The Temporomandibular Joint and Related Orofacial Disorders

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Signs and Symptoms

Textbooks and clinicians generally agree that there are four cardinal signs and symptoms of TM disorders.

- Pain in the TM joints, muscles of mastication, and adjacent soft tissues.

- TM joint sounds that occur during mouth opening and closing and moving the lower jaw to either side or forward.

- Tenderness of the TM joints, muscles of mastication, and adjacent soft tissues on digital palpation.

- Limitation on opening the mouth and moving the lower jaw to either side or forward.

Other signs and symptoms that have been associated with TM disorders include the following:

- Headache

- Neckache

- Ear problems (eg, tinnitus, earache, stuffiness, impaired hearing)

- Dizziness

- Visual disturbances

- Paresthesias (eg, burning mouth, tongue, throat)

- Gastrointestinal distress

- Sinus complaints.

However, there is limited evidence to establish a positive relation between these symptoms and the major signs and symptoms.

Pain: The Chief Symptom

Pain is the most obvious symptom identified by the person with a TM disorder. Usually, it is pain that brings the individual to the doctor. From a scientific perspective, pain can be thought of as an experience evoked by stimuli causing or warning of impending tissue damage and "defined introspectively by every person as that which hurts". According to the International Association for Study of Pin, pain is "an unpleasant sensory and emotional experience associated with either actual or potential tissue damage, or described in terms of such damage."

Dimensions of Pain

Pain is a subjective phenomenon, recognized either by verbal communication or by nonverbal behaviors of the person in pain. This subjective nature complicates diagnosis and treatment of the patient.

Different dimensions of the pain experience have been recognized, including the sensory, affective, and evaluative qualities. The sensory component reflects variation in intensity, quality, or duration of pain. The affective dimension deals with the degree of pain unpleasantness. Words that trigger tension, fear, or autonomic response fit this part of the pain experience. The evaluative dimension describes the overall subjective intensity of the experience. These dimensions depend on numerous cognitive and motivational experiences and are influenced by psychological factors. An astute clinician must recognize the differences among these dimensions, particularly when interviewing the patient with pain.

Attacks of Pain

Nearly everyone suffers pain from time to time. Fortunately, for most sufferers of TMD pain, each episode ends with resolution of the symptoms. If the pain resolves in a short period of time, the person is said to suffer from acute pain. If the pain persists for long periods of time, the person suffers from chronic pain. A small but significant number of individuals may have recurrent, frequent episodes of pain.

Chronic pain afflicts many individuals of the general population. Its course and the resulting disability are interwoven with social, psychological, economic, and cultural factors. Chronic pain also is related to changes in the body due to normal processes such as aging. These factors must be weighted in the diagnosis and treatment of the individual.

Chronic pain accompanies many musculoskeletal disorders. Musculoskeletal pain, primarily in the upper or lower back, accounted for the largest percentage of all disorders found at the initial visit by physicians. According to the 1980 National Ambulatory Medical Care Survey, approximately 70 million (41%) of 1.2 billion initial visits to nonfederal, office based physicians practicing in the USA were for these disorders.

Many work days are lost from pain. According to the *Nuprin Pain Report,* Americans lose 550 million work days each year because of pain. If routine activities besides full-time work are included, adults lose more than 4 billion days each year because of pain. The clinicians should be aware of the hierarchy of these numbers. Orofacial pain may arise from

joint, muscle, headache, or dental complaints. Based on the number of days lost, the typical patient is more likely to suffer from somatic pain of the joints than from pain of the muscles. Headache proved the most frequent pain problem. Some 73% of adults suffered one or more headaches in the past 12 months. The average patient would be expected to suffer less from dental pain than from other paints.

Significance: When the patient presents for examination or telephones with a complaint of pain, ask the patient to identify the tissue system responsible for the pain. The problem for the clinician is predicting the course that the pain will follow for each individual. No one can predict with any great degree of accuracy the likelihood of recovery, the response to different therapies, or the potential for rehabilitation. Any correlation between the severity of pain and the level of disability at any given time is difficult, if not impossible to predict. Each case must be assessed on an individual basis.

Pain of TM Disorders: Self Report

Clinicians have great difficulty in identifying the kind of pain present with TM disorders. Like musculoskeletal pain of the back, most TM pain is musculoskeletal in origin. This disorder has been classified as a group III craniofacial pain of musculoskeletal origin. A diagnosis of this syndrome may be made after dental disease has been excluded by history, physical examination, and appropriate diagnostic tests of the teeth.

The individual with a TM disorder may complain of various kinds of pain. They may speak of pain on chewing, pain when opening the mouth wide, pain of the ears, pain that incapacitates or prevents them from functioning with daily activities, sleeping pain, or headache pain.

Typically, TM disorders are characterized by pain just in front of the ear or preauricular area. The pain is usually aggravated by functional jaw movements. If the pain arises from the joint, it is termed *arthralgia.* The pain may originate from the muscles *(myalgia)* or from a nerve *(neuralgia).* Some orofacial pains arise from vascular sources. Although orofacial pain may result from neoplasia, few complaints arise from this morbid condition. If the condition does not fit these descriptions, it is defined as *atypical facial pain.*

Word Descriptors

Word descriptors are terms used by patients to express the kinds of pain they are experiencing. The intensity of TM pain generally has been referred to by patient self-report as dull and aching. The quality has been characterized as cramping, locking, and penetrating. Among 164 patients with myofascial pain of the head and neck, the most commonly expressed words were pressure (48%), dull (27%), throbbing (26%), sharp (18%), burning (16%), and heavy (14%). This list of descriptors does not fully embrace the gamut of words used by sufferers to describe TM pain. Research has shown a relation between certain symptoms and the kind of words chosen by the TMD sufferer.

Some word descriptors are clinically significant. Although there is some overlap in selection by patients, most words have diagnostic importance. An individual with joint noise or joint locking is annoyed by the persistence of these complaints. Pain on chewing is

annoying, but movement of the joints causes pain. Pain from headache or face-ache throbs or feels sharp. Knowledge of these associations is vital in communicating with patients.

The difficulty in identifying the source of the pain is complicated by patterns of referred pain. The individual with *referred pain* feels the pain at one anatomic site, such as the neck, ear, or shoulder, rather than at the site of the pathology (eg, the joint). An often overlooked pattern is the referral of pain of the masticatory muscles surrounding the joints to the teeth.

Numeric Index of Pain

The present level of any pain can be ascertained from a numeric rating index as follows: 1, mild; 2, discomforting; 3, distressing; 4, horrible; and 5, excruciating. In contrast to other known painful syndromes, such as back and cancer pain, the intensity of TM pain has proved low. Among 85 TM patients admitted to a TMJ-Orofacial Pain Center, the pain was rated as mild at low intensity, discomforting at usual intensity, and distressing at high intensity.

Frequency of Pain

The frequency of TM pain as reported by many sufferers varies greatly. In 85 TMD patients examined at a TMJ-Facial Pain Center, 42.5% rated their pain as frequent, 42.5% as constant, and 15% as occasional. These variable findings contribute to the problems that practitioners experience in diagnosing and treating these individuals.

Severity of Pain

In a study that attempted to estimate the severity of TM pain, severity was graded according to six categories. From this information, approximately two thirds of patients with TMD complaints could be expected to suffer from recurrent or severe, persistent pain which would not limit their activities. Nearly 16% would suffer a severe, persistent pain and would require considerable limitation of activities.

Significance: From a clinical standpoint, pain is difficult to assess in the suffering individual. There are few reliable ways to measure pain. What pain means to the patient may not mean the same to the doctor and vice versa. During an interview of the patient, the clinician must be aware that the patient may be talking about pain of the entire body and not specifically about the orofacial pain. This difference in interpretation can significantly influence diagnosis and treatment outcome.

Muscular Pain

The primary source of most TM complaints is masticatory muscle. The exact cause of pain is enigmatic. Patients may complain of fatigue, tension, aching (myalgia), discomfort, soreness, tenderness, or cramping in these jaw muscles. Like other skeletal muscle, masticatory muscle is subject to strains and sprains. *Strain* implies overstretching of tissue. *Sprain* involves rupture of fibers, but continuity of the muscle remains intact. Some strains

and sprains are acute; others become chronic. Most acute complaints are caused by single injuries, whereas chronic problems develop after repetitive or overuse activities.

For purpose of comparison, information about patients with TMD is interspersed with reviews of the present research about another musculoskeletal syndrome, so-called fibromyalgia, to extend the reader's knowledge on the extant literature. Fibromyalgia is a common rheumatologic syndrome. It manifests as chronic, diffuse musculoskeletal aching and soreness. The sufferer complains of fatigue and morning stiffness among other problems, many of which are identical to the complaints of patients with TMD. If these complaints reflect muscle pain and the pain reflects pathology, one would expect to find muscular abnormalities in both TMD and fibromyalgia patients.

Muscular Fatigue

Fatigue is defined as an inability to produce a preexisting level of tension, or velocity of contraction, after either static or dynamic activities. This subjective perception represents an increased exertion of physical effort. The sensation is one of localized slight discomfort and weakness. Although patients with either overuse or pain of the jaws may complain of fatigue, on an average, masticatory muscle fibers exhibit low fatigability (they do not tire easily).

Overuse Complaints

The experience of muscle pain within 1 or 2 days of unaccustomed or excessive exertion is a classic phenomenon. Localized tenderness and increased fatigability follow. Initially, fatigue results from muscles that contract concentrically. Concentric contractions shorten muscle fibers. Repetitive clenching of the teeth causes facial discomfort from this kind of isometric contraction. Among healthy adults, an average pain threshold is reached within 51 seconds of voluntary clenching. Swift recovery occurs after clenching: 80% of the pain decreases within 1 minute. Thus, static activities such as clenching give rise initially to fatigue and then to pain.

Grinding of the teeth is a dynamic activity that causes eccentric changes involving the lengthening of muscle fibers. After 20 minutes of working the jaw muscles by bruxing, there is gradual onset of pain. The pain evolves from negative work that has not been equalized by positive work. Usually, spontaneous pain develops within 8 hours; pain is detectable by digital palpation in approximately 20 hours. Weak to moderate deep pain may last for 2 or 3 days.

Loss of Muscle Strength

The effect of fatigue on the induction of pain has been demonstrated after eccentric contractions of the elbow flexor muscles. Strength was reduced 30% by exercise of muscles at long length and by 10% at short length. Force fell 65% at long length and 30% at short length. The level of pain was worse at long length than short length, and the authors concluded that the development of pain depended on length.

Patients with fibromyalgia had a 60% reduction in isometric and isokinetic muscle strength compared with healthy control persons. Fatigue was figured a significant factor in this loss.

Muscular Tension

Tension is defined as a tightening from prolonged contraction of muscle fibers. There is no motor unit activity associated with tension. Historically, hyperactivity leading to increased tension (hypertonicity) has been hypothesized as one source of jaw pain, although data supporting this hypothesis are meager. Extensive discussion on hyperactivity as the cause of muscle pain is included in Chapter 4.

Based on the hypothesis that hyperactivity leads to tension, electromyography (EMG) is a method to assess changes in muscular activity. Studies have revealed no differences that relate pain with hyperactivity. EMG patterns differed little between patients with fibromyalgia and those with no symptoms. Tonic and phasic EMG muscle tension of patients with fibromyalgia did not prove different from muscular tension of matched asymptomatic control patients. Finally, muscle tension was ruled out as a prominent factor in fibromyalgia. Compared with the EMG patterns of 9 healthy control patients, the EMG patterns of painful muscles of 22 women diagnosed with fibromyalgia showed only minor changes.

Muscular Tenderness and Soreness

Tenderness of the masticatory muscles is a common finding among patients with TMD. Generally, tenderness on palpation signifies muscle soreness. Soreness is a localized pain that can be induced by repeated strain or excessive movements of the muscles. It implies damaged muscle fibers. The damage is not related to metabolic energy demands but has been attributed to the mechanic forces of eccentric contractions.

Muscle Architecture

Microtrauma characterizes the muscle tissue of fibromyalgia patients. Microscopic examination of muscle reveals inflammatory changes, including increased tissue fluids, fat, and numbers of mast cells; swollen mitochondria; and degenerated or compressed muscle filaments.

The same kind of muscle fiber dissolution and extensive inflammation has been found in rat masseter muscle following repetitive lengthening contractions. The authors of this study speculated that the repetitive lengthening contractions may be the mechanism for pain in human masticatory muscle.

Tenderness on Palpation

Lateral joint soreness has been claimed to be of muscular origin. Evidence confirmed that patients with TMD decrease their use of tender muscles because of the pain. EMG recordings of several types of jaw movements showed that 43 patients with temporalis pain used the anterior fibers with less frequency and less intensity than did 17 control subjects. Furthermore, clenching of the teeth significantly related to the degree of muscular tenderness.

During maximal clenching, EMG activity of masseter muscle decreased as the severity of tenderness increased. This reduction in activity indicated that the patients avoided jaw closure as tenderness of the masseter increased.

Muscle soreness proved difficult to induce in healthy men after repeated sustained isometric protrusion. Neither jaw pain nor tenderness was found in the masseter and temporalis muscles of eight normal men who performed a series of protrusive exercises. The authors argued that soreness would result if previous underlying pathophysiologic changes occurred in the muscle.

Myofascial Pain

Myofascial pain is the hallmark of many generalized muscle pain syndromes. Hyperactivity leading to muscle spasm has long been considered important in the genesis of TM myofascial pain, but no true spasms have been found. Muscle spindle sensitivity has been demonstrated to increase, which suggests failure in the large contractile properties of the muscle. No pain has been related to this enhanced sensitivity.

The pain has been characterized as continuous, and usually dull, in one or more muscles. The regional aching seems to be associated with localized tenderness of firm bands of muscle and tendon termed *trigger points.* They are nodes of degenerated tissue. Usually dormant, they may become tender.

The belief that trigger points cause significant pain is controversial. Some clinicians believe that trigger points develop when the fibers are not stretched through the normal range of motion. This shortening can result from lack of use, mechanical overload, misuse, or repeated minor injury. Any excessive strain or overuse may contribute to already shortened fibers and lead to pain. The pain is faithful at a referred site on palpation of a point.

Trigger points have been found both in masticatory muscles and muscles of the neck and shoulders. Clinical judgments made by various health practitioners indicate that people with TM pain also have a high prevalence of neck and shoulder pain. Likewise, many individuals with neck and shoulder pain have a high prevalence of TM pain. The commonality of trigger points in these different anatomic locations suggests that muscle is the primary source of pain.

Each set of masticatory muscle tissue has its own referral pattern of pain. In the illustrations that follow, trigger points are marked by X, "essential" referred areas of pain are shown in black, and "spillover" areas are stippled. Pain does not occur in all patients with spillover areas. The muscle groups are masseter, temporalis, lateral pterygoid, and medial pterygoid. Other muscle groups with pain referral to or near the orofacial region are illustrated. These include the sternocleidomastoid, trapezius, occipitofrontalis, suboccipitalis, splenius capitis, splenius cervicis, medial posterior cervical muscles (semispinalis cervicis and semispinalis capitis).

Myositis

Myositis (myofibrositis) is defined as an inflammation or general change in consistency of muscle and attached connective tissue. No focal muscle spasms occur. Lesions are present along the borders of muscle fasciae and may result from rupture of collagen fibers. Damage occurs in the contractile fibers. Usually, acute pain is accompanied by some localized swelling. Tenderness may occur over the entire region of the muscle.

Myositis of the TMJ region must be differentiated from fibromyalgia or systemic myofibrositic pain. It is uncommon among TMD sufferers.

Myospasm and Trismus

Spasm implies cramping, a sudden involuntary contraction of the muscle. *Trismus* is a motor disturbance of the trigeminal nerve. Acute pain signifies spasm, a complication of trismus that is usually caused by overstretching or overuse. This painful state may prevent the affected individual from opening the mouth, and there may be marked limited range of mandibular motion. Fasciculation is identified by continuous muscular contraction. Palpation reveals a firm, linear band within the affected muscle.

Speculation is that microspasms cause masticatory muscle pain. Groups of fibers can spasm without immobilizing the entire muscle. Intermittent pain can be relieved by gentle stretching. Spasm is too painful to be considered a major cause of chronic jaw pain.

Contracture

Contracture is a chronic, usually painless, resistance to stretch that results from scarring or fibrosis. There is unyielding firmness on passive stretching. The limited range of motion is not associated with a joint disorder.

Up to 80% of patients with fibromyalgia have stiffness. The content of energy-rich phosphates is reduced in painful muscles, suggesting that this form of contracture results form an energy crisis.

Splinting

Splinting implies rigidity that is protective in nature. The patient guards against mandibular movement. Muscles may be tender on palpation. There is no evidence that prolonged splinting results in spasm.

Hypertrophy

Hypertrophy is a generalized abnormal enlargement of muscle tissue. Usually, no pain is involved. The condition is typically unilateral after persistent clenching. There may be a dull aching after extensive bruxing, and range of mandibular motion may be limited.

Significance: Muscle pain seems to account for most face and neck complaints of TMD sufferers. The presence of widespread tenderness on palpation suggests myofascial pain

of the masticatory muscles. This pain results from specific tender areas termed *active* trigger points of pain referral. Often, those trigger points of the head and neck regions are a manifestation of myofascial syndrome of somatic origin. The astute clinician must master the technique of muscle palpation for proper diagnosis. Details of palpation are covered in Chapter 7. Appropriate identification of trigger points enhances treatment.

Joint Pain

The source of joint pain is difficult to establish. Conclusions drawn from the literature indicate that comments about the source of pain would be inferential. Most sensations from joint structures have been considered proprioceptive in nature. The fibrous tissue covering the mandibular condyle, the articular eminence, and the large central area of the disk lacks nerves and blood vessels. Few nerves have been found in the synovial tissues.

Branches of the trigeminal nerve supply the joint. The mandibular nerve branches into the auriculotemporal and masseteric nerves. Finer branches innervate the collateral diskal ligaments that attach the disk to the medial and lateral poles of the condyle, the retrodiskal tissue interposed between the disk and posterior wall of the capsule, and the capsule of the joint.

Most receptors are found in the lateral and posterior parts of the joint. Terminations have been found to be of three types:

1. Free nerve endings occur in ligaments, the joint capsule, and, to some extent, the synovial membrane. Numerous sensory endings are pain-sensitive to tissue injury and are common near the junction of the capsule and periosteum.

2. Nonencapsulated endings are of the Rufini type and occur mainly in the capsule, primarily in regions that are compressed or otherwise deformed during movement. These structures are designated *Golgi tendon organs* when located within ligaments and *Rufini-type endings* when present in the capsule. They are sensitive to stretch induced by movement or tension or by increase of intra-articular pressure.

3. Encapsulated receptors are Pacinian-like corpuscles that are most involved in control of reflexes.

The actions of receptors are complex. Recordings of individual nerve fibers supplying the joint indicate two different kinds of receptors are involved with jaw movement. One type fires rapidly when the condylar hear is rotated and provides information about mandibular movement. The other kind is slow adapting and fires when the condylar head is at a specific position. The nociception probably derives primarily from contact or irritation of the free nerve endings.

Arthralgia may arise from several pain-sensitive structures. An early suggestion was that arthralgia resulted from stretching of the capsule during translation of the mandible. Stretching of the capsule was not considered as important as condylar force directed against the synovial structures behind the disk. Tearing of the fibers behind the disk from the condyle neck and poles has been hypothesized. Maldirected condylar force could produce joint pain

in cases of anterior disk displacement. This primary source of pain could lead to protective muscle splinting.

Pain has been associated with inflammatory aspects of arthropathy. Synovitis and joint effusion frequently accompany derangement of the disk. Pain referred to the face is a frequently observed symptom.

Significance: Although proof is lacking, irritation of free nerve endings is the likely cause of minor episodes of joint pain. Tenderness and soreness probably relate to stretching of tendinous attachments and localized muscle changes. Trauma to the joint may result in pain caused by swelling and effusion.

Arthritic Pain

Osteoarthrosis

Bony changes of the joint identify *osteoarthrosis,* a noninflammatory condition with neither pain nor associated tenderness. A hallmark is crepitus on mandibular movement.

Osteoarthritis

Osteoarthritis is often referred to as degenerative joint disease. It is a disease of both articular cartilage and subchondral bone. The joint lesion is primarily noninflammatory, characterized by deterioration of articular cartilage and remodeling of underlying bone. Erosion results from pitting and fissuring of the cartilage. This destruction progresses to spiculation of subchondral bone. The bone collapses then undergoes sclerosis, with concomitant clinical disability of the joint. Advanced joint disease may lead to atrophy of associated muscles.

The pain of osteoarthritis has been hypothesized as arising from several sources. It has been attributed to synovitis, secondary to the original degeneration. Joint soreness correlates with cases of synovitis. Effusion results in swelling of surrounding tissues. Irritation of the attachments, capsule, and possibly the superficial bone has been suggested as another cause.

TMJ osteoarthritis may occur simultaneously with internal derangement of the disk or may follow it. Different stages of internal derangement with varying degrees of pain have been identified: a few episodes of pain occur in the early stage; multiple episodes characterize an intermediate stage, followed by chronic, variable episodes in the late stage.

Most of the degeneration occurs in the lateral part of the joint. Morphologic study of human TMJs at autopsy showed that 56% of 102 individuals had some form of articular remodelling or arthritic lesions. Twenty-two were judged with major arthritic changes, of which 4 involved all joint parts and 18 had local changes. Of the 18, 13 occurred laterally, 5 centrally, and none medially.

The clinical features of osteoarthritis are similar to those of other forms of joint dysfunction. Typically, pain and crepitation occur during mandibular movement. The joint and surrounding masticatory muscles become tender to palpation. Most people experience

restriction of the mandible. Usually, crepitation remains after the other symptoms disappear. It is one dominant clinical finding that can be expected at follow-up examinations, but it alone cannot be used as the determining clinical feature of TMJ arthritis. Other bilateral symptoms are common with both.

A long-term study of 119 patients observed for up to 10.5 years and examined for pathology of the TMJs revealed a well-defined stages of degeneration in osteoarthritis. The initial stage is characterized by mild symptoms. The intermediate stage follows with mandibular pain at rest and during lateral and opening movements, and restriction of movement of the mandible. Patients generally seek care at this stage. Individuals in the terminal stage may present with crepitation, slightly reduced mandibular movement, and joint tenderness on palpation. As the symptoms of arthropathy diminish, joint clicking may occur on the contralateral side.

Arthritic lesions develop slowly over many years without any symptoms. Sometimes, they cause acute painful inflammation. Acute symptoms last about 9 months then gradually subside; patients usually require treatment during these periods. Some severe symptoms may continue for 1 to 3 years. Study of 11 patients examined at a follow-up of 7 years showed 6 had no symptoms, 3 had slight symptoms, and 2 had moderate symptoms.

In another study, TM symptoms appeared less than 1 year after general joint complaints in 55% of a group of patients with osteoarthritis. They lasted for more than 2 years in 96% of these patients, compared with 12% for control patients. Other clinical findings are summarized in Table 3-6.

Some of these findings have been challenged by different groups of investigators. These clinicians concluded that the evidence for clicking leading to osteoarthritis is too meager to make definitive statements. This conclusion is supported by the findings in two other studies. Clicking was more common in individuals lacking complex joint problems than in patients with osteoarthritis or rheumatoid arthritis (see Table 3-6). Reciprocal clicking was found in joints with slight or no evidence of osteoarthritis.

Rheumatoid Arthritis

After long-term study of patients with rheumatoid arthritis, the results showed 1 in 3 experienced TM symptoms and 1 in 10 developed TM symptoms. TM symptoms appeared less than 1 year after general joint complaints in 38% of this group. The symptoms lasted for more than 2 years in 55%, compared with 12% for the control patients.

Skin surface temperature over the affected joint and the adjacent masseter muscle was low in patients with rheumatoid arthritis. It was lower in the joint areas of these patients than in the joints of healthy patients, but higher than in patients with internal derangement. The low temperature in the masseter has been attributed to disuse atrophy and decreased muscular blood flow.

Comparison of 40 juveniles with rheumatoid arthritis matched by age and sex with 40 asymptomatic individuals showed few differences in symptoms reported by questionnaire. Compared with control patients, arthritic patients more often experienced symptoms and

crepitation in the cheek. Clinical examination revealed that the arthritic patients had more signs considered to be dysfunctional, including reduced maximum mouth opening. Crepitation occurred only in the arthritic patients (total of 6). Tenderness on palpation was more common in the arthritic patients for the posterior aspect of the TMJs, temporalis tendon, and lateral pterygoid areas. No differences were found for eight other areas palpated.

Various textbooks mention anterior open bite as a classic sign of rheumatoid arthritis. A recent review of the literature on occlusion and TMD problems concluded that skeletal anterior open bite was strongly associated with osteoarthritic patients and was rare in other populations. Approximately 25% of 49 patients with TMJ arthrosis had anterior open bite. Analysis conducted on juveniles with rheumatoid arthritis matched against nonarthritic control patients showed a significant difference only for those with class II (postnormal) occlusion. Thirty-three percent of the arthritic group had occlusal problems compared with 8% of the control group. One cannot be too excited about this difference since the sample size was small. Also, for neutral occlusion, 93% of the control group had similar problems compared with 68% of the group with arthritis. Furthermore, just 7% to 11% of the 123 affected patients had open bites. Thus, although open bite may occur frequently in arthritic populations, it should not be considered a main diagnostic feature. Adaptive changes may occur in some individuals and prevent the open bite.

Significance: Pain may arise from more than one area of the joint. Inflammation must be present, most likely synovitis. Joint soreness may represent synovitis or capsulitis, which are difficult to differentiate. The presence of crepitation signifies potential joint pathosis and correlates with advanced osteoarthrosis. The lateral surface of the joint degenerates most frequently. Radiographic, arthroscopic, or magnetic resonance analysis is required to establish the extent of degeneration.

Neurogenous Pain

The sensation of pain is perceived in the brain. Receptors in the body transmit impulses along nerve fibers to the spinal cord and then to the brain. Neurogenous pain usually occurs along the course of the involved cranial nerve.

Nociceptive Afferent Pathway

The nociceptive afferent pathway forms a third type of conduction system in the body. Sympathetic and parasympathetic systems form the basis for the fight-or-flight mechanisms. When appropriately stimulated, nociceptive fibers can transmit pain or cause a mitigation of pain.

Three different types of nerve fibers detect pain and other sensations; they are designed C, alpha-beta, and alpha-delta. The thin C fibers form a network under the skin and transmit impulses slowly. The thick alpha-beta fibers are few in number but transmit impulses quickly. Alpha-delta fibers are relatively thin and transmit impulses more quickly than C fibers but more slowly than alpha-beta fibers.

C fibers carry pain to the spinal cord. Some alpha-delta fibers can carry pain and act in much the same way as C fibers. C fibers are capable of regeneration; alpha fibers are not.

In a damaged area, impulses are transmitted through C and alpha-beta fibers, alpha-beta fibers carry nonpainful sensations and block the painful signals of C fibers.

Spinal Gating System

This fibrous network participates in the operation of the "gate control system". A specific area of the spinal column, the substantia gelatinosa of the dorsal horns, functions as the gate control system. This area modulates pain by influencing the afferent patterns. The effect of alpha-beta fibers is to close the gate, and the effect of C fibers is to open the gate.

A simplified version demonstrates this system. When the mandible is abruptly displaced, the patient may feel a sharp sensation in the joint. This sensation travels quickly through the alpha-beta fibers to the brain. A negative feedback mechanism is activated in the brain that reduces the original sensation. Shortly afterward, a second slow pain travels through the C fibers to the brain. Transmission by C fibers activates a positive feedback mechanism that exaggerates this slow pain effect. Now, the patient recognizes the pain as a dull ache.

The gate control system may be preset or reset any number of times. Specific neurons of the dorsal horn, so-called transmission cells, form the action system. Once triggered, this system elicits both conscious perception and physical reaction to the pain. Both pain perception and reaction act together to create suffering in the individual. Continued suffering becomes experience and subject to marked changes in reaction.

Significance: The gate control system may be modified by therapeutic modalities. Nerve blocking by local anesthesia is one method of influencing transmission of pain. Another method is transelectrical nerve stimulation (TENS). With TENS, an electric current of low intensity stimulates both large and small fibers. Activation of large fibers closes the gate to pain transmission by small C fibers. Also, small fiber input, stimulated by the electrical current, activates the inhibitory areas of the brain and minimizes pain transmission.

Characteristics of Neurogenous Pain

Neurogenous pain is caused by structural abnormalities of nerves that innervate affected areas. Pain can arise without nociception. The quality of pain is typically described as stimulating, bright, and burning. An abnormal sensation may occur along the nerve pathway. The patient usually can locate the pain, but the perceived area may not be the source. A pattern of referred pain may be absent. The severity of pain is almost always more intense than the degree of stimulation.

Peripheral and central mechanisms may be involved. Peripherally, nociceptive neurons from primary afferent fibers may become hyperactive and elicit pain. Centrally, there may be less central inhibition or increased activity of the sympathetic efferent fibers. Neurogenous pain can be classified into neuropathic and deafferentation categories.

Neuropathy

Neuropathic pain is caused by a functional abnormality within a peripheral nerve. It can be divided into paroxysmal neuralgia, neuritic neuralgia, and traumatic neuroma pain. The last can also be classified as deafferentation pain.

Paroxysmal Neuralgia

Paroxysmal neuralgia represents a distinctive, severe, paroxysmal head pain that seems to arise from sudden episodic, intrinsic, and excessive discharges from a specific nerve. The quality of pain is described as electric-like, jabbing, burning, or stabbing. Unilateral pain travels along the distribution of the nerve. Two examples of the orofacial region are trigeminal neuralgia and glossopharyngeal neuralgia.

Trigeminal Neuralgia

Trigeminal neuralgia (tic douloureux) is unilateral pain that involves the trigeminal (V) nerve. The highest frequency occurs in the mandibular division. Ten of 18 patients had pain limited to the V_3 branch, 3 each to the V_2 and V_1-V_2 branches, and 1 each to the V_1 and V_2-V_3 branches (Table 3-8). A combination of peripheral and central factors seems to be involved. The International Association for the Study of Pain recognizes two secondary forms of trigeminal neuralgia. One arises from a lesion in the central nervous system and the other appears after facial trauma. Together, they account for less than 12% of all cases of this neuralgia.

The main features of trigeminal neuralgia are sharp, agonizing electric shock-like stabs of pain felt superficially in the skin or buccal mucosa. Touching a trigger zone around the lips or jaw precipitates the brief, electric-like bursts. The pain lasts from seconds to minutes and can be precipitated again after a refractory period of several seconds. Tic douloureux is probably one of the most intense of all acute pains.

Trigeminal neuralgia can be classified into symptomatic and idiopathic forms. The symptomatic form is experienced by most individuals who present to clinicians. A pathologic factor may be found, such as a tumor of the cerebellopontine angle, multiple sclerosis, or brain stem infarct. The pain is probably caused by compression of the trigeminal nerve by surrounding arteries of the posterior fossa. The idiopathic form has no known cause, but vascular, viral, neural, and dental origins have been suggested. There may be demyelination of the nerve.

An argument has been made for dental involvement. Tooth extraction is the chief cause according to one investigator. Presumably, extraction triggers ephaptic transmission (impulse conduction across nonsynaptic membranes) between the broken fibers for phasic pain of the tooth pulp and neighboring fibers of the epicritic (purposeful) and proprioceptive sensitivity. Remissions are explained by the theory of biorhythm neogenesis with the involvement of the antinociceptive and nociceptive subsystems. This proposition merits consideration, but it fails to explain affected individuals with no history of tooth removal. Also, few patients with typical trigeminal neuralgia present with toothache.

An early phase of the mature condition has been reported. This prodromal pain has been termed *pretrigeminal neuralgia* (see Table 3-8). Just 6 of 18 patients with pretrigeminal neuralgia had tooth symptoms. This pain appears to be different than the typical neuralgia. Some patients reported a toothache or sinus-like pain that was triggered by chewing, talking, yawning, or drinking hot or cold liquids. They had little to no dental disease. The typical condition appeared a few days to 12 years later.

Glossopharyngeal Neuralgia

Glossopharyngeal neuralgia follows the distribution of the glossopharyngeal (IX) and vagus (X) nerves. A diffuse pain emanates from the pharynx and postmandibular areas. It involves men and women equally, although there is a tendency for men more than women. Most cases start after patient age 40.

Swallowing, talking, and chewing are difficult and may trigger the pain. The trigger zone in the posterior pharynx causes pain to radiate toward the ear or angle of the mandible. The patient may sleep or swallow on the side opposite the trigger zone to avoid the discomfort. A painful episode is not as severe as that with trigeminal neuralgia.

A serious complication of glossopharyngeal neuralgia is syncope, which may occur during an attack. Probably, a spillover of impulses from the nerve to the dorsal motor nucleus of the vagus results in reflex bradycardia or asystole.

Neuritic Neuralgia

Neuritic pains result from inflammation of pain-conducting fibers. Symptoms follow dermatome patterns. All painful effects are anatomically related to the specific peripheral nerve, and no patterns of pain referral occur. The main clinical feature of neuritic neuralgia is constant, dull, burning, or stimulating pain. The inflammation causes abnormal sensations along the distribution of the nerve.

The inferior and superior branches of the trigeminal nerve may be affected within the oral cavity. Extraction of a mandibular third molar may traumatize the V_3 branch of the trigeminal nerve. An altered sensation or numbness may remain in the lip with neuritic-like pain in the teeth. Pain of this sort following extraction of the maxillary teeth may be confused with a pain caused by inflammation in the maxillary sinus.

Postherpetic Neuralgia

Postherpetic neuralgia is a neuritic pain that affects the face and mouth. A history of varicella-zoster virus is diagnostic. The acute herpes zoster persists as a chronic burning pain. The pain precedes a rash (shingles) by a few days. Dysesthesia along the involved dermatome confirms the diagnosis.

The ophthalmic branch of the trigeminal nerve may become involved. Lesions may form on the cornea. Vesicles occur on the tip of the nose. Postherpetic neuralgia generally follows shingles unless appropriate treatment is begun.

Geniculate Neuralgia

Geniculate neuralgia is extremely rare. Infection of the geniculate ganglia of the facial nerve (VII) follows an attack of acute herpes zoster. Painful skin eruptions may occur within the auditory meatus and tympanic membrane. An ipsilateral palsy may result. A severe lancinating pain is felt deep within the auditory canal within several days to a week after eruption of vesicles.

Deafferentation Pain

Deafferentation pain is a neuropathic pain due to damage of a peripheral or central somatosensory pathway. The sensory nerve supply to facial region may be partially or totally lost. The pain may arise after trauma as impulses are interrupted within afferent fibers. Highly emotional states tend to exacerbate the pain, which is severe and resistant to treatment.

Within the orofacial region, deafferentation symptoms may be common after trauma. Few elicit pain. Clinical features include burning pain and dysesthesia in the affected area. Occasionally, paresthesia or anesthesia develops after extraction of a posterior mandibular tooth. This altered feeling around the extraction site may not appear for several months. It may then spread to other orofacial fields. Trauma to the mandibular branch of nerve V must be suspected.

Deafferentation pain can be divided into traumatic neuromas, atypical odontalgia, and reflex sympathetic dystrophy.

Traumatic Neuroma Pain

Neuroma pain is rare in the orofacial area. Microscopy reveals an area of poorly integrated nervous tissue in the traumatized area, where successful healing has not occurred. Painful neuromas have been suspected for the trigeminal and facial nerves after surgery, fracture of the facial bone, or development of an area of scar tissue. The pain of traumatic neuromas has been described as deep, aching, and burning. It can be induced by compression or stretching the neuroma and is relieved by local anesthesia.

Neuromas may be present at the surgical site of the TMJ, but no one has proved that they contribute significantly to the painful complaints associated with the joint.

Atypical Odontalgia

Atypical odontalgia has been characterized as toothache or tooth-site pain, with chronic occurrences of 4 months or longer, normal radiographs, and no observable cause. This persistent pain occurs in apparently normal teeth and surrounding alveolar bone. Atypical odontalgia was formerly grouped under atypical facial pain or atypical neuralgia and included a host of unexplained disorders of the trigeminal-cervical 1 and 2 sensory fibers.

The tooth pain appears to be a localized form of this syndrome. It is marked by a dull, burning pain felt deep in the tooth that seems to radiate into soft tissues and bone (Table 3-9). The pain may be throbbing of varying intensity but continuous in nature. It may move from

tooth to tooth. Unpredictable responses to percussion and pulp testing occur. Usually the teeth are hypersensitive to heat and cold. Radiographs of affected teeth and surrounding gingiva are within normal limits.

Most patients relate the onset of pain with minor tooth trauma or pulpal extirpation. Unfortunately, the pain persists even after extraction of the offending tooth in nearly all cases. Most complaints involve premolars and molars. Maxillary teeth are affected more often than mandibular teeth (see Table 3-9).

A hypothesis has been suggested for the constancy of pain. Apparently, the pain is sympathetically maintained from tonic activity in myelinated mechanoreceptor afferents. In some way, trauma serves to establish a high rate of firing in specific neurons and triggers the response in susceptible individuals.

Reflex Sympathetic Dystrophy

Reflex sympathetic dystrophy is sometimes referred to as causalgia (hot pain), but the International Association for the Study of Pain distinguishes these dystrophies from each other. According to this classification, reflex sympathetic dystrophy does not involve a major nerve, whereas causalgia involves a nerve or its major branches.

The pain of reflex sympathetic dystrophy is continuous and has a burning quality. It usually follows mild trauma. Some authors have attributed the dystrophy to deafferentation or nerve injury. It is associated with sympathetic hyperactivity.

Areas of the limbs are affected first. The patient may experience severe hyperpathia on moving or touching the skin. The pain may spread to an entire extremity. Vasomotor changes may develop - first, vasodilation with increasing temperature and then later, vasoconstriction in the skin appendages. Disuse atrophy may follow.

Reflex sympathetic dystrophy has not been routinely associated with TMD. Degenerative TMJ disease was found in a 33-year-old woman who suffered from reflex sympathetic dystrophy. She presented with restricted mouth opening of 33 mm. Tomographic radiographs showed flattening of the left condyle. A second case involved a 34-year-old woman with a maximum jaw opening of 33 mm. She was diagnosed with otalgia, hypertonicity of the masticatory muscles, capsulitis, synovitis, and radiographic evidence of remodelling of the TMJs. These changes were considered secondary to the dystrophy.

A minor form may involve the mouth or face, but few patients suffer from this complaint. It may follow minor oral surgical procedures or postsurgical infection. Facial edema and elevated skin temperature followed by cool, cyanotic skin and edema may terminate in atrophy. Temporary reduction in pain may be achieved by stellate ganglion block.

Significance: Fortunately, orofacial neuralgias contribute little to most pain complaints associated with the TMJ apparatus. They are far less common than cervical neuralgias leading to frontal (cervicogenic-related) headache and facial pain. This discomfort originates from the occipital area and ascends facially. An unpleasant dullness or cranial pressure results.

Vascular Pain

Vascular disease, either local or remove, probably is an uncommon cause of TMJ pain. The degree to which vascular pain contributes to the genesis of TMD symptoms is unknown. Most of the information about head pain and face-ache has dealt with the involvement of vascular changes in headache. A few isolated cases have been reported in the literature involving hypertension, carotodynia, and cranial arteritis. These conditions are reviewed in Chapter 4.

Vascular pain has variable qualities. It has been the subject of various unconfirmed explanations for suffering in headache patients. Moreover, the presence of TMJ symptoms in patients with concomitant headache has caused diagnostic confusion in both directions. TMD pain and the pain of acute and chronic tension headaches have the same system of origin. Each has been classified differently as subcategories of craniofacial pain of musculoskeletal origin. Because the symptoms of patients with these separate pains share similar features, this review critiques the possible association between TM pain and pain of vascular origin.

Status of Fluid Exchanges in the Joint

Various areas of ischemia have been found in condyles subjected to trauma. These condyles had undergone avascular necrosis, a sequela of an inflamed, deranged joint. Generally, pain is a consequence.

The disk is capable of large volumetric fluctuations. Its posterior attachment seems to function as a device for rearrangement of blood, tissue fluid, and synovial fluid.

The superficial temporal and maxillary arteries supply the posterior attachment of the joint. A profuse vasculature forms as branches of these vessels enter the intermediate zone. Veins of this area are large and thin walled, and anastomose freely, forming a plexus mediolaterally. They dilate as the mandible opens. Vessels within the temporal and condylar parts of the posterior attachment are small and course randomly.

Theoretically, localized joint pain could arise as follows. Chronic overloading or frequent abrupt movement of the joint may compress the small arteries. Venous drainage slows, leading to swelling and pain. Alternately, excessive dilation of the veins might occur after sudden opening of the jaws. Arterial vasoconstriction could follow with the same consequences.

Alterations in the vascular layer of the synovial tissues may lead to pain. The synovium becomes expanded as the jaws open. Folds (villi) lining the superior synovial cavity permit the disk to translate anteriorly as much as 2 cm. Similar folds line the inferior synovial cavity; they intermesh with diskal fibers, permitting rotation posteriorly as the condyle translates anteriorly. Speculation is that reduction in viscosity of synovial fluid could lead to frictional resistance within the capsule followed by joint noise, inflammation, and dysfunction. Pain could develop in the associated masticatory muscles as a result of the dysfunctional joint.

Status of Vascular Changes and Some Head Pains

Headaches are viewed as a symptom, not a disease. They may originate from either peripheral or central mechanisms. Vascular structures about the head are pain sensitive, primarily the proximal cerebral arteries and the large veins and venous sinuses. Apparently, noxious stimulation of sensory receptors and afferent fibers in vascular and perivascular tissue produces the pain.

Stretching or pulsation of arterial walls and muscle contraction have been suggested as affecting peripheral nerve receptors in these vessels, leading to head pain. Since the trigeminal nerve is the major innervation for these vessels and the TMJs, overlap between TMD complaints and headache would be expected.

Migraine

The mechanism of the vascular headache, the so-called migraine, is not well understood. Migraine has been considered a familial disorder with vasomotor instability of the extracranial and intracranial arteries, leading to excessive vasoreactivity. Recent evidence shows that the symptoms of the migraine aura are caused by deficient cerebral blood flow, although some argue that migraine may represent a neurologic disorder rather than a vascular disorder. Little agreement exists about whether migraine is a single disorder or a set of related disorders. Hence, migraine has been used generically to include many chronic recurring forms of headache.

The throbbing, boring qualities released from nerve endings in the vessel walls lower the pain threshold, and pulsatile pain results. The release of vasoactive neurohumoral substances, including substance P, has been implicated in triggering the pain. Platelet dysfunction has been suggested as another theory.

Argument has been made for common migraine as a muscular disorder rather than a vascular disorder. Other evidence seems to support this belief. Comparison of three subgroups of headache patients reveal many similarities of the common signs and symptoms. Among 19 TMD parameters studied in patients with migraines, tension headaches, or combination headaches, jaw pain on movement and tenderness of the masseter and temporalis muscles differed significantly.

Tension-Type Headache

Tension headache (scalp muscle contraction headache) has been designated craniofacial pain of musculoskeletal origin, but some opinions favor origin from either vascular or central nervous systems. Continuous head pain in the form of nonpulsatile ache characterizes this myofascial disorder. Typically, the aching is symmetric and localized either frontally, frontotemporally, or occipitally. Patients may progress to photophobic, periorbital, or otologic complaints. Often, they are diagnosed as occipital neuralgia. Apparently, fibers of the occipital nerve entrapped among tense cerviconuchal muscles cause the pain.

Cluster Headache

Review of the literature about other forms of vascular headache contributes little to understanding a relationship with TMD pain. Several hypotheses have been suggested for the pathophysiology of cluster headache, including vasomotor disturbances and changes in cerebral blood flow, as well as altered levels of serotonin and histamine.

The pain of cluster headache may be stabbing, burning, or pulsating. It occurs unilaterally and almost always remains on the same side of the head. The eye and frontotemporal region are the usual sites. Attacks may be accompanied by tearing, nasal congestion, rhinorrhea, and a partial Horner's syndrome. Few of these symptoms fit TM pain.

Significance: There is the potential for pain of vascular origin to arise in the joint, probably after an inflammatory process. Attempts to differentiate joint complains from pain referred from headache is difficult. Similar symptoms can be produced by various conditions. Diagnosis of headache is complex even for headache specialists. There are few reliable tests for diagnosis. It is doubtful whether treatment performed by dentists, such as occlusal adjustment, insertion of an occlusal guard, or rebuilding the occlusion, can solve the problems of headache.

Dental Pain

Toothache may be due to dentino-enamel defects, pulpitis, or periapical periodontitis and abscess. Most have throbbing, sharp, boring, and sickening qualities. The boring and sickening qualities set toothache apart from other pains.

Compared with other types of pain, patients rated toothache less painful than postherpetic neuralgia and back pain and more painful than arthritic and menstrual pains (Table 3-10). Two characteristics make toothache rather dreadful: the sharp quality suggests that the intensity will last forever, and this sharpness causes considerable anxiety, which evokes the terror.

Toothache From Dentino-Enamel Defects

Pain from dentino-enamel defects is short-lasting, diffuse, and evoked by local stimuli such as heat, cold, or air. These stimuli cause movement of extracellular fluid that fills the dentinal tubules. Dentinal pain occurs intermittently and lasts from seconds to minutes. Usually it is easy to identify the source of this pain either by radiograph or clinical examination. An exception may involve the so-called split-tooth syndrome. This pain may be sharp, lancinating, and with abrupt onset. Because there may be pulpal involvement, it may be poorly localized by both the patient and the dentist.

Toothache From Pulpitis

Pain caused by pulpitis may vary from mild to spontaneous if no external stimulus triggers it. Throbbing or burning may occur with moderate to severe intensity, and bouts may last for minutes to hours. The tooth usually is not tender to percussion. Spontaneous pain

confirms the diagnosis. If pulpitis is untreated, the pulp may die, with infection spreading into periapical tissues. Severe pain may result from acute periapical periodontitis.

Abscess causes the tooth to be tender to palpation, and biting on the tooth makes the pain worse. The abscess may track to the mucosal surface and drain. The pain may be sharp or dull with some throbbing and may last for hours or days. Pulpitis is identified by examination - radiologic, clinical, or both.

Toothache From Periodontal Abscess

Unlike a gingival abscess, which is confined to the marginal gingiva, the periodontal abscess involves the supporting periodontal structures. Periodontal pain is well localized by the patient. Periodontal abscess can be differentiated from periapical abscess by the presence of a draining abscess on the lateral aspect rather than the apical part of the root. A radiolucent area along the lateral part of the root may be found in the early stages. A single lesion that can be probed from the gingival margin confirms the diagnosis.

Atypical Odontalgia

Atypical odontalgia is a form of toothache or tooth-site pain with no observable cause. Because the ache may persist even after the pulp of healthy teeth has been removed, this toothache is discussed in the section on neurogenous pain of deafferentation origin.

Significance: Most dental pains have observable causes. Peripheral stimuli evoke pathologic processes. The pain can be mitigated or eliminated by local anesthesia. Elimination of the pain leads to successful outcome. Some dental pains are obscured by the presence of simultaneous periapical and periodontal disease.

Psychogenic Pain

Many clinicians suspect a psychogenic component in the history in certain cases of TM pain. These conclusions usually are based on an absence of organic pathology following interview, clinical examination, and even special tests of the patient. The unanswered question in the minds of clinicians is, "Are these patients psychiatrically different from other non-pain patients that I treat, or is this a disorder of lesser behavior due to mental conflict?"

It is widely accepted that emotional processes play a central role in pain and that exposure to and manner of coping with stressful events contribute to the onset and exacerbation of a wide range of painful physical conditions, including TMD. If the pain is chronic, one must determine whether the pain is of somatic delusion or if there is an underlying psychophysiologic disorder.

The pain of somatic delusion occurs in patients with profound psychiatric disturbances, such as psychotic depression or schizophrenia. (These are terms used in psychiatry to describe a conversion of mental experiences into bodily symptoms.) There is no evidence of structural disorder. Few patients with TM pain fall into this category.

Individuals with psychophysiologic disorders have psychological factors that engender physiologic changes. Often they may deny that any psychic factors are involved in their pain. Emotional stress may be a dominant factor. Structural disorders may not be obvious. Typically, these patients present as polysymptomatic with a diffuse pattern of complaints. The concept of "self" refers to a mental representation of what an individual thinks of him- or herself as a total person - a psychophysiologic total. Such images derive from experience of emotions, sensations, and indirect perceptions of the bodily and mental self.

The term *personality* refers to the predictable ways - attitudes or habits - in which an individual responds to life situations. These ways evolve consciously and unconsciously and represent a compromise between a person's wishes and the need to restrict their expression. The chief function of personality is to maintain a stable relationship between the person and the surrounding environment.

A superb review of the literature that signaled the importance of psychological factors in TMD was published as part of the 1983 American Dental Association President's Conference on Temporomandibular Joint Disorders. Psychological factors were considered under a number of different headings (Table 3-11). Based on information from that publication and review of the current literature, the present state of knowledge has been updated as discussed in the following sections.

Personality Traits of Pain Subjects

Differences between *personality* and *self* are vital for understanding that no single trait predisposes to TMD. Clinicians recognize may stereotypes of personality, which have been divided into normals, hypernormals, and psychoneurotics. Normal persons are free of emotional problems, have a sense of humor, and relax easily. Hypernormal individuals are divisible into subgroups:

Overly responsible - a person who feels responsible even for situations and events that are patently not his or her responsibility

Career individual - a person with a high activity level who finds projects and is action oriented.

Explorer - a person who is driven to enhance himself or herself at the expense of others.

High approved motive - a person who carries the need for approval to extremes and who has an exaggerated fear of rejection.

Harridan - a person who rules by nagging, bullying, or cruelty.

The psychoneurotic is an individual handicapped in life by certain personality traits and is truly emotionally troubled. Although none of the three stereotypes has proved clinically useful, they give meaning to the way patients behave and enhance the diagnostic and treatment skills of the clinician. Every clinician knows individuals who subject themselves to

much more external stress than normal. Many individuals with TM pain seem to fall into this category.

Problems in Testing for Emotions

Numerous scales and inventories have been used to assess the psychological status of individuals with TMD. These include the Minnesota Multiphasic Personality Inventory, Cornell Medical Index, Interpersonal Adjective Checklist, Edwards Personality Profile, Thematic Apperception Test, Cattell Personality Factors Questionnaire, and Eysenck Personality Inventory. Since the 1983 review by the American Dental Association, these and other inventories have been used (Table 3-12).

There have been several criticisms of the clinical significance of these tests. The general feeling is that clinicians should not waste their efforts on analyzing specific psychological characteristics of individual patients. A second criticism asks the question whether higher scores reflect pain suffering caused by the disorder or if emotional disturbance leads to the suffering. In addition, these that inventory psychological factors have not yet been diagnostically applied successfully to patients with TMD.

The first criticism has merit. There are many methodological flaws in the interpretation of paper and pencil tests. Only a trend can be inferred by comparing results from a single profile against standardized values obtained from many profiles, and findings may differ between subjects even though the same inventory has been used. For example, consider the case for assessment with the Minnesota Multiphasic Personality Inventory (MMPI). About half of the patients with TM pain had an elevated score of anxiety on the MMPI compared with matched controls without TM pain. Although the level of anxiety was higher for pain patients, it fell within the normal range for this test. In another study, MMPI scores for TMD patients were similar to scores of patients visiting a medical clinic for other disabilities.

There may never be an answer to the second criticism. The problem of establishing cause and effect is not restricted to studies about TMD. Cross-sectional studies of 3023 individuals from the USA, aged 25 to 74 years, showed 14.4% suffered from well-defined chronic pain related to the joints and musculoskeletal system. Pain of uncertain duration was present in another 7.4%. About 83% of the total had received treatment. Of the total, 18% had some degree of depression, compared with only 8% of those who did not have chronic pain. Although these differences were statistically significant, the investigators concluded that they still did not know whether the chronic pain caused the depressive illness or the contrary, or if there was comorbidity of both.

Other attempts have been made to resolve the dilemma. A well-controlled study compared 163 chronic, nonmalignant pain sufferers with 81 control subjects to determine whether emotional disturbance was the cause or the consequence of chronic pain. Numerous measures, including personal history and psychological variables, were studied for both groups. The results suggested that emotional disturbances in the pain patients were more likely the consequence rather than the cause of chronic pain. The pain was related to more current depression and less current life satisfaction.

Evidence suggests that there may be some solutions to the third criticism, that tests that inventory psychological factors have not yet been diagnostically applied successfully to patients with TMD. Different psychological characteristics have been found among diagnostic subgroups of patients with orofacial pain. MMPI scores for hypochondriasis, depression, and hysteria were higher for patients suffering from myogenic facial pain and atypical facial pain than in patients suffering from internal derangement of the TMJ. Three subgroups of psychologically different TMD patients have been identified using the SCL-90-R inventory: normal, moderately distressed, and severely distressed. Patients with distress had high ratings of pain severity and interference with daily functioning.

Visual analogue scores for emotion in clinic patients with TM myogenous disorders were higher for disease conviction and inability to endure pain than for patients with TM arthrogenous disorders. Similar results have been found in another study.

Evaluation of three subgroups of TMD patients (primarily myogenic, primarily TMJ, and combination myogenic and TMJ) confirmed psychological differences among the subgroups. After differences in pain levels were adjusted for the groups, myogenic patients displayed higher concern for bodily functions and illness preoccupation compared with subjects of the other groups. Greater pain was not associated with anxiety and depression levels. Analysis of psychometric variables correctly identified 74% of structural patients and 46% of myalgia patients.

Anxiety

Anxiety is a feeling of apprehension, uncertainty and fear. Assessment with the same anxiety test has produced conflicting results in different groups (see Table 3-12). No difference was found between different subgroups of patients of orofacial pain with the State/Trait Test. Higher levels were reported for TMD patients than for asymptomatic subjects and for muscle-related TMD subjects than for TMJ-related subjects. This same result was previously found using a visual analogue scale (VAS) for emotions. Greater VAS levels were found in highly emotional TMD neurotics than in low neurotics.

Depression

Depression is defined as loss of hope or cheerfulness. It is emotional dejection accompanied by decreased functional activity. Scores derived from studies on depression among TMD patients have varied (see Table 3-12). Most assessments have been made with the Minnesota Multiphasic Personality Inventory. Using this instrument, no difference was found between TMD and asymptomatic subjects. Higher levels of stress were reported for TMD and medical clinic patients compared with pain-free subjects, for muscle-related TMD and atypical facial pain patients versus patients with internal derangement, and for muscle-related TMD patients versus patients with TMJ or combined muscle-joint problems using another scale.

No difference was found between orofacial pain patients and asymptomatic subjects with other depression scales. However, greater levels for depression and hypochondriasis discriminated TMD and other pain patients from pain-free subjects tested with one inventory and between TMD patients and asymptomatic subjects with another inventory.

Among 75 adult patients visiting a multidisciplinary orofacial pain clinic, 31% scored 16 or above on the CES-D scale, a score considered as being at risk for significant depression.

Stress and Coping

Previous studies have suggested that TMD patients handle stress poorly (see Table 3-9). Such results must be viewed with caution in light of differences in the kind of tests used. Amplification of symptoms and disturbed capacity for interpersonal contact were reported for orofacial myofascial pain dysfunction (MPD) patients. Other measurements of stress levels and ways of coping response showed TMD patients differed from asymptomatic subjects. Higher levels of stress or difficulties in coping were found with three separate instruments, but not with three others.

Clearly, the interference of pain and levels of distress associated with life situations depends on the coping ability of TMD patients. Patients have been classified as normal, moderately distressed, or severely distressed; as dysfunctional, interpersonally distressed, or adaptive copers; and as highly emotionally distressed, moderately emotionally distressed, or nondistressed. Somatization was the chief factor found by analysis of psychological variables among TMD patients. The authors of that study concluded that the extent of somatization was a potent contributor to pain chronicity. These findings are consistent with results of other studies. A greater tendency toward distress and psychological isolation has been found among patients with chronic MPD syndrome of the total body than in asymptomatic subjects.

Illness Behavior

Illness behavior embodies a broad range of pain-related complaints and responses. Several factors are embraced by the Illness Behavior Questionnaire: hypochondriasis, disease conviction, somatic versus psychological perception of illness, affective inhibition, affective disturbance, denial, and irritability.

Results with this instrument have been consistent for most studies (see Table 3-12). One or more factors have been identified as significantly different between TMD patients and asymptomatic subjects. TMD subjects had higher scores for somatic preoccupation, greater anxiety or depression, and inability to accept reassurance from the doctor easily, and were less likely to acknowledge psychological aspects of illness, as well as disease conviction. TMD patients with varying degrees of neuroticism differed significantly from TMD patients rated as extroverts.

No significant differences were found between TMD subjects and patients with other bodily pains. Populational differences have been reported. TMD patients from a Finnish population scored lower on affective disturbance than Australian patients.

Psychological Stress Relationship and Muscle Tension

Several clinicians have reported that TM pain results from intense muscular activity that derives from emotional states. If one type of stress predominates over another, the condition may become pathologic, as when increased tension in the jaw muscles occurs after an emotionally stressful event. This is referred to as response specificity. One theory is that

psychological stress elicits muscular hyperactivity terminating in TMD pain. This logic forms the basis for the psychophysiologic theory of myofascial pain dysfunction syndrome in TMD. The literature relating to this theory is divided and varies greatly in quality, and there is a persisting problem of distinguishing muscle pain syndromes from psychological illness.

Clinical Correlational Studies

Many clinical observations suggest a relationship between psychological stress, increased muscle tension, and parafunctional habits. The classic example is the presence of bruxism and symptoms of the masticatory muscles. Most of these studies have focused on the presence of greater muscular tenderness and greater tooth wear in bruxers than in nonbruxers. A clinically relevant study compared self-reported frequency of nine oral habits in individuals with (1) facial pain, (2) tension headaches, or (3) no facial pain. Clenching occurred most frequently in the facial pain group. Headache subjects reported a higher frequency of resting the hand on the chin and face than the group reporting no pain. It was hypothesized that one or a combination of oral habits could elicit muscular changes leading to head pain. In this case the mechanism by which patients became symptomatic seemed clarified, but not all casual relationships can be inferred from this kind of correlational study.

Experimental Studies

Numerous experimental studies suggest a relationship between stress-related muscular activity and TMD symptoms, and empirical findings support this notion. These studies fall into three categories: EMG activity of individuals in the natural environment; EMG activity under laboratory conditions; and the effect of experimental pain on individuals with muscular symptoms.

EMG activities of the jaw muscles of symptomatic individuals in the natural environment show that stressful life events create muscular tension. Bruxism and levels of nocturnal activity become elevated during episodes of daytime stress and anticipation of stress. Long-term study of patients diagnosed with chronic myogenous TMD showed that exacerbations of pain were preceded by increased EMG muscular activity.

Under laboratory-induced stress, differentially higher EMG levels occurred in the masseter and frontalis muscles of TMD patients compared with asymptomatic individuals. EMG temporalis activity of TMD patients was different from the activity of matched controls when both were presented with timed psychomotor tasks. Stress, identified by finger temperature and skin admittance monitoring, showed TMD patients responded differently from controls in terms of habituation to stressful stimuli. That is, they seemed to relax or became accustomed to the stimuli.

Other experimental findings suggest that chronic TMD pain sufferers differ from asymptomatic individuals. For example, some investigators found lower pain thresholds, but only trends toward lower pain tolerance among individuals with TM pain, compared with controls. Studies of young women (19 to 28 years of age) exposed to a slow-building pain under laboratory conditions tended to confirm psychophysiologic differences in women with myofascial pain. They had lower pain thresholds, poor discriminability, and greater proclivity to report pain, compared with asymptomatic controls. These laboratory studies have been

faulted for various reasons, including exposure of patients to unrealistic life situations and the argument that the response to stress has nothing to do with symptoms or etiology of TMD.

Treatment Outcome Studies

Outcome studies indicate that many TMD patients respond successfully to various kinds of treatments. Between 70% and 80% of all patients will improve regardless of the kind of treatment rendered. Because patients can be managed with so many different therapies, something other than structural changes seems responsible for this success. The findings do not necessarily mean that patients are rid of the pain or other clinical signs - they simply may not complain as much. An absence of evidence is not evidence of absence.

TMD pain complaints have been reduced using stress management techniques designed to reduce muscular activity, including biofeedback training and relaxation therapy. Assessment of TM patients randomly divided into a group receiving biofeedback and a group receiving relaxation therapy showed no significant differences in report of pain. Both therapies were moderately successful in reducing complaints of pain immediately posttreatment and at a follow-up 2 years later. Although outcomes were not different, some characteristics of the groups were not similar. Patients receiving relaxation therapy were young, had TM pain of short duration, and had other psyhophysiologic complaints. Those individuals treated by biofeedback were older, were married, and had pain of long duration. They also reported less tension of the facial musculature. Two predictors of outcome were presence of the disorder and pretreatment by occlusal adjustment of the teeth. Occlusal adjustment was negatively related to outcome and was not significant in the biofeedback group. Outcome of patients with bruxing habits was considered more successful in the relaxation group. No personality factors were associated with outcome. The study could have been improved by documentation of masticatory muscle tenderness to palpation before therapy, immediately posttreatment, and at the follow-up.

Another outcome study evaluated the effectiveness of psychotherapy and antidepressant medication on chronic, intractable, "psychogenic" pain. A total of 129 patients, most with head and neck pain, were divided into four groups. During 12 weekly sessions, the groups were treated as follows: (1) amitriptyline plus psychotherapy, (2) amitriptyline plus support (reassurance), (3) psychotherapy plus placebo tablet, and (4) support plus placebo tablet. Result showed little difference in the effect on pain. Psychotherapy alone was ineffective in reducing pain, psychotherapy plus placebo slightly increased pain, and psychotherapy plus amitriptyline reduced the duration but not the intensity of pain. Some results were difficult to interpret, possibly due to the undetermined differences in initial baseline levels of pain for the groups.

An impressive study compared psychosocial correlates of TM pain among three groups. TM patients, patients with other painful conditions, and a pain-free healthy group were subjected to a battery of psychological tests (see Table 3-12). This study showed no direct relationship between measures of personality, chronicity of pain, intensity of pain, or perceived severity of dysfunction. There was a weak relationship between chronicity and pain intensity of non-TM pain and perceived stress, hypochondriasis, depression, anxiety, and disease conviction. TM patients and patients with other painful conditions had significantly higher scores for hypochondriasis and depression than asymptomatic controls.

Furthermore, these and other illness traits decreased in patients who responded successfully to treatment. No changes occurred on the coping scales. This indicated that part of the distress resulted from the physical disability. It was suggested that although distress and symptoms may be unrelated, individuals seeking treatment may be more distressed and that reduction of pain after treatment lessens their total worry. The investigators concluded that TMD should not be considered a psychosomatic disorder. The patients did not differ from other pain patients on many psychological measures, but they did differ from pain-free controls. Neither did they differ from either pain patients or asymptomatic controls in illness behavior or manner of coping with stress. Furthermore, generalizations of greater emotional disturbances among TM patients have resulted from clinicians interacting with less manageable patients.

Significance: The findings suggest that there psychologically different subgroups of patients with TMD. Some have personality characteristics that are different from asymptomatic individuals. Patients from one population may be psychologically different from patients of other populations. Although the inventories use different terms, in most of them affected individuals are found to be distressed. Although pencil and paper tests have not always aided in diagnosis, it may be that the questions making up the test were inappropriate for diagnosis or were incomprehensible to the patient in the clinical setting. Certain questions appear to be of predictive value and give evidence of suffering or disability. The heterogeneity of these inventory findings shows no single specific pain-patient personality for the TMD patient.

Psychosocial Characteristics of Those Seeking Treatment

Practically every clinician has seen the patient who overreacts to rather minor symptoms. One of the mysteries about TMD is why some individuals cope effectively with the pain and others do not. It is unclear whether (1) TMD patients who are particularly maladaptive pain copers are confronted by a different class of intrinsically more problematic stressors that stretch their coping ability; (2) the stressors are of a particular type; or (3) the stressors are similar and the main problem is that TMD patients are poorer copers who habitually use ineffective strategies.

An early attempt to classify TMD patients by their capacity for interpersonal contact showed that patients scoring low on the scale were more apt to overreport symptoms. With increased severity of pain and other symptoms, there was an increasing severity of disturbed capacity for interpersonal contact. Patients with this disturbed capacity expected surgical treatment for their dysfunction and were less likely to improve with therapy. A very sophisticated attempt has been made to identify which individuals can and which cannot cope with TM pain. The West Haven-Yale Multidimensional Pain Inventory (MPI) was used because it is comprehensive, demonstratedly reliable, and valid with heterogenous populations of chronic pain patients. From this MPI, a taxonomy has been devised that provides a psychometrically sound heuristic framework for elucidating the mechanisms by which stress and coping processes influence TMD symptomatology. This typology was based on empirical integration of physical, psychosocial, and behavioral data. Three different groups of TMD patients were identified. The crucial difference between groups was largely behavioral and psychosocial, with physical factors playing a secondary role. Physical factors were identified as common symptoms, age, or radiographic findings.

Group one was classified "dysfunctional" individuals; they accounted for 46% of the population studied. Dysfunctional individuals reported the highest levels of pain and affective distress and the greatest amount of life interference. Group two was designated "interpersonally distressed" individuals, who made up 22% of the population. They had intermediate levels of pain and were lowest on perceived social support. Individuals of group three were designated "adaptive copers" and constituted 32% of the population. These individuals had the lowest levels of pain, affective distress, and life interference, and the greatest perceptions of life control. None of the groups differed significantly based on any measures of structural abnormalities or oral dysfunction. Although division of TMD patients into these subgroups is significant, this classification critically limits diagnosis based on clinical findings.

Gender has not proved to be a prepotent variable for symptom presentation at the initial examination of TMD patients seeking treatment. No significant differences were found between men and women with respect to pain experience or emotional state judged from numerous measures of pain intensity, pain unpleasantness, and psychological variables.

Significance: TMD patients differ in their ability to cope with the pain. Complaints may not relate to the number and degree of dysfunctional signs. Pain interference in daily living may relate to interpersonal conflicts.

Psychological Characteristics of Unsuccessfully Treated Patients

Clinicians worry about the 20% to 30% of patients who find little relief from complaints despite treatment. Clearly, these symptomatic individuals are more emotionally distressed than untreated individuals of the general population. Analysis of profiles of both successfully treated and unsuccessfully treated TMD patients showed elevated MMPI scores for both groups compared with untreated individuals. For treated groups, there was a tendency toward somatization and repression. Unsuccessfully treated patients had higher scores for depression, agitation, and anger than successfully treated patients. These findings underscore the psychophysiologic characteristic of TMD.

The presence of psychological problems among treated patients does not mean that the treatment was necessarily a failure. Not all unsuccessfully treated patients should be considered failures because of the presence of pain. Survey of the literature showed 80% of patients undergoing surgery for TMJ pain were satisfied and would have the surgery again. Approximately 70% were essentially pain free; 18% had pain on chewing tough foods. Another study showed 90% of 300 patients treated surgically for internal derangement were generally pain free. Judged by surgical criteria, including clinical measurements for mouth opening and lateral jaw motion, reasonably good functional success had been attained.

These findings contrast sharply with a news release describing a National Institute of Dental Research - funded study completed on 200 TMD patients at the University of Washington dental school. According to that release, 70% of the patients still reported TM pain after surgical treatment.

A central issue that must be addressed is whether clinicians can effectively identify psychosocial problems of TMD patients presenting for initial examination. Evaluation of the

dentist's ability to detect psychological disturbances in these patients is fraught with problems. One study compared the dentist's clinical impression of the existence of psychological problems with criteria determined from standardized psychological tests. Analysis of the results showed that judgments made from screening at the initial examination fail to satisfactorily identify psychosocial problems in these patients.

Comparison of pretreatment and posttreatment scores from five different kinds of personality questionnaires obtained on the same TMD patients illustrates the complexity of judging effectiveness of treatment. Significant differences were found with the Basic Personality Inventory and Illness Behavior Questionnaire, but not with the Perceived Stress Scale, Multidimensional Health Locus of Control, or the Ways of Coping scales. Lower scores were found after treatment for depression and hypochondriasis with the Basic Personality Inventory and the irritability, disease conviction, denial, and affective disturbance scales of the Illness Behavior Questionnaire. The authors concluded that some of the psychological distress may be a function of the physical condition.

Treatment approaches can be improved if dentists and physicians use some of the skills of psychologists. Helping patients with skills for solving problems, approaching life's stressful events in a more rational manner, and dealing with interpersonal relations are a few of the more important areas that can be addressed in this way.

Significance: There is no stereotypic personality that characterizes a typical TMJ profile. Stress and coping are inextricably tied to every aspect of these patients. When imposed demands are perceived to exceed the ability to cope, distress arises. Increases in physical distress do not appear to be linearly related to changes in psychological disturbance. Proof is lacking that negative events always trigger psychological distress.

The psychological status of TMD patients differs little from that of patients presenting with most other painful conditions. No major gender differences have been found in the pain experience and emotional state of subjects seeking treatment.

Many standardized tests are available for evaluating psychological status. This information may be used to determine whether the patient needs referral for psychological counseling. The practitioner should select a test that patients can complete easily and that can be graded easily.

Attempts have been made to clarify the problem of selecting an appropriate test for assessment. Eleven different kinds of questionnaires (seven for depression; four for anxiety) were given to approximately 132 TMD patients. Factor analysis revealed that the different questionnaires measured a single factor. The authors concluded that a simplified two-item assessment was an appropriate as more complex depression and anxiety scales. Half of this two-item test was the Single Question Depression Assessment (SQDA); the other half was the Single Question Anxiety Assessment (SQAA). For the SQDA, the patient responds to the question "How depressed are you?" by rating on a continuum from 0 (never) to 4 (often). For the SQAA, the patient rates himself on the question "Do you consider yourself more tense than calm or more calm than tense?" on a continuum from (0) calm to (4) tense. This short form may be useful for providing immediate information about the emotional status of TMD patients.

A larger and more systematic data base is needed for the assessment of this instrument.

Treatment of TM pain demands careful consideration. There must be clear understanding between clinician and patient before treatment begins. A clinical result acceptable to the clinician but unacceptable to the patient reflects differences in expectations of the two parties.