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FUNCTIONAL PATHOPHYSIOLOGY

Breathing and temporomandibular joint disease

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Summary Temporomandibular joint disease (TMD) refers to a collection of pain related conditions in the masticatory muscles and temporomandibular joint. Occlusal factors have been implicated in TMD pathogenesis, yet despite decades of research no causal relationship between occlusion and TMD has been found. The significance of psychosocial factors in both the assessment and the long-term management of patients with TMD is receiving increased recognition. The teaching of relaxation skills and coping strategies are effective, proven TMD therapies. The role of breathing re-education in temporomandibular joint (TMJ) disorders is rarely mentioned. A focus on breathing patterns and their disorders potentially explains how biomechanical factors associated with psychosocial influences might lead to pathophysiological changes within the TMJ as well as in the associated muscles. Attention to factors such as breathing and postural rehabilitation provides health professionals valuable, additional tools to help care for patients with TMD.

Introduction

Temporomandibular joint disease (TMD) refers to a collection of pain related conditions in the masticatory muscles and temporomandibular joint. Occlusal factors have been implicated in TMD pathogenesis, yet despite decades of research no causal relationship between occlusion and TMD has been found (Koh and Robinson, 2004; Mackie and Lyons, 2008). The tendency of TMD to improve with the passage of time, regardless of treatment modality (Sherman and Turk, 2001), has hampered research. The condition appears self-limiting and associated with no significant long-term disability in the majority of patients. Where patients have been followed up for 20 years, the end result of TMD is often not incapacitating (Magnusson et al., 2005). The significance of psychosocial factors in both the assessment and the long-term management of patients with TMD is receiving increased recognition (Suvinen et al., 2005). Physical and/or sexual abuse during childhood (Curran et al., 1995) as well as anxiety and depression are often associated with TMD (Madland et al., 2000; Manfredini et al., 2004; Wright et al., 2004). Significantly, the teaching of relaxation skills and coping strategies are effective, proven TMD therapies (Sherman and Turk, 2001; Suvinen et al., 2005; Orlando et al., 2007; Carlson, 2008).

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Breathing patterns

While the main function of respiration is the homeostatic regulation of arterial pO_2 and pCO_2 levels, breathing is also influenced by emotions and the limbic system (Homa and Masaoka, 2008). Respiratory symptoms are common in people suffering from anxiety and/or depression (Goodwin et al., 2004) and breathing re-education is an effective treatment for both depression (Tweeddale et al., 1994) and anxiety (Gilbert, 2003). In stressful situations the limbic system activates the "fight-or-flight" response (Von Schéele and von Schéele, 1999) and the breathing pattern can change from a diaphragmatic (or abdominal) breathing pattern to a thoracic (or chest) breathing pattern (Schleifer et al., 2002). The corresponding increase in respiratory rate and in minute volume (the volume of air inhaled and exhaled per minute) leads to a hyperventilation state where by definition the increase in respiratory rate exceeds the metabolic demands for oxygen (Schleifer et al., 2002) and arterial pCO_2 is lowered. TMD patients when compared to controls have a similar breathing rate, but a significantly lower end tidal pCO_2 (Carlson et al., 1998) indicating that TMD patients hyperventilate creating a respiratory alkalosis (Foster et al., 2001). End tidal pCO_2 refers to the peak concentration of CO_2 in a single breath of air at the end of expiration; it provides a close estimate of arterial pCO_2. (Schleifer et al., 2002).

The hyperventilation seen in TMD (Carlson et al., 1998) can be regarded as part of the "fight-or-flight" response (Von Schéele and von Schéele, 1999). In the 1930s, Cannon described the physiological changes associated with the "fight-or-flight" response as being characterized by increased sympathetic nervous system activation, increased central nervous system arousal and increased skeletal muscle activity (Jacobs, 2001). Hyperventilation by lowering arterial pCO_2 induces an acute respiratory alkalosis, with a corresponding increase in plasma pH and the movement of CO_2 from intracellular to extracellular fluids. The increased pH enhances the likelihood of motor unit neuron depolarisation or excitation (Schleifer et al., 2002) contributing to increased central nervous system arousal. The increased pH also improves muscle function as seen in short duration cycle sprints (Bishop et al., 2004) contributing to the increased skeletal muscle activity again necessary in a "fight-or-flight" response.

Ironically this same 'fight-or-flight" response when prolonged can contribute to musculoskeletal disorders (Jacobs, 2001). The increased neural excitation associated with the central nervous system arousal contributes to increased muscle tension and muscle spasm (Schleifer et al., 2002). A respiratory alkalosis reduces the release of oxygen from haemoglobin (Bohr effect) (Schleifer et al., 2002). Prolonged hyperventilation influences the alkaloid buffering system (Von Schéele and von Schéele, 1999). In chronic respiratory alkalosis renal compensatory mechanisms excrete HCO_3^- in order to return the plasma pH back towards the normal range (Foster et al., 2001). The systemic loss of bicarbonate from intracellular and extracellular fluids reduces the body's ability to buffer any build up of metabolic byproducts such as lactic acid in muscle tissue (Von Schéele and von Schéele, 1999; Schleifer et al., 2002). In this situation skeletal muscle fatigues more readily (Von Schéele and von Schéele, 1999). In an alkalotic state, muscle lactic acid efflux also increases leading to an increase in blood lactic acid levels (Galloway and Maughan, 1996). Lactic acid contributes to muscle pain (Mense, 2008).

In stressful situations the change in breathing pattern from a diaphragmatic (or abdominal) breathing pattern to a thoracic (or chest) breathing pattern (Schleifer et al., 2002) leads to the increased utilization of upper body muscles. When used as accessory muscles of respiration, the sterno (cleido) mastoid muscles not only lift the chest upward but can also pull the head forward (Simons et al., 1999a). In this situation the scalene and parasternal intercostal muscles also flex the neck (Simons et al., 1999b). Because the eyes need to keep looking forward, the head extends at the atlanto-occipital joint and the suboccipital muscles at the base of the skull shorten, to extend the head (Simons et al., 1999c ; Hruska, 1997). These suboccipital muscles, designed for fine eye movement coordination, are frequently tender to palpation in patients who suffer from migraine (Mongini, 2007; Fernandez-de-Las-Penas et al., 2008), tension type headache (Mongini, 2007; Fernandez-de-Las-Penas et al., 2006) and TMD (Chaves et al., 2005). In dysfunctional breathing, the pectoralis minor muscle lifts the chest upward, but in so doing pulls the shoulders forward (Hruska, 1997; Simons et al., 1999d). This action is opposed particularly by the upper trapezii and levator scapulae muscles (Simons et al., 1999d).

Patient with a dysfunctional breathing pattern typically adopt a posture, with rounded shoulders and a forward head position (FHP) (Hruska, 1997). The FHP has been implicated in TMD (Woda et al., 2001), but the quality of evidence supporting this association has been questioned (Olivo et al., 2006). The distance between the upper and lower teeth with the mandible in rest position is the freeway space (Woda et al., 2001). A FHP leads to a decrease in freeway space (Woda et al., 2001). Dental splints increase the freeway space and reduce the pressures on the TMJ generated by clenching (Nitzan, 1994). Correspondingly a reduction in freeway space through mandibular elevation results in an increased pressure on the temporomandibular joint itself (Hruska, 1997). A FHP is believed to alter occlusion, lead to increased posterior tooth contact and increased temporomandibular joint (TMJ) compression — all anatomical changes emphasized in TMD (Mannheimer and Rosenthal, 1991; Hruska, 1997). Mouth breathing can lead to a FHP with alteration in mandibular position (Neiva et al., 2009). In children, mouth breathing is associated with increased masticatory muscle tenderness (Chaves et al., 2005). Correction of head and cervical posture which increases freeway space (Woda et al., 2001) is considered important in TMD treatment (Olivo et al., 2006). The restoration of diaphragmatic breathing is also an important musculoskeletal and psychological therapy used in correcting the FHP (Simons et al., 1999e), and in helping patients suffering from TMD (Hruska, 1997; Sherman and Turk, 2001).

The temporomandibular joint

The temporomandibular joint is a combined hinge and sliding joint (Figure 1). A dense fibrous tissue disc called the articular disc, in the middle of the joint, separates the condyle of

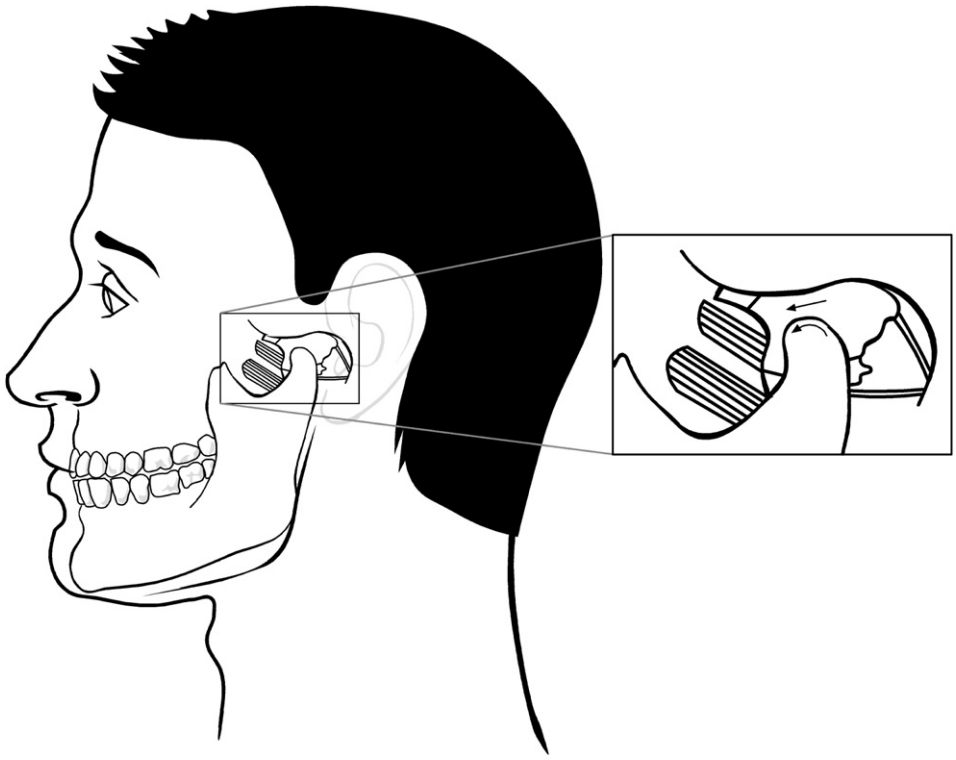

Figure 1 As the jaw joint opens, the condyle rotates on the disc and the disc slides forward on the mandibular fossa.

the mandible and the mandibular fossa. On opening and closing, the rotating action of the condyle is largely against the cartilaginous articular disc. While the lower joint has a rotating action, the upper joint has a sliding (or translating) action. The articular cartilage on the condyle slides forward against the articular cartilage of the mandibular fossa. The synovial fluid in the TMJ has important roles both in supplying nutrition to the avascular cartilaginous tissue within the TMJ and in lubrication (Nitzan, 2001). Whenever the pressure within the TMJ exceeds the capillary perfusion pressure a temporary tissue hypoxia ensues. An intra-articular pressure above 40 mm Hg surpasses the peripheral arteriolar pressure and can cause temporary hypoxia within the TMJ (Nitzan, 1994). Nitzan recorded the average intra-articular pressure on clenching as 73.70 ± 61.06 mm Hg in females as opposed to 31.42 ± 11.47 in males (Nitzan, 1994). Repetitive loading and unloading of the joint can lead to hypoxia-perfusion injury. Reactive oxidative molecules are released which can breakdown the molecules that facilitate joint lubrication (Zardeneta et al., 2000). Increased friction is frequently mentioned as a possible cause of TMJ clicking and possible disc displacement during jaw opening (Tanaka et al., 2008). Bruxism, the habitual grinding of the teeth and jaw clenching, has also been linked to TMD (Michelotti et al., 2010). The repetitive loading and unloading of the joint involved with bruxism would also lead to hypoxia-perfusion injury within the TMJ. Awake bruxism, as opposed to sleep bruxism, is associated with stress, anxiety and depression (Manfredini and Lobbezoo, 2009), once again emphasizing the importance of psychosocial factors in the development and treatment of TMD.

Lavage (or irrigation) of the upper joint compartment with saline using arthroscopy or arthrocentesis in closed lock situations or in osteoarthritic joints improves function and alleviates pain, although it does not change disc position (Brennan and Ilankovan, 2006). In this situation, lavage may be effective by removing reactive oxidative molecules that damage TMJ lubrication (Nitzan, 2003). Dental splints elevate the occlusal plane and reduce the pressures on the TMJ generated by clenching (Nitzan, 1994). This supports the concept that the reduction in the distance between the upper and lower teeth with the mandible in rest position (freeway space) brought about through a FHP (Woda et al., 2001) could result in increased pressure through the mandible on the temporomandibular joint leading to hypoxia-perfusion injury.

TMJ clicking without pain or disc displacement is common (Gesch et al., 2004). Clicking in this situation might occur as a result of increased friction between the disc and the eminence of the mandibular fossa — the clicking in this situation seems to occur with a jump by the disc and the condyle over the eminence (Tanaka et al., 2008). Prolonged frictional changes could lead to secondary pathology such as disc displacement where the disc is displaced from on the head of the condyle (Nitzan, 2003). If the disc moves off the condylar head the ligaments holding the disc in place stretch. The chemical inflammation from reactive oxidative molecules within the TMJ and the trauma associated with stretching of the ligaments holding the disc in place could sensitize local nocioceptive endings (Mense, 2008). These issues are discussed more fully in the next section.

Pain production

Muscle pain is produced by both peripheral and central mechanisms. Peripheral muscle pain is produced by the

sensitization of specific pain receptors called nociceptors. Nociceptors are activated by trauma, mechanical overloading and inflammatory mediators such as bradykinin, prostaglandins, adenosine triphosphate (ATP) and protons (H^+) (Mense, 2008). An acidic tissue pH, which would be aggravated by a loss of systemic bicarbonate and lactic acid production, is probably one of the main activators of peripheral nociceptors (Mense, 2008). Mechanical factors such as poor posture, which leaves some muscles in shortened positions and others under chronic tension in a lengthened position for prolonged periods, as well as muscle spasm can lead to trigger point development in these muscles (Simons et al., 1999f). The resulting drop in tissue pH would sensitize nociceptors (Mense, 2008). Systemic factors that compromise muscle and neural energy metabolism are also thought to be contributory. These include nutritional deficiencies in vitamin D, a number of the water-soluble vitamins including vitamins B_1, B_6, B_{12}, folic acid, vitamin C and certain elements such as calcium, magnesium, iron and potassium. Borderline hypothyroidism has also been implicated in muscle pain (Simons et al., 1999g; Gerwin, 2005).

Sensitization of muscle nocioceptive endings leads to the release of the neuropeptides substance P and calcitonin gene related peptide. These peptides created local oedema by dilating local blood vessels and increasing their permeability. The sensitization of muscle nociceptors is assumed to be the peripheral mechanism leading to muscle tenderness (Mense, 2008).

The influx of nervous impulses from muscle nociceptors into the spinal cord can also lead to central processing changes of pain signals at a dorsal horn level. This leads to a long lasting increase in excitability (central sensitization) due to the effect of glutamate on NMDA (*N*-methyl-D-aspartate) receptors. This is a possible mechanism for referred muscle pain, a contributory factor to increased peripheral muscle tenderness as well as an explanation for myofascial trigger points (Mense, 2008). Glial inflammation has also been implicated as an additional important factor in the initiation of acute pain and the perpetuation of chronic pain in a number of pain conditions including migraine (Bartley, 2009; Milligan and Watkins, 2009). In a rat model, the injection of capsaicin into the TMJ capsule has been shown to cause glial inflammatory changes within the trigeminal ganglion. Glial inflammation is thought to be an additional important factor in the development of a number of peripheral pain conditions as well as providing a potential explanation of the link between TMD and migraine (Thalakoti et al., 2007).

In an experimental situation, patients with a history of tension headache or age and sex matched controls were made to clench their teeth for 30 min. Sixty-nine percent of the patients and 17% of controls developed a tension type headache. Muscle tenderness after tooth clenching increased only in those people who then went on to develop a tension headache. The headache can be viewed as a central neural sensitization by tender muscles (Jensen and Olesen, 1996). Incoming pain messages from chronically contracted muscles holding the head and neck in a prolonged unnatural position such as a FHP could generate a central neural hypersensitivity. This is also the position adopted by many people when they sit at a computer (Pascarelli and Hsu, 2001) or hyperventilate using their accessory muscles of respiration (Hruska, 1997). The findings of tender muscles may indicate that not only the muscles are sore, but also that a central neural hypersensitivity is present.

Therefore the masticatory muscle pain seen in TMD may be secondary to the interplay of a number of factors.

- Postural changes associated with a FHP which place some masticatory muscles in shortened positions and others under chronic tension in a lengthened position for prolonged periods would be a factor in muscle trigger point and pain development (Simons et al., 1999f).
- Increased neural excitation associated with respiratory alkalosis contributing to increased muscle tension and muscle spasm (Schleifer et al., 2002).
- The systemic loss of bicarbonate compromising the body's ability to buffer the build up of metabolic byproducts such as lactic acid in muscle tissue leading to muscle fatigue (Von Schéele and von Schéele, 1999; Schleifer et al., 2002) and muscle pain (Mense, 2008).
- Chemical inflammation from reactive oxidative molecules sensitizing local nocioceptive endings in the TMJ (Mense, 2008).
- Prolonged peripheral pain leading to changes in pain processing at a central level (Mense, 2008).

Breathing patterns may also be linked to pain modulation at a central level (Rhudy, 2010). A number of experiments have also shown that anxiety, which is linked to alterations in breathing patterns (Gilbert, 2003), lowers pain thresholds (more sensitivity) (Rhudy and Meagher, 2000). In an experimental situation, slow breathing rate results in reduced pain intensity and unpleasantness ratings to pulses of painful heat (Zaustra et al., 2010).

Genetic polymorphisms

Catechol-*O*-methyl transferase (COMT) is an enzyme that metabolises catecholamines (adrenalin, noradrenalin and dopamine) through the methylation process. A genetic polymorphism means members of the population with reduced COMT activity are less able to degrade adrenalin, noradrenalin and dopamine. People with this genetic polymorphism have reduced pain thresholds and are more likely to develop TMD (Diatchenko et al., 2005). Rodent studies have shown that COMT inhibition increases pain sensitivity through activation of β2 and β3 adrenergic receptors (Nackley-Neeley et al., 2007). Propanolol, a non-selective β-adrenergic agonist, reduces pain more in those people with the COMT polymorphism and the reduced ability to degrade adrenalin and noradrenalin (Tchivilea et al., 2010). This would indicate that treatments that reduce stress levels and the sympathetic response could be beneficial in those people with reduced COMT activity. Diaphragmatic breathing and relaxation skills reduce the stress response (Martarelli et al., 2009).

Male /female TMD differences

While gender differences in pain sensitivity have been attributed to social conditioning and psychosocial factors,

increasing evidence suggests that other biological factors play an important role (Craft et al., 2004; Wiesenfeld-Hallin, 2005). Gonadal steroid hormones, in particular estrogen, are known to modulate opioid analgesia. In rats, increased estradiol levels appear to be risk factor for TMJ pain (Tashiro et al., 2009). TMD pain is 1.5—2 times more prevalent in women (Warren and Fried, 2001). If occlusal abnormalities alone were an important factor, TMD should occur equally in men and women. The incidence of TMD appears to peak around 20—40 years (Warren and Fried, 2001). In females, TMD incidence parallels reproductive function decreasing in menopause when ovarian production of estrogens decreases (LeResche et al., 2003).

Females are able to generate significantly higher TMJ pressures during clenching than males (Nitzan, 1994). During the second part of the menstrual cycle after ovulation has occurred women have a greater tendency to hyperventilate lowering arterial pCO_2 by 6.5 mm Hg (Hadziomerovic et al., 2008). As discussed the respiratory alkalosis as well as potential postural changes associated with hyperventilation could contribute to TMJ pain. In the second part of the menstrual cycle both progesterone and estrodiol are secreted. The administration of either progesterone or estradiol lowers arterial pCO_2 (Hadziomerovic et al., 2008). In females, TMD pain rises towards the end of the cycle and peaks in the first three days of menstruation. A secondary peak in pain also occurs around the time of ovulation (LeResche et al., 2003). TMD pain occurs at the time of low or fluctuating estrogen (LeResche et al., 2003). Estrogen and progesterone both influence minute ventilation and arterial pCO_2 levels (Slatkovska et al., 2006). The hormonal intricacies relating to pain and the menstrual cycle are still being untangled (Sherman and LeResche, 2010), but alterations in breathing pattern during the menstrual cycle could contribute to TMD.

Migraine, tension headache and TMD

TMD is frequently discussed as a risk factor in migraine (Bevilaqua Grossi et al., 2009) and tension headache (Lupoli and Lockey, 2007). Occlusal treatment may be suggested as a therapeutic option (Stapleman and Turp, 2008). TMD is common in the community (Gesch et al., 2004), migraine sufferers and tension headache sufferers (Ballegaard et al., 2008). Anxiety, depression, and muscle tenderness are often common factors in jaw joint pain, tension headache and migraine (Mongini, 2007). Rather than jaw joint problems per se causing tension headache and migraine, it may be that they share a similar underlying central nervous system pathophysiology (Bevilaqua Grossi et al., 2009). The teaching of relaxation skills and coping strategies as well as being effective in TMD (Sherman and Turk, 2001; Suvinen et al., 2005) is also beneficial in migraine (Campbell et al., 2009) and tension headache (Smitherman et al., 2007).

Conclusions

The role of breathing re-education in TMJ disorders (Hruska, 1997) is rarely mentioned. A focus on breathing patterns potentially explains how biomechanical factors associated with psychosocial influences might lead to pathophysiological changes both within the TMJ as well as in the associated muscles. While it has been reasonably postulated that the FHP leads to increased pressure in the joint this part of the picture has not yet been scientifically proven. Attention to breathing and postural correction provides health professionals valuable additional tools to help care for patients with TMD.

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