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Pathoanatomical characteristics of temporomandibular dysfunction: Where do we stand? (Narrative review part 1)

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A B S T R A C T

Temporomandibular dysfunction (TMD) is a complicated and multifactorial condition that affects the temporomandibular joint (TMJ) and muscles of mastication, resulting in pain and disability in 5–12% of the population. The condition involves genetic, anatomic and hormonal factors and is propagated, in part, by trauma, habitual activity, psychosocial components and occlusal variation. Yet, the exact etiology of TMD is still unknown and the most strategic conservative management of the condition is still a topic of debate. The purpose of this paper, the first of a two part series, is to provide greater insight into the pathoanatomical factors associated with TMD. Consistent with Scully (2008, 2013), degenerative changes seem to disrupt the relationship between the TMJ capsule, articular disc and muscles of mastication. The resulting position of the articular disc coincides with three primary classifications of TMD: Type 1 (muscle disorders), Type 2a/b (disc displacement with and without reduction), and Type 3 (any joint pain). Given the association of the lateral pterygoid with both the joint capsule and articular disc, the superior and inferior head seem to play a key role in TMD. Both heads undergo biological changes associated with the vicious cycle, pain adaptation and integrated pain adaptation, making the muscle a key pain generator associated with TMD. Clinicians must understand the pathoanatomic features associated with TMD so as to choose appropriate treatment strategies, leading to optimal short and long-term outcomes. While the former is discussed in part 1 of this narrative review, the latter will be considered in part 2.

1. Introduction

Temporomandibular dysfunction (TMD) is a complicated and multifactorial condition that affects the temporomandibular joint (TMJ) and muscles of mastication, resulting in pain and disability (Mujakperuo et al., 2010). According to the National Institute of Health and Cranial Research, the prevalence of temporomandibular dysfunction (TMD) ranges from 5 to 12% (Ariji et al., 2015; Murray and Peck, 2007). While the exact etiology of TMD is still unknown, genetic (Pihut et al., 2016), anatomic (Murray et al., 2004; Peck et al., 2008) and hormonal factors (Hiraba et al., 2000; Saghafi and Curl, 1995) seem to predispose the joint to problems (Friedman, 1997; Pihut et al., 2016). While a number of studies have also identified trauma, habitual activity and occlusal variation as being precipitating factors for TMD, there also seems to be a strong psychosocial component that propagates the condition (Jayaseelan and Tow, 2016; Mapelli et al., 2016). TMD is a complex condition, and the pathoanatomical factors associated with its etiology require further consideration.

2. Muscles of mastication

The primary muscles of mastication are the masseter, temporalis and medial and lateral pterygoids. The masseter is attached to the maxillary process of the zygomatic bone and the zygomatic arch proximally and the angle and ramus of the mandible distally (Marieb and Hoehn, 2010). It primarily elevates and protracts the mandible (Moore and Dalley, 2006). While the temporalis also elevates the mandible, its proximal and distal attachments to the temporal fossa of the temporal bone and the coronoid process and anterior border of the ramus of the mandible, respectively, are better suited for retracting rather than protracting the mandible (Marieb and Hoehn, 2010; Moore and Dalley, 2006). Deep to the

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temporalis and masseter, the medial pterygoid further assists with mandibular elevation and protrusion via its attachments to the lateral pterygoid plate and medial surface of the ramus of the mandible (Marieb and Hoehn, 2010; Moore and Dalley, 2006). While the medial pterygoid also facilitates side-to-side grinding movements (Moore and Dalley, 2006), the synergistic actions of the temporalis, masseter and medial pterygoid vertically close the jaw during mastication (Marieb and Hoehn, 2010; Moore and Dalley, 2006).

In contrast, the lateral pterygoid is divided into two heads, both of which are intimately related to the TMJ. Classically, the superior head of the lateral pterygoid runs from the infratemporal crest of the sphenoid bone and inserts onto the anterior aspect of the articular disc (Stelzenmueller et al., 2016). Based on the attachments of the superior head, a number of researchers have suggested that it may contract to pull the disc forward during mandibular depression (Hiraba et al., 2000; Juniper, 1984; Reichert and Stelzenmuller, 2008; Schunke et al., 2006), consistent with the position of the mandibular condyle (Manfredini, 2009; Stelzenmueller et al., 2016). Given this function, hyperactivity of the superior head of the lateral pterygoid could easily be implicated in anterior disc displacement (Bakke et al., 2005; Taskaya-Yilmaz et al., 2005).

However, based on the relatively small number of superior head fibers directly attached to the articular disc (Carpentier et al., 1988) compared to the condylar neck and the limited number of studies demonstrating superior head activation during mandibular depression (Gibbs et al., 1984; Hiraba et al., 2000; Mahan et al., 1983; Manfredini, 2009; McNamara, 1973; Murray and Peck, 2007; Murray et al., 2004; Wood et al., 1986), some researchers have suggested an alternative function. The nonelastic quality of the ligaments connecting the disc to the condyle and the biconcave shape of the disc also makes it unlikely that the disc is able to migrate from the superior aspect of the condyle, making tracking unnecessary (Manfredini, 2009). Notably, the posterior aspect of the disc attaches to the posterior capsule via retrodiscal tissue, which maintains posteriorly directed traction via its superior layer as the disc moves anteriorly with jaw opening (Manfredini, 2009). The attachment of the disc anteriorly to the superior head may, therefore, passively drag the disc forward in concert with the actions of the inferior head during mandibular depression, while its primary purpose may be to counter the retrodiscal traction and provide an anterior braking force to the disc as it moves posteriorly into the condyle during mandibular elevation (Manfredini, 2009).

The inferior head of the lateral pterygoid runs from the lateral plate of the pterygoid process to the condylar process of the mandible (Bakke et al., 2005; Benninghoff, 2004; Schmolke, 1994; Stelzenmueller et al., 2016; Taskaya-Yilmaz et al., 2005; Usui et al., 2008). Classically, bilateral inferior head activation is responsible for mandibular depression and protrusion (Gibbs et al., 1984; Hiraba et al., 2000; Mahan et al., 1983; McNamara, 1973; Murray and Peck, 2007; Murray et al., 2004; Wood et al., 1986), functions that have been confirmed by single-motor-unit recordings conducted by Phanachet et al. (Phanachet et al., 2001, 2002). That is, the bilateral contraction of the inferior head works with the suprahyoid digastric muscles (Marieb and Hoehn, 2010; Stelzenmueller et al., 2016) to pull the mandible anteriorly and inferiorly out of the fossa during jaw opening (Miloro, 2004). Notably, while the masseter, temporalis, medial pterygoid and superior head of the lateral pterygoid all seem to be primarily active during mandibular elevation, the inferior head of the lateral pterygoid and the digastric muscles are the primary players during mandibular depression (Monemi et al., 1999; Peck et al., 2000).

While a number of studies support the notion that the superior head is primarily active during mandibular elevation and the inferior head is active during mandibular depression (Desmons et al., 2007; Hiraba et al., 2000; Mahan et al., 1983; McNamara, 1973; Murray et al., 2004), investigations using EMG have suggested a more synchronous relationship for the two heads (Hannam and McMillan, 1994; Murray et al., 2004). By incorporating image guided EMG electrode placement via computer tomography, Murray et al. discovered more overlap between the superior and inferior head of the lateral pterygoid than previously thought (Murray et al., 2004). In fact, the authors cite three unique regions of the superior head of the lateral pterygoid, a medial region that fires consistent with the inferior head (i.e. during mandibular depression, protrusion and contralateral excursion), a lateral region that is active during mandibular elevation, retrusion and ipsilateral excursion and a middle region that exhibits firing patterns consistent with both the superior and inferior head (Murray et al., 2004; Phanachet et al., 2001). Therefore, EMG electrodes mistakenly placed in the middle region would certainly suggest the superior and lateral pterygoid were functionally similar when, in fact, that appears to not be the case (Murray et al., 2004).

Notably, the ipsilateral activation of the superior and inferior head of the lateral pterygoid has also been implicated in ipsilateral and contralateral jaw movements, respectively (Murray et al., 2004). In doing so, the inferior head may provide horizontal forces required for mastication and parafunctional activities (Murray et al., 2004; Widmalm et al., 1987; Wood et al., 1986). The inferior head seems to progressively increase in activity with horizontal excursion of the mandible in the contralateral direction (Murray et al., 2004; Uchida et al., 2002). Importantly, while some activity of both the superior and inferior head of the lateral pterygoid has been shown to occur with intercuspal jaw clenching, this action may be an effort to stabilize the condyle and prevent slippage in the posterior direction (Murray et al., 2004; Widmalm et al., 1987). Alternatively, the superior head may be firing to tension the articular disk, thereby maintaining the position of the condyle (Murray et al., 2004). Thus, while the masseter, temporalis and medial pterygoid provides the forces required for mastication in the vertical plane, the inferior head of the lateral pterygoid seems to facilitate horizontal forces. The reciprocal actions of the superior and inferior heads in the horizontal plane further suggest a role in fine motor control of the mandible during jaw movements (Murray et al., 2004; Phanachet et al., 2001, 2002).

3. The lateral pterygoid: two distinct muscles

Treating the superior and inferior head of the lateral pterygoid as one or two muscle continues to be a source of debate in the literature (Hannam and McMillan, 1994; Murray and Peck, 2007; Murray et al., 2004). Perhaps the best argument for two functionally distinct lateral pterygoid muscles is their neural innervation (Desmons et al., 2007). According to Kim et al. the superior head is innervated by the buccal nerve, while the inferior head is innervated by the mandibular nerve trunk (Kim et al., 2003). While different fiber orientations of a multi-penniform muscle could alternatively explain various complex actions of a single muscle (Desmons et al., 2007; El Haddioui et al., 2005), the unique neural innervation and activity during both jaw opening and closing suggests that a 2 muscle system is more likely (Aziz et al., 1998; Juniper, 1981, 1984; Liu et al., 1989). Notably, the masseter and medial pterygoid have the same muscle fiber structure and only fire during mandibular elevation (Desmons et al., 2007).

4. Etiology of temporomandibular dysfunction with anterior disc displacement

While TMD is a complex condition with a multi-factorial

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etiology (Gauer and Semidey, 2015; Silva et al., 2015), a recent study by Kumar et al. looked at 44 patients with TMD via MRI and found that 81.8% had disc displacement with or without reduction, but only 2.3% of displacement occurred in the posterior direction (Kumar et al., 2015). That is, most disc displacement occurs in the anterior direction (de Leeuw and Klasser, 2013), suggesting a disruption of the delicate balance in the forces imposed on the posterior and anterior aspect of the articular disc that normally work together to ensure proper tracking of the condyle (Manfredini, 2009; Taskaya-Yilmaz et al., 2005). Interestingly, the predominant mechanism described by Scully (2008, 2013) involves trauma and/or degeneration of the posterior capsule, which allows the disc to slip anterior of mandibular fossa and places the connection between the superior head of the lateral pterygoid and the anterior aspect of the disc on slack (Scully, 2008, 2013). According to Scully (Scully, 2008, 2013), the superior head of the lateral pterygoid is no longer able to control the disk as it moves back into the fossa during mouth closing (Gauer and Semidey, 2015; Liu et al., 1989). As a result, the inferior head of the lateral pterygoid may attempt to compensate by becoming active both during mouth opening, when it normally should fire, and during mouth closing, when it is normally silent (Juniper, 1984; Lafreniere et al., 1997). The double duty imposed on the inferior head results in significant overuse, resulting in micro tearing (McCain et al., 1989; Perrott et al., 1990; Scrivani et al., 2008), trigger point formation (Gonzalez-Perez et al., 2015; Harrison et al., 2014; Sidebottom et al., 2013; Wright, 2000), and pain (Chantaracherd et al., 2015; Lund et al., 1991; Manfredini, 2009; Scrivani et al., 2008).

Scully (Scully, 2008, 2013) outlines 3 classifications of TMD based on the position of the articular disc, which is consistent with the recent diagnostic classification proposed by Harrison et al. (Gauer and Semidey, 2015; Harrison et al., 2014). With little to no movement of the disc (Type 1: muscle disorders), muscle pain may be the primary symptom, resulting directly from overuse and/or tension or indirectly via guarding and central-mediated myalgia (Harrison et al., 2014). However, if there is remarkable anterior displacement of the disk, the condyle would shift on and off the central aspect of the disk during mandibular depression (Type 2a: Disc Displacement with Reduction without limited opening), often causing clicking, clunking and popping (Harrison et al., 2014; Manfredini, 2009). In the event that the disc continues to slip anteriorly, the disc may prevent a full anterior glide of the condyle (close-lock), thereby limiting mandibular depression (Type 2b: Disc Displacement without Reduction with Limited Opening) (Harrison et al., 2014). Nitzan (2002) also reports a number of cases whereby the mandibular condyle shifts over the articular disc and cannot return to the fossa, resulting in an inability to elevate the mandible (open-lock) (Nitzan, 2002). The continuous loading of joint structures, especially the retrodiscal tissue, results in persistent inflammation, a precursor of osteoarthritis (Type 3: Any Joint Pain) (Harrison et al., 2014; Manfredini, 2009).

5. Etiology of temporomandibular dysfunction with anterior disc displacement: superior head of lateral pterygoid

Any disruption of the balance between the posterior force imposed on the disc from the superior layer of the retrodiscal tissue and the anterior force from the superior head of the lateral pterygoid could, therefore, result in anterior disc displacement and TMD (Manfredini, 2009). Interestingly, Mazza et al. reported a correlation between a greater concentration of superior head attachments onto the disc and TMD, which may predispose the disc to excessive braking (Mazza et al., 2009). Excessive braking may not be an issue under healthy conditions, but over involvement of the superior head combined with degenerative changes of the TMJ could additively result in TMD (Manfredini, 2009; Mazza et al., 2009). A number of studies have demonstrated increased signal intensity on T2 images in the posterior aspect of the disc in TMD patients, which indicates effusion and represents an early sign of arthritis (Omami and Lurie, 2012; Takaku et al., 1998; Tomas et al., 2006). With time, two distinct changes occur to the retrodiscal tissue. There is evidence that the fibers of the superior layer begin to rupture and the stiffer fibers of the inferior layer begin to stretch, resulting in an anterior shift of the articular disc (Eriksson et al., 1992; Manfredini, 2009). Interestingly, Finden et al. reported nonlinear changes in T1 and T2 image signal intensity of the superior head of the lateral pterygoid consistent with muscle spasms in patients with displacement of the anterior disk (Finden et al., 2007). Given the connection between muscle overload, fatigue and ion imbalance (Bergeron, 2008), the spasms may be indicative of superior head overuse. Ironically, however, the involuntary muscle activity likely works to further jerk the disc forward, propagating the cycle of dysfunction (Finden et al., 2007). An anteriorly shifted disc disrupts the disc-stabilizing function of the superior head of the lateral pterygoid, eventually leading to disuse and atrophy (Taskaya-Yilmaz et al., 2005). Notably, patients with TMD experience greater anterior disc displacement and subsequent superior head spasms, disuse and atrophy on MRI images, when the muscle primarily attaches to the articular disc and not the disc and the condyle (Taskaya-Yilmaz et al., 2005). Given that Yang et al. found more pathologic changes in the superior head than the inferior head of the lateral pterygoid, the superior head seems to be a primary player in TMD (Yang et al., 2001).

6. Etiology of temporomandibular dysfunction with anterior disc displacement: articular disc

Changes to the disc shape and joint lubrication could also disrupt the normal forces imposed on the articular disc (Manfredini, 2009). More specifically, a thickening of the posterior disk bands and a thinning of the anterior bands cause a gradual transition from a biconcave shape to a biconvex shape (Almasan et al., 2013; Hirata et al., 2007; Manfredini, 2009). According to Manfredini et al. a biconcave shape is advantageous, as it helps to seat and stabilize the disc on top of the mandibular condyle, especially during the translation phase of jaw closing (Manfredini, 2009). Notably, Hirata et al. found a significant relationship between anteriorly displaced discs without reduction and either folded or biconvex shapes, suggesting that a disc that has lost its shape may be easier to anteriorly displace (Hirata et al., 2007). In addition, a number of authors have suggested that the normal movement of the articular disc in and out of the mandibular fossa requires a low friction coefficient (Nitzan, 2001). According to Tanaka et al. (2008), repetitive overloading or “wear and tear” results in decreased joint lubrication between the articular disc and the mandibular fossa (Tanaka et al., 2008). As the inferior head of the lateral pterygoid pulls the condyle forward during jaw opening, the increase in friction causes the disc to lag behind, leading in the disruption of the disc-condyle attachments (Leonardi et al., 2011; Manfredini, 2009; Nitzan, 2001; Tanaka et al., 2008). As a result, the compression of the disc by the condyle during chewing combined with activity of the superior head of the lateral pterygoid displace the disc anterior of the fossa, thereby placing the retrodiscal attachments in a stretched position (Leonardi et al., 2011; Manfredini, 2009; Nitzan, 2001; Tanaka et al., 2004, 2008). Importantly, changes in the shape of the articular disc and increased friction are not mutually exclusive issues. According to Tanaka et al. the mere presence of the disc minimizes the frictional coefficient (Tanaka et al., 2006). While a healthy disk minimizes incongruity between joint surfaces, a misshaped disk (i.e. biconvex and folded) is less

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capable of doing so (Tanaka et al., 2006).

7. Etiology of temporomandibular dysfunction with anterior disc displacement: inferior head of lateral pterygoid

Per the Scully model of TMD (Scully, 2008, 2013), the inferior head of the lateral pterygoid becomes more active in TMD in an attempt to compensate for the superior head's failure to stabilize the disk on the mandibular condyle (Juniper, 1984; Lafreniere et al., 1997). Juniper et al. reported simultaneous activity in the superior and inferior head of the lateral pterygoid (when it should normally be silent) during mandibular closing in patients with TMD (Desmons et al., 2007; Juniper, 1984). Lafreniere et al. also found increased superior head EMG activity during molar clenching, suggesting a loss of disk stabilization function in patients with internal disc derangement (Lafreniere et al., 1997). At the same time, the authors measured increased activity in the inferior head during rest, resisted protraction and clenching, which likely represents an effort to compensate for the lost stabilization while continuing to maintain its normal function (Lafreniere et al., 1997). Notably, no EMG increases were found in the temporalis and masseter in any condition, suggesting that these muscles may play less of a role in symptoms related to TMD (Lafreniere et al., 1997).

Consistent with Lafreniere et al. (1997)., Yang et al. identified 136 MR images of dysfunctional TMJs whereby both the superior and inferior head of the lateral pterygoid were abnormal (Yang et al., 1992). The authors identified 51.47% of the joints as having hypertrophied superior and inferior lateral pterygoids (Lafreniere et al., 1997; Yang et al., 1992). Moreover, 27.2% and 11% of the TMDs presented with atrophy and contractures of the superior belly, respectively, and hypertrophy of the inferior belly. That is, while the superior belly initially attempts to manage the articular disc, it eventually undergoes pathological changes indicative of pain avoidance and disuse, and the inferior belly tries to compensate (Hannam and McMillan, 1994; Yang et al., 1992). In accordance with Scully (Scully, 2008, 2013), 89.7% of patients with TMD had an abnormal lateral pterygoid with a hypertrophied inferior head according to MRI (Hannam and McMillan, 1994), findings consistent with recent investigations by Rawlani et al (Rawlani S, 2013). and Lopes et al. (2015). It is perhaps worth mentioning that radiologists are taught to recognize a “double disc sign” to identify patients with anterior disc displacement (Bag et al., 2014). The name comes from the fact that an anteriorly displaced disc often looks like two discs due to hypertrophy of the inferior head of the lateral pterygoid (Bag et al., 2014).

Interestingly, a number of authors have reported improvements in the dislocations (Fu et al., 2010; Oztel et al., 2016; Stark et al., 2015), clicking (Bakke et al., 2005; Emara et al., 2013), pain (Karacalar et al., 2005; Sunil Dutt et al., 2015) and disability (Karacalar et al., 2005; Sunil Dutt et al., 2015) associated with TMD after injecting the lateral pterygoid muscle with botulinum toxin A. Bentsianov et al. also noted a 70% success rate in adults with severe bruxism by injecting botulinum toxin into the lateral pterygoid, temporalis and masseter (Bentsianov et al., 2004; Tan and Jankovic, 2000). However, perhaps the most convincing evidence connecting a hyperactive inferior head of lateral pterygoid and TMD comes from a recent study by Guerrero et al. (2015). By comparing cone-beam computed tomography images of 60 patients with TMD, the authors found that patients with TMD had significantly longer pterygoid plates than control subjects (Guerrero et al., 2015). Given that the distal insertion of the inferior muscle belly attaches to the lateral pterygoid plate of the sphenoid bone, Guerrero et al. hypothesize that muscle hyperactivity leads to lengthening of the plate via Wolff's Law, which may be a primary, or at least one of the etiologic factors, for the pain and dysfunction associated with TMD (Guerrero et al., 2015).

8. Temporomandibular pain: lateral pterygoid

Empirical evidence of patient symptoms during clinical evaluation further implicates the lateral pterygoid as playing a major role in TMD. Cooper and Kleinberg (2007) looked at 4528 patients with TMD and found that the most prevalent symptom was tenderness to palpation of the lateral pterygoid muscle (83.3%) followed by tenderness of the joint capsule itself (62.4%) (Cooper and Kleinberg, 2007). Similarly, the most common symptom (96%) of patients with TMD with and without occlusal abnormalities was sensitivity of the lateral pterygoid (Ai and Yamashita, 1992; Shinoda et al., 2008). D'Ippolito et al. (2010) also evaluated 50 patients with TMD using the lateral pterygoid muscle test, a test originally described by Dworkin and Le Resche in 1992 (Dworkin and LeResche, 1992), to assess pterygoid sensitivity and reported that 100% of the patients were positive compared to 28.6% in healthy controls (D'Ippolito et al., 2010). According to D'Ippolito et al. (2010), the lateral pterygoid muscle test attempts to provoke lateral pterygoid pain by asking patients to bite a toothpick using the protruded central incisor teeth for 1 min. The consistent report of pain in the lateral pterygoid strongly suggests involvement of the muscle group in TMD. Given the key role of the lateral pterygoid in TMD, it perhaps worth noting that studies that have conducted dry needling treatments targeting the lateral pterygoid muscle in patients with TMD have reported significant reductions in pain and disability (Cho and Whang, 2010; Dunning et al., 2014; Gonzalez-Perez et al., 2015; La Touche et al., 2010). The evidence for dry needling along with other conservative treatments used for TMD will be further explored in part two of this narrative review.

9. Temporomandibular dysfunction: an appropriate pain model

There are two primary models that account for the chronic pain and loss of function typically associated with TMD, the Vicious Cycle Model and the Pain Adaptation Model. According to the Pain Adaptation Model, pain results in decreased agonist muscle activity and increased antagonist muscle activity in an effort to avoid pain and protect the joint (Peck et al., 2008; Svensson and Graven-Nielsen, 2001). However, studies that have investigated experimental pain do not always result in this pattern of masticatory muscles (Svensson et al., 1996). In fact, experimental TMJ pain in rats has been shown to cause increased EMG activity in muscles associated with mandibular opening and closing (Ro et al., 2002). Given the mounting evidence for a hyperactive inferior head of the lateral pterygoid, it is likely that the Vicious Cycle Model may also play a role in TMD (Murray and Peck, 2007; Peck et al., 2008). According to the Vicious Cycle Model, hyperactive muscle results in excessive acetylcholine (ACh) and subsequent Ca^{2+} release from the sarcoplasmic reticulum, initiating a continuous cycle of localized muscle contraction (Gerwin et al., 2004). The localized hypertonicity begins to block blood flow to the muscle, resulting in a shortage of oxygen and nutrients and leading to ischemia and hypoxia (Gerwin et al., 2004). Local ischemia and hypoxia cause the release of chemicals responsible for propagating pain and inflammation such as bradykinin, prostaglandins, serotonin, calcitonin gene-related peptide (CGRP) and substance P along with a number of inflammatory cytokines, including tumor necrosis factor-α, interleukin 1-β, interleukin-6 and interleukin-8 (Gerwin et al., 2004; Noma et al., 2013; Shah and Gilliams, 2008). There is also a significant drop in pH, which inhibits the action of acetylcholinesterase and the Ca^{2+}-ATPase pump, resulting in hypertonicity and propagating the metabolic crisis (Butts et al., 2016; Shah and

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Gilliams, 2008).

Peck et al. further proposed an integrated pain adaptation model of TMD, which takes into account both biological and psychosocial components of the disorder (Peck et al., 2008). According to Lund (Lund et al., 1991), Nakamura and Katakura (Nakamura and Katakura, 1995), the ventromedial and dorsomedial reticular nuclei set the tone and rhythm of chewing and therefore act as a masticatory central pattern generator (Desmons et al., 2007). The reticular formation provides output to the trigeminal motor nuclei, setting a stereotypical open-close cycle. However, during normal chewing, sensory afferents are continuously sent through the cortex and the brain stem to adjust the mastication pattern, as needed (Desmons et al., 2007; Leite-Almeida et al., 2006). Previous studies in rats have established a connection between the medullary dorsolateral reticular formation to the thalamus and limbic system, suggesting that emotional input may also influence the masticatory pattern (Desmons et al., 2007; Leite-Almeida et al., 2006). Psychosocial factors such as stress and anxiety increase dopamine levels in the reticular formation (Gomez et al., 1999), disrupting the synchronization of the masticatory pattern generator and shortening the refractory period after muscle contraction (Desmons et al., 2007). The result is hyperactive masticatory muscles, which often presents clinically as clenching and grinding. Thus, there are significant psychosocial factors to TMD that may initiate and propagate the vicious cycle model of pain. A number of recent studies have confirmed the relationship between the myofascial pain associated with TMD and stress, anxiety and depression (Akhter et al., 2007; Diracoglu et al., 2015; La Touche et al., 2015).

10. Conclusion

Temporomandibular dysfunction is a complex condition with a multifactorial etiology that typically leads to degeneration and a subsequent imbalance between the articular disc, joint capsule and muscles of mastication. A number of studies in the literature seem to support the pathoanatomic model originally described by Scully, which suggests that the superior and inferior head of the lateral pterygoid are key players in TMD. Both heads of the lateral pterygoid undergo changes associated with the vicious cycle model, the pain adaptation model and the integrated pain adaption model, leading to the discomfort and loss of function associated with TMD.

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