



APPLIED PHYSIOLOGY

Neurological influences of the temporomandibular joint

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Summary This study reviews recent advances in temporomandibular joint (TMJ) or masticatory system related neurology, and suggests the TMJ as a neurological window and lever.

The TMJ is integrated with the brainstem centers via the sensorimotor system, including the body balance and coordination control systems. A dysfunctioning TMJ may reflect not only local problems, but also the underlying remote or systemic problems. Neurological examination, including balance testing, for example, may reveal the contributing imbalances and provide an additional evaluation of the appropriateness of TMJ therapeutics being attempted.

Repetitive or tonic sensory stimulations involving the TMJ may be related to therapeutic interventions, contributing to neural plasticity, which may be adopted as a therapeutic approach in treatment of neurological disorders, including dystonia and movement disorders.

TMJ related therapeutics, such as use of an occlusal splint, cranial manipulation, muscle/myofascial therapy, and acupuncture, ideally need to be practiced along with neurological monitoring, to ensure neurologically desirable effects.

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Introduction

What is the ideal occlusion?

Occlusion, the alignment of the maxillary teeth and mandibular teeth when brought together, is one of the most controversial and continuously

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evolving areas of prosthodontics (Baker et al., 2005). Centric relation (CR) is the beginning of occlusion, and all treatment modalities are based on it (Keshvad and Winstanley, 2000). But there is still no consensus regarding the definition of CR.

One of the current definitions of CR is “the maxillomandibular relationship in which the condyles articulate with the thinnest avascular portion of their respective disks with the complex in the anterior-superior portion against the shapes of the articular eminences.” (Jasinevicius et al., 2000).

Occlusal appliances are commonly used in the treatment of patients with temporomandibular disorders (TMD), and have been reported to improve TMD pain (Wahlund et al., 2003). Several factors influencing these effects have been discussed, such as reduced postural activity in the elevator muscles, elimination or alteration of the influence of noxious proprioceptive input from occlusal interferences, changes of the condyle-fossa relationship, the placebo effect, and the effect of stabilization of the occlusion, as well as an increase in the vertical dimension (Ekberg et al., 1998).

Occlusion related therapies, including use of occlusal appliances, treatment of masticatory muscles and cranial manipulation involve the posture of the temporomandibular joint (TMJ), which is under the control of, and relays proprioceptive signals to, the nervous system. This article reviews some of recent advances regarding the TMJ and tries to suggest a rationale for use of an occlusal splint, or TMJ related therapies, as being potentially useful modalities in the treatment of neurological disorders, including musculoskeletal dysfunctions.

Temporomandibular joint as a neurologic window and lever

The masticatory system is a functional unit composed of the teeth; their supporting structures, the jaws; the TMJs; the muscles involved directly or indirectly in mastication (including the muscles of the lips and tongue); and the vascular and nervous systems supplying these tissues. The importance of jaw movement has become apparent in fixed prosthodontics, periodontics, orthodontics, and in the diagnosis and treatment of pain disorders of the masticatory system (Soboleva et al., 2005). The TMJ serves, to some degree at least, as a window onto the nervous system, as well as a tool for influencing the nervous system. A window onto the

nervous system refers to examination and appreciation, of the orchestrated motor control of the TMJ and related muscles.

The concept of this being a tool for influencing the nervous system refers to the sensory afferentation related to the TMJ, and the tissues in its vicinity.

Motor efferents of TMJ

Mastication is oral motor behavior reflecting central nervous system (CNS) commands, with many peripheral sensory inputs modulating the rhythmic jaw movements. Observation of masticatory movements may be of diagnostic value for assessing disorders of the stomatognathic system (Soboleva et al., 2005). Mastication becomes well coordinated around 4–5 years of age, by which time the primary teeth have erupted. It is believed that each individual has a characteristic basic pattern of masticatory movement (van Eijden and Turkawski, 2001).

Jaw movements are among the most complex and unique movements performed by the human body. The most important muscles for mastication are the temporal (anterior and posterior), the masseter (superficial and deep), the medial pterygoid, the lateral pterygoid (superior and inferior), and the digastric muscles. However, mastication involves far more muscles than these ‘muscles of mastication’, innervated by the trigeminal nerve. Synergistic movements of muscles innervated by facial and hypoglossal nerves are equally important (Soboleva et al., 2005). In masticatory muscles, the organization of motor control is more localized, and the classification of motor unit types is less distinct. These features imply that, in masticatory muscles, a finer gradation of force and movement is possible than in limb and trunk muscles (van Eijden and Turkawski, 2001). The control and rapid coordination of tongue movements is essential for a number of complex orofacial behaviors, such as swallowing, mastication, respiration, speech, licking, gaping, coughing, gagging and vomiting (Svensson et al., 2003). See Figs. 1 and 2.

Masticatory muscles are under the control of masticatory motoneurons.

Jaw muscle motoneurons are activated by three sources: the motor cortex, the central pattern generator (CPG), and the peripheral input (Türker, 2002; Lund, 1991). Unlike homologous left and right muscles in the upper or lower limbs, the masticatory muscles on the left and right side share a common mechanical action around the TMJs and are co-activated during biting tasks (Jaberzadeh

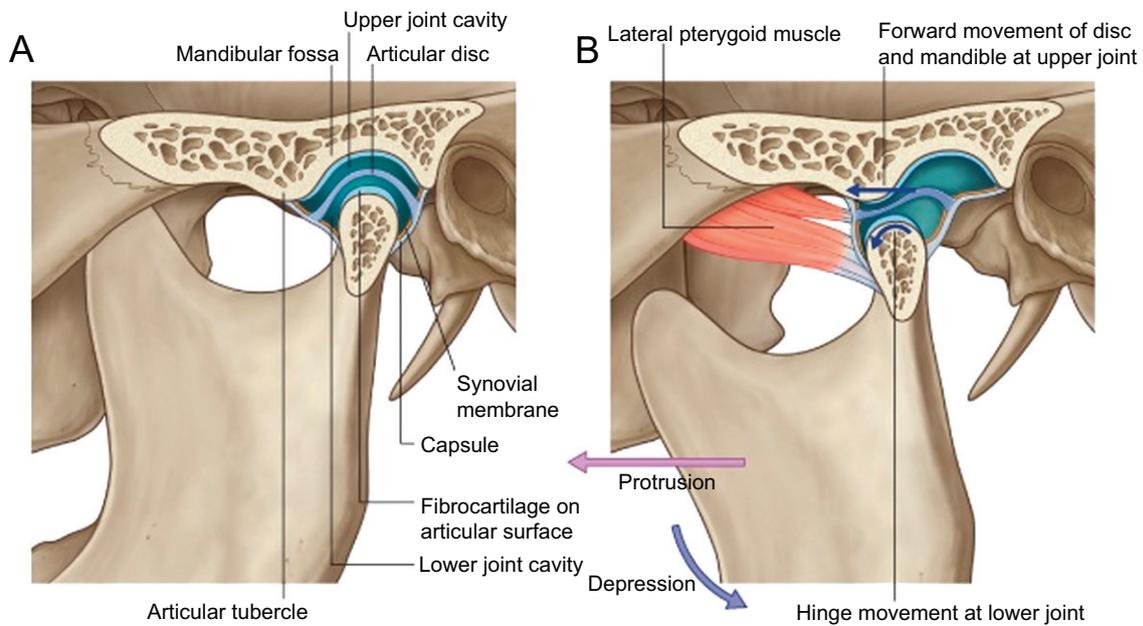


Figure 1 Opening and closing of the temporomandibular joint: from Gray’s Anatomy Student Edition (Blake).

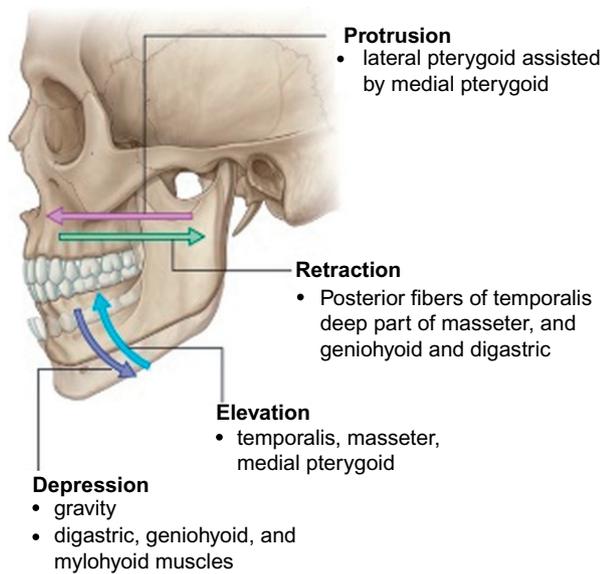


Figure 2 Aspects of movement of the jaw from Gray’s Anatomy Student Edition (Blake).

et al., 2006). For example, the corticotrigeminal projections to the masseter muscle are bilateral, with a stronger contralateral projection (Butler et al., 2001). And there exists synchronization of presynaptic neurons that project to the motoneuron pools (Jaberzadeh et al., 2006). For the normal mandibular posture to be maintained, absolute positional information is needed, requiring calibration of the muscle-spindle afferent information, with the exact time of tooth contact. Brainstem interneurons have been shown to control temporal, spatial, and quantitative aspects of jaw muscle

activity during natural orofacial behaviors (Stohler, 1999), and the cerebellum appropriately alters fusimotor activity and regulates the gain of the spindles in the jaw muscles (Türker, 2002).

The mandibular movements are reported to always be paralleled by head movements (initial head extension, followed by head extension-flexion movements), and in general the start of these head movements precedes the start of the mandibular movements (Eriksson et al., 2000). From observations such as this, close functional integration between the temporomandibular and the cranio-cervical motor systems during natural jaw activities, probably under the control of a common central nervous network, has been suggested (Eriksson et al., 2000). Lateral inclination of the occlusal plane, and imbalance between the right and left masticatory muscles antagonistically, were reported to act on displacement of the cervical spine, compensating for cervical postural control (Shimazaki et al., 2003).

During mastication and biting, the masticatory muscles generate forces that are responsible for the movements and deformations of the jaw and for the production of forces at the teeth and TMJs (van Eijden and Turkawski, 2001).

Injury or irritation occurs when the tissue’s threshold is surpassed by external load. The threshold is dependent on the individual’s level of fitness. A history of too little, or too much, external tissue load will create an environment conducive to tissue failure, and motor control is a key component in injury prevention (Liebenson, 2004). Loss of motor

control involves failure to control joints, commonly because of incoordination of the agonist-antagonist muscle co-activation (Chaitow, 2004). Coordination of agonist and synergist muscles, and not strength, plays a pivotal role in resisting injury (Liebenson, 2004). Such coordination essentially depends on the orchestration of the nervous system. If there is any defect or functional inefficiency of such neural orchestration, micro- or macro-injuries follow, which is, when cumulative, a possible process contributing to pathologic conditions, such as peripheral local lesions involving inflammation, pain, stiffness, or tenderness. In this case, prominent symptoms such as local muscle stiffness or joint pain are superficial presentations of the underlying process, while the motor incoordination-imbalance and resulting accumulation of injuries would be considered as being part of the underlying aetiological process.

TMD symptoms

For example, TMD is a collective term encompassing a number of clinical signs and symptoms of disorders that involve the masticatory muscles, TMJs, and other adjacent structures of the stomatognathic system (Kobs et al., 2005). Signs and symptoms of TMD are common in the population, with a higher prevalence in women than in men. Common clinical parameters include mandibular mobility, TMJ function, pain on movement of the mandible, TMJ pain on palpation and muscle pain on palpation. Oral parafunction, tooth wear, TMJ clicking, and deep bite in childhood have been discussed as possibly significant predictors of increased clinical dysfunction in a long-term perspective (Carlsson et al., 2002). Oral parafunctions such as clenching and bruxism have been suggested as an aetiological or accompanying factor of TMD (Kobs et al., 2005). Direct mechanical stresses loaded by oral parafunctions, associated with an altered motor control of the masticatory muscles, may be involved as an underlying factor in TMD. The altered sensory afferentation induced by oral parafunctions plays a role in developing a distorted pattern of neural organization. The distorted neural organization loses efficacy in conducting a harmonious symphony of muscle functions, or act as an aggravating or a perpetuating factor of TMD.

Sensory afferents of TMJ

The correct execution of a voluntary movement depends crucially also on peripheral sensory feed-

back. Peripheral pathways conveying sensory information project to cortical motor areas. Sensorimotor integration is the process whereby sensory input is integrated by the CNS, in order to assist motor program execution (Abbruzzese and Berardelli, 2003).

The term 'proprioception' was coined by Sherrington to describe the sensory information contributing to a sense of self-position and movement. Proprioception has been related to a distinct class of sensory receptors, most notably those found in the muscles and related deep tissues, in relation to unconscious or more automatic functions. In contrast, kinesthesia, or the conscious sense of position and movement, has been more closely associated with joint and cutaneous receptors (Bosco and Poppele, 2001).

Sensory receptors in the masticatory system comprise epithelial mechanoreceptor afferents, periodontal afferents, TMJ afferents, and muscle afferents including spindle afferents, golgi tendon organ afferents, and others (Lund, 1991). Signals from periodontal receptors are used in the fine motor control of jaw actions associated with biting, intraoral manipulation and the chewing of food (Trulsson, 2006). The jaw-closing muscles, not the jaw-opening muscles, contain muscle spindles, and the spindles have a strong proprioceptive impact on the control of human mastication (Türker, 2002). Oro-facial tissues such as the teeth, facial skin, TMJ and adjacent musculature are mainly supplied by branches of the trigeminal (V) nerve. The V brainstem complex (especially its subnucleus caudalis) may also receive afferent inputs from other cranial nerves such as VII, IX, X and XII, as well as from upper cervical nerves (Sessle, 2006). Functional coupling between the temporomandibular and the craniocervical motor systems have been suggested both at the neuroanatomical and neurophysiological levels (Valentino et al., 2002a; Browne et al., 1993). See Figs. 3–6.

Neck pain and TMD

Significant association between neck pain and temporomandibular symptomatology has also been reported (Ciancaglini et al., 1999). Sensory information from the cervical spine converges with trigeminal afferents within the spinal tract of the trigeminal nucleus, while fibers arriving in the subnucleus caudalis descend further down to C2–C3 and even C6. The superficial sensory distribution of the upper cervical nerves (the ventral cervical roots 2 and 3) also comprises parts of the face, especially the mandibular angle. Segmental

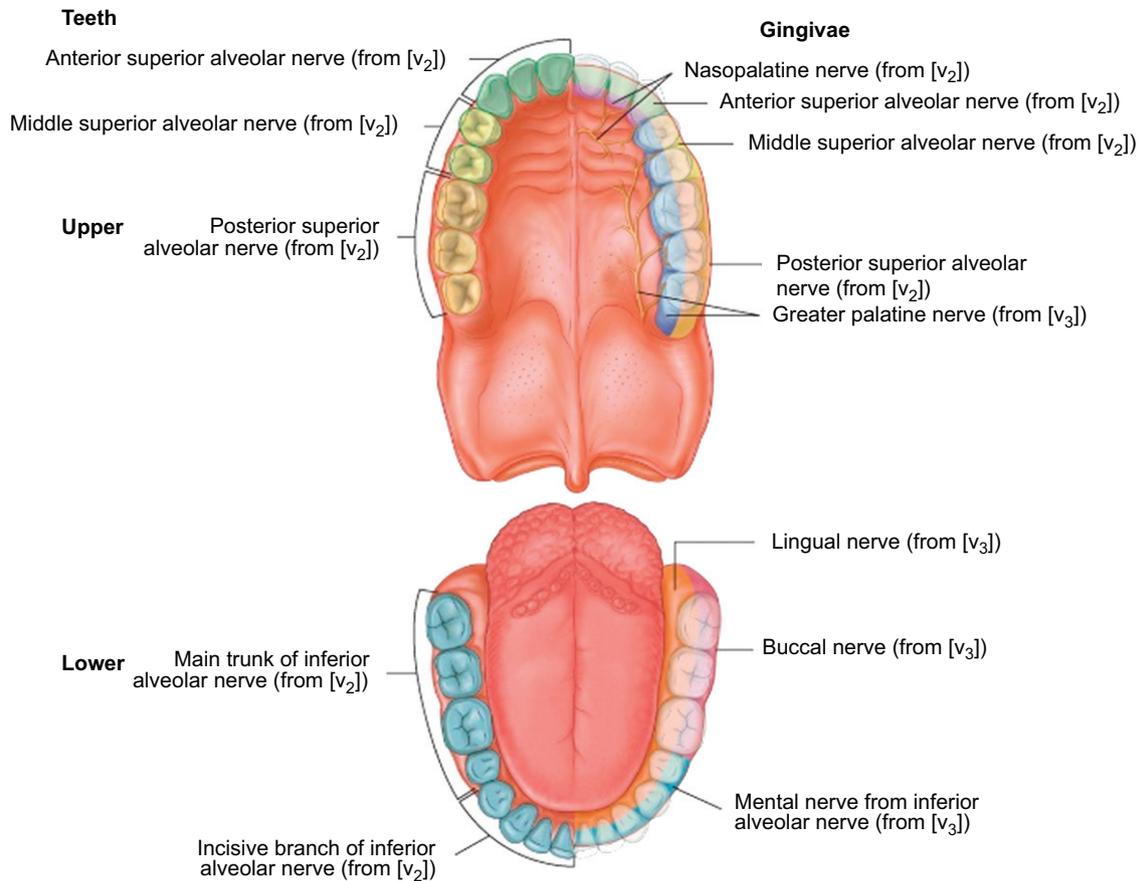


Figure 3 Nerve supply to the teeth and gums from Gray’s Anatomy Student Edition (Blake).

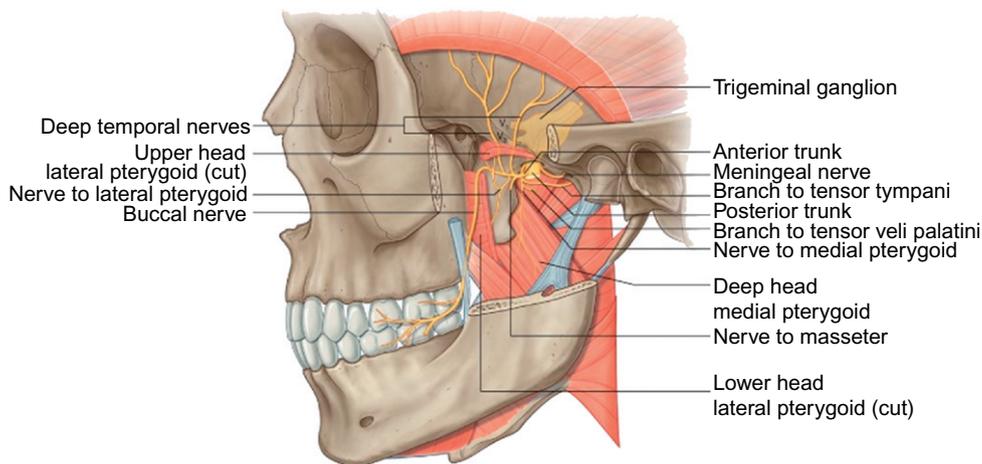


Figure 4 Nerves and muscles of the temporomandibular joint, showing trigeminal ganglion from Gray’s Anatomy Student Edition (Blake).

limitations (especially at the C0–C3 levels) and tender points (especially in the sternocleidomastoid muscle and upper trapezius muscle) were found to be significantly more present in patients with TMD symptoms, than in controls (De Laat et al., 1998).

The trigeminal motor nucleus receives afferents from premotor neurons widely distributed in the pons to the caudal level of the medulla oblongata (Xiong and Matsushita, 2000).

Considering the above-mentioned neural network between cervical muscles and masticatory

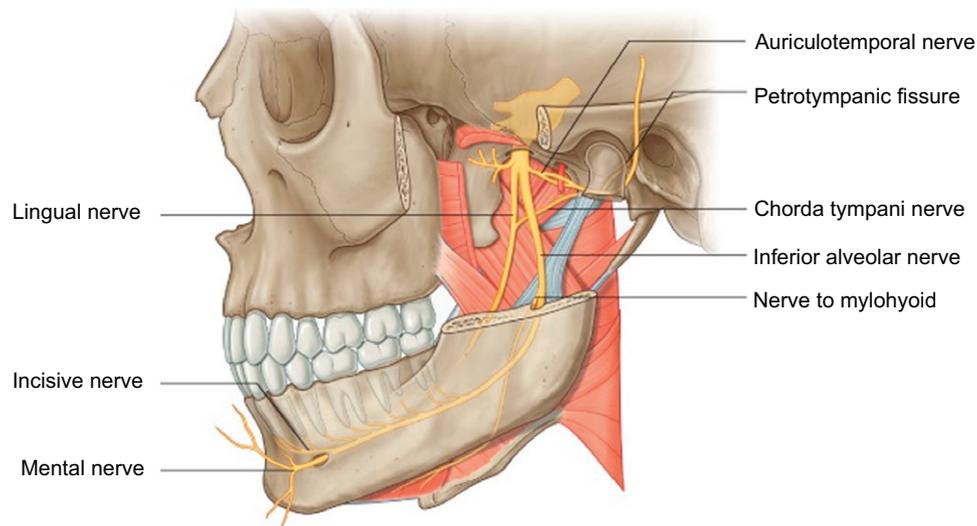


Figure 5 Additional nerves of the jaw from Gray's Anatomy Student Edition (Blake).

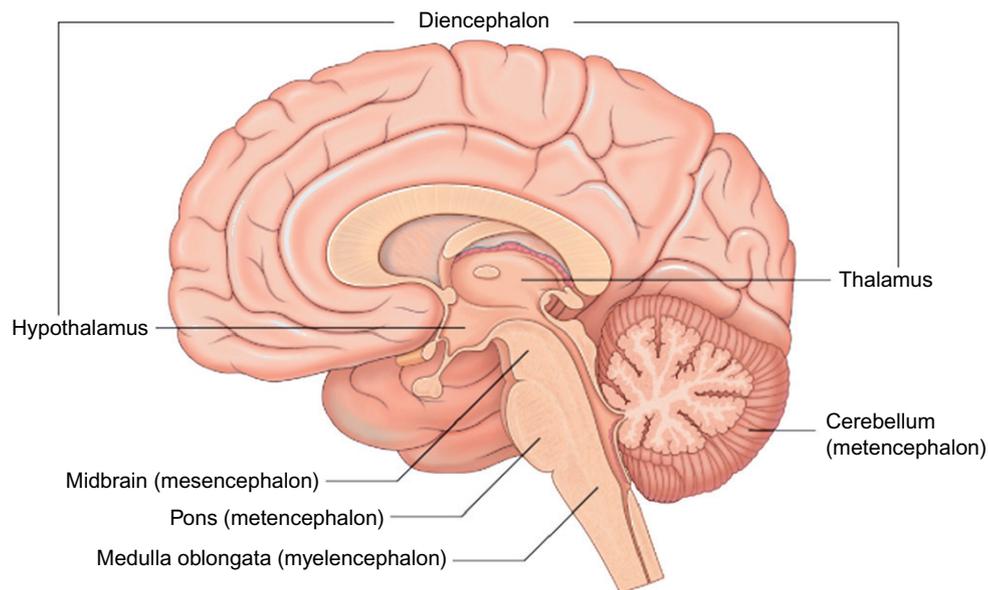


Figure 6 Sagittal section of the brain from Gray's Anatomy Student Edition (Blake).

muscles, occlusal interference or other masticatory problems may be an underlying factor of some chronic cervical complaints. Or, cervical dysfunctions may be an underlying factor of a masticatory or occlusal dysfunction.

Body posture influences

Surpassing the extent of the head and neck, body-posture control has been reported to be significantly related to different mandibular positions, such as centric occlusion (intercuspid occlusion),

resting, or myocentric positions (Bracco et al., 2004). Postural control and gaze stabilization quality have been reported to decrease, from the best to the worst, with splints in CR, intercuspal occlusion and lateral occlusion (Gangloff et al., 2000), although a contradictory report also exists (Perinetti, 2006).

Occlusion related proprioceptive afferents are reported to play a role in posture regulation, balance control, oculogyric stabilization, and sports performance. Dento-muscular-joint afferents of the masticatory system project to the accessory nerve nucleus controlling the sternoclei-

domastoid and trapezius muscles' motoricity. In the vestibular nuclei (VN), sensory information from facial receptors is added to that retrieved from proprioceptive afferents of the neck and body. Neurons in the caudal part of the trigeminal mesencephalic nucleus also project to the VN (Gangloff et al., 2000). Functional correlation between masticatory muscle function, the interdental occlusal plane, and changes of the plantar arch have been reported, supposedly through the connection from the afferent proprioceptive impulse of the muscles governing the configuration of the plantar arch to the trigeminal motor nucleus that innervates the masticatory muscles (Valentino et al., 2002b). In brief, the masticatory system is considered to be neurologically integrated with proprioceptive, visual, balance, and postural control of the whole body.

Sensory experience and plasticity

Plasticity is a property of a self-organizing CNS that is continually optimizing its own performance whether, for example, in sensory processing or information storage (Pinaud, 2004). Plasticity is an obligatory consequence of all neural activity (even mental practice), an intrinsic property of the human nervous system, and a most efficient way to utilize the brain's limited resources and the mechanism for development and learning, as well as being a potential cause of pathology and of clinical disorders (Pascual-Leone et al., 2005). Brain cortical representations are continually shaped by experience. Experimental manipulations of sensory experience can result in a variety of changes in cortical responsiveness, which are generally described as experience-dependent plasticity. Potential mechanisms that underlie such a cortical plasticity include changes in network, synaptic, or cell intrinsic properties. Although it is likely that effects at all three levels contributed to the observed changes in spatial and temporal response properties, the simplest explanation is that sensory experience differentially affected the balance of inhibition and excitation (Kilgard et al., 2001).

Several possible mechanisms underlying cortical reorganization following peripheral sensory deafferentation have been suggested: one is the synaptic alterations of corticocortical connections and another is the reorganization of thalamocortical projection (Masuda et al., 2002). Environmental pressures, functional significance, and experience are critical factors of plasticity (Pascual-Leone et al., 2005). Daily experience essen-

tially leads to the plasticity of the nervous system, while the plasticity necessarily entails differences or limitations according to the nature of experience, individual constitutional factors, pathologic changes or others.

Abnormalities in the peripheral afferent input, or in the brain response to sensory input, may interfere with the processing of motor programs in the cortical motor areas. Dysfunctional sensorimotor integration seems to play an important role in the disturbances of motor control (movement guide, muscle activation) (Abbruzzese and Berardelli, 2003). It has been reported that inaccurate perception of limb position may cause or contribute to abnormal movements in Parkinson's disease (O'Suilleabhain et al., 2001). The duration of a movement disorder, termed pseudo-choreoathetosis of abnormal movements was reported to correlate with the duration of proprioceptive sensory loss, and the abnormal movements were restricted to body parts with proprioceptive sensory loss, suggesting the mechanism of proprioceptive sensory loss and failure to process limb proprioceptive information in the striatum (Sharp et al., 1994). Dystonic patients show a reduced perception of the kinaesthetic sensations induced by muscle vibration, suggesting a dysfunction in the central elaboration of Ia afferent signals. The modulation of sensory cortical responses during preparation for movement was reported to be impaired in focal dystonia. In addition, sensory manipulation is known to modify (induce or attenuate) dystonia (Abbruzzese and Berardelli, 2003). Plasticity has also been discussed in relation to the development and treatment of neuropathic pain (Cooke and Bliss, 2006). Long-term changes in nerve cell activity at the level of the spinal cord and higher centers in the brain (central sensitization) are also a frequent result of nerve excitation or injury (Greene, 2001). Neuroplasticity in central somatosensory pathways may be induced either by an enhanced nociceptive afferent input (e.g. by direct stimulation of peripheral nerves by an injury or by inflammation), or by a decreased afferent input. This central sensitization is thought to contribute to persistent or chronic pain and to the spontaneous pain, allodynia, hyperalgesia and pain spread or referral that characterize many clinical cases of persistent pain following injury or inflammation (Sessle, 2006).

Oral sensations, especially periodontal afferents, are necessary for the control of jaw movement during mastication (Masuda et al., 2002). The details of the representation of the body surface in somatosensory cortical areas are modified by recent behavioral experiences, and the plasticity of

neuromotor mechanisms occurs to accommodate to the altered environment (Klineberg and Murray, 1999). Sensory input itself induces neural plasticity, be it in the cortex or the subcortex area. If the sensory input is appropriate, then a normal and efficient functioning-related neural plasticity would result. When the sensory input is not appropriate, be the cause bad habit, posture or pathologic process in related tissues, distorted plasticity develops, that may further aggravate the undesirable condition.

Sensory afferentations, especially when repetitive or continuous, may accompany a neural plasticity, and plasticity-induced neural reorganization innately leads to altered information-processing and motor output patterns, which may facilitate the learned skill, compensate for the underfunctioning state, or aggravate the undesirable state of the inflicted individual.

In other words, daily experience eventually expresses itself as a plasticity-related altered neurological or behavioral pattern. Posture in daily lifestyle involves a considerable portion of our sensory experience, in that posture involves a tonic proprioceptive sensory afferentation. If posture or daily habit alters, then sensory input changes. The changed sensory experience, via neural plasticity or reorganization, would eventually express itself as an altered neural functioning and motor output.

Therapeutic implications

The challenge is to modulate neural plasticity for optimal behavioral gain, which is possible, for example, through behavioral modification and through invasive and noninvasive cortical stimulation (Pascual-Leone et al., 2005). Repetitive non-invasive stimulation of the brain such as transcranial magnetic stimulation has been shown to exert long-lasting functional effects via neural plasticity mechanism (Cooke and Bliss, 2006). The eye position and head-body angle have been shown to represent important tonic signals for modulating the perception of body movement (Pettorossi et al., 2004).

Considering the neural network of the masticatory system, therapeutic approaches involving the masticatory system may be a tool to influence outside of the masticatory system, and diagnostic observations involving the masticatory system may reflect remote or systemic problems. In applied kinesiology (AK), for example, manual muscle testing or sensory-receptor based therapeutics has been hypothesized as a functional neurological assessment or stimulation method via the neurolo-

gical network of the muscle tested or stimulated (Motyka and Yanuck, 1999). In AK masticatory muscles are usually related with the stomach meridian. According to the acupuncture classic "*Huangdineijing*," the masticatory area is the converging site of life energies from all the meridians (Yin et al., 2005).

Therapies targeting the masticatory system, such as occlusal splints, masticatory muscle work, lifestyle intervention of oral habits, myofascial therapy, cranial manipulation, acupuncture, and others, may have significant neurologic implication via sensorimotor integration with the brainstem, subcortical and cortical centers, cervical region, proprioception, and body posture. If therapeutic approaches induce appropriate neural plasticity, then it is possible that considerable neurologic improvement of the patient may be achieved.

Modulating occlusion related proprioceptive afferents may be adopted as a way of enhancing posture regulation, balance control, oculogyric stabilization, sports performance such as shooting (Gangloff et al., 2000), the configuration of the plantar arch (Valentino et al., 2002b), physical quickness, and back strength (Ishijima et al., 1998), although the reaction may be variable depending on the individual or the stimulation factors (Ferrario et al., 2001).

Occlusal appliances are commonly used in the treatment of patients with TMD and have been reported to improve signs and symptoms in these patients (Ekberg et al., 1998), though systematic reviews have reported that there is a lack of evidence of occlusal splints offering benefit in TMD treatment (Al-Ani et al., 2004; Forssell et al., 1999).

TMJ related therapy such as use of an occlusal splint, episodically leads to dramatic clinical improvement of neurologic conditions such as dystonia (Yin et al., 2006), which might be the result of an appropriate neural plasticity in related sensorimotor systems. It has been reported that isometric strength of the cervical flexors and deltoids increased significantly from habitual occlusion as the vertical dimension of occlusion was increased, then diminished as the dimension was increased further (Chakfa et al., 2002). Varied vertical dimension of jaw posture entails an altered masticatory muscle activity (Michelotti et al., 1997).

Occlusal splint therapy was reported to have a differentially stabilizing effect on the postural contractile activities of jaw elevator and depressor muscles within one week in a study of small sample size (Carr et al., 1991). If the postural activities of the jaw muscles are altered and stabilized, the proprioceptive afferentations from such muscles would also be altered and stabilized. If such

therapy is repeated, or persists for a certain period, appropriate neural plasticity may develop, potentially providing lasting therapeutic benefits.

It is suggested that the result of any therapeutic mandibular condylar position should be an improvement in masticatory muscle function, for example, with respect to balance and activation (Hickman and Cramer 1998). Neurologic testing of balance, for example in a private clinical setting, may provide clinical information as to the appropriateness of the occlusal splint of a given patient. The occlusal splint may accompany additional positive or negative neurologic influences, which may be revealed by neurologic examination such as balance testing. To ensure best service to the patient, such a scenario suggests that a qualified dentist, and/or any bodywork physician/practitioner working with the TMJ or a related area, should be equipped with appropriate neurological examination skills.

Discussions

The concept of the TMJ as a neurological window leads us to consider the possibility that a dysfunctioning TMJ or masticatory muscle may be a reflection of remote or systemic problems. The disharmony of the motoneurons innervating masticatory muscles may eventually lead to the incoordination of masticatory muscles. The motoneurons are integrated with other sensorimotor centers. If the window gets dirty, it might be appropriate to cleanse the window. But if the window and the object over the window gets dirty, it would be best to clean both of them. This suggests it best to adopt an integrative approach comprising direct TMJ therapeutics, as well as other approaches. Therapeutic modalities may be selected, applied, or modified according to the neurological response of the patient. The concept of the TMJ as a neurological lever can provide a neurological base for the foundation of an holistic TMJ approach, expanding clinical application of existing therapeutic approaches involving the TMJ, as well as guiding physicians and practitioners to plan and revise individualized therapeutic interventions, by selecting or modifying appropriate modalities.

In that the TMJ is neurologically integrated with the body, neurological improvement observed in muscular control would be an expanded target of TMJ related therapeutics. In this regard, it would be desirable that therapies involving the masticatory system should be accompanied by close neurological monitoring. Neurological assessment, alongside TMJ related interventions, provide an

opportunity for selecting, reassuring, refining, improving, or dropping the therapeutic modality being tried. On the other hand, TMJ related interventions without neurological monitoring may inadvertently aggravate remote or systemic problems. TMJ related intervention, especially when carried out over a prolonged period, should be tried with caution to avoid any negative neural plasticity. Finally, a neurological improvement and a desirable neural plasticity would be considered to be a contributing factor to the probable answer to the question, 'what is the ideal occlusion?'

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