Temporomandibular Disorders: Epidemiologic and Etiologic Considerations (Part 1)

Ulises A. Guzman & Henry A. Gremillion, U.S.A.

Temporomandibular disorder is a collective term embracing a number of clinical problems that involve the masticatory musculature, the temporomandibular joint (TMJ), associated structures, or both that have many common symptoms. The term is synonymous with others frequently utilized such as myofascial pain and dysfunction syndrome, temporomandibular joint syndrome and craniomandibular disorders. Temporomandibular disorders are currently recognized as a major cause of non-dental pain in the orofacial region and are considered a subclassification of musculoskeletal disorders.1

Classic signs and symptoms associated with TMD are pain in the pre auricular region and contiguous areas to include, TM joint and masticatory and cervical muscles: limitation or deviation in mandibular movements; and TM joint sounds (clicking, popping, crepitus). The pain is usually aggravated by chewing or other jaw function.

Commonly associated co-existing/co-morbidity factors with TMD are headache, neckache, facial pain, ear and jaw ache. Non-painful masticatory muscle hypertrophy and abnormal occlusal wear resulting from oral parafunction activities such as bruxism may be related problems 1.2

A functional homeostatic balance between the various components of the masticatory system must be maintained for long-term stability. In addition, there are other contributing factors that can disrupt this dynamic balance. Anatomical, neurologic, physiologic and psychologic factors can, alone or in combination, be sufficient to disrupt this balance; thus reducing the adaptive capacity of the masticatory system with subsequent expression of symptoms of TMD.³

Epidemiology, the study of the distribution and determinants of health-related states and events in populations,4 should have a definitive application to the problem in question. Epidemiologic studies related to TMD have been primarily focused on prevalence and the associated signs and symptoms. Most of the studies are cross-sectional samples, meaning they are not necessarily representative of the general population. Therefore, their prevalence on a case-specific basis must be questioned.

Signs and symptoms of TMD are very common in the general population. They suggest that 40 to 75% of the general population have at least one sign of TMD (joint noise, tenderness, etc), 33% of the general population have at least one symptom (face pain, joint pain, etc.).⁵⁻⁸ The prevalence of signs and symptoms of TMD in childhood has been assessed but tends to be significantly lower than in adults.¹⁰⁻¹² Signs and symptoms

years suggesting that either biologic, neurological or psychological factors unique to women in this period of life could increase the risk of developing or maintaining this condition. It has been long recognized that females demonstrate a greater pain sensitivity during the menstrual cycle, at ovulation, and following menses. Functional estrogen receptors have been identified in most synovial joints of males and females in equal concentrations,

A relationship between a history of physical and/or sexual abuse and a range of psychological, functional, and physical factors has been suggested. Abuse history has been identified as a significant feature of TMD chronic pain patients populations as contrasted to non-chronic TMD patients. Research have found that an abuse history was likely to increase an individual's tendency to dwell on, amplify, and over interpret somatic symptoms.³⁹

"Historically, TM disorders have been on stage for confusion and disagreement about what constitutes proper diagnosis, treatment and management."

of TMD are more prevalent in the third or forth decade of life. 13-17 Studies related to the severity of pain between age groups have demonstrated no difference across all age groups. 18 However, the frequency of the morphologic changes and a marked continuous decrease in signs and symptoms is observed with advancing age. 19-25

Data indicate that significant gender differences in the TMD population exist. Importantly, this trend is observed in most chronic pain conditions. Factors that must be taken into consideration are behavioral factors such as the more stoic nature of males; social conditioning and care seeking behaviors have been proposed as possibly being responsible for the gender differences. Physiological factors related to hormonal influences are also reported. A natural tendency is for females to exhibit a greater potential of masticatory muscle fatigue has been suggested. This phenomenon has been attributed to a greater concentration of fast twitch, easily fatiguing white fibers versus slower twitch, endurant red fibers in the females.26-28 Recent data also suggests hormonal factors may be largely responsible for gender differences in the TMD population.

Exercise-induced fatigue must also be considered in gender differences associated with TMD. During moderate-intensity long-duration exercise, females demonstrated greater lipid utilization and less carbohydrate and protein metabolism than males, indicating differences in muscular physiology. TMD appears to peak in incidence during the reproductive

and there exists a significant difference in the number of estrogen receptors within the TM joint. Male TM joints have been found to have few, if any, estrogen receptors while female TM joints exhibited significant numbers of these receptors. 34-36

Psychosocial factors have been proposed to be related to TMD experience. The relationship of the psychological factor(s) either directly or indirectly as causative must be determined on a case-specific basis. Catastrophizing (thinking of the worst) has been identified as

The value of proper nutrition and exercise must be recognized, particularly in patients living with chronic pain where withdrawal from normal daily activities may have compromised 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, enhancing the production of endogenous opiods (enkephalins, dynorphins, endorphins). Balanced nutrition can enhance the body's pain mechanism by maximizing anti-eicosanoid effects and aiding in the production of

"The role of various types of trauma in the etiology of TMD has been debated for many years."

a significant impediment to successful management of pain conditions. Studies have demonstrated that pain severity to be significantly related to the degree of life interference and to negative affect (depression, anxiety, anger).37,38 Additionally, depressed mood is associated with a decrease in the concentration of central nervous system neurotransmitters norepinephrine and serotonin. A 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, thus lowering individual host

antioxidants, which limit the damage cause by free radicals in both joints and muscles. Certain vitamin deficiencies have been linked to enhanced inflammatory process activities and free radical formation, including magnesium and B vitamins. 40

Historically, TM disorders have been on stage for confusion and disagreement about what constitutes proper diagnosis, treatment and management. The literature has demonstrated that most of the management and treatment of TM disorders has been based on belief systems and testimonials. Why so much controversy? The controversy may exists because of the limited

knowledge regarding the etiology and the natural history of the course of TMD. Probably the difficulty is in establishing clinically, a significant direct cause/effect relationship because of the many variables involved that probably are too difficult, if not impossible, to exclude.

Many early theories emphasized dental morphological factors of malocclusion, occlusal disharmony, and bad mandibular alignment as being primarily responsible for the development of TMD symptoms. Based on the definition, evaluation and analysis of occlusion are important aspects in the diagnosis and treatment of TMD. The question is, Is occlusion the most important factor? Little evidence is available to strongly implicate occlusion in the etiology of TMD.43-46

Several studies have demonstrated that the presence of predisposing factors such as structural, metabolic, and/or psychological conditions could be sufficient to increase the risk of developing TMD related problems if they are affecting the masticatory system in a negative way. It has been reported than an extreme anterior open bite, overjet greater than 6 to 7 mm, discrepancy between the retruded contact position and the intercuspal position greater than 4 mm, five or more missing posterior teeth, and unilateral maxillary posterior lingual crossbite in children may be associated with TMD.41-46

Some contributing etiologic factors are only risk factors, others are causal in nature, and others result from, or are purely coincidental to the problem. These factors are classified as predisposing, initiating (precipitating), and perpetuating (factors that interfere with the healing or enhance the progression) of a disease process.⁴⁷ The contribution of specific occlusal factors to the multifactorial etiology of TMD have been demonstrated in studies to be only 10 to 25%.48 Associated existing factors should be considered and not assume a direct cause effect due to their presence. The dentist must consider each of these potential contributing factors on a casespecific basis.

The role of various types of trauma in the etiology of TMD has been debated for many years. Trauma is described as any force applied to the mastication structures that exceeds that of normal

DENTAL TRIBUNE Asia Pacific Edition

Trends & Applications

functional loading. Factors such as intensity and duration must be considered. Most trauma can be divided into three types: direct trauma (the result of a sudden and usually isolated blow to the structures), indirect (sudden blow without direct contact), micro-trauma (the result of prolonged, repeated forces over time due to parafunctional habits or adverse loading through postural imbalances).

Forces leading to structural failure, loss of function may follow. Stretching, twisting, or compressing forces during eating, yawning, yelling, or prolonged mouth opening have also been reported to trigger or aggravate TMD.^{49,50} The results of several studies indicate that the majority of TMD patients experience a more gradual and mostly unperceived onset of their symptoms, likely related to micro-trauma or a repetitive stress response. Micro traumatic factors include bruxing, clenching, postural dysfunction, and any other habitual repetitive behaviors. Experimentally induced parafunction has shown to cause pain similar to that reported by those with TMD.53,54

The importance of sleep has been underestimated by the majority of the population, 63% of American adults do not obtain the recommended amount of daily sleep. Sleep is a basic human need, and must be considered as important as diet and exercise. Getting the right amount of sleep is vital, but just as important is the quality of our sleep. Sleep disturbances have been reported in many epidemiological studies in persons experiencing not only acute but also chronic pain. It is estimated than one in seven Americans suffers from some kind of sleep disturbance. Studies have shown that disturbed sleep has significant physiological effects and a number of psychological relationships have been demonstrated.53-56

Sleep bruxism is reported by 8% of the adult population and is mainly associated with rhythmic masticatory muscle activity, characterized by repetitive muscle contractions primarily during the REM stage of sleep. The reduction in the inhibitory controls while sleeping makes forces during nocturnal bruxism 3 to 4 times greater than during waking hours, forces that potentially exceed the normal capacity of the system.55 Sleep bruxism may eventually lead to many signs and symptoms of dysfunction, including pain, and/or structural changes in the masticatory system.

It has been demonstrated that the metabolic activity of the brain significantly decreases after 24 hours of sustained wakefulness. A number of sleep dependent activities have been recognized, leading us to a better understanding of how sleep deprivation or interruption may result in a decrease in body temperature, a decrease in immune system function (T-cells and lymphocyte function), a decrease in the release of growth hormone (necessary for repair and regen-

eration of damaged tissues) and a reduction in serotonin (neurotransmitters involved in pain modulation and mood) in the central nervous system. A thorough sleep history must be obtain in all orofacial pain patients due to the significant implications of disrupted sleep and nocturnal bruxism.

Acceleration-deceleration injury (whiplash) with no direct blow to the face can cause symptoms consistent with TMD. However, a direct causal relationship between jaw symptoms and indirect trauma has yet to be established. 57,58,59 Studies have failed to demonstrate a jaw movement to cause mandibular strain in a flexion-extension type of injury.

The TMD examination requires a comprehensive approach understanding all potential factors. The physical examination should consist of a review of systems including not only a patient's actual chief concern(s), but also the chronological history, history of present illness(es), medical history, dental history, and personal history (social, family), general inspection of the head, neck and cervical spine, neurovascular evaluation, comprehensive orthopedic evaluation of the TM joints, evaluation and palpation of the masticatory and cervical muscles, gross screening of the cranial nerves, and intraoral evaluation of hard and soft tissues including occlusal analysis.

Basic assessment of all TMD patients should include behavioral and psychological screening by the dentist during the history taking process. The history should include questions to evaluate behavioral, social, emotional, and cognitive factors that may initiate, sustain or result from the patient's condition. Consideration to relevant factors such as oral habits, signs of depression, anxiety, stressful life events, lifestyle, secondary gain, and overuse of health care should also be given. Imaging of the TM joint and orofacial structures may be necessary to rule out structural disorders, and must be prescribed primarily when the clinical examination suggests some form of disorder.60

Heretofore clinical practice in the area of TMD has been based on anecdotal reporting. Individual and group interpretation of the limited scientific evidence has led to a marked variation in the philosophy of practice in this complex area. Empiricism and rationalism has at times resulted in disregard for the valid scientific evidence-base that does exist. With the recent explosion of knowledge regarding pain mechanisms and pathways, the effect of pain on quality of life, and an enhanced appreciation for the multifactorial nature of TMD, today's dentist can better apply science to the art of practicing evidencebased dentistry. Evidence-based dentistry is the conscientious, explicit and judicious use of current best evidence in making decisions about the care of each patient. "The purpose of using the evidence-based approach is to close the gap between what is

known and what is practiced and to improve patient care based upon informed decision making". 61 Albert Einstein said, "Science without religion is lame, religion without science is blind".

Literature

- 1. Okeson JP. Bell's Orofacial Pains, 5th ed.Chicago: Quintessence, 1995:123–133.
- Bell WE. Temporomandibular Disorders. Classification, Diagnosis, Management, 3rd ed. Chicago: Year Book, 1990:166–176.
- 3. Parker MH. A dynamic model of etiology in temporomandibular disorders. J Am Dent Assoc 1990; 120:283–289.
- 4. Taber's Cyclopedic Medical Dictionary, Ed 20:2001:722.
- 5. Solberg WK. Epidemiology, Incidence and Prevalence of Temporomandibular Disorders: A Review in: The President's Conference on the Examination, Diagnosis and Management of the Temporomandibular Disorders. Chicago: American Dental Association, 1983:30-39.
- 6. Rugh JD, Solberg WK. Oral health status in the United States. Temporomandibular disorders. J Dent Educ 1985;49:398-404.
- 7. Schiffman E, Fricton JR. Epidemiology of TMJ and craniofacial pain. In: Fricton JR, Kroening RJ, Hathaway KM (eds). TMJ and Craniofacial Pain: Diagnosis and Management. St Louis: Ishiaku Euro American, 1988:1–10.
- 8. De Kanter RJAM, Truin GJ, Burgersdijk RCW, et al. Prevalence in the Dutch adult population and a meta-analysis of signs and symptoms of temporomandibular disorders. J Dent Res 1993;72: 1509–1518.
- 9. Dworkin SF, Huggins KH, Le Resche L, et al. Epidemiology of signs and symptoms in temporomandibular disorders: Clinical signs in cases and control. J Am Dent Assoc 1990;120:273–281.
- 10. Keeling SD, McGorray S, Wheeler TT, et al. Risk factors associated with Temporomandibular joint sounds in children 6 to12 years of age. Am J Orthod Dentofac Orthop 1994;105:279–287.
- 11. Verdonck A, Takada K, Kitai N, Kuriama R, et al. The prevalence of cardinal TMJ dysfunction symptoms and its relationship to occlusal factors in Japanese female adolescents. J Oral Rehabil 1994;21:687-697.
- 12. Motegi E, Miyasaki H, Oguka I. An orthodontic study of temporomandibular disorders. Part I: Epidemiological research in Japanese 6-18 year olds. Angle Orthod 1992;62:249–256.
- 13. Egermark-Ericksson I, Carlsson GE, Magnusson T. A long-term epidemiologic study of the relationship between occlusal factors and mandibular dysfunction in children and adolescents. J Dent Res 1987;67:67–71.
- Agerberg G, Bergenholz A. Craniomandibular disorders in adult populations of West Bothnia, Sweden. Acta Odontol Scand 1989;47: 129–140.
- 15. Salonen L, Hellden L. Prevalence of signs and symptoms of dysfunction in the masticatory system: An epidemiologic study in an adult Swedish population. J Craniomandib Disord Facial Oral Pain 1990;4:241–250.
- 16. Nilner M, Kopp S. Distribution by age and sex of functional disturbances and diseases of the stomatognathic system in 7-18 year olds. Swed Dent J 1983;7:191-198.
- 17. Magnuson T, Egermak-Ericksson I, Carlsson GE. Five year longitudinal study of signs and symptoms of mandibular dysfunction in adolescents. J Craniomand Pract 1986;4:338–344.
- 18. Levitt SR, McKinney MW. Validating the TMJ scale in a national sample of 10,000 patients: Demographic and epidemiologic char-

- acteristics. J Orofacial Pain 1994;
- 19. Widmalm SE, Westesson P-L, Kim I-K, et al. Temporomandibular joint pathology related to age, sex, and dentition in autopsy material. Oral Surg Oral Med Oral Pathol 1994;78:416–425.
- 20. Peirera FJ Jr, Lundh H, Westesson P-L. Morphologic changes in the temporomandibular joint in different age groups: An autopsy investigation. Oral Surg Oral Med Oral Pathol 1994;78:279–287.
- 21. Howard JA. Temporomandibular joint disorders, facial pain and dental problems of performing artists. In: Sataloff R, Brandfonbrener A, Lederman R (eds). Textbook of Performing Arts Medicine. New York: Raven,1991: 111–169.
- 22. Koidis PT, Zarifi A, Grigoriadou E, et al. Effect of age and sex on craniomandibular disorders. J Prosthet Dent 1993;69:93-101.
- 23. Lipton JA, Ship JA, Larach-Robinson D. Estimated prevalence and distribution of reported orofacial pain in the United States. J Am Dent Assoc 1993;125:125-135.
- 24. Osterberg T, Carlsson GE, Wedel A, et al. A cross-sectional and longitudinal study of craniomandibular dysfunction in an elderly population. J Craniomandib Disord Facial Oral Pain 1992; 6:237–246.
- 25. Greene CS. Temporomandibular disorders in the geriatric population. J Prosthet Dent 1994;72: 507-509.
- 26. Clark GT, Beemsterboer PL, Jacobson IL. The effect of sustained sub maximal clenching on maximum voluntary bite force in Myofascial pain dysfunction patients. J Oral Rehab 1984; 11:387.
- 27. Clark GT, Carter MC. Electromyographic study of human and recovery at various isometric force levels. Arch Oral Biol 1985;30:563.
- 28. Miller A. Mandibular muscle pain and craniomandibular muscle function. In: Craniomandibular muscles: their Role In Function and Form. Boca Raton: CRC Press 1991:181-206.
- 29. Miller AE, MacDougall JD, Tarnopolsky MA, Sale DG Gender differences in strength and muscle fibers caracteristics. Eur J Appl Physiol Occup Physiol. 1993;66(3):254–62.
- 30. Tarnopolsky LJ, MacDougall JD, Atkinson SA, Tarnopolsky MA,

- Sutton JR. Gender differences in substrate for endurance exercise. J Appl Physiol. 1990;68(1):302–8.
- 31. Tarnopolsky MA, Saris WH. Evaluation of gender differences in physiology: an introduction. Lippincott Williams & Wilkins. 2001;1363–1950.
- 32. Anderson HI, Ejlertsson G, Leden I, et al. Chronic pain in a geographically defined general population: studies in differences in age, gender, social class, and pain localization. Clin J Pain 1993; 9:174–82.
- 33. Meisler JG. Chronic pain conditions in women. J Womens Health 1999;8(3):313–20.
- 34. Milam SB, Aufdemorte TB, Sheridan PJ, et al. Sexual dimorphism in the distribution of estrogen receptors in the temporomandibular joint complex of the baboon. Oral Surgery Oral Med Oral Path1987:64:527–32.
- 35. Aufdemorte TB, Van SI, Dolwick MF, et al. Estrogen receptors in the temporomandibular joint of the baboon (Papio cynocephalus): an autoradiographic study. Oral Surg Oral Med Oral Path 1986; 61:307–14.
- 36. Abubaker AO, Raslan WF, Sotereanos GC. Estrogen and Progesterone receptors in the temporomandibular joint discs of the symptomatic and asymptomatic persons: a preliminary study. J Oral Maxillofac Surg 1993;51: 1096-1100.
- 37. Brown FF, Robinson ME, Riley JL, Gremillion HA. Pain severity, negative affect, and microstressors as predictors of life interference in TMD patients. J Craniomandib Pract 1996;14(1):63–70.
- 38. Holzberg AD, Robinson ME, Geisser ME, Gremillion HA. The effects of depression on psychosocial and physical functioning. Clin J Pain 1996;12(2):118–125.
- 39. Riley JL, Robinson ME, Kvaal SA, Gremillion HA. Effects of physical and sexual abuse in facial pain: direct or mediated? J Craniomandib Pract 1998;16(4):259-66.
- 40. Travell JG, Simmons DG, Myofascial Pain and Dysfunction: The trigger point manual. Vol 1, Baltimore: Williams and Wilkins 1983:115.
- 41. Pullinger AG, Seligman DA, Gornbein JA. A multiple logistic regression analysis of the risk and relative odds of temporo-

→ DT page 10

Medesy is the result of know-how spread over 6 centuries of the Maniago smith's art, from the Renaissance to today and the expression of a culture of ingenious and industrious craftsmen, dedicated to their profession as if it were an art.

Make a test!
www.medesy.it

HIGH QUALITY SURGICAL INSTRUMENTS

10 Trends & Applications

DENTAL TRIBUNE Asia Pacific Edition

←DT page 9

mandibular disorders as a function of common occlusal features. J Dent Res 1993;72:968–979.

- 42. McNamara JA, Seligman DA, Okeson JP. Occlusion, orthodontic treatment, and temporomandibular disorders: A review. J Orofacial Pain 1995;9:73–90.
- 43. Tanne K, Tanaka E, Sakuda M. Association between malocclusion and temporomandibular disorders in orthodontic patients before treatment. J Orofacial Pain 1993;7:156–162.
- 44. Gesch D, Bernhardt O, Kirbschus A. The association of malocclusion and functional occlusion with temporomandibular disorders (TMD) in adults: A systematic review of population based studies. Quintessence Int. 2004;35(3): 211-21
- 45. Gremillion HA. TMD and Maladaptative Occlusion: Does a Link Exist? The J Craniomandib Pract 1995;13(4):205–206.
- 46. Gesch D et.al. Association of Malocclusion and Functional Occlusion with Signs of Temporomandibular Disorders in Adults: Results of the Population-based
- study of Health in Pomerania. Angle Orthodontist 2004;74(4)
- 47. Fricton JR, Kroening RJ, Hathaway KM (eds). TM Disorders and Craniofacial pain: Diagnosis and Management. St Louis: Ishiaku Euro America 1988.
- 48. Pullinger AG, Seligman DA, Gornbein JA. A multiple logistic regression analysis of the risk and relative odds of temporomandibular disorders as a function of common occlusal features. J Dent Res 1993;72:968–979.
- 49. Parker MW. A dynamic model of etiology in temporomandibular
- disorders. J Am Dent Assoc 1990:120:283–289.
- Harkins SJ, Marteney JL. Extensive Trauma: A significant precipitating factor in temporomandibular dysfunction. J Prosthet Dent 1985;271–272.
- 51. Christensen L. Some effects of experimental hyperactivity of the mandibular locomotor system in man. J Oral Rehabil 1975;2:169–178.
- 52. Scott DS, Lundeen TF. Myofascial Pain involving the masticatory muscles: An experimental model. Pain 1990;8:207.
- 53. Roehrs T, Roth T. Sleep and Pain: Interaction of Two Vital Func-
- tions. Seminars in Neurology 2005;25(1):106-116.
- 54. Lavigne GJ, Kato T, Kolta A, Sessle B.J. Neurobiological Mechanisms Involved in Sleep Bruxism 2003; 14(1):30–46.
- 55. Ware JC, Rugh JD. Destructive Bruxism: Sleep stage relationship Sleep 1988-172-181
- ship. Sleep 1988;172–181.
 56. Bonnet MH. Sleep deprivation.
 In: Principles and practice of sleep medicine. Philadelphia:
 W.B. Saunders, 2000:53–71.
- 57. Burgess JA, Kolbinson DA, Lee PT, et al. Motor vehicles accidents and TMD's: assessing the relationship. J Am Dent Assoc 1996; 127(12):1767-72.
- 58. Goldberg HL. Trauma and the improbable anterior displacement. J Craniomandib Disord Facial Oral Pain 1990;4:131–134.
- 59. Kronn E. The incidence of TMJ dysfunction in patients who have suffered a cervical whiplash injury following a traffic accident. J Orofacial Pain 1993;7:209-213.
- 60. Howard JA. Imaging techniques for the diagnosis and prognosis of TMD. J Calif Dent Assoc 1990; 18:61–71.
- 61. Turpin DL. Consensus builds for evidence-based methods. Am J Orthod Dentofacial Pain Orthop 2004; 24:30–1.

Risk management publications



Focus on what matters most

Hot tips for problem free practice

Enhance your knowledge



www.ingentaconnect.com/content/dps

Titles available for download

- 36 clinical risk
 management modules
- 12 ethical modules
- Publications on team working, communications, patient management, handling complaints and much more

Contact Info

Ulises A. Guzman, D.D.S., FAGD

D.D.S., FAGD
Dr. Ulises A. Guzman is a Fellow in the Craniofacial Pain program in the Parker E. Ma-

low in the Craniofacial Pain program in the Parker E. Mahan Facial Pain Center at the University of Florida College of Dentistry. He graduated from Marquette University School of Dentistry. He served as a Captain in the United States Air Force Dental Corps. He maintained a private general dental practice in Cooper City, Florida for 12 years where his clinical interests included restorative dentistry, orthodontics, and temporomandibular disorders. You may contact him at: uguzman@dental.ufl.edu.

Henry A. Gremillion, D.D.S., MAGD

Dr. Henry A. Gremillion is a Professor in the Department of Orthodontics at the University of Florida College of Dentistry. He holds an affiliate appointment in the Department of Prosthodontics at UFCD. He is the Director of the Parker E. Mahan Facial Pain Center and directs a Fellowship in Craniofacial Pain program. He has expertise in the diagnosis and management of orofacial pain. He has authored or co-authored numerous scientific articles, abstracts, and book chapters. He lectures internationally in the field of temporomandibular disorders and orofacial pain. You may contact him at:

Parker E. Mahan Facial
Pain Center
P.O. Box 100437
University of Florida College
of Dentistry
Gainesville, FL 32610-0437
E-mail:
hgremillion@dental.ufl.edu

