Functional Cranial Analysis of the Mandibular Angular Cartilage in the Rat

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INTRODUCTION

Classical craniology emphasizes the role of cranial cartilaginous areas as primary growth sites. Specifically, they view such regions as the spheno-occipital synchondrosis, the cartilaginous nasal septum and the mandibular condylar cartilage as primary sites of interstitial (and sometimes perichondral) growth whose volumetric and linear expansion causes the subsequent separative growth of adjacent bones.[1] There is a point of view diametrically opposed to such concepts derived from the method of Functional Cranial Analysis.[2-9] This newer theory suggests that the growth of all skeletal tissues, osseous as well as cartilaginous, is secondary and compensatory to the primary growth of related soft tissue matrices. It is felt that the growth of the cranial cartilages, without exception, is a secondary event following in a mechanically obligatory manner the primary growth of related soft tissue (functional) matrices.

The theoretical basis for this statement is as follows. The head consists operationally of a series of relatively independent *functional cranial components*. Each component consists of: a) all of those soft tissues (muscles, glands, nerves, blood vessels, etc.) which carry out a given function (termed the functional matrix); and of b) the bone and cartilage (termed the skeletal unit) whose function it is to protect and support a specific functional matrix. Further, this analytic method has shown repeatedly that the osteological elements of formal anatomy have no biological reality, but rather that a given "bone" may well be formed by a number of contiguous microskeletal units whose size, shape, position in space and, indeed, maintenance in being are relatively independent of each other. This has been demonstrated specifically in the case of the mammalian mandible, both ontogenetically and phylogenetically.[5]

It is implicit in these statements that the growth of the mammalian mandible is: a) not produced by the primary expansion of its intrinsic condylar cartilages; and b) that the removal of the mandibular condylar cartilages (skeletal units) of immature animals will not affect the dimensional growth or spatial relocation of the remaining non-condylar mandibular skeletal units whose functional matrices are intact. This has been demonstrated to be true for the mandibular condylar cartilages of both rodents and man. While seemingly admitting that the rodent condylar cartilage is not a primary growth site responsible for the spatial repositioning of the growing mandible nor for the linear growth of this "bone," Manson[10] feels that the angular cartilage of the rodent mandible is, in some way, "much more likely to be important to the longitudinal growth of the mandible than is the condylar cartilage." Presumably he feels this because the angle of the rat mandible is an essentially horizontal process, parallel to the long axis of the mandibular corpus. Since the role of rodent angular cartilage in mandibular growth has never been experimentally studied, to the best

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of my knowledge, the present work was undertaken in conjunction with other studies of the growth of the human angular process[8] and of the growth of the rat mandible (Moss and Simon, unpublished).

In this paper data are presented demonstrating that the experimental extirpation of the angular cartilage of the neonatal rat results *only* in the loss of the angular process, while the growth of all other mandibular skeletal units remains normal, as does the spatial relocation of the growing lower facial skeletal complex.

Materials and Methods

Observational The angular process of the rat mandible was obtained at the following ages: 1, 2, 3, 5, 8, 14, 18, 23, 29 and 34 days. They were fixed in neutral formalin, paraffin embedded, serially stationed in the sagittal plane and stained in haematoxylin and eosin. Selected angular processes were also sectioned in the coronal plane. Mandibles of similarly aged rats were obtained by dissection of boiled heads, bleached in 3% hydrogen peroxide, and dried for further study. All were x-rayed on dental intraoral films, magnified to a constant degree, and tracings made on acetate-matte paper. Identical treatment was given to the mandibles of operated rats and the experimental and unoperated sides so compared.

Extirpative techniques Ten two-day-old rats had their right angular processes removed either by electric cautery or by excision of the process with an iridectomy scissors. In either case the skin was incised, the masseter and medial pterygoid muscle attachments to this process carefully reflected prior to surgery. No muscle tissue was removed and the freed ends were allowed to fall back into place postoperatively. Closure was affected with 4% celloidin. Sacrifice occurred on the 17th and 34th day of life.

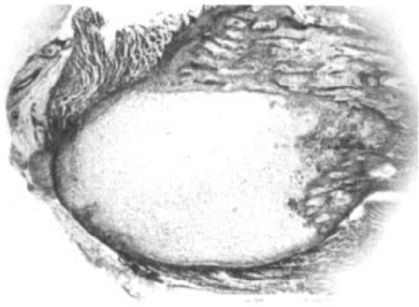

Fig. 1 The neonatal rat angular process is composed almost entirely of a mass of secondary cartilage.

Results

Histology of the Angular Cartilage The neonatal angular process essentially is a mass of secondary cartilage (Fig. 1). Bhaskar[11] provides prenatal data. The tissue is remarkably uniform in structure having large, closely packed chondrocytes with well-marked lacunar walls and very sparse intercellular substance. At its most posterior end, an active perichondrium is found with a sizable layer of small cells, presumably newly formed (Fig. 2). At the anterior end of this tissue mass which extends forward to the developing molar tooth buds at

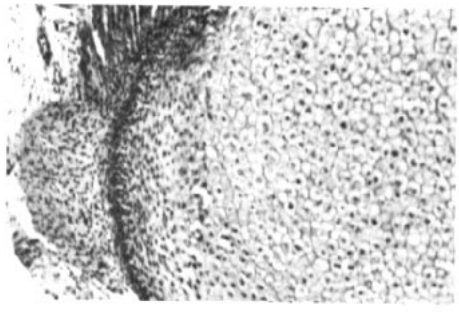

Fig. 2 A higher power view of Fig. 1 showing an active layer of chondroblastic proliferation at the posterior border of the process. Note that no columns of hypertrophic chondroblasts are seen, in distinction to the appearance of an epiphyseal growth plate in a long bone.

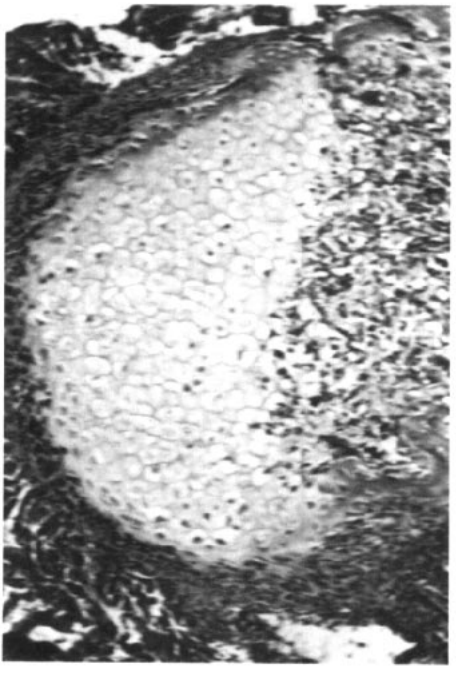

Fig. 3 The marked diminution in relative size of the angular cartilage by the fifth postnatal day is shown.

Fig. 4 Vascular invasion, from the posterior border, is evident by the 14 day.

this age, active vascular erosion occurs. There is no evidence of any cartilaginous organization resembling an epiphyseal growth plate of a long bone. The outer surfaces of this cartilage are covered by a thin lamella of bone to which it blends insensibly on its inner surface. By the fifth day active vascular erosion has reduced greatly the anteroposterior dimension of the cartilage. The relative size of the now reduced angular cartilage is maintained at the eighth day (Fig. 3). Here the posterior zone of perichondral chondrogenesis is much thinner, while vascular erosion anteriorly occurs without evidence of any chondrocytic hypertrophy or provisional calcification. At day 14 vascular penetration from the posterior region is noted, accompanied by apparent metaplasia of neighboring secondary cartilage cells into osteocytes (Fig. 4). The cartilage is still present sparsely at day 29 (Fig. 5) and almost gone at day 34.

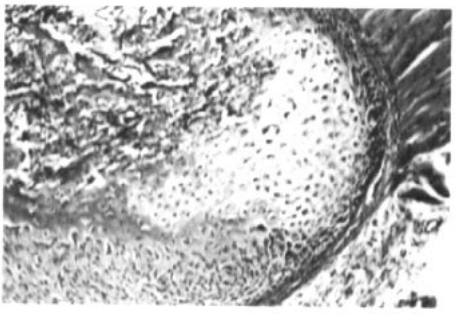

Fig. 5 At day 29, the cartilage is rapidly disappearing. Note that in this, as in the preceding photographs, cell columns do not appear, nor does a zone of provisional calcification. The picture is entirely one of vascular erosion.

Effect of Extirpation All mandibles which had one angular process removed grew normally. That is *both* sides of the mandible were virtually identical save that on the operated side that process was missing. The most effective way of demonstrating this is to compare the operated and unoperated hemimandi-

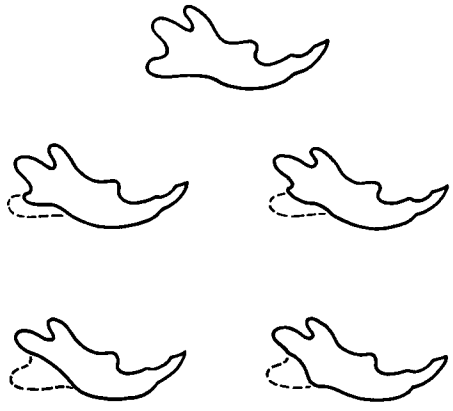

Fig. 6 Five 34-day-old rat mandibles are depicted. The tracings were made, at identical magnifications, from x-ray plates. The control figure is shown at top. The middle row shows two cases in which partial reattachment of both the masseter and medial pterygoid muscles occurred with a resulting partial reformation of a type of angular process. In the bottom row we see two mandibular halves in which such reattachment did not occur.

bles of the same rat (Fig. 6). It is of interest to note those cases in which a rudimentary angular process seems to be present. In these cases, dissection at sacrifice showed a partial reattachment of both the masseter and medial pterygoid muscles, while in others without such "reconstructed" processes no such muscular reattachment was noted. Comparison of the several dimensions of both hemimandibles with control data, derived from a study to be published elsewhere (Moss and Simon, unpublished), showed that the absolute growth of mandibular complex of operated rats was normal, as was the relative growth of the several contiguous mandibular skeletal units.

Discussion

Previous work has shown that the mammalian mandible consists of a number of contiguous, yet relatively independent, functional cranial components. The size, shape, spatial position, and maintenance in being of each single skeletal unit is a secondary response to the demands of its related skeletal unit. Mandibular growth has two aspects: the first is the dimensional change of individual skeletal units in response to the direct action of periosteal matrices; the second is the movement through space of the entire mandibular complex in response to the indirect action of capsular matrices. Concisely, the mandible, i.e., the sum of individual and contiguous mandibular microskeletal units, moves in space during growth because the orofacial capsule within which the mandible is embedded expands in response to the primary morphogenetic expansion of the enclosed and protected orofacial functioning space.[6] Condylar cartilage growth is compensatory, and secondary, to such capsular expansion. Usually, but *not* necessarily, the several mandibular periosteal matrices grow simultaneously. As a result of the direct effect of their increased functional demands, the several skeletal units respond in a secondary manner by increasing their dimensions. Accordingly we expect the alveolar and angular processes, for example, to "grow" as teeth erupt and as the periosteal functional matrix of the angular process grows. The matrix of the angular process consists of the masseter and medial pterygoid muscles. The work of Avis,[12] in particular, is cited to show that following the removal of either of these muscles the size of the angular process decreases approximately in half, while following removal of both muscular matrices the entire angular process disappears. These results have significance in comparison with our data. In those rats in which the angular functional matrices did not reform anything resembling a normal anatomical relationship, the angular process was completely gone, while in those animals where these muscles did so reposition themselves, a rudimentary reformation of the angular process oc-

curred. We interpret these data as indicating that in the latter cases the functioning matrices were capable of calling forth at least partially a neoformation of the process.

A semantic problem is associated with the term growth. The literature makes clear the need for a precise differentiation between the spatial relocations of a "bone" as a whole (in the sense of formal osteology) and the dimensional increases of its constituent skeletal units. It is true that the dimensional and volumetric growth of the angular process depends upon the interaction between a specific functional matrix and a specific skeletal unit. But it is also true that neither the spatial relocation nor the dimensional changes of any other mandibular skeletal unit is *in any way* dependent upon the angular functional cranial component or indeed upon its presence in being. Regardless of its histological composition, the angular process is a skeletal unit response only to its own periosteal matrix demands. In other words, the dimensional growth of the angular process is a secondary and compensatory osseous tissue response to the demands of its specific periosteal functional matrix.

The growth change, in length, of the corpus of the rodent mandible, on the other hand, is a response to the functional demands of other periosteal matrices (teeth, neurovascular bundles, muscle attachments, etc.). Quite apart from all of these direct skeletal unit responses to their periosteal matrices, there is the additional passive response of all contiguous mandibular cranial components to the translative growth of the orofacial capsule; a response to the volumetric expansion of the oropharyngeal functioning spaces.[6]

Since these data support the thesis of compensatory growth by cranial cartilages, it is well to inquire into the significance of the presence of such cartilage in the neonatal and juvenile angular process in the rat. The tissue in question is classified as secondary cartilage. This cartilage type arises independently of the primary cartilage of either the chondrocranium or the primary cartilaginous postcranial or appendicular skeleton. A review of this tissue, and of its subtypes, has been published previously.[13] Secondary cartilage has been noted in many cranial areas, usually considered to be intramembranous in origin. Two recent reports note its presence in the palate of the fetal and juvenile cat, and in the temporal squama of the bat.[14,15,16] Accordingly, the presence of secondary cartilage at the angular process of the rat is not unusual and its presence does not signify anything in terms of spatial growth processes. Without citation in depth, it seems reasonable to assume that the formation of secondary cartilage is related physiologically to local vascular sparsity. However, the consequent relative anoxia does not seem to be the causal factor leading to the formation of secondary cartilage tissues in intramembranous bones, but rather the lowered oxygen partial pressure may be the process by which a biochemically desirable result is obtained. De Beer[17] has summarized this aspect in suggesting that this tissue type "appears to be an adaptation for resistance to precocious strains and stresses on the part of rudiments of membrane-bones." I interpret this quotation as but another way of stating the basic premise of Functional Cranial Analysis, i.e., that the skeletal units respond secondarily to the morphogenetically primary demands of their related functional matrices. The differential processes by which periosteal (muscular) matrices call forth either cartilage or osseous tissue (both of which are encompassed in the term skeletal unit) remain unknown.

SUMMARY

The role of the cartilaginous angular process in the growth of the rat mandible is studied. This secondary cartilaginous tissue occupies virtually the entire extent of the process at birth and rapidly diminishes in extent by vascular erosion. No epiphyseal-plate apparatus is present and the tissue is replenished by perichondral chondrogenesis. Surgical removal of the neonatal angular process results in a mandible whose configuration and dimensions are normal, save for the absence of an angular process. It is concluded that the angular cartilage plays no primary role in the growth of the mandible as a whole, but rather that the form, position, and maintenance in being of the angular process is a secondary response to the primary morphogenetic demands of its specifically related muscles. These data are discussed in terms of the method of Functional Cranial Analysis which they support and extend.

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