

The effects of neuromuscular function on craniofacial growth

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This paper has two purposes: (1) To give some orthodontic perspectives to the problem of the functional relationships between neuromuscular activities and bone growth and (2) To identify some basic concepts about muscle-bone biological problems *viz.*, how do muscle and bone affect one another during growth and function and how can the clinician take advantage of the situation?

I. Concept of models

In attempting to understand any complex biological problem, attack is often by one of two strategies: (1) Division of the larger problem into component parts for analysis or (2) Identification of models which represent the interacting factors in the problem. In either instance it is assumed and hoped that clarity comes from analysis after manipulation of variables or from having a less obscured view of the entire matter. In the problem at hand, three kinds of models are available for study: natural, experimental and accidental.

Natural models. Our preoccupation with experimentation has often caused us to neglect nature's own experiments. Some natural models shed light on the relationship between the orofacial neuromusculature and craniofacial growth, but few have been studied extensively. The commonest natural model of all is seen in the extreme natural variability

encountered randomly in the population. We tend to study the norm or central tendency of large populations perceiving it as the model for the universe while ignoring the particular lessons to be learned from the instances a few standard deviations from the mean. The gross craniofacial syndromes are often accompanied by developmental aberrations in sensory distribution and motor function (Henkin *et al.*, 1970). Much could be learned by further detailed serial studies of gross craniofacial syndromes particularly if neuromuscular parameters are included. Extensive unilateral loss of teeth results in unilateral skeletal adaptation. Such clinical problems abound in practice but are rarely studied.

Experimental models. Many experimental models have been used to study this problem, among which are the classic studies of denervation and extirpation of the orofacial musculature, the effects of varying the texture of food, altering the functional relationships of the jaws and altering the sensory input to parts. Our own current work is based on contrived experimental alterations in functional relations.

Accidental models. Before the discovery of the Salk and Sabin vaccines, poliomyelitis provided us with an extensive population of children showing the accidental effects of neuromuscular disease on bone growth. It is interesting to note in these studies that the length of the long bones altered less than the diameter during growth after poliomyelitis (Tower, 1939; Scott, 1954). Extensive studies have also been made of bone growth after amputation of various limbs or parts of limbs. Since several models for studying the relationship between muscles and bone are available, it would seem that much more use could be made of natural and accidental models.

II. Concept of different ways of measuring bony change

Changes in bone during experimentation, growth, and disease are studied and measured in many ways. It should not be assumed that changes recorded in one way in one study will be reflected in another method although our literature abounds with many such assumptions. An experiment which results in alteration in the external morphology of a bone does not necessarily bring about changes in the chemistry, the specific gravity or internal structure of the bone. Watt & Williams (1951), for example, in studying two groups of rats which were fed the same diet from a nutritional standpoint, but varied from the standpoint of texture, noted localized bony changes in the regions of muscle attachment and a generalized increase in the mass of bone. Care must be taken

in drawing inferences from studies, or in making comparisons between studies with methodological differences.

The neuromuscular side of the muscle-bone equation has been rather more neglected in the orthodontic literature probably because of the difficulties in quantifying neuromuscular changes. Many of the methods are difficult to effect without altering the muscles themselves. There is no procedure for muscles analagous to that of serial cephalograms. Nevertheless the methods of muscle study are far more sophisticated and varied than have generally been applied in correlated studies of muscle-skeletal relations in human craniofacial growth.

III. Concept of the uniqueness of the mandible

Orthodontists are most concerned with the mandible even though all bones of the craniofacial complex are of interest. Since there is abundant evidence that the mandible is a unique bone, care must be taken in adopting for the mandible findings which are proven on other bones. The human mandible in development, shape and function, is remarkably different from that of most lower mammals, although it does exhibit similarities to some of the higher primates. The mandible is unique in other ways. It is the only single midline bone activated by bilateral sets of muscle. Furthermore, the sensory and functional relationships of the teeth are unique. The temporomandibular joints, in addition to being bilateral, provide unusual sensory guidance for the mandible as well as providing growth centers in the articular cartilages. Finally, the role of the face in human psychology must not be forgotten, for the muscles of the face and jaws reflect our emotions and psychic state in a far more sensitive fashion than do any other muscle sets in the body. Because of these unusual features, general studies of skeletal and neuromuscular relations must be applied to the facial region with care and understanding.

IV. Concept of the influence of varying control systems

The intrinsic factors influencing development are present very early, providing an interrelated system of controls on growth. These controls are usually described as genetic, endocrinal, neurovascular and neurotrophic, though of course there are other controlling mechanisms as well.

Genetic. We may draw two important conclusions from Hunter's (1965) finding that the heritability of vertical facial dimensions is consistently

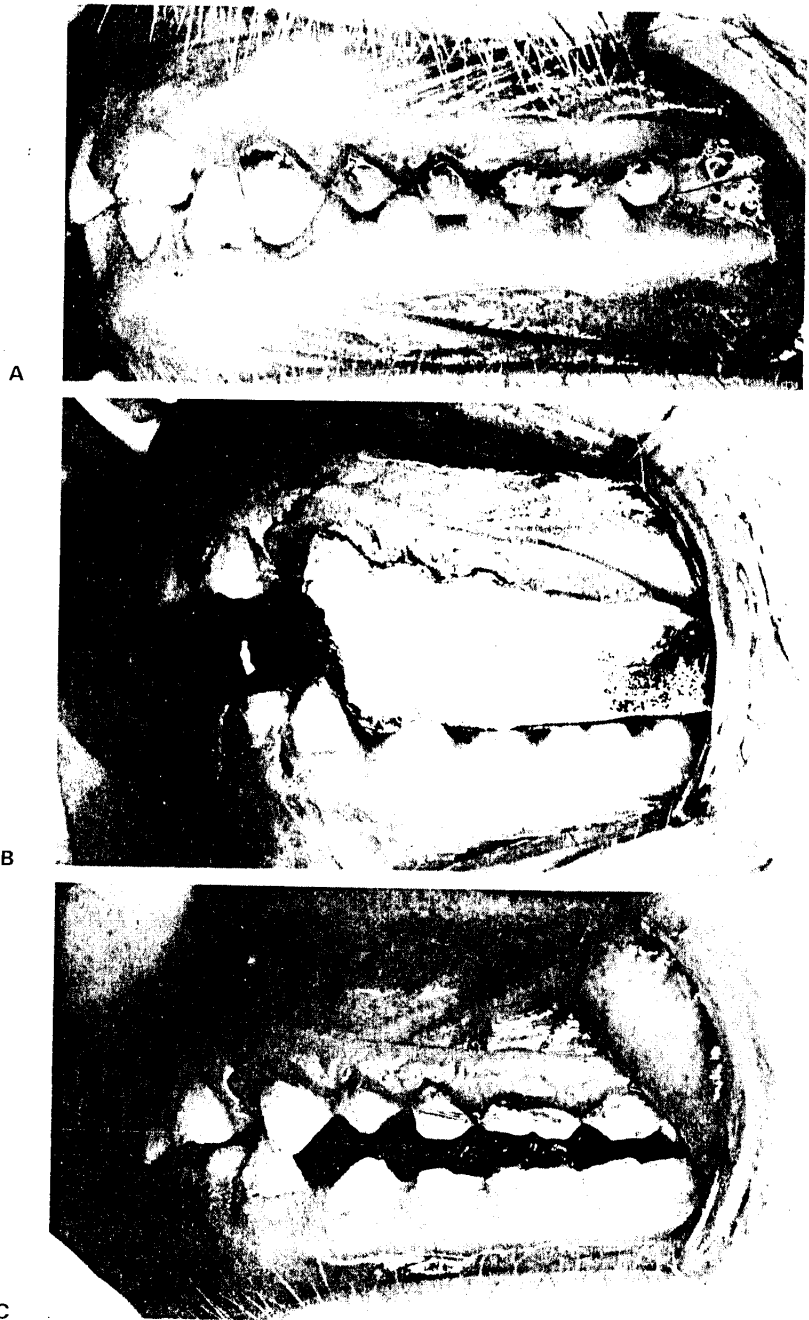


Fig. 1 Studies of increased vertical dimension in rhesus monkeys. The vertical dimension of a young adult female with normal occlusion (A) was opened 15 mm through the use of a cast gold maxillary onlay (B). After 6 months the appliance was removed and a Class II molar relationship was evident (C). This anteroposterior occlusal change occurred even though only the vertical dimension was altered.

higher than anteroposterior dimensions: (1) Environmental influences on the etiology of malocclusion will probably cause more anteroposterior variability than vertical and (2) Clinical alteration of function should affect anteroposterior growth more than vertical growth. The first is well documented in the literature, and our own research supports the second conclusion, since anteroposterior changes can be effected independently, but experimental vertical changes are usually accompanied by horizontal adaptations as well (Fig. 1). Further evidence of regional genetic effects is reported by Watnick (1972) and Harris, Kowalski & Watnick (1973) who demonstrated that the areas of greatest and least heritability in the mandible are highly localized.

Endocrinal. Acromegaly and the pubescent growth spurt are well known examples of endocrinal control dominance which have implications for facial growth. Much is made of timing treatment to coincide with the pubescent spurt, which poses problems in prediction. On the other hand, altering the functional environment earlier makes treatment possible during a period of growth increments significantly greater than that seen during the spurt, and obviates problems in timing. Definitive studies of the effects of the spurt on response to altered function have not been reported. Should we expect a greater response to functional therapy during the spurt or is the inertia of the pubescent spurt more difficult to overcome? Assumptions are often made but clear answers are not yet at hand.

Neurovascular. Boyd *et al.* (1967a, b) consider that neurovascular factors are probably as significant as neuromuscular factors in determining the rate and direction of growth in the gonial and coronoid regions of the mandible. Warner (1969) disputes their findings, considering that functional stimulation is essential in attaining the normal size and shape of a bone.

Neurotrophic. Neurotrophism is the word used to describe non-membrane conductive neurocellular activity which controls modifications and activities in other cells, regulating their growth and the general integrity of body structure. Moss (1972), who has done so much to advance the ideas of functional matrices in facial growth, has recently suggested that neurotrophic effects probably operate by regulating and controlling the genetic mechanisms of the target cells, and thus play a role in the growth and regulation of the functional matrix. Moss (1972) perceives neurotrophism, which must be clearly differentiated from neural transmission, as part of the homeostatically controlled series of processes in which the brain and other neural centers regulate the peripheral tissues and the

periphery in turn has feedback mechanisms to influence the regulatory centers. The concepts of neurotrophism are well known in developmental biology, particularly as they relate to muscle development and limb regeneration. Koski (1973) has recently shown a stable relationship through time of bony landmarks dependent on neural factors, indicating a possible postnatal neurotrophic effect. However, workers in craniofacial growth generally have done a better job of transferring basic concepts of skeletal growth to the orofacial region than they have the basic concepts of developmental neurobiology.

Our concern is the role of function as a controlling mechanism in the light of the varying influence of these other controlling factors just mentioned, i.e. genetic, endocrinal, neurovascular and neurotrophic.

V. Stage of development of observation

Neuromuscular and bony tissue have an intimate and changing relationship from the earliest days of differentiation; indeed, at the very start, they may be considered the same. The original genetically determined mass is affected by a continuing order of developmental mechanisms which become interrelated to a great extent before the more random external influences are seen. We must understand the internal predispositions and controls of the tissues and the effects of the various tissues and organs on each other during growth, before we can understand adaptation to random or contrived clinical changes and external influences.

Little is known of the 'signal' or 'message' which passes between neuromuscular tissue and bone at their interface, but it is inoperable until the initial muscle mass is neurally innervated and contractility is made possible. Even prenatally, after contractility appears, bone is influenced by the intimate presence and activity of muscle. Our own research has shown clearly that, postnatally, the nature and extent of specific skeletal adaptations depend upon the level of maturation.

In an experimental study of anterior mandibular displacement McNamara (1972, 1973) found mandibular adaptations of a general nature in younger monkeys, while in older animals adaptation in the mandible was more apt to be localized in the dento-alveolar region. It is significant that the marked changes in condylar growth rates and vectors seen in infants and juveniles were not observed in adolescents or young adults (Table 1). In contrast, maxillary skeletal adaptations were seen at all ages, though the response decreased with age. These findings suggest why clinicians may expect extra-oral traction appliances to the maxillary complex to effect changes over a longer period of time than functional jaw appliances used to promote mandibular growth.

TABLE 1 Summary of skeletal and dental responses to contrived functional protrusion. Note particularly the differences in maxillary and mandibular skeletal response (McNamara 1972, 1973).

	Mandible		Maxillary complex	
	Skeletal	Dento-alveolar	Skeletal	Dento-alveolar
I. Infant	++	o	+++	+
II. Juvenile	+++	o	+++	+
III. Adolescent	o	+++	++	o
IV. Adult	o	+++	+	o

VI. Concepts of changes other than in bone

As orthodontists we are interested in bone because it has seemed to us to be the most important tissue which our therapy changes and we perceive it as the raw material with which the face is formed; we also have better methods of studying bone and teeth than we do muscle, consequently we know more about bone and teeth. As growth and the environment (including the orthodontist) change the bone, what happens to other tissues, specifically the neuromusculature?

The interrelationship of muscle function and structural adaptations can be investigated by interrelating the findings from studies using different techniques, e.g. electromyographic, microscopic and cephalometric. By

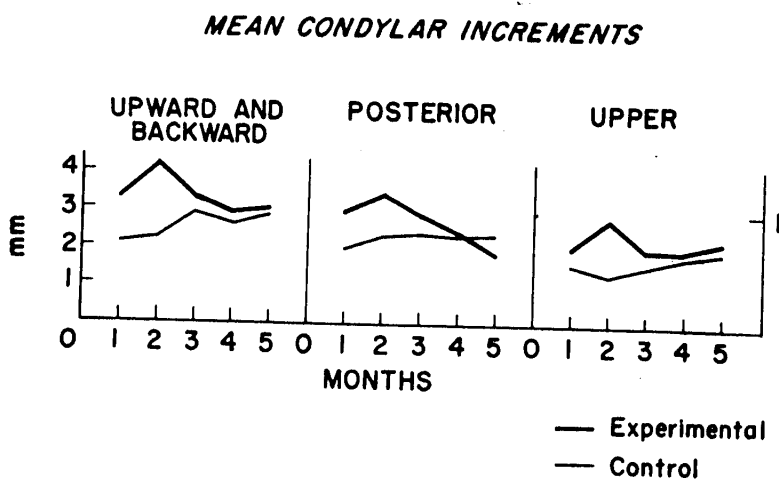


Fig. 2 Mean monthly condylar increments during contrived functional protrusion. Note the return to normal rates of growth by the fourth month (Elgoyhen *et al.*, 1972).

correlating findings from different data on the same experiments, the timing of the appearance and disappearance of altered functional patterns can be related to the rate and extent of skeletal and dento-alveolar adaptations. In our initial protrusive study (Moyers *et al.*, 1970; Elgoyhen *et al.*, 1972), using six juvenile monkeys, monthly growth increments were measured and a statistically significant increase in the extent of growth within the mandibular condyle was noted. Increased growth rates of the experimental animals tended to occur during the first three months, with a peak in the second. By four months the growth rate of the experimental animals was not significantly different from that of the controls (Fig. 2). It is important to note that the maximum rate of skeletal growth in these animals was during the second month. Similarly McNamara (1972, 1973) noted that maximum adaptive neuromuscular activity appeared in the lateral pterygoid muscle during the 4th to 8th weeks (Fig. 3). The decreasing rate of adaptive skeletal growth subse-

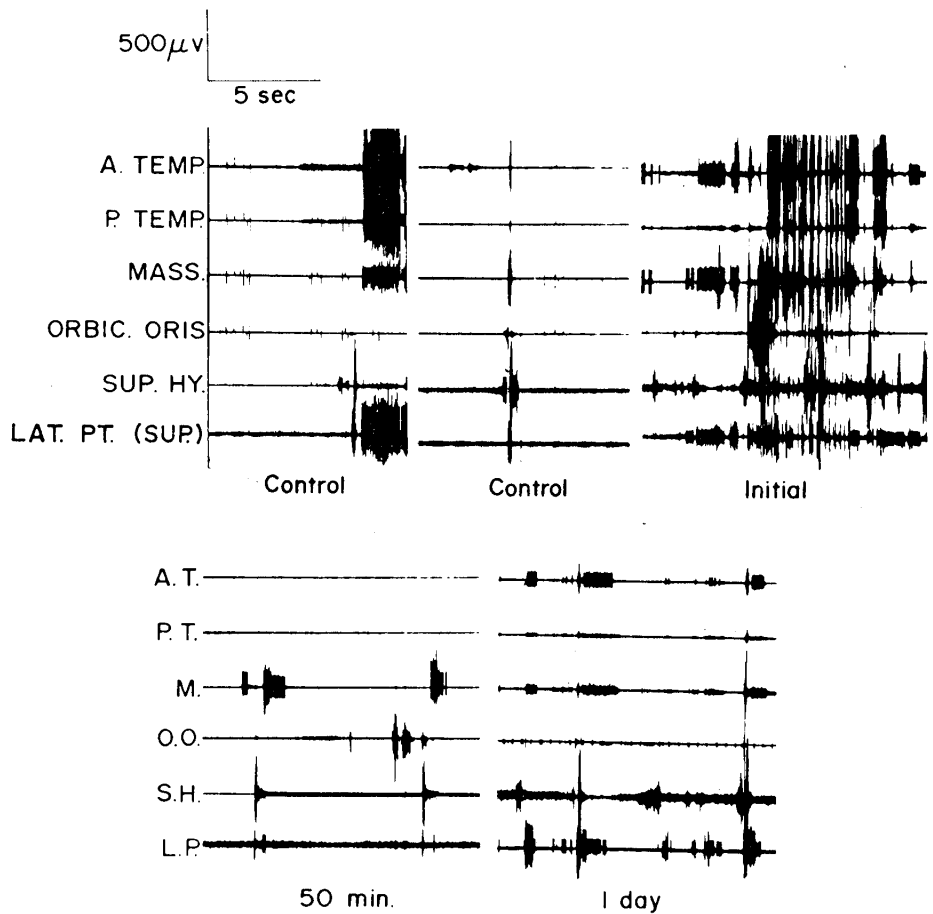


Fig. 3

quently seen was coincident with a reduction in lateral pterygoid activity. A similar correlation in time can be made with the microscopic findings of Stöckli & Willert (1971).

A chronological correlation exists between the occurrence and disappearance of altered neuromuscular function and the re-establishment of

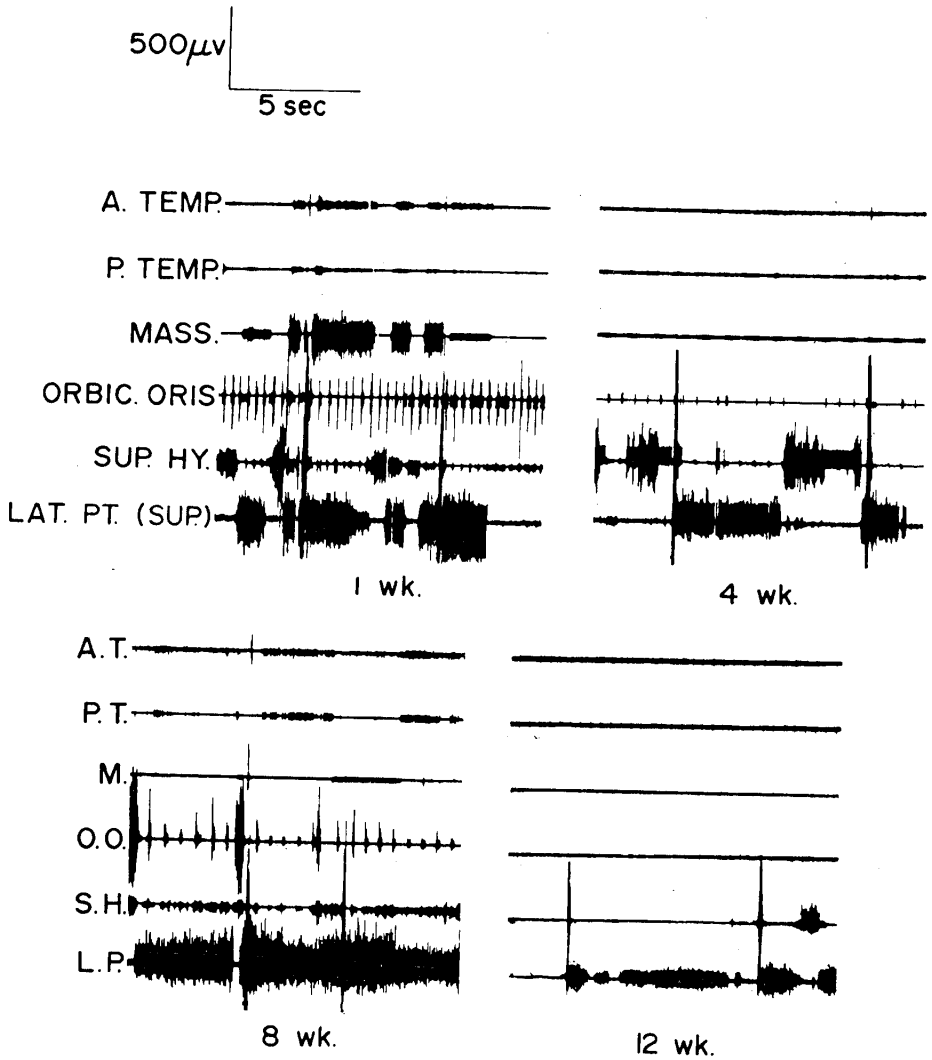


Fig. 3. Overview of typical muscle activity during the contrived protrusive function. In the *control* records, note the synergistic function of the elevators and superior head of the lateral pterygoid during such functional movements as clenching (left control) and swallowing (right control). *Initially*, coordinated muscle function was disrupted. By one week, lateral pterygoid activity is observed not only during movement but also during postural maintenance. This activity reaches a maximum at eight weeks and then becomes reduced by the end of the 12th week (McNamara, 1972, 1973).

structural balance, even though a direct cause and effect relationship is not conclusively shown. The initial reflex alteration in muscle function appears to be directly associated with the experimentally induced changes in the oral environment. Neuromuscular function, after the initial reflex response, reorganizes to compensate for the alteration in structural relations. Then as skeletal balance is restored through specific structural growth and remodelling adaptations, the neuromusculature re-establishes more efficient functional patterns. The final electromyograms are similar to those seen before the experiment, though there have now been extensive bony adaptations (McNamara, 1972, 1973).

Any single muscle or muscle group which has been elongated has several adaptive mechanisms available to re-establish functional homeostasis:

- (1) *Elongation of the muscles themselves.* Crawford (1954, 1961) has shown that the functional length of a growing muscle can be increased by increasing the amount it must shorten during contraction. He states that experimentally lengthened muscles increase in length either through the addition of sarcomeres or through reorientation of the muscle fibers themselves. This helps to explain our findings on growing animals. However, the effect of functional lengthening of the musculature in the adult has not been examined in a similar way.
- (2) *Altered neuromuscular feedback mechanisms.* Reflex control of tonus of the involved musculature can be modified through the activity of the fusimotor or gamma efferent system without direct obvious anatomical changes of the muscles themselves. The previous presumption that the stretch reflex served to regulate muscle length has recently been challenged on both experimental and theoretical grounds. Nichols & Houk (1973) have shown that autogenetic reflexes play an important role in muscular control since they compensate for variations in muscle properties.
- (3) *Migration of muscle attachments along bony surfaces.* Enlow (1962) and Hoyte & Enlow (1966) have described the nature of the moving muscle attachments to growing bone. Our own experimental data show that this change in muscle-bone relationships does occur as an adaptive mechanism.
- (4) *Change in muscle dimensions due to displacement and/or rotations of the bony elements.* The conditions of our experiments alter the functional length of certain muscles as the bony elements are separated and the mandibulo-maxillary relationship altered anteroposteriorly. There is lengthening of some muscles and shortening of others, creating differential stress within attached connective tissue which is presumably transmitted to bone.

All of the above suggests the following chain of events. Experimentally created occlusal configurations prompt a reflex positioning of the mandible to avoid occlusal interference. The reflexly adopted position of the mandible alters the length of a number of the muscles and neuromuscular function is reorganized to compensate for the alteration in structural relations. Skeletal growth and remodelling gradually adapts itself to the new neuromuscular environment. As the skeletal adaptations appear, the neuromuscular compensatory functions diminish until finally neuromuscular balance is seen, though an altered skeleton exists.

References

- Boyd, T. G., Castelli, W. A. & Huelke, D. F. (1967a) Removal of the temporalis muscle from its origin: effect on the size and shape of the coronoid process. *Journal of Dental Research*, **46**, 997-1001.
- Boyd, T. G., Castelli, W. A. & Huelke, D. F. (1967b) Arterial supply of the guinea pig mandible. *Journal of Dental Research*, **46**, 1064-1067.
- Crawford, G. N. C. (1954) An experimental study of muscle growth in the rabbit. *Journal of Bone and Joint Surgery*, **36B**, 294-303.
- Crawford, G. N. C. (1961) Experimentally induced hypertrophy of growing voluntary muscle. *Proceedings of the Royal Society (London)*, **154B**, 130-138.
- Elgoyhen, J. C., Moyers, R. E., McNamara, J. A., Jr. & Riolo, M. L. (1972) Craniofacial adaptation to protrusive function in young rhesus monkeys. *American Journal of Orthodontics*, **62**(5), 469-480.
- Enlow, D. H. (1962) Functions of the Haversian system. *American Journal of Anatomy*, **110**, 269-306.
- Harris, J. E., Kowalski, C. J. & Watnick, S. S. (1973) Genetic factors in the shape of the craniofacial complex. *Angle Orthodontist*, **43**, 107-111.
- Henkin, R. I., Christiansen, R. L. & Bosma, J. F. (1970) Facial hypoplasia, growth retardation, impairment of oral sensation and perception and hyposmia: a new syndrome. In *Second Symposium on Oral Sensation and Perception*, ed. Bosma, J. F., Springfield, Illinois: Thomas.
- Hoyte, D. A. N. & Enlow, D. H. (1969) Wolff's law and the problem of muscle attachment on resorptive surfaces of bone. *American Journal of Physical Anthropology*, **24**, 205-214.
- Hunter, W. S. (1965) A study of the inheritance of craniofacial characteristics as seen in lateral cephalograms of 72 like-sexed twins. *Transactions of the European Orthodontic Society*, **41**, 59-70.
- Koski, K. (1973) Variability of the craniofacial skeleton. *American Journal of Orthodontics*, **64**, 2, 188-196.

- McNamara, J. A. Jr. (1972) *Neuromuscular and skeletal adaptations to altered orofacial function*. Monograph No. 1, Craniofacial Growth Series, Center for Human Growth and Development, The University of Michigan, Ann Arbor.
- McNamara, J. A., Jr. (1973) Neuromuscular and skeletal adaptations to altered function in the orofacial region. *American Journal of Orthodontics*, **64**, 578-606.
- Moss, M. L. (1972) An introduction to the neurobiology of orofacial growth. *Acta biotheoretica*, **21**, 236-259.
- Moyers, R. E., Elgoyhen, J. C., Riolo, M. L., McNamara, J. A. Jr. & Kuroda, T. (1970) Experimental production of Class III in rhesus monkeys. *Transactions of the European Orthodontic Society*, **46**, 61-74.
- Nichols, T. R. & Houk, J. C. (1973) Reflex compensation for variations in the mechanical properties of a muscle. *Science*, **181**, 182-184.
- Scott, J. H. (1954) The growth and function of the muscles of mastication in relation to the development of the facial skeleton and of the dentition. *American Journal of Orthodontics*, **40**, 429-449.
- Stöckli, P. W. & Willert, H. G. (1971) Tissue reactions in the temporomandibular joint resulting from anterior displacement of the mandible in the monkey. *American Journal of Orthodontics*, **60**, 142-155.
- Tower, S. S. (1939) The reaction of muscle to denervation. *Physiological Review*, **19**, 1948.
- Warner, W. M. (1969) *The relationship of the temporal muscle to the form and architecture of the skull of the rat*. M.S. Thesis, University of Michigan.
- Watnick, S. S. (1972) Inheritance of craniofacial morphology. *Angle Orthodontist*, **42**, 339-351.
- Watt, D. G. & Williams, C. H. M. (1951) The effects of the physical consistency of food on the growth and development of the mandible and the maxilla of the rat. *American Journal of Orthodontics*, **37**, 895.