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# The differential roles of periosteal and capsular functional matrices in orofacial growth

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SUMMARY Historically students of craniology believed, erroneously, that only the active processes of surface deposition and resorption and of interstitial expansion were involved in skull growth. The introduction of the method of functional cranial analysis placed primary emphasis on the morphogenetic role of the functional matrix. The two principal types of functional matrices, the periosteal and the capsular, are defined. The former alter the size and shape of the skeletal tissues while the latter alter spatial position. The majority of facial skeletal growth is shown to be due to the passive translation of the skeletal tissues within the orofacial capsule, responding to the prior and primary volumetric expansion of the oro-nasopharyngeal functioning spaces, acting as capsular matrices.

## Introduction

The biological mechanisms of orofacial growth remain a topic of perennial interest to all students of craniology. The skeletal tissues, cartilage and bone, are the usual focus of both clinical attention and therapeutic intervention, as well as the primary objects of roentgenographic observation. Further, osseous tissues are the only materials available to the vertebrate palaeontologist and human osteologist.

For these reasons, among others, it is customary linguistically and conceptually to regard skeletal tissues as possessing primary and intrinsic growth processes; self-sufficient to account for the observed phenomena of normal and abnormal orofacial growth.

Two centuries of experimentation on skeletal tissues with both vital dyes and metallic markers established, very early, that osseous tissues alter their form only by surface accretion and resorption, while cartilaginous tissues have an additional and significant process of interstitial expansion. Earlier controversy regarding the role of periosteum and perichondrium as vital sources of these surface phenomena was resolved finally only four decades ago (Keith, 1919). Utilizing these data, an attempt was made some 25 years ago to describe the biological processes of orofacial growth as follows: a) bone and cartilage alter their size and shape (form) by surface deposition and removal; and b) skeletal organs (bones) move relative to each other as a result of an expansive, 'pushing,' force primarily generated by the interstitial expansion of either sutural or cartilaginous tissues. In this formulation interosseous sutural tissues were held to be analogous to the growth plates of long bones; while the nasal septal, mandibular condylar and basal cranial synchondroses were thought to be homologous to such growth plates.

In recent decades, an increasing corpus of experimental data conclusively disproved the generation of any primary expansive force within either sutural or splanchnocranial cartilaginous tissues. Contemporary residual statements purporting to re-establish these erroneous concepts in orthodontics are due, in part, to the less than satisfactory biological background of some individual investigators and, in part, to a continued and provincial adherence to earlier and incorrect conceptions of cranial growth mechanisms long since discarded by the mainstream of modern craniological thought. Papers continue to appear written by authors who are obviously still entrapped in the spurious dichotomy between the data of Brash (1934) and of Massler and Schour (1951) on the mechanism of cranial vault growth; the former describing a thick-skulled mammal and the latter a thin-skulled form. The mutually confirmatory work of other modern investigators refute such a biologically unsophisticated thesis (cf: Hoyte, 1966).

Returning to our discussion, the older, classic, concepts attempt to describe how the size and shape of skeletal tissues altered, but not why. Further they do not describe satisfactorily how skeletal organs change their relative positions, not even when we accurately quantify the amount of 'drift' or 'translocation' of bones produced by the net effect of surface appositions and resorptions. The operational method of functional cranial analysis is a significant contribution to the solution of these problems. The experimental and theoretical bases of this technique are published extensively elsewhere (Moss, 1962, 1968a,b, 1969a, b; 1969a,b; Moss and Young, 1960). Succinctly, the head is a region in which a number of functions are carried out (respiration, vision, olfaction, digestion, etc.). Each function is accomplished completely by a functional cranial component which has two parts. The totality of any function is performed by a functional matrix, and each such matrix is protected and supported biomechanically by a skeletal unit.

Recent work permits us to define two types of functional matrices and two related types of skeletal units. The first is the periosteal matrix. Muscles, glands, neurovascular

bundles and teeth are excellent examples. These matrices act directly upon individually related micro-skeletal units. Such matrices produce morphological expression of their operational demands by the active processes of deposition and resorption, thus altering the size and shape (i.e., the form) of their micro-skeletal units by the processes of transformation. In varying degrees, transformative growth changes produce 'drift' or 'translocation' of the micro-skeletal units. For example, the primary growth of the medial pterygoid and masseter muscles, of the temporalis muscle and of the lateral pterygoid muscle causes a secondary transformative growth change in the mandibular angular, coronoid and condylar processes respectively. Such changes not only alter their form, but also the position of these contiguous micro-skeletal units relative to each other. However none of these processes is responsible for the motion of the totality of the mandibular micro-skeletal units (i.e. of the mandibular macro-skeletal unit) away from either the cranial base or from the maxillary skeletal units.

All microskeletal units are entirely dependent upon the morphogenetically primary demands of their functional periosteal matrices for changes in size and shape as well as for maintenance in being. It is established that there is no direct genetic determination of either the form or position of any skeletal unit. Morphological genetic activity is directed primarily to functional matrices and so only indirectly and secondarily to skeletal tissues (Grüneberg, 1963).

All orofacial micro-skeletal units, together with their periosteal matrices exist within a series of orofacial capsules (oral, nasal, pharyngeal), which surround and protect the functioning spaces of the oral, nasal and pharyngeal cavities. Our second type of functional matrix is the capsular; consisting of these functioning spaces. The neural, orbital and otic masses are other cephalic capsular matrices. Capsular matrices act indirectly on both the totality of all the embedded micro-skeletal units (i.e. on macro-skeletal units) and on their periosteal matrices. Capsular matrices do not act by the processes of resorption and deposition. Rather, as the volume of the functioning spaces increases, the surrounding capsule expands and the embedded macro-skeletal units are passively translated in space. Since periosteal matrices simultaneously are translated, they in turn alter their functional demands, thus producing synchronous transformations of their respective micro-skeletal units. The apparently simultaneous appearance of both translative and transformative growth changes plays a major role in creating the confusion now existing concerning the mechanisms of orofacial growth.

Experimentally and clinically it is possible to isolate these two aspects of growth. We can demonstrate conclusively that neither nasal septal nor mandibular condylar cartilages are primary sites of expansive orofacial growth. As the surfaces of micro-skeletal units respond secondarily by transformation to periosteal matrices, so sutural margins and cephalic cartilages respond transformatively in a secondary, compensatory, manner to translations produced by volumetric expansion of capsular matrices.

Complete removal of sutural tissues results only in sutural dehisence (i.e., by a local loss of secondary transformation). Similarly, removal of facial cartilages (or their congenital absence) does not interfere with orofacial translative growth. The normal translation of the upper jaw in arrhinencephalic and bilateral cleft palate infants, as well as those missing a septum, are clinical proof that the nasal cartilage is not a primary site of such translative growth. The normal growth of all non-condylar mandibular micro-skeletal units in patients with congenital absence of their condylar processes establishes the non-translative role of these cartilages. So true is this that young patients with either congenital or acquired ankylosis of the temporomandibular joint (unilateral or bilateral) are treated successfully by bilateral condylectomy (Moss and Rankow, 1968). We can study quantitatively the role of both translation and transformation (the role of periosteal and capsular matrices) in orofacial growth. Using multiple vital staining in the rat, Cleal *et al.* (1968) provide data on both the magnitude and direction of orofacial and neuro-cranial transformative bone growth. Comparing these data with our own, which measures the sum of both translation and transformation, we note the following results of analyzing adolescent vertical growth in the sagittal plane:

a) anterior nasal aperture height—transformation 35.4 per cent, translation 64.6 per cent;
b) height from middle of frontal bone to plane of hard palate—transformation 22.5 per cent, translation 77.5 per cent;
c) height from centre of parietal bone to lower border of basi-sphenoid, transformation, 32.4 per cent, translation 67.6 per cent.

Obviously the 'drifts' or 'translations' due to direct active transformation of micro-skeletal units in response to their growing periosteal matrices ranges from 25 to 33 per cent, while indirect passive translation of macro-skeletal units in response to the volumetric expansion of capsular matrices ranges from 66 to 75 per cent.

The effect of transformative and translative growth are not always additive. For example, Cleal *et al.* (1968), note that the cerebral surface of the basi-sphenoid transforms upwards while being translated downward. Further, the occipital squama transforms anteriorly while being translated posteriorly. Recently we demonstrated methods to quantitate both the magnitude and direction of the vectors of transformative and translative growth (Moss and Salentijn (1969a,b, 1970). Here we introduced two terms requiring clarification. Tracings of successive stages of either longitudinal or cross-sectional growth data film can be superimposed; correctly on the anterior cranial base and alternately and less meaningfully by any cephalometric technique. What is observed is interosseous growth, the sum of both active transformation and passive translation. It is possible now to distinguish between them. Re-registration

of mandibular tracings on the mental foramina has been shown to be as accurate as that obtained by Björk (1964, 1968) using metallic implants. Now we observe intraosseous growth, which is totally transformative. Superimposition of both sets of tracings permits the visual, biological and verbal subtraction of the transformation of intraosseous growth from the sum of both the transformation and translation of interosseous growth, leaving translation alone. Our published data indicate that about 66-75 per cent of normal vertical mandibular growth is due to the passive translation of the mandibular macro-skeletal unit as a secondary response to the primary volumetric expansion of the orofacial functioning spaces acting as a capsular matrix. The change in position of ramal micro-skeletal units, as expected, is due to transformation. A striking example of the dominance of capsular matrices in vertical mandibular growth is shown in Figure 1, taken from the data of Björk and Kuroda (1968).

At this time we present the first similar analogies of mid-facial skeletal growth, with longitudinal growth data published by Björk (1964; 1968), using metallic implants. It has become customary to consider the vertical lowering of the hard palate relative to the anterior cranial base as being either totally transformative in nature, or having a variable contribution from supposed expansive forces generated by interstitial growth within the nasal septal cartilages. Having disproved the latter previously (Moss and Greenberg, 1967; Moss and Bromberg, 1968; Moss and Simon, 1968), we turn now to the possible role of capsular matrices.

Graphic analysis of Björk's data for the mean growth of his sample population shows that on the average almost all the vertical growth of the hard palate is translative (Fig. 2). In any given case, this generalization is not true. The examples in Figures 3 and 4 make it clear, even as a first approximation, that passive translation accounts for a significant proportion of total vertical growth of the hard palate. Transformation is responsible for the posterior 'relocation'. Continuing work in our laboratory, to be published elsewhere, will deal with this topic more definitively.

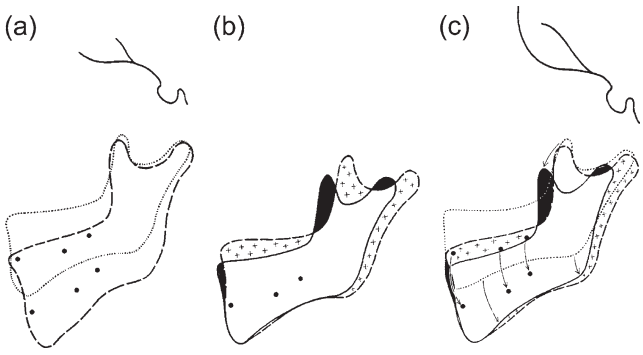



**Figure 1** The dominance of capsular matrices and of translative growth are shown in this case of 'congenital bilateral hypoplasia' of the mandibular condyles (after Björk and Kuroda, 1968). a) Interosseous growth is shown in the period from 11 years 5 months to 19 years 5 months. This is the sum of both translation and transformation. Registration is on the anterior cranial base. b) Intraosseous growth, totally transformative is shown, with resorption in black and deposition with crosses. c) The superimposition of (a) and (b) now shows that almost all of the vertical motion here is translative. The oro-pharyngeal functioning spaces, being unable to express their normal direction of growth, cause this deformation since the magnitude of the growth force of these capsular matrices is unaltered.

## Discussion

Professor Tulley congratulated Professor Moss on his paper: a great many of the things that had been said he personally had believed for some time. He hated to disagree with Professor Moss but nevertheless he intended to do so. Most people would agree with a great deal of what Professor Moss had said. They would not agree however that this was a new concept. The suggestions that were being put forward were at least 50 years old. He agreed that those people who are just transplanting bones are not studying the entire concept of growth but many people had been transplanting limb buds and not just bone structures. He felt that although 95 per cent of what Professor Moss had put forward was correct, it was the other 5 per cent that was dangerous. First of all he would question whether it was reasonable to use the mental foramen as a landmark on which to superimpose tracings of the mandible several years apart in growth. We all know that the functional matrix and the way in which nervous structures emerge make the position of the mental foramen entirely different at these two stages.

Professor Moss was not a clinician, most of his audience were clinicians, but he would agree, as a scientist, that it would be unwise to place too much emphasis on a single case of absence of the condyles.

There would be many people in the audience who have seen numerous cases of early condylectomy without the success that Professor Moss had claimed for the case that he had shown. He had said yesterday that we were doing children a disservice if the condyles were not removed early. Professor Tulley had had the opportunity to study cases over 15 years - twice as long as those shown in Professor Moss' paper - but he could not see quite the same end result and no doubt this would be supported by others in the audience with a great deal more experience than he had.

We all see a lot of pathological cases and he would like to ask whether excessive endocrine secretion in certain specific instances, such as acromegaly, act entirely on the functional matrix.

Another dangerous point that had been made, and here Dr Fränkel's paper is relevant, is the thought that we can stimulate both growth beyond its genetic potential. Professor Tulley felt that in his paper Professor Moss had given the ready-made answer to people who believed that this was a possibility and he would like to ask Professor Moss whether, in all honesty, he believed that it was possible to stimulate bone growth to this extent.

Professor Moss said that he was going to be very blunt. If everyone had known about these concepts for 50 years why

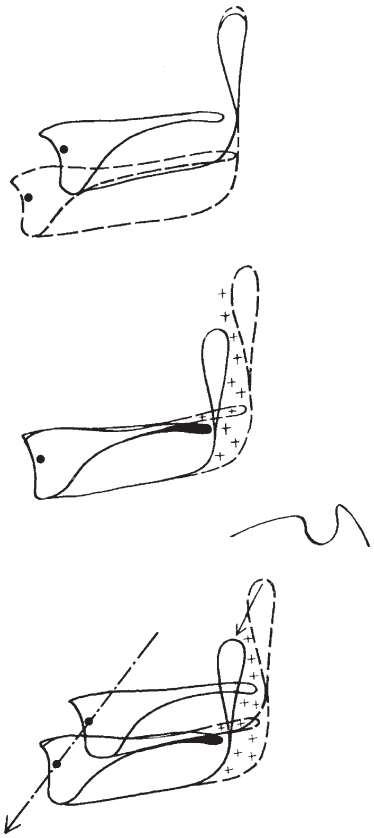


**Figure 2** The mean growth of the mid-face is shown in this data derived from that of Björk (1964) for a sample of 32 Danish boys. The registrations are as indicated in Figure (1). See text for details.

hadn't they done something about it? Why were their treatments and their concepts not any better? With respect to the President and to Professor Tulley, he did not think that the things that he was talking about in terms of functional matrix were the same as what they were talking about. The concepts that he was developing were possibly more sophisticated biologically than many speakers had realised. He was sorry that the name, Functional Matrix, seemed to have caught on and he was unhappy at the rather facile use that some people were making of the concepts that he was developing. The followers of Dr Fränkel claimed that their undoubtedly good manipulative technique was based on his beliefs and theories, but he doubted whether these workers were using either his terminology or concepts and he must disclaim any responsibility for their work on the basis of his own theories. As to the use of the mental foramen, Professor Tulley was wrong. He knew, as his audience all knew, that the shape and direction of the mental foramen alters with growth and development but the position of the original endosseous site of the foramen, as in any nutrient foramen in any bone, does not alter and this can be proved by metallic implant studies. He thought that he and Professor Tulley were talking about different things. He agreed that that the external position changes but the internal site of the foramen does not migrate at all and he was perfectly justified in using it. Furthermore having established that he could reproduce this tracing accurately he had been able to apply this to Dr Björk's magnificent implant study and had produced the same results. Now, condyles! Why hasn't everyone else been able to reproduce these results? The problem is one of surgical technique. The situation was comparable to the early days of cleft palate surgery when unfortunate operative techniques produced extensive scarring and the result on growth of the mid-face was more serious than was an unoperated cleft. There are many ways of taking condyles out and lots of reasons for doing so but the *sine qua non* of good results is the absence of post-operative scarring. You may observe cases for 15 years, or for 50, but it is the first three weeks that count in terms of results. He hoped that Professor Tulley did not consider him to be so poor a scientist as to rely on one case. He showed only one because his paper was limited to 30 minutes but he had an extensive series of cases that had been followed for quite a few years, both adults and children, and the results have been uniform. The technique in the United States is being utilised in a wide number of major surgical centres. The method was a 'no-scar technique'. The lateral pterygoid is cut and allowed to retract. All the condylar head is taken off, including the neck, and provision is made for no scarring, including, at times, application of a series of plastic films over the cut edges, but this is a no-scar technique with immediate motion – not fixation.

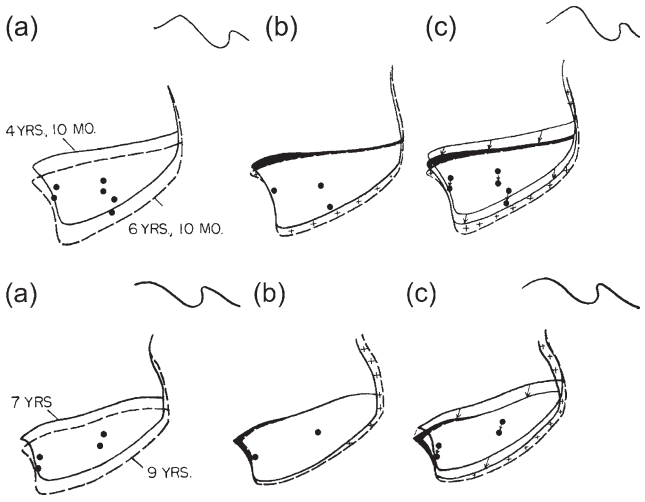
Now as to acromegaly, the clinical sign that most patients will demonstrate first is enlargement of the tongue. The question is asked from time to time, does growth hormone affect the cartilage *per se* or is it acting primarily on the soft tissues. Everything that he had discussed on functional matrix had made certain assumptions – namely that we have been dealing within that so-called 'normal'. The primary site of somatotrophic hormone action he believed was in the soft tissues but this was an interesting point.

Dr Logan said he heartily concurred with Professor Moss when he said that we are not in agreement on the meaning of Functional Matrix. He felt that Professor Moss was probably not informed about what we were thinking in Europe and he would be interested to learn what he thought about the Fränkel cases in the demonstrations later in the programme.

Professor Moss said that Dr Logan should not necessarily assume a state of intellectual provincialism in the speaker. On the contrary, he was very well informed about anatomical, anthropological, and orthodontic thought in the United Kingdom, as well as the Continent.

Concerning Professor Fränkel, his previous comments would serve in this instance also.

Professor Baume said that Professor Moss had stated that genes do not affect cartilage. Professor Baume quoted the work of Stockard on cross-breeding of dogs to establish which cartilages were genetically determined; by showing malformations he had indicated that there was some genetic




**Figures 3–4** The original data are those of Björk (1964, 1968) and are shown in each figure as (a). In both cases (b) shows the amount and direction of active transformation, while (c) shows the vectors of passive translation. The original figure did not, unfortunately, show the oral surface of the hard palate, so that all we can demonstrate are the changes of the alveolar borders and of the nasal surface. In both children translation in response to capsular matrices accounts for the majority of the totality of vertical growth.

control of bone growth through cartilages and that these cartilaginous centres were sometimes genetically lacking. He had previously discussed the chronology of fusion of cartilages in various animals and had referred to the rat which Evans had called a genetically dwarfed animal. Some rat cartilages never fuse and if you administer growth hormone they will start to grow again. Professor Moss had said that growth hormone acts primarily on the soft tissues but he would recall that growth hormone was once called chondrotropic hormone and it was believed that it acted initially on cartilage. There is experimental evidence in adult rats and dogs. Evans gave growth hormone to Dachshunds and they increased in length but not in height because there were no epiphyseal cartilages in the tibia because the Dachshund is an achondroplastic dwarf. This is further evidence that the growth hormone acts on cartilage. The theory of the functional matrix left a great deal unexplained. Professor Moss had not shown the profile of his patient but we know that ankylosis produces the bird faced profile. He had carried out histological sections systematically during growth in monkeys and this had shown that when the mandible grows it grows in width as well as in length. How can this occur? There is resorption at the attachment of the mesial pterygoid and apposition at the attachment of the masseter muscle. How could Professor Moss explain this? The temporal muscle is inserted into the anterior part of the ramus but this part of the mandible resorbs. The initial growth centre is the condylar cartilage and this leads and directs the growth in length and width. He would accept that this might be secondary but, in his view, it undoubtedly plays an important role in jaw growth.

Professor Moss said in reply that the photographs of the profile that Professor Baume wished to see had been published over 18 months previously in the *Angle Orthodontist.* In the short paper that he had just given he did not claim to have presented even a moderate synopsis of the method of functional cranial analysis and if his theories were to be fully understood it was necessary to read the previous relevant literature.

Professor Moss could not accept the work of Stockard on hybridisation in dogs as scientific data to be used to establish the role of cartilage in growth in humans. His own work on functional matrix is not a philosophy, it is the result of experimental anatomical investigation over a period of about 15 years. It began as, and still is, experimental morphology with conclusions derived on the basis of experimental data only. Professor Moss did not know originally whether what he was presenting in terms of functional analysis and functional matrix was good, bad or indifferent in the clinical treatment of the patient. It was never devised with the concept of having anything to do with clinical techniques. It was originally a craniological investigation. It has, however, proved valuable clinically. He could not allow his presentation at this meeting to be interpreted as an attempt to give the sum and substance of the method. He did believe that dentistry has passed from a restorative to a preventive phase and, in his school at least, from a purely technical profession to one with a truly scientific basis. He did not believe that orthodontists in any country are adequately trained in the biological sciences and this was as true in the United States as elsewhere. The scientific basis could only be achieved by individuals who with appropriate clinical background are then prepared to devote the major portion of their time as serious scientists and it was inevitable that such people would have very solidly based concepts that would be very disturbing to present clinically oriented beliefs. They would be rejuvenating and refreshing, and orthodontists would only benefit from these workers if they first got rid of the concept that everything that they had done in the past was right. Professor Moss said that he was something of an educator in dentistry and it was his policy to educate by irritation. To judge from the reaction in the discussion he had taught well today.

Dr Stockli said that Professor Moss had suggested that the mandible grows without a condyle and quoted as evidence the fact that the mandible continues to grow after resection of the condyle. This is not a new observation but the question really is whether it is the same mandible, the same size and in the same relation in space as it would have been without resection. No-one, as far as Dr. Stockli was aware, has ever suggested that we need the condylar cartilage for the growth of the alveolar process.

Professor Moss replied that in his series of bilateral condylectomies the now acondylar mandible did not assume ‘the same relation it would have had without resection’ since pre-operative position, obviously, would be abnormal.

However, in his series, the acondylar mandibles did alter both their form and position in a manner and to a degree homologous with those of normal patients. Finally, Dr. Moss agreed with Dr. Stockli that the condylar cartilage was not necessary for the growth of the alveolar process. Indeed, one of the basic points of functional cranial analysis was precisely the independence of such mandibular functional cranial components.

## Acknowledgement

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