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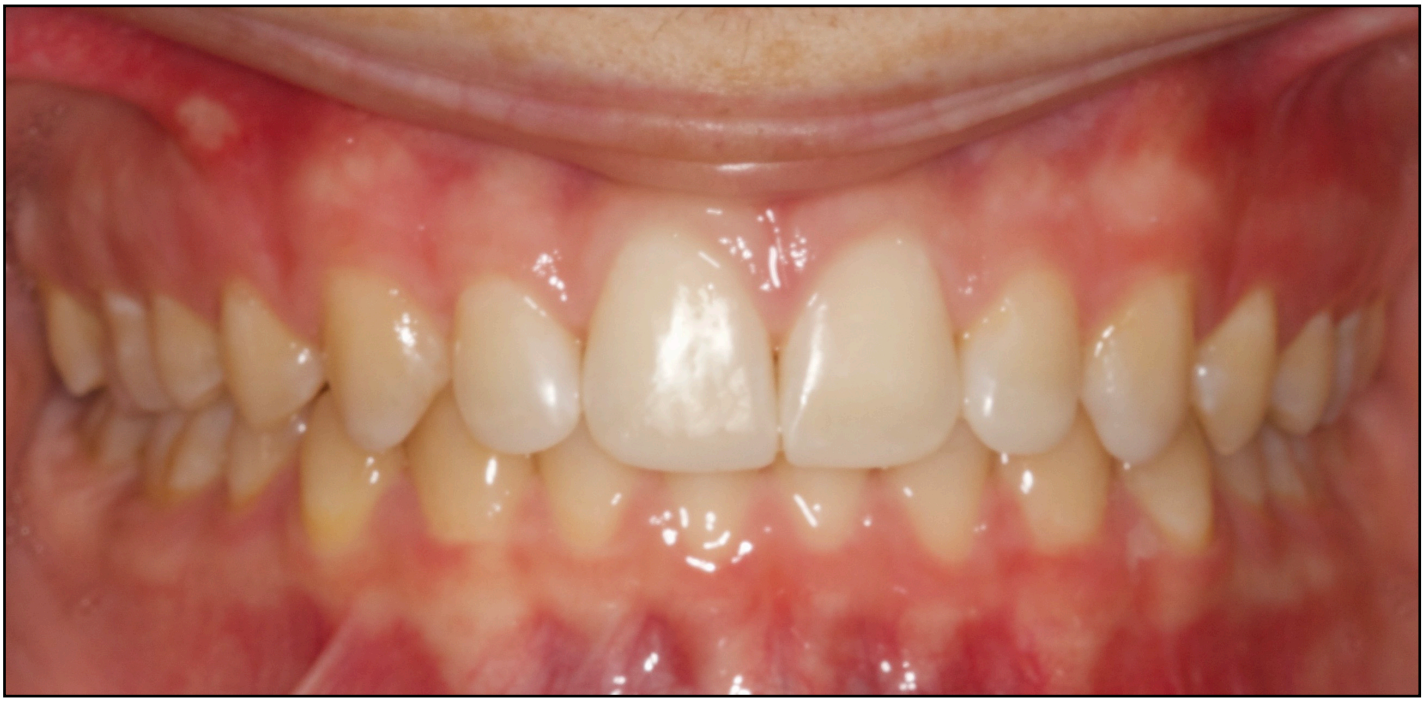

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Teeth are complex organs that have evolved to nourish and sustain life. The ability of human teeth to endure the rigors of a lifetime of masticatory function is based on their durable stone-like structure and on the complex neural machinery that controls them. During mastication, the teeth explore the texture and consistency of foodstuffs before crushing them into small easy to digest pieces. To accurately control the exploratory and destructive functions of the teeth, the brain must obtain accurate descriptions of the mechanical properties of the objects that they encounter. The mechanoreceptive system of the teeth plays a crucial role in providing the tactile sensory feedback necessary for the teeth to endure, relatively unscathed, the rigors of pulverizing the vast quantities of food that humans require for survival into old age. It is ironic, however, that this exquisitely sensitive system also plays an important role in activating the destructive oromotor behaviors that occur when occlusal and central nervous system conditions are pathologic.

DEFINITIONS

Mechanoreception: The unconscious or conscious perception of touch or mechanical displacement arising from stimuli outside of the body, (exteroreception).

Proprioception: The unconscious perception of movement or spatial orientation arising from stimuli within the body itself.

Nociception: The conscious perception of pain from noxious (tissue damaging) or potentially noxious stimuli.

Functional Cortical Map: The representation of somatosensory stimuli in the somatosensory cortex, whereby peripheral sensory neurons have orderly mapped connections to cells in the somatosensory cortex.

Receptive Field: the area on the body surface that has connections to a neuron.

Vibrotactation: The ability to recognize textures using tactile vibration sense.

Stereognosis: The ability to recognize objects by touch alone, using size, shape, texture, weight and other physical features.

Oromotor Behaviors: Activities relating to the muscles of the mouth and/or mouth movements.

Masticatory Efficiency: a) the effort required achieving a standard degree of comminution (as defined in the 8th edition of Glossary of Prosthodontic terms)[1] b) The ability to apply the minimum amount of bite force needed to fracture the food bolus, thereby minimizing stress and damage to the teeth, joints, muscles and supporting orofacial structures (i.e. as defined in this publication by the author).

Centric Relation:
The maxillomandibular relationship in which the condyles articulate with the thinnest avascular portion of their respective disks with the complex in the anterior-superior position against the shapes of the articular eminencies. This position is independent of tooth contact. This position is clinically discernible when the mandible is directed superior and anteriorly. It is restricted to a purely rotary movement about the transverse horizontal axis. [1]

Maximal Intercuspal Position: The complete intercuspation of the opposing teeth independent of condylar position, sometimes referred to as the best fit of the teeth regardless of the condylar position called also maximal intercuspation. [1]
Centric Occlusion: The occlusion of opposing teeth when the mandible is in centric relation. This may or may not coincide with the maximal intercuspal position. [1]

SENSORY MOTOR INTEGRATION

Occlusion (as defined in the 8th edition of Glossary of Prosthodontic terms)[1]:
1: the act or process of closure or of being closed or shut off
2: the static relationship between the incising or masticating surfaces of the maxillary or mandibular teeth or tooth analogues

During the process of jaw closure, the muscles of mastication generate static and dynamic forces that act through the teeth. These forces may function to fracture the food bolus or to stabilize the mandible during swallowing and head movements. Occlusion of the jaws causes maxillary and mandibular teeth to be touched (i.e. by opposing teeth or by an object such as the food bolus). The static and dynamic characteristics of each touching tooth surface (e.g., force intensity direction, velocity, acceleration and vibration components) provide *tactile cues* that activate specialized mechanoreceptive sensory neurons located within the periodontal ligaments and the dentin/pulp complexes of the teeth. During mastication and swallowing, tactile cues arising from the occlusion of opposing tooth contacts serve as timing signals that

trigger deactivation of the jaw closing muscles and activation of the jaw opening muscles.

Once triggered, periodontal and intradental mechanoreceptors convert mechanosensory information into waves of action potentials that are conveyed through the trigeminal ganglion to synapse and communicate with other neurons in the brainstem.[2] Afferent mechanosensory information from the teeth is distributed widely within the brainstem. Some neurons within the brainstem relay mechanosensory information up the neural axis to the thalamus and somatosensory cortex for higher order processing. Others synapse directly with brainstem efferent motor neurons that innervate the muscles of mastication thereby eliciting some of the most rapid reflex behaviors found in the human body.

Efferent neurons in the motor cortex generate descending control signals that project to brainstem motor neurons and the muscles of mastication to affect jaw movements and other oromotor behaviors. These cortically initiated efferent signals, often volitional, are integrated with ascending mechanoreceptive and proprioceptive sensory information from the teeth, muscles, soft tissues and temporomandibular joints (TMJs). The activation of dental mechanoreceptors and their respective afferent signals during biting and chewing is thus integrated with command signals originating in the cortex in order to elicit complex, coordinated jaw movements.

In other words: the *will* to chew comes from the cerebral cortex of the brain, the *rhythms* of chewing are orchestrated in the brainstem central pattern generator (CPG) and *feedback from the teeth* adapts chewing rhythms and bite force levels to the task at hand (e.g. chewing, swallowing and even dysfunctional activities such as bruxism and clenching).

The process of combining sensory signals from the teeth, soft tissues, joints and muscles with central nervous system commands is termed "sensory motor integration".[3] During mastication, sensory motor integration of tactile cues, arising from the texture and consistency of the food bolus modifies chewing and swallowing behaviors. Many oromotor behaviors (e.g. chewing patterns, maximum intercuspation during swallowing and bruxism) are learned and continuously altered as streams of current sensory information are integrated with past behavioral strategies that have been stored in the cerebral cortex.

JAW REFLEXES

Reflex behavior of the jaws has been studied extensively. A number of reflexes have been documented following the mechanical stimulation of the teeth, jaws and

perioral region. These reflexes include jaw-opening, jaw-closing, lateral-jaw, jaw-unloading and jaw-jerk reflexes.[4]

Jaw-opening and jaw-closing reflexes play important roles in the occlusion of the teeth. In human beings the jaw-opening reflex involves rapid inhibition of the jaw closing muscles (i.e. masseter, temporalis and medial pterigoid muscles) and reduction in bite force following a tooth tap. The jaw-opening reflex is thought to serve a protective function in situations where a tooth encounters an unexpected obstacle during chewing. The jaw-closing reflex involves activation of the jaw closing muscles resulting in an increase in bite force. The jaw-closing reflex may function to increase bite force levels to hold and fracture the food bolus

The jaw-unloading reflex serves to prevent opposing teeth from accelerating and forcefully striking each other after the food bolus has fractured and resistance to jaw closing forces has been eliminated. The jaw-unloading reflex is initiated by muscle spindles that are located within the bellies of the jaw closing muscles. Muscle spindles are specialized sensory receptors that detect changes in muscle length. Because they monitor the length and position of muscles and attached tissues, muscle spindles are categorized as proprioceptors.

Yang and Türker (1999)[5] demonstrated that the net response of all jaw muscles to mechanical tooth stimulation depends on the rate of force application (i.e. rapidly or slowly rising rate) as well as background clenching levels (i.e. a 5% of maximal surface electromyogram (SEMG) clenching level or 10% of maximal SEMG clenching level). In this study, rapidly rising tooth stimulation forces always induced the jaw-opening reflex (i.e. net decrease in bite force) regardless of the background clenching level (i.e. 5% or 10% SEMG clenching levels). At lower, 5% SEMG clenching levels, slowly rising tooth stimulation forces induced only the jaw-closing reflex (i.e. increased bite force). Whereas, at higher 10% SEMG clenching levels, slowly rising tooth stimulation forces induced the jaw-opening reflex followed immediately by the jaw-closing reflex (i.e. reduced bite force followed by increased bite force).

In Summary, Yang and Türker's results showed that:

- Rapid tooth contacts *always* inhibit jaw closure.
- When clenching levels are low, slowly applied tooth contacts encourage further jaw closure.
- When clenching levels are high, slowly applied tooth contacts at first inhibit and then encourage jaw closure.

The results suggest that patients who present with centrally initiated clenching and or bruxing behaviors may improperly regulate bite force levels. For example, gentle tooth stimulation at high background clenching levels in bruxing patients would elicit cycles of jaw-opening and jaw-closing reflex behaviors. Whereas, gentle tooth stimulation in normal patients with low background clenching levels would elicit only jaw-closing reflex behavior. The increased numbers of cycles of jaw opening and closing in the bruxing patient would result in a cumulative increases in biting forces and tooth stresses causing accelerated tooth wear and breakdown.

An important secondary finding of Yang and Türker's study is that SEMG recordings of jaw muscles are highly variable and demonstrate poor fidelity in representing jaw reflex behavior. Jaw reflex behaviors are represented with greater fidelity by recording net bite force levels using calibrated bite force sensors. Whereas, SEMG samples only a few myofibril bundles specific to electrode placement, different groupings of myofibril bundles within the masticatory musculature can be recruited to produce equivalent net biting forces, oromotor behaviors or postural positions.

In a similar fashion, shifting your weight without moving your feet recruits different groupings of myofibrils in leg and back muscles without changing the transmission of your net weight to the floor. If only a few bundles of myofibrils were sampled then SEMG data would have very little to do with your net weight or posture. The variability of SEMG data and the inability of SEMG to reflect net muscular recruitment strategies associated with postural changes, urges caution in the use of SEMG in clinical applications where it may be improperly used to establish a new postural jaw position (e.g. so called *Neuromuscular Position*).[6,7]

MECHANORECEPTION OF SKIN

Perception and recognition (i.e. stereognosis) of finely textured objects encountered by the skin relies on the ability to encode and integrate tactile cues arising from the size, shape and roughness of objects that are grasped and handled (i.e. the duplex theory of tactile texture perception).[8,9] The encoding of tactile cues within the skin takes place in slowly adapting (i.e. Merkel's disks and Ruffini's endings) and rapidly adapting (i.e. Meisner's and Pacinian Corpuscles) mechanoreceptors.

Slowly adapting (SA) mechanoreceptors fire a continuous stream of action potentials as long as the stimulus (e.g. touch) remains active. Because they fire continuously during contact, SA mechanoreceptors are best suited for providing an awareness that an object is still in your grasp.

Rapidly adapting (RA) mechanoreceptors fire briefly upon the initiation of a stimulus, shut off quickly and are able to re-fire rapidly in response to a new stimulus. Vibrations are produced as textured objects rub against the surface of the skin. The rapid on/off firing characteristics of RA mechanoreceptors makes them well suited for sensing the vibrations associated with textural assessment.

The somatosensory cortex processes information relating to the shape and textural qualities of objects encountered by SA and RA mechanoreceptors in the skin by creating neural imprints called functional cortical maps.[10,11] When a new object is encountered, the stream of sensory information generated by mechanoreceptors in the skin is compared to a library of previously stored maps widely distributed in the cortex and other brain structures. An integrated motor response may then occur as an appropriate behavioral strategy occurs (e.g. if the sensory information acquired reveals that a glass of water is full, the glass is grasped more tightly and drawn up to the mouth and the water is consumed).

In summary, mechanoreception in the skin involves a "dual channel" process whereby tactile sensory information acquired by both slowly and rapidly adapting mechanoreceptors updates the central nervous system about the physical condition of the surrounding tactile world, enabling coordinated activity to occur.

MECHANORECEPTION OF TEETH

Historically, the tactile function of the teeth has been broadly ascribed to periodontal mechanoreceptors. However, many studies now indicate that intradental mechanoreceptors play an important role in contributing to the exquisitely sensitive tactile and vibrotactile abilities that are required to efficiently assess texture and perform precise oromotor behaviors.

The primary mechanoreceptors in the periodontal ligament are mainly Ruffini endings, which are categorized as low-threshold, slowly adapting (SA), type II mechanoreceptors.[12] Most periodontal mechanoreceptors are tuned to specific force and directions of force application enabling them to reliably encode tactile information about force direction.[13] In monkeys, periodontal mechanoreceptors are more numerous around the anterior teeth than the posterior teeth,[14] which may in part account for reports that indicate that tactile thresholds of anterior teeth are lower than posterior teeth.[15,16]
In 1955, experiments performed by Lowenstein and Rathkamp[14] compared the tactile sensory thresholds of nonvital (i.e. root canal treated teeth) to vital teeth and found that the tactile thresholds of nonvital teeth were 57% higher than the thresholds of

contralateral vital teeth. They concluded that a specialized mechanosensory mechanism that is located within the teeth contributes to the tactile sensitivity of the teeth. In 1975, a study by Linden[17] failed to identify differences in thresholds of vital and nonvital teeth. Even though the two studies used radically different modes of tooth stimulation, the more recent study by Linden served to sway the scientific community into supporting the hypothesis that periodontal mechanoreceptors serve as the principle receptors of tactile tooth stimulation.[18]

Once thought to contain only Aδ and C pain fibers, recent physiologic investigations have discovered that the tooth pulp also contains more rapidly conducting Aβ mechanoreceptive nerve fibers. Using methods that stimulate nerves within the teeth and determine the elapsed time for impulses to propagate over measured lengths of nerve fibers, physiologists have shown that some intradental afferent nerves possess very rapid conduction velocities.[19] These rapidly conducting intradental nerve fibers conduct impulses faster impulses than Aδ and C pain fibers and are categorized as Aβ mechanoreceptive fibers.

Dong and Chudler (1984)[20] used electrophysiological recording techniques in cats to measure the elapsed time for impulses to travel up the neural axis from stimulated intradental nerves through the brainstem, thalamus and somatosensory cortex. They determined that some intradental afferents convey mechanosensory information all the way to the somatosensory cortex at rapid Aβ conduction velocities and signal transmission over the trigeminal leminiscal pathway.

Dong et al (1985)[21] mechanically stimulated canine teeth in cats and recorded from intradental afferent nerves in the trigeminal ganglion. Rubbing sandpaper of different grit size on the tooth evoked distinct frequency encoded discharge patterns in ganglion cells. This suggested that intradental mechanoreceptive afferents have the ability to faithfully encode mechanical vibrations. In a later study Dong et al (1993)[22] recorded from both intradental and periodontal mechanoreceptors in the trigeminal ganglion. They found that rapidly adapting, fast conducting Aβ intradental mechanoreceptors encoded a wide frequency range of tooth vibrations and that periodontal mechanoreceptors were able to encoded only lower frequency vibrations. Intradental mechanoreceptors had rapidly adapting response characteristics and did not exhibit preferred direction sensitivity whereas the response magnitude of individual periodontal mechanoreceptors depended on the direction of force application.

Olgart et al (1988)[23] monitored intradental nerve activity and the jaw-opening reflex in cats in response to bending forces applied to a canine tooth. Their results

demonstrated that bending forces applied to vital teeth evoked the jaw-opening reflex and that subsequent endodontic procedures abolished this reflex behavior. They concluded that a specialized sensory transducer mechanism exists in dentin and that it is activated by deformation of the clinical crown of a tooth.

Vibration perception threshold testing of vital and nonvital human incisors in this author's laboratory suggests that low threshold intradental mechanoreceptors are capable of encoding vibrotactile tooth stimulation.[24,25,26,27] In a series of psychophysical experiments the ability of subjects to detect vibrations over a wide range of stimulation frequencies was tested.

The results of these vibrotactile experiments indicate that:

- Maxillary and mandibular incisors have similar vibration perception thresholds between 40 and 315 Hz.
- Nonvital maxillary incisors have higher vibration perception thresholds than vital maxillary incisors between 40 and 315 Hz.
- Maxillary incisors have a "U" shaped vibrotactile threshold tuning curve between 10 Hz and 315 Hz and are most sensitive to vibrations at approximately 55 Hz..

We surmised that RA, fast conducting, A-β nerve fibers within the teeth are activated when teeth are subjected to mechanical vibrations because subjects were able to detect vibrations at low amplitudes through vital teeth and not through nonvital teeth. Subjects with nonvital teeth suffered sensory deficits in vibrotactile sensation. Without intact vibration sensitive mechanoreceptors within their teeth, the ability to perceive and assess finely texture objects would likely be compromised. Furthermore, the absence of intradental mechanoreceptors in nonvital teeth may unwittingly allow the use of excessive biting forces which could lead to excessive tooth wear and even catastrophic fractures of these teeth over a period of time. This hypothesis is supported by the excessively high fracture rate associated with nonvital teeth reported by Aquilino and Caplan (2002).[28]

These studies suggest that dental mechanoreception is remarkably similar to mechanoreception in the skin. As is the case in the skin, dental mechanoreception involves a "dual channel" process whereby tactile sensory information is acquired by slowly adapting mechanoreceptors located in the periodontal ligament and rapidly adapting mechanoreceptors located within the dentin/pulp complex of the tooth. These two channels of dental mechanosensory information update the central nervous system about the texture and physical characteristics of the food bolus as

well as the topography and texture of interacting tooth forms during occlusion of the jaws.

The data obtained in this author's laboratory revealed that the frequency range of dental mechanoreceptors is tuned to somewhat lower frequencies range than skin mechanoreceptors. Whereas, the skin relies on Paccinian and Meisner corpuscles for vibrotaction, the teeth rely on intradental and periodontal mechanoreceptors. Our data indicated that the most sensitive frequency for dental vibrotaction through a maxillary incisor is tuned to approximately 55 Hz. The most sensitive frequency for skin vibrotaction is tuned to between 150 and 200 Hz.[29] This difference in tuning may arise from biophysics and stiffness properties of two physically different sensory systems. Being tuned to lower frequencies than the skin may allow for better perception of specific surface textures or objects using the teeth. For example, the calcified surface of a pearl is perceived as smooth when touched by a fingertip and gritty when touched by a tooth.

In the light of these findings the teeth may be considered to be tactile sensory organs. In this capacity the entire dentition may further be considered to be a tactile sensory array. All vital teeth in this array are able to convey rich textural and force information that describes the physical properties of solids, liquids or gases that they encounter. This information is integrated with streams of sensory information elicited from mechanoreceptors in the tongue and oral mucosa as well as proprioceptors located in the masticatory muscles and the TMJs. Armed with years of experience in the form of functional cortical maps (i.e. many acquired from the early years of childhood exploration), new streams of textural and force information are compared with previous experiences and integrated to establish oromotor behaviors.

OROMOTOR BEHAVIORS

The ability to position the jaws and accurately regulate biting forces relies on the sensory motor integration of a wide range of sensory input from mechnoreceptors, proprioceptors and nociceptors in the orofacial region. Sensory input from the visual system is also integrated to mediate assessment of foodstuffs and preplanning of chewing behaviors. Once planned, tooth contacts have been initiated, sensory information from intradental mechanoreceptors, periodontal mechanoreceptors and nociceptors in and around the teeth plays a critical role in adapting chewing behaviors to specific physical properties of the food bolus and opposing tooth morphology.

Biting:

Trulsson and Gunne (1998) observed "striking disturbances in the control of certain jaw motor behaviors in subjects lacking periodontal receptors".[30] This study compared the oromotor performance of edentulous subjects that were restored with complete dentures or fixed full arch implant supported prostheses (i.e. in both the maxilla and mandible) to subjects with natural teeth by measuring the forces that were generated to hold a peanut between maxillary and mandibular incisors. The subjects in the 'denture' or 'implant' groups averaged using four times the force to hold the peanut than subjects in the 'natural' teeth group. The elevated forces utilized by the 'implant' and 'denture' groups were comparable to those used by subjects in 'natural' teeth group after local anesthesia was administered to their teeth. Moreover, because the subjects in the 'denture' or 'implant' groups could not feel the peanut they found it difficult to coordinate the simple task of holding it between the incisal edges of their prosthetic teeth resulting in the peanut frequently escaping.

Trulsson and Gunne (1998) broadly categorized all dental mechanoreceptors involved in the tactile perception of the peanut as periodontal receptors even though their study was not designed to determine specific receptor location (e.g. intradental or periodontal). Nonetheless, their study clearly shows the important role that dental mechanoreceptors play in the fine motor control of tooth contacts. Because they showed that patients with dental implants use greater than normal biting forces, these findings imply that implant supported prostheses would be expected to suffer greater rates of wear and fracture than natural teeth. Implant supported prostheses would therefore require that protective measures be taken to minimize the risk of damage.

Occlusal equilibration and occlusal splints are two protective measures that may be used to minimize the risk of prosthetic tooth fracture. These procedures reorganize bite force vectors on the teeth thereby altering the tactile cues that guide jaw closing behavior. Additionally, occlusal splints redistribute bite force vectors evenly among the teeth they overlie masking them from tactile sensations thereby minimizing jaw reflex activation (e.g. jaw opening or closing reflexes). When properly used, both of these procedures generate clear tactile cues that favorably redirect oromotor behavior during the occlusion of the jaws.

Nocifensive Behaviors:

Pain fibers (from Aδ and C nociceptive neurons) located in the dentin-pulp complexes of the teeth provide an additional channel of sensory input to the teeth. These fibers signal warnings of damaging or potentially damaging forces or temperatures encountered by the teeth. Mild activation of pain fibers produces pre-pain sensations[31] that can warn of mechanical dangers. Pain and pre-pain can lead to powerful avoidance behaviors (i.e. nocifensive reflexes).[32] Forward posturing of the

mandible to avoid a painful molar is an avoidance behavior that may be seen in clinical practice. If this pain is not eliminated in a timely manner, chronic habitual forward posturing of the mandible to avoid the painful tooth can lead to anterior tooth damage (e.g. wear, fracture, mobility, orthodontic tooth movement).

Centric Relation:

Centric relation (CR) is a postural position of the mandible that is achieved when inferior and superior heads of right and left lateral pterygoid muscles are in their resting state and are not being activated by motor commands from the central nervous system.[33] Dawson suggested that an occlusion will experience long-term stability when centric occlusion occurs coincidently with approximately equal intensity contacts of all teeth, without contraction of the lateral pterigoid muscles. (i.e. articulation of the teeth coincides with CR condylar position).[34]

Eriksson and Zafar[35] ond others[36] described how during chewing both the mandible and maxilla rotate about different axes. Makofsky expanded on the concept of Centric Relation by describing how the maxilla moves during opening and closing movements based on the arthrokinematics of the atlanto-occipital joints. (i.e. AOJs).[37] He described in his "Sliding Cranium Theory" that the motion of the maxilla is based on rotations and slides of the cranial condyles bearing on the superior articular facets of the atlas. The arthrokinematic movements of the AO joints result in the cranium and maxilla sliding and rotating during opening and closing movements of the jaws. The effect of cranial slides on the atlas is to change the position of the mandibular condyle in the TMJs and the positions of occluding tooth contacts. In other words, Makofsy points out that CR is dependent on head posture.

In an unstable occlusion a single pair of interfering molars may be the only occlusal contact that occurs when the mandible is positioned in CR. Whereas a single molar may be strong enough to resist gentle biting forces, it may not have sufficient strength to endure the stronger forces needed to chew and swallow. During swallowing, isometric contraction forces of the mandibular elevator muscles stabilize the mandible against the cranium to resist forces generated by the suprahyoid muscles. This occurs either with concurrent occlusal clenching or biting with the tongue being interposed between the teeth. Clenching forces during swallowing and chewing range between 13 and 26 lb.[38]

The activation of mechanoreceptors and nociceptors in and around interfering molars initiates protective oromotor behaviors. One or two swallows or bites taken with the jaws postured in centric relation may be all that is needed for interfering molars to be overloaded and stressed to the point of generating pre-pain sensations. Once

warned of potentially damaging tooth contacts in CR, the cerebral cortex generates oromotor behaviors that search for jaw positions where functional closing behaviors (e.g. chewing or swallowing) can be accomplished without causing excessive tooth forces or pain. These behaviors frequently involve lateral pterigoid muscle activation leading to protrusion into a maximal intercuspal position (MIP) jaw posture. In time, chronic protrusion into MIP from CR occlusion can lead to traumatized anterior teeth, sore masticatory muscles, headaches and temporomadibular disorders (TMD).

Excursive Jaw Behaviors:
Lateral and protrusive mandibular excursions are coordinated jaw movements occurring with the teeth in contact. They are generally initiated in the cerebral cortex and are modified by sensory motor integration of afferent signals from a wide range of proprioceptors and mechanoreceptors in the orofacial region. Afferent mechanosensory information from intradental and periodontal mechanoreceptors is important to the execution of these movements as patients lacking these receptors use much higher functional bite force levels.[30]

Occlusal interferences are "tooth contacts that inhibit the remaining occluding surfaces from achieving stable and harmonious contacts".[1] Occlusal interferences compete with canine tooth contacts for control of the masticatory muscles during excursive jaw movements. Canine guidance, anterior guidance and group function are excursive jaw behaviors that depend on the sensory motor integration of tactile cues arising from interactions of moving maxillary and mandibular natural or prosthetic tooth surfaces. Streams of mechanosensory information are continuously generated by rapidly and slowly adapting dental mechanoreceptors as each contacting tooth rubs past the tooth that opposes it during excursive jaw movements. As more teeth are encountered during excursive movements (e.g. group function or nonworking side interferences), more mid-course corrections are needed to complete the excursion. As additional teeth come in contact, each tooth increases the complexity of the stream of mechanosensory information that is processed to complete the movement.

In disorganized, maloccluded dentitions force vectors on teeth continuously change magnitude and direction as cusps engage opposing cusps and rub over varied tooth topography during jaw excursions. Changing force vectors produce concomitant changes in the mechanosensory signals produced by intradental and periodontal mechanoreceptors. The integration of these changing streams of sensory signals acts as a feedback mechanism that continuously modifies motor commands that evoke muscle contraction patterns that effect jaw movements. Occlusal schemes that have fewer interacting teeth during excursive jaw movements have less mechanosensory

information to process (e.g. canine guided occlusions) and are able to complete excursive movements more rapidly. Kerstein (1994)[39,40] has reported that the elimination of working and nonworking side interferences using occlusal equilibration methods leads to faster lateral jaw movements (i.e. disclusion time reductions) and reduction of myofascial pain symptoms.[41] In occlusal schemes with multiple working and nonworking side occlusal interferences, an excessive amount of mechanosensory feedback from multiple tooth contacts may cause muscle hyperactivity, jaw pain, elevated biting forces, slower jaw movements and excessive tooth wear.

Occlusal equilibration of pre and post canine lateral tooth interferences eliminates competing tactile sensory information from the interfering teeth, thereby simplifying the muscle recruitment strategies that are required to execute excursive jaw movements. This therapeutically established canine guided occlusal scheme allows decisive, rapidly executed excursive jaw movements to occur as observed by Kerstein (1994, 1997).[35,36,37,42]

SUMMARY

One of the primary goals of occlusal therapy is to improve oromotor behaviors so that stress, strain and pain in all components of the masticatory system are minimized. These approaches typically alter tooth morphology and position or use occlusal splints to mask tooth contacts. Altering the morphology and position of occluding tooth surfaces (i.e. using a variety of techniques including occlusal equilibration, orthodontics and prosthetics) changes the sensory stream that is integrated by the central nervous system during the occlusion of the teeth, thereby altering oromotor behaviors. Changes made to the occlusion (i.e. occlusal program as defined by Dr Niles Guichet[43] in this manual), either intentionally through occlusal therapy or accidentally by fabricating high or low dental restorations, causes altered streams of sensory information to be integrated into new oromotor behaviors.

Dr Niles Guichet coined the term “neuromuscular release” to explain his clinical observation that elimination of CR occlusal interferences causes more rapid jaw closing behavior. And while the research presented in this chapter has provided a clearer understanding of the general processes and pathways responsible for this behavioral change, a great deal of additional scientific study is needed.

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Vibration perception thresholds of human maxillary and mandibular central incisors

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KEYWORDS
Vibration perception thresholds; Psychophysical thresholds of teeth; Oral sensory function; Dental mechanoreceptors; Normative data

Summary Tactile information from dental mechanoreceptors contributes to the perception of food bolus textures and the control of mastication. While numerous studies have measured the light-touch sensory thresholds of teeth, little information is available about the vibrotactile perception thresholds of teeth. This study uses an adaptive psychophysical procedure to determine thresholds of vibratory stimulation of maxillary and mandibular central incisors in 16 healthy human subjects. An electromechanical vibrator delivered labiolingual forces perpendicular to the long axis of the maxillary and mandibular incisors at 10 stimulation frequencies between 40 and 315 Hz. The median thresholds ranged between 44 and 104 mN. A linear regression analysis revealed a significant increase in the vibrotactile thresholds with increasing frequencies for stimulation of the maxillary and mandibular incisors. No significant differences were found between regression slopes of the thresholds of the maxillary and mandibular incisors. These results indicated that maxillary and mandibular incisors should be able to discriminate effectively among a variety of textures based on their ability to encode a wide range of vibration frequencies.

Introduction

Tactile sensory information from dental mechanoreceptors contributes to the perception of form, texture, and hardness of a food bolus within the oral cavity and to the motor control of the mandible during mastication.[1–3] Abnormalities of the tactile sensitivity of teeth may decrease oral stereognosis[4] and increase bite force.[5,6] Numerous psychophysical studies have attempted to determine the tactile thresholds of teeth to mechanical stimulation [see reviews by Jacobs and van Steenberghe[7] and Linden[1]]. It is generally agreed that most of the mechanoreceptors for light touch sensation involve the slowly adapting mechanoreceptors that are located within the periodontal ligament.[2,8] However, neurophysiological studies in the cat indicate dental mechanoreceptors can also encode vibrotactile stimulation of the teeth[9–11] that likely would involve rapidly adapting mechanoreceptors.[10]

Vibrotactile stimulation of the skin has been implicated in the perception of textured surfaces of objects and as part of the diagnoses of various neuropathies,[12–14] but little information is available about the vibrotactile perception thresholds of

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teeth. Jacobs et al.[15] measured the vibrotactile thresholds of canine teeth at 32, 128, and 256 Hz in three human subjects. The subjects detected all three frequencies, although the thresholds ranged from 85.6 to 105.9 g (839.4–1033.6 mN), which was considerably higher than the reports for thresholds of the light touch mechanoreceptors.[7] Jacobs et al.[15] also found that the threshold at 128 Hz was significantly lower than the thresholds at 32 and 256 Hz, which suggests that dental mechanoreceptors encoding vibration may have a U-shaped, tuning curve similar to Pacinian corpuscle receptors located in the skin.[16] The purpose of this study was to determine the frequency-tuning curve for vibratory mechanoreceptors of human central incisors to mechanical stimulation of a series of frequencies between 40 and 315 Hz.

The vibratory mechanoreceptors may be affected by the biomechanics of the periodontal ligament, since large morphologic differences exist in the root surface areas among different tooth types. Different response characteristics of the biomechanics of the periodontal ligament may explain why the tactile thresholds of anterior teeth are lower than posterior teeth.[17,18] There also are large differences of the root surface areas between maxillary and mandibular teeth. For example, the mean root surface area of maxillary central incisors is approximately 32% larger than the area of the mandibular incisors.[19] By comparing the vibrotactile thresholds of maxillary and mandibular central incisors in this study, the possible influence of tooth root morphology on the dental mechanoreceptors to vibration may also be revealed.

Materials and methods

Subjects

Vibration perception thresholds were determined for a vital maxillary central incisor (#8 or 9) and a vital mandibular (#24 or 25) central incisor in 16 healthy human subjects (13 males and 3 females, 23–35 years of age). The tested teeth were free of dental restorations and the subjects had no dental or orofacial pain, healthy periodontal tissue, and no evidence of peripheral or central neurological disorders. The central incisors were also assessed for vitality with an electric pulp tester (Model 2001, Analytic Technology, 1717 West Collins Orange, CA, USA) and were responsive within the normal range of stimulus intensities. No subjects were excluded based on occlusal relationship, although one subject (# 36) presented with an anterior open-bite occlusal relationship that prohibited anterior tooth contact in maximum intercuspation and in other jaw positions. The Institutional Review Board of the Oregon Health and Science University approved the experimental protocol, which was explained to the subjects who gave their written, informed consent to participate in this investigation.

Apparatus

Fig. 1 shows the components of the experimental set-up. An acrylic stimulus probe with a 2 mm diameter tip was used to deliver labio-lingual forces to the tooth. An electromechanical stimulator (Ling Dynamic Systems Ltd., Heath Works, Baldock Road Royston, Herts SG8 5BQ, England, UK) was used to generate calibrated mechanical stimulus forces. A frequency generator with the impedance matched to the recording attenuator (Model E326A, Grason-Stadler, 5225 Verona Road, Madison, WI, USA) drove the electromechanical stimulator. Dynamic forces were monitored with a piezoelectric force transducer (Model 8001, Bruel & Kjaer, Heinrich-Hertz-Strasse 26, Langen, D-63225, Germany) electrically coupled to a charge amplifier (Model CH-1100, Ono Sokki, 1-16-1 Hakusan, Midori-ku, Yokohama, Japan). Calibration of the piezoelectric force transducer was performed periodically using a reference vibration signal source, which consisted of an electromechanical exciter driven by a stabilised oscillator at 159.2 Hz (Model 84294, Bruel & Kjaer, Heinrich-Hertz-Strasse 26, Langen, D-63225, Germany). The static force of the probe was monitored using a load cell system that included a digital meter and a power supply (ELFS-T3E-2L/RQ and MM50, Entran Devices Inc., 10 Washington Avenue, Fairfield, NJ, USA) and periodically calibrated using known masses. Stimulus force profiles were displayed on a digital storage oscilloscope and the subjects' responses were recorded on strip chart paper. All data were also recorded on magnetic tape.

Procedures

The 90-min experimental sessions included a brief clinical examination and the experimental testing of a maxillary and a mandibular central incisor. After a brief review of the subject's health history, the clinical exam consisted of an inspection of the teeth and periodontal tissues, and palpation of the temporomandibular joints. The data collection took place in a quiet room with the subjects reclined comfortably in a dental chair. The subject's incisors were held in an orthogonal relation to the stimulating probe. The stimulus probe and the teeth were held in a stable relation with silicone rubber impression material (Blu Mousse, Parkell, Farmindale, NY,

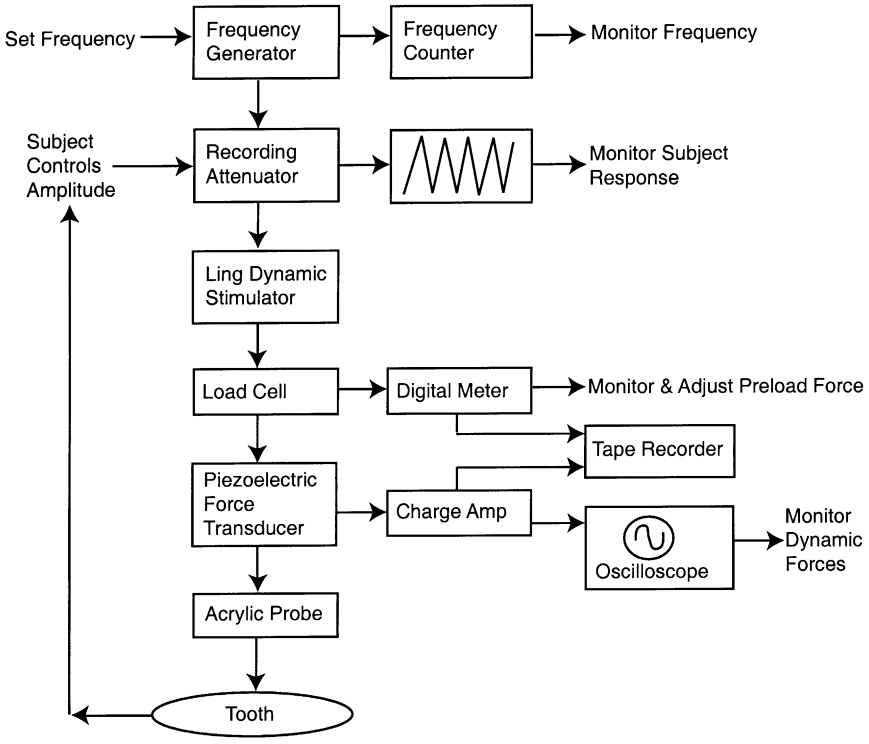

Figure 1 The experimental apparatus consisted of a Ling electromechanical stimulator that was controlled by a frequency generator, in which the experimenter randomly set the frequencies, and an attenuator, which was under the control of the subject. Between the electromechanical stimulator and the acrylic stimulus probe was a load cell, which was used to set a preload force on the tooth, and a piezoelectric force transducer that measured the dynamic stimulus force.

USA) that was attached to a stainless steel bite-fork, which was connected to the stand supporting the stimulus probe. The impression material was trimmed from the central incisors, thereby, allowing the incisors to move freely in response to mechanical stimulation forces.

The stimulus was applied 3 mm from the middle of the incisal edge at force levels between 0.0 and 637 mN. During the application of dynamic stimulation forces, contact of the stimulus probe with the tooth was maintained with approximately 150 ± 50 mN static force, which was based on pilot data that showed lower static force levels resulted in a loss of tooth-probe contact at all but the highest dynamic force levels. Most studies of vibrotactile sensitivity of skin surfaces employ some preload force, although high contract force (e.g. >490 mN) can lead to lower thresholds.[20,21] During data collection, the subjects wore isolation headphones and listened to white noise to mask any auditory components of the test stimuli.

Sinusoidal stimulation forces were presented at ten randomly assigned frequencies between 40 and 315 Hz in 1/3 octave intervals. Subjects were given several practice trials to learn to quickly depress a switch when they first felt the vibration of their tooth. Vibration perception thresholds of maxillary and mandibular incisors were determined using a modification of the von Békésy[22] adaptive psychophysical method, which enabled us to determine the vibration perception thresholds of teeth for a wide range of frequencies within a relatively short time. This stimulation titration method requires the subject to continuously adjust the stimulus amplitude to converge on the upper and lower limits of the stimulus threshold, which is different than the ascending method of limits or the staircase methods where the experimenter adjusts the stimulus amplitude each time the subject does or does not respond to the stimulus. With the von Békésy psychophysical method, the subjects controlled a recording attenuator so that when they sensed the vibration of the tooth they depressed a switch, which decreases the stimulus amplitude, and they released the switch upon cessation of sensation, whereupon the attenuator would increase the signal amplitude until the subject again detected the stimulus. At each frequency tested, the subjects continually adjusted the stimulus amplitude to the high and low limits of threshold range, which typically required about five high and low amplitude oscillations before the subjects achieved a stable level. Once the subjects' high and low oscillations stabilised, three high and three low stimulus

amplitudes were measured. The midpoint of the high-low excursion was considered as the vibration perception threshold.[23] A 5-min rest period was provided between the testing of the maxillary and the mandibular incisors.

Data analysis

The means and standard deviations (S.D.) were calculated for thresholds of the maxillary and mandibular teeth for each subject at the 10 test frequencies. A linear regression analysis was used to measure changes across frequencies or between tooth types. An *F* test was used to test the probability that the slope of the linear relationship between threshold and frequency was different than zero (no change) and the differences between the threshold slopes of maxillary versus mandibular incisors. A probability value of less than 0.05 was considered to represent a significant difference.

Results

Across all frequencies and for both tooth types, the vibrotactile perception thresholds were mainly between 44 and 104 mN of peak force, with a few subjects having thresholds between 108 and 441 mN. Consequently, the distribution of responses was skewed toward the lower threshold values, so the data are presented as medians and percentiles (Table 1).

The main question of this study was to determine the type of relationship that exists between the frequency of stimulation and the vibrotactile perception threshold for either the maxillary or mandibular teeth. A linear relationship was found between the stimulus frequency and the thresholds for stimulation of both maxillary and the mandibular incisors (Fig. 2A and B). Significant increases in thresholds across frequencies were evident for both the maxillary ($F_{27.91}$, d.f.$_8$, $P < 0.001$) and the mandibular ($F_{11.49}$, d.f.$_8$, $P < 0.01$) stimulation. The median thresholds ranged from 44 mN at 40 Hz to 104 mN at 315 Hz for the maxillary incisors, which was reflected as a 58% increase in force. The distribution of thresholds to mandibular stimulation was similar to the stimulation of the maxillary incisors, although there was a slight increase in intersubject differences.

A second question of this study was whether the vibrotactile perception thresholds differed between the maxillary incisor with a relatively large root surface area and the mandibular incisor with a smaller root surface area. A comparison was made of the slopes of the thresholds to stimulation of the maxillary and mandibular incisors to determine if the thresholds to the two types of teeth respond differentially to the 10 stimulation frequencies tested (Fig. 2C). The tooth type had no significant effect overall on the threshold levels ($F_{0.403}$, d.f.$_{17}$, $P < 0.533$).

While a goal of this study was to determine the vibrotactile thresholds of normal central incisors (i.e. in dental students with excellent dentention and healthy periodontal tissue), some intersubject variability was evident, particularly among the subjects with high sensory thresholds. Fig. 3A shows representative threshold curves of a subject whose thresholds to maxillary and mandibular stimulation were similar to the group average, Fig. 3B shows the threshold curve of a subject with thresholds generally below the group average, and Fig. 3C shows a threshold-frequency plot of subject with thresholds in the upper quartile for both maxillary and mandibular incisor stimulation. Except for subjects in the upper quartile, the variance was generally consistent across frequencies, except for an increase at

Table 1 Median thresholds (mN force) and percentiles to tactile stimulation of maxillary or mandibular incisors at 10 frequencies for 16 subjects.

Stimulation frequency (Hz)	Maxillary stimulation			Mandibular stimulation		
	Median	25% percentile	75% percentile	Median	25% percentile	75% percentile
40	43.8	26.2	140.5	67.8	41.2	130.5
50	51.5	26.5	105.0	49.9	35.7	97.1
63	70.8	38.5	85.4	37.9	24.7	70.6
80	76.3	34.4	101.7	65.9	25.8	149.6
100	67.1	21.2	148.0	63.9	43.8	106.3
125	80.8	40.0	156.7	77.1	45.2	155.9
160	78.3	33.2	158.8	70.4	30.0	116.7
200	75.0	40.4	171.3	92.1	61.1	135.5
250	102.1	52.9	182.2	108.8	87.1	193.4
315	103.8	56.1	163.4	83.4	45.8	149.2

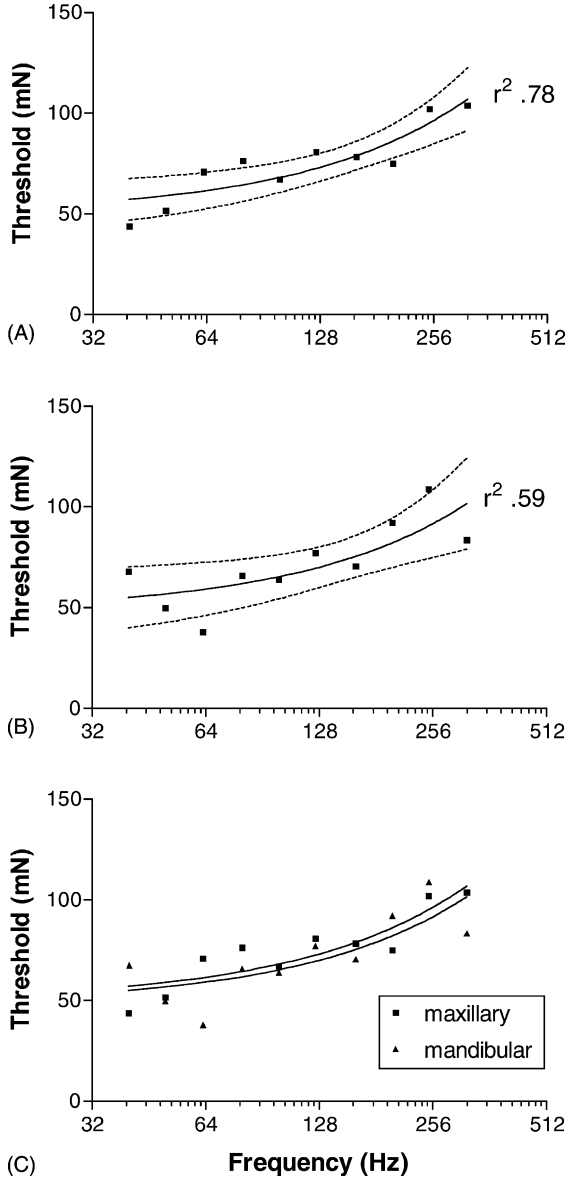

Figure 2 The threshold curves for the maxillary (A) and mandibular (B) incisors at 10 stimulation frequencies on a log scale. The median vibrotactile threshold for 16 subjects is shown at each frequency. Both linear regression lines are significantly different from a horizontal line. The thin dashed lines represent the 95% confidence intervals of the regression line. The linear regressions were not significantly different between maxillary and mandibular stimulation (C).

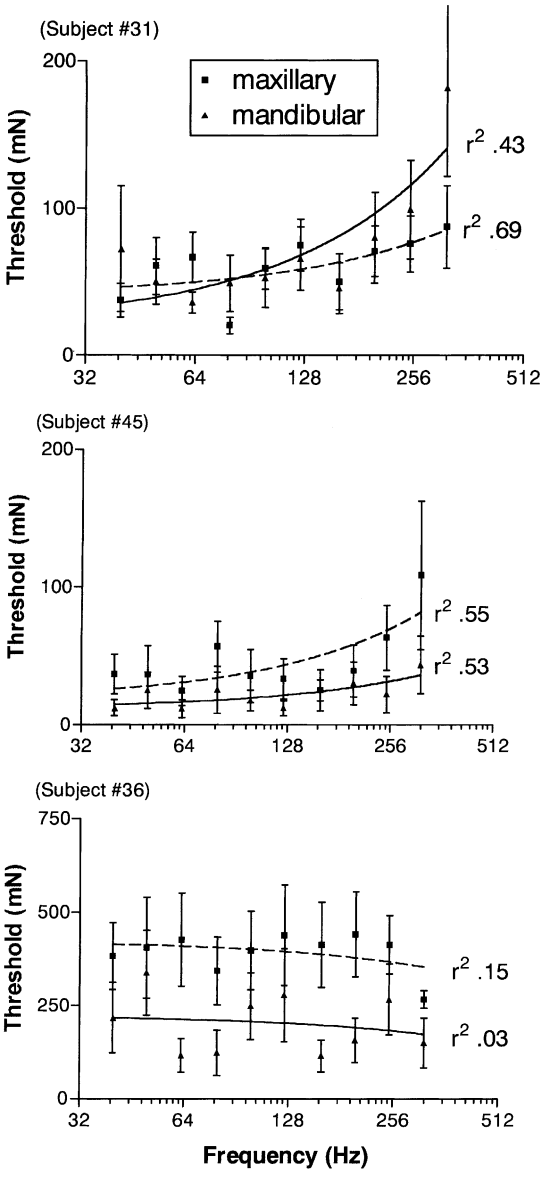

Figure 3 The mean and standard deviations of the vibrotactile perception thresholds of three subjects to stimulation of a maxillary incisor (dashed line) and a mandibular incisor (solid line) at 10 stimulation frequencies. The linear regressions revealed a significant increase with frequency for stimulation of both the maxillary and mandibular teeth for subjects 31 (A) and 32 (B), whereas for one subject (C) the linear regressions did not vary significantly from zero for stimulation of either the maxillary or mandibular incisors.

the two higher frequencies (Fig. 3A and B). Occasionally, a subject had an unexplained high threshold at a particular frequency (Fig. 3A at 32 Hz, 3B at 80 Hz). However, the subjects with thresholds in the upper quartile showed considerable variance at all frequencies, the thresholds did not vary significantly with changes in stimulation frequencies, and the thresholds of the maxillary incisors were an average of 48% higher than the thresholds for the mandibular incisors (Fig. 3C).

Discussion

This study provides the first demonstration of vibration perception thresholds of maxillary and

mandibular incisors to a systematic application of sinusoidal vibration between 40 and 315 Hz in young healthy subjects with excellent dentition and periodontonal tissue. Most subjects had low thresholds, between 44 and 104 mN of force, which were considerably lower than the threshold range of 839–1034 mN to vibrotactile stimulation of canine teeth at three frequencies as reported by Jacobs et al.[15] The differences in vibration perception thresholds of this study and the results by Jacobs et al. may be due to several factors. Different types of teeth may have different vibrotactile thresholds that coincide with distinct sensory functions. The maxillary central incisors have lower thresholds than canine teeth to mechanical taps and pressure.[17,24] Different static force levels might affect the vibrotactile thresholds. Most studies of the quantitative testing of vibration threshold employ some static force in order to maintain probe contact with the stimulus site.[20] While the effects of static force on vibration thresholds have not been systematically studied for the teeth, the static force level used in this study or the 49 to 78 mN used by Jacob et al.[15] are considerably lower than the levels used during vibrotactile testing of skin.[20,21] Different psychophysical methods were also employed. Studies that used the ascending or staircase methods of determining thresholds may include errors in threshold determinations due to loss of attention while the subject waits for the stimulus to appear, whereas an advantage of the adaptive psychophysical method, which was used in this study, minimizes errors related to loss of attention by requiring the subject to actively search for the threshold.[23] However, the trial-to-trial variance of both studies was low, which suggests the subjects maintained attention.

Relation between vibrotactile perception thresholds and stimulation frequency

A significant relation between increasing thresholds and higher frequencies of vibratory tooth stimulation was evident for most of our subjects. We found a linear increase in thresholds for stimulation rates between 40 and 315 Hz, which was similar to the frequency tuning thresholds curves of PDL and intradental mechanoreceptors in the cat canine.[10] However, our psychophysical observations and the electrophysiological observations in the cat[10] are in contrast to the observations of vibratory tuning thresholds associated with Pacinian corpuscle receptors that encode cutaneous vibrotactile stimulation.[25] The frequency-tuning threshold curves of Pacinian receptors and vibratory perception are U-shaped and not linear, with maximum sensitivity between 200 and 300 Hz.[16,26] Our findings are also in contrast to observations of Jacobs et al.,[15] who observed that the threshold at 128 Hz was lower than at 32 or 256 Hz, although the frequency tuning curves are difficult to ascertain with data for only three frequencies.

Influence of tooth morphology on vibrotactile perception thresholds

Since the natural or harmonic frequencies of a dynamic mechanical system depend on the mass and viscoelastic damping characteristics of a particular system,[27] we expected that the difference in mass between maxillary and mandibular incisors and the likely difference in the viscoelastic properties of the PDL of the two teeth with different root surface areas would result in differences in thresholds across the three-octave range for the two tooth types. Our data did not support this hypothesis. The vibrotactile perception thresholds and the threshold-frequency linear regressions between maxillary and mandibular incisors were not significantly different, which is similar to previous findings that used a ramp-hold stimulation of the incisor teeth.[17] However, it is probably appropriate that both tooth types convey the similar vibrotactile thresholds, since vibrotactile stimulation of the maxillary and mandibular incisors usually would occur simultaneously when assessing the texture of a food bolus during normal incisor biting.

Other factors that may influence vibrotactile thresholds

Numerous studies of sensory thresholds have described individual differences. Differences in attention, motivation, and cooperation can produce increased variance among individual responses and increased intersubject variance.[28] In the present study, some individuals had low variance at all frequencies, whereas other individuals had large variations in their thresholds at various frequencies, particularly for the three subjects with thresholds in the upper quartile. Our subjects appeared to be very cooperative, motivated, and appeared to attend to the task, so factors such as occlusal relationships, past sensory experiences (including trauma), or subclinical peripheral or central neuropathic changes may explain large threshold variances.

The subject with maxillary thresholds that were more than two standard deviations above the mean (Fig. 3C) had a class I open-bite occlusal relation. Since it was not possible for this subject to occlude his maxillary and mandibular incisors, these teeth would have a different tactile sensory experience

than in a normal occlusion. The absence of the normal everyday tooth contacts that occur during biting, chewing, swallowing and speech may have resulted in a deficit in making vibratory discriminations with his incisors. There is a growing body of evidence that various levels of the somatosensory system are dependent on environmental experiences.[29,30] Consequently, the perception of vibration of the central incisors may vary among individuals depending on the past sensory experiences.

Possible mechanoreceptors

Perceptually, vibration of the human skin is typically divided into two separate sensations—flutter that occurs at frequencies less than 40 Hz and vibration that spans the range from 40 to 400 Hz—that are generally considered to be conveyed to the nervous system by large, myelinated Aβ fibres, with conduction velocities of more than 30 m/s.[25] The mechanoreceptors of the human skin that best encode flutter are the Meissner corpuscle, whereas the Pacinian corpuscles respond to vibration.[25] However, neither Meissner nor Pacinian corpuscles have been identified in the PDL or the tooth, so it is not yet clear how vibration sensation of the human teeth is achieved. The mechanoreceptors within the PDL are Ruffini endings that have slowly and less often, rapidly adapting discharge properties.[10,31] It is also likely that the slowly adapting mechanoreceptors were not responsive to vibratory stimuli, since these mechanoreceptors would be expected to respond to the static qualities of the preload force. This suggests that a distinct population of dental mechanoreceptors can encode vibration. Dong et al.[10] suggested that free nerve endings of the Aβ fibers within the tooth may be capable of encoding the transmission of vibration through enamel to the dentinal fluid, although other investigations have not supported this hypothesis.[32,33]

The sinusoidal stimulation may have also elicited, via bone conduction, receptors in the middle ear, stretch receptors in the jaw closing muscles, or mechanoreceptors located in other structures of the oral and perioral areas, such as in the hard palate and the temporomandibular joint capsule.[34–37] It is unlikely that any auditory receptors were elicited, since these receptors were probably masked by the white noise presented during testing. Mechanoreceptors located solely within the palate, jaw-closing muscles, or joint probably are not sufficient to encode vibrotactile stimulation for both the maxillary and mandibular incisors, since the vibrotactile thresholds were similar between the maxillary and mandibular incisors. However, the vibration perception thresholds may have been influenced by interdental contact of the test-incisor with neighbouring teeth,[38,39] since interdental contacts may have allowed the activation of neighbouring mechanoreceptors or may have resulted in the spatial summation of afferent input.

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Teeth as Sensory Organs

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ABSTRACT:
Mastication triggers a unique and complex neural control system that is designed to protect teeth's structure. As part of this process, mechanoreception is the unconscious sensing or conscious perception of touch or mechanical displacement caused by stimuli such as tension, pressure, and vibration. Endodontically treated teeth and dental implant-retained prostheses provide less mechanosensory information than vital teeth. Consequently, tooth wear and catastrophic failures of nonvital teeth may ensue. The author proposes a new paradigm in dentistry called *teeth as sensory organs;* its application may alter treatment plans in the future.

Seventeen years ago, as a student of Peter Dawson, DDS, this author first observed in his patients that the elimination of occlusal interferences enabled many of them to open and close their jaws faster, move more freely through excursive jaw movements, and feel more comfortable. Consequently, the author was motivated to investigate the causes of these phenomena by exploring the scientific literature. Research demonstrated that, while an enormous volume of relevant scientific publications existed, the scientific basis for occlusal therapy remained unclear. To understand occlusal therapy's success, information would have to be integrated from widely divergent fields, such as neurophysiology, biomechanics, and histology, and individual interdisciplinary research protocols designed. This article encapsulates the findings to date.

MECHANORECEPTION

Teeth are specialized organs that function to nourish and sustain life. While people eat, the brain rapidly compares food's texture and hardness in the mouth to previous encounters and determines the best chewing strategy. Optimal chewing forces and rhythms are developed based on tactile sensory feedback from the food bolus's contact with the teeth and soft tissues as the bolus progressively becomes smaller. The ability of a tooth to endure the rigors of mastication depends on having a durable stone-like structure and a complex neural control system to maintain the tooth's integrity. The cornerstone of this neural control system is an exquisitely sensitive network of mechanoreceptors within the tooth and its periodontal ligament. Dental mechanoreceptors play a crucial role in providing tactile sensory feedback that minimizes the stresses that teeth endure while they pulverize vast quantities of food in a lifetime. Under the influence of pathologic conditions such as malocclusion or central nervous system disease, the teeth's mechanosensory system can play a key role in promoting destructive oromotor behaviors, such as bruxism and clenching.

Mechanoreception is the unconscious sensing or conscious perception of touch or mechanical displacement arising from stimuli outside the body. Mechanoreceptors are sensory end organs that respond to mechanical stimuli such as tension, pressure, or vibration.

Perception and recognition of a finely textured object that is handled or bitten relies on the ability to encode tactile cues arising from its size, shape, and roughness. The encoding of these cues occurs primarily as a result of two types of mechanoreceptors, which include slowly adapting (SA) and rapidly adapting (RA) mechanoreceptors. SA mechanoreceptors, such as Merkel disks and Ruffini endings, fire continuous streams of action potentials as long as the stimulus (eg, touch) remains active. Because

they fire continuously during contact, SA mechanoreceptors are best suited for providing awareness that an object is between the teeth.

Vibrations are produced when textured objects rub against the surfaces of the skin or teeth. RA mechanoreceptors, such as Meisner and Pacinian corpuscles, fire briefly upon initiation of vibrating or rapidly accelerating stimulation, stop quickly, and are able to re-fire rapidly in response to a new stimulus. The rapid on/off firing characteristics of RA mechanoreceptors make them well suited for sensing the vibrations associated with textural assessment.

Historically, the tactile sensory function of the teeth had been ascribed solely to periodontal mechanoreceptors and pain perception to the richly innervated tooth pulp. However, studies indicate mechanoreceptors within teeth play an important role in their tactile sensory function. In 1955 a study by Lowenstein and Rathkamp compared tactile sensory thresholds of nonvital (ie, root canal treated teeth) to vital teeth and found tactile thresholds of nonvital teeth were 57% higher than those of contralateral vital teeth.[1] The authors concluded that a specialized mechanosensory mechanism within the teeth contributed to tactile sensory function. In 1975 Linden[2] failed to identify significant differences in the thresholds of vital and nonvital teeth. Although the method of tooth stimulation used by Linden was quite different than Lowenstein and Rathkamp's, Linden's study convinced the scientific community to support the hypothesis that periodontal receptors served as the principal receptors involved in dental mechanosensation.[3]

Once thought to contain only Aδ and C pain nerve fibers (ie, nociceptive), physiologic investigations have discovered the tooth pulp contains numerous rapidly conducting Aβ mechanoreceptive fibers.[4] Dong and Chudler[5] used electrophysiologic recording techniques in cats to measure the elapsed time for impulses to travel up the neural axis from stimulated intradental nerves through the brainstem and thalamus to the somatosensory cortex. They determined some intradental nerves conveyed mechanosensory information to the somatosensory cortex much faster than Aδ pain fibers. These intradental nerves were classified as Aβ mechanoreceptive fibers based on their rapid conduction velocities. Similar results have been repeated in monkeys[6] and more recently in humans

LEARNING OBJECTIVES

After reading this article, the reader should be able to:

- describe the role of mechanoreception and sensory motor integration.
- recognize how some dental procedures may adversely affect this complex process.
- describe the *teeth as sensory organs* paradigm.

in whom Aβ pulpal nerves have been mapped to a specific location in the somatosensory cortex, effectively adding mechanoreceptive pulpal nerves to the classic "sensory homunculus"[7] first described by Penfield and Jasper.[8]

Studies in cats have shown the neurophysiologic properties of intradental and periodontal mechanoreceptors are functionally different.[9,10] Rubbing sandpaper with different grit sizes on canine teeth causes frequency-encoded discharge patterns to arise in trigeminal ganglion neurons. These discharge patterns are unique to grit size, indicating that intradental mechanoreceptors are able to encode mechanical vibrations. Intradental mechanoreceptors have rapidly adapting response characteristics and encode vibrations throughout a wide frequency range. Periodontal mechanoreceptors have slowly adapting response characteristics and encode only lower vibration frequencies (Figure 1). Intradental mechanoreceptors respond to forces applied to the tooth from all directions (ie, omnidirectional) whereas periodontal mechanoreceptors respond

Intradental Mechanoreceptors	*Periodontal Mechanoreceptors*
Rapidly Adapting	Slowly Adapting
Fire Intermittently	Fire Continuously
Wider Frequency Range	Lower Frequency Range
Omnidirectional	Unidirectional
Perception of Texture	Perception of 1st Contact

FIGURE 1 **Dental mechanoreception relies on two channels of sensory input.**

only when forces are applied from specific directions (ie, unidirectional). In monkeys, periodontal mechanoreceptors are more numerous around the anterior teeth than the posterior teeth,[11] which may partly account for reports that indicate tactile thresholds of anterior teeth are lower than those of posterior teeth.[12]

Vibration perception through human skin is essential for accurate perception of textured objects that are grasped.[13] Similarly, vibration perception through the teeth enables accurate assessment of textured objects placed in the mouth. The author has developed a test to assess vibration perception thresholds of human teeth. The results show intradental mechanoreceptors encode vibrotactile tooth stimulation at amplitudes low enough to help discern textural differences in objects.[14-17] These experiments demonstrate vital maxillary and mandibular incisors encode vibrations between 10 Hertz (Hz) and 315 Hz at low amplitudes and endodontically treated teeth lack the ability to encode vibrations.

The author's research confirms the presence of intradental mechanoreceptors and suggests endodontic procedures may limit patients' abilities to perceive vibrations associated with textural assessment of objects with their teeth. In addition, the results show vibration perception thresholds are related to stimulation frequency, suggesting the conflicting results of earlier studies by Lowenstein and Rathkamp[1] and Linden[2] may be attributed to the different vibration frequencies delivered by their respective tooth stimulation methods.

Having lost intradental mechanoreceptors, nonvital teeth may unwittingly allow the use of stronger-than-normal biting forces. Eventually, elevated occlusal forces may lead to tooth wear and catastrophic fractures in nonvital teeth. This hypothesis is supported by the excessively high fracture rate associated with nonvital teeth.[18]

SENSORY MOTOR INTEGRATION

Sensory motor integration is a feedback process during which sensory inputs from peripheral parts of the body modify actions initiated by the central nervous system. This process occurs primarily in the brainstem, thalamus, and cortex (Figure 2). In the masticatory system, sensory motor integration coordinates fundamental activities such as breathing, eating, and swallowing with sensations that arise during their performance.

Occlusion of the jaws and teeth, as defined in *The Glossary of Prosthodontic Terms,* is "the act or process of closure."[19] Occlusion is a dynamic process during which willful and rhythmic jaw movements are integrated with sensations experienced during movement and memories of prior movements. Efferent motor commands from the cortex, cerebellum, and brainstem are integrated with peripheral sensory feedback from the teeth, muscles, temporomandibular joints, bones, and soft tissues. Occlusion relies on sensory motor integration to coordinate the activities of the muscles of mastication.

Movement coordination in most of the body's joint systems (eg, arm and leg) is facilitated by proprioceptors

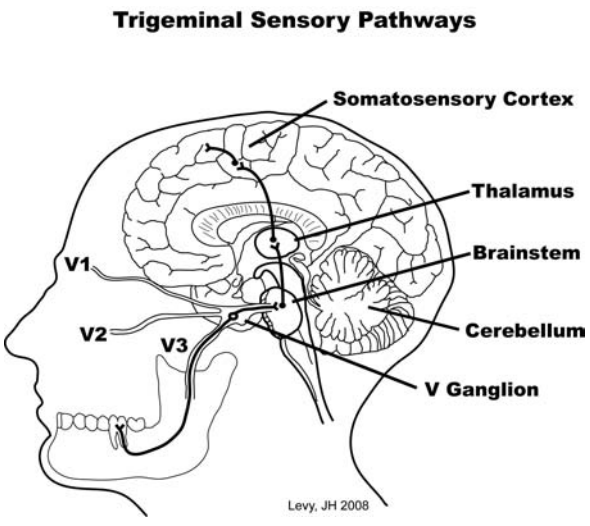

FIGURE 2 The main pathways of dental mechanosensory information to the somatosensory cortex.

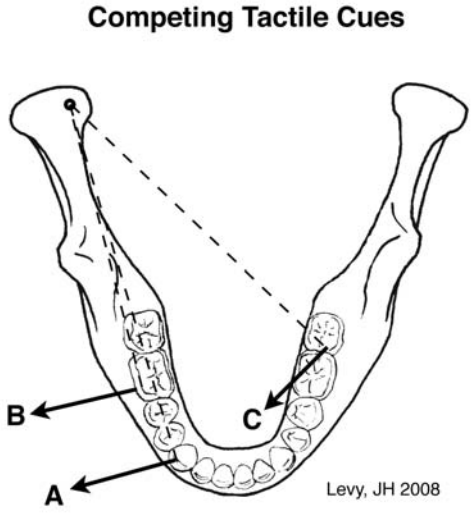

FIGURE 3 Working and nonworking side occlusal interferences create competing tactile sensory information.

(eg, muscle spindles) in antagonist muscle groups (ie, abductor *and* adductor muscles) and sensory receptors in the skin and joints. The masticatory system is unique in that *only* its adductor muscles (ie, jaw-closing muscles) are innervated by muscle spindles.[20,21] As a result of this unique neural architecture, control of the jaw-opening muscles may be more reliant on tactile sensory feedback from mechanosensory receptors (ie, intradental and periodontal mechanoreceptors) than in other joint systems.

Mechanical tooth contacts produce very rapid jaw reflex behaviors. Jaw reflexes are thought to protect the teeth from excessively strong biting forces. Whether tooth contacts induce inhibition or excitation of the jaw-closing muscles depends on several variables, including rate of force application and background clenching level.[22] Reflex inhibition of the jaw-closing muscles after mechanical tooth stimulation may be referred to as the jaw-opening reflex or silent period. In humans, the jaw-opening reflex is characterized by the rapid inhibition of the jaw-closing muscles (ie, masseter, temporalis, and medial pterygoid) and bite-force reduction, following tooth contact. When an unexpectedly high biting force occurs (eg, a stone in lentil soup) the jaw-opening reflex may prevent tooth fracture by rapidly shutting down the jaw-closing muscles.

Olgart et al[23] monitored the jaw-opening reflex in cats in response to bending forces applied to their canine teeth. Their results demonstrated that bending forces applied to vital teeth evoke the jaw-opening reflex and subsequent endodontic procedures abolish this reflex. In conclusion, Olgart et al speculated a specialized sensory transducer mechanism exists in dentin that is activated by deformation or bending of the crown of a tooth.

Trulsson and Gunne observed "striking disturbances in the control of certain jaw motor behaviors" in people lacking dental mechanoreceptors.[24] Participants with dentures and implants could not position their jaws as precisely as participants with vital teeth and used four times the biting force to hold a peanut between their teeth. These findings imply implant and denture prostheses are likely to undergo mechanical damage as a consequence of poor biting control.

RA intradental and SA periodontal mechanoreceptors generate streams of mechanosensory information as the teeth are maneuvered past each other during excursive jaw movements. Sensory motor integration of mechanosensory information from these receptors regulates the course and speed of the excursive movement. Patients with canine and incisive guidance have fewer contacting teeth during excursions and have less mechanosensory information to integrate than patients with group function and nonworking side interferences. Increasing the number of interfering tooth contacts during excursions compels the central nervous system to integrate *more* mechanosensory information because additional midcourse corrections are needed to accomplish the movement.

Having lost intradental mechanoreceptors, nonvital teeth may unwittingly allow the use of stronger-than-normal biting forces. Eventually, elevated occlusal forces may lead to tooth wear and catastrophic fractures in nonvital teeth.

Posterior occlusal interferences compete with canine tooth contacts for control of masticatory muscles during excursive jaw movements (Figure 3). Competition for jaw-closing muscle activity occurs when posterior-interfering tooth contacts evoke muscle recruitment patterns that differ from those initiated by working-side anterior tooth contacts. Occlusal tooth contact competition can induce muscle hyperactivity in the orofacial region as jaw muscles become overworked. This may cause pain in these muscles. Reduced competition for muscle recruitment may explain why the elimination of working- and nonworking-side occlusal interferences can increase the speed of lateral jaw excursions, reduce muscle hyperactivity, and alter bruxing behaviors.[25,26]

A PARADIGM SHIFT IN DENTISTRY

Kuhn applied the term *paradigm* to the evolution of science. Kuhn wrote, "…a paradigm is an accepted model or pattern.…The new paradigm implies a new and more rigid definition of a field.…In the absence of a paradigm or some candidate for paradigm, all of the facts that could possibly pertain to the development of a given science are likely to seem equally relevant."[27] Controversies that shroud the field of occlusion may be resolved in time as irrelevant facts are pared away by the acquisition of new paradigms.

Teeth as sensory organs is a new paradigm in dentistry. In this paradigm, tooth contacts are understood to initiate

streams of mechanosensory information that shape oromotor behavior. Endodontically treated teeth and dental implant-retained prostheses provide less mechanosensory information than vital teeth. It becomes clear that bite force magnitude is affected by mechanosensory feedback that can restrain muscle activity and limit structural damage to the teeth, temporomandibular joints, and periodontal apparatus. The function of occlusal therapy can be interpreted as the manipulation of mechanosensory streams to change jaw muscle activity and oromotor behavior. The goal of such therapy is to foster changes in oromotor behavior that reduce functional occlusal forces and positively affect the health and longevity of the masticatory system.

In the future, the application of the paradigm *teeth as sensory organs* may alter prosthetic treatment plans. Strategies may be developed that incorporate the fact that dental implants and nonvital teeth are more likely to be exposed to higher bite force levels because they are deficient in protective mechanosensation. Bias toward conservative tooth preparation may increase with widespread knowledge of how operative procedures affect intradental mechanoreception and vital teeth's abilities to protect themselves from adverse biting forces.

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Teeth as Sensory Organs

JAY HARRIS LEVY, DDS

1. **Optimal chewing forces and rhythms are developed based on:**
   - **a** stimulation of various taste neurons.
   - **b** tactile sensory feedback.
   - **c** visual feedback gleaned while putting food into the mouth.
   - **d** an alternating closed-open-closed proprioceptive dynamic.

2. **Which mechanoreceptors, such as Merkel disks and Ruffini endings, fire continuous streams of action potentials as long as the stimulus (eg, touch) remains active?**
   - **a** SA
   - **b** RA
   - **c** LA
   - **d** ATP

3. **Which mechanoreceptors, such as Meisner and Pacinian corpuscles, fire briefly upon initiation of vibrating or rapidly accelerating stimulation, stop quickly, and are able to re-fire rapidly in response to a new stimulus?**
   - **a** SA
   - **b** RA
   - **c** LA
   - **d** ATP

4. **In 1955 a study by Lowenstein and Rathkamp compared tactile sensory thresholds of nonvital (ie, root canal treated teeth) to vital teeth and found tactile thresholds of nonvital teeth were how much higher than those of contra lateral vital teeth?**
   - **a** 12%
   - **b** 18%
   - **c** 23%
   - **d** 57%

5. **Vibration perception through the teeth enables accurate assessment of what type of objects placed in the mouth?**
   - **a** textured
   - **b** liquid
   - **c** hot
   - **d** cold

6. **The conflicting results of earlier studies by Lowenstein and Rathkamp and Linden may be attributed to:**
   - **a** the use of cats vs humans for experimental study.
   - **b** the use of cats vs dogs for experimental study.
   - **c** the different vibration frequencies delivered by their respective tooth stimulation methods.
   - **d** the fact that the more recent Linden study used current information on C pain nerve fibers.

7. **Occlusion relies on sensory motor integration to coordinate:**
   - **a** salivary stimulation.
   - **b** the activities of the muscles of mastication.
   - **c** horizontal chewing forces only.
   - **d** vertical chewing forces only.

8. **Increasing the number of interfering tooth contacts during excursions compels the central nervous system to integrate *more* mechanosensory information because:**
   - **a** the wear of the dentition over time isn't accounted for.
   - **b** additional midcourse corrections are needed to accomplish the movement.
   - **c** posterior contacts are mathematically closer to the hinge axis of the jaw.
   - **d** anterior contacts are mathematically farther from the hinge axis of the jaw.

9. **In the absence of a paradigm or some candidate for paradigm, all of the facts that could possibly pertain to the development of a given science:**
   - **a** are ranked by chronological appearance in the literature.
   - **b** are ranked by a scientific team approach to determine plausibility.
   - **c** are likely to seem equally relevant.
   - **d** must be supported by level 1 or level 2 scientific evidence.

10. **It becomes clear that bite force magnitude is affected by what that can restrain muscle activity and limit structural damage to the teeth, temporomandibular joints, and periodontal apparatus?**
    - **a** mechanosensory feedback
    - **b** proprioceptive bioanalysis
    - **c** efferent motor feedback
    - **d** afferent motor feedback

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The Effect of Head Posture on Muscle Contact Position: The Sliding Cranium Theory

Howard W. Makofsky, P.T.

Abstract

The effect of head posture on initial occlusive contacts has been studied extensively by researchers in the basic sciences, dentistry, and physical medicine. The purpose of this paper is to review their theories and propose a new mechanism that attributes the effect of head posture to a change in the upper to lower jaw relationship. This mechanism is referred to as the sliding cranium theory. To understand how head posture alters initial tooth contact or muscle contact position, the arthrokinematics of the occipital-atlantal joint are covered in detail. The implications of the proposed new theory have relevance for dentists concerned with occlusal function and the treatment of temporomandibular joint dysfunction with temporomandibular repositioning, as well as for physical therapists who effect a change in head posture through mobilization procedures and therapeutic exercise. To conceptualize the proposed theory two easily performed tests are described. The sliding cranium theory presents a mechanical model that explains the interrelationship between the head-neck complex and the craniomandibular system in a way that has not been previously done.

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The relationship between head posture and the muscle contact position[1] (initial tooth contact) is of great interest to all disciplines concerned with the treatment of patients with cranio-facial pain as well as to those dentists concerned with bite registration for full denture, fixed reconstruction, and orthodontic diagnosis.[2]

The purpose of this paper is to review existing theories on the relationship between head posture and muscle contact position and to propose a new concept that demonstrates the direct relationship between the cranio-vertebral system and the initial contact position of the mandibular and maxillary teeth.

Literature Review

Many studies have been done investigating the relationship between head posture and mandibular function. It is well established that head-neck backward bending increases the electromyographic (EMG) activity of the masticatory elevator muscles, especially the temporalis muscle.[2–4] The possible mechanisms mediating this phenomenon include the tonic neck reflex,[4] the role of gravity,[5,6] and body position.[6]

The influence of head posture on the rest position of the mandible has also been studied extensively[7–10]; Kraus[11] has stated that head-neck posture has the most immediate and long-lasting effect on the mandibular rest position.

Solow and Tallgren[12,13] demonstrated a relationship between head posture and craniofacial morphology. Kraus' review of the effect of head posture on the development of the mandible concludes that a high correlation exists between an extended head-neck posture and the development of a retrognathic mandibular posture.[11]

Of greatest relevance to this paper, however, is the role of head posture as it influences the mandibular pathway of closure into the fully intercuspated position of mandibular and maxillary teeth. Mohl[14] suggests that a change in head posture will likewise alter the habitual closing path from rest position to maximum intercuspation. The consensus of most studies[14–16] is that initial tooth contacts are more retruded when the head is positioned in backward bending (extension or dorsiflexion), or when a subject is supine.[1,5,6,17] Ramfjord and Ash[18] have stated that initial contact will depend on posture.

On the contrary there is no evidence that body position or head posture can alter such structural relationships as tooth position in maximum intercuspation[1,6,15,16] or the vertical dimension of occlusion[19] (teeth maximally intercuspated).

Current Theories on the Influence of Head Posture on Muscle Contact Position (MCP)

With the exception of practitioners of cranial manipulative therapy who assert that MCP can be altered by small intracranial movements of the maxilla and/or temporal bones,[20,21] other theories on the influence of head posture on MCP deal exclusively with the change in mandibular position.[2–6,11,14–16,18,22]

Mohamed and Christensen[1] state that neck dorsiflexion (backward bending) causes the mandible to move away from the maxilla with resultant retrusion/depression of the mandible; in ventroflexion (forward bending) the opposite occurs. Other researchers[7–9,22] attribute the influence of head-neck backward bending on the mandible, i.e., down and back movement to increased inframandibular soft tissue tension (supra/infrahyoid muscles and fascia). This retrusive force is one attempt to explain the posterior occlusal contacts observed with the head-neck backward bent.[14–16]

Another popular theory involves the effect of head-neck backward bending on the temporalis muscle. Assuming increased EMG activity in this posture, a force of elevation and retrusion on the mandible would account for initial occlusal contacts that are posterior to the intercuspal position.[11]

The research on body position is also worth noting. In the supine position the MCP is consistently retruded.[1,5,6,17] According to McLean et al., mandibular position is affected by the position of the body in space through the activity of neuromuscular mechanisms.[6]

To demonstrate the principle of head-neck backward bending causing a posterior MCP and forward bending causing an anterior MCP, one need only perform a simple test. While lightly tapping the teeth (2–3 taps per second) with the patient sitting or standing, one can easily detect a change in contact pattern as the head-neck is moved from neutral to backward bending and from neutral to forward bending. This phenomenon is exactly what the aforementioned researchers/authors have studied, albeit in a nonexperimental fashion.

The Sliding Cranium Theory

Goldstein et al.[22] state, "Although the exact mechanism by which head position affects the movement of the mandible is not completely understood, proper

head-neck positioning appears important to all phases of dentistry."

One of the reasons why the "exact mechanism" remains unclear is that research to date has focused primarily on how head position affects the mandible. The sliding cranium theory suggests that changes in head posture are able to produce a change in MCP by altering the position of the maxillary teeth relative to the mandibular teeth. This is not to say that the previously mentioned mechanisms acting on the mandible do not play an important role but that there is an additional mechanism that deserves consideration. This theory only applies to a change in initial occlusal contacts and not to maximum intercuspation, which is a structural position and is therefore not affected by head posture.[1,6,15,16] To appreciate how maxillary occlusal position is altered by changes in head posture, a review of occipito-atlantal (O-A) joint arthrokinematics (intimate joint mechanics) is helpful. Kapandji[23] states that in extension or backward bending of the cranium the occipital condyles slide anteriorly on the lateral masses of the atlas (C-1); in forward bending the opposite occurs. An understanding of synovial joint mechanics will serve to elucidate this concept. When a convex joint surface moves on a concave surface, the rotary movement or roll and the translatory movement or slide occur in opposite directions simultaneously.[24] Consequently, when the occiput backward bends, the convex occipital condyles simultaneously slide anteriorly on the concave atlas, and during forward bending they slide posteriorly[23–26] (**Figure 1**). According to Steindler[25] the total excursion of the convex occipital condyles on the lateral masses of atlas is 10 mm. This 10-mm slide in the joint is associated with a total rotary range of motion of 24.5° with 21° in O-A backward bending and 3.5° in forward bending.[27]

When the cranium slides forward on the atlas during backward bending the maxillary teeth also slide forward (being structurally joined to the cranium through the periodontal membrane system) relative to the mandibular teeth. Consequently the MCP shifts posterior to the intercuspal position (**Figure 2A**). However, as the teeth assume maximum intercuspation, the maxillary teeth will guide the mandible forward (through cusp-fossa relationships) such that in maximum intercuspation mandibular position in centric occlusion remain unchanged regardless of head position.

When the cranium slides backward on the atlas during forward bending the situation is reversed, i.e., MCP shifts anterior to the intercuspal position (**Figure 2B**). Ideally with the head in neutral, orthostatic posture[28] and the teeth free of interferences, MCP will be in direct alignment with the intercuspal position[1,29] (**Figure 2C**).

Regarding the arthrokinematics of the O-A joint during rotation and side bending[23,25,26] and the effect of these movements on MCP, the literature is inconclusive.[30,31] Therefore the proposed theory will not address the effect of these less understood movements of the cranium on initial tooth contact patterns.

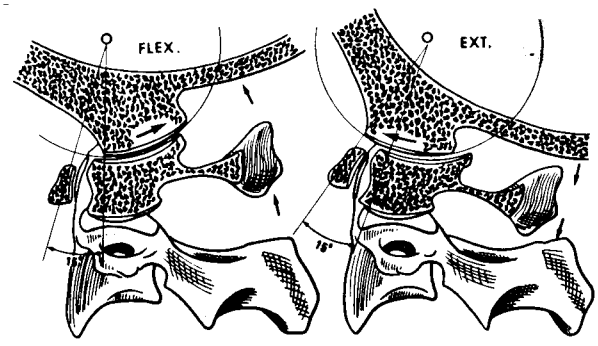

Figure 1
Lateral view of occipito-atlantal joint. A. Flexion (forward bending) of occiput on atlas is associated with posterior slide of the convex condyle on concave atlas. B. Extension (backward bending) of occiput on atlas is associated with anterior slide of convex condyle on concave atlas. (Figures taken from Kapandji IA, *The Physiology of the Joints* Vol 3. Edinburgh: Churchill Livingstone, 1974, with permission.)

Clinical Implications

Because of the profound implications of the effect of head posture on the initial tooth contacts and on craniomandibular function, numerous authors have studied this relationship.[1–4,7–16,18,19,22,28–31]

The sliding cranium theory builds on what is already known about the influence of head posture on craniomandibular function and adds to it the role of the O-A joints. In addition to the role of gravity, body position, tonic neck reflexes, and soft tissue factors all influencing mandibular position and function, there is yet another significant factor influencing the relationship of the maxilla to the mandible in response to head posture changes.

The sliding cranium theory offers a timely explanation of how the dental management of temporomandibular joint (TMJ) dysfunction affects the physical therapy management of associated head-neck dysfunction and vice versa. For example, a patient with forward head posture (FHP) received an anterior repositioning splint to manage an anterior TMJ disk dis-

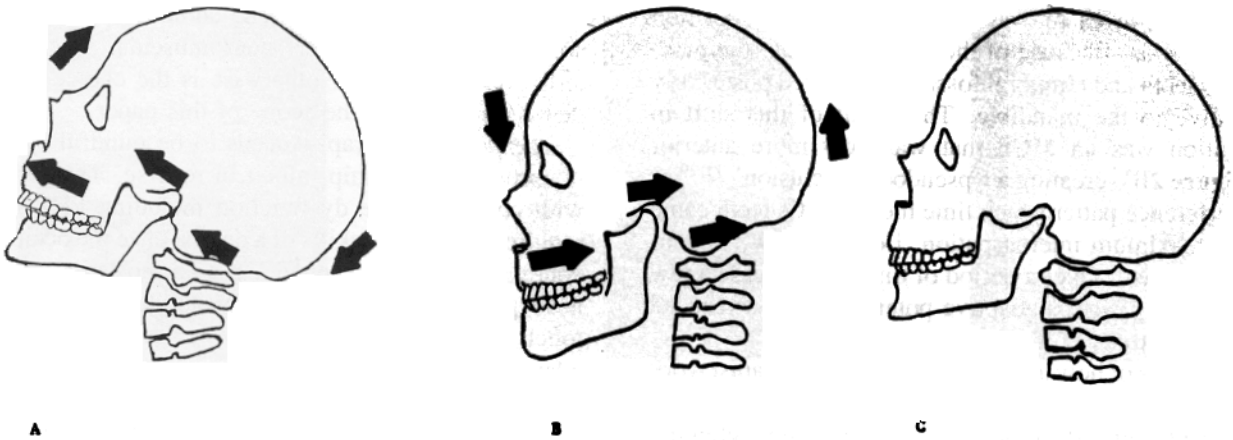

Figure 2
A. Cranial backward bending is associated with anterior translation of occiput on atlas. This slide shifts muscle contact position (MCP) posterior. B. Cranial forward bending shifts MCP anterior secondary to posterior slide of occiput on atlas. C. Neutral head posture is ideally associated with MCP in direct alignment with maximum intercuspation.

placement with reduction. The patient responded well to splint intervention, but developed suboccipital pain. Conversely, a patient with head-neck dysfunction responded well to physical therapy procedures including the correction of FHP, but developed facial pain. While many theories attempt to explain these reciprocal relationships, the sliding of the cranium on the atlas offers another theoretical perspective.

In our example of the patient with FHP and a TMJ disk displacement, the anterior repositioning splint used not only affected mandibular position, but also head posture. As the mandible was repositioned anteriorly/inferiorly relative to the maxilla, the cranium hypothetically attempted to forward bend on the atlas (opposite to the head position in FHP). Providing that this patient had a long-standing FHP, the occipital condyles would have likely developed restrictions in posterior slide secondary to adaptive shortening of the soft tissues (joint capsule, musculature, connective tissue, etc.). Consequently, this limited ability of the occipital condyles to slide posteriorly as the cranium attempted to reposition itself in more relative forward bending resulted in suboccipital pain.

This theoretical explanation is based on neurophysiologic mechanisms mediated through the peridontal mechanoreceptors. It is postulated that a tooth interference activates the periodontal mechanoreceptors, which are capable of changing the habitual closing pathway of the mandible into centric occlusion.[18,29,32] This response is a function of supraspinal reflexes to the muscles of mastication and is an attempt by the body to eliminate the interference. It is also possible, although not well researched, that activation of the periodontal mechanoreceptors is able to effect changes in head-neck muscle function and thereby produce changes in head posture.[33,34] This reflex mechanism mediated through the trigeminocervical nucleus[11,35,36] is another attempt by the body to align the upper and lower jaws for the purpose of eliminating an undesirable interference pattern between one or more teeth.

Returning to the aforementioned hypothetical patient, the anterior repositioning of the mandible produced an interference that did not previously exist. As the patient closed into his new centric occlusion, he encountered a hit and slide forward. The periodontal mechanoreceptors activated by this interference act on the masticatory as well as the cervical musculature to reposition the mandible and maxilla such that the mandibular teeth (in the case of a maxillary splint) close directly into the occlusal splint. Because the patient was able to make this correction in mandibular position, the splint was able to obtain a successful result. However, the O-A restriction in posterior slide prevented the cranium from making a similar adjustment and symptoms resulted.

The second patient scenario involved the correction of head-neck dysfunction in a patient with neck pain. Using specific physical therapy procedures,[10,28,31] the patient responded well to treatment with improved head-neck mobility, posture, and reduction in symptoms. Why, however, did this patient develop facial pain? Let us assume that this patient not only had a significant FHP of long duration, but also had a class II malocclusion, which is often the case.[12,13,37] As this patient responded to physical therapy and began approaching an orthostatic head-neck posture[28,31] the oc-

cipital condyles moved to a more posterior position on the atlas. Because of this posterior slide, the maxillary teeth and temporal fossae also moved posteriorly relative to the mandible. The result of this shift in position was an MCP that was now more anterior (**Figure 2B**), creating a "pseudomalocclusion"[11,29] or interference pattern each time the patient's teeth came into maximum intercuspation, i.e., swallow, clench, bruxism, etc. Over a period of time (in this case a few days) the patient's adaptive potential[38] was exceeded and symptoms of TMJ/facial pain ensued.[29]

In terms of how a dentist and physical therapist work together with a team approach to manage TMJ and cervical spine dysfunction,[39–41] the sliding cranium model functions as a type of "bridge" in spanning this interdisciplinary relationship. The dentist who uses occlusal splint therapy must recognize that an adjustment of mandibular position necessitates associated movement of the O-A joints. Consequently, a physical therapist trained in manipulative therapy is needed to evaluate the head-neck region and render the appropriate treatment if indicated. The dentist who recognizes the need for an in-depth assessment of the craniovertebral region at the outset of occlusal splint therapy must also appreciate that altered head-neck mobility/posture necessitates ongoing occlusal splint adjustment. If the splint is not adjusted as head posture changes then no allowance for an altered MCP is being made. The result of this oversight will be either interferences in the appliance (from MCP to centric occlusion) or a tendency for head-neck posture to remain as it was at initial splint fabrication. This clinical dilemma is frustrating to the dentist, physical therapist, and most importantly the patient who is not recovering.

There are several suggestions regarding the type of splint that is best suited for a patient who is experiencing a change in MCP as a result of changing head posture, i.e., a patient receiving orthopedic physical therapy.[42]

A splint that repositions the mandible into a predetermined position may provide relief of TMJ/facial pain; but will it allow for a correction in head posture simultaneously? Our data and experience suggest that repositioning, if necessary, should await the correction of head-neck dysfunction. Once head posture is normalized, or at least improved upon, then mandibular repositioning will be more easily tolerated by the patient and a superior result obtained. If, however, splint therapy is warranted in the presence of head-neck dysfunction (including FHP), then the appliance of choice is one with shallow inclines to allow for a changing MCP, i.e., long centric.[16] The choice of hard versus soft, upper versus lower, neuromuscular versus anterior repositioning or otherwise is the choice of the dentist and beyond the scope of this paper.

The physical therapist needs to be mindful of this reciprocal relationship, albeit in reverse. The patient with cervical spine dysfunction including FHP may require the occlusal skills of a dentist once the occipital condyles are "repositioned" in an orthostatic, neutral head posture. This patient may notice that his teeth touch differently than previously. If this patient's adaptive potential[38] for change is not compromised he may remain asymptomatic. However, if it has been compromised by physical, biochemical, and/or emotional factors, then he will either develop TMJ/facial pain or relapse to his former head posture in an attempt to eliminate his tooth interferences.

Testing the Model

There are a few simple tests that can be done to add credence to the sliding cranium theory. One test is to compare the amount of cranial backward bending present with the TMJ in neutral, and in retrusion. With a lateral radiograph, the space between the occiput and the posterior arch of atlas (O-A space) is measured (millimeters) in head-neck backward bending and then remeasured following passive mandibular retrusion by the patient. As noted in **Figure 3** head backward bending is essentially blocked if the mandible is retruded beforehand. This is because the cranium is unable to slide forward on the atlas secondary to a bony stop between the posterior temporal fossa and the posterior aspect of the mandibular condyle.

A second simple test that can be done without radiographs is to compare passive mandibular retrusion with the head first backward bent, then in neutral, and lastly in forward bending. With the head in backward bending, retrusion of the mandible is blocked as the condyle abuts the temporal bone, whereas in head forward bending, it is free to retrude even more so than in neutral. This is because of the increase in posterior TMJ space created by a posterior slide of the cranium during forward bending.

Once the mechanism is understood, many such tests can be performed to confirm the mechanics that prevail at the occipito-atlantal junction.

Summary

Many theories have been proposed in the literature to explain the mechanism by which the head-neck

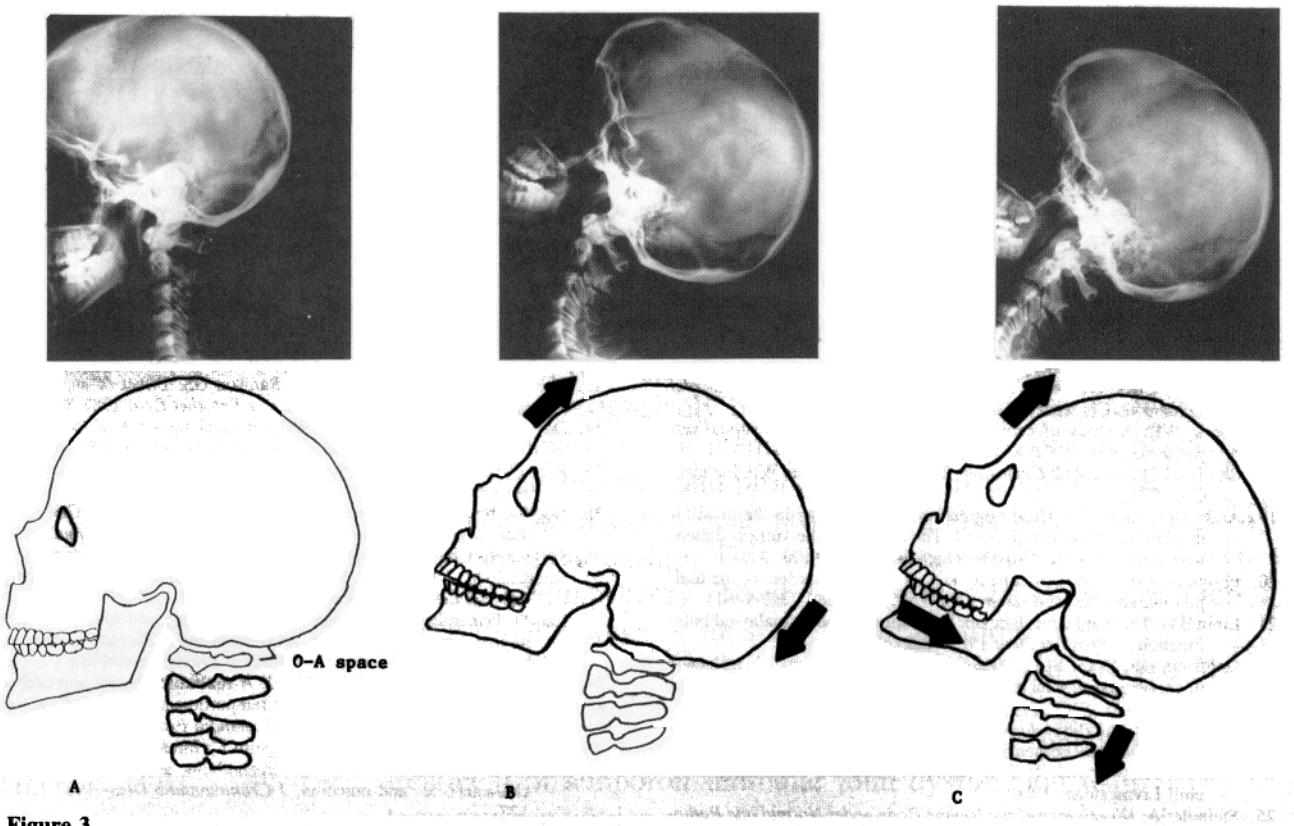

Figure 3
A. With the mandible relaxed and head-neck in neutral note the occipito-atlantal (O-A) space. B. With the mandible relaxed but the head-neck backward bent note the decreased O-A space. C. With the mandible passively retruded, note the inability of the occiput to backward bend on the atlas by virtue of a "boney block" between the posterior aspect of the condyle and the posterior aspect of the temporal fossa (backward bending occurs in the lower cervical spine instead).

complex influences the muscle contact position of the teeth as well as the rest position and movement behavior of the mandible. What has been lacking, however, is the influence of head posture on the maxillary component of MCP. To describe the mechanisms whereby changes in head posture influence the position of the maxillary teeth, the sliding cranium theory has been elucidated. The forward and backward slide of the cranium on the cervical spine follows the joint mechanics of the occipito-atlantal articulation. Implications for treatment concern the dentist who must recognize the relationship between mandibular repositioning therapy and its effect on head posture, as well as the physical therapist who must appreciate the influence of head posture on initial tooth contact patterns. Two simple tests are discussed for the purpose of illustrating the mechanics of the sliding cranium theory, which adds a new dimension to the understanding of how head posture and craniomandibular function are inextricably linked together.

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Concomitant mandibular and head-neck movements during jaw opening–closing in man

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SUMMARY **To test the hypothesis of a functional relationship between the human mandibular and cranio-cervical motor systems, head-neck movements during voluntary mandibular movements were studied in 10 healthy young adults, using a wireless optoelectronic system for three-dimensional (3D) movement recording. The subjects, unaware of the underlying aim of the study, were instructed to perform maximal jaw opening–closing tasks at fast and slow speed. Movements were quantified as 3D movement amplitudes. A consistent finding in all subjects was parallel and coordinated head-neck movements during both fast and slow jaw opening–closing tasks. Jaw opening was always accompanied by head-neck extension and jaw closing by head-neck flexion. Combined movement and electromyographic recordings showed concomitant neck muscle activity during head-neck movements, indicative of an active repositioning of the head. No differences in 3D movement amplitudes could be seen with respect to speed. The head movement was 50% of the mandibular movement during jaw opening, but significantly smaller (30–40%), during the jaw closing phase. In repeated tests, the 3D movement amplitudes of the concomitant head movements were less variable during slow jaw movement and during the jaw opening phase, than during fast and jaw closing movements, suggesting speed- and phase-related differences in the mechanisms controlling the integrated mandibular and head-neck motor acts. The present results give further support to the concept of a functional trigemino-cervical coupling during jaw activities in man.**

Introduction

Animal studies have demonstrated close connections between the trigeminal and the neck neuromuscular systems, through documentation of trigeminal somatosensory afferent projection to the upper cervical spinal cord, both with neurophysiological (Sherrington, 1898; Kerr & Olafson, 1961; Kerr, 1972; Abrahams & Richmond, 1977; Sumino, Nozaki & Katoh, 1981; Westberg & Olsson, 1991; Alstermark *et al*., 1992; Abrahams *et al*., 1993) and neuroanatomical (Matsushita *et al*., 1981; Ruggiero, Ross & Reis, 1981; Sumino *et al*., 1981; Chang *et al*., 1988; Tellegen & Dubbeldam, 1994) techniques. Mechanical, thermal and electrical stimulation at different levels of the trigeminal nerve, the trigeminal ganglion, and the trigeminal sensory nuclei, has been found to readily elicit neck motoneurone activity. The existence of a trigemino-neck reflex was reported by Manni *et al*. (1975) and Sumino & Nozaki (1977), and convergence of neck muscle and trigeminal somatosensory afferents onto neck motoneurones has been demonstrated (Abrahams *et al*., 1979; Chudler, Foote & Poletti, 1991). It has also been shown that these trigemino-spinal pathways within the brainstem are relatively direct and fast (Abrahams & Richmond, 1977; Alstermark *et al*., 1992; Abrahams *et al*., 1993).

Connections between the trigeminal system and the neck motoneurones is of importance for head withdrawal reactions in all species, as any sudden or unex-

pected stimulus in the oro-facial region leads to fast head aversion, hence paralleling the flexor reflex of the limbs (Abrahams & Richmond, 1977). Such connections are also likely to be critical in coordinating jaw and head motions in timing of jaw opening and closing with head-neck movements during daily activities such as eating and communication. Furthermore, from a phylogenetic perspective, it should be of considerable survival value in all species to optimally direct the mandibular system, for example during the catching of a prey and defence behaviour. Taken together, a line of evidence suggest that afferent activity from the oro-facial region must be considered as a significant input to the head-neck motor control mechanisms (Donevan & Abrahams, 1993).

In man, a close functional coupling between the temporomandibular and the cranio-cervical regions is suggested by their intimate anatomical and biomechanical relationships (Thompson & Brodie, 1942; Brodie, 1950; Kraus, 1988; Sobotta, Staubesand & Taylor, 1990). Free neck movements are a prerequisite for natural maximal mouth opening, and a reduced head extension ability may limit the three-dimensional space for the mandibular movement, due to impingement of the mandible with suprahyoid and airway structures. Furthermore, studies in humans have reported reflex activities in the neck muscles following electrical stimulation of trigeminal nerve branches (Broser, Hopf & Hufschmidt, 1964; Goor, 1984; Sartucci, Rossi & Rossi, 1986; Browne *et al.*, 1993; Di Lazzaro *et al.*, 1996), corroborating findings in animal studies of trigeminal projections onto the motoneurones of the neck muscles. Notably, the earliest reflex found in the human embryo is the trigemino-neck reflex, which consists of contraction of neck muscles elicited by light touch of the perioral region (Humphery, 1952). Finally, simultaneous activation of jaw and neck-shoulder muscles have been demonstrated during mandibular movements (Halbert, 1958; Davies, 1979), during jaw clenching (Hagberg, Agerberg & Hagberg, 1985; Widmalm, Lillie & Ash, 1988; Clark *et al.*, 1993) and following change in body posture (Forsberg *et al.*, 1985).

As early as in 1748, Ferrein observed that the upper jaw takes part in human jaw-opening movement (Ferrein, 1748). Theoretical analysis by Mollier (1929) supported Ferrein's observations and later Bauer (1964) verified this concept by means of a photographic method. In a recent short communication we have reported, from kinesiographic analyses, that natural voluntary jaw movements in man are accompanied by finely tuned and reproducible movements of the head and neck (Zafar *et al.*, 1995). The purpose of the present study was to further test the hypotheses of a functional relationship between the human mandibular and cranio-cervical motor systems by quantifying the relative amplitude of mandibular and head-neck movements during maximal jaw opening–closing tasks, performed at fast and slow speeds. A high precision wireless optoelectronic system for three-dimensional movement recording was used to monitor mandibular and head-neck movements in healthy young adults. Preliminary data of some of the material have earlier been reported in abstract form (Eriksson *et al.*, 1993; Nordh *et al.*, 1993).

Materials and methods

Subjects and movement recording

Ten healthy young adults, five males and five females (aged 22–45 years; median age 24 years), volunteered to take part in the study, according to the principles given by the World Medical Association's declaration of Helsinki. The subjects were unaware of the underlying aim of the investigation. They were comfortably seated in an upright position, with firm back support up to mid-scapular level, without head support. Movements of the mandible and the head-neck complex (here denoted head) were simultaneously recorded in three dimensions (3D) using a wireless optoelectronic recording system of high accuracy and reliability*; for technical details see Josefsson, Nordh & Eriksson, 1996. The system registered the 3D movements of stroboscopically illuminated retro-reflective markers, fixed to the mandible and the head (see below), at a frequency of 50 Hz. The camera set-up allowed mandibular and head-neck movements to be recorded within a working volume of 45 cm × 55 cm × 50 cm, and with a spatial resolution of ±0·02 mm. During the movement recording, the two-dimensional locations of each marker's geometric centroid, as viewed by each camera, were determined on-line by the system hardware and digitally sampled, whereas the 3D location of the markers were computed off-line. The latter procedure included a display of each marker's trajectory for visual inspection and verification of marker identification.

* MacReflex®; Qualisys AB, Sävedalen, Sweden.

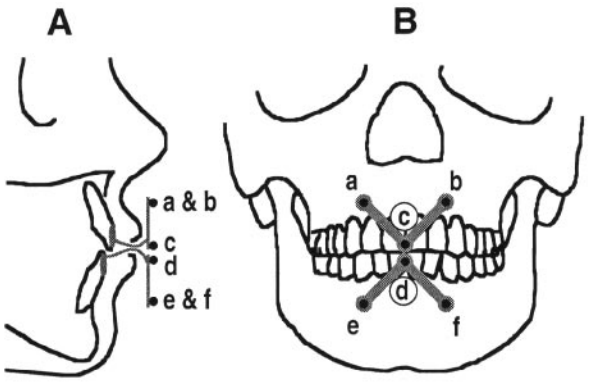

Fig. 1. Fixation of mounts to the labial surfaces of upper (with reflex markers a, b and c) and lower (with reflex markers d, e and f) incisors, is schematically shown in lateral (A) and frontal (B) views. Markers a and b on the head mount and e and f on the mandibular mount overlap in the lateral view.

Individual mounts, made of acrylic plastic and dental steel wire, with triplets of spherical (5 mm diameter) retro-reflective markers were attached to the lower and upper incisors, respectively (Fig. 1). The mounts were glued to the labial surfaces of the teeth with composite material[†]. They were fixed with care to avoid interference with occlusion and lip closure. The weight of the mount with markers was 1·5 (0·02) g [mean (standard deviation)]. One of the markers on each mount (markers c and d in Fig. 1) was positioned in the midline of the face, in front of the lower and upper central incisors, respectively. The mean distance between the upper incisors and marker c was 24 (2·0) mm, and between the lower incisors and marker d it was 23 (2·6) mm. The three markers on each mount determined two arbitrarily oriented planes, each with a rigid relation to the mandible and the head, respectively. Specially developed computer algorithms for calculations of relative body segment movements (cf. Selvik, 1974; Söderkvist, 1990) were used for computation of the 3D mandibular movements relative to the head. Thus, movements of the mandibular triplet were expressed within the coordinate system of the head triplet, in spite of simultaneous head-neck movements. After this segmental movement compensation, the data for the markers c and d were used to describe the head-neck and the mandibular positions in subsequent analyses and displays.

[†] Prismafil®; Densply, Germany.

Test procedure and definitions of observation parameters

The subjects executed a test sequence containing two maximal jaw opening–closing tasks, one at a fast and one at a slow speed, recorded during a 12 s period. They were instructed to perform two maximal jaw opening–closing movement cycles, one 'as fast as possible' and one 'slow'. The movements were self-paced and performed without feedback or detailed instructions. In the same recording session, this test sequence was recorded twice for every subject. In addition, the subjects were instructed to perform a sequence of consecutive self-paced maximal jaw opening and closing movement cycles. This test was supplemented by surface electromyography (EMG) monitoring of myoelectric activity in the masseter, suprahyoid, sternocleidomastoid and upper trapezius muscles. Analysis of these consecutive movement patterns was not pursued in detail for the present report.

A graphical illustration including definitions of the positions used to quantitatively assess the individual 3D movement amplitudes is given in Fig. 2. All movements started with the teeth close together in the intercuspal position and ended in the same position. The start and the end of the jaw opening–closing movement cycle were defined as the time-points at which the mandible moved away from the pre-movement position (Mand-pre) and at which it returned again to the Mand-pre, respectively. The position of the mandible at maximal jaw opening was termed Mand-max. The opening phase of the mandibular movement started at the Mand-pre and ended at the Mand-max. The jaw closing phase started at the Mand-max and ended at the Mand-pre. The head movement corresponding to the jaw opening phase was defined to start at the time-point when the head marker started to move from the pre-movement position (Head-pre), and to end at the time-point when the head reached its maximally displaced position (Head-max). Similarly, the head movement corresponding to the jaw closing phase started at the Head-max and ended at the time-point of the mandibular closing phase (Head-post).

The maximal amplitude of the 3D mandibular movement (in relation to the head), and the 3D head movement (in space), during the jaw opening and closing phases, were termed 3D-Mand and 3D-Head, respectively. The 3D-Mand and 3D-Head were calculated as the shortest 3D distance between the defined

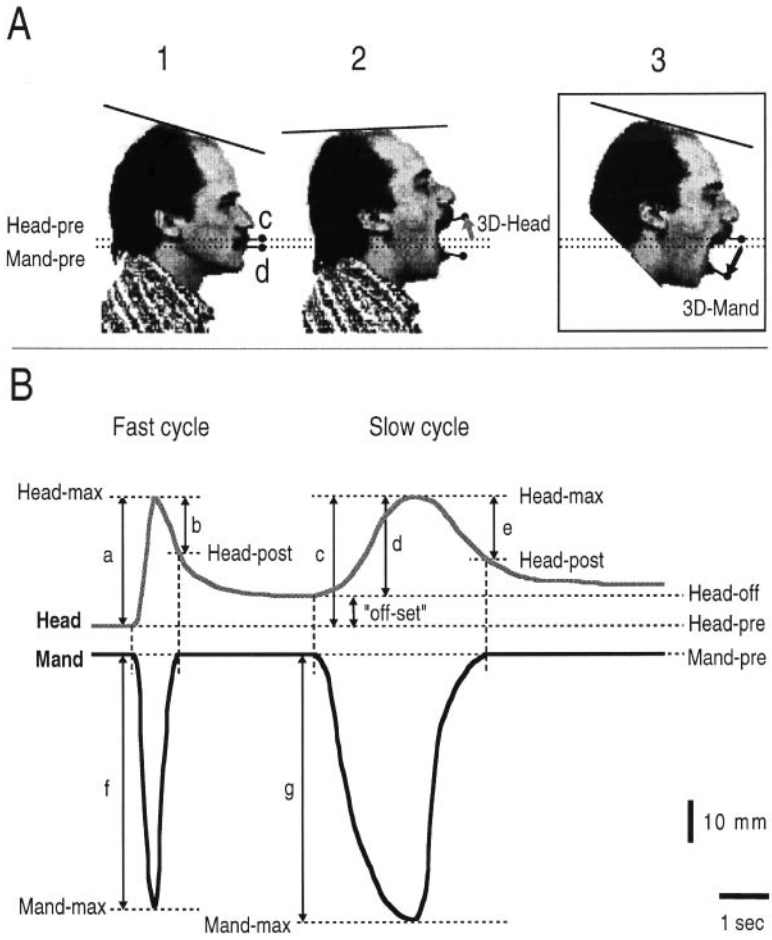

Fig. 2. A: Illustration of head (3D-Head, arrow in 2) and mandibular (3D-Mand, arrow in 3) 3D movement amplitudes. Line above the head indicates change in head position. (1) Head marker c and mandibular marker d (cf Fig. 1) at start positions Head-pre and Mand-pre (dotted lines). (2) Position of head and mandible at maximal jaw opening. Note relatively small movement of mandible in space. (3) The concept of mathematical compensation for head movement to calculate 3D mandibular movement in relation to the head (3D-Mand). B: Length–time relation of 3D movement amplitudes of the mandible in relation to the head (Mand), and the head-neck in space (Head), during fast and slow jaw opening-closing movements (data from subject no. 5 in Table 1). Double head arrows indicate the maximal amplitude of 3D movement of the mandible and the head for different phases of movements. a = 3D-Head during the fast jaw opening phase; b = 3D-Head during the fast jaw closing phase; c = 3D-Head during the slow jaw opening phase (from Head-pre position); d = 3D-Head during the slow jaw opening phase (from Head-off position); e = 3D-Head during the slow jaw closing phase; f = 3D-Mand during the fast jaw opening and closing phases; and g = 3D-Mand during the slow jaw opening and closing phases. The start and end positions of mandibular and head 3D movement amplitudes have been labelled (for explanation, see text). Length and time bars in lower right corner.

start and end positions of each phase (Fig. 2) according to the formula:

$$\sqrt{(x_b - x_a)^2 + (y_b - y_a)^2 + (z_b - z_a)^2}$$

where x, y and z denote the coordinate values in the x-, y-and z-dimensions, respectively, and a and b indicate the pre-movement and the maximally displaced positions. The 3D-Mand for the opening phase was defined as the shortest 3D distance between the Mand-pre and the Mand-max positions. By definition of the Mand-pre and Mand-max positions, the 3D-Mand values of the jaw opening and the closing phases were equal. The 3D-Head during the jaw opening phase was defined as the shortest 3D distance between the Head-pre and the Head-max positions. Similarly, the 3D-Head during the jaw closing phase was defined as the shortest 3D distance between the Head-max and the Head-post positions. For each subject the mean of two recordings was estimated for analyses.

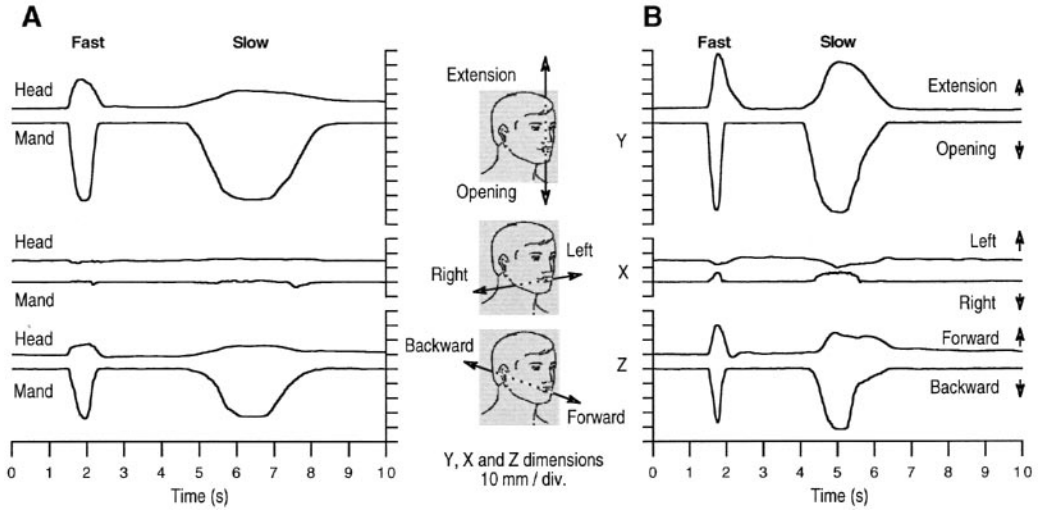

Fig. 3. Traces of position-time data of mandibular (Mand) and associated head-neck (Head) movements during fast and slow jaw opening-closing tasks in two subjects (A) and (B). Upper, middle, and lower panels show the 3D movement as viewed in superio-inferior (*y*), medio-lateral (*x*) and ventro-dorsal (*z*) dimensions, respectively. Note the individual movement patterns, despite the overall similarity.

Statistical analyses

The precision of the estimation of the maximal jaw opening movement was assessed from duplicate recordings of the 3D-Mand. The standard deviation (*S*) of repeated measurements on the same subject was estimated according to the formula (Bland, 1988):

$$s = \sqrt{\frac{1}{2n} \Sigma (x_i - y_i)^2}$$

where the first and the second readings of the 3D-Mand are x_i and y_i for i = 1 to *n* (*n* = number of subjects). The measurement error of the 3D-Mand, expressed as the coefficient of variation, was 2·4% for the fast and 1·7% for the slow jaw opening movement. The variability of the recorded head movements associated with jaw opening–closing was assessed by the same method (see results section).

Computer off-line analyses of 3D movement characteristics were performed with conventional mathematical and statistical descriptions and tests, using standard computer software. Mean, range and standard deviation were used for descriptive statistics. The hypothesis of no difference between the amplitudes of the 3D mandibular and the 3D head movements, and with respect to the phase of movement, speed and gender, was tested by the Wilcoxon signed-rank test with a significance level of 0·05.

Results

A consistent finding in all tests and subjects was the parallel and coordinated head-neck movements occurring during both jaw opening and jaw closing, at fast as well as at slow speed (Fig. 2). Jaw opening was always accompanied by head-neck extension, and jaw closing by head-neck flexion. All subjects showed a basic similarity in behaviour, although individual differences were apparent. In the repeated recordings, individual characteristics of the mandibular and the head-neck movement patterns allowed identification of subjects from their unique 'jaw-head traces'. Figure 3 shows typical mandibular and head-neck movement patterns from two subjects displayed in the, superio-inferior (*y*), medio-lateral (*x*) and ventro-dorsal (*z*) dimensions.

During the test with consecutive jaw opening–closing movements, simultaneous EMG activity of the suprahyoid and neck muscles was observed during the jaw opening phase. During the jaw closing phase, there was an increase in EMG activity in the masseter muscle accompanied by a decrease in neck and suprahyoid muscle activity (Fig. 4).

A general observation during both the fast and the slow jaw opening–closing tasks was that the head did

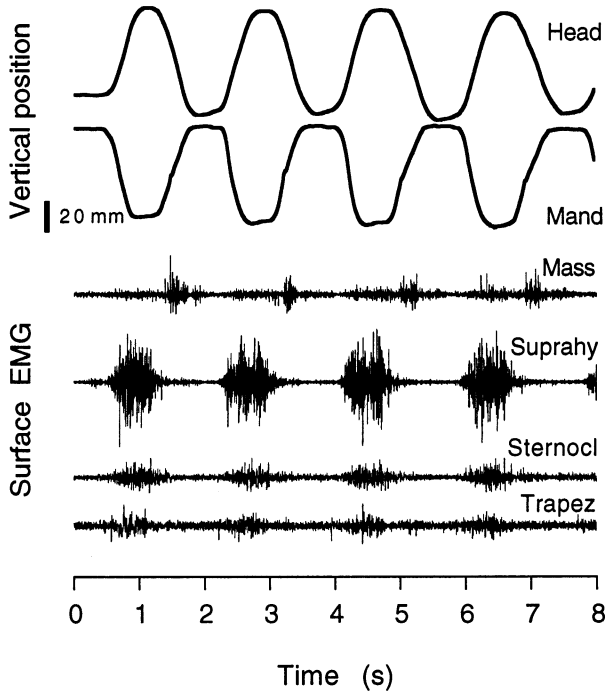

Fig. 4. Kinesiographic and EMG records (subject no. 3 in Table 1) of mandibular and associated head-neck movements during four consecutive voluntary jaw opening–closing tasks. The traces show (from top): vertical head-neck (Head) and mandibular (Mand) positions, surface EMG records from the right masseter (Mass), suprahyoid (Suprahy), sternocleidomastoid (Sternocl), and upper trapezius (Trapez) muscles. Note the consistent activation of sternocleidomastoid and upper trapezius muscles along with suprahyoid muscles during jaw opening and the decrease in activity of these muscles along with an increase in masseter muscle activity during jaw closing.

not return to its starting position (Head-pre) after completion of the fast jaw movement cycle, but instead remained in an 'offset' position (Head-off) (Fig. 2). At the start of the subsequent slow cycle, this 'offset' averaged 15·2% of the 3D-Head during the fast jaw opening phase for males and 15·4% for females. For this reason, the 3D-Head during the slow jaw opening phase was calculated both from the Head-pre and the Head-off positions (Fig. 2, Table 1).

The variability of the recorded head movements, expressed as the coefficient of variation (CV), was 14% during the fast jaw opening phase and 28·9% during the fast jaw closing phase. For the slow jaw opening phase, the corresponding values were 8·7% (calculated from Head-pre) and 7·2% (from Head-off), and for the slow jaw closing phase, 15·9%.

Figure 5 and Table 1 show the 3D-Mand and 3D-Head, during jaw opening–closing tasks at fast and at slow speed for all subjects. The mean values in the female group were lower than those of the male group, although no statistically significant differences were found.

For the entire group, the 3D-Head was approximately 50% of the 3D-Mand during the jaw opening phase, and 30–40% during the jaw closing phase, both at fast and at slow speed (Fig. 5 and Table 1). The relationship between the 3D-Mand and 3D-Head during the jaw opening and jaw closing phases was also estimated as the ratio (3D-Head/3D-Mand). The ratio was less than 1·0 for all subjects except for one male, and significantly smaller for the jaw closing phase than for the jaw opening phase, during both fast and slow speeds ($P = 0{\cdot}005$) (Fig. 6).

Discussion

The present experiments were aimed at quantifying head-neck movements associated with voluntary jaw movements in man. The results provide the first systematic documentation that jaw opening–closing movements are paralleled by active concomitant head-neck movements, extension during jaw opening, and head-neck flexion during jaw closing, albeit the instructions to the subject concerned mandibular movements only. The finding emphasizes the concept of a close functional relationship between the mandibular and the head-neck motor systems, as suggested by their anatomical and biomechanical inter-relationships, as well as by previous neuroanatomical, neurophysiological and clinical studies.

For mathematical as well as biomechanical reasons, two-dimensional recordings and/or projections may introduce error in estimations of the actual three-dimensional movement amplitudes. To avoid such error, the amplitudes of the mandibular and head movements were quantified as the 3D movement amplitude, since this parameter is unbiased by projection errors. The reliability of recording maximal jaw opening–closing movements, i.e. the task which the subjects were instructed to perform, was found to be good, as shown by the low coefficient of variation in repeated tests. In the present study the reflex markers were attached about 20 mm in front of the incisors. For biomechanical reasons, this marker arrangement resulted in an overestimation of the actual jaw gape of about 25%. However, this did not affect the interpretations of the present result, since our aim was to study the relative

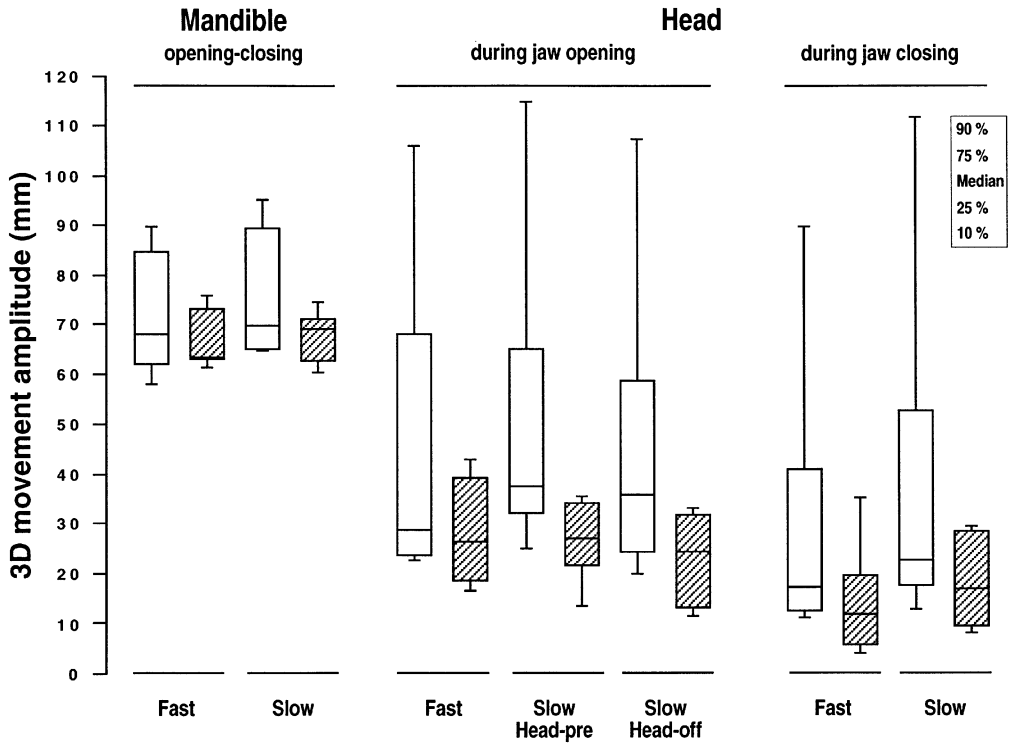

Fig. 5. Box and whisker plots summarizing the 3D movement amplitudes of the mandible and the head-neck (Head), during jaw opening–closing at fast and slow speed. Open boxes indicate males ($n = 5$) and hatched boxes females ($n = 5$). The 3D movement amplitude of the head during the slow jaw opening phase is shown from two positions, i.e. from Head-pre and Head-off (see 'Methods' and Fig. 2).

mandibular-head movement patterns, not the absolute jaw gape.

It could be argued that the observed head-neck movements are due to passive mechanical adjustments of the head relative to the cervical spine, as a result of variation in gravitational effects on the head mass. However, this is not likely, because in an upright position, the centre of gravity of the head lies in front of the atlanto-occipital junction, and the neck extensor muscles counteract the gravity, thus preventing the head from tilting forward (Kapandji, 1974; Vig, Rink & Showfety, 1983). This biomechanical situation does not change during jaw opening. Furthermore, an active repositioning of the head was suggested by the finding in the EMG records of concomitant jaw and neck muscle activity during the jaw opening–closing tasks, corroborating results of previous EMG studies of human jaw and neck muscle activation during jaw function (Halbert, 1958; Davies, 1979).

A consistent observation in all subjects and tests, irrespective of the speed of jaw movement, was that the head had not returned to its starting (premovement) position at the time-point when the jaw closing phase was completed. Thus, a larger amplitude of the head-neck movement was found during jaw opening than during jaw closing. This observation suggests that the setting parameters of the neuromuscular mechanisms controlling the functional interaction of the head-neck and jaw movements differ between the jaw closing and jaw opening phases. No differences in head movement amplitudes were observed with respect to speed of movement. However, the differences in CV values between the recorded head movements indicate differences in the stability of head movement control related to speed and phase of jaw movement. Lower head CV values seen for slow speed, suggest that associated head movements are activated in a more precise manner during slow than during fast jaw movements. The lower head CV values seen for the jaw opening phase may reflect a general phenomenon in mammalian jaw-head-neck behaviour; a requirement of well-controlled jaw opening movement in vital actions such as catching a prey and defence. The inter-individual variation in the movement amplitudes probably reflects individual

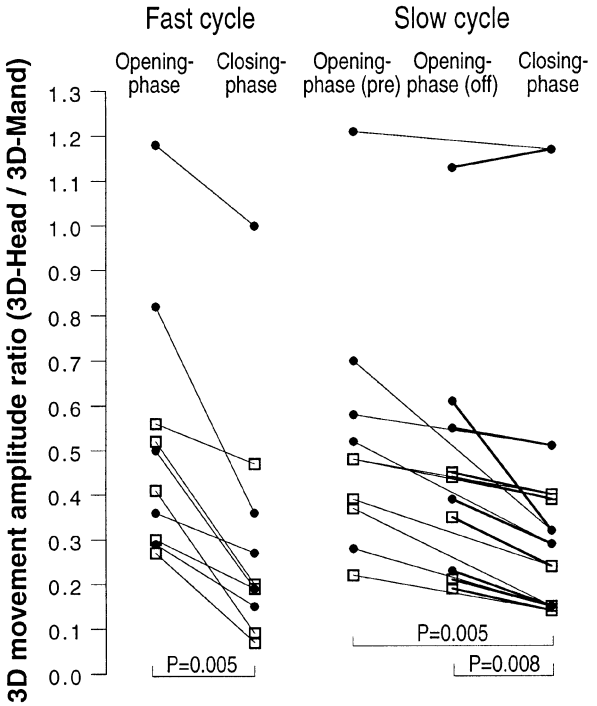

Fig. 6. Ratio of head versus mandibular 3D movement amplitudes for the jaw opening and closing phases during fast and slow speed. Data from five male (solid circles) and five female (open squares) subjects. For the slow cycle, the ratio of the opening phase is calculated from two positions, Head-pre (pre) and Head-off (off) (bold lines) (see 'Methods' and Fig. 2). Note that the ratio is below 1·0 for all subjects but one male (subject no. 3 in Table 1), and the significant differences in ratio between the jaw opening and the jaw closing phases.

differences in anatomy as well as in coordination mechanism of integrated jaw and the head-neck function. However, the observation of smaller movement amplitudes for the female group than for the male group may suggest gender differences in normative data, and merits further investigation.

It has been proposed that the neck muscles directly moving the head will require a control system at least as complex, if not even more, than the better understood mechanisms for limb motor control (Abrahams *et al.*, 1993). The human head-neck movements require an adequate control of at least 27 pairs of muscles (Kendall & McCreary Kendall, 1983) and 37 joints (Bland & Boushey, 1992) in the cranio-cervical region. As the neck region seems to possess a relatively small representation in the sensori-motor cortex areas (Penfield & Rasmussen, 1950), it can be assumed that subcortical mechanisms are of significant importance in the control of head-neck movements. Numerous studies in various species have indeed shown that the function of the neck motor system is significantly influenced by visual and vestibular inputs (cf. Dutia, 1991; Berthoz, Grawf & Vidal, 1992). However, most species exhibit a wide range of head-neck movements in activities that are influenced or initiated by input from oro-facial structures (Abrahams *et al.*, 1993). Trigeminal primary afferents do not only project to the trigeminal sensory nuclei, but also to cells in the dorsal horn of the upper cervical spinal cord, and may reach the C7 segment and many other 'non-trigeminal' areas in the central nervous system (Marfurt & Rajchert, 1991). In fact, trigeminal projections to all levels of the spinal cord have been reported in the rat (Ruggiero *et al.*, 1981). In decerebrate cats even treadmill locomotion has been induced by mechanical stimulation of various parts of the face, after microinjection of neuroactive substances into the area of the trigeminal spinal nucleus (Noga, Kettler & Jordan, 1988). Thus, trigeminal afferents seem to strongly affect neck motoneurons, through widespread connections. These connections should allow critical trigeminal modulation of head-neck movements in specific tasks, such as head orientation in aversive movements, feeding and tactile or olfactory exploration. The results of the present study in man corroborate this notion.

Several underlying mechanisms may account for the consistent nature of the head-neck movements. It can be speculated that descending activation from cortical or subcortical structures, modulated by proprioceptive and somatosensory reflexes, may contribute to the final movement pattern. There is evidence of trigeminal proprioceptive input to the cerebellum, directly (Jacquart & Strazielle, 1990; Donga & Dessem, 1993) as well as indirectly via the mesencephalic nucleus (Eller & Chan-Palay, 1976; Ito, 1984; Nomura & Mizuno, 1985; Elias, 1990), which in turn may act on neck motoneurones via a variety of pathways (cf. Hirai, 1988). Strong reflex modulation of neck motoneurones may also be initiated by proprioceptors in the jaw closing muscles, which are known from studies in man to be of extraordinary large size and complexity (Eriksson & Thornell, 1987; Eriksson, Butler-Browne & Thornell, 1994), and by intricate somatosensory input from the oro-facial region (Trulsson, 1993). Recent anatomical (Cowie, Smith & Robinson, 1994) and functional (Cowie & Robinson, 1994) studies in adult rhesus monkey have identified a functional zone within the medullary reticular formation, denoted the gigantocellular reticular nucleus, with an important role in head

Table 1. 3D movement amplitudes (mm) of the mandible (3D-Mand) and the head-neck (3D-Head) during maximal jaw opening-closing movements at fast and slow speed, summarized for five male and five female young adults. For the slow cycle, the 3D-Head during the mandibular opening phase was calculated at two starting positions, Head-pre and Head-off (see 'Methods' and Fig. 2). Mean ($\overline{X}$) and range of duplicate tests. Mean ($\overline{X}$) and standard deviation (s.d.) for group values

	3D-Mand				3D-Head									
	Opening-closing				During jaw opening						During jaw closing			
	Fast		Slow		Fast		Slow Head-pre		Slow Head-off		Fast		Slow	
Subjects	$\overline{X}$	Range	$\overline{X}$	Range	$\overline{X}$	Range	$\overline{X}$	Range	$\overline{X}$	Range	$\overline{X}$	Range	$\overline{X}$	Range
1 Male	83·0	3·7	87·5	2·1	23·7	3·3	24·9	2·0	19·9	0·9	12·5	0·7	12·8	2·5
2 Male	68·2	2·7	70·0	0·9	55·7	19·1	48·9	10·1	42·8	4·3	24·6	11·9	22·6	14·0
3 Male	89·8	2·6	95·3	1·9	106·3	2·3	114·9	6·0	107·5	0·5	89·8	22·2	111·9	11·8
4 Male	63·1	0·8	64·8	2·1	22·6	2·0	37·4	7·6	35·9	6·3	17·3	11·5	32·9	4·5
5 Male	58·2	3·3	65·0	1·2	28·9	7·3	34·1	2·6	25·3	3·3	11·3	5·1	18·5	6·1
6 Female	63·6	1·9	63·4	1·1	26·3	2·7	23·8	0·8	13·4	3·4	5·9	3·2	9·6	3·7
7 Female	72·3	2·2	74·7	0·0	37·9	9·0	35·6	1·6	33·2	0·3	14·5	6·7	29·4	2·7
8 Female	63·3	1·1	60·4	3·2	18·9	4·9	13·5	0·5	11·6	0·7	11·8	2·6	8·2	0·4
9 Female	75·9	2·6	70·3	2·0	42·8	3·8	33·5	4·4	31·3	5·9	35·3	1·5	28·1	1·2
10 Female	61·4	0·4	69·2	0·6	16·7	1·3	27·0	2·1	24·3	2·3	4·1	1·9	16·8	3·3
Males (n = 5)														
$\overline{X}$	72·5		76·5		47·4		52·0		46·3		31·1		39·8	
s.d.	13·4		14·0		35·6		36·2		35·4		33·2		41·0	
Females (n = 5)														
$\overline{X}$	67·3		67·6		28·5		26·7		22·8		14·3		18·4	
s.d.	6·4		5·7		11·5		8·8		10·0		12·5		10·0	
Group (n = 10)														
$\overline{X}$	69·9		72·1		38·0		39·4		34·5		22·7		29·1	
s.d.	10·3		11·1		26·8		28·2		27·4		25·3		30·3	

orientation as well as in concomitant jaw, facial, tongue and upper limb movements. The region has numerous afferent inputs from subcortical and cortical systems, and it projects to motor and interneurons of the trigeminal, facial, hypoglossal and propriospinal nuclei, the caudal medulla and the cervical spinal cord. It is supposed to operate as a generator of head movement patterns and the motor activity that accompanies head movements. Cortical inputs might elicit movements through activation of the gigantocellular region, which integrates different afferent signals to generate the synergistic patterns of head and associated oro-facial and upper limb movements (Cowie & Robinson, 1994). Such an integration would then be an important basis for coordinated actions involving the head, jaw, arms and hands, for example in eating behaviour.

Finally, the clinical implications of the present results for dental and medical practice should be emphasized. Clinical studies have reported pain and dysfunction in the jaw and oro-facial regions in association with cranio-cervical pain and dysfunction following head-neck injury. Similarly, pain in the cervical region has been demonstrated in relation to functional disorders in the jaw system (for review, see Mannheimer & Dunn, 1991). There is also evidence that treatment of temporo-mandibular disorders may significantly reduce pain and disorder even in the cervico-brachial region (Kirveskari & Alanen, 1984; Kirveskari *et al*., 1988; Karppinen *et al*., 1996). Moreover, studies in rat have demonstrated a sustained activation of both jaw and neck muscles following stimulation of cervical paraspinal tissues by an inflammatory irritant (Hu *et al*., 1993). These experiments suggest that noxious stimulation, e.g. following head-neck injuries, can produce a sustained excitation of jaw muscles. In the light of previous data and the present findings, a multidisciplinary assessment and

management of patients suffering from pain and dysfunction in the jaw, face and cranio-cervical regions seems justified.

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Influences of head positions and bite opening on collapsibility of the passive pharynx

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Isono, Shiroh, Atsuko Tanaka, Yugo Tagaito, Teruhiko Ishikawa, and Takashi Nishino. Influences of head positions and bite opening on collapsibility of the passive pharynx. *J Appl Physiol* 97: 339–346, 2004. First published March 12, 2004; 10.1152/japplphysiol.00907.2003.—A collapsible tube surrounded by soft material within a rigid box was proposed as a two-dimensional mechanical model for the pharyngeal airway. This model predicts that changes in the box size (pharyngeal bony enclosure size anatomically defined as cross-sectional area bounded by the inside edge of bony structures such as the mandible, maxilla, and spine, and being perpendicular to the airway) influence patency of the tube. We examined whether changes in the bony enclosure size either with head positioning or bite opening influence collapsibility of the pharyngeal airway. Static mechanical properties of the passive pharynx were evaluated in anesthetized, paralyzed patients with sleep-disordered breathing before and during neck extension with bite closure ($n = 11$), neck flexion with bite closure ($n = 9$), and neutral neck position with bite opening ($n = 11$). Neck extension significantly increased maximum oropharyngeal airway size and decreased closing pressures of the velopharynx and oropharynx. Notably, neck extension significantly decreased compliance of the oropharyngeal airway wall. Neck flexion and bite opening decreased maximum oropharyngeal airway size and increased closing pressure of the velopharynx and oropharynx. Our results indicate the importance of neck and mandibular position for determining patency and collapsibility of the passive pharynx.

obstructive sleep apnea; upper airway; closing pressure; neck positions; mouth opening

A PATENT PHARYNGEAL AIRWAY IS crucial for stable breathing. Pharyngeal airway size is regulated by a precise interaction between neural regulation of pharyngeal airway dilator muscle activities (neural mechanisms) and structural properties of the pharyngeal airway (anatomical mechanisms) (7). Through elimination of the neural mechanisms by administration of a muscle blockade under general anesthesia, we demonstrated that closing pressures of the passive pharynx were distinctively higher in patients with sleep-disordered breathing (SDB) than in normal subjects (3). Furthermore, specific structural abnormalities, such as obesity and craniofacial anomaly, contributed to increased collapsibility of the passive pharynx (24).

Structurally, the pharyngeal airway is surrounded by soft tissue such as the tongue, which is enclosed by bony structures such as the mandible and cervical vertebrae (Fig. 1). Consequently, a collapsible tube surrounded by soft material within a rigid box was proposed as a two-dimensional mechanical model for the pharyngeal airway (24). Pharyngeal bony enclosure size, anatomically defined as cross-sectional area bounded by the inside edge of the bony structures and being perpendicular to the airway, corresponds to the box size of the mechanical model. The contribution of obesity and craniofacial anomaly such as a small maxilla and mandible to the increased collapsibility of the passive pharynx was well explained by the mechanical model (24).

Within one subject, the pharyngeal bony enclosure size varies with head and mandible positioning changes, which may be an influential factor of the pharyngeal airway patency. Cervical extension with bite closure (neck extension) increases the distance between the mentum and cervical column, which consequently increases the bony enclosure size, whereas cervical flexion with bite closure (neck flexion) decreases it. Bite opening with neutral neck position decreases the distance between the mentum and cervical column, which consequently increases the bony enclosure size without head positioning change. Accordingly, the purpose of this study was to evaluate influences of head positions and bite opening on static mechanical properties of the passive pharynx in anesthetized and paralyzed patients with SDB.

MATERIALS AND METHODS

Subjects and overnight oximetry. The study consisted of 24 male patients with SDB who were interested in undergoing uvulopalatopharyngoplasty and were scheduled to undergo endoscopic pharyngeal assessment to determine their indications for this procedure (4). All had histories of excessive daytime sleepiness, habitual snoring, and witnessed repetitive apnea. SDB was evaluated by a pulse oximeter (Pulsox-5; Minolta, Tokyo, Japan). All subjects were instructed to attach an oximetry finger probe before sleep and to remove the probe on awakening. Digital readings of arterial oxygen saturation (Sa_{O_2}) and pulse rate were stored every 5 s in a memory card. The stored data were displayed on a computer screen to check the quality of the recordings. The computer calculated oxygen desaturation index, defined as the number of oxygen desaturation exceeding 4% from the baseline, and the percent of time spent at $Sa_{O_2} < 90\%$. Table 1 lists all nocturnal oximetry data and anthropometric characteristics. Although the oximetry evaluation alone does not clarify the nature of SDB, we believe that all patients can be safely diagnosed as having obstructive sleep apnea (OSA) on the basis of the oximetry results and the clinical symptoms (1).

Informed consent was obtained from all subjects after the aim and potential risks of the study were fully explained to each. The investigation was approved by the hospital ethics committee of our institution.

Preparation of the subjects. Each subject was initially premedicated with 0.5 mg of atropine and placed in the supine position on an operating table, where a modified tight-fitting nasal mask was attached. Care was taken to prevent air leaks from the mask, particularly

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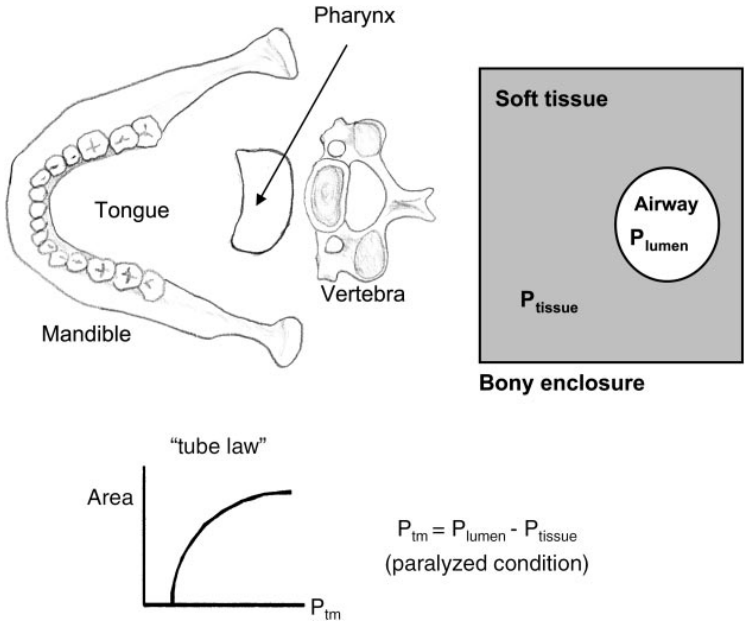

Fig. 1. Anatomic arrangements of the pharynx, tongue, mandible, and cervical vertebra (*left*) and a mechanical model for the structures (*right*). P_{lumen}, pressure inside the collapsible tube; P_{tissue}, pressure surrounding the collapsible tube; Ptm, transmural pressure. In the mechanical model, the luminal size of the collapsible tube is determined by mechanical properties of the tube (tube law) and Ptm. Ptm is defined as the pressure difference between pressures inside (P_{lumen}) and outside the tube (P_{tissue}). For a given P_{lumen}, an increase in P_{tissue} results in reduction of Ptm and therefore narrows the lumen. P_{tissue} is determined by the balance between the amount of soft material inside the rigid box and the size of the surrounding rigid box.

when the airway was pressurized above 20 cmH_2O. General anesthesia was induced and maintained by intravenous infusion of propofol, and intravenous injection of a muscle relaxant (vecuronium 0.2 mg/kg) produced complete paralysis throughout the experiment while the subject was ventilated with positive pressure through an anesthetic machine. Sa_{O_2}, electrocardiogram, and blood pressure were continuously monitored. The tip of a slim endoscope (FB10X, Pentax, Tokyo, Japan, 3 mm OD) was inserted through the modified nasal mask and the naris down to the upper airway to visualize the velopharynx (retropalatal airway), and the oropharynx (retroglossal airway). A closed-circuit camera (ETV8, Nisco, Saitama, Japan) was connected to the endoscope, and the pharyngeal images were recorded on a videotape. Reading of airway pressure (Paw), measured by a water manometer, was simultaneously recorded on videotape.

Experimental procedures. To determine the pressure-area relationship of the pharynx, after disconnection from the anesthetic machine the nasal mask was connected to a pressure-control system capable of accurately manipulating Paw from −20 to 20 cmH_2O in a stepwise fashion. At cessation of mechanical ventilation of the subject under complete muscle paralysis, apnea resulted. Paw was immediately increased up to 20 cmH_2O to dilate the airway and then gradually reduced, within a 2- to 3-min span, from 20 cmH_2O to the closing pressure of the retropalatal airway in a stepwise fashion. The latter represented the pressure at which complete closure of the retropalatal airway occurred, as evident on the video screen. The apneic test was terminated when Sa_{O_2} fell below 95%. This procedure of experimentally induced apnea allowed construction of a pressure-area relationship of the visualized pharyngeal segment. The subject was manually ventilated for at least 1 min before and after the apneic test. Distance between the tip of the endoscope and the narrowing site was measured with a wire passed through the aspiration channel of the endoscope.

Table 1. *Anthropometric characteristics and results of nocturnal oximetry*

Age, yr	47.0 (38.0–54.5)
Weight, kg	77.7 (69.0–82.8)
Height, m	1.72 (1.66–1.77)
BMI, kg/m^2	26.4 (23.7–28.4)
ODI, h^{-1}	27.7 (20.8–49.5)
CT_{90}, %	12.0 (2.0–23.1)
Nadir Sa_{O_2}, %	87.9 (84.9–90.3)
Lowest Sa_{O_2}, %	72.0 (61.0–80.5)

Values are medians (25–75 percentiles) of all patients (n = 24). BMI, body mass index; ODI, oxygen desaturation (Sa_{O_2}) index defined as number of desaturations exceeding >4% per hour; CT_{90}, percent of time spent Sa_{O_2} <90%; Nadir Sa_{O_2}, mean of the nadir Sa_{O_2} values in all desaturation events; Lowest Sa_{O_2}, a lowest Sa_{O_2} value among the desaturation events.

Each patient participated in either the head position study (n = 13) or the bite opening study (n = 11) (Fig. 2). In the head position study, in addition to the control measurement (head and neck in neutral position with bite closed by a chin strap), the apneic tests were repeated during both neck extension (neck maximally extended by placing cushions under the shoulders with bite closed by a chin strap) and neck flexion (neck maximally flexed by placing cushions under the head with bite closed by a chin strap) in seven patients, whereas the tests were performed either during only neck extension (n = 4) or neck flexion (n = 2) in the remaining six patients. The control measurement was performed once per patient. In the bite opening study, the bite was widened by inserting a mouthpiece between the upper and lower incisors in head and neck in the neutral position, producing a 15-mm distance between the incisors (Fig. 2). The apneic test was initiated immediately after establishment of each experimental condition. On the completion of the experiment time span of 30–60 min, atropine (0.02 mg/kg) and neostigmine (0.04 mg/kg) were administered to reverse muscle paralysis.

Data analysis. To convert the monitor image to an absolute value of the pharyngeal cross-sectional area, magnification of the imaging system was estimated at 1.0-mm interval distances between the endoscopic tip and the object in the range of 5–30 mm. At a defined value of Paw, the image of the pharyngeal lumen was traced and pixels included in the area were counted (SigmaScan version 2.0, Jandel Scientific Software, San Rafael, CA). The pixel number was converted to pharyngeal cross-sectional area according to the distance-magnification relationship. Using known-diameter tubes, we tested the accuracy of the cross-sectional area measurements. For constant distance, the measured areas were systematically deviated from actual areas (Fig. 3); the largest known area tested

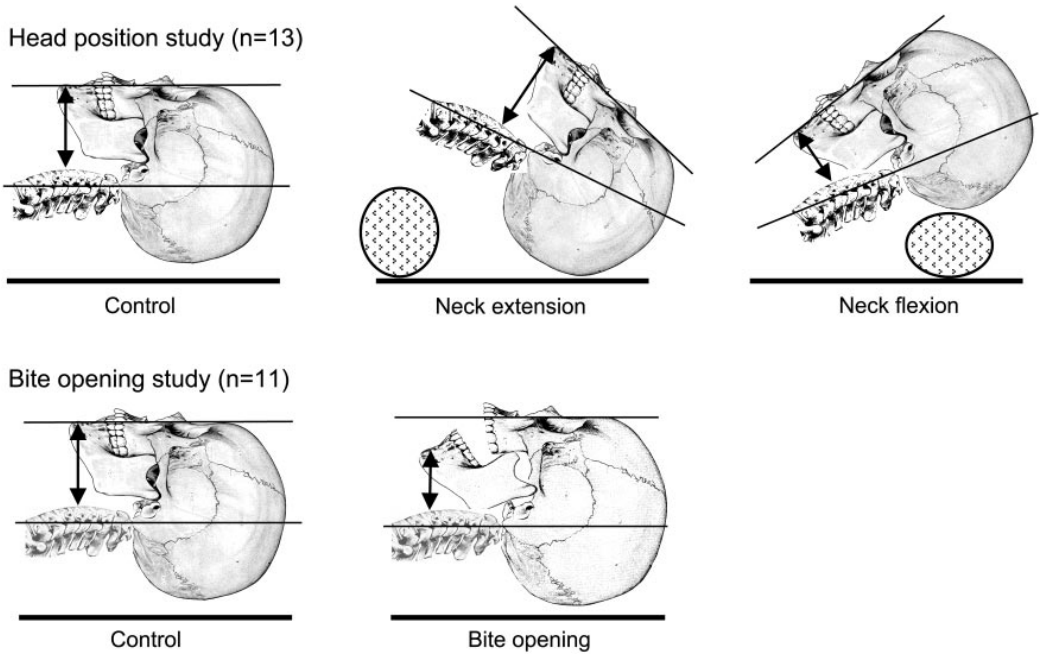

Fig. 2. Illustrative configuration of head, neck, and mandible positions for each experimental condition. Note that differences of the distance between the mentum and cervical column resulted from the configuration differences. A horizontal straight line for each condition represents the experimental table, and cushions were placed under the shoulders for the neck extension and placed under the head for the neck flexion.

(0.95 cm^2) was underestimated by 11% because of image deformation of the outer image area, and the smallest known area tested (0.03 cm^2) was overestimated by 13% because of reduction of the image resolution (5).

The measured luminal cross-sectional area was plotted as a function of Paw. The closing pressure was defined as pressure corresponding to the zero area. At high values of Paw, relatively constant cross-sectional areas were revealed; therefore, maximum area (A_{max}) was determined as the mean value of highest three Paw (18, 19, and 20 cmH_2O). The pressure-area relationship of each pharyngeal segment was fitted by an exponential function, $A = A_{max} - B \times \exp(-K \times \text{Paw})$, where B and K are constants. A nonlinear least square technique was used for the curve fitting, and the quality of the fitting was provided by the coefficient R^2 (SigmaPlot version 2.0, Jandel Scientific Software, San Rafael, CA). A regressional estimate of closing pressure (P'_{close}), which corresponds to an intercept of the curve on the Paw axis, was calculated from the following equation for each pharyngeal segment: $P'_{close} = \ln(B/A_{max})K^{-1}$. The shape of the pressure-area relationship was described by the value of K. When pressure-area relationship is curvilinear, compliance of the pharynx defined as a slope of the curve varies with changes in Paw; therefore, a single value of compliance calculated for a given Paw does not represent collapsibility of the pharynx for the entire Paw ranges. In contrast, K represents the rate of changes in the slope of the curve; therefore, when K is high, a small reduction in Paw results in a significant increase in compliance, leading to remarkable reduction in cross-sectional area. Consequently, collapsibility of the pharynx increases with increasing K. We suggest that both P'_{close} and K values represent collapsibility of the pharynx, whereby the former determines the position of the exponential curve and the latter characterizes the shape of the curve.

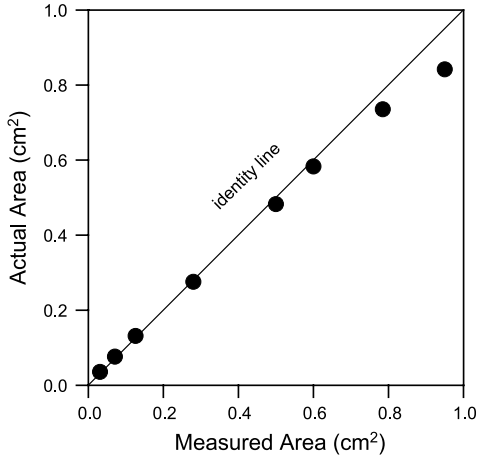

Fig. 3. Accuracy of our area measurement for a constant distance. The measured area was systematically deviated from actual area.

Statistical analysis. All values are expressed by median (25–75 percentiles). The Wilcoxon's signed-rank test was used for comparison between the control and other conditions. Linear regression analysis was performed between observed and estimated closing pressures. $P < 0.05$ was considered to be significant.

RESULTS

Effects of neck extension on static pharyngeal mechanics. Changes in static mechanical variables before and during neck extension are presented in Fig. 4. Neck extension approximately doubled A_{max} and significantly decreased both K and P'_{close} at the oropharynx. In addition to the significant influ-

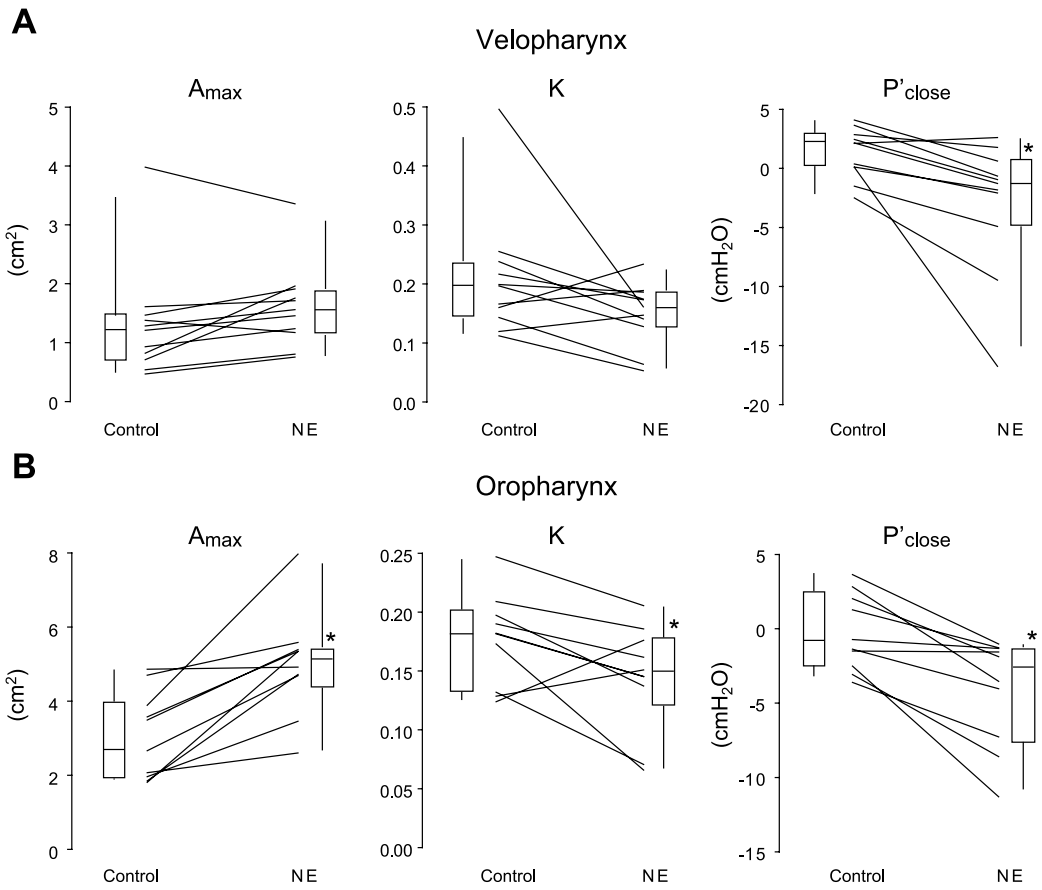

Fig. 4. Changes in static mechanical variables in response to neck extension (NE) at the velopharynx (*A*) and oropharynx (*B*). Each line represents a different patient. A_{max}, maximum cross-sectional area; *K*, a constant obtained by an exponential fitting the pressure-area relationship and representing stiffness of the pharyngeal airway wall; P'_{close}, estimated closing pressure calculated from the fitted exponential function. Lower and upper boundaries of the box indicate 25th and 75th percentages. Solid line within the box marks the median, and vertical lines indicate the 90th and 10th percentages. *$P < 0.05$ vs. control.

ences on the oropharyngeal segment, P'_{close} at the velopharynx also significantly decreased during neck extension. Notably, there were tendencies of increase in A_{max} ($P = 0.054$) and decrease in K ($P = 0.067$) at the velopharynx, as shown in Fig. 4, although these are not statistically significant. The results indicate that neck extension dilates and stiffens the velopharyngeal and oropharyngeal airway, improving pharyngeal airway patency.

Effects of neck flexion on static pharyngeal mechanics. Changes in static mechanical variables before and during neck flexion are presented in the Fig. 5. Neck flexion significantly decreased A_{max} and increased P'_{close} at the oropharynx. Velopharyngeal P'_{close} also increased during neck flexion. Neck flexion significantly reduced oropharyngeal airway size and increased airway collapsibility at both the velopharynx and oropharynx. K did not change in response to neck flexion.

Effects of bite opening on static pharyngeal mechanics. Changes in static mechanical variables before and during bite opening are presented in the Fig. 6. Bite opening significantly decreased A_{max} and increased P'_{close} at the oropharynx. Velopharyngeal P'_{close} also increased during bite opening. K did not change in response to bite opening. Notably, the pattern of changes in the pharyngeal mechanics during bite opening is similar to that during neck flexion.

Comparison between observed and estimated closing pressures. Figure 7 demonstrates the correlation between P'_{close} and observed P_{close} (the highest airway pressure at which complete closure of either the retropalatal or retroglossal airway was seen on the video screen) at the primary site of closure for all experimental conditions. Most data points are located below the identity line. A linear relationship between the variables (observed $P_{close} = -0.56 + 1.0 \times P'_{close}$, $R^2 = 0.966$) was obtained form a linear regression analysis. Accordingly, the P'_{close} is significantly greater than the observed P_{close} approximately by 0.5 cmH_2O on average.

DISCUSSION

Major findings in this study are as follows: *1*) neck extension decreased closing pressures of the velopharynx

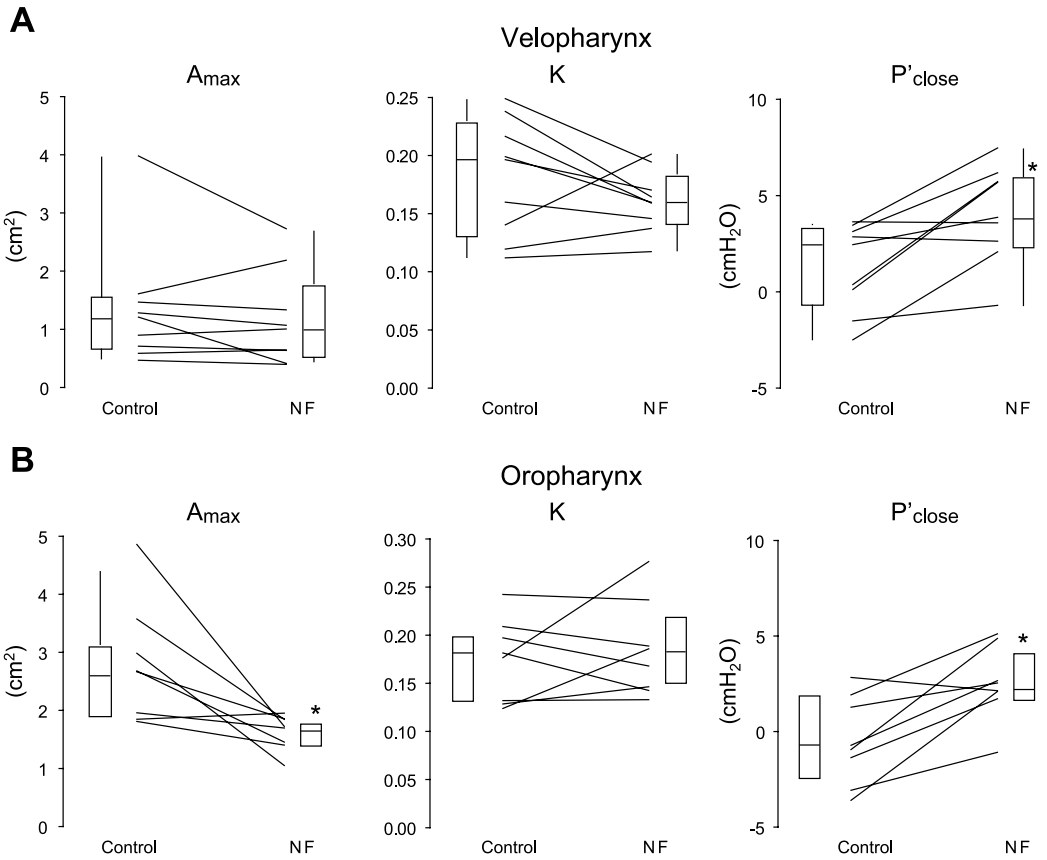

Fig. 5. Changes in static mechanical variables in response to neck flexion (NF) at the velopharynx (*A*) and oropharynx (*B*). Each line represents a different patient. Lower and upper boundaries of the box indicate 25th and 75th percentages. Solid line within the box marks the median, and vertical lines indicate the 90th and 10th percentages. *$P < 0.05$ vs. control.

and oropharynx and increased maximum oropharyngeal airway size, *2*) neck flexion and bite opening increased closing pressures of the velopharynx and oropharynx and decreased maximum oropharyngeal airway size, and *3*) oropharyngeal airway compliance decreased during neck extension. These findings support the concept of the mechanical model presented in Fig. 1 to predict upper airway patency and collapsibility of the passive pharynx.

Design and limitations of the study. Although many previous studies have reported significant influences of neck positions (9, 10, 13, 16, 17, 25) and mouth opening on pharyngeal airway patency (11), this is the first study that purely evaluates regional structural changes of airway collapsibility by mechanical interventions under the elimination of neural mechanisms. Because techniques in obtaining closing pressures, study population, and amount of structural changes by the interventions differ between previous studies and this study, it is inappropriate to compare the amount of changes in closing pressures between the studies. However, it should be noted that the amount of increase in the closing pressure with bite opening in the active pharynx during sleep (3 cmH_2O) (23) is never smaller than that obtained in the passive pharynx (2–2.7 cmH_2O), suggesting no recruitment of the pharyngeal dilator muscles for compensation of the structural pharyngeal narrowing with bite opening during sleep. In contrast, the fact that bite opening did not influence upper airway collapsibility during wakefulness (23) strongly suggests the presence of neural compensatory mechanisms for the structural detrimental effects of bite opening during wakefulness.

Because of the systematic error of cross-sectional area measurement in our experimental setting, as shown in Fig. 3, the pressure-area relationship obtained in this study may systematically deviate from a true relationship. A true A_{max} is considered to be ~10% greater than the measured A_{max}. Because A_{max} error mainly results from deformation of the endoscopic image at the outer area, statistical A_{max} differences before and during mechanical interventions may be valid. In contrast, P'_{close} may be minimally influenced by the measurement error due to the nature of the exponential curve. We consider that the significant discrepancy between observed P_{close} and estimated P'_{close} demonstrated in Fig. 7 is a result of cross-sectional measurement error, but rather of discontinuity of airway pressure changes (1-cmH_2O step changes) during the apneic test.

Mechanical model of the pharyngeal airway. Assuming that neck extension increases the pharyngeal bony enclosure size, the two-dimensional model presented in Fig. 1 predicts

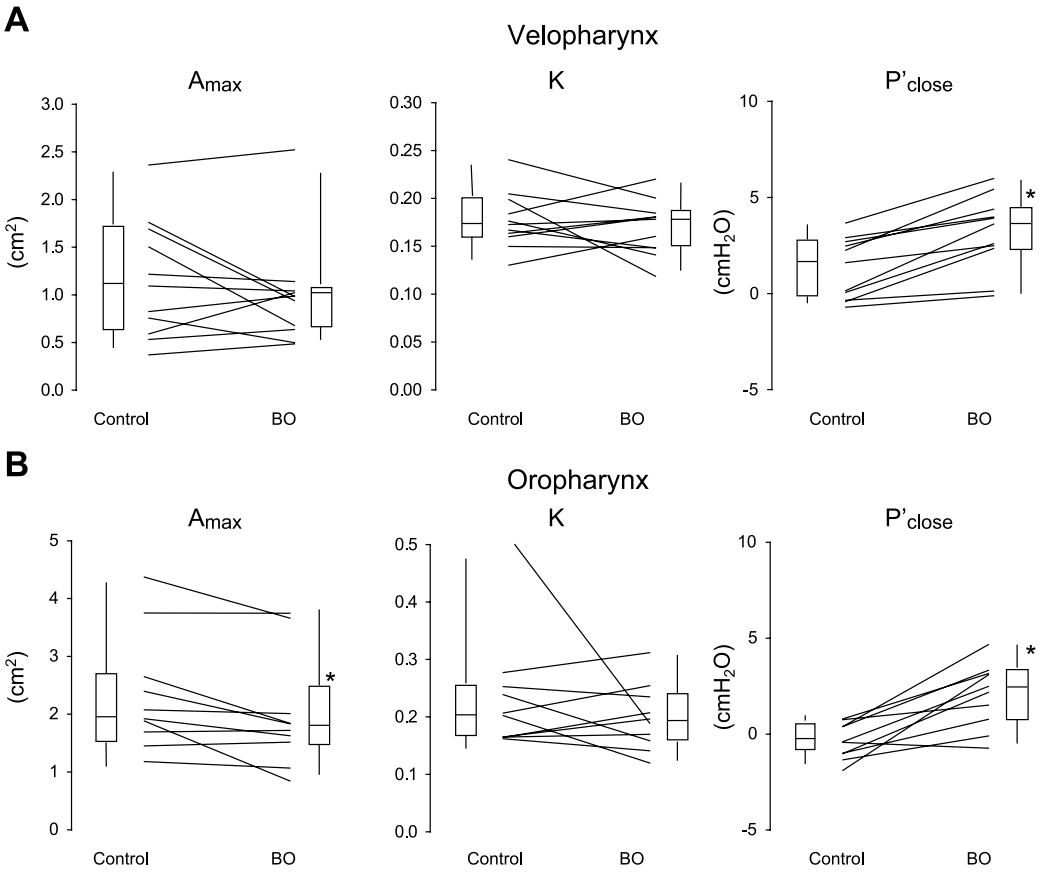

Fig. 6. Changes in static mechanical variables in response to bite opening (BO) at the velopharynx (*A*) and oropharynx (*B*). Each line represents a different patient. Lower and upper boundaries of the box indicate 25th and 75th percentages. Solid line within the box marks the median, and vertical lines indicate the 90th and 10th percentages. *$P < 0.05$ vs. control.

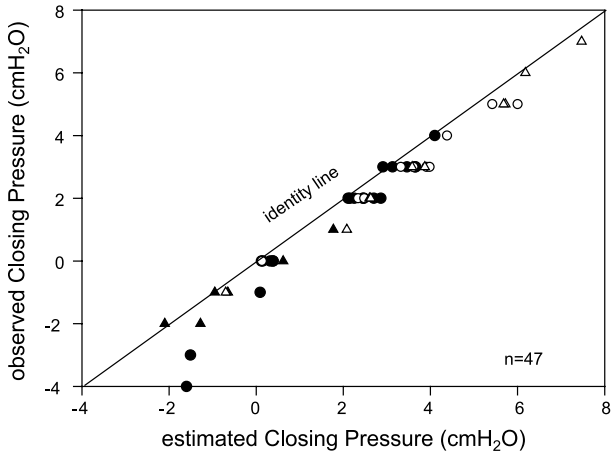

Fig. 7. Correlation between estimated closing pressure and observed closing pressure at a primary site of closure for all experimental conditions. ●, Control condition; ▲, neck extension; △, neck flexion; ○, bite opening.

reduction of pressure surrounding the collapsible tube (P_{tissue}) and increase of the airway size for a given pressure inside the collapsible tube (P_{lumen}). In contrast, neck flexion and bite opening increase P_{tissue} and decrease the airway size, with the assumption that these interventions decrease the pharyngeal bony enclosure size. In fact, the results of this study confirmed these predictions, although this does not indicate that change in the bony enclosure size is the only predominant mechanism for the observed changes in pharyngeal airway collapsibility during the mechanical interventions.

Reduction of pharyngeal airway compliance during neck extension. One notable finding in this study is reduction of the oropharyngeal *K* value, i.e., reduction of the oropharyngeal airway wall compliance, during neck extension. This can be interpreted as flattening of the "tube law" curve of the pharyngeal airway in the two-dimensional mechanical model (Fig. 1). However, the mechanisms causing the tube law change are not clearly presented by the model. In anesthetized dogs, van de Graaff (22) previously demonstrated increase in the tracheal tension decreased upper airway resistance. Recently, Thut et al. (21) found significant changes of critical closing pressure during head positioning in association with changes in upper airway length in cats with isolated upper airways. In anesthe-

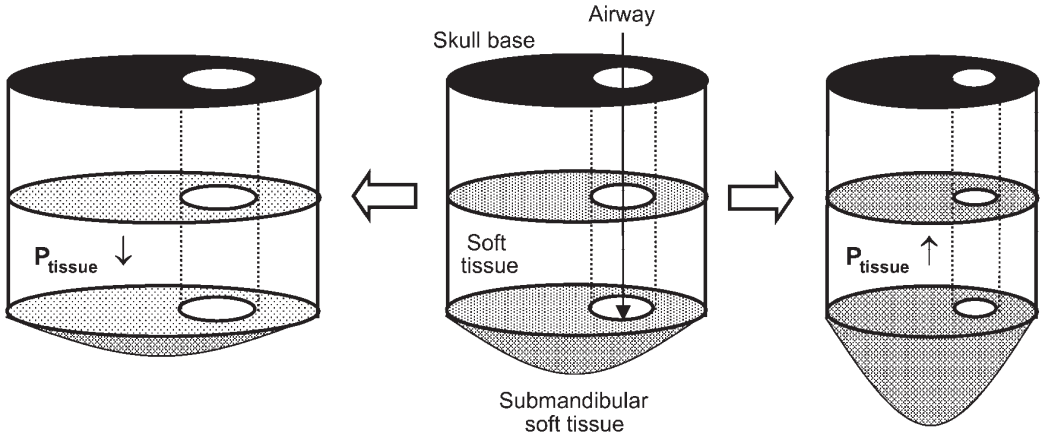

Fig. 8. Advanced 3-dimensional mechanical model of the pharyngeal airway explaining the results of this study. Neck extension (*left*) possibly produces increase in box size and displacement of soft tissue into the box in addition to airway lengthening, resulting in decreasing P_{tissue} and increasing longitudinal force. In contrast, neck flexion and bite opening (*right*) may produce reduction of box size and displacement of the soft tissue out of the box, resulting in increasing P_{tissue}.

tized humans, the distance between the tip of an endotracheal tube and the carina of the trachea increases during neck extension (20), suggesting airway lengthening, and therefore an increase in the longitudinal force during neck extension. Accordingly, significant reduction of the oropharyngeal airway compliance found in this study can result from an increase in longitudinal force during neck extension. A three-dimensional model including airway length and interaction between the soft tissue structures along the airway, which may significantly influence longitudinal force altering airway compliance, may be an advanced mechanical model for the pharyngeal airway and should be tested in the future by measuring P_{tissue} and longitudinal forces (Fig. 8). Furthermore, in a nonparalyzed condition like natural sleep, the role of pharyngeal airway dilator muscles also needs to be included in the model as an alternative mechanism.

Regional differential effects of mechanical interventions. This is the first study that demonstrates that neck extension, neck flexion, and bite opening influence airway collapsibility at both velopharynx and oropharynx. Because tongue musculature originates from and is enclosed by the mandible, changes in the mandibular position by mechanical interventions should result in displacement of the tongue base toward the same direction; therefore, it is not surprising to find significant influences of mechanical interventions on the oropharyngeal airway patency. In contrast, soft tissue at the level of the velopharynx is not enclosed by the mandible and there is no direct structural connection between the mandible and soft palate, which implies less or no influence of the mandibular position changes on velopharyngeal airway patency. Nevertheless, we found significant influences by mechanical interventions on the velopharyngeal patency. Structurally, the dorsum of the tongue is in close apposition with the anterior wall of the soft palate and is enclosed by the maxilla, suggesting mechanical interaction between the tongue and soft palate as we recently reported (6). Changes in the mandibular position may displace the tongue soft tissue at the level of the velopharynx, resulting in changes in the amount of soft tissue enclosed by the maxilla and therefore the P_{tissue} at this segment. Soft tissue interaction along the airway during a variety of mechanical interventions needs to be evaluated in future studies.

Clinical implications. Craniocervical extension with a forward head posture is reported to be common in severe OSA patients during wakefulness (14, 19). Our results suggest that head posture compensates for increased collapsibility of the pharyngeal airway in these patients. Head position during sleep is mainly determined by the height and design of the pillow. Although use of a higher pillow often flexes the neck, which is not advantageous for airway maintenance, placement of a higher pillow with maintenance of the head straight up (sniffing position) produced higher cervical extension and improvement of pharyngeal airway patency in sedated children (18). Recently, Kushida et al. (8) found significant improvement of SDB in mild OSA patients with use of a pillow that promoted neck extension, whereas the improvement was not evident in severe OSA patients. Their finding agrees with our observation that 3 of 11 patients presented P'_{close} above atmospheric pressure even after neck extension, whereas neck extension significantly decreased P'_{close}. Accordingly, neck extension alone may not completely establish patent airway and normalize breathing in severe OSA patients.

Miyamoto et al. (12) reported that the total time spent with mouth opening during sleep was greater in OSA patients than in normal subjects, in accordance with previous observations by Hollowell and Suratt (2). They further reported that bite opening progressively increased during the apneic period and decreased at termination of apnea (12). Our results suggest that bite opening partly contributes to the occurrence and persistence of obstructive apnea; moreover, bite closure partly contributes to reestablishment of the patent airway. Furthermore, bite closure with the use of oral appliances can contribute to efficacy of oral appliances for treatment of SDB, although a recent clinical investigation did not support this (15).

We conclude that head positions and bite opening influence collapsibility of the passive pharynx in patients with SDB. The

observations are well explained by a mechanical model for the pharyngeal airway (a collapsible tube surrounded by soft material within a rigid box).

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Influences of head positions and bite opening on collapsibility of the passive pharynx

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Isono, Shiroh, Atsuko Tanaka, Yugo Tagaito, Teruhiko Ishikawa, and Takashi Nishino. Influences of head positions and bite opening on collapsibility of the passive pharynx. *J Appl Physiol* 97: 339–346, 2004. First published March 12, 2004; 10.1152/japplphysiol.00907.2003.—A collapsible tube surrounded by soft material within a rigid box was proposed as a two-dimensional mechanical model for the pharyngeal airway. This model predicts that changes in the box size (pharyngeal bony enclosure size anatomically defined as cross-sectional area bounded by the inside edge of bony structures such as the mandible, maxilla, and spine, and being perpendicular to the airway) influence patency of the tube. We examined whether changes in the bony enclosure size either with head positioning or bite opening influence collapsibility of the pharyngeal airway. Static mechanical properties of the passive pharynx were evaluated in anesthetized, paralyzed patients with sleep-disordered breathing before and during neck extension with bite closure ($n = 11$), neck flexion with bite closure ($n = 9$), and neutral neck position with bite opening ($n = 11$). Neck extension significantly increased maximum oropharyngeal airway size and decreased closing pressures of the velopharynx and oropharynx. Notably, neck extension significantly decreased compliance of the oropharyngeal airway wall. Neck flexion and bite opening decreased maximum oropharyngeal airway size and increased closing pressure of the velopharynx and oropharynx. Our results indicate the importance of neck and mandibular position for determining patency and collapsibility of the passive pharynx.

obstructive sleep apnea; upper airway; closing pressure; neck positions; mouth opening

A PATENT PHARYNGEAL AIRWAY IS crucial for stable breathing. Pharyngeal airway size is regulated by a precise interaction between neural regulation of pharyngeal airway dilator muscle activities (neural mechanisms) and structural properties of the pharyngeal airway (anatomical mechanisms) (7). Through elimination of the neural mechanisms by administration of a muscle blockade under general anesthesia, we demonstrated that closing pressures of the passive pharynx were distinctively higher in patients with sleep-disordered breathing (SDB) than in normal subjects (3). Furthermore, specific structural abnormalities, such as obesity and craniofacial anomaly, contributed to increased collapsibility of the passive pharynx (24).

Structurally, the pharyngeal airway is surrounded by soft tissue such as the tongue, which is enclosed by bony structures such as the mandible and cervical vertebrae (Fig. 1). Consequently, a collapsible tube surrounded by soft material within a rigid box was proposed as a two-dimensional mechanical model for the pharyngeal airway (24). Pharyngeal bony enclosure size, anatomically defined as cross-sectional area bounded by the inside edge of the bony structures and being perpendicular to the airway, corresponds to the box size of the mechanical model. The contribution of obesity and craniofacial anomaly such as a small maxilla and mandible to the increased collapsibility of the passive pharynx was well explained by the mechanical model (24).

Within one subject, the pharyngeal bony enclosure size varies with head and mandible positioning changes, which may be an influential factor of the pharyngeal airway patency. Cervical extension with bite closure (neck extension) increases the distance between the mentum and cervical column, which consequently increases the bony enclosure size, whereas cervical flexion with bite closure (neck flexion) decreases it. Bite opening with neutral neck position decreases the distance between the mentum and cervical column, which consequently increases the bony enclosure size without head positioning change. Accordingly, the purpose of this study was to evaluate influences of head positions and bite opening on static mechanical properties of the passive pharynx in anesthetized and paralyzed patients with SDB.

MATERIALS AND METHODS

Subjects and overnight oximetry. The study consisted of 24 male patients with SDB who were interested in undergoing uvulopalatopharyngoplasty and were scheduled to undergo endoscopic pharyngeal assessment to determine their indications for this procedure (4). All had histories of excessive daytime sleepiness, habitual snoring, and witnessed repetitive apnea. SDB was evaluated by a pulse oximeter (Pulsox-5; Minolta, Tokyo, Japan). All subjects were instructed to attach an oximetry finger probe before sleep and to remove the probe on awakening. Digital readings of arterial oxygen saturation (Sa_{O_2}) and pulse rate were stored every 5 s in a memory card. The stored data were displayed on a computer screen to check the quality of the recordings. The computer calculated oxygen desaturation index, defined as the number of oxygen desaturation exceeding 4% from the baseline, and the percent of time spent at $Sa_{O_2} < 90\%$. Table 1 lists all nocturnal oximetry data and anthropometric characteristics. Although the oximetry evaluation alone does not clarify the nature of SDB, we believe that all patients can be safely diagnosed as having obstructive sleep apnea (OSA) on the basis of the oximetry results and the clinical symptoms (1).

Informed consent was obtained from all subjects after the aim and potential risks of the study were fully explained to each. The investigation was approved by the hospital ethics committee of our institution.

Preparation of the subjects. Each subject was initially premedicated with 0.5 mg of atropine and placed in the supine position on an operating table, where a modified tight-fitting nasal mask was attached. Care was taken to prevent air leaks from the mask, particularly

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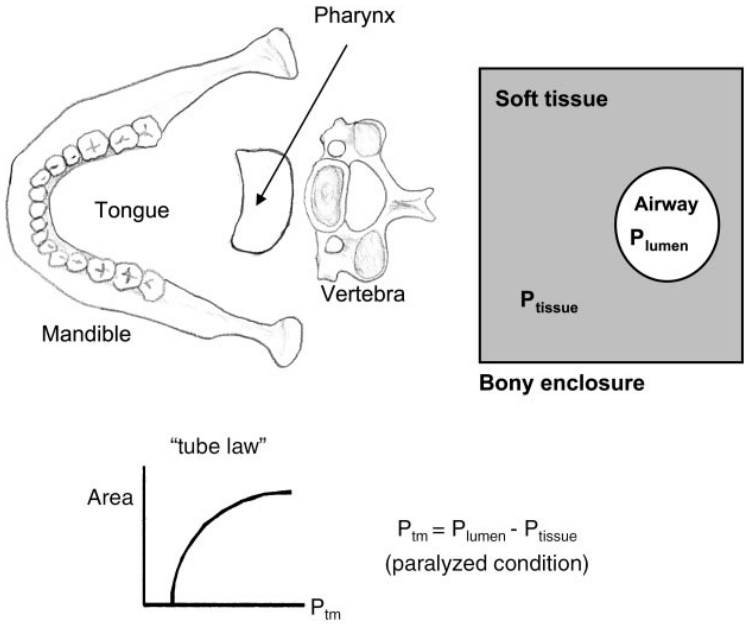

Fig. 1. Anatomic arrangements of the pharynx, tongue, mandible, and cervical vertebra (*left*) and a mechanical model for the structures (*right*). P_{lumen}, pressure inside the collapsible tube; P_{tissue}, pressure surrounding the collapsible tube; Ptm, transmural pressure. In the mechanical model, the luminal size of the collapsible tube is determined by mechanical properties of the tube (tube law) and Ptm. Ptm is defined as the pressure difference between pressures inside (P_{lumen}) and outside the tube (P_{tissue}). For a given P_{lumen}, an increase in P_{tissue} results in reduction of Ptm and therefore narrows the lumen. P_{tissue} is determined by the balance between the amount of soft material inside the rigid box and the size of the surrounding rigid box.

when the airway was pressurized above 20 cmH_2O. General anesthesia was induced and maintained by intravenous infusion of propofol, and intravenous injection of a muscle relaxant (vecuronium 0.2 mg/kg) produced complete paralysis throughout the experiment while the subject was ventilated with positive pressure through an anesthetic machine. Sa_{O_2}, electrocardiogram, and blood pressure were continuously monitored. The tip of a slim endoscope (FB10X, Pentax, Tokyo, Japan, 3 mm OD) was inserted through the modified nasal mask and the naris down to the upper airway to visualize the velopharynx (retropalatal airway), and the oropharynx (retroglossal airway). A closed-circuit camera (ETV8, Nisco, Saitama, Japan) was connected to the endoscope, and the pharyngeal images were recorded on a videotape. Reading of airway pressure (Paw), measured by a water manometer, was simultaneously recorded on videotape.

Experimental procedures. To determine the pressure-area relationship of the pharynx, after disconnection from the anesthetic machine the nasal mask was connected to a pressure-control system capable of accurately manipulating Paw from −20 to 20 cmH_2O in a stepwise fashion. At cessation of mechanical ventilation of the subject under complete muscle paralysis, apnea resulted. Paw was immediately increased up to 20 cmH_2O to dilate the airway and then gradually reduced, within a 2- to 3-min span, from 20 cmH_2O to the closing pressure of the retropalatal airway in a stepwise fashion. The latter represented the pressure at which complete closure of the retropalatal airway occurred, as evident on the video screen. The apneic test was terminated when Sa_{O_2} fell below 95%. This procedure of experimentally induced apnea allowed construction of a pressure-area relationship of the visualized pharyngeal segment. The subject was manually ventilated for at least 1 min before and after the apneic test. Distance between the tip of the endoscope and the narrowing site was measured with a wire passed through the aspiration channel of the endoscope.

Table 1. *Anthropometric characteristics and results of nocturnal oximetry*

Age, yr	47.0 (38.0–54.5)
Weight, kg	77.7 (69.0–82.8)
Height, m	1.72 (1.66–1.77)
BMI, kg/m^2	26.4 (23.7–28.4)
ODI, h^{-1}	27.7 (20.8–49.5)
CT_{90}, %	12.0 (2.0–23.1)
Nadir Sa_{O_2}, %	87.9 (84.9–90.3)
Lowest Sa_{O_2}, %	72.0 (61.0–80.5)

Values are medians (25–75 percentiles) of all patients (n = 24). BMI, body mass index; ODI, oxygen desaturation (Sa_{O_2}) index defined as number of desaturations exceeding >4% per hour; CT_{90}, percent of time spent Sa_{O_2} <90%; Nadir Sa_{O_2}, mean of the nadir Sa_{O_2} values in all desaturation events; Lowest Sa_{O_2}, a lowest Sa_{O_2} value among the desaturation events.

Each patient participated in either the head position study (n = 13) or the bite opening study (n = 11) (Fig. 2). In the head position study, in addition to the control measurement (head and neck in neutral position with bite closed by a chin strap), the apneic tests were repeated during both neck extension (neck maximally extended by placing cushions under the shoulders with bite closed by a chin strap) and neck flexion (neck maximally flexed by placing cushions under the head with bite closed by a chin strap) in seven patients, whereas the tests were performed either during only neck extension (n = 4) or neck flexion (n = 2) in the remaining six patients. The control measurement was performed once per patient. In the bite opening study, the bite was widened by inserting a mouthpiece between the upper and lower incisors in head and neck in the neutral position, producing a 15-mm distance between the incisors (Fig. 2). The apneic test was initiated immediately after establishment of each experimental condition. On the completion of the experiment time span of 30–60 min, atropine (0.02 mg/kg) and neostigmine (0.04 mg/kg) were administered to reverse muscle paralysis.

Data analysis. To convert the monitor image to an absolute value of the pharyngeal cross-sectional area, magnification of the imaging system was estimated at 1.0-mm interval distances between the endoscopic tip and the object in the range of 5–30 mm. At a defined value of Paw, the image of the pharyngeal lumen was traced and pixels included in the area were counted (SigmaScan version 2.0, Jandel Scientific Software, San Rafael, CA). The pixel number was converted to pharyngeal cross-sectional area according to the distance-magnification relationship. Using known-diameter tubes, we tested the accuracy of the cross-sectional area measurements. For constant distance, the measured areas were systematically deviated from actual areas (Fig. 3); the largest known area tested

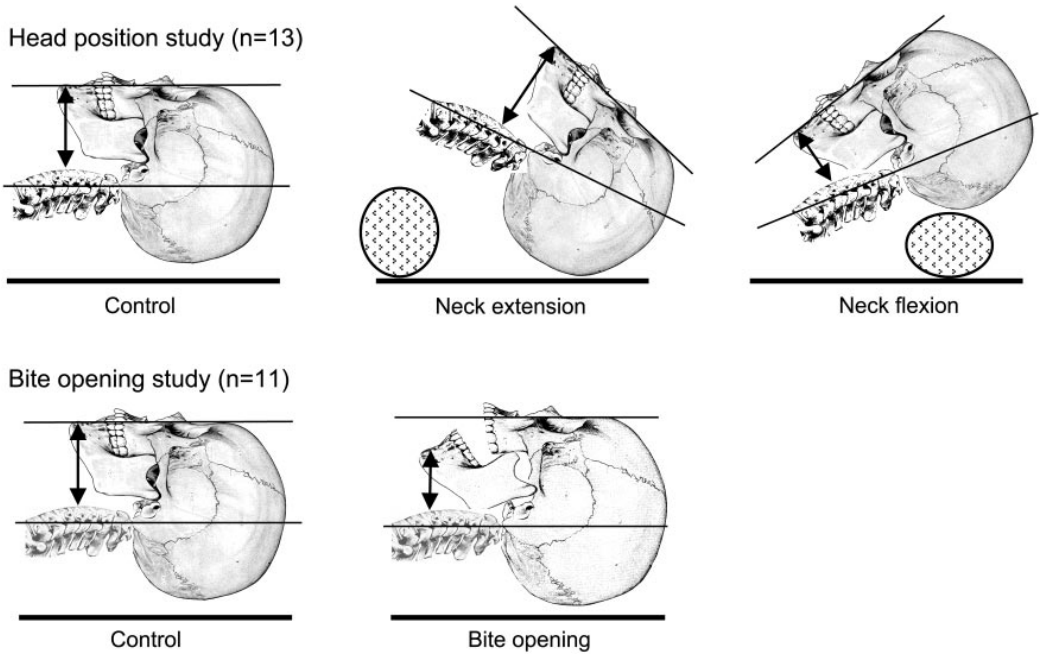

Fig. 2. Illustrative configuration of head, neck, and mandible positions for each experimental condition. Note that differences of the distance between the mentum and cervical column resulted from the configuration differences. A horizontal straight line for each condition represents the experimental table, and cushions were placed under the shoulders for the neck extension and placed under the head for the neck flexion.

(0.95 cm^2) was underestimated by 11% because of image deformation of the outer image area, and the smallest known area tested (0.03 cm^2) was overestimated by 13% because of reduction of the image resolution (5).

The measured luminal cross-sectional area was plotted as a function of Paw. The closing pressure was defined as pressure corresponding to the zero area. At high values of Paw, relatively constant cross-sectional areas were revealed; therefore, maximum area (A_{max}) was determined as the mean value of highest three Paw (18, 19, and 20 cmH_2O). The pressure-area relationship of each pharyngeal segment was fitted by an exponential function, $A = A_{max} - B \times \exp(-K \times \text{Paw})$, where B and K are constants. A nonlinear least square technique was used for the curve fitting, and the quality of the fitting was provided by the coefficient R^2 (SigmaPlot version 2.0, Jandel Scientific Software, San Rafael, CA). A regressional estimate of closing pressure (P'_{close}), which corresponds to an intercept of the curve on the Paw axis, was calculated from the following equation for each pharyngeal segment: $P'_{close} = \ln(B/A_{max})K^{-1}$. The shape of the pressure-area relationship was described by the value of K. When pressure-area relationship is curvilinear, compliance of the pharynx defined as a slope of the curve varies with changes in Paw; therefore, a single value of compliance calculated for a given Paw does not represent collapsibility of the pharynx for the entire Paw ranges. In contrast, K represents the rate of changes in the slope of the curve; therefore, when K is high, a small reduction in Paw results in a significant increase in compliance, leading to remarkable reduction in cross-sectional area. Consequently, collapsibility of the pharynx increases with increasing K. We suggest that both P'_{close} and K values represent collapsibility of the pharynx, whereby the former determines the position of the exponential curve and the latter characterizes the shape of the curve.

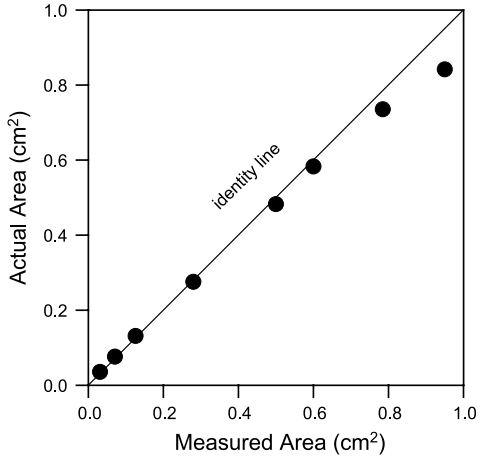

Fig. 3. Accuracy of our area measurement for a constant distance. The measured area was systematically deviated from actual area.

Statistical analysis. All values are expressed by median (25–75 percentiles). The Wilcoxon's signed-rank test was used for comparison between the control and other conditions. Linear regression analysis was performed between observed and estimated closing pressures. $P < 0.05$ was considered to be significant.

RESULTS

Effects of neck extension on static pharyngeal mechanics. Changes in static mechanical variables before and during neck extension are presented in Fig. 4. Neck extension approximately doubled A_{max} and significantly decreased both K and P'_{close} at the oropharynx. In addition to the significant influ-

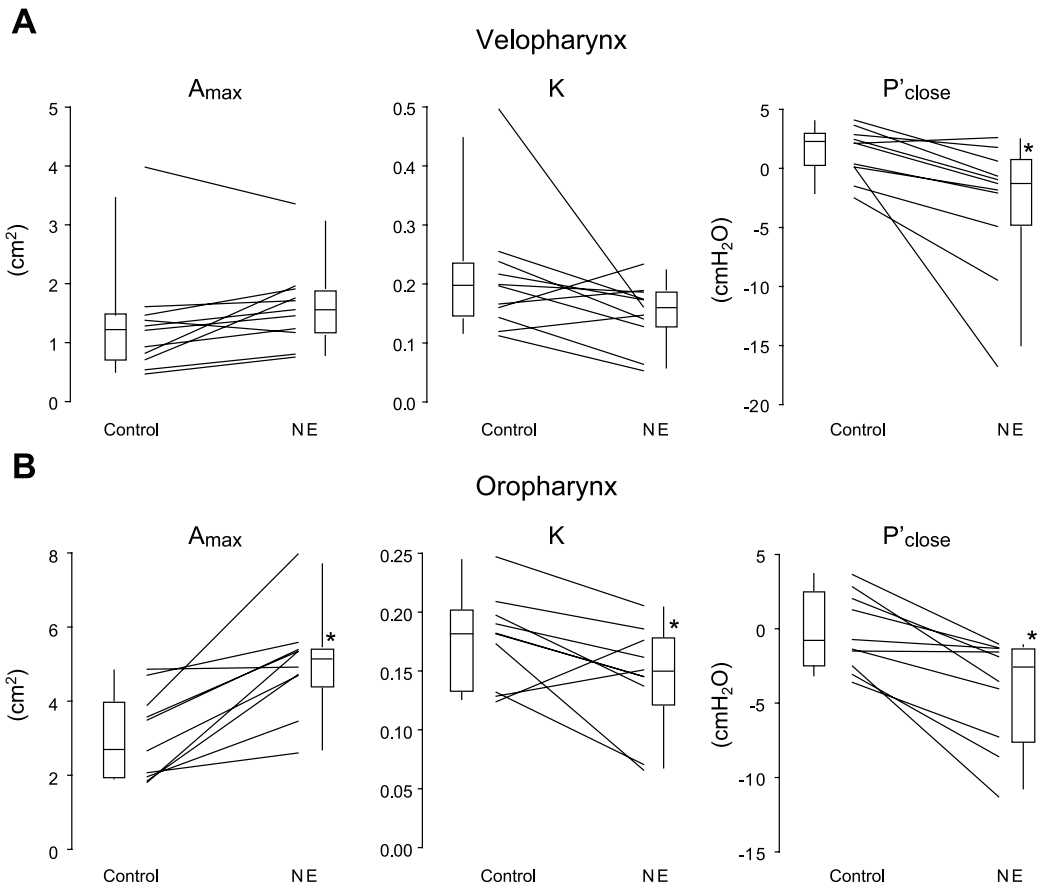

Fig. 4. Changes in static mechanical variables in response to neck extension (NE) at the velopharynx (*A*) and oropharynx (*B*). Each line represents a different patient. A_{max}, maximum cross-sectional area; *K*, a constant obtained by an exponential fitting the pressure-area relationship and representing stiffness of the pharyngeal airway wall; P'_{close}, estimated closing pressure calculated from the fitted exponential function. Lower and upper boundaries of the box indicate 25th and 75th percentages. Solid line within the box marks the median, and vertical lines indicate the 90th and 10th percentages. *$P < 0.05$ vs. control.

ences on the oropharyngeal segment, P'_{close} at the velopharynx also significantly decreased during neck extension. Notably, there were tendencies of increase in A_{max} ($P = 0.054$) and decrease in K ($P = 0.067$) at the velopharynx, as shown in Fig. 4, although these are not statistically significant. The results indicate that neck extension dilates and stiffens the velopharyngeal and oropharyngeal airway, improving pharyngeal airway patency.

Effects of neck flexion on static pharyngeal mechanics. Changes in static mechanical variables before and during neck flexion are presented in the Fig. 5. Neck flexion significantly decreased A_{max} and increased P'_{close} at the oropharynx. Velopharyngeal P'_{close} also increased during neck flexion. Neck flexion significantly reduced oropharyngeal airway size and increased airway collapsibility at both the velopharynx and oropharynx. K did not change in response to neck flexion.

Effects of bite opening on static pharyngeal mechanics. Changes in static mechanical variables before and during bite opening are presented in the Fig. 6. Bite opening significantly decreased A_{max} and increased P'_{close} at the oropharynx. Velopharyngeal P'_{close} also increased during bite opening. K did not change in response to bite opening. Notably, the pattern of changes in the pharyngeal mechanics during bite opening is similar to that during neck flexion.

Comparison between observed and estimated closing pressures. Figure 7 demonstrates the correlation between P'_{close} and observed P_{close} (the highest airway pressure at which complete closure of either the retropalatal or retroglossal airway was seen on the video screen) at the primary site of closure for all experimental conditions. Most data points are located below the identity line. A linear relationship between the variables (observed $P_{close} = -0.56 + 1.0 \times P'_{close}$, $R^2 = 0.966$) was obtained form a linear regression analysis. Accordingly, the P'_{close} is significantly greater than the observed P_{close} approximately by 0.5 cmH_2O on average.

DISCUSSION

Major findings in this study are as follows: *1*) neck extension decreased closing pressures of the velopharynx

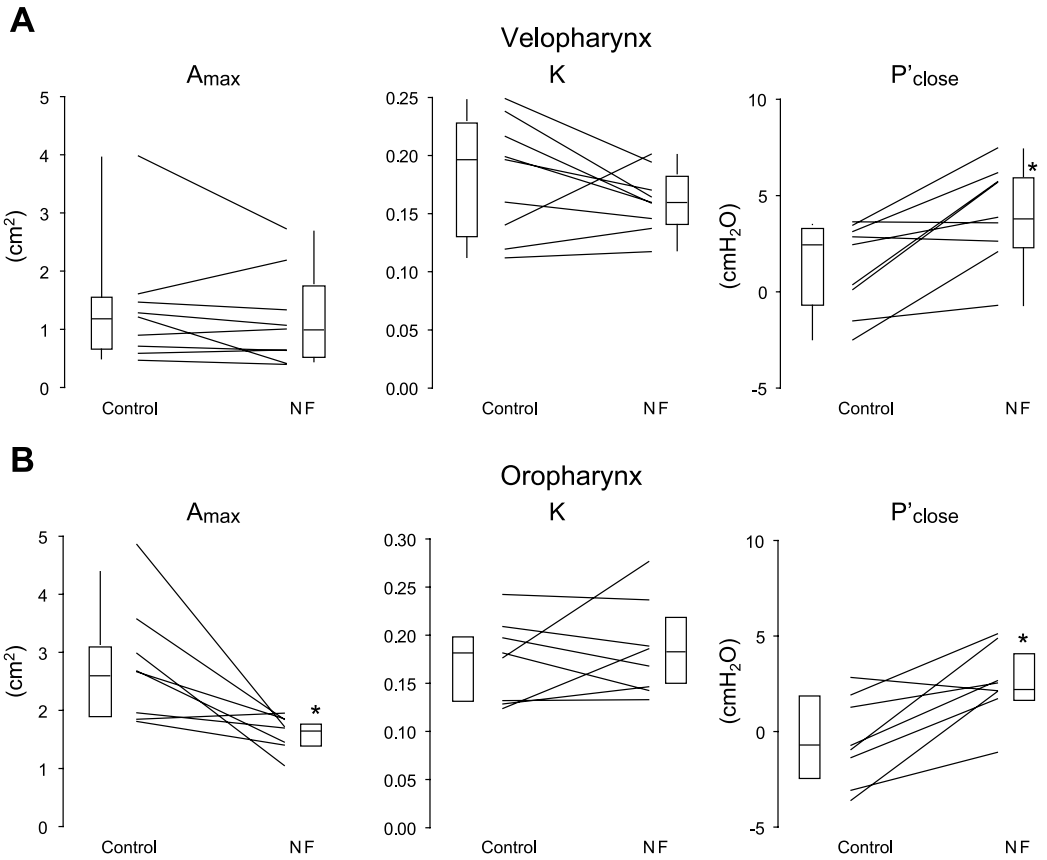

Fig. 5. Changes in static mechanical variables in response to neck flexion (NF) at the velopharynx (*A*) and oropharynx (*B*). Each line represents a different patient. Lower and upper boundaries of the box indicate 25th and 75th percentages. Solid line within the box marks the median, and vertical lines indicate the 90th and 10th percentages. *$P < 0.05$ vs. control.

and oropharynx and increased maximum oropharyngeal airway size, *2*) neck flexion and bite opening increased closing pressures of the velopharynx and oropharynx and decreased maximum oropharyngeal airway size, and *3*) oropharyngeal airway compliance decreased during neck extension. These findings support the concept of the mechanical model presented in Fig. 1 to predict upper airway patency and collapsibility of the passive pharynx.

Design and limitations of the study. Although many previous studies have reported significant influences of neck positions (9, 10, 13, 16, 17, 25) and mouth opening on pharyngeal airway patency (11), this is the first study that purely evaluates regional structural changes of airway collapsibility by mechanical interventions under the elimination of neural mechanisms. Because techniques in obtaining closing pressures, study population, and amount of structural changes by the interventions differ between previous studies and this study, it is inappropriate to compare the amount of changes in closing pressures between the studies. However, it should be noted that the amount of increase in the closing pressure with bite opening in the active pharynx during sleep (3 cmH_2O) (23) is never smaller than that obtained in the passive pharynx (2–2.7 cmH_2O), suggesting no recruitment of the pharyngeal dilator muscles for compensation of the structural pharyngeal narrowing with bite opening during sleep. In contrast, the fact that bite opening did not influence upper airway collapsibility during wakefulness (23) strongly suggests the presence of neural compensatory mechanisms for the structural detrimental effects of bite opening during wakefulness.

Because of the systematic error of cross-sectional area measurement in our experimental setting, as shown in Fig. 3, the pressure-area relationship obtained in this study may systematically deviate from a true relationship. A true A_{max} is considered to be ~10% greater than the measured A_{max}. Because A_{max} error mainly results from deformation of the endoscopic image at the outer area, statistical A_{max} differences before and during mechanical interventions may be valid. In contrast, P'_{close} may be minimally influenced by the measurement error due to the nature of the exponential curve. We consider that the significant discrepancy between observed P_{close} and estimated P'_{close} demonstrated in Fig. 7 is a result of cross-sectional measurement error, but rather of discontinuity of airway pressure changes (1-cmH_2O step changes) during the apneic test.

Mechanical model of the pharyngeal airway. Assuming that neck extension increases the pharyngeal bony enclosure size, the two-dimensional model presented in Fig. 1 predicts

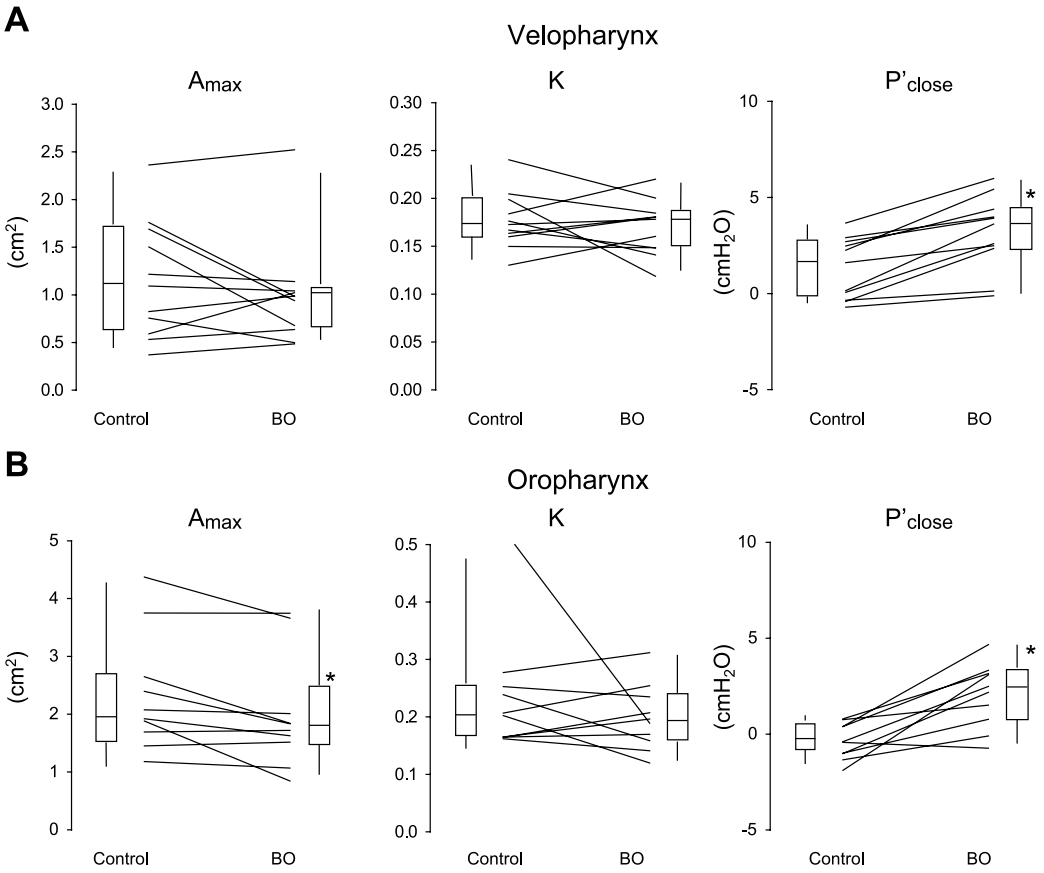

Fig. 6. Changes in static mechanical variables in response to bite opening (BO) at the velopharynx (*A*) and oropharynx (*B*). Each line represents a different patient. Lower and upper boundaries of the box indicate 25th and 75th percentages. Solid line within the box marks the median, and vertical lines indicate the 90th and 10th percentages. *$P < 0.05$ vs. control.

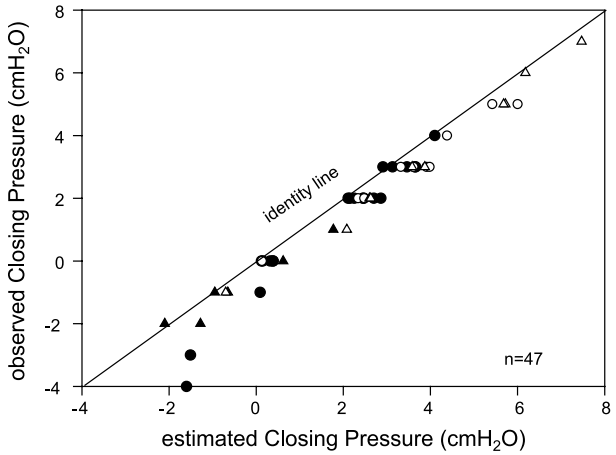

Fig. 7. Correlation between estimated closing pressure and observed closing pressure at a primary site of closure for all experimental conditions. ●, Control condition; ▲, neck extension; △, neck flexion; ○, bite opening.

reduction of pressure surrounding the collapsible tube (P_{tissue}) and increase of the airway size for a given pressure inside the collapsible tube (P_{lumen}). In contrast, neck flexion and bite opening increase P_{tissue} and decrease the airway size, with the assumption that these interventions decrease the pharyngeal bony enclosure size. In fact, the results of this study confirmed these predictions, although this does not indicate that change in the bony enclosure size is the only predominant mechanism for the observed changes in pharyngeal airway collapsibility during the mechanical interventions.

Reduction of pharyngeal airway compliance during neck extension. One notable finding in this study is reduction of the oropharyngeal *K* value, i.e., reduction of the oropharyngeal airway wall compliance, during neck extension. This can be interpreted as flattening of the "tube law" curve of the pharyngeal airway in the two-dimensional mechanical model (Fig. 1). However, the mechanisms causing the tube law change are not clearly presented by the model. In anesthetized dogs, van de Graaff (22) previously demonstrated increase in the tracheal tension decreased upper airway resistance. Recently, Thut et al. (21) found significant changes of critical closing pressure during head positioning in association with changes in upper airway length in cats with isolated upper airways. In anesthe-

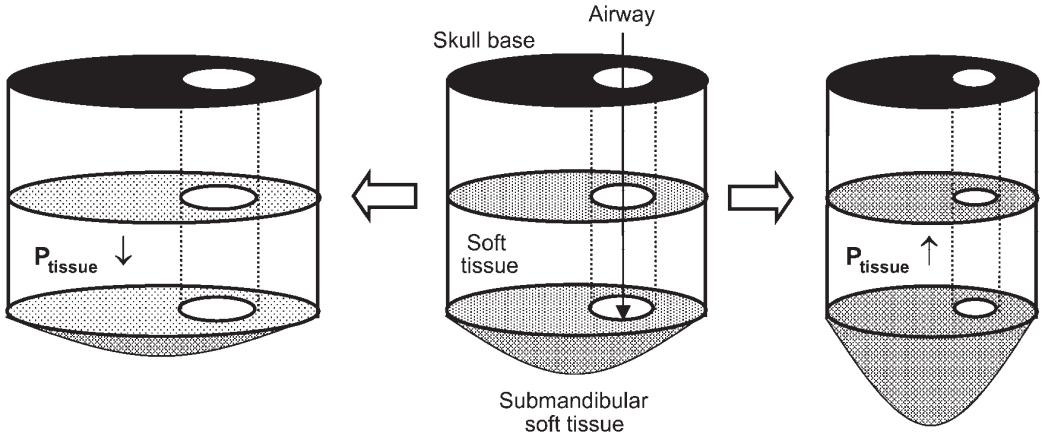

Fig. 8. Advanced 3-dimensional mechanical model of the pharyngeal airway explaining the results of this study. Neck extension (*left*) possibly produces increase in box size and displacement of soft tissue into the box in addition to airway lengthening, resulting in decreasing P_{tissue} and increasing longitudinal force. In contrast, neck flexion and bite opening (*right*) may produce reduction of box size and displacement of the soft tissue out of the box, resulting in increasing P_{tissue}.

tized humans, the distance between the tip of an endotracheal tube and the carina of the trachea increases during neck extension (20), suggesting airway lengthening, and therefore an increase in the longitudinal force during neck extension. Accordingly, significant reduction of the oropharyngeal airway compliance found in this study can result from an increase in longitudinal force during neck extension. A three-dimensional model including airway length and interaction between the soft tissue structures along the airway, which may significantly influence longitudinal force altering airway compliance, may be an advanced mechanical model for the pharyngeal airway and should be tested in the future by measuring P_{tissue} and longitudinal forces (Fig. 8). Furthermore, in a nonparalyzed condition like natural sleep, the role of pharyngeal airway dilator muscles also needs to be included in the model as an alternative mechanism.

Regional differential effects of mechanical interventions. This is the first study that demonstrates that neck extension, neck flexion, and bite opening influence airway collapsibility at both velopharynx and oropharynx. Because tongue musculature originates from and is enclosed by the mandible, changes in the mandibular position by mechanical interventions should result in displacement of the tongue base toward the same direction; therefore, it is not surprising to find significant influences of mechanical interventions on the oropharyngeal airway patency. In contrast, soft tissue at the level of the velopharynx is not enclosed by the mandible and there is no direct structural connection between the mandible and soft palate, which implies less or no influence of the mandibular position changes on velopharyngeal airway patency. Nevertheless, we found significant influences by mechanical interventions on the velopharyngeal patency. Structurally, the dorsum of the tongue is in close apposition with the anterior wall of the soft palate and is enclosed by the maxilla, suggesting mechanical interaction between the tongue and soft palate as we recently reported (6). Changes in the mandibular position may displace the tongue soft tissue at the level of the velopharynx, resulting in changes in the amount of soft tissue enclosed by the maxilla and therefore the P_{tissue} at this segment. Soft tissue interaction along the airway during a variety of mechanical interventions needs to be evaluated in future studies.

Clinical implications. Craniocervical extension with a forward head posture is reported to be common in severe OSA patients during wakefulness (14, 19). Our results suggest that head posture compensates for increased collapsibility of the pharyngeal airway in these patients. Head position during sleep is mainly determined by the height and design of the pillow. Although use of a higher pillow often flexes the neck, which is not advantageous for airway maintenance, placement of a higher pillow with maintenance of the head straight up (sniffing position) produced higher cervical extension and improvement of pharyngeal airway patency in sedated children (18). Recently, Kushida et al. (8) found significant improvement of SDB in mild OSA patients with use of a pillow that promoted neck extension, whereas the improvement was not evident in severe OSA patients. Their finding agrees with our observation that 3 of 11 patients presented P'_{close} above atmospheric pressure even after neck extension, whereas neck extension significantly decreased P'_{close}. Accordingly, neck extension alone may not completely establish patent airway and normalize breathing in severe OSA patients.

Miyamoto et al. (12) reported that the total time spent with mouth opening during sleep was greater in OSA patients than in normal subjects, in accordance with previous observations by Hollowell and Suratt (2). They further reported that bite opening progressively increased during the apneic period and decreased at termination of apnea (12). Our results suggest that bite opening partly contributes to the occurrence and persistence of obstructive apnea; moreover, bite closure partly contributes to reestablishment of the patent airway. Furthermore, bite closure with the use of oral appliances can contribute to efficacy of oral appliances for treatment of SDB, although a recent clinical investigation did not support this (15).

We conclude that head positions and bite opening influence collapsibility of the passive pharynx in patients with SDB. The

observations are well explained by a mechanical model for the pharyngeal airway (a collapsible tube surrounded by soft material within a rigid box).

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