Masticatory muscle influence on craniofacial growth

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The influence of the masticatory muscle function on craniofacial growth has been recorded in a series of animal experimental and clinical studies. The common characteristic of these investigations is that the elevator muscles of the mandible influence the transversal and the vertical dimensions of the face. The increased loading of the jaws due to masticatory muscle hyperfunction may lead to increased sutural growth and bone apposition, resulting in turn in an increased transversal growth of the maxilla and broader bone bases for the dental arches. Furthermore, an increase in the function of the masticatory muscles is associated with anterior growth rotation pattern of the mandible and with well-developed angular, coronoid, and condylar processes. 

Biomechanics; bone remodeling; cranial sutures; masticatory muscles; periosteum

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During the postnatal growth of bones, a continuous remodeling process takes place to maintain a form appropriate to their biomechanical function. The discussion about the functional adaptation of bone—that is, the remodeling response of bone to alterations in mechanical demands—was started already last century, and many scientists (Meyer, Culmann, Roux, Wolff, Koch) have contributed to developing the theory known today as Wolff's law (for a review, see Hall (1)).

Effects of load changes on long bones

Bone remodels to reach the shape that can best withstand the mechanical loads applied. Thus, prolonged application of a bending force on a long bone stimulates bone apposition on the surface that becomes more concave and bone resorption on the surface that becomes more convex (2). This remodeling process would lead to a bone configuration that reduces the deformation during physiologic functioning (3, 4).

An increase in the mechanical load on long bones was found to augment the thickness of the cortical bone in young pigs (5) and in adult rats (6). Besides, the trabecular bone mass was increased in adult rats as an effect of overload (7). The increase in bone mass was found to have a linear relationship with strain magnitude (8). The above-mentioned examples were results of an intermittent application of strain which, according to Lanyon & Rubin (9), had a greater remodeling effect than static application. Continuous application of load in animals induced periosteal apposition of bone with reduced mineralization and altered bone structure (10). Mechanical stress also influences the mineral structure of bone. Stress in the long bones induced hydroxyapatite crystals to orientate in a vertical direction in the mid-shaft of femora. This phenomenon has a peak around 2–3 years of age, when a child rises to the standing position and starts walking, thus dramatically increasing the load of weight-bearing bones (11).

The reduction or absence of mechanical stress on usually load-bearing bone induced a general remodeling and reduction of bone mass resembling a disuse osteoporosis (12). Experimental data from rats confirmed these observations, showing a halt in periosteal bone formation and an accelerated bone marrow expansion (13) with a poor appearance of the trabecular architecture (14). The weightless state caused bone formation to stop in weight-bearing limbs in growing animals (15). Furthermore, loss of bone mass was observed, being greater in trabecular than in cortical bone (16, 17). The non-weight-bearing bones of rats did not show any alteration in the periosteal apposition rate after a weightless state (18), although more recent studies affirm that bone matrix maturation nevertheless was affected in all skeletal structures (19).

Effects of muscular function on long bone growth and structure

The close relation between the muscle and the bone is due to the fact that the muscle influences the growth of the latter both as a tissue affecting the vascular supply of bone and as a force element. The vascular influence is either direct, because muscular arteries provide the main blood supply, or indirect, because of volume and pressure changes during muscle contraction and movement (20). Muscles are inserted into the bone by means of their aponeuroses through the periosteum or tendons, and through them they exert tensile forces on the bone (21, 22). Thus, the effect of muscle contraction is both
local—that is, exerted in the area of the muscle insertion—and general, changing the loading level of the bone which acts as a lever or beam.

In young pigs a long period of physical training induced morphologic changes in the femur, such as the increase in thickness of cortical bone by 17%, whereas no changes in the quality of the bone and its mechanical properties were observed (23). However, it was shown that prolonged moderate exercise in mature female rats can increase calcium deposition in both axial and appendicular bones (24). Furthermore, it was demonstrated that physical exercise can increase bone mass or reduce bone loss related to age in humans (25). Conversely, general bone atrophy was noted in cast-immobilized limbs of human subjects, characterized by a thinning of cortical bone (26).

Experimental studies in growing rats showed an alteration in the tibia cross section and elimination of its longitudinal curve caused by cutting the nerve supply to surrounding muscles (sciatic neurectomy). The total mineral density also appeared to be reduced. However, no changes were found in the length of the bone (27). Similar results were caused by suspension of hindlimbs and fibula was reduced, while the length of these bones was also affected (28).

Masticatory muscle function and craniofacial growth

Animal experiments

In one of the animal experimental models used to test the influence of the masticatory muscles on craniofacial growth the loads applied on the skull during mastication were reduced by feeding the animals a soft diet. This model was used mainly in small animals such as the rat (29–36), but its significance was also proved to some extent in primates (37, 38). The advantage of this model was the possibility of observing the effects of an altered function in an intact masticatory system. The decreased functional demand on the animals fed a soft diet caused structural changes in the masticatory muscles and alteration in their contractive capacity, as indicated by changes in the muscle fiber types and the smaller size of the fibers (39). These changes could explain the low biting forces measured in the animals with reduced function (40). The low biting and chewing forces applied on the bones of the craniofacial region may have been the causal factor for induced changes in bone remodeling and the sutural growth. Thus, studying the factors that locally control the osteogenesis of the sutures in monkeys, Behrents et al. (41) proposed that the bite forces could provide tensile strain sufficient to cause a separation of the sagittal suture of the skull, as previously suggested by Herring (42). This could be the underlying mechanism for the observations in histologic sections that the width of the sutures in the anterior part of the snout was reduced, and that several synostoses appeared in the internasal suture in rats with less tension in the lateral part of the nasal bones due to a reduced masticatory and biting function (43). A morphometric study was able to verify a narrower sutural space and a more parallel orientation of bony surfaces of the facial sutures in the rats after a reduced functional demand (44). The changes in the functional demands of the masticatory muscles, induced by feeding the animals a soft diet, also influenced the remodeling of the bone structures of the upper viscerocranium and the mandible. Reduced function caused changes in the size and dimensions of the alveolar process, such as the width, height, and thickness of alveolar bone, resulting in a smaller cross-sectional area of the bones. Thus, changes in bone apposition were observed not only in the vicinity of the insertion of the elevator muscles but also in areas where increased bone apposition reinforces the bone so that it is able to withstand higher bending forces in animals fed a hard diet (36, 45).

The mandible is composed of different functional and morphogenetic units, such as the mandibular body, the alveolar complex housing molars and incisor, the gonial region, the coronoid process, and the condyle (46–48). The response of the different functional units of the mandible to changes in the functional strain was evaluated by measuring the bone mass on roentgenograms in relation to an aluminum step wedge, using a computer-aided image-analysis system. The total bone mineral content was calculated as ‘aluminum equivalent’ mass. Reduced muscle function was found to decrease the bone mineral mass in all functional units of the mandible, possibly in accordance with the strain distribution of the masticatory forces—that is, in the alveolar bone of the molars and the incisors—in the angular process, which is the area of the insertion of the masseter and pterygoideus medialis, and at the bony bulk ascending to condylar processes of the mandible. These changes in the areas with a high bone apposition rate seem to be the result of decreased total bone mass, whereas in areas with a low bone apposition rate they seem to be an effect of decreased bone density (49).

These alterations in bone remodeling and sutural growth may be the factor underlying the radiographically observed morphologic changes—that is, the more upward rotation of the upper viscerocranium and the reduced growth of the angle of the mandible (34, 50) and the reduced transversal palatal width observed in dry skulls of animals fed a soft diet (29, 31). The important effect of the muscle function on the form, growth, and structure of the mandible was also tested in rats with hypocalcemia. Changes in the mechanical strain environment of the mandible, caused by feeding the animals a soft diet, thereby reducing their vigorous mastication, were a powerful deterrent of remodeling behavior at certain sites, even when a metabolic bone disturbance was present (34).

The condyle is considered to be a mandibular growth
zone directly affected by the functional alterations. Several investigations have concerned the effect on the temporomandibular joint in growing animals, after altering the function by changing the consistency of the diet (32, 51, 52). Significant differences were found in the condylar length between groups, the soft-diet group having a smaller condyle. The morphometric analysis of the thickness of the condylar cartilage on histologic sections at the sagittal level showed significant differences between the two groups, being thicker at the posterior part and thinner at the anterior part of the condyle in the soft-diet group (S. Kiliaridis et al. Unpublished observations). The results of this study indicate that a low masticatory function induces a decreased condylar growth and changes in the thickness of the cartilage. This might be related to the alteration in the stress distribution in the temporomandibular joint area, due to the absence of big loads exerted on the condylar tissues.

Human studies

The results of the animal experimental studies illustrated the influence of the masticatory muscles on the basic mechanisms of craniofacial growth—that is, on bone remodeling and condylar and sutural growth. However, direct extrapolations to humans are not valid. The importance of the masticatory muscle function has been observed in anthropologic studies, in which a low frequency of malocclusions was found in populations with primitive living conditions (53-56). The frequency of malocclusions seems to have increased since these populations came under the influence of industrialized civilization (56, 57). This was thought to be either a result of the premature loss of deciduous teeth due to caries or an effect of the hypofunction of the masticatory muscles due to alteration in the consistency of the diet, reducing the forces applied to the jaws. Similar findings were observed in studies of medieval skulls, in which a lower frequency and a lower severity of malocclusions than in contemporary individuals were found (58-60). Characteristic of the morphology of the skulls was a small intermaxillary angle, a small gonial angle, and broad jaws (59, 61-65). The excessive attrition observed on the teeth of the medieval skulls indicated an extensive function of the masticatory muscles.

However, these findings are not limited to the primitive populations and medieval skulls. Among contemporary individuals there are subjects showing advanced stages of dental wear and an increased level of masticatory muscle activity, mainly due to bruxism. These individuals show a low incidence of malocclusions and are characterized (66-68) by a short lower face, a small intermaxillary angle, and a small gonial angle (Fig. 1) as compared with adult norms. Similar results were
found in a group of wrestlers, who, in general, are characterized by a well-developed muscular system and have, furthermore, undergone heavy resistance training. They had wide and well-developed dental arches with a low frequency of malocclusions. Their craniofacial morphology was characterized by a short lower face, small intermaxillary angle, and decreased gonial angle of the mandible, possibly a result of increased bone strain on the dentofacial structures during their growth period (69). This has been caused either by the forces exerted directly during the sport or by the occlusal forces generated by their well-trained masticatory muscles.

The possibility of training the masticatory muscles was tested in a group of young adults by introducing them to systematic chewing exercises using a hard chewing gum. After 1 month's training their maximal bite force was increased (70). The clinical utilization of this mechanism in the treatment of the anterior open bite had already had a positive effect (71, 72). In contrast, the craniofacial morphology had deteriorated in a group of patients with myotonic dystrophy (Fig. 2). The patients had a vertical growth pattern and a high incidence of malocclusions, such as distal occlusion, anterior open bite, lateral crossbite, and crowding (73). This group was characterized by a low bite force level and reduced masticatory muscle electromyographic activity (74).

Furthermore, in adults with jaw deformaties, the thickness of the masseter muscle was less than in the control group, as measured by ultrasonography. In the deformity group, thin masseter muscles were related to individuals with a long anterior face (75). Weak masticatory muscles could be one of the reasons for the development of jaw deformity. However, the possibility cannot be ruled out that the weakness of the masseter muscle was an effect of its unfavorable functional condition due to the existing jaw deformity. Postsurgical investigations may elucidate this question and define the possible relation between the muscular factor and the reported postsurgical relapse observed in some patients after orthognathic surgery.

One of the methods of investigating the possible relation of masticatory muscles function to facial morphology has been to relate the functional capacity of the subjects' masticatory apparatus to their facial morphology. Different methods have been used in adults to evaluate the relation between facial characteristics and their functional capacity, such as recording the maximal bite force (76–78), computer tomography (79, 80), magnetic resonance imaging (81), and ultrasonography (82, 83). A general consensus in all these studies is that subjects with strong or thick mandibular elevator muscles have wider transversal head dimensions. Tendencies towards parallelism between jaw bases and between occlusal and mandibular lines, a small gonial angle, small lower facial height, and a rectangular shape of the face were also pointed out in some of the studies.
However, the finding of Proffit & Fields (84), that no statistical differences could be detected between the bite force of long-face children when compared with normal ones, caused some confusion, since the bite force of adults with a long face was comparable to that of the long-face children and much lower than that of normal-face adults (78). Of course, a closer view of the results of these studies on long-face children and adults may show that a considerable variation (SD) exists in the normal group when compared with the long-face children or adults. This was also the case when the masseter cross-sectional area was measured in normal and long-face adult groups (85) (Table 1).

A possible explanation is that the long-face groups were more homogeneous, with thin muscles and low bite force, than normal-face individuals, who varied widely both in the thickness of the muscles and the bite force level. This means that individuals with weak masticatory muscles could belong to either the long-face or the normal group, whereas scarcely any individual with strong masticatory muscles could be found in the long-face group.

The findings of Ingervall & Helkimo (77), that individuals with strong masticatory muscles had a homogeneous facial morphology, in contrast to the individuals with weak masticatory muscles, who showed great inter-individual variation, lends support to this explanation. Thus, it may be concluded that the masticatory muscles are able to influence the craniofacial growth of man provided that the tension they apply to the facial bone structures is above a certain strain level, reaching what Frost (86) calls ‘the mild overload window’. The principle that is valid for long bones, that ‘modeling drifts can adapt bone to overloads by changing bone architecture and adding bone, but apparently cannot adapt it to underloads or disuse . . .’ (86), could be also applied to the craniofacial complex. Thus, the epigenetic influence of the masticatory muscles, in their role as force-generating elements, on craniofacial growth, may be valid in the presence of the increased muscle activity but not necessarily when this activity is reduced.

### Table 1
Descriptive statistics of the cross-sectional area of masseter and maximal bite forces (MBF) in normal and long-face individuals, as supplied by the literature (SD = standard deviation; \( n \) = number of individuals; M = males; F = females)

<table>
<thead>
<tr>
<th></th>
<th>Normal Mean</th>
<th>SD</th>
<th>n</th>
<th>Long-face Mean</th>
<th>SD</th>
<th>n</th>
</tr>
</thead>
<tbody>
<tr>
<td>From Van Sprosen et al. (85)</td>
<td></td>
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</tr>
<tr>
<td>Right masseter (cm(^2))</td>
<td>4.64</td>
<td>0.83</td>
<td>35 (M)</td>
<td>3.64</td>
<td>0.43</td>
<td>13 (M)</td>
</tr>
<tr>
<td>Left masseter (cm(^2))</td>
<td>4.64</td>
<td>1.03</td>
<td>35 (M)</td>
<td>3.41</td>
<td>0.50</td>
<td>13 (M)</td>
</tr>
<tr>
<td>From Proffit et al. (78)</td>
<td></td>
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<tr>
<td>MBF adults, 2.5-mm opening (kg)</td>
<td>31.00</td>
<td>20.00</td>
<td>21 (M, F)</td>
<td>11.20</td>
<td>7.90</td>
<td>19 (M, F)</td>
</tr>
<tr>
<td>MBF children, 2.5-mm opening (kg)</td>
<td>17.43</td>
<td>19.16</td>
<td>18 (M, F)</td>
<td>10.05</td>
<td>5.94</td>
<td>12 (M, F)</td>
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