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THE UNIVERSITY OF ALBERTA

THE POSSIBLE ROLE OF PERIODONTAL RECEPTORS IN THE PRODUCTION OF MASSETERIC SILENT PERIODS IN DOG

BY

© DONALD KEVIN GIBSON

A THESIS

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FACULTY OF GRADUATE STUDIES AND RESEARCH

The undersigned certify that they have read, and recommend to the Faculty of Graduate Studies and Research, for acceptance, a thesis entitled:

The Possible Role of Periodontal Receptors in the Production of Masseteric Silent Periods in Dogs

submitted by Donald Kevin Gibson in partial fulfilment of the requirements for the degree of Master of Science in Oral Biology.

Supervisor

External Examiner

Date: March 4th 1976

To my wife, Marie, and our sons

Kevin, Timothy and Steven

ABSTRACT

The purpose of this study was to investigate the role of periodontal receptors in producing the silent period associated with tooth contact in the masseter muscles of the dog. Two mongrel dogs were used.

Occlusal interferences were placed on the right cuspid teeth thereby disoccluding the remaining teeth. The animal was induced to produce a chewing motion causing contact at the cuspid interference which completed a circuit to mark the time of occlusion. The electromyographic activity was recorded before and after infiltration of local anesthetic around the occluding cuspid teeth.

The preanesthetic silent periods had a mean latency of 26.0 ± 3.4 milliseconds and a mean duration of 23.5 ± 2.9 milliseconds. The local anesthetic infiltration abolished the silent periods.

This study suggests that silent periods seen upon tooth contact in the masseter muscle of dog may be attributed to periodontal receptors.

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TABLE OF CONTENTS

LIST OF FIGURES

CHAPTER ONE

INTRODUCTION

The physiologic process of mastication is a complex neuromuscular process, the control of which is not entirely understood. In addition to the active force generating mechanism of the masticatory system there also appears to be a self protection apparatus (Ahlgren, 1969). In an electromyogram of the mandibular elevating muscles (superior head of lateral pterygoid (Grant, 1973; McNamara, 1973), masseter, medial pterygoid and temporalis) there appears a silent period in the electrical activity of the muscles associated with initial tooth contact during the occlusal phase of the masticatory cycle (Griffin and Munro, 1969). It has been assumed (Hannam et al, 1970) that the silent period mechanism prevents excessive and destructive forces from being delivered to the teeth, their supporting structures and the temporomandibular joint. The silent periods in the mandibular elevator muscles in humans with good occlusion of the teeth are fewer in number, and of shorter duration than those in patients with temporomandibular joint dysfunction and occlusal dysharmonies (Bessette et al, 1971). Therefore, the clinical significance of an understanding of the origin of the silent period in the mandibular elevator muscles is apparent.

Initiation of the silent period in the mandibular elevator muscles has been attributed to the following:

(1) temporomandibular joint receptors (Kawamura,

(1967). Three functional types of mechanoreceptors have been demonstrated in the capsule and ligaments of the temporomandibular joint (Greenfield and Wyke, 1966).

(2) stimulation of muscle spindles either by mandibular rebound upon tooth contact (the myotatic reflex) or by physical vibration from tooth contact transmitted through bone (Hannam et al, 1969).

(3) Golgi tendon organ response (Matthews, 1964).

(4) mechanoreceptors in the periodontal ligaments of the teeth (Hannam et al, 1969)."

Studies into the possible role of the periodontal ligament receptors have produced conflicting results. The initiation of the silent period has been attributed to stimulation of periodontal ligament receptors (Hannam and Matthews, 1969; Griffin and Munro, 1969), while Hannam et al (1970) and Matthews and Yemm (1970) have indicated that the periodontal ligament receptors may not be associated.

It is the purpose of this study to investigate the role of the periodontal receptors in producing the silent period in the masseter muscles of the dog.

[redacted]

[redacted]

A. Neuromuscular Physiology

Skeletal muscle contraction normally occurs only in response to nerve impulses transmitted to the muscle from the central nervous system along motor nerve fibers. The basic unit of muscular contraction is the [redacted] motor neuron in the central nervous system, its axon and terminal branches and the muscle fibers supplied by these branches.

The motor axons synapse with the muscle fibers at the neuromuscular junction. This synaptic junction is called the end plate. Light and electron microscope investigations have shown that the axon ends as a spherical enlargement called the "synaptic knob" or presynaptic terminal. The presynaptic terminal is separated from the postsynaptic side by a narrow gap, the synaptic cleft.

The presynaptic terminal contains a large number of vesicles known as synaptic vesicles. These vesicles contain the transmitter substance, acetylcholine, which is released into the synaptic cleft upon excitation. This triggers depolarization of the muscle fiber. Depolarization of the muscle fiber occurs producing an action potential that spreads along the muscle fiber. As the action potential passes along the muscle fiber it mobilizes the calcium, in the form of calcium ions, which is bound to the reticulum extending along the sides of the muscle fiber.

The calcium ions initiate chemical interactions between the actin and myosin filaments in the myofibrils causing muscle contraction (Guyton, 1966). The duration of the action potential in skeletal muscle is 1 to 10 milliseconds with a latency of about 3 milliseconds, measured from the time the action potential has spread along the muscle fiber until the fiber begins to contract.

The activity of the motoneurons is largely influenced by stimulation from peripheral receptors such as Golgi tendon organs and muscle spindles. The Golgi organs are usually located at the musculotendonous junction of the origin and insertion of a muscle (Barker, 1967). They have been described (Matthews, 1933) as slow adapting tension receptors with a high threshold to stretch. Recent studies of the Golgi organ (Stuart et al., 1970; Alnaes, 1967) have revealed a low threshold to stretch indicating a regulatory role for the receptor. When a muscle is subjected to extreme stretch, the Golgi organ response produces inhibition of the motoneurons innervating the muscle. The sudden relaxation of the muscle causes a rapid increase in its length. This mechanism apparently protects the tendonous attachment of the muscle to the bone (Guyton, 1966).

The muscle spindle is a specialized receptor which contains nerve and muscle structures and therefore has afferent and efferent innervation. The afferent nerves carry proprioceptive information from the muscle to the central nervous system. The efferent innervation produces contrac-

tion of the intrafusal fibers. The muscle spindles maintain muscle tonus and initiate the reflex response to muscle stretch (Guyton, 1966; Bessette et al, 1971).

Considerable work has been done to determine the neural mechanism which controls contraction and coordination of the masticatory muscles. Sherrington (1917) described the jaw opening and the jaw closing reflexes in the decerebrate cat preparation. Electrical stimulation or blunt pressure applied to the gingiva or the anterior region of the hard palate as well as pressure applied to the crown of a tooth evoked the jaw opening reflex. Putting fluid in the mouth to elicit a reflex swallow or stroking the dorsum of the tongue near its tip evoked the jaw closing reflex. Sherrington considered these jaw reflex movements to be the basic mechanism of mastication.

A jaw jerk reflex evoked by striking the lower jaw has been described by Corbin and Harrison (1940) as a true stretch reflex initiated by proprioceptive stimuli. They traced proprioceptive impulses from stretched masseter muscles into the mesencephalic root of the trigeminal nerve. A discrete lesion in the caudal portion of the mesencephalic root of the trigeminal nerve abolished the jaw jerk reflex on the ipsilateral side. There was no affect on the other jaw reflexes such as jaw closing during reflex swallowing or jaw opening by noxious stimulation (Harrison and Corbin, 1942).

The spastic condition of the mandibular elevator

muscles in the decerebrate dog was attributed to muscle supersensitivity to stretch and not to active jaw clenching (Kawamura et al, 1958). Destruction of the mesencephalic trigeminal nucleus abolished the spastic state of the mandibular elevator muscles by interrupting the transmission of proprioceptive impulses from the jaw muscles to the motor nucleus of the trigeminal nerve. Jaw depression evoked a response of short latency and slow adaptation that was assumed to result from stimulation of the muscle spindle afferents in the jaw closing muscles. The control of tonus of the jaw closing muscles and the degree of mandibular depression upon opening the mouth was attributed to the mesencephalic nucleus of the trigeminal nerve.

Two innervation patterns for the mandibular cuspid teeth of the cat have been described by Kizior et al (1968). They include small free ending fibers and specialized ovoid encapsulated receptors that are innervated by large myelinated and nonmyelinated fibers. The free ending or alveolar fibers are associated with the pain response resulting from heavy forces, in excess of 1100 grams, applied to the teeth. They enter the periodontal ligament through foramen in the alveolar bone and are located throughout the ligament. The fibers innervating the specialized receptors are responsive to light forces, less than 500 grams, applied to the teeth. They enter the ligament from the apical region of the alveolus with the receptors confined to the apical third of the ligament.

Anderson et al (1970) suggested that periodontal receptors could initiate reflex jaw opening only when forces on the teeth reached potentially damaging levels.

Kawamura and Nishiyama (1966) determined the topographic organization of the locations in the trigeminal sensory nuclei that respond to pressure on each tooth in the cat. Most locations in the sensory nuclei of the trigeminal nerve responded to pressure on the teeth applied from any direction but some locations only responded to pressure in a specific direction. Some spots in the sensory nuclei received afferents from several teeth with the cuspid tooth having the most densely distributed neurons. This suggests that the periodontal ligament of the canine tooth in the cat has a larger number of sensory receptors than the other teeth.

The trigeminal mesencephalic nucleus of the cat has three types of neurons (Jerge, 1963). One group innervates the muscle spindles of the masseter, temporalis and medial pterygoid muscles. Another group innervates the dental pressoreceptors of a single tooth. The last group innervates the dental pressoreceptors of two or more adjacent teeth and in some cases the contiguous gingival areas. Activation of the cells described was only from homolateral fields.

The pressoreceptors responsive to a single tooth were termed type I and were mainly slow adapting receptors, requiring 0.65 to 3.0 seconds for the spike frequency

to decline to zero. The threshold of these receptors was dependent upon the direction of the stimulus. Pressoreceptors innervating two or more teeth were termed type II and had a more complex peripheral field, probably requiring branching axons. From two to six teeth were innervated by this type of pressoreceptor with each tooth displaying a preferred direction of stimulus application. The thresholds were higher for this type of pressoreceptor compared with type I and they were classified as rapid adapting receptors but the adaptation time was not presented. Both receptor types were associated with the same tooth types, in contrast to findings by Thomas (1970). He found that fast pressoreceptor afferents were associated with the incisor periodontium and the monosynaptic reflex involved in positioning the incisor teeth for incision. Slow pressoreceptor afferents were associated with the molar periodontium and a polysynaptic reflex involving the mandibular depressor musculature which probably effect mastication. According to Thomas (1970), "two functionally distinct pressoreceptors project to the mesencephalic nucleus V via fast and slow fibers respectively", providing flexibility to the mastication process. He also stated that "the muscle spindle afferent neurons located in the mesencephalic nucleus V exert an inhibitory influence over oral receptor afferent neurons in the same nucleus". This would facilitate alternation from the opening to the closing reflex.

The reciprocal relationship in the brain stem

among afferent impulses from each jaw muscle in the cat has been reported (Kawamura et al, 1960). The increase in the electrical activity in the trigeminal motor nucleus evoked by stretching a jaw muscle was reciprocally inhibited by stretching the antagonistic muscle. However, the reciprocal inhibitory responses leading to the trigeminal motoneuron interactions were not observed in the mesencephalic trigeminal nucleus. The inhibitory effects were assumed to be mediated by afferent nerve fibers from the muscle spindles. However, the proprioceptive mechanism of the temporomandibular joint may also exert a strong influence on the control of the activities of the jaw muscles (Kawamura and Majima, 1964).

Matsunami and Kubota (1972) used monkeys to determine whether the muscle spindle afferent activity resulting from efferent fusimotor neuron activity in a voluntary movement, preceded skeletomotor neuron activity. They found that fusimotor activity occurred during both isometric and isotonic masseter muscle contraction. Their results demonstrated that efferent spindle innervation did not precede skeletomotor innervation of these muscles following stimulation of the motor cortex in primates.

Yemm (1971) has revealed that the activity of the masseter and temporal muscles increases during experimental stress. This was attributed to activity in the gamma motor system initiated by higher centers of the central nervous system. Alpha and gamma fibers are often activated simul-

taneously (Matthews, 1972). Taylor and Davey (1968) have demonstrated alpha-gamma co-activation in the jaw muscles of the cat. The discharge of spindle afferents increased during muscle contraction and decreased when the muscle relaxed. This indicated that alpha and gamma fibers were firing together since the spindles would otherwise have been silent during muscle contraction (Sears, 1964). The possible sympathetic innervation of muscle spindles was unlikely (Matthews, 1972). Boyd (1962) failed to demonstrate such an innervation although Barker (1948) reported that the spindles had a rich blood supply with capillaries adjacent to the intrafusal fibers.

Lund and Dellow (1971) studied the effect of interactive stimuli on rhythmical masticatory movements in rabbits. They found that rhythmical mandibular movements were induced when subthreshold stimulation at central neural sites known to establish the jaw rhythm, were coupled with facilatory concurrent peripheral stimulation such as an intraoral input simulating a food bolus. Inhibition of jaw rhythm was obtained when peripheral stimulation such as noxious paw-pinching, strong pressure to a maxillary incisor or rectal distension was coupled with the central neural stimulation. The described augmentatory and inhibitory effects were unaffected by decerebration and removal of the cerebellum, which demonstrated that the interactions were not dependent on ascending-descending loops involving higher centers. The rhythmical chewing resulting from central

stimulation of the back of the mouth are assumed to be through activation of a brain stem pattern generator. Such a mechanism would be important in initiation and maintenance of rhythmical mastication and sucking.

Several workers (Goldberg, 1972; Schaerer et al, 1967) have demonstrated that the activity of the mandibular elevator muscles is influenced by the occlusal relationship of the teeth. Occlusal interferences increase the number (DeBoever, 1969) and duration (Bessette et al, 1971) of silent periods. Atkinson and Shepherd (1973) demonstrated the need for an occlusal stop which provides for a correct vertical dimension in order to obtain a normal masticatory cycle. The occlusal stop may be natural or artificial and the presence of a periodontal membrane did not appear essential for normal mastication. Temporomandibular joint dysfunction may be directly related to occlusal dysfunction (Bessette et al, 1971). Griffin and Munro (1971) have shown that the latency of the silent period in the masseter muscle is significantly shorter in patients with temporomandibular joint dysfunction indicating that a facilatory mechanism may be operating.

Ahlgren (1967) described the chewing cycle in three phases as follows:

(1) the opening phase consisting of mandibular depression;

(2) the closing phase consisting of mandibular elevation; and

(3) the occlusal phase during which time the teeth are in contact in habitual occlusion. Ahlgren and Owall (1970) studied the relationship between muscular activity and chewing force during mastication in human subjects and found that the maximal chewing force occurred during the occlusal phase. The mean time interval between maximum force and the start of the opening phase was 69 milliseconds. The chewing force outlasted the occlusal phase. Furthermore, the peak electromyographic activity preceded the maximal chewing force by 41 milliseconds, with the chewing force outlasting the electromyographic activity.

Jaw reflex pathways are illustrated in figure one. The jaw jerk reflex has been described as a monosynaptic reflex initiated by sudden stretch of the mandibular elevator muscles (Goldberg, 1971). Proprioceptive impulses from the muscle spindles are carried to unipolar neurons located in the mesencephalic trigeminal nucleus in the mid brain. Processes from these neurons synapse in the trigeminal motor nucleus producing excitation of the alpha motoneurons that innervate the mandibular elevator muscles. The resulting muscular response is a synchronized contraction of the muscle fibers (Goldberg, 1971).

Stimulation of periodontal receptors produces inhibition of the mandibular elevator muscles. Afferent impulses from peripheral periodontal receptors are carried to higher centers by the superior and inferior dental branches of the maxillary and mandibular divisions of the trigeminal

JAW REFLEX PATHWAYS

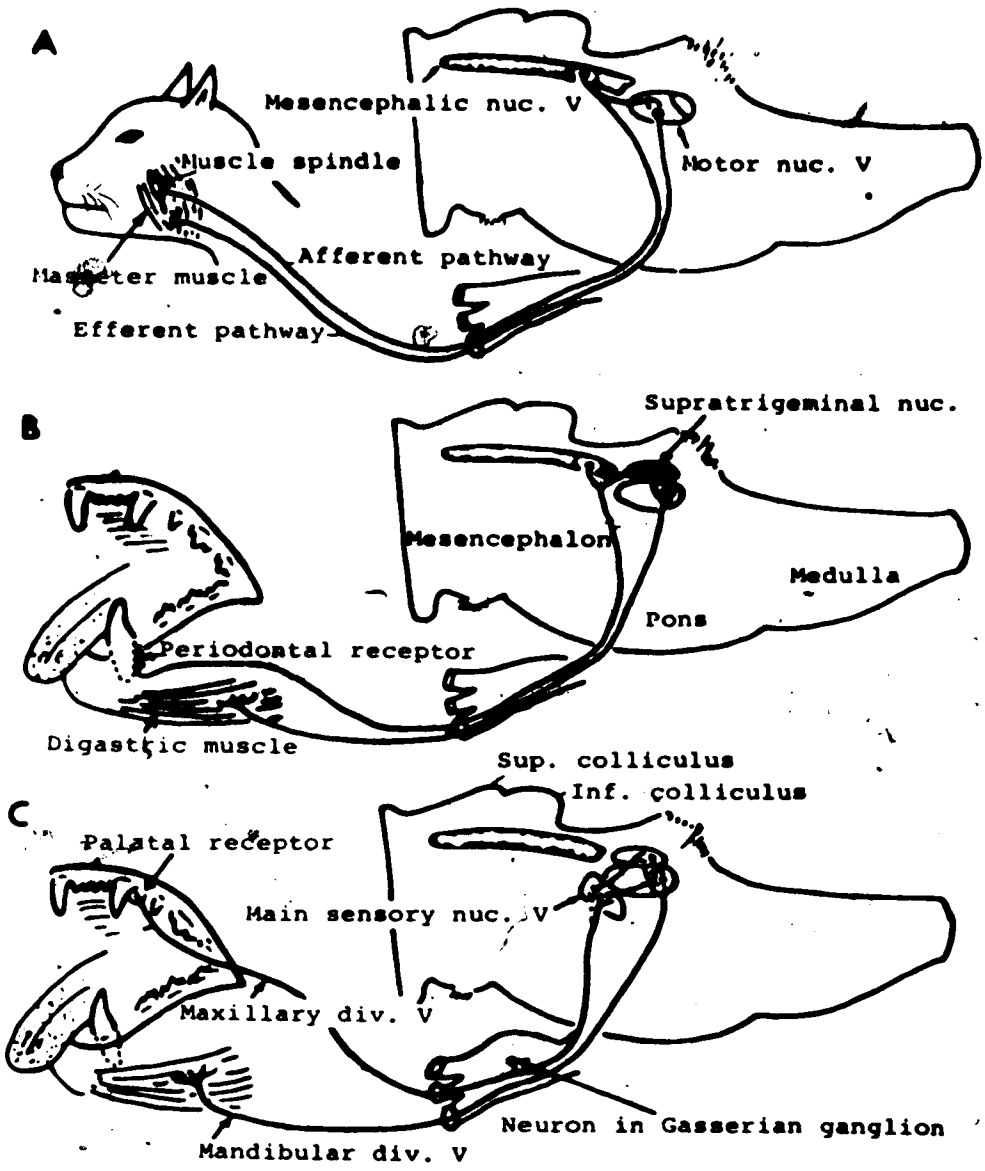


Figure 1: A. Muscle spindles initiate the myotatic reflex of the jaw elevator muscles. The reflex pathway is monosynaptic involving mesencephalic nucleus cells and motoneurons in the trigeminal motor nucleus. B and C illustrate possible pathways of the jaw opening reflex. B. The cell bodies of periodontal receptors are located in the mesencephalic nucleus. Interneurons in the supratrigeminal nucleus make the reflex polysynaptic. C. Receptors with cell bodies located in the Gasserian ganglion project to the main sensory nucleus V. The disynaptic pathway involves motoneurons in the trigeminal motor nucleus. A more complicated reflex pathway may involve interneurons in the supratrigeminal nucleus.

nerve (Griffin and Munro, 1969). At least two pathways may be involved in this process. In one pathway the periodontal receptors have their cell bodies in the mesencephalic nucleus V. Processes from these neurons synapse with neurons in the supratrigeminal nucleus which project to the trigeminal motor nucleus. Impulses from the periodontal receptors cause inhibition of the alpha motoneurons that innervate the mandibular elevator muscles.

In the other pathway, the cell bodies of the periodontal receptors are located in the Gasserian ganglion. Processes from these neurons synapse with neurons in the main sensory nucleus V which project to the trigeminal motor nucleus. A more complex pathway may involve interneurons in the supratrigeminal nucleus (Jerge, 1964).

B. Electromyography

The inherent electrical activity associated with muscle contraction provides the basis of electromyography. The asynchronous depolarization of many muscle fibers during muscle contraction produces many action potentials. The algebraic summation of these electrical potentials can be detected by sensitive electrodes in or near the muscle (Basmajian, 1967).

Fine wire or needle electrodes may be placed in the muscle but action potentials are only received from the immediate muscle fibers which may not accurately represent the muscle function (Carlsoo, 1952). Larger subcutaneous plate electrodes may be attached directly onto the muscle

to record action potentials from a larger number of muscle fibers. The wire or needle and plate electrodes have the disadvantage that they may be painful to the subject and that during function, the muscle under investigation is disturbed by the electrode which may alter muscle function. Surface electrodes attached to the skin over the muscle to be studied eliminate the disadvantages of the former electrodes. The surface electrodes receive electrical activity from a larger area of the muscle which more accurately describes the function of the entire muscle.

Since electromyographic techniques permit the study of muscle activity without disturbing the muscle, they were of value in determining the relationship between dental occlusion and the activity of the muscles of mastication (Ahlgren, 1966; Moller, 1966). Electromyography has been used to determine changes in jaw muscle activity associated with orthodontic correction of malocclusion (Moyer, 1949). Griffin and Munro (1971) used electromyography to study the action of the masseter and anterior temporalis muscles in patients with temporomandibular joint dysfunction.

Since occlusal dysfunction alters the activity of the masticatory muscles (Bessette et al, 1971), electromyography assumes an important role in diagnosis.

C. The Silent Period

The term, silent period, is used to describe the electrical inactivity seen in the electromyogram of skeletal muscle following adequate electrical or mechanical stimula-

tion of the muscle. The phenomenon was first described by Hoffman (1922) who considered it to be a refractory state of the motoneurons in the spinal cord, a theory supported by Moldaver (1936). Gasser (1939) attributed the silent period to the positive after potential following the discharge of the motoneurons.

Denny-Brown (1928) found that deafferenting the muscle greatly reduced the silent period. He concluded that the refractoriness of the motoneurons could only be implicated with a small portion of the silent period and that the greatest part of the silent period was due to afferent discharge caused by the tendon jerk. Matthews (1933) supported this theory when he described a pause, similar to the silent period, in the regular discharge of impulses from the muscle spindles during a muscle twitch. However, Hoff et al (1934) demonstrated that deafferenting a muscle did not alter the duration of the silent period.

More recently, Matthews (1964) stated other factors in addition to muscle spindles which may be responsible for the silent period. They include the activation of Golgi tendon organs either electrically or by tension during muscular contraction which produce inhibitory impulses that are carried via group 1B fibers to the motoneurons. In addition, the innervation of Renshaw cells by motor axons through recurrent collaterals may provide inhibitory impulses to the motoneurons. However, Renshaw loops have not been demonstrated in the mandibular elevator muscles.

Granit (1955) stated that tendon organs produce inhibition in synergist motoneurons and excitation of antagonist motoneurons. Angel, Eppler and Iannone (1965) using human subjects showed that the silent period could not be attributed to the discharge of Golgi tendon organs, inhibition through the Renshaw cells or the synchronised discharge of motoneurons. They also reported that reciprocal innervation was not responsible for initiation of the silent period since it often occurred before action potentials appeared in the antagonistic muscle. The silent period only occurred when the muscle was permitted to shorten, which presumably unloaded the muscle spindles.

In addition to the mechanisms mentioned, receptors located in the temporomandibular joint (Kawamura et al, 1964; Greenfield and Wyke, 1966) and in the periodontal structures of the teeth (Hannam and Matthews, 1969) may also elicit silent periods in the main elevator muscles of the mandible (Goldberg, 1971).

Ahlgren (1969) determined that the silent period in the masseter and temporalis muscles appeared at a time corresponding to initial tooth contact during chewing. He supported the view of Angel et al (1965) that the Golgi tendon organs were not responsible for the silent period. Granit (1955) found that the duration of the silent period following the myotatic reflex of leg muscle was from 50 to 100 milliseconds and Ahlgren (1969) reported a similar duration of 100 milliseconds for the jaw muscles. Ahlgren

(1969), considered the jaw opening reflex to be the mechanism underlying the silent period seen in the electromyogram during chewing and biting. However, Ahlgren (1969) found that the mean silent period associated with tooth contact was only 17.8 milliseconds during chewing and 15.3 milliseconds during biting. Furthermore, the silent period in the masseter and temporalis muscles usually appeared before the peak tension developed in these muscles while the discharge from Golgi tendon organs increased with increased tension. Renaudgeau et al (1969) stated that the anatomical homologue between the Golgi tendon organ and periodontal receptor and the inhibitory effect of the latter suggest the possibility that periodontal ligament receptors perform for the masticatory muscles some, if not all, the functions Golgi tendon organs do for other somatic muscles.

Griffin and Munro (1969) described the electromyographic activity of the jaw closing muscles in the open-close-clench cycle in man. In each cycle they found a silent period in each of the main mandibular elevator muscles of about 13 milliseconds with a latent period of the same duration measured from the time of initial tooth contact. Both the onset and the termination of the silent period were abrupt.

Hannam et al (1969) studied the changes in the activity of the masseter muscle following tooth contact in man and reported that the return of activity following the silent period (elicited by having the subjects tap their

teeth together in centric occlusion) was variable, being dependent upon the voluntary efforts of the subject.

Munro and Griffin (1970) supported Ahlgren's (1969) observations concerning the silent period in the masseter and anterior temporalis muscles in man. They believed that tooth contact produced reflex inhibition of the mandibular elevator muscles.

In a study of the electromyograms of the masseter muscles in edentulous subjects, Matthews and Yemm (1970) demonstrated a silent period following tooth contact in subjects wearing full dentures similar to that in subjects with natural teeth. They concluded that the silent period does not necessarily depend upon stimulation of the mechanoreceptors of the periodontal ligaments of the teeth. Further work by Hannam et al (1970) added support to that conclu-

They found that mechanical stimulation of a tooth produced a silent period in the masseter muscle while the muscle maintained steady activity with the subject biting on a hollow rubber bung. Local anesthetic applied around the tooth failed to abolish the silent period. A similar response in the masseter muscle was obtained by tapping the chin to evoke a jaw jerk reflex and by tapping a metal disc attached to the forehead of the subject. This result was explained as follows: "In those experiments in which a single upper tooth was tapped, the spindles appear to have been stimulated by vibration transmitted through bone since a similar response could be obtained by vibrating the skull

over the frontal bone. During chewing and tooth tapping the spindles may have been excited similarly by vibrations set up at the moment of tooth contact or alternatively by slight rebound of the mandible which could have stretched the elevator muscles."

Hannam and Matthews (1969) evoked a jaw opening reflex upon mechanical stimulation of a maxillary canine tooth in cat. The response proved independent of stimulus duration, and was not altered by removal of gingiva including palatal mucosa from around the tooth or by extirpation of the pulp tissue. However, the response to the mechanical stimulus was abolished following infiltration of local anesthetic over the root of the canine. They concluded that jaw muscle activity following tooth stimulation was influenced by mechanoreceptors in the periodontal ligament and not by receptors in the gingiva, palate or pulp.

Yemm (1972) reported a similarity between the silent period seen upon tooth contact and that seen following the jaw jerk response. The silent period response evoked in the masseter muscle by electrical stimulation of oral mucus membrane in man had a latency and duration similar to the silent period seen in the jaw jerk response. Yemm (1972) suggested that the silent period associated with tooth contact may have a dual origin with contributions from both the muscle spindles of the mandibular elevator muscles and the periodontal ligament receptors.

The unloading reflex in the masticatory muscles

has been examined (Hannam et al, 1968; Gill, 1970) as well as electrical and mechanical stimulation of various oral structures including the teeth, palate and gingiva (Thexton, 1973; Bessette et al, 1974, Sessle and Schmitt, 1972). However, the results of the studies to date do not clearly demonstrate the mechanism responsible for the silent periods observed in the electromyograms of the mandibular elevator muscles in man during mastication. The role of periodontal receptors in the production of silent periods in the elevator muscles of the mandible is not clearly understood.

CHAPTER THREE

METHODOLOGY

A. Subjects

The subjects for this study consisted of two, two year old mongrel dogs, one of each sex. The average weight of the male dog was [redacted] kilograms while that of the female dog was 15 kilograms. The animals were maintained by the Animal Services Department of the University of Alberta.

B. Appliances

The intraoral appliances employed in this study consisted of a cast gold band, custom made to fit the right maxillary canine tooth and a preformed human deciduous incisor stainless steel crown (#77 - Unitek Corp., Monrovia, California), which was adapted to the opposing mandibular canine tooth.

The gold band was constructed with a platform directed anteriorly and ending one millimeter from the adjacent anterior tooth as illustrated in Figure 2. Three feet of hook-up wire with thermoplastic insulation (#8523, Belden Corp., Chicago, Illinois) was soldered to the labial surface near the gingival border of each appliance.

In order to fabricate the gold band extraorally, an impression was required so that a positive reproduction of the maxillary arch could be obtained. The tray for the impression material was fabricated directly in the dog's mouth. Two sheets of baseplate wax (Truwax, Dentsply In-

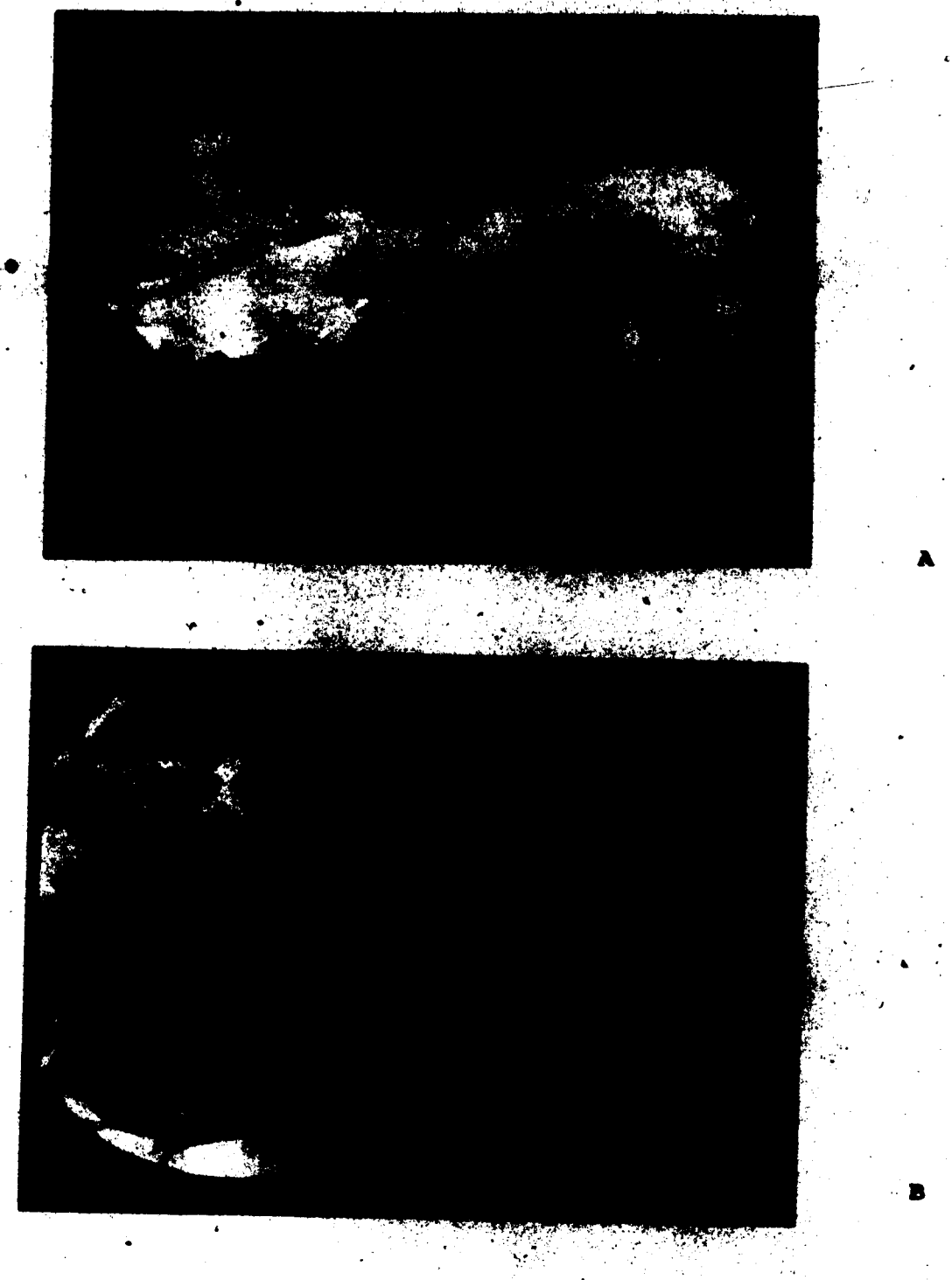


Figure 2: Illustration of maxillary canine appliance in position on the model. A, lateral view. B, occlusal view.

ternational Inc., York, Pennsylvania) were softened under warm water and placed to cover the maxillary arch and palate. The powder and liquid of an orthodontic acrylic resin (L.D. Caulk Co., Toronto, Ontario) were mixed to the dough stage and then adapted over the wax in the dog's mouth. The wax prevented the acrylic from becoming trapped in the undercut areas of the teeth and provided an acrylic tray of adequate size to take the impression. After the acrylic began to set it was removed from the mouth, along with the wax, and placed in hot water to hasten the set. When the acrylic was set all of the wax was removed and the tray was trimmed to the desired form. The impression portion of the tray was perforated with numerous two millimeter holes that were uniformly distributed to provide for the retention of the impression material in the tray. The impression material was an irreversible hydrocolloid (Jeltrate, L.D. Caulk Co.).

The impression was poured with stone (Velmix, Kerr Sybron Corp., Romulus, Michigan) to provide the reproduction of the maxillary arch. The gold band was first made in wax on the model, removed from the model and invested, burned out and then cast in gold. After polishing, the band was ready for use in the dog's mouth. The appliances were held in place on the canine teeth by a zinc phosphate cement (Orthocem, L.D. Caulk Co.).

C. Instrumentation

Silver-silver chloride surface electrodes (#650-951, Beckman Instruments Inc., Fullerton, California) were

employed to pick up the electrical activity in each of the masseter muscles and provide the input to two differential preamplifiers (Grass Model P15B). The amplifier frequency response range was set at a lower limit of 100 Hz and an upper limit of 1000 Hz and the amplifier gain was set at 1000. The amplifier outputs were recorded on separate channels of a four track FM tape recorder (Thermionic Model TDR3 with a bandwidth of 2500 Hz) at a tape speed of 15 inches per second and monitored through a four channel amplifier (Tektronix Type 3A74, with time base Type 2B67) on a four trace storage oscilloscope (Tektronix Type 564).

The direct current circuit indicating the time of occlusion consisted of the wire leads from the intraoral appliances forming a simple series circuit operated by a 1.5 volt battery. The intraoral appliances produced an open-close switch action. This circuit was connected directly to one channel of the tape recorder and monitored along with the masseter muscles. A microphone provided input to the fourth channel of the tape recorder permitting an audio record of experimental progress. A block diagram illustrating the apparatus arrangement during recording is presented in Figure 3.

The data was averaged by an analogue computer (Biomac 1000, Data Laboratories Ltd., London, England). The recorded muscle activity was full wave rectified to provide a signal which was unidirectional from ground potential in order that all components of the signal would sum in the

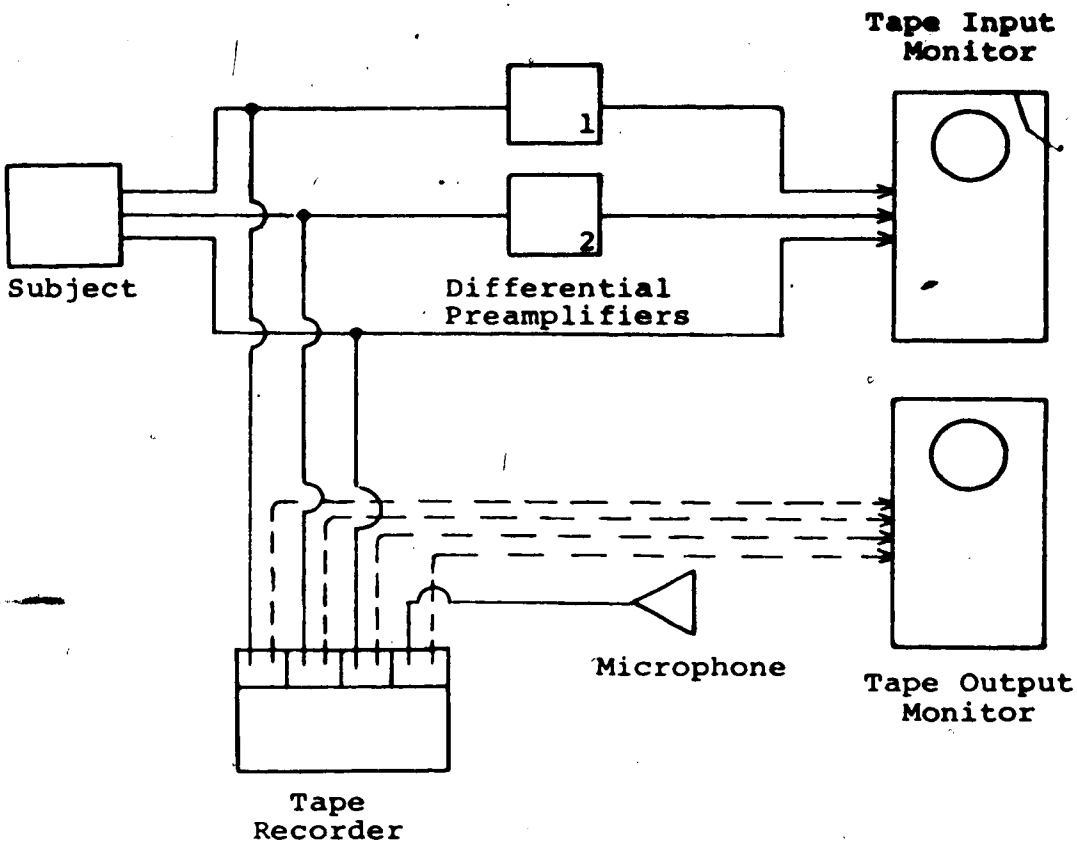


Figure 3: Block diagram of equipment arrangement for recording.

averaging process. The full wave rectifier also doubled the amplifier output voltage amplitude. After rectification, the signals from the two masseter muscles served as two inputs to the computer averaging program, while the direct current pulse generated by contact of the intraoral appliances served to trigger the sweep phase of the averaging process. The computer was set in the external trigger mode. This meant that if the time interval between successive trigger pulses was less than the sweep time, the second pulse would initiate a new sweep cycle. The duration of the sweep cycle was 80 milliseconds. Each sample consisted of 32 consecutive sweeps (consecutive tooth taps) averaged by the computer. The averaged result was displayed on the cathode ray tube of the computer and photographed with an oscilloscope camera (Tektronix Model C12) to provide a permanent visible record.

D. Procedure

One half hour prior to each work session with the dog, 10 milligrams of acetpromazine (Atravet, Ayerst Laboratories, Montreal, Quebec) was administered intramuscularly using a disposable 25 gauge needle and a 5 cubic centimeter syringe (Becton Dickinson Co., Clarkson, Ontario).

As the animal became familar with the laboratory environment and experimental procedures, the administration of acetpromazine was not required. During recording of data on tape, local anesthetic was the only drug administered.

The impression of the maxillary arch was obtained

with the animal under general anesthesia. Pentothal sodium (Nembutal, Abbott Laboratories, Montreal, Quebec) was administered intravenously (30 milligrams per kilogram of body weight) using a disposable 21 gauge needle and a 10 cubic centimeter syringe, to render the animal unconcious. This permitted intubation and connection to the general anesthesia apparatus (The Ohio Mfg. Co., Minneapolis, Minnesota), supplying oxygen and fluothane (Halothane, Halocarbon Ltd., Malton, Ontario).

The following procedure was employed for each electromyographic trial and recording session. The hair over the masseter muscles and the saggital crest was removed with an electric clipper to permit attachment of the surface electrodes to the skin. Early attempts to remove the hair to the skin level by using depilating agents or shaving cream and safety razor produced a skin surface with a film of clear inflammatory exudate due to the irritation of the procedure. The exudate interferred with the adhesion of the electrodes and the reception of the electrical activity from the muscles. The areas of electrode attachment were thoroughly cleaned with ninety-five percent ethanol and dried. To facilitate conduction of electrical action potentials from the skin to the electrode, a conducting medium (Synapse, Med Tek Corp., Northbrook, Illinois) was placed in the concavities of the electrodes. The electrodes were attached to the skin using adhesive collars (#650451, Beckman Instruments Inc.) in a standardized manner

with two electrodes overlying each masseter muscle and a single ground electrode attached over the saggital crest, as illustrated in Figure 4A. The interelectrode distance between centers was three quarters of an inch for the muscle electrodes, which were centrally located on the long axis of each masseter muscle and equidistant from the transverse axis.

The intraoral appliances were placed on the right cuspid teeth while the animal was biting on a wooden stick to keep the mouth open. The cuspid teeth involved were dried with gauze sponges and then the appliances were lined inside with a zinc phosphate cement (Orthocem, L.D. Caulk Co.) to supply appliance retention. They were then seated into place on the teeth and held until the initial setting of the cement occurred, usually 3 to 5 minutes. The intraoral appliances are illustrated in Figure 4B.

The electrical leads from the surface electrodes and the intraoral appliances were connected to the recording equipment. The animal was induced to produce a chewing motion by inserting a few drops of local anesthetic, mepvicaine, (Carbocaine, Cook Waite Laboratories, Aurora, Ontario) into its mouth. The electrical activity of both masseter muscles as well as the time of tooth contact during the jaw movements were recorded on tape. Then 54 milligrams of mepevicaine were administered by infiltration around each right cuspid tooth, using a 25 gauge needle, 1.8 milliliter anesthetic carpules and a regular aspirating type dental

Figure 4: A, illustration of surface electrode placement. B, illustration of intraoral appliances in place in the mouth.

syringe (Cook Waite Laboratories).

Ten minutes after the local anesthetic agent was administered, a pulp tester (Vitapulp, The Pelton and Crane Co., Charlotte, North Carolina) was used to provide an indication of the degree of anesthesia obtained. The stimulus intensity of the pulp tester could be increased on a scale from 1 to 10. The contralateral cuspid in each arch served as the control. All cuspid teeth were dried for the test. While the animal responded to a setting of 2 for the control teeth, a setting of 6 on the anesthetized teeth failed to elicit a response. Mechanical probing of the gingiva around the cuspid teeth on both sides of each arch provided similar responses. This was interpreted as having adequate anesthesia for this study.

The animal was again induced to produce a chewing motion as before, and the recording procedure was repeated. At the end of each experimental session with a dog, all electrodes and appliances were removed and the areas of their attachment were cleaned.

CHAPTER FOUR

RESULTS

The preanesthetic results of the latency and silent period for the right and left masseter muscles are presented in Figure 5.

PREANESTHETIC RESULTS

Sample	Latent Period (milliseconds)		Silent Period (milliseconds)	
	Right Masseter	Left Masseter	Right Masseter	Left Masseter
1	30	26	18	24
2	26	24	22	24
3	27	20	21	28
4	26	22	30	26
5	34	24	22	20
$\bar{x}$ ± S.D.	28.6 ± 3.4	23.5 ± 2.3	22.6 ± 4.4	24.4 ± 2.9

Figure 5: Preanesthetic results for latent and silent periods for right and left masseter muscles.

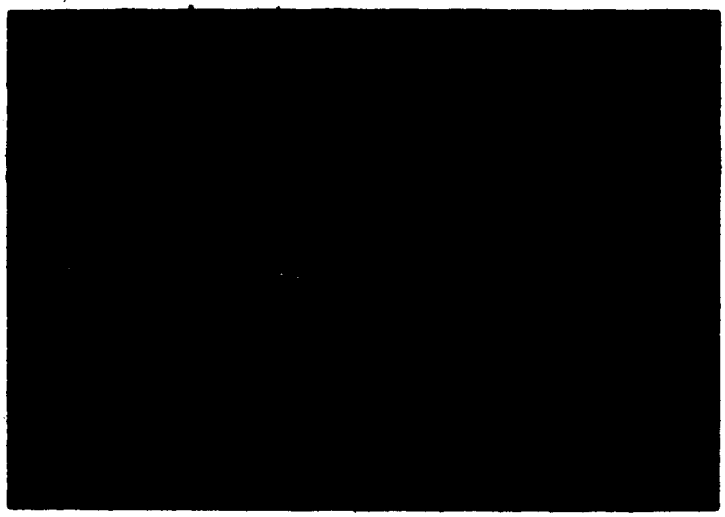
The mean latency of the right masseter muscle was 28.6 ± 3.4 milliseconds. This was 5.1 milliseconds longer than the mean latency of the left masseter muscle. The mean duration of the silent period in the right masseter muscle was 22.6 ± 4.4 milliseconds which was 1.8 milliseconds shorter than that in the left masseter muscle.

An illustration of the preanesthetic results is

presented in Figure 6 which demonstrates the initial myotatic reflex, the latency and silent period of both masseter muscles. The mean time of the myotatic reflex response was found to be 9.7 ± 1.8 milliseconds in the preanesthetic animal and 13.0 ± 1.7 milliseconds at ten minutes post anesthesia. In each case the left masseter muscle revealed a stretch reflex response that was slightly shorter (1 millisecond) than that of the right masseter muscle.

Figure 7A is inserted simply to illustrate silent periods associated with normal mastication in the dog. This electromyogram shows raw data obtained by monitoring both masseter muscles of the dog during mastication of dog biscuits. No local anesthetic or intraoral appliances were involved at that time. Figure 7B is included to demonstrate a preanesthetic unrectified sample which has been averaged by the computer.

The post anesthetic findings are illustrated in Figure 8. No silent periods are present in the electrical activity of either masseter muscle. The initial myotatic response to tooth contact is still present in the post anesthetic record. This stretch reflex was increased in amplitude and time due to loss of periodontal afferent activity. This result was consistently obtained in both dogs and is based on 244 recordings.



0.17 mv
8.0 msec

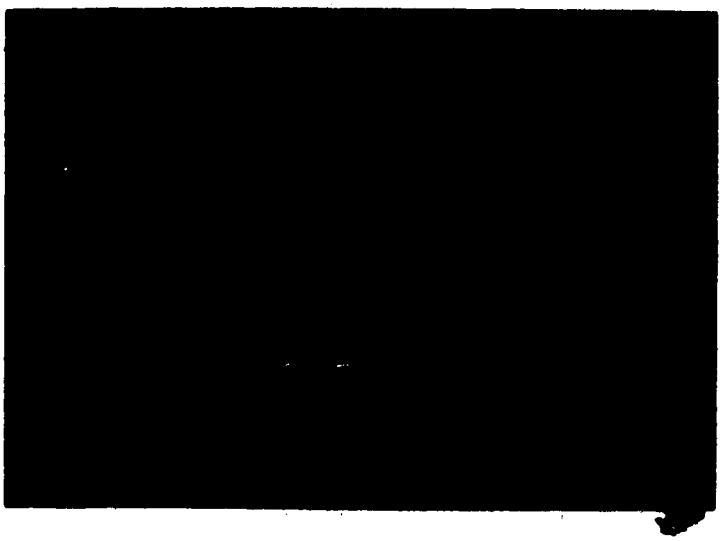
0.17 mv
8.0 msec

Figure 6: Preanesthetic records of two computer averaged samples illustrating the latent and silent periods. The top display in each photograph is from the right masseter muscle (appliance side). The bottom display is from the left masseter muscle.

A.

0.5 mv

20.0 msec



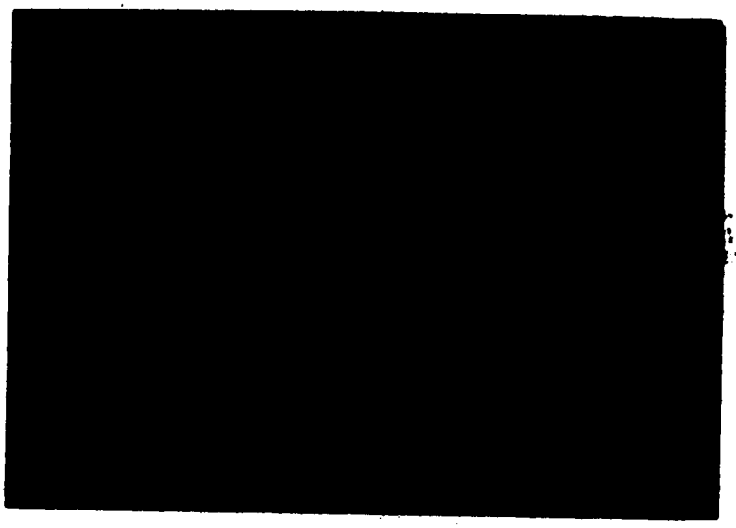
B.

0.17 mv

8.0 msec

Figure 7: A, illustration of silent periods (see arrows) in the dog recorded during normal mastication. B, illustration of preanesthetic unrectified computer averaged sample.

0.17 mv
8.0 msec



0.17 mv
8.0 msec

Figure 8: Postanesthetic records of two computer averaged samples illustrating the absence of silent periods. The top display in each photograph is from the right masseter muscle (appliance side). The bottom display is from the left masseter muscle.

CHAPTER FIVE

DISCUSSION AND CONCLUSION

The results of this study were in general agreement with the findings of Sessle (1972), Sessle and Schmitt (1972) and Goldberg (1971) which support the view that periodontal receptors initiate the silent period in the masseter muscles associated with tooth contact. The masseteric silent periods associated with tooth contact in this study were abolished by local anesthetic infiltration around the occluding cuspid teeth. Sessle and Schmitt (1972) demonstrated reduction and abolition of the silent period in human masseter muscles following administration of local anesthetic to mechanically stimulated maxillary central incisors. Masseteric silent periods in the present study on dogs were evoked by tooth stimulation resulting from functional mandibular movements which more closely represents natural mastication. Goldberg (1971) found a marked decrease in the masseteric silent period duration following infiltration of local anesthetic around mechanically stimulated maxillary central incisors. During stimulation of the incisor, the subject was biting on cotton wood sticks placed between the molar teeth. The stimulus was applied in a labio-lingual direction to the crown of the incisor, normal to its long axis. The method of stimulus application and biting on the sticks may have permitted stimulation of some receptors of adjacent or posterior teeth thus preventing abolition of the silent period.

In contrast to these results, Hannam et al (1970) found that local anesthetic failed to abolish the inhibition of the masseter muscles following stimulation of maxillary incisor teeth in human subjects. However, they used only 0.5 milliliters of local anesthetic (lidocaine) which was about one quarter of that used by Sessle and Schmitt (1972) or Goldberg (1971) and may have been an insufficient amount to anesthetize all of the involved periodontal receptors. In the present study 2.7 milliliters of 2 percent mepivacaine hydrochloride containing 1:20,000 neocobefrin (a vasoconstrictor) was infiltrated around both right canine teeth in the dog. Mepivacaine was used in this study because it remains more localized in the tissues, compared with lidocaine (Sutherland, 1970). In addition, it is claimed to be less toxic, more potent and longer acting than lidocaine even though both local anesthetics are xylidides (Grollman and Grollman, 1970).

Matthews and Yemm (1970) found silent periods with similar latency and duration in subjects with natural dentitions and those wearing full dentures. No attempt was made to anesthetize all of the denture bearing area to determine receptor locations. Their study raises the question concerning the fate of periodontal receptors upon extraction of the teeth. Do all of the receptors degenerate or do some adapt to a new role such as increasing gingival sensitivity? Matthews and Yemm (1970) suggested that receptors in the mucus membrane were stimulated by contact between the full

dentures to produce silent periods in the mandibular elevator muscles.

Hannam and Matthews (1969) have demonstrated in cat that the receptors associated with the jaw opening reflex were not located in the pulp, gingiva or palatal mucosa of the teeth. They found that mechanical stimulation of a maxillary canine always elicited a reflex jaw opening response. The gingival tissue was removed to expose 2 millimeters of bone around the canine. The response to the mechanical stimulation of the canine was undiminished. The pulp tissue of the canine was then removed and stimulation of the tooth was repeated. There was no alteration in the response to the stimulus. At this point in the experiment, with the pulp and gingival tissues removed, local anesthetic was infiltrated over the root of the canine. The tooth was mechanically stimulated as before and the response was found to be abolished.

The results of that study were important to the present investigation by indicating that the receptors associated with jaw muscle activity were not located in the pulp or gingival tissues. With these tissues excluded, the remaining periodontal structures assume more importance as possible sites of the receptors associated with the silent period. Since the masticatory systems of the cat and dog are anatomically similar, it is assumed, and remains to be demonstrated, that the findings of Hannam and Matthews (1969) in the cat are true for the dog.

Yemm (1969, 1971) has shown that muscle activity in humans was influenced by emotional stress. The alteration in muscle activity was attributed to activity in the gamma motor system initiated by higher centers in the central nervous system. Hannam et al (1969) have demonstrated that masseter muscle activity following tooth contact was variable and dependent upon the voluntary efforts of the subject.

This study attempts to avoid those problems by using experimental animals. The dog was chosen as the subject for this study because the temporomandibular joint in the dog is a simple hinge type joint with restricted mandibular movement. The absence of lateral jaw movements avoids extraneous muscle activity which may otherwise have altered the electromyographic results of this study. The simple open-close movement of the joint permits placement of occlusal interferences on the teeth such that occlusal contact always occurs at the same spot. The dog is a readily available animal and was of adequate size for this study. The author is not aware of previous masseteric silent period studies in the dog. This study provides the basis for future investigations into pathways and higher centers associated with the silent period in the masseter muscles of dog.

The abolition of silent periods in this study supports the view that the Golgi tendon organs were not responsible for the silent period seen in the masseter muscle upon tooth contact. The nature of the local anesthetic to be

localized in the tissues and the distant location of the infiltration from the masseter muscles in the dog indicates the Golgi organs and their nervous innervation were not affected by the anesthetic. In addition, the presence of the myotatic reflex before and after local anesthetic administration verifies that the masseter muscles and their neural innervation were not affected by the local anesthetic. This is in contrast to the results of Bessette et al (1974). They administered block anesthesia to the four dental quadrants in human subjects. Following anesthesia the jaw jerk response in the subjects was greatly diminished. This could be attributed to diffusion of the local anesthetic from the area of the mandibular foramen to the mandibular notch. Direct involvement of the masseteric nerves may have occurred as they pass laterally through the mandibular notch to reach the medial surface of the masseter muscles.

Munro and Griffin (1970) found the mean latent and silent period of the masseter muscle in man to be about 13 milliseconds, while Yu et al (1973) reported a latency of 15 to 20 milliseconds and a duration of 8 to 18 milliseconds following innocuous stimulation of facial and intraoral sites. Noxious stimulation of the same areas produced a response with a latency of 40 to 50 milliseconds and a duration of 15 to 35 milliseconds. Bessette et al (1971) reported that patients with temporomandibular joint dysfunction resulting from occlusal dysharmonies had a mean silent period of 60 milliseconds (with a range from 23 to 152

milliseconds.

The silent period in this study had a mean latency of 25.9 ± 3.9 milliseconds and a mean duration of 23.5 ± 3.7 milliseconds in the dog with occlusal dysfunction. This duration was within the range stated by Bessette et al (1971) and agreed with that found by Yu et al (1973) during noxious intraoral stimulation. The latency of this study was less than that found by Yu et al (1973) during noxious stimulation but greater than their latency associated with innocuous stimulation.

Munro and Basmajian (1971) reported that during the silent period in the mandibular elevator muscles, the digastric muscle revealed electromyographic activity. However, the mandibular depressor muscles (anterior and posterior digastric, inferior head of lateral pterygoid and mylohyoid) were without muscle spindles (Dmytruk, 1974). Thomas (1975) has suggested that periodontal receptors may serve for the mandibular depressor muscles the function that muscle spindles provide for the mandibular elevator muscles.

Goldberg (1972) found that the jaw jerk response was diminished when subjects clenched in the intercuspal position. According to Goldberg (1972) the periodontal receptors produced inhibition of the mandibular elevator muscles thereby reducing the jaw jerk response. He stated that "If inhibitory influences from periodontal receptors suppress the stretch reflex, then the reflex would be smallest when many receptors were activated (clench) and largest when

no receptors were activated (mass[redacted] muscle contralateral to biting side in the lateral position)". However, Moller (1966) reported that during chewing the masseter muscle on the chewing side displayed the greatest activity, and this study is in agreement with that report. The masseter muscle on the side ipsilateral to the intraoral appliances in this study had the longer latency and shorter silent period than the nonfunctioning side. The reduced stretch response during clenching (Goldberg, 1972) may be attributed to the restricted mandibular movement during the jaw jerk. The stimulation of muscle spindles would be reduced, thereby causing a reduced response.

In the present study, the shorter latency on the side contralateral to the side with the appliances may be an adaptive response to protect the integrity of the temporomandibular joint by preventing torquing of the mandible. Innervation of such a response may originate from receptors in the fibrous capsule of the temporomandibular joint (Greenfield and Wyke, 1966). Kawamura and Majima (1964) have described regions of the trigeminal sensory nuclei which respond to condylar movements. The activities of these points in the trigeminal sensory nuclei had corresponding spots of activity in the homolateral motor nucleus of the trigeminal nerve that were reciprocally inhibited or activated. The longer duration of the silent period on the side contralateral to the appliances may similarly act as a protective mechanism for the temporomandibular joint as well

as the involved teeth and their supporting structures.

In conclusion, this study has demonstrated the silent period associated with tooth contact in the masseter muscle of the dog. Administration of local anesthetic around the occluding teeth caused abolition of this silent period. This result indicates that silent periods seen upon tooth contact in the masseter muscle of the dog may be attributed to periodontal receptors. The unilateral occlusal interference in the animal produced an apparent adaptation to the foreign object. This was evident by the shorter latent period and the longer silent period on the side contralateral to that of intraoral appliances. It is suggested that this response prevents torquing of the mandible, thus protecting the temporomandibular joint and decreasing the force applied to the occluding teeth.

As with all research, this study leads to further work. The apparent location of the peripheral receptors associated with the masseteric silent period seen upon tooth contact, has now been determined. Further studies should investigate the pathways and higher centers of the central nervous system associated with this silent period. Electrolytic lesions in the trigeminal nuclei and nerve resections may be utilized to determine the pathways and synaptic locations involved with the silent period. The work of Hannam and Matthews (1969) should be repeated using the dog to ascertain the role of the pulp and gingiva in the production of masseteric silent periods. Future studies should investi-

gate the affect of long term unilateral occlusal interferences to determine the adaptation response.

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