Postnatal development of the human temporomandibular joint

I. A histological study

BIRGIT THILANDER, GUNNAR E. CARLSSON & BENGT INGERVALL

Departments of Orthodontics and Stomatognathic Physiology, Odontological Faculty, University of Gothenburg, Sweden

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Temporomandibular joints from 61 humans, aged 2 days to 27 years, were examined histologically. Four layers of the condyle were studied in detail. The outermost layer was richly vascularised in new-borns but by 3 years of age it had become avascular and contained few cells. In neonates the cartilage layer constituted a large part of the condyle but soon decreased in thickness and by 5—6 years of age it constituted only a thin zone of the top of the condyle. In the proliferative zone, mitoses occurred up to 13—15 years of age. This zone then decreased in thickness; the number of cells decreased, while the amount of intercellular substance increased. At birth, the temporal component was flat and was lined by vascularised connective tissue which became richer in collagen with increasing age. The cartilage layer was lacking in the fossa but was present on the tuberculum. A proliferative zone in this cartilage could be seen up to the age of 17—18 years and cartilage having only few cells was found in adults.

Remodelling processes were seen in all components of the joints. The significance of the remodelling seen in the fossa and on the mandibular neck is discussed with relation to condylar and periosteal growth of the mandible.

Key-words: Anatomy; growth; temporomandibular joint

Birgit Thilander, Department of Orthodontics, Odontological Faculty, University of Gothenburg, Fack, S-400 33 Göteborg, Sweden

The anatomy and development of the temporomandibular joint has received much attention in studies of facial growth. Several investigations of the prenatal as well as postnatal development in various experimental animals are available (*Öberg*, 1964; *Symons*, 1965; *Blackwood*, 1966; *Duterloo*, 1967; *Hall*, 1968; *Kanouse*, *Ramfjord & Nasjleti*, 1969; *Bremers*, 1973; *Durkin*, *Heeley & Irving*, 1973). The embryology and prenatal development of the human temporomandibular joint has also been the subject of several investigations (*Symons*, 1952; *Macalister*, 1955; *Baume*, 1962, 1970; *Levy*, 1964; *Yuodelis*, 1966). However, apart from descriptions in textbooks by *Moffet* (1962) and *Öberg* (1973) no comprehensive systematic investigation of postnatal development was available at the start of the present study. After that a comprehensive paper has been published (*Wright & Moffett*, 1974).

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Opinions differ as to the role played by the temporomandibular joint in the growth of the mandible and of the orofacial system. According to a survey by *Durkin et al.* (1973), at least two schools can be distinguished, *viz:* the *classicists* and the so-called *modernists*. According to the former, the condylar cartilage is identical in structure and function with the growth plate of the long bone and is a major primary growth center of the mandible. The latter claim that the main function of the condylar cartilage is adaptive remodelling to maintain the integrity of the joint when changes in the relationships of the surrounding structures occur.

The present paper is a survey of the development of the human temporomandibular joint from birth to adulthood, based on histological sections made from necropsy specimens.

MATERIAL AND METHODS

The material consisted of specimens from 61 subjects (36 males and 25 females), aged 2 days to 26 years 10 months. The age distribution is given in Table I.

No specimens were accepted from individuals who had died from diseases known to be capable of influencing growth. Most of the deaths were accidental.

The specimens were removed *en bloc*. They comprised the right temporomandibular joint, including the fossa, tuberculum, condyle and mandibular neck, capsule and disk, as well as contiguous soft tissue. The specimens were fixed in neutral formalin, decalcified in a mixture of 50 % formic acid and 15 % sodium formiate, and embedded in paraffin. One half of the material was serially sectioned at 12 μ in the sagittal plane and the other half in the transverse plane; consecutive sets of seven sections were stained with haemalun-eosin (Mayer), resorcin fuchsin (Weigert), picrofuchsin (van Gieson), azan (Heidenhain), htx-eosin (Bock), toluidine-blue and with PAS (Schiff reaction).

Table I. *Age and sex distribution of subjects from whom tissue samples were obtained*

Age (Years, months)	Male	Female
0.0— 4.11	9	8
5.0— 9.11	6	2
10.0—14.11	4	4
15.0—19.11	10	4
20.0—26.10	7	7
Total	36	25

RESULTS

The condyle varied in appearance from one part of the condyle to another and its histo-morphological picture varied from birth to adulthood (Fig. 1 a—f). Four layers could, however, usually be seen (Fig. 1 b).

1) A fibrous connective tissue layer, which constituted the articular surface (surface articular zone).
2) A highly cellular intermediate layer containing proliferating cells, which in a deeper layer, were found to be in a transitional stage between undifferentiated and cartilage cells (transitional or proliferative zone).
3) A cartilage layer with hypertrophic cartilage cells (hypertrophic zone) and a deeper layer of mineralized cartilage.
4) A zone with subchondral bone formation (bone formative zone).

In neonates, the fibrous connective tissue layer (surface articular zone) was well vascularised and the vessels continued down through the different cartilage

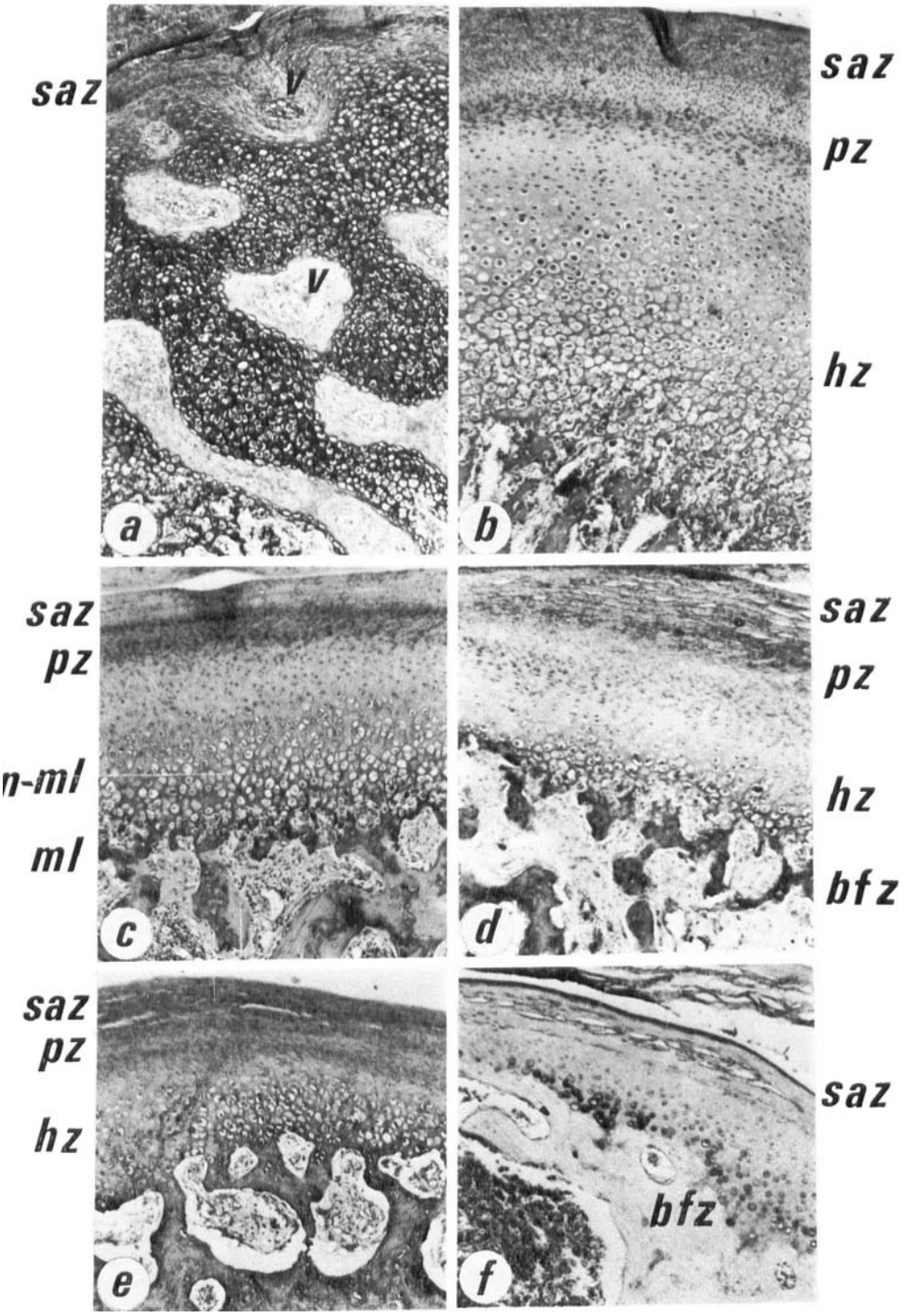


Fig. 1. The condyler layers at various ages from the central and upper part of the condyle (sagittal plane) showing the decrease in thickness of the proliferative and hypertrophic zones of the cartilage with increasing age.
Magnification: × 40
a. newborn ♂ (Bock) with richly vascularised cartilage (v)
b. 6 months ♂ (Bock)
c. 5 y. 8 m. ♂ (Azan)
d. 10 y. 6 m. ♂ (Bock)
e. 15 y. 3 m. ♂ (Bock)
f. 19 y. 7 m. ♂ (Toluidin)
saz (surface articular zone)
pz (proliferative zone)
hz (hypertrophic zone) { n—ml (non-mineralized layer) / ml (mineralized layer) }
bfz (bone formative zone)

zones (Fig. 1 a). The vascularity decreased soon after birth and by 6 years of age this zone was avascular, less cellular, while the collagen fiber bundles became successively coarser and denser (Fig. 1 c—e).

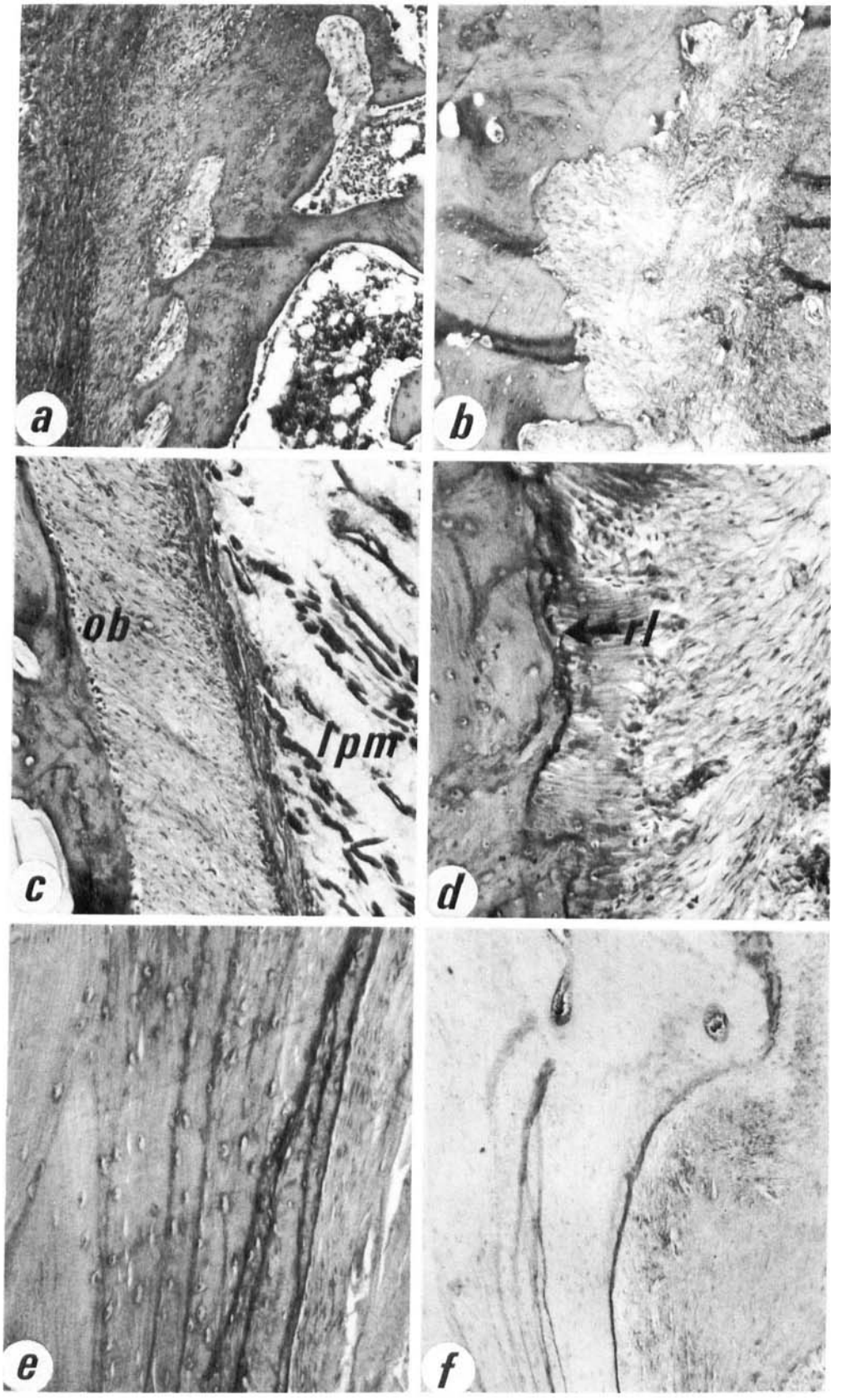
In neonates, the cartilage constituted a large part of the head of the condyle and was, as mentioned, highly vascular (Fig. 1 a). As vascularity decreased, the cartilage decreased in thickness, and was at 5—6 years of age (Fig. 1 c) only half as broad as at 6 months (Fig. 1 b) and continued to diminish in thickness progressively thereafter (Fig. 1 d—e). The proliferative zone was relatively narrow even in the youngest individuals, but thickest at the top of the condyle. It could be divided into an upper and a lower layer according to its cellular organization. The upper part was wider and contained many small cells in mitotic division and only little intercellular substance. In the lower layer the cells were somewhat larger, round, and were in a transitional stage between undifferentiated and cartilage cells. The histomorphological picture was largely the same up to 13—15 years of age, *i.e.* during the major part of the growth period (Fig. 1 e). Later, the zone decreased in thickness, while the cells decreased in number and the intercellular substance increased in amount. The top of the condyle showed this change last.

In contrast to the epiphyses of the long bones, the chondrocytes in the hypertrophic zone were not arranged in columns, but irregularly. During the first year of life this zone was quite broad (Fig. 1 b) but later gradually decreased in thickness and by 10—11 years was not much wider than the proliferative zone (Fig. 1 d). The cells in the upper part of the zone were small and round, but successively increased in size towards the center of the joint and become hypertrophic. The deepest part of the zone underwent pericellular mineralization. The hypertrophic zone was divided into two distinguishable layers, an upper non-mineralized layer and a deeper, mineralized layer (Fig. 1 b—e). Until about 13—15 years of age, the mineralized zone had a constant width because the deepest layer was resorbed by chondroclasts. This zone of erosion was fairly even. After 13—15 years, the hypertrophic zone decreased in thickness and by 19—27 years, only some cartilage cell islands were seen in the superior and anterior part of the condyle (Fig. 1 f).

Mandibular neck. In young specimens, apposition was observed laterally while resorption occurred medially (Fig. 2 a—b). With increasing age, the pattern changed (Fig. 2 d) and in young adults resting lines, indicative of remodelling, were seen on both sides (Fig. 2 f). Remodelling processes were also observed in sagittal sections, especially anteriorly (Fig. 2 e). The lateral pterygoid muscle was attached to the capsule, whose collagenous fibers had the same direction as the muscle fibers (Fig. 2 c). Osteoblastic activity was observed in most specimens on the anterior bone surface (Fig. 2 c).

Temporal component. At birth, the articular surface was almost flat (without the S-shaped profile seen in adults)

Fig. 2. The mandibular neck in the transverse plane (a, b, d, f) and sagittal plane (c, e)
a. 1 y. 9 m. ♀ (Mayer). Lateral apposition. Magnification: x 40
b. 3 y. 8 m. ♂ (Bock). Medial resorption. × 40
c. 2 y. 10 m. ♂ (Bock). Fibers from the lateral pterygoid muscle (lpm) inserting into the capsule anteriorly. Osteoblastic activity (ob) at the bone surface. × 40
d. 6 y. 5 m. ♀ (Bock). Apposition starting medially at resting line (rl). × 100
e. 21 y. 4 m. ♂ (Bock). Resting lines indicating remodelling anteriorly. × 100
f. 23 y. 4 m. ♀ (PAS). Resting lines, indicating remodelling medially. × 40


a
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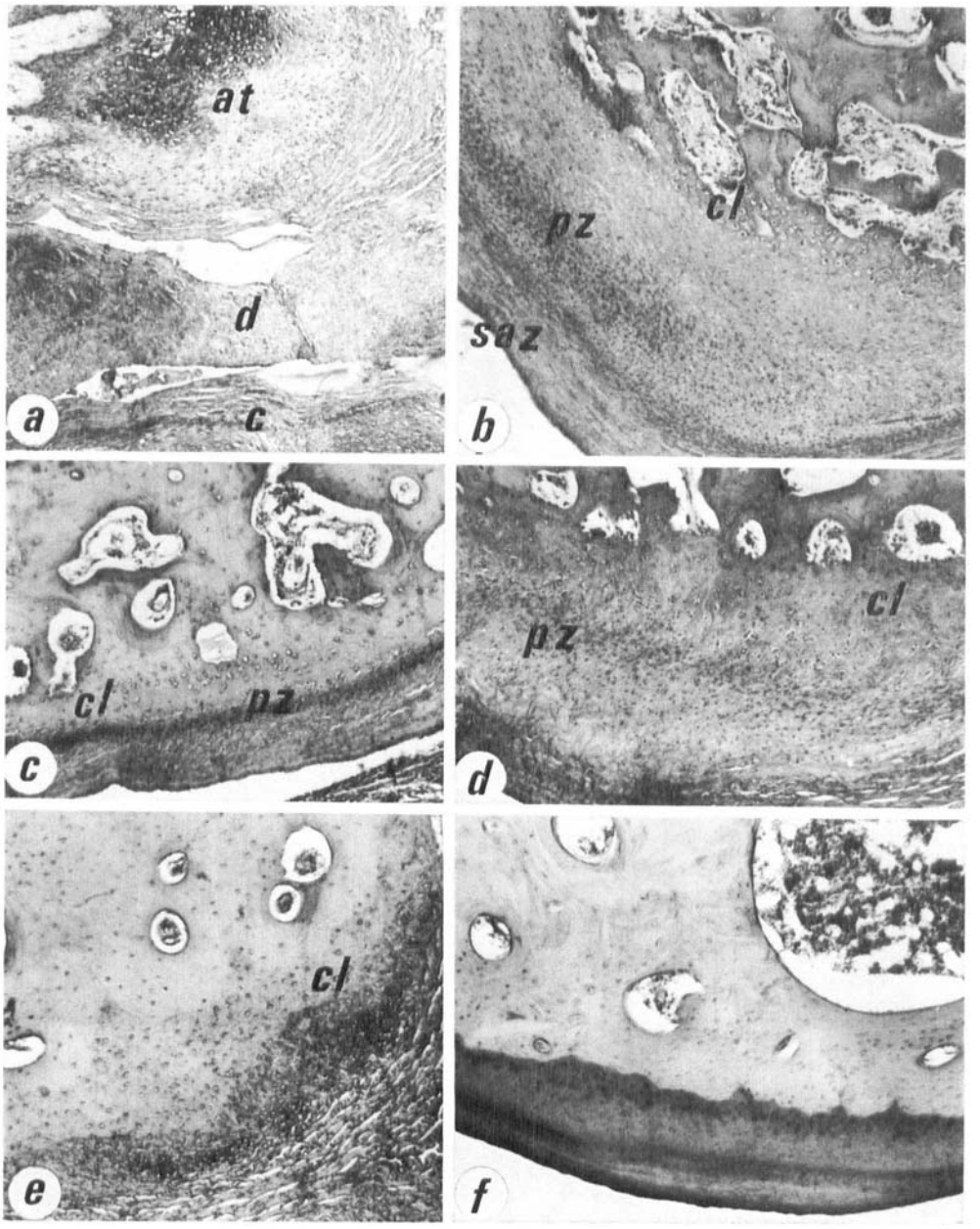

Fig. 3. The articular tubercle as seen in a central section in the sagittal plane. Anterior to the right.
a. newborn ♂ (Bock). ×25
b. 3 y. 4 m. ♂ (Bock). ×40
c. 6 y. 0 m. ♂ (Bock). ×40
d. 13 y. 10 m. ♂ (Bock). ×40
e. 16 y. 7 m. ♂ (Bock). ×40
f. 21 y. 0 m. ♀ (PAS). ×40
at = articular tubercle
d = disk
c = condyle
saz = surface articular zone
pz = proliferative zone
cl = cartilage layer

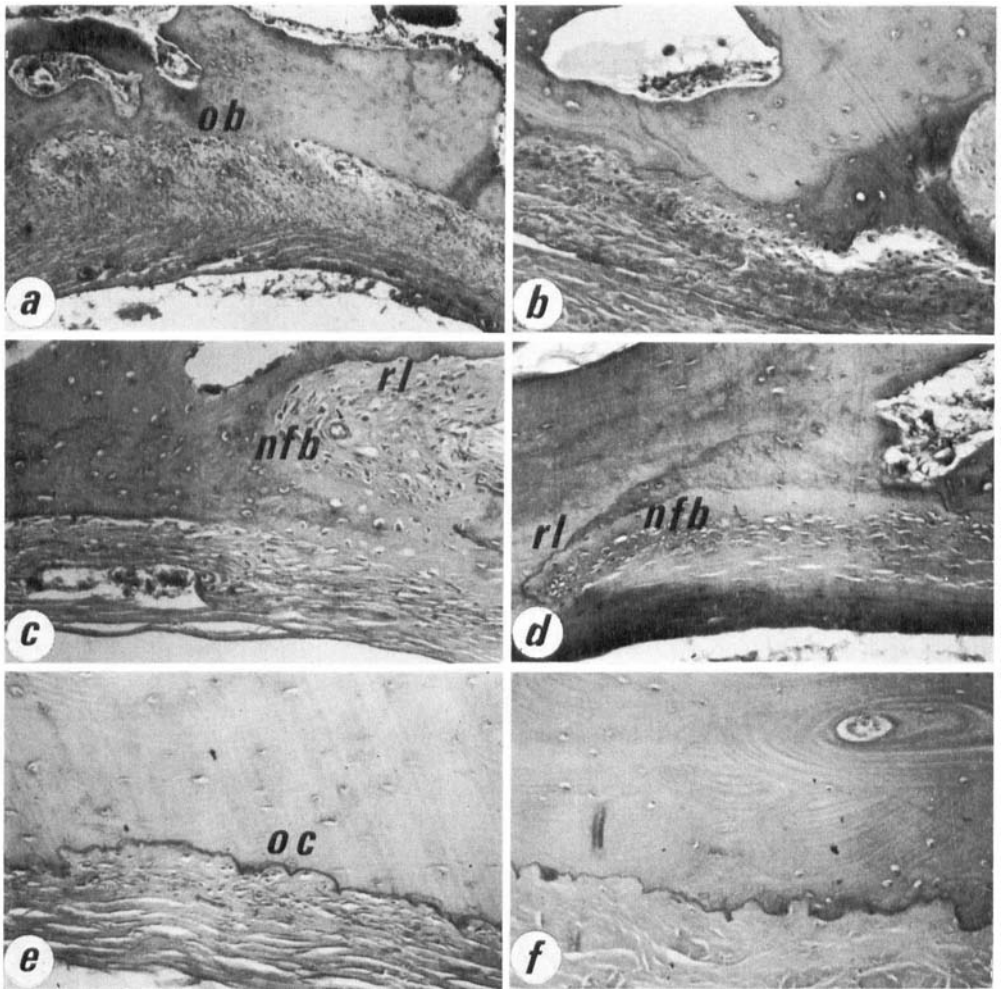

Fig. 4. Remodelling processes in the fossa in sagittal (a, c, e) and transverse (b, d, f) planes
a. 6 months ♂ (Bock). Osteoblastic activity (ob), indicating apposition. Magnification: × 40
b. 1 y. 9 m. ♀ (Mayer). × 100
c. 6 y. 10 m. ♂ (Bock). Resting line (rl) with newly formed bone (nfb). × 100
d. 7 y. 11 m. ♀ (Bock). × 100
e. 16 y. 7 m. ♂ (Bock). Osteoclastic activity (oc), indicating resorption. × 100
f. 25 y. 10 m. ♀ (Mayer). × 100

because the articular tubercle had just begun to form (Fig. 3 a). The articular surface was lined with a layer of fibrous connective tissue, which resembled the fibrous connective tissue layer of the condyle. It was thus vascular at birth and remained so for some years afterwards. With increasing age, it became richer in collagen with fibers running in an antero-posterior direction (Fig. 3 b—e).

At birth a transitory cartilage was observed at the future site of the tubercular eminence (Fig. 3 a). This cartilage soon decreased in thickness, was missing in the fossa (Fig. 4) and occurred only as a thin layer on the articular tubercle, mainly on the inferior part (Fig. 3 b—e). The cells were fewer and smaller than those in the condylar cartilage. An increase in the thickness of the proliferative zone could

be seen at puberty (Fig. 3 d). A proliferative zone could be seen up to 17—18 years of age (Fig. 3 e). Cartilage having few cells was demonstrable in the lower part of the tuberculum up to adulthood (Fig. 3 f). In the fossa, remodelling was observed from early childhood to adulthood (Fig. 4).

The disk. At birth, the disk was relatively uniform in thickness, but the central part very soon became thinner, while the anterior and particularly the posterior parts became much thicker. During the first few years of life the disk was very well vascularised and highly cellular.

Later it became richer in collagen and had fewer cells while the vessels disappeared from the central part. At birth, the cells consisted mainly of fibroblasts. With increasing age, the proportion of chondroid cells increased. The collagen fibres were first oriented in an anteroposterior direction and were very dense in the central part. With increasing age they formed a strong netlike pattern in three dimensions in the anterior, but especially in the posterior part.

DISCUSSION

The specimens of the temporomandibular joints studied represented the age period from birth to the first few years of adulthood, and both sexes. All the specimens were obtained from individuals who had died sudden deaths, usually accidents, suicide, or acute diseases. None of them had had chronic diseases that might have influenced the growth of the joint. The specimens may thus be regarded as representative of a normal population in the age range under consideration.

The histological procedures entailed problems regarding the orientation, decalcification and sectioning. Some controversial conceptions of the anatomy of the temporomandibular joint can probably be explained by such histo-technical difficulties which have been solved in different ways by different examiners. Here special care was taken to obtain sections from the central part of the joint for study and illustrations.

The morphological picture of the human condylar cartilage with its age changes was very similar to that seen in earlier animal studies. In monkeys, the proliferative zone and the zones of erosion and new bone formation showed a marked radioactivity in young animals after administration of ^{3}H-thymidine, an activity that decreased rapidly with age (*Kanouse et al.*, 1969). The site of the main growth activity was in the proliferative zone, which has also been pointed out in more recent investigations in the rat (*Bremers*, 1973; *Durkin et al.*, 1973). In humans the proliferative and cartilage zones decreased in thickness relatively soon after birth. No change in morphologic characteristics of these layers was observed during the pubertal growth spurt. The same finding was reported by *Wright & Moffett* (1974). It was thus not possible to produce micro-morphologic support for the assumption that the condylar cartilage is a very active growth center during the period between 10 and 15 years of age. However, in the articular tubercle, an increase in the proliferative zone was seen early in the teen-age period. In the late teen-age period the hypertrophic condylar cartilage layer decreased further, suggesting that growth was stopping. It must, however, be pointed out that the histological method used did not permit any definite conclusions concerning the degree of activity.

The growth of the mandible is rapid in the first years of life. It is interesting

to note that the condylar cartilage is penetrated by highly vascularized connective tissue columns during that period, which *Blackwood* (1965) and *Wright & Moffett* (1974) also observed. They postulated that the function of this vascularization is to nourish the cartilage, thus enabling more rapid growth during the period when the mandible has to accommodate to developing and erupting deciduous teeth. That is a plausible explanation for the cartilage becoming avascular at the same time as the deciduous dentitions is completed.

After 20 years of a age, the superior and anterior parts of the condyle and the postero-inferior parts of the articular tubercle still contain cartilage with only a few cells. This has been considered to constitute the basis for remodelling processes which occur later in life in response to various stimuli (*Carlsson & Öberg*, 1974). The localization of these cartilaginous remnants may also be interpreted as an adaptation of the tissues to functional loading of the joint. The morphologic changes with increasing age in the articular surface of the condyle and that of the temporal component, as well as in the central part of the disk, also support this interpretation.

Other factors than the cartilage thus must play an important role in remodelling processes of the joint. This can be seen in the fossa and the neck of the mandible. In the neck the periosteum is probably mainly responsible for the remodelling. This suggests that periosteal growth is of great importance in the entire growth pattern of the mandible; after puberty it may even be of greater importance than cartilage growth.

The results of this investigation thus seem to indicate that, unlike the growth plates of the long bones, the condylar cartilage is not a primary growth center and that growth of the temporomandibular joint is of adaptive nature. The condylar cartilage seems to act as a secondary cartilage, that is, to resist intermittent pressure and movement and to produce rapid growth during early life. This conclusion is in correspondence with the findings of *Koski & Rönning* (1965); *Duterloo & Jansen* (1969); *Durkin et al.* (1973) and *Koski* (1974).

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