Multidisciplinary Diagnosis & Management of Orofacial Pain

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Dentistry has enjoyed a remarkable period of technological and scientific growth over the past several decades. With the increase in life expectancy, the number of individuals seeking dental care also has escalated. One of the most common reasons for seeking care is because of pain and/or dysfunction, usually involving the teeth or periodontal tissues. However, musculoskeletal, vascular, and neuropathic causes of orofacial pain occur frequently. The need to understand pain and all of its ramifications is of utmost importance in diagnosis and case-specific, evidence-based management of conditions afflicting the masticatory system. This article reviews current concepts with regard to the multiple etiologic and/or perpetuating factors now thought to be associated with myogenous and arthrogenous orofacial pain. Important distinctions between acute and chronic pain are discussed. The rationale for consideration of multidisciplinary evaluation and management is highlighted.

Received: January 3, 2001
Accepted: February 20, 2001

The new millennium heralds an exciting time in dentistry. Technological advances coupled with a rapidly growing foundation of scientific knowledge have brought the field of dentistry to the forefront with regard to the provision of evidence-based care. Over the past several decades, the general population has gained a greater appreciation for the importance of quality oral health as a significant component of optimum overall health. The number of individuals seeking dental care on a routine basis has increased and continues to rise. However, the stark reality is that the primary reason for patients seeking care in medicine and dentistry is due to pain.

Pain is the most frequent cause of suffering and disability. It is estimated that 20% of the United States population experiences significant acute pain and 33% experiences chronic pain each year.[1] Unfortunately, health care professionals may lack essential training that would facilitate accurate recognition, detailed assessment, and effective management of many acute and chronic pain conditions. Inadequate pain relief is associated with enormous socioeconomic consequences, so much so that pain is now viewed as the fifth vital sign.[2,3] The Joint Commission on Accreditation of Health Care Organizations recently adopted standards directed at ensuring all patients the right to appropriate assessment and management of their pain.

Orofacial pain occurs frequently in the general population. A study by Lipton et al of 45,711 households revealed that 22% of the U.S. population experienced orofacial pain on more than one occasion in a six-month period.4 Certainly it comes as no surprise that the most commonly experienced orofacial pain is odontogenic in nature. Dworkin and Massoth reported that the most prevalent non-odontogenic orofacial pains are musculoskeletal in origin.[5]

Temporomandibular disorders (TMDs) comprise a broad subgroup of musculoskeletal disorders that affect the temporomandibular joint (TMJ), the muscles of mastication, and/or the associated structures.6 TMD has been identified as one of the most commonly occurring non-odontogenic pain complaints.4 Recent studies indicate the prevalence of TMD-related pain to be 12%.7 It has been reported that 10 million Americans suffer from TMD-related complaints each year.8,9 One general population study indicated that 75% percent of those evaluated exhibited at least one sign, such as joint noise or palpation tenderness, and 33% of this non-patient population exhibited at least one symptom that potentially would prompt that individual to seek evaluation and care.[10,11]

Signs and symptoms in the general population have been found to occur more frequently in females than in males, at a ratio of approximately 2:1.[11-13] However, females are three to nine times more likely to be represented in patient populations with an age range of 15-45.[10,14,15] The age-sex prevalence patterns of TMD are consistent with a possible etiological role for female reproductive hormones in these pain conditions. Several peripheral and central mechanisms through which estrogen could operate to increase pain have been postulated for TMD and other painful

conditions, including joint laxity, enhanced inflammatory responses, and actions of prostaglandin release or serotonin receptors.[16]

Primary headache (tension-type and vascular) occurs commonly and frequently is associated with TMD. Approximately 45 million Americans experience headache pain on a recurrent basis. Some of the mechanisms associated with these conditions may overlap those mentioned above. For example, the increased rates of occurrence of migraine headaches during the menstrual cycle and the adverse effects of oral contraceptives on migraine are well documented in clinical studies.[16] Other maladies that commonly are expressed in the orofacial region include trigeminal neuralgia, trauma-induced neuropathy, herpes zoster (shingles), and post-herpetic neuralgia. Autoimmune disorders such as rheumatoid arthritis, scleroderma, systemic lupus erythematosis, and psoriatic arthritis also are associated with symptoms in the head and neck region.

The diagnosis and management of TMDs and other non-odontogenic orofacial pain complaints may be one of the most challenging yet rewarding aspects of dental practice. Recent expansion of knowledge with regard to the many ramifications of the pain experience has generated an explosion of inquiry into the complex arena of pain mechanisms and pathways. This has resulted in a dramatic paradigm shift in the diagnosis and management of pain. Therefore, it is mandatory for dental professionals to develop the necessary clinical expertise and a scientific knowledge base on which they may determine case-specific diagnostic and management approaches.

The complexity of understanding orofacial pain may be related to underlying neurophysiological mechanisms such as activation of peripheral receptors, alteration of the size of receptive fields, neurotransmitter release, transmission and projection of nociceptive information, and convergence of nociceptive afferents onto common central neurons.[17] Orofacial pain can be classified into four categories – myogenous, arthrogenous, neurologic, and vascular. Mechanisms implicated in the pathogenesis in each of these subgroups involve the communication of a multitude of neurotransmitters and neuromodulators, which may play key roles in the perception of and response to pain.[18]

Diagnosis of painful conditions involving the head and neck frequently is complicated by referred pain, which is characterized by the site of the pain differing from the actual source of the pain.[19] When assessing the orofacial pain patient, it is important to remember that the site in which the patient reports the pain is based on perception and interpretation and may not truly reflect the area in which pathology is present. Several hypotheses have been proposed regarding this commonly experienced phenomenon.

Central excitatory effects resulting from ongoing peripheral nociceptive input may occur. Nociception refers to electro-chemical impulses that are transmitted from the periphery and are interpreted as pain in the upper brain. Continuous and/or recurrent nociceptive input into the central nervous system (CNS) may promote the release of neurotransmitter and vasoactive substances in the spinal trigeminal nucleus (subnucleus caudalis).[20,21] This central excitation may decrease the threshold of adjacent second order neurons that receive input from sites other than nociceptive sources. As signals on excited second order neurons are transmitted to the higher centers (thalamus, limbic system, and somatosensory cortex), nociception is interpreted as pain.[22] Nociception from deep structures (muscle, vasculature, and joint) is nontopographic in nature and often difficult for a patient to localize. When central excitation sensitizes adjacent second order neurons, the site versus source of deep tissue nociception may be even more difficult for the individual to distinguish.

This shared neurologic circuitry may make the true source of pain difficult to diagnose.[23] Confusion with regard to diagnostic and clinical decision-making is compounded by the fact that signs associated with TMD occur quite commonly in the general population. TMD also is associated with a number of signs and symptoms affecting other areas of the head and neck. Headache and ear-related symptoms are frequent complaints among TMD patients. A recent publication found that 75% of a TMD patient population also reported concomitant neck pain, 72% indicated that they were experiencing pain in areas of the head other than the masticatory region, and 72% reported back pain.[24]

The trigeminal convergence-projection theory (Fig. 1) is based on the fact that nociceptive input from virtu-

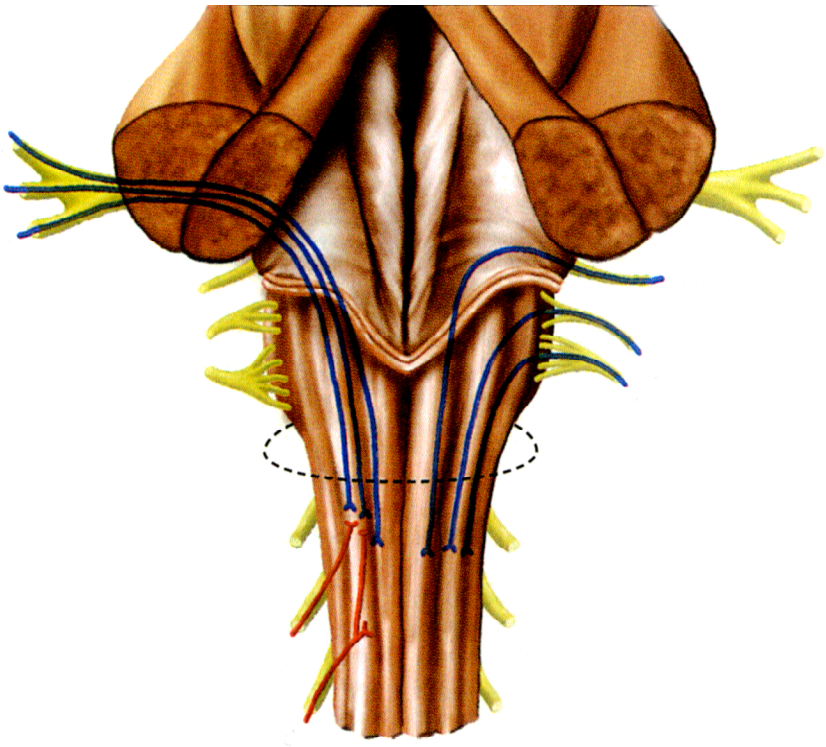

Figure 1 - Convergence of primary small fibers subserving nociception.

ally the entire head and neck (cranial nerves V, VII, IX, and X and cervical nerves 1-4) converge on the trigeminal spinal nucleus (subnucleus caudalis).[25-27] This area is where primary nociceptive nerves synapse with second order neurons. This concept suggests that it is in this area where central excitation occurs. The number of primary pain-transmitting neurons is far greater than the number of second order neurons. Therefore, sensory input from multiple regions, including the cervical region, may project onto the same second order neuron for transmission to the higher centers in the pain pathways where pain localization, interpretation, and response occur.

Currently, it is recognized that chemical changes and alterations in neurotransmitter receptor systems take place in response to pain. This may result in a decreased tolerance to pain, compromised sleep quality and/or quantity, depression, and other behavioral changes. Over the last decade, a tremendous amount of insight regarding neural plasticity in the peripheral and central nervous systems has been explained.[28,29] Neural plasticity refers to how the transmission properties of nerves are altered due to peripheral and central excitatory effects. When neural plasticity is induced, input on non-nociceptive nerves may be interpreted by the brain as pain. For example, light touch or wind blowing across hair is conveyed by proprioceptive nerves. Neuroplastic changes at primary receptors and second order neurons may allow input from such sites to be interpreted as painful.

The extent to which neuroplastic changes occur is controlled by the excitatory and inhibitory pain modulating systems. Continuous nociception may stimulate excitation and increase the patient's pain experience. However, nociception also stimulates descending pathways (serotonergic, adrenergic, and enkephlinergic), which modify the spinal trigeminal nucleus and regulate sensory input into the nervous system. Whether excitation or inhibition prevails may be largely dependent on accurate diagnosis and expeditious case-specific treatment. This may be the difference between a quick resolution of the patient's pain problem and the development of a potentially devastating chronic pain condition.

Pain is defined as an unpleasant sensory and emotional experience associated with actual or potential tissue damage or described in terms of such damage (see Table 1).[30] Acute pain serves a protective function by warning the body of imminent danger.[31] It is characterized by a sudden onset, usually recogniza-

Table 1. Differences Between Acute & Chronic Pain

	Clinical Acute Pain	Chronic Pain
Definition	Unpleasant sensation associated	Sensation that persists when the other aspects of the disease/disorder have been resolved
Duration	Limited to normal healing time required to overcome the causative mechanisms	Beyond the typical time for healing; not self-limiting
Neurophisiologic Implications	Serves as a protective mechanism	No useful purpose; may become self-perpetuating
Psychological Implications	Associated anxiety but no persisting psychological reactions	Frequently accompanied by psychological changes; appears to be permanent
Management	Respond predictably to traditional approaches	May be refractory to traditional modalities; requires a multidisciplinary approach

ble pathology, short duration, predictability of treatment outcome, and if present, reversible psychological distress.

Chronic pain may be defined as pain that persists a month beyond the usual course of an acute disease process or the reasonable time of healing for a specific injury.[22] Chronic pain is characterized by its extended duration, poorly defined pathology, poor response to treatments, and associated disability.[32] Whereas acute pain usually may be managed effectively by traditional monodisciplinary approaches to care, chronic pain typically requires a multidisciplinary approach to management involving biologic and psychologic components. Chapman and Gavrin recently postulated that severe, chronic pain is an extended and destructive stress response characterized by neuroendocrine dysregulation, fatigue, dysphoria, myalgia, and impaired mental and physical performance.[33] It has been stated that chronic pain may become self-perpetuating and may be considered a disease in itself.[34,35]

The implications of chronic pain in the United States are enormous. Recent estimates suggest that one-third of the population (97 million Americans) suffers from chronic pain.[36] It has been found that chronic pain disables more Americans each year than cancer or heart disease and that the cost of chronic pain in diagnosis management and all factors related to it are greater than both heart disease and cancer put together.[22] Lost workdays, direct and indirect health care costs, and worker's compensation benefits account for approximately $79 billion annually.[36] The impact on individuals and their families may be even more devastating.

Data indicate that the face, head, and neck are one of the most common regions of the body in which chronic pain is experienced.[37] Von Korff et al reported that the risk of developing chronic head/facial pain is 24.3% by age 50 and 33.8% by age 70.38 Importantly, McKinney et al suggested that chronic TMD pain patients with a symptom duration of more than six months are behaviorally and psychologically similar to non-TMD chronic pain patients. However, they differ in their perceptions of their disorder, rendering them less handicapped by their problems.[39] Unpublished data regarding comparison of patients in a facial pain center, spine pain center, and anesthesia pain center found no statistical differences between patients with regard to pain levels, depression, and anxiety.[40] Therefore, understanding the clinical relevancy of central excitation, trigeminal convergence, and neural plasticity may allow the practitioner to coordinate care most effectively.

Disruptive Factors

A functional homeostatic balance exists between the various components of the masticatory system, including the teeth, periodontium (hard and soft tissue-supporting structures), masticatory and cervical musculature, TMJ structures, and the psyche of each individual. This adaptive balance may be disrupted by a number of factors acting alone or in combination, resulting in the expression of signs and symptoms associated with TMD. New scientific information has provided an enhanced understanding of pathogenesis, those cellular events and reactions and other pathologic mechanisms occurring in the development and maintenance or recurrence of TMD. Slavkin has stated, "Understanding these interrelationships should improve how we promote

health, reduce disease, and enhance diagnosis and treatment." The following interrelating factors may disrupt the dynamic balance of the masticatory and orofacial region.

Gender Differences

Data indicate that there is a significant sexual disparity in the TMD patient population and, more globally, in the vast majority of human pain conditions.[41] Behavioral factors such as the more stoic nature of males, social conditioning, and care-seeking patterns have been proposed as being responsible for gender differences. Studies in which age-matched males and females were exposed to laboratory stressors have found that anxiety has an effect on both sexes. However, a significant difference in the levels of various psychophysiologic responses in males and females was revealed.[42] Females demonstrated a more robust response, exhibited by a greater decrease in pain tolerance and threshold, more disrupted self-control strategies, increased electromyographic activity of the facial musculature, and more pain behavior than the male subjects.[43] Other researchers also have identified gender differences in response to anxiety as well as laboratory-induced pain.[44-46]

Physiologic factors related to sexual structural differences have been reported. The propensity for females to exhibit a greater ease of masticatory muscle fatigue has been suggested.[47,48] This phenomenon has been attributed to gender differences in the concentration of fast twitch, easily fatiguing white fibers versus slower twitch, more endurant red fibers.[49] Fibromyalgia, a chronic skeletal muscle condition that is associated with disruption in pain modulation, commonly is found to co-exist with TMD. An increase in the number of red, ragged fibers (a pathologic fiber state) in areas approximating diagnostic tender points is reported to occur in fibromyalgia.[50,51] This finding also is noted in postexercise muscle soreness and myositis.[52] Class II skeletal subtypes (high angle cases) have been reported to demonstrate greater propensity for masticatory muscle fatigue.[53] It has been suggested that individuals with a history of systemic joint laxity or certain collagen vascular diseases are predisposed to the development of arthrogenous TMD.[54,55]

Recent data also suggest hormonal factors may largely be responsible for gender differences in the TMD patient population.[56,57] TMD appears to peak in incidence during the reproductive years, suggesting that either biologic or psychosocial factors unique to women in this period of life could increase the risk of developing and maintaining this condition.[58] Studies have linked female reproductive hormones with the occurrence of migraine in some females.[58] It has long been recognized that females demonstrate greater pain sensitivity during the menstrual cycle, at ovulation, and following menses. The relationship of estrogen and, to a lesser degree, prolactin to pain sensitivity has been explained. It has been reported that the use of estrogen supplement significantly increased the odds of having TMD.[58] Studies have shown that although functional estrogen receptors have been identified in many synovial joints of males and females in equal concentrations, a definitive difference exists in the number of estrogen receptors within the TMJ. Male TM joints have been found to have few if any estrogen receptors while female TM joints exhibited significant numbers of these receptors.[59-61]

Implications regarding hormonal variables relate to their potential to modify the adaptive capacity of the TMJ. Haskin et al reported that:[62]

- estrogen may inhibit glycosaminoglycan degradation and synthesis
- estrogen may promote degenerative changes in the TMJ by increasing the synthesis of specific cytokines (polypeptide hormones, which can evoke a variety of cellular responses)
- estradiol enhances the synthesis of interleukin-1 (IL-1) and interleukin-6 (IL-6) by peripheral blood mononuclear cells, whereas testos terone may inhibit the release of these cytokines from stimulated monocytes
- prolactin, released from the pituitary in response to estrogen, exacerbates cytokine production by lymphocytes and macrophages.

Nerve growth factor (NGF) is expressed during tissue injury. It is recognized that NGF is capable of activating nociceptive nerve endings in the peripheral as well as the central nervous system. NGF levels also have been found to be elevated in synovial fluid from joints of arthritic patients and have been associated with hyperalgesia (exaggerated pain response) in humans.

Petty et al have reported that when human skeletal muscles were injected with NGF, diffuse myalgia was induced.[63] The onset of myalgia occurred in six to nine minutes; peak pain levels were reached in four to six hours and lasted two to nine days. In some cases, the pain persisted up to seven weeks after a single injection. It was found that women were significantly more sensitive to the effects of NGF than men.

Measurement of TMJ pressure differentials in the superior joint space on patients experiencing several subtypes of internal derangement revealed tremendously elevated pressures associated with clenching.[64] These elevated pressures, which far supercede the end capillary perfusion pressure, can slow or stop the flow of vital nutrients into the TMJ, resulting in a hypoxic condition. Decreased oxygen saturation with a subsequent reperfusion, the return of flow to the area, has been reported to stimulate the production of tissue-degrading substances such as free radicals and cytokines.[65,66] A trend toward significantly greater TMJ pressures associated with clenching was found to occur more frequently in female subjects than in males.[64] This finding is interesting in light of the fact that males routinely have been found to develop greater biting forces than females.[67] Further study in this area may provide additional explanation with regard to the preponderance of female TMD patients.

Psychosocial Issues

Psychosocial factors are suggested to be related to TMD/orofacial pain. There is little doubt that some psychological factors are associated with every pain experience. However, the relationship of these psychologic factors as a cause, either direct or indirect, must be determined on a case-specific basis. Additionally, the degree of response must be assessed since psychological response in acute pain states typically is a short-lived, normal reaction. It is well recognized that anxiety, stress, negative affect, and depression may compromise physical and mental well being. Catastrophizing (thinking the worst) has been identified as a significant impediment to successful management of pain conditions. Brown et al found pain severity to be significantly related to degree of life interference and to negative affect (depression, anxiety, and anger).[68] A direct relationship between depression and both the physical and psychosocial functioning of patients with facial pain has been reported.[69] Additionally, a depressed mood is associated with a decrease in the concentration of CNS neurotransmitters norepinephrine and serotonin. Decrease in these neurotransmitters is associated with impairment of endogenous pain inhibition and disrupted sleep patterns. Anxiety and stress have been found to cause compromise in the immune system, lowering individual host resistance.

Importantly, a relationship has been suggested between a history of physical and/or sexual abuse and a range of psychological, functional, and physical factors. Abuse history has been identified as a significant feature in TMD chronic pain patient populations as contrasted to non-chronic TMD patients. Riley et al found that an abuse history was likely to increase an individual's tendency to dwell on, amplify, and over interpret somatic symptoms.[70]

The vast majority of individuals suffering from TMD/orofacial pain may be managed successfully in the private setting when evidence-based principles of diagnosis and treatment are followed. However, the complex patient may not be discerned so readily. It has been reported that when patients have both a somatic complaint and a co-existing psychological problem such as depression, the psychological distress will be overlooked in most cases.[71,72] Key features of the complex patient profile include a history of multiple failed procedures, polypharmacy, biomedical mentality, nonspecific clinical features, and a "fix me without my participation" attitude.

Nutrition & Exercise

In today's health-conscious society, the value of proper nutrition and exercise is recognized. However, many patients with TMD/orofacial pain have withdrawn from normal activities of daily living, which may compromise not only their mental well-being but also their neurophysiological well-being. Exercise on a regular basis boosts the body's natural pain defense mechanisms by enhancing the production of endogenous opioids (enkephlins, dynorphins, and endorphins).

Likewise, balanced nutrition can enhance the body's pain defense mechanisms by maximizing anti-eicosi-

noid effects and aiding in the production of antioxidants, which limit the damage caused by destructive free radicals in both joint and muscle disorders. Chronic muscle pain disorders such as myofascial pain and fibromyalgia have been associated with a decrease in serum magnesium. Magnesium deficit also is associated with an enhanced inflammatory process, enhanced free radical (superoxide) formation, an enhanced excitatory state in the CNS, and enhanced calcium mobilizing potential (abnormal calcium handling). Travell reported that B-vitamin deficiency is a common perpetuating factor of myofascial pain.[73]

Trauma

The role of various types of trauma in the etiology of TMD has been debated for many years. A study of 400 consecutive TMD clinical patients assessed the incidence of jaw injury in relation to onset of symptoms.[74] Only 24.5% of the study population could relate the onset of pain and dysfunction directly to an identifiable macrotraumatic event, primarily extension/flexion injury. Similarly, a study of patients with degenerative TMJ disease found that only 31.6% reported previous trauma to the head and/or neck.[75] These data indicate that the vast majority of patients with TMD experience a more insidious onset of their symptoms, likely related to microtrauma or a repetitive stress response. Putative microtraumatic factors include bruxing/clenching, postural dysfunction, and other habitual repetitive behaviors.

Occlusion

One of the areas of greatest debate relates to the association between occlusal factors and TMD. Although occlusion has been recognized as an important etiologic or perpetuating co-factor, the degree to which it plays a role has not been definitively delineated.

Few terms in dentistry are used in as broad a context as malocclusion. Malocclusion is defined as any deviation from acceptable contact of opposing dentitions or any deviation from normal occlusion.[76] This definition begs the question, "What is normal occlusion?" An average of the results of 14 studies regarding the prevalence of malocclusion reveals that 42% of the population displayed a Class I malocclusion, 23% exhibited a Class II malocclusion, and 4% had a Class III malocclusion.[77] Therefore, only 31% have what would be considered "normal" occlusion. One may ask whether these occlusal relationships truly are aberrant or whether this simply is a static relationship.

An association between open bite, posterior crossbite, and deep bite and the occurrence of TMD has been reported.[53] Additionally, a multiple logistic regression analysis of 11 common occlusal features in asymptomatic controls and five different TMD subgroups found that five occlusal factors demonstrated an odds-risk ratio of at least 2.[78,79] These occlusal features included anterior open bite, overjet greater than 6.0 mm, centric relation/intercuspal position slide greater than 4.0 mm, unilateral lingual crossbite, and five or more missing posterior teeth. No other occlusal schemes were found to be statistically significant.

These studies may not reveal the total story. While it may be said that the manner in which teeth fit is important, what patients do with their teeth may be more important. Dynamic occlusal function affects multiple interfaces, including tooth interface, tooth/supporting structure interface, TMJ relationship, and muscle activity (functional and parafunctional). Mechanical stresses at each of these interfaces have been shown to be associated with a compromise in the integrity of tissues. Additionally, we must consider the various case-specific factors that may affect adaptability, such as variable directions of muscular loading forces, selective action of multiple dental and articular components, the length of time that the load is imposed, the amount of load, and the individual's host resistance.

It may be more appropriate to view TMD cases where occlusal function serves as a significant factor in TMD as a maladaptive occlusion. This term takes into consideration peripheral and central sensory, motor, and autonomic nervous system factors involved in masticatory system pathofunction on a case-specific basis.

Posture

Postural imbalances have been suggested as an etiologic variable in TMD.[80] While there is little doubt that craniocervical dysfunction is common in the

TMD patient population, a cause-and-effect relationship has not been definitively established. Studies indicate the existence of a dynamic relationship between the cervical and masticatory musculature. Injection of local an esthetic into the trapezius myofascial pain trigger point not only was associated with a decrease in the electromyographic (EMG) activity in the injected muscle, but also with decreased EMG activity in the masseter muscle on the same side.[81] Results from a study of patients in motor vehicle accidents suggest that TMJ or masticatory muscle injury may be associated with various postural relationships.[82] Therefore, it appears that the complex innervation of the head and neck creates an environment in which sensory and motor systems may interact to result in musculoskeletal compromise involving the masticatory and cervical regions.

Sleep Quality & Quantity

It is estimated that one in seven Americans suffers from a diagnosable sleep disturbance.[83] Disturbed sleep has significant physiological effects and a number of psychological relationships. It is well-recognized that a number of sleep-dependent processes are necessary for health maintenance.

During the deeper, restorative stage of sleep, growth hormone is produced. Growth hormone is necessary for repair and regeneration of damaged tissues such as joint or muscle. Additionally, T-cell and lymphocyte function is enhanced by quality, restorative sleep. A compromise in the amount of the deeper stage of sleep also results in a decrease in serotonin in the CNS. The ramifications of diminished serotonin levels are widespread involving altered pain modulation and mood. Unfortunately, disturbed sleep patterns are common in TMD patient populations. Associated with these disrupted sleep cycles may be an increase in nocturnal masticatory system parafunctional activities such as clenching and bruxing. Nocturnal bruxism has been reported to be carried out at levels three to four times more forceful than the maximum voluntary force during waking hours due to the reduction in inhibitory controls while sleeping.[84] Because of the significant implications of impaired sleep and nocturnal bruxism, it is essential that the sleep history of each patient with TMD/orofacial pain be reviewed thoroughly.

Management

Management of TMD/orofacial pain must be viewed on a case-specific basis. To achieve optimum treatment outcomes, practitioners must address the specific pathophysiology. The traditional model of monodisciplinary management has proven to be effective in cases in which definitive cause-and-effect relationships can be established. However, the multifaceted nature of these conditions, combined with the associated features of recurrent and/or chronic pain, add a significant degree of complexity to management decisions. Utilization of a multidisciplinary model of diagnosis and management encourages the integration of a management plan with input from all team members. This approach would enhance outcomes by addressing physical, somatic, psychological, environmental, and behavioral factors in a well-orchestrated fashion.

The goals of management include reducing or eliminating pain, halting the disease process when possible, normalizing function, improving quality of life, and reducing the need for long-term care. Implementation of this multidisciplinary model requires the team to first arrive at a complete diagnosis encompassing all physical and psychological factors. Goals must be established regarding treatment duration, pain management approaches, patient involvement, and a plan for the patient to return to activities of daily living. Success is dependent on regular communication between the team members.

Key members of the clinical service team for multidisciplinary diagnosis and management of TMD/orofacial pain are dentist(s), physical therapist(s), clinical and health psychologist(s), and a network of consultants in various disciplines of medicine. Table 2 delineates the roles of the various key members of the team. The network of consultants typically includes pharmacy, neurology, otolaryngology/ENT, rheumatology, internal medicine, neurosurgery, and anesthesia pain disciplines.

Conclusion

The prevalence of orofacial pain mandates that greater emphasis be placed on educating all health profession students and practitioners in appropriate pain assessment and management protocols to

Table 2. Roles of Core Team Members in the Diagnosis & Management of TMD/orofacial Pain

Dentist
Evaluation/Diagnosis
Patient education
Pharmacologic management
Dental care
Occlusal orthosis therapy
Coordination of appropriate consults
Team interaction

Physical Therapist
Evaluation/Diagnosis
Education
Modalities/Techniques
Rehabilitation
Team interaction

Clinical & Health Psychologist
Evaluation/Psychometric testing
Identification of underlying & resultant psychological problems
Cognitive-behavioral therapy
Pain & stress management
Team interaction

enhance clinical decision-making skills. It is incumbent on today's health care professionals to have current, comprehensive knowledge of recognized principles of multi-modal management strategies that include well-directed psychological pain management and rational pharmacological regimen. It is clear that the multi-causal nature of TMD/orofacial pain and the number of conditions with similar signs and symptoms mandate consideration of a multidisciplinary approach to diagnosis and management. Etiologic and perpetuating factors can be elusive. Both local and systemic disorders must be considered in differential diagnosis. Importantly, we must recognize that pain transcends all health care disciplines. A multidisciplinary approach to pain management has been demonstrated to be most efficacious.[85] The January 1996 Florida Pain Management Commission report stated, "Pain sufferers noted the vast difference in the success of their treatments when a multidisciplinary approach was used."[86]

With methodologies and techniques validated by scientific inquiry, the incidence of chronic pain ultimately may be reduced. By utilizing sound biomechanical and technical skills together with a firm understanding of anatomy, physiology, neurology, and psychology, we will achieve optimum treatment outcomes and enhance the quality of life for our patients. As stated by Liebeskind and Melzack, "By any reasonable code, freedom from pain should be a basic human right limited only by our knowledge to achieve it."[87]

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