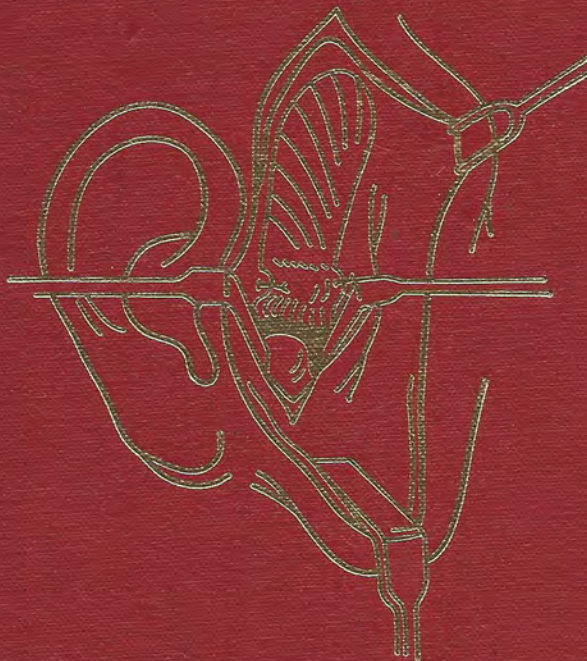


FONSECA

Oral and Maxillofacial Surgery

*Temporomandibular
Disorders*



BAYS ■ QUINN

4

Oral and Maxillofacial Surgery

VOLUME

4

Temporomandibular Disorders

Edited by

Raymond J. Fonseca, D.M.D.

Dean

University of Pennsylvania

School of Dental Medicine

Philadelphia, Pennsylvania

Volume Editors

Robert A. Bays, D.D.S.

Peter D. Quinn, D.M.D., M.D.

Illustrated by William M. Winn, Medical Illustrator

W.B. Saunders Company

A Harcourt Health Sciences Company

Philadelphia London New York St. Louis Sydney Toronto

Physical Therapy Management of Temporomandibular Disorders

Steven L. Kraus

Nonsurgical treatment is the primary focus for patients experiencing symptoms and a limitation in function due to temporomandibular disorders (TMDs). Nonsurgical treatment options and the application and sequencing of the treatment options for TMD are controversial. Nonsurgical treatment options for TMD may include medication, mouth appliances, injections, stress management, and physical therapy. Physical therapy is both a conservative and a noninvasive form of management for TMD. This chapter has four objectives: first, to introduce clinical practice guidelines for physical therapy in the management of TMD; second, to familiarize the reader with physical therapy; third, to discuss TMD conditions that will best respond to physical therapy management; fourth, to give an overview of the cervical spine and its significance in head and neck pain management.

Clinical Practice Guidelines for Physical Therapy

An overview of clinical practice guidelines as they pertain to physical therapy intervention for TMD is important because current practice guidelines for physical therapy in TMD management are, at best, misleading. Clinical practice guidelines ex-

ist in all areas of medicine. Guidelines may be used as a means to understand the benefits of a certain intervention. The development of clinical practice guidelines is heralded by some experts as a prelude to a golden age of health care accountability and effectiveness.¹ Other experts, however, view the guidelines as a main element in a dangerous campaign to interpose governmental and health care managers between practitioners and patients—a campaign that could lead to diminished quality of care.¹ There are thousands of guidelines in existence. Many of these guidelines are proprietary, not based on science, or slanted toward a particular organization's or subspecialty's point of interest.²

Pertinent to this discussion are guidelines from the Agency for Health Care Policy and Research (AHCPR).³ The AHCPR was created in 1989 as an extension of the U.S. Public Health Service to develop clinical practice guidelines in accordance with AHCPR regulations and procedures.¹ A majority of physical therapists and medical doctors are aware of the AHCPR recent guidelines, *Acute Low Back Problems in Adults*.³ There have been many legitimate questions raised as to the agency's own guidelines used to acquire and review evidence for establishing these guidelines for acute low back pain.^{1, 2} Weaknesses inherent in the AHCPR guideline development process would increase the likelihood of misinterpretation.^{2, 4-6}

Dentists and dental organizations have become aware of the AHCPR guidelines on acute low back pain. Several task forces in dentistry refer to the AHCPR comments on modalities commonly used by physical therapists.^{7, 8} Several task forces in dentistry also have conducted their own review of the literature pertinent to "physical therapy" management of TMD,⁷⁻⁹ providing dentists or dental organizations the means to evaluate modalities commonly used by the physical therapist in the management of TMD.

A technical assessment conference (April 29–May 1, 1996) on TMD, sponsored by the National Institutes of Health (NIH), produced a Technology Assessment Statement.⁷ Neither the conference planning committee, the expert panel, nor the conference itself included representation from the physical therapy profession.¹⁰ The following is the final statement by the NIH on physical therapy:⁷

Physical Therapy: Physical therapy applications to TMD include a wide variety of evaluative techniques and treatment modalities that have been commonly used in other neurological and musculoskeletal disorders. These therapies generally are conservative and noninvasive. Benefits to TMD patients have been described, although few data are available to document these results.

A paper by Dr. Feine and colleagues⁸ critiques the field of physical therapy on the basis of a Medline search of data from 1976 to 1996. Some modalities reviewed by Feine and coworkers⁸ should have been excluded. For example, the use of diathermy is contraindicated for application to the temporomandibular joint (TMJ) area because the TMJ is located close to the eye. The U.S. Food and Drug Administration (FDA) has not yet approved laser treatment for use by a physical therapist, except in a research capacity. Transcutaneous electrical nerve stimulation is known to most physical therapists as an adjunctive modality and is considered only as a part of a comprehensive approach, not as a treatment unto itself.

The NIH position and Dr. Feine's paper on physical therapy are extremely narrow in perspective. They neither accurately nor adequately represent the profession of physical therapy and refer to physical therapy

in the generic sense to mean a modality. This is a clear misrepresentation of the physical therapy profession. *Physical therapy*, as defined by the American Physical Therapy Association (APTA), is as follows:¹¹

Physical therapy, which is the care and services provided by or under the direction and supervision of a physical therapist, includes:

1. *Examining patients with impairments, functional limitations, and disability or other health-related conditions in order to determine a diagnosis, prognosis, and intervention.*
2. *Alleviating impairments and functional limitations by designing, implementing, and modifying therapeutic interventions.*
3. *Preventing injury, impairments, functional limitations, and disability, including the promotion and maintenance of fitness, health, and quality of life in all age populations.*
4. *Engaging in consultation, education, and research.*

The APTA¹² advocates that

only physical therapists provide or direct the provision of physical therapy. Therefore the use of modalities, therapeutic techniques and exercises, unless provided by or under the direction of physical therapists, is not physical therapy. Other health care providers who use modalities and therapeutic techniques or exercises similar or identical to those used by physical therapists should be held to comparable educational and clinical standards.

The validity of research investigating physical therapy intervention for TMD should be seriously questioned when physical therapy is not defined and the credentials of the clinician are unclear. Conclusions based on research that investigates a single modality or therapeutic technique for single-tissue involvement also should be seriously questioned. Physical therapists seldom offer a singular treatment option for a patient who has myriad tissue and functional involvements. Physical therapists combine and sequence various treatment parameters, which may include modalities, therapeutic (manual) techniques, and exercise, as well as patient education and instructions in behavioral modifications.

Treatments offered are modified on the basis of the physical therapist's reassessment of the patient's signs and symptoms.

In summary, guidelines can appear to be beneficial to the public and the practicing clinician. Guidelines, however, can be incomplete and misleading.

Patient Accessibility to Physical Therapy

Patients have access to a physical therapist by way of self-referral or physician referral. Currently, more than 30 states have direct-access legislation. Patients in these states can consult physical therapists without a physician's referral. Patients can be assured that anyone who has a license in physical therapy must have graduated from an accredited program. Physical therapist education and training in the United States are uniform and standardized. Academic programs must meet the rigorous standards of an accreditation process. The Commission on Accreditation of Physical Therapy Education (CAPTE) is the independent accrediting agency for physical therapy education.

Though TMD patients can be seen directly by a physical therapist in those states that have direct access or by way of a medical referral to the physical therapist, the majority of patients with TMD are currently referred to a physical therapist by a dentist. Instead of consulting a medical doctor or physical therapist, patients often consult a dentist for TMD treatments for several reasons:

- Lay articles published in magazines and newspapers that describe in detail the reader's symptoms (e.g., headaches, facial, jaw, and neck pain); prospective patients conclude from these articles that the TMJ is the source of the patients' symptoms and help will be found by consulting a dentist.
- Word of mouth from neighbors or friends who received treatment from a dentist
- Patients' perception of the "cause" of their problems, i.e., teeth causing jaw pain
- TMD discovered during a routine dental visit

Once a dentist sees a patient, he or she may elect to initiate conservative and non-invasive care for the patient's TMD condition. Beyond this initial approach by the dentist, the patient can be referred to a physical therapist. When to refer to physical therapy is often a matter of clinical judgment. This author prefers physical therapy intervention before or during the nonsurgical dental care or before and then shortly after surgical intervention for the TMJ. The following guidelines can alert the dentist to those patients who should be referred to physical therapy at the earliest time possible:

- Chronic symptoms (more than 3 months in duration)
- Significant limitations with functional activities involving chewing, talking, and opening of the mouth
- Adjacent tissues to the TMJ, such as the muscles of mastication and cervical spine, that are involved are contributing to symptoms and limiting function of the jaw or neck
- Symptoms that have not responded to or are worsening after self-care management and/or the use of a mouth appliance and/or medication
- Patient demonstration of a lack of understanding and willingness to follow through with self-care management initiated by the dentist
- Postoperative arthroscopy or arthrothomy to the TMJ and postoperative orthognathic surgery
- "Significant" trauma received to the jaw, head, and/or neck

Management of TMD by a Physical Therapist

The widely used and accepted definition of TMD is as follows:

Temporomandibular disorders refer to a cluster of disorders characterized by the following main symptoms: pain and tenderness in the region of the muscles of mastication and the temporomandibular joint (TMJ), sounds during condylar movements, and limited or asymmetric mandibular movements.⁹

The term *TMD* evolved over time since previous terms were deemed too limiting, for example, “temporomandibular joint disturbances,” “temporomandibular joint dysfunction,” and “functional temporomandibular joint disturbances.”¹³ This definition includes not only problems related to the TMJs but also “all functional disturbances of the masticatory system.”¹³ Therefore, the term *TMD* implies a category of disorders, such as the TMJ and muscles of mastication, rather than a single diagnosis. There does exist a subclassification system for both the TMJ and muscles of mastication.⁹

For clinical and research purposes, three subgroups of TMD have been distinguished, according to the guidelines of the American Academy of Oral Facial Pain⁹:

- TMD with an arthrogenous component (TMD—A)
- TMD with an arthrogenous and myogenous component (TMD—A/M)
- TMD with a myogenous component (TMD—M)

Several studies have been completed, using these three subgroups of TMD.^{14, 15} Physical therapy management of these three subgroups of TMD is covered in the next section.

Initially, TMD was thought to be caused by occlusal factors and therefore best treated by occlusal adjustments, prosthetic rehabilitation, and orthodontic treatment.¹⁶ Occlusion is now considered to play a secondary role as an etiologic factor to TMD.^{16–18} TMD is now thought to be a medical disorder in which diagnosis and treatment are based on principles used to treat other joints and muscles in the body.¹⁹ Physical therapists treat nondiseased neuromuscular-skeletal conditions, which include the temporomandibular joint and associated muscles of mastication.

Other than doing nothing at all, treatment for any of the three subgroups of TMD should initially focus on instructing the patient in self-care management. This includes patient education, heat and/or cold application, awareness and control of oral habits, and, if necessary, a nonchew diet and over-the-counter nonsteroidal anti-inflammatory drugs (NSAIDs), such as aspirin and ibuprofen. Self-care management can be taught by the doctor or someone in the doctor’s office who has proper training

and can respond appropriately to the patient’s questions. Otherwise, the physical therapist can provide the home care instructions for TMD management. To understand the role of the physical therapist in TMD management is to go beyond the self-care treatment.

PHYSICAL THERAPY MANAGEMENT FOR TMD—A

A subclassification scheme for the patient population having TMD—A is the following⁹:

- Deviation in form
- Disk displacement
- Dislocation
- Inflammatory conditions
- Arthritides
- Ankylosis

Identifying the role for physical therapy intervention for the subclassifications of TMD—A listed is difficult. Identifying the role for physical therapy intervention for other medical diagnoses of the neuromuscular-skeletal system is also difficult. Physical therapists in the majority of cases do not treat medical diagnoses even though they may imply involvement of the neuromuscular-skeletal system.^{20–24} For example, physical therapists do not treat degenerative disk disease of the spine, herniated nucleus pulposus, rheumatoid arthritis, and spondylosis. Likewise, physical therapists do not treat ankylosis, arthritides, or deviation in form of the TMJ. *Physical therapists treat impairments and functional limitations that are related to the disability of the patient, which may or may not have a correlation to the medical diagnosis.* An example is a loss of mouth opening (impairment). The patient’s loss of mouth opening results in an inability to perform tasks involving talking (functional limitations). The patient’s occupation demands extensive talking; thus the inability to open the mouth may prevent the patient from performing some aspects of his occupational duties (disability). The extent to which the patient perceives how the impairments and functional limitations affect his or her social roles will further impact the level of disability.²⁵ After physical therapy treatments, patients often have a return of function without symptoms, yet their medical or dental diagnosis—her-

niated disk, rheumatoid arthritis, spondylosis, or TMJ disk displacement/degenerative joint disease—still exists. *For research and outcome data to be meaningful, the diagnosis must match the skills of the clinician treating the diagnosis.*

The role of physical therapy in the treatment of TMD—A is to determine whether one or more of the following *physical therapy subclassification diagnoses* for TMD—A is present²⁶:

- Inflammation
- Hypermobility
- Hypomobility

Inflammation

When a patient's primary complaint is "pain" of the TMJ, inflammation should be considered as one of the more probable sources of this complaint. TMJ tissues commonly inflamed are the posterior attachment (retrodiscal pad, superior and inferior stratums), collateral ligaments, and periarticular tissue (capsule/synovium and TMJ ligament). Generally speaking, tissues that are inflamed have varying degrees of vasodilatation (redness), swelling (exudate and bleeding from torn vessels), increase in blood flow and/or chemical and metabolic activity (heat), and irritation of nerve endings (pain). Inflammation can be acute, subacute, or chronic. Categorizing conditions in one of the three stages is difficult and is often subjective and is not considered here.

The cause of inflammation can be infection, disease, or trauma. Inflammation secondary to infection or disease requires dental and/or medical treatment. Infection or disease, e.g., various polyarthritides, are not common conditions of the TMJ. From the physical therapist's perspective, treatment for inflammation related to arthritides does not differ significantly from treatment offered for inflammation caused by trauma.

By far, the majority of inflammation of the posterior attachment and/or periarticular tissue is due to *either macrotrauma or microtrauma*. Macrotrauma to the TMJs may result from open joint surgery or a blow to the mandible. Microtrauma may occur when the TMJ tissues' physiologic range has been exceeded. An example would be opening wide during a dental procedure or

by yawning. Duration, force, and repetition of opening would affect whether such an event would lead to inflammation. The TMJ is a load-bearing joint; however, microtrauma may result from TMD—M (masticatory muscle hyperactivity), which involves excessive or prolonged loading. TMD—M is covered later.

Inflammation is often accompanied by hypomobility (limited mouth opening) because the patient is unwilling to move his or her mouth in a functional range as a result of pain, associated muscle guarding, and/or joint effusion. However, inflammation of the periarticular tissue may lead to periarticular tissue tightness that also results in hypomobility. Acute inflammation of the periarticular tissue if left untreated progresses to the chronic stage, at which fibroblasts are formed, more collagen is produced, and periarticular tissue tightness develops.²⁷ Treatment of periarticular tissue tightness and a disk displacement without reduction, both of which result in hypomobility, are discussed later.

Diagnosis of Inflammation

A medical history focuses on events that may have caused macro- or microtrauma to the jaw. *Subjectively*, the patient reports having pain in the area of the TMJ with possible radiation into the temple, masseter, ear, and angle of the jaw. Patients often report that their pain is either increased or decreased by functional and/or parafunctional movements of the mandible. *Objective* confirmation of inflammation is made by the clinical examination, incorporating various palpation and selective loading procedures to the TMJs.^{28, 29}

Treatment for Inflammation

Modalities

Modalities are used as part of a comprehensive treatment and not as a single approach. Primary goals for modalities are to lower pain and to enhance the healing process by reducing the effects of inflammation. Modalities discussed for the treatment of inflammation are moist or dry heat, ultrasound, cold, iontophoresis, and transcutaneous electrical nerve stimulation (TENS). These modalities are commonly

used by this author. The duration and sequencing of modalities in conjunction with other procedures offered by the physical therapist are a matter of clinical judgment, experience of the clinician, and specific signs and symptoms of the patient. Before applying any modalities, the clinician must know specific indications and contraindications. More research is needed on the therapeutic effects of modalities on the TMJ. For now, research on other joint tissues is inferred to be applicable to the TMJ tissues. The following is only an overview; the reader is referred to the references for additional information.³⁰⁻³²

Thermal Agents

Moist/Dry Heat—Superficial Moist/dry heat is superficial heating. Changes in tissue temperature resulting from superficial heating depend on the intensity of the heat applied, time of heat exposure, and thermal medium (thickness of adipose tissue). When using tolerable superficial heat, temperature can be elevated up to 1°C at a depth of 3 cm. Superficial heating produces a therapeutic effect by elevating pain threshold, altering nerve conduction velocity, and decreasing muscle tension.

Superficial heating of the skin may produce a counterirritation effect to reduce pain. Superficial heating over a peripheral nerve (auriculotemporal nerve for primary TMJ innervation) can increase the threshold of sensory nerve conduction velocity, thus producing an analgesic effect. In both cases, superficial heating reduces the patient's perception of pain associated with inflammation.

Unrelated to inflammation but pertinent to the discussion of TMD—M are the effects of superficial heating on muscle. Heat decreases firing of the alpha motoneurons, thereby reducing tonic extrafusal fiber activity based on reflex mechanisms that are not totally understood. Muscle relaxation may also result from the analgesic effect of heating because the patient perceives less pain and as a result does less muscle guarding, which is a response to pain.

Increasing tissue temperature is usually associated with vasodilation, causing an increase in blood flow to the area. If the clinician suspects "significant" inflammation, superficial heat applied to the skin may be a relative contraindication. The clinician

would have to weigh the benefits of the analgesic and neuromuscular effect in relation to the possible negative effects of vasodilation of inflamed tissues of the TMJ.

Ultrasound—Deep Ultrasound has been used for more than 50 years in medicine. Ultrasound has similar effects to superficial heating of tissues. However, unlike in superficial heating, the biophysical effects result from the interaction of ultrasound with tissues at depths of 5 cm or more. Ultrasound has an anti-inflammatory effect. Ultrasound produces an increase in cell membrane and vascular wall permeability with enhanced blood flow to improve tissue repair and to reduce inflammation.³² Physiologic effect is very dependent on the delivery parameters. For TMJ inflammation, pulsed ultrasound is applied. Pulsed ultrasound produces a nonthermal effect, therefore negating the effects that may be produced with continuous ultrasound in the presence of inflammation. Pulsed ultrasound is applied with a 5-cm or smaller sound head at a frequency of 3 MHz and low intensity of 0.5 to 0.8 W/cm² for 5 to 8 minutes.³² Ultrasonic energy is frequency-dependent, so most absorption occurs at 1 to 2 cm below the skin surface at 3 MHz. This depth of ultrasound would be appropriate for the TMJ. Low-intensity continuous ultrasound may be used as inflammation decreases as determined by the reassessment of the patient's signs and symptoms.

Low-intensity continuous or pulsed ultrasound can be used in the treatment of acute and chronic wounds to enhance the reparative process.³³ Ultrasound applied within the first week may hinder the early stages of wound healing.³³ During the proliferation phase, which usually occurs in the second week, ultrasound would be beneficial. Ultrasound of higher intensity of 1.5 W/cm² is significant in the healing of traumatized soft tissue.³⁴ Timing and dosage of ultrasound are important considerations for facilitating the healing process after arthroscopy and arthrotomy.

When exercise was combined with ultrasound versus exercise alone, patients who had ultrasound reported a higher percentage of pain relief than those who received exercise only.³⁵ When appropriate, ultrasound should precede jaw exercises, when exercises are done to assist in the treatment

of symptomatic periarticular tissue tightness or disk displacement without reduction.

Unrelated to inflammation but pertinent to the discussion of TMJ hypomobility are the biophysical effects of ultrasound on connective tissue and muscle. Elevation of tissue temperature by ultrasound preceding or during passive or active stretching has been postulated to alter the viscoelastic properties of collagen tissue and collagen molecular bonding, thus enhancing ease of stretch.³³ Unless sufficient intensity is used, the benefits of ultrasound are no greater than those of superficial heating. Ultrasound of 1.0 to 2.0 W/cm² was used on scar tissue secondary to lacerations, x-ray burns, and Dupuytren's contracture to reduce the size of keloids.³⁶ Ultrasound plays an important adjunctive role in the treatment of hypomobility secondary to capsular tightness and disk displacement without reduction by increasing collagen tissue extensibility.

Like superficial heating, ultrasound can decrease extrafusal fiber activity via a reflex mechanism. However, it may also affect the muscle spindle activity more directly because of its deeper penetration.³³ Ultrasound can elevate skeletal muscle temperature at the bone-muscle interface. Superficial or deep heating agents play an important adjunctive role in the treatment of TMD—M.

The following are general contraindications of superficial and deep heating:³⁰

- Circulatory impairments that are prone to increased bleeding or hemorrhage (e.g., hemophilia)
- Post acute trauma with bleeding not controlled
- Potential capillary fragility in patients on long-term steroid therapy
- Elevation of tissues at the site of malignancy

Cold

Cryotherapy is used for reducing fever, controlling bleeding, decreasing muscle guarding, and, pertinent to this discussion, helping reduce inflammation and associated pain. Cold reduces bleeding by arteriolar vasoconstriction; decreases inflammation by decreasing metabolism and levels of vasoactive agents, e.g., histamine; and elevates the pain threshold by decreasing nerve-con-

duction velocity.³⁷ Cold to control inflammation can be applied to the TMJ by ice packs, ice cubes/cups for ice massage, or ice-soaked towels. During ice massage, the patient often experiences four distinct sensations: cold, burning, aching, then analgesia.

Unrelated to inflammation but pertinent to the discussion of TMJ—M are the biophysical effects of cold on muscle. Cold reduces muscle tension by decreasing pain and by decreasing the sensitivity of the muscle spindle afferent fiber discharge.³⁷ When applied by a vapocoolant to the affected muscle on passive stretch, cold is postulated to stimulate cutaneous afferents, producing a reflex decrease in gamma motoneuron firing and thus permitting more passive stretch of the muscle.³⁸

Cold should be avoided in patients with cold-sensitivity conditions, including cold urticaria, cryoglobulinemia, cold intolerance, Raynaud's phenomenon, and paroxysmal cold hemoglobinurias.³⁷ Cold should not be applied over circulation-compromised areas (e.g., in peripheral vascular disease). Care should also be taken when the patient is hypertensive because cold can cause a transient increase in systolic and diastolic blood pressure.³⁷ Prolonged cold application for 1 hour or more over a superficial peripheral nerve can lead to neurapraxia or axonotmesis. Finally, the psychological response of the patient should be taken into account when applying cryotherapy.

There are certain clinical situations in which either heat or cold is clearly preferred. Often the choice is empirical. The following factors should be considered:³⁹

1. Stage of injury
2. Area of body treated
3. Medical status
4. Patient preference

Iontophoresis

In iontophoresis a battery-powered system is used to deliver water-soluble ionizing drugs through the skin. The effects are dependent on the drugs used. Common drugs used for inflammation of the TMJ are dexamethasone sodium phosphate, methylprednisolone sodium succinate (Solu-Medrol), and lidocaine hydrochloride. Comparison of iontophoresis to systemic therapy and local injection showed that tissue concentration of the administered ion was higher for the

iontophoresis than that obtained with systemic therapy but lower than that obtained by local injection.⁴⁰ Multiple treatments of iontophoresis would seem to be needed to be comparable to a single injection. However, the author is not aware of any research systematically comparing the ability of iontophoresis and joint injection to deliver medication to the TMJ. Therefore, it is not known whether these two treatment modalities are comparable.

Case reports, case series, and a clinical trial have shown patients who have been clinically diagnosed with capsulitis or tendonitis to have a decrease in signs and symptoms after the application of iontophoresis.⁴¹⁻⁴⁴ One study concluded that iontophoresis was no more effective than a saline placebo in providing pain relief or improvement in mandibular range of motion in patients with TMJ pain.⁴⁵ A recent double-blind randomized study investigated the effects of iontophoresis on limited mouth opening secondary to pain and a disk displacement without reduction.⁴⁶ All patients were asked to discontinue over-the-counter and prescribed medication for their condition. Use of intraoral appliances, jaw exercises, and self-administered heat or ice treatments was also discontinued. Results suggested that iontophoresis was effective in improving mandibular function, but not in reducing pain, in patients who had concurrent TMJ capsulitis and disk displacement without reduction. Iontophoresis is used by this author when the examination suggests localized inflammation of the periarticular tissue of the TMJ and/or masseter muscle.

Transcutaneous Electrical Nerve Stimulation

Transcutaneous electrical nerve stimulation (TENS) employs a small, portable, battery-operated unit. The unit has controls allowing the clinician to adjust the frequency, pulse width, and amplitude of an alternating current that is administered to the patient by electrodes of a variety of sizes and shapes. TENS has long been used as a means of pain control, largely on the basis of the "gait control theory" of Melzack and Wall. Gait control selectively activates large-diameter proprioceptive afferents to inhibit or balance small-diameter nociceptive input at the dorsal horn to block the

patient's perception of "pain."⁴⁷ TENS has also been shown to produce reflex vasodilatation and decrease muscle guarding, thereby enhancing circulation and lymphatic drainage.⁴⁷ Optimal stimulation parameters for TMJ inflammation are a frequency of 1 Hz, pulse width of 75 to 100 μ sec, and amplitude to the point of mild visible and rhythmic mandibular elevation without tooth or appliance contact.³² Mannheimer states, "TENS performed in this manner is not designed to produce pain relief as its primary goal but to enhance the healing process by reducing the effects of inflammation."³² Contraindications to TENS are epilepsy, transient ischemic attacks, and postcerebrovascular accidents.⁴⁷

Hypermobility

The cause of hypermobility of the TMJ is unknown. Potential predisposing factors that have been suggested range from joint laxity to psychiatric disorders to skeletal abnormalities.⁴⁸ One study suggests that systemic hypermobility (ligament laxity) may be closely related to TMJ hypermobility.⁴⁹ Two studies on systemic hypermobility investigated whether a correlation exists between systemic hypermobility and disk displacements/osteoarthritis of the TMJ.^{50, 51} In one of the studies, the authors concluded that disk displacements of the TMJ are a sign of "joint hypermobility syndrome."⁵⁰ The other study showed that generalized joint hypermobility was not a predisposing factor to TMJ disk displacements and osteoarthritis.⁵¹ Regardless of the proposed etiologic factors for hypermobility, it is more often than not asymptomatic.

Diagnosis of Hypermobility

Hypermobility of the TMJ is identified when the condyle functions beyond the articular crest—an anatomic landmark on the temporal bone. The condyle moving past the articular crest is then functioning on the articular tubercle. A definitive diagnosis can only be made by a radiograph of the TMJ when the patient's mouth is fully opened. Hypermobility is such a benign condition that the expense and exposure to radiation do not justify radiographs for the sole purpose of identifying the condition. Often the

clinical examination is sufficient to determine whether hypermobility is present.²⁶

Subjectively, the patient often describes the jaw as “going out of place.” This may be perceived as the patient touches the lateral poles of the TMJ. As the patient opens his or her mouth, the condyle(s) translates a significant distance anteriorly, causing the patient to interpret the sensation as the joint’s “going out of place.”

Objectively, the clinician palpates the lateral pole(s) of the TMJs to identify whether one or both of the following are present:

- Excessive anterior excursion of the lateral pole(s) during mandibular opening
- A “juddering” movement of the condyle(s) at the end of mandibular opening and at the beginning of mandibular closing, as a result of the condyle’s going past the articular crest onto the articular tubercle during opening and closing movements of the mandible

This clinical examination for hypermobility has obvious flaws that contribute to false-positive or false-negative conclusions. Hypermobility, however, is one condition in which overdiagnosis or underdiagnosis is acceptable. The reason is that the condition is benign and the treatment is conservative, cost-effective, and reversible.

Treatment for Hypermobility

Only when hypermobility is accompanied by inflammation is treatment required, especially if pain occurs or is intensified at the end of full-mouth opening. Even if hypermobility were not present, avoiding or controlling the end range of joint movement would be therapeutic for the management of inflammation. Controlling asymptomatic hypermobility would only be necessary if the patient insisted that he or she did not want to experience the sensation of the jaw’s “going out of place.”

Primary treatment is simply telling the patient, “Do not open your mouth so wide” (conservative); if the patient applies this awareness exercise on a daily basis, only one treatment is needed (cost-effective). Patients only open wide if they eat a large sandwich, receive dental work, or have surgery requiring intubation. Because situations other than nonelective dental or surgi-

cal situations can be avoided, the only time a patient needs to open the mouth wide is in yawning. Yawning can be controlled by having the patient yawn with tongue pressed up against the palate of the mouth. Pressing the tongue up limits the patient’s mouth opening to about 25 mm. The therapist can teach the patient additional cognitive awareness/neuromuscular exercises if necessary.²⁶

Hypomobility

Hypomobility is limitation in functional movements of the mandible secondary to arthrogenous involvement. A review of the physiologic and neurophysiologic sequelae of hypomobility affecting the articular cartilage and mechanoreceptor activity of diarthrodial joints can be found in the reference section.²⁶ Functional movements affected by hypomobility include chewing food, opening the mouth to receive food, talking, and yawning.

There are various causes of TMD—A hypomobility. Ankylosis (bony or fibrous) results in complete restriction of movement. Hypomobility can be a result of fractures involving the TMJ or severe degenerative joint disease, neoplasia, aplasia, hypoplasia, and dysplasia of the bony components of the TMJ. If needed, treatment for these previous causes for hypomobility of TMD—A is rendered by the oral surgeon. Hypomobility may also stem from previous TMJ or orthognathic surgery. The more common causes of TMD—A hypomobility, which also respond best to physical therapy treatments, are *periarticular tissue tightness* and a *disk displacement without reduction*. Before a discussion of periarticular tissue tightness and disk displacement without reduction, the diagnosis of hypomobility is discussed.

Diagnosis of Hypomobility

Mandibular depression, protrusion, and lateral excursions are osteokinematic (movement of bone, e.g., mandible) movements frequently observed by the clinician to diagnose hypomobility. Mandibular hypomobility is present when any one or a combination of the following limited osteokinematic movements are present:

Mandibular depression: Actively, the patient is unable to achieve 36 mm of interincisal opening.

Functional interincisal opening is 36 mm and greater.

Mandibular protrusion: Actively, the patient is unable to achieve end-to-end position with the central incisors (this does not apply to class III patients).

Functional protrusion occurs when the lower central incisors can move past the upper central incisors.

Mandibular lateral excursions: Actively, during mandibular movement to the right, the patient is unable to achieve end-to-end position of the right bottom canine to the right upper canine; this also applies to the left canines when moving left.

Functional lateral excursion to the right occurs when the right bottom canine can move past the right upper canine; this also applies to the left canines during left lateral excursion.

Limitations and aberrant movements of the mandible are classically the same for periarticular tissue tightness and a disk displacement without reduction. The following is what the clinician typically sees in unilateral hypomobility of the TMJ:

Depression: Not functional, less than 25 mm of interincisal opening with the mandible deflecting to the side of the involved joint; deflection is movement of the mandible away from midline.

Protrusion: Not functional, with the mandible deflecting to the side of the involved joint.

Lateral excursion: Not functional to the opposite side of the involved joint with lateral excursion of the mandible to the same side functional.

Since limited mandibular dynamics are the same for periarticular tissue tightness and a disk displacement without reduction, history is the key to making the differential diagnosis. *Periarticular tissue tightness* is usually associated with a history of chronic inflammation resulting from trauma, long-term limited mobility, or strict immobiliza-

tion. *Disk displacement without reduction* usually has a sudden onset preceded by a history suggesting a disk displacement with reduction.

Limited opening can also be caused by a nonarthrogenous condition, such as TMD—M. Hypomobility of TMD—A origin and hypomobility of TMD—M origin can be differentiated by having the patient move the mandible into protrusion and lateral excursions. If protrusion and lateral excursions are normal, then TMD—M should be suspected. If it is limited, then TMD—A is suspected. Myogenous involvement typically does not restrict protrusive and lateral excursions as does arthrogenous involvement. A complete history and clinical examination confirm whether hypomobility is of an arthrogenous or myogenous origin.^{26, 28} The treatment for hypomobility secondary to TMD—M is discussed later.

Periarticular Tissue Tightness

Periarticular tissue is the capsular-ligamentous tissue of joints. The entire lateral aspect of the TMJ capsule is thickened, forming the temporomandibular ligament. The TMJ ligament is regarded as part of and inseparable from the capsule. Here the terms *periarticular tissue* and *capsule* can be considered synonymous. Biomechanical and biochemical changes that occur with periarticular tissues of diarthrodial joints as a result of hypomobility have been well documented in the literature.⁵²⁻⁵⁶

Disk Displacement

Disk displacement is a very common condition affecting the TMJs. Experts disagree over the cause and treatment of disk displacements. The role of physical therapy in the treatment of disk displacement is based on the recognition and acceptance that the morbidity of the disk functioning off the condyle has not been demonstrated to be entirely pathologic and that patients can function in a pain-free state with minimal limitations in function with a disk displacement.⁵⁷⁻⁶⁴ However, some researchers believe that the disk should not function off the condyle and that all efforts should be made to reposition the disk to its "normal" position on the condyle. Repositioning the disk to its normal position often requires

various mouth appliances and/or arthro-tomy and possible orthognathic surgical procedures. Long-term studies have shown that in a high number of patients who receive treatment to recapture the disk, the disk is often displaced again.⁶⁵⁻⁶⁸

I believe both treatment approaches are appropriate when based on rigid patient selection criteria. Criteria for making a decision to treat the disk off or on the condyle include the patient's age, traumatic or insidious onset, amount of time the disk has been displaced, symptomatic or asymptomatic, degree of functional limitations, and patient's response to previous treatment. Essentially, the decision to treat the disk on or off the condyle is based on the clinician's judgment and the patient's being well informed. *The patient needs to be well informed* of the pros and cons of all nonsurgical (no treatment, use of mouth appliance, and physical therapy) and surgical treatments (arthroscopy, arthro-tomy, and orthognathic).

There are three well-defined stages of a disk displacement.⁹ A discussion of the role of physical therapy for each stage follows.

Disk Displacement with Reduction—Stage I The diagnosis of a stage I disk displacement with reduction is classically made by the presence of joint noises, e.g., the reciprocal click. Additional clinical examination procedures can be found in the literature.^{26, 28} The key to understanding the treatment of a stage I displacement is to know that it is functional. Patients can chew, talk, and yawn; they simply have joint noises as they perform these activities. Some patients with a stage I displacement may have a catch on opening related to the disk, or the opening becomes an effort because the "reduction" of the disk has become difficult. A treatment choice for this specific patient population may be progression to a disk displacement without reduction that is functional. Some patients with a stage I displacement may have inflammation and/or TMD—M. Patients with a stage I displacement and inflammation and/or TMD—M should be approached with treatment(s) as though the conditions simply coexist and are not related.

Patient Education The primary treatment offered by the physical therapist for a stage I displacement is education of the patient about the condition. If the decision

to treat the disk off the condyle is made, patients need to be told that a stage I displacement and the associated joint noises do not mean that the joint is bad, diseased, or pathologic. The patient needs to know that he or she can function pain-free even with the disk(s) displaced. If the patient is consulting both a dentist and a physical therapist, it is important that the two professionals communicate as to what the emphasis should be on joint noises. Once agreement has been achieved, the two professionals can respond to the patient's questions in a consistent manner.

Inflammation If a stage I displacement is painful, pain results because inflammation is present. The initial approach that should be taken by the physical therapist is to assume that the stage I displacement and inflammation are independent and are simply coexisting. Treatment for inflammation has been covered. More often than not, as the inflammation is resolved, the patient returns to functioning in a pain-free state with joint noises. If the inflammation recurs or is not resolved to the patient's satisfaction, TMD—M is suspected.

TMD—M Treatment for TMD—M or masticatory muscle hyperactivity is covered later. TMD—M can be a source of the patient's symptoms, e.g., myofascial pain. As in the case of inflammation, a stage I displacement and TMD—M can also be independent of one another and simply coexisting. Often as TMD—M is treated, the patient states that the joint noises have lessened or have gone away. In such cases, the decrease in joint noises is assumed to be secondary to reduced joint loading because masticatory muscle hyperactivity has decreased.

Regardless of the inflammation and/or TMD—M that may be associated with a stage I displacement, resolution of the inflammation and/or TMD—M should occur in or around a 4- to 6-week period, with physical therapy intervention two to three times a week. If inflammation and/or TMD—M continues, a referral to a dentist for an appropriate mouth appliance to assist in controlling TMD—M is indicated. If the patient already has an appropriate mouth appliance and has undergone physical therapy treatments, he or she should be referred to an oral surgeon. In this situation, the stage I displacement is perpetuating the inflam-

mation and/or TMD—M, and surgery may now be indicated.

Disk Displacement without Reduction—Stage II The diagnosis of a stage II disk displacement without reduction is made by history and altered mandibular dynamics. Patients often report having had joint noises prior to their limited function, suggesting a stage I displacement preceded the stage II displacement. Stage II displacement interferes with the patient's ability to perform functional activities. Objectively, altered mandibular dynamics associated with a unilateral stage II displacement are observed. The description of the altered mandibular dynamics seen with a stage II displacement is given in the section on hypomobility.

Physical therapists treating patients with a stage II displacement from a nondental referral are advised to have the patient consult a dentist who specializes in the treatment of TMD for a second opinion. For liability reasons, the patient should have a second opinion stating that physical therapy is a suitable option in the treatment of the stage II displacement. Treatment for a stage II displacement is covered later.

Disk Displacement with Osteoarthritis—Stage III The diagnosis of a stage III disk displacement with osteoarthritis is made by the presence of crepitus throughout the full range of opening and closing movements of the mandible. Stage III displacement, degenerative joint disease, and osteoarthritis are considered synonymous in regard to the disk's being permanently displaced and undergoing significant changes. Though the integrity and mechanics of the TMJ are disturbed, the TMJ seems to adapt adequately to this altered disk position.⁶⁹ Patients who have a stage III displacement can have pain-free and near-normal mandibular dynamics.

Treatment—Stage III As in the treatment of a stage I displacement, physical therapy intervention centers on patient education and, if present, the treatment of inflammation and/or TMD—M.

Treatment for Periarticular Tissue Tightness and Stage II Displacement

The following therapeutic procedures and home exercises are used for either a capsu-

lar tightness or a stage II displacement. What is different in the application of the treatments for these two conditions is the degree of aggressiveness with which they are applied. For example, capsular tightness secondary to arthrotomy involving a repair to the disk warrants a less aggressive treatment approach than that used when discectomy is performed. Though capsular tightness can be the result in both cases, the arthrotomy with the disk repair adds another variable to the postoperative rehabilitation. The disk repair often involves the "shortening" of disk attachments. Therefore, limitation in function may also be due to the surgical repair itself. This should not detract from the need to establish early mobility as long as the therapeutic procedures are applied in a controlled manner by the patient and physical therapist.⁷⁰ The time between the surgery and physical therapy would also dictate how aggressive the physical therapist would be in restoring functional movements of the mandible. Regarding stage II displacement, the only limitation in treatment is the patient's tolerance of the therapeutic procedures, which is discussed next.

Regardless of whether the patient has capsular tightness or a stage II displacement, an active interincisal opening of 25 mm should be expected.²⁶ Rotation of the condyle without any condylar translation allows up to 25 mm of opening. If the patient is unable to achieve 25 mm, this limitation is usually secondary to joint inflammation and/or TMD—M. Before therapeutic procedures are initiated for joint hypomobility, inflammation and/or TMD—M must be managed.

Modalities previously discussed can be used before, during, or after each therapeutic procedure. The choice and sequencing of modalities and therapeutic procedures as well as the degree of aggressiveness and length of time the therapeutic procedures are applied are determined by the patient's history and current signs and symptoms.

Finger Spread Stretch The patient rests supine with the cervical spine properly supported. The clinician stands at the head of the patient. Using the hand opposite the involved joint, the clinician places the thumb on the tip of the patient's lower central incisors and the index finger on the tip of the top central incisors. The patient is

asked to open actively as the clinician follows with pressure of the fingertips on the patient's incisors. At the end of the available range, additional pressure is applied with the clinician's fingertips. While performing this exercise, the patient can be asked to extend the head to facilitate mouth opening.

An important modification to this manual exercise is performed at the time the patient can actively achieve opening to 25 to 30 mm. The patient is instructed to protrude the mandible forward and then open the mouth. Protruding the mandible translates the condyle to prevent forcing rotation beyond 25 mm. Valuable feedback from the therapist to the patient during the stretch helps the patient to understand what is expected when he or she does this exercise at home.

Joint Mobilization Techniques *Joint mobilization* is a general term that might be applied to any active or passive attempt to increase movement of a joint. Passive joint mobilization has been a popular treatment for the restoration of joint motion for many years. For better patient relaxation, the following techniques for the TMJ are best applied with the patient supine rather than sitting.

Distraction Distraction is a force applied parallel to the longitudinal axis of the bone—in this case, the neck of the condyle. Distraction is the first choice among intraoral techniques because of safety, ease, and effectiveness.

The patient is lying supine with appropriate support given to the cervical spine. The clinician stands on the opposite side of the involved joint. The clinician's thumb is placed on the patient's mandibular molars, on the side of the involved joint. If limitation of jaw opening prevents the placement of the thumb on top of the molars, the thumb may be positioned in the premolar area. The remaining fingers are wrapped around the chin comfortably. The clinician's other hand stabilizes the patient's cranium, with the middle or index finger palpating the lateral pole of the condyle for movement. While performing the distraction technique, the patient can actively open and close on command, using minimal muscle contraction. When the patient relaxes, additional distraction forces can be applied. Performing the distraction technique with active participation by the patient allows the patient to experience a less stressful, less painful movement of the joint. While performing

distraction, the patient can be asked to extend the head to facilitate mouth opening.

Translation The clinician's body position and hand placements are the same as in the distraction technique. The clinician's mobilizing hand translates the condyle in an anterior direction by passively pulling on the mandible. Translation is not performed only in the sagittal plane but also slightly across midline. Translation should be performed during varying degrees of distraction. Distraction simultaneous with translation helps the patient tolerate this technique better.

Lateral Glide The clinician's body position and the stabilizing hand are placed as in the distraction technique. The thumb contact of the mobilizing hand, however, is different. Thumb contact for this technique is on the top/inside of the molars. The rest of the fingers wrap around the mandible comfortably. Lateral glide is performed by pressing laterally with the thumb, while force is directed toward the table and patient's feet (final direction of the force generated by the thumb is as in following the slope of the patient's shoulder). This direction of force minimizes the discomfort on the contralateral side a patient would experience if a lateral force alone were applied.

Graded rhythmic oscillatory movements may be applied simultaneously with distraction, translation, and lateral glide joint mobilization techniques. Rhythmic oscillatory movements aid in pain inhibition and enhancement of joint mechanoreceptor activity.²⁶

General rules to follow when applying joint mobilization techniques are as follows:

1. Patient and clinician must be properly positioned and relaxed.
2. Patient should be stabilized firmly.
3. Clinician must be willing to modify the technique on the basis of the tissue's response and patient's tolerance.
4. Clinician should use minimal force consistent with achieving the objective of the technique, which is to restore function.

Mobilization techniques when performed incorrectly or not indicated may result in the following:

1. Increase in pain
2. Increase in muscle guarding
3. Decrease in mobility

Static Tongue Blade Technique

Tongue blades are used to apply a low-load prolonged stretch (LLPS) to promote long-lasting elongation of the periarticular tissue. The effectiveness of LLPS has been well documented by laboratory studies.⁷¹⁻⁷³ The patient should be positioned supine for this procedure. The patient is instructed to place tongue blades on the side of the involved joint in the area of the molars. Using the tongue blades, the patient is told to "take up the slack and then some." If more than five tongue blades are used, try taping or gluing all but three of them together. The last tongue blade can be slid in or out between the remaining tongue blades. Actual duration of the LLPS is dependent on patient tolerance, but working up to a 10-minute stretch is adequate.

Studies have shown that raising the temperature of the stretched tissue and allowing the tissue to cool in a loaded position produce greater elongation of the treated tissue(s).⁷³⁻⁷⁵ Ultrasound is used to enhance tissue extensibility with tongue blades in place.

Continuous Passive Motion The importance of initiating early motion during the inflammatory phase, especially after surgical intervention, is widely accepted in the rehabilitation of other joints, such as the knee.⁷⁶ Use of passive motion on a continuous basis, as introduced by Salter and associates in 1970, has become an important area of study.⁷⁶ Salter has shown that continuous passive motion (CPM) applied in experimental animal studies is beneficial to soft tissue healing, bone and cartilage healing, swelling, hemarthrosis, and joint function.⁷⁶ It is reported that CPM in humans has resulted in positive effects on joint effusions, wound edema, pain, and reduction of capsular contractures and joint stiffness.^{77, 78} Even though CPM has been used in a variety of orthopedic conditions, present indications and clinical studies for the use of CPM have largely focused on the rehabilitation of various knee disorders.

Though the potential benefit of CPM is known, there is insufficient clinical research to define the most appropriate device and protocol after arthroscopy or arthrotomy to the TMJ.⁷⁹ The term *CPM* may need to change because it indicates continuous motion. Motorized devices used on the TMJ are short term. Measurable outcomes in terms

of therapeutic value and cost-effectiveness of CPM for TMD have not been fully researched. Timely physical therapy for TMD—A hypomobility produces therapeutic outcomes acceptable to the patient, surgeon, and insurance companies while avoiding the inconvenience and cost of CPM.

Home Exercise Program for Hypomobility

Tongue Up and Open/Close Patients having limited mobility with associated inflammation should be instructed to open and close the mouth with the tongue against the palate. Opening and closing with tongue up limit the condyle to rotation and prevent translation. This exercise creates controlled movement while increasing the patient's confidence that movement can occur without an increase in pain. This exercise can be performed numerous times throughout the day. Once inflammation is decreased, opening and closing can be performed with tongue down to allow opening beyond 25 mm.

Finger Spread Stretch and Static Tongue Blade Stretch After proper instructions to the patient, finger spread stretch and static tongue blade stretch therapeutic procedures can be continued at home. Patients do not have access to various modalities available in the physical therapist clinic. They can, however, apply heat and/or cold and, if available, a home TENS unit. Finger spread stretch can be applied with few repetitions many times a day, whereas static tongue blade exercise is applied for up to 10 minutes one to three times per day.

Dental Roll Distraction The dental roll distraction exercise is designed to distract the condyle. This is a *passive*, not an active exercise. The patient sits in a comfortable supported position. The patient places a dental roll ¼ inch in diameter between the back molars on the side to be distracted. The patient places the palm of the hand under the chin. The other hand is placed on the top of the head toward the forehead. Both hands are in front of the dental roll. With the patient relaxing the jaw, the chin hand applies a slight pressure superiorly as the other hand stabilizes the head. Hand pressure in the presence of relaxed jaw muscles creates a pivot over the dental roll, resulting in distraction of the

condyle. The patient may elect to perform active protrusion several times while hand pressure is maintained. This procedure can be done 10 to 15 times each session, repeating 3 to 6 times per day.

Horizontal Tongue Blade Exercise The patient places seven tongue blades horizontally between the upper and lower central incisors. The patient does not bite on the tongue blades; rather the tongue blades are held in position by the patient's grasping the ends of the tongue blades. Seven tongue blades equals approximately 11 mm when placed between the central incisors. Translation normally begins at approximately 11 mm.⁸⁰ With tongue blades in place, the patient moves the mandible forward and backward repetitively 30 times per session, repeating 1 to 3 times per day. The forward and back motion of the mandible improves condylar translation. Should repetitive translation be performed with less than 11 mm of jaw opening, the patient usually feels pain or discomfort.

Capsule and Stage II Tissue Changes in Response to Treatment

Capsule Connective tissue responds to mechanical stress in a time-dependent or viscoelastic manner. The force/load applied needs to exceed the elastic range of a tissue; otherwise, the tissue returns to its original shape and prevents permanent elongation of the tissue.^{81, 82} Beyond the elastic range or yield point is the plastic range of the tissue.^{81, 82} Long-lasting elongation is dependent upon the intensity and duration of force applied.^{83, 84} If loading is continued too far into the plastic range (fatigue point), tissue damage may result. The therapeutic procedures and home exercises previously discussed need to exceed the elastic range of the tight capsular tissue for permanent elongation to be achieved.

Stage II Displacement It is nearly impossible to predict whether a patient will regain function by going to a stage I displacement or progress to a stage III displacement. *Patients must be well informed and in agreement as to the possibilities.* If inflammation and/or TMD—M is present and is treated, it is not unusual for a stage II displacement to progress to a stage I or to

a stage III displacement with no therapeutic procedures performed. When therapeutic procedures and a home exercise program are performed, they have been shown to be highly effective as a conservative, noninvasive treatment for a stage II displacement.^{85–91} Signs and symptoms improved by these procedures are not the result of the disk's reducing to its normal position.⁹² Rather, a return to functional movements of the mandible may be caused by elongation of the posterior attachment (PA) with further anterior displacement and advanced deformation of the disk, progressing from a stage II to a stage III displacement.⁹³ The condyle then is capable of functioning on the PA that has remolded to become fibrotic.⁹⁴ This change in the PA allows it to withstand loading from the condyle. However, in some patients, the PA does not remold, remolds inadequately, or remolds adequately for a time and then fails.⁹⁵ In such failed cases, an oral surgery consultation is indicated.

PHYSICAL THERAPY MANAGEMENT FOR TMD—A/M

The vast number of patients with TMD have both an arthrogenous and myogenous involvement. The interrelationship between arthrogenous and myogenous components is unclear.^{96, 97} It is not unlikely that the arthrogenous and myogenous components are simply coexisting entities in a considerable number of patients.⁹⁶ As a general rule, if the patient's condition has both an arthrogenous and a myogenous component, the myogenous component should be treated first, except when *significant* inflammation or stage II displacement is present. These two conditions may require treatment at the same time the myogenous component is treated.

PHYSICAL THERAPY MANAGEMENT FOR TMD—M

The American Academy of Orofacial Pain lists six subset diagnoses for masticatory muscle disorders, such as myofascial pain, myositis, spasm, protective splinting, contracture, and neoplasia, which are applica-

ble to TMD—M.⁹ Fibromyalgia, also termed *myofascitis*, *myofibrositis*, or *fibrositis*, is often confused with myofascial pain.⁹⁸ Tests used to differentiate among the various masticatory muscle disorders and between myofascial pain and fibromyalgia have had poor reliability and validity. Attempts to differentiate the different muscle conditions are more academic and offer little clinical value. Even if reliability and validity were established for differentiating the various muscle conditions, the treatment(s) offered would be the same in most cases. This chapter condenses all masticatory muscle disorders in one classification, referred to as *masticatory muscle hyperactivity* (MMH). It is considered in the following discussion as a source of the patient's head and jaw symptoms.

Examination for Masticatory Muscle Hyperactivity

Symptoms associated with diurnal or nocturnal MMH range from pain or soreness to tightness and/or fatigue in the jaw and temple areas of the head. Some patients may have MMH symptoms only during the day and not at night. Other patients may not have symptoms during the day; symptoms may wake the patient at night or be present first thing in the morning.

Clinical examination for MMH involves digital palpation of the muscles of mastication. Numerous muscles are involved in the act of mastication. From a clinical point of view this chapter focuses on only the three muscles that are responsible for elevating the mandible—namely, the medial pterygoid, masseter, and temporalis muscles. The specificity of palpating the medial pterygoid is low because of the location of the medial pterygoid and the overlying of adjacent muscles to the medial pterygoid. Reliability and validity are better when palpating the temporalis and masseter muscles. This author relies on the masseter and temporalis as “scout muscles” to signify whether MMH is present. Palpation may reveal objective tightness of these muscles and/or the patient may subjectively state that he or she experiences tenderness or pain during the palpation examination. The amount of opening is not a reliable sign for diagnosis of MMH.

Treatment for Masticatory Muscle Hyperactivity

Oral Modification and Awareness

It is essential that patients are made aware of bad habits that require use of the muscles of mastication. Patients must be educated in how they can control and/or modify their bad habits.⁹⁹ Patient awareness of a problem over which he or she has some degree of control may be one of the essential and common components of a successful outcome.¹⁰⁰ Habits such as fingernail biting, or gum or ice chewing result in MMH if done repetitiously. The awareness and avoidance of such habits can therefore help in reducing MMH. Minimizing essential functional activities, such as talking, chewing food (nonchew diet), and drinking liquid (versus sucking fluid out of a straw), assists in reducing MMH.

Modalities

Modalities previously reviewed can also be used to decrease MMH. Superficial heat can be applied to the masseter and temporalis. Ultrasound and iontophoresis are limited to the masseter since hair may compromise the application of these modalities to the temporalis. When hyperactivity of intraoral muscles is suspected, intraoral electric stimulation rendered by a hand-held probe has been clinically helpful in achieving relaxation of intraoral muscles.¹⁰¹

Massage

Extraoral or intraoral massage or pressure point techniques may be offered to the muscles of mastication. The specific application of massage or pressure point techniques to the muscles is more an art than a science.

Therapeutic Exercises

Contracting one muscle or group of muscles causes reflex inhibition of the antagonist muscle(s). Contraction of the depressor muscles of the mandible causes a reciprocal inhibition of the elevator (antagonist) muscles. This can be done by having the patient place one fist under the chin to resist mouth

opening. Questions of duration and the amount of resistance needed to initiate inhibition of the antagonist muscles in a dysfunctional state remain unanswered.^{102, 103}

A maximum isometric contraction of a muscle can also induce relaxation of the contracting muscle. To inhibit the elevator muscles of the mandible effectively, the elevator muscles should be in a position of slight stretch. The patient positions the mouth slightly open and places the index and middle fingers of one hand over the lower central incisors. The patient attempts to close the mouth against an unyielding force: the fingers. For the elevator muscles to relax reflexly, a great amount of tension is required to stimulate the Golgi tendon organs (GTOs). Information on how long this relaxation/inhibition persists is limited.¹⁰⁴ I am concerned about performing a maximum isometric contraction of the elevator muscles. Depending on the degree of myofascial pain stemming from the elevator muscles and the degree of inflammation of the TMJ, such a maximum contraction often increases the patient's signs and symptoms. Good clinical decision making is mandated in this instance. I prefer minimal isometric contraction. Minimal contraction does not stimulate the GTOs. Minimal contraction gives the patient cortical awareness of the muscles contracting and then relaxing. Over time, this form of light isometric contraction may provide relaxation if done in conjunction with other forms of treatment.

Exercising the muscles of mastication for the purpose of increasing strength is the exception rather than the rule. I seldom find weak jaw muscles. The idea that a jaw muscle can be isolated so as to "test" it to see whether it is weak is difficult to accept. Even if the muscle can be isolated, a false-positive muscle testing result can be caused by (1) the patient's unwillingness to contract maximally, (2) pain that limits maximum contraction, and (3) pseudo-weakness secondary to reflex inhibition.^{105, 106} Isometric exercise for the purpose of strengthening suspected jaw muscle weakness needs to be re-evaluated. However, muscle weakness can be suspected after strict immobilization, e.g., intermaxillary fixation, and may require instructing the patient on isometric, isotonic, and/or eccentric strengthening jaw exercises.

Neuromuscular Re-educational Exercises

If not a result of the oral habit(s) previously addressed, MMH is the result of bruxism. Bruxism and MMH are considered synonymous in this discussion. *Bruxism* has been defined as diurnal or nocturnal para-functional activity, including clenching, bracing, gnashing, and/or grinding of the teeth.¹⁹ At one time, stress was considered a leading cause of bruxism. Researchers have questioned the relationship between the stress-prone personality and bruxism, which may progress from normal behavior to abnormal behavior.^{96, 107, 108} Though epidemiologic surveys indicate positive correlations between bruxism and TMD—A, any cause-and-effect relationship is still speculative.⁹⁶

Since the cause of bruxism is unknown, the treatment becomes palliative. From the physical therapist's perspective, diurnal parafunction is controlled through a neuromuscular awareness exercise, known as *tongue up/teeth apart/breathe* (TTB). *Tongue up* is the normal rest position of the tongue.²⁶ Patients need to be educated about the normal rest position, which is up and forward with the tip of the tongue lightly touching the back side of the upper central incisors. The tongue in this position should help patients develop an awareness that their back teeth are slightly apart. Instructing and helping patients know how to breathe with the diaphragm will further encourage relaxation. Often, patients are observed breathing with their accessory muscles (e.g., scaleni and sternocleidomastoids), which only contributes to muscle hyperactivity. Motivating patients to perform TTB is largely fueled by the clinician's understanding of the physiologic mechanisms underlying TTB and the ability to demonstrate and communicate the application of this neuromuscular re-educational exercise in a meaningful way.²⁶

Myofascial pain resulting from bruxism can be reduced through oral modification/awareness, modalities, massage, therapeutic, and neuromuscular re-educational exercises. In another approach to treating diurnal and nocturnal bruxism the physical therapist treats the cervical spine. Because the cervical spine's contribution to bruxism is theorized, the reader must draw his or

her own conclusions. Managing bruxism by treating the cervical spine is discussed in the following section.

Cervical Spine Considerations

The cervical spine has been overlooked by the dental profession in both the clinical and research areas. Only recently has the dental profession considered the cervical spine in clinical practice. The Dental Practice Act Committee of the American Academy of Orofacial Pain convened in 1995 to study the scope of TMD/orofacial pain and dental practice. The term *TMD and orofacial pain* became useful to reflect the expanded scope of dental practice which now includes neurovascular and neuropathic pain management.¹⁰⁹ The committee concluded that the scope of dental practice for TMD and orofacial pain “is expanding beyond the teeth and oral cavity to include the diagnosis and treatment of disorders affecting the head and neck.”¹⁰⁹ The term *orofacial pain* was judged to be too limiting because it implies anatomic limitations that are not consistent with the scope of dental practice.¹⁰⁹ To be consistent with contemporary practice, the term *head and neck pain management* is recommended to emphasize the expanding scope of dentistry.¹⁰⁹ Head and neck pain management encompasses TMD and orofacial pain.¹⁰⁹

I believe viewing TMD as only a part of head and neck pain management is a step in the right direction. Physical therapists have been evaluating and treating patients' neuromuscular-skeletal involvement from this point of view for decades (e.g., treatment of medial knee pain may require supporting the arches of the foot, and relaxing back muscles may require stretching the hamstrings). However, my concern related to this expanding scope in dentistry is expressed in the following statement from the report of the Dental Practice Act Committee of the American Academy of Orofacial Pain:¹⁰⁹

As a result of the comprehensive nature of dental education and the experience of clinical practice, only the dentist is able to assess whether intraoral pain, jaw pain, and facial pain originate from local causes or as a result of referred pain from cervical musculoskeletal

structures, neurovascular pain, or neuropathic pain.

This position on the cervical spine is extremely narrow in thought and practice. The report goes on to state that dentists' main focus for evaluating and treating the cervical spine is trigger-point injections to the muscles.¹⁰⁹ Cervical musculoskeletal structures include not only muscles but soft tissue, facet joints, ligaments, disks, and neural tissues (i.e., nerve roots and peripheral nerves). *All of these cervical musculoskeletal tissues can be a source of cephalic symptoms, not just muscles.*¹¹⁰⁻¹¹⁶ The cervical spine requires a comprehensive approach to evaluation and management. The medical doctor manages disease or pathosis stemming from the cervical spine as well as from the cranium, eyes, and ears. Fortunately for the patient, the vast majority of patients' symptoms originating in the cervical spine are of nondisease/nonpathologic origin.^{117, 118} *It is the physical therapist who has the skills to perform a comprehensive evaluation of a nondiseased cervical spine condition.* Noninvasive treatment is thereby offered not only to the cervical muscles, but also to facet joints, central and peripheral nerves, and cervical disks. If the physical therapist is having difficulties managing the cervical spine, consultation with a medical professional is indicated.¹¹⁹ The medical doctor can assist the physical therapist in the treatment of nonresponding muscle, facet joint, disk, and/or neural tissue involvement of the cervical spine.¹¹⁹

Head and neck pains are best evaluated by a team approach. For the nondiseased patient, the team must include the dental, medical, and physical therapy professions.

Prevalence of Cervical Spine Disorder Coexisting with TMD The coexistence of TMD and cervical spine disorder (CSD) is more prevalent than one might expect.^{14, 15, 120-123} One study assessing the incidence of cervical pain/dysfunction in a TMD population found that cervical spine involvement is associated with TMD 70% of the time.¹²⁴ Another study indicates that bruxism is more common in patients with myofascial pain in both the muscles of mastication and the cervical spine.¹²⁵ A recent thesis by De Wijer concluded that patients with TMD report complaints in the neck area more frequently than controls, whereas

patients with cervical spine disorder report more signs and symptoms of TMD than healthy controls.¹⁵ When TMD is divided into TMD—M and TMD—A, neck and shoulder pain is more prevalent in TMD patients with a myogenous component than in TMD patients with an arthrogenous component.^{126, 127} The coexistence of CSD and TMD—M complaints is real. The interrelationship as to cause and effect between CSD and TMD—M is still unknown. Clearly, research is needed to investigate whether such a cause-and-effect relationship exists between CSD and TMD.

There are two theories as to how a CSD may be a predisposing, precipitating, or perpetuating factor to bruxism. One theory is based on neurophysiologic influences of the tonic neck reflex on the muscles of mastication. The other theory suggests patients may brux in response to somatic pain originating from the cervical spine.

Bruxism Resulting from Tonic Neck Reflex Activity The role of the tonic neck reflex (TNR) in reflexly orienting the limbs in relationship to the head-body angle was described by Magnus in 1926.¹²⁸ Magnus,¹²⁹ in his classic work, analyzed the postural reaction of 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,¹³⁰ 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, which was not labyrinthine in origin but occurred secondary to activation of neck proprioceptors. The neck proprioceptors that are implicated consist of the facet joint receptors (the mechanoreceptors) and muscle spindle receptors.¹³¹ In 1951 McCouch and colleagues¹³¹ showed that the TNR of the decerebrate labyrinthectomized cat was 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 effect of the TNR is to induce postural changes on trunk and upper/lower extrem-

ity musculature. Excellent reviews of the extensive body of literature on the mechanisms of TNR have been published.^{132, 133} Pertinent to this discussion is the TNR influence on jaw muscle activity.

The TNR increases and decreases mandibular muscle tone through the trigemino-neck reflex, which consists of motor neurons located in the subnucleus caudalis and probably in the dorsal horn of the upper cervical spine.^{134–136} 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.¹³⁴ From these studies, *there appears to be a closely organized neurophysiologic reflex relationship between TNR activity and trigeminal motor neuron activity.*

A number of studies have confirmed that temporalis, masseter, suprahyoid, and infrahyoid muscle activity increases and decreases in response to extension and flexion of the head on the cervical spine.^{134, 137–140} Changes in masticatory muscle activity in response to extension and flexion positions of the head on the neck can be explained by the neurophysiologic reflex relationship between TNR activity and trigeminal motor neuron activity. Changes in masticatory muscles may also occur in response to the gravitational pull on the mandible when the head assumes different positions on the neck. The effect of TNR on jaw muscle activity can be appreciated in a study that observed animals biting on hard objects, such as a bone or nut, between the molars.¹³⁴ When biting on the right molars, the animal rotated its head to the left and tilted right. Such head positioning facilitated the elevator muscles of the mandible through the TNR.¹³⁴

In normal, real-life conditions TNR influences on muscle tone are expressed in complex postural responses that also reflect sensory information from other sources, such as vestibular, visual, somatic, and proprioceptive areas, all of which are likely to interact strongly with one another.¹⁴¹ The existence of the trigemino-neck reflex is real, whereas the clinical significance of bruxism is theoretical. Can sustained, aberrant mechanoreceptor activity due to facet joint dysfunction in the upper cervical spine be sufficient to activate trigeminal motor neuron activity that would result in brux-

ism? Does treatment rendered to a dysfunctional cervical spine help to manage bruxism? Though the scientific response to both these questions is not known, the clinical answer to the latter is yes.

Bruxism Resulting from Somatic Pain

Although the stress-prone personality may no longer be considered the primary or only cause of bruxism, a patient's emotional state and response to environmental stress cannot be ignored as possible predisposing, precipitating, and perpetuating factors in bruxism. Another form of stress often overlooked is somatic stress, which can appear in the form of pain, experienced by the patient as the result of nociceptive afferent activity stemming from dysfunctional muscles, facet joints, and so on, of the cervical spine. The simplified concept that I am proposing is that patients may brux in response to a "pain in the neck."

This concept may be appreciated in the studies investigating cervical whiplash and temporomandibular joint injuries. It is clear that injuries to the TMJ can be attributed to direct impact of the mandible with hard structures, as well as direct impact with a deployed air bag. However, in cervical whiplash when there is no direct trauma to the mandible, injuries to the TMJ are still reported.^{142, 143} The proposed cause-and-effect relationship is based on a series of forces acting on the mandible via the anterior neck muscles' tensing in response to posterior rotation of the cranium.¹⁴² This movement is postulated to cause hypertranslation of the condyles. Condylar hypertranslation then results in damage to the TMJ structures and eventually causes disk displacement(s).¹⁴² Several questions come to mind concerning the mechanism by which cervical whiplash has been theorized to cause injuries to the TMJ.

1. Were all the whiplash injuries the result of a rear-end impact, or were some the result of a side-impact injury? Does the postulated series of events previously described occur in a side-impact whiplash?
2. Whiplash can occur at an impact of 6 to 8 km/hr.¹¹⁸ This impact subjects the cervical spine to a force of 4.5 G, which may result more in a compression-tension force than in a hyperextension-hyperflexion force.¹¹⁸ The result is a

mild cervical spine injury. Can such a low impact initiate the postulated series of events previously described to injure the TMJ? Were the impact forces known in these studies?

3. Are the TMJ injuries recorded immediately post whiplash by magnetic resonance (MR) imaging? The majority of studies on whiplash and TMJ injuries do not record the interval between the date of the accident and time the MR imaging was done. In a recent article on this topic,¹⁴² the time between the accident and the MR imaging ranged from a few days to more than 1 year. Only 15% of the 87 patients in this article received imaging within 1 to 30 days.¹⁴²

The conclusion that "MR imaging clearly demonstrates the relationship between post MVA cervical whiplash and TMJ injuries"¹⁴² is not warranted if these questions are not addressed. This is not to say that a higher incidence of disk displacements is seen in patients with postcervical whiplash. The mechanism postulated simply may be different.

It would be interesting to know how many whiplash patients brux. If bruxism (neuromuscular factors) is still one of several heterogeneous etiologic factors for disk displacements, could bruxism in these whiplash patients be a response to pain originating from the patient's neck? I will not go as far as to state that the cervical spine causes a disk displacement via bruxism. I am, however, suggesting that a symptomatic cervical spine can cause masticatory muscle hyperactivity that may be expressed as bruxism.

Summary Physical therapy diurnal management of masticatory muscle hyperactivity comprises oral modification/awareness exercises, modalities, massage, and mandibular and neuromuscular re-educational exercises. Physical therapy management of the symptomatic cervical spine is helpful in diurnal and nocturnal bruxism. Supporting the cervical spine with an appropriate cervical pillow either supine or side-lying also helps in the management of nocturnal bruxism. A number of patients have been observed to have a decrease in symptoms related to bruxism when no lifestyle or treatment changes occurred except that treatments were offered to the cervical spine.

The following section discusses the mechanism by which the cervical spine can be a source of two common cephalic symptoms: headache and dizziness.

Cervical Spine as a Source of Cephalic Symptoms

It is evident that serious disease or pathology can cause cephalic symptoms that require a thorough neurologic and often otolaryngologic examination. It is just as evident that the cervical spine has not been recognized as it should as a source of non-diseased/pathologic cephalic symptoms. In order for the clinician to appreciate the cervical spine as a common source of cephalic symptoms, the neuroanatomic characteristics are now discussed.

The following is the 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.¹¹⁰

The spinal nucleus of the trigeminal nerve consists of three parts: pars oralis, par interparalis, and pars caudalis. The pars caudalis extends caudally 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 possibly 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 that of the upper cervical nerves.

From the preceding description, Bogduk summarizes, "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."¹¹⁰ Bogduk states that this region of gray matter can legitimately be viewed as a single or combined nucleus, for which he prefers to use the term *trigeminocervical nucleus*.¹¹⁰ Trigeminocervical nucleus incorporates the essential central nervous structures responsible for the transmission of pain. The trigeminocervical nucleus receives afferents from the trigeminal and up-

per cervical nerves. *This convergence of trigeminal and cervical afferents in the trigeminocervical nucleus is viewed by Bogduk as the nociceptive nucleus for the entire head and upper neck.*¹¹⁰ Essentially, nociceptive information from the 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.^{110, 116, 144-147} Numerous human studies have clearly demonstrated the existence of the trigeminocervical nucleus when experimental stimuli to the upper neck tissues produced referred pain in the head.^{148, 149}

Common cervical spine tissues that can be a source of nociceptive information to the trigeminocervical nucleus would include those tissues innervated by cervical nerves C1, C2, C3, and C4. The more common tissues are the longus capitis and cervicis, the rectus capitis anterior and lateralis, and the posterior neck muscles: semispinalis capitis, longissimus capitis and splenius capitis, multifidus, sternocleidomastoid, and trapezius.¹⁵⁰ Facet joints innervated by C1, C2, C3, and C4 include the occipitoatlantal, C1-2, C2-3, and C3-4 facet joints.

In addition to the neuroanatomic connection explaining cephalic symptoms of cervical origin, there also is a peripheral nerve entrapment origin for cephalic symptoms. The greater occipital nerve (GON) is the main sensory nerve in the occipital area, deriving most of its fibers from the C2 nerve root.¹⁵⁰ Involvement of the C2 nerve root or greater occipital nerve has been collectively referred to as *occipital neuralgia*.¹⁵¹ GON compression or irritation has been attributed to various causes, ranging from post-traumatic lesions and cervical degenerative arthrosis to muscle spasm in the upper cervical spine.¹⁵²⁻¹⁵⁴

Symptoms associated with GON entrapment would be located in the area innervated by cutaneous branches of the GON. Such complaints may be located in the occipital area, at the top of the skull, and/or around/in the ear or TMJ.¹⁵⁰

HEADACHE

John Edmeads¹⁵⁵ points out that in order for the cervical spine to be a source of headaches, the following three conditions must be present:

1. There should be pain-sensitive structures within the neck.
2. There should be identifiable pathologic processes or physiologic dysfunction 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 from previous discussion that all of Edmeads' criteria are present for the cervical spine to be a source of headaches.

The term used to specify the cervical spine as the source of headaches is *cervicogenic*. It was first used by Sjaastad and associates in 1983.¹⁵⁶ *Cervicogenic* only indicates the region; it does not indicate the structure primarily affected.¹⁵⁶ Since 1983, numerous articles have appeared in the literature providing documentation of cervicogenic headache.¹⁵⁷⁻¹⁶² Only recently have relatively clear criteria for the diagnosis of cervicogenic headache been published.¹⁶² The following are the primary criteria used to diagnose cervicogenic headache:¹⁶²⁻¹⁶⁴

1. Pain localized to neck and occipital region, which may project to the forehead, orbital region, temples, vertex, or ears
2. Pain precipitated or aggravated by special neck movements or sustained neck posture
3. At least one of the following:
 - a. Resistance to or limitation of passive neck movements
 - b. Changes in neck muscle contour, texture, tone, or response to active or passive stretching and contraction
 - c. Abnormal tenderness of neck muscles

The examination by the dentist to diagnose nondiseased cervical spine involvement is covered later. Not included in the dental cervical spine examination are 3.a and 3.b, for practical reasons (patient positioning and manual skills of the clinician).

Cervical spine involvement needs to be considered even when other forms of headache have been implicated. Headaches that may be mistaken for cervicogenic headaches

are the classic or common migraine, tension-type headache, and post-traumatic headache. Evidence suggests that cervicogenic headache is more common than migraine.¹⁶⁴ There is no doubt that diagnostic categories overlap because most headaches are diagnosed by location of symptoms.¹⁶⁵ The term *cervicogenic headache* is not suggested to supersede a more popular headache label (i.e., common migraine or tension-type headache). However, at the very least, cervical spine involvement should be considered as a primary feature or epiphenomenon.

DIZZINESS

Like headache, dizziness is a common complaint of patients that can originate in the cervical spine. Dizziness "is a general term, implying only the sense of a disturbed relationship to the space outside oneself."¹⁶⁶ *Vertigo* is the illusion of motion or position, either of the patient or of the patient's environment, and the term has been used more specifically to connote rotation.¹⁶⁷ Both *dizziness* and *vertigo* refer to a false sensation of motion of the body and in the discussion that follows are considered synonymous.¹⁶⁸ Dizziness often is described by the patient as unsteadiness, imbalance, floating, light-headedness, and spinning.¹⁶⁶⁻¹⁶⁸ Dizziness results from discrepancy or conflict in positional information from the cerebellum, vestibular nuclei, ears, eyes, proprioceptors, and other peripheral receptors.^{110, 168} Troost^{169, 170} identifies the cause of dizziness as either a peripheral (benign paroxysmal positional vertigo) or central disorder (ischemic or reflex vertigo). Pertinent to this discussion is reflex vertigo that contributes to dizziness.

Reflex cervical vertigo originates from neck proprioceptors whose input affects vestibular nucleus activity, resulting in dizziness.^{110, 171} 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."¹¹⁰ Neck proprioception origi-

nates from muscle spindles and mechanoreceptor activity in the cervical spine.¹⁷²

Though orientation of the head in space is the special role of the vestibular apparatus, there is, as Cohen states,¹⁷³ “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 be achieved only by neck proprioception. To have only the vestibular system indicating a position of the head in space, without information from the neck proprioceptors indicating the relationship of the head on the neck, would prove to be insufficient to function in the upright position.¹⁷³

Vestibular catastrophes, such as vertigo and nystagmus, can be caused solely by abnormalities related to neck proprioceptors.¹⁷³ Various studies show that damage to deep cervical tissues, such as facet joints and neck muscles, produces a generalized ataxia, with symptoms of imbalance, disorientation, and motor incoordination.^{174–176} Vertigo, ataxia, and nystagmus were induced in animals and humans by injecting local anesthetic into the neck.¹⁷⁷ The injections presumably interrupted the flow of afferent information from neck muscles and joint receptors. Hinoki,¹⁷⁸ through his experiments and clinical studies, suggests that vertigo after 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, tinnitus, and diminished hearing, could be related to cervical spine involvement.¹⁷¹

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 neck and cephalic symptoms.

Dental Examination for Cervical Spine Disorder

Spinal research has largely focused on the lumbar spine. For this discussion, conclusions drawn about the lumbar spine are considered applicable to the cervical spine.

A clinician faces dilemmas in diagnosing and treating the lumbar and cervical spine. In many cases, it may be difficult or impossible to establish the correct diagnosis for a patient with spinal complaints.¹⁷⁹ An expert panel has estimated that a precise diagnosis probably cannot be determined for up to 80% to 90% of patients with spinal pain, depending in part on how we regard radiographic degenerative changes.¹¹⁷ Clinical practice, despite the major advances in knowledge and technology, is largely an art.¹⁷⁹ What is known from clinical research is that the majority of patients seek help for *nonspecific* acute or chronic spinal complaints.¹¹⁷ *Nonspecific* implies that no underlying disease can be established.¹¹⁷

Inspired in part by the Quebec Task Force publication, the medical and physical therapy professions have pursued research and clinical studies classifying patients having a nonspecific spinal involvement according to signs and symptoms (versus a radiologic diagnosis or physiopathologic hypothesis).^{180–185} Classifying/diagnosing patients according to signs and symptoms should provide a direct treatment plan and a better understanding of treatment outcomes.^{184–186} Physical therapists can also diagnose/classify patients according to signs and symptoms that identify the condition. A diagnosis based on symptoms and functional limitations can be the focus of the physical therapist's treatment and reassessment of the patient's condition.²¹ Diagnosis by a physical therapist has been defined by Sahrman:²⁰

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.

Jette states, “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.”²¹ A physical therapy diagnosis should not be used to reflect ownership of the condition.²¹

This author proposes the following classi-

fication and its subcategories for nonspecific complaints of the cervical spine. A similar list of categories has been published by the Quebec Task Force on Whiplash-Associated Disorders.¹¹⁸

Classification

Movement Dysfunction of the Cervical Spine

Categories

1. Neck symptom(s) (central) without radiation
2. Neck symptom(s) with radiation into proximal upper extremity
 L R
 Radiation related to radiculopathy
 Y N
3. Neck symptom(s) with radiation into distal upper extremity
 L R
 Radiation related to radiculopathy
 Y N
 Neurologic signs Y N
4. Neck symptom(s) with cephalic radiation L R

Utilizing the categories should foster understanding and reduce confusion between the dental/medical professions and the physical therapy profession when discussing what the patient's problem is. Future examination methods that prove reliable and valid will specify the tissue(s) causing the patient's symptoms and functional limitations. Once the predictive value of tests is established, the preceding categories may have their own subcategories that name a specific tissue(s) that has undergone physiopathologic changes. An example is the following:

Classification

Movement Dysfunction of the Cervical Spine

Category

Neck symptom(s) with radiation cephalic to the left

Subcategories

C2-3 left facet joint dysfunction
 Left occipital neuralgia
 Muscle hyperactivity of the left suboccipital, levator scapulae, and trapezius muscles

Dentists can diagnose a nondiseased cervical spine condition on the basis of symptom locations. The following section indicates the minimum data a dentist should obtain through the history and physical examination for determining whether the patient has movement dysfunction of the cervical spine. The dentist may elect not to perform the examination and refer the patient to a physical therapist.

HISTORY

During the history interview the dentist should be alert to "red flags" suggesting a medical referral is indicated. Examples are a previous cancer history, trauma sufficient to cause a fracture, unexplained weight loss, intravenous drug abuse, and diplopia, dysarthria, dysphagia, and drop attacks with or without dizziness. The history interview also should include *mode of onset* (traumatic, insidious, iatrogenic), *progression of pain behavior* (pain better, worse, or unchanged), *past treatment and response to past treatment*, and *aggravating factors* (positions or movements of the cervical spine that increase or decrease the symptoms).

The following are symptoms that can result from cervical spine involvement. The list has been published by the Quebec Task Force on Whiplash-Associated Disorders.¹¹⁸ These questions pertain to patients involved in a whiplash. However, I considered these same questions to be appropriate for those experiencing trauma from a nonwhiplash injury, as well as symptoms that have an insidious onset. I have placed the questions into three groups.

Do you have any of the following symptoms?

Group One

Neck/shoulder pain
 Reduced/painful neck movements

Headaches
 Numbness, tingling, or pain in arm or hand
 Dizziness/unsteadiness
 Nausea/vomiting

Group Two

Difficulty in swallowing
 Ringing in the ears
 Memory problems
 Problems in concentrating
 Vision problems
 Reduced/painful jaw movement

Group Three

Numbness, tingling, or pain in leg or foot
 Lower back pain

Group one contains the key questions asked in order to diagnose “movement dysfunction of the cervical spine.” These same questions allow the dentist to classify the patient into categories 1 to 4. Recording the patient’s response to *group two* symptoms helps the clinician appreciate the role of the cervical spine as a source of symptoms normally thought to originate elsewhere. Medical exclusion of disease/pathosis as a source of *group two* symptoms makes the cervical spine a primary suspect. Patients experiencing *group two* symptoms should not be given false hope that the symptoms will diminish once the cervical spine condition is treated. Instead, a wait and see attitude should be applied. *Group three* symptoms are not as important for the dentist to ask about as they are for the physical therapist. They are listed here for completeness.

Patients can be asked to fill out a pain drawing (Fig. 10–1). The common pain drawing is used to describe presenting symptoms and can also be scored for research purposes according to the method of Margolis and associates.¹⁸⁷

PHYSICAL EXAMINATION

The physical examination as illustrated in Figure 10–2 has been published by the Quebec Task Force on Whiplash-Associated Disorders.¹¹⁸ I have modified several areas of the physical examination for use by the dentist. This examination is not intended to

give the dentist a clear understanding of the absence or presence of radiculopathy, i.e., nerve root irritation/compression. Radiculopathy is included for completeness. The reader should refer to the reference for further understanding of radiculopathy.¹¹⁹

Completion of the history, pain drawing, and physical examination should provide the dentist with enough information to implicate the cervical spine as a source of the patient’s head and neck symptoms, indicating a referral to a physical therapist. The following is a general overview of what the dentist should expect from the physical therapist in the management of movement dysfunction of the cervical spine.

Physical Therapy Management for Movement Dysfunction of the Cervical Spine

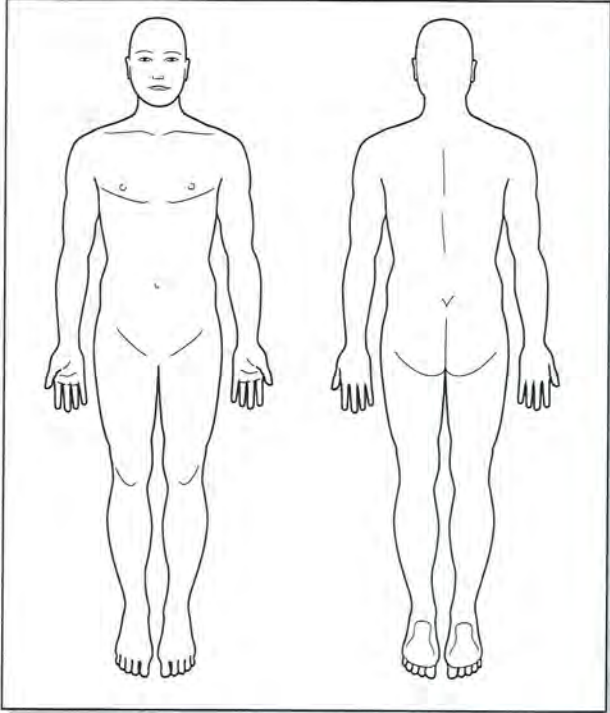
The following is a general overview of physical therapy management for movement dysfunction of the cervical spine. The reader is referred to the reference section for additional information on this topic.^{188–195} As discussed in treatment of TMD, physical therapists seldom offer a single treatment option for a patient who has myriad tissue and functional involvements of the cervical spine. Treatment options considered by the physical therapist in the management of movement dysfunction of the cervical spine are the following:

- Patient education
- Sleeping postures
- Upright postures
- Exercise
- Cervical traction
- Cervical collar
- Manual therapy

The physical therapist’s knowledge of anatomy, physiology, neurophysiology, and mechanics assists in the application of these treatment options. Cervical traction and cervical collar treatment are the only two options that are not offered to every patient. *Every* patient is educated about the condition along with precipitating and perpetuating factors that can aggravate it. *Every* patient is instructed on proper sleeping postures either supine or sidelying and if necessary the use of a cervical support. *Ev-*

Name: _____

PAIN DRAWING
Carefully shade or mark in the areas where you feel any pain on the drawing below.



FORM COMPLETED BY:
 Patient
 Clinician
 Other, specify _____

FIGURE 10-1. Pain drawing.

ery patient is shown exercises to improve flexibility, strength, and endurance. *Every* patient receives manual therapy. Physical therapists have knowledge and skills in all treatment options listed. An area of special interest is the manual skills of the physical therapist.

Manual skills are incorporated into the examination and consist of palpation and passive mobility testing. Treatment via manual therapy may consist of hands-on, repetitive oscillations; steady stretch or high-velocity thrusts of joints; and application of various forms of soft tissue massage, muscle stretching, or shortening.¹⁹⁰ The potential therapeutic value of manual therapy revolves around its mechanical, neurologic, neurophysiologic, and psychological effects.¹⁹⁰⁻¹⁹⁵ A comprehensive understanding of clinical viewpoints and scientific research

on manual therapy can be obtained from the reference section.¹⁹⁰⁻¹⁹⁹

Chapter Summary

Guidelines for the management of TMD can be misleading, especially if the treatment under study is misunderstood. Research on the therapeutic value of physical therapy treatments must incorporate the role of the physical therapist in performing the treatments. This basic concept can help in future clinical and research studies that investigate the therapeutic value of physical therapy for TMDs.

Physical therapists play an important role in the conservative and noninvasive

Dental Examination of the Cervical Spine

Examination indicated but deferred to: ___ PT and / or ___ MD

Date of Examination: Day ___ Month ___ Year ___

A. PAIN / LIMITATION

	No	Pain	Limitation
Flexion	___	___	___
Extension	___	___	___
Right rotation	___	___	___
Left rotation	___	___	___
Right lateral flexion	___	___	___
Left lateral flexion	___	___	___

B. PALPATORY TENDERNESS

	___ Yes	___ No		
If yes:	Left	Midline	Right	
Cervical spine	___	___	___	
Thoracic spine	___	___	___	
Other, specify	_____			

C. NEUROLOGIC EXAMINATION

___ Normal or ...	Sensory deficit		Motor weakness		Decreased deep tendon reflexes	
	Right	Left	Right	Left	Right	Left
C5	___	___	___	___	___	___
C6	___	___	___	___	___	___
C7	___	___	___	___	___	___
C8	___	___	___	___	___	___
Other, specify	_____		_____		_____	

D. DIAGNOSTIC TESTS

Plain radiographs
(Medical report available)
___ Yes ___ No

If yes:
___ Normal

___ Degenerative changes
Specify levels _____

Other specialized tests, specify:

F. MANAGEMENT PLAN

- Reassurance
___ Yes

- Activation
___ Return to usual activities ASAP
___ Delayed return to recreational activities

- Treatments
___ Medications, specify: _____
___ Home care, specify: _____
___ Referral to Physical Therapy: _____
___ Referral to a medical specialist: _____

E. DIAGNOSIS

CLASSIFICATION: Movement dysfunction of the cervical spine ___ Yes ___ No

If yes, patient categorized as:

- ___ 1. Neck symptom(s) (central) without radiation
___ 2. Neck symptom(s) with radiation into proximal upper extremity ___ L ___ R; Radiation related to radiculopathy ___ Y ___ N
___ 3. Neck symptom(s) with radiation into distal upper extremity ___ L ___ R; Radiation related to radiculopathy ___ Y ___ N
___ 4. Neck symptom(s) with radiation cephalic ___ L ___ R

REMARKS: _____

FIGURE 10-2. Form for dental examination of the cervical spine.

treatment of TMD: TMD—A, TMD—A/M, and TMD—M.

Subcategories of TMD—A that are diagnosed and treated by a physical therapist are inflammation, hypermobility, and hypomobility. Modalities for the treatment of inflammation have been covered, as well as the management of hypermobility. Hypomobility secondary to capsular tightness or a disk displacement without reduction can be managed by therapeutic procedures offered by a physical therapist. Physical therapy in the treatment of a disk displacement without reduction is gaining wider acceptance. Through the application of modalities, therapeutic procedures, and a home exercise program, a physical therapist can restore mobility of the mandible to a pain-free state with the disk remaining out of place. In conditions that do not respond to nonsurgical treatment for TMD, TMJ surgery and/or orthognathic surgery may be necessary. Postoperative physical therapy is important in pain control, promotion of healing, and restoration of functional mandibular movements.

Physical therapy management of TMD—M/bruxism focuses on oral modification, modalities, massage, and therapeutic and neuromuscular re-educational exercises. Decreasing symptoms and improving functional limitations associated with cervical spine involvement may also help reduce masticatory muscle hyperactivity. Theories that bruxism results from aberrant tonic neck reflex activity and cervical somatic pain have been discussed.

It is important to view TMD as only a component of head and neck pain management. Dentists cannot overlook the cervical spine as a major source of nondisease cephalic symptoms. Headaches, dizziness, as well as symptoms in the area of the eye, ear, and TMJ can originate in the cervical spine. Considering the number of muscles and facet joints of the cervical spine that can be a source of cephalic symptoms, the dentist alone cannot assess and treat referred pain from cervical musculoskeletal structures. A number of patients may unnecessarily continue to suffer from pain and functional limitations because the cervical spine was overlooked or treatments to the cervical spine were given by a clinician who did not have clinical skills or knowledge of a physical therapist.

The evaluation of the cervical spine that can be done by the dentist has been reviewed. The diagnosis of a nondiseased cervical spine is *movement dysfunction of the cervical spine*. Essentially, the diagnosis is made by the *exclusion* of disease/pathosis. The cervical spine evaluation primarily provides the clinician with functional information and a baseline to determine the effectiveness of treatment. A referral to a physical therapist is in order should the evaluation confirm the diagnosis of movement dysfunction of the cervical spine.

Physical therapists have various treatment options for managing the cervical spine. Manual therapy is a key treatment option. Should the physical therapist require assistance in managing nondiseased cervical spine-related complaints, he or she can consult a dentist and/or physician. Cervical pain secondary to inflammation of cervical dura, nerve roots, facet joints, or muscles may be relieved or reduced by localized injection by a medical doctor.

Patients experience multiple-tissue involvement that contributes to varying levels of symptoms and functional limitations of the head and neck. Whether or not the TMJ, muscles of mastication and occlusion, and cervical spine are interrelated or simply coexist remains to be seen. What is essential to head and neck pain management is a team approach consisting of a physical therapist, dentist, and physician. Physical therapists are key players in the conservative and noninvasive management of nondisease head and neck pains stemming from the TMJ, muscles of mastication, and cervical spine.

REFERENCES

1. Rothstein J, Delitto A, Scalzitti D: Understanding AHCPR Clinical Practice Guideline No. 14: Acute low back problems in adults. Prepared for the American Physical Therapy Association. *J Am Phys Ther Assoc*, Sept 1995, p 1.
2. Connolly J: AHCPR Guideline No. 14: Acute Low Back Problems in Adults: A commentary. *Phys Ther Magazine* 1995;3(9):89.
3. Bigos S, Bowyer O, Braen G, et al: Acute Low Back Problems in Adults. Clinical Practice Guideline No. 14. AHCPR publication 95 - 0642. Rockville, MD: Agency for Health Care Policy and Research, Public Health Service, US Department of Health and Human Services, 1994.
4. Tygiel PP: A critical analysis of the AHCPR acute low back pain guidelines. *Orthop Pract* 1995;7:3.
5. Ellis J: Concern still exists over guidelines for

- treatment of acute low back pain. *Phys Ther Bull* July 21, 1995, p 10.
6. Woolf SH: Practice guidelines: A new reality in medicine. Part III. Impact on patient care. *Arch Intern Med* 1993;150:2464.
 7. Judith Albino, Chairperson: National Institutes of Health Technology Assessment Conference on Management of Temporomandibular Disorders. April 29–May 1, 1996, Bethesda, MD.
 8. Feine J, Widmer C, Lund J: Physical Therapy: A Critique. National Institutes of Health Technology Assessment Conference on Management of Temporomandibular Disorders. April 29–May 1, 1996, Bethesda, MD, p 75.
 9. McNeil C (ed): Temporomandibular Disorders, Guidelines for Classification, Assessment and Management, ed 2. Lombard, IL, Quintessence, 1993.
 10. Letter of communication from Marilyn M. Moffat, President of the APTA to William H. Hall, Director of Communications, National Institutes of Health. May 29, 1996. Letter on file with the American Physical Therapy Association.
 11. A guide to physical therapist practice. Volume I: A description of patient management. *Phys Ther* 1995;75:707.
 12. APTA House of Delegates Policies HOD #06-93-22-43, June 1996, APTA, Alexandria VA.
 13. Okeson Jeffrey P: Current terminology and diagnostic classification schema. In: National Institutes of Health Technology Assessment Conference on Management of Temporomandibular Disorders. April 29–May 1, 1996, Bethesda, MD, p 21.
 14. De Wijer A, Rob J, de Leeuw J, et al: Temporomandibular and cervical spine disorders: Self-reported signs and symptoms. *Spine* 1996;21:1638.
 15. De Wijer A. Temporomandibular and cervical spine disorders. Dissertation, Utrecht University, The Netherlands, 1995. Pub Elinkwijk BV, Utrecht, The Netherlands.
 16. McNamara JA Jr, Seligman DA, Okeson JP: The relationship of occlusal factors and orthodontic treatment to temporomandibular disorders. In Sessle BJ, Bryant PS, Dionne RA (eds): Temporomandibular Disorders and Related Pain Conditions. Seattle, IASP Press, 1995; pp 399–427.
 17. Seligman DA, Pullinger AG: The role of intercuspal occlusal relationships in temporomandibular disorders: A review. *J Craniomandibul Disord Facial Oral Pain* 1991;5:96.
 18. Seligman DA, Pullinger AG: The role of functional occlusal relationships in temporomandibular disorders: A review. *J Craniomandibul Disord Facial Oral Pain* 1991;5:265.
 19. Okeson JP (ed): Orofacial Pain: Guidelines for Assessment, Diagnosis, and Management. Chicago: Quintessence, 1996.
 20. Sahrman S: Diagnosis by the physical therapist: A prerequisite for treatment. A special communication. *Phys Ther* 1988;68:703.
 21. Jette A: Diagnosis and classification by physical therapists: A special communication. *Phys Ther* 1989;69:967.
 22. Delitto A, Guccione A, Jette A, et al: Diagnosis in physical therapy. *PT Magazine*, June 1993, pp 58–65.
 23. Dekker J, van Baar M, Curfs E, et al: Diagnosis and treatment in physical therapy: An investigation of their relationship. *Phys Ther* 1993;73:568.
 24. Delitto A, Snyder-Mackler L: The diagnostic process: Examples in orthopedic physical therapy practice. *Phys Ther* 1995;75:203.
 25. Fitzgerald G, McClure P, Beattie P: Issues in determining treatment effectiveness of manual therapy. *Phys Ther* 1994;74:227.
 26. Kraus SL: Physical therapy management of TMD. In Kraus SL (ed): Clinics in Physical Therapy, Temporomandibular Disorders, ed 2. New York, Churchill Livingstone, 1994, p 161.
 27. Brian R, Zarro V: Inflammation and repair and the use of thermal agents. In Michlovitz S (ed): Thermal Agents in Rehabilitation: Contemporary Perspectives in Rehabilitation, Philadelphia, FA Davis, 1990, p 1.
 28. Benoit PW: History and physical examination for TMD. In Kraus SL (ed): Clinics in Physical Therapy, Temporomandibular Disorders, ed 2. New York, Churchill Livingstone, 1994, p 71.
 29. Kraus SL: Evaluation and management of temporomandibular disorders. In Evaluation, Treatment and Prevention of Musculoskeletal Disorders. Minneapolis, The Saunders Group, 1993.
 30. Michlovitz S (ed): Thermal Agents in Rehabilitation: Contemporary Perspectives in Rehabilitation, ed 2. Philadelphia, FA Davis, 1990.
 31. Mannheimer JS: Physical therapy concepts in evaluation and treatment of the upper quarter: Therapeutic modalities. In Kraus SL (ed): TMJ Disorders: Management of the Craniomandibular Complex. New York, Churchill Livingstone, 1988, p 311.
 32. Mannheimer J: Postoperative physical therapy protocols. In Kraus SL (ed): Clinics in Physical Therapy: Temporomandibular Disorders, ed 2. New York, Churchill Livingstone, 1994, p 277.
 33. Ziskin MC, McDiarmid T, Michlovitz SL: Therapeutic ultrasound. In Michlovitz S (ed): Thermal Agents in Rehabilitation: Contemporary Perspectives in Rehabilitation, Philadelphia, FA Davis, 1990, p 134.
 34. Stratton SA, Heckmann R, Francis RS: Therapeutic ultrasound: Its effect on the integrity of a nonpenetrating wound. *J Orthop Sports Phys Ther* 1984;5:278.
 35. Munting E: Ultrasonic therapy for painful shoulders. *Physiotherapy* 1978;64:180.
 36. Bierman W: Ultrasound in the treatment of scars. *Arch Phys Med Rehabil* 1954;35:209.
 37. Michlovitz S: Cryotherapy: The use of cold as a therapeutic agent. In Michlovitz S (ed): Thermal Agents in Rehabilitation: Contemporary Perspectives in Rehabilitation. Philadelphia, FA Davis, 1990, p 63.
 38. Travell J, Simons D: Myofascial Pain and Dysfunction: The Trigger Point Manual. New York, Williams & Wilkins, 1983.
 39. Michlovitz S: Cryotherapy: Biophysical principles of heating and superficial heat agents. In Michlovitz S (ed): Thermal Agents in Rehabilitation: Contemporary Perspectives in Rehabilitation. Philadelphia, FA Davis, 1990, p 88.
 40. Glass J, Stephen R, Jacobson C: The quality and distribution of radiolabeled dexamethasone delivered to tissue by iontophoresis. *Int J Dermatol* 1980;19:519.
 41. Harris P: Iontophoresis: Clinical research in musculoskeletal inflammatory conditions. *J Orthop Sports Phys Ther* 1982;4:109.
 42. Bertolucci L: Introduction of antiinflammatory drugs by iontophoresis: Double blind study. *J Orthop Sports Phys Ther* 1982;4:103.

43. Braun BL: Treatment of an acute anterior disk displacement in the temporomandibular joint. *Phys Ther* 1987;67:1234.
44. Lark M, Gangarosa C Sr: Iontophoresis: An effective modality for the treatment of inflammatory disorders of the temporomandibular joint and myofascial pain. *J Craniomandibul Pract* 1990;8:108.
45. Reid K, Dionne R, Sicard-Rosenbaum L, et al: Evaluation of iontophoresis applied dexamethasone for painful pathologic temporomandibular joints. *Oral Surg Oral Med Oral Pathol* 1994;77:605.
46. Schiffman E, Braun B, Lindgren J, et al: Temporomandibular joint iontophoresis: A double-blind randomized clinical trial. *J Orofac Pain* 1996;10:157.
47. Mannheimer JS, Lampe GN: *Clinical Transcutaneous Electrical Nerve Stimulation*. Philadelphia, FA Davis, 1984.
48. Keith DA: *Surgery of the Temporomandibular Joint*. Boston, Blackwell Scientific Publications, 1988, pp 139-141.
49. Buckingham RB, Braun T, Harenstein DA, et al: Temporomandibular joint dysfunction syndrome: A close association with systemic joint laxity (the hypermobile joint syndrome). *Oral Surg Oral Med Oral Pathol* 1991;72:514.
50. Westling L, Mattiasson A: General joint hypermobility and temporomandibular joint derangement in adolescents. *Ann Rheum Dis* 1992;51:87.
51. Dijkstra PU, Lambert GM de Bont, Stegenga B, et al: Temporomandibular joint osteoarthritis and generalized joint hypermobility. *J Craniomandibul Pract* 1992;10:221.
52. Akeson WH: An experimental study of joint stiffness. *J Bone Joint Surg* 1961;43A:1022.
53. Akeson WH, Amiel D, LaViolette D, et al: The connective tissue response to immobility: An accelerated ageing response. *Exp Gerontol* 1968;3:289.
54. Akeson WH, Amiel D, Woo S: Immobility effects of synovial joints: the pathomechanics of joint contracture. *Biorheology* 1980;17:95.
55. Salter RB, Simmonds DI, Malcolm BW, et al: The biological effect of continuous passive motion on the healing of full-thickness defects in articular cartilage. *J Bone Joint Surg* 1980;62A:1232.
56. Salter RB, Hamilton WH, Wedge JH, et al: Clinical applications of basic research on continuous passive motion for disorders and injuries of synovial joints, a preliminary report of a feasibility study. *J Orthop Res* 1983;3:325.
57. Toller P: Non-surgical treatment of the temporomandibular joint. *Oral Sci Rev* 1976;7:70.
58. Rasmussen OC: Description of population and progress of symptoms in a longitudinal study of temporomandibular arthropathy. *Scand J Dent Res* 1981;89:196.
59. Clark GT, Mulligan RA: A review of the prevalence of temporomandibular dysfunction. *J Gerodontology* 1986;3:231.
60. Greene CS, Turner C, Laskin DM: Long-term outcome of TMJ clicking in 100 MPD patients. *J Dent Res* 1982;61:218.
61. Turell J, Ruiz HG: Normal and abnormal findings in temporomandibular joints in autopsy specimens. *J Craniomandibul Disord Facial Oral Pain* 1987;1:257.
62. Hellsing G, Holmlund A: Development of anterior disk displacement in the temporomandibular joint: An autopsy study. *J Prosthet Dent* 1985;53:397.
63. Westesson PL, Eriksson L, Kurita K: Reliability of a negative clinical temporomandibular joint examination: Prevalence of disk displacement in asymptomatic temporomandibular joints. *Oral Surg Oral Med Oral Pathol* 1989;68:551.
64. Ribeiro RF, Tallents RH, Katzberg RW, et al: The prevalence of disc displacement in symptomatic and asymptomatic volunteers aged 6 to 25 years. *J Orofac Pain* 1987;11:37.
65. Zamburlini I, Austin D: Long-term results of appliance therapies in anterior disk displacement with reduction: A review of the literature. *J Craniomandibul Pract* 1991;9:361.
66. Orenstein ES: Anterior repositioning appliances when used for anterior disk displacement with reduction—a critical review. *J Craniomandibul Pract* 1993;11:141.
67. Vichaichalmvong S, Nilner M, Panmekiate S, et al: Clinical follow-up of patients with different disc positions. *J Orofac Pain* 1993;7:61.
68. Moloney F, Howard JA: Internal derangements of the temporomandibular joint: Anterior repositioning splint therapy. *Aust Dent J* 1986;31:30.
69. de Leeuw R, Boering G, Stegenga B, et al: Clinical signs of TMJ osteoarthritis and internal derangement 30 years after nonsurgical treatment. *J Orofac Pain* 1994;8:18.
70. Braun BL: The effect of physical therapy intervention on incisional opening after temporomandibular joint surgery. *Oral Surg Oral Med Oral Pathol* 1987;64:544.
71. Lehmann J, Masock A, Warren C, et al: Effect of therapeutic temperature on tendon extensibility. *Arch Phys Med Rehabil* 1970;51:481.
72. Warren C, Lehman J, Koblanski J: Heat and stretch procedures: An evaluation using rat tail tendon. *Arch Phys Med Rehabil* 1976;57:122.
73. Lentell G, Hetherington T, Eagan J, et al: The use of thermal agents to influence the effectiveness of a low-load prolonged stretch. *J Orthop Sports Phys Ther* 1992;16:200.
74. Warren C, Lehmann J, Koblanski J: Elongation of rat tail tendon: Effect of load and temperature. *Arch Phys Med Rehabil* 1971;52:465.
75. McCarthy MR, Yates CK, Anderson MA, et al: The effects of immediate continuous passive motion on pain during the inflammatory phase of soft tissue healing following anterior cruciate reconstruction. *J Orthop Sports Phys Ther* 1993;17(2):96-101.
76. Salter RB: The biological concept of continuous passive motion of synovial joints: The first 18 years of basic research and its clinical application. *Clin Orthop* 1989;242:12.
77. Gossman MR, Sharmann SA, Rose SJ: Review of length-associated changes in muscle. *Phys Ther* 1979;62:1799.
78. McCarthy MR, O'Donoghue PC, Yates CK, et al: The clinical use of continuous passive motion in physical therapy. *J Orthop Sports Phys Ther* 1992;15:132.
79. McCarty WL, Darnell MW: Rehabilitation of the temporomandibular joint through the application of motion. *J Craniomandibul Pract* 1993;11:289.
80. Osborn JW: The disc of the human temporomandibular joint: Design, function and failure. *J Oral Rehabil* 1985;12:279.
81. Wright V, Dowson D, Longfield M: Joint stiffness - its characterization and significance. *Biomed Eng* 1969;4:8.
82. Randall T, Portney L, Harris BA: Effects of joint

- mobilization on joint stiffness and active motion of the metacarpal-phalangeal joint. *J Orthop Sports Phys Ther* 1992;16:30.
83. Stromberg D, Wiederhielm CA: Viscoelastic description of a collagenous tissue in simple elongation. *J Appl Physiol* 1969;26:857.
 84. LaBan MM: Collagen tissue: Implications of its response to stress in vitro. *Arch Phys Med Rehabil* 1962;43:461.
 85. Chung S-C, Kim H-S: The effects of the stabilization splint on the TMJ closed locked. *J Craniomandibul Pract* 1993;11:95.
 86. Farrar WB: Characteristics of the condylar path in internal derangements of the TMJ. *J Prosthet Dent* 1978;39:319.
 87. Murakami K-I, Segami N, Moriya Y, et al: Correlation between pain and dysfunction and intra-articular adhesions in patients with internal derangement of the temporomandibular joint. *J Oral Maxillofac Surg* 1992;50:705.
 88. Dolwick MF, Katzberg RW, Helms CA: Internal derangements of the temporomandibular joint: Fact or fiction? *J Prosthet Dent* 1983;49:415.
 89. Martini G, Martini M, Carano A: MRI study of a physiotherapeutic protocol in anterior disk displacement without reduction. *J Craniomandibul Pract* 1996;14:216.
 90. de Leeuw R, Boering G, Stegenga B, et al: Radiographic signs of temporomandibular joint osteoarthritis and internal derangement 30 years after nonsurgical treatment. *Oral Surg Oral Med Oral Pathol* 1995;79:382.
 91. Mongini F, Ibertis F, Manfredi A: Long-term results in patients with disc displacement without reduction treated conservatively. *J Craniomandibul Pract* 1996;14:301.
 92. Scapino RP: The posterior attachments: Its structure, function, and appearance in TMJ imaging studies, Part 1. *J Craniomandibul Disord Facial Oral Pain* 1991;5:83.
 93. Scapino RP: Histopathology of the disc and posterior attachment. In Disc Displacement, Internal Derangements of the TMJ. In Palacios E (ed): *Magnetic Resonance Imaging of the Temporomandibular Joint*. Stuttgart, Georg Thieme Verlag, 1990.
 94. Van Dyke AR, Goldman SM: Manual reduction of displaced disk. *J Craniomandibul Pract* 1990;8:350.
 95. Minagi S, Nozaki S, Sato T, et al: Manipulation techniques for treatment of anterior disk displacement without reduction. *J Prosthet Dent* 1991;65:686.
 96. Labbezoo F, Lavigne G: Do bruxism and temporomandibular disorders have a cause-and-effect relationship? *J Orofac Pain* 1997;11:15.
 97. Mao J, Stein R, Osborn J: Fatigue in human jaw muscles: A review. *J Orofac Pain* 1993;7:135.
 98. Russell JI (ed): Clinical overview and pathogenesis of the fibromyalgia syndrome, myofascial pain syndrome, and other pain syndromes. *J Musculoskeletal Pain* 1996;4.
 99. Lawrence ES, Razzoog SJ: Nonsurgical Management of Mandibular Disorders. In Kraus S (ed): *Clinics in Physical Therapy, Temporomandibular Disorders*, ed 2. New York, Churchill Livingstone, 1994, p 125.
 100. Schnurr RF, Rollman GB, Brooke RI: Are there psychologic predictors of treatment in temporomandibular joint pain and dysfunction? *Oral Surg Oral Med Oral Pathol* 1991;72:550.
 101. Bertolucci LE: Physical therapy post-arthroscopic TMJ management (update). *J Craniomandibul Pract* 1992;10:130.
 102. Smith AM: The coactivation of antagonist muscles. *Can J Physiol Pharmacol* 1981;59:733.
 103. Burke D: Critical examination of the case for or against fusimotor involvement in disorders of muscle tone. In Desmedt JE (ed): *Motor Control Mechanisms in Health and Disease*. New York, Raven Press, 1983, p 133.
 104. Tanigawa MC: Comparison of the hold-relax procedure and passive mobilization on increasing muscle length. *Phys Ther* 1972;52:725.
 105. DeAndrade J, Grant C, Dixon A: Joint distention and reflex muscle inhibition in the knee. *J Bone Joint Surg* 1965;47(A):313.
 106. Larsson L, Thilander B: Mandibular positioning, the effect of pressure on joint capsule. *Acta Neurol Scand* 1964;40:131.
 107. Hathaway KM: Behavioral and psychosocial management. In Pertes RA, Gross SG (eds): *Clinical Management of Temporomandibular Disorders and Orofacial Pain*. Chicago, Quintessence, 1995.
 108. Slater M, Brooke RI, Merskey GF, et al: Is the temporomandibular pain and dysfunction syndrome a disorder of the mind? *Pain* 1983;17:151.
 109. Robert Rosenbaum, Committee Chairman: The scope of TMD / orofacial pain (head and neck pain management) in contemporary dental practice. *J Orofac Pain* 1997;11:78.
 110. Bogduk N: Cervical causes of headache and dizziness. In Grieve G (ed): *Modern Manual Therapy*. New York, Churchill Livingstone, 1986, p 289.
 111. Everett N: *Functional Neuroanatomy*, ed 6. Philadelphia, Lea & Febiger, 1972.
 112. Kerr F: Mechanism, diagnosis and management of some cranial and facial pain syndromes. *Surg Clin North Am* 1963;43:951.
 113. Kerr F, Olafson R: Trigeminal and cervical volleys. *Arch Neurol* 1961;5:171.
 114. Kerr F: A mechanism to account for frontal headache in cases of posterior fossa tumors. *J Neurosurg* 1961;18:605.
 115. Kerr F: Structural relation of the trigeminal spinal tract to upper cervical roots and the solitary nucleus in the cat. *Exp Neurol* 1961;4:134.
 116. Allen ME (ed): Musculoskeletal pain emanating from the head and neck. *J Musculoskeletal Pain* 1996;4.
 117. Spitzer WO (ed): Scientific approach to the assessment and management of activity-related spinal disorders. A monograph for Clinicians Report of the Quebec Task Force on Spinal Disorders. *Spine* 1987;12(7).
 118. J. David Cassidy, Editorial Coordinator: Scientific monograph of the Quebec Task Force on Whiplash-Associated Disorders: Redefining "whiplash" and its management. *Spine* 1995;20(85).
 119. Lester JP, Windsor RE, Dreyer, SJ: Medical management of the cervical spine. In Kraus SL (ed): *Clinics in Physical Therapy: Temporomandibular Disorders*, ed 2. New York, Churchill Livingstone, 1994, p 413.
 120. Braun BL, DiGiovanna A, Schiffman E, et al: A cross-sectional study of temporomandibular joint dysfunction in post-cervical trauma patients. *J Craniomandibul Disord Facial Oral Pain* 1992;6:24.
 121. Clark GT: Examining temporomandibular disorder patients for craniocervical dysfunction. *J Craniomandibul Pract* 1983;2:55.

122. De Laat A, Meuleman H, Stevens A: Relation between functional limitations of the cervical spine and temporomandibular disorders (abstract). *J Orofac Pain* 1993;1:109.
123. Kirveskari P, Alanen P, Karskela V, et al: Association of functional state of stomatognathic system with mobility of cervical spine and neck muscle tenderness. *Acta Odontol Scand* 1988;46:281.
124. Padamsee M, Mehtan N, Forgione A, et al: Incidence of cervical disorders in a TMD population. (IADR; Abstract No. 680). *J Dent Res* 1994;73.
125. Isaccsson G, Linde C, Isberg A: Subjective symptoms in patients with temporomandibular joint disc displacement versus patients with myogenic craniomandibular disorders. *J Prosthet Dent* 1989;61:70.
126. Lobbezoo-Scholte AM: Diagnostic subgroups of craniomandibular disorders. PhD dissertation, Utrecht University, The Netherlands, 1993.
127. DeLeeuw JRJ: Psychosocial aspects and symptom characteristics of craniomandibular dysfunction. PhD dissertation, Utrecht University, The Netherlands, 1993.
128. Magnus R: Some results of studies in the physiology of posture. *Lancet* 1926;2:531.
129. Magnus R: *Korperstellung*, pp xiii, 740, Berlin, 1924, Julius Springer, *Animal Posture*, Croonian Lecture. *Proc R Soc B* 98(690):339-353.
130. Magnus R, DeKleijn A: Die Abhangigkeit des Tonus der Extremitätenmuskeln von der Kopfstellung. *Pflugers Arch* 1912;145:455.
131. McCouch G, Deering I, Ling T: Location of receptors for tonic neck reflexes. *J Neurophysiol* 1951;14:191.
132. Wilson VJ: The tonic neck reflex: Spinal circuitry. In Peterson B, Richmond F (eds): *Control of Head Movement*. New York, Oxford University Press, 1988.
133. Pompeiano O: The tonic neck reflex: Supraspinal control. In Peterson B, Richmond F (eds): *Control of Head Movement*. New York, Oxford University Press, 1988.
134. Funakoshi M, Amano N: Effects of the tonic neck reflex on the jaw muscles of the rat. *J Dent Res* 1973;52:668.
135. Sumino R, Nozaki S, Katoh M: Trigemino-neck reflex. In Kawamura Y, Dubner R (eds): *Oral-Facial Sensory and Motor Functions*. Tokyo, Quintessence Books, 1981, p 81.
136. Wyke BD: Neurology of the cervical spinal joints. *Physiotherapy* 1979;65:72.
137. Funakoshi M, Fujita N, Takehana S: Relations between occlusal interference and jaw muscle activities in response to changes in head position. *J Dent Res* 1976;55:684.
138. Bratzlavsky M, Vander Eecken H: Postural reflexes in cranial muscles in man. *Acta Neurol Belg* 1977;77:5.
139. Mohl N: Head posture and its role in occlusion. *NY State Dent J* 1976;42:17.
140. Forsberg CM, Helsing E, Linder-Aronson S, et al: EMG activity in neck and masticatory muscles in relation to extension and flexion of the head. *Eur J Orthod* 1985;7:177.
141. Aiello I, Rosati G, Sau GF, et al: Tonic neck reflexes on upper limb flexor tone in man. *Exp Neurol* 1988;101:41.
142. Garcia R, Arrington JA: The relationship between cervical whiplash and temporomandibular joint injuries: An MRI study. *J Craniomandibul Pract* 1996;14:233.
143. Kronn E: The incidence of TMJ dysfunction in patients who have suffered a cervical whiplash injury following a traffic accident. *J Orofac Pain* 1993;7:209.
144. Everett N: *Functional Neuroanatomy*, ed 6. Philadelphia, Lea & Febiger, 1972.
145. Kerr F: Mechanism, diagnosis and management of some cranial and facial pain syndromes. *Surg Clin North Am* 1963;43:951.
146. Kerr F, Olafson R: Trigeminal and cervical volleys. *Arch Neurol* 1961;5:171.
147. Kerr F: Structural relation of the trigeminal spinal tract to upper cervical roots and the solitary nucleus in the cat. *Exp Neurol* 1961;4:134.
148. Feistein B, Langton J, Jameson R, et al: Experiments on referred pain from deep somatic tissues. *J Bone Joint Surg* 1954;36A:981.
149. Bogduk N, Marsland A: C3 headaches. Paper presented at the International Meeting on Pain and Regional Anaesthesia. Australian Pain Society, Perth, February, 1983.
150. Bogduk N: The clinical anatomy of the cervical dorsal rami. *Spine* 1982;7:319.
151. Cox C, Cocks G: Occipital neuralgia. *J Med Assoc State Ala* 1979;48:23.
152. Saadah H, Taylor F: Sustained headache syndrome associated with tender occipital nerve zones. *Headache* 1987;27:201.
153. Jansen J, Markakis E, Rama B, et al: Hemispheric attacks or permanent hemispheric attacks—a sequel of upper cervical root compression. *Cephalalgia* 1989;9:123.
154. Magnusson T, Ragnarsson T, Bjornsson A: Occipital nerve release in patients with whiplash trauma and occipital neuralgia. *Headache* 1996;36:32.
155. Edmeads J: The cervical spine and headache. *Neurology* 1988;38:1874.
156. Sjaastad O, Saunte C, Hovdahl H, et al: "Cervicogenic" headache: An hypothesis. *Cephalalgia* 1983;3:249.
157. Sjaastad O, Fredriksen T, Stolt-Nielsen A: Cervicogenic headache, C2 rhizopathy, and occipital neuralgia: A connection? *Cephalalgia* 1986;6:189.
158. Pfaffenrath V, Dandekar R, Pollmann W: Cervicogenic headache - the clinical picture, radiological findings and hypotheses on its pathophysiology. *Headache* 1987;27:495.
159. Fredriksen T, Hovdahl H: "Cervicogenic headache": Clinical manifestation. *Cephalalgia* 1987;7:147.
160. Fredriksen T, Fougner R, Tangerud A, et al: Cervicogenic headache: Radiological investigations concerning head/neck. *Cephalalgia* 1989;9:139.
161. Jaeger B: Are "cervicogenic" headaches due to myofascial pain and cervical spine dysfunction? *Cephalalgia* 1989;9:157.
162. Classification and diagnostic criteria for headache disorders, cranial neuralgias and facial pain, ed 1. International Headache Society. *Cephalalgia* 1988;8(suppl 7):61.
163. Olesen J (ed): *Classification and Diagnostic Criteria for Headache Disorders, Cranial Neuralgias and Facial Pain*. Copenhagen: The International Headache Society, 1990.
164. Nilsson N: The prevalence of cervicogenic headache in a random population sample of 20-59 year olds. *Spine* 1995;20:1884.
165. Rose F: Headache: Definition and classification. In Vinken PJ, et al (eds): *Handbook of Clinical Neurology*, Vol 48, No 4: Headache. New York, Elsevier, 1986, p 1.

166. Smith D: Dizziness, a clinical perspective. *Neurol Clin* 1990;8:199.
167. Adams R, Victor M: *Principles of Neurology*, ed 3. New York, McGraw-Hill, 1985.
168. Brown J: A systemic approach to the dizzy patient. *Neurol Clin* 1990;8:209.
169. Troost B: Dizziness and vertigo in vertebrobasilar disease, Part I. *Stroke* 1980;11(3):301-303.
170. Troost B: Dizziness and vertigo in vertebrobasilar disease, Part II. *Stroke* 1980;11(4):413-415.
171. De Jong J, Bles W: Cervical dizziness and ataxia. In Bles W, Brandt T (eds): *Disorders of Posture and Gait*. Amsterdam, The Netherlands, Elsevier, 1986, p 1856.
172. Abrahams V: The physiology of neck muscles: Their role in head movement and maintenance of posture. *Can J Physiol Pharmacol* 1977;55:332.
173. Cohen L: Role of eye and neck proprioceptive mechanisms in body orientation and motor coordination. *J Neurophysiol* 1961;24:1.
174. Manzoni D, Pompeiano O, Stampacchia G: Tonic cervical influences on posture and reflex movements. *Arch Ital Biol* 1979;117(2):81.
175. Cope S, Ryan GMS: Cervical and otolith vertigo. *J Laryngol* 1959;73:113.
176. Gray LP: Extralabyrinthine vertigo due to cervical muscle lesions. *J Laryngol* 1956;70:352.
177. De Jong PTVM, De Jong JMBV, Cohen B, Jongkees LBW: Ataxia and nystagmus induced by injection of local anesthetics in the neck. *Ann Neurol* 1977;1:240.
178. Hinoki M: Vertigo due to whiplash injury: A neurological approach. *Acta Otolaryngol Suppl (Stockh)* 1985;419:9.
179. Liang M, Andersson G, Bombardier C, et al: Strategies for outcome research in spinal disorders. *Spine* 1994;19(S):2037S.
180. Coste J, Paolaggi J, Spira A: Classification of nonspecific low back pain. II. Clinical diversity of organic forms. *Spine* 1992;17:1038.
181. Guccione A: Physical therapy diagnosis and the relationship between impairments and function. *Phys Ther* 1991;71:499.
182. DeRosa C, Porterfield J: A physical therapy model for the treatment of low back pain. *Phys Ther* 1992;72:261.
183. 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* 1993;73:216.
184. Dekker J, van Baar M, Curfs E, et al: Diagnosis and treatment in physical therapy: An investigation of their relationship. *Phys Ther* 1993;73:568.
185. Riddle D, Rothstein J: Intertester reliability of McKenzie's classification of the syndrome types present in patients with low back pain. *Spine* 1993;18:1333.
186. McKenzie R: *The Lumbar Spine: Mechanical Diagnosis and Therapy*. Waikanae, New Zealand, Spinal Publications, 1981.
187. Margolis RB, Tail RC, Krause SJ: A rating system for use with patient pain drawings. *Pain* 1986;25:57.
188. Kraus SL: Cervical spine influences on the management of TMD. In Kraus SL (ed): *Clinics in Physical Therapy: Temporomandibular Disorders*, ed 2. New York, Churchill Livingstone, 1994, p 325.
189. *Grieve's Modern Manual Therapy*. Boyling & Palastangat. 1994; New York, Churchill Livingstone.
190. Zusman M: Spinal manipulative therapy: Review of some proposed mechanisms, and a new hypothesis. *Aust J Physiother* 1986;32.
191. Korr (ed): *The Neurobiologic Mechanisms in Manipulative Therapy*. New York, Plenum Press, 1978.
192. Idczak R (ed): *Aspects of manipulative therapy. Proceedings of a Multidisciplinary International Conference on Manipulative Therapy*, Lincoln Institute of Health Sciences, Melbourne, Aug 1979.
193. Rothstein J (ed): *Manual therapy: Special issue. Phys Ther* 1992;72.
194. 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.
195. *Manipulative Physiotherapists Association of Australia, 7th Biennial Conference*. Nov 27-30, 1991, Blue Mountains, New South Wales. Published by Manipulative Physiotherapists Association, Australia, North Fitzroy Victoria, Australia. Scientific Programme Convenor, Jenny McConnell.
196. Bogduk N: A scientific approach to cervical diagnosis. *Proceedings from the Fourth Biennial Conference of Manipulative Therapist Association of Australia*, Brisbane, May 22-25, 1985, pp 151-158.
197. Grieve G (ed): *Modern Manual Therapy of the Vertebral Column*. New York, Churchill Livingstone, 1986, pp 270-282.
198. Jull G, Bogduk N, Marsland A: The accuracy of manual diagnosis for cervical zygoapophyseal joint pain syndrome. *Med J Aust* 1988;148:223.
199. Shepard K, Jensen G, Schmoll B, et al: Alternative approaches to research in physical therapy: Positivism and phenomenology. *Phys Ther* 1993;73:88.