ELECTRICAL STIMULATION OF THE MASSETER MUSCLE

by

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Abstract

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Functional Electrical Stimulation (FES) of skeletal muscle has been used to treat idiopathic scoliosis. To determine the feasibility of using FES to treat malocclusion thin, coiled, multistranded wire electrodes were placed bilaterally in the masseter muscles in two groups of adult volunteers. A portable pulse generator (Neu Rx Is/4) was used to stimulate the masseter muscle at half the stimulus current required to effect a painful sensation in five subjects. In a second group of five subjects the stimulus amplitude was adjusted to produce three pounds of bite force. Morning and afternoon bite force measurements were taken on weekdays for a two week time period. Results indicated that a bite force of three pounds can be sustained over a period of two weeks using this method. Pain and discomfort was rated as minimal. In conclusion, the use of FES to treat malocclusion is theoretically possible and technically feasible. Further tests are needed to determine the utility of using FES to treat growing patients.

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DEDICATED

TO

MR. and MRS. G. G. DI BATTISTA

My Parents, who made my
education possible.

TABLE OF CONTENTS

Page

ABSTRACT ii

ACKNOWLEDGEMENTS iii

DEDICATION. iv

LIST OF TABLES vi

LIST OF FIGURES. x

INTRODUCTION. 1

LITERATURE REVIEW 3

STATEMENT OF THESIS 56

METHODS AND MATERIALS 58

RESULTS. 78

DISCUSSION. 111

SUMMARY AND CONCLUSIONS 118

APPENDIX I 119

APPENDIX II 168

REFERENCES CITED. 171

LIST OF TABLES

TABLE		Page
1.	Amplitude (mA) used for simultaneous chronic electrical stimulation of the bilateral masseter muscles for Subjects in Part A	.67
2.	Amplitude (mA) used for alternating chronic electrical stimulation of the bilateral masseter muscles for Subjects in Part B	.67
3.	Daily average bite forces at T1 (morning) and T2 (afternoon) during simultaneous, chronic electrical stimulation of the bilateral masseter muscles for Subject 1 in Part A	.80
4.	Daily average maximum voluntary bite forces before (T1, morning) and (T2, afternoon) simultaneous, chronic electrical stimulation of the bilateral masseter muscles for Subject 1 in Part A	.82
5.	Average % change of daily bite forces during simultaneous, chronic electrical stimulation of the bilateral masseter muscles for all Subjects in Part A	.84
6.	Daily average bite forces right, at T1 (morning) and T2 (afternoon) during alternating, chronic electrical stimulation of the right masseter muscle for Subject 1 in Part B.	.87
7.	Daily average bite forces left, at T1 (morning) and T2 (afternoon) during alternating, chronic electrical stimulation of the left masseter muscle for Subject 1 in Part B.	.89
8.	Daily average maximum voluntary bite forces right, before (T1, morning) and after (T2, afternoon) alternating, chronic electrical stimulation of the bilateral masseter muscles for Subject 1 in Part B	.91
9.	Daily average maximum voluntary bite forces left, before (T1, morning) and after (T2, afternoon) alternating, chronic electrical stimulation of the bilateral masseter muscles for Subject 1 in Part B	.93
10.	Daily average bite forces right, at T1 (morning) and T2 (afternoon) during alternating, chronic electrical stimulation of the right masseter muscle for all Subjects in Part B	.96

11. Daily average bite forces right, at T1 (morning) and T2 (afternoon) during alternating, chronic electrical stimulation of the left masseter muscle for all Subjects in Part B 99

12. Average % change of daily maximum voluntary bite forces, right and left, before (T1, morning) and after (T2, afternoon) alternating, chronic electrical stimulation of the bilateral masseter muscles for all subjects in Part B 103

13. Electromyographic results of the right temporalis muscle following simultaneous or alternating, chronic electrical stimulation of the bilateral masseter muscles in Part A and Part B. 105

14. McGill Pain Questionnaire results for Subjects in Part A that received no local anesthesia at the time of electrode insertion 107

15. McGill Pain Questionnaire results for Subjects in Part A that received local anesthesia at the time of electrode insertion 108

16. McGill Pain Questionnaire results for Subjects in Part B 109

17. McGill Pain Questionnaire word descriptive by number of Subjects selecting each descriptor in Part A and Part B 110

18. Daily average bite forces at T1 (morning) and T2 (afternoon) during simultaneous, chronic electrical stimulation of the bilateral masseter muscles for Subject 2 in Part A 120

19. Daily average maximum voluntary bite forces before (T1, morning) and (T2, afternoon) simultaneous, chronic electrical stimulation of the bilateral masseter muscles for Subject 2 in Part A 122

20. Daily average bite forces at T1 (morning) and T2 (afternoon) during simultaneous, chronic electrical stimulation of the bilateral masseter muscles for Subject 3 in Part A 124

21. Daily average maximum voluntary bite forces before (T1, morning) and (T2, afternoon) simultaneous, chronic electrical stimulation of the bilateral masseter muscles for Subject 3 in Part A 126

22. Daily average bite forces at T1 (morning) and T2 (afternoon) during simultaneous, chronic electrical stimulation of the bilateral masseter muscles for Subject 4 in Part A 128

23. Daily average maximum voluntary bite forces before (T1, morning) and (T2, afternoon) simultaneous, chronic electrical stimulation of the bilateral masseter muscles for Subject 4 in Part A 130

24. Daily average bite forces at T1 (morning) and T2 (afternoon) during simultaneous, chronic electrical stimulation of the bilateral masseter muscles for Subject 5 in Part A 132

25. Daily average maximum voluntary bite forces before (T1, morning) and (T2, afternoon) simultaneous, chronic electrical stimulation of the bilateral masseter muscles for Subject 5 in Part A 134

26. Daily average bite forces right, at T1 (morning) and T2 (afternoon) during alternating, chronic electrical stimulation of the right masseter muscle for Subject 2 in Part B 136

27. Daily average bite forces left, at T1 (morning) and T2 (afternoon) during alternating, chronic electrical stimulation of the left masseter muscle for Subject 2 in Part B 138

28. Daily average maximum voluntary bite forces right, before (T1, morning) and after (T2, afternoon) alternating, chronic electrical stimulation of the bilateral masseter muscles for Subject 2 in Part B 140

29. Daily average maximum voluntary bite forces left, before (T1, morning) and after (T2, afternoon) alternating, chronic electrical stimulation of the bilateral masseter muscles for Subject 2 in Part B 142

30. Daily average bite forces right, at T1 (morning) and T2 (afternoon) during alternating, chronic electrical stimulation of the right masseter muscle for Subject 3 in Part B 144

31. Daily average bite forces left, at T1 (morning) and T2 (afternoon) during alternating, chronic electrical stimulation of the left masseter muscle for Subject 3 in Part B 146

32. Daily average maximum voluntary bite forces right, before (T1, morning) and after (T2, afternoon) alternating, chronic electrical stimulation of the bilateral masseter muscles for Subject 3 in Part B 148

33. Daily average maximum voluntary bite forces left, before (T1, morning) and after (T2, afternoon) alternating, chronic electrical stimulation of the bilateral masseter muscles for Subject 3 in Part B 150

34. Daily average bite forces right, at T1 (morning) and T2 (afternoon) during alternating, chronic electrical stimulation of the right masseter muscle for Subject 4 in Part B 152

35. Daily average bite forces left, at T1 (morning) and T2 (afternoon) during alternating, chronic electrical stimulation of the left masseter muscle for Subject 4 in Part B 154

36. Daily average maximum voluntary bite forces right, before (T1, morning) and after (T2, afternoon) alternating, chronic electrical stimulation of the bilateral masseter muscles for Subject 4 in Part B 156

37. Daily average maximum voluntary bite forces left, before (T1, morning) and after (T2, afternoon) alternating, chronic electrical stimulation of the bilateral masseter muscles for Subject 4 in Part B 158

38. Daily average bite forces right, at T1 (morning) and T2 (afternoon) during alternating, chronic electrical stimulation of the right masseter muscle for Subject 5 in Part B. 160

39. Daily average bite forces left, at T1 (morning) and T2 (afternoon) during alternating, chronic electrical stimulation of the left masseter muscle for Subject 5 in Part B. 162

40. Daily average maximum voluntary bite forces right, before (T1, morning) and after (T2, afternoon) alternating, chronic electrical stimulation of the bilateral masseter muscles for Subject 5 in Part B 164

41. Daily average maximum voluntary bite forces left, before (T1, morning) and after (T2, afternoon) alternating, chronic electrical stimulation of the bilateral masseter muscles for Subject 5 in Part B 166

42. E.M.G. of the right temporalis muscle during simultaneous electrical stimulation of the bilateral masseter muscles in Subject I, Part A . . . 169

43. E.M.G. of the right temporalis muscle during electrical stimulation of the masseter muscles at rest and during voluntary mouth opening for all subjects. 170

LIST OF FIGURES

FIGURE		PAGE
1.	Neu Rx Is/4 stimulation system	.59
2.	Programmer	.61
3.	Pusle Specifications	.62
4.	Standard Electrode	.64
5.	Pulse train specifications	.66
6.	Daily average bite forces during simultaneous, chronic electrical stimulation of the bilateral masseter muscles for Subject 1, Part A . . .	.81
7.	Daily average maximum voluntary bite forces before and after simultaneous, bilateral chronic electrical stimulation of the masseter muscles for Subject 1, Part A	.83
8.	Average % change of daily bite forces during simultaneous, chronic electrical stimulation of the bilateral masseter muscles for all Subjects in Part A	.85
9.	Daily average bite forces right, at T1 (morning) and T2 (afternoon) during alternating, chronic electrical stimulation of the right masseter muscle for Subject 1, Part B	.88
10.	Daily average bite forces left, at T1 (morning) and T2 (afternoon) during alternating, chronic electrical stimulation of the left masseter muscle for Subject 1, Part B	.90
11.	Daily average maximum voluntary bite forces right, before (T1, morning) and after (T2, afternoon) alternating, chronic electrical stimulation of the bilateral masseter muscles for Subject 1, Part B	.92
12.	Daily average maximum voluntary bite forces left, before (T1, morning) and after (T2, afternoon) alternating, chronic electrical stimulation of the bilateral masseter muscles for Subject 1, Part B	.94

13. Daily average bite forces right, at T1 (morning) and T2 (afternoon) during alternating, chronic electrical stimulation of the right masseter muscle for all Subjects in Part B 97

14. Average % change of daily maximum voluntary bite forces, right, at T1 (morning) and T2 (afternoon) during alternating, chronic electrical stimulation of the right masseter muscle for all subjects in Part B 98

15. Daily average bite forces left, at T1 (morning) and T2 (afternoon) during alternating, chronic electrical stimulation of the left masseter muscle for all Subjects in Part B 100

16. Average % change of daily maximum voluntary bite forces, left, at T1 (morning) and T2 (afternoon) during alternating, chronic electrical stimulation of the left masseter muscle for all subjects in Part B . . . 101

17. Average % change of daily maximum voluntary bite forces, right and left, before (T1, morning) and after (T2, afternoon) alternating, chronic electrical stimulation of the bilateral masseter muscles for all subjects in Part B 104

18. Daily average bite forces during simultaneous, chronic electrical stimulation of the bilateral masseter muscles for Subject 2, Part A . . 121

19. Daily average maximum voluntary bite forces before and after simultaneous, bilateral chronic electrical stimulation of the masseter muscles for Subject 2, Part A 123

20. Daily average bite forces during simultaneous, chronic electrical stimulation of the bilateral masseter muscles for Subject 3. Part A . . 125

21. Daily average maximum voluntary bite forces before and after simultaneous, bilateral chronic electrical stimulation of the masseter muscles for Subject 3, Part A 127

22. Daily average bite forces during simultaneous, chronic electrical stimulation of the bilateral masseter muscles for Subject 4, Part A . . 129

23. Daily average maximum voluntary bite forces before and after simultaneous, bilateral chronic electrical stimulation of the masseter muscles for Subject 4, Part A 131

24. Daily average bite forces during simultaneous, chronic electrical stimulation of the bilateral masseter muscles for Subject 5, Part A . . 133

25. Daily average maximum voluntary bite forces before and after simultaneous, bilateral chronic electrical stimulation of the masseter muscles for Subject 5, Part A 135

26. Daily average bite forces right, at T1 (morning) and T2 (afternoon) during alternating, chronic electrical stimulation of the right masseter muscle for Subject 2, Part B 137

27. Daily average bite forces left, at T1 (morning) and T2 (afternoon) during alternating, chronic electrical stimulation of the left masseter muscle for Subject 2, Part B 139

28. Daily average maximum voluntary bite forces right, before (T1, morning) and after (T2, afternoon) alternating, chronic electrical stimulation of the bilateral masseter muscles for Subject 2, Part B 141

29. Daily average maximum voluntary bite forces left, before (T1, morning) and after (T2, afternoon) alternating, chronic electrical stimulation of the bilateral masseter muscles for Subject 2, Part B 143

30. Daily average bite forces right, at T1 (morning) and T2 (afternoon) during alternating, chronic electrical stimulation of the right masseter muscle for Subject 3, Part B 145

31. Daily average bite forces left, at T1 (morning) and T2 (afternoon) during alternating, chronic electrical stimulation of the left masseter muscle for Subject 3, Part B 147

32. Daily average maximum voluntary bite forces right, before (T1, morning) and after (T2, afternoon) alternating, chronic electrical stimulation of the bilateral masseter muscles for Subject 3, Part B 149

33. Daily average maximum voluntary bite forces left, before (T1, morning) and after (T2, afternoon) alternating, chronic electrical stimulation of the bilateral masseter muscles for Subject 3, Part B 151

34. Daily average bite forces right, at T1 (morning) and T2 (afternoon) during alternating, chronic electrical stimulation of the right masseter muscle for Subject 4, Part B 153

35. Daily average bite forces left, at T1 (morning) and T2 (afternoon) during alternating, chronic electrical stimulation of the left masseter muscle for Subject 4, Part B 155

36. Daily average maximum voluntary bite forces right, before (T1, morning) and after (T2, afternoon) alternating, chronic electrical stimulation of the bilateral masseter muscles for Subject 4, Part B 157

37. Daily average maximum voluntary bite forces left, before (T1, morning) and after (T2, afternoon) alternating, chronic electrical stimulation of the bilateral masseter muscles for Subject 4, Part B 159

38. Daily average bite forces right, at T1 (morning) and T2 (afternoon) during alternating, chronic electrical stimulation of the right masseter muscle for Subject 5, Part B 161

39. Daily average bite forces left, at T1 (morning) and T2 (afternoon) during alternating, chronic electrical stimulation of the left masseter muscle for Subject 5, Part B 163

40. Daily average maximum voluntary bite forces right, before (T1, morning) and after (T2, afternoon) alternating, chronic electrical stimulation of the bilateral masseter muscles for Subject 5, Part B 165

41. Daily average maximum voluntary bite forces left, before (T1, morning) and after (T2, afternoon) alternating, chronic electrical stimulation of the bilateral masseter muscles for Subject 5, Part B 167

INTRODUCTION

Most orthodontists conceptually agree that facial form is influenced by the musculature of the orofacial region. However, it is the rare instance in which the treatment plan includes an appliance designed to change muscle function. In fact, the majority of orthodontic treatment provided today is remarkably similar to that prescribed fifty years ago. To help close the gap between theoretical knowledge and clinical practice we need to dramatically improve our ability to change muscle function. Ideally, appliances that seek to change facial morphology by changing muscle function should: 1) Provide reliable operator control of both the force and duration of muscle activity, 2) Allow stimulation or inhibition of selected muscles, and 3) Be able to be used alone or as an adjunct to traditional mechanical techniques. If appliances meeting these criteria could be developed, the range of malocclusions that could be effectively treated by orthodontic practitioners in the 21st century would undoubtedly increase. Fortunately, devices designed to stimulate skeletal muscle exist and have been used in orthopedic medicine for the treatment of idiopathic scoliosis. (Axelgaard 1984; McCollough 1986). Similar devices have been designed and built in the Applied Neural Control Laboratory at CWRU. However, before electrical stimulation devices can be developed for orthodontic uses, we need to determine some parameters for their use on the masticatory musculature. In particular, we plan to study the efficacy of using a compact, battery powered electrical stimulator (Neu Rx Is/4) to cause contraction of the masseter muscle in a group of 10 adult volunteers. Using hypodermic needles

coiled, multistranded, intramuscular electrodes will be placed and electromyographic activity (EMG) as well as bite force will be measured to achieve the following specific aims:

1. Determine the pulse width, frequency, amplitude, and duty cycle of electrical stimuli necessary to produce bite forces in the therapeutic range (1-10 lbs.).
2. Determine if such forces can be sustained over a sufficiently long period of time so as to make it practical and useful to use electrical stimulation of muscles to alter facial morphology.

If successful, there would be many potential clinical uses for this new technique. One application would be to limit vertical facial development in the growing child. The stimulator could be used to improve the quality and/or quantity of muscular activity of the elevator muscles of the mandible. In this way, eruption of posterior teeth and subsequent development of the alveolar process may be modified. The ability to control changes in vertical alveolar height would allow orthodontic treatment of a greater number of severely handicapping open bite malocclusions in growing adolescent patients. These open bite malocclusions are now treated most often by surgical LeFort I osteotomy after the pubertal growth spurt. No valid treatment option currently exists for treatment of these children during the growth years. Although initially this technique will be used for patients who have no other valid treatment option, our long term objective is to use electrical stimulation of the facial muscles as an adjunct to traditional mechanical force delivery systems. For example, wouldn't it be beneficial to have all our high angle open bite malocclusions have the muscle physiology of low angle deep bite subjects? Operator control of the muscular environment will benefit our patients by allowing clinical application of biologic knowledge that has been largely of scientific value.

LITERATURE REVIEW

Growth of the craniofacial skeleton is influenced by neuromuscular function. Specifically, factors such as migration and attachment of muscle, variations in neuromuscular functions, growth of the muscles, and abnormal function (e.g., mouth breathing) influence craniofacial growth and form. Moss' Functional Matrix Theory states that

> *"the origin, growth and maintenance of all skeletal tissue and organs are always secondary, compensatory and obligatory responses to actual and prior events that occur in specifically related non-skeletal tissues, organs or functioning spaces, the functional matrices."*

The cranial and mandibular muscles enclose the skeletal structures of the face and mandible and can be divided into the muscles of facial expression and the muscles of mastication. These muscle groups assist in swallowing and respiration. During function muscles exert force on their anatomical points of insertion. For example, the muscles of mastication exert force on the mandible, provide stability, allow precise jaw movements and maximize loading forces to the teeth. The intimate relationship of the skeletal structure and supportive musculature indicates that bone, as a tissue, is modified in its shape and size by the muscle tissue with which it interacts.

Current intervention strategies do not directly increase muscle contraction. Instead, appliances are designed to alter the postural position of the mandible. Thereby,

potentially inducing change in muscle. Low level current applied to the neuromuscular system, called Functional Electrical Stimulation, is used in rehabilitation technology (Hambrecht and Reswick, 1977). Functional Electrical Stimulation is the use of electricity to evoke a skeletal muscle response. The electrical excitability of nerve and muscle tissue provides the basis for its therapeutic use. Zoll (1952) developed the first successful artificial cardiac pacemaker, using external cardiac stimulation. This marked one of the first therapeutic applications of electricity which demonstrated effectiveness. Liberson et al. (1961) described a technique for treating foot drop in stroke patients, which consisted of stimulating the affected foot to prevent the drag during the swing phase of gait. The stimulus was turned on each time the foot was lifted off the floor by a switch placed in the heel of the patient. The success of this procedure stimulated additional research, which focused on the possibility of using electrical stimulation to control paralyzed extremities. This term, Functional Electrical Stimulation, coined by Moe and Post in 1962, has been broadly applied to encompass this field ever since.

Orthodontic treatment has focused on changing the position of teeth and bone, using mechanical systems. Using fixed orthodontic appliances, forces are applied to the teeth. In a like manner, changes in neuromuscular activity generated by functional orthodontic appliances alter stress on the teeth and bones. Theoretically, the functional appliances induce change in the pattern of muscle function. This change in muscle function is postulated to lead to the development of a correspondingly new morphologic pattern in the skeletal structure (Carels et al., 1987).

Facial Growth - General Overview

Over the years, many theories have been postulated to explain not merely what happens when the face grows, but how it is accomplished. In trying to explain local control mechanisms, many regulators have been suggested. These include the genome, neurotrophic substances, pH levels, oxygen tension, and muscle function. In addition, a multitude of enzymatic, as well as localized hormonal factors, and physiologic demands have been mentioned (Enlow, 1973).

An interrelationship of muscle function and bone growth has been shown in many experimental studies. But the actual role muscle function plays in controlling growth still remains obscure. Brodie (1941) has stated that facial form is genetically predetermined and environmental factors are secondary. Hunter (1965) stated that the antero-posterior dimensions of the face appear to reflect the effects of environmental influences to a greater extent than do height dimensions.

Van der Klaaw (1945) views the craniofacial complex as being comprised of at least thirty independent functional units, each governed by its own growth director. Moss (1962) developed the "Functional Matrix" concept which places primary importance upon the adaptability of structure to functional patterns and intrinsic environmental conditions. Van Limborgh (1972) notes the variable roles of intrinsic genetic factors and local epigenetic factors in the differentiation of specific areas of the craniofacial complex.

It appears that, in a complex structure such as the craniofacial skeleton, many factors may be concerned with the determination of form. It is known that certain parts of the cranial skeleton depend on the degree of activity of the attached muscles for their full development. Wolff's law of bone transformation, introduced in 1899, is a useful

concept (Enlow, 1968a). Growing muscles and other mechanical agents exert specific stresses on a bone, thereby bringing about and controlling the lengthening of the whole bone. When muscle growth ceases an equilibrium is established and the stimulus for osteoblastic activity ceases (Enlow, 1973).

A relationship between muscle and facial growth has been advanced by several investigators (Moss and Simon, 1968; Moss, 1969). Certain factors such as migration and attachment of muscle, variations in neuromuscular function, growth of the muscles and abnormal function are now known to influence craniofacial growth and form. Moss (1981) describes the Functional Matrix theory and states that the origin, growth and maintenance of all skeletal tissue and organs are always secondary, compensatory and obligatory responses to actual and prior events that occur in specifically related non-skeletal tissues, organs or functional spaces.

According to Moss' method of functional cranial analysis, a Functional Matrix, along with a skeletal unit, make up an individual functional cranial component. Any function is carried out by a matrix while the necessary role of providing protection and support is assumed by the skeletal unit. The periosteal and the capsular matrices make up the "functional matrices". The periosteal functional matrix can be exemplified by the effect of muscles on the skeletal units upon which they act. The periosteal matrices act by osseous deposition and resorption (transformation). All skeletal units and functional matrices arise, grow and operate, and are maintained within the cranial capsules. Enlargement of the capsular matrices (e.g., oronasopharyngeal cavities) with the resultant increase in volume causes movement of the functional cranial components (translation), (Moss and Rankow, 1968; Moss and Salentijn, 1969; Moss and Feldman, 1969; Moss and Salentijn, 1969b).

Muscle and Facial Growth

The interaction of the soft tissue with the cranio-skeleton has focused on two aspects: 1) the effect that muscle has on modeling, and 2) the effect of muscle on the growth pattern of the cranio-skeleton. The relationship between muscle function and cranio-skeletal growth has been vastly studied. Loss or impaired function of the craniomandibular muscles dramatically alters the form and growth of the rapidly changing mammalian skeleton, but has little effect on adult facial form.

Many investigators have reported bony changes associated with surgical removal of a muscle or portion of a muscle. For example, Pratt (1943) described extreme skeletal distortions following removal of the masseter muscle and a portion of the zygomatic arch in one day old rats. In a similar experiment Washburn (1947) removed the temporal muscle unilaterally on rats. The operated animals showed deviation from the normal. The coronoid process of the mandible was entirely absent or represented as a small tubercle. The temporal line was either absent altogether or was far down on the temporal fossa at the margin of the residual infratemporal fibers. Horowitz and Shapiro (1951) reported similar findings.

Historically, one of the alternative concepts advanced to explain the control of bone growth included the concept that skeletal morphology is the direct product of the sum of all mechanical stresses placed upon that skeletal component. Many workers presumed that bone growth and biomechanics were synonymous and that mechanical stresses were the principal factors governing the course of skeletal morphogenesis. However, it is generally realized that the calcified portion of bone represents a product of growth and that the enclosing soft tissues associated with the bone (periosteum and endosteum or other comparable tissues) are the agents that produce growth and modeling of bone.

Horowitz and Shapiro (1955) removed the masseter muscle on 30 day old rats. The masseter muscle was removed unilaterally. Removal of the masseter muscle resulted in severe alterations of the skull, jaws and dental arches. Molar tooth occlusion was affected on both sides of the mouth, and an "open bite" resulted on the side of the operation.

Avis (1959) removed the superficial masseter and internal pterygoid muscles in a series of young rats. The angular process was greatly reduced in all dimensions when either the masseter muscle or the internal pterygoid muscle was removed. The angular process was entirely absent when both the superficial masseter and internal pterygoid muscles were both completely removed.

Soni and Malloy (1973) removed the temporal muscle on the coronoid process in guinea pigs. The study was based on observations in 14 guinea pigs which were separated at random into two groups: seven operated and seven sham operated. All guinea pigs received demethylchlorotetracylcine (DCTC) 24 hours before surgery and 24 hours before they were killed. Hematoporphyrin was administered to each animal at 30 days of age and 2,4-bis fluorescein (DCAF) at 45 days of age. The entire origin of the temporal muscle and fascia was removed from the skull. The results showed that unilateral removal of the temporal muscle in infant guinea pigs was followed by a decrease in size of the coronoid process.

The extirpation experiments are sometimes criticized based on the assumption that the morphologic changes following resection would be rather due to secondary factors such as interruptions of the vascular supply to the bony environment, sensory loss and scar tissue.

In order to decrease general trauma to the muscle and bone and in order to leave the

muscle bone interface intact, a number of experiments have been designed to study the form/function relationship by paralyzing the jaw muscles.

Byrd (1988) determined the effects of alteration of the facial neuromuscular complex on craniofacial postnatal development by creating a lesion in the facial motoneurons within the brainstem of actively growing rats. Basicranial dimensions of experimental animals were least affected by the lesion, whereas zygomatic arch, dorsal facial region, and mandibular condyle dimensions were most affected.

Phillips et al. (1982) studied the morphologic alterations in Macaca Mulatta following destruction of the motor nucleus of the trigeminal nerve. Alterations in craniofacial form were noted clinically in both animals and included paralysis and atrophy of the muscles of mastication on the lesion side and mandibular asymmetry. Shape included dental and mandibular asymmetry, the appearance of an anterior open bite on the affected side, and a decrease in size of the infratemporal fossa on the affected side.

Behrents and Johnston (1984) demonstrated that skeletal alterations are primarily related to the effects of denervation. Behrents stereotaxically created unilateral electrolytic lesions in three different locations in growing rats. Results indicated that the effects on growth were dependent on lesion location, with the greatest effect occurring in animals that had a Gasserian ganglion lesion. Interestingly, both sensory and motor nerve deficits produced mandibulofacial growth alterations. Behrent's observations of the skeletal structure may be related to the elimination of contractile muscle activity with a decrease in muscle tension. In addition, these changes may be related to the elimination of neurotrophic substances. These conclusions are drawn from the fact that both sensory and motor portions of the nerve were destroyed.

Gardner et al. (1980) studied the alterations in the facial skeleton of the guinea pig following a experimental lesion of the trigeminal motor nucleus. Gardner's study avoided sensory denervation. The changes involved a substantial reorientation of the circummaxillary sutures, remodeling in several areas of bone underlying atrophic muscles and marked resorption of the condylar process and glenoid fossa on the operated side. There were significant differences in several dimensions of the mandible on the operated side versus the unoperated side and control animals.

The nature of neuromuscular adaptations in the orofacial system and their relation to skeletal alterations has also been studied. One of the first studies to investigate this was designed by McNamara. In 1973 McNamara investigated the nature of skeletal adaptations resulting from experimental alteration of the orofacial environment. A new occlusal configuration was devised which modified oral sensory stimuli. The experimental device positioned the mandible of rhesus monkeys in an anterior position during functional jaw movements. The study reported that 10 of the 12 experimental monkeys demonstrated anterior-posterior alteration in molar relation. Carlson and Schneiderman (1983) cephalometrically analyzed adaptations after postural lengthening of the masseter muscle in adult rhesus monkeys. The functional length of the elevator muscles of the mandible was increased with a bite opening splint cemented to the maxillary dentition. The major adaptations to increased vertical dimension were: 1) marked superior and some anterior displacement of the maxillary complex, and 2) dental intrusion.

Muscle size and function has also been correlated to bone size and facial type. Weijs and Hillen (1984) determined the relationship between masticatory muscle cross section and skull shape. Cross sectional areas of the masseter, temporalis

and medial, and lateral pterygoid muscles were determined in 16 subjects by means of computed tomography. In each subject three scans were made, intersecting the thickest part of the muscles at right angles to the fiber direction. The masseter and medial pterygoid muscles were large in persons with brachycephalic skulls, short faces and small jaw angles. The cross sectional areas of the temporalis and lateral pterygoid muscles showed no correlation with facial dimensions. Bloem and Van Hoof (1971) have demonstrated that increased muscle function of the masseter, such as in muscular hypertrophy, produces a corresponding increase in bone size. Lowe et al. (1983) demonstrated that high anterior temporal amplitude (using electromyography) was correlated with flat palatal planes and large ramus heights. High masseter amplitude was also correlated with upright maxillary incisors. Lowe (1980) also studied the correlation of orofacial muscle activity and craniofacial morphology in a sample of control and anterior open bite subjects. They observed that low threshold values for the masseter muscle were associated with low overbite measurements.

The studies mentioned above, as well as other similar studies, are often used to prove that muscle "stress" is the key factor controlling bone development. However, such a conclusion is premature since many variables have not been taken into account. For example, the muscles of the face, especially the muscles of mastication, have been shown to influence the morphogenesis of the craniofacial region (Scott, 1954). Yet Hoyte and Enlow (1966) and Enlow (1968a, 1968b) have also shown that the patterns of muscle attachment do not necessarily coincide with patterns of resorption and deposition. In many instances, the vector of force of the musculature is opposite to the direction of bone growth. There is no single agent directly responsible for the master control of growth. The growth control process must be multifactorial with specific

control pathways that can follow many routes and involve many agents. Control is a function of a system of feedback pathways.

Another approach to alter the facial growth process is to change the neuromuscular system by increasing or decreasing muscle contraction. Woodside et al. (1983) studied the hypothesis that muscle function influences bone formation and morphology and that the movement of bone within a muscle system results in a re-arrangement of the stress distribution and re-organization of shape and internal structure. Using powered muscle stimulation short periods of hyperactivity were induced in the masseter and temporalis muscles of juvenile and adult monkeys. No malocclusion was produced in the adult monkeys after 3 - 5 months of unilateral contraction. In contrast, similar levels of bilateral stimulation in the juvenile monkey resulted in a malocclusion.

Miller et al. (1989) had muscle leads implanted in the left temporalis muscle of 16 animals and were studied for 9 months. The animals were either stimulated in the mesencephalic or motor trigeminal nucleus. Miller studied the electromyographic properties of the muscles and found that the muscle most readily recruited with central stimulation is the masseter muscle. They also found that the mandibular response most readily observed with stimulation was vertical elevation of the mandible. Kantona and Ronning (1982) stimulated the lateral pterygoid muscle of 30 day old rats with intermittent shocks. After sixteen days of stimulation, the left mandibular half was 0.4 mm longer than the non-stimulated right side.

Yamada (1992) studied the influence of chewing function on craniofacial structure and development. A 24 hour electromyographic recording and analyzing system was developed to investigate masticatory activity in daily life. This method was applied to monkeys with experimental nasal obstruction. The electromyographic recording was

analyzed during 24 hours. The results showed a one-third decrease in the masseter muscle activity in obstructed animals compared to controls. Electric stimuli were applied to the masseter and temporal muscles to increase the frequency and duration of chewing to five to ten times greater than normal. The cephalometric superimposition tracing showed intrusion of the upper and lower molars, as well as the forward and upward rotation of the mandible.

In summary, acute and chronic stimulation of the masseter and temporalis muscle in the rat and primate has been produced. Electrical stimulation has also been applied to humans to determine muscular reflexes. However, the parameters of chronic electrical stimulation of the muscles of mastication in the human have not been delineated.

Muscle Adaptation

The largest single tissue in the body, skeletal muscle, comprises 40% of mammalian body weight. In addition to its mass skeletal muscle also has the capacity to influence other organs or tissues (Close, 1972). Functional demands imposed on skeletal muscle fibers are capable of inducing adaptive changes. For example, orthognathic surgery changes the position of bone and induces stress on the muscles of mastication. These muscles adapt to the new skeletal positions to maintain the desired alterations.

Tissue, organs, and cells have an intrinsic capability of adaptation to environmental change (Barbashova, 1964; Bajusz, 1969). The rate and magnitude of change determines the adaptive response induced (Brooks, 1969; Prosser ,1964). Adaptations can be defined as a change in the function or structure of an organism which favors its survival in an altered environment (Leake, 1964). Continuous change to better meet environmental requirements or maintain homeostasis is defined as adaptation.

Muscular adaptation can affect the neuromuscular junction, muscle contractile proteins and metabolic capabilities.

The Neuromuscular Junction

The physiologic properties of slow and fast facial muscles have been shown to be influenced by nervous impulse activity. For example, Barany and Close (1971) clearly demonstrated the transformation of myosin in cross innervated rat muscles. They found the transformation taking place in both directions, that is, myosin of cross innervated extensor digitorum longus is very similar to that of normal soleus muscle, and myosin of the cross innervated soleus muscle resembles that of normal extensor digitorum longus muscles. Salmons and Sreter (1976) subjected the extensor digitorum longus (EDL) of rabbits to a continuous low frequency discharge (25 Hz). The results indicated that the stimulated EDL contracts even more slowly than a classical slow muscle in the same animal. The stimulated muscles also appeared to be more fatigue resistant. They also cross innervated the soleus muscle with the extensor digitorum longus muscle and then subjected the cross innervated soleus muscle to a continuous low frequency discharge of 10 Hz tetani. They found that the cross innervated and stimulated soleus muscle contracts and relaxes even more slowly than the control soleus muscle. In summary, they concluded that a change in pattern of impulse activity unaccompanied by a change in the motor innervation can produce effects equalling or surpassing those brought about by nerve cross union.

Muscle Contractile Proteins

Fast and slow twitch motor units contain different properties of myosin which develop during the late fetal period or early postnatal period of life (Perry, 1970)

Myosin purified from fast twitch muscles exhibits both structural and enzymatic differences from the myosin of slow twitch muscles Barany et al. (1965) analyzed the properties of myosin prepared from fast (extensor digitorum longus and medial head of gastrocnemius) and slow (soleus and currells) muscles of the rabbit. They concluded that the myosin of slow muscles of the rabbit differs significantly from that of fast muscles. Fast twitch muscles also differ from slow twitch muscle in ATPase activity. Eccles et al. (1958) systematically investigated the intracellular action potentials of over 260 motorneurons belonging to 20 different species of muscles. They found considerable variability both within a species and between species, in the time necessary for transition from fetal to adult myosin. Those motor units subjected to low sustained frequency activation develop slow twitch characteristics, whereas motor units developing fast twitch characteristics are activated intermittently, but with a high frequency.

The effect of aging and endurance training on the oxidative capacities and the contractile machinery of the plantaris, soleus and psoas muscles was investigated by Maxwell et al. (1973). During the first two weeks after birth, all plantaris fibers have high succinic dehydrogenase (SDH) activity. The percentage of fibers with high SDH activity decrease with age until by 14 weeks of age 53% of the plantaris fibers are classified as low-oxidative fast . In guinea pigs of all ages all soleus fibers have high SDH activity. At one to two weeks of age, the soleus is composed of both high oxidative fast and high oxidative slow fibers, but by 14 weeks of age all fibers are slow. In the plantaris muscle the 8 week program of endurance training resulted in an increased proportion of high oxidative fast fibers and a decreased proportion of low oxidative fast fibers. In the soleus muscle, endurance training produced no changes in

composition or total number of fibers present. The soleus muscle responded to endurance training with hypertrophy of individual skeletal muscle fibers and of the total muscle.

Transitional muscle fibers have different levels of myofibrillar ATPase activity from those of fast twitch or slow twitch fibers. Ringqvist (1974) observed intermediate levels of myofibrillar ATPase activity in masseter muscles of humans.

Contractile Properties

Changes in the contractile properties of muscle are evidenced by changes in the following five parameters: 1) the time to reach peak twitch tension; 2) half relaxation time following a twitch; 3) maximum velocity of shortening; 4) the half relaxation time from maximum tetanus; and 5) the frequency of stimulation necessary to produce maximum efforts (Faulkner et al., 1978). Low frequency stimulation of skeletal muscle by implanted electrodes resulted in adaptive changes of their contractile properties (Salmons and Sreter, 1976). For example, fast muscles which receive sustained low frequency stimulation (10Hz) have a longer time to reach peak tension than do the slow twitch soleus muscles.

The extensor digitorum longus muscle has a high percentage of fast twitch fibers. whereas the masseter muscle has a high percentage of slow twitch fibers (Faulkner et al., 1978). The twitch characteristics are due to different myofibrillar ATPase levels. There are no data on adaptation of the contractile properties of the muscles of mastication to experimental procedures. However, Maxwell et al. (1978) demonstrated that compared to controls, the masseter muscles of edentulous monkeys have higher percentages of fast twitch fibers, smaller mean fiber area and lower succinate oxidase activity. Based on these characteristics, one would predict increased velocity of

shortening, decreased development of absolute maximum tetanus tension and increased fatigability compared to slow twitch fibers.

Metabolic Capabilities

The cytoplasmic and mitochondrial protein content determine the type of physical work for which a muscle is best suited. The biochemical properties of limb skeletal muscles adapt to changes in habitual frequency or type of usage. Adaptations which have been documented in response to high repetition-low tension endurance activities are most relevant to the muscles of mastication.

Holloszy and Booth (1976) dealt with the hindlimb muscles of rodents compared to endurance running on a treadmill. Overall, the skeletal muscles respond by becoming more like heart muscle on its biochemical profile and uses predominantly oxidative pathways to meet its energy needs. MacDougall (1977) studied nine healthy subjects under control conditions and following 5 months of heavy resistance training and 5 weeks of immobilization in elbow casts. Biochemical assays of adenine triphosphate, adenosine diphosphate, creatine, creatine phosphate, and glycogen concentrations were performed. Training resulted in significant increases in resting concentrations of muscle creatine, creatine phosphate, adenosine triphosphate and glycogen. Sargeant et al. (1977) demonstrated that muscles in disuse or immobilized limbs of humans show a decrease in maximum oxygen consumption.

Maxwell et al. (1978) state that the masseter muscle of a normal Rhesus monkey has a high oxidative capacity in comparison to the limb muscles and can be considered physically trained. In other words, the masseter muscle is used more frequently than any individual limb muscle. The high levels of succinate oxidase activity and succinic acid dehydrogenase activity, demonstrated histochemically, indicate the high oxidative

capacity of the masseter muscle (Faulkner et al., 1978). Due to the high oxidative capacity and predominantly slow twitch characteristics the muscles of mastication in monkeys with normal dentition are extremely resistant to fatigue. The masseter muscle of the monkey shows no decline of tension with a tetanic stimulating pattern which would result in a 50% decrease in tension development of monkey limb muscle with 3 minutes.

Faulkner et al. (1978) state that muscle inactivity will result in a significant loss in oxidative capacity, as data from muscles of edentulous monkeys demonstrate. Electromyographic studies of edentulous monkeys indicate a significant decrease in recruitment of the masseter. McNamara et al. (1978) used an appliance to open the bite of monkeys 15 mm and at the same time, detached the masseter muscle at its insertion. This experimental procedure resulted in a 50% decrease in succinate oxidase activity of the whole muscle homogenates in both the masseter and temporalis muscles.

In general, any intervention that reduces the habitual level of activity of the muscles of mastication results in a decrease in oxidative capacity. A restoration of muscle recruitment results in an increase in oxidative capacity and a gradual return to the control values. These observations emphasize the need for exercise rehabilitation programs for the muscles of mastication as an adjunct to clinical procedures (Faulkner et al., 1978).

Muscle Morphology and Physiology

Skeletal muscle, comprising the muscles of the limbs, trunk, neck, and head, differs in its properties from cardiac and smooth muscle in being more dependent upon neuronal control. Each skeletal muscle is comprised of numerous cells or fibers. Within each muscle cell or fiber are contractile proteins capable of developing tension or

force at the attachment sites of the fiber. Muscle fibers are 10 to 100 microns in diameter and up to 30 centimeters in length. Each fiber is surrounded by endomysium, a loose connective tissue sheath. Small diameter blood vessels and capillary beds are intertwined in the endomysium. Varying numbers of fibers are grouped into bundles or fascicles which are delineated by a dense connective tissue called perimysium. Finally, the fascicles of a given muscle are ensheathed in the epimysium, which defines the extent of the muscle. The ends of a muscle are attached to bone by extended lengths of collagen bundles arising from the perimysium and epimysium (tendons). Tendons act to transmit the force of muscle contraction to the attached bones.

Each muscle fiber is composed of hundreds to thousands of myofibrils, which are cylinders extending along the long axis of the fiber. The myofibrils are about 1 to 2 microns in diameter and comprise about 80% of total muscle mass. Each myofibril is composed of a series of repeating segments called sarcomeres. The sarcomere is the basic functional unit of skeletal muscle.

Z lines separate adjoining sarcomeres such that two successive Z lines defines the limit of one sarcomere (Huxley, 1957; 1965). The Z lines run transverse to the longitudinal axis of the myofibrils and serve as attachment points for thin filaments of the two adjoining sarcomeres. The thin filaments are composed of actin and are one half of the contractile apparatus. The second half being the thick filaments which are composed of myosin.

The contractile properties of muscle fibers are generally associated with the arrangement of overlapping filaments of actin and myosin. Studies of the length-tension relationship of single fibers using electron microscopy led to the sliding filament model for the generation of tension (Huxley, 1965; Gordon et al., 1966).

According to this model, tension developed during a stimulus is a function of the number of bridges that may be formed along certain zones of overlapping actin and myosin filaments. However, several points about this model need amplification. First, differences in fiber length, mean cross sectional area and fiber arrangement may markedly affect the development of tension in the entire muscle (Gans and Bock ,1965; Close, 1972; Burke and Tsairis, 1973; Maxwell et al., 1977). Second, if the fiber is allowed to shorten (contract) during stimulation, the tension will drop, depending upon the velocity of shortening. This produces the characteristic force-velocity relationship in which the tension generated approaches zero from the isometric values as the velocity of shortening increases (Edman and Hwang, 1978). Third, these tension values are isometric, meaning that the values derived are for fibers that are maintained at a fixed length while they are being stimulated.

The interaction between actin and myosin proteins results in skeletal muscle contraction. This interaction occurs very often and requires a continuous supply of adenosine triphosphate (ATP). Hydrolysis of ATP by the myofibrillar ATPase enzyme system and rate of release and uptake of calcium from the sarcoplasmic reticulum determines the rate and contraction and relaxation of muscle fibers. Skeletal muscle fibers that experience faster contraction and relaxation times have a high activity of myofibrillar ATPase and rapid calcium movements (Barany, 1967: Brody, 1976). If the rate of muscle contraction is low, most of the steady rate energy flow occurs via the oxidative pathway. The concentration of creatine phosphate and glycogen decrease initially and then remain fairly constant.

The concentration of ATP changes little. If the contraction exceeds the rate and intensity that can be maintained by oxidative phosphorylation, then glycogen and

creatine phosphate will continue to contribute to energy flow until depletion. Depletion of muscle glycogen is one correlate of muscle fatigue (Bergstrom et al., 1967).

The glycolytic pathway is activated rapidly and has the enzymatic capacity for rapid production of ATP. Pyruvate, a product of glycolysis, will enter subsequent oxidative pathways competitively with metabolites principally from free fatty acid metabolism. At sub-maximum intensities of contraction, the pyruvate from glycolysis readily enters the oxidative pathway. As the rate of contractions increases and metabolic demands approach the maximum which can be maintained by oxidative phosphorylation, greater amounts of pyruvate are produced that can immediately enter the oxidative pathway. Under these circumstances, excess pyruvate is reduced to lactate and the concentration of lactate in muscle fibers and plasma increases. Free fatty acids are a preferred substitute for the oxidative pathway and contribute greatly to energy for continued contractions of skeletal muscle fibers at sub-maximum intensities (Havel, 1970; Wahren et al., 1975).

The craniomandibular muscles are innervated by the motor nerve fibers with cell bodies situated within the motor trigeminal nucleus. Motor nerve fibers innervate skeletal muscle fibers through a neuromuscular junction so that the neural activities translate into the release of a chemical transmitter, which modifies the post synaptic membrane of the muscle fiber.

The masseter muscle has been divided anatomically into two or three overlapping and only partially separated layers. It originates from the zygomatic arch and extends caudally to attach to the lower mandible on its lateral side. DuBrul (1954) suggested that the masseter has two layers, with a superficial layer arising from the lower border of the maxilla and the zygomatic arch, and a deep layer attached to the entire deep and

medial surface of the zygomatic arch. Both superficial and deep layers fuse to attach to the lateral surface of the ramus and gonial angle of the mandible. The deep layer sends a fibrous attachment to the temporomandibular disk. The two layers seem to be most separate in their posterior region near the temporomandibular joint in which the deep portion of the masseter is directed almost vertically, while the fibers of the superficial layer are directed more diagonally. Orientation of the fibers suggests that the masseter muscle is involved in the elevation, ipsilateral movements and protrusion of the mandible.

Effect of Nervous System on Muscle Contraction

"The nervous system grades muscle contraction in two ways: (1) Through recruitment it increases the number of active motor units and thereby increases the force of contraction. (2) Through rate coding it increases the frequency of activation of individual motor neurons. When the motor neurons fire faster, they increase muscle tension.

Recruitment

"When motor neurons are activated by a synaptic drive, they are recruited in an orderly fashion according to their size. Neurons with the smallest cell bodies have the lowest threshold for synaptic activation and are recruited by the weakest afferent input. As the afferent input increases in strength, it recruits progressively larger motor neurons. Because larger motor neurons innervate fast muscle fibers which develop greater tension, each motor unit that is recruited adds a larger increment in force than the last one. This stereotypical recruitment order is known as the size principle and applies equally to reflex activation and to voluntary contraction.

Rate Coding

"The second way the nervous system can command greater muscular force is by increasing the firing rate of motor neurons. When muscles are activated by successive action potentials at intervals that are less than successive action potentials at intervals that are less than the time it takes for the muscle to contract and relax again (called muscle twitch time), the forces generated by each impulse summate until a plateau is reached. This state of maintained contraction is called tetanus.

"The rate of stimulation of a muscle nerve affects the tension produced isometrically in the muscle.

"At low intermediate rates some relaxation can take place between impulses and the tension record shows ripples corresponding to the peaks of each twitch. This is called unfused tetanus. At very high frequencies a smooth tension record is observed. This is called fused tetanus. Complete fusion, however, takes place only with the most rapid and forceful natural muscle contractions. Movements requiring less than maximal contraction have smoother trajectories because motor units are always activated asynchronously. When one motor unit is at the peak of its twitch, others are relaxed.

"The force developed by a motor unit also depends on the unit's pattern of activity. Insertion of one extra action potential at the onset of a relatively low frequency stimulus train produces a remarkable enhancement of the tension output. The tension produced when the pair of spikes occurs close together (called doublet) is much greater and develops much more rapidly than the sum of the tensions that would be produced by either spike alone. Moreover, the affect of the second impulse can last many seconds. This mechanism for increasing both the tension in a muscle and the rate at which the tension increases is thought to be used for producing rapid movement. Recruitment and rate coding are not mutually exclusive strategies for grading muscular force." (Carew and Chez, 1985).

Muscle Fiber Characteristics

The function of entire skeletal muscles can best be approached by an understanding of the types of skeletal fibers. Histochemically, the muscle fibers of the craniomandibular muscles represent a range that can be grouped into three major categories. Ranvier (1874) noted differences in the microscopic properties of individual skeletal muscle fibers. Each craniomandibular muscle has different proportions of these muscle fiber types and their properties can change with age, gender and level of muscle activity. Classification of the muscle fiber depends upon the biochemical composition which is revealed by using different stains. Fatigue and contraction speed characteristics of muscle fibers have been revealed by staining with two types of enzymes. The contractile enzyme, myofibrillar adenosine triphosphate (ATPase) is emphasized to determine contraction speed (Ringqvist et al., 1982). To determine muscle fatigue, mitochondrial enzymes, including succinic acid dehydrogenase and nicotinamide adenine dinucleotide tetra-zolium reductase (NADH-

TR), are utilized (Ringqvist, 1974; Eriksson et al., 1981). Although many classifications for skeletal muscle fibers have been proposed, the most useful is the functional classification described by Burke and co-workers (1971) which is based on data from the medial gastrocnemius muscles of cats. The three major categories are slow twitch fatigue resistant, fast twitch fatigue resistant and fast twitch fatiguable. Ringqvist (1974) stated that the muscles of mastication are composed of various combinations of these fiber types. Most investigators designate craniomandibular muscle fibers by their level of staining for the enzyme, contractile or myofibrillar ATPase. Type I fibers exhibit low activity and light staining for myofibrillar ATPase with an alkaline preincubation. Type II fibers stain darkly with alkaline ATPase, while IM fibers stain intermediately between these two ranges.

Muscle fibers can be further subdivided if they are pre-incubated before staining in two different acid mediums. Type I fibers stain weakly with alkaline ATPase and strongly for NADH-TR; Type IIA fibers stain strongly for both alkaline ATPase and NADH-TR; and type IIB fibers stain strongly for alkaline ATPase and weakly for NADH-TR.

Eriksson and Thornell (1983) showed that the temporalis, masseter and medial pterygoid muscles of the human differ in proportion of fiber types. The masseter muscle is composed mainly of type I fibers in three of the four major regions. However, the posterior superficial region of the masseter muscle contains an equal amount of type I and type IIB fibers. They also related the fiber types to the cross sectional areas of the muscle fiber to determine the proportion of the overall muscle cross sectional area occupied by a specific fiber type. The analysis indicated that all three jaw closing muscles have a majority of their cross sectional area occupied by type

I fibers with the masseter having the highest proportion of type I fibers in the superficial and deep anterior regions.

Edgerton (1978) demonstrated that skeletal muscle fibers are functionally associated with motor units. An alpha-motoneuron and the skeletal muscle fibers it innervates make up a motor unit. According to Burke et al. (1973), the skeletal fibers within a motor unit are of similar histochemical profile and presumably of similar contractile properties. The number of muscle fibers per motor unit varies greatly and, therefore, provides for varying degrees of neural control of muscle activity. Granit et al. (1957) and Henneman et al. (1965) concluded that the size of the motor unit is inversely related to the frequency of recruitment. In humans, slow twitch motor units appear to be recruited first, both for reflex (Buchthal and Schmalbrach, 1970) and voluntary movements (Milner-Brown et al., 1973). The contractile response of whole skeletal muscles represents an average response of the individual fiber types making up that muscle.

The influence of nervous impulse activity or the physiologic properties of slow and fast twitch muscles are demonstrated by cross-innervation experiments in which physiologic properties are largely reversed (Barany and Close, 1971; Salmons and Sreter, 1976). However, the degree of adaptation has not been determined.

Muscle Tension as Related to EMG

Manns et al. (1979) and Haraldson et al. (1985) state that the total tension developed by the muscle is directly related to the EMG signal when alteration in the length of the muscle does not occur (i.e., isometric tension). The length of the muscle, the velocity of shortening, and the number and frequency of motor units recruited influences the force developed by a craniomandibular muscle.

Guelinckx et al. (1986) studied the tension of the superficial temporalis in both normal rabbits and in rabbits in which the masseter muscle had been removed. Direct electrical stimulation was applied to the temporalis muscle to determine how tension was developed. The superficial temporalis was detached near the zygoma and attached to a transducer in order to record twitch tensions. Incisal bite force was also evaluated, using a bite force transducer following bilateral stimulation of the superficial temporalis and medial pterygoid muscle. Over an 8 week experimental period, the bite force steadily increased in the developing animal with an average of 34.3 N at 10 weeks and 72.5 N at 16 weeks.

Bite forces were also evaluated in 6 young rabbits over an 8 week period following bilateral removal of the masseter muscles. The total bite force developed by stimulating the bilateral temporalis and medial pterygoid muscles decreased by approximately 60% with bilateral loss of the masseter muscles.

The twitch tension of the temporalis was also evaluated for fatigue following 3 Hz of repeated stimulation over five minutes. The superficial temporalis of the experimental animals developed a significantly lower peak twitch tension, suggesting some muscle fatigue. In animals without masseter muscles, histochemical studies revealed changes in the superficial temporalis with an increase in the area of type IIA fibers. Following removal of the masseter the cross sectional area of the superficial temporalis muscle did not increase. However, a large proportion of the temporalis muscle contained fatigue resistant fibers with high oxidative capacities.

Evoked Potentials with Central Stimulation

From electrical stimulation of the neural pathway, activity during muscle recruitment can be obtained electromyographically. Evoked potentials can be elicited by

stimulating the peripheral nerve innervating the muscle, as is often done in human studies of the masseteric reflex, or under experimental conditions in which the trigeminal pathway is stimulated (Miller et al., 1989).

Evoked potentials within the jaw closing muscles and within the lateral pterygoid were assessed following electrical stimulation of the brainstem trigeminal pathway on the region of the mesencephalic sensory nucleus and with the region of the motor trigeminal nucleus. All four unilateral muscles are recruited when the area around the mesencephalic nucleus is stimulated. Stimulation around the motor trigeminal nucleus induced potentials in one muscle before stimulating other muscles. Before muscle contraction is visually detected and vertical raising of the mandible is observed, the lowest electrical currents can induce potentials within the temporalis, masseter, medial and lateral pterygoid muscles. An interference pattern is elicited when the stimulus frequency is raised above 100 Hz. Maximum clenching also elicits an interference pattern similar to patterns resulting from high frequency stimulation. Central stimulation of the mesencephalic and motor trigeminal nuclei usually elicits evoked potentials in several muscles during the raising of the mandible, but direct stimulation of the temporalis muscle is sufficient to raise the mandible to occlusal contact.

Masseter Muscle Function

Surface electromyography has been used extensively to provide information on the two superficial masticatory muscles, the temporalis and masseter. Alternatively, the application of intramuscular electrodes has provided insight into the function of each of the jaw-closing and jaw-opening muscles.

Gibbs et al. (1984) studied the superficial masseter of the human using surface electrodes. Maximum clenching in centric relation with full occlusal contact when the

second molars were separated by 1.5 to 1.0 mm elicited maximum EMG activity. Near maximum activity of the masseter was induced while clenching on the canines. Low masseter recruitment resulted only when clenching in the most retruded position.

Belser and Hannam (1986) completed an EMG study of the superficial and deep layers of the masseter muscle using both surface and intramuscular fine wires. The greatest dissimilarity in use of the two layers was produced when clenching in the intercuspal position with a retrusive intent. The superficial layer was minimally active at 5.5% of the maximum activity. In contrast, the deep layer exhibited EMG activity around 50% of the maximum activity. The side chosen for chewing also affected the superficial layer of the masseter. The superficial layer was highly active with ipsilateral chewing (78% of the maximum EMG activity) and minimally active with contralateral chewing (25% of the maximum EMG activity). In contrast, the deep layer was active at the higher level during contralateral chewing registering 50% of the maximum EMG activity.

The recruitment pattern on 20 adult subjects for 16 responses using electromyography was studied by Nielsen and Miller (1988). The highest intensity of the masseter muscle was evident in intercuspal chewing and mastication. However, it was also used with high probability (78%) in any clenching or masticatory response regardless of the position of the contacts. The masseter was used with less probability and intensity during protrusion and fast mandibular closure (60%). During retrusion, laterotrusion and mandibular opening the masseter was used with the least probability (<50%).

Pancherz (1980) studied the effect of maturation on the use of the masseter muscle between males of 10 to 13 years of age and males of 23 to 33 years of age. The EMG

activity of the masseter was greater during both clenching and mastication with the adult group, while the temporalis muscle did not change its amplitude of recruitment with age. The adult males had a full complement of teeth while the boys were in the mixed dentition. Ahlgren (1966) and Ahlgren et al. (1973) suggest that the transition of the dentition might significantly alter the recruitment of the primary force developing muscle, the masseter.

Masseteric Reflex

A rich supply of sensory fibers are located in the craniomandibular muscles, oral muscles, temporomandibular joint, periodontal ligament and tongue muscles. The muscle spindle of the jaw closing muscles respond to changes in length. The muscle spindle can be characterized as a large myelinated sensory fiber. Delicate touch or pressure are detected by the medium size sensory fibers. These fibers can be divided into those that respond to a constant stimulus and those that are excited only when the stimulus is initially placed or removed. Tissue damage and changes in temperature excite the smallest myelinated nerve fibers. The smallest of the sensory fibers are unmyelinated fibers and include some that respond to mechanical stimuli, a few that respond to changes in temperature and many which are excited by chemicals that induce the sensation of pain.

The stretch reflex of the masseter muscle can be elicited by direct electrical stimulation of the masseteric nerve. Godaux and Desmedt (1975) studied the electromyographic activity of the masseter muscle following direct electrical stimulation. The stimulating electrodes were placed through the sigmoid notch to a depth of about 20 mm in 17 normal subjects. A short latent evoked potential within the muscle (i.e., masseteric reflex), was observed following direct stimulation of the motor

fibers innervating the masseter muscle. A larger latent evoked potential within the muscle was also evident. This larger evoked potential is due to excitation of sensory fibers which synaptically excite the motor neurons of the masseter to excite muscle fibers (i.e., H reflex).

The level of stimulation to the masseteric nerve determined which potential appeared. The lowest stimulus used produced the longer latent H reflex potential. Electrical currents excite the sensory fibers, inducing the H reflex because sensory fibers have larger diameters compared to motor fibers in the masseteric nerve. This size differential results in different thresholds for the two induced potentials.

Clenching

Gibbs et al. (1984) demonstrated electromyographically that anterior and middle temporalis and the superficial and deep masseter muscles develop their maximum EMG activity when the subject clenches in the intercuspal position. The medial pterygoid and superior head of the lateral pterygoid and digastric muscles are also recruited , but only a low level of EMG activity.

Increasing the vertical dimension between occlusion using a splint does not alter this co-activation potential for the EMG intensity of these five muscles. Belser and Hannam (1986) have demonstrated that changing the direction of force development, while maintaining the same intercuspal position, changes the use of the muscle. Clenching in the intercuspal position with an anteriorly directed effort decreases the activity of the anterior temporalis and eliminates posterior temporalis activity completely. The superficial masseter muscle remains at maximum intensity, while the deep masseter decreases in intensity. Clenching in the intercuspal position, but with a posteriorly directed effort, shifts the recruitment of the temporalis muscle with the

posterior temporalis having its maximum activity. The deep masseter maintains its maximum level of EMG activity, but the superficial masseter, medial pterygoid, and inferior head of the lateral pterygoid muscles cease discharging.

Relation between Muscle Activity and Bite Force

Haraldson et al. (1985) demonstrated the relationship between myoelectric activity and bite force in the muscles of mastication. Myoelectric activity in the anterior and posterior temporalis and in the masseter muscle was assessed in five subjects, biting unilaterally on a bite fork at 5, 100 and 200 N for 60 seconds. EMG activity for all muscles tested increased with increasing bite force. The temporalis muscle was the only muscle that demonstrated linearity. An endurance test was also performed in which four of the five subjects exerted maximum bite force on the right side for as long as possible. The EMG activity of the right masseter muscle was closely related to bite force in contrast to the other tested muscles.

Kawazoe et al. (1979) and Rasmussen and Moller (1975) demonstrated linearity between myoelectrical activity and tension in the muscles of mastication. Furthermore, Ahlgren and Owall (1970) proposed that integrated EMG can be used as a reliable index to both chewing and biting forces.

Haraldson and Ingervall (1979) state that when data from "mechanical" bite force studies are compared with those of EMG studies, the suggested linearity between bite force and EMG activity is not always evident. Christensen (1981) states that muscular fatigue in prolonged biting is a complicating factor. Merton (1954) observed that force or tension and EMG activity decreased linearly at maximum muscular effort.

Lindstrom et al. (1977) and Lindstrom and Hellsing (1983) state that the development of muscular fatigue correlate well with the myoelectric power spectrum of EMG activity registered during isometric contractions.

Relation between Facial Morphology and Activity of the Muscles of Mastication

Indirect measurements of the muscle force developed by the jaw-closing muscles have been determined by measuring forces between the occlusion. Profitt and Fields (1983) and Profitt et al. (1983) used piezo-electric transducers based on quartz crystals which require a thicker casing. An alternative piezo-electric material is polyvinylidine fluoride foil which is available in sheets which are only 30 microns thick. Using both quartz and foil based piezo-electric force transducers, occlusal forces during swallowing, simulated chewing and maximum biting effort were evaluated in 19 long-face and 21 normal adults. Forces were measured at 2.5 and 6.0 mm molar separation. They concluded that long-face individuals have significantly less occlusal force during maximum effort, simulated chewing and swallowing than do individuals with normal vertical facial dimensions. No differences in forces between 2.5 and 6.0 mm jaw separation were obtained for either group.

Profitt and Fields (1983) also studied occlusal forces in children aged from 6 to 11 years, during swallowing, simulated chewing, and hard biting. Forces in the normal- and long-face children are similar to those in long-face adults. It appears that individuals with the long-face pattern fail to gain strength normally in the mandibular elevator muscles.

A difference in body build and general muscle strength certainly can contribute to occlusal force differences. Another explanation for the differences in bite force is the mechanical advantage of the lever system. Throckmorton et al. (1980) demonstrated that there is a greater mechanical advantage for the elevator muscles of the mandible if the ramus of the mandible is upright and the gonial angle is relatively acute. As the gonial angle increases, the mechanical advantage of the muscles decrease.

The forces of occlusion may also serve as a direct control mechanism for eruption of the teeth (Profitt et al., 1983). A common finding in long-face individuals is excessive eruption of posterior teeth, particularly the maxillary molars. As the long-face individuals grow, there is often a tendency for the mandibular plane angle to increase as the mandible rotates downward and backward. Extremes of this pattern of growth can be seen in syndromes which cause generalized muscular weakness, implying that relative muscular weakness may precede and contribute to the long face pattern.

Ingervall and Thilander (1974) studied the activity in the anterior and posterior parts of the temporalis muscle, the masseter muscle, and upper lip in 52 children. The activity was recorded bilaterally with the mandible in postural position and during chewing, swallowing and maximal bite. The average mean voltage amplitude in the temporalis and masseter muscles was greatest during maximal bite, smaller during chewing and smallest during swallowing. The highest correlations between muscle activity and facial morphology were found during chewing and maximal bite. During these functions the amplitudes in the temporal and masseter muscles were large in cases with a tendency to parallelism between the jaw bases and between the mandibular occlusal line and the mandibular line.

The relationship between muscle function and craniofacial morphology was studied in the patients with myotonic dystrophy (Kiliaridis et al., 1989). The dental arches and the occlusal traits were analyzed on dental casts. Lateral cephalograms were taken in the patients with myotonic dystrophy to study the craniofacial morphology. It was found that patients suffering from myotonic dystrophy had weak masticatory muscles which might be caused by the disease. A high prevalence of malocclusions

characterized by anterior open bites and lateral cross bites was found among these patients. Their craniofacial morphology showed a vertical aberration, characterized by a large angle between the mandibular and palatal planes and a steep mandibular plane. These findings were most pronounced in patients with early onset of the disease supporting the hypothesis that reduced muscle function may cause changes in the craniofacial morphology. Myotonic dystrophy may be one of the etiological factors in "long face syndrome". Profitt et al. (1968) also presented a case report of an unusual syndrome of generalized muscular weakness and described combined orthodontic-surgical correction of the severe open bite malocclusion with dental crowding and facial asymmetry.

Kreilborg et al. (1978) studied craniofacial growth in a case of congenial muscular dystrophy. The patient was born at term. At the age of 4 years and 9 months she was referred to the Institute of Orthodontics because of severe malocclusion. Here she was followed annually or biannually until the age of 12 years, 8 months. An electromyographic analysis of the action of the temporal, masseter and digastric muscles including maximal activity, chewing and swallowing was performed at the age of 6. The electromyographic recordings at the age of 7 demonstrated almost no activity in the temporal and masseter muscles during natural chewing of an apple. No sign of rhythmic activity was seen in the temporal muscles while the masseter muscles displayed weak bursts of activity. During swallowing, activity in the masseter muscles was negligible. Clinical and roentgen cephalometric findings at 4 years 9 months of age indicated extreme anterior facial height caused by a pronounced backward inclination of the mandible. The intraoral examination revealed a severe skeletal open bite with occlusion limited to the second deciduous molars. At the age of 12 years 9

months the mandible was extremely retrognathic and exhibited a marked backward inclination, leading to excessive anterior face height. The gonial angle was obtuse.

Bakke et al. (1991) concluded that muscle thickness was systematically and significantly correlated with bite force, occlusal tooth contact, anterior facial height, vertical jaw relation, and mandibular inclination. The thickness of the human masseter muscle was measured by ultrasound scanning at three sites 1 cm apart. Thirteen women, ages 21 - 28 years with a minimum of 29 teeth and without craniomandibular disorders comprised the sample. The study showed a connection between measures of masseter thickness and function of the muscle, as well as parameters generally associated with masseter muscle function. Bite force and amplitude of electrical activity were positively correlated to masseter thickness and increase of thickness from contraction. Still, the strongest correlations were found between masseter thickness and facial morphology and masseter thickness and the number of front and total teeth contracting.

Strong activity in the masseter muscles during maximal bite in the intercuspal position is associated with an anteriorly inclined, prognathic mandible with a curved base and a tendency to a large overbite. On the other hand, in normal subjects, weak activity in the masseter muscles is associated with a mandible which is posteriorly inclined and retrognathic. The base of the mandible is flattened and there is a tendency to an anterior open bite (Kreilborg et al., 1978). Ringqvist (1973) states that strong activity in the masseter muscles during swallowing coincides with prognathism and anterior inclination of both jaws.

Muscle Fatigue

Neuromuscular fatigue is defined as a decrease in force generated by the

neuromuscular system during repeated or continuous recruitment (Miller, 1991). Christensen (1981a) describes physiological muscle fatigue as the inability of a skeletal muscle to produce, on either natural or artificial stimulation, a tension that was pre-existing subsequent to dynamic or static contractile activity of the muscle. In other words, a decrease in muscle tension following a fairly short period of sustained stimulation is defined as fatigue.

Muscle fatigue can be elicited experimentally by several paradigms. One paradigm measures the electromyographic signal and force decay during a sustained contraction. Another measures the duration of maintaining a given force level. A third measures the force and electromyographic activity developed during brief voluntary contractions. These voluntary contractions usually occur during sustained clenching on a transducer while a subject maintains some level of muscle contraction (Van Steenberg et al., 1978).

> "Maximum stress maintained during prolonged contraction or during a series of brief tetani elicited by direct stimulation of the motor nerve or muscle can lead to fatigue of motor units. Direct stimulation allows adequate perfusion when the circulation is intact. Tetanic stress decays rapidly to a level that can be maintained for long periods. Failure of the fast motor units leads to this rapid decay. Lactic acid production, creatine phosphate, and glycogen depletion results in the decline of tetanic stress. This implies that ATP depletion leads to a failure of contraction. Slow motor units which can meet the energy demands of the stimulus regimen do not exhibit significant fatigue. Evidently, some factor associated with energy metabolism can inhibit contraction. There is some evidence that neuromuscular fatigue can occur in the largest fast motor units in which the ability of the motor nerve to synthesize and release acetylcholine may be the limiting factor." (Berne and Levy, 1983)

Intolerable pains and total subjective exhaustion of one or more jaw muscles may force a human subject to stop the isometric muscle activity of tooth clenching. Disturbed homeostasis produced by exercise can lead to general physical fatigue. Most persons cease exercise long before there is any muscle fatigue. Lowering of plasma

glucose levels and accumulation of metabolites can lead to discomfort or even pain. Trained athletes are able to tolerate the discomfort and will exercise to the point where some motor unit fatigue occurs. Berne and Levy (1983) state that part of the enhanced performance observed after training involves motivational factors.

Christensen (1981b) had ten adult male subjects exercise maximal voluntary tooth clenching to determine the onset of subjective fatigue, the subjective endurance of fatigue and pain in the masseter muscle. Onset of subjective fatigue occurred on average 21 seconds after onset of muscle hyperactivity and the isometric endurance time of the masseter muscle, during which periods of severe muscle pains were present was on average 82 seconds. The data from electrical muscle activity of the threshold and endurance tests suggested that progressive physiological or peripheral muscle fatigue accompanied the psychological endurance of tooth clenching.

Christensen (1979) studied some subjective experimental parameters in experimental tooth clenching in 14 human subjects. During production of maximal voluntary isometric tension by the elevator jaw muscles fatigue threshold (31 s), the pain threshold (55 s) and the pain tolerance (118 s) of tooth clenching did not vary significantly intra-individually, whereas this was so inter-individually. On a command signal, the subject exercised maximal voluntary tooth clenching or maximum voluntary isometric contractions of the elevator jaw muscles and the very moment a subjective sensation of fatigue appeared in the jaw muscles, this was reported by the subject. Christensen follows and describes fatigue as:

> "Initial subjective or psychological muscle fatigue is defined as an initial urge after onset of muscle activity to cease the activity because of a feeling of slight and localized discomfort. There must be a subjective conviction that the muscle activity can be continued voluntarily. An initial sensation of muscle pain (muscle pain threshold of tooth clenching) is defined as the first intense and localized discomfort that urges the subject after onset of muscle activity to cease the muscle

> contractions, although it is possible to continue the activity voluntarily. The subjective muscle pain tolerance of tooth clenching is defined as the moment that severe and localized discomfort, after onset or muscle activity forces the subject to stop the muscle activity because of subjective sensations of intolerable pain and possibly exhaustion of muscle."

Christensen (1980) studied the electrical activity in the right masseter during different isometric exercises. A consistent decrease in muscle activity from onset of fatigue to onset of pain to the endurance of pain was witnessed, possibly because of progressive jaw muscle fatigue.

In man, a few minutes following exhaustive muscle fatigue recovery of force of skeletal muscle is almost complete. However, a subjective feeling of weakness and some inability to produce voluntarily a pre-existing level of tension prevails (Edwards et al., 1977). Central muscle fatigue or psychological fatigue may also be a cause of peripheral muscle fatigue. It is conceivable that physiological fatigue is the central result of at least two peripheral phenomena. The first, Wedinsky inhibition is the result of a transmission block at the neuromuscular junction. The second is repetitive alpha motor firing, and/or a depletion of the muscle energy stores. Together these events partially or totally prevent the skeletal muscle from contracting (Gollnick et al., 1974; Davson and Segal, 1975). Psychological muscle fatigue precedes that of physiological fatigue in that artificial stimulation of a skeletal muscle results in contractile activity despite the presence of a subjective sensation of extreme fatigue or exhaustion of the muscle (Ikai, Yabe and Ischii, 1967; Ikai and Yabe, 1969).

Davson and Segal (1975) and Christensen (1981a) concluded that the purpose of the subjective sensation of muscle fatigue might be that of warning the skeletal muscle against being overloaded. While the purpose of progressive physiological muscle fatigue might be that of protecting a skeletal muscle against excessive tissue injury.

For example, necrosis can be restored by restoring to a greater or lesser extent the flow of blood in the muscle. Increase in blood flow in turn allows initial payment of the oxygen debt incurred during the period of heavy and prolonged contractile activity (Petersen and Christensen, 1973; Christensen, 1979; 1981b).

Van Steenberghe et al. (1978) studied the resistance of jaw-closing muscle to fatigue during repetitive maximal voluntary tooth clenching in three male volunteers. A gnathodynamometer was used to record all bite forces. The volunteers were examined for the forces they could develop during successive maximal voluntary contractions of their jaw muscles, their arm flexor muscles and their handgrip muscles. A typical decrease in the developed force during successive isometric contraction was noted. In contrast, clenching at least eight teeth on each side produced nearly constant force. The increased resistance to fatigue of jaw muscles could be due to higher oxygenation of the muscle or to differences in slow twitch/fast twitch fiber composition.

Most of the physiologic studies on muscle fatigue have been done on the limb muscles. Two different effects of muscle fatigue at sub-maximal isometric force have been demonstrated by DeLuca (1985) and Maton (1991). One condition is associated with local conditions and the other is associated with central motor control. Lindstrom et al. (1970) and Lindstrom and Petersen (1983) described fatigue due to central motor control as an increased recruitment of additional motor units. Local conditions are evidenced as a reduction in the mean or median frequency of the power density spectrum of the electromyographic signal. An increase in the r.m.s. (root mean square) power of the signal reflects a change in central motor controls.

During fatiguing contractions, so many motor units are recruited that the observed signal in general shows an interference having the character of random noise. Power

spectrum analysis is a method for noise analysis. This is a useful tool as it has been shown that the general shape of the spectrum varies little with the contraction level (Lindstrom, Magnusson, and Petersen, 1974). However, the spectrum varies strongly with the development of muscle fatigue (Kadevors, Kaiser, and Petersen, 1968). Kaiser and Petersen (1962) demonstrated a high frequency power decrease and a low frequency power increase during fatiguing contractions. The shift of the spectral curve along the frequency axis towards lower frequencies showed that this was caused by a decrease in conduction velocity of the action potentials of the muscle fibers (Mortimer, Magnusson, and Petersen, 1970: Lindstrom, Magnusson, and Petersen, 1970).

Palla and Ash (1981) studied muscle fatigue in the anterior temporal and masseter muscle of 13 normal subjects following maximum clenching in the intercuspal position. The power spectra obtained during fatigue was statistically significantly shifted to lower frequencies and narrower than those obtained at the beginning of the clench. Studies on limb muscles showed that metabolic changes occurring during fatigue decrease the frequency of the EMG and thus its power spectrum shifts to lower frequencies (Viitasalo and Komi, 1978). This shift is due to a reduction of the conduction velocity of the action potential along the muscle fiber and, therefore, to an increased duration of the motor unit action potential (Lindstrom et al., 1970: Viitasalo and Komi, 1977). During stationary isometric contraction, there is usually a rise in integrated electromyographic activity. The average motor unit potential shows sensitivity to fatigue with an increase in amplitude, rise time, and number of spikes counted. In contrast, the power density function of the electromyogram usually shifts to lower frequencies and synchronization in the motor unit discharges also occurs as a result of fatigue. Lindstrom and Hellsing (1983) studied masseter muscle fatigue in 17 healthy

persons. Under the influence of fatiguing contractions, a gradual shift of the myoelectric spectrum occurred. The rate of change was taken as a measure of the development of fatigue.

Maton et al. (1992) studied the fatigue characteristics in masseter and temporalis muscles. Each subject was instructed to maintain maximal and six levels of sub-maximal incisal bite forces for as long as possible. The decrease in endurance time with an increase in bite force followed a pattern similar to that of limb muscles. Each muscle was characterized by a decrease in the mean power frequency of the power density spectrum. However, they reported variability in the changes observed in root mean square (r.m.s.) power. This finding was inconsistent with changes in r.m.s. power found in the limb muscles. This difference was explained in terms of either changes in motor unit firing rate or the muscle's relative contribution to the generation of bite force.

Clark et al. (1988) recorded surface EMG activity from the temporal and masseter muscles during sustained isometric clenching not only at the maximum force, but at 75, 50 and 25% of that maximum force. The normal subjects could maintain their maximum force on a single transducer for less than one minute and increase that time about 4 times when contracting at a quarter of the maximum force. They indicate that the dominant factor for subjects ceasing a sustained clench resides with intolerable pain. They suggest that the cause of the pain could be the resulting ischemia with decreased blood flow occurring in skeletal muscle when sustaining a contraction. However, the concept that biochemical end-products from the sustained contraction are also effective in stimulating noxious sensory endings within the muscle and around the blood vessels

cannot be disregarded (Barcroft and Miller, 1939; Moller et al., 1979; Richardson, 1981).

The Long Face Syndrome

The growth pattern leading to an increased vertical dimension of the face is commonly referred to as "skeletal open bite", but "long face syndrome" is a more appropriate term because it provides a more complete description of the facial, dental and skeletal features (Opdebeeck and Bell, 1978; Schendel et al., 1976). Schendel et al. (1976) also state that an open bite is not a constant characteristic and does not define the syndrome.

Etiology

Profitt (1978) describes the different factors suspected to influence the vertical dimension of the face.

He divided them as follows:

A. Primary factors in equilibrium

1) Intrinsic forces by the tongue and lips
2) Extrinsic forces. These are habits (thumb sucking, etc.), orthodontic appliances, etc.
3) Forces from dental occlusion

4) .Forces from the periodontal membrane. Eruption forces

B. Secondary factors in equilibrium

These are factors influencing the primary forces. The resulting posture of the head and jaws may influence the amount of pressure exerted on the teeth and jaws. In turn, the position of the head and neck is thought to be influenced by respiration, nasal versus oral.

Characteristics

Muller (1983), Sassouni (1969), and Schendel et al. (1976) described the long face syndrome as having the following characteristics:

- Anterior lower face height is increased, as expressed by the line subnasal soft tissue menton.
- Anterior middle face height and anterior upper face height are unchanged.
- The face appears long and narrow from the frontal view.
- The nose tends to be long and narrow, the nasal apertures are narrow and often there is a bump on the nose.
- Lip incompetence; the lips at rest are not in contact. When the lips are together mentalis strain is evident.
- Interlabial distance is increased and the gingiva is exposed upon smiling.
- Lack of a mental protuberance making the lower profile rather convex.
- Open bite may or may not be present.
- Angle Class I, II or III dental relation may be present. Although a Class II relation is most prevalent (Fish et al., 1978; Epker and Fish, 1972; Subtelny and Sakuda, 1964).
- The palatal vault is high and narrow often leading to a mandibular buccal cross bite (Fish et al., 1978; Epker and Fish, 1977).

Cephalometric Findings

According to the Sassouni analysis (Muller, 1983; Sassouni, 1969) the supra-orbital, palatal, occlusal and mandibular plane are steep to each other. The mandibular plane relative to SN or FH is steep (Cangialosi, 1984; Epker and Fish, 1977; Fish et al., 1978) as well as to the palatal plane (Arvystas, 1977; Nahoum, 1975; Sassouni, 1969). The cranial base angle is obtuse. The gonial angle is obtuse (Cangialosi, 1984; Ellis and McNamara, 1984; Nahoum, 1975). The interincisal angle between the incisors is small (Sassouni, 1969; Sassouni and Nanda, 1964).

Dimensional Deviation

The increased dimension of the anterior total face height with a concomitant decrease in posterior total face height seems to be a constant finding (Cangialosi, 1984; Nahoum, 1975). The anterior lower face height nearly always shows a considerable increase where as the anterior upper face height is reported to be essentially normal.

The anterior cranial base is of normal length (Cangialosi, 1984; Ellis and McNamara, 1984) while the posterior cranial base is shortened. The palatal length in normal (Subtelny and Sakouda, 1964), but the premaxillary area is reported to be tipped up toward the base of the skull or the posterior part of the palate is tipped downward. This would increase the palate mandibular plane angle.

One of the most important features of the long face syndrome is the vertical maxillary excess (Opdebeeck et al., 1978; Schendel et al., 1976; Sassouni and Nanda, 1964). The distance occlusal plane-palatal plane exceeds 19 mm (normal). The occlusal plane-palatal plane was measured as the distance between the superior cortex of the palate perpendicular to the occlusal plane through the mesiobuccal cusp of the first maxillary molar. Epker and Fish (1977), Schendel et al. (1976), Subtelny and

Sakouda (1964), and Sassouni and Nanda (1964) observed not only an increased vertical drift of the maxillary molars, but also the maxillary incisors despite the possible presence of an open bite.

Subtypes

Although the positional and dimensional deviation of all the LFS patients follow the same trend, two patterns can be distinguished according to Opdebeeck (1978) and Schendel et al. (1976). Subtype I is characterized by a rather long ramus and an increased occlusal plane-palatal plane distance. The increased maxillary dentoalveolar height is the dominant feature and, as a consequence, these patients tend to show more gingiva upon smiling. These patients manifest very long faces with a moderately increased SN-MP angle. The palatal plane is usually not angulated. The upper and lower incisors are more upright than in Subtype II.

Subtype II is characterized by having a short ramus. Occlusal plane-palatal plane distances are normal or slightly increased with less gingiva showing upon smiling. The anterior lower face height is increased, but to a lesser extent than in Subtype I. The high value for SN-PP is the dominant feature indicating that the posterior rotation is extreme. The ANS-PNS plane is frequently angulated at the level of the incisive canal, resulting in a reverse curve of the occlusal plane. Open bite is more likely to occur in this subtype.

Children

Most studies of long face syndrome were performed on post pubertal persons. It can be extrapolated that children would show different morphological characteristics. However, Fields and Profitt (1984) in an investigation of a sample of long-faced

children from 6 to 12 years old also found an increase in anterior total face height, mandibular plane angle, gonial angle and palatal plane-mandibular plane angle. In contrast, due to the absence of the pubertal growth spurt, ramus height was not increased.

Functional Electrical Stimulation

Functional electrical stimulation (FES) is a system that refers to more than two dozen different clinical applications in which electricity is applied to the body to produce a specific outcome. The term "functional" is often used in conjunction with "electrical stimulation" because the goal of the stimulation is to restore or improve an individual's ability to function in activities of daily living. Cardiac pacing is an example of FES in which electrical stimulation is used to initiate and control heart muscle contractions. "TENS" (transcutaneous electrical nerve stimulation) is another example which is used by many physicians and therapists to provide relief of chronic pain. All FES systems either mimic or adjust the tiny electrical currents that are naturally generated in the body. These currents are caused throughout the body by certain tissues and nerve fibers which provide muscle (motor and sensory - sight, hearing, touch, etc.) function.

FES systems use devices called "stimulators" to generate electrical current which is transmitted to the body through "electrodes". The electrodes are attached to a particular part of the body, depending on the specific application. The electrodes may be placed on the skin, inserted through the skin, or implanted in the body. The electrodes are connected to the stimulator by thin cables and are placed directly on the skin over the nerves or muscles to be stimulated. All FES systems require some method of controlling the amount of electrical current that is passed through the electrode and into

the body. The stimulator interfaces to a detachable programmer to accept stimulation protocol specifications. The protocol is followed until changed.

Presently, functional electrical stimulation is being used in many applications. These include auditory prostheses, bladder evacuation (incontinence), bone growth acceleration, bowel evacuation, cardiac pacing, cardiomyopathy, cardiovascular exercise, circulatory pooling inhibition, diaphragm (phrenic pacing), electro-ejaculation, epilepsy control, hand grasp and arm movement, joint contracture management, laryngeal pacing, muscle strengthening, neural regeneration, pain control (TENS), postural maintenance, scoliosis correction, spasticity control, spinal cord stimulation, standing and gait, tactile stimulation, visual prosthesis and wound healing.

Disuse atrophy with muscle may occur in paralyzed muscles. If the neurons innervating the muscles (lower motor neurons) are damaged, the process is irreversible and disuse atrophy continues. If, however, the paralysis involves the neurons originating in the central nervous system (upper motor neuron) and the lower motor neurons are intact, electrical stimulation can reverse the muscle atrophy and can be used to produce functional muscle contractions. This stimulation can be applied directly to a motor nerve (Glenn et al., 1977) by means of electrodes placed on or in the muscle. However, activation is still accomplished by stimulation of motor nerve fibers within the muscle (Crago et al., 1974). McNeal and Reswick (1976) point out that, if the electrical pulse is applied slowly the muscle responds in a series of course twitches. Above a certain stimulation frequency (fusion frequency), the response becomes smooth.

Most muscles consist of several muscle fiber types with different dynamic properties. The groups of units activated include a mixture of fast contracting, high

force output, rapidly fatiguing units and slow, low force, fatigue resistant units. This can result in response variability when periods of sustained muscular contraction are desired. If the fast-fatiguing fibers can be converted to fatigue-resistant type fibers, this problem can be overcome. Mortimer and Peckman (1973); Peckman and Mortimer (1977) have shown that conversion is possible with 10 Hz of stimulation. The stimulated muscle fibers of the tibialis anterior muscle were studied histochemically and had altered enzyme staining consistent with conversion of the fiber type when compared with the unstimulated contralateral control muscle.

Peckman and Mortimer (1977) have shown that chronic electrical stimulation in quadriplegic patients can overcome disuse atrophy. The finger flexor muscles were exercised for 2 to 6 hours a day over a period of 7 to 28 weeks at 10 Hz in 10 patients. The strength of the muscles increased 33% to 88% compared with the starting force to a state usable for functional tonic activation.

A technique developed to reduce the effects of fatigue is to place multiple electrodes in a muscle and then to activate them sequentially. Since the rate of fatigue of a muscle is proportional to the frequency of stimulation, the sequential technique permits each groups of fibers stimulated by the different electrodes to be activated at a lower frequency, while the whole muscle maintains a smooth fused contraction. The principal problems with this technique are increased complexity of activated fiber population by the different electrodes and differing dynamic contraction responses of the population. Peckman and Mortimer (1977) state that, if conversion to slow contracting, fatigue resistant fibers proves feasible in paralyzed human muscle, a single electrode system might be preferable.

Electrochemical Considerations

Current flow within metal conductors occurs by movement of electrons, whereas current flow in tissue is by movement of ions. Charge transfer between an electronic conductor and an ionic conductor can occur by oxidation-reduction reactions and by capacitive mechanisms that involve changing and discharging of the so-called electrode double layer (Hambrecht, 1979). Oxidation reduction reactions can be reversible or irreversible. The reversible reactions are those such as oxide formation in which the reaction products are immobilized from the electrode surface. Irreversible reactions generate new potentially toxic chemical species that leave the electrode. To prevent irreversible tissue damage, these reactions should be avoided.

The recommended materials at present are platinum, platinum-iridium alloy and 316 stainless steel. Platinum is listed first because it is the most well known. In addition, the passive tissue response to platinum is considered acceptable for use in both the central nervous system and the peripheral motor system. Platinum-iridium alloys are also acceptable, but show a greater tendency toward surface erosion when subjected to high pulsing currents (Brummer and McHardy, 1977). Stainless steel is commonly used in surgical implants. It is acceptable for electrode use in the peripheral motor system, but insufficient data are available to recommend it for electrode use in the central nervous system (Mortimer, 1981). Stainless steel has been used in intramuscular electrical stimulation because of its practical availability and high strength compared to platinum and its commonly available alloys (Caldwell, 1970; Mortimer and Kaufman, 1980).

Monopolar electrodes are efficient and selective if located close to the nerve entry point, but their performance declines as the distance between the nerve entry point and

the electrode increases. Bipolar intramuscular stimulation is more efficient and selective than monopolar stimulation, but only if the nerve entry point is between the electrodes (Popovic et al., 1991).

Stimulation Parameters

Electrical stimulators offer varying adjustable features including: current amplitude; pulse duration; pulse frequency; total "on" time of the stimulus train; total "off" time between stimulus train; and treatment time.

Current amplitude determines the magnitude of the sensory as well as motor response evoked by stimulation. For most patients, electrical stimulation is a new sensation and requires an accommodation period.

Pulse duration affects the current intensity required to generate a motor response and, therefore, can play a role in the effectiveness and comfort of electrical stimulation. As pulse duration decreases, the amount of charge passed with each pulse decreases. Therefore, the intensity of current required to evoke a particular motor response must be increased. With regard to pulse duration and patient comfort, pulses of 300 microseconds were preferred when compared to pulses of either 50 or 1000 microseconds duration (Gracanin, 1972). Crago et al. (1974) determined that the optimal pulse duration for reducing the possibility of tissue damage was less than or equal to 0.01 milliseconds for intramuscular stimulation in the test situation. Van den Honert and Mortimer (1979) studied tissue damage occurring following a stimulation protocol of 50 Hz, 20 mA with varying pulse durations of 50, 100, 500 and 1000 microseconds in 19 adult cats. Microscopically, the damaged areas stimulated at 50 and 100 microseconds were indistinguishable from the controls. At pulse durations of 500 to 1000 microseconds, an electrode would occasionally fail.

Stimulus frequency affects the quality of muscle contraction and also the rate of muscle fatigue during a stimulation program. For most stimulation programs a frequency sufficient to cause tetanization is desirable, but frequencies markedly higher than this can cause rapid muscle fatigue. Frequencies between 30 and 35 pulses per second are usually sufficient to achieve a tetanized contraction is most muscles of the upper and lower extremities (Benton et al., 1981).

Ferguson et al. (1989) measured the changes in the contractile properties induced by a 30 Hz phasic stimulation paradigm compared with the changes induced by a 10 Hz continuous stimulation paradigm. The study was performed on the tibialis anterior muscle of cats with one paradigm applied to one hindlimb muscle and the other to the contralateral limb. Both hindlimb muscles received the same number of stimuli in a day, making the average stimulation frequency 10 Hz. Two periods of daily stimulation were studied, 8 and 24 hours per day. Muscles stimulated at 30 Hz produced greater overall tetanic tension and during a prolonged stimulation test, exerted a greater mean tension than muscles stimulated at 10 Hz. Muscle mass was least reduced and fewer pathological abnormalities were observed in the muscles stimulated at 30 Hz. There were no apparent differences in the histochemistry or biochemistry between the muscles stimulated at 10 and 30 Hz. These results indicate that the 30 Hz paradigm may be better suited than the 10 Hz continuous stimulation for application requiring sustained muscle tension such as correction of scoliosis.

During initial treatments a full amplitude "on" period which is one-third of the stimulus "off" time generally will avoid rapid muscle fatigue during a treatment session. By gradually extending the time during which the stimulation is "on", it is possible to make a two- or three-fold increase in the amount of stimulation given during a single

treatment session without causing functional muscle fatigue. As the stimulation program progresses, the "off" time may be reduced gradually as the stimulated muscle becomes less fatigable. This reduction in rest time in conjunction with an increased "on" time, should proceed slowly (Benton et al., 1981). The desirable length of the stimulation treatment session depends on program goals and the status of the patient within the program.

Stimulating Waveform

A nerve fiber may be excited by a broad range of electrical waveforms, all of which elicit the same physiological response. The reaction of the tissue surrounding the electrode, however, shows a marked dependence on the parameters of the stimulating waveform. Van den Honert and Mortimer (1979) studied the response of a single node of Ranvier to short duration biphasic stimulating currents. Such stimulus patterns are known to be less likely to induce tissue damage than are longer monophasic stimuli. The presence of the anodic phase of a pulse train was found to abolish excitation in cases where the cathodic phase alone was near threshold. In addition, peak twitch force from the tibialis anterior muscle of cat was found to be reduced by the use of biphasic motor nerve stimulation in place of monophasic stimulation. The abolition phenomenon could be eliminated by the introduction of a delay of 100 microseconds. between the phases of biphasic stimulus waveform. Mortimer and Kaufman (1980) evaluated coiled wire electrodes for intramuscular electrical stimulation. They concluded that balanced charge biphasic stimulation allows greater charge densities to be used without the effect of increased tissue damage. Balanced charged biphasic stimulation is considered safer than monophasic stimulation and recommended for chronic use (Crago et al., 1974). Unequal ion transfer may be reduced by the use of a

symmetrical, balanced, biphasic pulses. Although the active electrode pulse is of greater amplitude in one than the other, the total current flow is the same in both directions. Therefore, the average flow is zero.

Functional Electrical Stimulation And Scoliosis

Idiopathic scoliosis is among the most common developmental ailments of children. It appears as a lateral deviation of the spine at the level of the thoracic or lumbar vertebrae in up to 10% of the population. The onset of scoliosis can be at any stage of growth from birth to skeletal maturity, but it is primarily discovered in otherwise healthy adolescents. Fifty to 80% of curves will progress at least 5 degrees (O'Donnell, 1988). Most small curves are treated with simple observations, however, approximately 0.2% of adolescents warrant further treatment because of a high likelihood of continued curve progression (Bunnell, 1986).

Electromyographic studies of the spinal muscles in patients with scoliosis have been performed for several decades with the same result; the electrical activity recorded at the level of the apex, on the convex side of the curve, is consistently higher than that recorded from the concave side (Riddle and Roaf, 1955; Le Febvre et al., 1960; Alexander and Season, 1978; Reuber et al., 1983). Fidler and Jowett (1976) measured the length of the multifidus muscle on each side of the curve at the level of the apex and found that the muscle is consistently shorter on the convex side. The combination of these results and the EMG findings reveals a consistent picture of muscle imbalance around the spine. It follows that electrical activation of the longer, less active muscles on the concave side of the curve should balance the forces and effect correction of the curve.

Animal studies have demonstrated that electrical stimulation of the spinal muscles

produces sufficient force to alter the curvature of the spine, without producing degenerative changes in muscle fiber types (Olsen et al., 1975; Monticelli et al., 1975; Bobechko et al., 1979). The most common form of treatment for curves 20 to 30 degrees is mechanical bracing. Cervical-thoracic-lumbar-sacral orthoses, such as the Milwaukee brace, the thoraco-lumbar-sacral orthoses, such as the Boston and Miami braces are successful in arresting curve progression in approximately 85% of cases (Bunnell, 1986). These methods, however, rarely provide correction of the deformity and have major drawbacks in cosmetics, comfort and psychological stress. For these reasons, the adolescent often refuses to comply with the bracing procedures, resulting in a potentially significant curve progression (Herbert et al., 1976). Induced contraction of the spinal muscles has been attempted as an alternative to mechanical bracing. Animal studies have demonstrated that electrically-activated spinal muscles can exert sufficient forces to alter curvature of the spine (Monticelli et al., 1975; Olsen, 1977; Bobechko et al., 1979). Several investigators have reported success in partially correcting or halting progression of scoliosis, using functional electrical stimulation of the muscles of the spine (Axelgaard, 1984; McCollough, 1986; Herbert and Bobechko, 1988).

Three methods of electrical stimulation are currently available for treatment of scoliosis in the United States and Canada. Each is designed to induce muscle contraction on the concave side of the curve, for 8 hours at night, in order to produce

the forces required to realign the spinal column. The three methods are the Isomed Electrospinal Orthosis (ESO) developed by McCollough, the Isomed Electrospinal Instrumentation (ESI) developed by Bobechko et al. and Neuromedic's Scolitron which was developed by Axelgaard. The ESO system applies stimulation to the deep and superficial paraspinal muscles, using surface electrodes. The ESI is a totally implanted system which primarily activates the deep paraspinal muscles. Scolitron evokes contraction of the peripheral spinal musculature with lateral electrical surface stimulation (LESS).

The effectiveness of each of these methods has not been firmly established. McCollough (1985) reported that 25% of patients treated with ESO improved more than 4 degrees, 52% exhibited no change in spinal curvature (+/- 4 degrees), and 23% progressed more than 4 degrees. Herbert and Bobechko (1988) reported that 86% of children treated with ESI had their curves halted or improved with stimulation and 14% had curves that progressed to the point of requiring spinal fusion for stabilization. Axelgaard (1984) reported a success rate of 93% in preventing further progression of curves below 30 degrees and 73% success for curves greater than 30 degrees.

STATEMENT OF THESIS

There appears to be substantial evidence that facial form is influenced by the musculature of the orofacial region. Whether this influence is strictly functional remains debatable; however, it appears from experimental evidence that this may be the case.

With this in mind, it is the rare instance in which an orthodontic treatment plan includes an appliance designed to directly change muscle function. Ideally, appliances that seek to change facial morphology by changing muscle function should: 1) provide reliable operator control of both the force and duration of muscle activity; 2) allow stimulation or inhibition of selected muscles; and, 3) be able to be used alone or as an adjunct to traditional mechanical techniques.

The long term objective of this line of research is to alter facial growth by functional electrical stimulation (FES) of craniofacial muscles. In particular, FES could be used to reduce facial asymmetries in growing children with vertical skeletal discrepancies.

Muscle contraction will be accomplished using coiled, multi-stranded intramuscular electrodes. The specific aims of the study are: 1) To evaluate the efficacy of the stimulator (Neu Rx Is/4) in producing sustained muscle contraction of the masseter muscle. 2) To determine fatigue properties of the masseter muscle following chronic stimulation. 3) To determine the selectivity of individual muscle contraction. Electromyography will be used to determine if electrical stimulation of one muscle is

associated with muscle contraction of a neighboring muscle.

Therefore, we would expect that:

1) The electrical stimulator (Neu Rx Is/4) will produce contraction of the masseter muscle.

2) Bite forces will remain constant following chronic electrical stimulation of the masseter muscle.

3) Recruitment will be confined to the masseter muscle.

4) The course study will involve minimal subject discomfort.

5) Functional Electrical Stimulation of the masseter muscle is clinically applicable.

6) Percutaneous electrodes can be used safe and effectively.

MATERIALS AND METHODS

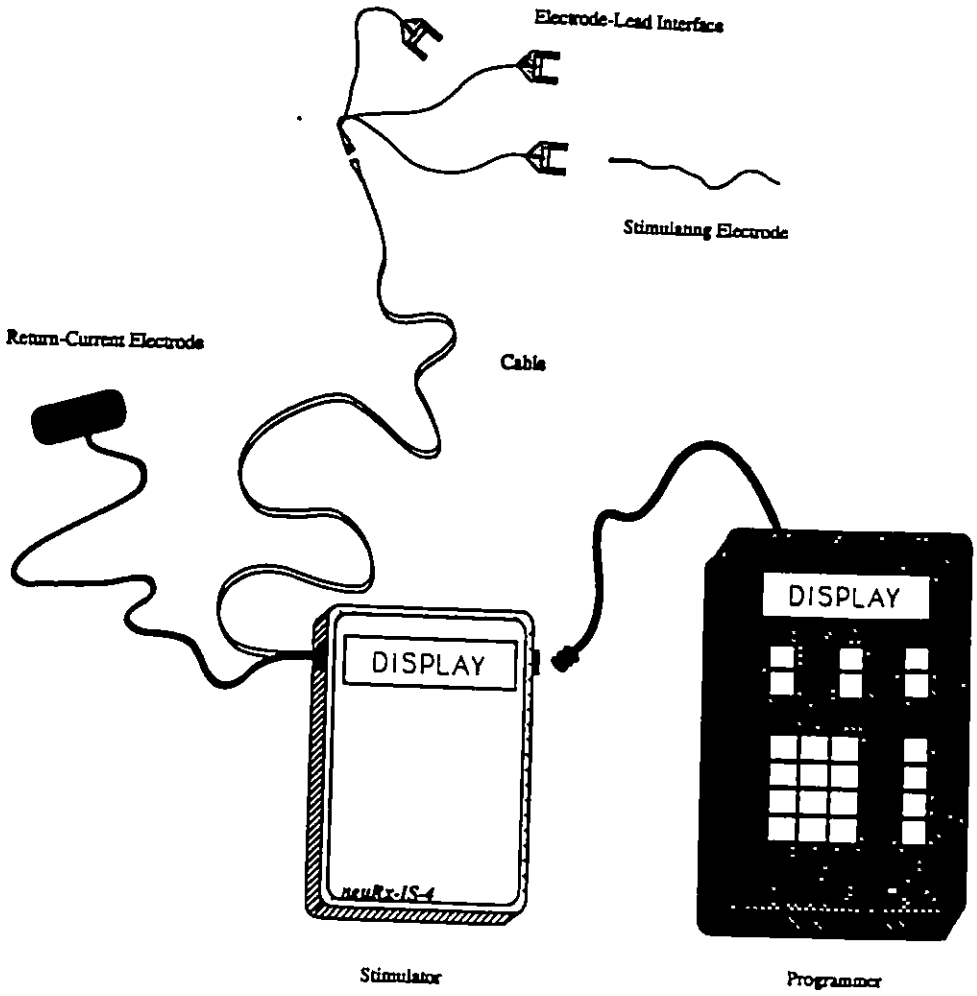
Patient Population

The present investigation recruited 10 volunteers from Case Western Reserve University School of Dentistry. Candidates for the study were young, healthy adults, either male or female. Candidates were excluded from the study if they were pregnant, had a cardiac condition, neuromuscular disease, a temporomandibular joint disorder, or any significant physical or mental disease. Medical and dental histories were evaluated to ensure safety. Selection of subjects was made independent of malocclusion and facial form. Subjects wishing to participate in the study were required to sign a consent form approved by the Institutional Review Board of Case Western Reserve University. The study had no personal benefit to the subjects in the study. The study will help obtain a better clinical understanding of electrical stimulation of the masseter muscle.

Electrode System and Stimulus Waveform

The intramuscular electrode system developed at Case Western Reserve University, Biomedical Engineering Department was used for all stimulation of the masseter muscles described in this experiment. This system allows a high degree of selective muscle contraction in the limbs without many problems associated with other methods of electrical stimulation. All electronics are externalized and the electrodes are implanted intramuscularly. The system consists of a stimulus generator (Neu Rx Is/4), electrodes and leads (figure 1). The stimulator interfaces to a detachable programmer to

Figure 1. Neu Rx Is/4 stimulation system



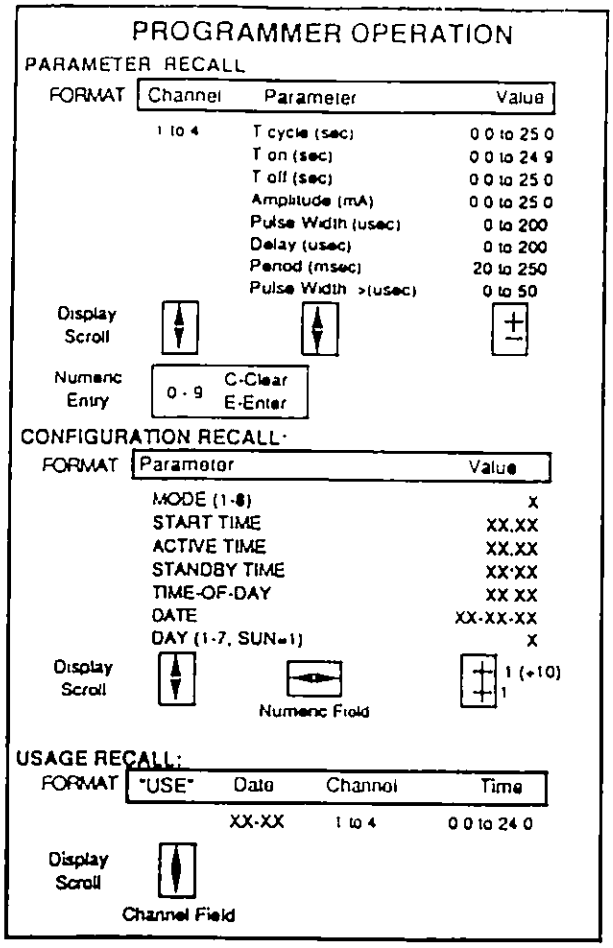
Courtesy of FDA Approval for scoliosis; Applied Neural Control Laboratory, Case Western Reserve University, 1991.

accept stimulation protocol specifications. The stimulation protocol is followed until changed (figure 2). There are no patient accessible controls. The device provides operational status information through a liquid crystal display.

The stimulator delivered a biphasic, balanced charge waveform (figure 3). This kind of stimulation is considered safer than monophasic stimulation and is recommended for chronic use (Mortimer and Kaufman, 1980). A nerve fiber may be excited by a number of different waveforms, all producing the same physiological response. However, the risk of tissue damage surrounding the electrodes depends highly on the parameters (voltage levels, energy, power, injected charge, timing parameters) of the stimulating waveform. In order to reduce the accumulation of potentially toxic reaction products, a balanced charge biphasic stimulating waveform was employed (Crago et al., 1974). Partial reversal of the electrochemical reaction at the electrode surface was achieved by applying a reversal current pulse following the stimulus which passes an equal charge backwards through the electrode. The net action of reaction products was thereby, reduced.

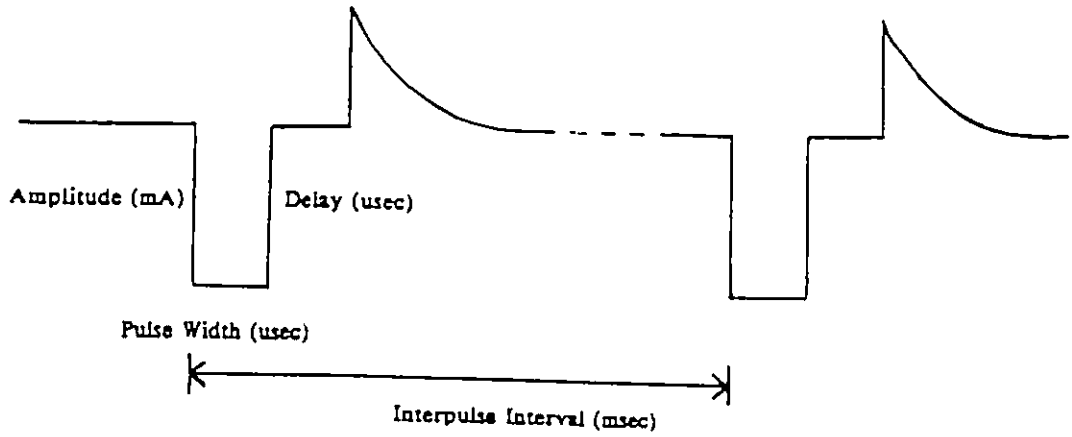
All subjects received regulated current, balanced charge biphasic stimulation. The duration of the primary cathodic pulse was fixed at 100 micro-seconds (μsec); the subsequent anodic pulse consisted of a capacitive discharge such that the net charge injected balanced the cathodic pulse. A delay of 100 μsec was introduced between the primary (cathodic) and secondary (anodic) pulses to prevent the secondary from partially reversing the depolarizing effect of the primary pulse (Ferguson et al., 1989).

Figure 2. Progammer



Courtesy of FDA Approval for scoliosis; Applied Neural Control Laboratory, Case Western Reserve University, 1991.

Figure 3. Pulse specifications



Courtesy of FDA Approval for scoliosis; Applied Neural Control Laboratory, Case Western Reserve University, 1991.

Description of Electrodes

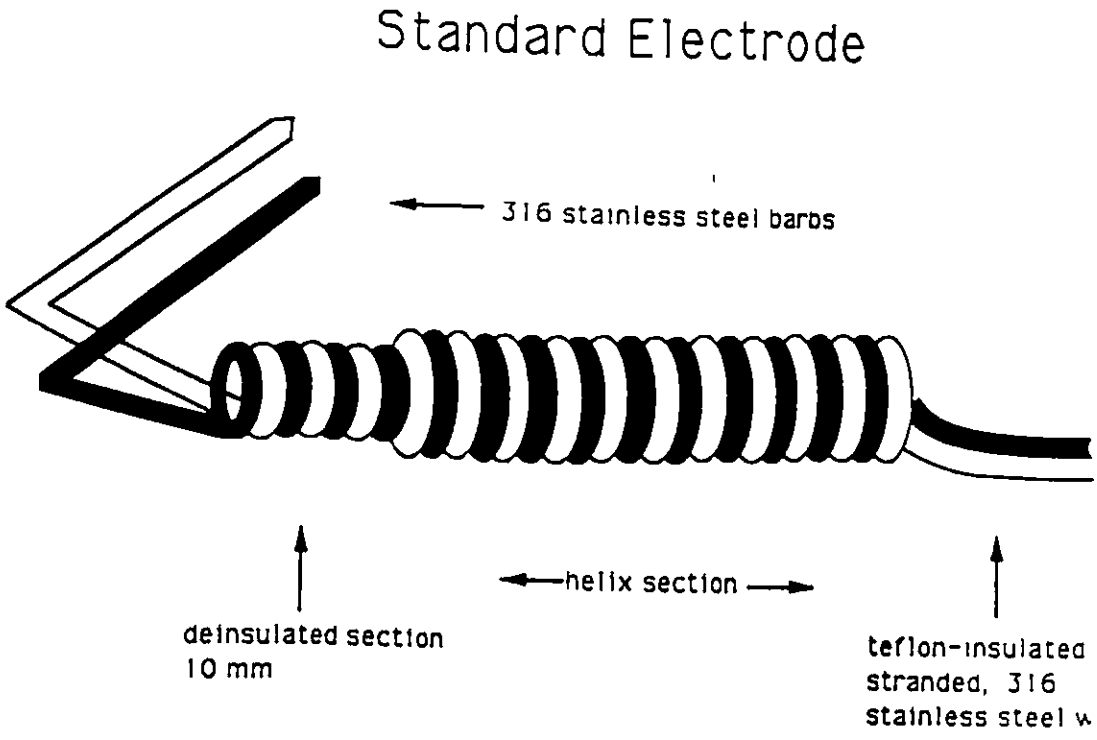
The intramuscular electrode is made of 316L stainless steel which is insulated except at the tip with teflon. Stainless steel has been used in intramuscular electrical stimulation because of its practical availability and high strength compared to platinum and its commonly available alloys (Caldwell, 1970; Mortimer and Kaufman, 1980). The electrode configuration used in this study was the multi-strand, coiled, monopolar electrode (figure 4). Monopolar electrodes are efficient and selective if located close to the nerve entry point, but their performance declines with distance from it. Bipolar intramuscular stimulation is more efficient and selective than monopolar stimulation, but only if the nerve entry point is between the electrodes (Popovic et al., 1991). The latter requirement makes bipolar stimulation impractical for the muscles of mastication.

Placement of Electrodes

The electrode will pass through the shaft of a 18-gauge injection needle and the de-insulated end of the wire which protrudes through the beveled end of the needle, was bent to form a hook of approximately 3 mm in length. The wire and needles were sterilized by autoclaving prior to use. Before and after electrode implantation, the electrode site was disinfected with betadine solution. Subjects 1 and 2 (Part I) received a topical anesthetic (Lidocaine Hydrochloride) at the site of electrode placement. All remaining subjects received injections of local anesthesia (3% Mepivicaine with no Epinephrine) at the site of electrode insertion.

All electrodes were placed using an extra-oral approach. Prior to insertion, the masseter muscle was palpated to determine the optimal direction for electrode implantation. The electrode was inserted perpendicular to the mandibular corpus in the region of the gonial angle. The needle was advanced 1 cm and then directed obliquely,

Figure 4. Standard electrode



Courtesy of FDA Approval for scoliosis; Applied Neural Control Laboratory, Case Western Reserve University, 1991.

antero-superiorly 2-3 cm following the middle belly of the masseter muscle. Following insertion, finger pressure was applied over the beveled area of the injection needle, the needle was then withdrawn leaving the hook to hold the electrode wire in the muscle. Following placement, the area was sterilized and the site covered with plastic surgical tape to protect the area from mechanical abuse.

Stimulation Protocol

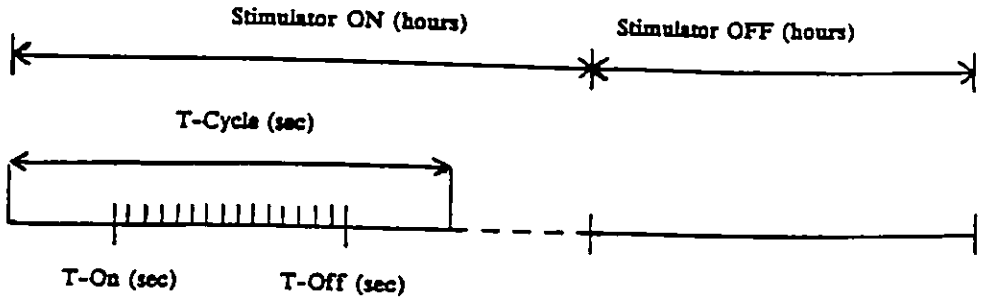
The stimulator was attached to a detachable programming device (figures 1,2) in order to define the stimulation protocol. Although two stimulation parameters were evaluated, each protocol had the following common parameters; Frequency=20 Hz, Pulse Duration=100 μsec, Interpulse Interval=50msec. Electrical stimulation was started two days after placement of the electrodes for a total of 10 days over a two week period. Pulse amplitude (mA) (figure 3) and duty cycle (figure 5) varied for the two groups.

The following protocols were tested:

Group A- Simultaneous, bilateral chronic electrical stimulation of the masseter muscles. Stimulation of the masseters was applied for four continuous cycles per day with 0.5 hours of rest after 1.5 hours of stimulation. The pulse amplitude (mA) was adjusted to half the value of the initial pain threshold and kept constant for the duration of the experiment (Table 1).

Group B- Alternating, bilateral chronic electrical stimulation of the masseter muscles. The duty cycle consisted of a continuous 5 second "ON" period followed by a 5 second "OFF" period alternating between the right and left masseter muscles for 8 hours per day. The pulse amplitude (mA) was adjusted to produce an initial interocclusal bite force of 3 pounds, on the side of electrical stimulation, and was kept constant for the

Figure 5. Pulse train specification



Courtesy of FDA Approval for scoliosis; Applied Neural Control Laboratory, Case Western Reserve University, 1991.

Table 1.--Amplitude (mA) used for simultaneous chronic electrical stimulation of the bilateral masseter muscles for Subjects in Part A

SUBJECT	RIGHT (mA)	LEFT (mA)
1	6.0	5.5
2	4.0	4.0
3	7.0	7.0
4	6.0	6.0
5	7.0	7.0

Constant: Frequency= 20 Hz, Pulse Duration = 100 µsec., Interpulse Interval = 50 msec, Duty Cycle = Simultaneous 15 sec on and 5 sec off.

Table 2.--Amplitude (mA) used for alternating chronic electrical stimulation of the bilateral masseter muscles for Subjects in Part B

SUBJECT	RIGHT (mA)	LEFT (mA)
1	4.1	2.3
2	1.8	4.0
3	4.0	4.3
4	7.0	7.0
5	5.0	4.8

Constant: Frequency= 20 Hz, Pulse Duration = 100 µsec., Interpulse Interval = 50 msec, Duty Cycle = Alternating 5 sec on and 5 sec off

duration of the experiment (Table 2).

The daily, duty cycle, "off" times were adjusted to the study subjects personal schedule so that the 8 hours "on" time coincided with early morning and afternoon. Maximum voluntary and stimulated bite forces were recorded twice daily for 10 days. Bite forces were recorded daily at the beginning and end of the 8 hour stimulation period period to determine the fatigue properties of the masseter muscles following chronic electrical stimulation.

Occlusal Force Transducers

Bite forces were recorded using a miniature quartz transducer (Kistler, Model 9211) and a charge amplifier (Kistler, Model 5010) (Profit et al., 1983; Profit and Fields, 1983). Bite forces were recorded on a chart recorder (Gould and Channel Chart Recorder, Model RS3400). The transducer was weight calibrated prior to experimentation. Bite forces were recorded in volts and translated to pounds.

Piezo-electric transducers, based on quartz crystals, produce a signal in the form of a small electrical charge. Therefore, to be useful these transducers must be used with a charge amplifier. If a steady load is applied to a piezo-electric material, a charge will appear when the load is applied, but then will fade away. When the load is released, an equivalent charge spike is observed in the opposite direction. Selection of an appropriate time constant in the amplifier system is important if signals which portray the dynamic course of occlusal loading are desired. Since occlusal loads may be maintained for a few hundred milliseconds, a relatively long time constant (seconds) works best for occlusal bite force measurements (Profit and Fields, 1983).

Procedure for Fabricating the Occlusal Stent and Location of the Bite Force Transducer

One transducer was used for this study. Study models were made of every subject and mounted on an articulator in centric occlusion. A vacuum molded plastic stent was fabricated over the mandibular teeth. The transducer was placed over the distobuccal cusp of the lower first molar parallel to the occlusal plane. The right mandibular first molar was used in group I while both right and left mandibular first molars were used in Group II. The models were analyzed to avoid teeth with large restorations.

The transducer was positioned on top of the plastic stent, over the distobuccal cusp tip of the mandibular first molar with the lead wire extending anteriorly. The vertical adjustment screw of the articulator was then set to provide approximately 1.5 mm clearance between the transducer and the central fossa of the maxillary first molar. Acrylic resin was mixed and placed over the maxillary first molar and transducer. The articulator was closed and the acrylic formed a matrix to protect the teeth from injury at high bite forces. The acrylic resin was trimmed parallel to the mandibular occlusal plane to avoid any interferences. Acrylic resin was also applied to the mandibular plastic stent surrounding the bite force transducer to provide stability and reproducibility.

Electromyography

Electromyography has enabled researchers to study masticatory movements concurrent with a direct monitoring of muscular activity. Muscle function has been studied extensively using electromyography.

This electrical recording technique records electrical signals which arise from the muscle fibers and can show various deviations, durations and shape. The recording image is dependent upon the type of electrode and whether it is placed over the muscle

as a surface electrode or within the muscle for intramuscular recordings. The primary source of the recording signal is the individual muscle fiber. However, electromyography using surface recording electrodes to intramuscular electrodes detect information from a functional group of muscle fibers known as motor units.

Electromyography offers a precise instrument for determining whether active recruitment of muscle by the central nervous system is occurring and furnishes information as to when craniomandibular muscles are used. The level of EMG activity is usually proportional to bite force, but muscle force or tension may not be directly related to the level of EMG activity.

Sources of the EMG records differ among investigators. Some prefer to record from a single muscle fiber, while others favor recordings from a group of muscle fibers innervated by one motor unit (Ekstedt and Skelberg, 1973; Burke and Tsairis, 1973). In an intramuscular electrode recording the position and size of the recording field determines the duration of the spike. Ekstedt (1964) and Ekstedt and Skelberg (1973) studied the effect of changing electrode configuration and its relation to the shape and frequency of the recorded signal. They concluded that spikes from individual muscles can be differentiated.

Function and the level of recruitment of the muscle influence the rate of spike discharge in the craniomandibular muscle. Eriksson et al. (1984) analyzed the discharge of spikes in EMG recordings of the craniomandibular muscles in five normal adult subjects during two different functions. The superficial anterior and middle temporalis, the anterior superficial masseter and deep masseter regions were recorded during clenching in the intercuspal position or retruded mandible. Although the study showed that single spike potentials exhibit different firing patterns in the two different

functions of clenching and retrusion, there was no significant trend. Some subject discharged their spike potential more during clenching, while others would discharge their potential more during retrusion. The lowest rate of discharge was 5 to 8 Hz, and the highest recognizable rate was 20 to 25 Hz. The lack of consistently higher discharge by a motor neuron during one response (i.e., clenching) suggests that control of the discharge rate may depend upon which regions of a muscle are recruited maximally for a given response.

Electromyographic Recordings

Electromyographic recordings were made for four subjects in Group A of the experiment and 2 subjects in Group B, using a Dantec Neuromatic 2000M (Denmark) with two channels for input. Recordings from the right anterior temporalis muscle were performed using concentric intramuscular needle recording electrodes and filters with an upper frequency of 10kHz and a lower frequency limit of 20 Hz. Paper speed was set at 10 cm/second and sweep speed at 10ms/division.

Electromyography was used to determine the selectivity of electrical stimulation in producing muscle contraction. Is the right temporalis muscle recruited during electrical stimulation of the right or left masseter muscle? Electromyography allowed us to determine whether the decrease or increase in bite force was strictly due to masseter muscle activity.

The parameters (amplitude, frequency, pulse duration, and interpulse interval) used for the electromyographic study were the same as those used for the chronic electrical stimulation protocol. Muscle activity in the right temporalis muscle was recorded during electrical stimulation of the masseter muscles when the study subject was asked to relax and when the study subject was asked to open. Results were recorded as an all

or none response.

Subjective Pain Evaluation

The McGill pain questionnaire was developed by Ronald Melzack in 1975 to measure dimensions of pain experience. The questionnaire consists primarily of 3 major classes of word descriptors- sensory, affective and evaluative- that are used by patients to specify subjective pain experience. It also contains an intensity scale to determine the properties of pain experience. There are 102 pain descriptors which are qualitatively grouped into 20 subclasses. The subject is asked to pick one word in each subclass that most accurately describes the pain present. If no words in a particular group are relevant, then none are chosen.

The 2 major measures used in this study are: (1) the Pain Rating Index, based on numerical values assigned to each word descriptor, and (2) the Present Pain Intensity based on a 1-5 intensity scale. Pain rating index is based on the rank values of each word. In this scoring system, the word in each subclass implying the least pain is given a value of 1, the next word is given a value of 2, etc. The values of the words chosen are then added up to obtain a score for each category, and a total score for all categories.

Subjective pain was evaluated for all study subjects during electrode insertion, days 1,5 and 10 of chronic electrical stimulation, and electrode removal. Study subjects who participated in the electromyographic study were also assessed for subjective pain. Subjects in Part A were assessed retrospectively, where as subjects in Part B were evaluated at the time of the procedure. Subjects were asked to complete the McGill pain questionnaire and the pain intensity scale (0=no pain to 5=excruciating).

Data Analysis

Bite Force Measurements

Means and standard deviations were calculated for all bite force measurements. In addition, individual subject variability was examined to determine possible sources of variation.

Electromyographic Analysis

Recruitment of the temporalis muscle was studied following electrical stimulation of the ipsilateral and contralateral masseter muscles. Results were recorded as a "all or none" response.

Subjective Pain Analysis

Because the ultimate goal of this study was to produce a clinical tool, each subject completed the McGill pain questionnaire. The level of discomfort experienced was recorded during placement of electrodes, during the stimulation period, electromyography and during removal of the electrodes.

RISK / BENEFIT ANALYSIS

All subjects were volunteers from the Case Western Reserve University School of Dentistry. Ten subjects were required to perform the study. The volunteers were healthy adults. Medical histories were completed by all subjects to ensure safety. Volunteers were excluded from the study if they were pregnant, had a neuromuscular disorder, had a cardiac condition, had any TMJ symptoms or had any significant physical or mental disease.

The information gained in this study had no direct benefits to the study subjects. The information gained will be used to improve the quality of treatment given to patients with dentofacial deformity. The risks associated with acute or chronic use of the electrical stimulating device are minimal and include the following:

1) Electrodes may Break Beneath the Skin

There is a possibility, 1/500, that electrodes will break beneath the skin because of their very small size. The fragments cannot always be visualized with radiography. The fragments do not migrate and do not present a risk to the patient when left in the muscle. X-ray photographs have been taken of identical broken electrodes in the arms of spinal cord injured patients. The broken sections appear to remain in constant location throughout several years. The metal used in these electrodes is medical grade stainless steel which is used for implants and has been judged to be non-toxic and not dangerous to body tissues.

2) Infection

There is possibility of infection at the site where the electrode enters the skin. Of the more than 500 electrodes that have been implanted in the last eight years, there has been no documented case of infection at the electrode site. All electrodes will be sterilized prior to use and sterile procedures will be used for implantation.

3) Skin Irritation

There have been instances of skin irritation due to the adhesive use of surface electrodes. However, the incidence of irritation with intramuscular electrodes is estimated to be less than 1 in 500 cases.

4) Granuloma Formation at the Electrode Entry Site

The presence of the electrode may induce granuloma formation at the skin junction in some patients. The presence of severe granuloma formation will warrant removal of the electrode. Small granulomas may be treated with additional cleansing or application of silver nitrate. No link has been made between granuloma formation and infection.

5) Tissue Damage

Tissue damage can be caused by the passive presence of the electrode or by the stimulus current. Tissue damage can destroy the muscle fiber.

Passive Presence -- materials implanted into the body initiate a foreign body response consisting of encapsulation by fibrous tissue. The more compatible the material, the thinner the encapsulation layer. A thick layer of fibrous tissue is detrimental because greater stimulus currents are required to excite the neural tissue (Mortimer 1981) and fibrous tissue may replace the viable muscle fibers. The duration of implantation in this study does not present such a possible risk.

Damage Due to Stimulus -- studies of the tissue reaction at the intramuscular site in cats (Mortimer 1980) have shown that the damage with balanced charge, biphasic stimulation was negligible when the charge density was less that or equal to 0.4 microcoulombs which is equivalent to a 20 mA pulse with 200 microsecond duration. It has been found that, if stimuli are restricted to these values, the area surrounding the stimulating tip would be indistinguishable from the surrounding tissue.

6) Electrical Hazard

With proper use, the electrical safety of the device is extremely good. The power source is external to the body and uses an isolated battery as the power supply. With normal use, no current pathways will cross the heart. The Association of Advancement of Medical Instrumentation (AAMI) Standards (1986) suggest limiting the charge injection to 25 microcoulombs per pulse. The charge injected by our system never exceeds 0.4 microcoulombs per pulse. Thus it is highly unlikely that cardiac function could ever be affected.

Misuse of the device, however, can cause side effects (electric shock). The stimulator should not be tampered with. To ensure this, the device will be mechanically sealed.

7) Discomfort Due to Stimulation

Stimulation with percutaneous electrodes rarely elicits a sensation that is uncomfortable because skin afferents are not excited. However, if painful sensations are expressed by the subject, the electrode will be withdrawn.

8) Jaw Joint Damage

Extreme and prolonged contraction of the muscles of mastication can have

deleterious effects to the temporomandibular joint. Due to the minimal charge injected by the system, it is unlikely that jaw joint damage will occur.

In the opinion of the investigators, the above complications are judged to be unlikely and the risk to patients involved in this study is minimal.

All subjects were fully informed about the study and were required to sign a written consent form.

RESULTS

The results of the present study will be presented under the following headings:

I. Part A

A) Individual Response - Subject 1

1) Stimulated Bite Forces

2) Maximum Voluntary Bite Forces

B) Group Response

1) Average % Change of Stimulated Bite Forces and Maximum Voluntary Bite Forces

II. Part B

A) Individual Response - Subject 1

1) Stimulated Bite Forces - Right

2) Stimulated Bite Forces - Left

3) Maximum Voluntary Bite Forces - Right

4) Maximum Voluntary Bite Forces - Left

B) Group Response

1) Daily Average and % Change of Stimulated Bite Forces - Right

2) Daily Average and % Change of Stimulated Bite Forces - Left

3) Average % Change of Maximum Voluntary Bite Forces - Right and Left

III. Electromyography

IV. Subjective Pain Evaluation

Part A

Individual Response - Subject 1

Stimulated Bite Forces

The results of the tests for subject 1 are presented in Table 3 and Figure 6. The amplitude used for simultaneous chronic electrical stimulation of the bilateral masseter muscles for subject 1 was 6.0 mA and 5.5 mA, right and left respectively. There was a decrease in bite force on all days except Day X. Days IV and IX demonstrate a statistically significant decrease ($p \leq 0.05$) of 22.20 and 18.10 percent respectively. Bite forces varied uniformly, morning and afternoon, from Day I to X with a progressive increase in bite force from Day VI to X.

Maximum Voluntary Bite Forces

The daily average maximum voluntary bite forces before (morning) and after (afternoon) electrical stimulation of the bilateral masseter muscles is shown in Table 4 and Figure 7. There was no statistically significant difference between T1 (morning) and T2 (afternoon) on any day. Figure 7 illustrates that maximum voluntary bite force changed very little over the course of the study.

Group Response

Average % Change of Stimulated Bite Forces and Maximum Voluntary Bite Forces

The average percentage change of daily bite forces for all subjects in Part A are depicted in Table 5 and Figure 8. Stimulated bite forces only showed an increase on Day III, while the greatest change was observed on Day IV.

Table 3.--Daily average bite forces at T_1 (morning) and T_2 (afternoon) during simultaneous, chronic electrical stimulation of the bilateral masseter muscles for Subject 1 in Part A

DAY	T_1 (lbs.)	S.D.	T_2 (lbs.)	S.D.	p VALUE	% CHANGE
I	6.90	2.00	4.50	0.60	0.09	-34.80
II	6.80	1.80	4.90	1.30	0.11	-27.90
III	2.70	0.20	3.30	0.40	*0.05	22.20
IV	3.60	0.30	2.80	0.30	*0.03	-22.20
V	7.90	0.70	8.40	0.80	0.50	6.30
VI	4.90	0.70	4.70	0.50	0.68	-4.10
VII	6.80	1.10	5.60	0.60	0.12	-17.60
VIII	6.70	1.20	5.90	0.40	0.27	-11.90
IX	7.20	0.40	5.90	0.70	*0.05	-18.10
X	8.30	0.60	8.28	1.10	0.81	-20.0

* Statistically significant difference ($p \leq 0.05$).

Constant: Frequency= 20 Hz, Pulse Duration = 100 µsec., Interpulse Interval = 50 msec, Duty Cycle = Simultaneous 15 sec on and 5 sec off

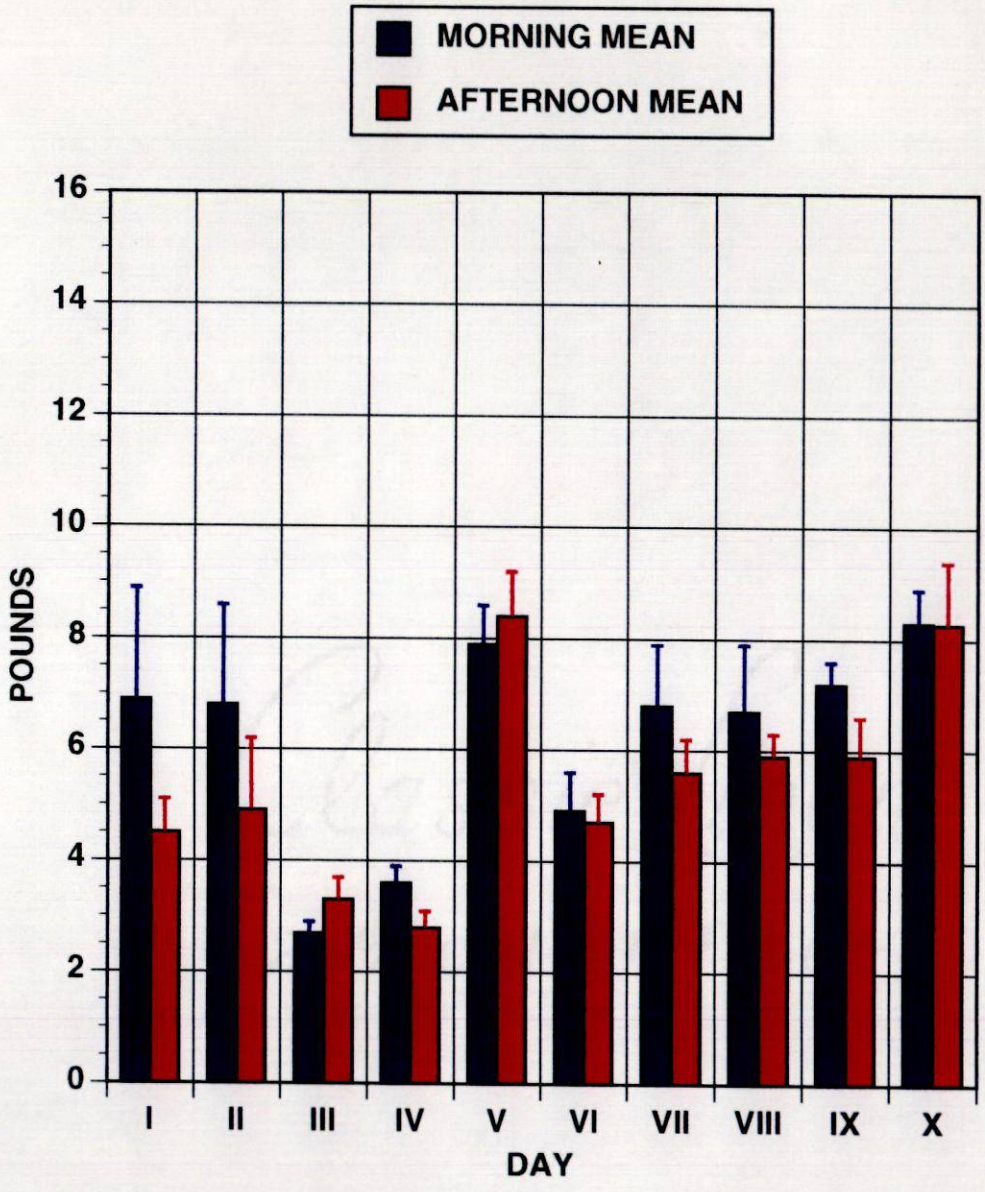


Figure 6. Daily average bite forces during simultaneous, chronic electrical stimulation of the bilateral masseter muscles for Subject 1, Part A

Table 4.--Daily average maximum voluntary bite forces before (T_1, morning) and after (T_2, afternoon) simultaneous, chronic electrical stimulation of the bilateral masseter muscles for Subject 1 in Part A

DAY	T_1 (lbs.)	S.D.	T_2 (lbs.)	S.D.	p VALUE	% CHANGE
I	88.54	7.10	87.60	4.85	0.71	-1.10
II	83.39	3.14	85.73	2.67	0.39	2.70
III	86.67	1.65	87.60	5.87	0.78	1.10
IV	86.20	3.47	85.27	4.85	0.73	-1.10
V	87.14	5.34	87.14	1.95	1.00	0.00
VI	87.14	4.19	90.42	15.05	0.63	3.60
VII	89.01	3.31	89.01	3.31	1.00	0.00
VIII	92.76	6.90	86.20	3.47	0.07	-7.60
IX	84.79	5.34	87.60	3.91	0.42	3.20
X	92.29	5.13	92.29	10.15	1.00	0.00

* Statistically significant difference ($p \leq 0.05$).

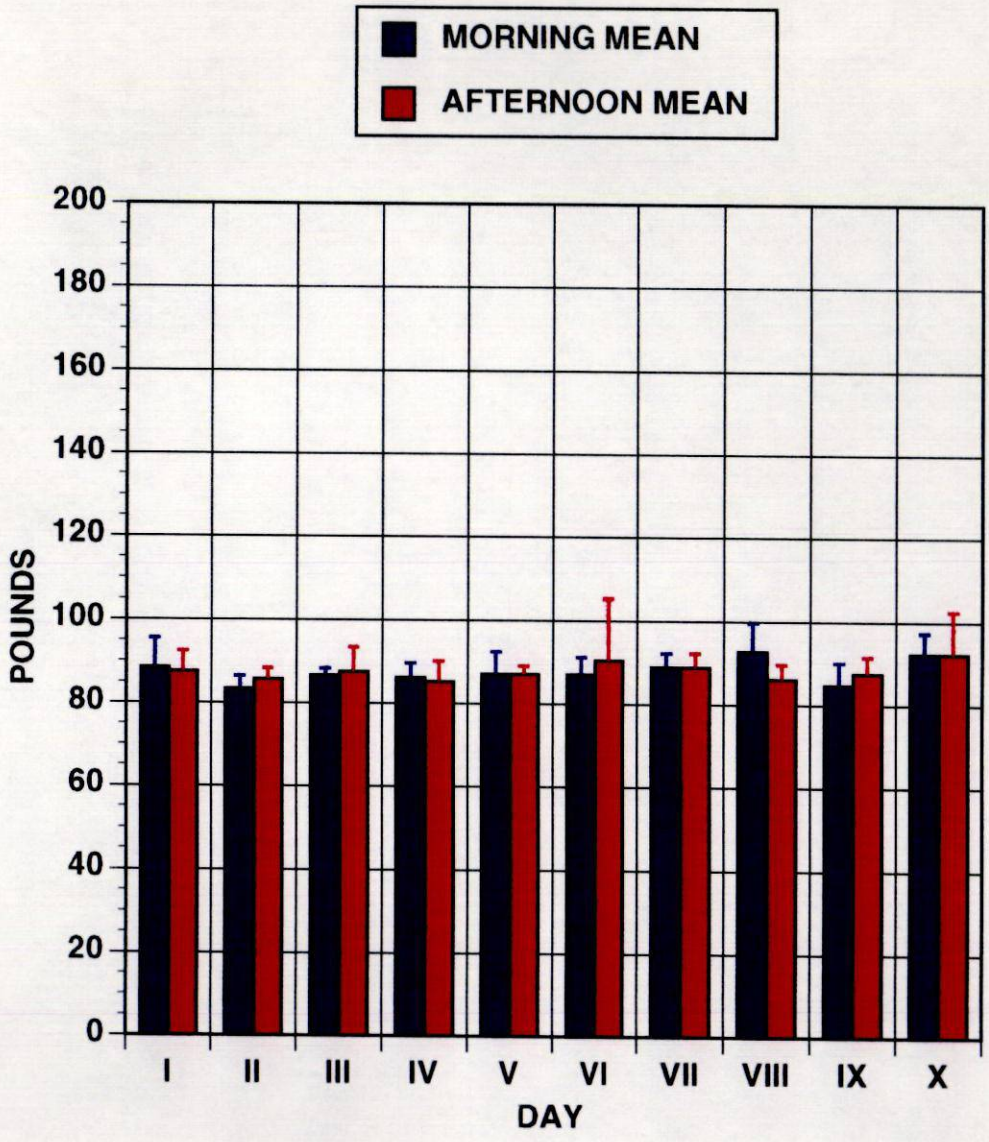


Figure 7. Daily average maximum bite forces before and after simultaneous, bilateral chronic electrical stimulation of the masseter muscles for Subject 1, Part A

Table 5.-- Average % change of daily bite forces during simultaneous, chronic electrical stimulation of the bilateral masseter muscles for all Subjects in Part A

DAY	% CHANGE (STIM. B.F.)	S.D.	% CHANGE (MAX. B.F.)	S.D.
I	-9.10	22.39	1.38	9.87
II	-6.74	20.81	-16.46	26.55
III	12.02	18.12	16.24	37.61
IV	-19.68	15.49	3.08	12.74
V	-10.66	20.28	0.48	2.17
VI	-6.40	8.36	-0.60	11.10
VII	-13.76	6.49	2.74	14.02
VIII	-7.30	4.62	-4.86	8.13
IX	-7.64	11.60	-0.08	9.47
X	-2.72	12.22	4.60	6.23

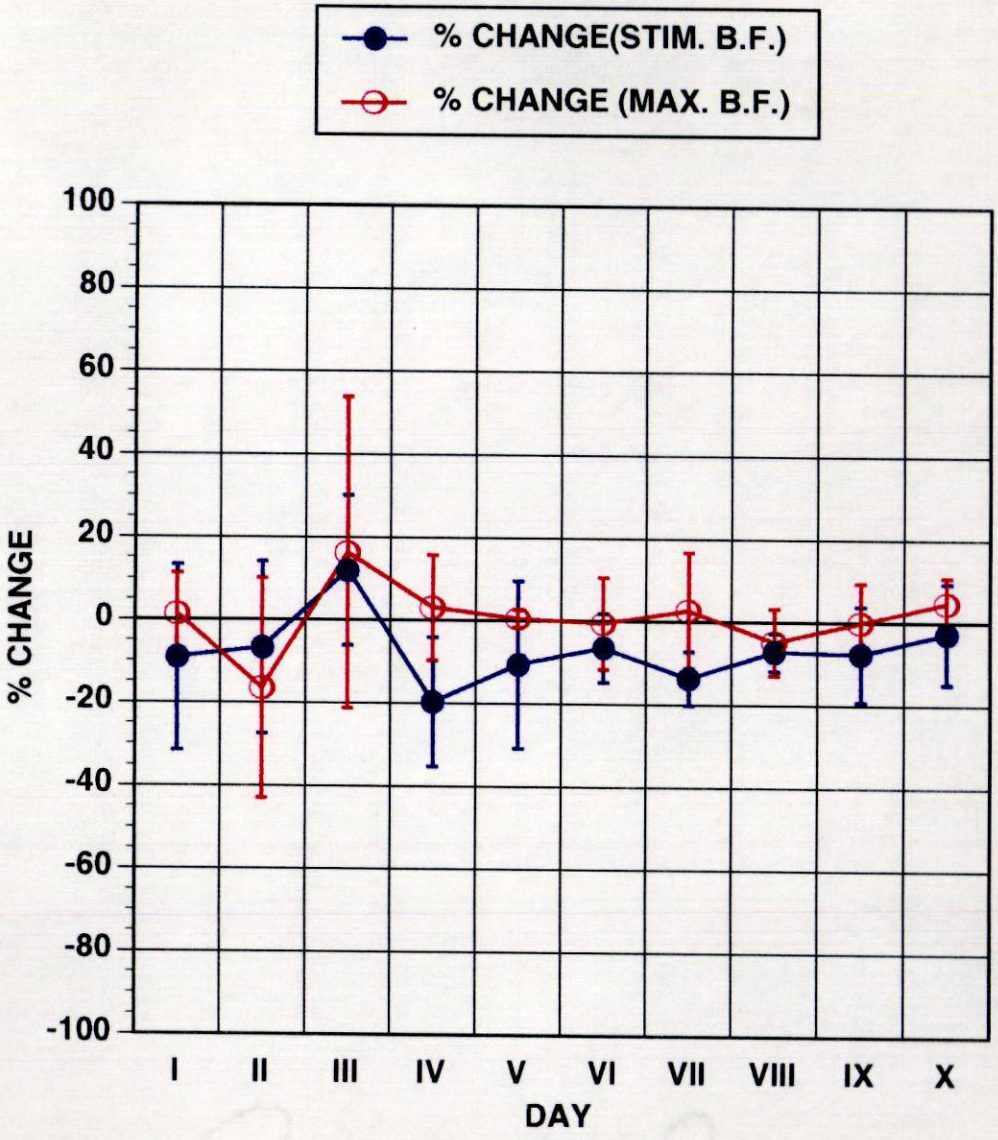


Figure 8. Average % change of daily bite forces during simultaneous, chronic electrical stimulation of the bilateral masseter muscles for all Subjects in Part A

The average % change of daily maximum voluntary bite forces demonstrate an increase on Days I, III, IV, V, VII, and X whereas Days II exhibited the largest decrease of 16.46%.

Part B

Individual Response - Subject 1

Stimulated Bite Forces - Right

The results of the experiment for subject 1 are presented in Table 6 and Figure 9. The amplitude used for alternating, chronic electrical stimulation of the bilateral masseter muscles for Part B was 4.1 and 2.3. There was a negative % change on all days except Days VII and IX. Days I, II, III, IV, and VIII exhibit a statistically significant decrease. Day IX demonstrated a statistically significant increase of 29.90 percent. Bite forces in the morning and afternoon increased relatively uniform from Day I to IX, from 3.33 to 6.66 pounds respectively.

Stimulated Bite Forces - Left

Daily average bite force left, at T1 (morning) and T2 (afternoon) are shown in Table 7 and Figure 10. A statistically significant decrease in bite force was evident on Days II and X of 18.20 and 25.20 percent respectively. Days I and IX exhibited no change in bite force from T1 to T2 while stimulated bite force increased during Day V. Figure 10 shows the increase in bite force from Day I to IX.

Maximum Voluntary Bite Forces - Right

The daily average maximum voluntary bite forces on the right side for subject 1 are presented in Table 8 and Figure 11. Bite forces remained relatively stable except for

Table 6.--Daily average bite forces right, at T_1 (morning) and T_2 (afternoon) during alternating, chronic electrical stimulation of the right masseter muscle for Subject 1 in Part B

DAY	T_1 (lbs.)	S.D.	T_2 (lbs.)	S.D.	p VALUE	% CHANGE
I	3.33	0.00	2.51	0.12	*0.00	-24.60
II	3.92	0.25	3.25	0.00	*0.03	-17.10
III	4.88	0.00	4.66	0.00	*0.00	-4.50
IV	4.88	0.00	3.18	0.12	*0.00	-34.80
V	4.36	0.71	4.07	0.33	0.66	-6.70
VI	5.77	0.22	5.55	0.00	0.22	-3.80
VII	6.21	0.22	6.51	0.25	0.38	4.80
VIII	6.43	0.22	5.47	0.12	*0.02	-14.90
IX	6.66	0.00	8.65	0.58	*0.02	29.90
X	4.07	0.33	3.33	0.00	0.06	-18.20

* Statistically significant difference ($p \leq 0.05$).

Constant: Frequency= 20 Hz, Pulse Duration = 100 µsec., Interpulse Interval = 50 msec, Duty Cycle = Alternating 5 sec on and 5 sec off

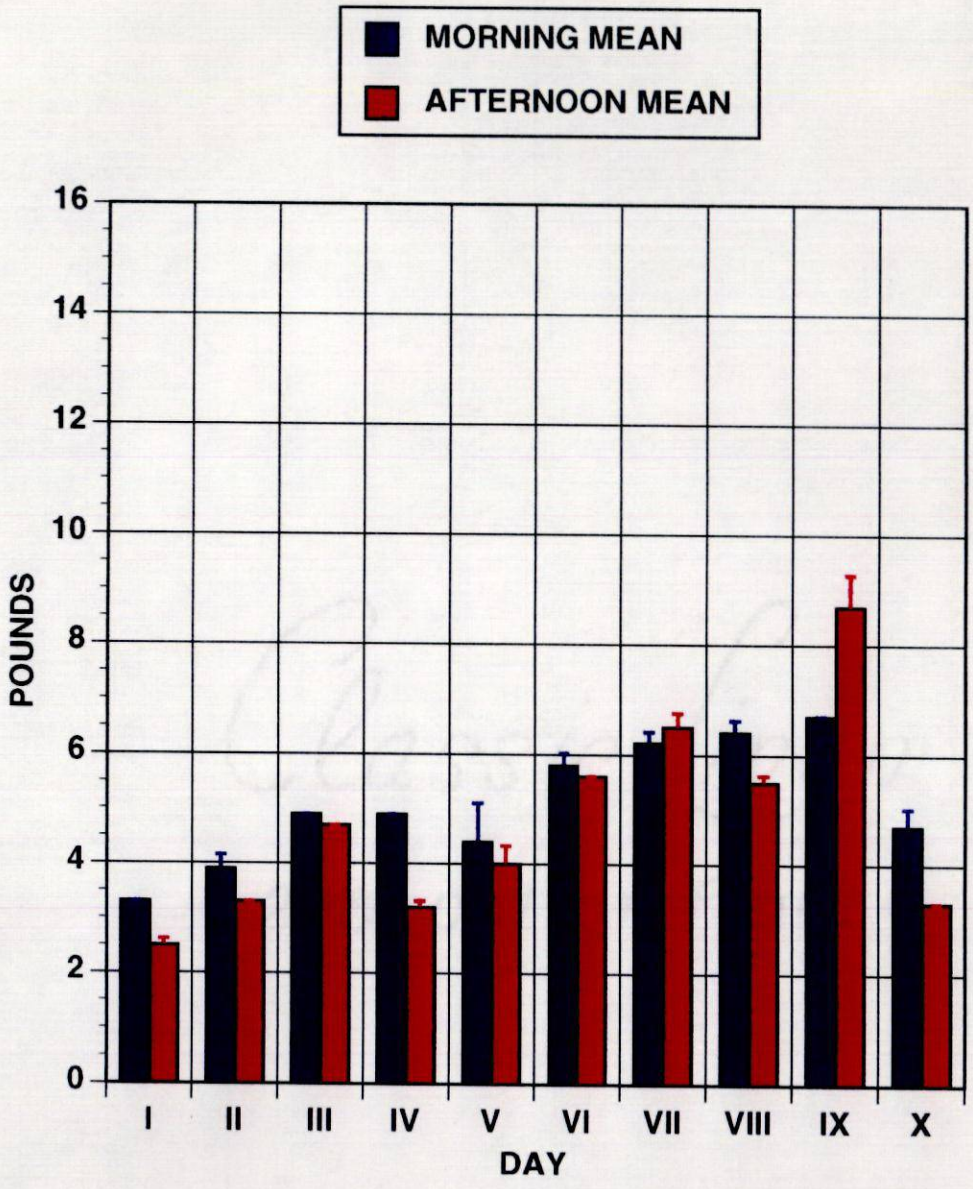


Figure 9. Daily average bite forces right, at T_1 (Morning) and T_2 (afternoon) during alternating, chronic electrical stimulation of the right masseter muscle for Subject 1, Part B

Table 7.--Daily average bite forces left, at T_1 (morning) and T_2 (afternoon) during alternating, chronic electrical stimulation of the left masseter muscle for Subject 1 in Part B

DAY	T_1 (lbs.)	S.D.	T_2 (lbs.)	S.D.	p VALUE	% CHANGE
I	3.40	0.12	3.40	0.12	1.00	0.00
II	3.25	0.12	2.66	0.00	*0.01	-18.20
III	4.21	0.00	3.99	0.22	0.22	-5.20
IV	4.29	0.12	4.07	0.12	0.22	-5.10
V	4.51	0.12	4.58	0.12	0.42	1.60
VI	4.73	0.12	4.58	0.12	0.18	-3.20
VII	5.47	0.67	5.10	0.00	0.44	-6.80
VIII	5.62	0.12	5.18	0.12	0.07	-7.80
IX	6.66	0.00	6.66	0.00	*0.00	0.00
X	6.43	0.22	4.81	0.25	*0.01	-25.20

* Statistically significant difference ($p \leq 0.05$).

Constant: Frequency= 20 Hz, Pulse Duration = 100 µsec., Interpulse Interval = 50 msec, Duty Cycle = Alternating 5 sec on and 5 sec off

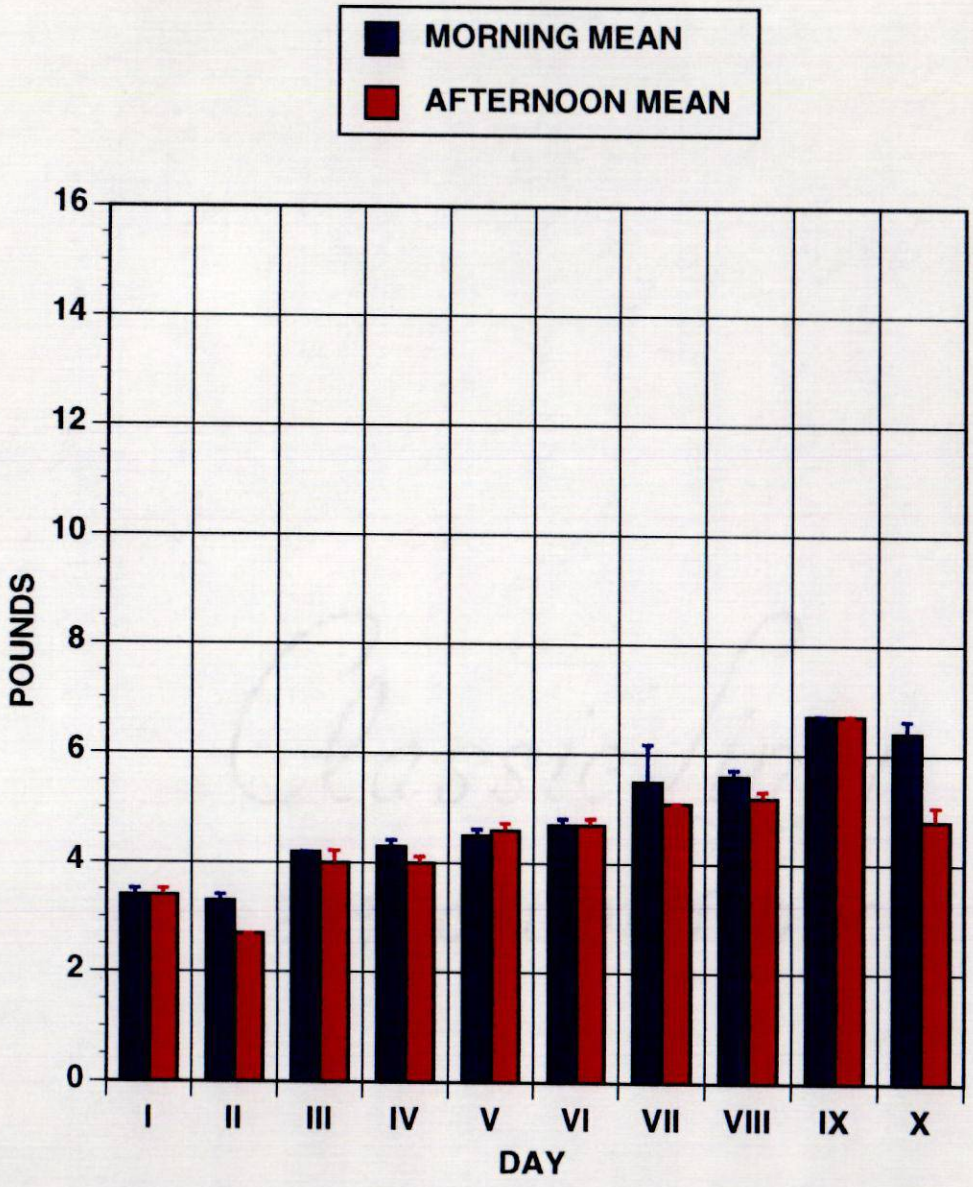


Figure 10. Daily average bite forces left, at T_1 (Morning) and T_2 (afternoon) during alternating, chronic electrical stimulation of the right masseter muscle for Subject 1, Part B

Table 8.--Daily average maximum voluntary bite forces right, before (T_1, morning) and after (T_2, afternoon) alternating, chronic electrical stimulation of the bilateral masseter muscles for Subject 1 in Part B

DAY	T_1 (lbs.)	S.D.	T_2 (lbs.)	S.D.	p VALUE	% CHANGE
I	129.30	1.95	112.44	3.31	*0.00	-13.00
II	119.93	2.56	122.74	3.55	0.10	2.30
III	127.43	3.55	122.27	7.10	0.09	-4.00
IV	122.74	6.10	126.02	3.84	0.40	2.70
V	120.40	3.55	125.08	3.91	0.14	3.90
VI	126.96	9.99	127.90	6.94	0.90	0.70
VII	99.32	3.55	126.49	0.00	*0.00	27.40
VIII	125.55	5.13	123.21	8.04	0.60	-1.90
IX	129.30	1.95	121.34	4.80	*0.03	-6.20
X	124.62	1.95	126.49	7.59	0.54	1.50

* Statistically significant difference ($p \leq 0.05$).

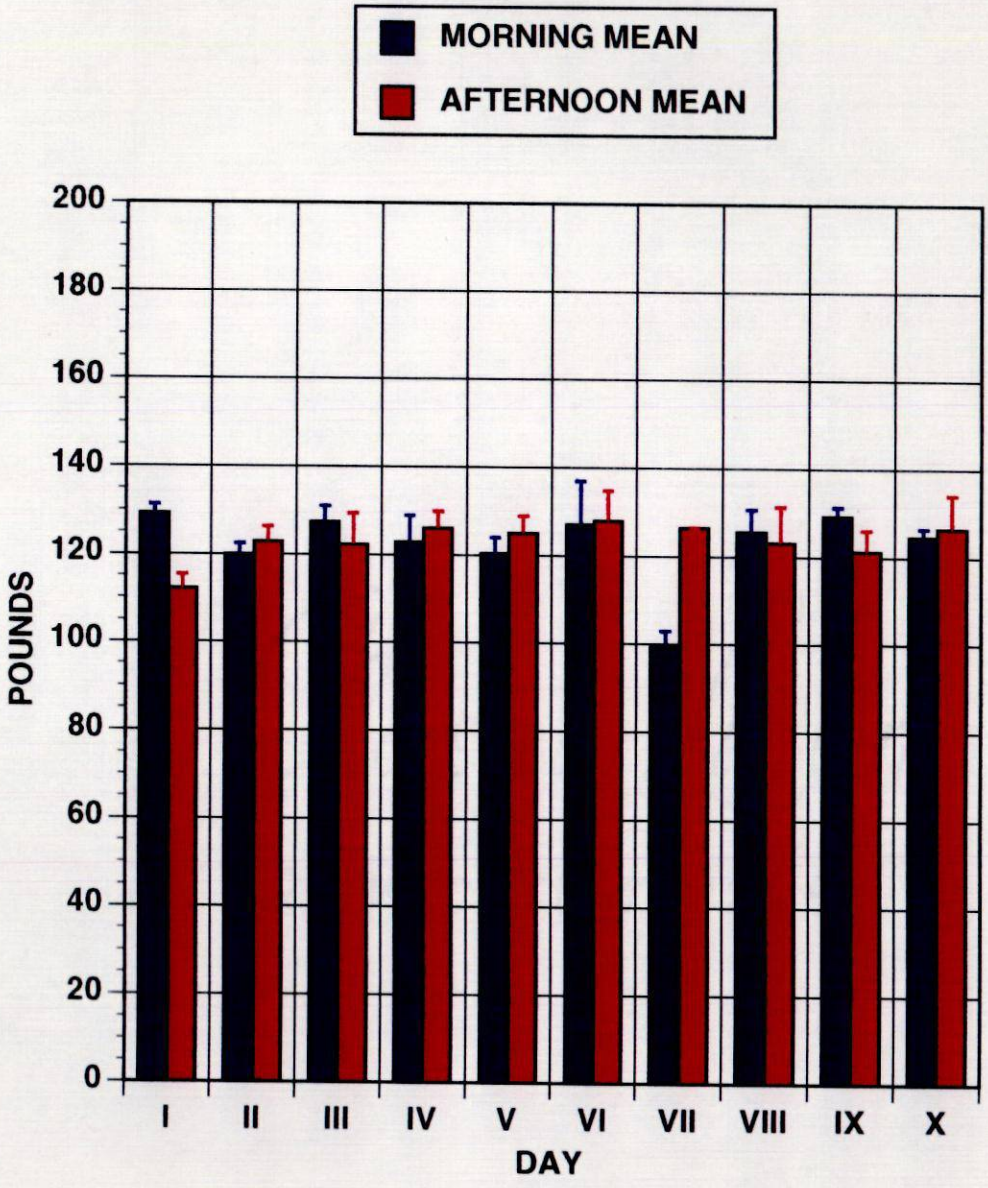


Figure 11. Daily average maximum voluntary bite forces right, before (T_1, morning) and after (T_2, afternoon) alternating, chronic electrical stimulation of the bilateral masseter muscles for Subject 1, Part B

Table 9.--Daily average maximum voluntary bite forces left, before (T_1, morning) and after (T_2, afternoon) alternating, chronic electrical stimulation of the bilateral masseter muscles for Subject 1 in Part B

DAY	T_1 (lbs.)	S.D.	T_2 (lbs.)	S.D.	p VALUE	% CHANGE
I	122.74	3.91	124.62	6.28	0.65	1.50
II	110.09	3.31	108.22	4.19	0.56	-1.70
III	110.09	3.13	108.22	4.19	0.52	-1.70
IV	105.41	11.35	109.62	4.50	0.48	4.00
V	104.47	2.09	108.69	12.01	0.45	4.00
VI	96.97	2.67	104.47	2.67	*0.02	7.70
VII	106.34	4.85	107.28	5.34	0.81	0.90
VIII	112.90	1.95	160.22	11.10	*0.00	41.90
IX	182.24	10.78	172.40	11.30	0.07	-5.40
X	161.16	6.50	178.03	6.62	*0.01	10.50

* Statistically significant difference ($p \leq 0.05$).

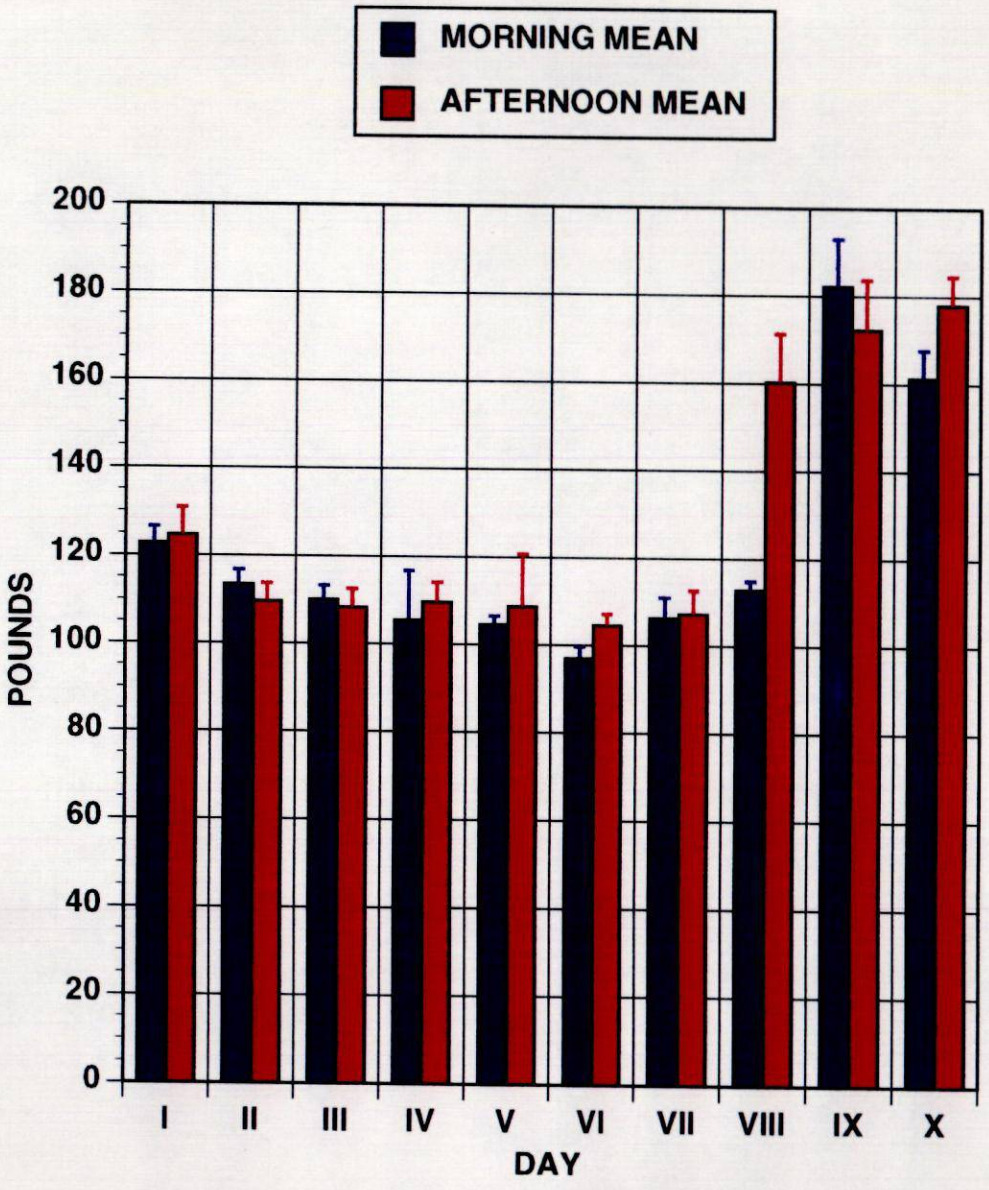


Figure 12. Daily average maximum voluntary bite forces left, before (T_1, morning) and after (T_2, afternoon) alternating, chronic electrical stimulation of the bilateral masseter muscles for Subject 1, Part B

Days I, VII, and IX. A statistically significant decrease in bite force occurred during Days I and IX while Day VII exhibited a significant increase in bite force from T1 to T2.

Maximum Voluntary Bite Forces - Left

Maximum bite forces on the left side during maximum effort exhibited no significant decrease in bite force, however Days VI, VIII, and X showed a significant increase. Bite forces remained comparatively the same from Day I to VII. An increased maximum voluntary bite force occurred on Days IX and X at both T1 and T2.

Group Response

Daily Average and Percentage Change of Stimulated Bite Forces - Right

The results of the tests for all subjects are presented in Table 10 and Figures 13 and 14. Days VI and IX exhibited an increase in bite force, however, all other days showed a decrease in bite force from T1 to T2. Days II, V, and VII demonstrated a statistically significant decrease from T1 to T2 of 12.94, 7.72, and 7.72 respectively. Figure 13 demonstrates the day to day consistent increase at both T1 and T2. For example, average bite force at T1 increased from 3.21 lbs. (SD = 0.27) to 5.53 lbs. (SD = 1.60) during Day IX.

Figure 14 illustrates the relatively constant decrease in bite force from Day I to VIII followed by a sudden increase in bite force on Days IX and X.

Daily Average and Percentage Change of Stimulated Bite Forces - Left

Table 11 and Figure 15 demonstrate the steadily increasing bite force from Day I to X at both T1 and T2. Bite forces recorded in the afternoon increased steadily along

Table 10.--Daily average bite forces right, at T_1 (morning) and T_2 (afternoon) during alternating, chronic electrical stimulation of the right masseter muscle for all Subjects in Part B

DAY	T_1 (lbs.)	S.D.	T_2 (lbs.)	S.D.	p VALUE	% CHANGE	S,D,
I	3.21	0.27	3.02	0.76	0.69	-3.86	34.15
II	3.89	1.06	3.36	0.82	*0.05	-12.94	9.73
III	4.47	0.42	4.13	0.89	0.25	-0.83	14.16
IV	4.43	0.68	4.00	0.86	0.41	-8.52	24.88
V	4.47	0.70	4.12	0.64	*0.05	-7.72	5.34
VI	5.75	1.27	5.85	1.38	0.48	1.84	5.16
VII	6.63	1.41	5.78	1.17	0.47	-8.20	28.88
VIII	5.75	0.95	5.04	0.97	*0.02	-7.72	5.34
IX	5.53	1.60	7.28	1.92	0.18	40.98	58.10
X	4.77	0.85	6.02	3.16	0.36	20.35	44.68

* Statistically Significant Difference ($p \leq 0.05$).

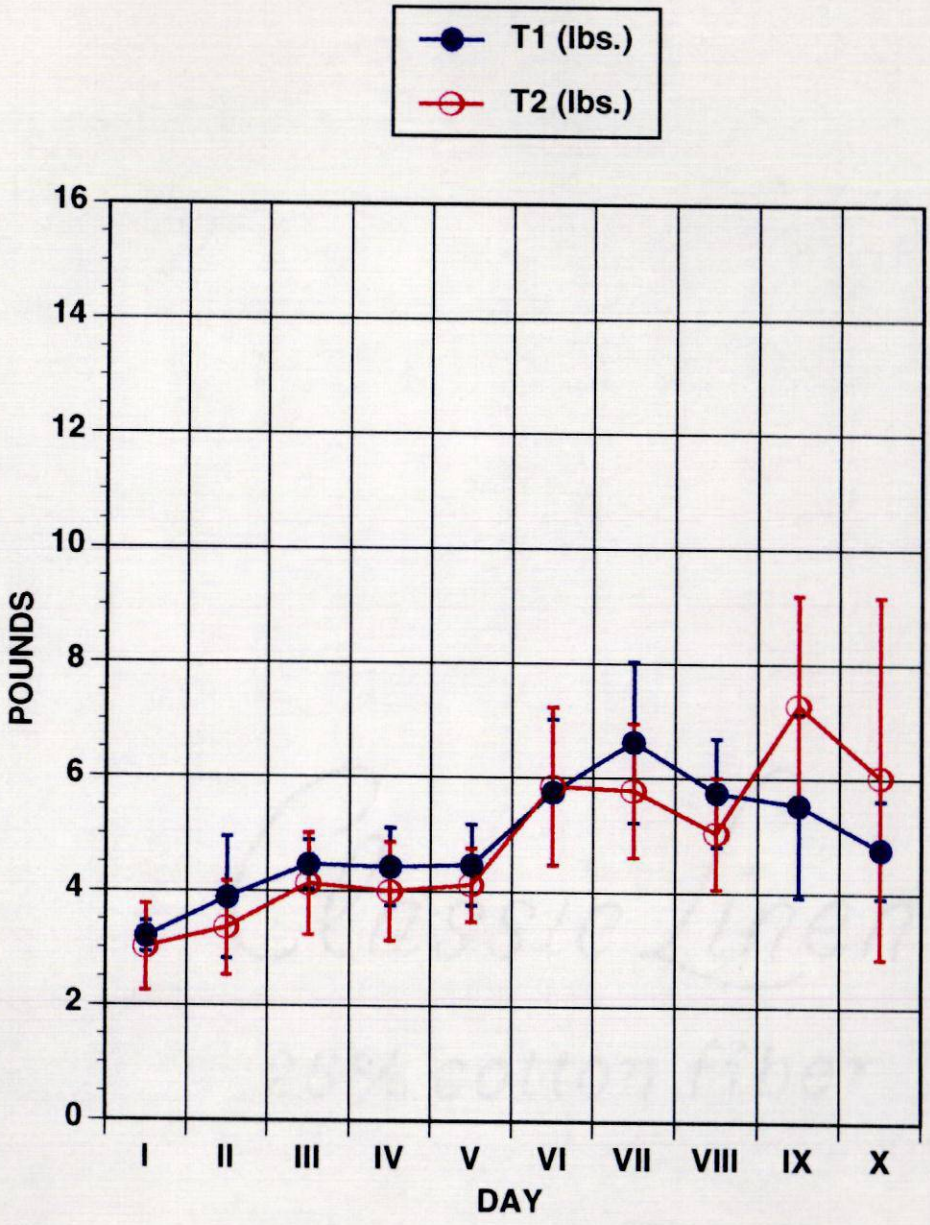


Figure 13. Daily average bite forces right, at T_1 (morning) and T_2 (afternoon) during alternating, chronic electrical stimulation of the right masseter muscle for all Subjects in Part B

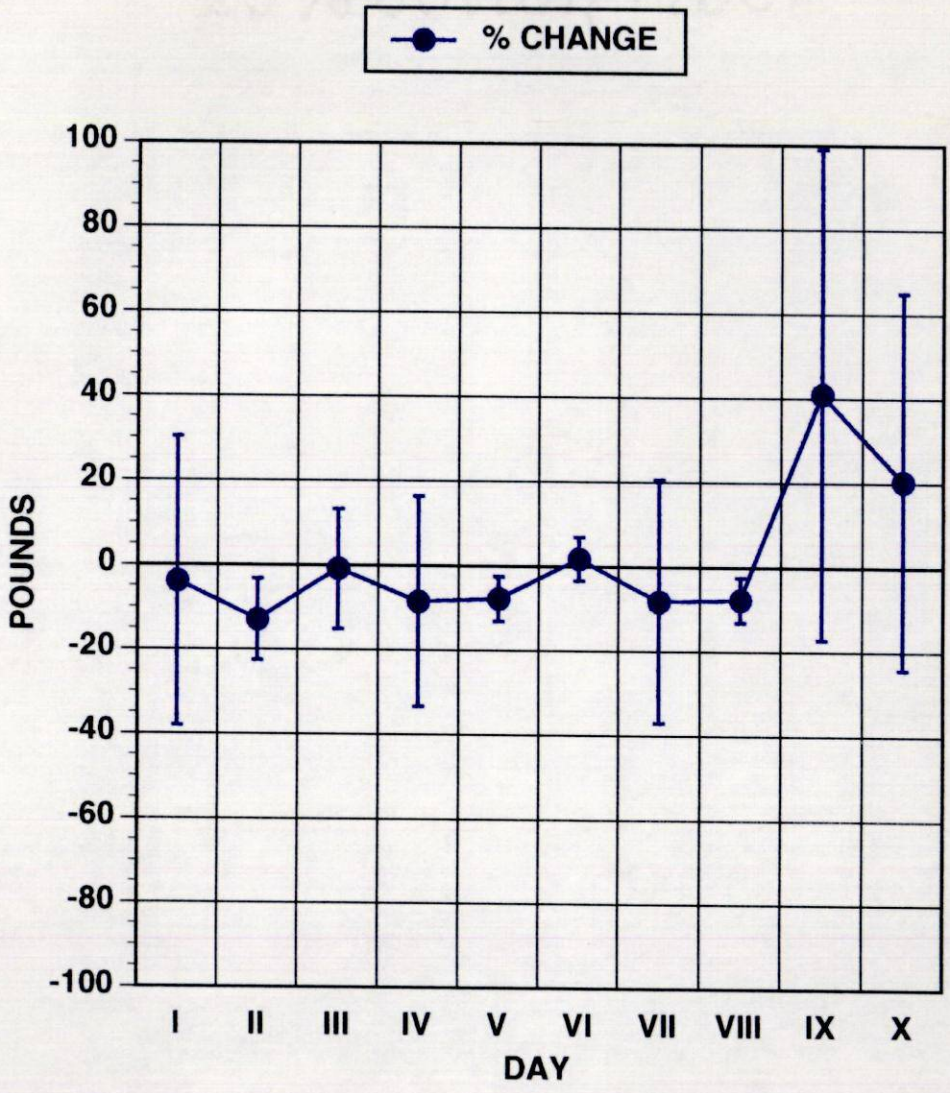


Figure 14. Average % change of daily bite forces right, at T_1 (morning) and T_2 (afternoon) during alternating, chronic electrical stimulation of the right masseter muscle for all Subjects in Part B

Table 11. --Daily average bite forces left, at T_1 (morning) and T_2 (afternoon) during alternating, chronic electrical stimulation of the left masseter muscle for all Subjects in Part B

DAY	T_1 (lbs.)	S.D.	T_2 (lbs.)	S.D.	p VALUE	% CHANGE	S.D.
I	3.53	0.66	3.54	0.91	0.99	3.10	34.67
II	3.60	0.78	2.91	0.37	0.12	-17.04	15.27
III	4.50	0.92	4.13	0.68	0.32	-8.82	15.27
IV	4.35	0.49	3.70	1.06	0.25	-11.14	24.05
V	5.65	2.21	5.63	2.37	O.84	-1.00	18.52
VI	4.47	0.38	3.60	1.07	0.13	14.62	16.45
VII	5.41	1.37	4.96	0.84	0.32	-6.90	14.20
VIII	5.85	1.79	4.51	0.87	0.15	0.30	9.62
IX	6.50	3.11	5.91	2.36	0.32	-9.10	22.39
X	6.24	2.57	5.59	1.59	0.65	-6.74	20.80

* Statistically Significant Difference ($p \leq 0.05$).

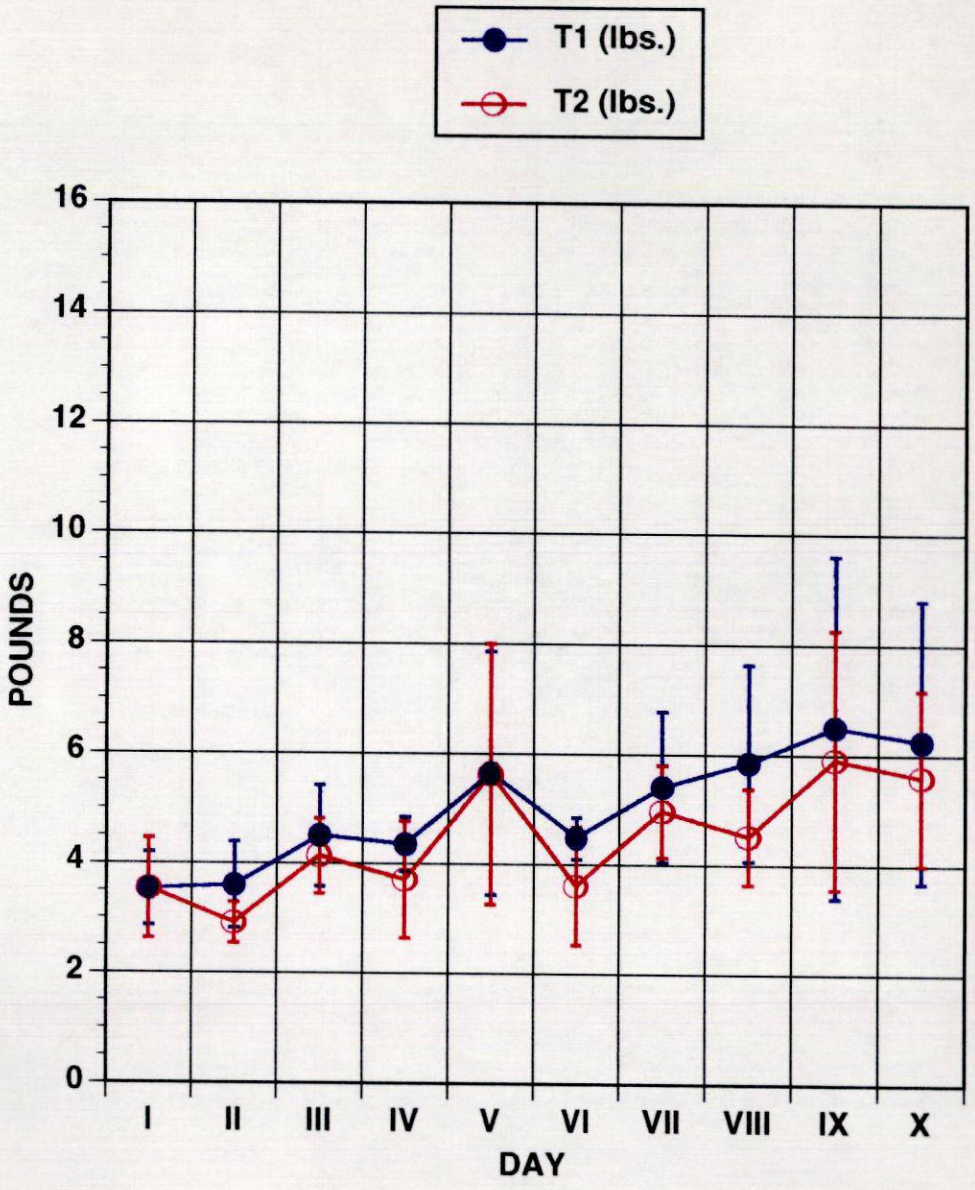


Figure 15. Daily average bite forces left, at T_1 (morning) and T_2 (afternoon) during alternating, chronic electrical stimulation of the left masseter muscle for all Subjects in Part B

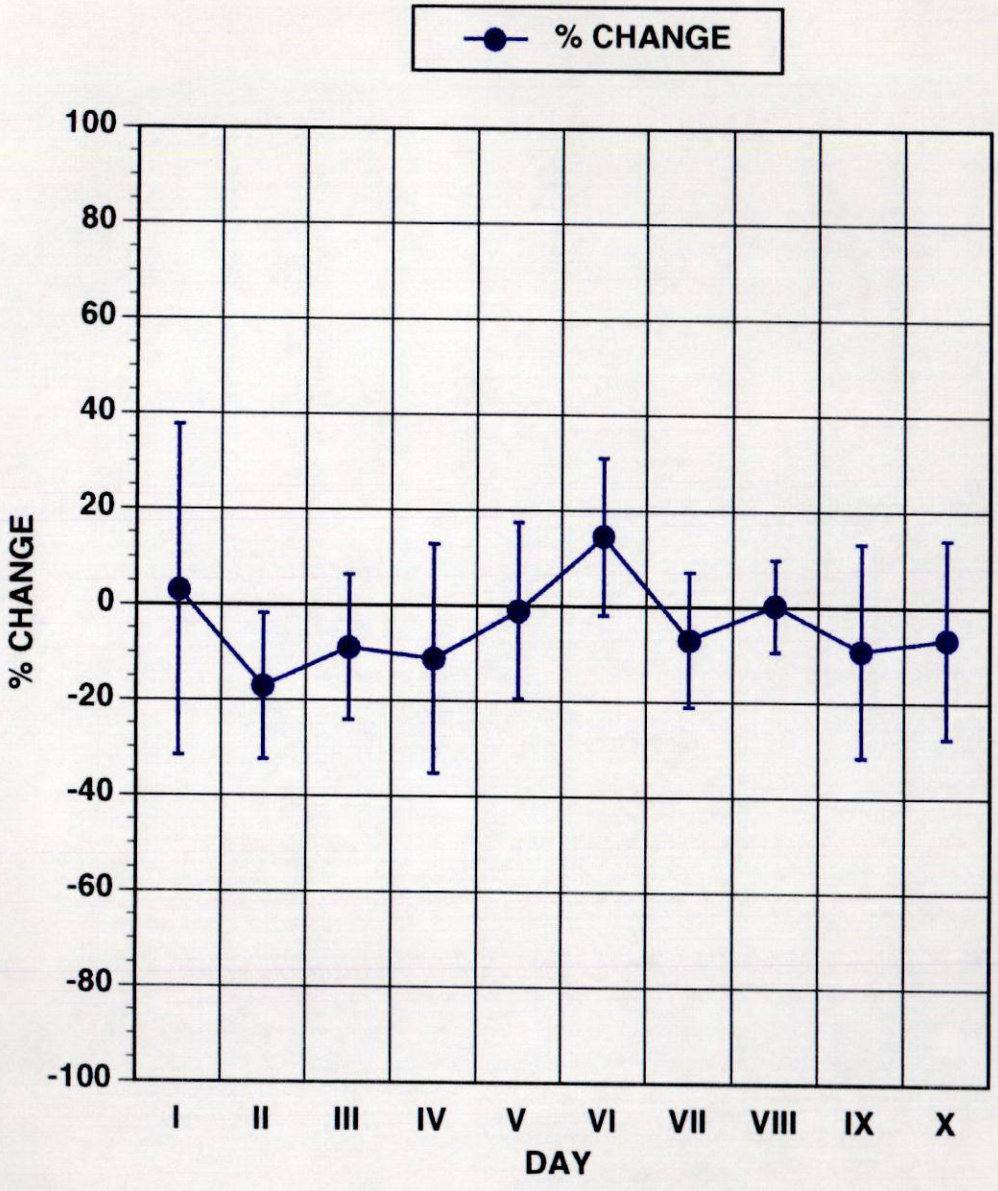


Figure 16. Average % change of daily bite forces left, at T_1 (morning) and T_2 (afternoon) during alternating, chronic electrical stimulation of the left masseter muscle for all Subjects in Part B

with the morning from 3.54 lbs. (SD = 0.91) during Day I to 5.59 lbs. on Day X.

Days II, III, IV, V, VII, IX, and X showed decreased bite forces at T2. No statistically significant increase or decrease in bite force was witnessed during chronic stimulation.

The largest decrease in bite force from T1 to T2 was recorded during Day II (17.04%) while the largest increase occurred on Day VI (14.62%). Figure 16 illustrates the relative variability of percentage change from T1 to T2.

Average % Change of Maximum Voluntary Bite Forces - Right and Left

The average percentage change of daily maximum voluntary bite forces on the right and left, before and after chronic electrical stimulation of bilateral masseter muscles is shown in Table 12 and Figure 17. The percentage change for maximum right indicated a decreased bite force on Days II to VII and X. Maximum voluntary efforts on the left side show a negative percentage change from T1 to T2 on Days IV, VI, VII, and IX of -0.60, -4.86, -0.08, and -6.90 respectively.

Electromyography

Electromyographic recordings were performed on four subjects in Part A and two subjects in part B. Results are shown on Table 13. The anterior temporalis muscle was recruited only in one subject (subject 1, Part A). However, the temporalis muscle was never recruited during voluntary mouth opening.

Subjective Pain Evaluation

All subjects in Part A and Part B were asked to complete the McGill pain questionnaire and a pain intensity scale at different times during the course of the study.

Table 12.--Average % change of daily maximum voluntary bite forces, right and left, before (T_1, morning) and after (T_2, afternoon) alternating, chronic electrical stimulation of the bilateral masseter muscles for all Subjects in Part B

DAY	% CHANGE (MAX. R)	S.D.	% CHANGE (MAX. L)	S.D.
I	12.02	18.12	16.24	37.61
II	-19.68	15.49	3.08	12.74
III	-10.66	20.28	0.48	2.17
IV	-6.40	8.36	-0.60	11.10
V	-13.76	6.49	2.74	14.02
VI	-7.30	4.62	-4.86	8.13
VII	-7.64	11.60	-0.08	9.47
VIII	-2.72	12.22	4.60	6.23
IX	1.38	9.87	-6.90	14.21
X	-16.46	26.55	0.30	9.63

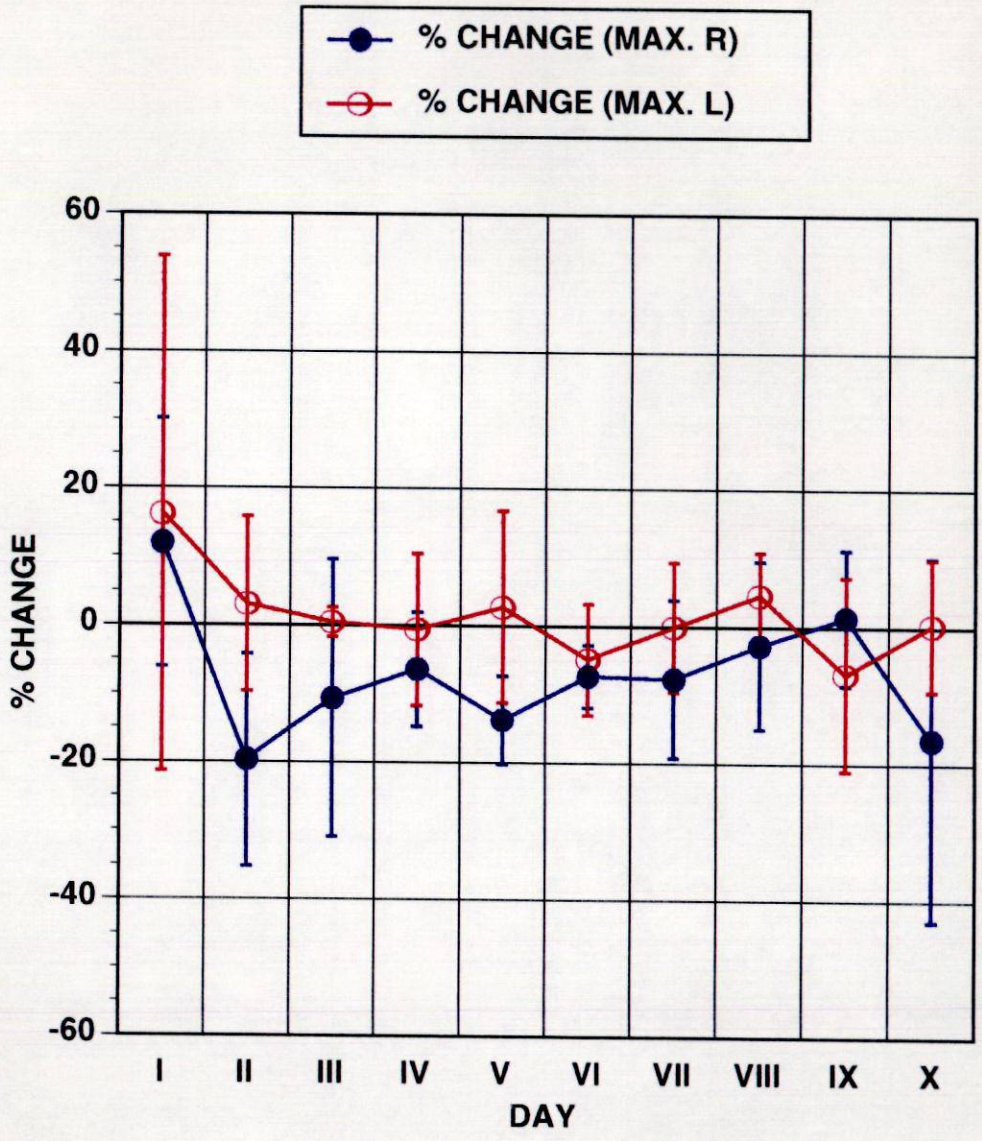


Figure 17. Average % change of daily maximum voluntary bite forces, right and left, before (T_1, morning) and after (T_2, afternoon) alternating, chronic electrical stimulation of the bilateral masseter muscles for all Subjects in Part B

Table 13.-- Electromyographic results of the right temporalis muscle following simultaneous or alternating, chronic electrical stimulation of the bilateral masseter muscles in Part A and Part B

	RELAXED POSITION		MOUTH OPENING	
SUBJECT	YES	NO	YES	NO
1	√			√
2		√		√
3		√		√
4		√		√
5		√		√
6		√		√

Table 14 depicts the results from subjects 1 and 2 in Part A that received no local anesthetic at the time of electrode insertion. The pain rating index (based on numerical values assigned to each word descriptor) indicates a mean of 17.0 during electrode insertion followed by man values of 5.0 and 4.0 during Day I of chronic electrical stimulation and electrode removal respectively. The present pain intensity (based on a 1- 5 intensity scale) indicates the greatest discomfort during electrode insertion followed by Day I of chronic electrical stimulation and electrode removal.

The remaining three subjects in part A received local anesthetic at the site of electrode insertion. Results in Table 15 indicate the largest mean value of the pain rating index during electromyography followed by electrode insertion with values of 11.0 and 9.3 respectively. Present pain intensity values indicate the largest discomfort during electrode removal.

Table 16 depicts the McGill questionnaire results for subjects in Part B. All Subjects received local anesthetic at the site of electrode insertion. The pain rating index indicates the largest subjective pain during Day I of electrode insertion followed by electrode insertion with mean values of 6.4 and 4.8 respectively. However, the mean values of pain intensity reveal equal increased discomfort during electrode insertion and removal.

The number of subjects charting each word descriptor is shown in Table 17. The most popular words chosen for all groups were pricking, sharp, and pinching.

Table 14.-- McGill Pain Questionnaire results for Subjects in Part A that received no local anesthesia at the time of electrode insertion

VARIABLE	n	TIME	MEAN
		ELECT. INS.	17.0
PAIN		DAY 1	5.0
DESCRIPTOR,77		DAY 5	0.5
(RANGE 0-74)		DAY 10	1.5
		EMG	1.0
	2	ELECT. REM.	4.0
		ELECT. INS.	4.0
PAIN		DAY 1	2.5
INTENSITY		DAY 5	0.5
(RANGE 0-5)		DAY 10	0.0
		EMG	0.0
	2	ELECT. REM.	1.0

Table 15.-- McGill Pain Questionnaire results for Subjects in Part A that received local anesthesia at the time of electrode inserction

VARIABLE	n	TIME	MEAN
		ELECT. INS.	9.3
PAIN		DAY 1	3.0
DESCRIPTOR, 77		DAY 5	1.3
(RANGE 0-74)		DAY 10	1.0
		EMG	11.0
	3	ELECT. REM.	3.6
		ELECT. INS.	1.0
PAIN		DAY 1	0.6
INTENSITY		DAY 5	0.0
(RANGE 0-5)		DAY 10	0.0
		EMG	1.0
	3	ELECT. REM.	1.3

Table 16.-- McGill Pain Questionnaire results for Subjects in Part B

VARIABLE	n	TIME	MEAN
		ELECT. INS.	4.8
PAIN		DAY 1	6.4
DESCRIPTOR,77		DAY 5	1.8
(RANGE 0-74)		DAY 10	2.0
		EMG	1.8
	5	ELECT. REM.	4.6
		ELECT. INS.	1.2
PAIN		DAY 1	0.8
INTENSITY		DAY 5	0.0
(RANGE 0-5)		DAY 10	0.0
		EMG	0.2
	5	ELECT. REM.	1.2

Table 17.-- McGill Pain Questionnaire word descriptors by number of Subjects selecting each descriptor in Part A and Part B

WORD	A NO A.	A	B	WORD	A NO A.	A	B
PRICKING	1	2	4	DULL	1	0	0
SHARP	1	3	3	PINCHING	2	1	3
HURTING	1	1	0	STABBING	0	1	0
RASPING	1	0	0	CRUSHING	0	1	0
INTENSE	1	1	0	HEAVY	0	2	0
PENETRAT-ING	2	0	1	TENDER	0	1	2
TEARING	1	0	1	TROUBLE-SOME	0	1	1
TUGGING	2	1	1	TINGLING	0	1	1
ANNOYING	1	1	1	PRESSING	0	1	3
TIGHT	1	1	1	THROBBING	0	1	0
CUTTING	1	0	0	SPLITTING	0	1	0
PULLING	1	1	3	FEARFUL	0	1	1
SHOOTING	1	1	1	PIERCING	0	1	1
LANCINAT-ING	1	1	0	SQUEEZING	0	1	0
LACERATING	1	0	1	BURNING	0	0	1
STINGING	1	0	2	TIRING	0	0	1
FLICKERING	1	0	2	RADIATING	0	0	1
BORING	1	0	2	NAUSEATING	0	0	1
ITCHY	1	1	1	QUIVERING	0	0	2
SORE	1	1	2	JUMPING	0	0	1
NAGGING	1	0	1	NUMB	0	0	2

DISCUSSION

The purpose of this investigation was to determine: 1) the fatigue properties of the masseter following chronic electrical stimulation; 2) the selectivity of electrical stimulation; and, 3) the subjective pain sensation during the course of the experiment. The design of the experiment also made it possible to draw inferences on the clinical application of electrical stimulation for the treatment of vertical dysplasia in growing children.

Several extensive studies have been conducted on the histochemical properties of the craniomandibular muscles (Ringqvist, 1974; Eriksson et al., 1981; Eriksson and Thornell, 1983). The masseter muscle is composed mainly of type I fibers accompanied by a lower frequency of type IIA (type IM) and type IIB fibers. Tuxen et al. (1992) histochemically characterized muscle fibers from biopsies of the anterior part of the superficial masseter muscle in 13 healthy females, aged 21-28 years old. The relative mean fiber area of muscle fibers were: type I 82.21%; type IM 9.5%; and, type II 8.3%.

Previous studies have been concerned primarily with the development, growth, and form of the craniofacial skeleton. None has been concerned with concomitant adaptations within the muscles of mastication of the adult human following chronic electrical stimulation. In the present experiment morning and afternoon bite force measurements were taken daily for ten days to determine the fatigue properties of the masseter muscles following chronic electrical stimulation.

Analysis of our results in Part A and B reveal a generalized increase of stimulated bite force from Day I to X while maximum voluntary bite forces remained relatively constant over the same ten days. For example, average daily bite force on the right side immediately following alternating, chronic electrical stimulation of the right masseter muscle for all subjects in Part B increased from 3.21 pounds at T1, Day I to 4.17 pounds at T1, Day X. These results agree with Guelinckx et al. (1986) who found an increase in maximum stimulated bite force over the course of 8 weeks following masseter removal and stimulation of the superficial temporalis muscle in controls and experimental groups of rabbits. An increase in the percent cross sectional area composed of fast fatigue resistant fibers, a slower time to reach twitch tension, and a decrease in fatiguability were observed by Guelinckx et al. (1986). They concluded that muscles used for highly repetitious activities with submaximal loadings adapt to increased functional requirements by increasing fatigue resistant fibers.

More recently, stimulation paradigms employing bursts of higher frequency stimulation have yielded muscles that can produce more force than those stimulated at a continuous rate at a lower frequency, with the same number of stimuli. One possible explanation for this is that the muscle responds to an increased load as well as increased activity.

Following a 30 Hz burst of phasic stimulation, all stimulated muscles showed evidence of conversion on the anterior tibialis muscles of cats (Ferguson et al., 1989). The combination of 30 Hz and 24 hour per day produced twice the average sustained tension achieved by the combination of 10 Hz and 8 hour per day.

The effect of induced exercise, histochemically, is an apparent decrease in fast

twitch-glycolytic fibers and a concomitant increase in fast twitch-oxidative and slow twitch-oxidative fibers (Peckham et al., 1973; Pette et al., 1976; 1975; 1973; Riley and Allin, 1973).

Endurance training seems to increase the frequency of type IIA fibers (Salmons and Henriksson, 1981). A large number of type IIA fibers thus suggests intense and frequent jaw muscle activity on behalf of a specific motoneuron pool. It may indicate individual differences in the jaw muscle contraction pattern related to craniofacial morphology (Moller, 1960; Ingervall and Thilander, 1974).

In adult animals the motorneurons innervating slow twitch skeletal muscle generate a sustained low frequency pattern of activity. In contrast, motorneurons supplying fast twitch muscle subject them to intermittent bursts of more intense activity (Eccles et al., 1958). The characteristic physiological and biochemical properties of muscle emerge late in development, usually after birth, and in slow muscle this seems to depend on the establishment of the adult pattern of motoneuron activity. If the motoneurons are rendered quiescent by isolation of the spinal cord a few days after birth, muscles which would have normally have acquired slow contractile characteristics develop as fast muscles (Buller et al., 1960). This capacity for change would enable a muscle to accommodate to changing functional demands. According to this view the change resulting from chronic stimulation of muscle in the adult represents an adaptive response to bursts of higher frequency stimulation.

Fatigue is an integral part of all forms of physical activity and the factors responsible for it start to appear immediately after the onset of exercise. When one makes a sustained maximal effort the force that can be developed starts to fall at once.

Work at submaximal levels, whether it is sustained or intermittent, can be carried on at the same rate for some time before either the speed or intensity is reduced (Bigland-Ritchie et al., 1986). The rate at which fatigue develops depends on the force generated, how long each contraction is held, and the period of rest between contractions. These factors also determine the degree to which blood supply is impeded and the rates of substrate utilization.

Stimulated bite forces were evaluated in all subjects to determine the level of masseter muscle fatigue following chronic electrical stimulation.

Part A of the study involved simultaneous, chronic electrical stimulation of the bilateral masseter muscle. Subject 1 experienced a decrease in stimulated bite force on all days, however, only Days IV and X had a statistically significant difference between T1 and T2 (Table 3). The observed decrease in bite force indicatied minimal muscle fatigue.

Part B of the study involved alternating, chronic electrical stimulation of the bilateral masseter muscles. Stimulated bite forces were recorded twice daily (morning and afternoon) to determine the level of muscle fatigue. A statistically significant decrease in stimulated bite force for subject 1 occurred on the right side on Days I, II, III, IV, and VII (Table 6). Left stimulated bite forces decreased significantly between T1 and T2 on Days II and X (Table 7). Again, results indicating minimal muscle fatigue.

Daily average bite forces, right and left, at T1 and T2 during alternating, chronic electrical stimulation of the right and left masseter muscles for all subjects in Part B are presented in Tables 10 and 11. Our results indicate that only 3 days (II, V, and VIII)

among all subjects had a statistically significant decrease in stimulated bite force. These observations only occurred for stimulated right bite forces.

Rasmussen et al. (1977) states that the high degree of substrate depletion, blood flow impairment, and a simultaneous decrease in oxygen transport to the contracted muscle in combination with a prominent lactate accumulation, may include a decrease in bite force production. A high pre-contractional content of glycogen, as revealed histochemically (Strom, 1990) and architectural factors such as pinnation and fiber type direction (Strom, 1986; 1988) may contribute to the longer endurance times. Differences in the distribution of fiber types can also be a critical factor in explaining differences in bite force production. Ferguson et al. (1989) stated that the fatigue resistance of an electrically activated muscle can be markedly improved by subjecting it to electrically induced exercise.

Subjective Pain Evaluation

Pain is viewed as a normal reaction to an abnormal stimulus or as an abnormal response to a normally harmless stimulus (Rasmussen, 1965). Rasmussen further describes pain as an intense feeling of discomfort or displeasure in a localized part of the human organism.

The purpose of this study was to describe the study subjects' perception of pain at different times (electrode insertion, Day I, V, and X of chronic electrical stimulation, electromyographic study, and electrode removal) during the course of the study. This enabled us to determine one of the parameters necessary for clinical application. The relationship of work descriptors chosen by subjects in Part A (with and without local

anesthesia) and part B was determined. Pricking, sharp, and pinching were the word descriptors most often chosen in the study. The above descriptors were most often chosen during electrode insertion at the electromyographic study and electrode insertion during chronic electrical stimulation.

Subjects in Part A which received no local anesthesia experienced significantly more discomfort compared to all other subjects that received local anesthesia before electrode insertion. The use of local anesthesia before electrode insertion is an important reccommendation to reduce discomfort prior to electrode insertion.

A significant decrease in the pain rating index and present pain intensity scales was experienced by subjects as electrical stimulation progressed from Day I to X. This was attributed to the patients accommodation to the electrical stimuli, described most often as a pressure sensation.

The pain threshold of the skin of the human subject determined by electrical stimulation is almost the same throughout the body, but the face and neck exhibit the relatively lowest values (Notermans, 1966).

Electromyography

Electromyographic recordings were made for four subjects in Part A of the experiment and two subjects in part B. The recordings from the right anterior temporalis muscle were preformed using concentric intramuscular recording electrodes. Electromyography was used to determine the selectivity of muscle contraction through electrical stimulation. This allowed the determination of whether the decrease or increase in bite force was strictly due to masseter muscle activity.

Only one subject experienced activity of the temporalis muscle. This occurred in the relaxed mandibular position. This activity was attributed to a minor level of apprehension or anxiety since no activity was noted during mouth opening. Mortimer (1981) designates the electric field generated by an intramuscular electrode as roughly circular and approximately 2 cm in diameter. The border of the circular area represents the hypothetical threshold limits. At distances greater than these limits the intensity of the stimulus field is less than threshold for nerve excitation. All motor nerves in this area become active upon stimulation and these nerve fibers in turn excite their respective muscle fibers.

The present results are consistent with the assumption that functional electrical stimulation can be used as an orthodontic tool involving minimal muscle fatigue, good muscle selectivity, and little discomfort.

The sample was chosen not to include events in facial growth. The technique for electrode insertion proved to be successful in that, of the ten subjects only one subject

showed signs of infection. The area over the mid-belly of the right masseter muscle of subject 2 in Part Bwas edematous and erythematous following Day VIII of chronic electrical stimulation. No trismus was present and the infection was localized. The electrode was withdrawn immediately and the patient was prescribed Keflex 500 mg for seven days. Over the course of four days the infection resolved.

The accuracy of the present study depended on variations in: the anatomic dimensions of the mandible, muscle origin and insertion, the skill and precision with which the electrodes were inserted, the overall precision of the bite force transducer, the chart recorder, the amplifier, and fabrication of occlusal stents. Since variation of the anatomical components in humans can not be controlled, error is unavoidable. The accuracy of the present study depended on variations in: the anatomic dimensions of the mandible, muscle origin and insertion, the skill and precision with which the electrodes were inserted, the overall precision of the bite force transducer, the chart recorder, the amplifier, and fabrication of occlusal stents. Since variation of the anatomical components in humans can not be controlled, error is unavoidable.

CONCLUSION

Functional Electrical Stimulation (FES) has been used to treat several orthopedic conditions. To determine the fatigue properties of the masseter muscle, thin, coiled, multistranded electrodes were placed bilaterally in the masseter muscle in two groups of adult volunteers. A portable pulse generator (Neu Rx Is/4) was used to stimulate the masseter muscle at half the milli-amperage of pain threshold in five subjects. In a second group of five subjects the milli-amperage was adjusted to an initial three pound interocclusal force. Morning and afternoon bite forces were recorded daily for ten days. Results indicated that a bite force of the milli-amperage of half pain threshold and a bite force of three pounds can be sustained over a period of two weeks using this method. Pain and discomfort was rated minimal and muscle stimulation was selective. In conclusion, the use of FES to treat malocclusion is theoretically possible and technically feasible.

APPENDIX I

Table 18.--Daily average bite forces at T_1 (morning) and T_2 (afternoon) during simultaneous, chronic electrical stimulation of the bilateral masseter muscles for Subject 2 in Part A

DAY	T_1 (lbs.)	S.D.	T_2 (lbs.)	S.D.	p VALUE	% CHANGE
I	3.97	0.68	2.93	0.43	0.06	-26.20
II	3.30	0.12	2.93	0.62	0.25	-11.20
III	3.84	0.32	3.77	0.32	0.77	-1.80
IV	5.7	0.36	4.59	0.38	*0.00	-19.50
V	5.77	0.58	5.21	0.65	0.13	-9.70
VI	7.12	0.91	5.70	0.59	*0.02	-19.90
VII	8.12	1.12	6.61	0.55	*0.03	-18.60
VIII	7.01	0.25	7.01	0.25	0.50	0.00
IX	6.83	0.48	7.10	0.72	0.49	4.00
X	6.48	0.37	7.23	0.28	*0.00	11.60

* Statistically significant difference ($p \leq 0.05$).

Constant: Frequency= 20 Hz, Pulse Duration = 100 μsec., Interpulse Interval = 50 msec, Duty Cycle = Simultaneous 15 sec on and 5 sec off

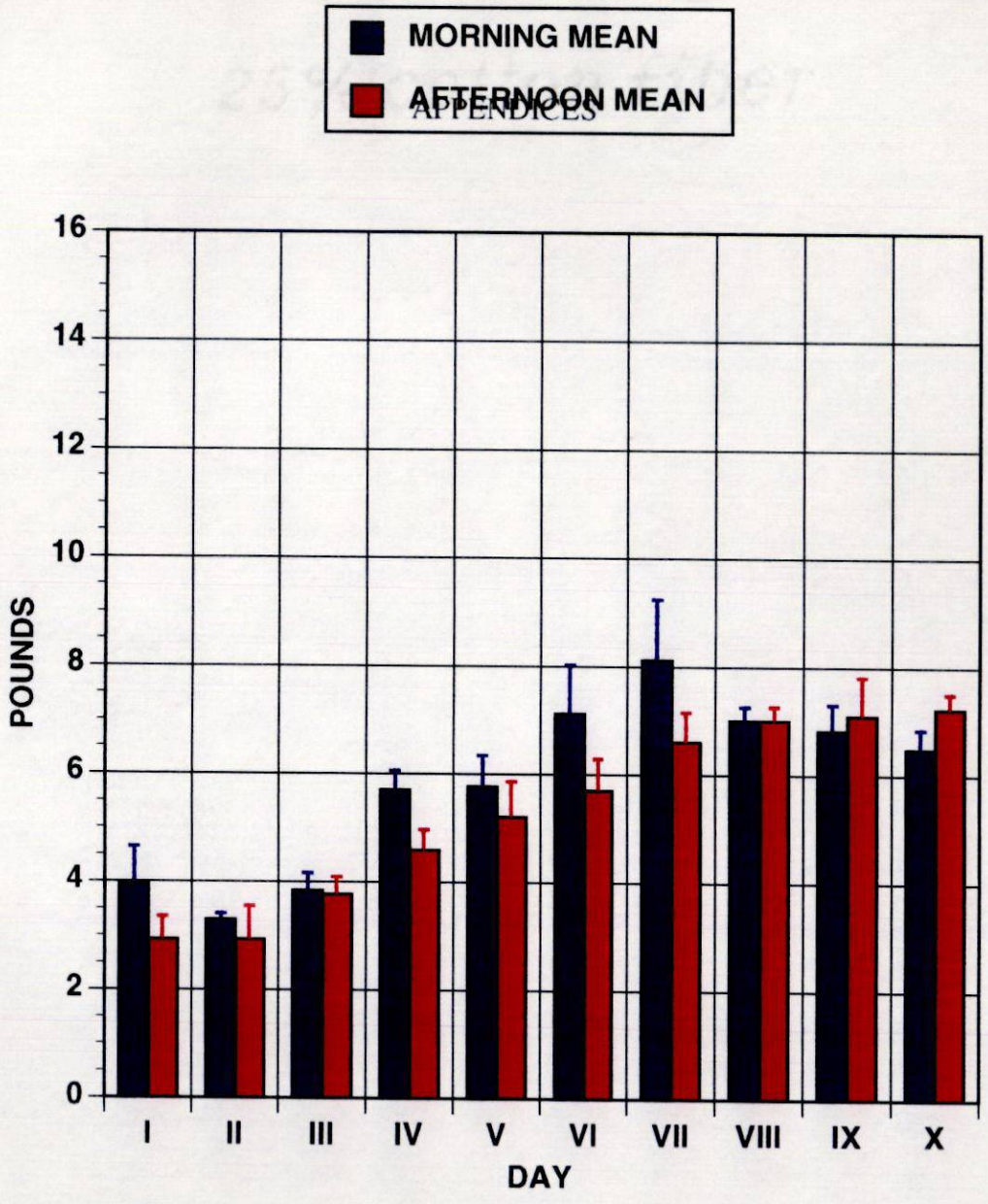


Figure 18. Daily average bite forces during simultaneous, chronic electrical stimulation of the bilateral masseter muscles for Subject 2, Part A

Table 19.--Daily average maximum voluntary bite forces before (T_1, morning) and after (T_2, afternoon) simultaneous, chronic electrical stimulation of the bilateral masseter muscles for Subject 2 in Part A

DAY	T_1 (lbs.)	S.D.	T_2 (lbs.)	S.D.	p VALUE	% CHANGE
I	173.81	7.66	160.22	11.06	0.12	-7.80
II	121.81	17.13	133.05	9.13	0.19	9.20
III	133.99	19.13	126.49	12.93	0.46	-5.60
IV	132.11	13.00	121.81	7.40	0.14	-7.80
V	142.42	5.34	141.08	6.28	0.47	-0.90
VI	161.63	4.05	171.47	6.06	*0.01	6.10
VII	145.23	12.61	171.93	12.67	*0.03	18.40
VIII	173.34	6.82	175.21	3.84	0.67	1.10
IX	132.58	3.55	135.39	4.50	0.34	2.10
X	195.36	5.64	194.42	4.05	0.58	-0.50

* Statistically significant difference ($p \leq 0.05$).

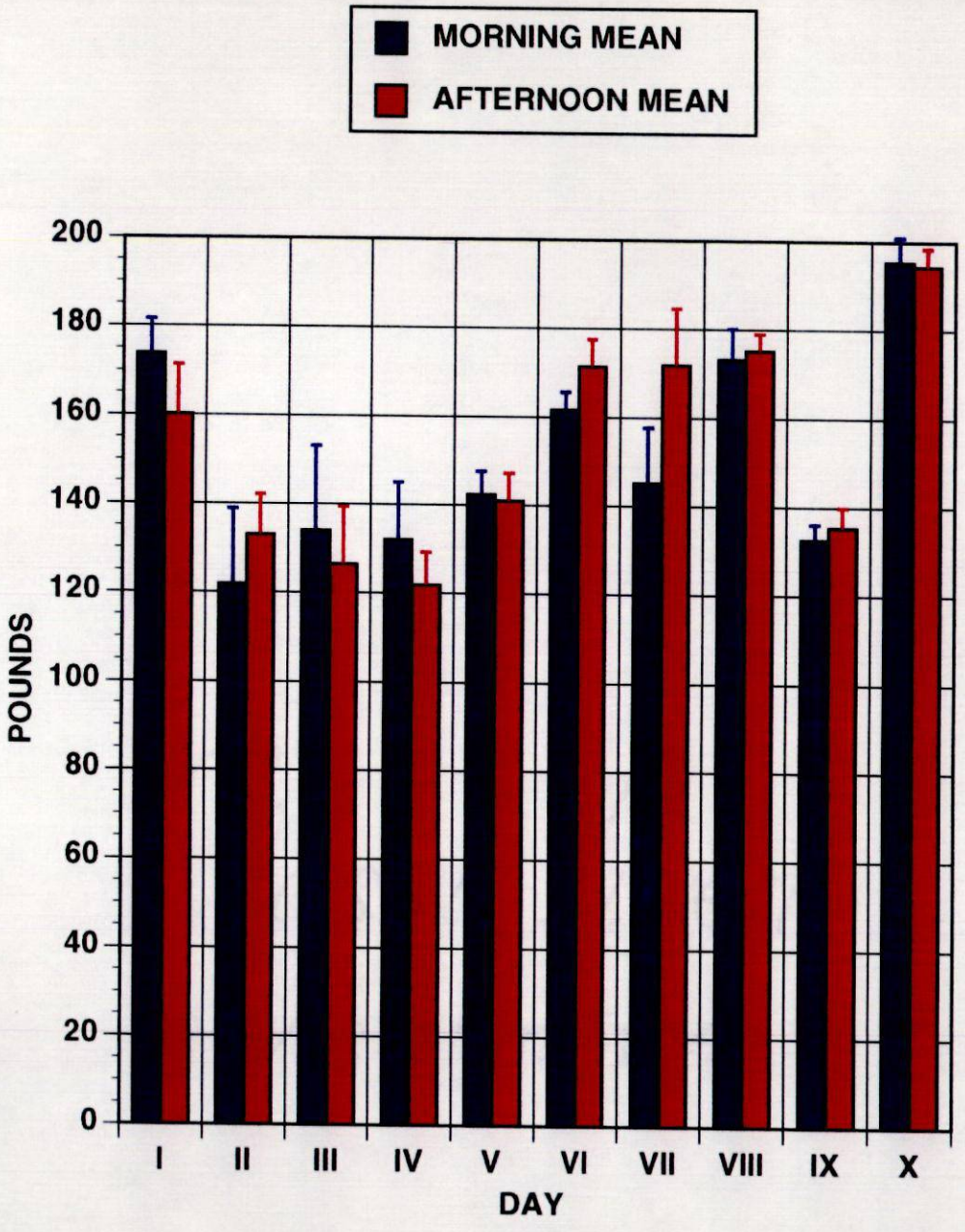


Figure 19. Daily average maximum bite forces before and after simultaneous, bilateral chronic electrical stimulation of the masseter muscles for Subject 2, Part A

Table 20.--Daily average bite forces at T1 (morning) and T2 (afternoon) during simultaneous, chronic electrical stimulation of the bilateral masseter muscles for Subject 3 in Part A

DAY	T_1 (lbs.)	S.D.	T_2 (lbs.)	S.D.	p VALUE	% CHANGE
I	5.49	0.79	6.71	0.30	0.40	22.20
II	10.32	0.49	11.10	0.39	*0.00	7.60
III	7.54	0.30	7.65	0.60	0.74	1.50
IV	10.48	0.41	9.10	0.49	*0.02	-13.20
V	9.65	0.63	9.43	0.87	0.70	-2.30
VI	13.48	0.37	12.37	0.99	0.13	-8.20
VII	11.43	0.84	10.21	0.77	0.06	-10.70
VIII	14.31	0.31	13.43	0.72	*0.03	-6.10
IX	13.20	0.57	10.71	0.37	*0.00	-18.90
X	11.48	0.37	11.26	0.46	0.47	-1.90

* Statistically significant difference ($p \leq 0.05$).

Constant: Frequency= 20 Hz, Pulse Duration = 100 µsec., Interpulse Interval = 50 msec, Duty Cycle = Simultaneous 15 sec on and 5 sec off

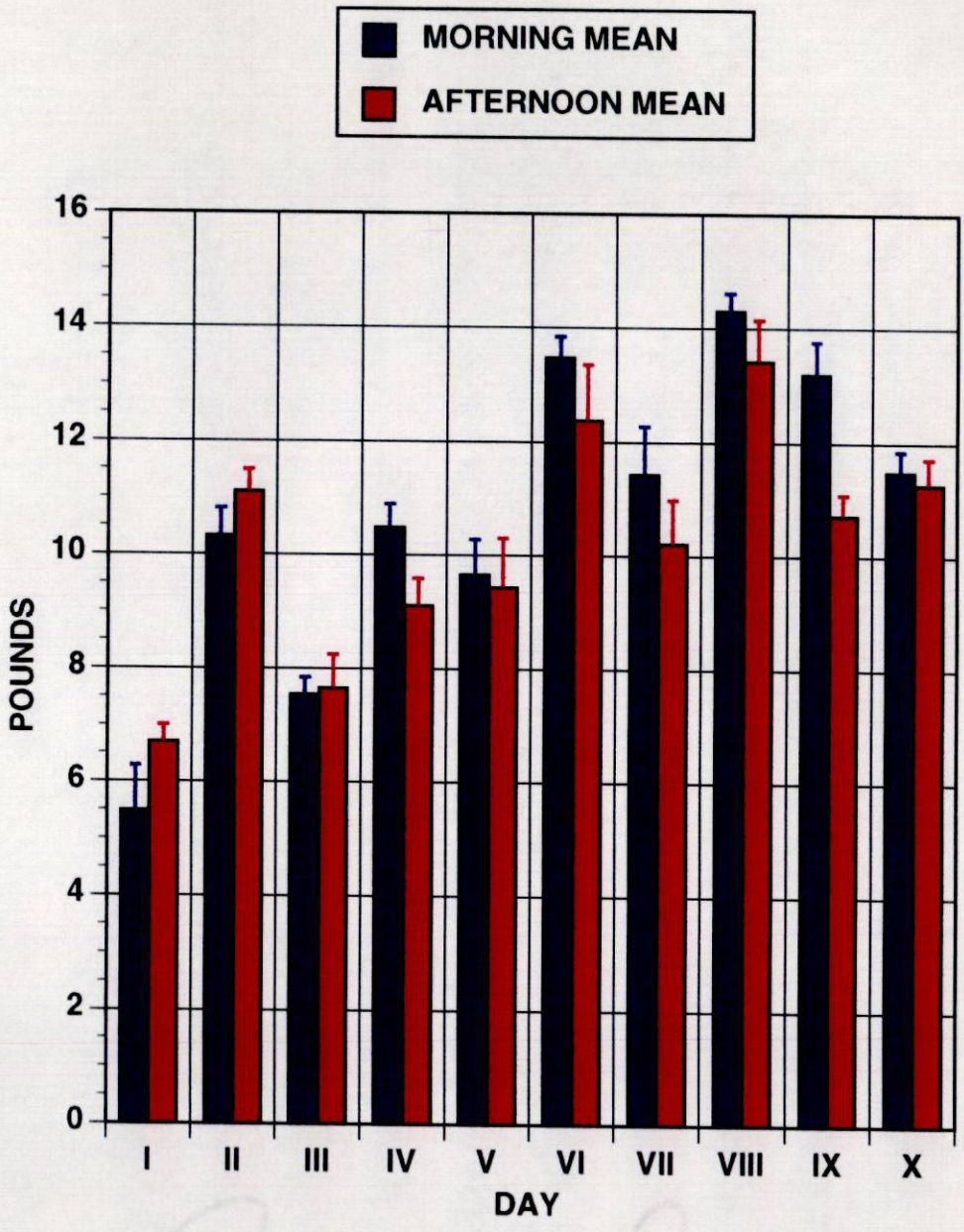


Figure 20. Daily average bite forces during simultaneous, chronic electrical stimulation of the bilateral masseter muscles for Subject 3, Part A

Table 21.--Daily average maximum voluntary bite forces before (T_1, morning) and after (T_2, afternoon) simultaneous, chronic electrical stimulation of the bilateral masseter muscles for Subject 3 in Part A

DAY	T_1 (lbs.)	S.D.	T_2 (lbs.)	S.D.	p VALUE	% CHANGE
I	152.26	2.86	154.13	3.05	0.40	1.20
II	154.60	2.34	145.23	6.62	*0.03	-6.10
III	146.17	6.94	148.98	5.87	0.38	1.90
IV	154.60	6.62	157.41	11.28	0.57	1.80
V	157.41	7.10	161.16	5.34	0.37	2.40
VI	165.84	5.34	170.06	5.64	0.20	2.50
VII	151.74	5.07	168.19	7.29	*0.03	10.80
VIII	167.25	9.88	158.82	5.34	0.11	-5.00
IX	159.29	6.82	167.25	3.55	0.10	5.00
X	148.98	4.85	156.94	7.40	0.12	5.30

* Statistically significant difference ($p \leq 0.05$).

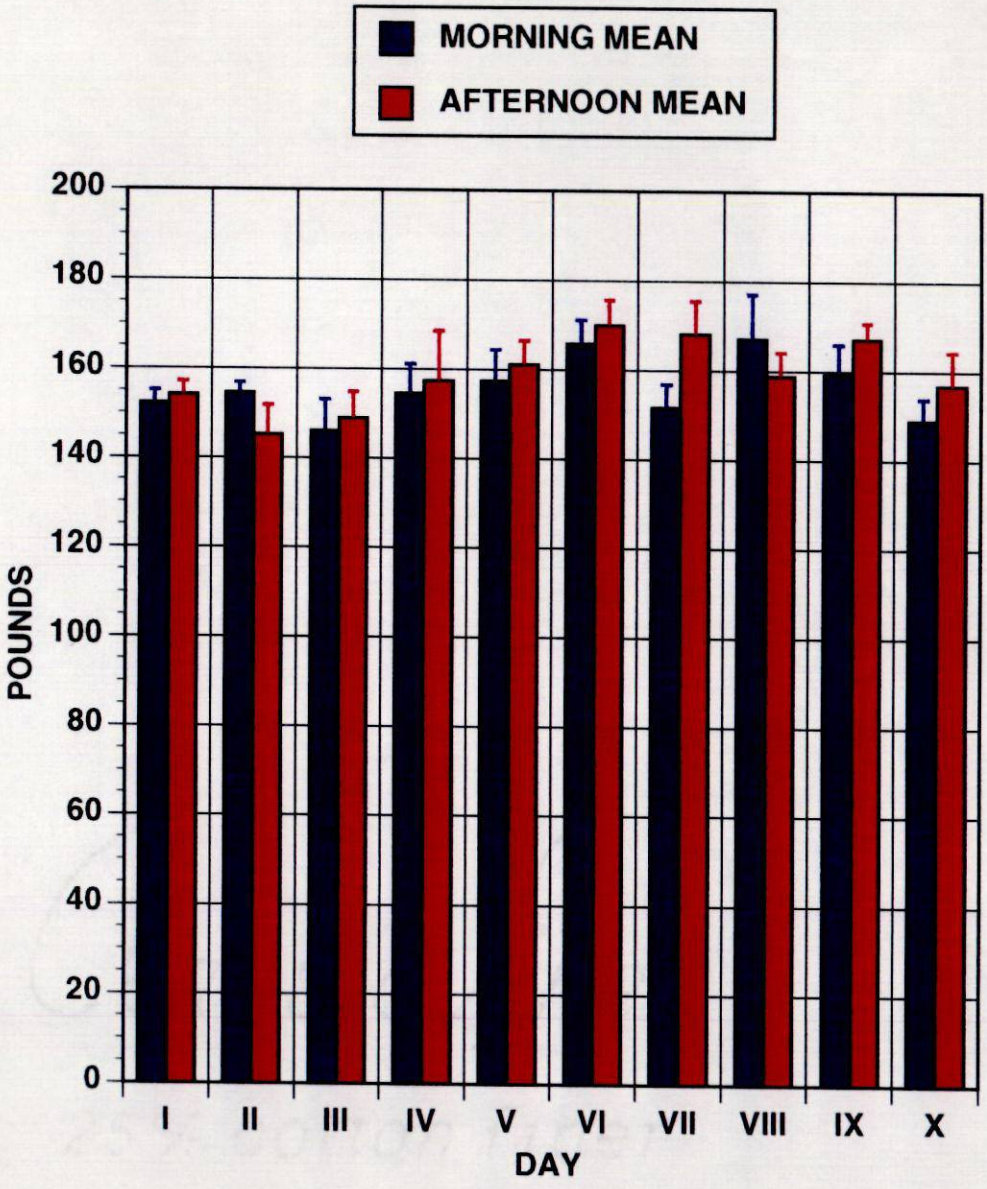


Figure 21. Daily average maximum bite forces before and after simultaneous, bilateral chronic electrical stimulation of the masseter muscles for Subject 3, Part A

Table 22.--Daily average bite forces at T_1 (morning) and T_2 (afternoon) during simultaneous, chronic electrical stimulation of the bilateral masseter muscles for Subject 4 in Part A

DAY	T_1 (lbs.)	S.D.	T_2 (lbs.)	S.D.	p VALUE	% CHANGE
I	5.94	0.24	5.63	0.32	0.18	-5.20
II	6.19	0.75	7.55	0.12	0.16	21.20
III	3.39	0.28	4.72	0.29	*0.00	39.20
IV	5.37	0.16	3.06	0.23	5.63	-43.00
V	6.92	0.38	3.77	0.27	9.54	-45.50
VI	6.77	0.97	6.88	0.78	0.80	1.60
VII	7.54	0.47	6.17	0.65	*0.03	-18.20
VIII	6.16	0.32	5.65	0.32	*0.04	-8.30
IX	5.10	0.27	4.58	0.48	0.12	-10.2
X	6.06	0.34	6.37	0.48	0.24	5.10

* Statistically significant difference ($p \leq 0.05$).

Constant: Frequency= 20 Hz, Pulse Duration = 100 µsec., Interpulse Interval = 50 msec, Duty Cycle = Simultaneous 15 sec on and 5 sec off

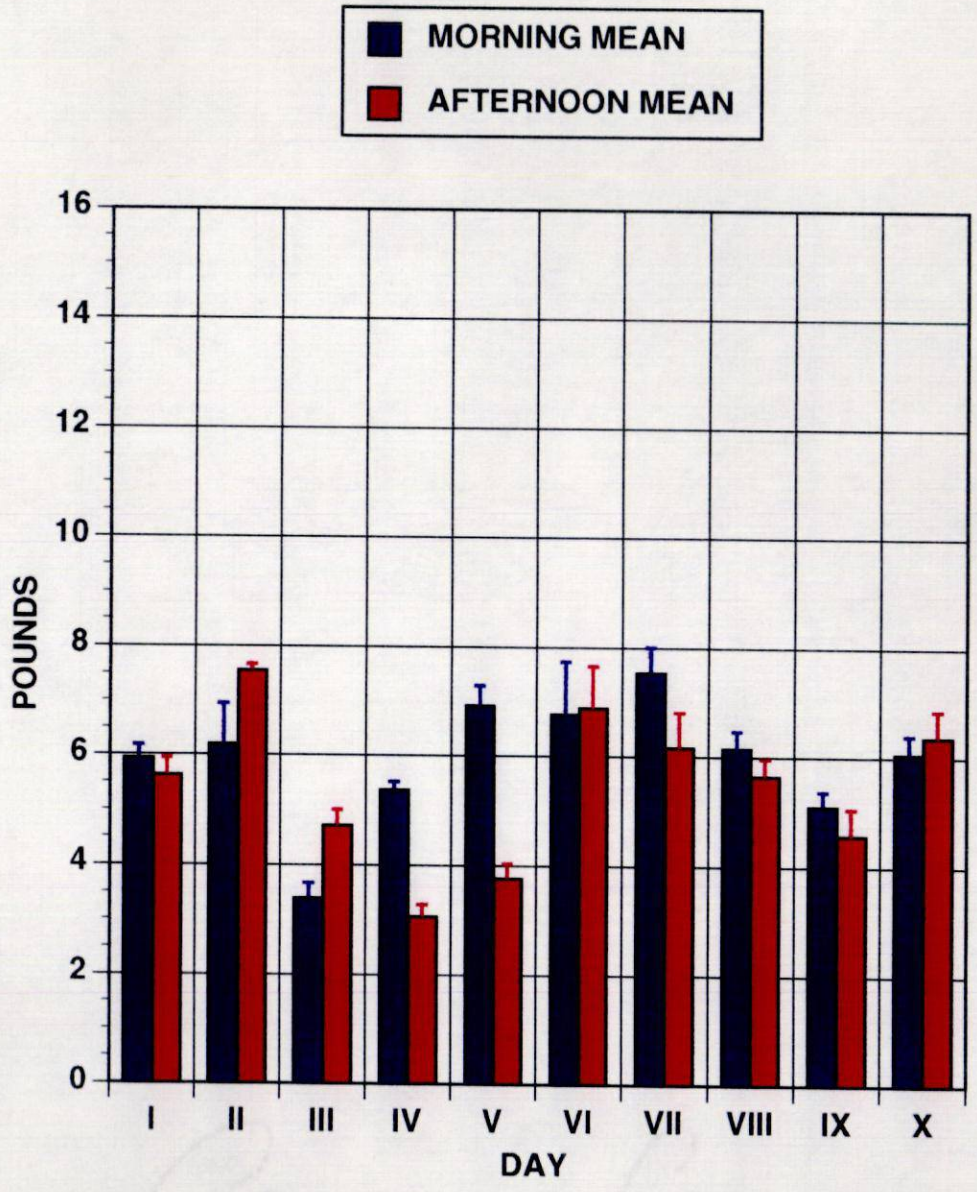


Figure 22. Daily average bite forces during simultaneous, chronic electrical stimulation of the bilateral masseter muscles for Subject 4, Part A

Table 23.--Daily average maximum voluntary bite forces before (T_1, morning) and after (T_2, afternoon) simultaneous, chronic electrical stimulation of the bilateral masseter muscles for Subject 4 in Part A

DAY	T_1 (lbs.)	S.D.	T_2 (lbs.)	S.D.	p VALUE	% CHANGE
I	90.88	5.34	107.28	3.05	*0.00	18.00
II	114.78	1.04	53.87	1.65	3.26	-53.10
III	88.54	4.19	89.01	3.70	0.89	0.50
IV	94.16	7.10	91.82	3.05	0.32	-2.50
V	94.16	4.19	96.97	4.56	0.49	3.00
VI	101.66	4.56	106.81	2.09	*0.02	5.10
VII	116.65	4.50	94.63	5.13	*0.00	-18.90
VIII	92.76	4.86	96.51	2.57	0.20	4.00
IX	91.83	1.96	97.45	4.86	0.08	6.10
X	99.80	2.10	103.07	5.74	0.30	3.30

* Statistically significant difference ($p \leq 0.05$).

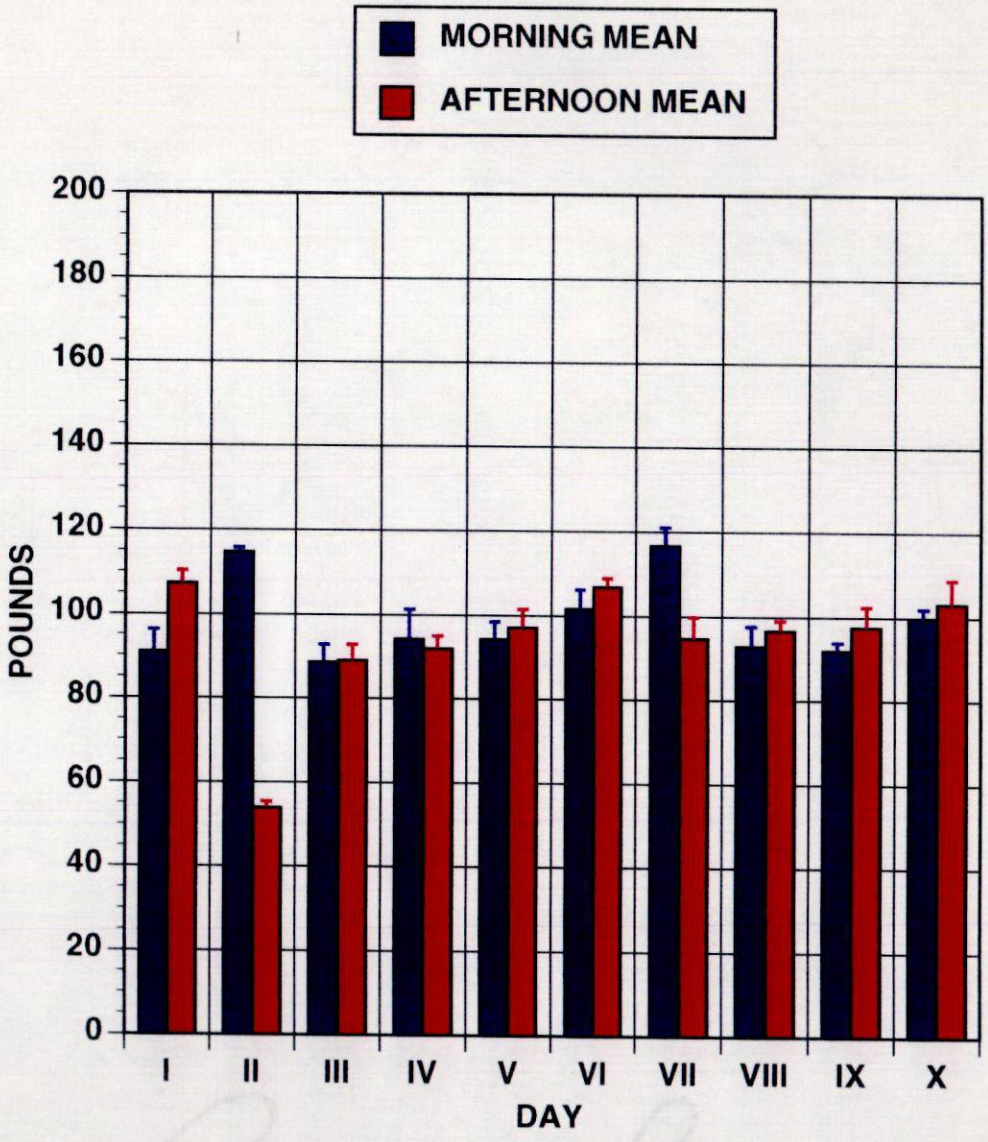


Figure 23. Daily average maximum bite forces before and after simultaneous, bilateral chronic electrical stimulation of the masseter muscles for Subject 4, Part A

Table 24.--Daily average bite forces at T_1 (morning) and T_2 (afternoon) during simultaneous, chronic electrical stimulation of the bilateral masseter muscles for Subject 5 in Part A

DAY	T_1 (lbs.)	S.D.	T_2 (lbs.)	S.D.	p VALUE	% CHANGE
I	2.04	0.48	1.97	0.40	0.77	-3.40
II	1.97	0.40	1.28	0.16	*0.04	-35.00
III	2.03	0.09	3.72	0.83	*0.02	83.30
IV	4.41	0.19	5.52	1.25	0.11	25.20
V	6.19	0.37	6.06	0.14	0.28	-2.10
VI	5.46	0.32	4.35	0.34	*0.01	-20.30
VII	6.74	0.37	6.97	0.55	0.57	3.40
VIII	5.41	0.62	4.83	0.18	0.08	-10.70
IX	6.61	0.32	5.50	0.45	*0.01	-16.80
X	4.44	0.00	5.10	0.15	*0.00	14.90

* Statistically significant difference ($p \leq 0.05$).

Constant: Frequency= 20 Hz, Pulse Duration = 100 µsec., Interpulse Interval = 50 msec, Duty Cycle = Simultaneous 15 sec on and 5 sec off

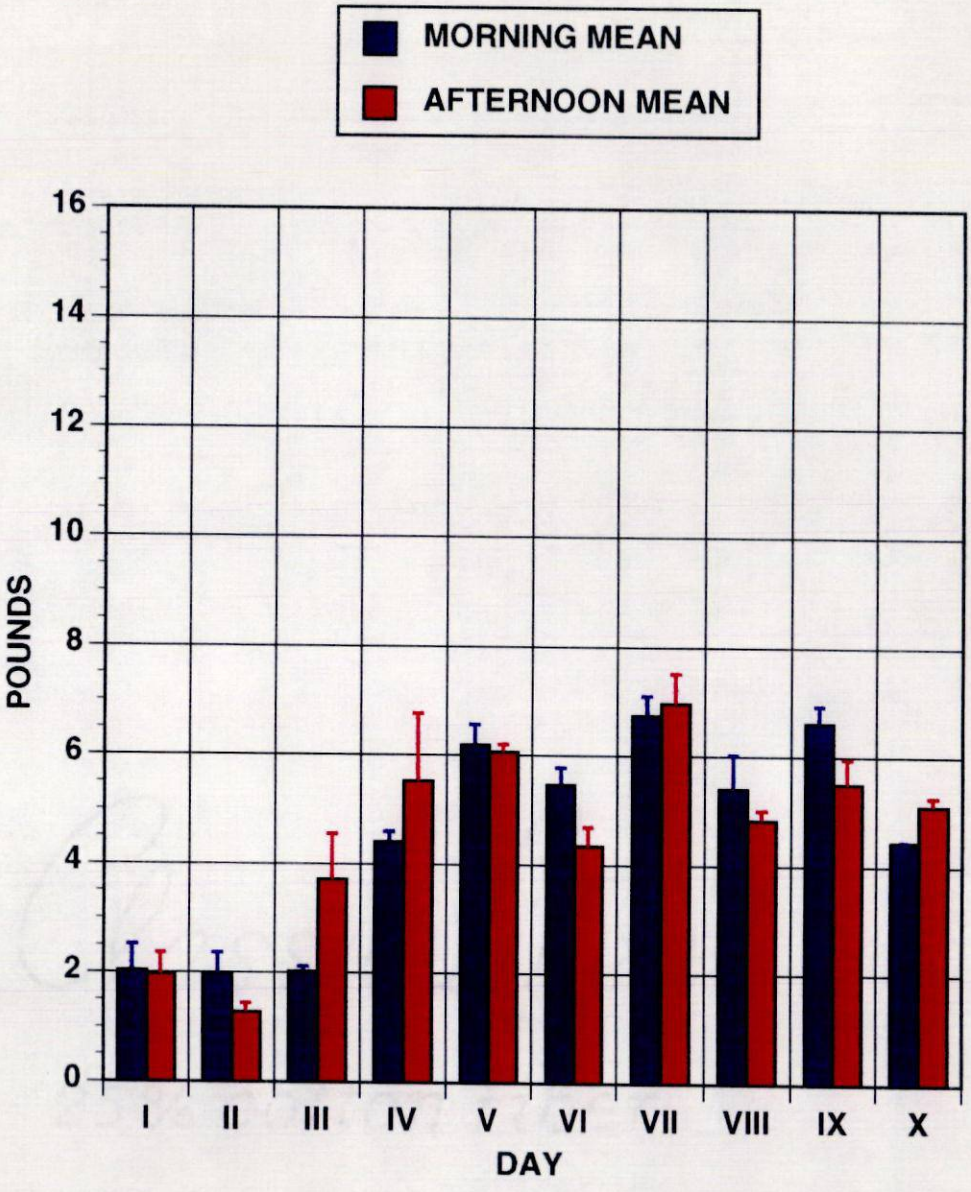


Figure 24. Daily average bite forces during simultaneous, chronic electrical stimulation of the bilateral masseter muscles for Subject 5, Part A

Table 25.--Daily average maximum voluntary bite forces before (T_1, morning) and after (T_2, afternoon) simultaneous, chronic electrical stimulation of the bilateral masseter muscles for Subject 5 in Part A

DAY	T_1 (lbs.)	S.D.	T_2 (lbs.)	S.D.	p VALUE	% CHANGE
I	94.16	2.56	92.76	2.67	0.37	-1.50
II	110.09	2.34	84.33	3.38	*0.00	-23.40
III	96.04	1.65	95.10	5.39	0.67	-1.00
IV	92.29	6.10	91.82	3.05	0.89	-0.50
V	90.88	4.19	89.01	0.00	0.37	-2.10
VI	100.72	4.96	99.32	5.87	0.71	-1.40
VII	100.25	4.19	96.51	5.34	0.24	-3.70
VIII	105.88	4.19	95.10	3.14	*0.02	-10.20
IX	92.76	2.09	97.44	9.01	0.32	5.00
X	111.50	8.38	102.13	9.60	0.12	-8.40

* Statistically significant difference ($p \leq 0.05$).

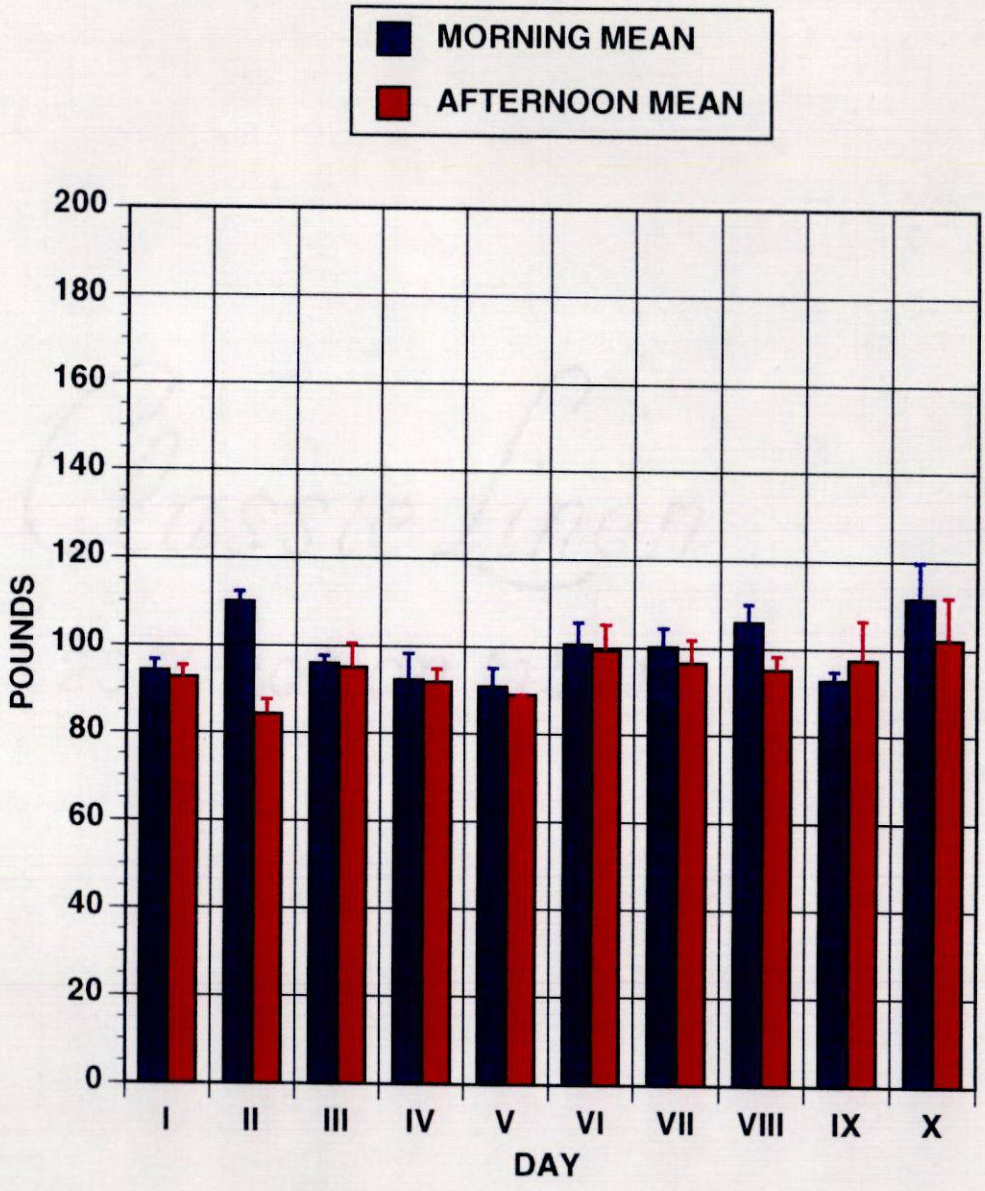


Figure 25. Daily average maximum bite forces before and after simultaneous, bilateral chronic electrical stimulation of the masseter muscles for Subject 5, Part A

Table 26.--Daily average bite forces right, at T_1 (morning) and T_2 (afternoon) during alternating, chronic electrical stimulation of the right masseter muscle for Subject 2 in Part B

DAY	T_1 (lbs.)	S.D.	T_2 (lbs.)	S.D.	p VALUE	% CHANGE
I	3.33	0.00	2.51	0.12	*0.00	-24.60
II	2.66	0.00	2.22	2.98	1.50	-16.50
III	3.88	0.00	2.77	0.19	*0.00	-28.60
IV	4.51	0.12	5.10	0.00	*0.01	13.10
V	5.40	0.33	4.62	0.16	*0.02	-14.40
VI	5.07	0.06	5.38	0.36	0.33	6.10
VII	5.05	0.05	5.73	0.20	*0.01	13.50
VIII	5.66	0.30	5.66	0.15	1.00	0.00
IX	-	-	-	-	-	-
X	-	-	-	-	-	-

* Statistically significant difference ($p \leq 0.05$).

Constant: Frequency= 20 Hz, Pulse Duration = 100 µsec., Interpulse Interval = 50 msec, Duty Cycle = Alternating 5 sec on and 5 sec off

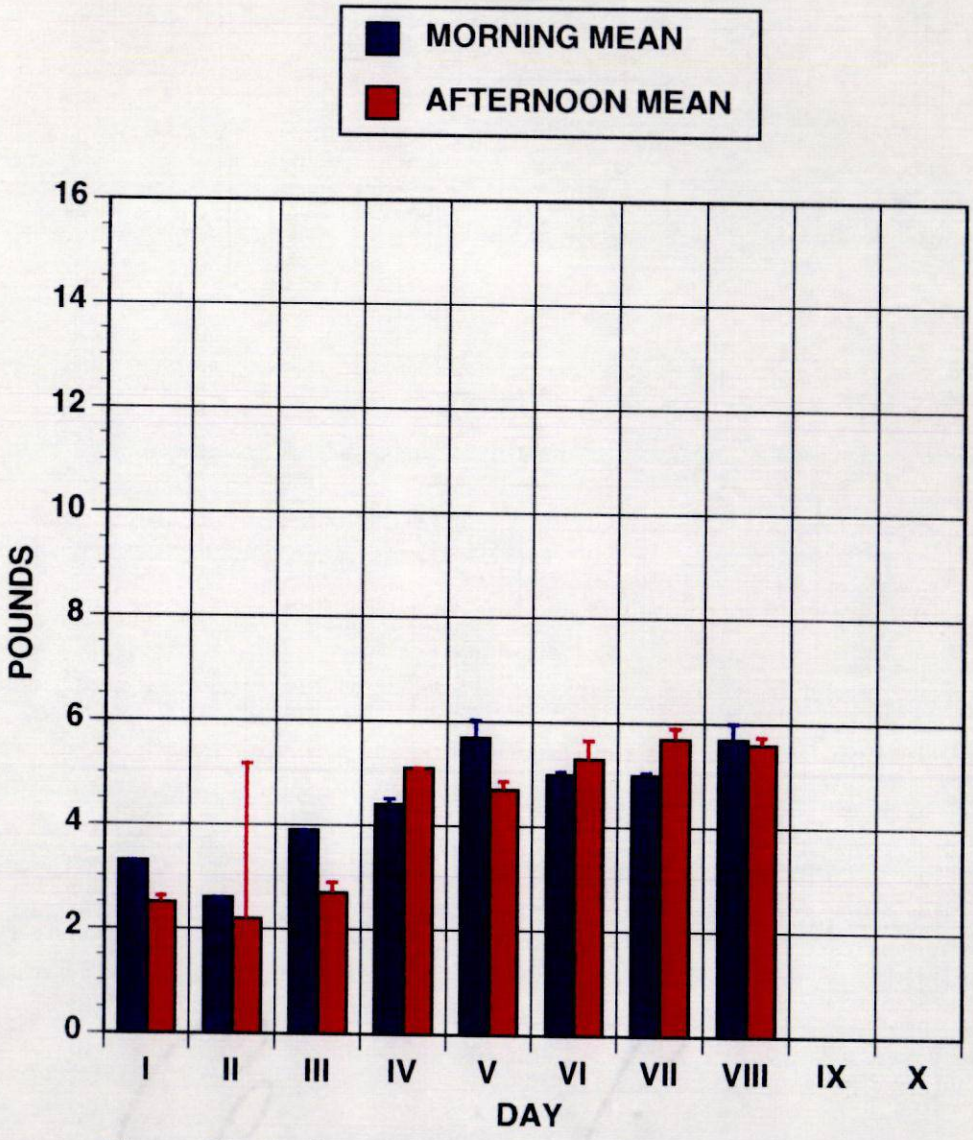


Figure 26. Daily average bite forces right, at T_1 (morning) and T_2 (afternoon) during alternating, chronic electrical stimulation of the right masseter muscle for Subject 2, Part B

Table 27.--Daily average bite forces left, at T_1 (morning) and T_2 (afternoon) during alternating, chronic electrical stimulation of the left masseter muscle for Subject 2 in Part B

DAY	T_1 (lbs.)	S.D.	T_2 (lbs.)	S.D.	p VALUE	% CHANGE
I	2.78	0.00	4.44	0.00	*0.00	59.70
II	4.81	0.00	2.77	0.00	*0.00	-42.40
III	4.66	0.22	4.81	0.32	0.26	3.20
IV	4.83	0.34	2.29	0.12	*0.00	-52.60
V	4.90	0.30	4.79	0.00	*0.00	-2.20
VI	4.95	0.25	3.18	0.12	*0.00	-35.80
VII	7.54	0.00	5.55	0.00	*0.00	-26.40
VIII	5.55	0.00	3.33	0.00	*0.00	-40.00
IX	-	-	-	-	-	-
X	-	-	-	-	-	-

* Statistically significant difference ($p \leq 0.05$).

Constant: Frequency= 20 Hz, Pulse Duration = 100 µsec., Interpulse Interval = 50 msec, Duty Cycle = Alternating 5 sec on and 5 sec off

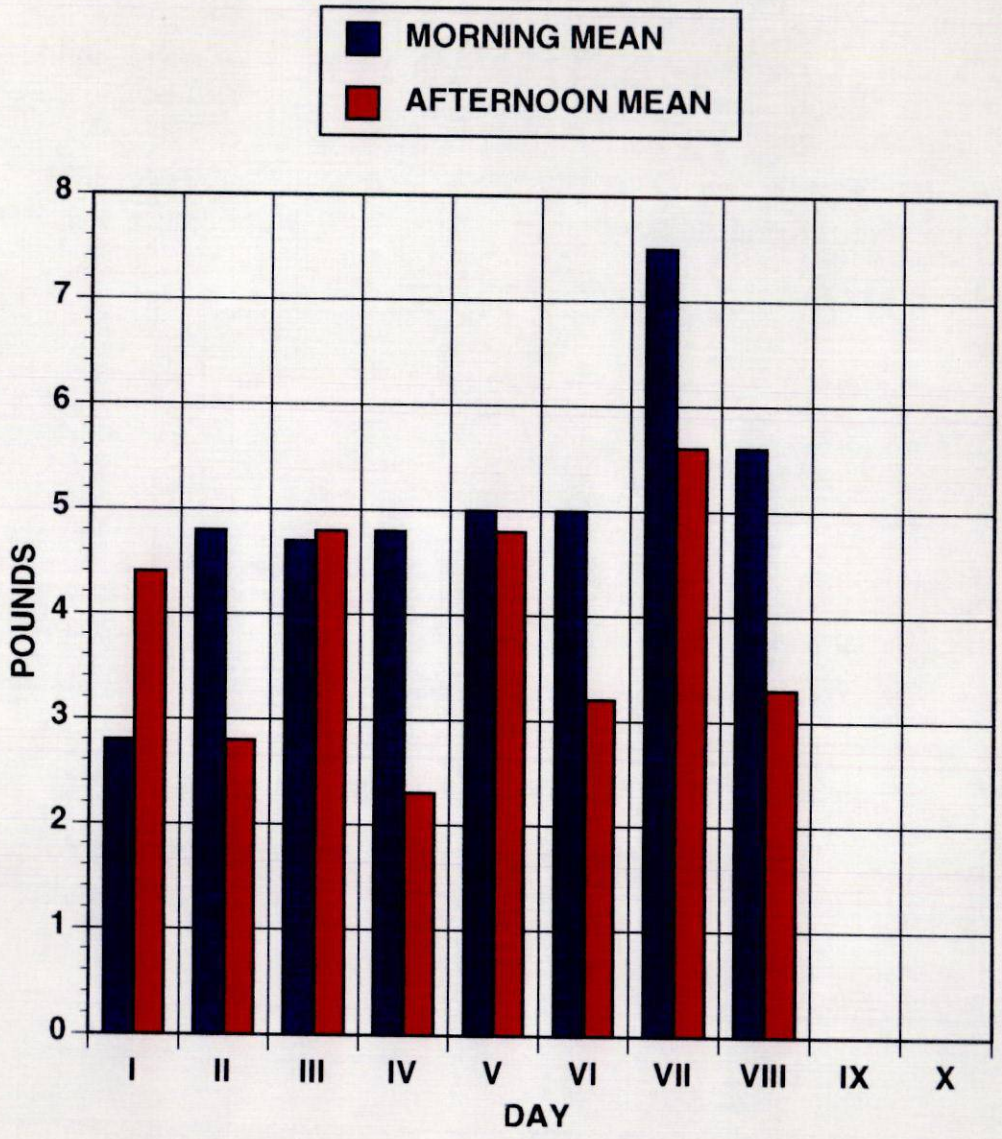


Figure 27. Daily average bite forces left, at T_1 (morning) and T_2 (afternoon) during alternating, chronic electrical stimulation of the left masseter muscle for Subject 2, Part B

Table 28.--Daily average maximum voluntary bite forces right, before (T_1, morning) and after (T_2, afternoon) alternating, chronic electrical stimulation of the bilateral masseter muscles for Subject 2 in Part B

DAY	T_1 (lbs.)	S.D.	T_2 (lbs.)	S.D.	p VALUE	% CHANGE
I	127.43	19.71	159.76	9.72	*0.03	25.40
II	141.96	14.40	90.89	5.34	*0.00	-36.00
III	91.36	7.22	95.57	13.90	0.52	4.60
IV	108.22	7.11	89.48	6.29	*0.02	-17.30
V	98.85	11.99	76.37	8.38	*0.03	-22.70
VI	91.36	4.06	99.79	10.29	*0.05	9.20
VII	93.23	8.51	92.76	2.67	0.91	-0.50
VIII	93.72	8.11	83.86	8.34	0.13	-10.50
IX	-	-	-	-	-	-
X	-	-	-	-	-	-

* Statistically significant difference ($p \leq 0.05$).

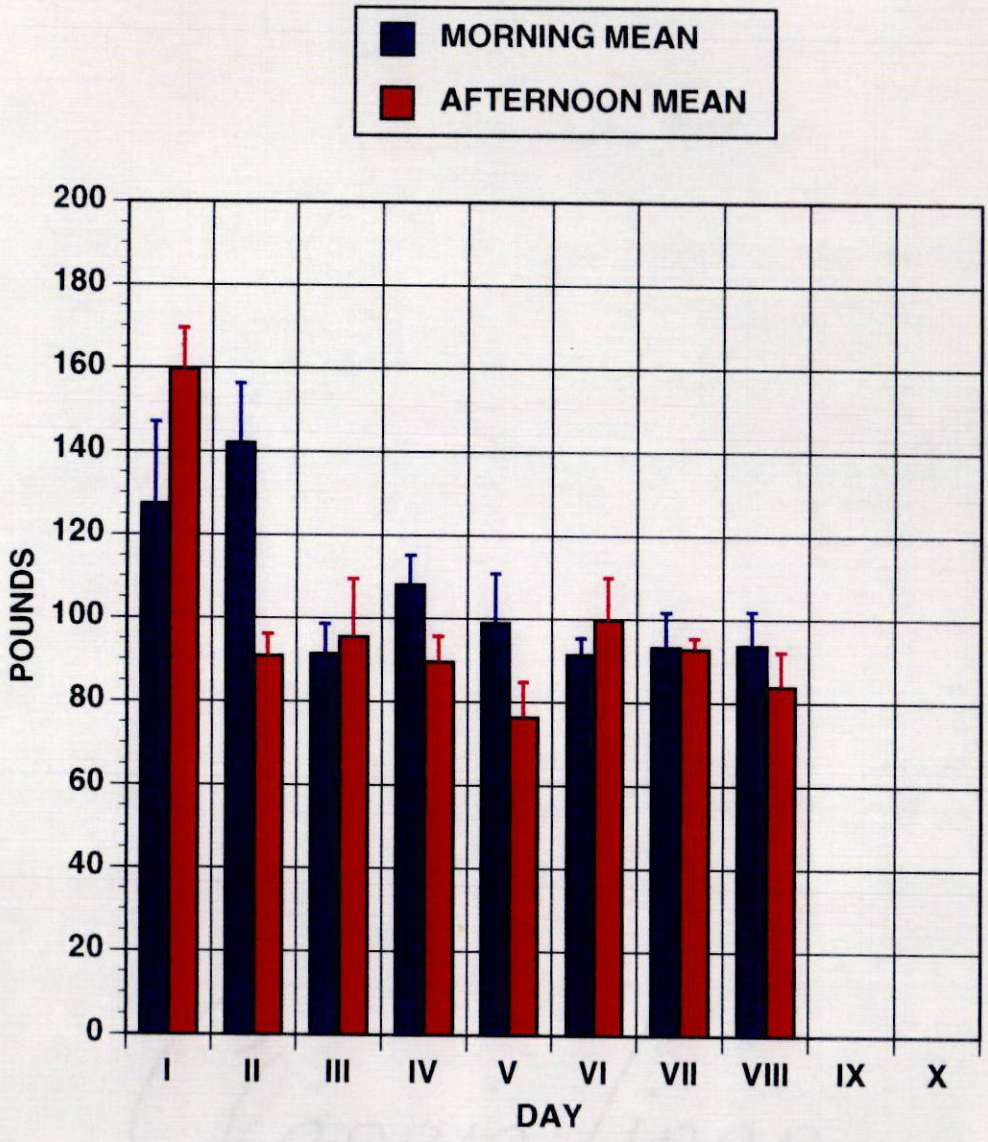


Figure 28. Daily average maximum voluntary bite forces right, before (T_1, morning) and after (T_2, afternoon) alternating, chronic electrical stimulation of the bilateral masseter muscles for Subject 2, Part B

Table 29.--Daily average maximum voluntary bite forces left, before (T_1, morning) and after (T_2, afternoon) alternating, chronic electrical stimulation of the bilateral masseter muscles for Subject 2 in Part B

DAY	T_1 (lbs.)	S.D.	T_2 (lbs.)	S.D.	p VALUE	% CHANGE
I	67.93	7.40	66.99	2.67	0.77	-1.40
II	72.61	5.49	113.37	12.01	*0.00	56.10
III	101.19	18.91	129.77	5.64	*0.01	28.20
IV	76.36	6.74	93.70	1.65	*0.00	22.70
V	84.33	9.37	87.60	3.14	0.42	3.90
VI	93.23	3.84	77.77	4.50	*0.00	-16.60
VII	104.94	1.95	82.92	6.94	*0.00	-21.00
VIII	77.77	6.90	76.36	8.38	0.69	-1.80
IX	-	-	-	-	-	-
X	-	-	-	-	-	-

* Statistically significant difference ($p \leq 0.05$).

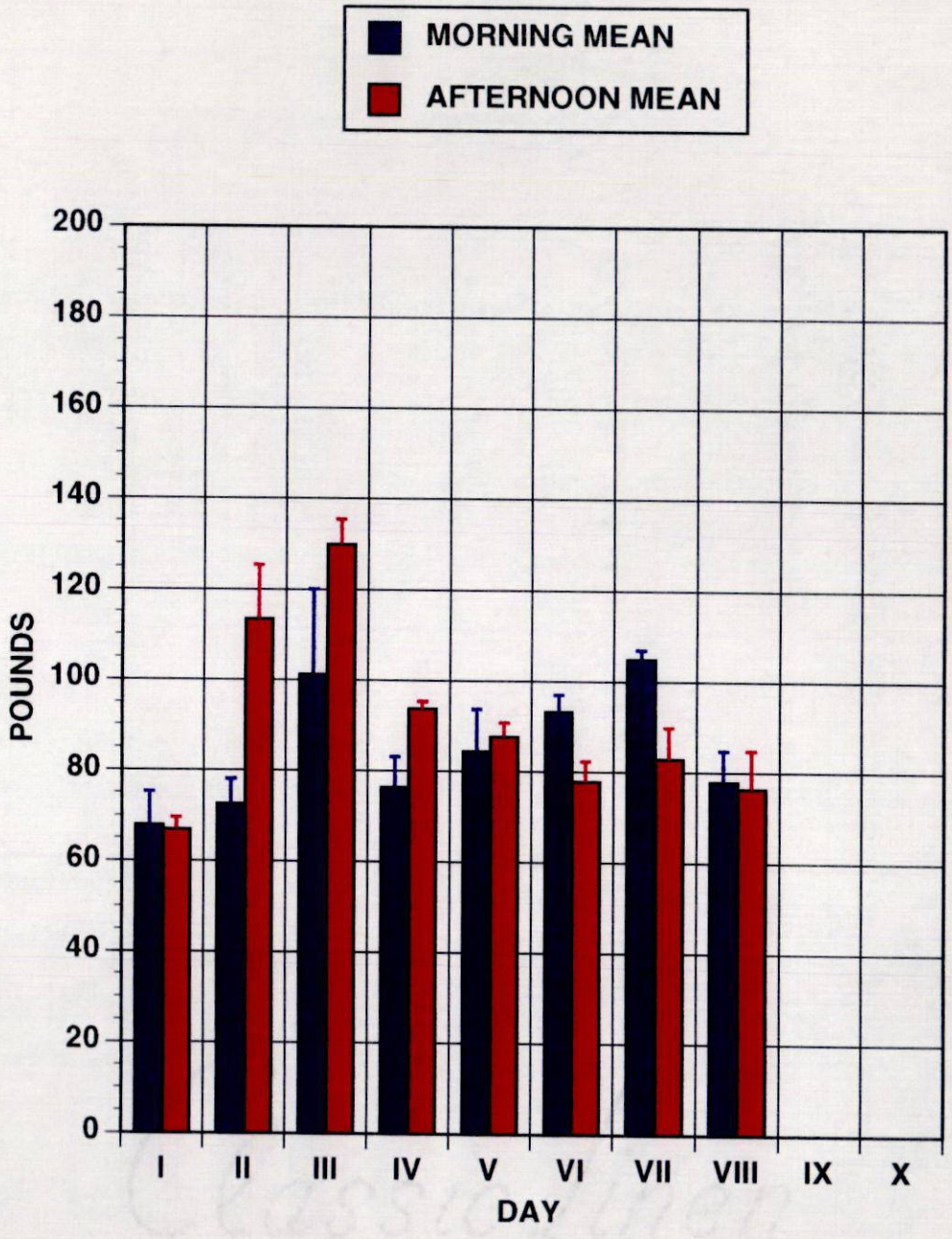


Figure 29. Daily average maximum voluntary bite forces left, before (T_1, morning) and after (T_2, afternoon) alternating, chronic electrical stimulation of the bilateral masseter muscles for Subject 2, Part B

Table 30.--Daily average bite forces right, at T_1 (morning) and T_2 (afternoon) during alternating, chronic electrical stimulation of the right masseter muscle for Subject 3 in Part B

DAY	T_1 (lbs.)	S.D.	T_2 (lbs.)	S.D.	p VALUE	% CHANGE
I	2.73	0.12	4.21	0.00	*0.00	54.20
II	5.55	0.00	4.51	0.33	*0.03	-18.70
III	4.29	0.33	3.7	0.25	0.30	-13.80
IV	4.66	0.22	4.58	0.33	0.74	-1.70
V	4.73	0.33	4.73	0.25	1.00	0.00
VI	4.36	0.33	4.58	0.25	0.22	5.00
VII	6.80	0.51	6.36	0.25	0.22	-6.50
VIII	6.06	0.12	5.25	0.12	*0.03	-13.40
IX	5.55	0.00	4.58	0.12	*0.00	-17.50
X	5.18	0.12	7.99	0.22	*0.00	54.20

* Statistically significant difference ($p \leq 0.05$).

Constant: Frequency= 20 Hz, Pulse Duration = 100 µsec., Interpulse Interval = 50 msec, Duty Cycle = Alternating 5 sec on and 5 sec off

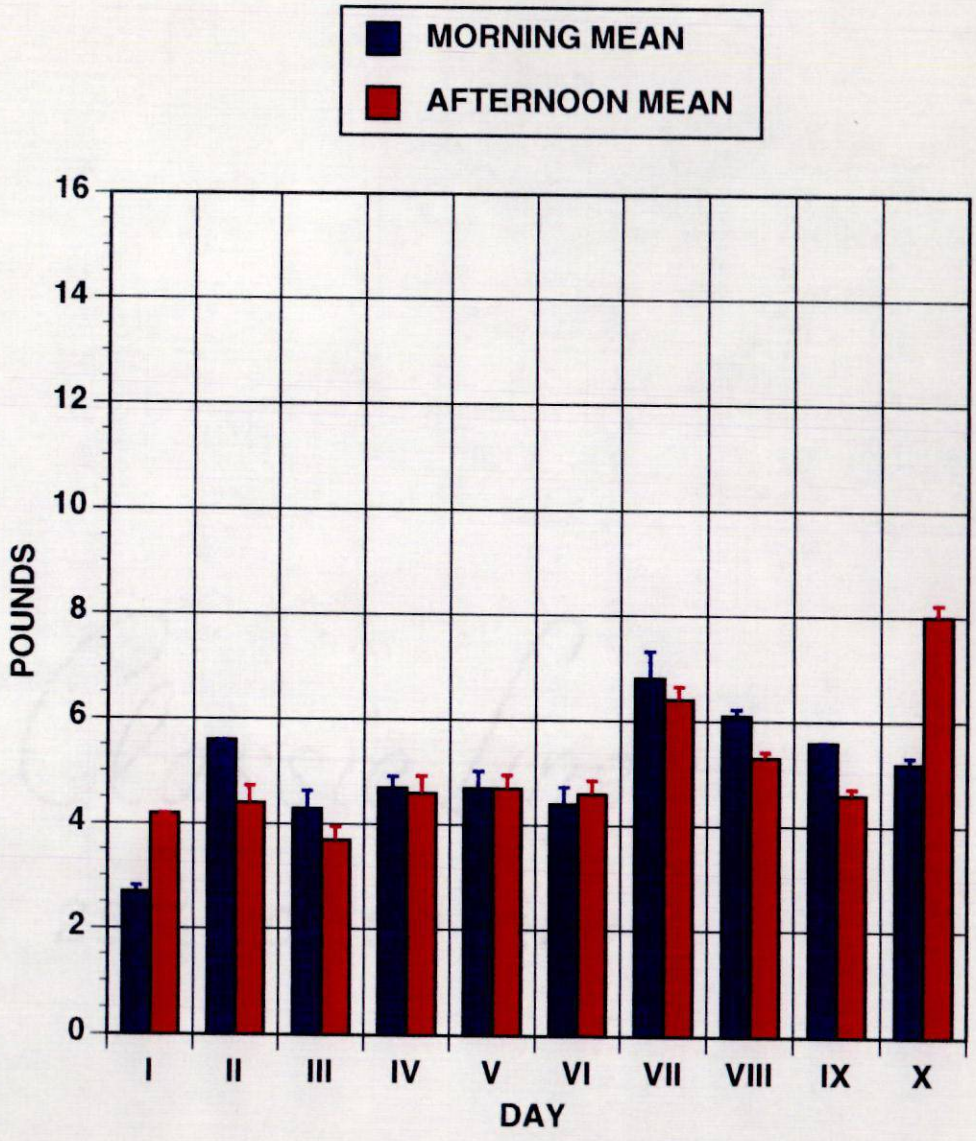


Figure 30. Daily average bite forces right , at T_1 (morning) and T_2 (afternoon) during alternating, chronic electrical stimulation of the right masseter muscle for Subject 3, Part B

Table 31.--Daily average bite forces left, at T_1 (morning) and T_2 (afternoon) during alternating, chronic electrical stimulation of the left masseter muscle for Subject 3 in Part B

DAY	T_1 (lbs.)	S.D.	T_2 (lbs.)	S.D.	p VALUE	% CHANGE
I	4.44	0.00	4.51	0.13	0.42	1.60
II	3.85	0.13	3.55	0.59	0.53	-8.50
III	5.99	0.00	4.44	0.00	*0.00	-34.90
IV	4.66	0.00	4.96	0.13	0.06	6.00
V	6.44	0.22	6.51	0.13	0.74	1.10
VI	4.44	0.00	4.66	0.22	0.23	4.70
VII	4.66	0.22	4.81	0.26	0.53	3.10
VIII	4.96	0.13	5.18	0.13	0.23	4.20
IX	5.11	0.00	5.18	0.13	0.42	1.40
X	4.51	0.13	7.30	0.22	*0.00	38.20

* Statistically significant difference ($p \leq 0.05$).

Constant: Frequency= 20 Hz, Pulse Duration = 100 µsec., Interpulse Interval = 50 msec, Duty Cycle = Alternating 5 sec on and 5 sec off

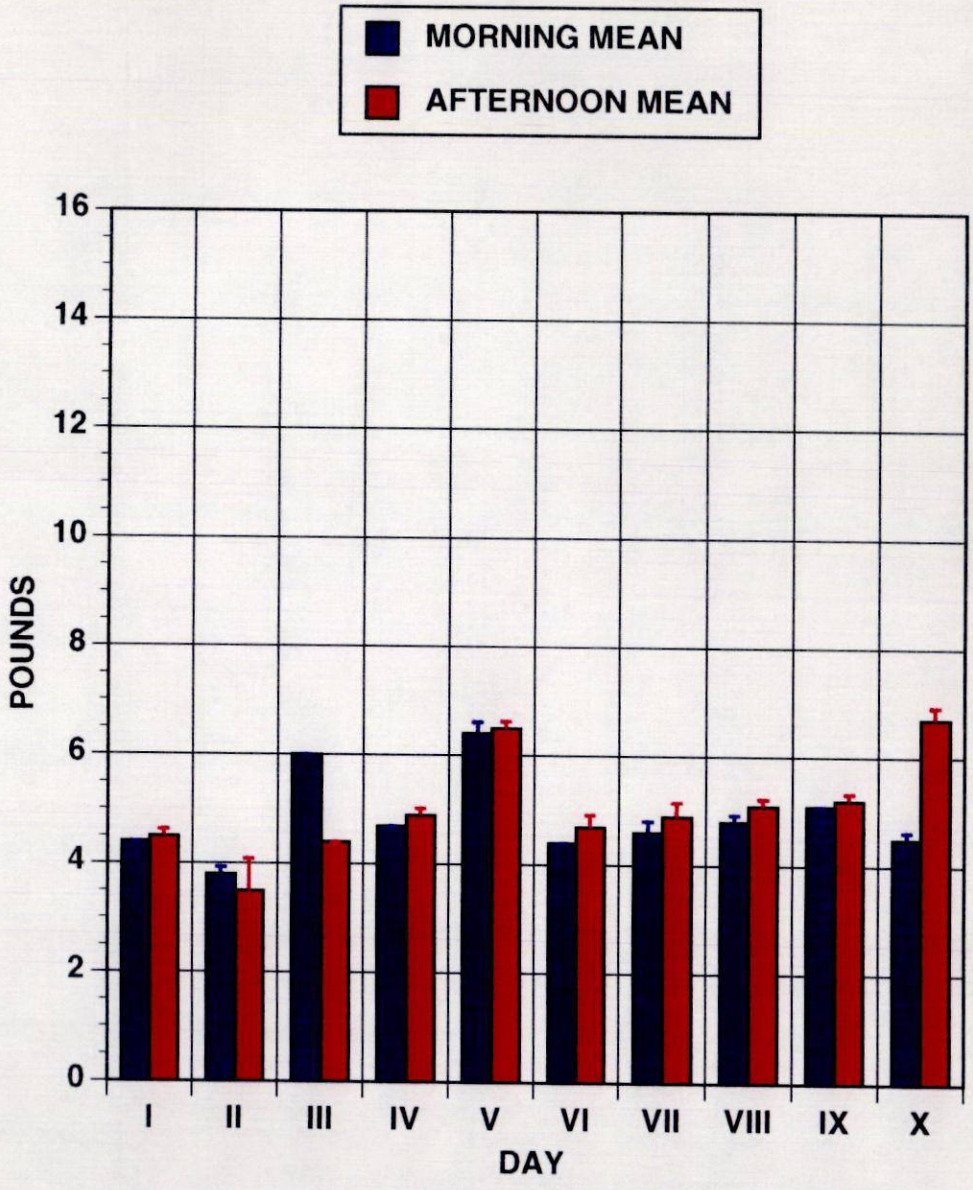


Figure 31. Daily average bite forces left, at T_1 (morning) and T_2 (afternoon) during alternating, chronic electrical stimulation of the left masseter muscle for Subject 3, Part B

Table 32.--Daily average maximum voluntary bite forces right, before (T_1, morning) and after (T_2, afternoon) alternating, chronic electrical stimulation of the bilateral masseter muscles for Subject 3 in Part B

DAY	T_1 (lbs.)	S.D.	T_2 (lbs.)	S.D.	p VALUE	% CHANGE
I	136.80	8.22	119.94	11.64	0.09	-12.30
II	138.68	20.65	151.79	11.76	0.32	9.50
III	158.35	5.13	121.81	10.99	*0.00	-23.10
IV	141.96	10.16	136.80	14.96	0.51	-3.60
V	130.24	8.54	132.59	11.06	0.51	1.80
VI	134.46	7.87	130.24	6.33	0.26	-3.10
VII	130.71	18.77	158.35	10.68	0.08	21.10
VIII	143.36	8.18	135.87	14.44	0.38	-5.20
IX	159.76	8.67	164.44	9.86	0.52	2.90
X	150.39	3.47	165.85	20.31	0.18	10.30

* Statistically significant difference ($p \leq 0.05$).

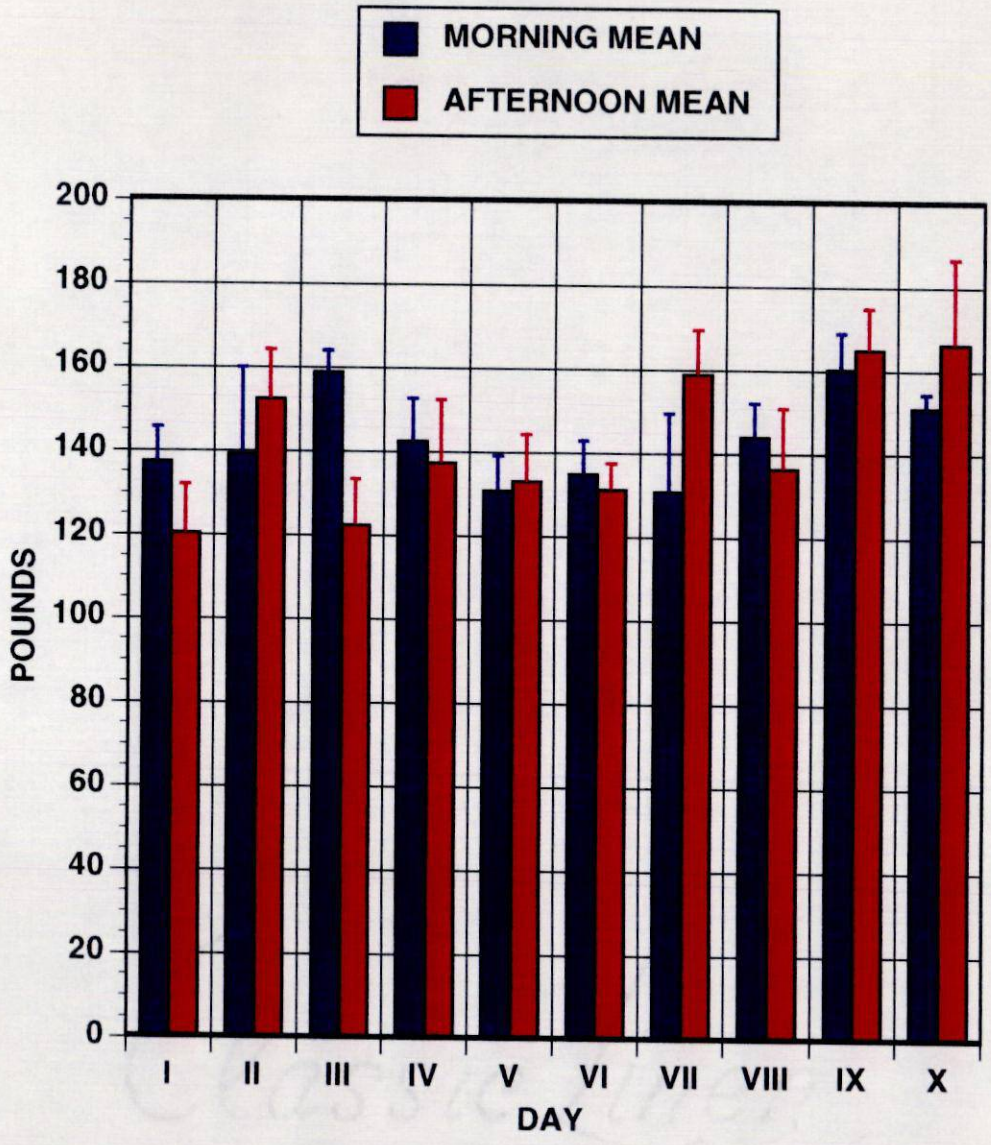


Figure 32. Daily average maximum voluntary bite forces right, before (T_1, morning) and after (T_2, afternoon) alternating, chronic electrical stimulation of bilateral masseter muscles for Subject 3, Part B

Table 33.--Daily average maximum voluntary bite forces left, before (T_1, morning) and after (T_2, afternoon) alternating, chronic electrical stimulation of the bilateral masseter muscles for Subject 3 in Part B

DAY	T_1 (lbs.)	S.D.	T_2 (lbs.)	S.D.	p VALUE	% CHANGE
I	118.06	8.86	138.68	5.59	*0.01	17.50
II	110.10	2.34	108.69	8.86	0.77	-1.30
III	96.04	6.63	81.05	5.13	*0.01	-15.60
IV	113.38	3.92	119.00	4.19	0.11	5.00
V	148.05	3.85	135.87	14.06	0.10	-8.20
VI	171.47	5.59	170.07	9.01	0.77	-0.80
VII	117.13	8.28	150.39	3.47	*0.00	28.40
VIII	111.97	6.06	119.94	3.85	0.15	7.10
IX	163.04	17.34	118.53	11.31	*0.00	-27.30
X	123.22	2.67	130.24	8.54	0.06	5.70

* Statistically significant difference ($p \leq 0.05$).

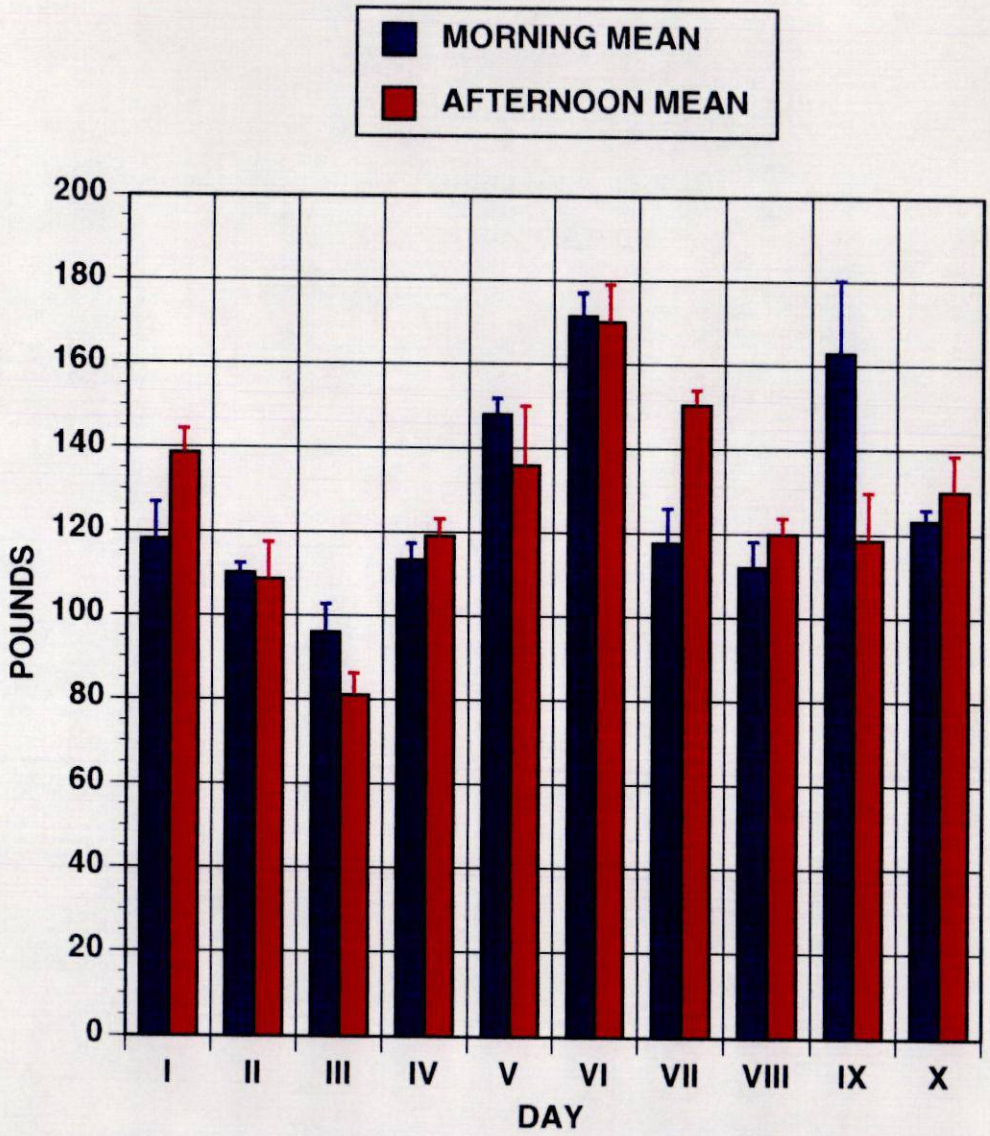


Figure 33. Daily average maximum voluntary bite forces left, before (T_1, morning) and after (T_2, afternoon) alternating, chronic electrical stimulation of the bilateral masseter muscles for Subject 3, Part B

Table 34.--Daily average bite forces right, at T_1 (morning) and T_2 (afternoon) during alternating, chronic electrical stimulation of the right masseter muscle for Subject 4 in Part B

DAY	T_1 (lbs.)	S.D.	T_2 (lbs.)	S.D.	p VALUE	% CHANGE
I	3.33	0.38	3.33	0.22	1.00	0.00
II	3.40	0.13	3.55	0.22	0.42	4.40
III	4.44	0.00	4.88	0.38	0.18	9.90
IV	3.26	0.13	3.77	0.00	0.12	15.60
V	3.48	0.13	3.11	0.00	*0.04	-10.60
VI	7.77	0.00	8.21	0.44	0.23	5.70
VII	8.88	0.00	3.77	0.00	*0.00	-57.50
VIII	4.14	0.26	3.33	0.00	*0.03	-19.60
IX	3.26	0.13	7.22	0.00	*0.00	121.50
X	5.77	0.22	9.44	0.00	*0.00	63.60

* Statistically significant difference ($p \leq 0.05$).

Constant: Frequency= 20 Hz, Pulse Duration = 100 µsec., Interpulse Interval = 50 msec, Duty Cycle = Alternating 5 sec on and 5 sec off

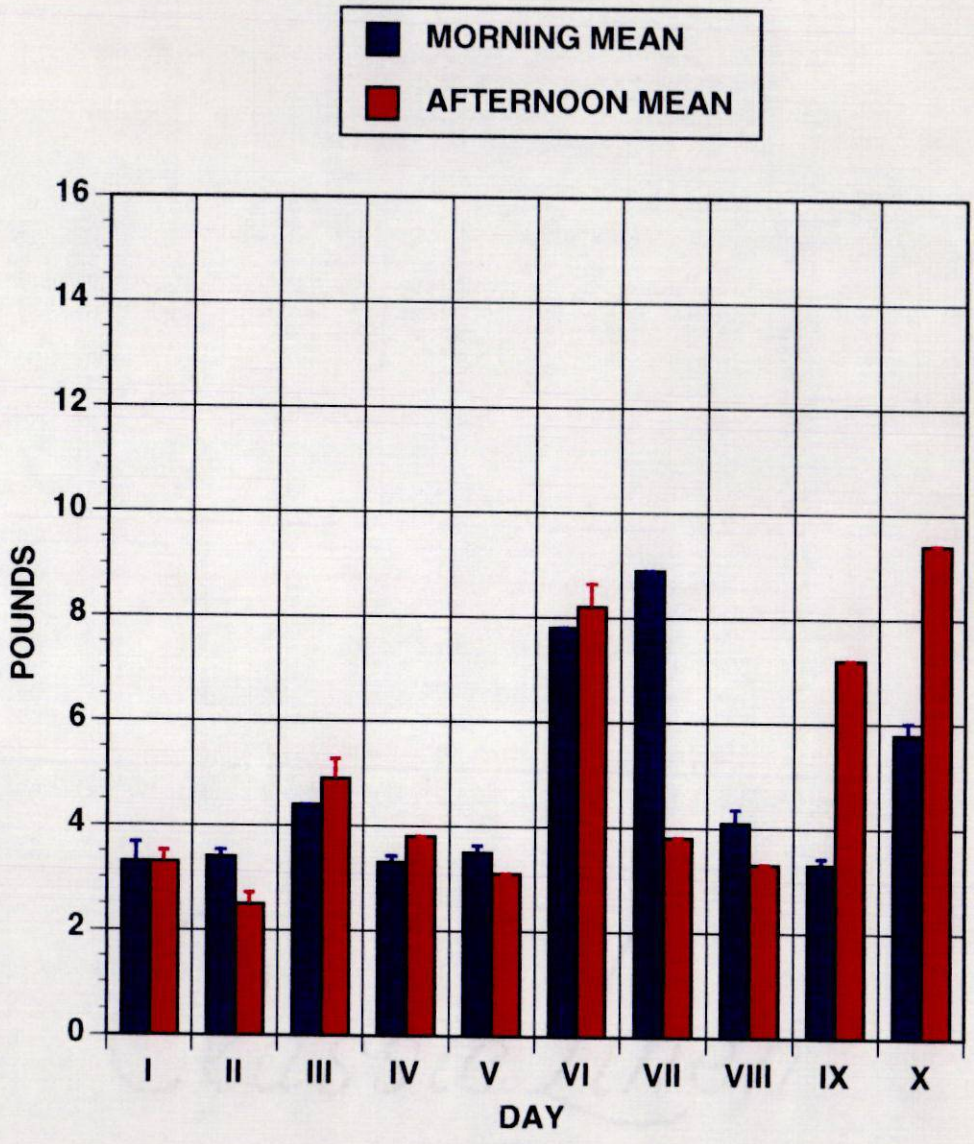


Figure 34. Daily average bite forces right, at T_1 (morning) and T_2 (afternoon) during alternating, chronic electrical stimulation of the right masseter muscle for Subject 4, Part B

Table 35.--Daily average maximum voluntary bite forces left, before (T_1, morning) and after (T_2, afternoon) alternating, chronic electrical stimulation of the bilateral masseter muscles for Subject 4 in Part B

DAY	T_1 (lbs.)	S.D.	T_2 (lbs.)	S.D.	p VALUE	% CHANGE
I	3.92	0.13	2.59	0.13	*0.01	-33.90
II	2.74	0.13	2.66	0.22	0.42	-2.90
III	4.07	0.13	4.37	0.13	0.06	7.40
IV	4.44	0.00	4.22	0.22	0.23	5.00
V	9.07	0.64	9.25	0.85	0.81	2.00
VI	4.22	0.00	2.07	0.26	*0.00	-50.90
VII	5.55	0.55	5.74	0.85	0.84	3.40
VIII	8.88	0.00	5.03	0.13	*0.00	-43.40
IX	10.73	0.64	8.70	0.32	*0.01	-18.90
X	9.81	0.32	6.48	0.85	*0.04	-33.90

* Statistically significant difference ($p \leq 0.05$).

Constant: Frequency= 20 Hz, Pulse Duration = 100 µsec., Interpulse Interval = 50 msec, Duty Cycle = Alternating 5 sec on and 5 sec off

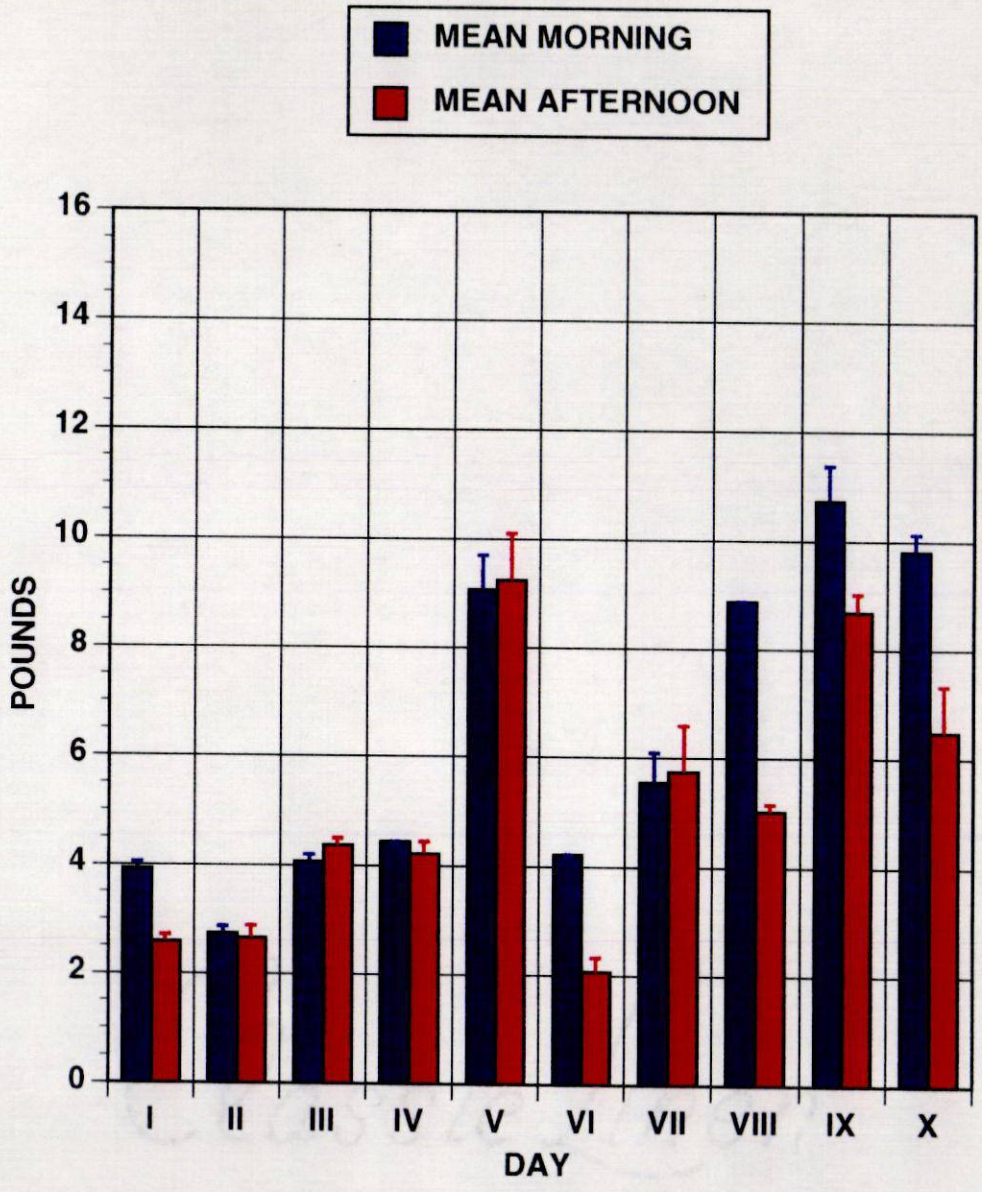


Figure 35. Daily average bite forces left, at T_1 (morning) and T_2 (afternoon) during alternating, chronic electrical stimulation of the left masseter muscle for Subject 4, Part B

Table 36.--Daily average maximum voluntary bite forces right, before (T_1, morning) and after (T_2, afternoon) alternating, chronic electrical stimulation of the bilateral masseter muscles for Subject 4 in Part B

DAY	T_1 (lbs.)	S.D.	T_2 (lbs.)	S.D.	p VALUE	% CHANGE
I	88.08	4.86	88.08	6.11	1.00	0.00
II	94.17	3.47	93.23	4.19	0.78	-1.00
III	91.36	5.49	92.29	3.14	0.79	1.00
IV	100.73	0.00	96.98	4.57	0.14	-3.70
V	89.95	6.33	89.48	3.47	0.87	-0.50
VI	91.83	6.06	96.51	6.50	0.29	5.10
VII	96.98	4.57	89.95	4.26	0.11	-7.20
VIII	92.76	3.55	97.45	4.26	0.12	5.10
IX	96.51	4.19	94.17	1.05	0.33	-2.40
X	108.69	6.75	106.35	7.87	0.61	-2.20

* Statistically significant difference ($p \leq 0.05$).

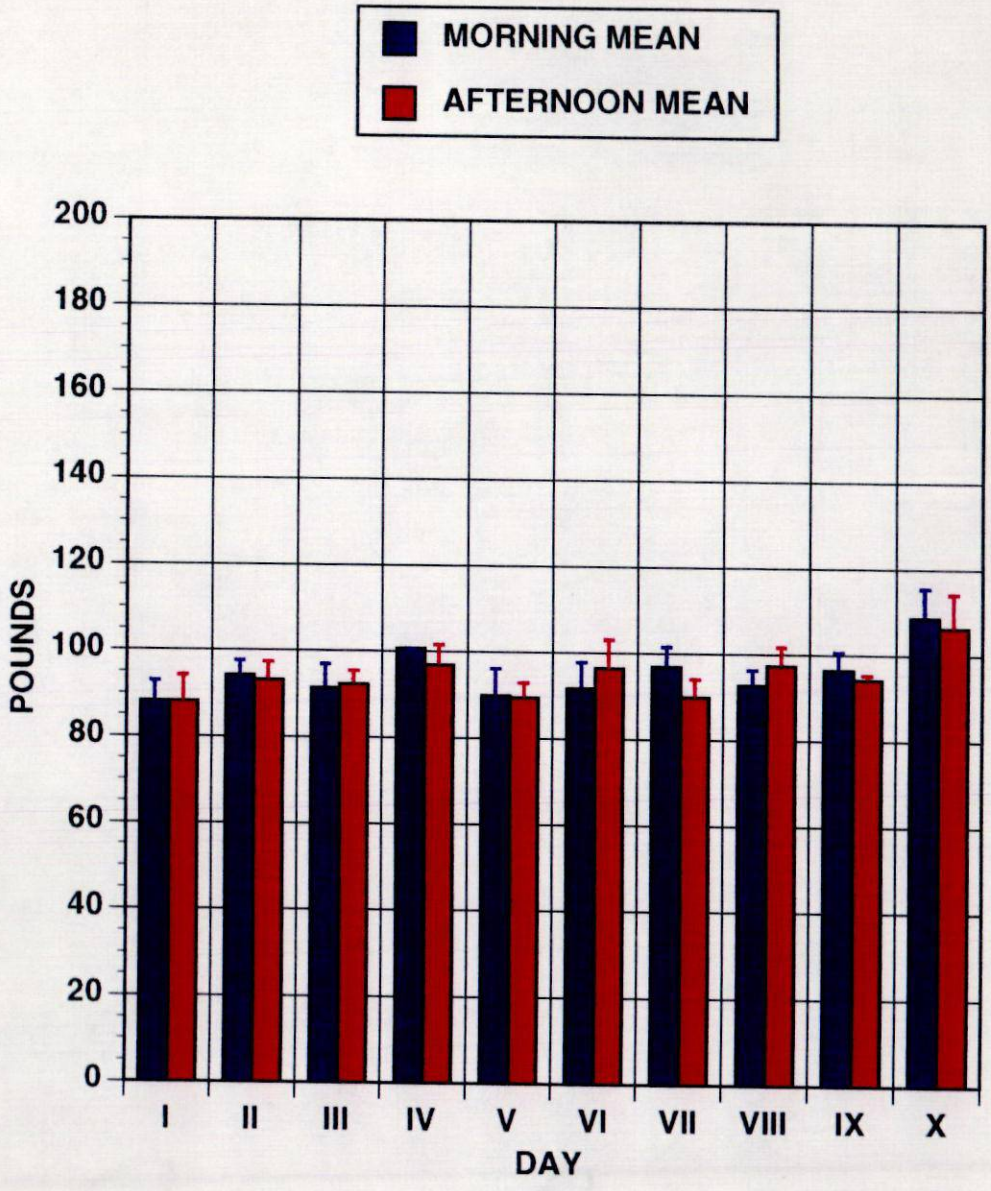


Figure 36. Daily average maximum voluntary bite forces right, before (T_1, morning) and after (T_2, afternoon) alternating, chronic electrical stimulation of the bilateral masseter muscles for Subject 4, Part B

Table 37.--Daily average maximum voluntary bite forces left, before (T_1, morning) and after (T_2, afternoon) alternating, chronic electrical stimulation of the bilateral masseter muscles for Subject 4 in Part B

DAY	T_1 (lbs.)	S.D.	T_2 (lbs.)	S.D.	p VALUE	% CHANGE
I	98.39	6.83	90.89	8.01	0.15	-8.30
II	98.39	3.31	98.39	10.48	1.00	0.00
III	100.26	9.57	100.28	7.26	1.00	0.00
IV	92.76	5.13	98.85	7.67	0.29	6.20
V	89.95	6.95	101.20	4.19	*0.02	11.10
VI	101.20	3.05	109.63	10.66	0.14	7.70
VII	108.69	4.26	106.35	2.10	0.33	-2.20
VIII	89.02	11.94	100.26	4.19	0.11	11.20
IX	97.45	7.87	102.13	8.05	*0.05	4.60
X	96.04	7.77	92.29	3.55	0.40	-4.10

* Statistically significant difference ($p \leq 0.05$).

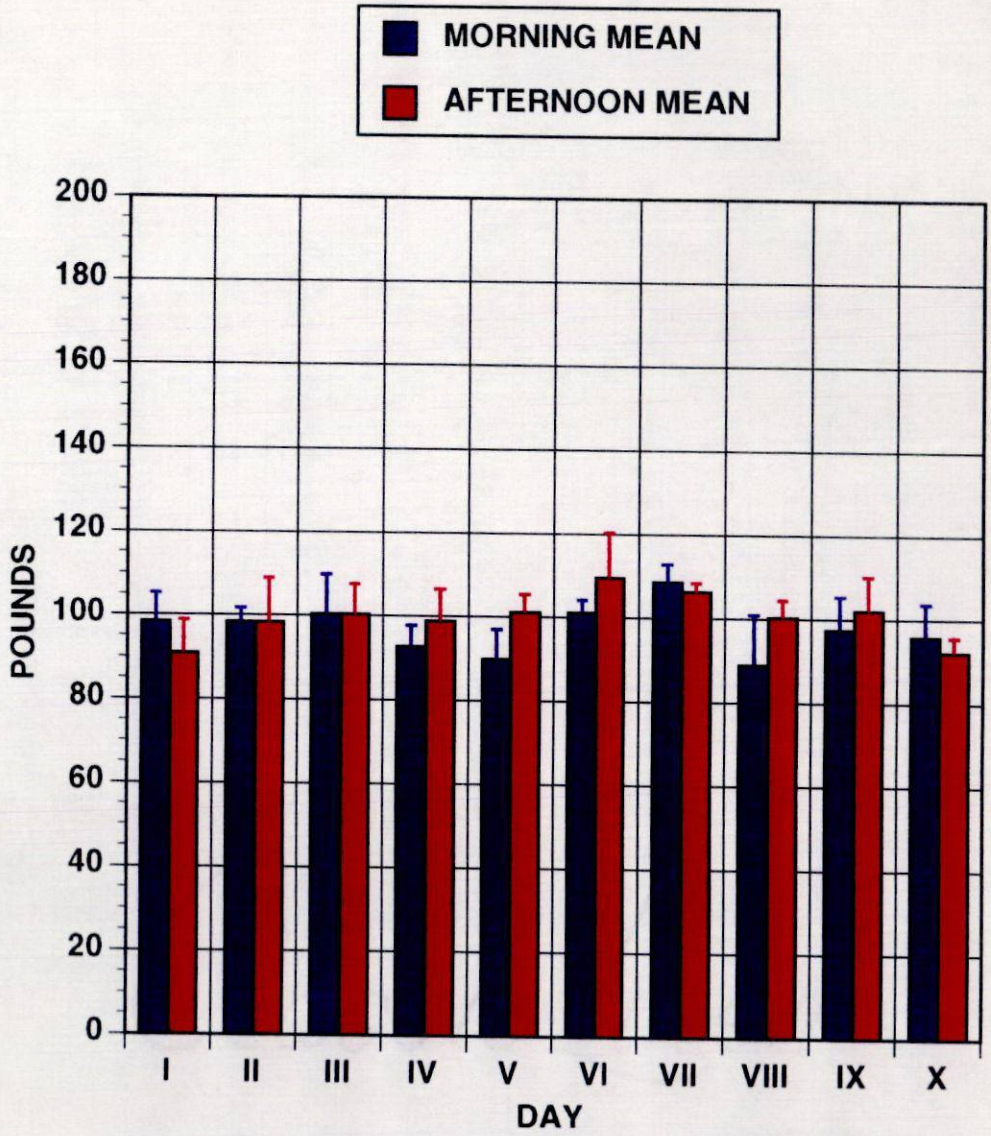


Figure 37. Daily average maximum voluntary bite forces left, before (T_1, morning) and after (T_2, afternoon) alternating, chronic electrical stimulation of the bilateral masseter muscles for Subject 4, Part B

Table 38.--Daily average bite forces right, at T_1 (morning) and T_2 (afternoon) during alternating, chronic electrical stimulation of the right masseter muscle for Subject 5 in Part B

DAY	T_1 (lbs.)	S.D.	T_2 (lbs.)	S.D.	p VALUE	% CHANGE
I	3.33	0.00	2.52	0.13	*0.01	-24.30
II	3.92	0.26	3.26	0.13	*0.04	-16.80
III	4.88	0.00	4.66	0.00	*0.00	-4.50
IV	4.88	0.00	3.18	0.13	*0.00	-34.80
V	4.37	0.71	4.07	0.34	0.67	-6.90
VI	5.77	0.22	5.55	0.00	0.22	-3.80
VII	6.22	0.22	6.51	0.26	0.38	4.70
VIII	6.44	0.22	5.48	0.13	*0.02	-14.90
IX	6.66	0.00	8.66	0.59	*0.03	30.00
X	4.07	0.34	3.33	0.00	0.06	-18.20

* Statistically significant difference ($p \leq 0.05$).

Constant: Frequency= 20 Hz, Pulse Duration = 100 µsec., Interpulse Interval = 50 msec, Duty Cycle = Alternating 5 sec on and 5 sec off

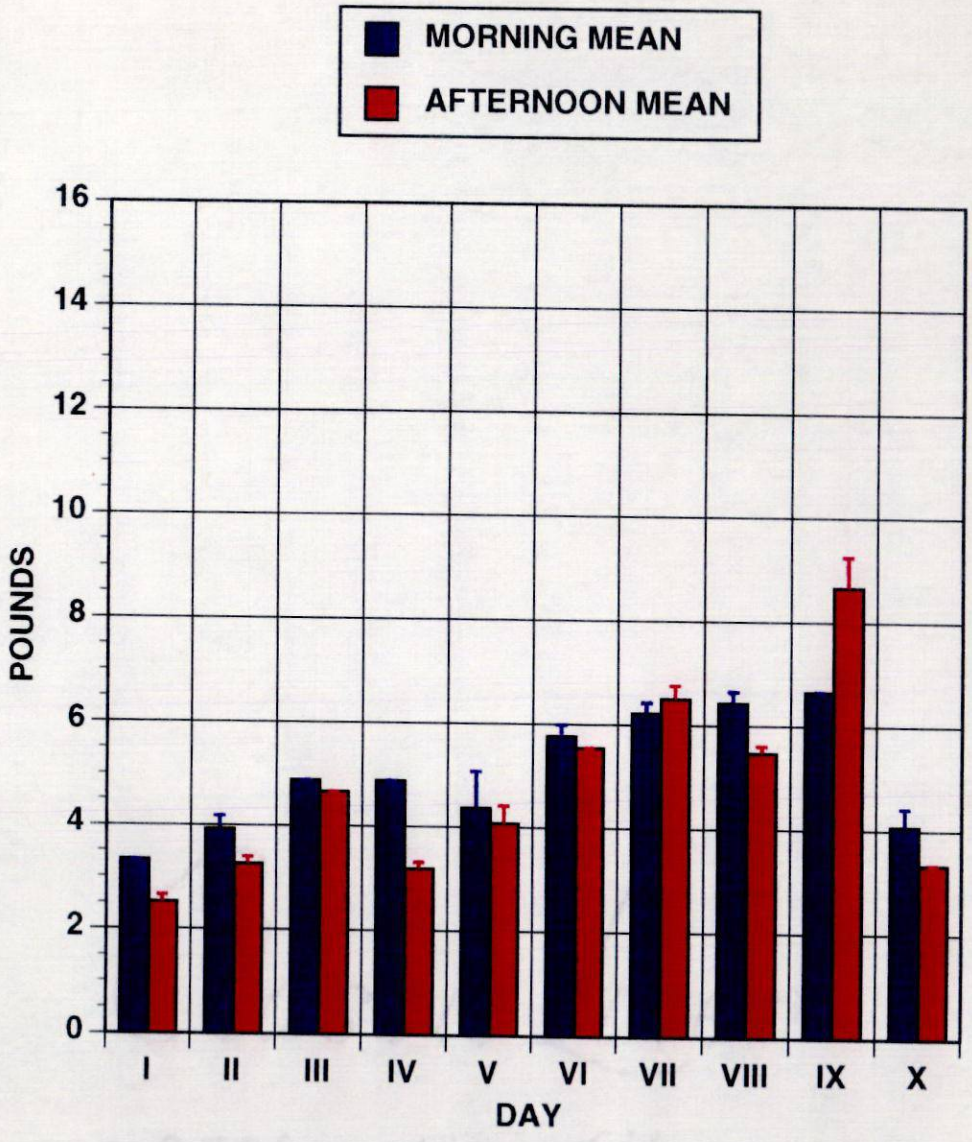


Figure 38. Daily average bite forces right, at T_1 (morning) and T_2 (afternoon) during alternating, chronic electrical stimulation of the right masseter muscle for Subject 5, Part B

Table 39.--Daily average bite forces left, at T_1 (morning) and T_2 (afternoon) during alternating, chronic electrical stimulation of the left masseter muscle for Subject 5 in Part B

DAY	T_1 (lbs.)	S.D.	T_2 (lbs.)	S.D.	p VALUE	% CHANGE
I	3.11	0.22	2.74	0.46	0.13	-11.90
II	3.33	0.00	2.89	0.22	0.07	-13.20
III	3.55	0.22	3.03	0.26	0.12	-14.60
IV	3.552	0.00	2.96	0.13	*0.02	-16.70
V	3.33	0.22	3.03	0.46	0.18	-9.00
VI	3.99	0.00	3.48	0.13	*0.02	-12.80
VII	3.85	0.26	3.60	0.26	1.00	-6.50
VIII	4.22	0.38	3.85	0.26	*0.04	-8.80
IX	3.48	0.13	3.11	0.22	*0.04	-10.60
X	4.22	0.22	3.78	0.44	0.07	-10.40

* Statistically significant difference ($p \leq 0.05$).

Constant: Frequency= 20 Hz, Pulse Duration = 100 µsec., Interpulse Interval = 50 msec, Duty Cycle = Alternating 5 sec on and 5 sec off

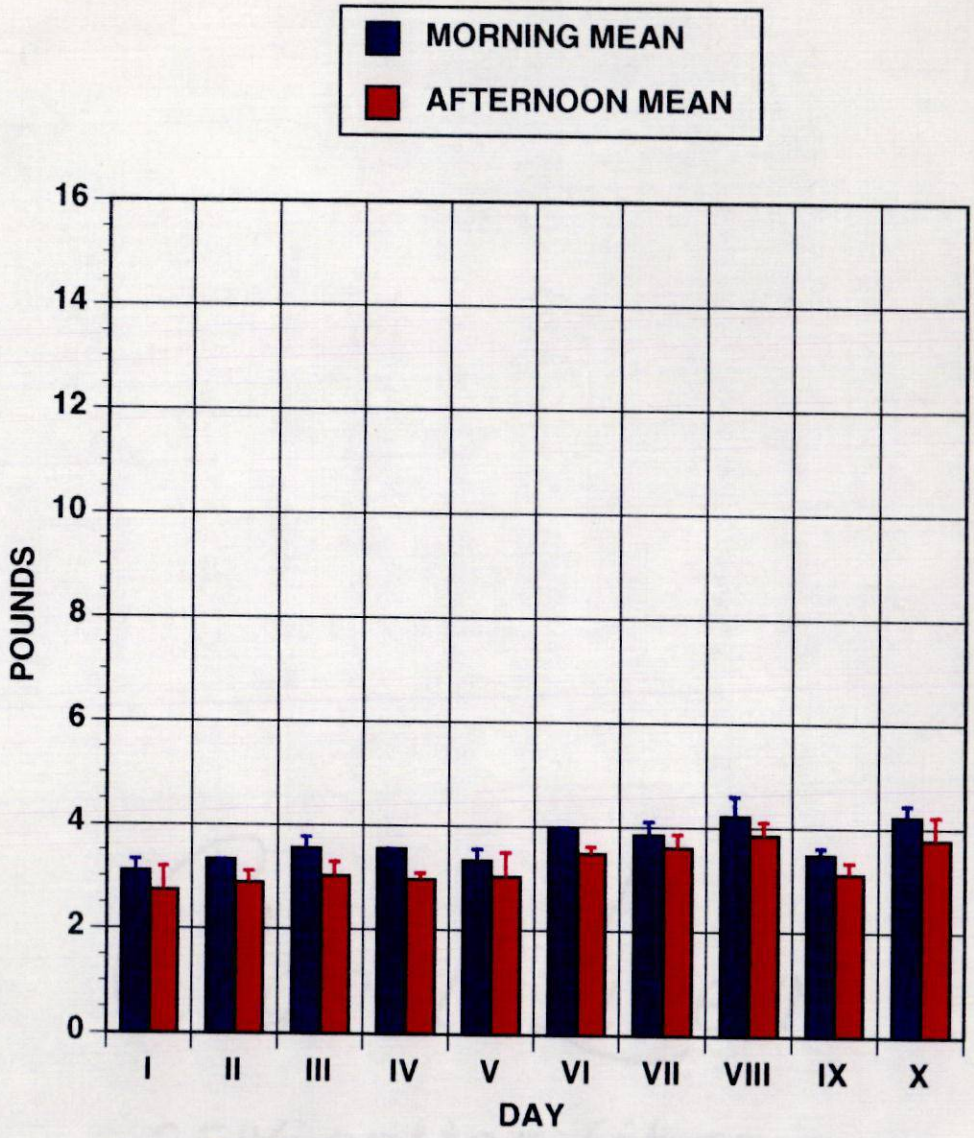


Figure 39. Daily average bite forces left, at T_1 (morning) and T_2 (afternoon) during alternating, chronic electrical stimulation of the left masseter muscle for Subject 5, Part B

Table 40.--Daily average maximum voluntary bite forces right, before (T_1, morning) and after (T_2, afternoon) alternating, chronic electrical stimulation of the bilateral masseter muscles for Subject 5 in Part B

DAY	T_1 (lbs.)	S.D.	T_2 (lbs.)	S.D.	p VALUE	% CHANGE
I	70.28	1.66	69.81	1.96	0.70	-0.70
II	64.18	1.28	67.93	5.49	0.20	5.80
III	70.28	7.94	67.93	15.09	0.81	-3.30
IV	68.40	29.51	66.06	11.28	0.88	-3.40
V	59.03	15.66	61.37	7.84	0.75	4.00
VI	61.37	13.19	65.12	8.18	0.63	6.10
VII	59.97	9.74	61.84	4.57	0.60	3.10
VIII	60.44	11.76	64.65	9.01	0.65	7.00
IX	69.81	6.91	66.06	5.08	0.08	-5.40
X	68.87	10.42	65.59	8.11	0.54	-4.80

* Statistically significant difference ($p \leq 0.05$).

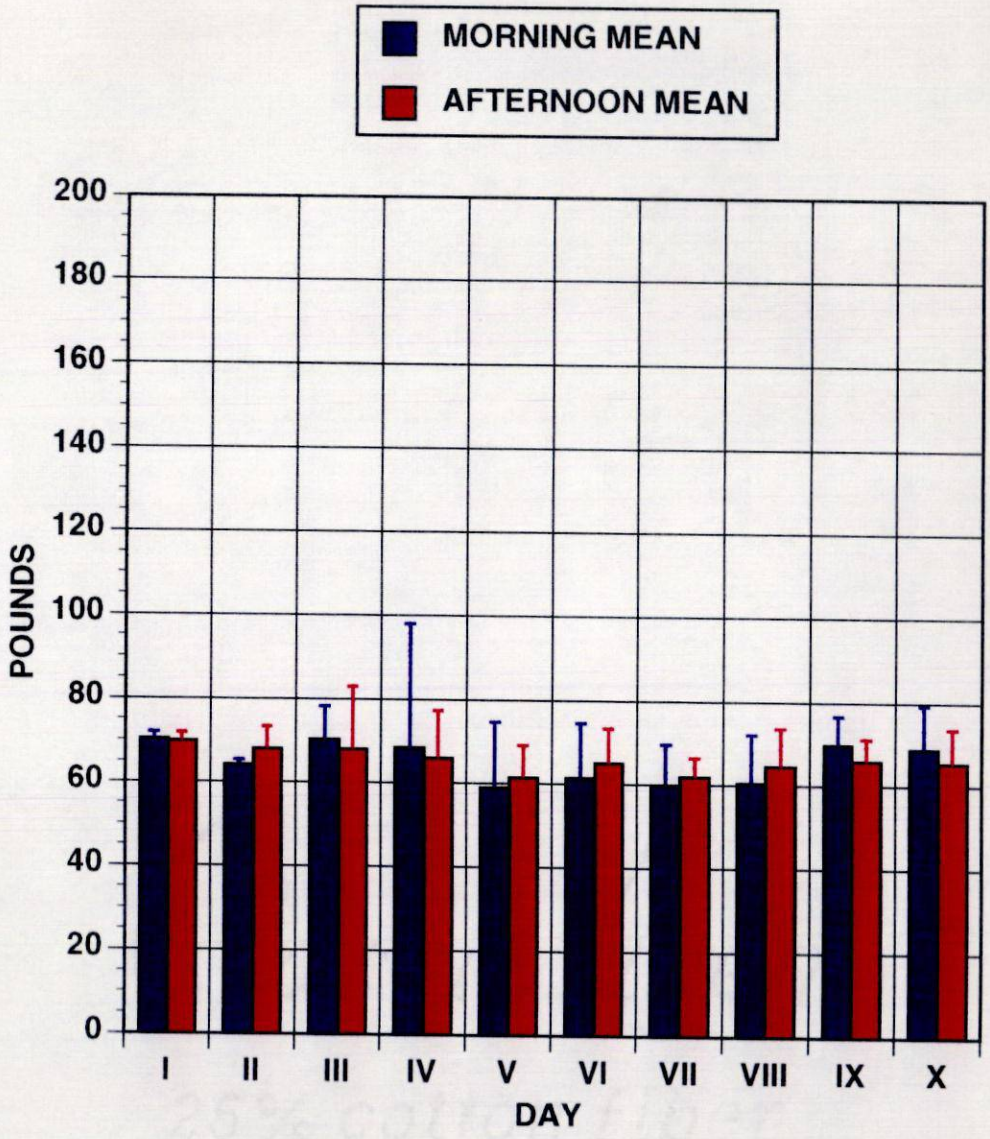


Figure 40. Daily average maximum voluntary bite forces right, before (T_1, morning) and after (T_2, afternoon) alternating, chronic electrical stimulation of the bilateral masseter muscles for Subject 5, Part B

Table 41.--Daily average maximum voluntary bite forces left, before (T_1, morning) and after (T_2, afternoon) alternating, chronic electrical stimulation of the bilateral masseter muscles for Subject 5 in Part B

DAY	T_1 (lbs.)	S.D.	T_2 (lbs.)	S.D.	p VALUE	% CHANGE
I	75.90	2.10	67.46	4.19	0.40	-11.10
II	65.12	14.67	71.21	7.52	0.52	9.40
III	71.21	3.55	63.72	7.48	0.12	-10.50
IV	59.50	5.39	66.06	8.18	0.30	11.00
V	73.09	4.51	68.40	6.50	0.10	-6.40
VI	73.09	8.67	79.65	15.18	0.11	9.00
VII	75.90	2.10	67.46	4.19	*0.04	-11.10
VIII	67.00	4.86	76.83	6.29	0.06	14.70
IX	89.02	0.00	89.48	7.66	0.90	0.50
X	81.52	1.96	72.62	6.52	*0.03	-10.90

* Statistically significant difference ($p \leq 0.05$).

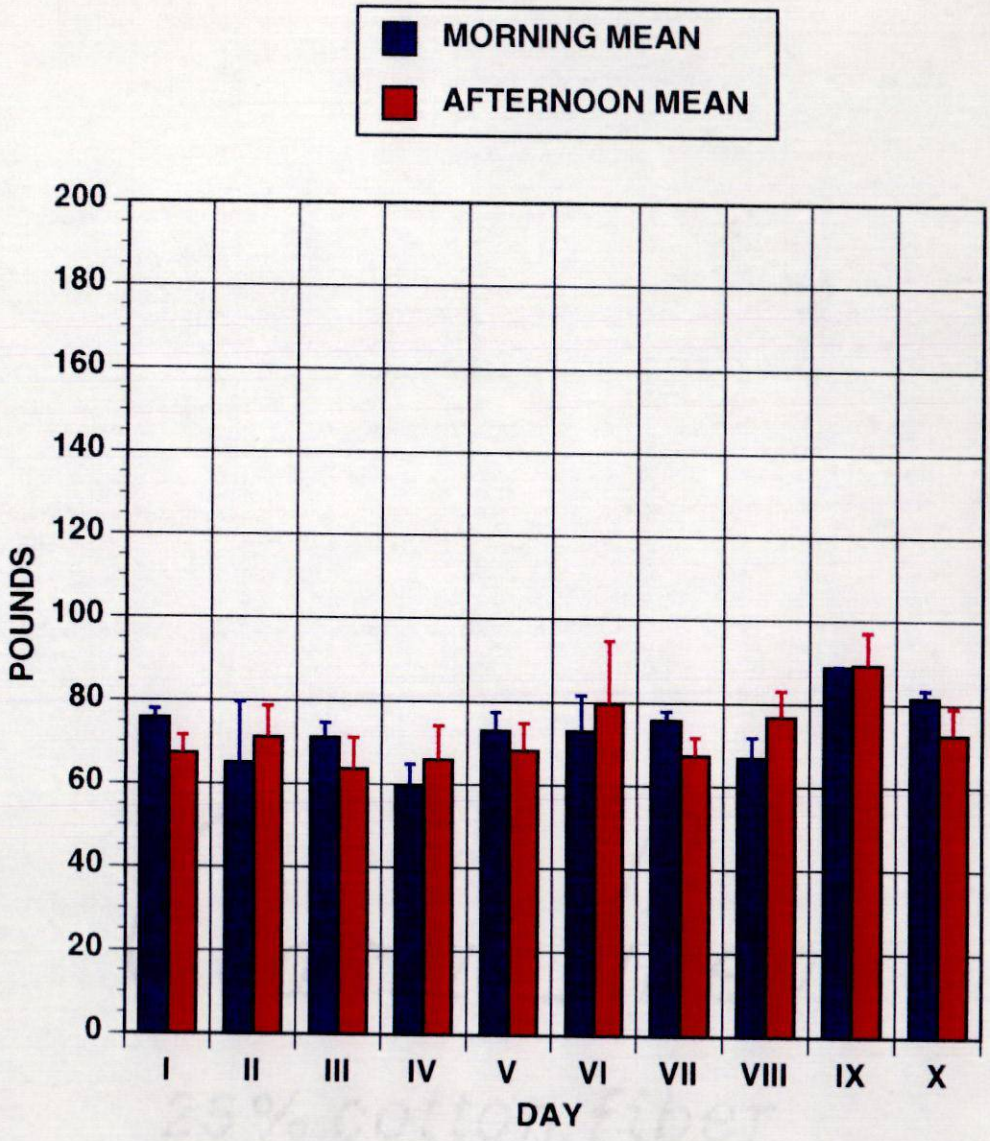


Figure 41. Daily average maximum voluntary bite forces left, before (T_1, morning) and after (T_2, afternoon) alternating, chronic electrical stimulation of the bilateral masseter muscles for Subject 5, Part B

APPENDIX II

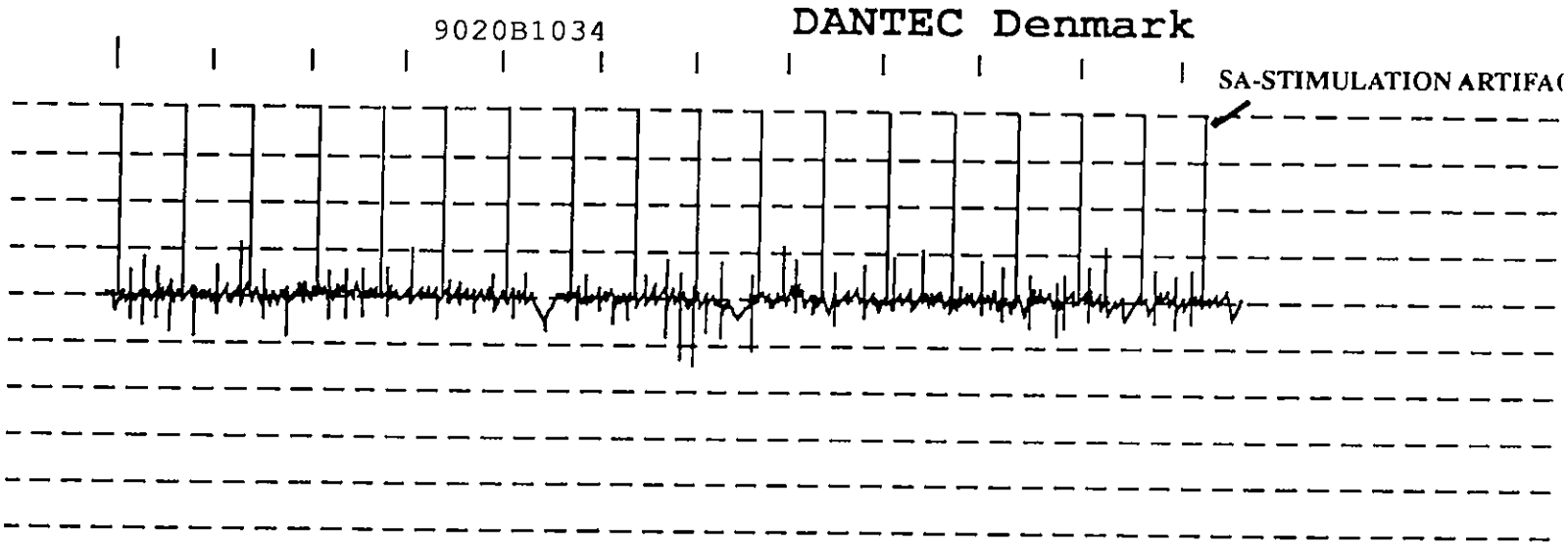


Figure 42. E.M.G. of the right temporalis muscle during simultaneous electrical stimulation of the bilateral masseter muscles in Subject I, Part I

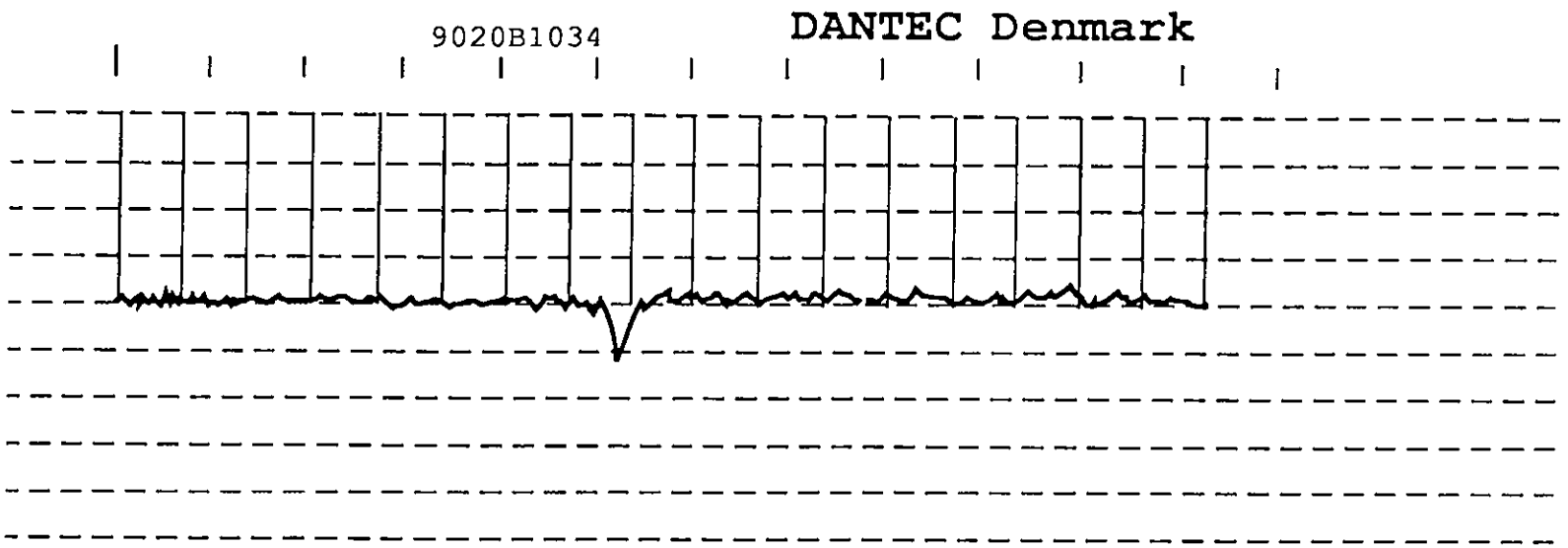


Figure 43. E.M.G. of the right temporalis muscle during electrical stimulation of the masseter muscles at rest and during voluntary mouth opening for all subjects

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