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Tutorial

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Sleep Cycles, TMD, Fibromyalgia, and Their Relationship to Orofacial Myofunctional Disorders

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Abstract

Poor quality sleep is caused by many factors including orofacial myology disorders. TMJ and fibromyalgia patients demonstrate a variety of similar symptoms making diagnosis difficult. A team approach utilizing appropriate referrals is critical to successful patient treatment.
Key Words: TMJ; Fibromyalgia; Orofacial Myology; Sleep Disorders; Sleep Cycles

Sleep, an essential component to health and well being, is rarely discussed in the orofacial myofunctional literature. However, quality of sleep has the potential to influence the health status of orofacial myology patients. Poor quality sleep, or non-restorative sleep, is often indicative of an underlying problem in need of resolution. As health-behavioral practitioners, orofacial myologists are in a unique situation of detecting any suspicious or suspect sleep abnormalities through thorough screening. The purpose of this article is to present information on sleep and its relevance to orofacial myology.

Sleep Cycles

There are two types of sleep which comprise a night's rest: slow wave sleep (SWS), and parodoxal/dreaming sleep, also called rapid eye movement sleep (REM). SWS is composed of four definitive stages of sleep. Various degrees of muscular activity, ease of wakefulness, and relative depth of sleep characterize each stage. REM sleep is also characterized as the bridge between Stage 1 and 2 of SWS sleep. A specific pattern of increasing Stage 1 and Stage 2 sleep, and decreasing Stage 3 and Stage 4 sleep (delta-deep), occurs in very specific cycles and levels of frequency in individuals who experience normal, complete sleep from the initial onset of the sleep cycle to the awakening process (Circadian.com, 1999; Chuder,1999).

When a person lies down, closes his eyes and becomes drowsy, it is referred to as the alpha state. People spend only five minutes in alpha before drifting into Stage 1 sleep. During Stage 1, or semiconscious sleep, the muscles relax, brain waves slow further, and the pulse slows. This stage lasts only a few minutes. Next Stage 2, is a light Stage of sleep, when the brain waves slow even further. This stage lasts 15-30 minutes in the non-sleep-deprived person.

During the deepest stages of sleep, Stages 3 and 4, brain waves slow way down, (delta waves). It is believed that during these sleep stages the body carries out its reparative and restorative work. These stages normally last a total of 30 to 40 minutes in the first sleep cycle, gradually decreasing as the night progresses. Once Stage 4 is reached the person descends gradually back to Stage 3, then 2, then 1; ultimately arriving at REM sleep-where dreaming occurs. In REM sleep, brain waves speed to the same pattern typically present during wakefulness, and the eyes move rapidly. It is in REM that the body is at its most muscularly relaxed state, and its most active brain-wave state. In fact, EEG's taken in this stage of sleep reveal brain activity similar to that encountered during wakefulness. However, during REM sleep the brain is active, and the body is motionless. If this did not occur, people would be acting out their dreams. REM sleep lasts from a few minutes to an hour. This sequence continues in a healthy individual, for four or five cycles of REM and NON-REM sleep. Each cycle lasts approximately 90 to 100 minutes. In essence, we rest our brains in NON-REM sleep, and rest our bodies in REM sleep (Circadian.com,1999; Chuder, 1999)

In Stage 4 SWS, the body produces and regulates various biochemicals essential to an individual's well being. Cortisol, noriepinephrine, and four chemicals in particular, Substance P, serotonin, dopamine, and growth hormone regulate various pain mediators within the body. The presence of poor quality sleep will influence the production and level of these specific and essential hormonal mediators within the body (Bennett, "Advanced Fibromyalgia," 1996).

Typical sleep disruption can occur as the result of central nervous system (CNS) disorders, stress, temporomandibular disorders (TMD), sleep apnea, snoring, enlarged tonsils and adenoids, illness and injury, or excessive caffeine, nicotine, or alcohol consumption. Alcohol and other central nervous system depressants decrease the time we spend in Stages 3 and 4 of the sleep cycle. (Institute for Natural Resources, 1997)

Sleep Cycles and Their Relationship to Orofacial Myology

It is in Stage 1 sleep where therapists attempt to teach the unconscious swallow and habituate night rest posture. During Stage 1, the subconscious mind is most receptive to suggestion. A variety of tapes and exercises addressing these issues are readily available. In this subconscious training, the patient is making an effort to dwell on thoughts of correct mouth and rest posture, or is listening to an audio tape repeating various related suggestions as he progressively drifts off to sleep (Peachey, 1993; Pierce, 1993; Zickefoose, 1995).

Children with adenoid and tonsil problems, and adults who snore and experience sleep apnea, do not sleep in these deeper stages of sleep. They may be affected by the dysregulation of these hormonal mediators, experiencing chronic fatigue, and lowered immune response. They may also experience an increased incidence of aches and pains. It is reported in the literature that many children with sleep apnea due to mouthbreathing and tonsil issues are "small in stature". This may be the result of altered levels of growth hormone in these children due to decreased time spent in sleep Stage 4, where this essential hormone is produced (Biegert, 1999).

Orofacial myology patients with TMD often report that they awaken themselves from sleep momentarily. They grind and brux, initiating the pain cycle, and triggering wakefulness. This behavior has the potential of altering the normal transition from the lighter stages to the deeper stages of sleep. In fact, Kampe et al (1997) found 72% of long-standing bruxing subjects presented with complaints of sleep disorders. He indicated that there was a significant correlation between frequent clenching and back, neck, head, shoulder pain, and sleep disorders. In addition, high scores on the clinical dysfunction index were noted. He feels more studies are warranted in this relatively unexplored field of odontology, and that sleep laboratory investigations would be beneficial, as well.

To better understand this mechanism, in Stages 1 and 2 of the sleep cycle, people are the most active muscularly. As they slip into the deeper stages, they experience less muscular activity. In REM., they literally become paralyzed, exhibiting muscular activity limited to the breathing cycle (Chuder, 1999). One could easily conclude that bruxers brux in the lighter stages of sleep, when muscular activities are at their peak. They may have difficulty arriving at or easing themselves into the deeper stages of sleep consistently. The cycles of sleep are defined by specific amounts of time in which individuals advance from one stage to the next. It is entirely possible that when sleep cycle disruption occurs in the TMD patient (i.e. a bruxer awakens after a few minutes in Stage 2 sleep), they must start the entire cycle over again. Therefore, they spend more time in lighter sleep, repeating the cycle often. They do not maintain Stage 2 sleep long enough to attain the deeper stages of sleep adequately throughout the course of the night. My personal theory is that as the TMD patient arrives at the natural point at which they begin to drift off to deeper sleep, the bruxing occurs, or pain ensues. This awakens them, thus preventing them from fully entering deeper sleep consistently. This may be why many TMD patients feel drained physically and emotionally. Again, Kampe et al (1997) states that more research would be beneficial in this area. If this theory is correct, TMD patients are truly lacking productive sleep. They are further perpetuating their pain, as they lack the deep sleep essential to the production of the body's own natural pain regulators.

In contrast, the child with a retained digit sucking habit has the opposite problem. According to Van Norman (1999) children with nocturnal digit sucking behavior spend more time in the deeper stages of sleep. As they enter the cycle of Stage 2, they actively suck, and this propels them into Stages 3 and 4. They spend an increased amount of their sleep time in the deeper stages of sleep. This is why many children who are thumbsuckers are also bedwetters. Deep sleep alters muscular reactions. They are sleeping so soundly they do not awaken to get up to go to the bathroom. It is also to important to note that during the REM

portion of the sleep cycle, the thumb and mouth are loose and limp as muscular inactivity predominates. This behavior is often confirmed by parents. Parents report that they have personally observed their child with a thumb in their mouth, in the absence of sucking (i.e., the thumb is near the mouth or gently within it). At this stage, the thumb or finger can be easily removed or falls out limply. In Stage 2 sleep, muscular sucking activity intensifies. The suction may be firm, and definitive muscular activity is observable. Parents who attempt to remove the thumb from their child's mouth at this point in the sleep cycle encounter firm resistance. This confirms the tendency of thumbsuckers to actively suck in the lighter stages of sleep to achieve deep sleep. Once they arrive at that ultimate state of muscular abandon, REM, they will discontinue their active sucking behavior until their next inevitable round in the sleep cycle occurs.

In addition, serotonin is produced during the deepest stages of sleep. Serotonin is a highly addictive substance, which binds to opiate receptors in the brain (Institute for Natural Resources, 1997). It is craved by children with a thumb habit and interestingly enough, contributes to the addictive nature of thumbsucking. When the thumbsucking child is actively sucking in an effort to descend to the deeper stages of the sleep cycle, they ultimately increase the amount of time they spend in that very portion of sleep which specifically and coincidentally produces elevated levels of serotonin. If this is the case, one can easily observe a link between additional time spent in the deeper stages of sleep, and elevated levels of serotonin contributing to the child with a thumb habit becoming addicted to the relaxed and pleasurable feelings thumbsucking appears to encourage. These relaxed and pleasurable sensations are easily seen in an infant who cries, slips a convenient thumb into their mouth, and drifts readily off to sleep. This same link can also be postulated in the child who may be emotionally upset and utilizes the thumb to "calm" down their emotions.

One may conclude that if a child is attempting to overcome their thumb habit, their sleep and thus body chemistry, will be altered as a result. No longer is the thumb used to assist them in transitioning to and increasing time spent in Stages 3 and 4 sleep. Instead, awakening can occur in light sleep, as these children no longer have the thumb to assist in their descent into deep sleep. They must learn to do this naturally, in the absence of the thumb (Van Norman, 1999). In my own experience, I have found that keeping a child physically active during the day in the course of thumbsucking cessation therapy is beneficial to overcoming their habit biochemically. During exercise and activity, serotonin is produced in increased amounts (Fibromyalgia Network, 1997). It is my personal observation that this physiologic "craving" for serotonin decreases as other activities besides sucking produce this addictive substance the child or teen thumb-sucker craves. This appears to help ease the transition from habit to cessation in many of my patients. Appropriate levels of physical activity also help one to spend more time in these deeper stages of sleep (Fibromyalgia Network, 1997).

Brief Overview of Fibromyalgia Syndrome

Fibromyalgia syndrome is described by Dr. Wesley Shankland II (1998), Director of the TMJ and Facial Pain Center of Columbus, Ohio as a syndrome exhibiting general muscle soreness or stiffness lasting longer than 3 months duration, characterized by poor sleep, morning fatigue, tenderness in 11 to 18 specific points of the body, and normal blood tests.

Additional symptoms common to fibromyalgia include: tension or migraine headaches (50% of all fibromyalgia patients report this symptom, according to the Fibromyalgia Network, 1997), irritable bowel, cognitive or memory impairment, post-exertional malaise and muscle pain, morning stiffness, numbness and tingling sensations, dizziness or lightheadedness, and skin and chemical sensitivities (The Fibromyalgia Network, 1998a). According to Starlanyl and Copeland (1996) "Fibromyalgia syndrome is a sensitivity-amplification syndrome. Individuals with fibromyalgia can be sensitive to smells, sounds, lights, odors, pressure, and temperature fluctuations and vibrations. Fibromyalgia sensitizes nerve endings as well as the rest of the autonomic nervous system, which means that the ends of the nerve receptors may have changed shape. Because of this, a patient with fibromyalgia might interpret touch, light, or sound as pain".

A proper screening to assess the TMD patient for these symptoms is crucial to comprehensive care and success in therapy. If a patient walks

into the office complaining of TMD discomfort, fatigue, headaches, itchiness, excitability when the phone rings, needing to wear a sweater when it is 70 degrees out, numbness or tingling in the hands, a poor memory, soreness in the lower as well as the upper extremities, and frequent trips to the restroom, the orofacial myologist needs to start asking more questions, and/or pick up the telephone and relay these findings to their primary dental or medical referral source!

The Fibromyalgia Network article "Fibromyalgia Basics-Symptoms, Treatment, and Research" (1997) states that it has been postulated that some "triggering event" does not cause fibromyalgia, but rather awakens an underlying physiologic abnormality already present in the form of genetic predisposition. In the official publication of the Arthritis Foundation, author Mary Anne Dunkin (1997) relates, "a few researchers have linked fibromyalgia to genetic markers. But both those markers have been different in different studies. In one of the most recent genetic studies... Dr. Muhammed Yunus, (noted fibromyalgia researcher), found a linkage between HLA's (human leukocyte antigens) in 40 families in which more than one member had fibromyalgia. Another study found an association with another genetic marker called HLA-DR4."

It should also be noted that Evengard (1998) concluded that chronic fatigue syndrome (CFS) and fibromyalgia are different disorders although both exhibit overlapping symptomatology. This conclusion was based on the fact that Substance P levels in the cerebrospinal fluid in patients with fibromyalgia were elevated, but patients diagnosed with chronic fatigue syndrome did not exhibit elevated Substance P levels. Elevated Substance P levels are associated with an increase in reported pain, and are regulated via depletion in deep sleep. This further reinforces the theoretical relationship of a CNS and hypothalamus/pituitary axis (HPA) link to fibromyalgia.

There is no "cure" for fibromyalgia, nor no "magic bullet". Patients must learn to manage their pain, and a combination of therapies and self-help techniques utilizing an interdisciplinary approach appear to have the most favorable outcome (Fibromyalgia Network, 1997).

Sleep Cycles and Hormonal Regulation-The TMD-Fibromyalgia Connection

As mentioned earlier, lack of restorative sleep has a profound impact on hormonal regulation, particularly for serotonin, Substance P, cortisol, noriepinephrine, dopamine, and growth hormone. Pain regulation, which occurs in deep sleep, is achieved mainly by the production of serotonin, growth hormone, and the depletion of Substance P (Dunkin, 1997; Fibromyalgia Network, 1997). Thus, the delicate balance of these hormones can be severely disrupted by altered sleep.

In patients with fibromyalgia, some dysregulation of the CNS is now believed to be present. This may be a factor in the ensuing lack of deep sleep (Mountz, 1998). Russell (1998) concurs, relating that the most widely accepted model for the pathogenesis of fibromyalgia now invokes CNS mechanisms, rather than pathologically painful muscles. He confirms that levels of platelet serotonin and Substance P appear to be abnormal in directions that could logically amplify pain perception.

Patients with altered sleep appear to spend most of their time in the lighter stages of sleep where the muscles are active and hormonal regulation is not addressed. By focusing on these sleep anomalies, and through an appropriate level of aerobic exercise which regulates the ratios of these same hormonal mediators, patients can greatly improve their pain and fatigue levels. Exercise depletes Substance P, increases the production of serotonin and growth hormone, and is coincidentally the same biochemical event which occurs during Stage 4 sleep (Fibromyalgia Network, 1997). Likewise, TMD patients can have a similar improvement with similar efforts (Pettengill, 1997).

What is the relationship between TMD and fibromyalgia? If the patient experiences TMD and fibromyalgia concurrently, treating only the TMD and not referring for rheumatological assessment to address the fibromyalgia syndrome may yield limited success. The comprehensive treatment plan for these patients should address both TMD and fibromyalgia. Interestingly, statistics show as high as 80-90% of all fibromyalgia patients have concurrent symptomatology of TMD according to an article written in 1997 for the Fibromyalgia Network a

non-profit group dedicated to research and education of fibromyalgia for patients and health care professionals. Thus, it stands to reason that as orofacial myologists, discerning what is truly TMD and what is truly fibromyalgia will help these patients immensely, because proper referrals can be instituted.

Dr. Herbert Gordon, D.D.S. (1995), a noted TMJ specialist states that a "major reason for the high prevalence of TMD in fibromyalgia and CFS could be the association of the jaw musculature with the 5th cranial (trigeminal) nerve that feeds directly into the limbic system of the brain". The 5th cranial nerve supplies the muscles of mastication, the TMJ, a portion of the throat, ears, back of the eye, sinus cavities, and muscles on the side of the head. Dr. Gordon indicates that recent studies show that the trigeminal nerve also supplies sensory input to the blood vessels of the brain. He postulates that this may explain why TMJ may not only be involved in headaches, but also how dysfunction of the TMJ muscular complex could amplify problems in the limbic system.

In the fibromyalgia patient, tricyclic antidepressants at night can help correct the alpha-delta (light stage to deep stage) sleep abnormality (Starlanyl and Copeland, 1996). Many researchers in the field of chronic pain feel that improving sleep in the TMD and fibromyalgia patient is essential. Improved sleep cycle regulation will be quite beneficial for those patients who experience TMD and fibromyalgia concurrently. Given Yunus' theory of TMD and FMS comprising a family of overlapping syndromes, or a "Dysregulation Spectrum Syndrome", addressing a sleep dysregulation issue in both these patients makes perfect sense."(The Fibromyalgia Network, "Overlapping Syndromes", 1998).

Productive sleep and moderate activity would thus be factors which have the potential to help both patient groups by hormonal regulation. L.J. Crofford (1998), noted rheumatologist at the University of Michigan, related that interventions including exercise provide symptomatic improvement in patients with fibromyalgia and directly or indirectly affect the HPA axis. These interventions include exercise, tricyclic antidepressants, and serotonin re-uptake inhibitors.

Finding the appropriate level of aerobic exercise for each individual is paramount. A patient should be supervised medically to determine their appropriate level, gradually striving to achieve a consistent level of 20 minutes per day, 3 times a week. Appropriate levels of exercise will improve sleep cycles, as well as regulate hormones directly, especially if done 4-5 hours prior to bedtime. However, one should avoid exercise directly before bed, as this may have a negative effect. It is more advantageous to exercise in the morning than too close to bedtime. The result of properly timed exercise is more time spent in critical Stages 3 and 4 of the SWS sleep cycle. In addition, as mentioned earlier, Substance P is depleted during exercise, while growth hormone and serotonin are secreted which confirms the absolute necessity of proper exercise and activity levels in these patients(Fibromyalgia Network, 1997; Institute For Natural Resources, 1997).

In addition, Bennett (1998) stated that growth hormone deficiency occurs in 30% of fibromyalgia patients. Treatment with recombinant growth hormone appears to improve tender point count, aiding once again, in symptomatic relief. Growth hormone is linked to fat metabolism, and most physicians who treat fibromyalgia suggest the patient regulate their dietary fat intake. These patients may crave carbohydrates as well, which makes sense since carbohydrates encourage serotonin production (Institute For Natural Resources, 1997).

Growth hormone is produced during peak muscular activity, as well as in Stage 4 sleep. An appropriate level of activity is necessary to help regulate this important substance (Bennett, 1998).

Serotonin re-uptake inhibitors appear to improve symptoms by working synergistically with the tricyclic antidepressants administered at night. (Smith, 1998). Measures which regulate hormonal levels based in the hypothalamic-pituitary-adrenal stress axis appear to be the key to symptomatic relief. In addition, tricyclic antidepressants are postulated to increase cerebral blood flow to the areas of the brain related to pain. Recent research in this area by Mountz et al (1995), appears to suggest that the fibromyalgia patient may have decreased regional cerebral blood flow to the areas of the brain that are related to pain.

Stress, Depression, and TMD/Fibromyalgia

Parker, Holmes, and Terezhalmy (1993) researched personality characteristics of the TMD patient and other chronic pain patients. They found that both groups exhibited similar personalities. Both patient groups seem to have stress. Stress over chronic pain, stress over illness, and stress from the poor sleep they experience which would tax anyone's system. TMD is linked to stress via bruxing and grinding. Fibromyalgia has previously been linked strongly to stress, but research efforts in the last five years have been directed towards discovering if there may be an actual centrally-mediated physical link to fibromyalgia (Mountz,1998; Simms,1998), as opposed to antiquated theories of it being a solely stress-related disorder. A multi-faceted model proposed by Bennett (1996a) suggests that a barrage of noxious stimuli assist in maintaining the neuroplastic changes which contribute to the chronicity of fibromyalgia, i.e., pain leads to the stress response which leads to a circular feedback which maintains, amplifies, and perpetuates chronic pain.

Studies conducted recently at the University of Michigan by Korszun (1997) and his associates concluded that the vast majority of TMD patients in this study met the criteria for clinical depression ranging from mild to severe, as measured by the Diagnostic and Statistical Manual of Mental Disorders. In addition, TMD disorders were demonstrable in 71% of these subjects who met the criteria for depression. Fibromyalgia is also correlated with a high incidence of depressive symptoms. It is estimated that over 20% of fibromyalgia patients exhibit depressive symptoms. (I feel this estimate is conservative). It is postulated that this may either be due to a chemical imbalance in the brain or due to the development of chronic pain (Shankland, 1998).

Is the high incidence of depressive issues in both of these population groups a result of hormonal factors, (perhaps influenced by CNS-involved altered sleep), or is it a result of the chronic pain and non-restorative sleep they experience which yields the altered hormonal levels? Interestingly, The Fibromyalgia Network (1997) mentions that although the two entities may co-exist, sleep patterns are distinctly different between the depressed patient and the patient diagnosed with fibromyalgia. (Food for thought.)

Statistics and Female Hormonal Issues

It is estimated that 8% of the female population has fibromyalgia, and over 75% of all fibromyalgia patients are women. It is also estimated that three to six million people have been diagnosed with fibromyalgia. Hormonal issues are postulated to be a key component (Smith, 1998; Fibromyalgia Network, 1997). The highest prevalence of TMD occurs in women of reproductive age (Le Resche, et al, 1997). This prevalence decreases in the post-menopausal years. Studies conducted in 1997 at the University of Washington by Le Resche, Saunders, Von Korf, Barlow, and Dworkin concluded that female reproductive hormones may play a role in TMD. Usage of oral contraceptives increased risk of TMD by 20%. The incidence of a patient experiencing TMD was 30% higher among patients receiving estrogen compared to those not exposed. These results suggest that female reproductive hormones may play an etiologic role in orofacial pain. Fibromyalgia is also most prevalent during the reproductive years, mainly affecting women, whose pain levels and tender points are commonly affected by the fluctuating hormonal levels during the menstrual cycle. Circulating serotonin decreases in the latter portion of the menstrual cycle. This corresponds with the propensity to increased pain perception in this same juncture in the menstrual cycle for both population groups.

Immune Related Factors and Their Relationship to Fibromyalgia and TMD

In addition, patients with fibromyalgia appear to have a higher incidence of allergies than the normal population (Fibromyalgia Network, 1998a). Hormonal, and thus immune factors, appear to play a role, which may also be based in the sleep cycle. Deep sleep encourages proper hormonal and immune regulation. Deep sleep is altered in the patient with fibromyalgia, by what current research postulates may involve some form of dysregulation of the central nervous system. Thus, hormonal and immune issues may often result from this centrally-involved disrupted sleep.

The presence of allergy background in patients diagnosed with fibromyalgia was frequently

found as well in a study by Tuncer et al (1997) as compared to matched controls. Allergic skin tests were positive in 66% of his test subjects with primary fibromyalgia. According to Baraniuk et al (1998) rhinitis symptoms are present in approximately 70% of subjects with fibromyalgia and chronic fatigue syndrome. Interestingly, only 35-50% of their subjects tested positive to allergy skin tests, suggesting some non-allergic mechanism may also play a role. They feel that alternative mechanisms such as inducible, irritant-activated, or reflex-mediated effects should be studied further.

Regardless of the cause, allergic or non-allergic rhinitis both encourage mouthbreathing which is a major factor in the symptomatology of myofunctional disorders (Barrett and Hanson, 1988). Therefore, if patients presented with either a hormonal dysregulation which would tend to encourage allergic tendencies, or a non-allergic mechanism which resulted in chronic rhinitis, wouldn't the development of myofunctional disorders including TMD difficulties be expected?

Boshart (1998) postulates through multiple sources, who report that the vast majority of TMD patients have altered tongue and jaw rest postures. Specifically, Boshart indicates that Dr. Jack Lynn of the TMJ Pain Clinic in Pittsburgh, Pennsylvania states, "Most of my patients, about 99% of them, have a low tongue resting posture." In addition, Boshart also says that Donald Chapton of the Advanced Dentistry TMD Treatment Clinic in Murrieta, California similarly concludes, "At least 86% of my patients have flaccid tongues that rest low and forward." Although this is an observation most practicing myologists would confirm in patient populations, it is nice to see this confirmed by noted TMD researchers. Thus, is it possible to link CNS altered sleep to TMD and mouthbreathing in a percentage of orofacial myofunctional patients? In my own practice, I have frequently observed this very link. For example, an adult patient presented for treatment with a childhood history of allergies or rhinitis, current low and forward postured tongue, tongue thrust swallow pattern, and TMD with sleep alterations. Perhaps this relationship warrants further investigation.

Pain and Perception-What Distinguishes TMD from Fibromyalgia?

Under normal circumstances, the body is capable of filtering out or blocking pain signals. However, TMJ disorder specialist, Herbert Gordon, D.D.S. (1995), states that "people with chronic pain conditions like fibromyalgia may have lost some of their ability to filter out pain at the spinal cord level and block it out at the higher regions in the brain. This is one of the effects of the limbic system/CNS deregulation that many believe to be the cause of fibromyalgia." In addition, he relates that the longer the pain exists in a particular site, the more the brain becomes actively involved in the pain process, and the larger the area of the brain dealing with the pain becomes.

Similar to fibromyalgia, pain radiates from a central location in the TMD patient. This can be to the jaw, head, neck, shoulder, face, scalp, etc. via highly sensitized and tight trigger points within the muscle tissue which indicates circulatory stasis, and causes pain.

According to Robert Bennett (1996b), one of the foremost researchers in fibromyalgia, in the fibromyalgia patient, there is a reduced pain threshold present (allodynia), an increased response to painful stimuli is present (hyperalgesia), and an increased duration of after nociceptor stimulation (persistent pain). These are all characteristics of central pain amplification states.

Korzun et al (1998) found that patients appearing for treatment of chronic facial pain/temporomandibular disorders show a high comorbidity with fibromyalgia. He concludes that clinical overlap between these conditions may reflect a shared underlying pathophysiologic basis involving dysregulation of the HPA axis in predisposed patients. This correlates with other research which postulated centrally regulated HPA axis link to fibromyalgia. Simms (1998) concludes that the weight of evidence suggests that fibromyalgia syndrome has a central rather that a peripheral or muscular basis.

It should be noted that patients with TMD and fibromyalgia may both experience myofascial pain. In fact, patients with fibromyalgia and TMD appear to have a high incidence of myofascial pain syndrome, and commonly exhibit overlapping syndromes. Dr. Muhammed Yunus, a noted researcher in the field of fibromyalgia, terms the overlap of a variety of syndromes Dysregulation Spectrum Syndrome, or DSS.

Included in this DSS grouping are fibromyalgia, chronic fatigue, myofascial pain, TMD, and other conditions that form a family of overlapping syndromes. Dr. Yunus uses the term dysregulation to mean biophysiological abnormalities, possibly in the neuro-hormonal system. (Fibromyalgia Network, "Overlapping Syndromes", 1998).

Body pain appears more episodic in nature in the myofascial pain patient, and more constant and severe in the patient with fibromyalgia. Dao et al (1997) found that facial pain is strongly correlated to the pain-body index in fibromyalgia, but not in myofascial syndrome. He concluded that facial pain in the fibromyalgia patient is most likely a clinical manifestation of fibromyalgia, but that it was unlikely to be related to body pain in the patient with myofascial pain syndrome. This finding suggests that myofascial pain syndrome and fibromyalgia have different etiologies. Dao et al (1997) recommends that fibromyalgia should be included in the differential diagnosis of myofascial pain of the masticatory muscles.

In response to TMD, fibromyalgia, and postulated central nervous system issues, research was conducted by Maixner, Fillingim, Sigurdsson, Kincaid, and Silva (1998). Their findings revealed that pain is more widespread, and appears to be felt to a greater degree in both the TMD patient and the patient with fibromyalgia than the level of pain intensity typically found in the normal population. However, due to more complex central nervous system issues, patients with fibromyalgia report pain to be more severe than the patient with TMD who has no underlying fibromyalgia. They found evidence of enhanced sensitivity to experimental pain in TMD patients, a common finding in fibromyalgia. These findings suggest that alteration in the CNS processes which contribute to enhanced pain sensitivity may indeed be present in patients with TMD as well as fibromyalgia.

This research also confirms the study conducted at the Stockholm Institute by Hedenberg, Ernberg, and Kopp (1997) that pressure and pain threshold and tolerance levels are lower in fibromyalgia than patients with local myalgia, but both experienced levels of pain tolerance which were lower than the control groups. Basically, the research concludes that the TMD or local myalgia patient will complain of lesser response to noxious stimuli than a person with fibromyalgia, but both groups will report higher pain perception than a non-effected patient. There appear to be several links which require investigative work by the orofacial myologist to successfully treat TMD. The key points include the patient's perception of pain. The diagnostic criteria for the evaluation of fibromyalgia hinges on whether the patient experiences pain, which is reported as severe or sustained, in a variety of areas of the body, as opposed to more localized pain in the jaw/head/shoulder region.

In myofascial pain syndrome, the pain is often more localized to a more specific body area. Bennett (1996a) postulates that an initial specific or localized myofascial injury may be a precipitating factor. Shortly thereafter, regional distribution occurs, and the pain spreads, ultimately sensitizing and altering the nervous system. More regional pain symptomatology then ensues. In fibromyalgia, the radiation of pain tends to be more severe and occurs in more individual and distant sites, and tends to be more generalized to the majority of the body. Arroyo and Cohen (1993) found that when the upper extremities of fibromyalgia patients are exposed to electrocutaneous stimulation, they experienced secondary hyperalgesia. In fibromyalgia, highly sensitive and effected sites are called tender points. Even with conscious control of relaxation, these points are difficult to eradicate due to a more complex cause. Local injections of anesthetic, however, will eradicate tender point pain, and lends credence to the theory that fibromyalgia is not solely a CNS, but a multi-faceted issue (Bennett, "Advanced Fibromyalgia" 1996). The use of trigger point injections has also become common in the management of TMD pain. Since current research suggests that TMD is principally musculoskeletal, orthopedic, and neurologic in nature, it is not surprising that these particular modalities, which have been utilized successfully in management of similar disorders such as fibromyalgia, would be utilized in TMD pain management.

The Orofacial Myologists Role-What We Can Do to Help

Success in treating these patients is best accomplished via a multidisciplinary team approach. Orofacial myologists should play a definitive role in this approach.

In my experience, I have found that addressing any correctable sleep or related issues in a multidisciplinary approach is essential to success. It is not beyond the scope of the orofacial myologist, (in addition to any primary recommendations from a patient's physician), to suggest behaviors that will assist these patients in obtaining restful and restorative sleep. Suggestions may include sleeping in a darkened room, wearing earplugs or eye masks, utilizing a comfort pillow suggested by their physician or physical therapist, listening to white noise or relaxation tapes prior to bedtime, and keeping exercise activity at a minimum 2-3 hours prior to bedtime. Soft oral splints are often utilized in the team approach by many of the orofacial myology patient's dental practitioners. Afternoon "quiet time" of 20-30 minutes can also be helpful in improving night-time sleep. An extended nap, however, tends to disrupt sleep at night. If the patient is capable of using relaxation/meditation techniques during this "down time", they will experience the synergistic effect of improved night time sleep, increased serotonin, and increased muscle relaxation (Institute For Natural Resources, 1997).

orofacial myofunctional disorder via orofacial myofunctional therapy, may all prove beneficial.

Conclusion

Treating the patient with chronic pain is not a clear cut nor easy process. Many factors appear to be involved, and much research in this area still needs to be initiated. However, taking into account the multi-faceted dynamics of chronic pain, the orofacial myologist can provide an invaluable service to patients as a member of an interdisciplinary team in the treatment and management of these disorders. Each factor in this approach is a necessary and complimentary/synergistic piece of the puzzle. The combination of addressing all these factors will bring the optimum degree of improvement in these very special patients.

In addition, this interdisciplinary approach may justify the orofacial myologist's need to suggest resources or provide care in a variety of areas. The emphasis must be on a course of treatment that involves educating and assisting patients in the physical and behavioral management of their own problem (McNeill, 1997). It would certainly be prudent to refer the patient to a rheumatologist if an underlying fibromyalgia syndrome is suspect. In addition, exploration with other medical and health care professionals to determine: appropriate levels of aerobic activity; educating patients in ergonomic correctness in the workplace and in their daily lives; informing patients on the role of caffeine and other various dietary substances on sleep; providing referrals to nutritionists and other qualified dietary professionals who can assist patients in supplementing their diet as needed with the essential vitamins and minerals necessary for improved hormonal function and optimum health; addressing relaxation and stress; referring aching taut muscles and trigger points for massage or directly performing related myofascial release (Institute For Natural Resources, 1997) and of course, treating the underlying

Whether a patient has a nocturnal thumbsucking habit, mouthbreathes due to possible ENT complications, exhibits symptoms of TMD, or complains of excessive snoring at night, orofacial myologists are in a unique position to identify symptoms of potential or underlying sleep disorders in our patients.

As always, a proper assessment and a keen eye will help us to help our patients to once again help themselves.

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