A Functional Cranial Analysis of Centric Relation

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We previously have used the method of functional cranial analysis to good effect in the study of cranial growth.[9] A fundamental attribute of this method is its operationalism, that is, it is predicated on an analysis of function. When we observe empirically that a biological operation or function is being carried out, we may then inquire into how it is carried out. In this effort we attempt first to identify the several anatomical tissues and organs involved in this function and then proceed to study the nature of the interaction between the skeletal and nonskeletal elements of the operational system as it carries out that function.

Since considerable study previously was devoted to mandibular growth[7] it seemed reasonable that a similar sort of analysis of the centric relation might be valuable.

A review of a variety of current clinical thought on the nature of centric relation as well as of the techniques advocated for the determination of this mandibular position, leaves the impression that many clinical workers regard centric relation, at least in adults, as real, functional, fixed, and reproducible.

However desirable such a position may be, particularly with reference to any given technique of clinical therapy, it is not correct. This article will suggest, on the basis of the biomechanics of the temporomandibular joint, that centric relation is never a functional position nor is it habitual or common. Its clinical demonstration appears to be iatrogenic. Further, current knowledge of the mode of growth and responsiveness of skeletal tissues to dynamically fluctuating functional environments suggests that the temporomandibular joint is significantly adaptive to alterations in oral function occurring in real time. Accordingly, possible, and biomechanically permissible, mandibular positions are not immutably fixed.

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This is not an academic problem. Many aspects of clinical therapy involve direct attempts at the regulation of either: (a) the mechanics, (b) the form (size and shape), or (c) the growth of the several anatomical components of the temporomandibular joint. Alternately, other therapeutic intervention is predicated indirectly upon an understanding of these same processes. To the extent that any therapy is based upon a correct comprehension of relevant biological principles, it is more likely to be successful. It is hoped that the material presented below will serve to clarify this matter, and so aid in the development of a more biologically sound therapy.

A Note to the Reader

The format of this symposium prohibits the more extensive type of bibliographic reference characteristic of monographic or scientific journal presentation. Operating within this restriction the author is aware that the following pages may appear to some readers as a series of *ex cathedra* pronouncements and seemingly not fully substantiated. Lacking appropriate references, the reader is unable to determine accurately the data upon which the views of the author are based. This problem is all the more acute since the positions taken below frequently diverge from what is, in certain circles at least, currently accepted clinical doctrine. Equally aware that the *argument ad hominum* is false, the author can only assure the reader that he has attempted to deal judiciously with these matters and that the principal statements have all met, at least, the necessary equivalent of interpersonal objective validification, insofar as presently available knowledge permits.

TEMPOROMANDIBULAR JOINT AS A DIARTHROSIS

There are few topics in dentistry about which so much misunderstanding and misinterpretation exist as that of the biomechanics of the temporomandibular joint. Despite this, it is possible to present a clear and simple understanding of this topic in a few basic statements.[7]

1. The temporomandibular joint is a typical diarthrodial joint. It does not differ from a typical diarthrosis in any way.
2. All diarthroses consist of concave and convex functional surfaces. The head of the condyle and the tubercular eminence, both convex, are the functioning osseous surfaces of this joint. The articular disc provides the reciprocal concave surfaces. The mandibular fossa is *not* a functional portion of this joint.
3. A typical diarthrosis contains both functioning and nonfunctioning joint surfaces. The former are covered with articular cartilage, the latter are not. The entire joint with both functioning and nonfunctioning surfaces is enclosed within a synovial membrane.
4. All movements at diarthrodial joints are rotations about a mechanical axis.
5. If an articular surface is perfectly hemispherical, its axis of rotation will be fixed. If the articular surface is ovoid, as is the case in almost all human mandibular condyles,[15] the axis of rotation will constantly shift, producing what is termed an evolute, that is, a constantly shifting axis of rotation (Fig. 1).
6. The first action of muscles, which produce motion at a diarthrosis, is to

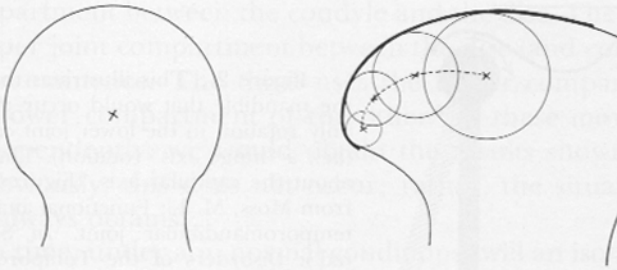

Figure 1. When a joint surface represents some portion of a perfect sphere, its axis of rotation will be a fixed position (*left*). However, when the joint surface has a constantly changing contour as in the femoral and mandibular condyles, the location of the axis of rotation changes instantaneously as rotation occurs. This causes the infinite number of instantaneous centers (axes) of rotation to form an evolute, such as is seen here (*right*).

produce compression of the articular surfaces, following which motion occurs. During function (i.e., during either the isometric or isotonic contraction of any muscle which tends to produce motion at that joint) there is always a compressive force exerted upon the functioning joint surface; the temporomandibular joint is "load-bearing." Such compressive loading is necessary for two reasons: (1) it permits the morphology of the functioning joint surfaces to guide and control movements; and (2) the act of compression plays an important role in permitting the synovial fluid within the joint membrane both to nourish the articular cartilage and to lubricate the joint surfaces.

7. Once the muscles producing motion at a joint contract and compress the reciprocal functioning joint surfaces, the envelope of motion occurring at the joint is determined entirely by the specific morphology of these same joint surfaces.

Further Implications

Gross anatomical, comparative, histological, and analytical biomechanical studies correlatively establish that the almost cartilage-free mandibular fossa is a nonfunctioning joint surface. Normally, no compression ever exists between the condylar head and this fossa, nor in man is there ever any functional articulation between the condylar head and the posterior osseous "wall" of this joint (the postglenoid tubercle). By any functional definition, utilizing the commonly accepted principle of biomechanics, the centric relation (defined as the position of the mandible relative to the maxilla when the condylar heads are in their most retruded position) is *not* a functional position. Indeed, it may be doubted if this "relation" of the condyle is other than iatrogenic.

MOVEMENTS AT THE TEMPOROMANDIBULAR JOINT

The clearest modern restatement of the mechanics of the temporomandibular joint is given by Hjortsjö[4] (cited also in Moss[7]). Three pairs of motions are possible at this joint: opening-closing; protrusion-retrusion; and lateral shift. The first occurs as a rotation in the lower

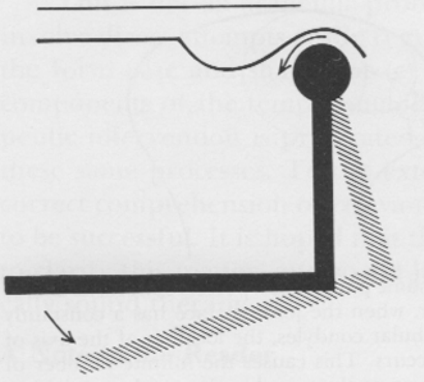

Figure 2. This illustrates the motion of the mandible that would occur *if* there were only rotation in the lower joint compartment (i.e., a "hinge axis" rotation). This is rotation about the capitular axis. (Figures 2, 3, and 4 from Moss, M. L.: Functional anatomy of the temporomandibular joint. *In* Schwartz, L. (ed.): Disorders of the Temporomandibular Joint. Philadelphia, W. B. Saunders Co., 1959, pp. 73–88.)

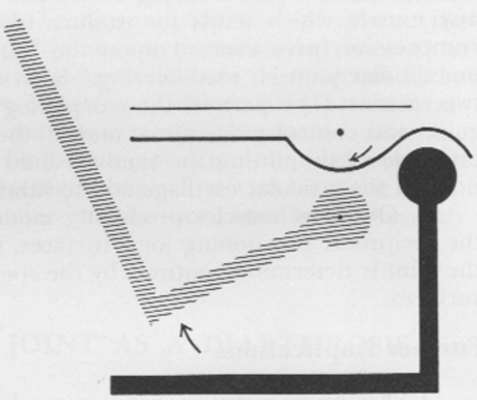

Figure 3. If rotation occurred only in the upper joint (rotation about the tubercular axis), this is the type of mandibular motion that would occur.

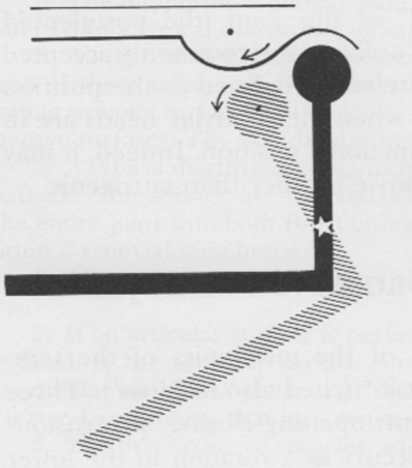

Figure 4. When simultaneous rotation occurs about both the capitular and tubercular axes simultaneously, a resultant axis of rotation occurs at the site of the mandibular foramen. This is the normal event in motion at the temporomandibular joint.

joint compartment between the condyle and the disc. The second occurs in the upper joint compartment between the disc (and condyle) and the tubercular eminence. The third uses the upper compartment of one side and lower compartment of the other.[5] If these movements could occur independently, we would obtain the results shown in Figures 2 and 3. Obviously, this does not occur; rather, the situation shown in Figure 4 always obtains.

At no time, under any normal conditions, will an isolated ("hinge") rotation of the condyle occur in the lower joint compartment without a simultaneous rotation in the upper joint compartment about the tubercular axis.[10]

We emphasize that the same statement can be made if we study the normal motion of mandible from centric relation to centric occlusion (the position of maximal intercuspation of the dentulous patient), and it is equally true for the motions between "physiological rest position" and centric occlusion.

Note that in any mechanical situation where rotation occurs simultaneously about two or more axes of movement, one can always describe a resultant axis of rotation. Such an axis exists for all motions of the temporomandibular joint at the mandibular foramen (Fig. 4).

The usual clinical descriptions of movements at this joint arose at a time when dentistry in general was not cognizant of the earlier German work on biomechanics; and by the process of repetitive and circular citation, coupled with an apparent inability to integrate more recent studies, the field remains troubled. The incorrect view holds that rotation occurs only in the lower joint compartment (i.e., between the condyle and the articular disc), while "translation" occurs in the upper joint compartment (i.e., to account for the relative motion between the anteriorly placed articular tubercle, or eminence, and the disc and condyle moving together).

By strict definition, rotation is a movement during which an object revolves about one of its axes of movement. The object (the mandible) changes its *position* is space (as measured in degrees) but does not change its *place* in space (as measured linearly). During translation an object changes its place but not its position. It has been repeatedly demonstrated by many workers independent of each other that what appears to be a "translation" of the condylar head during protrusive and retrusive positioning of the mandible, for example, is, in fact, a motion of rotation at the upper joint compartment with an axis of movement passing transversely through a given articular tubercle (Figs. 3 and 4).

From this work two critical points easily emerge. First, that all motions at this joint are rotations; there is no "translation" (or "slide") within this joint during function; and, second, at no time does a "hinge-axis" rotation occur at the condylar head (lower compartment) without a simultaneous rotation at a mechanical axis passing through the tubercular eminence (upper compartment).

In passing, we note that the normal range of condylar position relative to the tubercular eminence is greater than it is usually thought to be. The ability to place the condylar head on the anterior slope of the tubercle is found normally in a majority of people (70 per cent in one study), and so the concept of all clinical subluxation being equivalent to pathology is to be questioned seriously.

THE CENTRIC RELATION

We have repeatedly used the word normal in discussing joint motions, and for good reason. Normal motion at the temporomandibular joint is produced by the action of any combination of the muscles of mastication, the muscles of facial expression, and the muscles of the suprahyoid compartment. When these muscles contract voluntarily, they bring the functioning joint surfaces into compressive articulation. *Hence, as any normal mandibular function begins, by definition, then the condylar heads are not, and cannot be, in a centric relation.* Admittedly by the use of external (manual) force, local anesthesia, or under operator instruction, with or without the aid of a variety of intraoral and extraoral mechanical appliances, a patient may be trained (or exhausted) into producing a nonphysiological, "trained," and iatrogenic centric relationship.

However, there is no scientifically satisfactory demonstration by any worker that the centric relation is either a position usually (or habitually) assumed by a normal individual or, more importantly, that it is ever a normal functioning position; indeed, it is neither.

Without entering further into the clinical implications of a centric relation (which are discussed fully in Dr. Levy's article in this symposium), it is fair to note that the principal use of, even the concept of, a centric relation in dentistry is to aid reproduction of the envelope of motion of a specific patient's temporomandibular joint. To the extent that a dentist believes that this relation is static, constantly reproducible, and unchanging, even in the adult, he bases his therapy on poor grounds. If he further adds to this a belief in an isolated "hinge-axis" rotation, he is totally incorrect.

The facts are otherwise. As shown below, there is every reason to believe that the temporomandibular joint is significantly responsive to a dynamically altering environment and capable of significant morphological (and hence biomechanical) adaptation.

FUNCTIONAL MATRIX HYPOTHESIS OF MANDIBULAR GROWTH

The method of functional cranial analysis and its derivative concept, the functional matrix, is one of current interest in the broad area of craniofacial growth studies.[9] Based on an operational view of the head as a region where certain *functions* are carried out, the method then describes for each function: (a) all of the tissues, organs, and functioning spaces (oral, nasal, pharyngeal) necessary to completely carry out a given function; as well as (b) all of the skeletal tissues (bone, cartilage, tendon) required biomechanically to support and/or protect the former; (a) is called the functional matrix, and (b) the skeletal unit.

More than two decades of experimental and analytical studies support the claim that the size and shape (i.e., the form), the position in space, the changes in these attributes with time (i.e., the growth), as well as the very maintenance in being of all skeletal units are always, and without exception, secondary, compensatory and mechanically obligatory responses to the temporally (and functionally) prior demands of the specifically related functional matrices.

INTRINSIC AND EXTRINSIC FACTORS

In clinical circles often there is a misunderstanding of the processes by which the form and growth of skeletal tissues and organs (bones) are regulated. Certainly some continue to believe that intrinsic (genetic) factors play, if not a sole, at least a predominant, role. To maintain such a position it is necessary to hold that there is encoded within the genetic apparatus of each skeletal tissue cell specific information that will, of itself, regulate the amount, rate, and site of skeletal tissue deposition and resorption. This and only this could permit one to speak of, for example, the form and growth of the temporomandibular joint as being genetically (intrinsically) predetermined.

Available data from many fields and from many workers make it more reasonable to suggest that extrinsic (environmental) factors primarily regulate skeletal unit form and growth. We may subsume such extrinsic factors within the concept of the functional matrix. In this view, for example, the mandibular teeth would be a functional matrix and its supporting alveolar bone its skeletal unit. The formation, growth, and maintenance of alveolar bone is a secondary and compensatory response to the functional demands of teeth (or at least of tooth roots). Similarly, the angular process of the mandible is a response to the functional demands of both the medial pterygoid and masseter muscles. In the total absence (or loss) of the functional matrix, the skeletal unit will disappear (or not appear). For example, in a totally and congenitally anodontic patient, alveolar bone will never appear, whereas the response of alveolar bone following total tooth loss is clinically commonplace.

Let it be clearly understood that this position does not deny the role of intrinsic factors in development. There can be no doubt that the ability of an osteoblast, osteocyte, or osteoclast, for example, to perform their several differing functions can occur only when the intracellular encoded genetic apparatus is functioning appropriately. The selective repression and de-repression of one or another genetically regulated cellular metabolic pathway must occur for active skeletal tissue growth to occur. What we do deny is that the temporal and spatial regulation of these genetic processes in the skeletal tissue cells also is a *primary*, intrinsically-regulated event of the same genetic apparatus in the same cells. Rather, the regulation is extrinsic. In a phrase, the intrinsic factors in skeletal tissue cells provide only the possibility of an appropriate response to an extrinsic stimulus.

Finally, we only wish to note that other studies strongly suggest that the *primary* site of genetic regulation does not exist within the genes of the functional matrices either. It is now quite reasonable to suggest that the central nervous system, via neurotrophic regulatory process, itself constantly monitors the genetic controlled metabolic activity and phenotypic expression of all innervated cells and tissues.[8]

It is inappropriate to consider this matter further here. The reader is referred to the publications of skeletal geneticists for further data on the nonexistence of "skeletal genes."[2, 3, 13, 14]

The further developmental effects of function in a diarthrodial joint are well known, among which are: (a) it has been demonstrated repeatedly that in the organogenesis of all diarthrodial joints the formation of a concave joint surface is a secondary response to the presence of a convex surface; and (b) once the primary joint cavity has appeared in early fetal stages, unless function (produced by prenatal contraction of related musculature) occurs at that joint, the joint itself will tend to ankylose. In summary, there is no reason to believe that any attribute of the morphology of the temporomandibular joint, in particular, or of any diarthrodial joint, is under primary intrinsic control; rather, it appears that joint morphology is a secondary response to altering functioning demands.

ROLE OF THE CONDYLE IN MANDIBULAR GROWTH

At one time it was quite popular to assert that the mandibular condyle contained a cartilage that was functionally, if not morphologically, identical to that of the growth plate of a long bone. It was held, further, that this condylar cartilage was a most important site of mandibular growth; that it was by the processes of cartilagenous multiplication and subsequent endochondral bone replacement that the mandible was, literally, moved (or better "pushed") "downward and forward," as it were, as the upgrowing condylar cartilage "pushed" against the mandibular fossa and so, by a resultant force, lowered the mandible in space.

This classic view is as attractive in its mechanistic simplicity as it is biologically and demonstrably false. As proposed by us earlier and subsequently objectively verified by a number of workers, using the data of both a variety of experimental models and of clinical experience, the secondary and compensatory nature of the mandibular condylar cartilage and the extrinsic regulation of its growth are now established clearly.[1]

The condylar cartilage is an articular cartilage and histologically quite unlike the growth plate of a long bone. As numerous studies have demonstrated, removal of the condylar cartilages in young, growing animals does not interfere with subsequent changes in size, shape, or spatial position of the remainder of the mandible. Essentially all that is produced by such condylectomy is a lack of further growth (or presence) of a condylar process (the regeneration of such condyles is discussed below).

During orofacial growth the mandible as a whole is passively and literally lowered in space. This occurs as the volume of the oral (and nasal and pharyngeal) functioning spaces is increasing with age. Since the mandible orginates, grows, and exists completely embedded within an orofacial capsule, and since this capsule surrounds these functioning spaces, the volumetric enlargement of these spaces causes the surrounding capsule to expand. Although the analogy might superfically resemble increasing air volume causing a balloon to expand, such growth, in fact, is the result of epithelial mitosis neurotrophically regulated via afferent nerves.[8]

The mandible, then, is passively lowered in space during orofacial capsular expansion. One result of this is a tendency to distract the head of the condyle away from its superior, functional articulating surfaces. As a response to this distraction, the condylar cartilage proliferates. This effect is not at all mystical, but is well known in orthopedics as the Hueter-Volkmann law. This law states that when compressive loading on any cartilage is increased, chondrocytic multiplication decreases, and when compressive loading is decreased, such cartilage growth increases.

The rate of secondary, compensatory, and mechanically obligatory responsive growth by the condylar articular cartilage to the passive lowering, caused by the expansion of its functional matrix, is usually closely coordinated with the rate of such lowering. In effect, it is rather like a man running upwards on a "down" escalator at precisely the same rate and, as a result, appearing to stand still in space. That is why we cannot note this process clinically by observing the position of the growing condylar head in the temporomandibular joint of a given patient. However, experimental histological studies, using appropriate "markers" of skeletal growth, have demonstrated the correctness of this view repeatedly

ADAPTATION AT THE TEMPOROMANDIBULAR JOINT

It is significant to note that while the body of the mandible develops as intramembranous bone, the condylar process originates as a separate mass of cartilage that later fuses to the rest of the lower jaw. Further, this cartilage is of a type known as secondary cartilage. For our present purposes it is sufficient to know that experimentally secondary cartilage has been shown to develop as a response to functional movements between bones. Hence, the very origin of the condyle is functionally related. It is not surprising, therefore, that joint function plays a major role in its further growth and development.

POSTNATAL COMPENSATION

Given the reactive, compensatory nature of normal mandibular condylar growth, it is not surprising to find that this same cartilage seems well able to adapt its morphology to altered functional and spatial demands.

Abundant data clearly demonstrate that all components of the temporomandibular joint are capable of significant normal remodeling of form with increasing age.[6] Further, in both experimental and clinical studies, when the *appropriate* methods are used, it has been shown by many workers that when the compressive loadings (brought about by normal joint functioning) are reduced, as by *appropriate* repositioning of the lower jaw, adaptive condylar growth occurs; whereas conversely, when such compressive loadings are increased, an inhibition of condylar articular cartilage growth ensues. Both are obvious examples of the clinical application of the Hueter-Volkmann law. Parenthetically, an inappropriate method is one which does not apply this law to the specific clinical situation.

REGENERATION OF CONDYLES

An interesting example of condylar adaptation is seen in the now frequent reports of regeneration of a function mandibular condyle after condylectomy, both in animals and in man. The theoretical basis for this phenomenon is clear. If we leave the appropriate functional matrix intact and, if we also have a source of potentially chondrogenic cells in the condylectomy site to respond to the demands of the functional matrix, then such regeneration should and, in some cases, does occur. It would appear that either one or both conditions fail to exist in the condylectomies not followed by regeneration. Clearly, this is an excellent example of both the adaptiveness of the condyles as well as of the primary morphogenetic role of functional matrices.[12]

ROLE OF THE LATERAL PTERYGOID MUSCLE

Related to the general statement that function and joint motion are significant extrinsic (environmental) determinants of condylar form and growth, recent data clearly demonstrate that the lateral pterygoid muscle, which attaches to the condylar process and disc and acts as a type of functional matrix, plays a significant role in the regulation of the mandibular condylar cartilage. While the details cannot be discussed here,[11] it is certain that this muscle plays a role in the continuing adaptive morphological changes at this portion of the temporomandibular joint.

In another article in this symposium, Dr. McNamara discusses other aspects of neuromuscular physiology as they relate to adaptation of mandibular position and function. Suffice it to indicate here that alterations in the neuromuscular apparatus, either primary or as responses to other external environmental alterations, are quite capable of influencing modifications of temporomandibular joint morphology. Moreover, the relatively greater variation existing in the neuromuscular apparatus of any given individual, day by day, if not hour by hour, make it reasonable to presume that condylar position is subject to significant intra-individual variation in position during relatively short time periods.

CENTRIC RELATION

Despite its theoretical clinical utility, the search for an immutable condylar position, defined as a centric relation, within the temporomandibular joint unfortunately is an ephemeral undertaking. This is so for three reasons, as strongly suggested by the data above.

1. In biomechanical terms the centric relation is a nonfunctional position. This is so at any age and in any normal condition.

2. Over relatively long periods of time, the morphology of all functional surfaces of the temporomandibular joint is capable of significant adaptive alterations. These are normal compensatory responses of skeletal units to the prior alterations of functional matrices.

3. In much shorter time periods, the dynamically fluctuant state of the neuromuscular apparatus makes it reasonably certain that intra-individual variation in condylar positions can exist.

Any clinical procedure that potentially demands the location of a given patient's "centric relation" position within the tolerances of a millimeter or two must ignore the considerations given above. If we add to that the possibility that an isolated "hinge-axis" rotation of the condyle also is being sought clinically at that "centric relation" position, then we are even further removed from sound biology.

With a full understanding of the necessity for appropriate articulator registration of the potential range of a patient's temporomandibular joint motions, the clinician is urged not to convert the articulator into another Procrustean bed.

REFERENCES

1. Durkin, J., and Irving, J. T.: The cartilage of the mandibular condyle. Oral Sci. Rev., *2*:29–99, 1973.
2. Grüneberg, H.: The Pathology of Development. New York, J. Wiley and Sons, Inc., 1963.
3. Gruneberg, H., and Wickramaratne, G. A.: A re-examination of two skeletal mutants of the mouse, vestigeal tail (*vt*) and congenital hydrocephalus (*ch*). J. Embryol. Exp. Morphol., *31*:207–222, 1974.
4. Hjortsjö, C.-H.: Studies on the mechanics of the temporomandibular joint. Kung. Fysiograf. Sällsk. Handl., *66*:1–24, 1955.
5. Hjortsjö, G.-H., and Sonesson, B.: Notes on the mechanism in the temporomandibular joint. Odont. Rev., 5:167–175, 1954.
6. Moffet, B. C., Johnson, L. C., and McCabe, J. B. et al.: Articular remodelling in the adult temporomandibular joint. Am. J. Anat., *115*:119, 1964.
7. Moss, M. L.: Functional anatomy of the temporomandibular joint. *In* Schwartz, L. (ed.): Disorders of the Temporomandibular Joint. Philadelphia, W. B. Saunders Co., 1959, pp. 73–88.
8. Moss, M. L.: An introduction to the neurobiology of oro-facial growth. Acta Biotheoret., *22*:236–259, 1972.
9. Moss, M. L., and Salentijn, L.: The primary role of functional matrices in facial growth. Am. J. Orthod., *55*:566–577, 1969.
10. Nevakari, K.: "Elapsio praearticularis" of the temporomandibular joint. Acta Odont. Scand., *18*:123–170, 1960.
11. Petrovic, A., Ovdet, C., and Masson, N.: Effets des appareils de propulsion et de retropulsion mandibulaire sur le nombre des sarcomères en sirie du muscle pterygoïdien exteme et sur la croissance du cartilage condylien du jeure. L'Orthod. Franc., *44*:191–212, 1973.
12. Poswillo, D. E.: The late effects of mandibular condylectomy. Oral Surg., *33*:500–512, 1972.
13. Sawin, P. B., and Hamlet, M.: Morphogenetic studies of the rabbit. XL. Growth gradient interaction and functions in morphology. J. Morphol., *130*:397–420, 1970.
14. Sawin, P. B., Fox, R. R., and Latimer, H. B.: Morphogenetic studies of the rabbit. XLI. Gradients of correlation in the architecture of morphology. Am. J. Anat., *128*:137–146, 1970.
15. Yale, S. H., Allison, B. D., and Hauptfuehre, J. D.: An epidemiological assessment of mandibular condyle morphology. Oral Surg., *21*:169–177, 1966.

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