine is the proprioceptive system, which would include the muscles (muscle spindles) and tonic neck reflex (TNR). TNR pertaining to reflex vertigo has been previously addressed. Involvement of the proprioceptive system can have a significant impact on cervical spine dynamics in the achievement and maintainence of posture.[159,160,189–192] TNR will be mentioned again as a variable that can influence masticatory muscle tone.

Central Control System

The central control system (the brain) also influences the achievement and maintanence of posture on a continual basis.[193,194] Determination of head position is an interaction between the peripheral system and central control system[193,194] The central control system is comparing feedback from the peripheral system with pre-existing data that is based upon the central control's past and current proprioceptive experiences. Only when the feedback that the central control system anticipates is matched with the peripheral system's feedback does a certain head-neck posture exist.[195] This designates a type of reverse action of one system's dependence upon another. This reverse action of interdependence is the concept of reafference, promoted by the Nobel lauriate Nikolaas Tinbergen.[195,196] This concept of reafference strongly indicates that at various levels of integration from single muscle units up to complex behavior, the correct performance of many movements and positions is continuously checked by the central control centers.[196]

Peripheral and central control mechanisms constantly adjust, fine tune, and maintain the infinite number of head-neck postures used to fulfill the task at hand. Muscles and facet joints in the upper cervical spine provide important proprioceptive feedback for the peripheral system. Empirically, cervical spine management, especially manual therapy techniques, can influence the proprioceptive system in a therapeutic way. Empirically, the central control system is influenced by general and/or specific exercise programs that are designed to "put into touch" the patient's awareness on body movement and positioning.

Cervical spine dynamics are suggested to have the most immediate and long-lasting effect on the UPPM.[165,198–200] Patients present with various combinations and degrees of head-neck postures that may be flexed, extended, rotated or side bent. A three-dimensional model that displays the various effects the cervical spine can have on the UPPM is not available. What is available are conclusions derived from normal subjects in which the effects of head-neck postures in extension and flexion upon mandibular position and movement were investigated. Conclusions regarding cervical spine influences upon mandibular position and movement in a patient population are based entirely on empirical observations.

Tissue Elasticity Tone Affecting The UPPM

Tissue elasticity tone refers to the elastic tissue properties found in connective tissues both outside the epimysium and within each muscle.[201] The epimysium is the connective tissue sheath surrounding each muscle. Elastic properties consist of the connective tissue of the muscles, namely the fascial sheaths and the fibrillary muscle protein molecular aggregates (myosin and actin) within the individual muscle fibers.[201,202]

Tissue elasticity tone is present in all muscles. Slow closure of the mandible from a fully opened position to occlusion has been shown to take place without any change in muscle activity of the jaw closing muscles.[203] A study investigating jaw closing from a fully opened position showed that the closing was controlled by the elastic qualities of the digastric muscles.[204]

Cervical spine dynamics may affect tissue elasticity tone in muscle or connective tissue extending from the cervical spine or cranium to the mandible. During active or passive extension of the head on the neck, an increase in tension occurs in the supra and infrahyoid musculature that would result in depression of the mandible, thereby increasing the freeway space.[203,205] Ultimately, changes in head-neck relationships affect tissue elasticity tone about the mandible so as to alter mandibular position at rest.[206]

Muscle Tone Affecting The UPPM

The continuous state of tonic muscle activity occurring in the mandibular elevator muscles, namely the masseter, temporalis, and the medial pterygoids can influence UPPM. Resting muscle tone is primarily an expression of the number of firing motor units.[207,208] The motor unit consists of the anterior horn cell, the cell's axon, and all the muscle fibers innervated by the axon.[209] The anterior horn cell's activity will be influenced by both peripheral and corticospinal (central control) activity.[208] Under normal circumstances, the number of active motor units of the mandibular elevators should be minimal when the mandible is in the UPPM. The TNR can influence mandibular muscle activity.[210]

The role of the TNR in reflexly orienting the limbs in relationship to the head-body angle was described by Magnus in 1912.[211] Magnus,[212] in his classic work, analyzed the postural reaction of the decerebrate quadrupeds when their heads were experimentally turned to an extreme right or left position. He found extension of the forelimb on the side toward which the head was turned and flexion of the opposite forelimb.

Localizing the origin of the TNR to a specific area and tissue began with Magnus and DeKleijn,[213] who had limited the receptive field for the TNR to the first three cervical segments of the spine. They showed that the decerebrate cat possessed TNR that were not labyrinthine in origin but occurred secondary to activation of neck proprioceptors. The neck proprioceptors that are impli-

cated consist of the facet joint receptors (the mechanoreceptors) and muscle spindle receptors. McCouch et al.,[214] showed in 1951 that the TNR of the decerebrate labyrinthectomized cat were not abolished when the muscle mass of the neck was sectioned. The TNR was abolished only after the facet joints in the upper cervical spine were denervated, demonstrating that facet joint mechanoreceptors in the upper cervical spine are the origin of the TNR.

The TNR affects mandibular muscle tone through the trigemino-neck reflex. The trigemino-neck reflex has been demonstrated to occur via motor neurons located in the subnucleus caudalis and probably in the dorsal horn of the upper cervical spine. (Table 11-2)[40,215,216] This is verified when electric stimulation is applied to the central end of the ablated first cervical nerve and electromyographic activity is recorded from the masticatory muscles.[215] There appears then to be a closely organized neurophysiologic reflex relationship between TNR activity and trigeminal motor neuron activity. In the same study, EMG responses were abolished after the first three cervical nerves supplying the facet joints were cut.

Extension of the head on the cervical spine produces an increase in jaw muscle activity in the temporalis, masseter, and anterior digastric.[215,217] An investigation measuring anterior digastric and masseter muscle activity in response to head extension showed a marked increase in the masseter and a decrease in the anterior digastric muscle activity.[210]

Flexion of the head on the cervical spine causes a general decrease in jaw muscle activity, especially in the temporalis and masseter muscles.[215,217] Anterior digastric activity consistently increases with head flexion.[218,219]

In a study involving 30 normal adults, EMG was used to determine masticatory muscle activity in response to extension and flexion. Incremental movement of the head by 5°, 10°, and 20° into extension and flexion induced changes in EMG recording for the muscles under investigation (i.e., anterior temporalis, masseter, supra and infra hyoid muscles).[218]

The above studies conclude that during extension of the head on the neck, an increase in tone occurred in the supramandibular muscles under investigation. Flexion of the head on the neck caused a decrease in the same supramandibular muscles. Changes in head-neck relationships effects mandibular muscle tone, which will alter how the mandible is positioned at rest.

Cervical Spine Dynamics Influencing the Trajectory of Mandibular Closure

Mohl states:[219]

> we must logically conclude that, if rest position is altered by a change in head position, the habitual path of closure of the mandible must also be altered by such a change."

Table 11-2. Morphological and Functional Characteristics of Cervical Articular Receptor Systems[a]

Type	Morphology	Location	Parent Nerve Fibers	Behavioral Characteristics	Functions
I	Thinly-encapsulated globular corpuscles (100 μm × 40 μm) in clusters of 3 to 8	Fibrous capsule of joint (superficial layers)	Small myelinated (6–9 μm)	Static and dynamic mechanoreceptors; low threshold, slowly-adapting	(a) Tonic reflexogenic effects on neck, limb, jaw and eye muscles (b) Postural and kinaesthetic sensation (c) Pain suppression
II	Thickly-encapsulated conical corpuscles (280 μm × 100 μm), singly or in clusters of 2 to 4	Fibrous capsule of joint (deeper layers). Articular fat pads	Medium myelinated (9–12 μm)	Dynamic mechanoreceptors; low threshold, rapidly-adapting	(a) Phasic reflexogenic effects on neck, limb, jaw and eye muscles (b) Pain suppression
IV	Three-dimensional plexus of unmyelinated nerve fibers	Entire thickness of fibrous capsule of joint. Walls of articular blood vessels. Articular fat pads	Very small myelinated (2–5 μm) and unmyelinated (<2 μm)	Nociceptive (pain-provoking). High threshold, nonadapting	(a) Tonic reflexogenic effects on neck, limb, jaw and eye muscles (b) Evocation and pain (c) Respiratory and cardiovascular reflexogenic effects

[a] Please note the functions (a) of Type I, II, and IV receptors.
(From Wyke,[40] with permission.)

A change in the UPPM will influence the trajectory of jaw closure to the initial tooth or teeth contact. Altered cervical spine dynamics affects the UPPM, which results in a change in the trajectory of jaw closure.

McClean[220] studied changes in occlusal contact points during changes in body position by inclining the subject on a tilt table from a supine to upright posture while maintaining a constant head-to-thorax position. Electrical stimulation of the elevator muscle was done while the body position was changed from a supine to an upright posture. The results showed that in the supine position, contact points were more retruded. As the upright position was approached, the contact points moved forward into maximum intercuspation. Changes in occlusal contact patterns reflects changes in the trajectory path of mandibular closure during involuntary jaw closure. It was postulated that changes were due to gravitational influence on tissue elasticity tone and masticatory muscle tone. The position of the head-neck in the earth's gravitional field has been shown in other studies to change the UPPM and trajectory of jaw closure.[207,221]

The sound (acoustic energy) of occlusal contacts as maxillary and mandibular teeth come together provides information regarding the end-point of jaw closure.[220] The sound of occlusal contact is picked up via bone conduction by a contact microphone placed on the forehead of the subject. As the teeth meet, the sounds are recorded on tape, which constitutes the occlusogram. This technique is referred to as *gnathosonics*.[222] During voluntary and involuntary (electrically stimulated) jaw closure, occlusogram findings display either premature tooth contacts or prolonged intercuspal sliding contacts when present. Premature tooth contact occurred more often and in a more posterior position when subjects were in the supine position verses the upright position. Changes in occlusograms reflected changes in the trajectory of involuntary and voluntary mandibular closure. Graded changes in body position, from the supine to the upright position, appear to alter the function of the afferent-efferent loop that controls the finite and discrete neuromuscular coordination of a voluntary jaw closure to centric occlusion.[220,221,223,224]

Mohl[197] demonstrated habitual closing pathways are posture-dependent with subjects in the upright position. Extension of the head on the neck produces more posteriorly placed habitual closing pathways, with the initial occlusal contact occurring behind the maximum intercuspated position. Conversely, flexion of the head on the neck produces more anteriorly placed habitual closing pathways. Postural effects on occlusion in the upright position similar to Mohl's study were also found by Brenman and Amsterdam.[225] One study investigated the effects of normal subjects assuming different degrees of anterior to posterior positioning of the head-neck while maintaining the eyes in the horizontal plane. Vertical and anterior components of mandibular position and movement were altered.[226]

In summary, UPPM and trajectory of mandibular closure was altered in normal subjects when head-neck posture was moved from a supine to an upright posture, when the cervical spine was moved into extension, flexion, and anteroposterior movement with eyes kept horizontal in the sagittal plane.

Cervical Spine Dynamics and Vertical Dimension of Occlusion

Vertical dimension of occlusion (VDO) is the distance designated from the base of the nose to the base of the chin, when the teeth are in maximum intercuspation.[178] The freeway space encompasses the space between the UPPM and VDO. The freeway space is a physiologic necessity that allows the muscles of the oral-masticatory system to relax.[227] If the freeway space is infringed upon by increasing VDO, this may cause disturbances in the oral-masticatory system.[227]

Contrary to what has just been said, most patients adapt to changes in vertical dimension. The determination of the height of occlusion might not be a critical procedure as has often been stated.[228,229] However, these views may change if VDO is increased in patients with altered cervical spine dynamics.

There are many ways in which dentistry can change VDO. Pertinent to this discussion is the intraoral orthotic appliance, which will increase the vertical dimension. Appliance thickness may range from thin to thick according to the dentist's individual judgement and the various theories indicating use of an appliance.

Several studies support the view that the head is maintained by synergistic activity of the anterior head-neck and posterior head-neck muscles, as documented by EMG confirmation.[230,231] Studies have shown a close correlation between trigeminal inputs and neck muscle activity,[232,233] suggesting some degree of synergy exists between these two areas. Neural communication also has been shown to exist between the trigemino-neck reflex and the jaw opening reflex.[234] Research has shown[172,235] that as VDO is increased, EMG activity of mandibular muscles decreases until a resting range of approximately 11 mm is achieved. Speculation that a decrease in cervical spine muscle activity may occur as a response to the increase in VDO is inviting. I am not suggesting that intraoral orthotic appliances should be at a vertical dimension of 11 mm in order to decrease neck muscle activity. The preferred way to decrease neck muscle activity is to address the cervical spine directly through physical modalities and manual procedures.

Using a bite wedge that increased vertical dimension by 8 mm, Daly demonstrated the occurrence of cervical extension in 90 percent of his subjects (30 male students) within one hour.[236] This finding is sequential to the observation of Vig et al.[237] Two studies showed that when vertical dimension was increased, there was a tendency for the head to be raised from the horizontal plane, as measured by lateral cephalometric analysis.[238,239]

I hypothesize that the following head-neck posture events occur in response to an increase in VDO. In order to receive an object between the teeth two responses will occur. The most obvious response is depression of the mandible. The second and less obvious response is extension of the cranium on the cervical spine. When yawning, one will experience that opening the mouth is accompanied by extension of the head on the neck. Placing a thick object between the teeth (i.e., a half-inch wooden dowel), would result in depression of the mandible and extension of the head on the neck. Extension

of the head directs the eyes above the horizontal plane. The peripheral system (i.e., neck proprioceptors) will attempt to bring the eyes back to the horizontal plane. To return the eyes to a level position, the head-neck will either self-correct into an axial head-neck posture or move forward and slightly down, bringing the eyes level. Which way the patient self-corrects will often depend on the condition of the cervical spine (Fig. 11-6). A more dynamic cervical spine will experience less adverse reaction to any thickness of an intraoral orthotic appliance. In a more involved cervical spine, even a thin appliance can increase or reproduce the patient's symptoms.

Changes in cervical spine dynamics in response to the vertical dimension of an appliance may provide yet another explanation for symptomatic changes (improvement or aggravation) in response to an appliance. The anterior repositioning appliance used to "recapture" a disc displacement, often overcorrects jaw position in both the horizontal and vertical dimensions. The more the jaw is advanced anteriorly, the more the vertical dimension is increased. Williamson[243] relieved the symptom of vertigo (dizziness) in 25 patients (for 11 of the 25 patients, vertigo was the chief complaint) who had internal de-

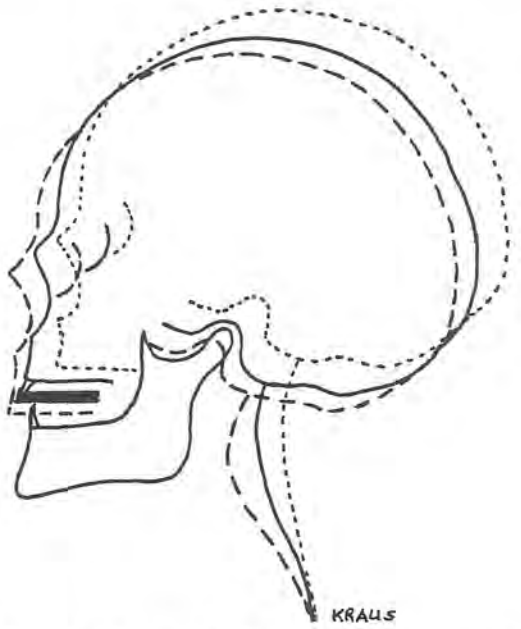


Fig. 11-6. The solid line depicts head-neck posture prior to a change in vertical dimension. Not shown is the initial response of the head extending on the cervical spine in response to an increase in vertical dimension by an intraoral orthotic appliance. Following the initial response of extension of the head on the cervical spine, in order for the eyes to return to the horizontal plane, one of two head postures may occur. The dotted line depicts an axial head-neck posture that would bring the eyes to the horizontal plane. The dashed line depicts a head-neck posture that is more forward and slightly down that would bring the eyes to the horizontal plane.

rangement with an anterior repositioning appliance.[240] He presented two hypotheses implicating the TMJ in the association of vertigo (dizziness) with internal derangement. In my opinion, a third explanation could be that the VDO of the anterior repositioning appliance altered cervical spine dynamics. The reader is reminded of an earlier section discussing dizziness, reflex vertigo, originating from the cervical spine. In this same study, dizziness reoccurred in 6 patients when they were taken off the anterior repositioning appliance after 3 months of wear. These same 6 patients underwent remission of their vertigo when placed back on the anterior repositioning appliance. It would be interesting to know if these 6 patients had cervical spine involvement. If cervical spine involvement was present it would be interesting to see if cervical spine management would have relieved the dizziness so as to avoid the continuation of the anterior repositioning appliance and subsequent occlusal work.

A patient's positive or negative response to the vertical dimension of an intraoral orthotic appliance will be dependent upon (1) the degree of cervical spine involvement, (2) thickness of the intraoral orthotic appliance, and (3) patients' individual physiologic adaptive range. The clinician should be aware that an intraoral orthotic appliance will effect mandibular posture which in return may directly or indirectly effect the cervical spine.

Clinical Significance of Cervical Spine Dynamics Influencing Mandibular Dynamics

The clinical interrelationship between the vertical dimension of an appliance and the cervical spine has been addressed. Attention will now focus on the clinical significance of the trajectory of jaw closure effected by an intraoral orthotic appliance and cervical spine dynamics. A discussion of this clinical hypothesis will begin with the occlusion, since it is the occlusion to which the appliance is fabricated.

The occlusion has been considered a popular etiology for the development and progression of TMD and/or MMH.[241] Reduction of MMH with an intraoral orthotic appliance is often used to minimize further aggravation to the TMD.[242,243] Appliance designs vary depending on the patient's occlusion, degree of TMD and/or MMH, and the dentist's preference. Although an intraoral orthotic appliance fits over an arch of teeth (mandibular or maxillary), symptomatic improvement from wearing an appliance may have nothing to do with the occlusion. A review of *intercuspal occlusal relationships* and *functional occlusal relationships* concluded that the etiologic role of occlusal factors has been overstated in the past, and in recent published research.[241,244] Intercuspal and functional occlusal interferences are too common and variable to offer sensitivity or specificity for defining a present or potential TMD population.[244] Intercuspal investigations consisted of skeletal anterior open bite, overbite, overjet, symmetry of retruded contact position, crossbite, and posterior occlusal support.[241] *Functional occlusal relationships* are occlusal contacts occurring when the teeth *are not in maximum intercuspation but contact is*

occurring during specific mandibular movements.[244] Functional occlusal relationships reviewed by Seligman consisted of balancing and working occlusal contacts, retruded contact position-intercuspal position (RCP-ICP) slide length and symmetry, occlusal guidance patterns, parafunction, and attrition.[244]

Although the role of occlusion in the development of TMD and/or MMH signs and symptoms remains controversial,[245–248] intraoral orthotic appliances have been and will no doubt continue to be a popular treatment choice offered by the dentist.[242,243,249]

Understanding the role of the cervical spine influencing *functional mandibular movements* may help in understanding yet another aspect of *functional occlusion*. Functional mandibular movements have been defined as "all natural, proper, or characteristic movements of the mandible made during speech, mastication, yawning, swallowing, and other associated movements."[250] The following discussion pertains to a single aspect of functional occlusion involving occlusal interference(s) as maximum intercuspation is approached. This aspect of functional occlusion is suggested to be influenced by cervical spine dynamics.

Searching/Avoidance Phenomenon

Regardless of cervical spine dynamics, the first contact between the upper maxillary teeth and the lower mandibular teeth during a series of jaw closures to maximum intercuspation is postulated to be a "searching" maneuver.[251] There is a registration of an afferent "engram" in the sensory cortex arising from the periodontal mechanoreceptors (the proprioceptors of the teeth).[251] The periodontal mechanorecepters are stimulated by the impacts and gliding contacts of the occlusal surfaces as the teeth move towards a position of maximum intercuspation. The afferent signals arising from the contacting occlusal surfaces interact with proprioceptive elements in the mandibular musculature to form a central sensory-motor "feedback-loop" that modifies the occlusal behavior of the individual.[223] Other investigators[252–254] demonstrated that the existence of occlusal interferences resulted in altered patterns of neuromuscular activity that tended to *circumvent* the occlusal interferences, and which were typified by *increased* EMG activity.

Similar to the searching maneuver, the "avoidance phenomenon" (Ch. 6) is believed to be a protective mechanism to prevent or minimize overloading of the joints or teeth. The avoidance of or the circumventing of occlusal interferences is done at the expense of altered (increased) muscle activity. A study investigated the introduction of an occlusal interference in 12 healthy subjects for a 1-week period.[255] The individual response to the interference varied substantially with regard to muscle tenderness to palpation. However, at the end of the experimental period, adaptation of the neuromuscular system to the interference was evident.

Avoidance maneuvers may be secondary to a "malocclusion" or "pseudomalocclusion." A pseudomalocclusion will infer an occlusal interference oc-

curring prior to maximum intercuspation, resulting from altered cervical spine dynamics. Therefore, it would seem as though altered cervical spine dynamics may trigger the "searching maneuvers"/"avoidance phenomenon" as maximum intercuspation is approached. This postulated series of events is as follows: (expanded from Mohl).[197,219]

Cervical spine dynamics affects the UPPM through alteration of tissue elasticity tone and mandibular muscle tone.
A change in the UPPM affects the trajectory of jaw closure.
A change in the trajectory of jaw closure results in tooth/teeth contact prior to maximum intercuspation (the pseudomalocclusion).
Teeth contact prior to maximum intercuspation are avoided but at the expense of altered (increase) muscle activity (searching/avoidance phenomenon).
Avoidance of tooth/teeth contact prior to maximum intercuspation continues as long as the patient's *physiologic adaptive range* is not exceeded.

An unlimited number of head-neck postures can be achieved throughout the day and night. Varying degrees of cervical spine involvement will accompany each individual patient. Therefore, it would be futile to attempt to predict a specific way that the mandibular posture and movement will be effected by cervical spine involvement.

The postulated series of events listed above can be experienced in the following exercise:

1. Sit or stand straight with jaw relaxed and begin to slowly look up and down. As previously described, tissue elasticity tone and muscle tone are affecting mandibular posture in response to extension and flexion of the neck.
2. Return the head to a neutral head posture. Now slowly and lightly tap the back teeth together. Be sure to relax and not allow cortical influences to dictate how the muscles move the mandible.
3. With the head straight continue tapping. Notice that in some individuals, occlusal contacts may be occurring in maximum intercuspation while others may be experiencing the initial occlusal contacts to be forward, back, left, or right to maximum intercuspation.
4. Continue tapping, and slowly look up and then down. What should be experienced is a change in the initial occlusal contacts from those contacts made in the neutral head posture.
5. Continue to tap with the head extended or flexed. Now avoid the initial tooth/teeth contacts and close straight into maximum intercuspation. The avoidance of occlusal contacts prior to maximum intercuspation can be done regardless of the head-neck posture. However, the avoidance of the pseudoocclusal interference(s) is done with an increase in masticatory muscle activity.

It can be surmised in the previous exercise that altered cervical spine dynamics, regardless of the occlusal state, may be a source of masticatory

muscle hyperactivity. I know of no studies confirming the previous observations in a symptomatic cervical spine patient population. The clinician, however, should be suspicious that MMH is originating from cervical spine involvement when (1) the history and physical examination is positive for cervical spine involvement, (2) the history indicates that the patient's cervical spine complaints preceded mandibular complaints, and (3) the patient's response to an intraoral orthotic appliance has been unfavorable.

Historically, bruxism is believed to be a result of occlusal prematurities and interferences[256] or stress.[257] However, both the etiology and the pathology are unknown. Could a symptomatic cervical spine be added to the list of multiple causes of bruxism? Is it possible for a patient to experience physical stress because of "neck pain," causing the patient to respond by clenching and/or bruxism?

There is speculation regarding the effects of cervical whiplash injury on the TMJ and MMH. Recent studies have shown that patient populations with a cervical spine whiplash injury have a higher incidence of signs and symptoms of TMD compared to a matched controlled group.[258,259] Unless there is a reported direct trauma to the mandible during a traffic accident, I have difficulty in accepting that jaw whiplash/jaw lash[260]/internal derangement whiplash[261] causes traumatic tearing of the posterior attachment at the time of the cervical spine whiplash. The high incidence of jaw-related symptoms to cervical whiplash may be explained by the patient clenching and/or bruxing in response to cervical pain occurring as a result of the cervical whiplash.

I have had the opportunity to see numerous intraoral orthotic appliance designs. The appliances were used for the treatment of TMD and/or MMH related symptoms and dysfunction. Many clinicians relate the success of splint therapy to breaking patterns of occlusal contact and deprogramming the musculature[242,243] that may be compensating for occlusal irregularities.[249] The design of the appliance should fit individual patient needs with full knowledge of the effects it may have on the TMJs, muscles, and teeth (Ch. 6). To be added to this list are the potential effects the appliance may have on the cervical spine. Splint designs should consider not only the occlusion effecting the UPPM and trajectory of jaw closure (Ch. 6)[262] but also that cervical spine dynamics affects the UPPM and trajectory of jaw closure. From the cervical spine perspective, an appliance that has the features listed below will minimize deleterious effects on the cervical spine yet maintain a therapeutic profile for the treatment of TMD and/or MMH. The appliance features listed below follow similar features mentioned in Chapter 6.

1. Full coverage to allow even contact simultaneously during closure
2. Maxillary coverage preferred though mandibular coverage may be required
3. Hard acrylic
4. Thin
5. Anterior portion of the splint provides immediate posterior disclusion in excursive movements

6. Shallow inclines leading into the centric stops still providing the features stated in #5
7. Avoid aggressive centric relation techniques
8. Appliance should ideally be balanced in more than one head posture (i.e., neutral and then extension or with patient reclining in dental chair)

Summary

Patients experiencing pain and/or dysfunction in the cervical spine may not be able to accommodate to mandibular changes as influenced by an intraoral orthotic appliance. An intraoral orthotic appliance may actually be a stimulus to the patient's symptoms in the presence of altered cervical spine dynamics. A healthy cervical spine is a more forgiving cervical spine when mandibular positioning and mobility is sufficiently influenced by the design and chairside balancing of the intraoral orthotic appliance. Comprehensive management of TMD and/or MMH will include cervical spine management. Dr. Mohl states:[197] "It therefore seems reasonable to consider that at least some of the dysfunctional problems involving the masticatory system could be in some way related to the adaptive requirements imposed by chronic or acute postural demands." The multifactorial etiology and pathophysiology of TMD and masticatory muscle hyperactivity is well accepted today. Cervical spine involvement may indirectly influence this complex topic. In select individuals, however, the cervical spine may be the primary feature contributing to pain and dysfunction.

DIAGNOSING "CERVICAL SPINE INVOLVEMENT"

The Diagnostic Dilemma

Previous sections discussed the cervical spine involvement as a source of neck, shoulder, upper extremity, and cephalic symptoms. The clinical implications of cervical spine involvement influencing mandibular position and movement have also been discussed. This section will discuss the criteria needed to diagnose the cervical spine condition that has, thus far, been referred to as cervical spine involvement.

Medical diagnoses that implicate cervical spine involvement as a patient's source of symptoms include such diagnoses as "neck pain," "cervical sprain," "cervical disc disease," "cervical osteoarthrosis," "thoracic outlet syndrome," "myofascial pain syndrome," "cervical fibromyalgia," and "radiculopathy." Such diagnoses may often rely on only anecdotal experience or general informed judgement gathered from the history and physical examination.[263] Yet for other diagnoses, the use of expensive paraclinical procedures including radiographic, electrodiagnostic, and laboratory procedures may be required.

Ideally, the diagnosis should indicate the source of the patient's symptoms

and should provide an insight into the treatment(s) required to resolve the patient's problem. The musculoskeletal diagnosis should be easily assessible by clinical tests and measures to determine effectiveness of treatment. The goal for the majority of all spinal pain patients is return to pain-free functional activity. The extent to which the treatment and the projected outcome/prognosis of treatment is influenced by the diagnosis emphasizes the importance of the diagnosis.[264]

Considering the emphasis placed on the diagnosis, hopefully the diagnosis will be correct. However, treatment, outcome of treatments, and prognosis of spinal disorders (lumbar, thoracic, and cervical), may seldom be based upon the previously described diagnostic scenarios. The following facts illustrate the problems and dilemmas of arriving at a medical diagnosis for spinal pain.

1. Radiographs, a common procedure used to diagnose spinal conditions,[265] may provide little, if any, insight into the source of the patient's symptoms.[266–268] Abnormal structural pathologic findings, including disc herniations, have been found in spines of asymptomatic patients.[269–272] Epidemiological studies show that the prevalence of most abnormalities seen in plain radiographs of subjects with spinal pain is similar to those abnormalities seen in subjects that never had spinal pain.[266,267,269,270]

2. New technology involving radiographs, electrodiagnostic, and laboratory procedures has not altered the overall incidence, morbidity, cost, or disability related to spinal pain disorders.[276] With the unnecessary cost and exposure to radiation yielding no additional information regarding etiology and prognosis of spinal pain,[273,274] some authors propose a selective use of radiographs in spinal pain patients.[275–277]

3. The diagnosis is unknown in 90 percent of spinal patients.[278,279] The vast majority of diagnoses that incriminate a particular tissue source are difficult to identify, because the physical signs and symptoms often have little correlation.[87] It is difficult to establish a correlation between tissue source with signs and symptoms because the degrees of dysfunction do not warrant the removal of tissues to be studied.[87]

4. Terminology and nosology of diagnosis currently used for spinal disorders are neither standardized nor validated.[87] The diagnosis thus becomes the fundamental source of error from which the clinician may formulate a treatment plan that may be done for an incorrect diagnosis.[280,281]

5. The ability of a physical therapist to evaluate the effects of physical therapy intervention for a medical diagnosis is hampered for reasons previously mentioned.[280,281] Physical therapy interventions cannot be shown to be effective for any diagnosis of a spinal condition unless there is a clear statement of the condition.[280,281] These are some of the reasons why there are contradictory findings in the literature and in rehabilitative therapy regarding criteria for evaluating the effectiveness of treatment.[87]

The literature continues to be deficient in scientifically admissible studies pertaining to the evaluation, treatment and prognosis in spinal diagnoses.[87]

Scientific studies need to provide better understanding of how to arrive at meaningful diagnoses for spinal conditions. With advanced diagnostic accuracy, more realistic insight as to the prognosis of returning the patient back to a functional and quality life style might be derived.[280,282]

Complexity of the spinal anatomy, physiology, neurophysiology, and the pain phenomenons are some of the inherent features of spinal pain that make it difficult to arrive at a diagnosis.[87,270,279] In an extensive review of the literature in 1987, the Quebec Task Force on Spinal Disorders concluded that of the numerous acute or chronic spinal complaints, *nonspecific* ailments of the lumbar, dorsal and cervical regions, with or without radiation of pain comprise the vast majority of problems for which the patient seeks help.[87] *Nonspecific* implies a nondiseased condition, meaning that no underlying disease can be established. The clinician should be alert to the possibility of disease or an unrelated musculoskeletal condition that can mimic a nonspecific ailment of the spine.[283] If the clinician is ever in doubt, a referral to the appropriate medical and or dental professional is in order.

Given the vast problems of diagnosing nonspecific complaints, the Task Force found it necessary to propose its own classification of spinal disorders.[87] This classification system was not based on radiological, physiopathological, or mechanistic entities, since they remain too vague in most cases. The Task Force proposed a classification based on simple clinical criteria (signs and symptoms) that represent the majority of cases seen in clinical practice. The Task Force proposed a classification called "Activity-Related Spinal Disorders" for patients having nonspecific complaints. The classification of activity related spinal disorders was divided into 11 categories.[87] (Table 11-3) The following is a summary of each category:

Categories 1–3. Based only on localization of pain (history)
Category 4. Based on the results of the clinical examination
Categories 5–7. Based on the result of paraclinical investigations
Categories 8–10. Based on response to treatment
Category 11. Based on conditions seldom seen or of little importance

The reader is strongly encouraged to read the full report of the Quebec Task Force on Spinal Disorders to appreciate the complexity of all factors (i.e., evaluation, diagnosis, treatment and prognosis) relating to spinal disorders with an initial literature review of 7,000 articles.[87] The report lists the 769 references used by the Task Force in reaching their conclusions.

A growing consensus among clinicians and researchers dealing with nonspecific spinal pain agrees that a new widely acceptable diagnostic classification is needed.[269,284–288] Inspired in part by the Quebec Task Force publication, research and clinical testing of various nonspecific classification systems, in which patients are primarily classified according to signs and symptoms, has been pursued in the medical and physical therapy professions.[268,280,283–286,289–291] Classifying/diagnosing patients according to signs and symptoms should provide a direct treatment plan and a better understanding of treatment outcomes.[289,292,293] Physical therapists would share in the domain of diagnosing

Table 11-3. Classification of Activity-Related Spinal Disorders

Classification	Symptoms	Duration of Symptoms From Onset	Working Status at Time of Evaluation
1	Pain without radiation	a (<7 days)	
2	Pain + radiation to extremity, proximally	b (7 days–7 weeks)	W (working)
3	Pain + radiation to extremity, distally[a]	c (>7 weeks)	I (idle)
4	Pain + radiation to upper/lower limb neurologic signs		
5	Presumptive compression of a spinal nerve root on a simple roentgenogram (i.e., spinal instability or frature)		
6	Compression of a spinal nerve root confirmed by Specific imaging techniques (ie, computerized axial tomography, myelography, or magnetic resonance imaging) Other diagnostic techniques (e.g., electromyography, venography)		
7	Spinal stenosis		
8	Postsurgical status, 1–6 months after intervention		
9	Postsurgical status, >6 months after intervention 9.1 Asymptomatic 9.2 Symptomatic		
10	Chronic pain syndrome		W (working)
11	Other diagnoses		I (idle)

[a] Not applicable to the thoracic segment.
[b] (From Spitzer et al.,[87] with permission.)

classifying patients according to signs and symptoms that identify the condition. A diagnosis based upon signs and symptoms would be the focus of the physical therapist's treatment and reassessment of the patient's condition during, immediately following and prior to subsequent treatments.[288,289] Diagnosis by a physical therapist has been defined by Sahrmann:[294]

> Diagnosis is the term that names the primary dysfunction toward which the physical therapist directs treatment. The dysfunction is identified by the physical therapist based on information obtained from the history, signs, symptoms, examination, and tests the therapist performs or requests.

Studies on the clinical efficacy of classifying patients with nonspecific complaints based upon signs and symptoms have largely addressed the lumbar spine. Therefore, the following classification for nonspecific ailments of the cervical spine is only a proposal. This classification is based upon the author's present clinical impression of nonspecific complaints of the cervical spine. As clinical research pertaining to classifying nonspecific activity related disorders of the cervical spine is expanded, both the name and criteria used in the history and physical examination will be modified.

The proposed classification and categories for nonspecific complaints of the cervical spine are:

Classification: Movement Dysfunction of the Cervical Spine (MDCS)

Category 1. Neck symptom(s) (central) without radiation.
Category 2. Neck symptom(s) and
  a. Radiation, but not into extremity
  b. Radiation into upper extremity
Category 3. Neck symptom(s) + radiation cephalic
Category 4. Patient perception of a limitation in mobility of the neck
Category 5. Patient perception of a limitation in mobility of the neck along with either Categories 1, 2 or 3

Jette states,[288] "the purpose of having a physical therapist establish a diagnosis is to name and communicate the primary impairment, disability, or handicap toward which the clinician directs his or her treatment within that professional's appropriate scope of practice." A physical therapy diagnosis should not be used to reflect ownership of the condition.[288] The following is an overview of the pertinent history and physical examination needed to arrive at the diagnosis of MDCS.

History

The patient's response to the following questions will direct the clinician towards suspecting MDCS. The series of questions will establish a base line as to how the patient subjectively and functionally is responding to treatments

offered for MDCS. Short-term and long-term goals will begin to be formulated during the history and will be determined after the physical examination is completed. Further "testing" may be needed to determine more specifics of impairment and functional limitation. The following questions are not meant to be an exhaustive outline of a detailed medical and/or clinical history.

1. What is your primary complaint(s)?

This question should document the chief complaint, the area of the symptoms, and the description of the symptoms. Patient will be asked if the previous features of their symptoms changed since they first become aware of their symptom(s).

Primary Symptoms can consist of any one or combination of:
Headaches
Facial
Jaw
Neck
Shoulder
Proximal or distal upper extremity symptoms

Secondary Symptoms can consist of any one or combination of:
Dizziness
Eye (pressure behind the eyes, eyes sensitive to light, difficulty in focusing
Ear (subjective hearing loss, fullness, ringing)
Throat (difficulty in swallowing, soreness that does not turn into a sore throat)

Insight as to how the cervical spine can be a source of secondary symptoms has been explained in the section Cervical Spine as a Source of Cephalic Symptoms.

Secondary complaints in the absence of "primary complaints" may suggest that the patient does not have MDCS. Secondary complaint(s) needs to occur in conjunction with a "primary complaint(s)" in order to suggest that the secondary complaints are originating from MDCS, although there are exceptions to this. Bell states[295] that the primary pain may be modulated and inhibited until it is not consciously felt by the patient, leaving the secondary referred pain as his complaint. When this occurs, the therapist finds himself with the task of locating the "silent" primary pain that is the source of the complaint.[295] If a secondary complaint(s) is verbalized as the patient's only complaint(s) with no "primary complaint" expressed by the patient, the clinician will have to rely upon the objectives of the physical examination to suggest that the secondary complaint(s) is stemming from MDCS. If the objectives of the physical examination are not achieved, such secondary complaints expressed as primary complaints may not be related to MDCS and a referral to the appropriate health professional is in order.

The clinician must take note that primary complaints such as headache, jaw, and facial symptoms can occur without other symptoms. In other words,

the patient may complain only of jaw symptoms with the source stemming from the silent cervical spine. Thus the importance of establishing a differential diagnosis is emphasized. If the TMJ and associated muscles of mastication are not suspected as a source of the patient's symptoms, then the clinician should investigate the cervical spine.

2. What do you believe caused your symptoms?

Any one or combination of the following are possible responses from the patient that should increase the clinician's suspicion that MDCS is present:

Physical trauma (i.e., MVA, fall, blow to the head and neck)
Patients who received a direct trauma involving the cervical spine usually take longer to recover and can continue to have symptoms for as long as 15 years.[2]
Occupational trauma (i.e., change in the physical working environment; travelling, lifting, reaching, etc.)
Emotional trauma (family or work related)
Recreational trauma (weekend athlete)
Insidious onset (detailed questioning will often indicate that an insidious onset is actually related to one or a combination of the conditions listed above)

3. When did you first notice the appearance of your symptoms?

Less than 7 days
7 days to 7 weeks
More than 7 weeks

According to the Quebec Task Force,[87] acute pain is restricted to 7 days duration, subacute is defined as lasting from 7 days to 7 weeks, and chronic is defined as lasting more than 7 weeks in duration. The Task Force recognizes that the outlook for recovery grows more ominous as time elapses, emphasizing the need to accurately diagnosis and begin appropriate treatments.[87] The Task Force suggests that symptoms that persist past the seventh week after onset (or treatment) require consultation with a specialist. However, the Task Force indicates even in the case of a patient whose symptoms have lasted 6 months, symptoms can still be treated without significant psychologic components.[87] Clinically, MDCS would best be treated within the first 7 weeks. However, MDCS can be managed well into the chronic stages based upon the skill level of the clinician. Variables as discussed in Chapter 13 may also need to be investigated.

4. Describe the intensity, frequency and duration of your symptom(s)

Intensity. Intensity can be graded on the visual analogue scale of 0 to 10.[88] MDCS can have an intensity anywhere from 1 to 10, most common between a 2 to 8.

Frequency. Frequency associated with MDCS can vary from daily to once a month. The less frequently a person experiences symptoms, the longer the patient will need to be followed to determine if treatments have any effect on symptomatic outcome.

Duration. Duration for MDCS can range from an intermittent "fleeting" symptom to a constant symptom that varies only in intensity. A fleeting primary or secondary symptom usually occurs in the presence of another primary symptom(s). As an example, "my jolting head pain" or "ringing in my ears" lasts for only a few seconds, "but my neck pain is constant." The patient may go on to say that the fleeting pain is noticed when the neck pain intensity increases.

This line of questioning, if appropriate, can be done for each primary and or secondary complaint. Some patients may be able to categorize the intensity, frequency, or duration of their symptoms in relation to morning, afternoon, and evening patterns. Asking the patient if symptom(s) are better, worse or no different since onset will provide an idea of the stage the patient is in at this time.

5. What increases, reproduces or decreases your symptoms?

A patient experiencing MDCS often will identify some form of movement and/or positioning of their neck as increasing, reproducing, or decreasing their symptoms. The clinician will recall the silent pain phenomenon explained earlier. If the patient has difficulty in answering this question, the clinician can ask lead questions such as:

Are your symptoms affected by movement or activities involving your head, neck, shoulders, or arms?

Are your symptoms affected by how you hold your head, neck, shoulders, or arms?

Are your symptoms affected by work-related, family-related or self-induced stress situations?

Do you have trouble in getting to sleep, or staying asleep, or are your symptoms present first thing in the morning? Do you find that you have to support your neck a certain way at night in order to get to sleep?

6. What can you not do since you have been experiencing your symptom(s)?

This line of questioning will determine how MDCS is effecting the patient's ability to function. Question the patient about functional activities both at work and at home, sleeping patterns, and recreational activities. If function is not affected then inquire about how their symptoms interfere with their quality of life (i.e., "I do not like going out," "I do not like being sociable or being with my spouse." Still other patients will state they can still do everything but are in pain while doing the activity.

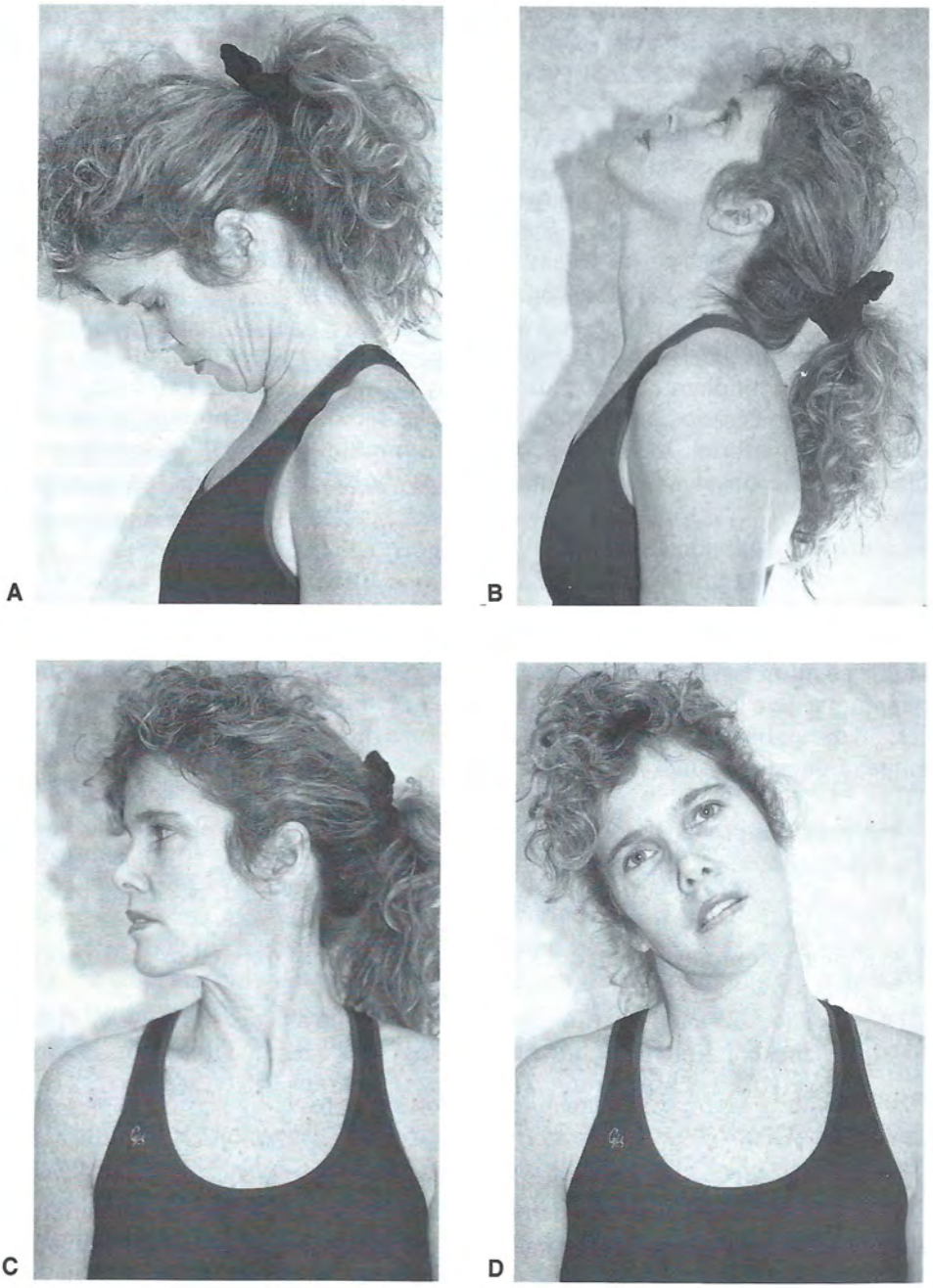


Fig. 11-7. Active movements in the cardinal planes of motion **(A)** Flexion. **(B)** Extension. **(C)** Rotation (right). **(D)** Side Bending (right).

Physical Examination

The physical examination will consist of the following three objectives. Any two of the three objectives along with the pertinent history questions previously covered will help in arriving at the diagnosis of Movement Dysfunction of the Cervical Spine.

Objective I

The patient's symptom(s) is either reproduced, increased or decreased (effected) by either or both (1) active movement(s) performed by the patient; (2) manual procedure(s) performed by the clinician.

Active Movement(s) Performed by the Patient. Active movements would include the cardinal plane movements of flexion, extension, rotation, and side bending (Fig. 11-7). Combined active movements of diagonal extension, right or left, and diagonal flexion, right or left, may be necessary to effect the patient's symptoms (Fig. 11-8). During and immediately following the active movements, the patient will be asked if the symptoms were affected by any of the active movements.

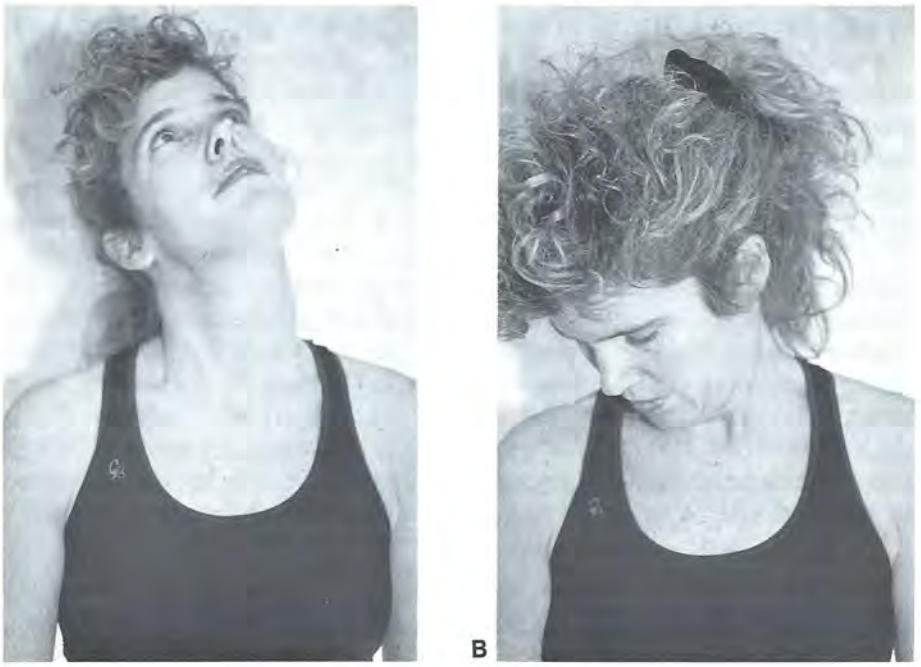


Fig. 11-8. Active Diagonal Movements. (**A**) Diagonal Extension (right). (**B**) Diagonal Flexion (right).

Manual Procedure(s) Performed by the Clinician. (The specifics as to the manual procedures will be discussed in Objective III.) Active or manual procedures may not affect the patient's symptom(s) all the time. Tissue "irritability" often determines whether or not a patient's symptoms will be affected with active and or manual procedures. Tissue irritability is defined as the clinician's observation as to the ease in which the patient expresses either verbally ("that hurts") or nonverbally (protective posturing) nociceptive activity caused by mechanical or chemical irritation of the involved tissue(s) in response to the active and or manual procedures.

Objective II

The active movements and or manual procedure(s) must incriminate an area that is commonly associated with a primary and or secondary symptom(s).

Active Movements. Active movements of the cervical spine would involve the cervical spine tissues (facet joints and nerve roots) and associated tissues (muscles and peripheral nerves). The clinician may want to stabilize the shoulders of the patient during cervical spine active movements, to ensure that active movements are occurring in the cervical spine area and not the thoracic area.

The clinician should record active range of motion (AROM) performed in the cardinal planes. If AROM is limited by MDCS then treatment of MDCS may result in symptomatic improvement. The patient's perception of improved mobility as well as an observed improvement in mobility by the clinician may also result from the treatment of MDCS.

Recording AROM provides objective data for the examination. The clinician is referred to Youdas and co-workers[297] for comparative analysis of three methods for reliability of measurements for active cervical range of motion.

Clinicians should avoid using previously reported singular values as estimates of normal cervical AROM for both genders and across all ages.[298] In a study involving 337 healthy subjects ages 11 to 97, Youdas and co-workers[298] concluded that AROM had a significant relationship to age and gender. Females usually have more AROM than males[298] and for each 10-year change in age, both females and males lose approximately 5 degrees of neck extension AROM and 3 degrees of AROM for the remaining cardinal planes of motion.[298,299] Due to the wide normal variation of active movements (age, gender, build, and habit) the value of AROM lies more in monitoring progress than in diagnosing.[300]

Manual Procedures. Manual procedures of palpation and passive mobility will be directed toward the six areas outlined in Figure 11-9. Empirical evidence indicates that manual procedures in these areas are frequently correlated with the patient's symptoms. Questions pertaining to specifics of hand placement, direction of force, amount of force, duration of force, and palpatory findings

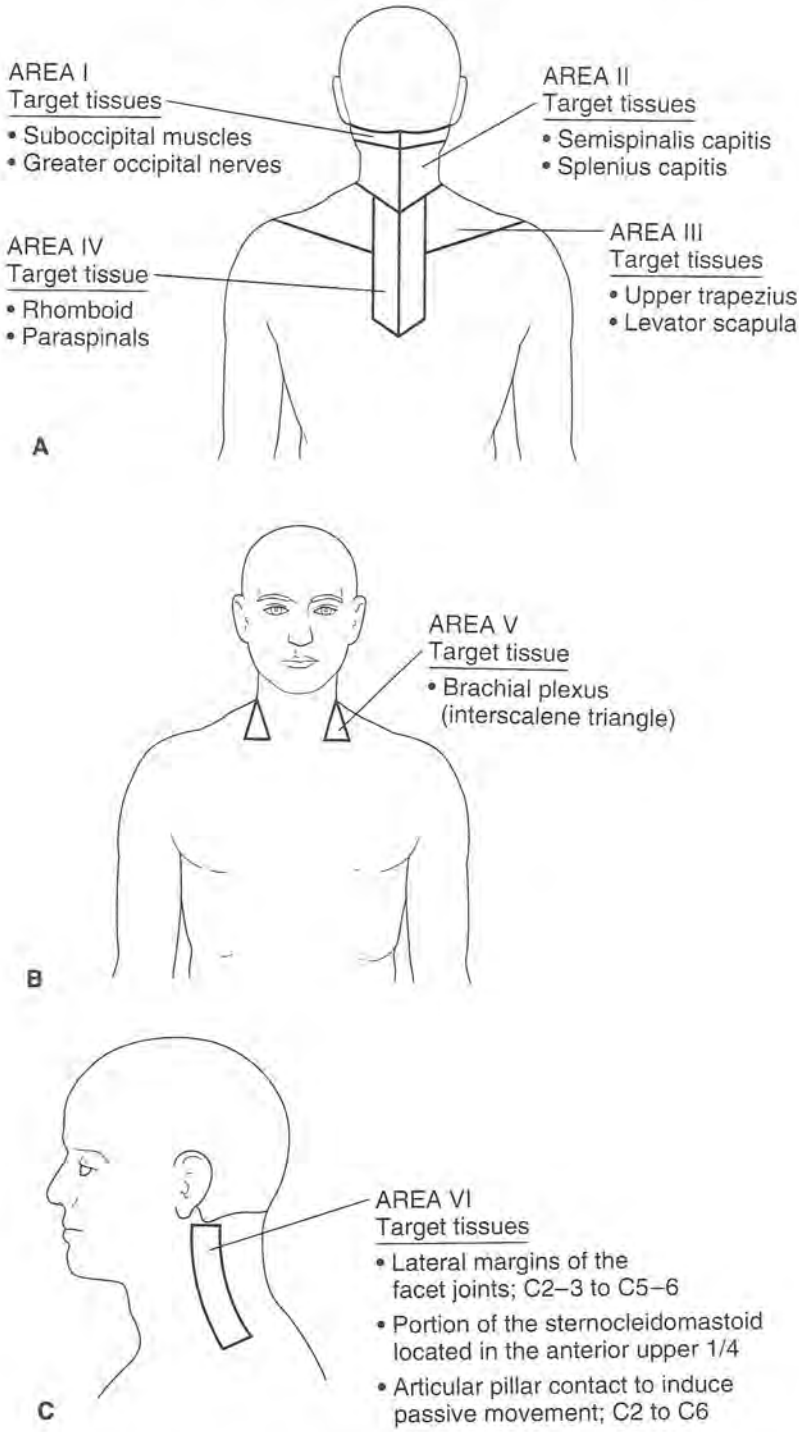


Fig. 11-9. Manual procedures are performed to these six areas.

are beyond the scope of this chapter. Knowledge of anatomy and clinical experience are variables that factor into the skilled application of manual procedures and into the interpretation of the subjective and objective findings.

Objective III

If a manual procedure(s) effects the patient's symptom(s), can the clinician identify altered mobility, position, and or tissue condition in the area(s) being examined?

Target Tissues. A clinical impression of altered mobility (joint, soft tissue), position (osseous positional faults) or tissue condition (increase, decrease of normal muscle, or soft tissue tension) may be determined by the clinician for the targeted tissue(s) in the appropriate area. There are, of course, other tissues that have not been named that are located in these areas. The purpose is not to identify all tissues, but to gather an impression about "key" tissue involvement that will help to arrive at the diagnosis of MDCS.

Area I (Fig. 11-10) Target Tissues
Suboccipital muscles
Greater occipital nerve

Fig. 11-10. Area I. Target tissues: suboccipital muscles, greater occipital nerve.

Fig. 11-11. Area II. Target tissues: semispinalis capitis, splenius capitis.

Area II (Fig. 11-11) Target Tissues
- Semispinalis capitis
- Splenius capitis

Area III (Fig. 11-12) Target Tissues
- Upper trapezius
- Levator scapula

Area IV (Fig. 11-13) Target Tissues
- Rhomboid
- Paraspinals

Area V (Fig. 11-14) Target Tissue
- Brachial plexus (interscaleni triangle)

Area VI Target Tissues
- Lateral margins of the facet joints C2–3 to C5–6 (Fig. 11-15A)
- Portion of the sternocleidomastoid in the anterior upper quarter of area VI
- Articular pillar (Fig. 11-1) contact to induce passive movement from C2 to C6 (Fig. 11-15B)

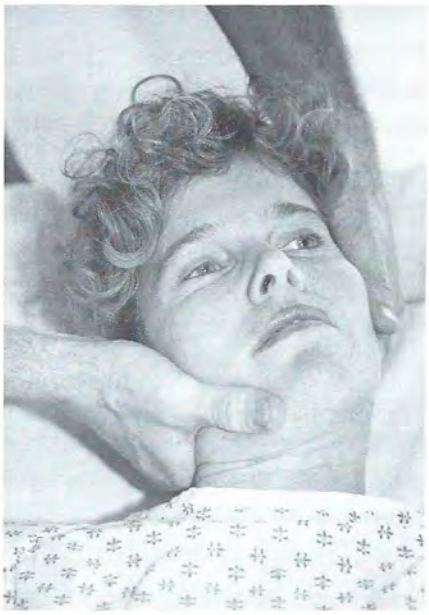
Areas I–VI (Fig. 11-16)
- Manual traction test.[79] This test is directed to all areas and tissues. It is a nonspecific test of the patient's subjective response to a traction force applied to the head and neck. Often the patient will report symptoms have

Fig. 11-12. Area III. Target tissues: upper trapezius, levator scapula.

Fig. 11-13. Area IV. Target tissues: rhomboid, paraspinals.

Fig. 11-14. Area V. Target tissue: brachial plexus.

A



B

Fig. 11-15. Area VI. Target tissues: **(A)** Lateral margins of the facet joints (C2–3) (a portion of the sternocleidomastoid can be palpated in the anterior upper quarter of area VI). **(B)** C4 articular pillar contact by the right metacarpal phalangeal joint, inducing a passive right side bending motion.

Fig. 11-16. Areas I–VI. Target tissues: a nonspecific passive cephalic traction force applied by the lateral aspect of the thumb of both hands to the mastoid processes.

decreased while the traction is applied. The amount of force is dependent upon the degree of tissue irritability and ability of the patient to relax.

Postural Evaluation. It has long been suggested that faulty posture may be involved in the etiology of spinal pain.[301] In a study of Refshauge and co-workers,[302] 25 subjects with pain in the cervical or trapezius region were age-matched with 25 volunteers. The study failed to show that the size of the cervico-thoracic kyphosis in habitual standing is associated with the presence of pain in the cervical and trapezius area.[302] Griegel-Morris and co-workers[303] looked at postural abnormalities in the thoraco–cervical–shoulder region of 88 healthy volunteers between the ages of 20 to 50 years. The relationship between the severity of postural deviations and the severity and frequency of pain in the thoraco–cervical–shoulder region was not significant.[303] Based upon these studies, ideal postural alignment may need to be redefined. My clinical observations have placed less emphasis on the evaluation of head–neck–shoulder (HNS) posture for the purpose of arriving at the diagnosis of MDCS. This is not to infer that postural instructions are not given to the patient. Patient education about maintaining an upright postural position during standing and sitting postures appears to be extremely therapeutic (Fig. 11-17). With reliability and predictability in question, it appears that an actual "measurement" of posture is not needed in order to diagnose MDCS. What may be more important than static posture is the dynamics of HNS relationship as well as active and passive segmental dynamics. If the patient has good regional and segmental dynamics and is instructed on concepts of good posture, then the HNS relationship obtained by the patient may be considered normal.

Fig. 11-17. An upright postural relationship of the HNS. Shown also is a line extending up and out from the chest to assist in the awareness of an upright postural position.

Comments on the Three Objectives of the Physical Examination

Additional objectives, areas, and tissues can be included in this form of an examination. For now, I find that if the above objectives are addressed, this will be sufficient to either include or exclude the diagnosis of MDCS. This form of examining is most reliable, valid, predictive, and teachable when combined with sound clinical reasoning. Clinical reasoning is here defined as the cognitive process or thinking used in the evaluation and management of a patient.[304,305] Clinical reasoning is the essential bridge between a history and physical examination and knowing the answer. I do not want to lead the reader to believe that all aspects of reliability, validity, and predictive value of the proposed physical examination have been worked out and documented, for they have not. It is recommended that the clinician read the "Standards for Tests and Measurements in Physical Therapy Practice," developed by a task force of the Committee on Research (1987) by the American Physical Therapy Association.[306] As a profession we need to achieve quality of our tests and measurements, ". . . not only among ourselves and our medical colleagues, but also, more relevantly, among those who must render decisions about our services and livelihood." (S Wolf)[306]. "Clinical practice cannot wait and testing will usually have to proceed; however, physical therapists should be aware of and should acknowledge the limitations of the measurements they are using."[306]

Special Findings of the Physical Examination

Special findings of the physical examination may modify the approach to treatment for MDCS. Manual therapy is a significant portion of the treatment approach. Manual therapy involves a wide spectrum of procedures that are aimed at specific objectives. Contraindications to manual therapy are all relative depending on the special findings of the individual patient and the clinician's skill level. The following special findings will influence the clinician's choice of manual therapy procedures.

Cervical Spondylosis

Cervical spondylosis is a condition involving degeneration[83] and is identified by radiographs. Patients with minimal degenerative changes as seen on x-rays can tolerate manual therapy to the cervical spine. Patients with severe degenerative changes may also benefit from manual therapy depending on which techniques are chosen and how they are delivered.

Standardization of radiographic interpretation for cervical spondylosis is needed so that both the medical and physical therapy professions will know what is meant by minimal, moderate, and severe spondylotic changes. Studies have shown the need to elaborate, validate, and standardize the criteria to improve on the reliability of interpreting plain radiographs[307] and CT scans.[269,307,308] Correlating degenerative changes with history and other physical findings of the examination for the symptomatic patient may provide insight into symptomatic and functional outcomes of physical therapy treatment for MDCS.

Radiculopathy

Upon examination, no finding by itself will validate the presence of radiculopathy. Radiculopathy will require a combination of the following findings on examination:

Neurologic Examination. Positive for incriminating a specific nerve root based upon muscle, reflex, and cutaneous testing. See neurologic examination section below for additional discussion.

Radiographic Findings of Cervical Spondylosis. The more severe the degenerative changes on x-rays, the more potential for stenosis of the intervertebral foramina resulting in radiculopathy.

Repeated Active or Passive Diagonal Extension Patterns. Extension of the cervical spine causes a statistically significant decrease in the intervertebral foramina diameter when compared to the intervertebral foramina diameter in a neutral cervical spine position.[309] Combining sagittal extension and ipsilateral rotation (diagonal extension—Fig. 11-8A), causes the most foraminal encroachment at C5, C6, and C7.[309] Diagonal extension patterns may be useful for eliciting pain as a provocative procedures for radiculopathy.[309]

Reproduction of neck symptoms with or without radiation into the shoulder and or upper extremity during diagonal extension patterns requires cautious interpretation. Reproduction of symptoms can be attributed to tissues (muscle, facet joint, peripheral nerve), other than nerve root irritation/compression. Pain in the arm is only a symptom and other tests need to be performed to confirm direct nervous tissue irritation. Reflex mechanisms causing referred symptoms are believed to be more common than mechanical nerve lesions.[310] Even in the presence of a known radiculopathy, I find that a majority of patients can do quite well with manual therapy to the cervical spine.

Upper Limb Tension Tests. The upper limb tension test (ULTT) has been termed the straight leg raise of the arm.[311] The relevant anatomy and biomechanics of the ULTT have been the focus of recent attention.[311–316] The ULTT can be used to test the mobility of cervical nerve roots and peripheral nerve mobility.[312] This procedure combines shoulder depression, shoulder abduction, extension, lateral rotation, forearm supination and elbow extension, and wrist/finger extension.[312] The major cervical nerve roots affected by the above procedure are C5 and C6,[314,315] which are also the main nerve roots involved with radiculopathy.[75] The absence, reproduction or increase in the patient's neck or upper extremity symptoms during or following the completion of the ULTT may help to determine the presence of neural tissue entrapment (central or peripheral). The clinician will need to do a differential examination of the glenohumeral, elbow, and wrist joints and their associated tissues so as to clear other sources of shoulder and upper extremity symptoms.

In summary, correlation of the neurologic assessment, radiographic findings of cervical spondylosis, diagonal extension patterns, and the patient's response to the ULTT will assist the clinician to conclude whether or not radiculopathy is present. If present, the clinician may need to modify certain manual therapy techniques. If the patient does not respond to the proposed management discussed in the next section, and uncertainty about the presence of radiculopathy persists, then additional radiographic and/or electrodiagnostic tests may need to be performed by the physician (Ch. 12).

Neurologic Assessment

A screening neurological examination is performed to assess for sensory, motor or reflex deficits (Ch. 12).

Sensory Testing. Sensory testing for signs and symptoms of nerve root involvement would be indicated in the presence of parasthesia in the dermatomal distribution of the nerve root (i.e., medial and lateral aspect of the upper arm—C5, thumb—C6, middle finger—C7, and ring and little fingers—C8 and T1).[4] Similarly, myotomal pain radiation in the nerve root distribution to distal muscles (C5–6 to the proximal shoulder muscle group, C7 to the triceps and pectoralis major, and C8–T1 to the distal hand intrinsics) may be factored into the clinician's reasoning.[4] Hypoalgesia may be a more valid index of nerve root involvement than hyperalgesia, which is thought to be only a reflection of severity of pain.[317,318]

Muscle Testing. Muscle testing can involve a number of false positives. The therapist's ability to determine the patient's ability to contract maximally in the presence of pain will factor into the clinician's interpretation of a manual muscle test. Research has demonstrated that muscle weakness may be due to pain inhibition that may be abolished by local anaesthetic.[319,320] Saal and Saal[321] found no statistically significant difference in outcome in low back patients with neurologic weakness or with extruded discs from the total study population. They found that the presence of muscle weakness does not adversely affect the outcome of nonoperative treatment, and should not be used as overwhelming evidence that surgery is needed.[321] Radiculopathy can be treated very successfully with aggressive nonoperative care.

Reflexes. Diminished reflexes as with muscle weakness have been shown to be abolished by local anaesthetic.[319,320] The clinician will be alert to false positives during this form of neurologic assessment.

Summary. Composite findings of radiographs and radiculopathy testing may suggest the deferral of manual therapy. Otherwise, I recommend commencing with manual therapy in the treatment of MDCS even in the presence of minimal to moderate neurologic findings, with the understanding that neurologic signs and symptoms will be reassessed prior to and following each treatment session.

Dizziness

As discussed earlier, dizziness can result from peripheral or central disorders. Ischemic dizziness that is caused by the extrinsic factor of vertebral artery involvement will need to be acknowledged before manual therapy is administered to the cervical spine. The vertebral artery test, though described different ways, would classically involve combining active or passive cervical spine rotation and extension.[322,323] The test position should be held for no less than 10 seconds so as to recognize any latent response. The patient's eyes should be observed for nystagmus and the patient should report any symptoms of dizziness. Aspinall[148] suggests a building block approach to clinical testing

that should reduce the risk to the vertebral artery. According to Aspinall, testing procedures are performed first in the sitting position, then in the lying position.[148] Positioning for the vertebral artery tests in the upper and lower cervical spine would then be performed individually for each position previously mentioned.[148] A vertebral artery test that is positive will be considered a strict contraindication for manual therapy, especially if significant cervical spondylitic changes are present on x-rays and the patient reports dizziness with certain neck positions (e.g., looking up). If a vertebral artery test is positive, with no prior history of dizziness and normal x-rays, then manual therapy may be only a relative contraindication in the hands of a skilled clinician. Questions arise about the specificity of this testing, as other tissues in the cervical spine can cause dizziness (i.e., reflex vertigo). As discussed in the section on Ischemic Vertigo, symptoms either associated or not associated with dizziness such as diplopia, drop attacks, dysarthria, and/or dysphagia are a strict contraindication to manual therapy or any physical therapy management until the etiology of such symptoms are investigated.

Upper Cervical Instability

The presence of upper cervical instability is usually suspected by history of trauma and rheumatoid arthritides.[324] Instability is confirmed by various radiographic views and or upper cervical ligament integrity testing.[325,326] However, clinicians should be skeptical when interpreting findings from ligamentous testing in the cervical spine because the reliability and validity of these tests have not been established in a clinical setting. The reader is referred to the references for additional information on upper cervical ligament integrity testing.[326,327]

MANAGEMENT OF MOVEMENT DYSFUNCTION OF THE CERVICAL SPINE

The natural history of nonspecific spinal disorders is that they usually improve spontaneously with time.[87,328,329] This known progression towards resolution has been used by doctors to support the advice of bed rest and the use of anti-inflammatories, analgesics and muscle relaxants.[330] Although there is a tendency for spinal pain to improve with time, Roland and Morris[328] concluded that up to one-quarter of patients report increasing disability during the first week after presentation, and one-quarter also report increasing disability during the subsequent 3 weeks. Given that 10 to 20 percent of patients who have a nonspecific spinal disorder do not follow the natural history of spontaneous recovery, a large number of patients continue to require treatment.[331,332] This smaller group of patients is growing and accounts for up to 85 percent of the total cost of spinal pain.[331,333] This group of symptomatic patients may progress from a simple neck pain problem to a chronic neck pain problem that becomes costly. Marbach et al.[334] speculated that because chronic

facial pain patients have a larger number of consultations and that most consultations result in no treatment or referral, chronic facial pain patients must resort to an emphasis on the sensory aspect of their complaints to obtain attention from health care providers.

Until criteria are established that will help to identify those patients who will not follow the natural history of resolution from those who do, early physical therapy intervention is suggested for the majority of patients with a nonspecific condition of the cervical spine. Often this may involve only a session on patient education pertaining to posture and activity.[330] Early conservative management can help reduce overall cost and may greatly decrease the chances that acute neck pain will become chronic. Waddell[332] states that ". . . acute and chronic pain are not only different in time scale but are fundamentally different in kind." The profile of a chronic patient is one of emotional distress, depression, failed treatment, and adoption of a sick role.[332] Early intervention by a physical therapist is strongly recommended to prevent such a catastrophe.

Research that investigates an *approach* using a variety of treatments may be more clinically relevant, as the application of one technique that will remedy the patient's total problem is rare.[335] The following are suggested treatments to be used in an *approach* to the management of an acute or chronic patient with MDCS.

Patient Education

Patient education may be the single most important area in physical therapy management of nonspecific symptoms. The first obstacle in patient education is overcoming the patient's fear, anxiety, misconception, and advice from friends about the meaning of their symptoms. Acknowledging that their feelings and confusion are real and normal may provide a level of comfort for the patient. In an essay by Norman Cousins, it is stated:[336]

> Somewhere in our early education we become addicted to the notion that pain means sickness. We fail to learn that pain is the body's way of informing the mind that we are doing something wrong, not necessarily that something is wrong.

Patients may also perceive "something to be wrong" not only from their symptoms but also from their diagnosis. A diagnosis may actually enhance the feelings of fear and anxiety, which in turn may intensify symptoms and lead the patient to believe that a cure is not available. For example, the diagnosis of "cervical arthritis" or "degenerative disc disease" may create an image of having to learn to live with pain. The diagnosis of "disc herniation" may influence some patients to believe the only cure for their symptoms is surgery. Patients need to be informed that unless there is explicit clinical criteria, their symptoms may originate from tissues other than the disc, which can be treated

conservatively. Only in selected patients have surgical procedures of discectomy or fusion demonstrated reduced pain and neurologic deficit.[273] Often, the results of surgically treated patients are less satisfactory than those of conservatively treated patients.[337] In a blinded controlled study of low back surgery, a follow-up at 4 and 10 years showed that patients who had undergone surgery were no better than those who had not.[338] Parry[339] has noted a general belief among specialists that surgery for lumbar pain syndromes has been widely overprescribed. By inference, the same concerns must be raised about cervical spinal surgery.[340] In a follow-up of nonsurgical patients the value of an educational program for potential surgery patients was demonstrated.[341] Education provided the patient with tools to make informed decisions as well as to motivate them to continue their home program and set new goals. In patients who avoided surgery, education provided the opportunity to increase activity without fear of increased pathology.[341]

Health professionals should be careful about the "label" they give the patient considering that the precise diagnoses in the majority of spinal pain patients are unknown.[87,342] Patients' interpretations of the meaning of their diagnosis along with the misconceptions associated with spinal pain suggest a great deal of patient education is needed. Patients should be told that the primary tissues that will be addressed with physical therapy are muscle and joint. Symptoms originating from nervous tissue (peripheral and central) and discogenic involvement will be helped indirectly by addressing the muscle and joint conditions. Patients need to know their symptoms may be managed by addressing the muscle and joint involvement regardless of the degenerative/discogenic involvement. Exceptions to this will be based upon the special objective findings previously covered.

Once the meaning of the diagnosis is understood by the patient, their role in the treatment process is explained to them. Improvement of symptoms and functional limitations is assisted by the patient's active participation in a treatment program. Focusing the patient's attention on ways to reduce and control abnormal stress on the cervical spine is important as unnecessary stress on the pathologic or normal cervical spine and surrounding tissues can exacerbate symptoms.[343] These stresses may occur nocturnally and diurnally. Patient awareness will be directed to sleeping and upright postures, cervical collars, and a specific and/or general exercise program.

Sleeping Postures

Primary and secondary symptoms may interfere with the patient's ability to sleep, stay asleep, or the symptoms may be present upon awakening in the morning. Nocturnal parafunctional activities often have been attributed to causing or aggravating headaches and jaw, and neck symptoms. Treatment for nocturnal parafunction has focused on the use of an intraocclusal appliance.

What should not be overlooked in controlling nocturnal symptoms is proper support to the cervical spine. The cervical spine may not only be a

source of the patient's symptoms, but also a possible cause of nocturnal parafunction, as discussed earlier in this chapter. Patient education regarding tongue-up, teeth apart, breathing, and swallowing (as covered in Ch. 7) may carry over into the night to minimize parafunction.

Sleeping positions should be supine and/or sidelying; stomach sleeping will usually exacerbate the patient's symptoms. In the supine or sidelying position, a cervical pillow will be used. The physical therapist may choose from among several cervical pillows. I have observed that pillows that are softer, with a larger diameter, are usually more therapeutic.

Supine Sleeping Posture

A cervical pillow is placed under the patient's neck. Depending upon the patient's anterior to posterior chest position and/or forward head posturing, a regular thin pillow may be placed under the cervical pillow, extending just under the patient's shoulders. (Fig. 11-18) The regular thin pillow may eventually be removed to allow the patient to be in a neutral head posture. However, the forward posturing of some patient heads may be normal; therefore, the regular pillow may always remain under the cervical pillow. Patients who have a long neck may lay with their head slightly in extension over the cervical pillow. If this is observed, a regular thin pillow should be placed under the cranium, which will support the head in a neutral position to the neck in combination with the cervical pillow.

Patients who regularly awaken with upper extremity symptoms secondary to a peripheral or central entrapment may need to have a pillow placed under their involved arm(s) while sleeping. Shoulder girdle depression and traction

Fig. 11-18. Cervical pillow used in the supine sleeping posture.

Fig. 11-19. Cervical pillow used in the sidelying sleeping posture.

through the extended arm are means of tensioning the brachial plexus and cervical nerve roots.[344] Elevating the involved arm will lessen tension of the neural tissue in the areas of potential peripheral and/or central entrapments. Placing several pillows under the patient's knees will complete the instructions in the supine sleeping posture.

Sidelying Sleeping Posture

Patients will be told to sleep on their uninvolved side. The cervical pillow will be placed under the cervical spine. (Fig. 11-19) Depending on the width of the patient's shoulders, a regular thin pillow will be placed under the cervical pillow. Depending on the length of the cervical spine, another regular thin pillow will be positioned behind the cervical pillow to support the head. A pillow or two should be placed under the top arm so as to decrease neural tissue tension. A pillow or two between the knees will complete the instructions in the sidelying sleeping posture.

Difficulty in these positions may be due to cervical restrictions that are advanced and cannot accommodate to the supported neck position. If such a situation occurs, manual therapy to the cervical spine for several treatment sessions should be offered first, followed by the use of the cervical pillow.

Upright Postures

Upright postures will focus on the relationship between the (HNS) areas. HNS postures can vary in the sagittal, horizontal and transverse planes. As discussed earlier, several studies showed no correlation between neck–shoul-

der–thoracic posture and symptoms.[302,303] This does not decrease the importance of educating the patient on postural concepts. Sedentary habits, poorly equipped work sites that result in prolonged positioning in poor postural alignment, and lack of postural awareness may be responsible for poor posture.[303] Tendencies to "yield to gravity" may become more pronounced with age.[345]

The easiest and most effective way for me to quickly educate the patient on the awareness of avoiding poor posture is instructing the patient on "chest up." The patient will visualize a string extending from their upper chest. (Fig. 11-17) The patient will be told to follow the string with their chest. By visualizing the string and the resulting chest position, the patient should be able to avoid a slumped HNS posture during the majority of activity carried out in the upright posture.

Avoiding the poor HNS posture in the sitting position is more difficult for the patient. Static sitting tends to allow gravity to force the patient away from an upright posture. The physical therapist will need to educate the patient about proper chair support and perhaps offer one of the various low back pillow supports that are available. (Fig. 11-20) Supporting the lumbar spine helps to avoid a poor HNS posture. Depending on the patient's work environment and job description, instructing the patient in the use of a pillow or two under their

Fig. 11-20. Low back pillow used in the sitting posture.

Fig. 11-21. Low back pillow and pillows under the arms used in the sitting posture.

arms will reduce the tractional force of the arms and shoulders on the neck. Elevating the arms will also decrease neural tissue tension. If pillows under the arms are not practical at work, the pillows can be placed under the arms at home while reading, watching television, etc. (Fig. 11-21) The patient will be told not to watch television or read in bed.

Manual Therapy

Manual therapy consisting of palpation, passive mobility testing, and manual traction was incorporated into the physical examination to assist in the diagnosis of MDCS. Manual therapy will now be discussed as a treatment tool to help in managing the symptoms and dysfunction associated with MDCS.

Manual therapy may consist of hands-on repetitive oscillations, steady stretch or high velocity thrusts of joints, or the application of various forms of soft tissue massage, muscle stretching, or shortening.[346] The potential therapeutic value in manual therapy evolves around the mechanical, neurologic, neurophysiologic and psychological effects of manual therapy.[346–353]

A comprehensive understanding of clinical and scientific viewpoints on manual therapy from clinicians and researchers can be obtained from the references.[346–353]

Clinical discussions pertaining to the cause and effect of mechanical, neurologic, and neurophysiologic involvement of a tissue, the identification of a tissue, and optimal techniques or treatments are diversified.[354] Grieve[355] describes the present state of manual therapy:

> We continue to sound as though we know so much, when we know comparatively little. It might be a good thing to admit this. We make much of clinical science, enthusiastically referring to this or that part of the massive mountain of literature which best serves our particular interest. . . . Much of what we do is simply what has been proven on the clinical shop floor to be effective in getting our patients better—we do not always know why.[355]

Therapeutic outcome resulting from the use of manual therapy does not necessarily mean that the tissue(s) causing the patient's symptoms have been identified.[356] The spine is a multisegmental system in which tissues work together and potentially fail together through either insidious or traumatic onsets. Singular tissue involvement is the exception rather than the rule. Clinical reasoning used to incriminate a tissue must be kept in proper perspective since the evaluation tools and manual procedures used by physical therapists have not been shown to be completely reliable and valid.[284,306] Steps have and continue to be taken to document the efficacy of the evaluation and treatment using manual therapy. DiFabio[356] in a review of the literature, identified 14 studies that were judged to be valid demonstrations of the efficacy of manual therapy in the treatment of patients who have somatic pain syndromes. DiFabio concluded there was clear evidence to justify the use of manual therapy in the treatment of patients who have back pain.[356] Recently a study has documented that a trained manual therapist is as objective and sensitive as controlled diagnostic blocks in identifying cervical facet joint dysfunction.[45]

Clinicians treat patients on a day-to-day basis and are looking for new directions in improving functional outcomes in patient care. More than we care to acknowledge, clinicians often treat by recalling what worked best for one patient or group of patients and applying that treatment approach to a current patient of similar history and physical findings. Guidelines in research should consider these clinical observations.

In an article by Shepard et al.,[357] entitled "*Alternative Approaches to Research in Physical Therapy: Positivism and Phenomenology,*" two philosophical approaches to research in physical therapy were discussed.[357] This article attempted to detail the subtle philosophical issues of quantitative (positivism) and qualitative (phenomenology) research methods in a comprehensive way. Justice to the contents of this article cannot be done in a few paragraphs. A brief definition of these two different philosophical perspectives is appropriate because they may assist the clinician's ability to formulate and research clinical observations.

The philosophical orientation of positivism is associated with empiricism.[357] The article of Shepard and co-workers quoted Polit and Hungler[358] defining positivism as:

> . . . the process whereby evidence rooted in objective reality and gathered directly or indirectly through the human senses is used as a basis for generating knowledge.

Examples of empirical evidence include heart rate, leg length discrepancy, and stride length. Cause and effect relationships are best researched through the positivistic perspective.[357]

Phenomenology tries to understand social phenomena or human activity from the viewpoint of the person being studied.[357] Reality is believed to be socially constructed by the individual, and thus multiple realities exist, not a finite number of objective truths.[357] In defining multiple realities, Sheppard and co-workers quoted Merriam as saying:

> The world is not an objective thing out there but a function of personal interaction and perception. It is a highly subjective phenomenon in need of interpreting rather than measuring. Beliefs rather than facts form the basis of perception.[359]

In a commentary to Shepard et al., Mattingly states:

> Often it is not enough to fix the "body" of the patient. Therapists are also asking the client to become an active participant in therapy, to practice exercises at home, to "buy on" to the goals of therapy. They may even set goals collaboratively, trying to individualize treatment to meet the needs and lifestyles of their clients. Phenomenological approaches have much to offer health practices, especially those such as physical therapy, whenever it matters not only what a patient's physical dysfunctions are but how those dysfunctions affect the patient's life. Put differently, phenomenological research is essential whenever the health professional must address the patient's 'illness experience'."[360] ". . . If treatment is 'treating a life,' so to speak, and not just a body, then it is important to do research that illuminates the phenomenological aspects of treatment. . . ."[360]

In order to document if manual therapy is better than doing nothing at all, both philosophical approaches to research in physical therapy will need to be addressed. *A classification system that offers consistency to an approach in treatment would be insignificant in manual therapy unless the physical therapist had the ability to modify the approach based upon the individual needs of the patient.* What follows is one of multiple manual therapy treatment approaches. Manual therapy by itself, without involving the other forms of management that have and will be discussed, will not be very effective. I have favored the following manual therapy approach for some time.

Three-Dimensional Movement Techniques

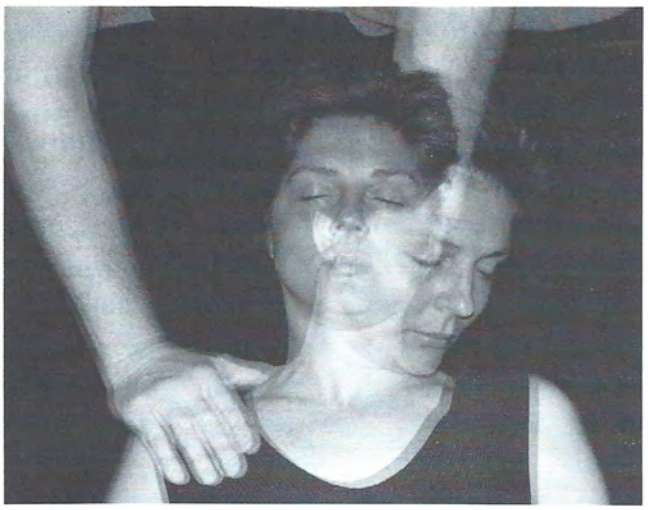
The identification/isolation of a spinal tissue is difficult if not impossible to achieve. Even if the tissue can be identified, it is difficult to know if the treatment is specific to the named tissue. This often leads to a major obstacle in deciding where to begin with a manual therapy approach. The inability to identify and name the tissue(s) does not imply that manual therapy cannot be specific to the dysfunction (restrictions) of a tissue(s) that is causing the patient's symptoms. The naming of a specific tissue (i.e., right upper trapezius muscle, left C2–3 facet joint, right GON or right C5 nerve root) for the majority of patients experiencing MDCS is often not necessary unless a positivistic perspective is strongly suggested from the history and physical examination.

The *initial* approach taken by this author is to use techniques that address *restrictions in movement patterns* of the cervical spine. The techniques are referred to as "three-dimensional movement techniques." Three-dimensional movement techniques are used when there is active and/or passive restrictions contributing to the patient's symptoms and dysfunction. The goal of three-dimensional movement techniques is to improve upon *passive functional movement patterns* that may result in the improvement of *symptoms and active functional movement patterns*.

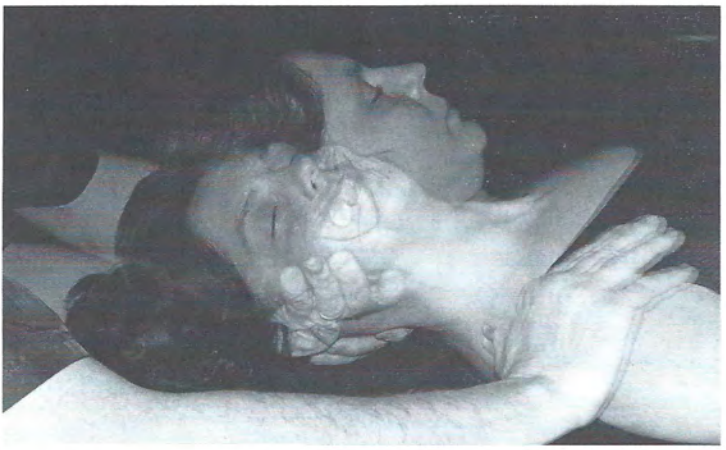
Before applying three-dimensional movement techniques or other manual therapy techniques, the following criteria is needed: (1) MDCS is present, identified by the history and physical examination; and (2) Acknowledge the presence of "special findings" that may modify the three-dimensional movement techniques.

The three-dimensional movement techniques involve any one or combination of *rotational, diagonal,* and *spiral* movement patterns. To detail hand placement, direction of movement, speed of movement, amount of stretch, duration of stretch, etc., is not the intent of this text. The reader is referred to the photographs in Figure 11-22 to obtain a general idea of the movement patterns created with these techniques. The application of these techniques and others by a skilled therapist will allow for the identification and treatment of specific or general restrictions of the tissue(s). During the application of the three-dimensional movement techniques, a continuum of treatment and reassessment occurs. The application of three-dimensional movement techniques may be all that is needed with some patients to manage symptoms and dysfunction associated with MDCS. For other patients these techniques will facilitate the application of other appropriate manual or medical (i.e., injections) techniques that are directed towards a named tissue or to a specific or general restriction that was not resolved with three-dimensional movement techniques.

Fig. 11-22. **(A–H)** Three-dimensional movement techniques involving any one or combination of *rotational, diagonal* and *spiral* movement patterns. **(A)** Upper arc. **(B)** Lower arc. **(C)** Melt. (*Figure continues.*)

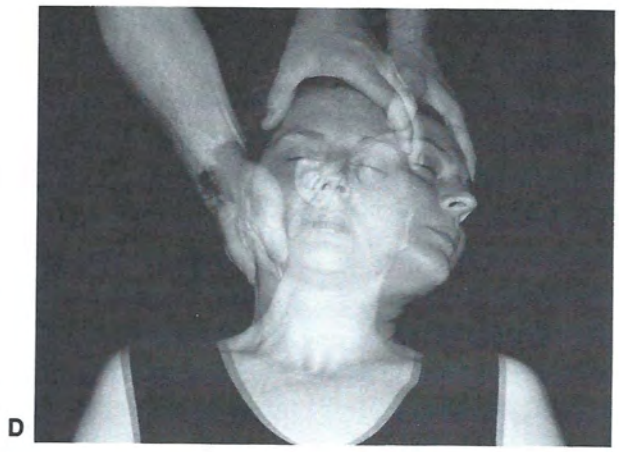


A

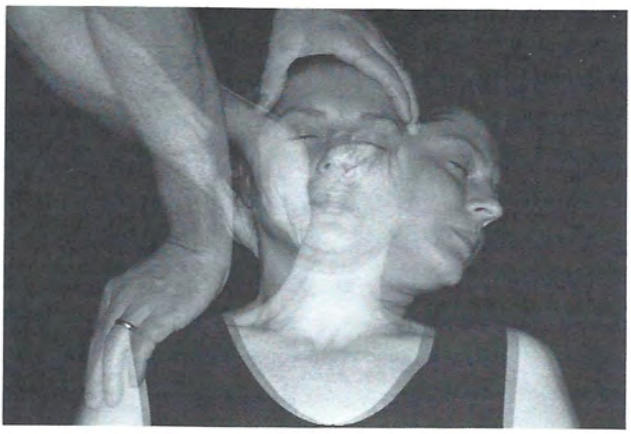


B

C



D



E

F

Fig. 11-22. (*Continued*) **(D)** Melt with thumb. **(E)** Melt with thumb cross over arm. **(F)** Bilateral lift. (*Figure continues.*)

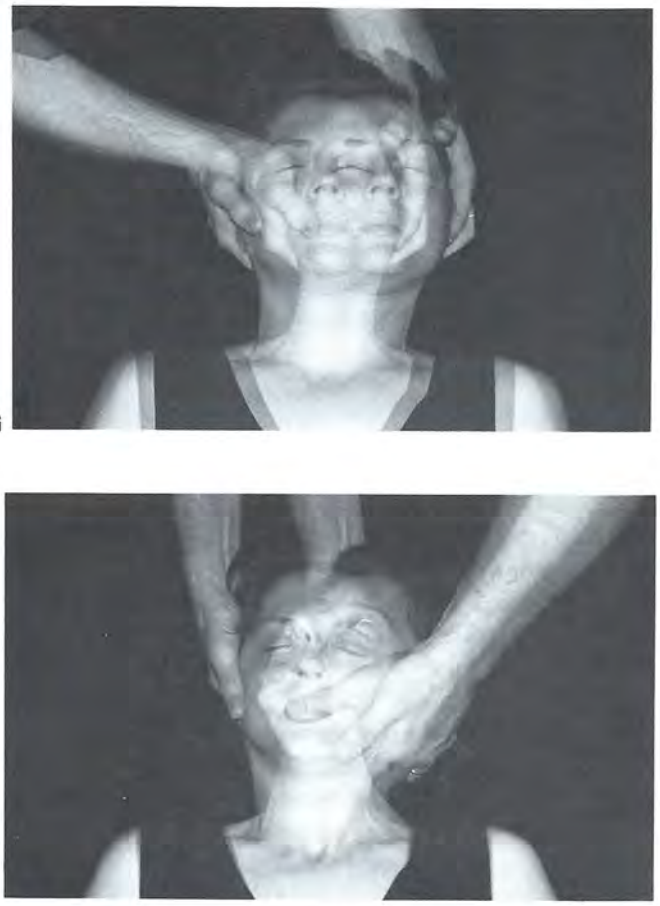


Fig. 11-22. (*Continued*). **(G)** Lateral arc. **(H)** Gap. The names indicate the movement patterns through which the cervical spine is moved. These photographs are not intended to detail hand placement, direction of movement, speed of movement, amount of stretch, or duration of stretch. The application of these techniques by a skilled therapist will allow for the identification and treatment of specific or general restrictions of the tissue(s). During the application of the three-dimensional movement techniques, a continuum of treatment and reassessment occurs.

Traction

Traction is often indicated when radiculopathy is suspected. The objective of traction is to separate the posterior intervertebral space in the presence of a compressed or irritated nerve root. When possible, manual therapy should be rendered over several treatment sessions before offering traction. Manual therapy will help to prepare the patient for traction by decreasing muscle and soft tissue tone and by improving the mobility of facet joints.

Fig. 11-23. Patient set up for the application of mechnical traction. Note the thin pillow positioned under the patient's shoulders and traction unit. Extra foam may be placed under the mastoid processes for the patient whose tissues are sensitive in this area and/or who is receiving a higher poundaged pull.

Traction can be applied through a variety of different cervical/head harnesses using weight or mechanical machinery. Mechanical traction using a head halter as shown in Figure 11-23 is my preferred way to ensure that a force of traction is occurring in the cervical spine. If the patient is set up correctly, minimal forces from the traction unit would arrive at the mandible and thus the TMJ. In my view, any home traction units or head halters other than that in Fig. 11-23 may not achieve the objectives of traction.

Colachis and Strohm[361,362] found that a traction force of 30 lbs for a duration of only 7 seconds can separate the cervical intervertebral space with the amount of separation increasing with flexion. Wong and co-workers found that traction either in the horizontal neck position or flexed to 30 degrees provided significant separation of the intervertebral space from C3–4 to C6–7 measured posteriorly and anteriorly.[363] Posterior intervertebral separation for all levels was best in the horizontal head position. The traction force used was 13.5 kg (approximately 30 lbs) for a duration of 8 seconds followed by unloading for 6 seconds alternately for a period of 20 minutes.[363] Traction was preceded by application of a heat pack. Clinically, a force of 20 to 25 lbs for a duration of 30 seconds and unloading for less than 5 secs for 15 minutes is therapeutic.

Cervical Collars

Cervical collars are seldom required in the treatment of MDCS, except in known traumatic injury. The most common traumatic injury is usually associated with a whiplash.[364] Cervical whiplash represents a collection of symp-

toms following injury to the neck, usually hyperextension/flexion occurring as a result of a rear-end accident.[365] The pathology of whiplash is poorly understood and described because the forces involved vary from incident to incident and fresh post-mortem cervical spines are not readily available for study.[364,366] Other than the apparent soft tissue and joint injury, Twomey[77] and Osti[367] describe "rim lesions" that may result from a whiplash injury. A rim lesion is best described as clefts in the cartilage plates and annulus fibrosus.[77,89] The clefts are in the peripheral part of the disc, near the vertebral rim and usually parallel with the end-plate.[77] Rim lesions principally involve the avascular cartilage plate, but have been observed to extend into the outer annulus, which contains nerves and blood vessels, and into the bony end-plate which is highly vascular.[77,89,368] Patients experiencing a significant whiplash injury may need to be supported with a collar. Twomey and Taylor state[77]; "It is essential that there be an early period of immobilization of the neck to allow for resolution of effusion and to ensure that bleeding into the damage areas ceases." However, when using a cervical collar, the importance of early mobility cannot be overlooked so as to avoid secondary problems associated with immobility. Patients who have become dependent upon the collar present with additional clinical concern. Questions as to how long the neck should be immobilized are largely an individual matter between patient and attending clinician. Collars should be designed and worn with the narrow section in front of the neck so that cervical hyperextension is avoided.[369]

Exercise

The passive application of modalities and manual therapy do not replace the need for a specific and general exercise program in the prophylaxis and treatment of nonspecific spinal disorders. Application of modalities and manual therapy procedures may often need to precede the initiation of an exercise program in order to decrease symptoms and increase passive and active mobility.

It is very clear that disuse has harmful affects on skeletal health.[370] Twomey indicates that bone and muscle both respond positively to exercise and adversely to disuse.[46] There is an increasing body of evidence supporting the value of regular exercise in preserving skeletal health.[46] Shepard[371] and Menard and Stanish,[372] indicate that the reduction in muscle mass that occurs with increasing age is due to disuse and can be substantially reversed by a program of activity. Pardini[373] has demonstrated that most elderly Americans can show up to 50 percent improvement in muscle strength after a relatively short exercise program. Aniansson and others[374] show that it is possible to increase muscle strength, endurance, and hypertrophy into old age. Aloia et al.[375] reported a significant increase in total body calcium in postmenopausal women who exercised regularly for 1 year, in contrast with a decline in total body calcium in a matched sedentary group. These findings were later supported by the work of Smith et al.[376]

Strength, endurance, and flexibility are important but without coordination

and proper recruitment of motion, spinal symptoms may still occur or reoccur. Irregular movement patterns with or without poor posture may contribute to repetitive microtrauma of cervical structures, including muscles, facet joints and nervous tissue.[377,378] Poor movement patterns contribute to habitual overuse of isolated motion segments while they minimize normal movement at others.[379] Patients experiencing pain originating from MDCS will require a comprehensive exercise program. Exercise programs will need to be individualized and consistent with personal goals.

SUMMARY

Muscles, facet joints, peripheral nerves, and nerve roots can cause neck, shoulder, and upper extremity symptoms. Each of these can prove to be a major source of cephalic symptoms, mimicking symptoms believed to be originating from a temporomandibular disorder and or from a masticatory muscle disorder. Cervical spine involvement needs to be acknowledged in patients who receive dental intervention such as equilibration, intraoral appliance, orthodontics, or orthognathic/joint surgical procedures. Dental intervention may need to come during or after the management of the cervical spine involvement.

The dilemma surrounding the inability to diagnose nonspecific spinal complaints is very real. Although it is preferable for a diagnosis/classification of a nonspecific spinal condition to be based on a unitary principle (i.e., anatomical [bone spur], pathologic [disc degeneration], or etiologic [cervical sprain], this is not always possible.[87] A diagnosis rarely exists as a pure entity explaining the patient's condition.[87] Caution therefore should be taken when the clinician, patient, and third party payers place too much emphasis on the medical diagnosis for a nonspecific condition of the spine.

Treatment strategies for nonspecific lumbar complaints based upon signs and symptoms are currently being developed. I propose a classification for nonspecific disorders of the cervical spine referred to as "Movement Dysfunction of the Cervical Spine." The pertinent history and criteria of physical examination that are needed to derive at the diagnosis of "Movement Dysfunction of the Cervical Spine" is outlined.

Management of movement dysfunction of the cervical spine begins with patient education to resolve the myths surrounding a specific condition and diagnosis. Patient education regarding proper sleeping and upright postures is vital so as to avoid further unnecessary stress to the cervical spine and associated structures. The therapeutic value of manual therapy, supported as needed with modalities, will decrease symptoms and improve function. Traction would be indicated when radiculopathy is present. Establishing a specific and/or general exercise program for individual patients of all ages will promote a more functional and independent quality of life.

ACKNOWLEDGMENT

I would like to thank Carolyn Law, M.P.T., for her invaluable assistance with manuscript preparation.

REFERENCES

1. Kelsey JL: Epidemiology of Musculoskeletal Disorders. p. 146. Oxford University Press, New York, 1982
2. Gore D, Susan B, Gardner G et al: Neck pain: a long-term follow-up of 205 patients. Spine 12:1, 1987
3. Accident Facts—1980 Edition. Chicago, National Safety Council, 1980
4. LaBan M: "Whiplash": its evaluation and treatment. p. 293. In: Saal J (ed): Physical Medicine and Rehabilitation: Neck and Back Pain: State of the Art Reviews, V. 4, No 2, Hanley & Belfus, Philadelphia, 1990
5. Insurance Bureau of Canada: Fact Book. p. 20. 15th Ed. Toronto, Canada, 1987
6. Ameis A: Cervical whiplash: consideration in the rehabilitation of cervical myofascial injury. Can Fam Phys 32:1871, 1986
7. Hodson SP, Grundy M: Whiplash injuries: their long-term prognosis and the relationship to compensation. Neuro-Orthop 7:88, 1989
8. Dvorak J, Valach L, Schmid S: Cervical spine injuries in Switzerland. Manual Med 4:7, 1989
9. Quarrier N: Performing arts medicine: the musical athlete. JOSPT, 17:90, 1993
10. Holmstrom E, Lindell J, Moritz: Low back and neck/shoulder pain in construction workers: occupational workload and psychosocial risk factors. Part 2: relationship to neck and shoulder pain. Spine 17:672, 1992
11. Hagberg M, Wegman D: Prevelance rates and odds ratios of shoulder-neck diseases in different occupational groups. Br J Ind Med 44:602, 1987
12. Leino P: Symptoms of stress predict musculoskeletal disorders. J Epidemiol Community Health 43:293, 1989
13. Linton S: Risk factors for neck and back pain in a working population in Sweden. Work and Stress 4:41, 1990
14. U.S. Department of Health and Human Services, Public Health Service, Center for Disease Control, National Institute for Occupational Safety and Health, HETA 89-299-2230 U.S. West Communications, July 1992
15. Rundcrantz B, Johnsson B, Mortiz U: Pain and discomfort in the musculoskeletal system among dentist. Swed Dent J 15:219, 1991
16. Goldenberg DL: Fibromyalgia syndrome: An emerging but controversial condition. JAMA 2783, 1987
17. Friction J: Myofascial pain syndrome, Neuro Clin 7:413, 1989
18. Awad E: Interstitial myofibrositis: hypothesis of mechanism. Arch Phys Med Rehabil 54:449, 1973
19. Thompson J: Subspecialty clinics: physical medicine and rehabilitation, Mayo Clin Pro 65:1237, 1990
20. Yunnus M, Kalyan-Raman U, Kalyan-Raman K: Primary fibromyalgia syndrome and myofascial pain syndrome: Clinical features and muscle pathology, Arch Phys Med Rehabil 69:451, 1988
21. Travell J, Simons D: Myofascial pain and dysfunction, the trigger point manual. New York: Williams & Wilkins, New York, 1983
22. Travell JG: On myofascial pain. J Clin Orthod 23:468, 1989
23. Bogduk N: Headaches and the cervical spine (editorial). Cephalalgia 4:7, 1984
24. Simons D: Muscular pain syndromes. Adv Pain Res Ther 17:1, 1990
25. Bennett R: Myofascial pain syndromes and the fibromyalgia syndrome: a comparative analysis. Adv Pain Res Ther 17:43, 1990
26. Campbell S: Regional myofascial pain syndromes. Rheum Clin North Am 15:31, 1989

27. Simons D: Myofascial pain syndromes: where are we? where are we going? Arch Phys Med Rehabil 69:207, 1988
28. Thompson J: Tension myalgia as a diagnosis at the Mayo Clinic and its relationship to fibrositis, fibromyalgia and myofascial pain syndrome. Mayo Clin Proc 65:1237, 1990
29. Yunas M, Masi A, Calabro J et al: Primary fibromyalgia (fibrositis): clinical study of 50 patients with matched normal controls. Semin Arthritis Rheum 11:151, 1981
30. Bennett R: Fibrositis: Does it exist and can it be treated? J Musculoskel Med, 57, 1984
31. Bennett R: Fibromyalgia. JAMA, 257:2802, 1987
32. Wolfe F: Fibrositis, fibromyalgia, and musculoskeletal disease: the current status of the fibrositis syndrome. Arch Phys Med Rehabil, 69:527, 1988
33. Hubbard D, Gregory B: Myofascial trigger points show spontaneous needle EMG activity. Spine 18:1803, 1993
34. Kent B: Anatomy of the trunk. Phys Ther, 1974
35. Hayashi K, Yabuki T: Origins of the uncus and of Von Luschka's joint in the cervical spine. J Bone Joint Surg, 67A:788, 1985
36. Carrera G, Williams A: Current concepts in evaluation of the lumbar facet joints. CRC Crit Rev Diagn Imaging 21:85, 1984
37. Mooney R: The facet syndrome. Clin Orthop 115:149, 1976
38. Dory A: Arthrography of the lumbar facet joints. Radiology 140:23, 1981
39. Leyshon A, Kirwan E, Parry C: Electrical studies in diagnosis of compression of the lumbar root. J Bone Joint Surg 36:71, 1981
40. Wyke BD: Neurology of the cervical spinal joints. Physiotherapy 65:72, 1979
41. Wyke BD, Polacek P: Articular neurology: the present position. J Bone Joint Surg 7B:401, 1975
42. Bogduk N, Marsland A: The cervical zygapophyseal joints as a source of neck pain. Spine 13:610, 1988
43. Dwyer A, Aprill C, Bogduk N: Cervical zygapophyseal joint pain patterns I: a study in normal volunteers. Spine 15:453, 1990
44. Aprill C, Dwyer A, Bogduk N: Cervical zygapophyseal joint pain patterns II: A clinical evaluation. Spine 15:458, 1990
45. Jull G, Bodguk N, Marsland A: The accuracy of manual diagnosis for cervical zygapophyseal joint pain syndrome. Med J Aus 148:223, 1988
46. Twomey L: A rationale for the treatment of back pain and joint pain by manual therapy. Phys Ther 72:885, 1992
47. Mercer S: The menisci of the cervical synovial joints. p. 109. Proceedings from the Fourth Biennial Conference of Manipulative Therapist Association of Australia, Brisbane, May 22–25, 1985
48. Bogduk N, Engel R: The menisci of the lumbar zygapophyseal joints: a review of their anatomy and clinical significance. Spine 9:454, 1984
49. Kos J, Wolfe J: Les menisques intervertebraux et leur role possible dans les blocages vertebraux. Ann Med Phys 15:203, 1972
50. Engel R, Bogduk N: The menisci of the lumbar zygapophyseal joints. J Anat 135: 795, 1982
51. Kos J, Wolf J: Die "Menisci" der Zwischenwirbelgelenke und ihre mogliche rolle lei Wirbelblockierung. Manuelle Medizine, 10:105, 1979
52. Yu S, Sether L, Haughton V: Facet joint menisci of the cervical spine: correlative MR imaging and cryomicrotomy study. Radiology 164:79, 1987
53. Lewin T, Moffett B, Viidik A: The morphology of the lumbar or synovial intervertebral joints. Acta Morphol Neerl-Scand 4:299, 1962

54. Lewit K: The contribution of clinical observation to neurobiological mechanisms in manipulative therapy. p 3. In Korr I (ed): The Neurobiologic Mechanism In Manipulative Therapy. Plenum Press, New York, 1978
55. Droz-Georget JH: High-velocity thrust and pathophysiology of segmental dysfunction. In Idczak RM (ed): Aspects of Manipulative Therapy. Proceedings of a Multidisciplinary International Conference on Manipulative Therapy. Melbourne, August 1979
56. Kopell H, Thompson W: Peripheral Entrapment Neuropathies. Williams & Wilkins, Baltimore, Maryland, 1963
57. Pratt N: Neurovascular entrapment in the regions of the shoulder and posterior triangle of the neck. Phys Ther 66:1894, 1986
58. Riddel D, Smith B: Thoracic and vascular aspects of thoracic outlet syndrome. Clin Orthop 207:31, 1986
59. Cuetter A, Bartoszek D: The thoracic outlet syndrome: controversies, overdiagnosis, overtreatment, and recommendations for management. Musc and Nerve, 410, 1989
60. Sunderland S: Traumatized nerves, roots and ganglia: musculoskeletal factors and neuropathological consequences. Editor Korr I. In Korr I (ed): The Neurobiologic Mechanisms in Manipulative Therapy. Plenum Press, New York, 1978
61. Sunderland S: Nerves and Nerve Injuries, 2nd Ed. Churchill Livingstone, New York, 1979
62. Schaumburg H, Spencer P: Pathology of spinal root compression. p 141. In Goldstein M (ed): The Research Status of Spinal Manipulative Therapy, U.S. Department of Health, Education, and Welfare. NINCDS Monograph No. 15, DHEW Publication No (NIH) 76-998, Feb 2–4, 1975
63. Seddon H: Three types of nerve injuries and repair. In Microsurgery Workshop Symposium. CV Mosby, St Louis, 1975
64. Bogduk N: The clinical anatomy of the cervical dorsal rami. Spine 7:319, 1982
65. Cox C, Cocks G: Occipital neuralgia. J Med Assoc State Ala 48:23, 1979
66. Hunter C, Mayfield F: Role of the upper cervical roots in the production of pain in the head. Am J Surg 78:743, 1949
67. Saadah H, Taylor F: Sustained headache syndrome associated with tender occipital nerve zones. Headache 27:201, 1987
68. Jansen J, Markakis E, Rama B, Hildebrandt J: Hemicranial attacks or permanent hemicrania—a sequel of upper cervical root compression. Cephalalgia 9:123, 1989
69. Perelson N: Occipital nerve tenderness: a sign of headache. South Med J 40:653, 1947
70. Bovim G, Bonamico L, Fredriksen T et al: Topographic variations in the peripheral course of the Greater Occipital Nerve; autopsy study with clinical correlations: Spine 16:475, 1991
71. Sjaastad O, Fredriksen T, Stolt-Nielsen A: Cervicogenic headache, C2 rhizopathy, and occipital neuralgia: a connection? Cephalalgia 6:189, 1986
72. Hildebrandt J, Jansen J: Vascular compression of the C2 and C3 roots—yet another cause of chronic intermittent henicrania? Cephalalgia 4:168, 1984
73. Rosenberg W, Swearingen B, Poletti C. Contralateral trigeminal dysaesthesias associated with second cervical nerve compression: a case report. Cephalalgia 10: 259, 1990
74. Sunderland S: Anatomical perivertebral influences on the intervertebral foramen. p. 129 In: The Research Status of Spinal Manipulative Therapy. U.S. Department of Health, Education, and Welfare. NINCDS Monograph No. 15, DHEW Publication No (NIH) 76-998, Feb 2–4, 1975

75. Dillin W, Booth R, Cuckler J et al: Cervical Radiolopathy. A review. Spine 11: 988, 1986
76. Mixter W, Barr J: Rupture of the intervertebral disc with involvement of the spinal canal. N Engl J Med 211:210, 1934
77. Twomey L, Taylor J: The whiplash syndrome: pathology and physical treatment. J Man Manipul Ther 1(1):26, 1993
78. Taylor J: Growth and Development of Human Intervertebral Disc. PhD Thesis, University of Edinburgh, 1973
79. Kramer J: Intervertebral disc lesions: Causes, Diagnosis, Treatment and Prophylaxis. Georg Thieme Verlag, Stuttgart, 1981
80. Bowden R: Cervical spondylosis. Proceedings of the Royal Society of Medicine, 59:62, 1966
81. Twomey L, Taylor J: Joints of the middle and lower cervical spine: age changes and pathology. In Jones H, Jones M, Milde M (eds): Sixth Biennial Conference Proceedings, Manipulative Therapists Association of Australia, Adelaide, South Australia, 1989
82. Lawrence J: Disc degeneration: its frequency and relationship to symptoms. Ann Rheum Dis 28:121, 1969
83. Parke W: Correlative anatomy of cervical spondylotic myelopathy. Spine 13:831, 1988
84. Friedenberg ZB, Edeiken J, Spencer HN et al: Degenerative changes in the cervical spine. J Bone Joint Surg 41A:61, 1959
85. Juntura E, Raininko R, Videman T et al: Evaluation of cervical disc degeneration with ultralow field MRI and Discography. Spine 14:616, 1989
86. Brain L, Wilkinson M: Cervical Spondylosis and other disorders of the cervical spine. WB Saunders, Philadelphia, 1967
87. Spitzer WO, LeBlance F, Dupuis M: Scientific approach to the asessment and management of activity-related spinal disorders: a monograph for clinicians. Report of the Quebec Task Force on Spinal Disorders. Spine 12(suppl 7):51, 1987
88. Mendel T, Wink C, Zimny M: Neural elements in human cervical intervertebral disc. Spine 17:132, 1992
89. Taylor J, Twomey L: Acute injuries to cervical joints. Spine 18:1115, 1993
90. Bogduk N: Cervical causes of headache and dizziness. p. 289. In Grieve G (ed): Modern Manual Therapy. Churchill Livingstone, Edinburgh, 1986
91. Everett N: Functional Neuroanatomy 6th ed, Lea & Febiger, Philadelphia, 1972
92. Kerr F: Mechanism, diagnosis and management of some cranial and facial pain syndromes. Surg Clin N Am 43:951, 1963
93. Kerr F, Olafson R: Trigeminal and cervical volleys. Arch Neurol 5:171, 1961
94. Kerr F: A mechanism to account for frontal headache in cases of posterior fossa tumors. J Neurosurg 18:605, 1961
95. Kerr F: Structural relation of the trigeminal spinal tract to upper cervical roots and the solitary nucleus in the cat. Exp Neurol 4:134, 1961
96. Cyriax J: Rheumatic Headache. Br Med J 2:1367, 1938
97. Campbell D, Parsons C: Referred head pain and its concomitants. J of Nervous and Mental Diseases 99:544, 1944
98. Feinstein B, Langton J, Jameson R et al: Experiments on referred pain from deep somatic tissues. J Bone Joint Surg 36A:981, 1954
99. Bogduk N, Marsland A: C3 Headaches. Paper presented at the International Meeting on Pain and Regional Anaesthesia. Australasian Pain Society, Perth, February, 1983

100. Bogduk N, Corrigan B, Schneider G et al: Cervical headache. Med J Australia 143:202, 1985
101. McNeill C (ed): Temporomandibular Disorders: Guidelines for classification, assessment, and management. The American Academy of Orofacial Pain. Quintessence Pub. Co., 1993
102. Ad Hoc Committee on the Classification of Headache. JAMA 179:717, 1962
103. Classification and Diagnostic Criteria for Headache Disorders, Cranial Neuralgias and Facial Pain. First Ed. International Headache Society. Cephalagia 8(suppl 7): 1988
104. Edmeads J: The cervical spine and headache. Neurology 38:1874, 1988
105. Barré JA: Le syndrome sympathique posterior. Rev Neurol (Paris) 33:248, 1926
106. Bartschi-Rochiax W. Headache of cervical origin. p. 192. In Vinken PJ, Bruyn GW (eds): Handbook of Clinical Neurology. Vol 5. Headache and cranial neuralgias. North Holland Publ Co., Amsterdam, 1968
107. Sjaastad O, Saunte C, Hovdahl H et al: "Cervicogenic" headache. An hypothesis. Cephalalgia 3:249, 1983
108. Pfaffenrath V, Dandekar R, Pollmann W: Cervicogenic headache—the clinical picture, radiological findings and hypotheses on its pathophysiology. Headache, 27:495, 1987
109. Fredriksen T, Hovdal H: "Cervicogenic headache": clinical manifestation. Cephalalgia 7:147, 1987
110. Pfaffenrath V, Dandekar R, Mayer E et al: Cervicogenic headache: Results of computor-based measurements of cervical spine mobility in 15 patients. Cephalalgia 8:45, 1988
111. Fredriksen T, Wysocka-Bakowska M, Bogucki A et al: Cervicogenic headache—pupillometric findings. Cephalalgia 8:93, 1988
112. Fredriksen T: Cervicogenic headache: the forehead sweating pattern. Cephalalgia 8:203, 1988
113. Sjaastad O, Fredriksen T, Sand T et al: Unilaterality of headache in classic migraine. Cephalalgia 9:71, 1989
114. Fredriksen T, Fougner R, Tangerud A et al: Cervicogenic headache. Radiological investigations concering head/neck. Cephalalgia 9:139, 1989
115. Jaeger B: Are "cervicogenic" headaches due to myofacial pain and cervical spine dysfunction? Cephalalgia 9:157, 1989
116. Farina S, Granella F, Malferrari et al: Headache and cervical spine disorders: Classification and treatment with transcutaneous nerve stimulation. Headache 26: 431, 1986
117. Ziegler DK, Hassanein R, Hassanein K: Headache syndromes suggested by factors analysis of symptom variable on a headache prone population. J Chronic Dis 25: 353, 1972
118. Rose F: Headache: definition and classification. p. 1. In Rose F (ed): Handbook of Clinical Neurology. Vol 4 (48): Headache. Elsevier, NY, 1986
119. Appenzeller O: Pathogenesis of vascular headache of the migrainous type: The role of impaired central inhibition. Headache 15:177, 1975
120. Rose F, Capildeo R: Migraine: definition and classification. Cephalalgia 3:225, 1983
121. Walters E: The epidemiological enigma of migraine. Int J Epidemiol 2:189, 1973
122. Bakal D, Kaganov J: Muscle contraction and migraine headache: Psychophysiologic comparison. Headache 17:208, 1977
123. Olesen J: Some clinical features of the acute migraine attack. An analysis of 750 patients. Headache 18:268, 1978

124. Kaganov J, Bakal D, Dunn B: The differential contribution of muscle contraction and migraine symptoms to problem headache in the general population. Headache 157, 1981
125. Carlsson J, Fahlcrantz A, Augustinsson L: Muscle tenderness in tension headache treated with acupuncture or physiotherapy. Cephalalgia 10:131, 1990
126. Sjaastad O: Cluster headache. p. 217. In Vinken PJ, Bruyn G, Klawans H, Rose F (eds): Handbook of Clinical Neurology. Vol. 4. Elsevier, Amsterdam, 1981
127. Jensen O, Nielsen F, Vosmar L: An open study comparing manual therapy with the use of cold packs in the treatment of post-traumatic headache. Cephalalgia 10: 242, 1990
128. Jensen O, Justesen T, Nielsen F et al: Functional radiographic examination of the cervical spine in patients with post-traumatic headache. Cephalalgia 10:295, 1990
129. Frykholm: Clinical picture. p. 5. In Hirsch, Zotterman (eds): Cervical Pain. Pergamon Press, Oxford, 1971
130. Smith D: Dizziness, a clinical perspective. Neurol Clin 8:199, 1990
131. Adams R, Victor M: Principles of Neurology. 3rd Ed. McGraw-Hill, New York, 1985
132. Brown J: A systemic approach to the dizzy patient. Neurol Clin 8:209, 1990
133. Uemura T, Suzuki J, Hozawa J: Neuro-otological examination with special reference to equilibrium function tests. Baltimore: University Park Press, 1977
134. Troost B: Dizziness and vertigo in vertebrobasilar disease. Part I. Stroke 11:301, 1980
135. Troost B: Dizziness and vertigo in vertebrobasilar disease. Part II. Stroke 11:413, 1980
136. Herdman S: Treatment of benign paroxysmal positional vertigo. Phys Ther 70: 381, 1990
137. Herr R, Zun L, Mathews J: A directed approach to the dizzy patient. Ann Emerg Med 18:664, 1989
138. Baloh R, Honrubia V, Jacobson K: Benign positional vertigo: clinical and oculographic features in 240 cases. Neurology 37:371, 1987
139. Schuknecht H: Cupulolithiasis. Arch Otolaryngol. 90:765, 1969
140. Hall S, Ruby R, McClure J: The mechanisms of benign paroxysmal vertigo. J Otolaryngol 8:151, 1979
141. Brandt T, Daroff R: Physical therapy for benign paroxysmal positional vertigo. Arch Otolaryngol 106:484, 1980
142. Shumway-Cook A, Horak F: Rehabilitation strategies for patients with vestibular deficits. Neurol Clin 8:441, 1990
143. Shumway-Cook A, Horak F: Assessing the influence of sensory interaction on balance. Phys Ther 66:1548, 1986
144. Fisher C: Vertigo in cerebrovascular disease. Arch Otolaryngol 85:529, 1967
145. De Jong J, Bles W: Cervical dizziness and ataxia. p. 185. In Bles W, Brandt T (eds): Disorders of Posture and Gait. Elsevier, Amsterdam, 1986
146. Coman W: Dizziness related to ENT conditions. In Grieve G (ed): Modern Manual Therapy of the Vertebral Column. Churchill Livingstone, Edinburgh, 1986
147. Hutchison M: An investigation of pre-manipulative dizziness testing. In Jones H, Jones M, Milde M (eds): Sixth Biennal Conference Proceedings. Manipulative Therapists Association of Australia. Adelaide, South Australia, 1989
148. Aspinall W: Clinical testing for cervical mechanical disorders which produce ischemic vertigo. JOSPT 11:5, 1989
149. Grant R: Dizziness testing and manipulation of the cervical spine. p. 111. In Grant

R (ed): Physical Therapy of the Cervical and Thoracic Spine. Clinics in Physical Therapy. Vol 17. Churchill Livingstone, New York, 1988
150. McCouch G, Deering I, Ling T: Location of receptors for tonic neck reflexs. J Neurophysiol 14:191, 1951
151. Abrahams V: The physiology of neck muscles; their role in head movement and maintenance of posture. Can J Physiol Pharmacol 55:332, 1977
152. Goodwin GM, McCloskey DI, Matthews PBC: The persistence of appreciable kinesthesia after paralysing joint afferents but preserving muscle afferents. Brain Res 37:326, 1972
153. Manzoni D, Pompeiano O, Stampacchia G: Tonic cervical influences on posture and reflex movements. Arch Ital Biol 117:81, 1979
154. Wyke B: Articular neurology—a review. Physiotherapy 58:94, 1972
155. Cohen L: Role of eye and neck proprioceptive mechanisms in body orientation and motor coordination. J Neurophysiol 24:1, 1961
156. Igarashi M, Watanabe T, Maxian P: Role of neck propriocepters for the maintenence of dynamic bodily equilibrium in the squirrel monkey. Laryngoscope 79:1713, 1969
157. Cope S, Ryan GMS: Cervical and otolith vertigo. J Laryng 73:113, 1959
158. Gray LP: Extralabyrinthine vertigo due to cervical muscle lesions. J Laryng 70: 352, 1956
159. Weeks VD, Travell J: Postural vertigo due to trigger areas in the sternocleidomastoid muscle. J Pediat 47:315, 1955
160. De Jong PTVM, De Jong JMBV, Cohen B, Jongkees LBW: Ataxia and nystagmus induced by injection of local anesthetics in the neck. Ann Neurol 1:240, 1977
161. Hinoki M: Vertigo due to whiplash injury: A neurotological approach. Acta Otolaryngol (Stockh). (suppl 419):9, 1985
162. Barlow D, Freedman W: Cervico-ocular reflex in the normal adult. Acta Otolaryngol (Stockh) 89:487, 1980
163. Kobayashi Y, Yagi T, Kamio T: Cervico-vestibular interaction in eye movements. Auris-Nasus-Larynx (Tokyo) 13:87, 1986
164. Carlsson J, Rosenhall U: Oclumotor disturbances in patients with tension headache treated with acupuncture or physiotherapy: Cephalalgia 10:122, 1990
165. Preiskel HW: Some observations on the postural position of the mandible. J Pros Dent 15:625, 1965
166. Kazis H, Kazis AJ: Complete mouth rehabilitation. Henry Kimpton, London, 1956
167. Thompson JR: The rest positic ı of the mandible and its significance to dental science. Am Dent Assoc 33:151 1946
168. Beyron HL: Characteristics of functionally optimal occlusion and principles of occlusal rehabilitation. J Am Dent Assoc 48:648, 1954
169. Atwood DA: A review of the fundamentals on rest position and vertical dimension. Internat Dent J 9:6, 1959
170. Murpy WM: Rest position of the mandible. J Pros Dent 17:329, 1967
171. Rugh JD, Drago CJ: Vertical dimension: A study of clinical rest position and jaw muscle activity. J Pros Dent 45:670, 1981
172. Garnick J, Ramfjord SP: Rest position. An electromyographic and clinical investigation. J Pro: Dent 12:895, 1962
173. Kawamura Y, Fujimoto J: Some physiologic considerations on measuring rest position of the mandible. Med J Osaka Univ 8:247, 1957
174. Wessberg GA, Epker BN, Elliott AC: Comparison of mandibular rest positions induced by phonetics, transcutaneous electrical stimulation, and masticatory electromyography. J Pros Dent 49:100,1983

175. Lund J, Widmer C: An evaluation of the use of surface electromyography in the diagnosis, documentation, and treatment of dental patients. J Craniomand Disord Facial Oral Pain. 3:125, 1989
176. Krajicek DD, Jones PM, Radzyminski SF et al: Clinical and electromyographic study of mandibular rest position. J Pros Dent 11:826, 1961
177. Turrell AJ: Clinical assessment of vertical dimension. J Prosthet Dent 28:238, 1972
178. Weinberg LA: Vertical dimension: a research and clinial analysis. J Prosthet Dent 47:290, 1982
179. Fieldman S, Leupold RJ, Staling LM: Rest vertical dimension determined by electromyography with biofeedback as compared to conventional methods. J Pros Dent 84:216, 1978
180. George JP, Boone ME: A clinical study of rest position using the kinesiograph and myomonitor. J Prosthet Dent 41:456, 1979
181. Basler FL, Douglas JR, Moulton RS: Cephalometric analysis of the vertical dimension of occlusion. J Pros Dent 11:831, 1961
182. Yemm R: Irrelevent muscle activity: D Pract 19:51, 1968
183. Atwood DA: A cephalometric study of the clinical rest position of the mandible. Part I: the variability of the clinical rest position following the removal of occlusal contacts. J Pros Dent 6:504, 1956
184. Carlsson GE, Ericson S: Postural face height in full denture wearers. A longitudinal x-ray cephalometric study. Acta Odontol Scand 25:145, 1967
185. Landa JS: Integration of structure and function of the temporomandibular joint. N York J Dent 24:290, 1954
186. Hackney J, Bade D, Clawson A: Relationship between forward head posture and diagnosed internal derangement of the temporomandibular joint. J Orofacial Pain 7:386, 1993
187. Shock P: Current concepts of the aging process. JAMA 175:108, 1961
188. Kiernander B: Discussion on postural re-education—a critical examination of methods. Proc R Soc Med Section of Physical Medicine 49:667, 1956
189. Igarashi M, Watanabe T, Maxian P: Role of neck propriocepters for the maintenence of dynamic bodily equilibrium in the squirrel monkey. Laryngoscope 79:1713, 1969
190. Manzoni D, Pompeiano O, Stampacchia G: Tonic cervical influences on posture and reflex movements. Arch Ital Biol 117:81, 1979
191. Fukuda T: Studies on human dynamic postures from the viewpoint of postural reflexes. Acta Otolaryng (Stockholm), Suppl. 161:1, 1961
192. Bizzi E. Polit A, Morasso P: Mechanisms underlying achievement of final head posture. J Neurophysiol 39:435, 1976
193. Gibbs C: The continuous regulation of skilled response by kinaesthetic feedback. Brit J Psychol 45:24, 1954
194. Eccles J, Sabah N, Schmidt R et al: Modes of operation of the cerebellum in the dynamic loop control of movement. Brain Res 41:73, 1972
195. Tinbergen N: Ethology and stress diseases. Science 185:20, 1974
196. Tinbergen N: Functional ethology and the human sciences. Proc R Soc Lond (Ser B) 182:385, 1972
197. Mohl DM: The role of head posture in mandibular function. p. 97: In Solberg WK, Clark GT (eds): Abnormal Jaw Mechanics Diagnosis and Treatment. Quintessence Publishing, Chicago, 1984
198. Posselt U: Studies on the mobility of the human mandible. Acta Ondontol Scand 10:1, 1952

199. Brill N, Lammie GA, Osborne J et al: Mandibular positions and mandibular movements. Br Dent J 106:391, 1959
200. Dombrady L: Investigation into the transient instability of the rest position. J Pros Dent 16:479, 1966
201. Basmajian JV: Muscles alive. Their functions revealed by electromyography. 2nd Ed. Williams & Wilkins, Baltimore, 1967
202. Granit R: The Basis of Motor Control. Academic Press, New York, 1970
203. Yemm R: The mandibular rest position: the roles of tissue elasticity and muscle activity. J the DASA, January 1975
204. Carlsoo S: An electromyographic study of the activity of certain suprahyoid muscles, and of reciprocal innervation of the mandible. Acta Anatomica, 26:81, 1956
205. Mohamed SE, Christensen LV: Mandibular reference positions. J Oral Rehab 12: 355, 1985
206. Darling DW, Kraus SL, Glasheen-Wray MB: Relationship of head posture and the rest position of the mandible. J Prosthet Dent 52:111, 1984
207. Wyke BD: Neuromuscular mechanisms influencing mandibular posture: a neurologist's review of current concepts. J Dent 2:111, 1972
208. Guyton AC: Textbook of Medical Physiology. 4th Ed. WB Saunders, Philadelphia, 1971
209. Goodgold J, Eberstein A: Electrodiagnosis of neuromuscular diseases. Williams & Wilkins, Baltimore, 1972
210. Bratzlavsky M, Vander Eecken H: Postural reflexes in cranial muscles in man. Acta Neurol Belg 77:5, 1977
211. Magnus R: Some results of studies in the physiology of posture. Lancet 2:531, 1926
212. Magnus R: Korperstellung, pp. XIII, 740, Berlin, 1924, Julius Springer, Animal Posture, Croonian Lecture, Proc. Roy. Soc., Series B., Vol 98, No B 690, pp 339–353
213. Magnus R, DeKleijn A: Die Abhangigkeit des Tonus der Extremitatenmuskeln von der Kopfstellung. Pflug Arch. Ges. Physiol., 145:455, 1912
214. McCouch G, Deering I, Ling T: Location of recepters for tonic neck reflexes. J Neurophys 14:191, 1951
215. Funakoshi M, Amano N: Effects of the tonic neck reflex on the jaw muscles of the rat. J Dent Res 52:668, 1973
216. Sumino R, Nozaki S, Katoh M: Trigemino-neck reflex. p 81. In Kawamura Y, Dubner R: Oral-Facial Sensory and motor functions. Quintessence, Tokyo, 1981
217. Funakoshi M, Fujita N, Takehana S: Relations between occlusal interference and jaw muscle activities in response to changes in head position. Journal Dent Res 55:684, 1976
218. Forsberg C-M, Hellsing E, Linder-Aronson S et al: EMG activity in neck and masticatory muscles in relation to extension and flexion of the head. Eur J Orthodon 7:177, 1985
219. Mohl N: Head posture and its role in occlusion. New York State J. 42:17, 1976
220. McClean LF: Gravitational influences on the afferent and efferent components of mandibular reflexes. PhD dissertation, Thomas Jefferson University of Philadelphia, 1973
221. Lund P, Nishiyama T, Moller E: Postural activity in the muscles of mastication with the subject upright, inclined, and supine. Scand J Dent Res 78:417, 1970
222. Watt DM: Gnathosonics—a study of sounds produced by the masticatory mechanism. J Pros Dent 16:73, 1966

223. Eberle WR: A study of centric relation as recorded in a supine position. J Am Dent Assoc 42:15, 1951
224. McLean LW, Brenman HS, Friedman MGF: Effects of changing body position on dental occlusion. J Dent Res 52:1041, 1973
225. Brenman HS, Amsterdam M: Postural effects on occlusion. Dent Prog 4:43, 1963
226. Goldstein DF, Kraus SL, Williams WB, et al: Influence of cervical posture on mandibular movement. J Prosthet Dent 52:421, 1984
227. Ramfjord SPM, Ash MM Jr: Occlusion. 2nd Ed. WB Saunders, Philadelphia, 1971
228. Carlsson GE, Ingervall B, Gulumser K: Effect of increasing vertical dimension on the masticatory system in subjects with natural teeth. J Prosth Dent 41:284, 1979
229. Rivera-Morales WC, Mohl N: Relationship of occlusal vertical dimension to the health of the masticatory system. J Prosthet Dent 65:647, 1991
230. Halbert R: Electromyographic study of head position. J Can Dent Assoc 24:11, 1958
231. Davis PL: Electromyographic study of superficial neck muscles in mandibular function. J Dent Res 58:537, 1979
232. Green JD, Groot GD, Sutin J: Trigemino-bulbar reflex pathways. Am J Physiol 189:384, 1957
233. Manni E, Palmier G, Marini R et al: Trigeminal influences on extensor muscles of the neck. Exp Neurol 47:330, 1975
234. Sumino R, Nozaki S: Trigemino-neck reflex: Its peripheral and central organization. In Anderson DJ, Matthews B (eds): Pain in the Trigeminal Region. Elsevier/North-Holland Biomedical Press, 1977
235. Manns A, Miralles R, Guerrero F: The changes in electric activity of the postural muscles of the mandible upon varying the vertical dimension. J Pros Dent 45:438, 1981
236. Daly PD, Preston CB, Evans WG: Postural response of the head to bite opening in adult males. Am J Orthod 82:157, 1982
237. Vig PS, Showfety KJ, Phillips C: Experimental manipulation of head posture. Am J Orthod 77:258, 1980
238. Root GR, Kraus SL, Razook SJ et al: Effect of an intraoral appliance on head and neck posture. J Prosth Dent 58:90, 1987
239. Salonen M, Raustia A, Huggare J: Head and cervical spine posture in complete denture wearers. J Craniomandib Pract 11:30, 1993
240. Williamson E: The interrelationship of internal derangements of the temporomandibular joint, headache, vertigo, and tinnitus: a survey of 25 patients. J Craniomandib Pract 8:301, 1990
241. Seligman D, Pullinger A: The role of intercuspal occlusal relationships in temporomandibular disorders: a review. J Craniomandib Dis Fac Oral Pain 5:96, 1991
242. Naeije M, Hansson T: Short-term effect of stabilization appliance on masticatory activity in myogenous craniomandibular disorder patients. J Craniomandib Dis Fac Oral Pain 5:245, 1991
243. Wilkinson T, Hansson T, McNeil C et al: A comparison of the success of 24-hour occlusal splint therapy versus nocturnal occlusal splint therapy in reducing craniomandibular disorders. J Craniomandib Dis Fac Oral Pain 6:64, 1992
244. Seligman D, Pullinger A: The functional occlusal relationships in temporomandibular disorders: a review. J Craniomandib Dis Fac Oral Pain 5:265, 1991
245. Seligman D, Pullinger A: Association of occlusal variables among refined TM patient diagnostic groups. J Craniomandib Dis Fac Oral Pain 3:227, 1989

246. Lipp M: Temporomandibular symptoms and occlusion: a review of the literature and the concept. NY State J Dent 56:58, 1990
247. Carlsson GE, Droukas BC: Dental occlusion and the health of the masticatory system. J Craniomand Pract 2:141, 1984
248. Kirveskari P, Alanen P, Jamsa T: Association between craniomandibular disorders and occlusal interferences. J Prosthet Dent 62:66, 1989
249. Nielsen I, McNeill C, Danzig W et al: Adaptation of craniofacial muscles in subjects with craniomandibular disorders. Am J Orthod Dentofac Orthop 97:20, 1990
250. Nomenclature Committee of the Academy of Denture Prosthetics: Glossary of prosthodontic terms, 5 Ed. J Prosthet Dent 58:713, 1987
251. Krough-Poulsen WB, Olssen A: Management of the occlusion of the teeth. p 236. In Schwartz L, Chayes CM (eds): Facial Pain and Mandibular Dysfunction. WB Saunders Co., Philadelphia, 1968
252. Pruzansky S: The application of electromyography to dental research. J Am Dent Assn 44:49, 1952
253. Jarabak JR: An electromyographic analysis of muscular and temporomandibular joint disturbances due to imbalances in occlusion. Angle Orthodont 26:170, 1956
254. Perry HT: Functional electromyography of the temporal and masseter muscles in Class II, Division I malocclusion and excellent occlusion. Angle Orthodont 26:49, 1955
255. Karlsson S, Cho S-A, Carlsson G: Changes in mandibular masticatory movements after insertion of nonworking-side interference. J Craniomand Disord Facial Oral Pain 6:177, 1992
256. Dawson PE: Evaluation, Diagnosis and Treatment of Occlusal Problems. p. 49. Baltimore: CL Mosby, Baltimore, 1989
257. Rugh J, Ohrbach R: Occlusal parafunction. p. 249. In Mohl ND, Zarb GA, Carlsson GE, Rugh JD (eds): A Textbook of Occlusion. Quintessence, Chicago, 1988
258. Kronn E: The incidence of TMJ dysfunction in patients who have suffered a cervical whiplash injury following a traffic accident. J Orofac Pain 7:209, 1993
259. Braun B, DiGiovanna A, Schiffman E et al: A cross-sectional study of temporomandibular joint dysfunction in post-cervical trauma patients. J of Craniomandib Disord Facial Oral Pain 6:24, 1992
260. Pullinger AG, Seligman DA: Trauma history in diagnostic groups of temporomandibular disorders. Oral Surg Oral Med Oral Pathol 71:529, 1991
261. Howard R, Benedict J, Raddin J et al: Assessing neck extension-flexion as a basis for temporomandibular joint dysfunction. J Oral Maxillofac Surg 49:1210, 1991
262. Maruyama T, Nishio K, Kotani M, Miyauchi S: The effect of changing the maxillomandibular relationship by a bite plane on the habitual mandibular opening and closing movement. J Oral Rehab 11:455, 1984
263. Begg C: Statistical Methods In Medical Diagnosis. CRC Critical Reviews in Medical Informatics, 1(1):1, 1986/1988
264. Kassirer J, Pauker S: Should diagnostic testing be regulated? New Engl J Med 299:947, 1978
265. Koch H, Gagnon R: Office visits involving x-rays. p. 79. National Ambulatory medical care survey: United States 1977. Advance Data from Vital and Health Statistics, No. 53, DHEW Publication, PHS, 1979
266. Boden S, Davis D, Dina T et al: Abnormal magnetic resonance scans of the lumbar spine in asymptomatic subjects. J Bone Joint Surg 72A:403, 1990
267. Deyo R: Plain roentgenography for low back pain: Finding needles in a haystack. Arch Inter Med 149:27, 1989

268. Coste J, Paolaggi J, Spira A: Classification of nonspecific low back pain. II. Clinical Diversity of organic forms. Spine 17:1038, 1992
269. Wiesel S, Tsourmas N, Feffer H et al: A study of computer-assisted tomography. I. The incidence of positive CT-scans in an asymptomatic group of patients. Spine 9:949, 1984
270. Torgerson W, Dotter W: Comparative roentgenographic study of the asymptomatic and symptomatic lumbar spine abstracted. J Bone Joint Surg 58A:850, 1976
271. Teresi L, Lufkin R, Reicher M et al: Asymptomatic degenerative disk disease and spondylosis of the cervical spine. MR Imaging. Radiology 83, 1987
272. Jackson R, Cain J, Jacobs R et al: The neuroradiographic diagnosis of lumbar herniated nucleus pulposus: II. Spine 14, 1989
273. Haldeman S: Failure of the pathology model to predict back pain: Presidential Address, North American Spine Society. Spine 1990; 15:718, 1990
274. Miles K, Maimaris C, Finlay D et al: The incidence and prognostic significance of radiological abnormalities in soft tissue injuries to the cervical spine. Skeletal Radiol 17:493, 1988
275. Deyo R, Diehl A: Lumbar spine films in primary care: Current use and effect of selective ordering criteria. J Gen Intern Med 1:20, 1986
276. Liang M, Komaroff A: Roentgenograms in primary care patients with acute low-back pain: a cost-effectiveness analysis. Arch Intern Med 142:1108, 1982
277. Scavone J, Latshaw R, Rohrer G: Use of lumbar spine films: Statistical evaluation at a university teaching hospital. JAMA 426:1105, 1981
278. Nachemson A: Advances in low-back pain. Clin Orthop 200:266, 1985
279. Spratt K, Lehmann T, Weinstein J et al: A new approach to the low-back physical examination. Spine 15:96, 1990
280. Guccione A: Physical therapy diagnosis and the relationship between impairments and function. Phys Ther 71:499, 1991
281. Binkley J, Finch E, Hall J et al: Diagnostic classification of patients with low back pain: report on a survey of physical therapy experts. Phys Ther 73:138, 1993
282. Jette A: Using health-related quality of life measures in physical therapy outcomes research. Phys Ther 73:528, 1993
283. Boissonnault WG: Examination in Physical Therapy Practice—Screening for medical disease. Churchill Livingstone, New York, 1991
284. DeRosa C, Porterfield J: A physical therapy model for the treatment of low back pain. Phys Ther 72:261, 1992
285. Coste J, Paolaggi J, Spira A: Classification of nonspecific low back pain. I. Psychological involvement in low back pain. Spine 17:1028, 1992
286. Delitto A, Cibulka M, Erhard R et al: Evidence for use of an extension-mobilization category in acute low back syndrome: A prescriptive validation pilot study. Phys Ther 73:216, 1993
287. Diagnosis in Physical Therapy. A roundtable discussion. Delitto A, Guccione A, Jette A, Sahrmann S, Moderator Magistro C. In PT Magazine, 58, 1993
288. Jette A: Diagnosis and classification by physical therapists: A special communication. Phys Ther 69:967, 1989
289. Dekker J, van Baar M, Curfs E et al: Diagnosis and treatment in physical therapy: an investigation of their relationship. Phys Ther 73:568, 1993
290. Riddle D, Rothstein J: Intertester reliability of McKenzie's classifications of the syndrome types present in patients with low back pain. Spine 18:1333, 1993
291. Khalil T, Asfour S, Martinez L et al: Stretching in the rehabilitation of low-back pain patients. Spine 17:311, 1992

292. Sikorski J: A rationalized approach to physiology for low-back pain. Spine 10: 571, 1985
293. McKenzie R: The Lumbar Spine: Mechanical Diagnosis and Therapy. Spinal Publications, Waikanae, New Zealand, 1981
294. Sahrmann S: Diagnosis by the physical therapist—a prerequisite for treatment. A special communication. Phys Ther 68:1703, 1988
295. Bell W: Orofacial Pains; Differential Diagnosis. 2nd Ed. p. 56. Year Book Medical Pub, Chicago, 1980
296. Tait R, Pollard C, Margolis R et al: The pain disability index: psychometric and validity data. Arch Phys Med Rehabil 68:438, 1987
297. Youdas J, Carey J, Garrett T: Reliability of measurements of cervical spine range of motion—comparison of three methods. Phys Ther 71:98, 1991
298. Youdas J, Garrett T, Suman V et al: Normal range of motion of the cervical spine: An initial goniometric study. Phys Ther 72:770, 1992
299. Dvorak J, Antinnes J, Panjabi M et al: Age and gender related normal motion of the cervical spine. Spine 17:5393, 1992
300. Burn L, Paterson J: Musculoskeletal Medicine. The Spine. p. 152. Kluwer Academic Publishers, Boston, 1990
301. Kendall H, Kendall F, Boynton D: Posture and Pain. Williams & Wilkins, Baltimore, 1952
302. Refshauge K, Bolst L, Goodsell: The relationship between cervicothoracic posture and the presence of pain. Proceedings from the International Federation of Orthopaedic Manipulative Therapists, Vail, Colorado June 1–5, 1992
303. Griegel-Morris P, Larson K, Mueller-Klaus K et al: Incidence of common postural abnormalities in the cervical, shoulder, and thoracic regions and their association with pain in two age groups of healthy subjects. Phys Ther 72:425, 1992
304. Hayes K: The effect of awareness of measurement error on physical therapists' confidence in their decisions. Phys Ther 72:515, 1992
305. Wolf S: Clinical Decision Making in Physical Therapy. FA Davis Company, Philadelphia, 1985
306. Standards for tests and measurements in physical therapy practice. Task Force members, Rothstein J, Campbell S, Echternach J, et al. Phys Ther 71:589, 1991
307. Coste J, Paolaggi J, Spira A: Reliability of interpretation of plain lumbar spine radiographs in benign, mechanical low-back pain. Spine 16:426, 1991
308. Deyo R, McNiesh L, Cone R: Observer variability in interpretation of lumbar spine radiographs, Arthritis Rheum 28:1066, 1985
309. Yoo J, Zou D, Edwards W et al: Effect of cervical spine motion on the neuroforaminal dimensions of human cervical spine. Spine 17:1131, 1992
310. Lewit K: The contribution of clinical observation to neurobiological mechanisms in manipulative therapy. In Korr I (ed): The Neurobiologic Mechanisms in Manipulative Therapy. Plenum Press, New York, 1978
311. Kenneally M, Rubenach H, Elvey R: The upper limb tension test: The SLR test of the arm: In Grant R (ed): Physical Therapy of the cervical and thoracic spine. Clinics in Physical Therapy 17. Churchill Livingstone, New York, 1988
312. Elvey R: Brachial plexus tension tests and the pathoanatomical origin of arm pain. p. 105. In Idczak R (ed): Aspects of Manipulative Therapy, Lincoln Institute of Health Sciences, 1980
313. Breig A: Adverse Mechanical Tension in the Central Nervous System. John Wiley and sons, New York, 1978

314. Butler D, Gifford L: The concept of adverse mechanical tension in the nervous system. Part 1: testing for "Dural Tension." Physiotherapy 75:622, 1989
315. Butler D, Gifford L: The concept of adverse Mechanical tension in the nervous system. Part 2: Examination and Treatment. Physiotherapy 75:629, 1989
316. Elvey R: Treatment of arm pain associated with abnormal brachial plexus tension. Austr J Physiother 32:225, 1986
317. Whitty C, Willison R: Some aspects of referred pain. Lancet ii:226, 1958
318. Lewis T, Kellegren J: Observations relating to referred pain, viscerомotor reflexes and other associated phenomena. Clin Sci 4:47, 1939
319. Mooney V, Robertson J: "The Facet Syndrome." Clin Orthop 115:149, 1976
320. Leyshon A, Kirwan E, Parry C: Electrical studies in diagnosis of compression of the lumbar root. J Bone Joint Surg 36B:71, 1981
321. Saal J, Saal J: Nonoperative treatment of herniated lumbar intervertebral disc with radiculopathy, an outcome study. Spine 14:431, 1989
322. Hutchison M: An investigation of premanipulative testing. p. 104. Proc of 6th Bienn. Conf MTAA, Adelaide, Australia, 1989
323. Powell V: An investigation of testing procedures for vertebrobasilar insufficiency. Aust J Physiother 36:31, 1990
324. Yochum T, Rowe L: Arthritides of the upper cervical complex. p. 22. In Idczak R (ed): Aspects of Manipulative Therapy. Proceedings of a Multidisciplinary International Conference on Manipulative Therapy, Melbourne, Aug 1979, Lincoln Institute of Health Sciences, 1980
325. Pettman E: Stress testing the anatomy. Proceedings from the International Federation of Orthopaedic Manipulative Therapists, Vail, Colorado, June 1–5, 1992
326. Paris SV: S3 Course Notes. p. 78. Institute of Graduate Physical Therapy, Inc. St. Augustine, Florida, 1992
327. Aspinall Wendy: Clinical testing for the craniovertebral hypermobility syndrome. JOSPT 12:47, 1990
328. Roland M, Morris R: A study of the natural history of low back pain. Spine 8: 145, 1983
329. Waddell G: A new clinical model for the treatment of low back pain. Spine 12: 632, 1987
330. Nachemson A: A critical look at the treatment for low back pain. Scand J Rehab Med 11:143, 1979
331. Benn R, Wood P: Pain in the back: an attempt to estimate the size of the problem. Rheumatol Rehabil 14:121, 1975
332. Berquist-Ullman M, Larsson U: Acute low back pain in industry. Acta Orthop Scand 170(suppl):1, 1977
333. Andersson G, Pope M, Frymoyer J: Epidemiology. p. 101. In Pope M, Frymoyer J, Andersson G (eds): Occupational Low Back Pain. Praeger, New York, 1984
334. Marbach et al: Psychother Psychocom 39:47, 1983
335. Farrell J, Twomey L: Acute low back pain, comparison of two conservative treatment approaches. Med J Aust 1:160, 1982
336. Cousins N: A nation of hypochondriacs. p. 88. Time, June 18, 1990
337. Greenough C, Fraser R: Assessment of outcome in patients with low-back pain. Spine 17:36, 1992
338. Weber H: Lumbar disc herniation: a controlled prospective study with ten years of observation. Spine 8:131, 1983
339. Parry W: The failed back. p. 341. In Wall PD, Melzack (eds): Textbook of Pain. Edited by PD Wall and Melzack. Churchill Livingstone. New York. 1989

340. Wetzel F: Chronic Benign Cervical Pain Syndromes. Surgical Considerations. Spine 17(suppl):S367, 1992
341. McCoy C, Selby D, Henderson R et al: Patients avoiding surgery; pathology and one-year life status follow-up. Spine 16(suppl)S198, 1991
342. Nelson M (ed): Low back pain and industrial and social disablement. Proceedings of symposium. London: Back Pain Association, 1983
343. McKenzie R: The Lumbar Spine: Mechanical Diagnosis and Therapy. Spinal Publications, Waikanae, New Zealand, 1981
344. Cooper D, Jenkins R, Bready L et al: The prevention of injuries of the brachial plexus secondary to malposition of the patient during surgery. Clin Orthop 228: 33, 1988
345. Russek AS: Diagnosis and treatment of scapulocostal syndrome. JAMA 150:25, 1982
346. Zusman M: Spinal manipulative therapy: review of some proposed mechanisms, and a new hypothesis. Austr J Physiother 32:89, 1986
347. Korr (ed): The Neurobiologic Mechanisms in Manipulative Therapy. Plenum, New York, 1978
348. Idczak R (ed): Aspects of manipulative therapy. Proceedings of a Multidisciplinary International Conference on Manipulative Therapy, Melbourne, Aug 1979. Lincoln Institute of Health Sciences, 1980
349. Burn L, Paterson J (eds): Musculoskeletal Medicine. Kluwer Academic Publishers, Boston, 1990
350. Rothstein J (ed): Manual Therapy: Special Issue. Phys Ther 72:(12), 1992
351. Goldstein M (ed): The Research Status of Spinal Manipulative Therapy. U.S. Department of Health, Education, and Welfare. DHEW Publication No. (NIH) 76-998. NINCDS Monograph No. 15, 1975
352. Zusman M, Edwards B, Donaghy: Investigation of a proposed mechanism for the relief of spinal pain with passive joint movement. J Manual Med 4:58, 1989
353. Manipulative Physiotherapists Association of Australia, 7th Biennial Conference. Nov 27–30, 1991, Blue Mountains, New South Wales. Manipulative Physiotherapists Association, Australia, North Fitzroy Victoria, Australia.
354. Bogduk N: A scientific approach to cervical diagnosis. p. 151. Proceedings from the Fourth Biennial Conference of the Manipulative Therapist Association of Australia, Brisbane, May 22–25, 1985
355. Grieve G (ed): Modern Manual Therapy of Vertebral Column. Churchill Livingstone, New York, 1986
356. Di Fabio R: Efficacy of manual therapy. In Rothstein J (ed): Manual Therapy: Special Issue. Phys Ther 72:853, 1992
357. Shepard K, Jensen G, Schmoll B et al: Alternative approaches to research in physical therapy: positivism and phenomenology. Phys Ther 73:88, 1993
358. Polit D, Hungler B: Nursing Research: Principles and Methods. 4th Ed. JB Lippincott, Philadelphia, 1991
359. Merriam S: Case study research in education: a qualitative approach. Jossey-Bass, San Francisco, 1988
360. Mattingly C: Commentary on Shepard K, Jensen G, Schmoll B et al: Alternative approaches to research in physical therapy: positivism and phenomenology. Phys Ther 73:98, 1993
361. Colachis S, Strohm B: Radiographic studies of cervical spine motion in normal subjections, flexion and hyperextension. Arch Phys Med Rehabil 46:753, 1965

362. Colachis S, Strohm B: Relationship of time to varied traction force with constant angle of pull. Arch Phys Med Rehabil 47:353, 1966
363. Wong A, Leong C, Chen C: The traction angle and cervical intervertebral separation. Spine 17:136, 1992
364. Twomey LT, Taylor JR: Damage to the cervical disc and facet joints following severe trauma. p. 25. In: Manipulative Physiotherapists Association of Australia, 7th Biennial Conference. Blue Mountains, New South Wales, Nov. 27–30, 1991, Manipulative Physiotherapists Association Australia, North Fitzroy Victoria, Australia, 1991
365. Pearce J: Whiplash injury: a reappraisal. J Neurol Neurosurg Psy 52:1329, 1989
366. Davis S, Teresi L, Bradley W et al: MRI Findings in cervical spine hyperextension injuries. p. 170. Proceedings of the 41st Annual Meeting of the Royal Australian College of Radiologists, 1990
367. Osti O, Vernon-Roberts B, Fraser R: Anulus Tears and intervertebral disc degeneration: an experimental study using an animal model. Spine 15:762, 1990
368. Taylor J, Twomey L: The development of the human intervertebral disc, p. 39. In Ghosh P (ed): The Biology of the Intervertebral Disc. Vol. 1. CRC Press, Boca Raton, 1988
369. Foreman S, Croft A: Whiplash injuries; The Cervical Acceleration/Deceleration Syndrome. Williams & Wilkins, Baltimore, 1988
370. St Pierre D, Gardiner P: The effect of immobilisation and exercise on muscle function: a review. Physiother Canada 39:24, 1987
371. Shepard R: Management of exercise in the elderly. Applied Sports Sciences 9: 109, 1984
372. Menard D, Stanish W: The aging athlete. Am J Sports Med 17:187, 1991
373. Pardini A: Exercise, vitality and aging. Aging 344:19, 1984
374. Aniansson A, Grimby G, Rundgren A et al: Physical training in old men. Age Ageing 9:186, 1980
375. Aloia J, Cohn S, Cane R et al. Prevention of bone loss by exercise. J Clin Endocrinol Metab 43:992, 1978
376. Smith E, Reddin N, Smith P: Physical activity and calcium modalities for bone mineral increase in aged women. Med Sci Sports Exerc. 13:60, 1981
377. Gustavsen R: Training Therapy: Prophylaxis and Rehabilitation. New York, Thieme, 1985
378. Saal JS: Flexibility training. Physical medicine and rehabilitation: state of the art reviews 1:537, 1987. Philadelphia, Hanley & Belfus, Inc.
379. Sweeney T, Prentice C, Saal J et al: Cervicothoracic Muscular Stabilization Techniques. In: Saal J (guest ed) Physical Medicine and Rehabilitation: State of the Art Reviews. Vol 4, No 2, June 1990. p 335–359, Philadelphia, Hanley & Belfus, Inc.
380. Jansen J, Spoerri O: Atypical frontoorbital pain and headache due to compression of upper cervical roots. p. 14. In Pfaffenrath V, Lundberg P-O, Sjaasdad O (eds): Updating in Headache. Springer, Berlin, 1985