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Factors that affect the magnitude and the direction of incisal bite forces: A possible role for pulpal mechanoreceptors

BY

Jarin Paphangkorakit ©

A thesis submitted to the Faculty of Graduate Studies and Research in partial fulfilment of the requirements for the degree of Doctor of Philosophy

IN

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To my parents, for their understanding.

To Wan, for her love and support.

ABSTRACT

It is generally accepted that pain is the only sensation aroused when pulpal nerves are stimulated. The stimuli used to evoke the sensation have always been artificial such as drilling a tooth, blowing air on exposed dentine or applying an electrical shock. Pain has never been evoked in response to the type of stimulus that would normally be experienced. It is very difficult to understand the functional advantage of encasing sensory nerves inside a tooth with the only function of registering pain in response to abnormal stimuli. It is postulated here that dentine and its contained nerves act as a "stress sensor." A transducer was designed that, when clenched between upper and lower teeth, could be used to measure the magnitude and direction of a bite force. The role of the putative mechanoreceptors was tested by varying the stress on incisors while measuring the maximum force subjects could develop when clenching on them. The force was increased as the stress was decreased. No nerves other than those in the pulp could have been used to detect the stress. In a situation which precluded the use of any other receptors than those in the incisor pulp it was found that subjects could discriminate between the hardness of materials clenched between upper and lower incisors. Finally when loads within the normal physiological range were applied to extracted human teeth, an amount of fluid was displaced sufficient to stimulate pulpal nerves. It is suggested that pulp may contain mechanoreceptors. The receptors may be *subconsciously* used to protect teeth from potentially harmful bite forces and probably help establish a precise cuspal interdigitation. They can also be *consciously* used to discriminate between the hardness of harder food. Forces applied to a tooth deform dentine, displace dentinal fluid into the pulp and subsequently activate mechanoreceptors inside the tooth either in dentine, predentine or pulp proper.

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Jarin Paphangkorakit

TABLE OF CONTENTS

CHAPTER 1

CHAPTER 2

CHAPTER 3

CHAPTER 4

CHAPTER 5

CHAPTER 6

CHAPTER 7

APPENDICES

LIST OF TABLES

LIST OF FIGURES

CHAPTER 1

INTRODUCTION

RATIONALE

Pulpal receptors have been widely considered as nociceptors and only give rise to pain. Most studies have been attempting to correlate their morphology, distribution and physiology with dentine sensitivity. Brännström (1963) proposed the "hydrodynamic mechanism of dentine sensitivity" in which fluid movement in the dentinal tubules is thought to account for observed pulpal responses. The theory helps to explain how most, although not all, of the stimuli applied to the dentine surface (for example, air blasting, hyperosmotic solution, tooth drilling, heat and cold application) evoke a pulpal response, specifically "pain."

It should be noted that air blasting, hyperosmotic solution, heat and cold are unlikely to have imposed any selection pressure on the evolution of dentitions, especially in mammals. Animals virtually never experience such stimuli and even if they did, receptors in the lips and tongue would more effectively monitor any potentially noxious stimulus. Yet the dentinal tubules and pulp remain throughout evolution. It makes little sense to have such a highly innervated tissue populated by nerves that are rarely or never activated and have the sole purpose of causing pain.

Some may argue that pulpal tissue is important for depositing secondary dentine. My opinion would be that it is more efficient to build a solid tooth that would presumably be more durable. It can be further argued that pulpal tissue also provides moisture for the tooth substance and strengthens the enamel-dentine complex. However, that is readily achieved by surrounding teeth with saliva and tissue fluid.

Osborn (1976) postulated that the tubular structure in enamel-dentine complex is a unique design of a force sensor and pulpal tissue may be necessary for analyzing bite forces. Dentine fluid is presumably displaced due to deformation of the enamel-dentine complex under occlusal loading. The deformation is presumably greater near the loaded area and may be sensed by pulpal receptors in the vicinity. This would enable the animal to detect forces imposed on individual cusps. Periodontal mechanoreceptors, unlike pulpal receptors, only detect the total amount of force or torque applied to a tooth. In addition, it is likely that discharge frequency of periodontal receptors reaches its maximum value when the tooth is compressed possibly by loads greater than 10 N. Larger bite forces may be detected by other sensory mechanisms.

The purpose of this thesis is to test whether the enamel-dentine complex together with the dental pulp can be used as a force, or more accurately a stress, sensor.

SPECIFIC AIMS

Five experiments have been designed to study whether tooth pulp may contain mechanoreceptors. A method was devised to exclude the use of information from periodontal receptors. Specifically, three hypotheses have been tested:

(1) The maximum bite force on an incisor is decreased if the stress (force per unit area) on the crown is increased.

(2) Stress on the crowns of incisor teeth can be used to detect the hardness of a hard object clenched between them.

(3) Forces within the range of possible bite forces deform the crowns of human teeth so that sufficient fluid is displaced to activate pulpal nerves.

BITE FORCE STUDIES

The study of human bite forces is necessary to understand the behavior of masticatory muscles in healthy and diseased conditions, and the effects of the facial skeleton and dental prostheses on the masticatory apparatus. It is the only way of investigating factors which control the production of bite forces.

Static bite forces can be studied in two ways. One is to investigate factors which affect the maximum bite force (MBF). The other is to investigate muscle activity at a particular submaximum bite force in different conditions.

The MBF varies considerably among subjects. At the incisal region the MBF is smaller than that in the premolar and molar regions. The incisal MBF ranges between 87-293 N (Ringqvist, 1973*a*; Hannam, 1976; Helkimo *et al.*, 1977; Helkimo and Ingervall, 1978) whereas the premolar MBF ranges between 350-650 N (Linderholm and Wennström, 1970; Widmalm and Ericsson, 1982; Dahlström *et al.*, 1988) and the molar MBF ranges between 209-680 N (Linderholm and Wennström, 1970; Floystrand *et al.*, 1982; Helle *et al.*, 1983). The variation is probably due to differences in jaw opening, types of measuring device, and the population being studied. All of these studies used uni-directional force transducers. The incisal MBF measured with a three-dimensional force transducer in our laboratory (117-368 N) is larger than that measured in previous studies. Similarly, van Eijden (1991) also measured a larger maximum molar bite force (724 ± 86 N) with a three-dimensional force transducer. The lower values in previous measurements are probably due to the inability to measure the components of the force at right angles to the axis of a uni-directional force transducer.

Many factors can contribute to the variation in bite force measurements. For example, the MBF increases with age in children (Helle *et al.*, 1983). However, as adults age the bite force decreases (Helkimo *et al.*, 1977; Bakke *et al.*, 1990). Males tend to have a higher MBF than females (Helkimo *et al*, 1977; Bakke *et al.*, 1990). The MBF, nevertheless, may nor may not be correlated with general body build (Linderholm and Wennström, 1970; Ringqvist, 1973*a*; Helkimo and Ingervall, 1978). Different populations may have different capabilities to produce a bite force. Inuits, for example, have much higher MBFs (up to 1500 N) than most other Western races (Waugh, 1937).

After some period of chewing exercises, the MBF can be increased but returns to normal after discontinuing the exercise (Brekhus *et al.*, 1941; Ingervall and Bitsanis, 1987). This may be due to either a temporary switch from type I to type II muscle fibres in jaw closing muscles (Edström and Grimby, 1986) or perhaps, to an increased threshold of periodontal nociceptors.

Facial morphology also affects the magnitude of bite forces. Long-faced subjects have a smaller MBF than normal ones (Proffit and Fields, 1983; Kiliaridis *et al.*, 1993). A small gonial angle (the angle between the posterior border of the ramus and the lower border of the mandible) has been shown to be an indicator of a strong bite force (Ingervall and Helkimo, 1978). This may be related to physiological or biomechanical differences of jaw closing muscles which may alter both the magnitude and direction of a bite force. It is not entirely clear whether strong jaw muscles cause a short facial skeleton or whether a short facial skeleton improves the biomechanics of bite force production, or both.

None of the above factors has been studied using a three-dimensional transducer in order to investigate their effect on bite directions.

PRODUCTION OF A BITE FORCE

A bite force is produced by contraction of jaw closing muscles (masseter, medial pterygoid, temporalis and lateral pterygoid muscles). It was believed that the lateral pterygoid muscle is involved solely in jaw opening. However, evidence has shown that the superior head of the muscle is also active during bite force production (McNamara, 1973). Osborn and Baragar (1985) and Osborn (1996) suggested that the superior head of the lateral pterygoid together with posterior temporalis muscles coactivate to stabilize the condyle against the almost frictionless articular eminence. This becomes very important during a forceful bite.

A bite force must be expressed both in magnitude and direction. In the past, the importance of bite force direction has been overlooked. Not until recently has the magnitude of a maximum bite force been shown to depend on its direction (van Eijden, 1991). The direction seems to depend on the nature of the contact between food and occlusal surface and on the positions of the upper and lower teeth. The differences in the orientation of the jaw muscles enable the production of bite forces in many directions.

The term "bite force" is preferably used for describing a static task, as opposed to "chewing force" which describes a dynamic task. Chewing forces could be visualized as a series of constantly changing bite forces. The direction of chewing forces presumably differs between species due to differences in jaw movement. For example, carnivores probably use vertical forces whereas ungulates use more horizontal forces.

A bite force is the resultant of forces produced by jaw closing muscles minus the temporomandibular joint (TMJ) reaction forces. The calculation is complicated by the fact that each muscle produces a force in a different direction. A three-dimensional vector summation is therefore necessary. Thus the orientation, the size of masticatory muscles and the shape of the articulating surfaces affect the production of a bite force. By considering the jaws as a rigid structure, both two- and three-dimensional mathematical models have been consistently used to study bite force mechanics (Hylander, 1975; Osborn and Baragar, 1985; Faulkner *et al.*, 1987; Koolstra *et al.*, 1988). Recently linear programming has been introduced to predict the most efficient way to exert a particular bite force (Osborn and Baragar, 1985; Koolstra *et al.*, 1988; Osborn, 1995, 1996).

DEVELOPMENT OF THREE-DIMENSIONAL BITE FORCE TRANSDUCERS

More than ten types of bite force transducers have been developed during the past 30 years. Most commonly used sensoring components are spring, strain gauge, pressure foil and piezoelectric materials. The transducer can be made in many forms for example, bite forks, bite foil, bite blocks *etc.*

In the past bite force transducers were designed to measure only the component of the bite force perpendicular to the recording surface. Graf *et al.* (1974) were the first to investigate the direction of a bite force, by incorporating a small three-dimensional piezoelectric force transducer (Kistler Instrument AG, Winterthur, Switzerland) in a dental bridge and later in dental implants (Mericske-Stern *et al.*, 1992). In their first study, the occlusal surface of an artificial tooth was placed on the top of the transducer. According to the manufacturer, the transducer suffered from cross-talk due to its very small size and is not now manufactured.

Later, Hylander (1978) bonded rosette gauges on both sides of a plastic block and used them to measure the incisal bite force direction, although only in the sagittal plane.

Van Eijden *et al.* (1988*a*) measured the direction of static bite forces using a commercial three-component piezoelectric load cell (Kistler Instrument). The load cell itself is about 10 mm thick and tooth separation is at least 14 mm. During the measurement, the load cell is sandwiched between two thin metal plates attached to the whole of the upper and lower dental arches by dental acrylic. The fact that all teeth are in contact with plates makes it impossible to exclude the effects of sensory feedback from individual teeth.

Osborn and Mao (1993) developed a three-dimensional bite force transducer having rosette strain gauges (Micro-Measurements Inc., Raleigh, NC, USA) as the sensors. The original stainless steel housing was H-shaped but has now been modified into a U-shape (Paper #1). Rosette strain gauges are bonded to both vertical sides of either the H or the U. Tooth separation can be reduced to 2 mm by adjusting the thickness of the horizontal plates of the transducer. Thin metal has the disadvantage of permanent deformation under load. Thick metal does not distort so readily but increases tooth separation.

The Osborn-Mao transducer has some advantages over the piezoelectric as follows.

(1) The jaw separation is reduced to only 2-4 mm.

(2) There is no need to sandwich the transducer or have the transducer locked in one position between the teeth during measurement. The transducer is attached to the tooth in only one arch (by acrylic overlays). Therefore, the study of forces directly applied to the opposing tooth is possible.

(3) The strain gauge technology is less expensive.

(4) The sensory input is limited to the teeth being studied rather than, as is the case with the van Eijden transducer, coming from all the teeth.

The only disadvantage of our transducer is its sensitivity to the position of load on its horizontal arm. In order to make the measurement reproducible, it is therefore necessary to load the transducer in the same way as when it is calibrated. A cone or a wedge made from acrylic resin attached to the horizontal arm of the transducer allows us to standardize the position at which a load is applied (see Papers #1, #2 and Appendix A).

Care should be taken when interpreting the results from uni-directional force studies. Measuring a decrease in the force magnitude in a new situation may not necessarily reflect the actual change since the direction of the new force could be different. A uni-directional force transducer can only measure the perpendicular component of a force and ignores its horizontal components with respect to the transducer.

Although three-dimensional transducers provide a more accurate bite force measurement, the bite direction is usually measured with respect to the plane of the transducer. Standardization of the reference plane is, therefore, necessary when comparing the directional data between studies. In some cases where the best placement of the transducer (parallel to the occlusal plane or the body reference planes) is not possible, an angular transformation is required (see Appendix C).

Interpreting a bite direction between molar teeth is more difficult since the direction presumably depends on the nature of tooth contact, shape and surface texture (Graf *et al.*, 1974). In other words, the direction being measured depends on cusps and inclines which are in contact. Most bite direction studies on multi-cusp teeth usually cover the occlusal surface with acrylic resin. The contact surface is hence flat.

STUDIES OF BITE FORCE DIRECTION

Graf *et al.* (1974) introduced a method to measure human chewing force in three dimensions and showed that the peak force magnitude is mainly vertical with small lateral and posterior components. During a chewing stroke, the bite force appeared to change from an inward to outward direction (with respect to the mandible) on molar teeth on the working side. They also showed that occlusal morphology affects the bite force direction. Their later study with dental implants, in contrast, showed that the occlusal and chewing forces are mainly directed in vertical, medial and anterior directions (Mericske-Stern *et al.*, 1992). The difference could be due to the difference in the biting surface. The first study covered the surface of the transducer with an occlusal surface shaped like a tooth whereas the later study did not.

MacDonald and Hannam (1984*a*, 1984*b*) used acrylic occlusal jigs of different inclines to control the bite force direction. They showed that the direction of the bite force, according to the inclination of the biting surface, was well correlated with the activity of the jaw muscles whose orientations were parallel to the direction of the force. For example, protrusive clenching is associated with more activity in the masseter and medial pterygoid muscles. Retrusive clenching is associated with temporalis muscle activity. Similar results were found in studies of clenching at the intercuspal position with different directed efforts (Wood, 1987).

Van Eijden (1991) showed the direction in which the MBF was produced was generally medial and posterior on canines, second premolars and second molars (with respect to the horizontal plane). With incisors, both submaximal bite forces (Hylander, 1978) and MBFs (Paper #1) were found to be anteriorly directed (with respect to the lower occlusal plane).

When the direction of a bite force is maintained, the activity in a jaw closing muscle seems to increase linearly with submaximal bite forces (Mao and Osborn, 1994). The linearity is maintained up to the MBF (van Eijden *et al.*, 1990). This, however, has not been found in other studies (Pruim *et al.*, 1978; Devlin and Wastell, 1985; Hagberg *et al.*, 1985; Wastell and Devlin, 1987). A change in bite direction as the bite force is increased, which was not measured by the latter workers, may explain the difference.

Our preliminary studies have shown that the measured bite direction is constant regardless of its magnitude at *discrete* submaximal levels. This seems to imply that the direction of submaximal bite forces is controlled by a neural circuit which is activated in a linear fashion. However, when a *continuously* increased bite force is tested up to its maximum with incisor teeth the bite direction changes slightly as the bite force is increased and the contribution from both masseter and anterior temporalis muscles is not always a constant ratio (unpublished results). The linearity may change at large bite forces.

It has also been shown that the direction of an incisal bite force shifts backwards during a clenching endurance test although its magnitude is maintained (Osborn and Mao, 1993). This seems to correspond with a decrease in the masseter activity and an increase in the anterior temporalis muscle (Hellsing and Linström, 1983).

Together with bite force magnitude, the bite force direction provides a comprehensive view on how jaw closing muscles function together as a synergistic system. Measuring the activity in all jaw muscles is not an easy task. Having obtained the bite force magnitude, bite force direction in conjunction with activities from some accessible superficial muscles we can input these data into a computerized linear programming model of the jaw (Osborn, 1995, 1996) and predict the activity of the deeply seated muscles.

MECHANORECEPTORS IN THE ORAL REGION

Mechanoreceptors are defined here as receptors which are activated by mechanical distortion. They provide information about forces, position and movement but not pain. Such receptors were classified as exteroceptors (receptors responsive to cutaneous stimuli) and proprioceptors (receptors responsive to position of body parts) according to Sherrington's (1906) classification.

In the masticatory system, several mechanoreceptors both inside and outside the mouth are important in regulating jaw function. They consist of periodontal receptors, TMJ receptors, muscle spindles, Golgi tendon organs, skin and mucosal receptors, and periosteal receptors. A role for pulpal nerves as mechanoreceptors has not been confirmed and is still far from being accepted.

PERIODONTAL MECHANORECEPTORS

Structure – The course of nerves supplying the periodontal ligament and the structure of their endings has been studied by light microscopy, electron microscopy, histochemistry, immunohistochemistry and by labeling axonally transported markers.

Different types of nerve endings in the periodontal ligament were described using light microscopy in early studies. Among these were Pacinian-like corpuscles (Black, 1887 cited by Lewinsky and Stewart, 1937*b*), spindles or knob-like endings (Simpson, 1966 [human]), coiled endings (Lewinsky and Stewart, 1936 [human]; Rapp *et al.*, 1957 [human]; Kizior *et al.*, 1968 [cats]), Ruffini-like and tree-like endings (Rapp *et al.*, 1957; Sato *et al.*, 1992 [dogs]). Free nerve endings have also been described (Bernick, 1952; Kubota and Osanai, 1977; Byers, 1985; Lambrichts *et al.*, 1992). This variation could be real or due to different planes and levels of sectioning.

More recent electron microscopic studies have consistently shown unencapsulated Ruffini-like endings in the periodontal ligament of several species (Byers, 1985 [rats]; Nakamura *et al.*, 1986 [human]; Millar *et al.*, 1989 [cats]; Kannari, 1990 [hamsters]; Sato *et al.*, 1992 [dogs]; Linden *et al.*, 1994 [cats]). A typical Ruffini ending in skin has a cylindrical shape, consisting of a single myelinated axon terminal with finger-like protrusions enclosed in a thin lamellated capsule. The protrusions are anchored to adjacent collagen fibres. Ruffini endings are known to respond to stretching of tissue similar to Golgi tendon organs.

The nerves supplying the periodontal ligament derive from the superior or inferior alveolar nerves (Linden *et al.*, 1995) and presumably from the lingual or palatine nerves. Nerve bundles run coronally from the apical region and are joined by small bundles coming out of the walls of the alveolar bone (Lewinsky and Stewart, 1936; Byers, 1985). About 14-22% of the inferior dental nerve fibres supplying cat periodontal ligament are unmyelinated (Brashear, 1936; Kizior *et al.*, 1968). The diameter of the unmyelinated fibres is about 0.5 μ (Hannam, 1982) whereas that of the myelinated fibres ranges from 2-16 μ (Brashear, 1936; Kizior *et al.*, 1968).

Nerves in the periodontal ligament travel close to both alveolar walls (Lewinsky and Stewart, 1936; Everts *et al.*, 1977) and close to cementum (Byers, 1985). In general, the endings are densely located in the apical region of the periodontal ligament (Bernick, 1959; Dubner *et al.*, 1978; Byers and Matthews, 1981). However, in cat canines, the nerve endings whose cell bodies are in the trigeminal ganglion are found primarily around the middle part of the root whereas those from the mesencephalic nucleus are found primarily around the apical region (Byers and Dong, 1989). In addition, more axons are present in the mesial and distal parts of the root than facially and lingually (Loescher and Holland, 1991).

The cell bodies of the first-order neurons supplying periodontal mechanoreceptors are in either the trigeminal ganglion or the mesencephalic nucleus. The mesencephalic neurons that respond to tooth stimulation are located in the caudal part of the nucleus (Gottlieb *et al.*, 1984*a* [cats]; Nomura and Mizuno, 1985 [cats]). The projection from teeth to the mesencephalic nucleus is ipsilateral (Corbin and Harrison, 1940; Jerge, 1963*a*) but bilateral representation has also been demonstrated (Smith and Marcarian, 1968). Mesencephalic neurons supplying the periodontal ligament have been shown to project to interneurons in the supratrigeminal nucleus and inhibit jaw closing motoneurons (Kidokoro *et al.*, 1968). Neurons from the trigeminal ganglion send axons to both trigeminal main sensory and spinal nuclei (Kawamura and Nishiyama, 1966; Woda *et al.*, 1983; Tabata and Karita, 1991*c*).

Electrophysiological studies have shown that some neurones within the VPM (ventralis posteromedialis) of the thalamus (Yokota *et al.*, 1988) and some cortical sensory neurones (Lund and Sessle, 1974) respond to forces applied to the teeth. Woda *et al.* (1983) found some neurones from the main sensory nucleus which responded to mechanical stimulation of the teeth, either ipsilaterally or contralaterally, projected to the VPM of the thalamus. A projection from periodontal receptors to the cerebellum has also been shown. Taylor and Elias (1984) recorded short-latency field potentials in the cerebellar cortex when electrically stimulating low-threshold nerves in the inferior alveolar nerve. They concluded that there was a direct projection from periodontal mechanosensitive afferents to the cerebellar cortex. This connection is supported by the anatomical finding that only neurons in the caudal part of the mesencephalic nucleus degenerate following cerebellar lesions (Brodal and Saugstad, 1965).

Neurophysiology – Periodontal mechanoreceptors are classified according to the way they respond to forces applied to a tooth, either rapidly adapting or slowly adapting. Spontaneously-active units have also been described (Pfaffmann, 1939*a* [cats]; Ness, 1954 [rabbits]; Wagers and Smith, 1960 [dogs]; Matthews, 1965 [dogs]; Hannam, 1969*b* [dogs]). Rapidly adapting units constitute about 20% of the population (Hannam, 1969*b*; Loescher and Robinson, 1989*a*) and fire a few spikes during a ramp-plateau force whereas slowly adapting units fire continuously throughout the stimulation. Spontaneously active units are slowly adapting but maintain a spontaneous discharge even when the stimulus is removed. Hannam (1969*b*) suggested that this spontaneous activity could be related to low-grade mechanical stimulation within the periodontal ligament. The slowly adapting units tend to be located in the apical third of the periodontal ligament and the rapidly adapting units tend to be located near the middle third (Cash and Linden, 1982*b*). In recordings from the inferior alveolar nerve in man all periodontal mechanoreceptors studied were slowly adapting, more than half of them being also spontaneously active (Trulsson *et al.*, 1992).

Linden (1978, 1990), on the other hand, suggested there is only one type of mechanoreceptor present in the periodontal ligament and the response characteristics depend on the distance of the receptor from the fulcrum of the tooth. In his studies, the receptor site was exposed by paring the labial alveolar wall until a thin layer of bone remained. The corresponding axons could be traced by applying an electrical current to a small area of the exposed bone while the electrical activity was recorded in a single nerve fibre teased from the inferior alveolar nerve. It was demonstrated that the closer the unit to the fulcrum, the more rapidly it adapted to the force and the higher the threshold. This relationship, however, was not found by Loescher and Robinson (1989*a*). Regarding the spontaneously active units, Cash and Linden (1982*b*) argued that it could be the discharge from cut sympathetic nerves. The idea of a single type of receptor is worth attention but the term "high threshold" for the units near the fulcrum may be incorrect. They may have the same threshold as the apical group but, being at the fulcrum of movement, the deformation of the periodontal ligament is too small for the receptors to be activated.

The nerve fibres from the mesencephalic nucleus have been shown to be only rapidly adapting (Linden, 1978; Linden and Scott, 1989*a*) whereas those from the ganglion are both slowly and rapidly adapting (Kerr and Lysak, 1964; Beaudreau and Jerge, 1968). This is in contrast to the finding that most periodontal receptors projecting to the mesencephalic nucleus are slowly adapting and also supply more apical regions of the tooth (Byers and Dong, 1989).

The conduction velocity of nerves supplying periodontal ligament determined by mechanical stimulation to a tooth ranges between 26-87 m/s with a mean of 54 m/s (Hannam, 1968; De Lange *et al.*, 1969; Linden, 1978; Linden and Scott, 1989*a*). The speed corresponds well with the A-β group. A-δ and C fibres have also been reported (Mei *et al.*, 1977; Byers, 1984; Mengel *et al.*, 1992, 1993*a*) but they are likely to be involved in nociception.

The minimum force applied to a tooth that activates periodontal mechanoreceptors is small, ranging from 10-800 mN (*e.g.* 10-20 mN [Linden, 1990]; 15-26 mN [Dong *et al.*, 1993]; 73-193 mN [Loescher and Robinson, 1989*a*]; 60-800 mN [Linden and Millar, 1988*a*]) The thresholds are not significantly different whether the force is applied vertically or horizontally to the tooth (Dong *et al.*, 1993). The rapidly adapting units have higher thresholds (~200-500 mN) than the slowly adapting units (~10-50 mN) (Hannam, 1969*a*; Linden and Millar, 1988*a*; Loescher and Robinson, 1989). The threshold also depends on the rate of force application, being smaller when a force is more quickly applied (Linden and Millar, 1988*b*; Loescher and Robinson, 1989*a*). This could not be simply explained by the viscoelastic property of the periodontal ligament since less intrusion has been observed in response to a rapidly applied force. A larger force would therefore be required to activate the receptors.

The mean firing frequency of periodontal mechanoreceptors seems to reach its peak well below a 5 N force (Linden *et al.*, 1995). The firing frequency has been shown to be about 140 Hz at 0.5 N (Linden and Millar, 1988*a*), 89-210 Hz at 0.5 N (Loescher and Robinson, 1989*a*), 30-100 Hz at 60 mN (Dong *et al.*, 1993). Such a small range of effective stimuli raises the question as to whether periodontal receptors can play the major role in bite force regulation during chewing when the bite forces are much larger. Kizior *et al.* (1968) observed two subgroups of response from the inferior alveolar nerve of the cat, one sensitive to small vertical forces (below 4 N) and the other sensitive to larger forces (over 12 N). The first response is probably from periodontal mechanoreceptors but the origin of the second response is unclear. As opposed to their speculation that this is the nociceptive response from the periodontal ligament, we think that the level of force was unlikely to cause pain and suspect it is the response from other mechanoreceptors supplied by the inferior alveolar nerve (possibly those in the tooth pulp).

Periodontal mechanoreceptors are directional-sensitive (Pfaffmann, 1939*a*; Wagers and Smith, 1960; Jerge, 1963*a*; Kerr and Lysak, 1964; Hannam, 1969*a*; Johansson and Olsson, 1976; Linden, 1978; Tabata and Karita, 1986; Loescher and Robinson, 1989*a*; Dong *et al.*, 1993). In other words, their maximum firing frequency is highest when a force is applied in a particular direction. Most of the receptors in human (86%) respond to axial loads (Trulsson *et al.*, 1992). For horizontal loads, Tabata and Karita (1986) and Loescher and Robinson (1989*a*) showed that most of the cat periodontal receptors fire maximally when a force is applied in a caudo-medial direction. Linden and Scott (1989*a*) and Dong *et al.* (1993), on the other hand, did not find directional sensitivity in the cat when recording from receptors having cell bodies in the ganglion. However, Linden and Scott (1989*a*) did find directional sensitivity in a rostro-lateral direction when recording from the mesencephalic nucleus. The difference in the arc of sensitivity is not clear but could be due to the difference of the recording site (Tabata and Karita and Loescher and Robinson recorded from peripheral nerves). Directional sensitivity is less frequently found in the thalamus (Tabata and Karita, 1991*c*).

It was thought earlier that the periodontal receptors were activated by compression. More recent electron microscopy (Byers, 1985 [rats]; Nakamura *et al.*, 1986 [human]; Millar *et al.*, 1989 [cats]; Kannari, 1990 [hamsters]; Sato *et al.*, 1992 [dogs]; Linden *et al.*, 1994 [cats]) has consistently shown unencapsulated Ruffini-like terminals anchored to collagen bundles and likely to respond to stretch or tension. In addition, a study of periodontal mechanoreceptor responses to horizontal forces applied to the tooth confirms that they fire maximally when they are stretched, not compressed (Linden and Millar, 1988*a*).

The receptive field of most periodontal mechanoreceptors is confined to one tooth (Kruger and Michel, 1962; Kawamura and Nishiyama, 1966; Tabata and Karita, 1986). Canine teeth have the largest representation in the trigeminal nuclei (Kawamura and Nishiyama, 1966; Tabata and Karita, 1991*c*) and in the superior alveolar nerve (Tabata and Karita, 1986). Multi-tooth sensitive neurons are also found (Woda *et al.*, 1983) and often project to anterior teeth (Tabata and Karita, 1986). The multi-tooth response is thought to be due to either a true branching of periodontal nerves or just the transmission of force between several teeth via interproximal contacts (Millar *et al.*, 1994; Linden *et al.*, 1995).

Roles in mastication – Periodontal mechanoreceptors are very sensitive to small forces applied to the teeth. In man, the tactile threshold for an axial force is about 1 gm (~10 mN) for the incisors and about 6-7 gm (~60-70 mN) for molars (Adler, 1947; Manly *et al.*, 1952; Loewenstein and Rathkamp, 1955; Tomeck and Howley, 1989). The tactile threshold of a pulpless tooth in man was shown to be higher than that for a normal tooth (Loewenstein and Rathkamp, 1955) but this was not confirmed by Linden (1975). It should be noted that forces used in the above studies were very small (< 100 mN) and likely to be detected by periodontal rather than pulpal mechanoreceptors. The tactile threshold is about eight times larger with endosseous implants (Hammerle *et al.*, 1995).

Periodontal mechanoreceptors may be used to discriminate between bite forces. The sensitivity is the same for vertical and horizontal forces (Bowman and Nakfoor, 1968; Bonaguro *et al.*, 1969). In normal subjects, the ability to discriminate is better at forces around 10 N than at 5 or 30 N (mean resolution = 2.75 N; Williams *et al.*, 1984). The ability to discriminate between small bite forces (around 5 N) is significantly reduced in denture wearers, but discrimination between large bite forces (around 10-30 N) is unaffected (Williams *et al.*, 1985). This suggests that periodontal receptors are primarily responsible for detecting small forces. Anaesthetizing the superior compartment of the TMJ (Williams *et al.*, 1989) does not alter discrimination sensitivity whereas anaesthetizing the teeth reduces the sensitivity (Williams *et al.*, 1984). However, subjects do not completely lose their ability to discriminate between bite forces (mean resolution = 3.56 N) even when the sensory inputs from both TMJ and periodontal ligament are impaired (Williams *et al.*, 1984). It should be noted that receptors in distant regions such as neck muscles which resist the external force applied to the teeth may also be involved in a bite force discrimination task.

A role for periodontal receptors in controlling bite forces is not clearly established. Several studies have compared the maximum bite force between control and anesthetized teeth but the results are controversial. Lund and Lamarre (1973) found a decreased activity in the masseter and anterior temporalis muscles after local anesthetics were given. A gradual reduction of the MBF was observed in three subjects tested during the 5-20 minute period when the anesthetic was most effective. They concluded that during voluntary biting the periodontal receptors provided a positive feedback to jaw closing muscles.

On the other hand, van Steenberghe and De Vries (1978) with nine volunteers and Orchardson and MacFarlane (1980) with 15 volunteers found an increase in the maximum bite force after local anesthesia. The profoundness of the anesthesia was, however, only tested in Orchardson's study, by an electrical pulp tester. In their discussion, Orchardson and MacFarlane suggested that not all periodontal nerve fibres had been anesthetized. The effect of the local anesthetics therefore depended on which receptor groups were anesthetized. Both pain receptors and mechanoreceptors are present in the periodontal ligament. Small fibres, presumably carrying nociceptive inputs, are generally more sensitive to local anesthetic agents (Nathan and Sears, 1961) than larger fibres. This could account for the increase in bite force after local anesthesia.

Teenier *et al.* (1991) measured bite forces and activities in masseter, anterior and posterior temporalis and digastric muscles at four predetermined forces (25%, 50%, 75% and MBF) in 20 subjects. They found no significant difference in (1) the MBF between the unanesthetized and anesthetized sides of the jaw, (2) the MBF on the anaesthetized side at different levels of anesthesia and (3) the %EMG at each predetermined bite force before and after anesthesia. The findings were similar regardless of the level and type of anesthesia (mandibular block alone, both maxilla and mandible, both maxilla and mandible with eyes closed). They attributed the changes observed in previous studies mainly to the lack of verifying anesthetic profoundness and inadequate statistical methodology.

The above controversial findings imply that periodontal receptors are not responsible for regulating the MBF.

The "jaw opening reflex" is generally evoked by applying a rapid tapping force to a tooth (4 N – Hannam and Matthews, 1968 [cats]; 0.25-1 N – Bonte *et al.*, 1993 [cats]; 1 N – Louca *et al.*, 1998 [man]). A similar reflex is also seen after sudden tooth contact in centric occlusion (Matthews, 1976; Brenman *et al.*, 1968; Hannam *et al.*, 1970; Munro and Griffin, 1970 [all in man]). During the jaw opening reflex the masseter and temporalis muscles are temporarily inhibited, causing a silent period in the EMG bursts. In cats (Olgart *et al.*, 1988; Bonte *et al.*, 1993) and monkeys (Sessle and Gurza, 1982) the digastric muscles are also excited but probably not in man (Beaudreau *et al.*, 1969; Gillings and Klineberg, 1975). The silent period is often preceded by a brief increase in muscle activity (Hannam *et al.*, 1970; Goldberg, 1971; Sessle and Schmitt, 1972 [all in man]).

Reflex horizontal jaw movement to the contralateral side has been demonstrated in decerebrate rabbits by applying light pressure on the labial surface of an incisor tooth (Lund and Dellow, 1971). In lightly anesthetized monkeys, the activity is seen in both superior and inferior heads of the muscle by a tap on the incisor or by sustained pressure on the canine tooth (Sessle and Gurza, 1982).

The jaw opening reflex has been demonstrated during chewing when teeth come into contact (Anderson and Picton, 1957; Ahlgren, 1969; Hannam *et al.*, 1969) or when teeth contact hard foods (Schwartz *et al.*, 1989 [rabbits]). Lavigne *et al.* (1987) placed a steel ball between the molar teeth on one side of the rabbit jaw and showed that the reflex only occurred in the first cycle and later disappeared, the pattern being confirmed by Schwartz *et al.* (1989). Together with the reduced jaw opening reflex, they also found an increased activity in jaw closing muscles during later cycles. The finding implies that the jaw opening reflex can be modulated and may be suppressed in chewing.

Stimulating periodontal receptors not only inhibits but also activates jaw closing muscles. The smaller MBF observed in denture wearers (Hellsing, 1980) and the reduction of the MBF after local anesthesia (Lund and Lamarre, 1973) support the positive feedback hypothesis for periodontal receptors. Greenwood and Sessle (1976) and Sessle (1977) showed that a jaw closing reflex could be elicited by stimulating periodontal afferent fibres which would happen when food is encountered between teeth during chewing (Lund and Olsson, 1983). In anesthetized monkeys, tapping the incisor tooth can evoke activity in the superior head of the lateral pterygoid (Sessle and Gurza, 1982), considered as one of the muscles associated with jaw closing. During cortically-evoked chewing in rabbits, placing an object between the teeth increases activity in the masseter muscle but the closing reflex is greatly reduced after anesthetizing both alveolar nerves (Morimoto *et al.*, 1989).

The level of background activity in the jaw muscles is important in determining the response from periodontal mechanoreceptors. Yamamura and Shimada (1992) showed that in the masseter muscle with large spontaneous activity the effect of periodontal stimulation is inhibitory whereas in the muscle with small or no spontaneous activity the effect is excitatory. In addition, the response may be affected by the rate at which force is applied to the tooth (Brodin *et al.*, 1993). A brief tap on an incisor tooth evokes an inhibitory response in the human masseter muscle whereas a slow push causes excitation. In this case, the rapidly applied force may be interpreted as a noxious stimulus.

It should be noted that tapping the tooth could also stimulate receptors in other structures such as auditory receptors (van der Glas *et al.*, 1988; Sato *et al.*, 1994), muscle spindles (Hannam *et al.*, 1970) and perhaps, periosteal or TMJ mechanoreceptors. However, the predominant role of periodontal receptors in the jaw opening reflex has been demonstrated by Bonte *et al.* (1993). They showed that inhibition of the cat temporalis muscle was greatly reduced when rapid forces were applied to an implant, as opposed to a natural tooth.

The number of teeth being stimulated may also play a part in controlling the jaw reflex. With fewer teeth the MBF is significantly reduced (Helkimo *et al.*, 1977; van Steenberghe and De Vries, 1978*b*; Waltimo and Könönen, 1994). From a mechanical standpoint, under a given bite force, forces on the periodontal ligament are larger when biting on a single tooth than when biting on several teeth. Biting on a single tooth may give a greater response from the single tooth whereas biting on several teeth may give a lesser response from each tooth but more receptors are activated. The ultimate response depends on how the central nervous system weights the information received. Thus, the number of teeth biting on a transducer may also contribute to the variation of MBFs measured in different bite force studies.

TEMPOROMANDIBULAR JOINT (TMJ) RECEPTORS

Structure – Like other diarthrodial joints in the body, four types of nerve ending have been generally described in the mammalian temporomandibular joint, namely (1) free nerve endings, (2) unencapsulated Ruffini-like endings, (3) Golgi tendon endings and (4) encapsulated Pacinian-like corpuscles (Thilander, 1961). The most numerous are free nerve endings. A recent immunohistochemical study, in contrast, could not demonstrate any specialized endings in human TMJs (Morani *et al.*, 1994) and only free nerve endings were found in the mouse TMJ, mainly in the dorsolateral part of the joint (Dreesen *et al.*, 1990). Nerve endings are conclusively found in the joint capsule and its capsular ligament but not in the articulating surfaces nor the central portion of the TMJ disc. The posterior-lateral region of the capsule is the most densely innervated (Tahmasebi-Sarvestani *et al.*, 1996). It has been shown that the human TMJ disc proper is highly innervated in the fetus but is gradually reduced during the last trimester, possibly in response to the growth of articular tissues (Ramieri *et al.*, 1996).

The primate TMJ is mainly supplied by the auriculotemporal branch of the trigeminal nerve. The anterior and lateral parts of the capsule are also innervated by small branches from the masseteric and posterior deep temporal nerves (Thilander, 1961). The diameter of most TMJ nerve fibres in man is less than 5 μ. Fibres larger than 10 μ are rare and presumably supply Pacinian corpuscles in the capsule and tendon organs in the temporomandibular ligament. Small-diameter nerves supply free nerve endings and could be autonomic fibres (Dubner *et al.*, 1978; Tahmasebi-Sarvestani *et al.*, 1996 [sheep]).

By injecting a compound of horseradish peroxidase (HRP) into the TMJ cavity, the cell bodies of those nerves supplying the joint were shown to be in the dorsolateral part of the ipsilateral trigeminal ganglion (Capra, 1987 [cats]; Chen and Turner, 1992 [rats]). Their central projections were found in the nucleus caudalis (Capra, 1987; Hathaway *et al.*, 1995) and rostral nuclei (Capra, 1987). None of the cell bodies was found in the mesencephalic nucleus (Chen and Turner, 1992).

Neurophysiology – Most receptors in the cat knee joint fire at the extreme joint positions rather than in the middle range of joint movement (Skoglund, 1956). This suggests that they may be used to protect the joint from overextension and overflexion. However, many slowly adapting endings in the joint increase their firing frequency as the joint rotates, although over a limited range (15-30°) (Skoglund, 1956).

Skoglund (1973) suggested that the tendon organs in joint ligaments are able to signal the exact position of the joint and the Pacinian corpuscles are able to detect very small movements and might signal the acceleration. Ruffini endings in joints signal the speed and direction of movements. They also respond to the contraction of muscles inserting at the joint and are probably used to distinguish between active and passive joint movements.

TMJ receptors in rabbits are inactive when the jaw is in its rest position and fire either phasically or continuously within a limited range of joint movement (Lund and Matthews, 1981). All receptors are active for a preferred direction of movement, presumably the one which stretches them. By recording from the auriculotemporal nerve, both slowly and rapidly adapting responses have been found in the isolated temporomandibular joint of the cat (Kawamura and Abe, 1974). The rapidly adapting units only fire at the start and the end of the jaw open-close cycle whereas some slowly adapting units fire throughout the cycle (Klineberg, 1971; Kawamura and Abe, 1974). The cat TMJ nerves fire more frequently as the velocity of jaw opening is increased (Klineberg, 1971). The above results suggest that some TMJ receptors are used to detect the position of the mandible as well as its velocity.

Since most nerve endings are found in the capsule of the temporomandibular joint they are probably not much stimulated during the initial (rotary) stage of jaw opening. Any awareness of a change in jaw position mediated by TMJ mechanoreceptors would be less sensitive during the early stage of jaw opening.

Roles in mastication – Joint receptors have been thought to be involved in kinesthesis (the recognition of position during movement – Sherrington, 1900) and also to protect the joint from movements beyond the physiological limit. During passive rotation of the TMJ isolated from a cat, where muscle receptors are unaffected, the background activity of the masseter muscle is reflexly decreased when the joint is rotated closed but increased when the joint is rotated open (Kawamura *et al.*, 1967; Clark and Wyke, 1974). This suggests that TMJ receptors may be used to signal the jaw resting position.

TMJ receptors are thought to detect the amount of jaw opening. Thilander (1961) showed that subjects could duplicate a preset jaw opening with an error of as small as 3.2 mm. The error was increased when one or both TMJs were anesthetized. Local anesthesia of the lateral pterygoid muscle alone (Christensen and Troest, 1975) reduces the accuracy of sensing jaw position, implying that receptors in TMJs and muscles may both be important. During these experiments, however, some anaesthetic might have leaked, either into the joint cavity or into the lateral pterygoid muscle.

TMJ receptors may be involved in detecting the thickness of objects, as thin as 8 μ, placed between upper and lower teeth. There is no significant difference in the sensitivity of incisor and molar teeth (Siirilä and Laine, 1963). This is interesting because a higher sensitivity would be expected with molar teeth if receptors in the TMJ and jaw muscles are primarily responsible (because the same interocclusal thickness on molar teeth results in a larger jaw opening and therefore stretches the tissues more). Caffesse *et al.* (1973) showed that after anaesthesia of both TMJs the detection threshold of 60% of subjects was significantly increased (mean from 16 μ to 32 μ). Nevertheless some subjects could still detect thicknesses as small as 8 μ. This suggests that TMJ receptors are important in detecting thickness although they are probably not the only ones involved. Anaesthetizing the teeth slightly increases the detection threshold (Siirilä and Laine, 1963; Fenton, 1973) implying that periodontal receptors could also be involved although it is difficult to explain how the change in thickness would affect periodontal input. It is also difficult to understand how such a small jaw opening would activate receptors in the joint capsule since the amount of condyle rotation would be extremely small.

It is also thought that TMJ receptors are used to detect the difference between two objects of different thickness placed between upper and lower teeth. Kawamura and Watanabe (1960) showed that the smallest difference of 0.2-0.3 mm could be detected with natural teeth. Riis and Giddon (1970) found the ability to discriminate was reduced when the teeth were anaesthetized. In contrast, the ability was not different for natural and artificial incisor teeth (Manly *et al.*, 1952). This observation discounts the involvement of periodontal receptors. It has also been shown that the absolute discrimination threshold is almost unchanged as the amount of jaw opening is increased (Ringel *et al.*, 1967; Siirilä and Laine, 1972). This does not seem to follow the psychophysics law which states that at higher stimulus intensity, a larger alteration in stimulus magnitude is required before a change can be detected (Weber's ratio, or Δs/s, is constant or decreased).

Discrimination between the thickness of objects placed between upper and lower teeth may be thought of as a simple comparison between two jaw separations. It should, however, be noted (Morimoto, 1983) that it also involves tooth contact. Since the error in reproducing a jaw position is as large as 3 mm, thickness discrimination by teeth (threshold for detection < 10 μ, threshold for discrimination < 0.5 mm) is unlikely to be just a comparison between two different jaw positions. Morimoto (1983) measured the time needed to perceive the difference in thickness between objects 6-13 mm thick placed between the teeth. He showed, in 12 subjects, that the perception of differences in thickness was reached before tooth contact when the difference was greater than 3 mm but after tooth contact when the difference was less than 3 mm. The finding suggests that for small differences (< 3 mm) the information from tooth contact is important for the discrimination of thickness. The brain presumably compares the jaw closing time before teeth get into contact in order to tell the thickness. The information about tooth contact is unlikely to come from periodontal receptors because the periodontal ligament was anaesthetized in Morimoto's study and the ability to discriminate has been shown to be unaltered in denture wearers (Manly *et al.*, 1952) or when the periodontal ligament is anaesthetized (Christensen and Morimoto, 1977). Impact or vibration at the moment teeth come into contact with an object is more likely to be used to signal tooth contact. The signals are probably detected by remote receptors such as those in the ear.

In man, it has been shown that locally anesthetizing the TMJ does not alter the accuracy of bite force discrimination (Hellsing, 1980; Williams *et al.*, 1989). Hellsing (1980) also showed that anesthetizing the TMJ does not alter the maximum incisal bite force. Although TMJ receptors are not thought to respond to joint loading, there is evidence that the discharge frequency of some receptors (presumably Ruffini type) can be altered by activating muscles that act around the joint (Skoglund, 1973).

There is no agreement on whether temporomandibular disorder (TMD) patients have lower MBFs than normal subjects (Marklund and Molin, 1972; Helkimo and Ingervall, 1978; Hagberg *et al.*, 1986*b*; Gelb, 1990). However, it should be noted that not only TMJs but also jaw muscles are affected in TMD patients. In addition, there is no clear explanation why TMJ pain, presumably originating from capsule and ligament, would affect the production of isometric bite forces since no nerve endings are found in the load-bearing area of the articular disc. It is possible that in the case of TMJ disc displacement (in which the disc is not properly centred between the articular surfaces) the richly innervated soft tissues in the periphery of the disc may be misplaced and crushed during biting. A well-known fact is patients with a painful TMJ prefer to chew on the painful side, implying that the joint load is greater on the balancing side. It could also be that the painful TMJ capsule and ligament are stretched more during an excursion to the opposite side of the jaw.

In patients with internal derangements of the TMJ, the chewing cycle was found to be slower especially during the closing phase and jaw opening was more restricted (Shiau and Chen, 1986; De Leeuw *et al.*, 1995; Kuwahara *et al.*, 1995*a, b*). The abnormal movement may result from either pain or from damaged joint tissue that mechanically disturbs the movement of the TMJ.

MUSCLE SPINDLES

Structure – A muscle spindle is an encapsulated fusiform structure lying parallel to the direction of muscle fibres in the muscle belly. It contains two types of modified muscle fibres, namely nuclear bag and nuclear chain intrafusal fibres each recognized by the arrangement of their nuclei. The equatorial regions of both intrafusal fibres are innervated by large-diameter group Ia (primary endings) and group II (secondary endings) muscle afferents. Both bag and chain fibres are also supplied by small γ motor (or fusimotor) nerves near the polar regions. Excitation of the fusimotor nerve contracts the spindle poles (which contain more myofilaments) and stretches its central portion which activates spindle afferents. Two types of fusimotor nerves have been shown in muscle spindles, static (γ_s) and dynamic (γ_d). Some fusimotor nerves may be branched from the α motor nerves which supply the extrafusal muscle fibres (Barker *et al.*, 1972).

Muscle spindles have been identified in jaw closing muscles (Kubota and Masegi, 1972*a*; Nagashima, 1989; Eriksson and Thornell, 1990). There are more muscle spindles in the temporalis than masseter (Kubota and Masegi, 1972*a* [Japanese shrew mole]; Lund *et al.*, 1978 [kittens]). Muscle spindles are mostly found in the deep portion of the human masseter (Karlsen, 1965; Kubota and Masegi, 1972*a*; Bredman *et al.*, 1991) and their density tends to increase with distance from the TMJ (Bredman *et al.*, 1991). They are found more in the horizontal than in the vertical portion of temporalis in the shrew mole (Kubota and Masegi, 1972*a*). Spindles are most numerous in the medial portion of the human medial pterygoid (Karlsen, 1965; Kubota and Masegi, 1972). The distribution of muscle spindles tends to be similar to the distribution of slow muscle fibres (Maier, 1979; Rokx *et al.*, 1984; Eriksson and Thornell, 1987), suggesting they have a role in postural control. The presence of muscle spindles in digastric (Muhl and Kotov, 1988) and lateral pterygoid muscles (Karlsen, 1969; Gill, 1971; Nozaki *et al.*, 1984) is still in question. The scarcity of muscle spindles in the jaw opening muscles may be due to the fact that these muscles are already protected from over-stretching by tooth contact.

The nerves supplying the spindles in jaw closing muscles travel in the motor root of the trigeminal nerve (Szenthgothai, 1948*a*; McIntyre, 1951). The cell bodies are found exclusively in the trigeminal mesencephalic nucleus (Szentagothai, 1948*a*; Jerge, 1963*a*) and have been shown to be distributed throughout the nucleus (Gottlieb *et al.*, 1984*a*; Nomura and Mizuno, 1985). It has been shown, by means of HRP injection, that there is an extensive projection from the mesencephalic neurons supplying muscle spindles onto the ipsilateral trigeminal motor nucleus and supratrigeminal nucleus (Shigenaga *et al.*, 1988; Dessem and Taylor, 1989). Spindles from a jaw closing muscle project monosynaptically (Nakamura *et al.*, 1967; Appenteng *et al.*, 1978; Nozaki *et al.*, 1985; Luo and Li, 1991) to a restricted number of motoneurons in that muscle (Appenteng *et al.*, 1978 [cat's masseter]; Nozaki *et al.*, 1985 [guinea pig's lateral pterygoid]), unlike those in limb extensor muscles in which the spindle afferents project to many synergistic muscles. Based on measurements of nerve conduction velocity, Shigenaga (1988) suggested that spindle afferents terminating in the motor nucleus could come from spindle primary endings whereas those terminating in the supratrigeminal nucleus could come from spindle secondary endings. Projections from spindle afferents from the mesencephalic nucleus to the nucleus oralis have also been reported using different tracing methods (Walberg *et al.*, 1984; Luschei, 1987).

The neural pathways from muscle spindle afferents to higher centres have been studied. Stimulating group I afferents from forelimb and hindlimb muscles elicits activities in the contralateral sensorimotor cortex (Oscarsson and Rosen, 1966). The relay sites are found in the cuneate nucleus (Rosen, 1969), thalamus (Andersson *et al.*, 1966) and brain stem nucleus Z (Landgren and Silfvenius, 1971). In jaw muscles, stimulating the masseteric nerve evokes activity in the frontal cortex (Lund and Sessle, 1974), implying there is a connection between muscle spindle afferents and the cortex. Collaterals of neurons in the mesencephalic nucleus are also found to project to the cerebellum (Weinberg, 1928; Cupedo, 1970; Donga and Dessem, 1993) and cerebellar field potentials evoked from masseteric nerve stimulation have been recorded in cats (Cody and Richardson, 1979) and ferrets (Taylor and Elias, 1984).

Neurophysiology – Muscle spindles respond to muscle stretch. The primary endings, with a greater dynamic index (*i.e.* firing at a higher frequency during the ramp phase), respond primarily to the speed of muscle stretch whereas secondary endings respond to the amount of muscle stretch (muscle length) (Matthews, 1964). Secondary endings are also sensitive to the speed of stretch (Matthews and Stein, 1969). Exciting γ motoneurons can also activate muscle spindles. This is thought to increase the sensitivity of the spindle and to keep it taut and functional during active muscle shortening. Stimulating a phasically active static fusimotor fibre (γ_s) increases the sensitivity of both primary and secondary endings at rest whereas stimulating a tonically active dynamic fusimotor fibre (γ_d) increases the sensitivity in only primary endings during a ramp stretch (Matthews, 1964; Gottlieb and Taylor, 1983). The primary endings in cats are more sensitive to a small than a large passive stretch whereas the secondary endings respond linearly as the amount of the stretch is increased (Matthews and Stein, 1969).

As in limb muscles, the activation of muscle spindles in jaw closing muscles causes a reflex contraction, the so-called "jaw closing" or "jaw jerk" reflex. The reflex is monosynaptic. It is, however, unclear whether the jaw closing reflex is accompanied by a reciprocal inhibition in the antagonistic muscles (jaw opening muscles). Kidokoro *et al.* (1968) could not find inhibition in the cat digastric muscle when the masseter Ia afferents were stimulated. In contrast, Hagbarth *et al.* (1976*b*) showed reciprocal inhibition of suprahyoid muscles in man when the jaw closing muscles were vibrated. Hellsing (1977) showed that vibratory stimuli applied to the jaw opening muscles inhibited the activities in jaw closing muscles.

Muscle vibration (at around 200 Hz) is known to be an effective stimulus for muscle spindles in cats, especially their primary endings. Tonic vibration reflex (TVR) is the excitation of a muscle in response to muscle vibration due to the activation of muscle spindles (De Gail *et al.*, 1966; Hagbarth and Eklund, 1966). It probably involves both monosynaptic and polysynaptic pathways (Rothwell, 1994). The reflex has been demonstrated in jaw closing muscles (Godaux and Desmedt, 1975; Hagbarth *et al.*, 1976*b*) and in jaw opening muscles (Hellsing, 1977). The latter suggests the possibility that muscle spindles or a similar structure exist in the jaw opening muscles.

Roles in mastication – Muscle spindles have been thought to play an important role in controlling jaw muscle tone and presumably the jaw rest position. It is now accepted that the jaw rest position is actively controlled by jaw closing muscles. Muscle hyperactivity may be caused by an increased fusimotor drive that would increase the discharge from muscle spindles and reflexly result in an increased activity in the jaw closing muscles at rest. The association between the two is, however, not confirmed. Sustained muscle hyperactivity has been suggested to be one of the factors that contribute to temporomandibular disorders.

As discussed before, jaw position could well be sensed by muscle receptors. The activity of the spindle is increased during both passive jaw opening (Lund *et al.*, 1979) and active jaw opening (Cody *et al.*, 1975; Larson, *et al.*, 1983). The findings that the ability to reproduce a preset jaw opening is not completely impaired by anaesthetizing TMJs (Thilander, 1961), that the ability is reduced when the jaw muscles are fatigued (Christensen, 1976), that muscle vibration causes an illusion of jaw movement (in the direction of opening; Hellsing, 1977), that the sense of finger joint position is maintained even after the joint is surgically removed (Cross and McCloskey, 1973) all suggest that muscle spindles are involved in detecting jaw position. The possible role of muscle spindles in detecting jaw position supports the finding in limb muscles (Eklund, 1967; Goodwin *et al.*, 1972) that input from the spindles can reach consciousness.

Discriminating between the size of objects placed between upper and lower teeth is presumably associated with the activation of muscle spindles in the jaw muscles. This is supported by the finding that vibratory stimuli applied to the mandible disrupt the ability to discriminate between the sizes of objects (Morimoto and Kawamura, 1976).

During chewing, muscle spindles in the masseter muscle fire maximally at the maximum jaw opening and continue firing, though with reduced frequency, during active jaw closure (Goodwin and Luschei, 1975 – in awake monkeys) presumably due to the fusimotor drive. There is no spindle firing during a passive jaw closure. The firing pattern of muscle spindles during chewing is studied in cats where two groups of muscle spindles are classified according to their firing frequency, high (220-400 Hz) and low (below 120 Hz) (Taylor and Cody, 1974). The high frequency group is thought to be related to primary endings and the low frequency to secondary endings. It is presumed that only secondary endings fire during active muscle shortening. But occasionally when jaw closure is slowed down during eating, the presumed primary endings are also active (Taylor and Appenteng, 1981). Goodwin and Luschei (1975) and Lund and Olsson (1983) observed an increased discharge in muscle spindles when teeth encounter food during chewing. The increased spindle discharge during jaw closure may indicate some assistance from the servo-control of the jaw closing muscles during chewing (Goodwin and Luschei, 1975) or they may merely reflect an over stretch of the spindle caused by a simultaneous fusimotor excitation and the abrupt disturbance of jaw closure when encountering food.

Morimoto *et al.* (1989) observed the activity in the masseter muscle of the rabbit during cortically evoked chewing on a plastic strip. The activity was increased as a thicker plastic strip was chewed. Most but not all of the increased activity could be abolished by anaesthetizing the teeth. However, the rest of the increased activity was completely removed after damaging the mesencephalic nucleus. This suggests that part of the masseter excitation results from an input from muscle spindles. Removing periodontal input could have an indirect effect on muscle spindle activity since it has been shown that stimulating periodontal receptors by pushing the rat incisor increases the activity in a fusimotor nerve supplying the masseter (Funakoshi and Nagasawa, 1980). It has been suggested that input from the periodontal ligament may be used to calibrate the sensitivity of spindles (Taylor and Elias, 1984).

The role of muscle spindles in detecting bite force is not known. During the ramp phase of an isometric bite force muscle spindles, as well as γ motoneurons, are very active in trained monkeys (Lund *et al.*, 1979). The activity is not changed or only slightly reduced while the bite force is maintained. Furthermore, it has been shown that the discharge of both presumed primary endings (high sensitivity to sinusoidal stretch) and presumed secondary endings (low sensitivity to sinusoidal stretch) does not increase even when the isometric force is increased from 4 N to 26 N (Larson *et al.*, 1981*a*). Vibration applied to the masseter muscle increases a sustained bite force (Hagbarth *et al.*, 1976*b*) but not a phasic MBF (Hellsing, 1980) whereas that applied to the digastric muscle decreases it (Hellsing, 1977).

When brittle food is suddenly broken muscle spindles are transiently silent. This “unloading reflex” produces a “silent period” in the jaw closing muscles (Hannam *et al.*, 1968). A similar response is still seen after the teeth are anaesthetized (Lamarre and Lund, 1975), and in full denture-wearing subjects when a simulated resistance force placed between the upper and lower arches is rapidly changed from large to small (Kobayashi, 1994). These observations indicate that the reflex is probably not mediated by periodontal receptors. Goodwin and Luschei (1974), however, showed that the reflex was not abolished even when the mesencephalic nucleus had been destroyed, suggesting that muscle spindles are probably not involved. Hannam *et al.* (1968) suggested that TMJ receptors might play a more important role in the unloading reflex.

GOLGI TENDON ORGANS

Structure – A mammalian tendon organ is a spindle-shaped connective tissue capsule which encloses a number of collagen fascicles, most of which are innervated by the endings of large diameter myelinated nerves (group Ib; diameter 8-12 μ). Tendon organs are usually found at the junction between the muscle and its tendon or between the muscle and an aponeurosis. On one end the collagen strands of the tendon organ are fused with those of the tendon. On the other end they can be attached to 3-50 individual muscle fibres, varying between species and muscles (Jami, 1992). An average of ten motor units (range 4-15) attach to one tendon organ in the cat soleus muscle (Houk and Henneman, 1967).

The presence of tendon organs in the jaw closing muscles is not well established. Only one histological study showed tendon organ-like structures in the cat masseter and temporalis muscles (Lund *et al.*, 1978). In all specimens, the tendon organs were found in association with muscle spindles, called a "dyad". They were limited to the deep portions of both muscles where slow tonic muscle fibres are usually found. Some neurophysiological studies have shown that a small number of muscle afferents from jaw closing muscles, recorded in the mesencephalic nucleus (Smith, 1969; Larson *et al.*, 1983) and in the trigeminal ganglion (Lund and Matthews, 1981), have some firing characteristics similar to tendon organs (*e.g.* they discharge during muscle twitch). Sumi and Hanai (1979) also found some endings associated with the rabbit mylohyoid nerve that fired during the contraction of digastric and mylohyoid muscles.

The failure to demonstrate tendon organs in jaw muscles means there are no data on the projection of their afferents. However, it has been suggested that tendon organs, like muscle spindles, have their cell bodies in the mesencephalic nucleus (Smith, 1969;

Larson *et al.*, 1983) and project to trigeminal motor nuclei. In limb muscles, inputs from tendon organs reach the cerebellum by travelling in the spinocerebellar tracts (SCT), especially the ventral spinocerebellar tract (VSCT) (reviewed by Oscarsson, 1973). Within the dorsal spinocerebellar tract, the axons give branches to the nucleus Z that relays the information to the cerebral cortex (McIntyre *et al.*, 1985).

Neurophysiology – The nerves supplying tendon organs are in the range of Ib muscle afferent group with conduction velocities between 70-120 m/s in the cat. Nerve terminals within the tendon organs are believed to be activated by the contraction of the muscle. The contraction stretches the collagen fascicles in the tendon organ, which subsequently deform the nerve endings. Since not all collagen bundles within the tendon organ are innervated, the tension developed in the non-innervated fascicles may unload some adjacent innervated fibres in the same tendon organ (Houk and Henneman, 1967). It was once thought that tendon organs were only activated by large contractile forces, which therefore protected the muscle from overcontraction. This view changed when it was demonstrated that individual tendon organs in cat soleus muscle can be excited by the contraction of just four motor units (Houk and Henneman, 1967). Forces in the range of 4.5-22 mg (Fukami, 1981) can initiate an action potential in an isolated tendon organ.

Tendon organs are very sensitive to active muscle contraction but they are not sensitive to a passive force applied to the muscle. About 65% of the tendon organs in cat hindlimb muscles do not fire when the muscles are passively stretched within their physiological range. The discharge frequency of the remainder does not change with further muscle extension (Houk *et al.*, 1971). Although, like muscle spindles, an isolated tendon organ can be stimulated by stretching (Fukami and Wilkinson, 1977) this response is not observed *in situ*. It is thought that when passively stretched *in situ* most of the tension is taken up by the in-parallel connective tissues within the same muscle.

Above a certain threshold, an isolated tendon organ tends to fire linearly as the hold force, during a ramp-and-hold stretch, is increased (Fukami and Wilkinson, 1977). However, when the isolated tendon organ is stimulated by the contraction of its in-series muscle fibres (Fukami, 1981), the linearity disappears once the contractile force is greater than 1 N, after which the slope declines. The behavior of tendon organs *in situ* is more complicated. It has been shown that stimulating an additional motor unit sometimes reduces the discharge frequency of a previously activated tendon organ (Horcholle-Bossavit *et al.*, 1989*a*). In studies of the maximal tetanic contraction of motor units, it is not uncommon to see a higher discharge frequency in small weak motor units rather than large strong units activating the same tendon organ (Jami and Petit, 1976; Gregory and Proske, 1979; Binder, 1981). Apart from the complex anatomical arrangement between motor units and tendon organs, the discrepancy in the response characteristics between the isolated and *in situ* receptors could be partly accounted for by the difficulty in accurately measuring forces acting on individual tendon organs in a muscle (Jami, 1992).

It is now clear that tendon organs not only provide information about forceful muscle contraction that may harm the muscle but also about a wide range of muscle contractile forces. The discharge frequency of tendon organs increases as the contractile force is increased (Crago *et al.*, 1982; Horcholle-Bossavit *et al.*, 1989*a*), although the response is not necessarily linear. In addition, the receptors have been shown to be extremely sensitive to a sinusoidal pattern of motor unit contraction up to 40 Hz (reviewed by Jami, 1992). It is likely that the discharge of tendon organs may be more important in signaling changes in muscle contraction than in recording a steady level (Rothwell, 1994).

Roles in mastication – The presence of tendon organs in jaw closing muscles and digastric muscles has not been confirmed. Even in those studies which have deduced their existence there has been no evidence regarding their role in mastication. Tendon organs in jaw muscles, if any, may be important for detecting the contraction force of individual muscles.

OTHER MUSCLE AFFERENTS

Structure – Non-spindle group II (diameter 4-12 μ; conduction velocity 24-72 m/s) and group III (diameter 1-4 μ; conduction velocity 6-24 m/s) muscle afferents supply mechanoreceptors in limb muscles. Although group II afferents are largely studied in the secondary endings of muscle spindles, they supply some Paciniform corpuscles (smaller and more elongated than typical Pacinian corpuslces) in the musculo-tendinous junction (Stacey, 1969) and some free nerve endings (Hník and Payne, 1965 [rats]). On the other hand, group III afferents seem only to terminate as free nerve endings. It should be noted that free nerve endings are the most numerous type of receptors in a skeletal muscle (Matthews, 1972).

Neurophysiology – The thresholds for electrical stimulation are about six times larger in group II and up to 24 times larger in group III (Eccles and Lundberg, 1959*b*) compared to that in group I afferents. They are likely to be excited by a large muscle stretch. Group III afferents have been shown to respond to punctate pressure applied to a restricted zone, often close to musculo-tendinous junctions in hind limb muscles (Paintal, 1960). They are also excited by muscle contraction (Paintal, 1960; Knifki *et al.*, 1981) although this was not observed in the neck muscle (*biventer cervicis*; Abrahams *et al.*, 1984).

Electrical stimulation of both group II and III muscle afferents generally excites ipsilateral flexors and inhibits ipsilateral extensors (Eccles and Lundberg, 1959*a*). The reflex is polysynaptic and has both short- and long-latencies (Jankowska *et al.*, 1967). The pattern of reflex response could, however, differ between different preparations being used in the study. For example, stimulating group II afferents instead inhibits flexor muscles in decerebrate cats with lower pontine lesions (Holmqvist and Lundberg, 1961). This suggests that there may be more than one reflex pathway from group II and III afferents to the motoneurons.

Non-spindle group II and group III afferents are thought to be involved in the "clasp-knife reflex." The reflex is best seen clinically in the knee extensor muscles of spastic patients. As the knee is flexed, there is a gradual increase in the resistance to the movement. Up to a certain level of flexion the resistance melts away. Beyond this point the reflex contraction due to the stretch in the knee extensor may be suddenly inhibited by the excitation of group II and III afferents.

Roles in mastication – Surprisingly, non-spindle group II and group III muscle afferents have never been studied in jaw muscles. If they exist, they would be suited to detect a high tension developed in the muscle and probably involved in protective reflexes.

MECHANORECEPTORS IN FACIAL SKIN AND ORAL MUCOSA

Structure – There are more than ten types of mechanoreceptors, judged by their physiological responses, in cat hairy and glabrous skin (Horch *et al.*, 1977). The simplest form of skin mechanoreceptor has tuft-like "penicillate endings" (Cauna, 1973). They are derived from unmyelinated nerves and are most commonly found in the dermis. Although penicillate endings exist in primates, they are unlikely to be mechanoreceptors since small unmyelinated nerve fibres (C fibres) in primates are nociceptive. Four other common types of mechanoreceptors are generally observed in primate skin, namely Merkel-cell neurite complexes, Ruffini endings, Meissner corpuscles and Pacinian corpuscles.

Merkel-cell neurite complexes are found within the stratum granulosum of the epidermis (Munger, 1971). The receptor consists of a nerve terminal (neurite) close to a specialized type of epithelial cell, a Merkel cell. Accumulations of Merkel-cell neurite complexes are found in the hairy skin known as touch domes (Iggo and Muir, 1969). However, a similar pattern is also found in glabrous (non hairy) skin (Dubner *et al.*, 1978).

Ruffini endings are derived from terminals of large myelinated fibres that are closely associated with collagen fibrils in the dermis. The endings are surrounded by a fluid-filled space and enclosed by a thin lamellated capsule.

Meissner corpuscles are found in the dermal papillae of glabrous skin. The corpuscle is oriented perpendicular to the surface of the skin. It can be innervated by more than one axon. Within the corpuscle the nerves lose their myelin and course tortuously through the cytoplasm of specialized lamella cells.

Pacinian corpuscles are onion-like structures found deep in the subdermal region of the skin. They are the largest receptors in skin. A single axon terminates in concentric lamellae. The lamellar structure is believed to prevent responses from slowly changing stimuli.

Hair mechanoreceptors play a very important part in skin proprioception such as detecting movement in the surrounding air. Thin body hairs in cats and primates (so called "down hairs") are innervated by small myelinated nerves (A-δ) and the receptors are presumably free nerve endings (Burgess and Perl, 1973). Thick and stiff hairs (so called "guard hairs") are innervated by A-β fibres that terminate as lanceolate or palisade endings. This specialized ending consists of a nerve terminal sandwiched between two flattened Schwann cells parallel to the hair shaft (Munger, 1971; Andres and von During, 1973). A single nerve usually innervates several hairs.

"Vibrissae or sinus hairs" are more complex sensory structures located in the perioral region of most mammals. They play a major role in tactile exploration. About one-third of all the trigeminal cutaneous afferents supply them (Burgess and Perl, 1973). The sinus hair follicles in rats and monkeys are innervated by several types of skin mechanoreceptors including Merkel-cell neurite complexes, Ruffini endings, lanceolate endings, Golgi-Mazzoni lamellated corpuscles and free nerve endings (Andres and von During, 1973).

The structure of mechanoreceptors in oral mucosa is probably comparable to that in glabrous skin where Meissner's corpuscles (Darian-Smith, 1973) and Pacinian corpuscles (Horch *et al.*, 1977) are abundant. Four types of mechanoreceptor have been detected in the cat lingual gingiva and alveolar mucosa including organized endings (both encapsulated and unencapsulated), Merkel cell-neurite complexes, intra-epithelial nerve endings (Sakada, 1981). Cat oral mucosa contains many different organized nerve endings ranging from type I (simple Paciniform) to type V (Meissner corpuscle) according to Malinovsky's (1966) classification. Merkel-cell neurite complexes are found in the epithelium of cat alveolar mucosa whereas other encapsulated and unencapsulated endings are found in the lamina propria. Intra-epithelial endings are also found in the gingiva and alveolar mucosa. Free nerve endings are found in the lamina propria and submucosa. They branch from both myelinated and unmyelinated fibres. The number of corpuscular endings such as Meissner corpuscles and Krause end-bulbs increases as one passes from the hairy facial skin to the mucocutaneous zone and to the oral mucosa (Darian-Smith, 1973).

Primary afferents from skin and vibrissae mechanoreceptors in the orofacial region have cell bodies in the trigeminal ganglion and terminate in all of the trigeminal spinal tract nuclei in a somatotopic manner (inverted dorsoventrally – Darian-Smith, 1973). The axons of nerves supplying complex corpuscular receptors in the oral mucosa, such as Meissner corpuscles, have usually been shown to terminate in the main sensory nucleus (reviewed by Capra, 1995).

Three types of neurones have been identified in the trigeminal nuclei when facial skin is stimulated, namely nociceptive-specific (NS), wide dynamic range (WDR) and low-threshold mechanosensitive (LTM) (Sessle, 1987). The NS neurones only respond to noxious whereas the WDR neurones respond to stimuli from light touch to noxious. The LTM neurones can only be activated by light touch applied to skin, mucosa or teeth. WDR neurones may play an important role in a pain gating mechanism.

Many mechanoreceptor neurons in the trigeminal spinal nuclei, including most of the nucleus oralis and interpolaris neurons, project to the contralateral ventroposteromedial (VPM) nucleus in the thalamus (Eisenman *et al.*, 1963; Rowe and Sessle, 1968; Darian-Smith, 1973). The projections from facial skin, hair and vibrissae terminate laterally in the VPM (Capra, 1995). Some of the neurons in the nucleus interpolaris also project to the cerebellum (Darian-Smith and Phillips, 1964). VPM neurons in cats send their projections from the thalamus to all somato-sensory cortical areas (SI, SII and SIII), half of them to SI (Darian-Smith, 1966).

Neurophysiology – Based on a study of mechanoreceptors in the glabrous skin of the hand (Westling, 1986), two major groups of nerve endings respond to mechanical stimuli, slowly (SA) and rapidly or fast (FA) adapting units. Slowly adapting units respond well to the magnitude of skin displacement when a static force is applied to the skin whereas fast adapting units respond well to the velocity of displacement. Both groups are further subdivided, based on the size of their receptive field, into type I (large phasic response with a small receptive field of about 13 mm^2 in skin) and type II (large tonic response with a large receptive field). The difference between these two subgroups could be, in part, due to the properties of the tissue in which they are invested and also their position in the tissue.

SA-II units fire regularly but SA-I units fire more irregularly under a static displacement. Unlike the SA-I units, the SA-II units are directionally sensitive and sensitive to lateral skin displacement. They fire maximally when a particular direction of stretch is applied and decrease the discharge when the force is applied in the opposite direction. At rest SA-II units also fire at 10-20 Hz. The threshold skin indentation is about 50 μ for SA-I units and 250 μ for SA-II units (Westling, 1986).

FA-I and FA-II units respond primarily to the velocity of stretch in the skin. Both have thresholds of about 10 μ. FA-I units can follow a sinusoidal stretch up to 100 Hz whereas the FA-II up to 400 Hz (Rothwell, 1994).

The physiological responses of skin mechanoreceptors can be related to structures of each subgroup by staining areas marked in the specimen at the end of a neurophysiological experiment and studying them under the microscope. SA-I units are shown to be closely associated with Merkel-cell neurite complexes (Iggo and Muir, 1969) and SA-II units are associated with Ruffini endings (Chambers *et al.*, 1972). FA-I units correspond to Meissner corpuscles and FA-II units correspond to Pacinian corpuscles (Westling, 1986).

The properties of mechanoreceptors in the oral mucosa can be deduced from studies of the receptors in glabrous skin. However, it must be kept in mind that the response characteristics of the same types of receptors can be different between two different tissues depending on the properties of the surrounding tissue (Sakada, 1983). Encapsulated and unencapsulated organized endings in the cat oral mucosa are rapidly adapting mechanoreceptors (Sakada, 1981). Type I receptors, simple Paciniform, respond well to vibratory stimuli up to 356 Hz. Merkel cell-neurite complexes are slowly adapting but probably also act as low frequency vibration detectors. Intra-epithelial endings are likely to be velocity or acceleration detectors. Free nerve endings in the oral mucosa are presumably nociceptors but some may be slowly or rapidly adapting mechanoreceptors similar to those in the cat periosteum (Sakada and Taguchi, 1971).

When a microelectrode is used to apply an electrical stimulus to a specific type of mechanoreceptor in the digits subjects usually report similar sensations. For example, stimulating FA-I units gives a "flutter" sensation and stimulating SA-I units gives a sensation of "pressure." The only exception is associated with stimulating SA-II units for which no clear sensation is felt (Gandevia and Burke, 1992). It is also found that the probability of evoking sensations is higher when stimulating the distal, as opposed to the proximal, part of the digit.

Hair mechanoreceptors are generally sensitive to rapid movement of the hair. Receptors in guard hairs can be subdivided into two groups, primary and secondary. The primary guard receptors only respond to rapid movement of the hair, both away from and toward the rest position. The secondary guard receptors only respond to slow movement of the hair. Receptors in down hairs best respond to slow movement of the hair but also show position sensitivity. Their displacement threshold is very low (Burgess and Perl, 1973). Receptors in the vibrissae of cats and monkeys are associated with both position (SA-I and SA-II) and velocity (FA) detection. The FA responses (velocity detection) may be associated with rapidly adapting free nerve endings or straight lanceolate endings (Dubner *et al.*, 1978).

Roles in mastication – Besides their role in detecting tactile stimuli such as touch and pressure, orofacial skin mechanoreceptors may help to sense position as elsewhere in the body (Edin and Abbs, 1991), in this case the position of the mandible. Ruffini endings are presumed to be responsible for this task since they are very sensitive to lateral stretch of the skin. There are no experiments studying the effect on the sense of jaw position of removing the input from facial skin receptors, probably due to technical difficulties.

Mechanoreceptors in the oral mucosa, together with the tongue and probably teeth (Landt, 1976*b*), play an important role in identifying the size and form of objects placed in the mouth, so called "oral stereognosis" (Landt, 1983; Garrett *et al.*, 1994). Without using teeth the ability to identify test pieces in the mouth is reduced (Landt, 1976*b*). A significant correlation between bite force discrimination and oral stereognosis (Williams and LaPointe, 1972) suggests the possible role of periodontal receptors in both tasks. On the other hand, Garrett and co-workers (1994) found no statistical difference between oral stereognosis in dentate and edentulous subjects, implying that periodontal inputs are not necessary. Oral stereognosis seems to be age-dependent, being more accurate and faster in children and young adults (Landt, 1983) but this was not found in a more recent study in which stereognostic ability remained good up to age 80 (Calhoun *et al.*, 1992).

Inside the mouth, the sensitivity to tactile stimuli (100 mN) tested by von Frey filaments is found to be greatest at the tip of the tongue and on the hard palate (Henkin and Banks, 1967). The tip of the tongue also has the greatest sensitivity to two-point discrimination (1.7 mm). Lips and the midline structures are more sensitive than lateral structures (Ringel, 1970). The two-point discrimination ability on the upper lip and cheek is reduced with age whereas that on the palate and tongue is not affected (Calhoun *et al.*, 1992). It is interesting to note that cutaneous receptors in the cheek are inevitably activated whenever the receptors in the cheek oral mucosa are activated since the two tissues are continuous with each other. The intra-oral receptors in the mucosa could be involved in detecting jaw position and the extra-oral receptors in the skin could be involved in oral stereognosis.

In completely edentulous patients, dentures are supported by gingival mucosa. The ability to detect bite forces on the denture may, in part, involve mechanoreceptors in the mucosa although their tactile threshold is much larger than periodontal receptors (1.25 N – Manly *et al.*, 1952). It is unlikely that residual periodontal receptors detect bite forces in denture wearers since no response to mechanical stimulation of the overlying mucosa over the extraction site seem to have been recorded in the mesencephalic nucleus after a tooth has been extracted (Linden and Scott, 1989*a*).

A tactile stimulus (a puff of air) over the skin around the mouth and over the oral mucosa (palate, tongue) during tooth clenching can elicit a powerful inhibitory effect on masseter and temporalis muscles in man (Di Francesco *et al.*, 1986). Similar responses (Yemm, 1972*a*) as well as the jaw opening reflex (Yemm, 1972*b*) have been shown when the oral mucosa is electrically stimulated.

During chewing about 60% of hair afferents in the facial area, most of which are near the corner of the mouth, are excited in rabbits (Appenteng *et al.*, 1982). The discharge frequency is dependent on the speed of jaw movement. Only the skin receptors near the corner of the mouth fire phasically during jaw movement. Passively moving the lower jaw of the cat, both opening and closing, excites neurons in the nucleus interpolaris that have receptive fields in the skin around the angle of the jaw and chin (Ro and Capra, 1995). These neurons probably encode the velocity of jaw movement.

PERIOSTEAL MECHANORECEPTORS

Structure – Free nerve endings are numerous in the periosteum of the rat mandible (Weddell and Harpman, 1940), human embryo orbital wall (Nozaki, 1955), dog mandibular alveolus (Tokumitsu, 1959) and the rat condylar region (Bernick, 1962). The nerves which supply the free endings are either myelinated (1-10 μ) or unmyelinated (Sakada and Maeda, 1967*a*). Some organized endings (both unencapsulated and encapsulated) have also been shown in the cat periosteum in the mental region of the mandible (Sakada and Maeda, 1967*a*; Sakada, 1971). Most encapsulated endings in the periosteum are composed of one corpuscle and one nerve terminal so-called Golgi-Mazzoni corpuscles (Sakada, 1971). The endings are branched from small myelinated nerves (1.6-4.8 μ). A large number of Pacinian corpuscles have been found in the periosteum (Carpenter, 1976). Aro *et al.* (1982, 1985) have shown a plexus of Pacinian corpuscles surrounding the distal end of the rat fibulae.

Neurophysiology – The properties of periosteal receptors have been studied by applying small mechanical stimuli to the overlying oral mucosa (Sakada, 1983) while recording in the nerve supplying the mandibular periosteum. The unencapsulated organized endings are slowly adapting. They fire irregularly during the application of a static force and the discharge frequency increases as the force is increased, like the SA-I units in skin. Encapsulated endings adapt rapidly and respond to vibratory stimuli as high as 400 Hz. The thresholds for pressure stimulation range from 10-50 μ of mucosa indentation.

Apart from nociceptive free nerve endings, many of those in the periosteum are classified as mechanoreceptors having either slowly or rapidly adapting responses. The rapidly adapting free nerve endings respond to vibratory stimulation up to 313 Hz (Sakada and Onoe, 1971*b*) whereas the slowly adapting units respond tonically to a static force and also to low frequency vibration.

Roles in mastication – During chewing food the mandible is distorted about the long axis of the body of the mandible (Hylander, 1984; Korioth and Hannam, 1994). Based on a finite element analysis the magnitude of deformation ranges from 0.46-1.06 mm during maximum clenches (Korioth and Hannam, 1994). The amount of jaw deformation seems sufficient to activate periosteal mechanoreceptors in the periosteum, although this has not been proved.

Studies of jaw reflexes in complete denture patients can give insight about the roles of both periosteal and mucosal mechanoreceptors. Studies of implant patients exclude inputs from oral mucosa and may be used to study the role of periosteal mechanoreceptors. It has been shown that implant patients are still able to detect forces applied to the implant although the threshold is larger than that for natural teeth, indicating that periosteal receptors may also be used to detect bite forces (Jacobs and van Steenberghe, 1993).

Pacinian corpuscles in the periosteum of a long bone may be used to detect tension along the bone surface and help bone healing after fracture (Aro *et al.*, 1982, 1985). A rigid fixation of the tibia is associated with an atrophic nonunion of a fibular fracture (Aro *et al.*, 1982). The fixation of the bone prevents the fibula from bending. This, in turn, prevents the activation of periosteal mechanoreceptors when the leg is loaded and the fracture does not heal. In a later experiment, Aro *et al.* (1985) removed about 8 mm of periosteum from the distal end of the fibula and a fracture was created in the middle shaft region. Deprivation of periosteal receptors resulted in non-union at the fracture site.

PULPAL MECHANORECEPTORS

Structure – Only free nerve endings have been found in tooth pulps. Most nerves supplying the pulp enter the tooth through the apical foramen and some through accessory foramina. Pulpal nerves branch little in the root canals (Byers and Matthews, 1981) but more extensively within the pulp chamber, especially near the pulp-dentine junction, to form the subodontoblastic plexus of Rashkow. Fibres from this plexus end either in the odontoblastic layer, predentine or dentine (Gunji, 1982). A considerable number reach the predentine and some extend as far as 0.2 mm into dentinal tubules (Byers and Kish, 1976; Lilja, 1979). Dentinal nerves are most frequently found near pulp horns (Fearnhead, 1957; Arwill, 1967; Lilja, 1979; Holland, 1981). Branches from one nerve can enter up to 100 tubules (Bradley, 1995). It is difficult to demonstrate nerves inside the tubules because they are so inaccessible to fixing solutions. This can be solved, to some extent, by fixing the animal intravenously before tooth extraction. For obvious reasons this is not possible in human studies.

The majority of pulpal nerves are unmyelinated near the apical foramen (Byers, 1984). The percentage of unmyelinated fibres increases inside the tooth due to axon branching. The number of myelinated fibres increases as the tooth matures (Johnsen *et al.*, 1983), probably because more bare nerves become myelinated. The size of myelinated fibres ranges from 1-4 μ, consistent with small diameter cutaneous A-δ fibres. The diameter of the unmyelinated fibres ranges from 0.2-1 μ consistent with cutaneous C-fibres. Both are commonly known to be nociceptive fibres. A separate group of large diameter A-β fibres has been found and may be involved in a non-nociceptive pathway (Holland and Robinson, 1983; Byers, 1984). A histochemical study on the effect of sympathectomy has shown that only a small number of unmyelinated fibres in the pulp are sympathetic nerves (Fehér *et al.*, 1979). These motor fibres are close to blood vessels and presumably regulate pulpal blood flow.

Pulpal nerves have cell bodies in the trigeminal ganglion and their central projections have been found in all trigeminal spinal nuclei (Nord, 1976; Sessle and Greenwood, 1976; Lisney, 1978; Arvidsson and Gobel, 1981; Hayashi *et al.*, 1984), even in the mesencephalic nucleus (Chiego *et al.*, 1980). Dong *et al.* (1990) demonstrated a short latency response (presumably due to a monosynaptic connection) from tooth pulp A-β afferents to some trigeminal nuclei neurons.

Neurophysiology – The tooth pulp has been widely used as a model for studies of pain. Most of these studies have been directed towards understanding tooth pain. It has not been unequivocally established how a stimulus activates pulpal nerves. Three mechanisms have been proposed:

(1) Direct contact between the stimulus and nerves inside the dentinal tubules initiates an action potential.

(2) Odontoblast processes are first excited and action potentials from them are transferred to the nerves via special cellular connections.

(3) Nerves either in dentine, in predentine or around the odontoblasts are activated by fluid moving through the dentinal tubules.

The finding that dentinal nerves only extend as far as the inner third of dentine may rule out Mechanism (1). Furthermore, many substances which cause pain when applied to skin do not cause pain when applied to outer dentine (Anderson and Naylor, 1962; Brännström, 1962; Dellow and Roberts, 1966).

Although histological studies have shown a close approximation between nerve-like and odontoblast-like structures in dentinal tubules (Frank *et al.*, 1972; Byers, 1984; Holland, 1985), the following observations seem to be contrary to the requirements of Mechanism (2): (a) No cholinergic activity is found in odontoblasts (Ten Cate and Shelton, 1966); (b) the resting membrane potential difference of odontoblasts is too low to be excitable (25-45 mV in mouse odontoblasts [Kroeger *et al.*, 1961] compared to 70 mV in nerve cells); (c) scraping the surface of the dentine still causes pain even when the odontoblast layer has apparently degenerated (Brännström and Aström, 1972).

The view proposed in Mechanism (3), that fluid moving through dentine activates pulpal nerves, is most widely accepted, the so-called "hydrodynamic mechanism of dentine sensitivity" (Brännström, 1963). The hypothesis is supported by the observation that most stimuli which evoke tooth pain also cause fluid to move through dentinal tubules.

C and A-δ sensory nerve fibres in the pulps of cats (Beasley and Holland, 1978; Lisney, 1978; Cadden *et al.*, 1983; Virtanen *et al.*, 1983), dogs (Wagers and Smith, 1960; Matthews, 1977; Närhi and Hirvonen, 1987) and man (Graf and Bjorlin, 1951; Matthews *et al.*, 1959; Reader and Foreman, 1981) have conduction velocities of < 2 m/s and between 2 and 30 m/s respectively (Lisney, 1978; Närhi and Haegerstam, 1983). Both are thought to exclusively mediate noxious sensations and contribute to pain sensation, as elsewhere in the body (Mumford and Bowsher, 1976; Sessle, 1987). A-β sensory nerves with conduction velocities greater than 30 m/s (Cadden *et al.*, 1983; Dong *et al.*, 1985, 1990) have also been described in pulps. It has been suggested that both A-β and A-δ might belong to the same functional group since they respond only to hydrodynamic stimuli whereas C-fibres do not (Närhi *et al.*, 1982, 1983). In addition, the threshold of electrical stimulation for the pulp-induced jaw opening reflex is close to that required to activate A-fibres, not C-fibres (Närhi *et al.*, 1983), suggesting that C-fibres are not involved in the reflex. The functional difference between C and A-fibres is further indicated by the finding that histamine or bradykinin applied to the pulp induce activity in C-fibres but not A-δ fibres (Närhi *et al.*, 1984; Närhi, 1985*a*). It was concluded that C-fibres only respond to stimuli that reach the pulp whereas A-fibres respond to stimuli that move fluid through the dentine.

The role of pulpal nerves as mechanoreceptors is not well established but the following findings tend to indicate their existence.

(1) A light touch applied to a paper thin layer of dentine overlying the pulp in cats (Scott and Tempel, 1963) or applied directly to superficial pulpal tissue in dogs (Närhi *et al.*, 1982) evokes a burst of discharges from pulpal nerves. These are presumably A-fibres.

(2) Some pulpal receptors in cats respond to small transient forces, 10-60 mN, rapidly applied to the crown of the tooth (Dong *et al.*, 1985). These mechanoreceptors are not directional sensitive and, unlike periodontal mechanoreceptors, only discharge a few spikes during the ramp phase of a transient force, indicating they are rapidly adapting. They are also very sensitive to vibratory stimuli applied to the tooth surface.

(3) A specially designed piston was used to aply forces (30-150 N) to the canine of a cat without moving it (Olgart *et al.*, 1988). This elicited activity from intradental A-fibres recorded inside the pulp. The receptors fired only at the onset and release of the force and their rate of discharge did not seem to increase as the load was increased. An excitation in the digastric muscle was also observed when forces larger than 60 N were used. Removing the coronal pulp or cooling the crown with ethyl chloride abolished the response. The authors concluded that the impulses and the withdrawal reflex might have been mediated via pulpal A-fibres stimulated by tooth deformation. However, it is not known from animal studies whether pain was involved.

(4) Lesioning nucleus oralis and nucleus interpolaris in cats causes cell degeneration within the cerebellum (Carpenter and Hanna, 1961) but none or very few of the cerebellar neurons die after destroying nucleus caudalis (Stewart and King, 1963). The lack of caudalis projections has also been shown by injecting HRP into the cerebellum (Steindler, 1977 [mice]; Watson and Switzer, 1978 [rats]). Since no pain pathways project to the cerebellum, some of the projections from pulpal nerves to the neurons in

the more rostral trigeminal spinal nuclei (Stephan, 1976; Cairns *et al.*, 1995) may be proprioceptive.

(5) The cortical potentials evoked by electrically stimulating pulps at threshold levels were normal in a patient with congenital absence of pain sensitivity (Manfredi *et al.*, 1981). This implies the existence of a non-nociceptive connection between tooth pulp and the cerebral cortex.

(6) Similarities between cornea and tooth pulp have cast doubt whether only pain can be sensed by the pulp. It has been demonstrated that although the cornea contains only free nerve endings it can differentiate between nociceptive, thermal and mechanical stimuli (Lele and Weddell, 1956, 1959).

(7) It has been shown that many of the neurones in the rostral nuclei that respond to pulpal electrical stimulation have low, non-noxious thresholds (Stephan, 1976; Cairns *et al.*, 1995). Unlike caudalis neurones, oralis neurones are not excited by the application of substance P (Henry *et al.*, 1980), suggesting they are probably not involved in pain transmission.

All of these findings tend to suggest that not all pulpal receptors are pain receptors but that some may have a non-nociceptive role and may be used in detecting forces applied to the tooth. The neural pathway is probably mediated via rostral trigeminal nuclei.

Roles in mastication – It has been widely accepted that intradental receptors are exclusively involved in the sensation of pain whereas their role in the control of mastication is far from being accepted.

The tactile threshold to mechanical force (10-45 mN) is found to be higher in pulpless teeth and in teeth covered by a metal cap than in vital teeth. (Loewenstein and Rathkamp, 1955). It has been argued that the difference may have been due to degeneration of some periodontal receptors around the apices of dead teeth (Linden, 1975). However, the periodontal ligament would not have been affected by covering the crown with a metal cap. The cap would, however, spread the load over the tooth crown. Pulpal nerves, sensitive to deformation of the crown, could have been involved in the detection of force.

A "pre-pain" sensation has been commonly reported when teeth are electrically stimulated at threshold levels (Hannam *et al.*, 1974; Matthews *et al.*, 1976; Brown *et al.*, 1985; Mason *et al.*, 1985). The sensation, described as "tingling," "cold," or "warm" (Mumford and Bowsher, 1976) may be mediated by non-nociceptive nerves inside the pulp. It is not known whether pain and pre-pain arise from the same group of pulpal nerves or whether they are two separate modalities. The former view seems to be supported by the fact that stimulating two teeth at a pre-pain level results in pain, not stronger pre-pain (Brown *et al.*, 1985). The latter view cannot be ruled out, however, since increasing the frequency of stimulation at pre-pain level does not alter the quality of the sensation (Matthews *et al.*, 1976). It has further been shown that stimulating human teeth at this pre-pain level evokes an inhibitory effect in the masseter muscle (Matthews *et al.*, 1976; McGrath *et al.*, 1981; Sharav *et al.*, 1982). Apparently this effect is not related to a nerve pathway associated with pain.

Periodontal receptors are thought to be responsible for the discomfort, or even pain, that is often felt when unexpectedly chewing on a piece of bone or metal. In fact, the force on the tooth at the moment the foreign body is encountered is not different from that used while chewing normally. Many studies of maximum bite force have shown that pain is not felt when teeth are subjected to forces much larger than those which would have been used while chewing food. A possible explanation for the pain is that biting on something hard, compared with something soft, causes pain because it suddenly increases the stress on the crown, not the force on the periodontal ligament. This indicates an association between occlusal stress and pulpal nerve activation.

Byers (1996) observed more CGRP (calcitonin gene-related peptides), a neuropeptide released by small sensory nerves, in functional teeth than in unused teeth of rats. The finding suggests a relationship between activity in pulpal sensory nerves and occlusal loads. Byers speculated that occlusal forces moved dentinal fluid and activated sensory nerves inside the tooth. This caused the release of CGRP which may have induced secondary dentine formation.

Some pulpal sensory nerves may play an important role in regulating microcirculation inside the pulp (reviewed by Olgart, 1996). The release of vasoactive neuropeptides increases blood flow and may be used to counteract the invasion of external noxious stimuli (Matthews and Vongsavan, 1994). However, the response seems to be associated with tooth pain and therefore mediated by nociceptive nerve fibres.

PULPAL MECHANORECEPTION FROM EVOLUTIONARY PERSPECTIVES

Bone and dentine were probably the first calcified tissue to have been evolved (Orvig, 1951). It was first seen in nodules covering the bony armour of ostracoderms (Ordovician jawless fish). Each nodule consisted of tubular dentine and was supported by bone. It has been suggested that the tubules might have been used to monitor changes in osmotic pressure of the water (Lumsden, 1981), thereby enabling fish to adjust their excretion of salts when migrating from salt water to fresh water habitats. It cannot be known whether the sensory information from the dentine was related to pain.

Osborn and Hillman (1979) studied the teeth of some mammal-like reptiles and early mammals from the Mesozoic. They observed a progressive increase in the number of enamel tubules as the animals acquired more precise occlusions. If the function of the tubular enamel was similar to that of dentine, it would imply more fluid movement in association with more complex mastication. It is unlikely that this would mean that teeth became more prone to pain.

The numerous fluid filled tubules in orthodentine could be interpreted as a uniquely designed sensor for detecting pressures and forces inside a hard tissue. Forces applied to the hard tissue move fluid through the tubules. The movement is detected by pulpal nerves. Thus mechanoreceptors in the pulp may be used to detect the amount and direction of occlusal forces on individual cusps (Osborn and Ten Cate, 1976). This sensory input probably enabled the small mammal-like reptiles and mammals that originally evolved an occlusion to correctly interdigitate the tiny cusps and ridges of their newly evolved teeth.

EFFECT OF FORCES ON TEETH AND SUPPORTING TISSUES

Most teeth consist of two layers of calcified tissue encasing a soft tissue pulp. Enamel covers coronal dentine and cementum covers root dentine. The root of the tooth is embedded in alveolar bone with the periodontal ligament between.

Enamel is formed by ameloblasts. It consists about 96% by weight mineral, 3% water and less than 1% organic material, making it the hardest tissue in the body. The hydroxyapatite crystals in enamel are oriented in a regular pattern, resulting in a prismatic appearance under the light microscope. Enamel prisms (or enamel rods) bend from side to side during their courses from the enamel-dentine junction toward the surface of the tooth. Due to this complex arrangement enamel is likely to be anisotropic (*i.e.* having different mechanical properties between its two or three orthogonal axes – Rasmussen *et al.*, 1976; Yettram *et al.*, 1976; Spears *et al.*, 1993; Spears, 1997) although this property has been poorly studied. Enamel is less resistant to fracture between its prisms than across them (Rassmussen *et al.*, 1976) and is more resistant to compression down the long axes of the prisms (Yettram *et al.*, 1976; Spears *et al.*, 1993). The elastic modulus of enamel varies widely in different studies, presumably due to differences in the plane of measurement and the region being measured: 9.65 GPa at the side of a tooth (Stanford *et al.*, 1960), 84.1 GPa in the cuspal region (Craig *et al.*, 1961), 40.8 GPa (Thresher and Saito, 1973). Poisson's ratio for enamel is about 0.3 (Haines, 1968; Grenoble *et al.*, 1972).

Dentine is formed by odontoblasts and consists of 72% mineral, 10% water and 18% organic material. It is unique among vertebrate hard tissues because it contains numerous tubules. Each tubule is an unmineralized trail left behind by an odontoblast and contains an odontoblast process. The tubules have a diameter of about 4 μ near the pulp and taper down toward the enamel-dentine junction. Apart from odontoblast processes, dentinal tubules contain extracellular fluid and, near the predentine, a few collagen fibrils and nerve fibres. The organic matrix of dentine is mainly composed of collagen fibres, laid parallel to the tubules in the outermost (mantle) dentine and perpendicular to the tubules in the remaining (circumpulpal) dentine. Most of the hydroxyapatite crystals are oriented parallel with the fibres. Dentine, like enamel, is anisotropic. It fractures more easily across the orientation of the dentinal tubules along the orientation of collagen fibres (Rasmussen *et al.*, 1976) and is probably more resistant to compression in this direction. The elastic modulus of dentine also varies although somewhat less than that of enamel: 16.6 GPa (Craig and Peyton, 1958); 6.9 GPa (Standford *et al.*, 1960); 13.6 GPa (Thresher and Saito, 1973); 17.7-29.8 GPa (Kinney *et al.*, 1996). The elastic modulus is smaller near the pulp than near the enamel-dentine junction (Kinney *et al.*, 1996). Poisson's ratio for dentine is also about 0.3 (Haines, 1968; Grenoble *et al.*, 1972).

Cementum consists of about 65% mineral and more than 20% organic material, most of which is collagen. The hydroxyapatite crystals and collagen fibres in cementum are similar to those in bone. A very thin layer of cementum adjacent to the dentine does not contain fibres (afibrillar cementum) and is well mineralized. The mineral in the fibrillar cementum is usually associated with the collagen fibres. When cement is rapidly deposited some cementoblasts become trapped in the so-called "cellular cementum". Most mechanical properties of cementum have not been studied.

The periodontal ligament consists of cells, matrix, nerves, blood and lymph vessels. The matrix contains two groups of collagen fibres. The "principal" fibres are embedded in cement at one end and alveolar bone at their other end. They suspend the tooth in its socket. "Indifferent" fibres pack spaces and surround neurovascular bundles. There are a few oxytalan and elastin fibres. The remaining matrix is primarily composed of proteoglycans and water. The periodontal ligament is generally considered to have visco-elastic properties comparable to those of a "Voigt element" (Picton and Wills, 1978; Coehlo and Moxham, 1989) in which the elastic and damping components are in parallel. The elastic modulus of the periodontal ligament seems to vary along the root, being greatest in the upper third (Korioth and Hannam, 1994). Values range from 0.0025-

0.0032 GPa (see Korioth and Hannam, 1994). The width of the periodontal ligament is variable (approximately 0.2 mm), depending on age, the type of tooth and its functional state. Its thickness is increased in teeth receiving larger occlusal forces.

Jaw bone, as elsewhere in the body, is arranged in either compact layers or in a cancellous pattern. Compact bone contains a much larger proportion of mineralized tissue whereas cancellous bone contains much more soft connective tissue. Compact bone constitutes the cortex of the maxilla and the mandible. Most of it consists of numerous concentric lamellated structures (called Haversian systems). The trabeculae of cancellous bone consist of spicules of lamellated bone forming a three-dimensional frame. Some of the bone lining the alveolar socket is a modified form of compact bone called "bundle bone." It contains fewer matrix fibres than normal together with numerous periodontal fibres. The bone cells in compact bone are connected to the surrounding soft tissue by a network of canaliculi. It has been suggested that the trabeculae of the cancellous bone are oriented along the direction of the highest stress, passing from the alveolar socket to the angle of the mandible and up to the ramus (Seipel, 1948; Ralph and Caputo, 1975). The elastic modulus of cortical bone is 13.7 GPa and of cancellous bone is 7.9 GPa. (Carter and Hayes, 1977; Carter, 1978).

EFFECT OF LOADS ON TOOTH STRUCTURES

Stress can be studied in either intact or altered teeth. Altered teeth are used to study the effect of restorations on tooth integrity. Several methods have been used including photoelastic techniques (Johnson *et al.*, 1968; Hood, 1972; Fisher *et al.*, 1975), measurements of strain (Reeh *et al.*, 1989), measurements of cuspal flexion (Malcolm and Hood, 1977) and finite element analysis (De Vree *et al.*, 1983; Williams *et al.*, 1987). Cervical cavities may be deformed by an occlusal load (Hood, 1972). Removing tooth substance reduces the stiffness of a tooth subjected to a 100 N occlusal load (Grimaldi and Hood, 1973; Malcolm and Hood, 1977; Reeh *et al.*, 1989). The more tooth substance that is removed the greater the tooth stiffness is reduced, ranging from 20-45% reduction in class I cavities to 63-195% in three-surface class II cavities. The stiffness is greatly reduced when the marginal ridges of the tooth are cut. Surprisingly, the stiffness of the tooth after root canal treatment is only reduced by about 5% (Reeh *et al.*, 1989). Reduction in stiffness is usually calculated in comparison with intact teeth. Filling teeth can restore tooth stiffness. Restorations with cuspal coverage (*e.g.* tooth onlays) strengthen a tooth more than those without cuspal coverage (Malcolm and Hood, 1977). Fluid moves out of the pulp chamber when a tooth containing a three-surface (MOD) cavity is loaded *in vitro* (Lutz *et al.*, 1991). Less fluid is displaced after the tooth has been restored. Composite filling materials more effectively reduce distortion than amalgam.

Studies of intact teeth provide some evidence about the distribution of stress in teeth. Both two- and three-dimensional finite element analyses have been based on either isotropic or anisotropic enamel. In isotropic models, tooth enamel is stressed by an occlusal load approximately two (Goel *et al.*, 1990) to approximately 20 times (Rubin *et al.*, 1983) more than dentine. Most of the stress is passed through an outer shell of the enamel down to the cervical region of the tooth (Yettram *et al.*, 1976; Goel *et al.*, 1990; Spears *et al.*, 1993). The stress in dentine is greatest centrally when the load is applied uniformly over the occlusal surface (Goel *et al.*, 1990). In an anisotropic model the elastic modulus is assumed to be greater along the direction of prisms. In this case more stress is passed into the dentine, especially beneath the loading point, and less stress is developed in the cervical enamel (Spears *et al.*, 1993). Yettram *et al.* (1976), however, did not find any significant difference between isotropic and anisotropic models. The reason for this discrepancy is not clear. Stress distribution in intact teeth has not been studied by other techniques. Nevertheless, the deformation of an intact tooth under loads has been observed by Grimaldi and Hood (1973) and probably also by Reeh *et al.* (1989). The former study showed that buccal and lingual cusps in an intact premolar are deflected 11 μ by an occlusal load whereas the latter study compared the strain of altered teeth with intact teeth. Sakaguchi *et al.* (1991) recently measured a significant strain in the cusps of loaded intact premolars.

EFFECT OF LOADS ON THE PERIODONTAL LIGAMENT

Parfitt (1960) was the first to study the effect of bite forces on tooth displacement. He demonstrated that a human maxillary incisor is maximally intruded (about 30 μ) with a force of 10 N although there is little change with forces greater than 1 N. The tooth moves less when the force is applied more rapidly. Repeatedly loading the tooth with the same force every 2-5 seconds does not allow the tooth to return to its original position. This results in a progressive shift of the baseline. The tooth fully returns to its original position in 1-2 minutes. A similar pattern of tooth intrusion was later observed in premolars and molars (Picton, 1963*a*) although a larger force (~5 N) was required beyond which a tooth could only be slightly intruded.

When a load is applied, a tooth is at first rapidly intruded but slows down as it approaches the maximum value. When the load is removed, the tooth is at first rapidly extruded but slows down as it reaches its original position. Picton and Willis (1978) attributed this pattern to the viscoelasticity of the periodontal ligament. The ligament becomes more elastic, however, if a short burst of loads similar to that during chewing is applied (Moxham and Coehlo, 1989).

The lateral displacement of loaded teeth has been studied by applying smaller forces in a bucco-lingual direction. A 5-N force applied bucco-lingually to a monkey maxillary incisor for 2 seconds displaces the tooth more than 150 μ labio-lingually (Muhlemann, 1954*a*). The lateral displacement is least in teeth heavily used. A more accurate holographic technique measured human maxillary incisors displaced around 60 μ labially under a maintained 3-N force (Burstone *et al.*, 1978). As in the case of axial forces, the rate of loading affects the tooth mobility. Teeth are more mobile when the load is applied slowly (Lear *et al.*, 1972). Bucco-lingual tooth displacement is also increased when contact areas are removed (Muhlemann, 1954*b*). The pattern of tooth displacement with horizontal loads again suggests the periodontal ligament is viscoelastic (Muhlemann, 1954*a*; Picton, 1964*b*; Parfitt, 1961; Picton, 1965).

The question of how the periodontal ligament supports teeth, by tension of its fibres or by compression of its matrix, has not been resolved. According to Moxham and

Berkovitz (1995) the following evidence supports a tension mechanism. (1) There is no change in the intrusive mobility of a tooth after the apical half of its root has been removed (Picton and Picton, 1987). A compression mechanism would suggest an increase in mobility. (2) Fibrobasts in the periodontal ligament are elongated with thin processes, as opposed to being round (Merrilees and Flint, 1980). (3) Most of the proteoglycan in bovine periodontal ligament consists of dermatan sulphate (Pearson, 1982) which predominates in other connective tissues under tension (Gillard *et al.*, 1979). (4) The mineralization front of the embedded periodontal (Sharpey) fibres is roughly perpendicular to the long axis of the fibres, similar to that between tendon and bone (Jones and Boyde, 1972). (5) Periodontal fibres have a "crimped", wavy appearance, when no load is applied (Sloan, 1979*b*). On the other hand the following favour a compression mechanism. (1) Removal of the coronal one-third of the root periodontal ligament does not affect tooth intrusive mobility (Picton, 1988). (2) The small size (40-50 nm), and unimodal distribution of collagen fibrils (Berkovitz *et al.*, 1981; Svoboda *et al.*, 1983) with short periodicity (57 nm – Moxham, 1985) are characteristic of tissues under compression (Merrilees and Flint, 1980). (3) There is a large amount of ground substance in the periodontal ligament (~65% by volume – Berkovitz *et al.*, 1981) as opposed to 27% found in tissues under tension (Merrilees and Flint, 1980) (4) The "heavy green" staining of periodontal collagen fibres with Masson's trichrome suggests they are usually compressed (Flint *et al.*, 1975).

Picton (1989) suggested that both tension and compression might play a part in the axial tooth support. Under small loads (< 1 N) the tooth is primarily cushioned by the matrix. Under larger loads the tooth is primarily supported by tension in periodontal fibres.

EFFECT OF LOADS ON JAW BONES

It has been shown in the monkey that the alveolar crest is deformed and the tooth socket is dilated either by an axial load (as low as 1 N – Picton, 1965) or a horizontal load (Picton and Davies, 1967). They attributed the effect to compression of the periodontal ligament. The alveolar bone responds to heavy loads by increasing the number and thickness of the bony trabeculae.

Forces applied to teeth during chewing and isometric biting deform maxillary and mandibular bones (Hylander, 1977; Hylander, 1984; Hylander and Johnson, 1992; Korioth and Hannam, 1994; Herring *et al.*, 1996). The anterior part of the zygomatic arch is the most strained region of the upper jaw of monkeys during chewing (Hylander and Johnson, 1992). A photoelastic technique showed that the base of the maxilla, where fractures are frequently observed, is the most stressed region in the maxilla, (Lehman, 1972). Unilaterally clenching on mandibular premolars increases the transverse width of the posterior end of the dental arch (Koeck and Sander, 1978). Hylander (1979) studied the deformation of the mandible by using strain gauges bonded to the mandibular cortex of monkeys. The working mandibular corpus is twisted inwards (*i.e.* its upper border is displaced medially), bent either medially or laterally, whereas the balancing corpus is twisted inwards, bent upwards and also laterally [Twisting is measured in the frontal plane, bending is measured in either horizontal or sagittal plane]. The corpus on each side of the mandible is also bent upwards and twisted inwards during incisal biting. The amount of strain (especially compression) along the lateral surface of the mandibular cortex in the molar region is proportional to the magnitude of the molar bite force on the same side (Hylander, 1986) and may be used to estimate chewing forces.

Deformation of the human mandible has been tested mathematically by a three-dimensional finite element analysis involving different clenching tasks (Korioth and Hannam, 1994). The forces acting on the mandible were derived from experimental EMG data. During simulated incisor clenching the corpus and the ramus were twisted outwards (*i.e.* the lower border of the mandible was displaced medially). During simulated intercuspal clenching, both the corpus and ramus were twisted inwards. During simulated unilateral molar clenching, the working side of the mandible was twisted outwards whereas the balancing side was twisted inwards.

Differences in the pattern of jaw deformation between studies may be attributed to differences in the pattern of muscle activation. An inward twist is presumed to be due to stronger activity in the masseter muscle whereas an outward twist may be related to stronger activity in the medial pterygoid muscle.

ORAL MECHANORECEPTORS WHICH DETECT BITE FORCES

The point of force application, magnitude and direction of forces need to be monitored in order to properly adjust them and prevent damage to supporting structures. Information can be in the form of nociception or proprioception (including exteroception) or both. Too large a bite force might be harmful to the teeth and their supporting tissues, to jaw muscles or to the temporomandibular joints.

All the commonly known mechanoreceptors exist in the masticatory system, namely low-threshold free nerve endings in joint capsule and periosteum, Golgi tendon organs in skeletal muscles, Pacinian corpuscles, Meissner corpuscles, Merkel cell-neurite and Ruffini endings in skin. In addition the masticatory system accommodates teeth which contain nerves both inside (pulpal nerves) and around them (periodontal nerves).

Periodontal mechanoreceptors are thought to be the primary receptors for monitoring the magnitude (Hannam, 1970; Tabata and Karita, 1986) and direction (Hannam, 1982; Tabata and Karita, 1986; Trulsson *et al.*, 1992; Linden *et al.*, 1995) of forces applied to the tooth. However, forces which have been shown to initiate both jaw opening (negative feedback) and jaw closing (positive feedback) reflexes are in the order of 10 mN (~1 gm) to 10 N (~1 kg). These forces are much smaller than those used in chewing. A human premolar tooth is completely pushed into its socket by a 10 N force (Picton, 1963*a*). It is, therefore, unlikely that any more periodontal receptors could be activated by forces larger than 10 N. Consider a force of 40 N on a molar tooth, one which is commonly used during chewing (Anderson,1956*a, b*). Under such a condition all periodontal mechanoreceptors are presumably maximally activated and they would be unable to monitor changes in the chewing force. Additionally the periodontal ligament is more deformed (Muhlemann and Zander, 1954; Parfitt, 1960; Picton, 1963*a*), and therefore the nerves are more highly activated, by forces smaller than 1 N. The periodontal ligament is increasingly less sensitive as the teeth are subjected to larger forces, such as those used during chewing.

We believe that periodontal mechanoreceptors are more likely to be used to locate the point of force application and detect the direction rather than to measure the magnitude of bite forces. Trulsson and Johansson (1996) suggested that the receptors may be important in detecting the amount of light forces required to hold food between upper and lower teeth.

TMJ mechanoreceptors are unlikely to be involved in bite force detection. All of the TMJ receptors are located within the capsule and ligaments and none is observed on the articular surfaces nor in the disc where the load is concentrated.

Muscle spindles in jaw closing muscles detect changes in muscle length as well as jaw position. They fire during active, but not passive, jaw closure. This suggests that muscle spindle activity during jaw closure is secondary to the fusimotor drive. They are also active during an isometric bite (sustained at 10 N – Larson *et al.*, 1981*a*) but the response does not increase if the bite force is increased. This tends to refute a role for muscle spindles in gauging the magnitude of bite forces.

Tendon organs, if present, could play an important role in detecting the tension in jaw closing muscles during active muscle contraction and provide a distant mechanism in monitoring bite forces. It is unlikely that they could be used to detect the point of application, magnitude and direction of a bite force.

Non-spindle group II and group III muscle afferents, if they exist in jaw muscles, would probably be used to detect moderate to large tension in the jaw muscle during the production of a bite force and indirectly gauge the amount of bite forces. Like tendon organs, they could not detect the point of force application.

Skin and mucosal mechanoreceptors are unlikely to detect the magnitude of an isometric bite force. It is true, however, that a muscle belly becomes increasingly more bulky when the muscle contraction increases. This could create a shearing force between the muscle and the tissue beneath the skin or mucosa which then activates resident mechanoreceptors, possibly Ruffini endings.

Mechanoreceptors in the periosteum could be used to detect all the parameters of a bite force. By monitoring the torsion in each jaw, especially the mandible, they could be used to assess the magnitude of a bite force. The spatial pattern of the distortion caused by the torsion presumably depends on the location, magnitude and direction of the bite force. Linden (1978) found some mesencephalic neurons that respond to force applied on the palate and all upper teeth namely type P-mechanoreceptors (because they responded to stimulation of the palatine nerve) and suggested that these receptors are likely to be located in the cranial sutures. Mechanoreceptors in the periosteum, like type-P mechanoreceptors, may be used to monitor the deformation of the maxillary and circum-maxillary bones caused by bite forces. It has been shown that many cranial sutures including the mandibular symphysis (Hylander, 1984) (although it is not a suture in man) may be stretched or compressed during chewing (Hylander and Johnson, 1992; Herring *et al.*, 1996). However, it is not known if the deformation is sufficient to activate periosteal receptors *in vivo*.

If the bones of the jaw contain mechanoreceptors they could be used to gauge the magnitude of bite forces. The central canals of the Harversian systems in compact bone are an interconnected system of fluid-filled canals. Stresses on the bone squeeze fluid from compressed regions into tensile regions (reviewed by Duncan and Turner, 1995). The moving fluid shears across cell membranes and activates bone cells to produce mediators that initiate bone formation. It is also possible that the moving fluid could activate nerve endings in the bone, similar to those associated with the nerve plexus in the tooth. The magnitude and pattern of the movement could be used to monitor the forces applied to the bone.

Pulpal mechanoreceptors, if they exist, could be used to detect all parameters of a bite force. Unfortunately, very little is known about the response of pulpal nerves to forces comparable to those used in chewing. The only experiment in cats (Olgart *et al.*, 1988) has shown that intradental A-receptors fire when a canine tooth is squeezed, but not moved, by 30-150 N forces. The deformation of the tooth was thought to stimulate the pulpal receptors but no detailed mechanism was proposed. Nor is it known whether the stimulus caused pain.

From a theoretical point, Peleg (1980) suggested that a hard receptor, rather than a soft receptor, was required to detect the hardness of a hard object. In other words, a soft receptor cannot be used to detect the magnitude of a large force because the receptors themselves have been maximally deformed by a smaller force. Thus, the pulpal mechanoreceptors encased in a hard dental cover would be more suitable to detect a larger bite force than soft periodontal receptors.

In conclusion, periodontal mechanoreceptors are extremely sensitive for detecting small forces. They can be used to signal tooth contact and to locate the point of force application. They may also be used for detecting the direction of a bite force. For moderate to large bite forces, the periosteal, sutural and, perhaps, bone mechanoreceptors would be more effective sensors. Tendon organs and other muscle afferents provide information about the activity of individual jaw muscles rather than the magnitude of bite force. Finally, only pulpal mechanoreceptors could provide information about the forces acting on the separate cusps of a tooth, and about the stress on the tooth crown.

The following experiments were designed to test the hypothesis that tooth pulp may contain mechanoreceptors.

(1) Effect of Jaw Opening on the Direction and Magnitude of Human Incisal Bite Forces [published in *J Dent Res* (1997), **76**, 561-567]

(2) The Effect of Pressure on a Maximum Incisal Bite Force in Man [published in *Archs oral Biol* (1997), **42**, 11-17]

(3) Discrimination of Hardness by Human Teeth Apparently Not Involving Periodontal Receptors [published in *Archs oral Biol* (1998), **43**, 1-7]

(4) Effects on Human Maximum Bite Force of Biting on a Softer or Harder Object [published in *Archs oral Biol* (1998), **43**, 833-839]

(5) Occlusal Loads Cause Fluid to Move Through Dentine *in vitro* [submitted to *Archs oral Biol*]

Paper #1 measures the effect of jaw separation on the magnitude and direction of incisal MBFs. The data on magnitude were used to estimate the effect on an incisal MBF of opening the jaw by capping the incisors in the experiment described in Paper #2. The data on direction were used to understand how the bite direction is affected by changes in the position of the lower jaw. The edge-to-edge jaw position was subsequently chosen for measuring MBFs in Papers #2 and #4.

Paper #2 measures the effect on a MBF of changes in the stress (the term "pressure" was originally used) on an incisor crown. The MBFs were compared for high- and low-stress conditions. In high-stress conditions the upper incisor tooth bit on an acrylic wedge. In low-stress conditions the same tooth was capped with an acrylic crown, spreading the load and reducing the stress, before biting.

Paper #3 tests whether a pulpal sensory mechanism alone can be used to detect the hardness of an object placed between upper and lower teeth. Subjects were asked to decide whether the upper incisors were biting on acrylic or rubber. The design of the experiment prevented subjects from using information from mechanoreceptors in TMJ, jaw muscles and periodontal ligament.

Paper #4 confirms the effect of stress on the MBF. MBFs were compared when the upper incisors bit on acrylic (high-stress) and on rubber (low-stress). The method developed in Paper #3 was used in order to prevent the MBFs being affected by other receptors.

Paper #5 tests whether sufficient fluid is squeezed through dentine to activate pulpal nerves when extracted teeth are compressed by forces in the range commonly used.

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CHAPTER 2

EFFECT OF JAW OPENING ON THE DIRECTION AND MAGNITUDE OF HUMAN INCISAL BITE FORCES[1]

(Paper #1)

J. Paphangkorakit and J. W. Osborn

INTRODUCTION

It has been reported that the maximum bite force (MBF) in man increases as the jaw is opened, reaches a maximum at between 14 and 20 mm of incisor separation, and then decreases as the jaw is further opened (Manns *et al.*, 1979; Mackenna and Turker, 1983; Fields *et al.*, 1986; Lindauer *et al.*, 1993). The change is generally attributed to a change in the length/tension relationship of the jaw closing muscles, a phenomenon which has been observed in single muscle studies. For example, the largest twitch tension in the masseter muscle develops at maximum gape in the cat (Mackenna and Turker, 1978) and each jaw closing muscle in the opossum develops its maximum twitch tension at a different degree of jaw opening (Thexton and Hiiemae, 1975).

In a less direct approach, Storey (1962) and Garrett *et al.*, (1964) both showed that the EMG activity required to sustain a given submaximum bite force is reduced as the jaw is opened. Manns *et al.*, (1979) kept masseter activity roughly constant and found that the MBF on a premolar peaked at 15-20 mm incisor separation. The EMG increment of masseter activity per unit molar bite force is least at 9-11 mm molar separation (Mackenna and Turker, 1983).

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The maximum incisal bite force (MBF) has always been measured by transducers which only recorded the single (vertical) component of the force, parallel to the measuring axis of the transducer. The change in MBF when opening could, however, be caused by a (unmeasured) change in the bite direction, no longer directed down the measuring axis of the transducer, rather than a change in the magnitude of the bite force. If the direction of the bite force changes as the jaw is opened all the above observations could be related to physical, as opposed to physiological, changes because a theoretical analysis suggests that if the direction of the bite force is changed in association with jaw opening the mechanical advantage of the jaw closing muscles may be increased (Osborn, 1995). The direction of the bite force has, however, been ignored except by Hylander (1978) who observed a different incisor bite direction at two different jaw openings.

The full effect of jaw opening on the MBF can only be clarified by simultaneous measurements of the magnitude and the direction in three dimensions of the bite force. We investigate both here in conjunction with the EMG activity in left and right masseter and anterior temporalis muscles.

MATERIALS AND METHODS

Bite force recording

The maximum bite force (MBF) between lower and upper incisors was measured by a transducer which simultaneously records the bite force direction in three dimensions. The transducer was similar to that used by Osborn and Mao (1993) except that it has been modified into a U shape which can be more easily placed on any teeth in the dental arch (Fig. 2-1A, see also Appendix A)

Impressions of the upper and lower jaws were taken. The lower horizontal plate of the transducer was attached to an acrylic resin overlay (Dura Lay, Dental Mfg, IL, USA), moulded over the lower incisors (Fig. 2-1C), which was made on a plaster model of the lower dentition. A small acrylic cone, 2 mm high, attached to the centre of the upper horizontal plate of the transducer ensured that the bite force from the upper incisor was always applied in the same position on the transducer.

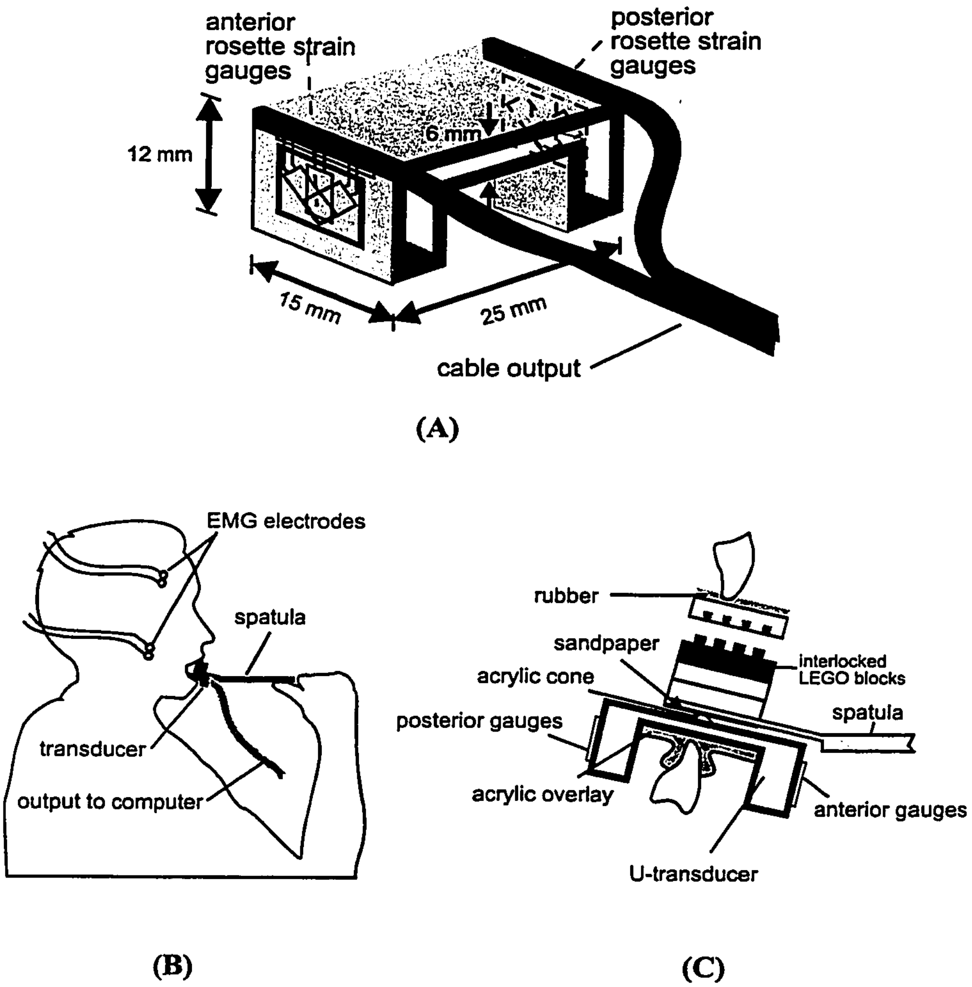

Figure 2-1 (A) The U-shaped force transducer. Rosette strain gauges were bonded to each of its vertical components. (B) The subject bites on LEGO blocks placed between upper and lower incisor teeth. The blocks were lightly supported by a subject's digit. (C) The bite block contained interlocked 3-mm thick plates of LEGO. A thin rubber sheet was glued to the top of the bite block. The transducer was fitted to the lower incisor teeth by moulded dental acrylic resin.

The minimum jaw opening was about 9 mm, being the thickness of the transducer with its lower incisor overlay and attached cone together with the incisor overbite. The opening was increased by placing a bite block between the upper incisor and the transducer. The bite block consisted of a stack of plates from an interlocking construction toy (LEGO) attached to a spatula (Fig. 2-1B,C). The 3 mm thickness of these plates determined the increments of jaw opening. The upper surface of the bite block was covered with a thin sheet of rubber, and the lower surface with a sheet of fine sandpaper, to prevent the surfaces from sliding.

The bite direction was measured with respect to the axes of the transducer. The orientation of the transducer to the lower occlusal plane was measured while the lower incisor overlay was being constructed on a plaster model of the lower dentition.

The transducer was connected to a strain gauge conditioner where the signals were digitized and then fed into an IBM personal computer. Bite force magnitudes and directions, viewed on the computer screen (see Osborn and Mao, 1993), were stored in files for off-line analyses.

EMG recording

After the skin was thoroughly cleaned, pairs of circular 16-mm skin electrodes (Beckman Instruments, Anaheim, CA, USA) were pasted by adhesive collars to the skin over the middle of the left and right masseter and anterior temporalis muscles, with an imaginary line joining their centres parallel to muscle fibre orientation. Each inter-electrode distance was 16 mm. The electrode cups were filled with electrolyte gel in order to improve conductivity. A ground electrode was pasted on the skin over the fifth cervical vertebra.

EMG signals were amplified with P15 Grass-preamplifiers, at a gain of 1000 and a bandwidth of 10-1000 Hz, and displayed on an 8-channel oscilloscope (5A14N, Tektronix, Beaverton, OR, USA). They were digitized with a 12-bit A/D converter (Dash-8, Metrabytes Ltd, Wilmington, DE, USA) and stored in a file using software (Labtech Notebook, Metrabytes Ltd, Wilmington, DE, USA) installed in an IBM-compatible personal computer. The signals were recorded for two seconds at 1000 Hz as soon as a subject reached the MBF after being instructed to bite as hard as possible on the transducer.

Experimental procedures

The study was approved by a Human Ethics Committee. Ten university students, eight males and two females, aged 28-36 with sound incisor teeth, without TMJ or muscle pain participated in the study. All gave consent before participating in the experiment. The subject was seated upright in a dental chair. The bite force display screen was hidden from the subject. After fitting the transducer to the lower teeth, the bite block was placed over the acrylic cone on the upper horizontal plate. The mandible was slowly closed with the help of guidance from the operator so that the upper incisors bit in the centre of the bite block. The operator ensured that the mandible did not deviate from the midline or protrude. The subject was now asked to lightly hold the spatula with his left hand and bite as hard as possible. As soon as he felt he was producing his MBF he tapped a bell which signalled the operator to begin recording. The MBF was maintained for about two seconds.

Subjects were allowed to practise a few times. The jaw opening was widened by gradually adding LEGO blocks until the subject felt that biting was uncomfortable (usually around 30-34 mm of incisal opening). Subjects rested until they felt comfortable (usually 3-5 minutes) after each trial. The whole series was completed twice.

A 2-second bite trial contained 12 bite force measurements (6 Hz × 2 seconds) and the MBF was chosen from the largest measurement in that trial. All bite forces were expressed as a percentage of the largest MBF (%MBF) throughout an experiment. The bite direction in the sagittal plane measured with the transducer was converted into a direction with respect to the lower occlusal plane (see above).

The raw EMG signal was rectified and smoothed by a moving averaging method involving 100 data points for each step. Two separate maximum clenches in centric occlusion were used to find the maximum EMG signal from each muscle. Customized computer software was used to calculate averages of the highest 50 smoothed EMG amplitudes in each of the two recordings. The higher value was used as 100% activity. All other rectified and smoothed EMG signals were averaged over the 2-second period and then normalized to the maximum values and expressed as %MVC (percent of maximum voluntary contraction).

Bite directions in the frontal and sagittal planes, %MBF and %MVC were each averaged from the two trials for each subject and plotted against incisal separations. The ratios of the activity of the temporalis and masseter muscles (T/M ratio) were also plotted against incisal separations.

Jaw opening was expressed as millimetres of separation between incisal edges plus the original overbite. Due to different overbites and opening capability, the minimum and maximum openings were different for each subject. However, since the height of each block was 3 mm, it was possible to group all incisal separations in 3-mm intervals for example, 8-10, 11-13, 14-16 mm *etc.* We also included preliminary results from two subjects who bit on another set of customized acrylic blocks whose height was different from those using the LEGO block. Therefore each opening position did not have the same number of data points.

Differences between the %MBF, bite direction, %MVC and T/M ratio at different openings were tested in pairs by a Mann-Whitney U test. A linear regression was also performed to investigate any relationship between the bite direction and jaw opening, as well as the T/M ratio and jaw opening.

RESULTS

Subjects used very little manual force to stabilize the bite block between the incisors and the transducer. It usually involved no more than a lightly placed finger or thumb (Fig. 2-1B) to check the position of the handle. For practical purposes therefore, the force recorded by the transducer was entirely produced by the jaw muscles.

The MBFs ranged from 117 N to 368 N with an average of 233 N (SD = 65 N). Such a considerable spread has been observed in all other equivalent studies. The MBFs for the pooled data showed a plateau between 14-16 mm and 26-28 mm of incisal separation (Fig. 2-2A). The average MBF significantly increased ($p < 0.01$) from 11-13 mm to 14-16 mm opening and dropped from 26-28 mm to 29-31 mm opening. In terms of individuals, five of the ten subjects recorded single peak MBFs at 15, 17, 20, 27 and 28 mm opening respectively. Three subjects recorded a plateau between about 14 and 28 mm opening. Two subjects recorded two separate peaks, the first at 14, 16 mm and the second at 23, 28 mm openings respectively. At the smallest and the largest jaw openings the MBFs were about 85% (SD = 11%) and 80% (SD = 15%) of the maximum recorded value for each individual.

The direction of the MBF changed from 11° forward to 5° backward with respect to the lower occlusal plane and was nearly symmetrical in the frontal plane throughout jaw opening (Fig. 2-2B). The initial forward bite with respect to the lower occlusal plane (range 2° to 28°) was gradually shifted backward (range 3° to -28°) in seven subjects. The remaining three subjects maintained a forward bite with respect to the lower occlusal plane (range 6° to 16°) throughout the experiment.

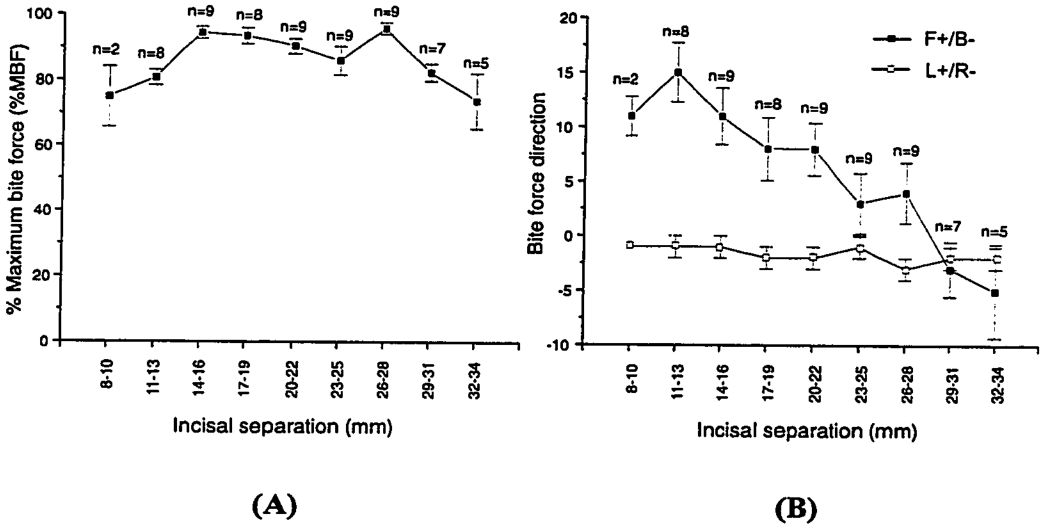

Figure 2-2 (A) The magnitude of the incisal maximum bite force (%MBF ± SE[2]) at different jaw openings. (B) The bite direction of the MBF (± SE) at different jaw openings. Note the backward shift of the bite force with respect to the mandible in the sagittal plane as the jaw was opened (forward = +, backward = -). The bite force was almost symmetrical in the frontal plane (left = +, right = -).

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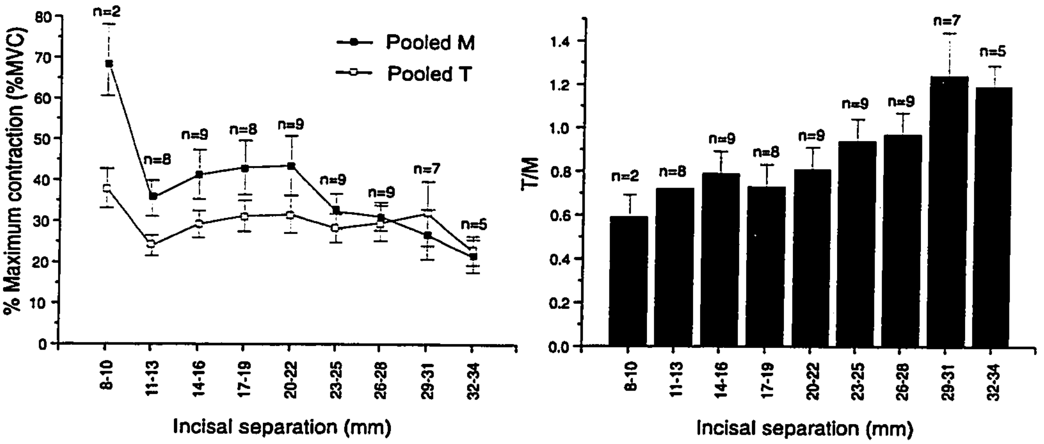
[2] SE = standard error of the mean ($SD/\sqrt{n}$)

Masseter activity gradually declined as the jaw was opened while temporalis activity was roughly constant (Fig. 2-3A). The T/M ratio therefore increased, from about 0.6 to 1.2, during jaw opening (Fig. 2-3B). There was no significant difference in either the T/M ratio, the bite direction or the %MVC between paired consecutive opening positions. Except for the %MVC those values at 11-13 mm opening, however, significantly differed from those at 32-34 mm opening ($p < 0.01$). Due to too small samples ($n = 2$), any data at 8-10 mm opening were not included in the statistical test. There was a significant correlation between bite direction and jaw opening ($r = 0.51$, $p < 0.001$) (Fig. 2-4A) and between T/M ratio and jaw opening ($r = 0.56$, $p < 0.001$) (Fig. 2-4B). The larger the opening, the larger the T/M ratio and the more backward the bite direction with respect to the lower occlusal plane.



(A) (B)

Figure 2-3 (A) The average pooled activity of the masseter and anterior temporalis muscles (%MVC ± SE) at different jaw openings. The masseter activity significantly declined at wider openings. (B) The ratio of the temporalis activity over the masseter activity (T/M ± SE) at different jaw openings.

DISCUSSION

In most previous studies of incisor MBFs the force transducer has been rigidly attached to an external frame. Furthermore, apart from Hylander (1978), previous workers have only measured the component of the bite force in the single dimension of the linear measuring axis of their transducer. A subject's true bite force is now the vector sum of (1) the bite force measured on the transducer plus (2) the unmeasured components of the bite force in the two dimensions perpendicular to the measuring axis of the transducer plus (3) the reaction force on the frame stabilizing the transducer. We have excluded the possibility of errors from (2) by using a transducer which measures forces in three dimensions and from (3) by minimizing stabilizing the transducer outside the mouth (with only light contact between a digit and the handle of the bite opener: Fig. 2-1B).

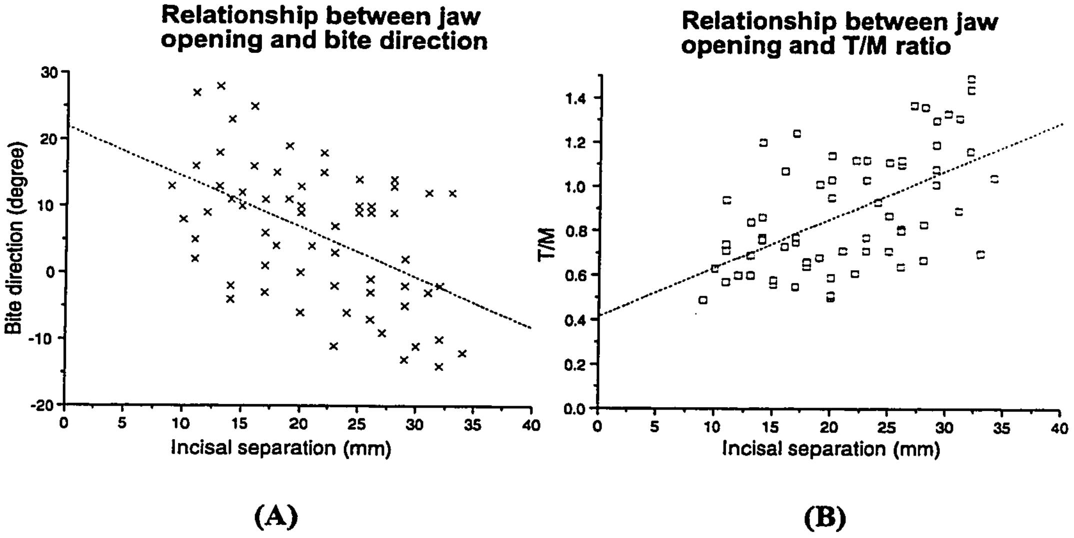


Figure 2-4 (A) All bite directions in the sagittal plane were plotted against jaw opening (forward = +, backward = -). The regression line is shown (r = 0.51, p < 0.001). (B) All ratios of the temporalis activity over the masseter activity (T/M) were plotted against jaw opening. The regression line is shown (r = 0.56, p < 0.001).

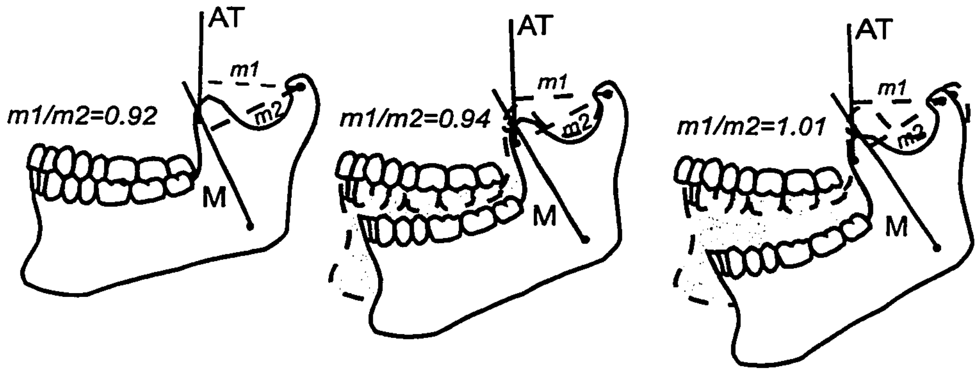
The %MBF showed a plateau roughly between 1/3rd and 2/3rd of the maximum opening. Gelb (1990) found two (or even more) peaks whereas Manns *et al.*, (1979) and Mackenna and Turker (1983) found only one peak. Boucher *et al.*, (1959) found more variability and described the shapes of the curves they plotted for MBF against jaw opening as plateau, concave, straight line and S-shaped.

There are several explanations for the observed changes in MBF associated with different jaw openings:

> (1) Increased activity in other jaw closing muscles could be responsible. Most studies of muscle activity and the MBF have only measured activity in the masseter and temporalis muscles because the other jaw closing muscles are not accessible to surface electrodes. An increase in the MBF associated with jaw opening might therefore be partly or wholly caused by an (unmeasured) increase in the activity of medial and lateral pterygoid muscles (Lindauer *et al.*, 1993).
>
> (2) There may be a change in mechanical advantage. Throckmorton *et al.*, (1980) concluded it is unlikely that the mechanical advantage of muscles would increase as the jaw is opened. However, using the data of muscle attachments from Osborn (1995), in our three-dimensional mathematical calculation we have found that the ratio of the length of the anterior temporalis muscle's moment arm to that of the superficial masseter muscle is increased during jaw opening (Fig. 2-5, see also Appendix B). Osborn (1995) has shown by means of a three-dimensional model that an MBF tilted 15° forward on the lower incisors of a jaw opened 15° is larger than a vertical MBF on lower incisors with the jaws closed. Koolstra *et al.* (1988) also showed in their model, that the MBF perpendicular to the upper occlusal plane was about 20% larger at 10° jaw opening than with jaw closed in an edge-to-edge position. These, however, do not explain the increase in the MBF observed in the present study because it was associated with a more backward directed bite force. It should be noted that a more backward incisal bite direction could be mechanically

associated with a larger bite force magnitude (Baragar and Osborn, 1987; Koolstra *et al.*, 1988).

(3) There may be a change in the length/tension relationship of muscles. The resting sarcomere length of masseter is greater than that of temporalis (Nordstrom *et al.*, 1974 in rats; Hertzberg *et al.*, 1980 in rabbits; Herring *et al.*, 1984 in pigs). Although the masseter sarcomere length is known for human jaw muscles (2.2-2.5 μ; van Eijden and Raadsheer, 1992), we do not have the datum for the temporalis muscle. It has been shown that human temporalis fibres are 30% longer than masseter fibres and both muscles have about 20° pinnation angles (Schumacher, 1961). Using the pig's resting sarcomere lengths (2.7 μ in the masseter and 2.1 μ in the temporalis; Herring *et al.*, 1984) together with a human geometric model



Closed

14-16 mm incisal separation (10-degree rotation)

26-28 mm incisal separation (7-mm translation and 17-degree rotation)

(A) (B) (C)

Figure 2-5 Geometrical diagrams were used to calculate the ratio of anterior temporalis' (AT) moment arm to that of masseter (M) (ml/m2). (A) Jaw closed position. (B) About 14-16 mm incisal separation. (C) About 26-28 mm incisal separation (plus a translation of 7 mm).

> (Osborn, 1995) we calculate that for a 10° opening rotation (roughly 16 mm incisal separation) temporalis is stretched 16% and masseter 11%. If the opening rotation is 17° and the condyle is simultaneously translated 7 mm forward (roughly 28 mm incisal separation) the stretches are roughly 32% and 16% respectively[3]. These figures can be manipulated to show that at 10° opening the sarcomeres of masseter are stretched 3 μ and those of temporalis 2.3 μ. Thus at 10° opening masseter sarcomeres have only just exceeded their optimum length (2.9 μ in pigs, Herring *et al.*, 1984). At 17° opening, the (shorter) sarcomeres of temporalis are getting closer to their (presumably shorter) optimum length and those of masseter are stretched beyond their optimum length.

The plateau-like curve in %MBF may result from a combination of the different optimum range in the masseter and later during jaw opening in the temporalis muscles. Some subjects might produce the largest MBF at the masseter optimum length whereas some at the temporalis optimum length. This may partly explain the variety of curves in previous studies but the unknown contribution of medial pterygoid may also affect the results.

The increase in the ratio of temporalis/masseter activity was associated with the backward shift in bite direction as the jaw was opened. Change in bite direction may be simply due to a change in the position of the upper relative to the lower bite points if the bite direction is presumed to lie along a line joining them. This line gradually tilts back with respect to the lower jaw during jaw opening. Therefore, the decline in masseter activity (also reported by Garrett *et al.*, 1964; Manns *et al.*, 1979; Lindauer *et al.*, 1993) is probably due to (1) a change in the bite direction determined by the altered bite points at larger openings[4] and (2) the fact that the masseter is probably stretched beyond its safe

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[3] The results were obtained by measurements from a two-dimensional diagram. See also Appendix C.

[4] See Appendix D.

limit and, being therefore more liable to muscle injury (Hasselman *et al.*, 1995; Hutchin *et al.*, 1995), is less active.

The backward shift of the bite direction was unlikely to be due to the muscle fatigue reported by Osborn and Mao (1993) during endurance tests which lasted about a minute. In this study subjects sustained their MBF for not longer than two seconds and a 3-5 minute rest was always allowed after each bite trial.

Although we did not observe a greater activity in the temporalis during incisor biting at large openings, some frugivorous anthropoid primates which make heavy use of their large incisors have larger anterior temporalis muscles (Cachel, 1979). It could be that these animals often use their incisors at the wide gapes required to incise large fruit and, like man at large gape, bite more backwards. In order to maintain the bite force in this backward direction more powerful temporalis muscles are needed (Osborn, 1995). Hylander and Johnson (1985) found that the anterior and posterior temporalis muscles were both very active in macaques and humans during apple incision compared with during chewing.

Synergistic muscles which cross a joint are presumably designed to maintain efficient working levels over a wide range of movement. This synergism might account for observed differences in resting sarcomere length, fibre length and/or pinnation angles (Richard *et al.*, 1990) as well as the different length-tension characteristics (Gareis *et al.*, 1992) of different muscles. All of these parameters may be gradually adjusted during growth in order to maintain the largest force with the minimum use of energy during the most frequently used range of movements (Herring *et al.*, 1984). This may explain variations among our subjects.

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CHAPTER 3

THE EFFECT OF PRESSURE ON A MAXIMUM INCISAL BITE FORCE IN MAN[1]

(Paper #2)

J. Paphangkorakit and J.W. Osborn

INTRODUCTION

It has been widely accepted that intradental receptors are exclusively involved in the sensation of pain whereas their role in the control of mastication is questioned. However, some pulpal receptors in cats have been reported to respond to small transient forces (10-60 mN) rapidly applied to the crown of the tooth (Dong *et al.*, 1985). These mechanoreceptors were not directional sensitive and, unlike periodontal mechanoreceptors, only discharged a few spikes during the ramp phase of a transient force. Transiently tapping (10-150 N) an immobilized cat canine tooth induced EMG activity in the digastric muscle (Olgart *et al.*, 1988). Removing the coronal pulp or cooling the crown with ethyl chloride abolished the response. The authors concluded that the withdrawal reflex might have been mediated via pulpal A-fibres stimulated by the deformed tooth. However, it is not known from animal studies whether pain was involved.

The human tactile threshold to mechanical force was found to be higher in pulpless teeth (10-45 mN) than in vital teeth and in teeth covered by a metal cap (Loewenstein and Rathkamp, 1955) although this was argued to be due to the effect of some degenerative periodontal receptors (Linden, 1975). Electrically stimulating human

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[1] A version of this chapter has been published. Paphangkorakit J. and Osborn J. W. (1997), *Archs oral Biol* **42**, 11-17.

incisor teeth at a non-painful level elicited inhibitory effects in the masseter muscle (Matthews *et al.*, 1976) and a successful stimulus to the dental pulp was not always associated with pain (Hannam *et al.*, 1974; Matthews *et al.*, 1976; Brown *et al.*, 1985; Mason *et al.*, 1985). A recent study in cats (Matthews and Vongsavan, 1994) has revealed the possible role of pulpal nerves in regulating pulpal pressure by means of vasodilation. All of the above evidence suggests that pulpal nerves could be involved in sensory mechanisms other than pain.

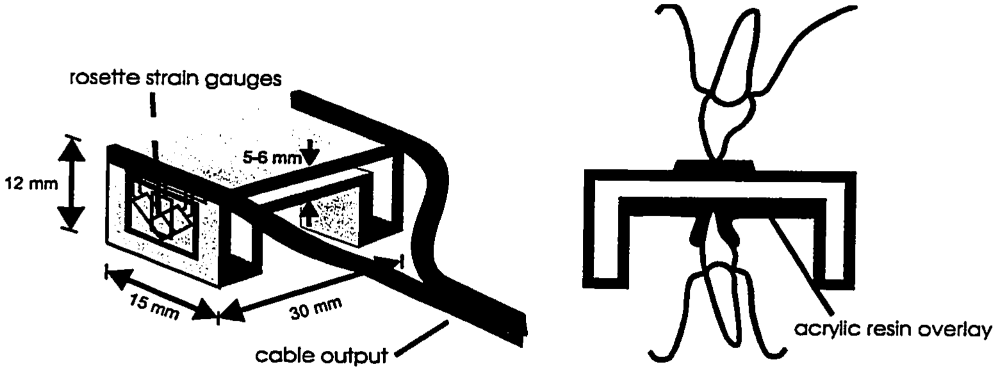
A-δ and C-sensory nerve fibres have been identified in the dental pulps of cats (Beasley and Holland, 1978; Lisney, 1978; Cadden *et al.*, 1983; Virtanen *et al.*, 1983), dogs (Wagers and Smith, 1960; Matthews, 1977; Närhi and Hirvonen, 1987) and man (Graf and Bjorlin, 1951; Matthews *et al.*, 1959; Reader and Foreman, 1981). The A-fibres are myelinated and have larger diameters and faster conduction velocities (> 2 m/s and usually < 30 m/s) than unmyelinated C-fibres (< 2 m/s) (Lisney, 1978; Närhi and Haegerstam, 1983). Both are thought to exclusively mediate noxious sensations and contribute to pain sensation, as elsewhere in the body (Mumford and Bowsher, 1976; Sessle, 1987). However, some fibres with faster conduction velocities (> 30 m/s), classified as A-β fibres, have been found and suggested to mediate non painful sensation (McGrath *et al.*, 1983; Virtanen *et al.*, 1987; Närhi *et al.*, 1992). Moreover, some pulpal afferents, like periodontal mechanoreceptors, were found to have their cell bodies in the trigeminal mesencephalic nucleus (Chiego *et al.*, 1980), suggesting their possible role in proprioception (Byers, 1984).

We investigate here whether some pulpal receptors could subconsciously control the production of bite force without involving pain. Instead of using the small loads associated with tactile thresholds we studied the response to heavy loads associated with maximum bite forces. For reasons which will be apparent later we test whether covering the crown of a tooth changes the maximum bite force which a subject can produce.

MATERIALS AND METHODS

A U-shaped transducer has been used to measure bite forces in three dimensions (Fig. 3-1A). Two stacked rosettes, each comprising three strain gauges (Micro-Measurements Inc, Raleigh, NC, USA), are bonded to the two vertical sides of the transducer. The six strain values are used to calculate the magnitude and direction in three dimensions of a bite force applied to the horizontal bars of the transducer (Fig. 3-1B).

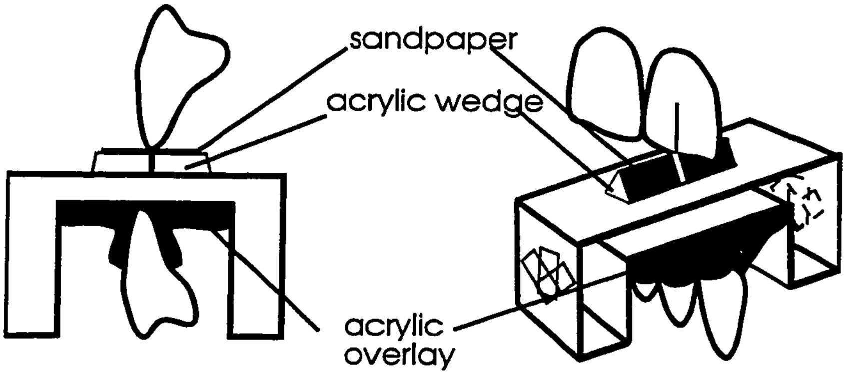
The transducer was stabilized by moulding an acrylic resin overlay (Dura Lay, Dental Mfg, IL, USA) between its lower horizontal plate and the lower incisal edges. A 2-mm high acrylic wedge was built on the upper horizontal plate so that the upper incisor could be loaded at a point, the apex of the wedge (Fig. 3-2). The wedge was covered by a strip of fine sandpaper to prevent the tooth from slipping. A line pencilled on the sandpaper was used to ensure that the upper incisal edge was centred on the transducer. The distance between the upper and lower incisal edges was about 8 mm.



(A) (B)

Figure 3-1 (A) The (inverted) U-shaped transducer. Note the orientation of the rosette strain gauges on both sides. The jaw separation caused by the transducer was 7-8 mm. (B) The lower incisors were covered by acrylic and the upper incisor tip bit on a wedge.

The study has been approved by an Ethics Committee. Having given informed consent, 18 individuals (13 males and five females), with no evidence of tooth pathology or history of joint pain participated. Upper and lower dental impressions were used to make plaster models on which two identical acrylic caps were made for an upper central incisor (Ortho-Resin, clear, Caulk, DL, USA) and acrylic overlays for lower incisors (Dura Lay). The choice of left or right upper incisor was random. The incisal edge of each cap was made less than 0.5 mm thick so as to minimise the increase in jaw opening with the cap in place. A vertical line was drawn in the same position down the centre of each cap (Fig. 3-2B). A hole was made in the centre of the incisal edge of one cap so that when the participant bit together the force would be transmitted directly to the incisal edge of the tooth rather than spread over its whole surface through the acrylic crown (Fig. 3-3). In this way the participant was made unaware of the true condition of the experiment (*i.e.* whether biting with no-cover or full-cover).



(A) (B)

Figure 3-2 The orientation of the transducer within the mouth. The lower jaw with its fitted transducer were moved so that the upper incisor and wedge were aligned with lines drawn on each.

The transducer with its lower overlay attached was fitted over the lower incisor teeth. The participant was guided to open and then lightly close with the lower jaw centred in the frontal plane (checked by the vertical line drawn on the acrylic) and the upper incisor biting at the point on the wedge marked by the pencilled line. Individuals were asked to rapidly clench as hard as possible on the occluding upper incisor under two different conditions: (1) on the full crown (full-cover) and (2) on the crown with a hole at its tip (no-cover). They practised biting before starting the measurements and were asked to try to use the same strategy for each trial.

Measurements of the maximum bite force (MBF) and its direction under each of the two conditions were repeated three times randomly and recorded at 5 Hz. A 3-minute break was provided between each trial. Recording began just before the trial started and ended as soon as the subject relaxed. The duration of time to the MBF was always less than a second.

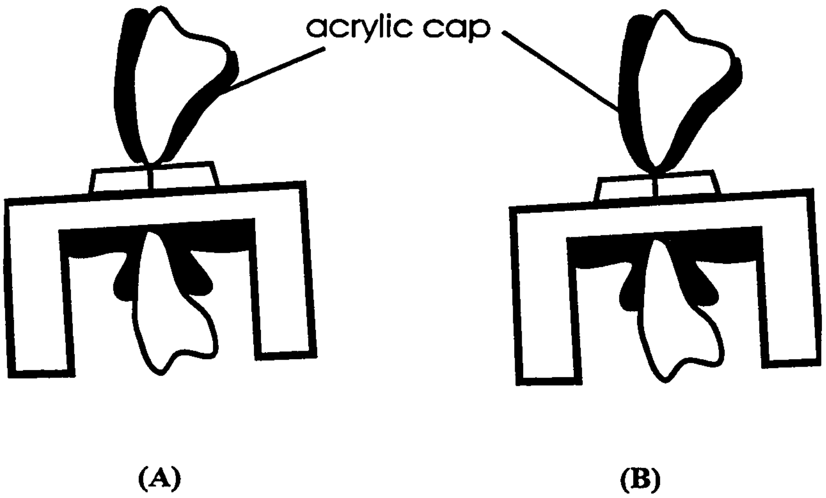
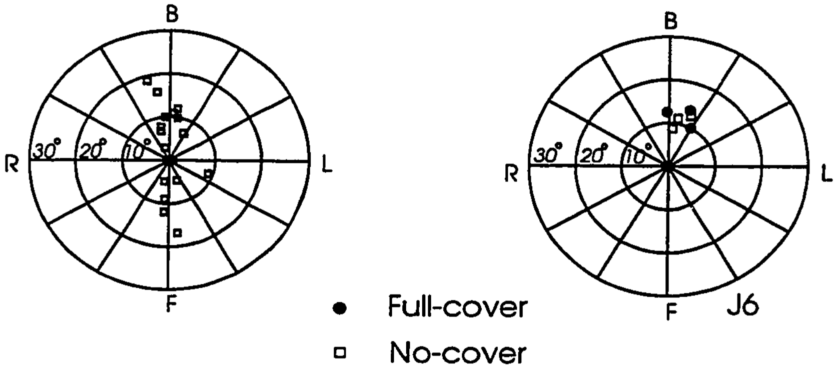


Figure 3-3 The relationship between the upper and lower incisor teeth with (A) no-cover, (B) full-cover. Note the mandibular protrusion required to center both upper and lower incisor teeth on the transducer. The transducer was sometimes tilted anteriorly to protect the soft tissues. The thickness of the acrylic cap at the contact point with the wedge was about 0.5 mm.

It was not always possible to orient the transducer on the plaster models so that its horizontal plates were parallel to the occlusal plane, because of interference between a vertical wall of the transducer and the lingual side of the alveolar process. Its spatial orientation with respect to the lower occlusal plane was measured on the models. The angles measured on participants were later transformed into angles with respect to the lower occlusal plane (see Appendix E).

Several individuals produced one MBF which was very small compared with the other MBFs due to some unknown discomfort. In order to eliminate these clearly abnormal results we rejected the smallest MBF for every participant and averaged only the larger two results. Each averaged MBF was normalised by expressing it as the percentage (%MBF) of the largest single MBF recorded for that individual. Bite force directions under different conditions were compared both in the frontal (left-right) and sagittal (front-back) planes by a paired t-test. The possibility of intraindividual differences in the %MBF for the two different conditions was also tested by a paired t-test.

RESULTS



(A) (B)

Figure 3-4 (A) Polar coordinate diagrams plotting the grand averages for all 18 participants. (B) The typical bite force directions for the six bite trials in an individual. Each circle represents 10° away from the vertical. (F = front, B = back, R = right, L = left of the participant)

Most participants reported a mild sensation of strain on the upper incisor after each trial but this disappeared during the 3-minute break; none reported pain. They took between 330-900 ms to reach their MBF, the rate of change ranging from 158-400 N/s.

All participants bit nearly vertically (symmetrically) in the frontal plane but the angle varied from about 20° forward to 20° backward in the sagittal plane (Fig. 3-4A). For a given individual, however, there was no significant difference in the bite direction under the two different conditions.

The variation of MBFs was large (90 to 370 N). The maximum MBFs and average %MBFs for the two different conditions are shown in Table 3-1.

Table 3-1 The average normalized maximum incisal bite force (expressed as percentage of the single largest MBF recorded in each participant) in all participants for no-cover and full-cover.

Participant	Sex	Maximum MBF (N)	No-cover	Full-cover	Increase
			(% MBF)		
R4	M	120	77.8	88.9	+11.1
A6	M	270	85.2	97.5	+12.3
W1	M	290	84.9	89.2	+4.3
C3	M	220	87.9	98.5	+10.6
F1	M	140	85.7	92.9	+7.2
E4	F	170	86.3	92.2	+5.9
N3	M	290	96.6	98.9	+2.3
D5	M	370	88.3	91.0	+2.7
T10	F	270	87.7	97.5	+9.8
K9	M	130	92.3	100.0	+7.7
S9	F	210	90.4	100.0	+9.6
Yi2	M	210	92.9	100.0	+7.1
Jm1	M	200	95.0	95.0	0
H1	M	200	90.0	90.0	0
B1	F	110	93.9	93.9	0
L3	F	120	91.7	91.7	0
J6	M	220	92.4	92.4	0
Y3	M	250	94.7	92.0	-2.7
	MEAN	**210.5**	**89.7**	**94.5**	**4.9**
	SD	**69.3**	**4.5**	**3.8**	**4.6**

M = male, F = female

The %MBF recorded for the full-cover was significantly larger than that for no-cover ($p < 0.001$). The MBF increased (range 2.3-12.3%) in 12 participants, was unchanged in five and decreased by 2.7% in one.

DISCUSSION

Hylander (1978) and Osborn and Mao (1993) have measured the directions of bite forces on incisor teeth. In both studies the bite force was tilted anteriorly although in the endurance tests of Osborn and Mao (1993) it moved toward vertical as the participants tired. Unlike those two experiments in which the bite forces were measured when the lower jaw was retruded, the bite force in this study was measured with the lower jaw protruded toward the edge-to-edge position. Different amounts of protrusion may account for the variability in the bite direction in the sagittal plane and the backward direction in some participants (Fig. 3-4). A more forward position of the lower incisors results in a more backward bite direction whereas a more backward position results in a more forward bite direction. In the frontal plane, all participants bit almost vertically as might be expected from the symmetrical jaw position when biting incisally.

There was little difference ($< 5^{o}$ in the cone of a three dimensional angle) between the bite force directions used by any given participant under different conditions (Fig. 3-4B). The result accords with those of Mao and Osborn (1994) who showed that the bite force direction on a given tooth used by a given individual is constant regardless of its magnitude.

Most participants, when asked, reported feeling mild strain on the upper incisor tooth but not on the lower teeth, probably because the bite force was supported by only one upper tooth as opposed to two to three lower teeth. An alternative explanation is the upper incisor tooth is more proclined and may receive a larger torque, depending on the direction of the bite force.

Participants were asked to increase their bite force very rapidly for two reasons. First, with a rapid bite force it seemed unlikely that a voluntary correction would be made during the experiment. A change in MBF would be an involuntary response. Second, it has been suggested that intradental mechanoreceptors are only activated by transient forces (Olgart *et al.*, 1988; Dong *et al.*, 1993).

The maximum incisal bite force ranged from 90 to 370 N. Different investigators have reported values from 50 N to 240 N (Helkimo and Ingervall, 1978; Hellsing, 1980; Osborn and Mao, 1993; Waltimo *et al.*, 1993). The variation may be due to muscle size, the shape of bones and the articulating joint tissues, the amount of jaw separation and/or instrumentation. The participant's motivation and sensitivity to discomfort (not necessarily pain) may also affect a maximum voluntary effort.

The MBF was significantly larger when the upper incisor crown was covered. We dismissed the following explanations for the results. (1) The MBF might be limited by a pain threshold in the periodontal ligament. No participant complained of pain and there is no reason for such a threshold to change if the crown of the tooth is covered. (2) The MBF might be limited by pain related to pulpal receptors (Robinson, 1964). But, again, no participant complained of pain and it was unlikely that the type of deformation studied by Robinson would have occurred in the present study. (3) Up to a certain limit, increasing jaw separation increases the MBF (Manns *et al.*, 1979). In our own recent study of the effect of jaw opening on incisal MBFs (Paphangkorakit and Osborn, 1997) the predicted increase in MBF for an increased jaw separation of 0.5 mm was < 1% as opposed to increases up to 12% observed here (Table 3-1). (4) There could be a training effect where participants perform better with practice. Our participants practised before the experiments but the sequence of tests was randomized. (5) The possibility of a change in bite direction, which would affect the MBF, was discounted by the observations (Fig. 3-4B). (6) A change in the point at which the bite force is applied to a transducer changes the measured force. We took care to ensure that each individual always bit in the same place on the wedge (Fig. 3-2).

The acrylic cap spread the load over the covered surface instead of allowing it to be concentrated at a point. Thus for a load of 100 N the pressure on the tooth enamel was roughly 100 N/mm^2 without a cap (contact area ~1 mm^2) and about 1 N/mm^2 with the full cover (contact area ~100 mm^2 when the labial or palatal surface is considered as the load bearing area for a backward or forward bite direction respectively).

The periodontal ligament detects the torque of a force applied to a tooth, not the pressure or the point of application. Thus a force of 75 N applied to a point on the crown (Fig. 3-5B) has exactly the same mechanical effect on the periodontal ligament as a 75 N force spread over a large area (Fig. 3-5A), provided the resultant is in the same direction and acts at the same point (as in our experiment). In the two cases the only difference is the pressure on the enamel and, through the enamel, on the dentine which supports it. We conclude that the recorded differences in MBF were probably related to the different pressures on the crown of the tooth. This implies the existence of intradental mechanoreceptors which reflexly control the MBF and can, perhaps, detect the differences between the two conditions shown in Fig. 3-5.

We postulate the existence of high threshold intradental coronal mechanoreceptors which are responsible for limiting the MBF when a tooth is subjected to a large transient bite force. They are unlikely to be the same as the low threshold receptors whose existence others have postulated (Loewenstein and Rathkamp, 1955: Dong *et al.*, 1985, 1993). The threshold would also presumably be lower than that of the pulpal nociceptors because none of our participants complained of pain.

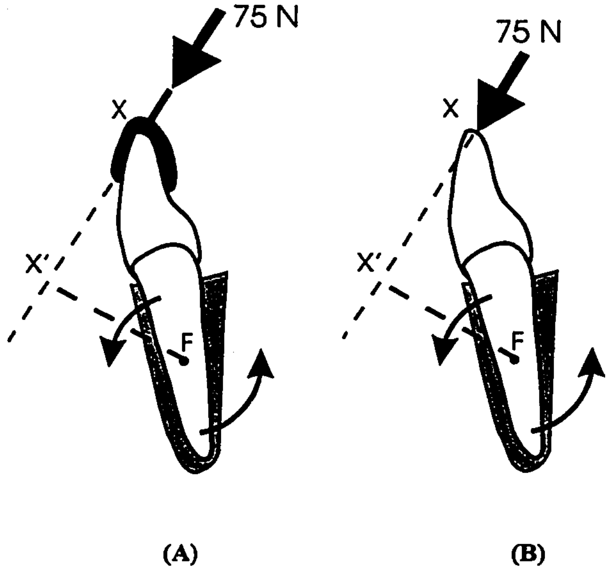


Figure 3-5 The periodontal ligament can receive the same torque (and therefore provide the same sensory input) under different conditions. In both diagrams the moment arm is X'F: (A) 75 N force spread around the tip of the tooth. (B) 75 N force concentrated at the tip of the tooth.

The generally accepted "hydrodynamic mechanism" of dentine sensitivity (Brännström, 1963) suggests that fluid moving through dentine tubules is the source of a successful stimulus. The movement of fluid through dentine has been demonstrated by placing solutions of high osmotic pressure on exposed dentine (Anderson *et al.*, 1967). Using finite element analyses Spears *et al.* (1993) have shown that, if tooth enamel is anisotropic, stress from a force applied to the crown of a tooth is dissipated across the enamel/dentine junction into the dentine. Presumably the stress strains the dentine, especially that close to the loading point, and fluid is displaced through the strained tubules (Fig. 3-6). Transient fluid movement may stimulate nerve endings in either dentinal tubules, predentine, or the subodontoblastic plexus. The speed and volume of moving fluid may both contribute to the pulpal response. First, rapidly applied forces (as in our experiment) move fluid more rapidly and might be expected to enhance a response. Second, the more a given force is localized on a tooth the larger and more localized is the pressure increase, and the larger the local strain and the volume of local fluid movement. If the same force is spread over a larger area the stress is reduced, but more widespread, and local flow is reduced thereby reducing the response.

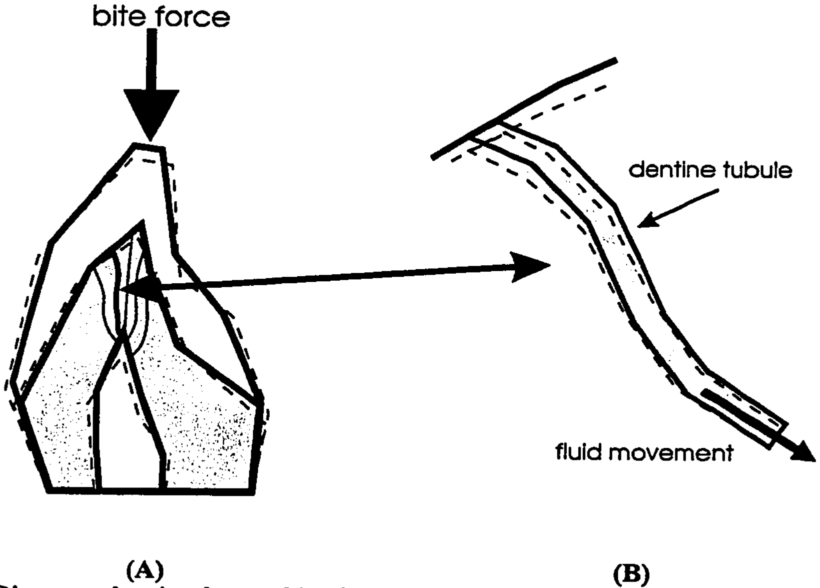


Figure 3-6 (A) Diagram showing how a bite force might deform dentinal tubules especially those around the load point. (B) An enlarged dentinal tubule close to the cusp tip is distorted and fluid is displaced into the pulpal end of the tubule. (Dashed lines represent the distorted condition)

The function of pulpal mechanoreceptors, if they exist, may be to protect a tooth by monitoring whether potentially harmful pressure might be used to break food. Excessive force as opposed to pressure would be monitored by periodontal mechanoreceptors. But pulpal mechanoreceptors could also be used to improve bite performance. For a given bite force the pressure on a nut, for example, can be increased by reducing the area of contact between it and the incisors. By encouraging an individual to reposition the nut, the mechanoreceptors could be used to increase the pressure on the nut without increasing the force on the tooth. Finally, most food substances are anisotropic and yield more readily to force in a particular direction. A combination of pulpal and periodontal mechanoreceptors might more readily find the most efficient position and orientation to place food between the incisors.

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CHAPTER 4

DISCRIMINATION OF HARDNESS BY HUMAN TEETH APPARENTLY NOT INVOLVING PERIODONTAL RECEPTORS[1]

(Paper #3)

J. Paphangkorakit and J. W. Osborn

INTRODUCTION

The hardness or softness of food affects the way it is eaten. When eating harder food, for example, a more horizontal chewing path is used (Aukes *et al.*, 1989; Huang *et al.*, 1993), the masticatory sequence is longer (Thexton *et al.*, 1980; Takada *et al.*, 1994), there are more silent periods in the masseter muscles (Watt *et al.* 1976), the temporalis and masseter muscles are more active and the bite force is larger (Slagter *et al.*, 1993) and there is greater stress on the cranium (Iwasaki, 1989). Finally, a short burst of digastric muscle activity is observed during jaw closing when rabbits eat hard food but not when eating soft food (Haraguchi and Yamada, 1992).

The above differences suggest there exists a sensory mechanism for detecting the hardness of food so that chewing is appropriately modified. Mioche and Peyron (1995) showed that participants could discriminate between elastic, plastic and brittle materials by biting on them with their incisors. The force used to detect the hardness was well correlated with the mechanical property of the materials. This implies that the sense of hardness might have been mediated by periodontal receptors. But placing a metal disc between the sample and the teeth reduced the ability to detect the difference. There is no

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[1] A version of this chapter has been published. Paphangkorakit J. and Osborn J. W. (1998), *Archs oral Biol* 43, 1-7.

reason to think that periodontal receptors were affected by the disc so that some other detector mechanism seems to have been compromised.

Several recent studies have tentatively identified receptors other than nociceptors in the pulps of teeth (Dong *et al.*, 1985; Olgart *et al.*, 1988; Matthews and Vongsavan, 1994). Paphangkorakit and Osborn (1997*b*) concluded that pulpal mechanoreceptors may exist and be used to detect differences in the pressure applied to the crown of human incisors. We test here whether such receptors might be involved in detecting differences in the hardness of materials.

MATERIALS AND METHODS

The magnitude and direction of an incisal bite force were measured by a transducer made of thinner metal than that used in the previous study (Paphangkorakit and Osborn, 1997*b*). The thinner new transducer enabled smaller forces to be more accurately measured but, because it was more fragile, the maximum force was limited to about 150 N. The magnitude and direction of the bite forces were displayed on a computer screen (Osborn and Mao, 1993) which was hidden from the participant.

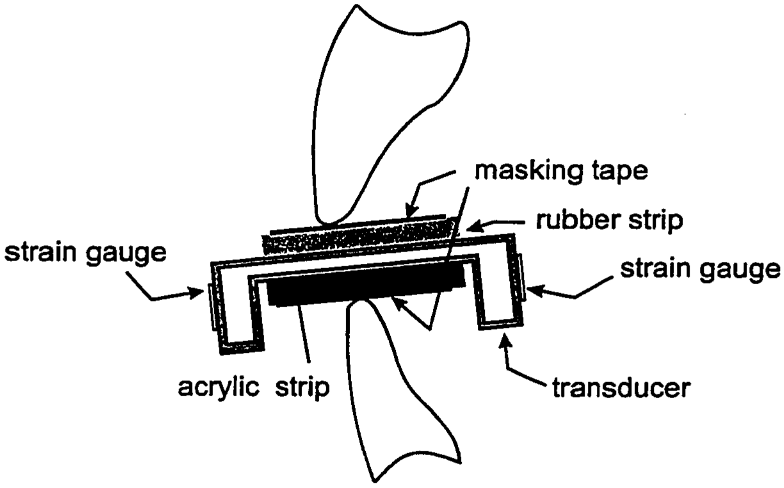
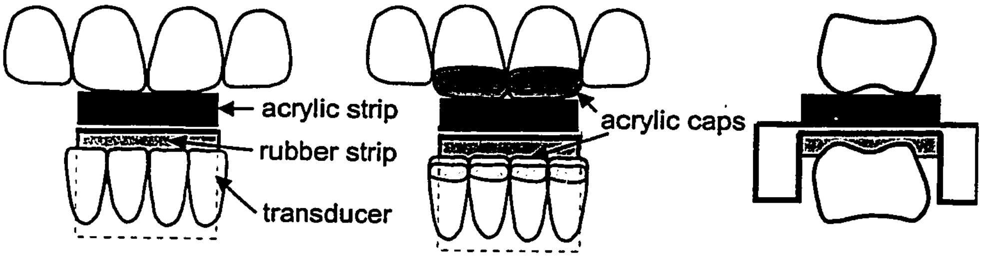


Figure 4-1 The (inverted U-shaped) transducer was sandwiched between a strip of acrylic on one side and rubber on the other side. Both were covered with masking tape to give them the same surface texture.

The study was approved by an Ethics Committee. Fifteen normal participants (11 male and four female university students), without dental, masticatory muscle and temporomandibular joint pain, participated in the study. The horizontal platform of the transducer was sandwiched between equal sized 2 mm thick strips of hard acrylic on one side and rubber, cut from a rubber bung, on the other side (Fig. 4-1).

Participants were told that one side of the platform on which they would bite was hard and the other was rubber and that their positions would be randomly chosen. Both strips were covered with masking tape to prevent participants from sensing a difference related to surface texture. The sandwich was placed so that the biting surfaces of the lower incisors were centered in the middle of the platform. The jaw was then gently closed, without lightly biting on the transducer. Participants were now told to gradually increase the bite force until they were confident they could detect the position of the harder surface, top or bottom, at which time they were to press a buzzer. They continued increasing the bite force until the computer screen, hidden from the participant, showed the bite force had reached 150 N, the safety limit for the transducer. Each participant undertook three different experiments during the same session. Each experiment included ten tests in which the positions of acrylic and rubber were randomly chosen. Unknown to the participant the rubber was five times each on the top and the bottom. The experiments involved (1) bare incisors, (2) incisors capped with acrylic resin overlays (~1 mm thick, Ortho Resin, Caulk. DL, USA) and (3) bare left first molars (Fig. 4-2). The sequence of the incisor experiments (bare or capped) was randomized whereas the molar experiments were always done last.

The results, either correct or incorrect identification or "don't know", were recorded. *Accuracy* was defined as the percentage of the correct answers from ten tests (mistaken identification and "don't knows" were both considered to be failures). The level of force being used when the participant pressed the buzzer was defined as the *(subjective) detection threshold* and was also recorded. When comparing detection thresholds, data were used in two ways. First, all detection thresholds were averaged and compared, regardless of whether the detection was correct or incorrect. Second, averages were only used for those participants whose accuracy was greater than 75% (*e.g.* Williams *et al.*, 1988; Mioche and Peyron,1995; Srinivasan and LaMotte, 1995).



(A) (B) (C)

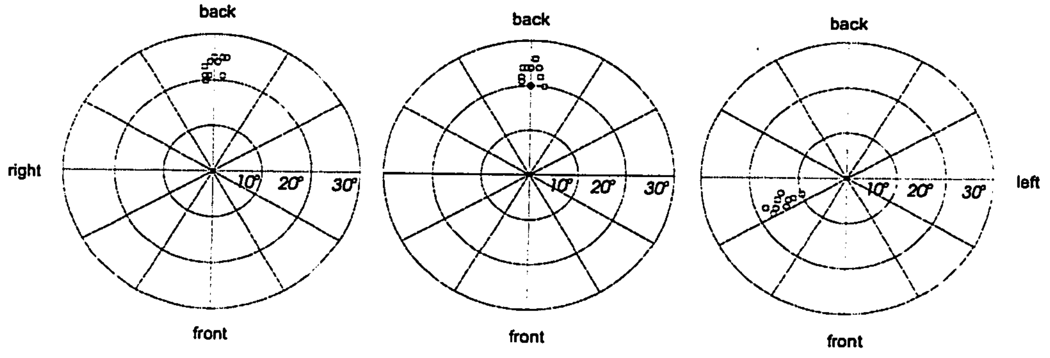
Figure 4-2 Diagrams in the frontal plane showing the hardness discrimination test with (A) bare incisors, (B) incisors capped with acrylic resin and (C) bare molar teeth.

In addition to recording the direction and magnitude of bite force when the hardness was detected and the accuracy of detection, participants were asked to describe how they thought they could feel the difference between surfaces. Early in the study some reported that they could feel the transducer moving against their lips. The incisor experiments were modified, and repeated for these early participants, to prevent contact between the transducer and the lips.

Differences in detection thresholds were analyzed intra-individually by independent t-tests. Differences in accuracy were analyzed by paired t-tests. Differences, if any, in the bite force direction within an experiment or between bare and capped incisors were tested by the Watson-Williams test for directional data (Mardia, 1972, see also Appendix F).

RESULTS

Participants did not significantly change their bite force direction during any experiment ($p > 0.05$) nor was the direction different when biting on bare and capped incisors ($p > 0.05$). All participants bit nearly vertically on their incisors in the frontal plane (Figs. 4-3A, 4-3B) but there was considerable variation in the direction in the sagittal plane (15° to 26° backward). Molar bite forces were directed somewhat medially (Fig. 4-3C).



(A) (B) (C)

Figure 4-3 Polar plots of bite force directions used during the hardness discrimination task between (A) bare incisors, (B) capped incisors and (C) bare molars in one participant. (○ = rubber on top, □ = acrylic on top). Note that the measured direction is related to the position of the transducer in relation to the incisal edges.

Average detection thresholds and the accuracy for all three experiments are summarized in Table 4-1. All participants except one performed well (accuracy >= 80%) when identifying the positions of the soft and hard surfaces with bare incisor teeth (mean 91% correct, SD = 11%). Performance was significantly reduced ($p < 0.01$) for capped incisors (mean 79% correct, SD = 19%) and ($p < 0.001$) for bare molars (mean 57% correct, SD = 24%).

For bare incisors the mean (subjective) detection threshold for all participants, whether detection was correct or incorrect, was 39 N (SD =18 N, range 13-71 N), for capped incisors it was 60 N (SD =18 N, range 31-89 N) and for molars it was 85 N (SD = 18 N, range 46-109 N). In six out of the 15 participants the mean detection threshold for capped incisors was significantly larger than that for bare incisors ($0.002 < p < 0.01$). Differences were not significant in the remaining cases although, with a single exception,

Table 4-1 Summary of the average (subjective) detection thresholds and %accuracy (with standard deviations in small figures) in all participants biting with bare incisors, capped incisors and bare molars. Each figure is an average of ten tests (n =10). The average threshold in each individual is calculated from both correct and incorrect detections. The italicized threshold values are those from participants who made > 75% correct answers. The percentage of "don't know" responses is also shown in brackets.

	Average Threshold (Newtons)				%Accuracy		
Participant	bare incisors	capped incisors	bare molars	Participant	bare incisors	capped incisors	bare molars
T10	*26*	67*	57*	T10	100 (0)	60 (0)	30 (10)
	13	17	23				
M5	*45*	47	101*	M5	90 (0)	80 (0)	50 (0)
	21	21	37				
E5	70	64	109*	E5	100 (0)	80 (10)	100 (0)
	20	11	39				
L8	41	44	96*	L8	90 (0)	100 (0)	40 (30)
	8	8	31				
Bd1	20	53	80*	Bd1	60 (30)	40 (60)	50 (50)
	9	38	46				
K10	15	87*	87*	K10	100 (0)	80 (10)	80 (0)
	7	13	28				
D8	71	81	87	D8	90 (10)	80 (20)	60 (30)
	16	12	17				
Dv1	32	34	46	Dv1	90 (10)	90 (10)	60 (10)
	12	14	32				
Ch1	46	73*	103*	Ch1	100 (0)	60 (10)	50 (20)
	12	18	16				
Jn1	60	89*	77	Jn1	80 (10)	50 (10)	80 (0)
	21	15	12				
Jk2	34	63*	104*	Jk2	80 (20)	90 (10)	70 (0)
	17	9	20				
Cm1	46	49	90*	Cm1	90 (10)	100 (0)	80 (20)
	8	23	18				
Cg1	20	31*	61*	Cg1	100 (0)	100 (0)	70 (10)
	6	5	12				
Yi3	13	43	81*	Yi3	100 (0)	100 (0)	10 (20)
	5	10	27				
An1	41	69	93*	An1	100 (0)	70 (30)	20 (70)
	20	6	5				
MEAN	**39**	**60**	**85**	**MEAN**	**91**	**79**	**57**
SD	**18**	**18**	**18**	**SD**	**11**	**19**	**24**

*significantly different ($0.002 < p < 0.01$) compared with bare incisors (independent t-test) the average threshold was always larger when the incisors were capped than when they were bare. The threshold was significantly larger for molars in 12 participants ($0.002 < p < 0.01$). When tested individually (Mann-Whitney test) there was no significant difference in the detection threshold whether the soft or the hard strip was on the top or the bottom ($p > 0.1$).

The number of detection thresholds which could be compared was considerably reduced when only experiments in which participants had an accuracy > 75% were used. When comparing incisor performance only four out of the 12 valid participants used significantly larger detection thresholds when the incisors were capped. Of the remaining eight participants, most used larger average detection thresholds but the differences were not significant. When comparing performances between bare incisors and molars four out of the six valid participants used significantly larger detection thresholds when using molars.

Almost all participants thought they could distinguish between the harder and softer sides of the sandwich because their teeth were "sinking" into the rubber but not into the acrylic. When asked, eight participants felt detection was easier with smaller bite forces, five felt it was easier with larger bite forces and two did not notice any difference.

DISCUSSION

It is obvious that when using bare incisors participants could correctly distinguish if the hard surface was on top of the sandwich (Table 4-1) even without statistics. We defined accuracy as the percentage of correct answers and used this measure to show statistically significant differences in the performance of participants when using bare incisors, capped incisors and bare molars. A poorer performance was not necessarily related to mistaken identities but due to the number of "don't knows". Performance in the sense of right *versus* wrong (ignoring "don't knows") was much better. For example, participant Bd1 with accuracies of only 40% on capped incisors and 50% on molars made only one mistake throughout the 30 tests, and that was when using bare incisors. Thus the difference between numbers of incorrect answers when using bare and capped

incisors did not reach statistical significance (p = 0.062). Nor was the difference between numbers of "don't knows" significant (p = 0.141). These differences were, however, significant (p = 0.0016 and p = 0.0036 respectively) when comparing between performances using bare incisors and bare molars.

An inadvertent difference in bite force direction as the cue for hardness discrimination (such as biting more backward when the rubber was below, for example) was excluded because the direction did not change significantly during any test. Moreover, it has been shown that discrimination is not affected whether a load is applied in a labial direction or down the long axis of an upper central incisor (Bowman and Nakfoor, 1968).

Hardness (or softness) has been defined in several different ways [see Peleg (1980) for a review]. Srinivasan and LaMotte (1995) described two basic mechanisms in skin for discriminating between the hardness or softness of materials. "Tactile information" is related to differences in force and pressure at the contact between the fingerpad and an object. "Kinesthetic information" is related to the sense of position and movement. For a given load the finger joints move the finger further into soft than hard material. The combination of both types of information is normally used to detect a variety of hardnesses, although kinesthetic information is not always necessary.

Detecting the hardness of food gripped between the upper and lower teeth probably involves assessing, directly or indirectly, the amount the teeth move into the food when a known load is applied. It must be emphasized that, unlike skin, periodontal tissues can only be used to assess *forces* applied to the crown of a tooth, not *pressures*. When participants successfully use their incisors to detect the difference in hardness between elastic, plastic and brittle materials (Mioche and Peyron, 1995) they probably compare the amount of jaw movement, with the force detected by periodontal mechanoreceptors. The lower jaw moves further and the receptors detect less force when incising softer material. This technique could not be used by our participants because (1) the jaw moved the same distance whether the rubber was on the top or the bottom and (2) the magnitude of the bite force was the same on both upper and lower incisors. Therefore upper and lower periodontal mechanoreceptors received the same information and the teeth moved the same distances into their sockets whether the rubber was on the top or the bottom (Fig. 4-4B, 4-4C).

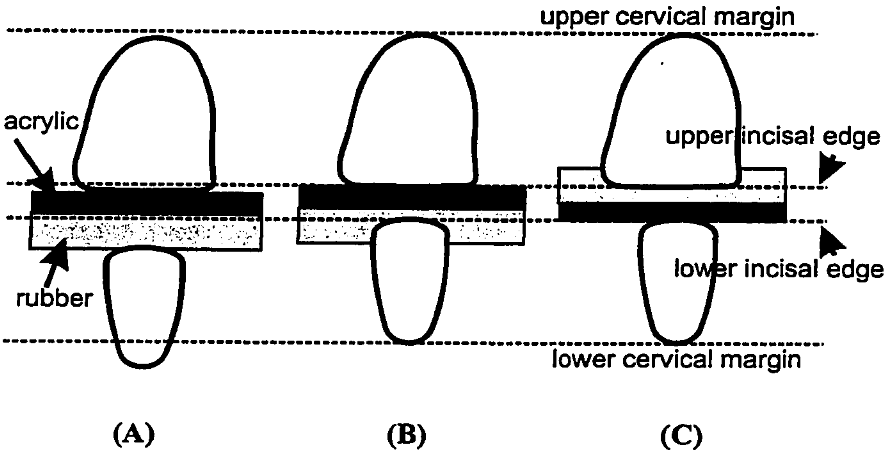


Figure 4-4 Upper and lower incisors are viewed from in front while biting on the sandwich of acrylic and rubber. (A) At the start of the experiment participants lightly held the sandwich between their incisors. (B) The bite force pushed each incisor into its socket. The lower tooth was pushed into the rubber as the jaw was closed on the sandwich. The highest and lowest dotted lines show the new height of the cervical margin of each tooth. The other dotted lines show the new heights of the upper and lower incisal edges. Note that the lower incisor moves up because the jaw is closed. (C) The situation when the sandwich is reversed. Note that the sandwich moves further up when the rubber is on top.

A simple experiment may help to explain why the periodontal mechanoreceptors could not be used to detect which surface was on top. A block of soft sponge was glued to a block of hard plastic (Fig. 4-5, see also Appendix G). The combined block was covered with masking tape to prevent participants feeling the surface texture. Blindfolded participants were asked to squeeze the block between thumb and forefinger and to guess which was pressing on the sponge, thumb or forefinger. Every participant guessed correctly every time. The surfaces of the block were now covered with thick glass microscope slides and the experiment repeated. Every participant found it impossible to detect the sides of the sponge and plastic[2] (Fig. 4-5B). The results of this experiment may

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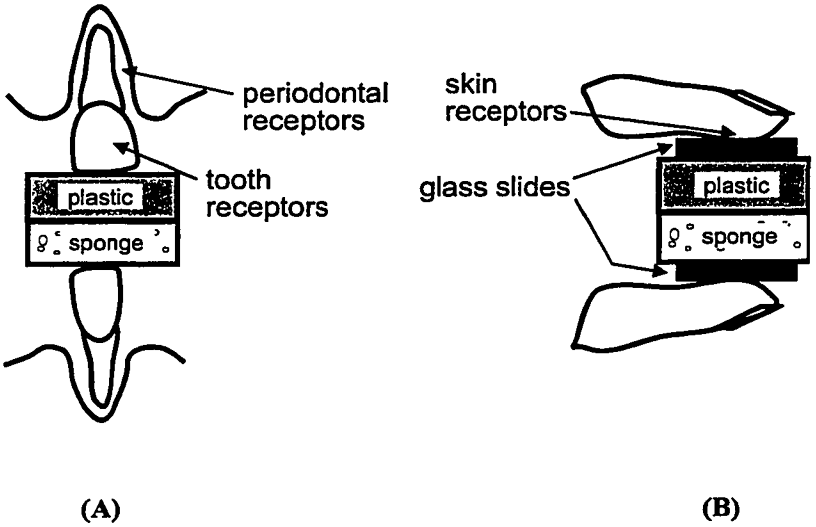
[2] Results shown in Appendix H

seem intuitively obvious but are awkward to explain. The hard tooth crown and the hard glass slide accurately transmit force to receptors in the periodontal ligament and the dermis. But the force is the same on the top and the bottom of the block and so it cannot be used to distinguish whether the hard surface is on the top or the bottom. The pressures on the tooth crown and on the glass are, however, different. The pressure is smaller on the soft side of the sandwich because it is spread over a larger area. This information cannot be transmitted through the glass and so participants cannot distinguish any difference between top and bottom. Unlike the glass, the tooth crown does contain sensory tissue. We conclude that the difference in pressure (in Fig. 4-5A), hence difference in hardness, is detected by receptors inside the tooth which are stimulated by the stressed dentine above.



(A) (B)

Figure 4-5 An analogy showing that only teeth, not periodontal tissue, could be used to discriminate between the soft sponge-hard acrylic assembly. Note that subjects found it impossible to discriminate between the acrylic and sponge with their fingers (B) but failed only 13 times out of 150 when using their teeth (A). For details see text.

The flat acrylic side of the sandwich did not accurately fit the incisal edges. It is to be expected that, at a microscopic level, an edge only touched the acrylic at high points. The pressure (force per unit area) on the incisor biting on the acrylic was therefore much larger than that on the incisor being embedded into the rubber. We think that participants were able to detect the softer surface because, although the periodontal input was the same for upper and lower incisors (the bite force was the same), the pressure on the tooth biting on the acrylic was much larger than that on the one cushioned by the rubber. The tooth subjected to the higher pressure was recognized as the one biting on the harder surface. In much the same way the difference in pressure was used by blindfolded participants to recognize whether the thumb or forefinger was pressing the sponge.

Another possible source of information, and one which our earlier participants appeared to use, was the movement of the sandwich. The sandwich moves upward less when the rubber is on the bottom (*cf.* Figs. 4-4B, 4-4C). It will be recalled that before finalizing the experimental protocol the results of some early tests were rejected because participants were able to detect the harder surface by feeling with their lips whether the sandwich moved up with the lower jaw and incisor (in which case the harder surface faced down).

The poorer performance when biting with capped incisors as opposed to bare incisors, although it did not always reach statistical significance, may be explained because the difference in pressure was more difficult to detect when the incisors were capped. The detection threshold was higher and accuracy was significantly lower when biting on molars. Although Bonaguro *et al.* (1969) did not find a significant difference in the ability of mandibular incisors, canine and first premolars to discriminate between bite forces, Coffey *et al.* (1989) found that incisors discriminated better than first molars. Additionally, both Kampe *et al.* (1987) and Torneck and Howley (1989) found that incisor perception was better than that of more posterior teeth.

In a study of maximum incisal bite forces, Paphangkorakit and Osborn (1997*b*) concluded that pulpal mechanoreceptors monitor the pressure on an incisor. Similar mechanoreceptors, but with smaller thresholds, would probably be the source of the input used by participants to detect whether the soft surface was on the top or bottom of the sandwich. Our participants used a range of thresholds which was similar to the range of forces used by Mioche and Peyron's (1995) participants to compare the hardness of materials. These pulpal mechanoreceptors apparently have smaller thresholds, in the range of 10-70 N forces applied to the surface of the tooth, than those which were involved in limiting the maximum bite force (110-370 N; Paphangkorakit and Osborn, 1997*b*) on incisors.

Eight of our participants thought it was easier to discriminate between the soft and hard sides of the sandwich with smaller bite forces while five thought it was easier with larger bite forces. It is easier to discriminate between small forces (*e.g.* 10 and 15 N) than between large forces (*e.g.* 1010 and 1015 N) (Bonaguro *et al.*, 1969; Coffey *et al.*, 1989). It will be recalled that after detecting the softer material participants continued increasing the bite force until it reached 150 N. Those who thought it easier to discriminate at larger forces may merely have been confirming what they had already identified at a smaller bite force. The subjective improvement in discrimination is unlikely to have been due to the teeth being closer together at larger bite forces because jaw separation does not affect the ability to discriminate between different bite forces (Williams *et al.*, 1988).

Many different detectors can be used in monitoring bite force. They are mechanoreceptors in the mucosa and tongue, in the periodontal ligament (*e.g.* Hannam, 1969; Hellsing, 1980; Williams *et al.*, 1985, 1987; Waltimo and Könönen, 1994), periosteum (Jacobs and van Steenberghe, 1993), tooth pulps (Paphangkorakit and Osborn, 1997*b*), and muscle spindles (Williams and LaPointe, 1987). For the present study we eliminated the use of soft tissue mechanoreceptors by allowing the participants to use only their teeth to detect hardness. The experimental method also eliminated the possibility of using the periodontium, periosteum and muscle spindles to help detect which material was on top of the transducer sandwich. The only variable which was changed during each test series was the pressure on the incisal or occlusal surfaces of teeth. Peleg (1980) showed it is theoretically necessary to deform a hard sensor to detect differences in hardness between hard substances and a soft sensor to detect differences in soft substances. Only a hard tooth, as opposed to a soft periodontal ligament, could be used to detect differences in the hardness of hard foods. We conclude that mechanoreceptors inside the tooth pulp were in some ways used to detect which material was on top of the sandwich.

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CHAPTER 5

EFFECTS ON HUMAN MAXIMUM BITE FORCE OF BITING ON A SOFTER OR HARDER OBJECT[1]

(Paper #4)

J. Paphangkorakit and J. W. Osborn

INTRODUCTION

Several studies have reported the possibility that the teeth of those animals investigated may contain low threshold mechanoreceptors (Dong *et al.*, 1985; Olgart *et al.*, 1988). The central destination of impulses was unknown although some of the cell bodies may be in the mesencephalic nucleus (Chiego *et al.*, 1980). Electrically stimulating human incisor teeth at a non-painful level elicited inhibitory effects in the masseter muscle (Matthews *et al.*, 1976). This again suggests that tooth pulps may contain sensory receptors not involved with the sensation of pain.

Subjects are able to increase their maximum bite force (MBF) if their incisors are covered with an acrylic cap (Paphangkorakit and Osborn, 1997*b*). We explained this result by concluding that the pulps of the teeth contain high threshold mechanoreceptors. These receptors may subconsciously protect a tooth from potentially damaging bite forces by monitoring the stress (force per unit area) on the dentine. When the tooth is capped the bite force is spread over a larger area instead of being concentrated at its incisal edge. This reduces the stress on the dentine, which in turn permits a larger bite force.

The hypothesis that tooth pulps contain mechanoreceptors is supported by the following observation. Subjects can detect whether their incisors are biting on hard

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[1] A version of this chapter has been published. Paphangkorakit J. and Osborn J. W. (1998), *Archs oral Biol* 43, 833-839.

acrylic or soft rubber when the input to all receptors, except those assumed to exist inside the tooth pulps, remains unchanged (Paphangkorakit and Osborn, 1998). We concluded that a person (consciously) detects the reduction in the stress on a tooth when biting with the same force into soft rubber as opposed to hard acrylic. The incisal edge sinks into the rubber. This spreads the load over the tooth crown and, for the same bite force, reduces the stress on the compressed dentine.

If the above conclusions are correct subjects should be able to develop a larger MBF when biting on a softer surface because the load is spread over a larger area and the dentine is therefore less stressed. But if the tooth is covered with a crown the load is already spread and there should be little or no difference in the MBF whether biting on a hard or a soft surface. We test here these predictions.

MATERIALS AND METHODS

A 2-mm thick piece of tough rubber was glued over the end of one side of a 1.5 cm wide thin metal spatula. A 2-mm thick piece of acrylic (Dura Lay, Dental Mfg, IL, USA) was glued over the other side (Fig. 5-1A). The sandwich was wrapped in masking tape to give all surfaces the same appearance and texture. Participants were asked to incise as hard as possible on the sandwich. By rotating the sandwich they could be made to bite with their upper incisors on either the hard acrylic or the soft rubber surface without being aware of any change in the experimental procedure. They would only be able to detect a difference by means of receptor mechanisms associated with either the jaws and/or the teeth.

The study was approved by the human ethics committee. Fifteen university students aged 21-36, without existing tooth and jaw muscle pain, took part in each of two experiments. Having given informed consent, alginate impressions were taken and plaster models of upper and lower jaws were made for each participant. A U-shaped transducer (Paphangkorakit and Osborn, 1997*a*) was fitted on to the cast of the lower incisors by means of an acrylic overlay. The transducer was positioned over the lower incisors with

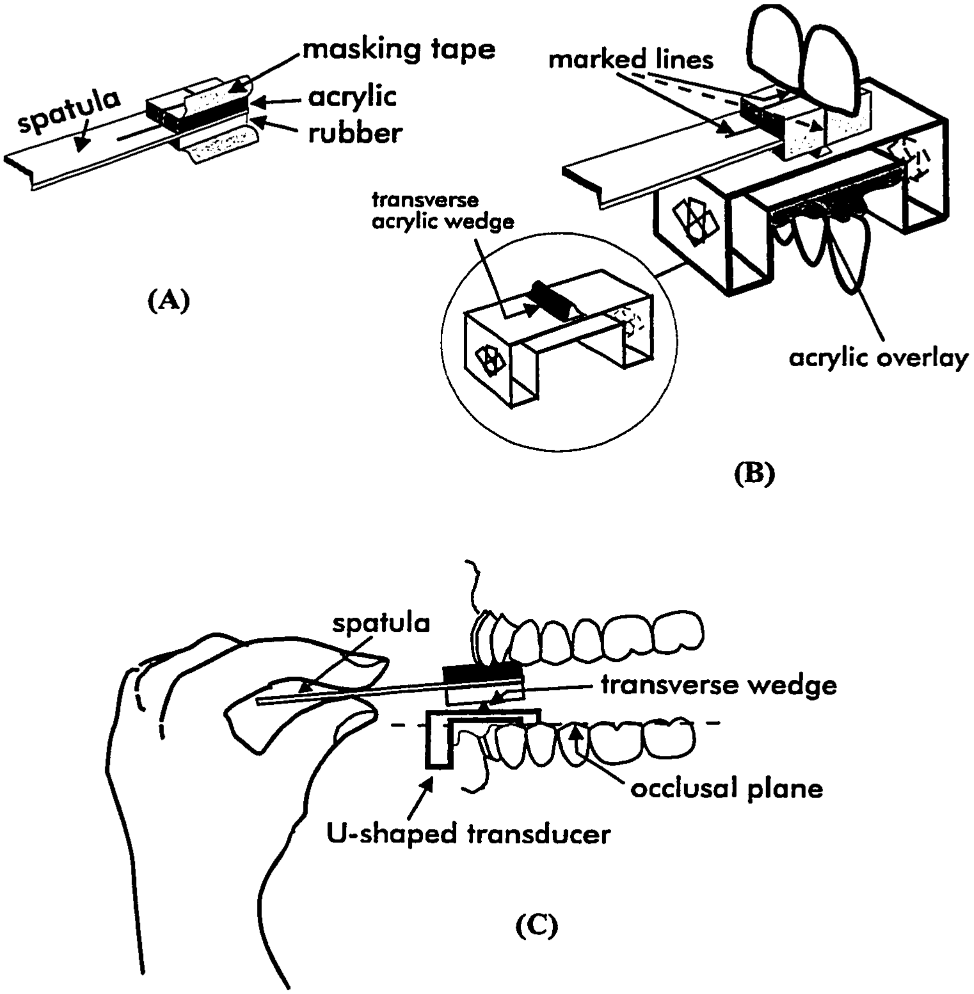

Figure 5-1 Diagrams showing experimental set-up. (A) The bite block. The acrylic and the rubber strips are glued to opposite sides of the metal spatula creating a bite block sandwich. The sandwich is wrapped with masking tape. (B) The U-shaped bite force transducer with the bite block symmetrically placed on top. The transducer is fitted over the lower incisors with an acrylic overlay. Note the upper and lower teeth during biting are edge-to-edge. Inset shows the transducer with the transverse acrylic wedge on top. The edge of the wedge is shaped like that of the upper incisor. (C) A lateral view of the transducer *in situ*. The free end of the spatula is lightly held by the participant.

its centre between the upper incisors and its horizontal platform as close as possible parallel to the lower occlusal plane (Fig. 5-1C). The directions of bite forces recorded from the transducer were therefore the same as those with respect to the lower occlusal plane. A wedge made from Dura Lay and shaped like the upper incisal edge was cemented to the centre of the upper surface of the transducer (Fig. 5-1B, C). This ensured that the bite force from the upper incisor, which was transferred to the tip of the wedge, was directed down the centre of the transducer in the frontal plane. Lines drawn transversely across the centre of both acrylic and rubber sides of the sandwich helped position the lower jaw so that the upper incisor maintained a constant antero-posterior relation to the lower jaw.

The transducer was fitted over a participant's lower incisors. Next, the bite sandwich was placed symmetrically in position with the line marked on either the hard or soft surface aligned with the line marked on the centre of the acrylic wedge on the transducer. Participants were guided to move their lower jaw until the transverse line marked on the upper side of the sandwich made contact with the edges of the upper incisors. They stabilized the block by lightly holding its handle (Fig. 5-1C). When they felt comfortable they were directed to bite rapidly as hard as possible. The direction and magnitude of the MBFs were measured with the transducer.

During earlier studies several participants complained of slight discomfort after they had exerted a maximum bite force (MBF) on the transducer more than about 12 times. We therefore set a limit of 12 on the number of maxima for a single recording session. Unfortunately some measurements had to be rejected because participants, for reasons unknown, occasionally used an unusual bite direction. A change in the direction of the bite force can greatly affect the MBF because it changes the moment arms and mechanical advantage of the system (Osborn, 1996). In order to standardize the procedure we therefore collected measurements of the first six MBFs whose associated bite directions clustered within a cone of 4^{o}, the resolution of the transducer obtained during calibration. As far as possible the sequence of hard and soft sides was randomly chosen but towards the end of an experiment we arranged things so that ultimately we obtained three MBFs with upper incisors biting on the soft side of the bite block and three biting on its hard side.

On a separate day, in order to limit the number of maxima produced in a single session, the whole experiment was repeated with the difference that the upper incisors were covered with a full acrylic crown (made from Ortho-Resin, Caulk, DL, USA) about 1 mm thick. Only 12 of the original 15 participants were available for this second experiment. Three new participants volunteered.

The percentage change in MBF when biting on (soft) rubber or (hard) acrylic was calculated in both bare-tooth and capped-tooth experiments. The chi-square goodness-of-fit method was used to test if the percentage changes were normally distributed (see Table H-4). A paired t-test for statistical differences was applied if the distribution was normal. Possible differences in bite directions between biting on acrylic and rubber were tested by the Watson-Williams test for directional data (Mardia, 1972).

RESULTS

Bite directions ranged from 14° forward to 16° backward (Tables 5-1, 5-2) and from 12° right to 11° left in the frontal plane. The change of biting surface from rubber to acrylic did not cause a significant change in the bite direction used by any participant ($p > 0.05$; Watson-Williams test).

Participants usually achieved their MBFs within 2-3 seconds. When asked if they had noticed any differences, 15 out of the 18 participants said they did not know they had been biting on either a hard or a soft surface. The accuracy of the remaining three participants was not tested.

The percentage changes in MBF were found to be normally distributed in both bare and capped tooth experiments ($p > 0.05$). The average MBFs with bare incisors biting on rubber (range 77-530 N; mean 213 N) were larger than those when biting on acrylic (range 93-460 N; mean 190 N) in 13 out of the 15 participants. The increase ranged from 3.4% to 29.0% (Table 5-1). The MBF was smaller in the remaining two participants. Incidentally these were also the participants with the smallest MBFs. The percentage increase in MBFs when biting with bare incisors on rubber and acrylic surfaces was statistically significant ($p = 0.004$; mean increase = 7.5%; SD = 13.8 %).

Table 5-1 Comparison of the average magnitude and direction of the MBFs (SDs in small figures) when bare upper incisors bit on soft rubber and hard acrylic surfaces. (F = front, B = back, L = left, R = right of the participants)

Participant	biting on rubber			biting on acrylic			% increase (rubber-acrylic)
	Average bite direction (degree)[§]		Average MBF (N)[§]	Average bite direction (degree)[§]		Average MBF (N)[§]	
	F/B (+/-)	L/R (+/-)		F/B (+/-)	L/R (+/-)		
BD	-7	0	193	-8	2	137	29.0
	1	1	5	4	1	33	
GL	7	-3	183	7	-1	143	21.9
	1	0	5	2	1	5	
JK[†]	13	2	267	9	1	220	17.6
	2	1	12	1	0	8	
RA[*]	8	-8	140	6	-3	120	14.3
	1	0	14	2	1	8	
AL	8	1	530	7	1	460	13.2
	2	1	29	2	1	43	
JI[*]	7	1	150	3	0	133	11.3
	1	3	29	2	3	24	
J	-3	6	383	-1	3	340	11.2
	2	0	17	1	2	8	
AN	2	-12	110	2	-12	100	9.1
	1	5	14	1	1	8	
M	-8	11	503	-9	10	460	8.5
	0	1	9	2	3	22	
E[†]	7	3	160	6	2	147	8.1
	2	0	8	0	1	12	
CM[†]	2	2	163	4	-2	153	6.1
	2	0	17	2	0	17	
LI[*]	-1	2	140	-1	4	133	5.0
	0	3	14	0	2	9	
W	-6	-2	117	-9	-2	113	3.4
	1	1	25	2	2	24	
L	-8	3	80	-9	5	93	-16.3
	5	1	10	2	2	5	
D	-1	2	77	1	3	100	-29.9
	1	1	5	0	1	22	
						MEAN % increase	**7.5**
						SD	**13.8**

[§] Average of three measurements. [†] Participants who took part in only one session. [*] Participants who did not significantly change their bite directions between two sessions.

Table 5-2 Comparison of the average magnitude and direction of the MBFs (SDs in small figures) when capped upper incisors bit on soft rubber and hard acrylic surfaces. (F = front, B = back, L = left, R = right of the participants)

	biting on rubber			biting on acrylic			% increase (rubber-acrylic)
Participant	Average bite direction (degree)[§] F/B (+/-)	L/R (+/-)	Average MBF (N)[§]	Average bite direction (degree)[§] F/B (+/-)	L/R (+/-)	Average MBF (N)[§]	
J	4	5	443	3	0	357	19.4
	1	1	52	1	0	5	
BD	-16	-3	180	-13	-2	157	12.8
	3	1	22	2	0	25	
K[†]	5	-3	225	6	-3	205	8.9
	0	0	15	0	1	5	
AN	11	-9	153	14	-8	143	6.5
	2	1	33	1	1.7	21	
LI[★]	-1	2	127	-2	7	120	5.5
	5	3	9	1	2	8	
RA[★]	5	-6	167	3	-6	160	4.2
	1	2	9	2	1	16	
M	-12	7	490	-10	10	473	3.5
	1	1	16	1	1	5	
GL	5	-4	187	5	-4	183	2.1
	1	1	19	2	1	29	
AL	10	-3	427	8	-1	423	0.9
	2	1	33	2	0	66	
JI[★]	3	0	133	3	-2	133	0.0
	1	4	5	2	1	17	
D	4	4	120	2	4	120	0.0
	2	0	8	0	0	16	
CS[†]	1	-3	150	3	0	150	0.0
	1	1	0	4	2	50	
DV[†]	6	2	163	2	3	177	-8.6
	2	1	21	1	2	19	
W	-12	-5	83	-9	-4	97	-16.9
	2	2	9	1	2	17	
L	-10	-1	60	-12	0	77	-28.3
	1	1	16	2	0	5	
				MEAN % increase			**0.7**
				SD			**11.2**

[§], [†], [★] see Table 5-1

The MBFs with capped incisors biting on rubber (range 60-490 N; mean 207 N), as opposed to acrylic (range 77-473 N; mean 198 N), were larger in nine participants (range 0.9% to 19.4%) (Table 5-2), smaller in three participants (range -8.6% to -28.3%) and unchanged in three participants. The percentage increase in MBFs when biting with capped incisors on rubber as opposed to acrylic surfaces was not statistically significant ($p = 0.18$; mean = 0.7%; SD = 11.2%).

In conclusion there was a statistically significant average percentage increase in MBFs when biting with bare incisors. The increase did not reach the significance level ($p > 0.05$) when the incisors were covered with crowns.

DISCUSSION

The decreased MBF when biting with bare incisors on hard acrylic as opposed to soft rubber might have been caused by central inhibition related to discomfort or anxiety, for example. We have rejected this possibility because 15 out of the 18 participants said they did not know whether they were biting on a hard or a soft surface. The probability that the difference was due to chance is remote ($p = 0.004$). We conclude that the difference was best explained by a reflex response based on input from as yet unidentified receptors.

The receptors indirectly responsible for the observed differences in MBF could be inside the tooth (pulpal mechanoreceptors) or outside it (*e.g.* muscle spindles or mechanoreceptors in the periodontal ligament, temporomandibular joint, or sutures). Consider the three different hypothetical conditions (X,Y, Z) in Fig. 5-2 where two hard blocks and one soft block are placed between the upper and lower incisors. X and Y could represent the two actual experimental situations (X equivalent to A and Y equivalent to B). Z has been included to make the point that for a given bite force the response from any receptors outside the teeth, including the upper and lower periodontal ligaments, is the same no matter where the soft block is placed (see also Paphangkorakit and Osborn, 1998). This leaves receptors inside the teeth as the only possible source of an input which could have been reflexly responsible for the observed differences in MBF when biting with bare teeth on soft and hard surfaces. Note that the environment of the lower incisors was the same for all experiments. Only the environment of the upper incisors was varied.

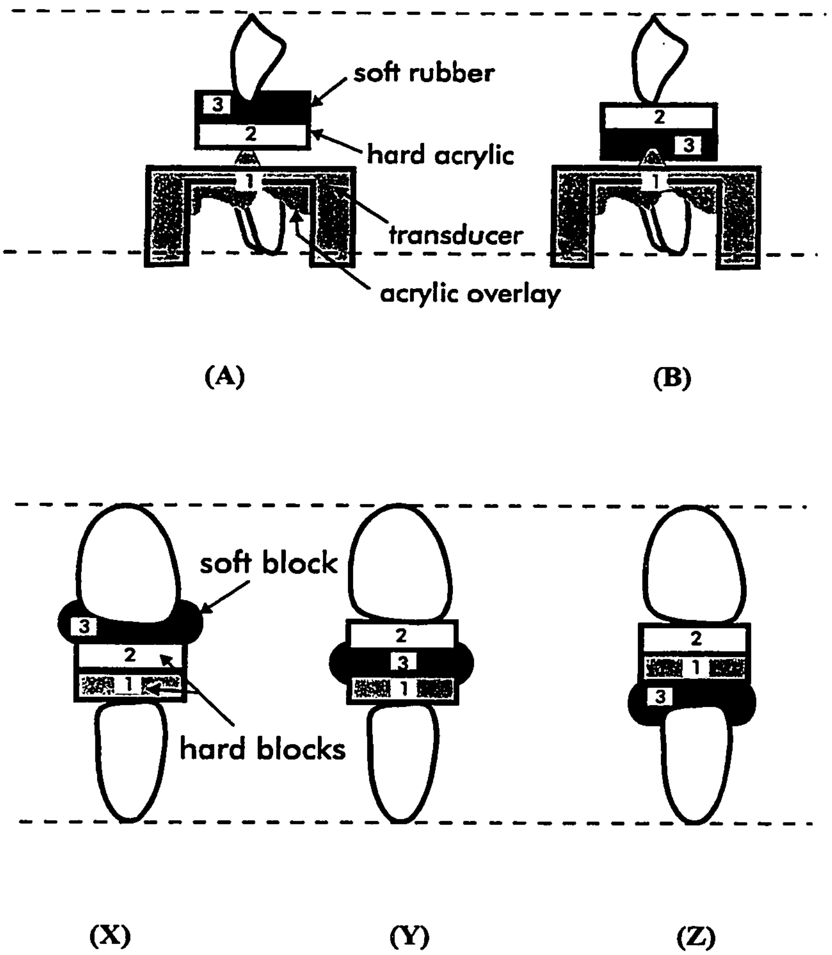

Figure 5-2 Three regions of different hardness in the experiments (A, B) and corresponding simplified diagrams (X, Y) can be identified between upper and lower incisor crowns. The lower incisors were covered with a hard acrylic overlay glued to the transducer (block 1) and the bite sandwich consisted of hard and soft blocks (blocks 2 and 3). Changing the position of the soft block (X, Y and Z, although condition Z was not tested) has no effect on any of the environment related to creating a bite force except that of the tooth crowns (note the positions of the upper and lower teeth marked by dotted lines). For a given bite force the input to all other receptors (*e.g.* in the periodontal ligament or jaw muscles) is the same no matter where the soft block is placed. The stress (force per unit area) on a tooth crown, however, is reduced when biting on soft rubber. Stress in the enamel-dentine complex could only be monitored by receptors inside the tooth.

We have suggested (Paphangkorakit and Osborn, 1997*b*) that pulpal mechanoreceptors may monitor the stress (force per unit area) on dentine. When the load was spread over a larger surface area of the upper incisor, as when biting with bare incisors on soft rubber (*i.e.* condition A in Fig. 5-2), the stress on the dentine was reduced. The reduced stress, when compared with biting on a hard surface (*i.e.* condition B in Fig. 5-2), allowed a larger MBF to be produced.

We predicted that when the upper incisor was capped the load would be spread more widely over the enamel surface instead of being concentrated on its incisal edge. The stress on the underlying dentine would now be much the same whether the capped tooth was biting on rubber or acrylic. In fact the average of the changes in MBF was less than 1% (larger average MBF with rubber) when the teeth were capped. The difference was not statistically significant and this tends to support the above hypothesis. It is, however, possible that such a small difference was real but we had too few subjects for the result to reach statistical significance. Biting on rubber may spread the load more evenly over the cap and tend to seat it more deeply than biting on acrylic. The improved seating may then more effectively spread the stress over the tooth and account for the small increase in MBF when biting on rubber.

We are reluctant to attach importance to individual results, as opposed to *global* averages, from different days because we have found (unpublished results) that measurements of a subject's performance often differ on different days but are far more consistent on a given day. The difference may be due to unrecognized differences in the experimental setup and the experimenter as well as differences in the subject related to unmeasurable factors such as motivation, well-being, muscle health, tooth status and so on. However, by considering the results from both days to be independent the overall values may be compared. The *global* average MBF increased by 8 N (from 190 N to 198 N, $p > 0.05$; independent t-test) when biting with capped incisors on acrylic compared with biting with bare incisors on acrylic. This trend is consistent with our hypothesis and confirms previous results (Paphangkorakit and Osborn, 1997*b*) although the difference was not statistically significant. On the other hand, the *global* average MBF when biting with bare incisors on rubber compared with biting with capped incisors on rubber increased by 6 N (from 207 N to 213 N, $p > 0.05$; independent t-test). Suppose the increase was real, although it was not statistically significant. It could be explained if the compressed rubber fits the incisor (spreads the load more effectively) a little better than an acrylic crown.

Some studies have shown a smaller MBF (Lund and Lamarre, 1973) or larger MBF (van Steenberghe and de Vries, 1978*b*; Orchardson and MacFarlane, 1980) when teeth are anaesthetized although others showed no change (Hellsing, 1980; Teenier *et al.*, 1991). The increased MBF, where observed, was thought to be in some way related to the removal of a periodontal input but it could equally have been interpreted as related to removal of a pulpal input. This does not account for the decrease observed by Lund and Lamarre.

Waltimo and Könönen (1994) showed that the MBF was larger when biting on the six anterior maxillary teeth than when biting on one central incisor and suggested the increase was due to spreading the load over more teeth. Although they favoured periodontal mechanoreceptors as responsible for the change in MBF, it is equally possible that pulpal receptors were responsible.

The choice between the pulpal or periodontal mechanoreceptors (or both) as a source of inhibition requires experiments where one of the inputs is eliminated. The present experiment eliminated the periodontal input because its environment was constant. This experiment now needs to be repeated using pulpless upper incisors. If the pulp is the only source of inhibition then there would be no difference in MBF whether biting on a hard or soft surface with bare pulpless upper incisors. But this experiment may be too dangerous because of the risk of fracturing the dead incisor.

The results of the present study are consistent with the hypothesis that the pulps of human incisor teeth contain mechanoreceptors whose input is subconsciously used to limit the MBF (Paphangkorakit and Osborn, 1997*b*).

Acknowledgement - The study was partially supported by the Fund for Dentistry at the University of Alberta.

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CHAPTER 6

OCCLUSAL LOADS CAUSE FLUID TO MOVE THROUGH DENTINE *IN VITRO*[1]

(Paper #5)

J. Paphangkorakit and J. W. Osborn

INTRODUCTION

It is generally believed that the movement of fluid through dentine activates sensory nerves supplying the pulps of teeth (Brännström, 1963). It is further believed that this causes the sensation of pain. In support of this hypothesis Brännström and co-workers demonstrated that fluid moved out of exposed dentine when it was blasted with a current of air (Brännström *et al.*, 1967) or covered with sugar solution (Linden and Brännström, 1967) or cut with a dental drill (Brännström *et al.*, 1968). In an *in vitro* experiment, Anderson *et al.* (1967) showed that the amount of fluid moved through dentine by an osmotic stimulus, saturated $CaCl_2$, applied to exposed dentine correlated well with the amount of pain evoked by the same stimulus applied to exposed dentine *in vivo* (Anderson and Matthews, 1967). The above studies measured volumes of fluid flow that varied from about 5,000 to 50,000 pL/s. This provides a rough indication of the volume of fluid that needs to be moved in order to activate sensory nerves in the pulp. Paphangkorakit and Osborn (1997*b*, 1998*a*, 1998*b*) concluded from a series of *in vivo* experiments involving clenched incisors that some of the sensory nerves in the pulps of human teeth are mechanoreceptors. We define mechanoreceptors as sensory nerves that respond by providing information about parameters of a mechanical stimulus without

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[1] A version of this chapter has been submitted for publication.

causing pain. We hypothesize that pulpal mechanoreceptors are activated by the movement of fluid squeezed out of compressed dentine toward the pulp. This requires that forces within the range normally applied to teeth are capable of compressing dentine and squeezing sufficient fluid (roughly 5,000 to 50,000 pL/s) toward the pulp to initiate action potentials in mechanoreceptors. The following *in vitro* experiment measures the amount of fluid that is moved by loads within the normal range.

MATERIALS AND METHODS

The pulp chamber of a tooth was connected by tubing to a capillary. Compressing the tooth squeezed fluid out of the pulp and through the capillary. The movement of fluid in the capillary was measured (Fig. 6-1).

Preparation of the tooth

Nine human teeth (five canines, two premolars and two third molars), extracted because of either periodontal problems or orthodontic treatment, were stored in 0.05% sodium azide in distilled water at 4° C. Immersing the crowns in methylene blue solution for five minutes did not reveal any fractures (a fracture line would be penetrated by the dye). The tooth was washed clean and any periodontal tissue adhering to the root surface was removed with hand scalers. About 3 mm of root was sliced from a tooth apex with a Carborundum disc. As much pulp as possible was removed through the small opening in the root canal by endodontic files (the opening was too small to admit a barbed broach). We did not wish to risk forming a smear layer or other barrier artifact by mechanically drilling open the pulp. Such a layer could block the movement of fluid out of the dentine. The apical opening was then sealed with dental sticky wax (Kerr, Emeryville, CA, USA) to prevent the tooth from drying. The tooth was now tightly fitted into an appropriately sized brass ring washer and sealed in place with self-cured dental acrylic resin (Dura Lay, Dental Mfg, IL, USA) (Figs. 6-1, 6-2A). The size of the washer chosen was one that fitted about 1-3 mm below the cement-enamel junction.

In preparation for an experiment the sticky wax was removed from a pulp opening. A suspension of instant coffee in cold tap water (1 teaspoon in 30 cc) was injected into the pulp chamber with a fine bore pipette. Coffee particles (size < 20 μ) suspended in the water acted as markers for measuring fluid movement, similar to the milk droplets used by Vongsavan and Matthews (1992). The tooth was vibrated during the injection to ensure all air bubbles had been removed. Injection was considered complete when no bubbles came out of the root canal and the opening was apparently filled. Excess fluid was removed leaving a drop, held in place by surface tension, over the root opening.

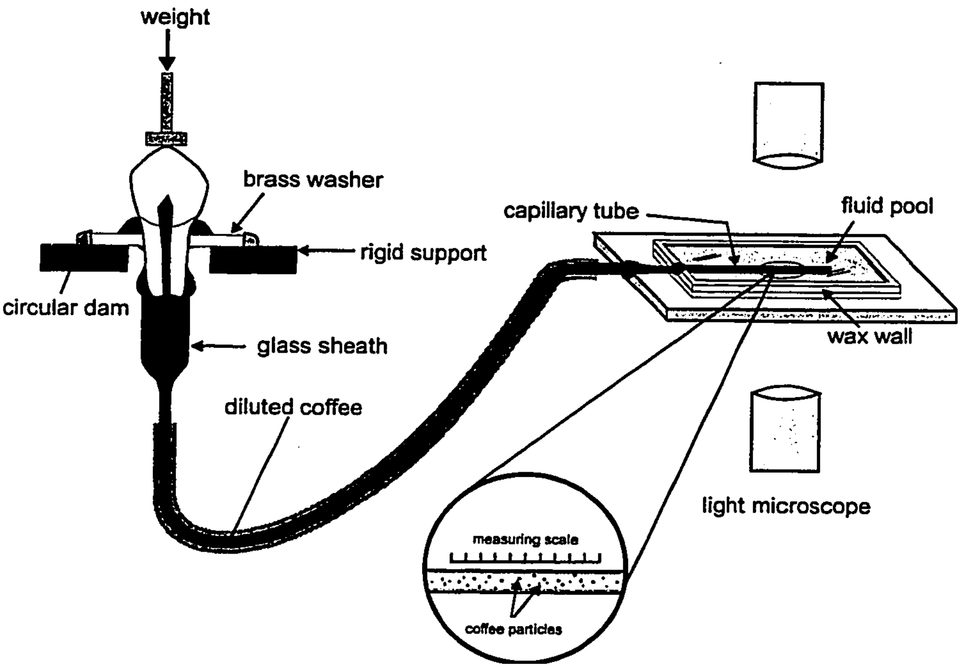

Figure 6-1 Diagram showing experimental set-up. An extracted tooth was placed firmly on a metal frame and loaded occlusally by 2-12 kg loads. The amount of fluid being displaced was measured by observing, under a light microscope, coffee particles suspended in water moving through a capillary continuous with the apical canal. Inset shows a magnified view of the capillary together with the graticule attached to the eyepiece.

A glass sheath, its tip drawn out to a smaller diameter, was sealed around the root of the tooth with pink baseplate wax. The tube was carefully filled with coffee from its narrow open end. All connections and the tooth surface were now coated with clear nail polish to prevent water leaking or possibly evaporating through the tooth enamel (Bergman and Siljestrand, 1962). The tooth, supported by its attached brass washer, was inserted into a hole in a heavy metal frame (see below).

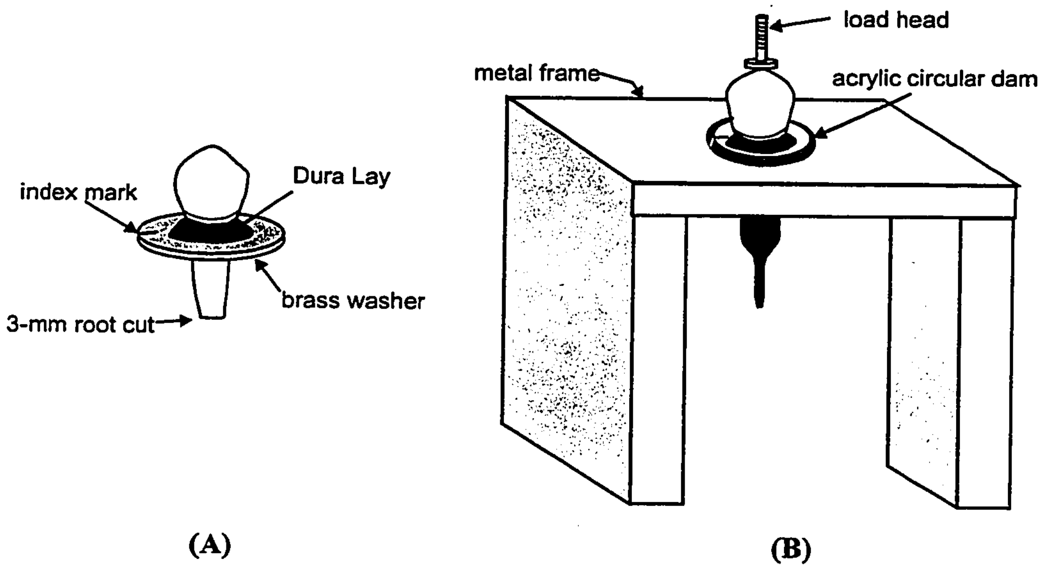

Figure 6-2 (A) About 3 mm of the root tip was removed and the tooth was attached to a brass washer by self-cured dental resin. (B) The tooth/washer assembly was then sealed to a glass sheath with dental wax and placed on the metal frame. The prefabricated circular acrylic dam and a line marked on the washer helped to replace the tooth in the same position for the control experiments.

Preparation of the measuring chamber

A capillary tube about 3 cm long with an internal diameter of 230 μ was prepared. Its wider undrawn end was connected to 0.8 mm diameter silicone tubing whose other end would later be connected to the root of the tooth.

The capillary tube was attached to the surface of a glass microscope slide with sticky wax. A 3-mm high wax dam was built around the slide and the open end of the capillary to create a shallow reservoir that could be filled with the coffee (Fig. 6-1). A light microscope with a fixed stage was chosen (the focussing system moved the lenses rather than the stage). The slide and its reservoir, together with the open capillary tube, were attached to the microscope stage with sticky wax. The capillary, silicone tubing and reservoir were filled with the coffee suspension and the free end of the tubing clamped with a hemostat.

The coffee particles in the capillary were viewed with a 10x objective lens and an eyepiece graticule. A stage micrometer showed that with the magnification being used a 100 μ interval separated the divisions on the graticule (Fig. 6-1). The sub-stage illumination was reduced to the minimum consistent with seeing the coffee granules in order to minimize the radiant heat that might have affected fluid movement.

The tooth was now connected to the measuring chamber. The hemostat was removed and the free end of the silicone tubing temporarily placed below the level of the reservoir to allow the fluid to fill the tubing, under the effect of gravity. The filled tubing was now carefully connected, without trapped air bubbles, to the glass-covered end of the pulp chamber.

Fluid always flowed towards the tooth immediately after the measuring chamber was connected to it, probably due to elastic recoil of the silicone tubing, to a difference in the temperature on each side of the system and/or to leakage between the tooth and the glass. The fluid usually stopped moving after the system had been left undisturbed for about half an hour, although a small movement along the capillary tube towards the tooth was sometimes observed. If this was greater than 50 μ/s all the connections in the system were broken apart and re-sealed. To overcome the temperature problem the apparatus was moved into a windowless room to reduce fluctuations in temperature and the movement of air.

A separate light source with adjustable intensity was used to stop any residual fluid flow toward the tooth. The light was directed towards the glass sheath. It slightly warmed the fluid and, by potentially displacing fluid away from the tooth, stopped the flow. The required intensity of illumination was low and, when properly adjusted, completely stopped movement for at least 30 seconds. This allowed enough time for the experiment.

Loading device

A loading device was designed to apply a vertical force on the crown of the tooth. The loading head was made from an inverted bolt. The platform supporting the tooth consisted of a 1-cm thick stainless steel frame with a 12-mm diameter hole drilled at its center. Initially an empty washer, the same size as that surrounding the root of the tooth, was coated with petroleum jelly and placed over the center of the hole. A small amount of self-cured acrylic (Dura Lay) was built around the edge of the washer (Fig. 6-2B) to create a circular dam. At the start of an experiment, the tooth-assembly was placed inside the acrylic dam. Pencilled lines were marked on the dam and on the washer. These, when aligned, ensured the sample could be replaced in the same position.

Weights were placed in a metal tray, itself weighing 2 kg, on top of the loading head. A screw joined two 12-cm long metal cylinders placed vertically under the load tray. By rotating the screw the height of the cylinders could be increased, thereby unloading the tooth, or decreased, thereby loading the tooth.

Experimental protocol

Loads of 2, 4, 7, 10 and 12 kg were used. Each load was applied five times, three times during one run of tests and twice during a second run. The sequence of loads was randomly chosen.

In control experiments each tooth was disconnected from its glass sheath and its root opening sealed with sticky wax. The glass sheath was then replaced and the tooth fitted back on the support (the pencilled guidelines on the dam ensured its orientation was unchanged) and connected to the silicone tubing as previously described. The sealed tooth was loaded in the same way as during the earlier test experiment and the movement of coffee particles was measured. Test experiments were always done before control experiments.

At the end of the control experiments the tooth was split open to observe how effectively the pulp tissue had been removed.

Calculation of the fluid displacement

Coffee particles moved along the capillary tube at different speeds, those in the middle being the fastest. The fluid displacement was assumed to be proportional to the movement of the fastest coffee particle. The volume of fluid moved (V) was calculated from the formula

$$V = \pi R^2 L$$

where R = the radius of the capillary ($115\ \mu$), and L = the observed displacement of the fastest coffee particle. Thus, the change in the volume of fluid in the tooth pulp needed to displace a coffee particle one interval along the measuring graticule was $3.14 \times (115)^2 \times 100 = 4 \times 10^6$ cu μ = 4,000 pL.

RESULTS

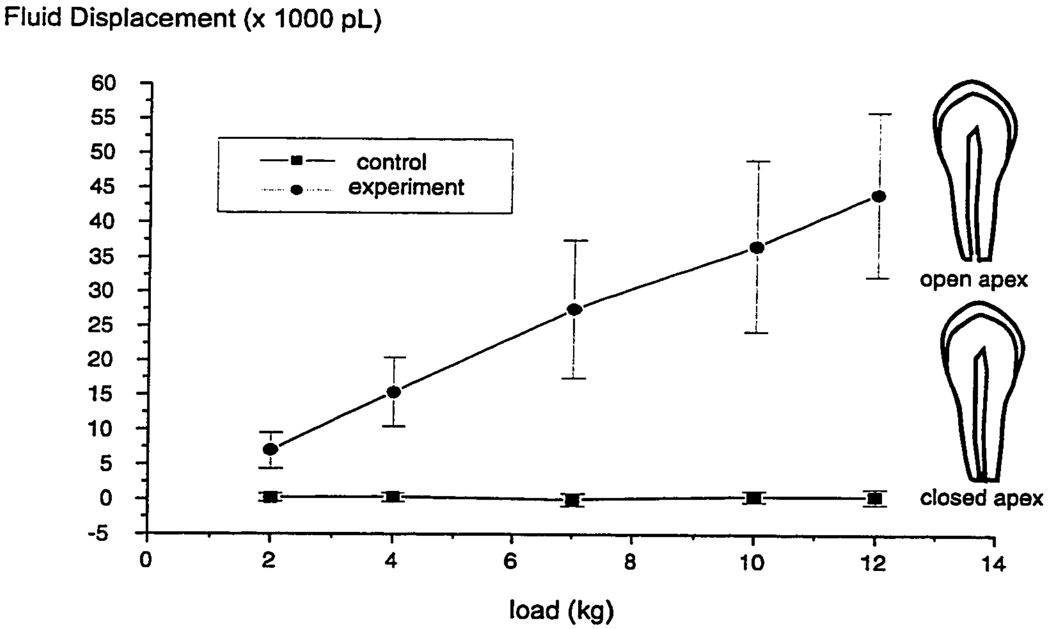

Figure 6-3 Graphs showing the amount of fluid displaced by occlusal loads. Each plotted point is the average of five measurements from each of nine teeth (n = 45). Standard deviations are shown. The upper curve shows the experimental group, the lower curve shows control measurements from the same teeth after the root opening had been sealed.

When each tooth was loaded coffee particles were displaced through the capillary. The movement started immediately after a tooth was loaded and stopped 1-3 seconds later. A similar movement was observed in the opposite direction when the load was removed. The volume of fluid moved is shown in Table 6-1. It increased linearly with an increase in the occlusal load (Fig. 6-3) and ranged from 4-12 × 10^3 pL with a load of 2 kg, to 26-70 × 10^3 pL with a load of 12 kg.

Table 6-1 The volume of fluid being displaced (from the tooth into the capillary) when different loads are applied to the occlusal surface in nine teeth. The small figures show the standard deviation from five measurements. The negative sign indicates fluid being displaced toward the opposite direction.

	Volume of fluid displaced (× 10^3 picolitres)									
	Control					**Experimental**				
LOADS	20N	40N	70N	100N	120N	20N	40N	70N	100N	120N
tooth 1	0.0	0.0	0.0	0.0	0.0	5.6	11.6	25.6	36.4	48.0
	0.0	0.0	0.0	0.0	0.0	1.5	0.8	2.0	2.0	2.5
tooth 2	0.0	0.0	-2.0	2.4	2.0	7.6	17.2	28.8	36.8	44.8
	0.0	0.0	0.0	0.8	0.0	0.8	2.4	3.0	3.0	3.0
tooth 3	0.0	0.8	0.0	0.8	0.4	4.4	16.0	23.2	31.6	42.0
	0.0	1.0	0.0	1.0	0.8	0.8	1.3	1.6	2.0	1.8
tooth 4	2.0	2.0	2.0	1.2	2.0	7.6	14.4	22.8	30.0	39.6
	0.0	0.0	0.0	1.0	0.0	0.8	1.5	2.0	3.6	2.0
tooth 5	0.0	0.0	0.0	0.0	0.0	8.4	16.0	24.8	38.8	50.0
	0.0	0.0	0.0	0.0	0.0	0.8	0.0	1.0	2.4	1.8
tooth 6	0.0	0.0	0.0	0.0	0.0	4.0	10.4	19.6	24.8	29.6
	0.0	0.0	0.0	0.0	0.0	0.0	0.8	0.8	1.6	2.0
tooth 7	0.0	0.0	0.0	0.0	0.0	9.2	18.8	33.6	44.8	48.8
	0.0	0.0	0.0	0.0	0.0	1.0	1.6	2.0	2.0	1.0
tooth 8	0.0	0.0	0.0	0.0	0.0	12.0	26.4	52.8	66.4	69.6
	0.0	0.0	0.0	0.0	0.0	0.0	2.0	3.9	2.0	3.2
tooth 9	0.0	0.0	0.0	0.0	0.0	4.0	8.4	16.8	21.6	26.4
	0.0	0.0	0.0	0.0	0.0	0.0	0.8	1.0	2.3	1.5
MEAN	**0.2**	**0.3**	**0.0**	**0.5**	**0.5**	**7.0**	**15.5**	**27.6**	**36.8**	**44.3**
SD	**0.6**	**0.6**	**0.9**	**0.8**	**0.8**	**2.6**	**5.0**	**10.0**	**12.4**	**11.9**

In contrast, when the root canal opening was sealed with sticky wax the coffee particles remained stationary in six teeth and moved fractionally in the remaining three teeth. Increasing the load had little or no effect on the initial values. The largest load displaced less than 3×10^3 pL fluid.

When the teeth were split open at the end of the experiments it was observed that remnants of pulp tissue, saturated with fluid, were still scattered over the surface of the predentine and in the pulp cavity.

DISCUSSION

We were able to measure the amount of fluid being displaced from the tooth pulp when its occlusal surface was loaded. The very limited movement in the control studies suggests the movement observed in the experiments was due to deformation of the teeth as opposed to deformation of other parts of the apparatus, a problem that we frequently encountered in our earlier experimental designs.

The suspension of coffee particles in water seemed to work as effectively as the milk droplets used by Vongsavan and Matthews (1992). Calculating the volume of fluid displaced from the movement of a particle in the fluid only approximates the true volume displaced. First, particles move more slowly than the fluid because of resistance. Second, we chose to measure the movement of the fastest particle. This represents flow near the middle of the tube. Towards the edges particles moved more slowly due to the frictional resistance related to laminar flow along a tube.

Several previous *in vitro* studies (*e.g.* Hood, 1972; Reeh *et al.*, 1989; Lutz *et al*, 1991) have shown that the stiffness of teeth is reduced after removing tooth substance for cavity preparation. It has also been demonstrated that cementing a full crown on a prepared tooth increases pulpal pressure (Wylie and Wilson, 1994). The increase might have been due either to forcing cementing material down the dentinal tubules or to compressing the dentine. All the above studies involved the action of compressive forces when a cut surface of dentine had been exposed. A study of loaded intact teeth demonstrated that cusps are deflected 1-4 μ by 10-200 N forces (Grimaldi, 1971).

It would be insufficient for us to demonstrate 5 pL of fluid movement, for example, under a load of 2,000 N. Our hypothesis required that first, the compressive forces be in the range normally applied to teeth and, second, enough fluid moves to activate pulpal nerves. Both requirements were fulfilled. It might seem intuitively obvious that compressing a tooth would squeeze fluid into the pulp. But if it were obvious it would have raised the following paradox. Moving fluid by heating the tooth activates nociceptors and causes pain but moving the same volume of fluid by compressing the tooth does not cause pain.

Enamel and dentine probably each resist compression differently in different directions: they are anisotropic (Spears *et al.*, 1993; Spears, 1997). Dentine, for example, may resist compression forces more strongly along the orientation of crystals (perpendicular to dentinal tubules) than transverse to them. Spears and co-workers also noted that the compressibility of both dentine and enamel has been poorly studied. The behavior of even a simple cylindrical model of a single tissue, dentine for example, is imperfectly known. But teeth are far more complex. Their shapes are very irregular and they consist of two different anisotropic materials connected by an interface whose mechanical properties are unknown. It is very difficult to predict with accuracy the force distribution in such a complex system and therefore impossible to predict how much fluid is moved when a tooth is loaded.

The fluid displacement observed in our study could be accounted for in three ways.

> (1) Enamel tufts, lamellae and/or enamel spindles are squeezed. Dentine fluid is continuous with enamel fluid (Bergman and Siljestrand, 1962; Bergman, 1963). Any deformation of the enamel (Young's Modulus = 4.1 $\times 10^4$ MPa; Rubin *et al.*, 1983) may compress these more richly organic regions and force fluid into the dentine and toward the pulp.

(2) Dentine is compressed. If dentine is compressed it is clear that fluid will be squeezed along the tubules because, compared with the intertubular region, the tubules cannot resist compression. Nevertheless the mineralized intertubular material will also be slightly compressed and fluid squeezed out.

(3) The pulp chamber is deformed. The pulp chamber has almost no resistance to compression from loads comparable to those used here. Its change in volume is almost entirely dependent on the distortion of the dentine.

It will be recalled that fluid immediately moved back toward the pulp when the load was removed. This was probably due to elastic recoil. If this is true it suggests that the outward flow was due to compressing dentine, rather than to compressing enamel because dentine is relatively elastic.

Apart from the compressibility of the dental tissues, the volume of fluid moved by loads is related to morphological features such as the length of the tooth, the height of its support, its shape and cross sectional area, the amount of secondary and peritubular dentine and the number of blocked dentinal tubules. Although we do not know how much each of these features influenced our results our aim was merely to measure whether the loads we used would displace a sufficient volume of fluid to activate pulpal nerves.

A quantitative prediction of the amount of fluid moved can be based on an analysis of the vertical deformation of dentine. We assume that the volume of the pulp chamber is small compared to that of dentine. We therefore analyze distortion of a (simplified) solid dentine cylinder, roughly the size of the crown of a premolar tooth (8.5 mm high, 4 mm radius). If a 100 N occlusal force is transmitted to the dentine, the stress is approximately 2 MPa. With a Young's Modulus of 1.9×10^4 MPa (Rubin *et al.*, 1983), the decrease in dentine volume due to purely vertical compression is 4.2×10^4 pL. Approximately this volume of fluid would be displaced. This theoretical value is close to the value we measured (mean = 3.7×10^4 pL for a 100 N force).

The distribution of stress beneath an occlusal load has recently been studied by finite element analysis. It demonstrates the inaccuracy of the above simple calculation. If enamel is isotropic (equally compressible in any direction), occlusal loads stress dentine approximately between two times (Goel *et al.*, 1990) and 20 times (Rubin *et al.*, 1983) less than enamel. Under these conditions Spears *et al.* (1993) predicted that the enamel would protect the coronal dentine from being compressed (Fig. 6-4A). More stress would be transferred through the rigid enamel down to the cervical region and thence to the root dentine. This suggests fluid would start moving into the pulp from compressed root dentine. But enamel is almost certainly anisotropic. In this case the stress would spread more directly through the enamel into the mantle dentine under the cusp (Spears *et al.*, 1993) (Fig. 6-4B). Most of the moving fluid would be squeezed from this region down those tubules that open into the underlying pulp horn.

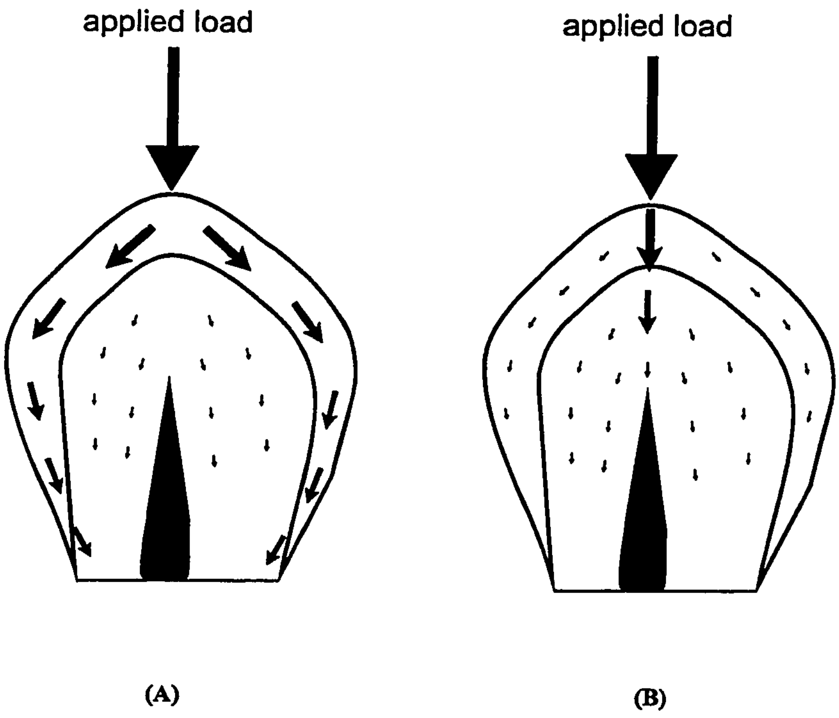

Figure 6-4 Theoretical distribution of stress in a tooth under the effect of a vertical occlusal load (modified from Spears *et al.*, 1993). (A) If enamel is isotropic more of the stress spreads down to the cervical enamel and thence to root dentine. (B) If enamel is anisotropic more of the stress spreads to the cuspal dentine. Fluid would be squeezed down the tubules to the pulp horn.

It must be remembered that a vital tooth *in situ* may respond differently to loads from a dead tooth *in vitro*. The permeability of human dentine in an extracted tooth increases with storage time (Goodis *et al.*, 1991). Opposing this enhanced movement, it is more likely that the dead tissue remaining in the pulp chambers of most our teeth would have reduced the volume of fluid movement. The dynamics *in vivo* are further complicated by the fact that vital pulps possess a tissue fluid pressure that might impede the inward movement of fluid (Beveridge and Brown, 1965; Vongsavan and Matthews, 1992).

The amount of fluid displaced in our study (range 7.5-45.8 × 10^3 pL) is comparable to that measured by Anderson *et al.* (1967) (up to 4.8 × 10^3 pL/s when saturated $CaCl_2$ is applied to exposed dentine) and by Brännström *et al.* (1968) (up to 4.9 × 10^4 pL/s during cutting a cavity with a high-speed hand piece). In both of these studies the fluid movement was almost certainly concentrated in the region where the stimulus was applied. This would be equivalent to the concentrated stress shown in Fig. 6-4B. The volume is also comparable to that measured by Brännström and Johnson (1970) (~1.7 × 10^4 pL/s) during the application of heat or cold to an intact crown (calculated from their Fig. 6-2). In this experiment fluid would start flowing from a much larger region, more like that shown in Fig. 6-4A, although the displaced fluid presumably came from the crown rather than root dentine.

It seems very unlikely that bite forces of 20-120 N would cause pain since they are within the range used during normal chewing (*e.g.* Anderson, 1956; Gibbs *et al.*, 1981; Lundgren and Laurell, 1986). Fluid movement due to loading might therefore activate both pulpal nociceptors and mechanoreceptors. The nociceptor input may not be perceived as pain because it could be suppressed (gated) by a simultaneous input from periodontal mechanoreceptors (Osborn and Ten Cate, 1976).

It has been postulated that pulpal mechanoreceptors may be unconsciously used to detect the amount and direction of occlusal forces on individual cusps (Osborn and TenCate, 1976). This ability enabled the small mammal-like reptiles and mammals that originally evolved an occlusion to finely tune the interdigitation between upper and lower teeth (Osborn and Hillman, 1979). The input may also be used to prevent a tooth being damaged by destructively large bite forces and to maximize the pressure on a hard object for a given bite force (Paphangkorakit and Osborn, 1997*b*). The more frequent tooth fracture observed after endodontic treatment (Gulabivala, 1995) could be in part due to the lack of these receptors. Finally, Paphangkorakit and Osborn (1998) have shown that pulpal mechanoreceptors, distorted by the movement of fluid out of the compressed dentine, may be consciously used to discriminate between the hardness of foods. From a theoretical analysis of hardness perception Peleg (1980) concluded that only a soft receptor, in this case the periodontal ligament, can be used to discriminate between the different hardness of soft objects, and only a hard receptor, in this case dentine, can be used to discriminate between the hardness of hard objects.

Periodontal mechanoreceptors are probably activated either by stretching the supporting periodontal collagen fibers (Linden and Millar, 1988*a*) or by compressing the surrounding tissues. A load of about 10 N fully intrudes a tooth in its socket (Parfitt, 1960; Picton, 1963*a*). Periodontal mechanoreceptors, therefore, cannot be used to distinguish between loads greater than 10 N because in every case the periodontal fibers are fully stretched and the soft tissues are fully compressed. Furthermore most (or all?) periodontal mechanoreceptors appear to be activated by a load of 1 N (Loescher and Robinson, 1989; Bonte *et al.*, 1993). Even if a tooth could be further intruded in its socket there are probably no more receptors to activate at loads greater than 10 N. The hard dentine together with its pulpal nerves may be a more effective receptor for gauging the amount a tooth is stressed under moderate to large occlusal forces.

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CHAPTER 7

GENERAL DISCUSSION AND CONCLUSIONS

STUDIES OF INCISAL BITE FORCE

Large variations in incisal MBFs have been measured with uni-directional bite force transducers (Table 7-1).

Table 7-1 Incisal MBFs measured with uni-directional bite force transducers.

Incisal MBF (N)	Investigators	Types of force transducer
216 N (males) 196 N (females)	Linderholm and Wennström, 1970	Bite fork (4 mm separation)
293 N	Ringqvist, 1973*a*	Bite fork (6 mm separation)
87 N	Hannam, 1976	Bite bars (2 mm separation)
157 N (females) 193 N (males)	Helkimo *et al.*, 1977	Bite fork (7.4 mm separation)
190 N	Helkimo and Ingervall, 1978	Bite fork (7.4 mm separation)
75-178 N	Helle *et al.*, 1983	Bite fork
158-172 N	Dahlström *et al.*, 1988	Bite fork (15 mm separation)
287 N (males) 243 N (females)	Waltimo *et al.*, 1993	Bite fork (22 mm separation)
139-491 N	Waltimo and Könönen, 1994	Bite fork (22 mm separation)

The incisal MBF has only once been measured with a three-dimensional bite force transducer. The values for three subjects ranged from 50-190 N (Osborn and Mao, 1993). Hylander (1978) measured the direction of incisal bite forces in the two dimensions of the sagittal plane but only at submaximal levels. Both the above studies found that incisal bite forces are directed approximately 10-15° anteriorly with respect to the lower occlusal plane.

In the retruded jaw position incisal MBFs from 117-368 N were recorded in Paper #1, with directions from about 30° backward to 30° forward with respect to the lower occlusal plane. Both bite force magnitude and direction appeared to be dependent on the amount of jaw separation (Paper #1), the bite force being larger and directed more posteriorly as the incisal separation is increased. The bite force direction at 8-10 mm incisor separation, the minimum separation with the present transducer, was approximately 10° forward.

Differences in the MBF measured in previous studies can be attributed to differences in the subjects and to differences in jaw separation related to different measuring devices. With a thinner transducer, the incisal MBF is smaller and directed more anteriorly (Paper #1). If an incisor bite force is more vertical its magnitude measured with a uni-directional transducer, most sensitive to vertical forces, is larger.

The direction of an incisal MBF seems to be dictated by the position of the lower incisor tooth with respect to the upper incisor tooth. If the system is in equilibrium the bite direction should lie along a line connecting the two incisal edges (see Appendix D). When the jaw is opened, the lower incisal edges move downward and backward and the lower occlusal plane rotates downward and backward. As a result, the connecting line becomes directed progressively more posteriorly with respect to the lower occlusal plane. The incisal bite direction in the sagittal plane observed in Paper #1 followed the predicted pattern (Fig. D-2). This was further confirmed by the later observations (Papers #2, #3) that the incisal bite force became more vertical when the lower jaw was protruded. The mean bite direction was 2° backward in Paper #2 and 0° in Paper #4, as opposed to 10° in Paper #1 (Tables H-2 and H-3). From these results it is clear that bite forces are not necessarily directed down the long axes of incisors. It would obviously not be possible for both upper and lower human incisors because both are tilted forward. Mathematical jaw models based on linear programming suggest that the maximum incisal MBF should be directed posteriorly (Baragar and Osborn, 1987; Koolstra *et al.*, 1988), more in line with the long axes of upper incisors.

Changes in the activity in masseter and anterior temporalis muscles, monitored by the T/M ratios measured in Paper #1, are probably secondary to changes in the bite direction. The first requirement when breaking food is to stabilize and retain it (Osborn and Lumsden, 1978). Stabilizing an object placed between upper and lower incisor teeth clearly requires a bite force passing through both incisal edges unless the object is firmly held by a hand. The jaw closing muscles are subsequently recruited to produce a force in this direction. Thus, the backward shift of the incisal force direction as the jaw is opened is facilitated by the anatomical arrangement of masseter and anterior temporalis (*e.g.* orientation, sarcomere length, moment arm), as discussed in Paper #1.

EXPERIMENTS INVOLVING INCISAL BITE FORCES

Advantages

(1) It is easy to place the bite force transducer and to adjust its horizontal orientation parallel to the lower occlusal plane (hence, angular transformation is not necessary, see Appendix E).

(2) Incisor bite forces are usually parallel to the sagittal plane (Tables H-1, H-2, H-3) with symmetrically active jaw muscles. Changes in bite force direction can be readily correlated with changes in the activity of jaw muscles in the sagittal plane (Fig. 2-3B).

(3) Incisor bite forces are smaller than molar bite forces and therefore less likely to permanently deform the transducer and thereby distort the calibration. This problem was encountered when measuring maximum molar bite forces (not reported here).

(4) The direction of incisal bite forces can be easily adjusted by protruding or retruding the lower jaw. A vertical force can be obtained by biting in an edge-to-edge relation and a backward force by protruding the lower jaw. A

vertical force minimized the tilt of the bite block (Paper #4) and therefore minimized the stabilizing force exerted on the handle of the bite block by a subject's fingers.

Disadvantages

(1) Bite directions are limited to the sagittal plane. Differences between the working and balancing side muscles cannot be investigated.

(2) Incisors are not often used during chewing.

We decided to use incisor bite forces to study the effect of pulpal stimulation. The bite force was measured with the incisors in the edge-to-edge position in Papers #2 and #4 because the bite point could be conveniently centred on the transducer (Figs. 3-1B, 4-1) at the same load point as that used when the transducer was calibrated. The retruded position was used in Paper #1 because it is the easiest to reproduce a jaw position at different jaw openings. In Paper #2 the upper incisor tooth was biting on an acrylic support attached to the centre of the transducer whereas in Paper #4 the bite block was placed between the incisor teeth and the transducer. The joint reaction force and jaw orientation (Goto *et al.*, 1995), and consequently the length of some jaw muscles, is changed during jaw protrusion. This may affect the excitability of the motoneurons supplying jaw closing muscles, as shown for limb muscles where the H-reflex is suppressed during muscle stretch (Robinson *et al.*, 1982; Etnyre and Abraham, 1986). The above effects were avoided by comparing MBFs at the *same* jaw position.

The magnitude of the bite force tended to increase during the first few bites, as observed in previous studies (Hellsing, 1980; Orchardson and MacFarlane, 1980; Waltimo and Könönen, 1994). This may have been due to subjects becoming more familiar with the procedures or less apprehensive of breaking their teeth (Orchardson and MacFarlane, 1980). These effects can be minimized by allowing subjects to practise a few times, by arranging the bite trials in a random order and by using *average* MBF values.

THE USE OF RAPID MAXIMUM BITE FORCES

In Papers #2 and #4, subjects were told to bite as fast as they could and then to relax. It usually took less than 3 seconds to reach MBF. The rapidity would prevent MBFs being influenced by a subject's motivation which has been shown to affect the value of maximum contraction (van Steenberghe and De Vries, 1978; Peacock *et al.*, 1981).

The 1 to 3-second ramp almost certainly allows sufficient time to initiate jaw reflexes since periodontal stimulation (Bonte *et al.*, 1993; Sato *et al.*, 1994) and electrical tooth stimulation (Matthews *et al.*, 1976) only take about 20 ms to evoke a response in jaw closing muscles. In addition, Lund and Lamarre (1973) suggested that a suddenly encountered stimulus during jaw closure tends to activate an inhibitory reflex at the brain stem level whereas a slow closure tends to activate excitatory cortical loops. However, Desmedt and Godaux (1978*a*) believed that during a ballistic contraction (muscle contraction time ≈ 70 ms) muscle activity was not regulated through proprioceptive loops.

In a preliminary study (see Table H-5) it was observed that the MBF during a rapid incisal clench (reaching the MBF within 1 second) tended to be smaller than that during a slower clench (reaching the MBF in 3-5 seconds). The bite direction was unchanged. The increased MBF may have been achieved by cortical reinforcement during the slow clench when subjects realized that the tooth was not being damaged. When a finger is rapidly contracted the discharge from a motor unit is not increased as the force is increased (Milner-Brown *et al.*, 1973). It is therefore not necessary to increase the firing frequency ("rate-coding") in order to increase the force of a rapid contraction. In this case recruiting more motor units may be more important. This is supported by the observation that the recruitment threshold of a motor unit is reduced in rapid contraction (Milner-Brown *et al.*, 1973; Desmedt and Godaux, 1979). A lack of rate coding may have contributed to the smaller MBF observed during a rapid tooth clench.

The recruitment order of motor units in the masseter muscle during a rapid clench on premolar teeth has been shown to obey Henneman's (1957) size principle (Desmedt and Godaux, 1979). Smaller motor units are recruited earlier. It has also been shown that muscle coactivation is different for slow and rapid tooth clenching. Desmedt and Godaux (1979) observed an increase in medial pterygoid activity and a decrease in temporalis activity during rapid tooth clenching. However, we did not find any significant difference in the incisal bite direction for slow and rapid tooth clenches. Had the difference existed it might have indicated changes in muscle coactivation.

The hypothetical MBFs calculated from jaw models are limited solely by muscle strength. They tend to be much larger than those actually measured (Koolstra and van Eijden, 1992). In real life, the MBF is presumably also limited by central drive and by peripheral feedback. In which case the measured MBF is not the maximum force jaw muscles can actually exert. Visual and auditory feedbacks, both of which indirectly influence the central drive, have been shown to influence the magnitude of isometric maximum bite forces (van Steenberghe and De Vries, 1978) and maximum isometirc contraction of the quadriceps muscle (Peacock *et al.*, 1981). Peripherally, a role for periodontal receptors in limiting the MBF has been based on the observation that MBFs can be increased by anaesthetizing teeth (van Steenberghe and De Vries, 1978; Orchardson and MacFarlane, 1980). A role for jaw muscle receptors in limiting the MBF (presumably tendon organs) has not been demonstrated. All of the above suggest that the MBF is not fixed. Its variability was exploited in present study.

EXPERIMENTAL DESIGN FOR STUDYING PULPAL RESPONSE TO FORCES

Studying a possible pulpal response to mechanical stimuli is complicated by the difficulty of excluding inputs from the periodontal ligament. Any force applied to the crown of a tooth in an attempt to stimulate pulpal receptors simultaneously stimulates periodontal mechanoreceptors. Attempts have been made to distinguish between the two possible sources of input by capping the experimental teeth (Loewenstein and Rathkamp, 1955), by using non-vital teeth (Loewenstein and Rathkamp, 1955; Linden, 1975), and by splinting the experimental teeth while force is applied (Olgart *et al.*, 1988).

The first method was used in Paper #2. Capping the tooth with a resin crown increased the contact area and consequently reduced the stress (force per unit area) at the incisal edge. A drawback of this method was that the cap increased jaw separation and jaw separation is known to affect the MBF. This could be rectified (1) by increasing the height of the acrylic wedge on the transducer to compensate for the crown thickness or (2) by adjusting results for differences caused by the increased separation. Adjusting the height of the wedge would have introduced another error because it would have changed parameters of the force applied to the transducer (see Appendix A). We therefore calculated the change in the MBF for the 0.5 mm difference in jaw separation, the thickness of the acrylic crown. Based on data from Paper #1 and a previous study (Mackenna and Türker, 1983) this might change the MBF by about 1-2%.

A new way of excluding differences in the input from receptors other than those in the pulp was used in Paper #3. A bite block, one side softer rubber and the other side harder acrylic was made. By turning the bite block around the upper incisor teeth either bit on rubber or acrylic. The environment of the lower incisors was kept constant. The stress at the incisal edge of the upper incisor depended on the size of contact area. With a harder surface the smaller contact area increased the stress. The method did not change the input from jaw muscles (because the jaw opening was not changed), from periodontal ligament (because the forces on the upper and lower teeth were the same), or from skin, mucosa or periosteum. During the hardness discrimination test (Paper #3), subjects were prevented from sensing the surface texture of the bite block by wrapping it in masking tape. The success of this precaution is verified by the fact that every subject had to increase the bite force before being able to detect which surface was uppermost. Had subjects been able to discriminate by using surface texture contact they could have signalled whether their upper incisors were biting on a hard or a soft surface when the bite block was being positioned.

ROLE OF PULPAL MECHANORECEPTORS IN LIMITING BITE FORCES

Elsewhere in the body, inputs from both nociceptors and non-nociceptors can be used to limit the maximum force produced by skeletal muscles, namely the "maximum voluntary contraction" (MVC). Free nerve endings in muscles, bone and joints are responsible for nociception whereas those responsible for non-nociceptive inputs include Golgi tendon organs associated with the contracting muscles (reviewed by Jami, 1992) and possibly mechanoreceptors in skin and bone. An isometric MVC is almost certainly influenced by central neural drive from the motor cortex. Verbal encouragement and visual feedback, for example, can increase the MVC (Peacock *et al.*, 1981).

The MVC produced by jaw closing muscles is thought to be mainly regulated by feedback from periodontal mechanoreceptors since the presence of tendon organs, either in jaw closing or jaw opening muscles, is poorly established. However, a tooth is likely to be maximally intruded into its periodontal ligament under a vertical load of 10 N (Parfitt, 1960; Picton, 1963*a*). A value of 60 N chewing force (Anderson, 1956*b*) would completely seat the tooth in its socket long before the force reaches its peak. It is therefore difficult to understand how the magnitude of large bite forces could be gauged by periodontal mechanoreceptors. Some other mechanoreceptors are required to limit the MBF. They could be in skin, periosteum or tooth pulp.

Any possible controlling input from periodontal, skin, or periosteal receptors was excluded by either covering the incisor tooth with an acrylic cap (Paper #2) or by using the rubber-acrylic bite block (Paper #4). Both acrylic caps and rubber increase the contact area and reduce the stress on an incisal edge without changing the periodontal input. Bite force directions were also monitored to ensure that they were not different in experiments whose results were to be compared. The possibility that subjects were aware of differences in experimental procedures was largely eliminated because most of them were unaware of what they were biting on and because the sequence of tests was randomized.

Electrical stimuli applied to the tooth at or just above the level of perception do not usually produce a sensation described as "pain." They do, however, evoke an inhibitory reflex in human jaw closing muscles (Matthews *et al.*, 1976; McGrath *et al.*, 1981; Sharav *et al.*, 1982) and activity in the digastric muscle of animals (Närhi *et al.*, 1983). Mechanical stimuli applied to the tooth pulp of cats have also been shown to evoke reflex activity in the digastric muscle (Olgart *et al.*, 1988). The tooth in this experiment was splinted to posterior teeth to prevent stimulating periodontal mechanoreceptors. Activity in the pulpal nerves was recorded from a cavity cut in the dentine. The origin of the response was supported by the absence of the reflex when the pulp was removed and when the tooth crown was cooled.

Papers #2 and #4 showed that the MBF on human incisor teeth is reduced when the stress on the incisal edge is increased. Robinson (1964) showed that forces bending the walls of an occlusal cavity could cause tooth pain. Such pain might have limited the MBF. But no subject involved in Paper #2 or #4 reported pain or discomfort.

The reduction in MBF observed in Papers #2 and #4 was presumably the result of an inhibitory reflex acting on the jaw closing muscles although their activity was not measured. Such an inhibition is comparable to that observed in the jaw opening reflex evoked by periodontal stimulation and electrical pulpal stimulation (Hannam and Matthews, 1968; Brenman *et al.*, 1968; Matthews *et al.*, 1976; McGrath *et al.*, 1981; Bonte *et al.*, 1993). Apart from the chosen techniques, which excluded other receptors, the pulpal origin of the response was confirmed by the insignificant change in MBF when the incisor teeth were capped in Paper #4. Caps spread the load more evenly over the incisal edges and eliminated the stress difference when biting on rubber and acrylic strips.

Although not statistically significant, the mean difference in MBF between bare and capped teeth biting on acrylic (4.9%, Paper #2) is smaller than that between bare teeth biting on acrylic and rubber (7.5%, Paper #4). The difference can be explained if the incisal edge adapts better to the rubber than to the inside of an acrylic cap. It does not perfectly fit the inside of a cap, but rather has multiple contacts.

A large stress concentrated on a cusp of a tooth may cause pain similar to that when accidentally biting on a piece of metal, or to that observed in Robinson's (1964) study. We conclude from the present study, however, that the response to a gradually increasing large stress may not necessarily be pain but a reflex reduction in the MBF.

This could subconsciously protect a tooth from being fractured especially in the cuspal region. The ability to detect stress may also be used to avoid biting on the harder parts of food where the stress on the teeth would be high. Instead a subject may be able to detect the softer part of the food where the stress is smaller for the same bite force magnitude and where the food can be broken up more easily.

ROLE OF PULPAL MECHANORECEPTORS IN HARDNESS DISCRIMINATION

The main finding from Paper #3 was that subjects could discriminate between soft and hard objects by using a sensory mechanism inside the teeth, pulpal mechanoreceptors. The range of bite forces used was far below that which might have been expected to cause pain. This is the first time pulpal receptors have been reported to be involved in detecting hardness. Although it is unlikely that the direction of a bite force could be used as a cue for detecting hardness, we measured bite directions to ensure that they did not change when biting on hard and soft objects.

Laboratory measurements of hardness involve comparing the amount of deformation caused by a known force, usually the size of a surface indentation. A softer object is indented more with a smaller force. The mechanism of hardness detection by fingers is more complex because the fingerpads not only detect force but also pressure. Srinivasan and LaMotte (1995) suggested that fingers provide two types of sensory input for hardness detection and hardness discrimination, "tactile information" and "kinesthetic information." Tactile information is associated with the force and pressure detected on the skin. Kinesthetic information is associated with the sense of joint movement. For a given force the pressure is smaller and the finger moves further when compressing a soft object than a hard object. Forces can be detected by mechanoreceptors in the skin or in the muscles. Pressure can only be detected by receptors in skin. Movement is probably detected by receptors in both joints and muscles.

Discrimination between rubbers of different hardness is best when both tactile and kinesthetic information are available (Srinivasan and LaMotte, 1995). Accuracy is slightly less without a kinesthetic input. However, after the fingerpad is anesthetized the ability to discriminate hardness is completely lost. They concluded that "pressure" and/or "the rate of pressure change" on the skin is a far more important cue for hardness discrimination than joint movement. The point is illustrated by the following experiment.

A piece of sponge was glued to a wooden spatula to make a soft-hard block (see Appendix G) similar to the one used in Paper #3. The block was covered with satin tape to produce a uniform surface texture. Ten subjects were asked to squeeze the block between their thumb and index finger and decide which surface was on the side of the index finger. Each subject was tested ten times in a random order. Every subject was accurate every time (Table G-1). This was also true when the block was compressed between right and left forefingers. In the latter case subjects could not have used kinesthetic information since there was no difference in the movement of left and right fingers. The experiment confirmed that tactile information alone is adequate to detect hardness.

The hardness of an object clenched between upper and lower teeth may also be detected by comparing the amount of jaw movement (closure) with the bite force used. A given bite force produces more movement into softer food. Receptors in the TMJ and/or in the jaw muscles can be used to monitor the amount of jaw movement. Periodontal mechanoreceptors might conceivably be used to detect the amount of force, provided it is small (we do not think they could be used to measure a 39-N force, the average detection threshold measured in Paper #3). The above mechanism may explain how subjects could discriminate between the hardness of different plastic and brittle materials by biting on them with their incisors (Mioche and Peyron, 1995). The bite force used was closely correlated with the hardness of the object. Interestingly, the bite force was almost constant when detecting the hardness of different elastic materials. In this case the amount of jaw closure might have provided the clue necessary to detect hardness.

The experimental design of Paper #3 prevented subjects using information from the periodontal ligament, muscle receptors, facial skin and bone to discriminate hardness (see Discussion in Paper #3). The only useful input was inside the tooth.

Just as when discriminating hardness with fingers, so also for a given bite force the contact area between tooth and object is larger when biting on a softer object. The larger contact area on the soft side reduces the stress (force/unit area) on the incisal edge. Two mechanisms may be used by subjects in Paper #3 to distinguish between the soft and hard sides of a block. First, although the magnitude of the force is the same on both sides at any given moment during biting, the stress on the soft side is always smaller than that on the hard side. The *instantaneous* stress on upper and lower teeth could therefore be compared. Second, the contact area on the soft side gradually enlarges as the bite force is increased and the tooth sinks into the rubber, up to a point where it cannot be further compressed. The contact area on the hard side remains constant with the result that the stress rises more steeply than that on the soft side. The *rate of change* in stress could therefore be compared. If this were the case, once the soft side had been fully compressed it would be more difficult to discriminate since the rate of change in stress would now be the same for both sides. In essence, the study suggested that tactile information alone can be used to detect hardness of an object being clenched.

The discrimination thresholds of 39 N for incisor teeth and 85 N for molar teeth are presumably the minimum differences in stress between upper and lower teeth required to detect the difference between rubber and acrylic surfaces. Molar teeth may have a higher threshold because they are better able to resist deformation and therefore more force is required to activate the pulpal mechanoreceptors.

Receptors with low compliance cannot be used to detect the hardness of a hard object because they are completely compressed before the hard object begins to be deformed enough to be detected (Peleg, 1980). The periodontal ligament cannot likely be used to detect the hardness of acrylic because it is so much more compliant than acrylic. The periodontal tissue is fully compressed before the acrylic begins to be distorted. In the reverse case, pulpal receptors encased in an ivory-hard shell cannot likely be used to detect the softness of a compliant object. The hard shell has not been sufficiently deformed to activate pulpal nerves before the soft object has been fully compressed.

In summary, hardness is most effectively detected when information about force, pressure and movement are combined. Of these, stress (or pressure) alone apparently provides adequate information for hardness discrimination. Pulpal mechanoreceptors are the only ones that can be used to measure the pressure on teeth.

FLUID MOVEMENT IN DENTINE UNDER PHYSIOLOGICAL FORCES

If human incisors contain mechanoreceptors that are stimulated by forces applied to the enamel it raises the question of where and how the nerves are activated. At present, it is generally believed that pulpal nerves are activated by fluid movement along dentinal tubules ("Hydrodynamic mechanism of dentine sensitivity" – Brännström, 1963). Activation of these pulpal nerves has been exclusively used to explain the mechanism underlying tooth pain.

Paper #5 demonstrated that fluid can be squeezed out of the pulp under loads ranging from 20 to 140 N. This is the first time a significant amount of fluid has been shown to be displaced when occlusal loads are applied to intact teeth. Forces within this range are unlikely to evoke tooth pain since they are normally used during chewing (Anderson, 1956*b*; Gibbs *et al.*, 1981*b*) but the volume of fluid moved was comparable to that observed when pain-producing stimuli are applied to exposed dentine. The amount of fluid being displaced was linearly proportional to the magnitude of loads.

Both *in vitro* experiments and theoretical analyses show that teeth can be stressed and deformed (*e.g.* Hood, 1972; Reeh *et al.*, 1989; Spears *et al.*, 1993). Most of these investigations studied the effect of tooth preparation on tooth stiffness and the pattern of stress distribution around a prepared cavity. Only one study has attempted to relate tooth deformation to the movement of dentine fluid. In this study, Lutz *et al.* (1991) measured changes in the stiffness of extracted teeth caused by different cavity preparations. They did this by comparing the amount of fluid displaced from the pulp when a constant load was applied to the occlusal surface.

The movement measured in Paper #5 could have been caused by fluid (1) squeezed out of organic structures in enamel (spindles, tufts) or dentine (either tubules or intertubular dentine), or (2) squeezed out of a compressed pulp. The volume of the pulp can only be changed if the dentine is deformed but the dentine can theoretically be deformed without changing the volume of the pulp. Compressed enamel and/or dentine squeeze fluid into the pulp.

If enamel is anisotropic occlusal forces tend to be dispersed directly into the coronal dentine and adjacent pulp horn (Spears *et al.*, 1993). In Paper #5 in which the loads were applied to the cusp of a tooth, most of the displaced dentine fluid would pass through the pulp horn where the tubules are densely packed.

Clearly it could have been predicted that fluid would be displaced out of dentine when it is compressed. It was, however, surprising that the amount of fluid was comparable to that which has previously always thought to be associated with pain.

PROPOSED MECHANISM

Nerve endings inside teeth are found in the inner third of dentine (dentinal nerves), in predentine, between odontoblasts and in the subodontoblastic nerve plexus (Gunji, 1982). Brännström's "hydrodynamic theory of dentine sensitivity" is widely accepted although a similar view was long ago proposed by Gysi (1900). It is thought that the nerves are stimulated by fluid flow but how they are activated has never been demonstrated. The theory is supported by a number of studies showing that most pain producing stimuli applied to the surface of dentine also cause fluid to move through the dentine. Most of these stimuli cause an outward flow, from the pulp into the dentine. Amongst these are $CaCl_2$ solutions (Anderson *et al.*, 1967), sugar solutions (Linden and Brännström, 1967), NaCl or NH_4Cl (Horiuchi and Matthews, 1973), cold stimuli (Brännström and Johnson, 1970) and drying the dentine surface (Brännström *et al.*, 1968). In contrast, heat (Brännström and Johnson, 1970), tooth drilling (Brännström *et al.*, 1968) and hydrostatic pressure applied to exposed dentine (Brännström *et al.*, 1969; Horiuchi and Matthews, 1973) move fluid inward, from the dentine into the pulp. About

10^3 - 10^4 pL/s of fluid flow is generally measured. Both outward and inward movement apparently produce pain although the intradental nerves are much more sensitive to outward flow (Matthews *et al.*, 1996). Brännström *et al.* (1969) suggested that the outward movement might be related to sharp pain and the inward movement to dull pain. An intradental nerve in the cat is excited by a flow rate of approximately 1-1.5 × 10^3 pL/s per 1 mm^2 of exposed dentine (Matthews and Vongsavan 1994). Nevertheless, there is no information about how much fluid movement is required to cause pain in human subjects.

Since occlusal forces can displace a substantial amount of fluid into the pulp (Paper #5), it is possible that pulpal mechanoreceptors are activated in a similar way to that suggested by the hydrodynamic theory. We do not know where the pulpal nerves are activated but in an anisotropic enamel model the dentine just below the loading point would be most deformed (Spears *et al.*, 1993). The dentine fluid would be squeezed from here and into the pulp chamber. The movement could activate nerve endings wherever they exist in dentine or pulp. Since the amount of the fluid being displaced is increased proportionally with the magnitude of force, the response from pulpal mechanoreceptors (discharge frequency and the number of firing nerves) would increase as the force is increased.

Those nerve endings near pulp horns may be best suited for detecting occlusal forces because occlusal loads would theoretically displace the greatest amount of fluid here. This is due to (1) the higher density of dentinal tubules (Pashley, 1996) and (2) the high stress around any acute-angled space (Johnson *et al.*, 1968; De Vree *et al.*, 1983). It is interesting that dentine is clinically more sensitive in the cervical than the occlusal region (Dowell and Addy, 1983; Flynn *et al.*, 1985). It is possible that most of the nerves observed in pulp horns (Fearnhead, 1957; Arwill, 1967; Lilja, 1979; Holland, 1981) are mechanoreceptors rather than nociceptors.

A-β fibres may carry non-nociceptive information from the pulp of a tooth (Dong and Chudler, 1984; Dong *et al.*, 1985, 1990; Olgart *et al.*, 1988) as they are known to carry tactile information elsewhere in the body (Dubner *et al.*, 1978). The possibility that pulpal A-δ fibres may mediate proprioceptive input cannot be excluded since many of these fibres supplying thin body hairs (D-hair receptors) are also low-threshold mechanoreceptors (Burgess and Perl, 1973). Both A-β and A-δ fibres in teeth respond to hydrodynamic stimuli whereas C-fibres do not (Närhi *et al.*, 1992*b*). Although A-β and A-δ fibres may belong to the same functional group (Närhi *et al.*, 1992*b*), A-β fibres do not respond to rapid heating or cooling (Dong *et al.*, 1985).

Mechanosensitive information from the tooth pulp would probably be carried by A-β nerves which project to low-threshold mechanoreceptive (LTM) neurones (Hayashi *et al.*, 1984) in the trigeminal spinal nuclei, particularly nucleus interpolaris and nucleus oralis which contain approximately 75% and 50% respectively of LTM neurones (Hayashi and Tabata, 1989). LTM neurones in both nuclei have been shown to respond to electrical pulp stimulation at low intensities (Stephan, 1976; Hayashi *et al.*, 1984; Cairns *et al.*, 1995) below the pain threshold in man (Matthews *et al.*, 1976). The responses have short latencies, in the range of 7-10 ms (Hayashi *et al.*, 1984; Cairns *et al.*, 1995). The axons of many LTM neurones from both nuclei project to the ventro-postero-medial (VPM) nuclei of the thalamus (Darian-Smith, 1966; Rowe and Sessle, 1972; Azerad *et al.*, 1982; Sunada *et al.*, 1990) while some probably project to cranial motor nuclei (Landgren *et al.*, 1986; Sessle, 1986). The jaw-opening reflex has been suggested to be mediated via inhibitory interneurons in the supratrigeminal nucleus and, with a longer latency, via reticular formation (Sumino, 1971). These neural pathways possibly account for the reduction in MBF in Papers #2 and #4.

There are some differences between the pattern of fluid movement caused by occlusal forces and by pain-producing osmotic solutions. First, occlusal forces create a transient movement as opposed to the prolonged flow observed with osmotic stimuli. Second, fluid is displaced towards the pulp when a tooth is loaded and drawn back when the load is removed. A-β fibres seem to be suitable for monitoring occlusal loads since they are rapidly-adapting and respond well to vibratory stimuli (Dong *et al.*, 1985).

If the measurement of fluid movement in Paper #5 and in previous studies is accurate then occlusal loads should apparently activate pulpal nociceptors. Perhaps nociceptors are activated but the information is suppressed (gated) by a central descending influence (see Sessle, 1987) or by peripheral inputs travelling in large-diameter nerve fibres simultaneously excited during biting (Bowsher, 1980*a*). Such an input might come from periodontal, muscle or skin mechanoreceptors. Modulation of pain by peripheral stimuli such as acupuncture and transcutaneous electrical nerve stimulation has been demonstrated (see Melzack, 1984). It has been shown that mechanical stimuli applied to a tooth can suppress the excitation of trigeminal brain stem neurons evoked by stimulating the pulp of the same tooth (Sessle *et al.*, 1977). A similar mechanism may account for the observation that tooth pain may be relieved when the painful tooth is pressed although the relief may be due to a diminished blood supply to the pulp.

An alternative explanation is that different mechanisms activate pulpal mechanoreceptors and nociceptors. For example, (1) pulpal mechanoreceptors might only be activated by bending dentinal tubules, an effect of stress, (2) the transient inward fluid movement caused by tooth loading might differentially activate only the rapidly-adapting pulpal mechanoreceptors.

The activation threshold of these putative pulpal mechanoreceptor was not tested. It is likely that the same receptors are used during hardness discrimination and when producing an MBF. In this case, they are activated by forces ranging from 13-71 N on incisor teeth (the detection thresholds used in hardness discrimination; Table 4-1). These moderate forces activate the mechanoreceptors and their input is consciously used to detect hardness. The same but larger input may also be used to reflexly suppress the activity of jaw closing muscles. It is not known, however, what level of occlusal force would initiate the reflex. The threshold of the reflex may be tested experimentally by comparing controlled ramps of two MBFs, one when biting on acrylic and the other when biting on rubber. Pulpal mechanoreceptors are most likely to be activated when the teeth bite on the acrylic. The point at which the bite force on the acrylic starts to deviate from that on the rubber would identify the threshold of pulpal mechanoreceptors involved in the inhibitory reflex.

Alternatively, assuming that the receptors involved in hardness discrimination and MBF tasks are the same, their activation threshold may be estimated from the detection threshold measured in the hardness discrimination test (Paper #3). This was about an average stress of 4 N/mm^2 for an upper incisor tooth and 17 N/mm^2 for a molar tooth (assuming the contact area is about 5 mm^2). These values, however, are those required to detect differences in hardness. The actual threshold is likely to be lower.

The pupal mechanoreceptors proposed by Loewenstein and Rathkamp (1955) respond to such small occlusal forces that we cannot see how they could be activated by deforming such hard tissues as enamel and dentine. Those proposed by Dong and co-workers (Dong *et al.*, 1985, 1993) also respond to very small forces.

Tubular dentine seems to be a perfect stress sensor. It is designed to equip a calcified tissue with the ability to detect the magnitude of large forces and the direction in which they deform it. The tiny tubules filled with fluid monitor the amount of force applied to a tooth. They are far more easily deformed than the surrounding intertubular dentine. The amount of force is related to the volume of fluid displaced. The point where force is applied to the tooth may be detected by monitoring the region where there is the largest inward movement of fluid.

RESEARCH IMPLICATIONS

The existence of mechanoreceptors inside the tooth certainly will have an impact on research in pulpal biology. Those neural responses observed previously (*e.g.* jaw opening reflex, pre-pain sensation, vasodilation *etc.*) following pulpal stimulation could as well reflect the activation of pulpal mechanoreceptors, as opposed to nociceptors. Future physiological and biochemical studies may be able to confirm the existence of the pulpal mechanoreceptors.

The presence of pulpal mechanoreceptors helps to explain the function of tubules in dentine as well as those in the enamel of most marsupials. It also provides an alternative explanation of why a dental pulp has been retained throughout mamalian evolution. Pulpal nerves may be necessary for learning how to masticate. The pointed pulpal horns in young teeth enhance stress concentration in that area and presumably increase the sensitivity of force detection in young mammals. The sensitivity may gradually decline as the tooth is worn down where peritubular dentine becomes thicker (reviewed by Pashley, 1996) and many tubules are blocked.

Bone and orthodentine, the type of dentine found in human teeth, evolved at about the same time during evolution, at a time when many tissues intermediate between bone and orthodentine existed (Orvig, 1951). A form of dentine comparable to bone, called osteodentine, still exists in many fish. The mechanism used to detect forces on dentine suggests a similar mechanism could exist in bone. The formation of a Haversian system (*i.e.* osteon) in compact bone is comparable to that of dentine (see Cormack, 1987). The osteoblasts secrete new bone matrix and recede inwards to become a Haversian canal which consists of tissue similar to that in the pulps of teeth. However, unlike the odontoblasts in orthodentine, osteoblasts are trapped in the bone matrix where they are referred to as osteocytes. Each Haversian system contains numerous canaliculi formed by the interconnected processes of the embedded osteocytes. It has been suggested that forces applied to bone move fluid along these canaliculi and activate bone remodelling (Duncan and Turner, 1995). Thus, when both dentine and bone are distorted by applied forces, fluid is moved through a tubular system and activates a response. Perhaps, like the pulps of teeth, the Haversian canals in bone also contain mechanoreceptors.

If dental pulp serves to detect large forces on the tooth, the removal of pulpal tissue during root canal treatment would result in a loss of jaw proprioception. It has been suggested that this loss may be the cause of the more frequent tooth fracture of root filled teeth (Gulabivala, 1995), presumably due to failure adequately to control large bite forces. Most operative procedures on teeth involve removing tooth tissue thereby reducing their stiffness. By itself, this enhances the sensitivity of the pulpal mechanoreceptors because a given applied force causes a greater deformation. But the response of the pulp, laying down more peritubular dentine and secondary dentine, partially compensates by stiffening the tooth. It is also possible that the trauma of cavity preparations may irreversibly damage some pulpal nerves and impair pulpal mechanoreception.

The present conclusion, that teeth may contain mechanoreceptors, is based on observations of human responses to tooth clenching. It is consistent with previous neurophysiological findings in animals (Dong *et al.*, 1985, 1990; Olgart *et al.*, 1988) although the forces we studied were several orders of magnitude larger. These receptors are activated without causing pain. Both pulpal nociceptors and mechanoreceptors seem to be activated by a similar mechanism. Future work may be directed towards looking for some form of gating mechanism to explain why activing pulpal mechanoreceptors does not cause pain.

TOPICS FOR FUTURE RESEARCH

(1) What is the threshold of pulpal mechanoreceptors in human subjects?

(2) Is the ability to discriminate between hardness (used in Paper #3) reduced with non-vital teeth?

(3) Is the MBF on non-vital teeth larger than that on vital teeth? (There is a risk of fracturing the tested teeth)

(4) Can periodontal stimulation modulate pulpal responses to pain-producing stimuli?

(5) Can periodontal mechanoreceptors alone be used to detect forces larger than 10 N?

(6) Do trigeminal LTM neurones preferably respond to stress in the tooth?

(7) Do the intradental nerves fire (recorded from electrodes in a cavity in dentine) in response to chewing in human subjects? Are some of the nerve fibres recorded in the inferior dental nerve which respond to electrical pulp stimulation also excited by force applied to the tooth?

(8) Are nerves inside dentine (particularly those near pulpal horn) more responsive to occlusal loads than nerves in other parts of tooth pulp? What are their parent axons and where are their central projections?

(9) Are there any differences in fluid displacement (volume and flow rate) *in vitro* between concentrated (high-stress) and distributed (low-stress) occlusal forces?

(10) Is the fluid flow caused by occlusal loads more localized (affecting a small area of the pulp) or more distributed than that caused by pain-producing stimuli?

(11) Is it possible that mechanoreceptors exist in Haversian canals ?

CONCLUSION

The results of the experiments reported here suggest that human incisors may contain pulpal mechanoreceptors. They may be *consciously* used to detect the hardness of objects clenched between the upper and lower teeth. They may be *subconsciously* used to monitor large stresses on the crowns and thereby limit the maximum bite force. Apparently, pain is not involved. Measurements *in vitro* show that forces within the range used in these studies displace fluid in dentine. Thus the putative pulpal mechanoreceptors may be activated by fluid movement similar to that associated with the hydrodynamic theory of dentine sensitivity.

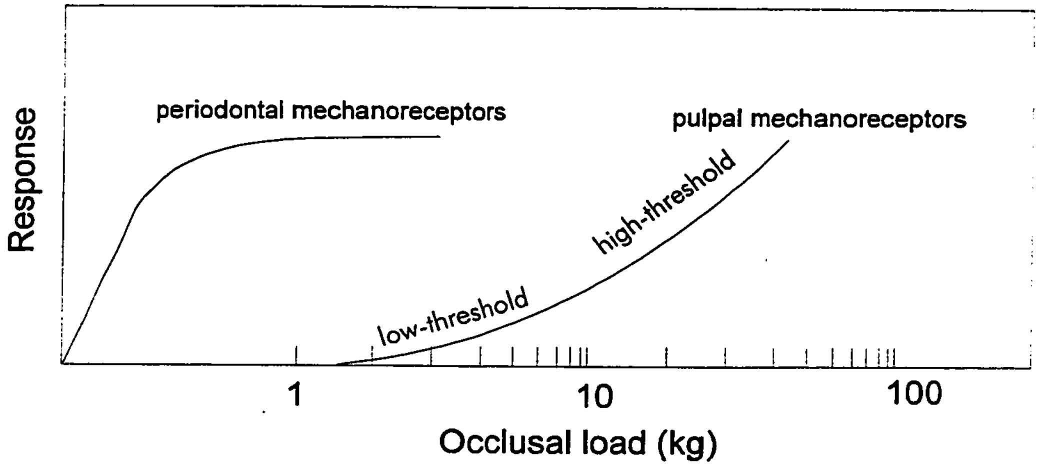

Figure 7-1 Theoretical responses expected from periodontal and pulpal mechanoreceptors when a tooth is loaded.

It is postulated (Fig. 7-1) that periodontal mechanoreceptors are most effective in detecting forces below 10 N (~ 1 kg) applied on a given tooth whereas pulpal mechanoreceptors are probably used to detect bite forces above 10 N. At about 10 N the response from periodontal receptors is likely to be saturated. Above 10 N pulpal receptors start to respond to an increasing stress on the tooth crown and probably continue firing until the MBF. There may be at least two different subgroups of pulpal mechanoreceptors, low- and high-threshold. Although other mechanoreceptors in the oro-facial region can indirectly help to detect the amount of bite force on the tooth (see Chapter 1) their affects were controlled during the experiments.

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APPENDIX A - TESTING THE BITE FORCE TRANSDUCER

1. Calibration of the transducer

Each new transducer requires a calibration for force direction and magnitude. It also needs a re-calibration after approximately 10-15 experiments or whenever the baseline strain (the strain when the transducer is not loaded) has changed >10 με[1] (microstrain). A thin transducer generally requires re-calibration more often than a thick one.

1.1 Method of calibration (Fig. A-1)

A metal nut (sized to fit the bolt on the tripod) was glued to the centre of the bottom of the horizontal plate by dental acrylic resin (Dura Lay, Dental Mfg, IL, USA). A small drop of unset Dura Lay was placed at the centre of the upper side of the plate. A shallow indentation of the loading pin was made in the resin in order to prevent the pin from slipping under loads. Once the resin hardened the transducer was mounted on a sturdy metal camera tripod.

The force direction was calibrated by tilting the transducer first in the front/back plane with an increment of 15° from the horizontal position (*i.e.* P = -30°, -15°, 0°, 15°, 30° ; forward direction = + and backward direction = –) and later in the right/left plane (*i.e.* Q = -30°, -15°, 0°, 15°, 30° ; right direction = + and left direction = – relative to the transducer). For each of the above positions, the baseline in all gauges was zeroed and strain values were recorded when a fixed 10-kg load was applied to the top of the pin.

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[1] 1 με = 1 μ of deformation per 1 m of length.

Finally, the transducer was calibrated when it was tilted simultaneously in both planes (*i.e.* P/Q = 15°/15°, 15°/-15°, -15°/15°, -15°/15°).

The force magnitude was calibrated by applying various loads (*i.e.* 2, 3, 5, 7, 8, 10, 12, 13, 15 kg) to the transducer mounted horizontally (P/Q = 0°/0°).

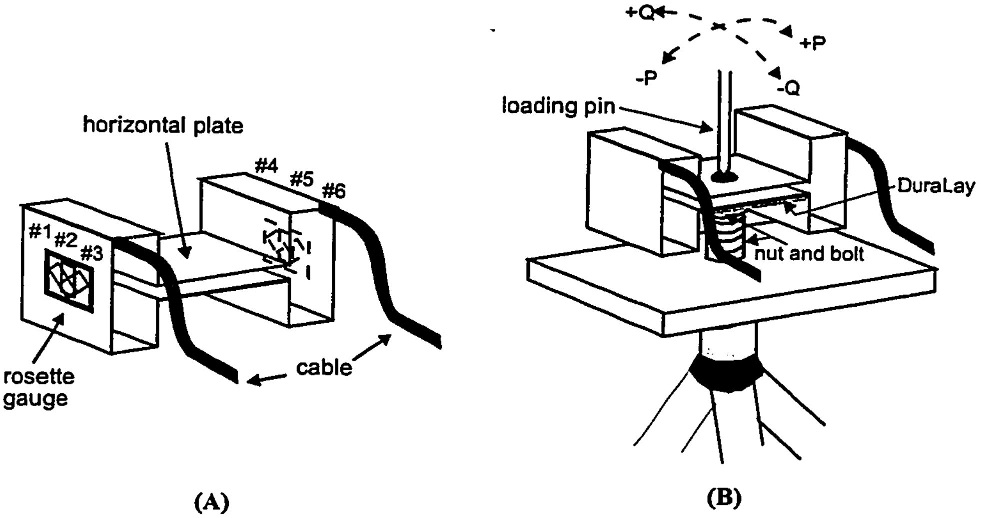


Figure A-1 Diagram showing the calibration of an H-shaped transducer. (A) H-shaped transducer with gauges #1 - #3 on the right and #4 - #6 on the left of the vertical arms. (B) The transducer is mounted on a camera tripod. The orientation of the transducer can be changed by tilting the tripod. Loads are applied to the top of the loading pin. (+P = right direction, -P = left direction, +Q = forward direction, -Q = backward direction)

1.2 Formulas for calibration (Table A-1)

The strain values were imported into a spreadsheet. Customized formulas were used in calculating ratios of the strains among gauges. If the strains in gauge #1, #2, #3, #4, #5, #6 are ε_1, ε_2, ε_3, ε_4, ε_5, ε_6 respectively, then

$$\text{front/back ratio (Q-ratio)} = \frac{(\varepsilon_1+\varepsilon_4)-(\varepsilon_3+\varepsilon_6)}{\varepsilon_1+\varepsilon_3+\varepsilon_4+\varepsilon_6} \qquad \text{(A-1)}$$

$$\text{right/left ratio (P-ratio)} = \frac{(\varepsilon_1+\varepsilon_3)-(\varepsilon_4+\varepsilon_6)}{\varepsilon_1+\varepsilon_3+\varepsilon_4+\varepsilon_6} \qquad \text{(A-2).}$$

P- and Q-ratios were used as dependent variables to run a linear regression between them and actual directions. A regression equation was used to calculate the direction from the ratio. The calculated directions were often slightly different from the actual values and needed to be adjusted by multiplying them with an arbitary number [ranging from -0.5 to 0.5; Table A-1(a)].

For force magnitude, the maximum strain on each side of the vertical plates was calculated by equations (Perry and Lissner, 1962),

$$\varepsilon_{\max}\,(\text{right}) = \frac{\varepsilon_1+\varepsilon_3}{2}+\frac{1}{2}\sqrt{(\varepsilon_1-\varepsilon_3)^2+[2\varepsilon_2-(\varepsilon_1+\varepsilon_3)]^2} \qquad \text{(A-3)}$$

$$\varepsilon_{\max}\,(\text{left}) = \frac{\varepsilon_4+\varepsilon_6}{2}+\frac{1}{2}\sqrt{(\varepsilon_4-\varepsilon_6)^2+[2\varepsilon_5-(\varepsilon_4+\varepsilon_6)]^2} \qquad \text{(A-4)}$$

The maximum strain on both sides were averaged at a given load. A regression equation (between average strain and actual loads) was used to calculate the loads from the average strain. Correction factors (the product of calculated direction and an arbitary number) were added to the original average strain to obtain a more accurate value [Table A-1 (b)].

All necessary equations were incorporated into a customized computer program written in BASIC (Osborn and Mao, 1993) which displayed instantaneously the direction and magnitude of forces on a computer screen.

Table A-1 Algorithms used in calibrating (a) force direction and (b) force magnitude. Note that the calculated P-direction of this transducer does not need to be corrected.

(a) Calibration of force direction

Actual direction P*	Actual direction Q*	P-ratio	Q-ratio	P-calculated (p)	Q-calculated (q)	P-corrected‡ 0.2(+q)	P-corrected‡ 0.1(-q)	Q-corrected‡ 0(+p)	Q-corrected‡ 0(-p)
0	-30	0.02	-0.46	2	-29		-1	-29	
0	-15	0.03	-0.21	3	-15		1	-15	
0	0	-0.01	0.03	-6	-2		-6		-2
0	15	-0.00	0.35	-4	15	-1			15
0	30	0.01	0.64	-2	31	5			31
-30	0	-0.12	0.02	-29	-3		-29		-3
-15	0	-0.06	0.01	-16	-3		-17		-3
0	0	0.01	0.05	-1	-1		-1		-1
15	0	0.09	0.08	16	1	16		1	
30	0	0.15	0.11	29	2	30		2	
-15	-15	-0.04	-0.19	-12	-14		-13		-14
-15	15	-0.07	0.33	-17	14	-14			14
15	-15	0.08	-0.17	14	-13		12	-13	
15	15	0.06	0.31	10	13	12		13	

(b) Calibration of force magnitude

P-calculated (p)	Q-calculated (q)	Average max. strain (με)	Correction factor -1.3(-p)	Correction factor 0.5(+p)	Correction factor -0.5(-q)	Correction factor 0.5(+q)	Corrected strain (με)†	Corrected load (kg)§
2	-29	248		1.1	14.4		264	9.6
3	-15	268		1.2	7.7		277	10.1
-6	-2	267	7.9		1.0		276	10.0
-4	15	262	5.1			7.6	274	10.0
-2	31	242	1.9			15.5	259	9.4
-29	-3	220	37.3		1.4		259	9.4
-16	-3	254	21.2		1.7		277	10.1
-1	-1	270	0.6	-0.2	0.5		271	9.8
16	1	264		8.1		0.3	272	9.9
29	2	248		14.7		1.0	264	9.6
-12	-14	252	15.3		7.2		274	10.0
-17	14	250	22.4			6.9	279	10.1
14	-13	255		6.8	6.5		268	9.7
10	13	263		4.9		6.6	274	10.0

[*] +P = right direction, -P = left direction, +Q = forward direction, -Q = backward direction (in degrees)

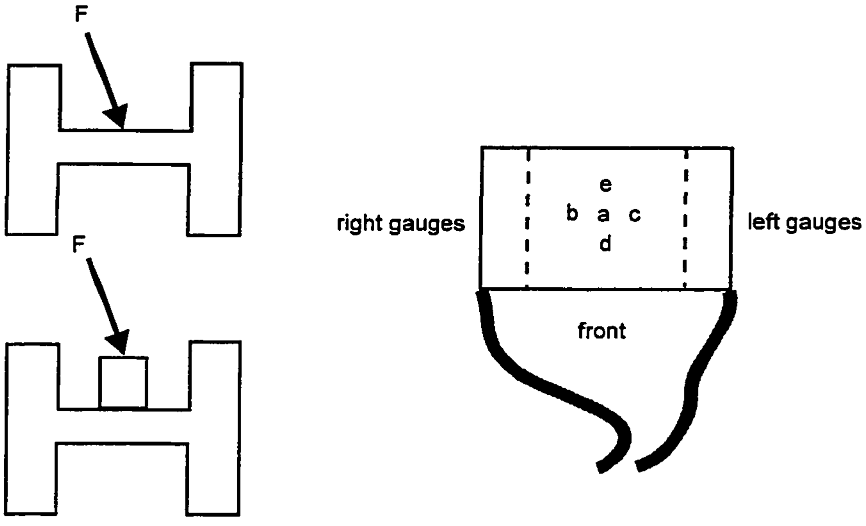
[‡] Corrected directions are the product between an arbitary number and the corresponding p or q.

[†] Corrected strains are the sum of the average maximum strain and correction factors.

[§] Corrected loads are calculated from corrected strain by a regression equation. The calibrating load is 10 kg.

2. Testing the effect of the position of the point of load application on the response of the H-shaped transducer

2.1 Vertical position of the loading point



(A) (B)

Figure A-2 Testing the effect of the point of load application on the responses from an H-shaped transducer. (A) Comparison made when the point of application is at (upper diagram) and 7-mm above (lower diagram) the horizontal plate. (B) Comparison made when the point of application is at the centre (a), 3-mm right (b), 3-mm left (c), 3-mm front (d) and 3-mm back (e) on the horizontal plate (viewed from above).

The height of loading point affects the way the vertical limbs are deformed and hence the accuracy in measuring a force direction. Table A-2 shows strain values and P, Q angles measured during the calibration when the tip of the loading pin was placed near the horizontal plate (Fig. A-2A, upper). Table A-3 shows the response from the same transducer when the tip was placed on the top of a 7-mm tall acrylic block attached to the horizontal plate (Fig. A-2A, lower). Increasing the height of the loading point tends to over-measure the angles, particularly for the Q-direction.

Table A-2 Calibration data when loads (10 kg) are applied at the horizontal plate of an H-shaped transducer (Hgreen 93) (height = 0 mm).

Actual direction		Strain (µε)								Values measured from the transducer		
		Right rosette†			Left rosette†							
P*	Q*	#1	#2	#3	#4	#5	#6	P-ratio$^{\Psi}$	Q-ratio$^{\Psi}$	P	Q	Load (kg)
0	-30	64	242	163	56	235	160	0.02	-0.46	-1	-29	9.6
0	-15	104	272	153	93	258	151	0.03	-0.21	1	-15	10.1
0	0	130	264	113	125	270	125	-0.01	0.03	-6	-2	10.0
0	15	166	248	73	158	262	83	-0.00	0.35	-1	15	10.0
0	30	181	217	30	163	230	45	0.01	0.64	5	31	9.4
-30	0	93	188	89	118	252	114	-0.12	0.02	-29	-3	9.4
-15	0	116	235	109	126	273	129	-0.06	0.01	-17	-3	10.1
0	0	141	269	115	125	270	125	0.01	0.05	-1	-1	9.8
15	0	154	282	118	116	245	111	0.09	0.08	16	1	9.9
30	0	157	277	108	98	217	97	0.15	0.11	30	2	9.6
-15	-15	91	237	131	96	263	145	-0.04	-0.19	-13	-14	10.0
-15	15	144	224	65	153	265	86	-0.07	0.33	-14	14	10.1
15	-15	117	272	144	84	234	139	0.08	-0.17	12	-13	9.7
15	15	175	267	81	142	248	85	0.06	0.31	12	13	10.0

* P and Q are angles (in degrees) measured in left/right and front/back planes of the transducer respectively (right = +, left = –, front = +, back = –). † Right and left sides of the transducer. $^{\Psi}$ P-ratio = $\frac{(\#1+\#3)-(\#4+\#6)}{\#1+\#3+\#4+\#6}$ and Q-ratio = $\frac{(\#1+\#4)-(\#3+\#6)}{\#1+\#3+\#4+\#6}$.

Table A-3 Calibration data from the same transducer used in Table A-2 but the loads are applied on the top of a 7-mm tall acrylic block attached to the horizontal plate (height = 7 mm).

Actual direction		Strain (με)								Values measured from the transducer		
		Right rosette†			Left rosette†							
P*	Q*	#1	#2	#3	#4	#5	#6	P-ratio$^{\psi}$	Q-ratio$^{\psi}$	P	Q	Load (kg)
0	-30	48	232	182	53	230	168	0.02	-0.55	-2	-34	9.7
0	-15	87	247	141	92	261	144	-0.02	-0.23	-8	-16	9.9
0	0	135	252	114	129	269	120	0.00	0.06	-3	-1	9.9
0	15	175	240	60	160	264	79	-0.01	0.41	-1	19	10.1
0	25	194	206	7	184	232	34	-0.04	0.80	-4	40	10.1
-30	0	106	182	74	129	281	123	-0.17	0.09	-38	1	9.8
-15	0	123	231	95	129	279	129	-0.08	0.06	-21	-1	10.2
0	0	132	250	107	128	273	124	-0.03	0.06	-9	-1	10.1
15	0	131	257	111	110	226	105	0.06	0.05	9	-1	9.8
30	0	117	240	105	85	171	84	0.14	0.03	25	-2	9.4
-15	-15	80	222	135	93	270	153	-0.07	-0.25	-19	-17	10.3
-15	15	162	214	46	162	274	92	-0.10	0.40	-21	18	10.5
15	-15	94	255	142	78	219	122	0.08	-0.21	13	-15	9.8
15	15	164	246	69	136	226	70	0.06	0.37	13	16	10.0

*, †, $^{\psi}$ see Table A-2

2.2 Horizontal position of the loading point

The measurement of both magnitude and direction of a force is also affected by the position of the loading point on the horizontal plate (Fig. A-2B). The strain on the side closer to the loading point tends to be increased (Table A-4). Moving the loading point toward the right or left side affects the values being measured more than moving it toward the front or back.

Table A-4 Comparison of strain values when a load (10 kg) is applied at the centre, 3 mm to the right, 3 mm to the left, 3 mm to the front and 3 mm to the back of the transducer. Note the values when the force is vertical (P=0, Q=0).

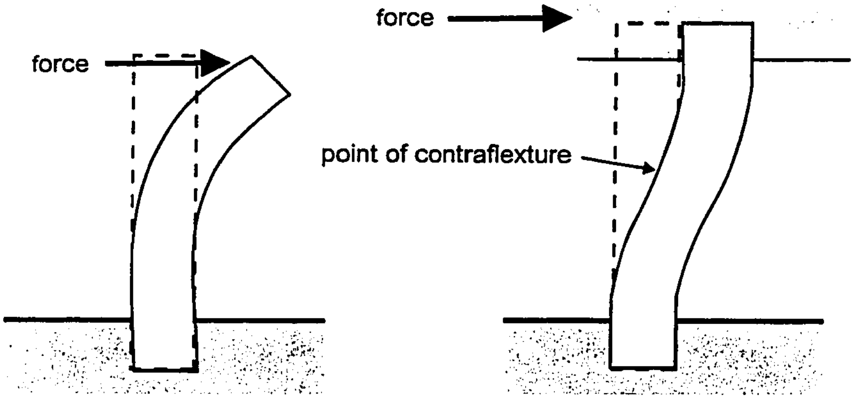
		Strain (με)										
		Right gauges[†]			Left gauges[†]					Values measured from thetransducer		
P[•]	Q[•]	#1	#2	#3	#4	#5	#6	P-ratio[ψ]	Q-ratio[ψ]	P	Q	Load (kg)
(a) Loads centred												
0	-15	191	412	245	203	407	220	0.02	-0.08	-1	-13	9.7
0	0	222	438	210	226	411	206	0.00	0.04	-2	-0	9.9
0	15	244	403	169	240	384	159	0.02	0.19	-1	16	9.7
-15	0	175	323	173	250	444	225	-0.15	0.03	-17	-1	9.4
15	0	256	477	251	162	328	157	0.23	0.01	20	-3	10.1
(b) Loads off-centred to the right												
0	-15	205	475	266	164	350	197	0.13	-0.11	10	-16	9.5
0	0	244	486	239	190	348	180	0.13	0.02	10	-2	10.1
0	15	264	437	199	229	347	122	0.14	0.21	11	18	9.4
-15	0	187	376	195	228	418	193	-0.05	0.03	-7	-1	9.4
15	0	263	527	281	155	262	132	0.31	0.01	27	-4	10.2
(c) Loads off-centred to the left												
0	-15	158	376	231	208	455	249	-0.08	-0.13	-10	-19	10.4
0	0	202	379	198	243	423	211	-0.06	0.04	-8	0	8.8
0	15	214	345	153	253	422	183	-0.09	0.16	-11	13	9.7
-15	0	147	277	152	263	476	240	-0.25	0.02	-27	-2	9.6
15	0	231	471	235	179	326	162	0.15	0.02	13	-3	9.7
(d) Loads off-centred to the front												
0	-15	188	410	238	199	415	210	0.02	-0.07	-0	-12	9.8
0	0	229	444	228	217	389	191	0.06	0.03	3	-1	9.7
0	15	248	407	173	230	361	134	0.07	0.22	5	19	9.4
-15	0	168	320	149	264	467	201	-0.19	0.10	-21	7	9.6
15	0	263	492	251	179	297	146	0.23	0.05	19	2	10.0
(e) Loads off-centred to the back												
0	-15	180	423	252	169	395	244	0.02	-0.17	-0	-23	10.0
0	0	219	440	239	214	393	200	0.05	-0.01	2	-5	9.8
0	15	243	409	193	224	369	159	0.06	0.14	4	11	9.2
-15	0	162	327	178	250	464	217	-0.16	0.02	-18	-2	9.8
15	0	243	497	270	167	317	162	0.22	-0.03	19	-7	10.0

•, †, ψ see Table A-2

3. Modification of the H-shaped to a U-shaped transducer

One drawback of the H-shaped transducer is that it is sometimes not sensitive to changes in forces applied in P-directions. The sensitiviy to detect force direction in this plane (as indicated by P-ratios) is improved if the two vertical plates are deformed in the opposite way, *i.e.*, tension on one side and compression on the other.

If a lateral force is applied to a vertical beam whose bottom end is fixed in the ground (a cantilever), a simple deflection occurs (Fig. A-3A). However, when the same lateral force is applied to a second beam with both ends are restrained, *i.e.* one end in the ground and the other end in another rigid structure (so called "sinking support"), a double deflection is observed (Fig. A-3B). The centre of the beam is least deformed (so called "the point of contraflexture"). This is because the upper end of the cantilever can be bent freely whereas that of the second beam is restrained from bending sideways. One can visualize the second beam as two cantilevers whose free ends are joined.



(A) (B)

Figure A-3 The bending of a beam when a lateral force is applied to (A) a simple cantilever beam and (B) a beam with both ends restrained. Note a double deflection in (B).

The deformation of the vertical plates of the H-shaped transducer is probably similar to the latter case. When a lateral force (F) is applied to the transducer both vertical limbs are bent in such a way that a double deflection occurs (Fig. A-4A) because its upper (x) and lower (y) corners are stiff. The point of contraflexture is probably located at the centre of each limb. Placing a strain gauge exactly at this position would measure little changes in the P-ratio as the direction of the force is changed in the right/left plane. This may account for the insensitivity of the H-shaped transducer to lateral forces.

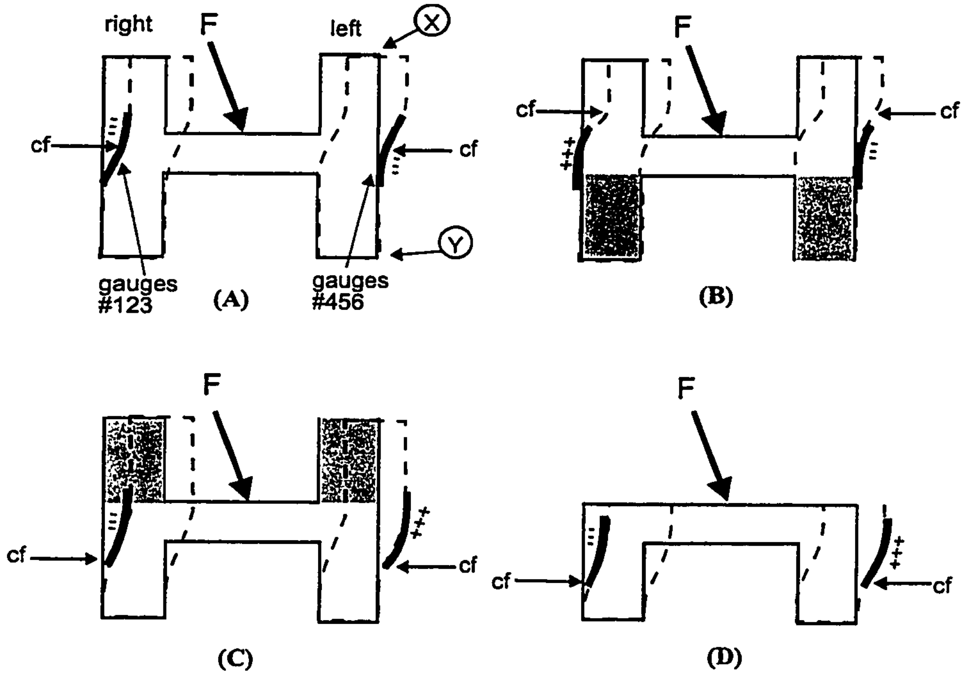


Figure A-4 Deformation (front view) of the vertical limbs of (A) an unfilled H-shaped, (B) an H-shaped with lower half filled, (C) an H-shaped with upper half filled, (D) a U-shaped transducer. Note that the gauges on both sides of an unfilled H-shaped transducer are compressed in the same way and therefore almost no difference in strain can be detected. Filling the upper half yields an opposite deformation to filling the lower half. Tension is + and compression is -; cf = point of contraflexture.

We have discovered that filling the lower half of the vertical limb improves the sensitivity (Fig. A-4B and Table A-5). The filler stiffens the lower half of the limbs, hence creating an asymmetry along the vertical limbs and probably moves the point of contraflexture upwards. There is more tension on the right and more compression on the left of the transducer (Fig. A-4B). The difference in the strain between both limbs is increased, resulting in a better sensitivity in detecting a lateral force. The opposite is true when the upper half is filled (Fig. A-4C).

With the notion that the transducer is more sensitive when the housing is not vertically symmetrical we modified the original H-shaped into a U-shaped transducer (Fig. A-4D). The U-shaped transducer behaves in the same way as the H-shaped with the upper half filled (Table A-6). The strain on the right side is gradually increased as the direction of the force shifts from 30° left to 30° right. The sensitivity is somewhat increased if the rosettes are not placed at the centre of the vertical limbs. This is probably because the position of the strain gauge is moved further away from the point of contraflexture.

Some advantages of the U-shaped transducer are that (1) it can be placed more easily on the dental arch because there is no interference between the upper limb and the opposing teeth and (2) the transducer could be made smaller, incorporated in a dental bridge and used to measure chewing forces.

Table A-5 Comparison of strain values of an H-shaped transducer before and after the lower half of the vertical limbs is filled with acrylic resin.

		Strain (με)							
		Right gauges†			Left gauges†				
P*	Q*	#1	#2	#3	#4	#5	#6	P-ratio$^{\psi}$	Q-ratio$^{\psi}$
(a) Unfilled									
0	-30	37	224	178	40	218	171	0.01	-0.64
0	-15	84	266	161	98	257	144	0.01	-0.25
0	0	127	283	142	142	265	117	0.02	0.02
0	15	151	271	100	171	254	73	0.01	0.30
0	25	163	257	80	179	237	50	0.03	0.45
-30	0	110	239	119	104	220	112	0.03	-0.04
-15	0	121	274	137	130	253	121	0.01	-0.01
0	0	123	278	139	142	265	116	0.01	0.02
15	0	117	276	138	142	263	112	0.00	0.02
30	0	103	146	129	133	229	89	0.02	0.04
-15	-15	86	258	155	91	248	149	0.00	-0.26
-15	15	148	258	92	162	242	74	0.01	0.30
15	-15	84	262	159	102	246	134	0.01	-0.22
15	15	145	266	101	176	247	69	0.00	0.31
(b) Filled									
0	-30	39	217	173	61	236	163	-0.03	-0.54
0	-15	69	242	160	87	258	151	-0.02	-0.33
0	0	117	248	125	127	266	124	-0.02	-0.01
0	15	147	237	81	151	252	82	-0.01	0.29
0	25	166	213	42	164	219	48	-0.01	0.57
-30	0	112	264	149	107	215	94	0.13	-0.05
-15	0	115	269	144	116	247	109	0.07	-0.05
0	0	116	250	127	125	265	121	-0.01	-0.01
15	0	101	213	105	124	264	125	-0.09	-0.01
30	0	80	167	74	113	243	122	-0.21	-0.01
-15	-15	75	257	175	83	239	138	0.06	-0.33
-15	15	147	256	96	143	233	75	0.05	0.26
15	-15	61	202	132	91	255	150	-0.11	-0.30
15	15	129	201	59	145	243	86	-0.10	0.31

*, †, ψ see Table A-2

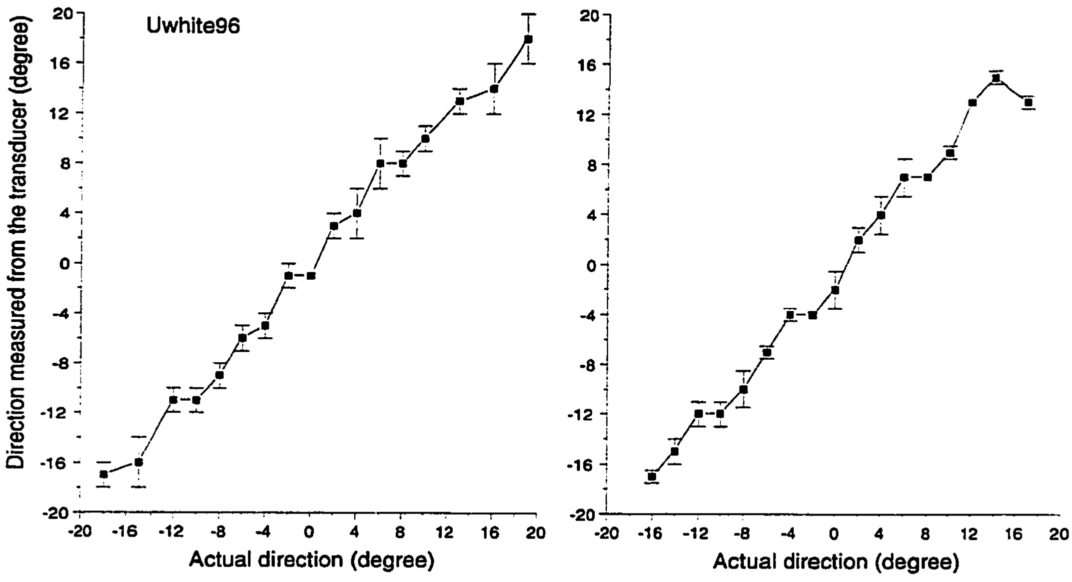
Table A-6 Comparison of strain values between (a) an H-shaped transducer with its upper vertical plates filled and (b) a U-shaped transducer.

		Strain (με)							
		Right gauges†			Left gauges†				
P*	Q*	#1	#2	#3	#4	#5	#6	P-ratio$^{\Psi}$	Q-ratio$^{\Psi}$
(a) Filled H-shaped									
0	**-30**	80	405	306	110	438	276	0.00	-0.51
0	**-15**	174	468	277	186	504	256	0.01	-0.19
0	**0**	240	498	228	246	516	220	0.00	0.04
0	**15**	279	463	158	280	475	163	-0.01	0.27
0	**30**	301	452	116	288	446	129	0.00	0.41
-30	**0**	183	364	178	283	623	256	-0.20	0.04
-15	**0**	223	456	217	246	536	215	-0.02	0.04
0	**0**	236	487	224	239	499	215	0.01	0.04
15	**0**	233	532	237	214	466	221	0.04	-0.01
30	**0**	254	586	240	181	380	184	0.15	0.01
-15	**-15**	138	398	260	206	561	283	-0.10	-0.22
-15	**15**	266	427	144	278	511	167	-0.04	0.27
15	**-15**	175	515	280	161	444	253	0.05	-0.23
15	**15**	283	535	175	256	435	159	0.05	0.23
(b) U-shaped									
0	**-30**	106	318	225	143	331	221	-0.05	-0.28
0	**-15**	122	318	205	167	350	208	-0.07	-0.18
0	**0**	159	315	180	188	344	184	-0.05	-0.02
0	**15**	171	309	147	189	318	153	-0.04	0.09
0	**30**	181	273	108	188	293	111	-0.02	0.26
-30	**0**	117	224	120	227	418	215	-0.30	0.01
-15	**0**	138	275	150	202	388	205	-0.17	-0.02
0	**0**	151	334	187	184	346	185	-0.04	-0.05
15	**0**	160	343	204	149	281	147	0.10	-0.06
30	**0**	169	347	193	120	220	110	0.22	-0.02
-15	**-15**	112	272	173	198	400	233	-0.20	-0.13
-15	**15**	153	258	116	211	365	174	-0.18	0.11
15	**-15**	134	346	223	127	274	167	0.10	-0.20
15	**15**	181	337	165	149	253	112	0.14	0.09

*, †, Ψ see Table A-2

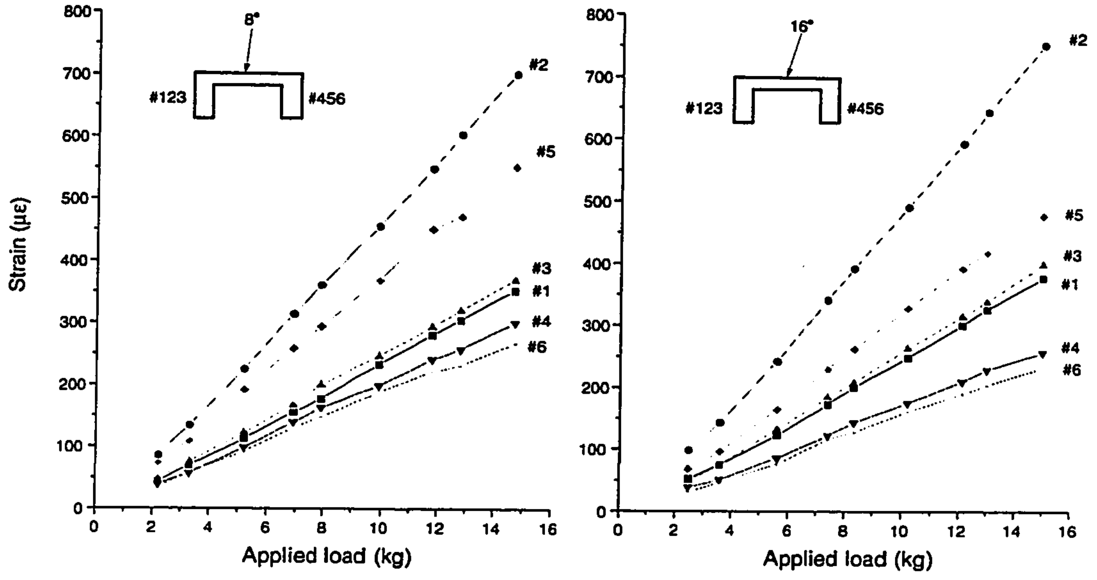
4. Testing the resolution of the U-shaped transducer

The resolution of the U-shaped transducer was tested separately in front/back (Q) and in right/left (P) directions. The directions were recorded when a 10-kg load was applied at an angulation varying from -16° to 16° away from vertical, with a 4° increment. The result showed that the transducer can discriminate a smallest difference of approximately 4° in both P and Q directions (Fig. A-5).



(A) **(B)**

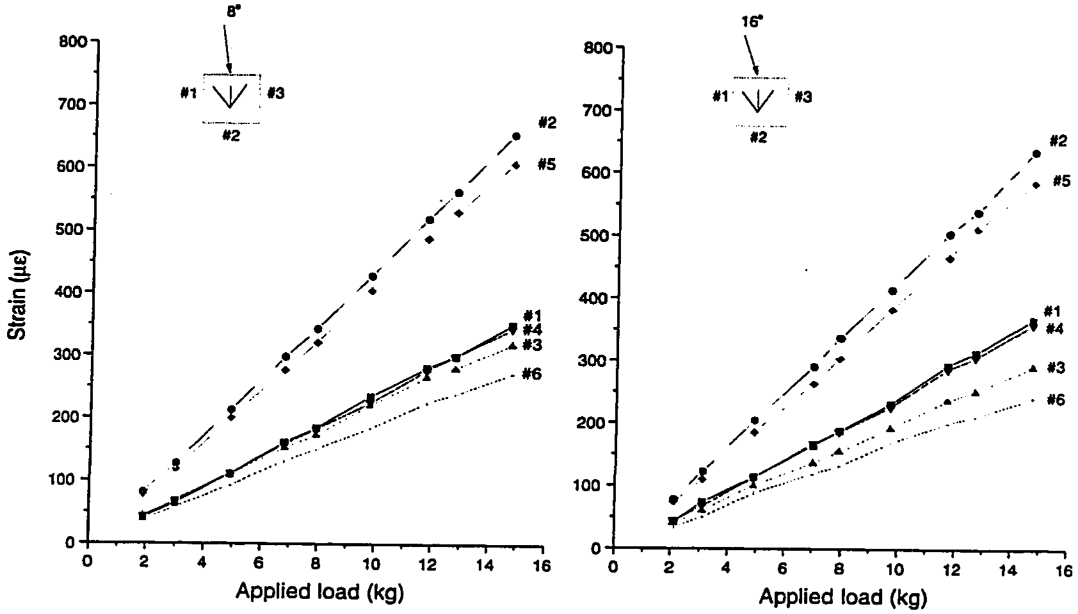
Figure A-5 Graphs showing the directions recorded by a U-shaped transducer when a 10-kg load is applied at angulations varying from -16° to +16° in (A) right/left (P-direction) and (B) front/back (Q-direction) planes. Each point is an average of four measurements.



(A) (B)

Figure A-6 Graphs showing the strains in all six gauges as the load is increased. (A) Loads applied at 8° to the right. (B) Loads applied at 16° to the right. Note the strain in the right gauges (#1-#3) is increased and the strain in the left gauges (#4-#6) is decreased as the force is shifted from 8° to 16°.

The response from each of the six gauges to different loads was tested by applying loads ranging from 2 to 15 kg, at 8° and 16° away from vertical. The results showed that the middle gauges of the rosettes (#2 and #5) are most sensitive in both right/left (Fig. A-6) and front/back (Fig. A-7) planes. In the right/left plane, the side towards which the force is directed is under tension whereas the opposite side is under compression. The larger the angulation, the greater the tension. In the front/back plane the gauges lying parallel to the direction of the force are under more tension than those lying perpendicular to it.



(A) **(B)**

Figure A-7 Graphs showing the strains in all six gauges as the load is increased. (A) Loads applied at 8° to the front. (B) Loads applied at 16° to the front. Note the strain in the back gauges (#1, #4) is increased and the strain in the front gauges (#3, #6) is decreased as the force is shifted from 8° to 16°.

References

Osborn J. W. and Mao J. (1993) A thin bite-force transducer with three-dimensional capabilities reveals a consistent change in bite-force direction during human jaw-muscle endurance tests. *Archs oral Biol* **38,** 139-144.

Perry C. C. and Lissner H. R. (1962) *The Strain Gage Primer.* McGraw-Hill, New York.

APPENDIX B - CALCULATING THE MOMENT ARM OF JAW MUSCLES

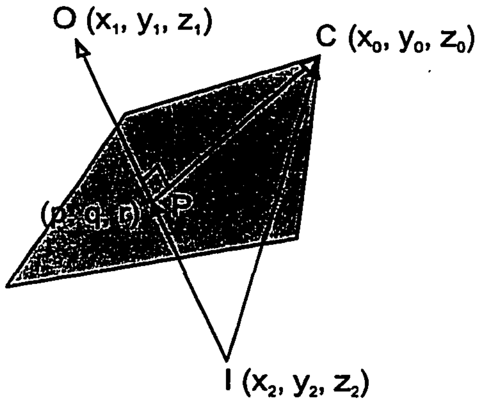


Figure B-1 Diagram showing vectors of a jaw muscle, the mandibular condyle (C), and the moment arm ($|P\bar{C}|$).

Let O and I be the origin and insertion of a jaw muscle respectively and C be the mandibular condyle. $|P\bar{C}|$ is the shortest distance from point C to $I\bar{O}$, *i.e.* the moment arm of the muscle.

The projection of $I\bar{C}$ on $I\bar{O}$, $I\bar{P}$, can be calculated from the equation (Nicholson, 1990)

$$\frac{I\bar{C}\cdot I\bar{O}}{|I\bar{O}|^2}\times I\bar{O} \qquad \text{(B-1)}$$

Hence, the length of the moment arm $P\bar{C}$ is simply $|I\bar{C} - I\bar{P}|$.

The moment arm of masseter and anterior temporalis muscles at two different jaw openings is shown in Table B-1.

Table B-1 The length of the moment arms of masseter and anterior temporalis muscles at two different jaw openings.

Jaw separation	Moment arm (cm)		$\frac{\text{AT's moment arm}}{\text{MA's moment arm}}$
	Masseter (MA)	Anterior Temporalis (AT)	
closed	3.52	3.21	0.91
14-16 mm incisal separation (10° rotation)	3.31	3.04	0.92
26-28 mm incisal separation (17° rotation + 7 mm translation)	2.62	2.55	0.98

References

Nicholson W. K. (1990) *Elementary Linear Algebra with Applications.* PWS-Kent Publishing, Boston.

APPENDIX C - CALCULATING CHANGES IN SARCOMERE LENGTH AS THE JAW IS OPENED SYMMETRICALLY

1. Data for muscle origins and insertions

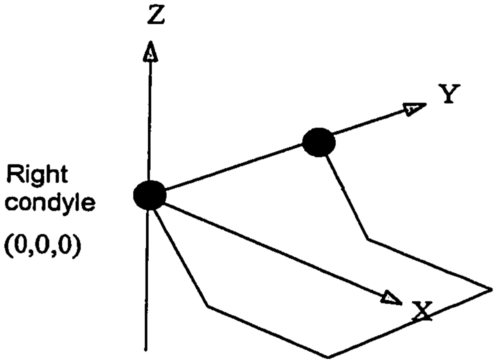


Figure C-1 Diagram of a mandible and its three reference axes.

Table C-1 Origins and insertions of some jaw muscles (modified from Osborn, 1995)

		S. Mass*	D. Mass*	M. Pter*	A. Temp*
Initial length (cm)		6.2	5.2	4.5	3.8
Origin	x	3.8	1.6	3.0	3.2
	y	0.3	0.0	2.8	0.1
	z	0.2	0.0	-2.2	2.2
Insertion	x	1.6	1.3	0.8	3.2
	y	1.6	1.3	1.1	0.4
	z	-5.4	-5.0	-5.8	-1.6

*S. Mass = superficial masseter, D. Mass = deep masseter, M. Pter = medial pterygoid, A. Temp = anterior temporalis. Coordinates are measured in cm.

2. Calculating changes in muscle length during a symmetrical jaw opening

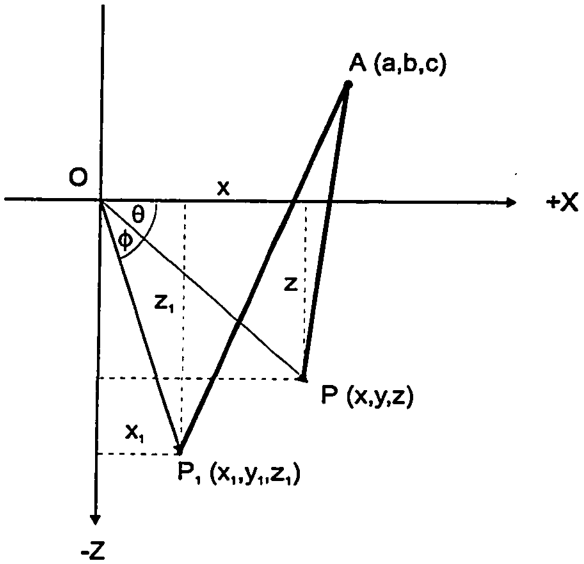


Figure C-2 Diagram used for calculating changes in muscle length during jaw opening.

Fig. C-2 shows the coordinates of the right condyle (O) and a jaw muscle in the sagittal plane. Let O (0, 0, 0) be the position of the right condyle, A (a, b, c) be the origin of the jaw muscle and P (x, y, z) be its insertion. θ is an angle measured from the x-axis to the line OP (θ is positive when measured counterclockwise and negative when measured clockwise) and can be derived from the equation,

$$\theta = -\tan^{-1}\left|\frac{z}{x}\right| \qquad \text{(C-1).}$$

P_1 (x_1, y_1, z_1) is the new position of the muscle insertion after a ϕ degree of jaw rotation (ϕ is positive when the jaw is rotated counterclockwise and negative when the jaw is rotated clockwise). Since the jaw is rotated symmetrically $y = y_1$ and its values can be ignored during the calculation whereas x_1 and z_1 are calculated from the equation,

$$x_1 = R\cos(\theta+\phi) \qquad \text{(C-2)}$$

$$z_1 = R\sin(\theta+\phi) \qquad \text{(C-3)}$$

where R is the length of OP which equals $\sqrt{x^2+z^2}$.

The initial muscle length, AP, is

$$\sqrt{(x-a)^2+(y-b)^2+(z-c)^2} \qquad \text{(C-4)}$$

and the final length, AP_1, is

$$\sqrt{(x_1-a)^2+(y_1-b)^2+(z_1-c)^2} \qquad \text{(C-5).}$$

For example, the superficial masseter whose origin and insertion are (3.8, 0.3, 0.2) and (1.6, 1.6, -5.4) respectively has an initial length of $\sqrt{(1.6-3.8)^2+(1.6-0.3)^2+(-5.4-0.2)^2}$ = 6.2 cm, $\theta = -\tan^{-1}\left|\frac{-5.4}{1.6}\right|$ = -73.5° and R = $\sqrt{(1.6)^2+(-5.4)^2}=5.63$.

After the 10° jaw opening (ϕ = -10) the new coordinates for the insertion are (5.63 cos -83.5°, 1.6, 5.63 sin -83.5°) = (0.6, 1.6, -5.6). Thus, the new muscle length is $\sqrt{(0.6-3.8)^2+(1.6-0.3)^2+(-5.6-0.2)^2}=6.7$ cm.

We assume that the slope of the articular eminence is 20°. If the condyle translates 7 mm along the eminence, the horizontal and vertical displacements will be 6.6 mm and 2.4 mm respectively. Using the data of the masseter muscle above, the final position of the muscle insertion will be (0.6 + 0.66, 1.6, -5.6 - 0.24) = (1.26, 1.6, -5.84) after a a 10° jaw opening combined with a 7-mm condylar translation.

3. Calculating changes in sarcomere length during a symmetrical jaw opening

The model assumes that in a given jaw muscle all muscle fibres have the same pennation angle and the same resting sarcomere length. The percentage elongation of a sarcomere from its resting position will be the same as that of the whole muscle if the pennation angle is 0°. But in a pennated muscle, with a pennation angle α, the percentage elongation of a muscle fibre, hence the sarcomere, is approximately cos α times the elongation of the whole muscle.

From the example above, if the resting sarcomere length of the masseter muscle at the closed-jaw position is 2.7 μ and the pennation angle of the muscle fibres is 20°, the sarcomere length after a 10° jaw opening will be increased by $\frac{(6.7-6.2)}{6.2} \times 100 \times \cos\, 20^{o} \approx 8\%$.

The sarcomere length of masseter and anterior temporalis muscles at two different jaw openings using pig's data is shown in Table C-2.

Table C-2 Sarcomere lengths at two different jaw openings. Both two- and three-dimensional models are used for pig's data (Herring *et al.*, 1984).

	2-D model		3-D model	
Jaw position	Masseter	Anterior Temporalis	Masseter	Anterior Temporalis
Pig's data				
closed	2.70 μ	2.10 μ	2.70 μ	2.10 μ
14-16 mm incisal separation (10° rotation)	2.95 μ	2.35 μ	2.90 μ	2.41 μ
26-28 mm incisal separation (17° rotation + 7 mm translation)	3.08 μ	2.66 μ	3.03 μ	2.67 μ

References

Herring S. W., Grimm A. F. and Grimm B. R. (1984) Regulation of sarcomere number in skeletal muscle : A comparison of hypotheses. *Muscle and Nerve* **7,** 161-173.

Osborn J. W. (1995) Biomechanical implications of lateral pterygoid contribution to biting and jaw opening in humans. *Archs oral Biol* **40,** 1099-1108.

APPENDIX D* - *RELATIONSHIP BETWEEN THE DIRECTION OF INCISAL MAXIMUM BITE FORCE AND THE LINE JOINING UPPER AND LOWER INCISAL EDGES

1. Materials and methods

Eight of the ten subjects who participated in the study of "Effect of Jaw Opening on the Direction and Magnitude of Human Incisal Bite Forces" were seated upright on a dental chair. A sticky pad, with a 1-cm line marked on it, was pasted on the cheek of the subject and the edge of the pad coincided with the upper occlusal plane (Fig. D-1A). The mandibular condyle at the closed-jaw position was palpated and the skin over the condyle was marked with a wash-off marker. Each subject was then asked to open their lower jaw symmetrically from small to large openings (usually five arbitary jaw positions were obtained). A side-view photograph was taken from each of the jaw openings, including the closed-jaw position, by a camera mounted on a tripod. The distance between the subject and the camera was kept constant.

In a given subject, the positions of the condyle, the upper and lower incisal edges, the orientation of the occlusal plane and the 1-cm line on the sticky pad from all openings were traced and superimposed onto a single transparency sheet. The tracing was scanned into an IBM-compatible personal computer and a drawing software (Corel Draw 3.0, Corel Corp, Ottawa, ONT, Canada) was used to enlarge the size of each tracing uniformly until the 1-cm line on the tracing was sized to the actual length. This ensured that the dimension of the mandible had been correctly duplicated.

A "jaw opening path" was drawn by connecting the position of lower incisal edges. The incisal separations corresponding to those measured in that subject (Paper #1) were marked along the opening path. Lines were connected between each of these marks and the upper incisal edge (Fig. D-1B). The inclination of the line joining the incisal edges in the sagittal plane (F+/B-) was measured with respect to the upper occlusal plane.

We assumed that the upper occlusal plane coincides with the lower occlusal plane at 0° jaw opening. The degree of jaw rotation can therefore be measured from the angle between the line joining the condyle and the upper incisal edge and the line joining the condyle and lower incisal edges (*e.g.* θ in Fig D-1B). Thus, the inclination of the line joining the incisal edges with respect to the lower occlusal plane at a given jaw separation can be estimated by subtracting the degree of jaw opening from the inclination with respect to the upper occlusal plane.

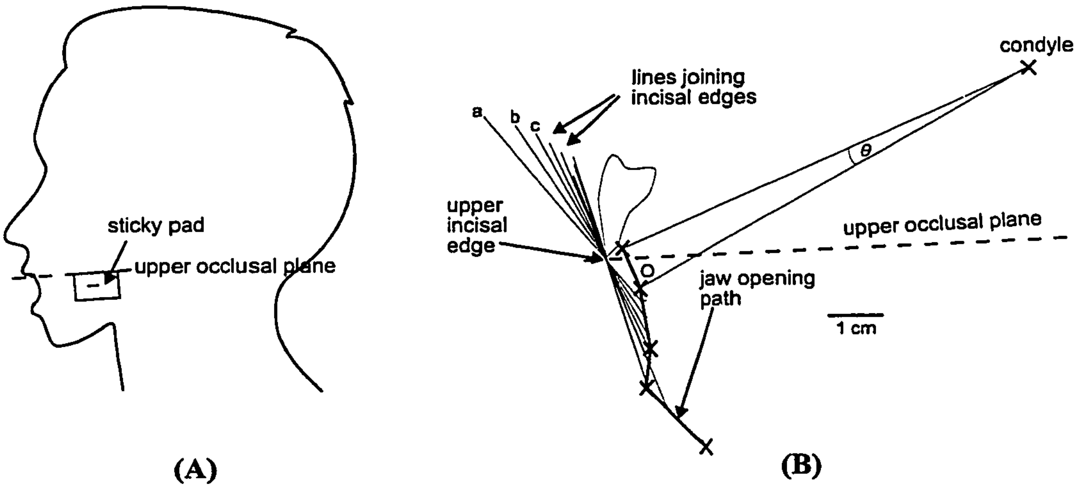


Figure D-1 Method used to trace the position of the lower and upper incisal edges. (A) A side-view photograph is taken at a particular symmetrical jaw opening. A sticky pad with a 1-cm scale is pasted on the cheek as a reference for dimension. (B) An example of the tracing. A "jaw opening path" is drawn by joining the photographed lower incisal edges (labelled as x). Lines are connected between the upper incisal edge and the positions of lower incisal edge along the "opening path". The inclination of these lines (*e.g.* a, b and c) are measured. θ is the degree of jaw opening at point O.

2. Results

Table D-1 and Table D-2 show the inclination of the lines joining the incisal edges from eight subjects at different jaw openings. The inclination of the line changes similarly to the bite direction as the jaw is opened (Fig. D-2).

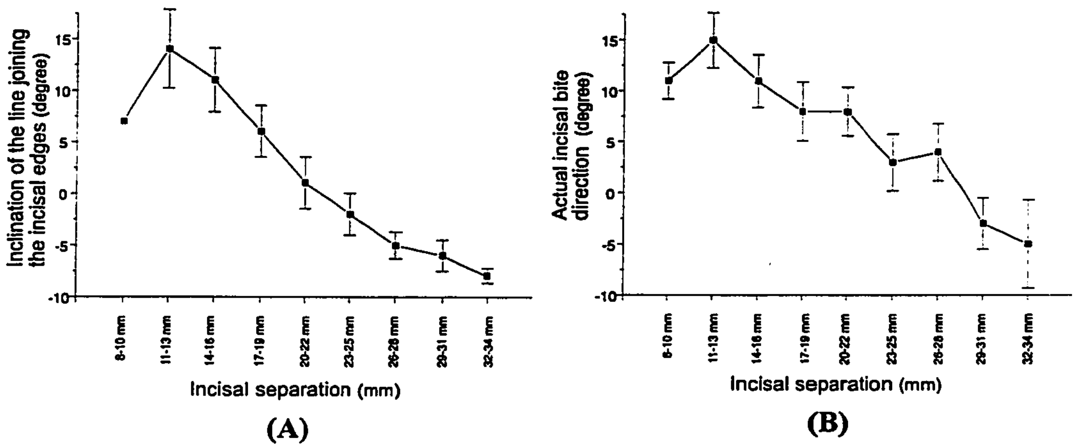


Figure D-2 Graphs showing (A) the inclination of the line joining the incisal edges and (B) the actual incisal bite direction (in the sagittal plane) as the jaw is opened. The directions are measured with respect to the lower occlusal plane. Front =+, Back = –.

Table D-1 The inclination of the lines joining the upper and lower incisal edges at different intervals of incisal separation.

	Inclinations at different intervals of incisal separation (F+/B-)								
	8-10 mm	11-13 mm	14-16 mm	17-19 mm	20-22 mm	23-25 mm	26-28 mm	29--31 mm	32-34 mm
	7	3	0	-3	-7	-9	-12	-14	-6
		8	8	3	-2	-5	-5	-6	-7
		27	22	16	12	8	-4	0	-7
		32	21	13	7	-1	-3	-8	-7
		20	13	7	1	-1	-6	-6	-11
		5	1	9	-7	-7	0	-5	
		18	14	-2	6	2	-6	-3	
		2							
mean	7	14	11	6	1	-2	-5	-6	-8
SE	0	4	3	3	3	2	1	2	1

Table D-2 The inclination of lines joining the upper and lower incisal edges at different jaw openings from eight subjects compared to the actual bite direction (with respect to the lower occlusal plane).

Incisal separation (mm)	% maximum jaw opening	Incisal bite direction (F+/B-)	Inclination of the line joining incisal edges (F+/B-)	Maximum jaw opening (mm)
subject D1				51
9	18	13	7	
12	24	9	3	
15	29	10	0	
18	35	4	-3	
21	41	4	-7	
24	47	-6	-9	
27	53	-9	-12	
30	59	-11	-14	
subject J27				61
11	18	27	8	
14	23	23	8	
17	28	11	3	
20	33	13	-2	
23	38	7	-5	
26	43	10	-5	
29	48	2	-6	
32	52	-14	-6	
subject K6				50
13	26	13	27	
16	32	16	22	
19	38	11	16	
22	44	15	12	
25	50	14	8	
28	56	13	3	
31	62	12	0	
subject R8				53
11	21	16	32	
14	26	11	21	
17	32	6	13	
20	38	9	7	
23	43	3	-1	
26	49	-1	-4	
29	55	-2	-8	
32	60	-2	-7	

Table D-2 (continued)

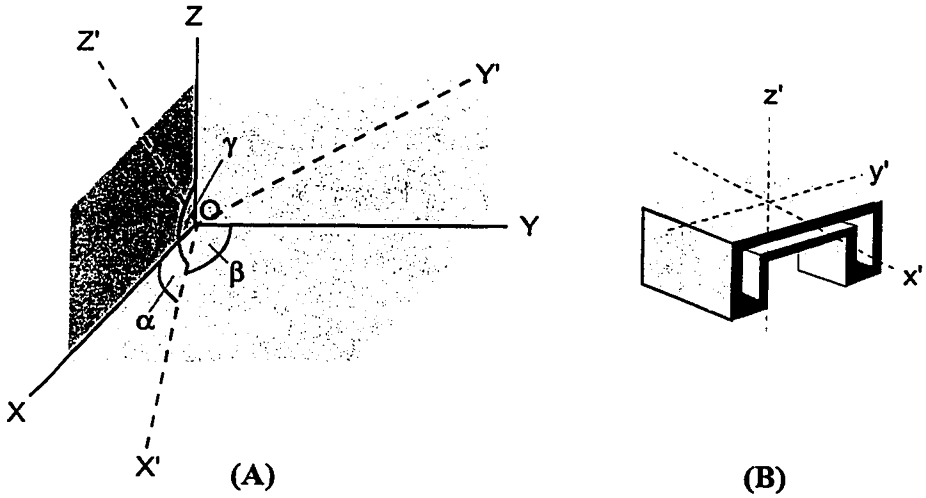
Incisal separation (mm)	% maximum jaw opening	Incisal bite direction (F+/B-)	Inclination of the line joining incisal edges (F+/B-)	Maximum jaw opening (mm)
subject S8				52
14	27	-4	13	
17	33	-3	7	
20	38	-6	1	
23	44	-11	-1	
26	50	-7	-3	
29	56	-13	-6	
32	62	-10	-7	
subject Y1				53
13	25	28	5	
16	30	25	1	
19	36	19	-3	
22	42	18	-7	
25	47	9	-7	
28	53	9	-8	
31	58	-3	-5	
34	64	-12	-7	
subject Z1				62
11	18	5	18	
14	23	-2	14	
17	27	1	9	
20	32	0	6	
23	37	-2	2	
26	42	-3	0	
29	47	-5	-3	
subject T6				50
13	24	18	2	
18	33	15	-2	
26	47	9	-6	
33	60	12	-11	

APPENDIX E - TRANSFORMATION OF A BITE DIRECTION WITH RESPECT TO THE TRANSDUCER INTO THE ONE WITH RESPECT TO OCCLUSAL PLANE

It is not always possible to place the transducer parallel to the mandibular occlusal plane, particularly in the posterior teeth. It is therefore necessary to transform the directions measured by the transducer into the ones with respect to the occlusal plane so they can be compared between subjects and between studies.

Two methods can be used as follows.

1. Method using direction cosines *(Spiegel, 1968)*



(A) (B)

Figure E-1 (A) Pure rotation of the axes x′, y′, z′ about the origin O. (B) U-shaped transducer and its corresponding axes.

Let XYZ be a reference frame in space in which the X-Y plane lies in parallel with the lower occlusal plane and X′Y′Z′ be the frame of the transducer which have already rotated about point O. Any given bite direction that is measured with respect to the transducer (X′Y′Z′ frame) can be transformed to that with respect to the XYZ frame by the following formulas:

$$\begin{aligned} x &= l_1 x' + l_2 y' + l_3 z' \\ y &= m_1 x' + m_2 y' + m_3 z' \\ z &= n_1 x' + n_2 y' + n_3 z' \qquad \text{(E-1)} \end{aligned}$$

where l_1, m_1, n_1 and l_2, m_2, n_2 and l_3, m_3, n_3 are the direction cosines of the x′and y′ and z′ axes relative to the x, y, z axes respectively. For example, $l_1 = \cos\alpha$, $m_1 = \cos\beta$ and $n_1 = \cos\gamma$ (see Fig. E-1A).

(x′, y′, z′) is the coordinate of a bite direction with respect to the transducer and (x, y, z) is the coordinate of the same vector with respect to the XYZ frame.

The formula looks simple but it is difficult to obtain the direction cosines since it is not practical to measure the necessary angles such as α, β, and γ.

2. Method using vectorial rotation *(Baragar and Osborn, 1984)*

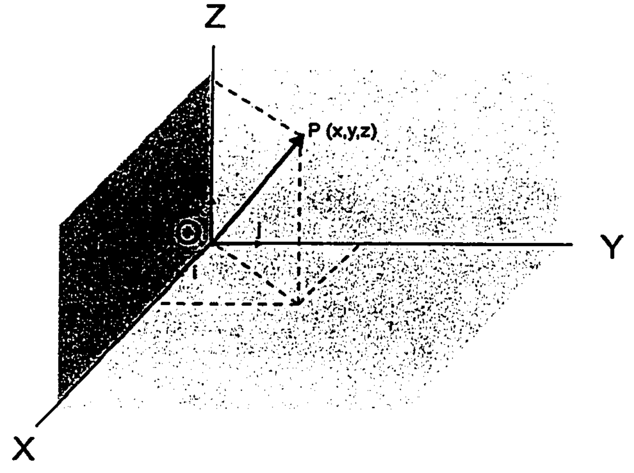


Figure E-2 An XYZ frame with its three mutually perpendicular unit vectors (i, j, k).

From Fig. E-1A, the transformation of the X′Y′Z′ frame to the XYZ frame can be achieved by rotating the X′Y′Z′ frame three times about its origin O. First, the X′Z′

plane is rotated while keeping the y′-axis fixed until the z′-axis meets the YZ plane. Second, the X′Y′ plane is rotated while keeping the z′-axis fixed until the x′-axis meets the XZ plane. Finally, the Y′Z′ plane is rotated while keeping the x′-axis fixed until the y′-axis meets the XY plane.

Let (**i**, **j**, **k**) be three, mutually perpendicular vectors, each of length one in the XYZ frame. P (x, y, z) is a point in space and the vector $\bar{OP}$ can be written as x**i** + y**j**+ z**k**. The same point P is seen as x′**i**′ + y′**j**′ + z′**k**′ in the rotated X′ Y′ Z′ frame in which **i**′, **j**′,**k**′ are its mutually perpendicular unit vectors. Therefore,

$$x\mathbf{i} + y\mathbf{j} + z\mathbf{k} = x'\mathbf{i}' + y'\mathbf{j}' + z'\mathbf{k}' \qquad \text{(E-2).}$$

By taking the dot product of each side with **i**, we obtain

$$x = x'\mathbf{i}'\cdot\mathbf{i} + y'\mathbf{j}'\cdot\mathbf{i} + z'\mathbf{k}'\cdot\mathbf{i}$$

since $\mathbf{i}\cdot\mathbf{i} = 1$, and $\mathbf{i}\cdot\mathbf{j} = \mathbf{i}\cdot\mathbf{k} = 0$.

Similar equations can be obtained for y (dot equation E-2 with **j**) and z (dot equation E-2 with **k**). The three equations derived can be written in matrix form

$$\begin{bmatrix} x \\ y \\ z \end{bmatrix} = \begin{bmatrix} \mathbf{i}\cdot\mathbf{i}' & \mathbf{i}\cdot\mathbf{j}' & \mathbf{i}\cdot\mathbf{k}' \\ \mathbf{j}\cdot\mathbf{i}' & \mathbf{j}\cdot\mathbf{j}' & \mathbf{j}\cdot\mathbf{k}' \\ \mathbf{k}\cdot\mathbf{i}' & \mathbf{k}\cdot\mathbf{j}' & \mathbf{k}\cdot\mathbf{k}' \end{bmatrix} \begin{bmatrix} x' \\ y' \\ z' \end{bmatrix} \qquad \text{(E-3).}$$

Equation E-3 yields the relationship between a point in the rotated X′Y′Z′ frame with respect to the original XYZ frame. If the original frame is rotated by an angle Ω_3 counterclockwise about its z-axis, then

$$\begin{bmatrix} x \\ y \\ z \end{bmatrix} = \begin{bmatrix} \mathbf{i}\cdot\mathbf{i}' & \mathbf{i}\cdot\mathbf{j}' & 0 \\ \mathbf{j}\cdot\mathbf{i}' & \mathbf{j}\cdot\mathbf{j}' & 0 \\ 0 & 0 & 1 \end{bmatrix} \begin{bmatrix} x' \\ y' \\ z' \end{bmatrix} = \begin{bmatrix} \cos\Omega_3 & \cos(\Omega_3 + \pi/2) & 0 \\ \cos(\pi/2 - \Omega_3) & \cos\Omega_3 & 0 \\ 0 & 0 & 1 \end{bmatrix} \begin{bmatrix} x' \\ y' \\ z' \end{bmatrix}$$

$$= \begin{bmatrix} \cos\Omega_3 & -\sin\Omega_3 & 0 \\ \sin\Omega_3 & \cos\Omega_3 & 0 \\ 0 & 0 & 1 \end{bmatrix} \begin{bmatrix} x' \\ y' \\ z' \end{bmatrix}$$

If $\Omega_3 << 1$, $\cos\Omega_3 \approx 1$ and $\sin\Omega_3 \approx \Omega_3$, thus

$$\begin{bmatrix} x \\ y \\ z \end{bmatrix} = \begin{bmatrix} 1 & -\Omega_3 & 0 \\ \Omega_3 & 1 & 0 \\ 0 & 0 & 1 \end{bmatrix} \begin{bmatrix} x' \\ y' \\ z' \end{bmatrix} \qquad \text{(E-4).}$$

Similarly, if the original frame is rotated by an angle Ω_1 counterclockwise about its x-axis,

$$\begin{bmatrix} x \\ y \\ z \end{bmatrix} = \begin{bmatrix} 1 & 0 & 0 \\ 0 & 1 & -\Omega_1 \\ 0 & \Omega_1 & 1 \end{bmatrix} \begin{bmatrix} x' \\ y' \\ z' \end{bmatrix} \qquad \text{(E-5)}$$

Similarly, if the original frame is rotated by an angle Ω_2 counterclockwise about its y-axis,

$$\begin{bmatrix} x \\ y \\ z \end{bmatrix} = \begin{bmatrix} 1 & 0 & \Omega_2 \\ 0 & 1 & 0 \\ -\Omega_2 & 0 & 1 \end{bmatrix} \begin{bmatrix} x' \\ y' \\ z' \end{bmatrix} \qquad \text{(E-6).}$$

If the rotation by Ω_3 (resulting in the $X_1'Y_1'Z_1'$ frame) is followed by the rotation by Ω_1 (resulting in the $X_2'Y_2'Z_2'$ frame) and followed by the rotation by Ω_2 (resulting in the $X_3'Y_3'Z_3'$ frame), *i.e.*

$$\begin{bmatrix} x \\ y \\ z \end{bmatrix} = \begin{bmatrix} 1 & -\Omega_3 & 0 \\ \Omega_3 & 1 & 0 \\ 0 & 0 & 1 \end{bmatrix} \begin{bmatrix} x_1' \\ y_1' \\ z_1' \end{bmatrix}$$

and

$$\begin{bmatrix} x_1' \\ y_1' \\ z_1' \end{bmatrix} = \begin{bmatrix} 1 & 0 & 0 \\ 0 & 1 & -\Omega_1 \\ 0 & \Omega_1 & 1 \end{bmatrix} \begin{bmatrix} x_2' \\ y_2' \\ z_2' \end{bmatrix}$$

and

$$\begin{bmatrix} x_2' \\ y_2' \\ z_2' \end{bmatrix} = \begin{bmatrix} 1 & 0 & \Omega_2 \\ 0 & 1 & 0 \\ -\Omega_2 & 0 & 1 \end{bmatrix} \begin{bmatrix} x_3' \\ y_3' \\ z_3' \end{bmatrix}$$

then the compound rotation can be rewritten as

$$\begin{bmatrix} x \\ y \\ z \end{bmatrix} = \begin{bmatrix} 1 & -\Omega_3 & 0 \\ \Omega_3 & 1 & 0 \\ 0 & 0 & 1 \end{bmatrix} \begin{bmatrix} 1 & 0 & 0 \\ 0 & 1 & -\Omega_1 \\ 0 & \Omega_1 & 1 \end{bmatrix} \begin{bmatrix} 1 & 0 & \Omega_2 \\ 0 & 1 & 0 \\ -\Omega_2 & 0 & 1 \end{bmatrix} \begin{bmatrix} x_3' \\ y_3' \\ z_3' \end{bmatrix}$$

$$= \begin{bmatrix} 1 & -\Omega_3 & \Omega_2 \\ \Omega_3 & 1 & -\Omega_1 \\ -\Omega_2 & \Omega_1 & 1 \end{bmatrix} \begin{bmatrix} x_3' \\ y_3' \\ z_3' \end{bmatrix} \qquad \text{(E-7)}$$

where $\Omega_1\Omega_3$, $\Omega_1\Omega_2$ and $\Omega_2\Omega_3$ are neglected because their values are small compared with Ω_1, Ω_2, Ω_3 and 1.

The result (equation E-7) is unchanged even if the rotations are performed in a different order as long as the terms $\Omega_i\Omega_j$ (where i, j = 1, 2, 3) are neglected.

This method simplifies the measurement of transducer orientation. The angular tilts ($\Omega_1, \Omega_2, \Omega_3$) in the formulas can be approximated by the "roll", "pitch" and "yaw" of the transducer[1]. The coordinate of a bite direction with respect to the transducer is represented by (x_3', y_3', z_3') and the coordinate of the same bite force with respect to the lower occlusal plane is (x, y, z).

Most incisal bite directions in the present study do not require the above angular transformation because the plane of the transducer can be manually adjusted to coincide with the lower occlusal plane. During the measurement the transducer was rotated 90° clockwise (yaw = $-\pi/2$). In this case, the direction is simply transformed by switching P and Q values, *i.e.* using P as the front/back (F+/B-) and using Q as the right/left (L+/R-) directions. Occasionally, it was not possible to place the transducer parallel to the lower occlusal plane, usually in the sagittal plane. In this case, the direction can be adjusted by

---

[1] "pitch" is the tilt in the sagittal plane, being positive when the transducer is rotated counterclockwise from the lower occlusal plane.

"roll" is the tilt in the frontal plane, being positive when the transducer is rotated counterclockwise from the lower occlusal plane.

"yaw" is the tilt in the horizontal plane, being positive when the transducer is rotated counterclockwise from the sagittal plane.

adding the sagittal tilt to or subtracting it from the front/back direction originally measured.

References

Baragar F. A. and Osborn J. W. (1984) A model relating patterns of human jaw movement to biomechanical constraints. *J Biomech* **17**, 757-767.

Spiegel M. R. (1968) *Mathematical Handbook of Formulas and Tables*. McGraw-Hill, New York.

APPENDIX F - STATISTICS FOR DIRECTIONAL DATA *(Mardia, 1972)*

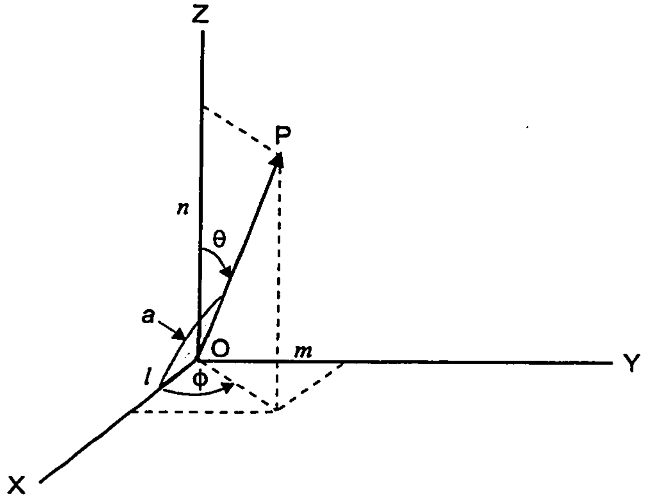


Figure F-1 Polar coordinates θ (co-latitude) and ϕ (latitude) of vector $O\bar{P}$.

Let $O\bar{P}$ be a vector in a three-dimensional space, one unit long. The orientation of $O\bar{P}$ is usually described by its co-latitude (θ) and latitude (ϕ). θ is the angle measured clockwise from the Z-axis to $O\bar{P}$ and ϕ is the angle measured counterclockwise from the x-axis to the projection of $O\bar{P}$ on the XY plane. The x-, y- and z-components of $O\bar{P}$; l, m, n are called the "direction cosines" in which

$$l = \cos a, \qquad m = \cos b, \qquad n = \cos c \qquad \text{(F-1)}$$

a, b and c are the angles $O\bar{P}$ makes with the x-, y- and z-axes respectively.

The direction cosines l, m, n can also be expressed in terms of θ and ϕ in which

$$l = \sin\theta\cos\phi, \qquad m = \sin\theta\sin\phi, \qquad n = \cos\theta \qquad \text{(F-2)}$$

where $0 < \theta < \pi$ and $0 < \phi < 2\pi$.

1. Mean direction and variance

Let (l_i, m_i, n_i), i =1, ..., n be n observed direction cosines. The mean direction (sometimes called the resultant) can be expressed in terms of its direction cosines $(\bar{l}, \bar{m}, \bar{n})$, whereas

$$\bar{l} = \sum_{i=1}^{n} \frac{l_i}{R}, \quad \bar{m} = \sum_{i=1}^{n} \frac{m_i}{R}, \quad \bar{n} = \sum_{i=1}^{n} \frac{n_i}{R} \qquad \text{(F-3)}$$

where R is the length of the resultant given by

$$R = \sqrt{\{(\sum l_i)^2 + (\sum m_i)^2 + (\sum n_i)^2\}} \qquad \text{(F-4).}$$

R will be as large as n if the observations are clustered about a direction (*i.e.* having a unimodal distribution) whereas if the observations are very dispersed as in the uniform distribution, R will be small. Hence R can be used as a measure of concentration about the mean direction. We may define the variance of directional data as

$$S^* = \frac{(n - R)}{n}, \qquad 0 \le R \le n, \qquad 0 \le S^* \le 1 \qquad \text{(F-5).}$$

2. A test for mean direction between two sample groups (Watson-Williams' test)

$$\boxed{\begin{array}{c}(l_{11}, m_{11}, n_{11})\\(l_{12}, m_{12}, n_{12})\\(l_{13}, m_{13}, n_{13})\\ \vdots \\(l_{1n_1}, m_{1n_1}, n_{1n_1})\end{array}}$$

size = n_1

$$\boxed{\begin{array}{c}(l_{21}, m_{21}, n_{21})\\(l_{22}, m_{22}, n_{22})\\(l_{23}, m_{23}, n_{23})\\ \vdots \\(l_{2n_1}, m_{2n_1}, n_{2n_1})\end{array}}$$

size = n_2

Suppose that (l_{ij}, m_{ij}, n_{ij}), $j = 1, ..., n_i$; $i = 1, 2$ are two independent random samples of sizes n_1 and n_2 respectively from a distribution, $F\{(\lambda_i, \mu_i, \nu_i), \kappa_1\}$; $i = 1, 2$ (κ = "concentration parameter", equivalent to standard deviation of the population). We assume that $\kappa_1 = \kappa_2 = \kappa$ where κ is unknown. We wish to test the null hypothesis,

$$H_o : (\lambda_1, \mu_1, \nu_1) = (\lambda_2, \mu_2, \nu_2)$$

against the alternative of unequal mean directions.

Let R_1 and R_2 be the lengths of the resultants of the first and second samples such that

$$R_i = \sqrt{R^2{}_{x,i} + R^2{}_{y,i} + R^2{}_{z,i}} \qquad \text{(F-6)}$$

where $R_{x,i} = \sum_{j=1}^{n_i} l_{ij}$, $\quad R_{y,i} = \sum_{j=1}^{n_i} m_{ij}$, $\quad R_{z,i} = \sum_{j=1}^{n_i} n_{ij}$, $\quad (i = 1, 2)$

and R is the length of the resultant of the combined sample such that

$$R = \sqrt{(R_x{}^2 + R_y{}^2 + R_z{}^2)} \qquad \text{(F-7)}$$

where $R_x = \sum R_{x,i}$, $\quad R_y = \sum R_{y,i}$, $\quad R_z = \sum R_{z,i}$.

When $\bar{R} = \dfrac{R}{n} > 0.70$ (which is true in all comparisons made in this thesis), the F-statistic given by

$$F_{2,2n-2} = (n-1)(R_1 + R_2 - R) / (n - R), \qquad n = n_1 + n_2 \qquad \text{(F-8)}$$

can be used to evaluate the null hypothesis. The null hypothesis will be rejected if the F-value obtained falls in the critical region.

However, if $\bar{R} < 0.70$, a test statistic $\bar{R}' = \frac{R_1 + R_2}{n}$ is instead calculated and compared to the critical values defined in special statistical tables (Mardia, 1972; Appendices 3.8a and 3.8b).

3. Transformation of P (right/left) and Q (front/back) angles into θ and ϕ

The direction measured from the U-shaped transducer is always expressed in the form of P and Q. It is therefore necessary to transform the direction into a standard form of θ and ϕ for statistical analyses.

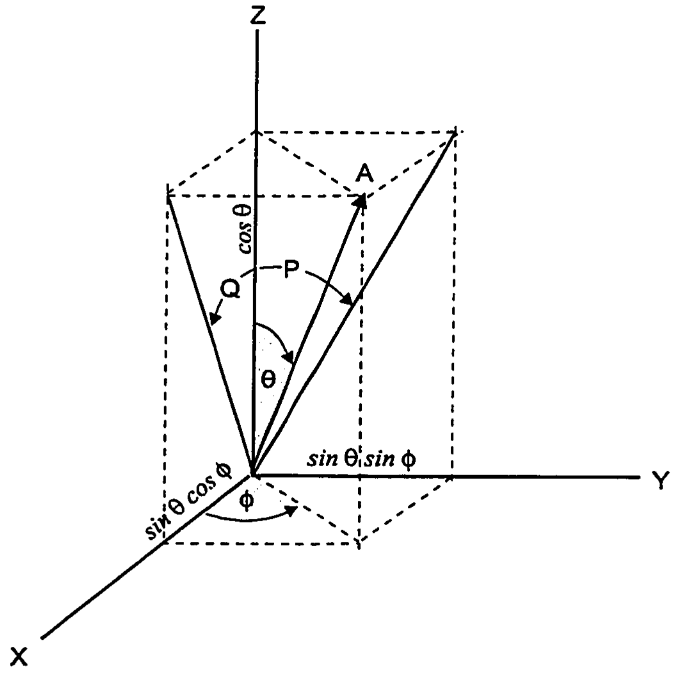


Figure F-2 Relationship between P, Q directions and θ, ϕ.

From Fig. F-2,
$$\tan \mathrm{P} = \frac{\sin\theta\sin\phi}{\cos\theta} = \tan\theta\sin\phi \qquad \text{(F-9)}$$

$$\tan \mathrm{Q} = \frac{\sin\theta\cos\phi}{\cos\theta} = \tan\theta\cos\phi \qquad \text{(F-10).}$$

By dividing equation (F-9) by equation (F-10) we have

$$\tan\phi = \frac{\tan \mathrm{P}}{\tan \mathrm{Q}} \qquad \text{(F-11)}$$

and subsequently

$$\cos\phi = \frac{\tan \mathrm{Q}}{\sqrt{\tan^2 \mathrm{P} + \tan^2 \mathrm{Q}}}.$$

By substituting cos ϕ in (F-10) we obtain

$$\tan\theta = \sqrt{\tan^2\mathrm{P} + \tan^2\mathrm{Q}} \qquad \text{(F-12)}$$

When the force direction is symmetrical in the frontal plane as observed with incisal bite forces (*i.e.* $\mathrm{P} \approx 0$ or $\phi = 0, \pi$), the mean direction can simply be calculated from $\frac{\sum \mathrm{Q}}{n}$.

References

Mardia K. V. (1972) *Statistics of Directional Data*. Academic Press, London, New York.

APPENDIX G - DISCRIMINATION OF HARDNESS USING FINGERPADS

1. Materials and methods

A piece of soft sponge, 2 × 2 × 1 cm (W × L × H), was glued to one side and a piece of flat Dura Lay, 2 × 2 × 0.5 cm (W × L × H), was glued to the other side of a wooden tongue depressor. The surfaces of the sponge and the acrylic were covered with a few layers of a Satin tape to ensure that the surface texture cannot be immediately noticed. In the first series of the experiment, ten subjects were asked to close their eyes and lightly hold the sponge-acrylic block with their thumb and forefinger (Fig. G-1A). Subjects who could not detect the difference immediately were told to slowly close their fingers until they could tell which side of the block touched the forefinger. Each subject was tested ten times. The position of sponge or acrylic was switched by turning the tongue depressor around in a pseudo-random order so that five tests had the sponge on the side of the forefinger. The accuracy of each test was determined by the number of correct matches a subject made. The minimum score of 75% was used to distinguish between a real detection and a guess. The experiment was repeated by placing a glass microscope slide between each fingerpad and the block (Fig. G-1B).

In the second series, the whole experiment (with and without glass slides) was tested between right and left forefingers. In the third series, subjects used unsharpened pencils to press the block instead of their fingers (Fig. G-1C).

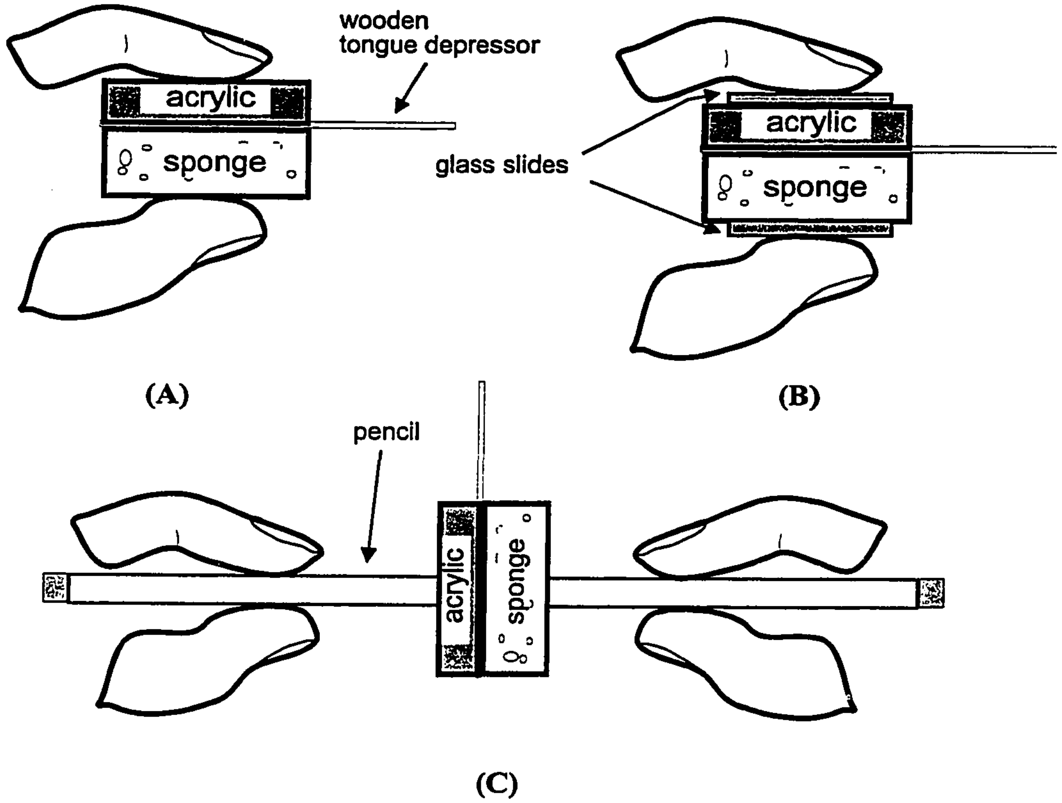


Figure G-1 Experimental set-up for the hardness discrimination tests using (A) bare fingers, (B) bare fingers covered with glass slides, and (C) pencils.

2. Results

Subjects could detect the material held between their bare fingers (either with one or both hands) with 100% accuracy. In contrast, when the fingerpads were covered with glass slides the correct detection was below 70%. The accuracy when pressing the block with pencils was not significantly different (p = 0.1; independent t-test) from when the fingers were covered with glass slides (Table G-1).

Table G-1 Detection accuracy (scored out of ten) during the hardness discrimination test with fingers.

Subject	With one hand		With both hands		With pencils
	bare finger	covered with glass slide	bare finger	covered with glass slide	
Ri	10	3	10	3	3
Ch	10	4	10	4	6
An	10	1	10	3	5
Al	10	6	10	6	4
Wn	10	3	10	2	3
T	10	4	10	0	3
To	10	0	10	0	7
Sa	10	4	10	1	6
Ni	10	1	10	4	3
Os	10	4	10	3	0

APPENDIX H - SOME TABULATED EXPERIMENTAL DATA

Table H-1 Detailed data from the study of "Effect of Jaw Opening on the Direction and Magnitude of Human Incisal Bite Forces" (Paper #1).

Incisal separation interval (mm)	Actual incisal separation (mm)	The direction of incisal MBF[*]		MBF (kg)	%MBF [§]	Muscle activity (% MVC) [†]				Average T[‡]	Average M[‡]	Average T/M[‡]
		F+/B-	L+/R-			RT	RM	LM	LT			
8-10	10	8	-1	30.3	87.8	18.1	38.9	72.4	52.4	35.3	55.6	0.6
	9	13	0	22.8	61.9	47.8	59.3	106.8	33.1	40.5	83.0	0.5
mean	**10**	**11**	**-1**	**26.5**	**74.9**	**33.0**	**49.1**	**89.6**	**42.8**	**37.9**	**69.3**	**0.6**
SE	**0.4**	**2**	**0**	**2.7**	**9.2**	**10.5**	**7.2**	**12.2**	**6.8**	**4.8**	**8.7**	**0.1**
11-13	12	9	-1	30.6	83.3	49.9	52.2	93.2	36.7	43.3	72.7	0.6
	11	27	-1	27.3	74.2	14.2	18.3	24.9	16.5	15.4	21.6	0.7
	13	13	-2	14.8	89.5	17.6	25.5	33.5	32.3	24.9	29.5	0.8
	13	18	-3	26.5	100.0	29.5	57.4	41.5	29.4	29.4	49.5	0.6
	11	16	2	20.1	76.6	14.9	34.0	37.8	38.3	26.6	35.9	0.7
	11	2	-3	21.7	86.0	17.5	37.8	29.3	20.4	19.0	33.6	0.6
	13	28	2	16.5	78.8	14.3	19.4	20.3	13.0	13.6	19.9	0.7
	11	5	-2	16.2	86.7	12.1	16.6	25.9	28.0	20.1	21.3	0.9
mean	**12**	**15**	**-1**	**21.7**	**84.4**	**21.3**	**32.7**	**38.3**	**26.8**	**24.0**	**35.5**	**0.7**
SE	**0.3**	**3**	**1**	**1.9**	**2.7**	**4.2**	**5.2**	**7.7**	**3.1**	**2.5**	**4.4**	**0.0**

[*] Direction is measured in degrees with respect to the lower occlusal plane. [§] %MBF is the normalized MBF to the maximum value in a given session.

[†] %MVC is the normalized EMG activity to the value during maximum intercuspal clenching (RT = right temporalis, RM = right masseter, LM = left masseter, LT = left temporalis). [‡] Average T = 1/2 (RT + LT), average M = 1/2 (RM + LM), average T/M = average T/average M. SE = standard error of the mean ($SD/\sqrt{n}$). The values in each line are from one individual.

Table H-1 (continued)

Incisal separation interval (mm)	Actual incisal separation (mm)	The direction of incisal MBF*		MBF (kg)	%MBF §	Muscle activity (% MVC) †				Average T‡	Average M‡	Average T/M‡
		F+/B-	L+/R-			RT	RM	LM	LT			
14-16	15	10	1	34.5	100.0	17.1	43.7	72.1	48.0	32.5	57.9	0.6
	15	10	0	33.0	89.7	64.2	77.6	133.2	58.1	61.2	105.4	0.6
	14	23	-0	32.2	87.4	17.0	18.6	31.7	21.9	19.5	25.2	0.8
	16	16	-1	16.3	98.7	21.7	20.7	28.9	31.5	26.6	24.8	1.1
	15	12	-3	21.8	100.0	16.7	37.2	52.3	34.8	25.7	44.7	0.6
	14	11	0	25.2	96.1	13.5	25.9	31.6	36.1	24.8	28.7	0.9
	14	-4	-4	22.4	88.8	29.9	48.6	43.3	40.4	35.1	46.0	0.8
	16	25	4	21.0	100.0	14.0	18.6	19.8	14.2	14.1	19.2	0.7
	14	-2	-3	16.6	88.8	11.2	14.0	25.1	34.8	23.0	19.5	1.2
mean	**15**	**11**	**-1**	**24.8**	**94.4**	**22.8**	**33.9**	**48.7**	**35.5**	**29.2**	**41.3**	**0.8**
SE	**0.3**	**3**	**1**	**2.2**	**1.8**	**5.2**	**6.4**	**11.2**	**4.1**	**3.3**	**6.0**	**0.1**
17-19	18	4	-2	34.0	92.6	71.4	71.0	143.2	65.4	68.4	107.1	0.6
	17	11	-0	35.9	97.5	23.7	39.0	48.2	24.7	24.2	43.6	0.6
	19	11	-2	14.0	84.8	20.1	22.0	30.7	33.0	26.5	26.3	1.0
	18	15	-5	25.8	97.1	36.0	60.7	41.7	31.3	33.7	51.2	0.7
	17	6	-0	21.4	81.7	14.8	30.9	43.1	40.4	27.6	37.0	0.7
	17	-3	-4	25.2	100.0	24.8	42.6	34.6	35.0	29.9	38.6	0.8
	19	19	3	20.3	97.0	13.0	19.2	19.8	13.7	13.3	19.5	0.7
	17	1	-3	18.2	97.3	11.7	16.2	27.0	42.0	26.9	21.6	1.2
mean	**18**	**8**	**-2**	**24.3**	**93.5**	**26.9**	**37.7**	**48.5**	**35.7**	**31.3**	**43.1**	**0.8**
SE	**0.3**	**3**	**1**	**2.7**	**2.4**	**6.9**	**7.0**	**13.9**	**5.3**	**3.8**	**6.6**	**0.1**

*, †, ‡, § see page 235.

Table H-1 (continued)

Incisal separation interval (mm)	Actual incisal separation (mm)	The direction of incisal MBF*		MBF (kg)	%MBF §	Muscle activity (% MVC) †				Average T‡	Average M‡	Average T/M‡
		F+/B-	L+/R-			RT	RM	LM	LT			
20-22	20	9	4	27.4	79.6	17.9	40.4	78.4	42.0	30.0	59.4	0.5
	21	4	-2	36.5	99.3	88.0	80.6	162.0	83.3	85.6	121.3	0.7
	20	13	-1	36.8	100.0	23.4	35.6	44.1	23.8	23.6	39.8	0.6
	22	15	-2	13.9	83.9	23.5	21.9	28.7	33.4	28.4	25.3	1.1
	20	10	-6	19.2	87.9	15.6	36.8	54.5	30.5	23.1	45.6	0.5
	20	9	1	22.5	86.0	18.9	29.8	39.2	52.4	35.6	34.5	1.0
	20	-6	-5	21.5	85.4	24.3	34.4	27.2	34.3	29.3	30.8	1.0
	22	18	2	20.5	98.1	12.1	18.4	20.4	11.5	11.8	19.4	0.6
	20	0	-4	17.3	92.4	9.7	14.9	16.3	25.7	17.7	15.6	1.1
mean	**21**	**8**	**-2**	**24.0**	**90.3**	**25.9**	**34.8**	**52.3**	**37.4**	**31.7**	**43.5**	**0.8**
SE	**0.3**	**2**	**1**	**2.5**	**2.3**	**7.5**	**6.1**	**14.3**	**6.5**	**4.6**	**7.3**	**0.1**
23-25	25	10	2	22.0	63.9	21.9	26.2	55.7	36.5	29.2	40.9	0.7
	24	-6	-2	35.3	96.1	60.8	48.9	88.5	66.5	63.7	68.7	0.9
	23	7	0	35.4	96.1	21.9	32.9	42.5	31.6	26.8	37.7	0.7
	25	14	-0	11.7	70.6	23.7	21.0	25.8	28.3	26.0	23.4	1.1
	23	3	-1	26.2	100.0	18.8	31.7	44.6	40.3	29.6	38.2	0.8
	23	-11	-4	20.6	82.0	11.7	18.0	12.5	19.8	15.8	15.2	1.0
	25	9	1	17.7	84.6	12.8	15.3	16.0	14.4	13.6	15.7	0.9
	23	-2	-4	17.6	94.4	11.0	16.6	22.1	32.2	21.6	19.4	1.1
mean	**24**	**3**	**-1**	**23.3**	**86.0**	**22.8**	**26.3**	**38.5**	**33.7**	**28.3**	**32.4**	**0.9**
SE	**0.3**	**3**	**1**	**2.8**	**4.4**	**5.3**	**3.7**	**8.4**	**5.2**	**3.5**	**4.3**	**0.1**

*, †, ‡, § see page 235.

Table H-1 (continued)

Incisal separation interval (mm)	Actual incisal separation (mm)	The direction of incisal MBF*		MBF (kg)	%MBF §	Muscle activity (% MVC) †				Average T‡	Average M‡	Average T/M‡
		F+/B-	L+/R-			RT	RM	LM	LT			
26-28	27	-9	-2	36.8	100.0	71.1	39.3	65.1	71.9	71.5	52.2	1.4
	26	10	-1	34.5	93.8	23.8	31.6	39.6	33.6	28.7	35.6	0.8
	28	13	1	16.5	100.0	13.3	12.5	15.1	24.3	18.8	13.8	1.4
	28	14	-5	21.2	97.4	18.4	31.6	41.1	30.1	24.2	36.3	0.7
	26	-1	-3	24.6	93.8	16.8	26.5	44.2	39.9	28.3	35.4	0.8
	26	-7	-5	21.0	83.6	27.0	35.7	24.8	39.8	33.4	30.3	1.1
	28	9	1	20.7	98.6	12.4	15.5	16.8	14.3	13.4	16.1	0.8
	26	-3	-4	18.7	100.0	11.7	14.6	24.5	32.0	21.9	19.5	1.1
	26	9	-5	25.3	95.4	32.3	47.9	34.5	20.4	26.4	41.2	0.6
mean	**27**	**4**	**-3**	**24.4**	**95.8**	**25.2**	**28.4**	**34.0**	**34.0**	**29.6**	**31.2**	**1.0**
SE	**0.3**	**3**	**1**	**2.3**	**1.8**	**6.2**	**4.1**	**5.2**	**5.5**	**4.3**	**3.4**	**0.1**
29-31	30	-11	-3	32.0	87.0	84.0	44.4	80.0	81.8	82.9	62.2	1.3
	29	2	-2	31.1	84.5	24.8	27.0	28.6	35.2	30.0	27.8	1.1
	31	12	-0	14.4	87.0	21.2	17.2	20.8	28.6	24.9	19.0	1.3
	29	-2	-3	21.0	80.2	15.1	16.3	38.7	40.2	27.7	27.5	1.0
	29	-13	-4	16.8	66.8	20.1	21.2	11.3	22.3	21.2	16.3	1.3
	31	-3	1	17.7	84.6	11.8	15.5	16.5	16.8	14.3	16.0	0.9
	29	-5	-4	16.4	87.7	10.9	14.1	23.2	33.5	22.2	18.7	1.2
mean	**30**	**-3**	**-2**	**21.3**	**82.5**	**26.9**	**22.3**	**31.3**	**36.9**	**31.9**	**26.8**	**1.2**
SE	**0.3**	**3**	**1**	**2.5**	**2.6**	**9.0**	**3.7**	**8.1**	**7.5**	**7.9**	**6.1**	**0.1**

*, †, ‡, § see page 235.

Table H-1 (continued)

Incisal separation interval (mm)	Actual incisal separation (mm)	The direction of incisal MBF*		MBF (kg)	%MBF §	Muscle activity (% MVC) †				Average T‡	Average M‡	Average T/M‡
		F+/B-	L+/R-			RT	RM	LM	LT			
32-34	32	-14	-0	34.0	92.5	28.2	24.7	28.8	34.0	31.1	26.7	1.2
	32	-2	-5	15.1	57.6	12.3	7.8	29.3	41.1	26.7	18.5	1.4
	32	-10	-2	12.6	50.1	13.8	11.8	9.2	17.6	15.7	10.5	1.5
	34	-12	1	15.0	71.4	10.7	13.7	13.2	17.2	13.9	13.5	1.0
	33	12	-2	25.9	97.6	32.3	41.5	35.9	21.9	27.1	38.7	0.7
mean	**33**	**-5**	**-2**	**20.5**	**73.8**	**19.4**	**19.9**	**23.3**	**26.4**	**22.9**	**21.6**	**1.2**
SE	**0.4**	**4**	**1**	**3.7**	**8.4**	**4.0**	**5.4**	**4.6**	**4.3**	**3.5**	**4.0**	**0.1**

*, †, ‡, § see page 235.

Table H-2 Detailed data from the study of "Effect of Pressure on a Maximum Incisal Bite Force in Man" (Paper #2).

	Bare incisor					Capped incisor			
Subject (tooth[§])	Bite direction[†]		MBF (kg)	%MBF		Bite direction[†]		MBF (kg)	%MBF
	F+/B-	L+/R-				F+/B-	L+/R-		
J6									
(#11)									
	-9	1	18	81.8		-12	4	18	81.8
	-11	2	21	95.5		-9	4	22	100.0
	-11	4	22	100.0		-12	0	21	95.5
mean	**-10**	**2**	**20.3**	**92.4**	**mean**	**-11**	**3**	**20.3**	**92.4**
SD	**1**	**1**	**1.7**	**7.7**	**SD**	**2**	**2**	**1.7**	**7.7**
L3									
(#11)	*	*	*			*	*	*	
	-8	-3	12	100.0		-7	1	11	91.7
	-6	-2	10	83.3		-9	4	11	91.7
mean	**-7**	**-2**	**11.0**	**91.7**	**mean**	**-8**	**2**	**11.0**	**91.7**
SD	**1**	**1**	**1.0**	**8.3**	**SD**	**1**	**1**	**0.0**	**0.0**
R4									
(#11)									
	-0	0	9	75.0		1	2	10	83.3
	-6	5	10	83.3		3	8	12	100.0
	-12	4	9	75.0		-18	-0	10	83.3
mean	**-6**	**3**	**9.3**	**77.8**	**mean**	**-5**	**3**	**10.7**	**88.9**
SD	**5**	**2**	**0.5**	**3.9**	**SD**	**9**	**3**	**0.9**	**7.9**
A6									
(#21)									
	-4	-1	25	92.6		-1	3	26	96.3
	-2	-3	22	81.5		0	-1	26	96.3
	-2	0	22	81.5		-2	-1	27	100.0
mean	**-3**	**-1**	**23.0**	**85.2**	**mean**	**-1**	**0**	**26.3**	**97.5**
SD	**1**	**2**	**1.4**	**5.2**	**SD**	**1**	**2**	**0.5**	**1.7**

[§] #11 = right upper central incisor, #21 = left upper central incisor. [†] Bite direction in degrees measured with respect to the lower occlusal plane. * Only the largest two MBFs are averaged.

Table H-2 (continued)

Subject (tooth§)	Bare incisor					Capped incisor			
	Bite direction†		MBF (kg)	%MBF		Bite direction†		MBF (kg)	%MBF
	F+/B-	L+/R-				F+/B-	L+/R-		
B1 (#11)									
	-6	-5	11	100.0		-8	-0	11	100.0
	-5	-1	11	100.0		-5	-5	9	81.8
	-9	1	9	81.8		-6	-1	11	100.0
mean	**-7**	**-2**	**10.3**	**93.9**	**mean**	**-6**	**-2**	**10.3**	**93.9**
SD	**2**	**3**	**0.9**	**8.6**	**SD**	**1**	**2**	**0.9**	**8.6**
W1 (#11)									
	-15	-5	25	80.6		-25	-7	26	83.9
	-22	-5	25	80.6		-20	-7	31	100.0
	-18	-5	29	93.5		-22	-5	26	83.9
mean	**-18**	**-5**	**26.3**	**84.9**	**mean**	**-22**	**-6**	**27.7**	**89.2**
SD	**3**	**0**	**1.9**	**6.1**	**SD**	**2**	**1**	**2.4**	**7.6**
S9 (#11)									
	15	3	19	90.5		15	1	21	100.0
	12	2	17	81.0		22	3	21	100.0
	23	2	21	100.0		26	1	21	100.0
mean	**17**	**2**	**19.0**	**90.4**	**mean**	**21**	**2**	**21.0**	**100.0**
SD	**5**	**0**	**1.6**	**7.8**	**SD**	**5**	**1**	**0.0**	**0.0**
Y3 (#11)									
	-20	-1	25	100.0		-14	-2	23	92.0
	-4	-1	24	96.0		-9	0	22	88.0
	-7	-2	22	88.0		-14	2	24	96.0
mean	**-10**	**-1**	**23.7**	**94.7**	**mean**	**-12**	**0**	**23.0**	**92.0**
SD	**7**	**0**	**1.2**	**5.0**	**SD**	**2**	**2**	**0.8**	**3.3**

§,†,* see page 240.

Table H-2 (continued)

	Bare incisor					Capped incisor			
Subject (tooth[§])	Bite direction[†]		MBF (kg)	%MBF		Bite direction[†]		MBF (kg)	%MBF
	F+/B-	L+/R-				F+/B-	L+/R-		
C3 (#11)									
	8	7	17	77.3		7	6	21	95.5
	0	7	20	90.9		4	8	22	100.0
	0	9	21	95.5		1	8	22	100.0
mean	**3**	**8**	**19.3**	**87.9**	**mean**	**4**	**7**	**21.7**	**98.5**
SD	**4**	**1**	**1.7**	**7.7**	**SD**	**2**	**1**	**0.5**	**2.1**
F1 (#11)									
	16	-1	11	78.6		15	-4	14	100.0
	12	-2	14	100.0		19	-5	11	78.6
	8	0	11	78.6		17	-5	14	100.0
mean	**12**	**-1**	**12.0**	**85.7**	**mean**	**17**	**-5**	**13.0**	**92.9**
SD	**3**	**1**	**1.4**	**10.1**	**SD**	**2**	**0**	**1.4**	**10.1**
E4 (#21)									
	8	2	14	82.4		6	2	14	82.4
	5	1	16	94.1		0	-1	17	100.0
	3	2	14	82.4		3	2	16	94.1
mean	**5**	**2**	**14.7**	**86.3**	**mean**	**3**	**1**	**15.7**	**92.2**
SD	**2**	**0**	**0.9**	**5.5**	**SD**	**2**	**1**	**1.2**	**7.3**
N3 (#11)									
	10	-1	28	96.6		24	-2	28	96.6
	12	-1	28	96.6		10	-2	29	100.0
	20	-1	28	96.6		15	-2	29	100.0
mean	**14**	**-1**	**28.0**	**96.6**	**mean**	**16**	**-2**	**28.7**	**98.9**
SD	**4**	**0**	**0.0**	**0.0**	**SD**	**6**	**0**	**0.5**	**1.6**

[§,†,*] see page 240.

Table H-2 (continued)

Subject (tooth§)	Bare incisor					Capped incisor			
	Bite direction†		MBF (kg)	%MBF		Bite direction†		MBF (kg)	%MBF
	F+/B-	L+/R-				F+/B-	L+/R-		
D5 (#21)									
	9	0	35	94.6		8	-1	31	83.8
	8	-2	33	89.2		4	-3	37	100.0
	10	-1	30	81.1		7	-2	33	89.2
mean	**9**	**-1**	**32.7**	**88.3**	**mean**	**6**	**-2**	**33.7**	**91.0**
SD	**1**	**1**	**2.1**	**5.6**	**SD**	**2**	**1**	**2.5**	**6.7**
T10 (#11)									
	-3	0	25	92.6		-2	3	27	100.0
	-1	1	23	85.2		4	2	27	100.0
	4	2	23	85.2		9	3	25	92.6
mean	**0**	**1**	**23.7**	**87.7**	**mean**	**4**	**3**	**26.3**	**97.5**
SD	**3**	**1**	**0.9**	**3.5**	**SD**	**4**	**0**	**0.9**	**3.5**
K9 (#11)									
	-19	-1	12	92.3		-14	-3	13	100.0
	-12	-4	12	92.3		-11	-4	13	100.0
	*	*	*			*	*	*	
mean	**-16**	**-3**	**12.0**	**92.3**	**mean**	**-13**	**-4**	**13.0**	**100.0**
SD	**4**	**2**	**0.0**	**0.0**	**SD**	**2**	**1**	**0.0**	**0.0**
Yi2 (#11)									
	-3	0	20	95.2		-9	2	21	100.0
	-18	1	19	90.5		-7	1	21	100.0
	*	*	*			*	*	*	
mean	**-11**	**1**	**19.5**	**92.9**	**mean**	**-8**	**2**	**21.0**	**100.0**
SD	**8**	**1**	**0.5**	**2.4**	**SD**	**1**	**1**	**0.0**	**0.0**

§,†,* see page 240.

Table H-2 (continued)

Subject (tooth§)	Bare incisor					Capped incisor			
	Bite direction†		MBF (kg)	%MBF		Bite direction†		MBF (kg)	%MBF
	F+/B-	L+/R-				F+/B-	L+/R-		
Jm1 (#11)									
	-5	0	19	95.0		-6	-2	20	100.0
	-10	-3	19	95.0		1	-5	18	90.0
	*	*	*			*	*	*	
mean	**-8**	**-2**	**19.0**	**95.0**	**mean**	**-3**	**-4**	**19.0**	**95.0**
SD	**3**	**2**	**0.0**	**0.0**	**SD**	**4**	**2**	**1.0**	**5.0**
H1 (#11)									
	-12	3	20	100.0		-13	0	19	95.0
	-14	0	17	85.0		-13	-1	18	90.0
	-11	3	17	85.0		-9	-1	17	85.0
mean	**-12**	**2**	**18.0**	**90.0**	**mean**	**-12**	**-1**	**18.0**	**90.0**
SD	**1**	**1**	**1.4**	**7.1**	**SD**	**2**	**0**	**0.8**	**4.1**
Grand mean	**-3**	**0**	**19.0**			**-2**	**0**	**20.0**	
SD	**10**	**3**	**6.4**			**11**	**3**	**6.7**	

§,†,* see page 240.

Table H-3 Detailed data from the study "Effects on Human Maximum Bite Force of Biting on a Softer or Harder Object" (Paper #4).

	Bare incisors						Capped incisors					
	Biting on rubber			Biting on acrylic			Biting on rubber			Biting on acrylic		
Subject	Bite direction†		MBF (kg)	Bite direction†		MBF (kg)	Bite direction†		MBF (kg)	Bite direction†		MB F (kg)
	F/B (+/-)	L/R (+/-)		F/B (+/-)	L/R (+/-)		F/B (+/-)	L/R (+/-)		F/B (+/-)	L/R (+/-)	
J44												
	-5	6	36	0	5	34	4	6	37	2	0	36
	-2	6	40	-1	1	33	5	6	48	2	1	36
	-1	5	39	-2	4	35	2	3	48	4	0	35
mean	**-3**	**6**	**38.3**	**-1**	**3**	**34.0**	**4**	**5**	**44.3**	**3**	**0**	**35.7**
SD	**2**	**0**	**1.7**	**1**	**2**	**0.8**	**1**	**1**	**5.2**	**1**	**0**	**0.5**
M5												
	-7	12	49	-11	8	44	-10	9	47	-11	8	47
	-8	11	51	-6	9	45	-13	6	51	-8	11	48
	-8	9	51	-10	14	49	-12	7	49	-11	10	47
mean	**-8**	**11**	**50.3**	**-9**	**10**	**46.0**	**-12**	**7**	**49.0**	**-10**	**10**	**47.3**
SD	**0**	**1**	**0.9**	**2**	**3**	**2.2**	**1**	**1**	**1.6**	**1**	**1**	**0.5**

n/a = missing data. † Bite directions in degrees measured with respect to the lower occlusal plane.

Table H-3 (continued)

Subject	Bare incisors						Capped incisors					
	Biting on rubber			Biting on acrylic			Biting on rubber			Biting on acrylic		
	Bite direction†		MBF (kg)	Bite direction†		MBF (kg)	Bite direction†		MBF (kg)	Bite direction†		MBF (kg)
	F/B (+/-)	L/R (+/-)		F/B (+/-)	L/R (+/-)		F/B (+/-)	L/R (+/-)		F/B (+/-)	L/R (+/-)	
L8												
	n/a	n/a	n/a	-10	2	9	-8	-2	6	-10	0	7
	-13	2	9	-6	6	9	-11	-1	4	-14	0	8
	-3	4	7	-11	7	10	-11	1	8	-13	0	8
mean	**-8**	**3**	**8.0**	**-9**	**5**	**9.3**	**-10**	**-1**	**6.0**	**-12**	**0**	**7.7**
SD	**5**	**1**	**1.0**	**2**	**2**	**0.5**	**1**	**1**	**1.6**	**2**	**0**	**0.5**
W5												
	-7	-4	15	-11	-2	13	-13	-3	9	-8	-5	9
	-6	-1	11	-10	0	13	-9	-8	7	-10	-6	12
	-5	-2	9	-6	-5	8	-14	-5	9	-9	-2	8
mean	**-6**	**-2**	**11.7**	**-9**	**-2**	**11.3**	**-12**	**-5**	**8.3**	**-9**	**-4**	**9.7**
SD	**1**	**1**	**2.5**	**2**	**2**	**2.4**	**2**	**2**	**0.9**	**1**	**2**	**1.7**

n/a = missing data. † Bite directions in degrees measured with respect to the lower occlusal plane.

Table H-3 (continued)

	Bare incisors						Capped incisors					
	Biting on rubber			Biting on acrylic			Biting on rubber			Biting on acrylic		
Subject	Bite direction†		MBF (kg)	Bite direction†		MBF (kg)	Bite direction†		MBF (kg)	Bite direction†		MBF (kg)
	F/B (+/-)	L/R (+/-)		F/B (+/-)	L/R (+/-)		F/B (+/-)	L/R (+/-)		F/B (+/-)	L/R (+/-)	
Bd1												
	-8	1	19	-8	3	10	-18	-3	13	-11	-2	15
	-7	-1	20	-4	2	18	-12	-1	18	-13	-2	13
	-5	0	19	-13	0	13	-19	-4	14	-16	-2	19
mean	**-7**	**0**	**19.3**	**-8**	**2**	**13.7**	**-16**	**-3**	**15.0**	**-13**	**-2**	**15.7**
SD	**1**	**1**	**0.5**	**4**	**1**	**3.3**	**3**	**1**	**2.2**	**2**	**0**	**2.5**
D8												
	0	4	8	1	4	11	3	4	11	2	4	10
	-2	2	8	1	2	12	7	4	12	2	3	12
	0	1	7	2	3	7	2	4	13	3	4	14
mean	**-1**	**2**	**7.7**	**1**	**3**	**10.0**	**4**	**4**	**12.0**	**2**	**4**	**12.0**
SD	**1**	**1**	**0.5**	**0**	**1**	**2.2**	**2**	**0**	**0.8**	**0**	**0**	**1.6**

n/a = missing data. † Bite directions in degrees measured with respect to the lower occlusal plane.

Table H-3 (continued)

	Bare incisors						Capped incisors					
	Biting on rubber			Biting on acrylic			Biting on rubber			Biting on acrylic		
Subject	Bite direction†		MBF (kg)	Bite direction†		MBF (kg)	Bite direction†		MBF (kg)	Bite direction†		MBF (kg)
	F/B (+/-)	L/R (+/-)		F/B (+/-)	L/R (+/-)		F/B (+/-)	L/R (+/-)		F/B (+/-)	L/R (+/-)	
All												
	8	2	49	9	0	40	13	-3	38	11	-2	33
	10	0	54	7	1	50	7	-5	45	7	-1	46
	5	0	56	4	2	48	11	-2	45	6	-1	48
mean	**8**	**1**	**53.0**	**7**	**1**	**46.0**	**10**	**-3**	**42.7**	**8**	**-1**	**42.3**
SD	**2**	**1**	**2.9**	**2**	**1**	**4.3**	**2**	**1**	**3.3**	**2**	**0**	**6.6**
Anl												
	2	-15	10	4	-14	10	8	-8	11	16	-6	12
	0	-4	13	2	-12	11	11	-9	19	13	-9	17
	3	-16	10	1	-11	9	14	-11	16	13	-10	14
mean	**2**	**-12**	**11.0**	**2**	**-12**	**10.0**	**11**	**-9**	**15.3**	**14**	**-8**	**14.3**
SD	**1**	**5**	**1.4**	**1**	**1**	**0.8**	**2**	**1**	**3.3**	**1**	**2**	**2.1**

n/a = missing data. † Bite directions in degrees measured with respect to the lower occlusal plane.

Table H-3 (continued)

Subject	Bare incisors						Capped incisors					
	Biting on rubber			Biting on acrylic			Biting on rubber			Biting on acrylic		
	Bite direction†		MBF (kg)	Bite direction†		MBF (kg)	Bite direction†		MBF (kg)	Bite direction†		MBF (kg)
	F/B (+/-)	L/R (+/-)		F/B (+/-)	L/R (+/-)		F/B (+/-)	L/R (+/-)		F/B (+/-)	L/R (+/-)	
Gl2												
	8	-3	18	7	-1	14	4	-5	20	5	-4	18
	6	-2	19	4	-2	14	5	-4	20	3	-5	22
	8	-3	18	9	1	15	7	-3	16	7	-2	15
mean	**7**	**-3**	**18.3**	**7**	**-1**	**14.3**	**5**	**-4**	**18.7**	**5**	**-4**	**18.3**
SD	**1**	**0**	**0.5**	**2**	**1**	**0.5**	**1**	**1**	**1.9**	**2**	**1**	**2.9**
Li2												
	-1	6	15	-1	2	14	9	-2	12	-4	7	11
	-1	0	12	-1	6	12	-4	4	14	-1	4	13
	0	0	15	0	3	14	2	5	12	-1	10	12
mean	**-1**	**2**	**14.0**	**-1**	**4**	**13.3**	**-1**	**2**	**12.7**	**-2**	**7**	**12.0**
SD	**0**	**3**	**1.4**	**0**	**2**	**0.9**	**5**	**3**	**0.9**	**1**	**2**	**0.8**

n/a = missing data. † Bite directions in degrees measured with respect to the lower occlusal plane.

Table H-3 (continued)

Subject	Bare incisors						Capped incisors					
	Biting on rubber			Biting on acrylic			Biting on rubber			Biting on acrylic		
	Bite direction†		MBF (kg)	Bite direction†		MBF (kg)	Bite direction†		MBF (kg)	Bite direction†		MBF (kg)
	F/B (+/-)	L/R (+/-)		F/B (+/-)	L/R (+/-)		F/B (+/-)	L/R (+/-)		F/B (+/-)	L/R (+/-)	
Ral												
	6	-9	15	8	-4	13	6	-3	16	4	-7	16
	9	-5	15	6	-1	11	4	-9	16	2	-4	18
	8	-9	12	3	-3	12	5	-7	18	5	-5	14
mean	**8**	**-8**	**14.0**	**6**	**-3**	**12.0**	**5**	**-6**	**16.7**	**3**	**-6**	**16.0**
SD	**1**	**2**	**1.4**	**2**	**1**	**0.8**	**1**	**2**	**0.9**	**2**	**1**	**1.6**
Jil												
	6	1	12	2	-3	10	4	-2	13	2	-1	11
	9	-2	19	2	4	15	1	-3	13	6	-2	14
	6	5	14	6	0	15	3	5	14	2	-3	15
mean	**7**	**1**	**15.0**	**3**	**0**	**13.3**	**3**	**0**	**13.3**	**3**	**-2**	**13.3**
SD	**1**	**3**	**2.9**	**2**	**3**	**2.4**	**1**	**4**	**0.5**	**2**	**1**	**1.7**

n/a = missing data. † Bite directions in degrees measured with respect to the lower occlusal plane.

Table H-3 (continued)

Subject	Bare incisors						Capped incisors					
	Biting on rubber			Biting on acrylic			Biting on rubber			Biting on acrylic		
	Bite direction†		MBF (kg)	Bite direction†		MBF (kg)	Bite direction†		MBF (kg)	Bite direction†		MBF (kg)
	F/B (+/-)	L/R (+/-)		F/B (+/-)	L/R (+/-)		F/B (+/-)	L/R (+/-)		F/B (+/-)	L/R (+/-)	
E6							K11					
	5	3	16	6	1	15	5	-3	21	6	-4	20
	8	4	17	6	3	13	5	-3	24	6	-2	21
	9	3	15	5	1	16						
mean	**7**	**3**	**16.0**	**6**	**2**	**14.7**	**5**	**-3**	**22.5**	**6**	**-3**	**20.5**
SD	**2**	**0**	**0.8**	**0**	**1**	**1.2**	**0**	**0**	**1.5**	**0**	**1**	**0.5**
Cm1							Dv1					
	4	2	14	1	-1	16	8	3	14	3	1	19
	0	2	17	6	-2	13	6	3	16	1	5	15
	1	1	18	4	-2	17	3	1	19	2	3	19
mean	**2**	**2**	**16.3**	**4**	**-2**	**15.3**	**6**	**2**	**16.3**	**2**	**3**	**17.7**
SD	**2**	**0**	**1.7**	**2**	**0**	**1.7**	**2**	**1**	**2.1**	**1**	**2**	**1.9**

n/a = missing data. † Bite directions in degrees measured with respect to the lower occlusal plane.

Table H-3 (continued)

Subject	Bare incisors						Capped incisors					
	Biting on rubber			Biting on acrylic			Biting on rubber			Biting on acrylic		
	Bite direction[†]		MBF (kg)	Bite direction[†]		MBF (kg)	Bite direction[†]		MBF (kg)	Bite direction[†]		MBF (kg)
	F/B (+/-)	L/R (+/-)		F/B (+/-)	L/R (+/-)		F/B (+/-)	L/R (+/-)		F/B (+/-)	L/R (+/-)	
Jk2							Cs1					
	16	3	27	8	1	22	1	-2	15	7	2	20
	12	1	25	10	1	21	0	-3	15	-1	-2	10
	10	2	28	8	1	23						
mean	**13**	**2**	**26.7**	**9**	**1**	**22.0**	**1**	**-3**	**15.0**	**3**	**0**	**15.0**
SD	**2**	**1**	**1.2**	**1**	**0**	**0.8**	**1**	**1**	**0.0**	**4**	**2**	**5.0**
Grand mean	**1**	**1**	**21.3**	**0**	**1**	**19.0**	**0**	**-1**	**20.5**	**0**	**0**	**19.8**
SD	**6**	**5**	**14.0**	**6**	**5**	**12.1**	**8**	**4**	**13.0**	**8**	**5**	**11.6**

n/a = missing data. [†] Bite directions in degrees measured with respect to the lower occlusal plane.

Table H-4 A goodness-of-fit test for the normal distribution of % difference in MBF observed in the study of "Effect on Human Maximum Bite Force of Biting on a Softer or Harder Object" (Paper #4).

Interval of %MBF difference	Observed frequency (O_i)	Z-interval	Probability of Z-interval	Expected frequency (E_i)	$\frac{(\lvert O_i - E_i \rvert - 0.5)^2}{E_i}$	χ^2
(a) Data from the BARE TOOTH experiment						
20.5~30.5	2	0.94~1.67	0.1261	1.89		
10.5~20.5	5	0.22~0.94	0.2393	3.59	0.19	
0.5~10.5	6	-0.51~0.22	0.2821	4.23		
-9.5~0.5	0	-1.23~-0.51	0.1957	2.94		
-19.5~-9.5	1	-1.96~-1.23	0.0843	1.26	0.01	
-29.5~-19.5	0	-2.68~-1.96	0.0213	0.32		
-39.5~-29.5	1	-3.41~-2.68	0.0034	0.05		**0.20**
(b) Data from the CAPPED TOOTH experiment						
20.5~30.5	0	1.77~2.66	0.0345	0.52		
10.5~20.5	2	0.88~1.77	0.151	2.27	0.12	
0.5~10.5	7	-0.02~0.88	0.3186	4.78		
-9.5~0.5	4	-0.91~-0.02	0.3106	4.66		
-19.5~-9.5	1	-1.80~-0.91	0.1455	2.18		
-29.5~-19.5	1	-2.7~-1.80	0.0324	0.49	0.48	
-39.5~-29.5	0	-3.59~-2.7	0.0033	0.05		**0.59**

Critical χ^2 at significance level of 0.05, $\nu = 1$, is 3.84.

Table H-5 Comparison between fast (ramp time ~ 1 s) and slow (ramp time ~ 3-5 s) incisal MBFs. Subjects bit in the edge-to-edge position.

Subject	Fast MBF				Slow MBF		
	Bite direction		MBF (kg)		Bite direction		MBF (kg)
	F+/B-	L+/R-			F+/B-	L+/R-	
J6				J2			
	-9	1	18		-10	-3	21
	-11	2	21		-13	-1	19
	-11	4	22		-12	0	19
mean	**-10**	**2**	**20.3**	**mean**	**-12**	**-1**	**19.7**
SD	**1**	**1**	**1.7**	**SD**	**1**	**1**	**0.9**
P2				P3			
	0	-16	9		3	-7	14
	5	-18	9		-4	-6	14
	2	-10	9		1	-5	15
mean	**3**	**-15**	**9.0**	**mean**	**0**	**-6**	**14.3**
SD	**2**	**4**	**0.0**	**SD**	**3**	**1**	**0.5**
L3				L1			
	*	*	*		-6	-0	16
	-8	-3	12		*	*	*
	-6	-2	10		-8	-4	14
mean	**-7**	**-2**	**11.0**	**mean**	**-7**	**-2**	**15.0**
SD	**1**	**1**	**1.0**	**SD**	**1**	**2**	**1.0**
T4				T2			
	-17	11	18		-8	13	21
	-12	11	19		1	16	19
	-13	11	19		-8	12	21
mean	**-14**	**11**	**18.7**	**mean**	**-5**	**13**	**20.3**
SD	**2**	**0**	**0.5**	**SD**	**5**	**2**	**0.9**
E3				E2			
	-2	5	15		-4	1	17
	*	*	*		-8	1	14
	0	4	15		*	*	*
mean	**-2**	**3**	**14.7**	**mean**	**-6**	**1**	**15.5**
SD	**1**	**0.5**	**0.0**	**SD**	**2**	**0**	**1.5**

* only the largest two MBFs are averaged.

Table H-5 (continued)

	Fast MBF				Slow MBF		
Subject	Bite direction		MBF (kg)		Bite direction		MBF (kg)
	F+/B-	L+/R-			F+/B-	L+/R-	
R4				R1			
	-6	5	10		-9	-4	10
	-12	4	9		4	-0	9
	-0	0	9		-2	-4	11
mean	**-6**	**3**	**9.3**	**mean**	**-2**	**-3**	**10.0**
SD	**5**	**2**	**0.5**	**SD**	**5**	**2**	**0.8**
A6				A2			
	-4	-1	25		0	5	39
	-2	-3	22		1	4	31
	-2	0	22		3	1	31
mean	**-3**	**-1**	**23.0**	**mean**	**1**	**3**	**33.7**
SD	**1**	**2**	**1.4**	**SD**	**1**	**2**	**3.8**
S2				S1			
	2	-3	15		3	-2	9
	6	0	14		2	-1	10
	2	0	14		2	-1	14
mean	**3**	**-1**	**14.3**	**mean**	**3**	**-1**	**11.0**
SD	**2**	**1**	**0.5**	**SD**	**1**	**1**	**2.2**
B1				B3			
	-6	-5	11		-4	0	11
	-5	-1	11		-5	-4	11
	-9	1	9		-3	-1	12
mean	**-7**	**-2**	**10.3**	**mean**	**-4**	**-1**	**11.3**
SD	**2**	**3**	**0.9**	**SD**	**1**	**2**	**0.5**
K3				K1			
	-8	-8	13		9	11	10
	-5	-7	10		5	10	14
	-8	-8	11		-2	10	14
mean	**-7**	**-8**	**11.3**	**mean**	**4**	**10**	**12.7**
SD	**1**	**0**	**1.2**	**SD**	**5**	**0**	**1.9**
Grand mean	**-5**	**-1**	**14.2**		**-3**	**1**	**16.4**
SD	**5**	**7**	**4.7**		**5**	**6**	**6.6**