
Coronoid process elongation in rhesus monkeys (*Macaca mulatta*) after experimentally induced mandibular hypomobility

A cephalometric and histologic study

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The present study provided an experimental model that allowed a cephalometric and histologic analysis of craniofacial growth in monkeys with induced translatory impairment of the mandibular condyle. Cauterization was performed anterior to the joint in nine experimental rhesus monkeys, while eight animals served as control subjects. The experimental procedure produced mandibular hypomobility in six animals, in three by means of temporomandibular joint ankylosis and in three by means of dense scar tissue formation anterior to the joint. In the remaining three experimental animals no restriction of mandibular mobility was created. Mandibular hypomobility was found to induce elongation of the coronoid process and was also associated with bone deposition in the gonial region. In contrast, normal remodeling of the gonial region was found in the experimental animals with normal range of movements and in the control animals. (ORAL SURG ORAL MED ORAL PATHOL 1990;70:704-10)

Clinical studies have demonstrated that the growth and the position of the mandible is affected by temporomandibular joint (TMJ) ankylosis.¹ Hohl and coworkers² experimentally induced TMJ ankylosis in juvenile monkeys and found it to result in an impairment of normal growth and a marked shortening of the mandibular ramus on the ipsilateral side, with tilting of the occlusal plane and major facial asymmetry. They noted that the tip of the coronoid process was seen above the zygomatic arch in one experimental animal. Whether this change in coronoid process position was due to the shortening of the condylar

process only or included an additional elongation of the coronoid process was not discussed. Mandibular asymmetry involving enlargement of the coronoid process was reported by Farrar and McCarty³ as a clinical finding at mandibular hypomobility associated with conditions such as TMJ disk displacement and osteoarthritis. Their clinical observation was supported by a computerized tomography study showing that the coronoid processes in patients with displaced TMJ disks were statistically significantly longer than those in control subjects.⁴

Extensive elongation of the coronoid process can result in its locking against the zygomatic bone on mouth opening. An association between coronoid process locking and permanent TMJ disk displacement on the ipsilateral side was demonstrated by Isberg and coworkers,⁵ who indicated that the aberrant dimension of the coronoid process was induced by mandibular hypomobility. In another study, cephalometric analysis of a group of patients with congenital coronoid process locking that had developed during puberty and of a group of patients with induced coronoid process locking revealed a statistically significantly smaller gonion angle in the congenital group

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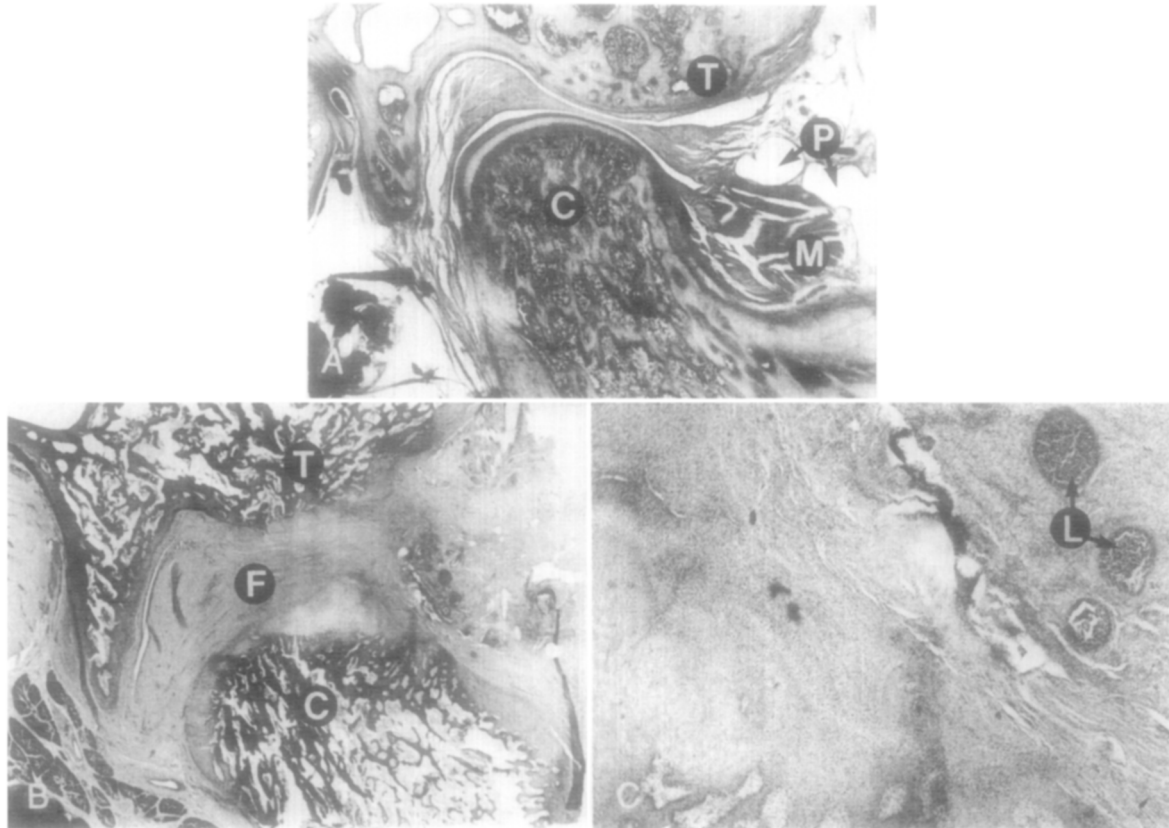


Fig. 1. A, Right TMJ from control animal with disk in normal position between condyle (C) and temporal bone (T). In region anterior to condyle, lateral pterygoid muscle (M), anterior disk attachment, and pterygoid venous plexus (P) are visible. (Hematoxylin-eosin stain.) B, Right TMJ from experimental animal with fibrous ankylosis (F). Condyle (C) and temporal bone (T) show signs of bone resorption. Joint spaces are occupied by dense collagenous tissue, and normal joint architecture is lost. Fibrous tissue extends into region anterior to joint. (Hematoxylin-eosin stain.) C, Detail from anterosuperior aspect of condyle of experimental animal. Lymphoid follicles (L) are seen in region of previous capsule. (Hematoxylin-eosin stain.)

than in the induced group and the control group.⁶ The authors found it reasonable to assume that the more prominent gonial region in the congenital group was due to bone deposition that had taken place in the growing individual as a result of the altered mandibular mobility.

The aim of the present study was to provide an experimental model that would allow an investigation of mandibular growth in rhesus monkeys (*Macaca mulatta*) after induced translatory impairment of the mandibular condyle.

MATERIAL AND METHODS

Seventeen juvenile male rhesus monkeys (*Macaca mulatta*), nine experimental and eight control animals were used in this study. The animals were 18 to 24 months of age at the beginning of the experiment, as determined by the presence of a deciduous dentition and erupted first molars.⁷ At least four implants were placed in the right side of the mandible by means

of an extraoral approach^{8,9}: one in the symphysis, two in the mandibular body, and one in the ramus. The implants provided a reference for measuring growth changes in the mandible. Additional implants were placed in the maxillary and premaxillary regions, in the frontal bone, and in the cranial base¹⁰ to allow for the evaluation of the growth of the maxilla and mandible relative to cranial base structures.

Surgical procedures

At the beginning of the investigation period, the experimental animals were subjected to cauterization anterior to the TMJ in the lateral pterygoid muscle area to influence mandibular mobility as is described below.

Each experimental monkey was preanesthetized with ketamine hydrochloride administered intramuscularly and was then brought to a surgical level of anesthesia with intravenous pentobarbital sodium. The region of the TMJ was scrubbed with a povidone-

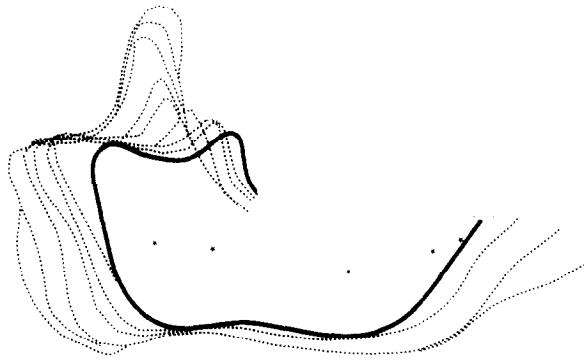


Fig. 2. Growth pattern of mandible in experimental animal with induced TMJ bony ankylosis. Experimental period lasted 3½ years. *Solid line* represents initial cephalometric tracing, and *dashed lines* represent successive tracings on metal implants superimposed. Mandibular growth was grossly affected, with major reduction in ramus height and elongation of coronoid process. Growth of coronoid process continued during young adult life. Note bone deposition in gonial region.

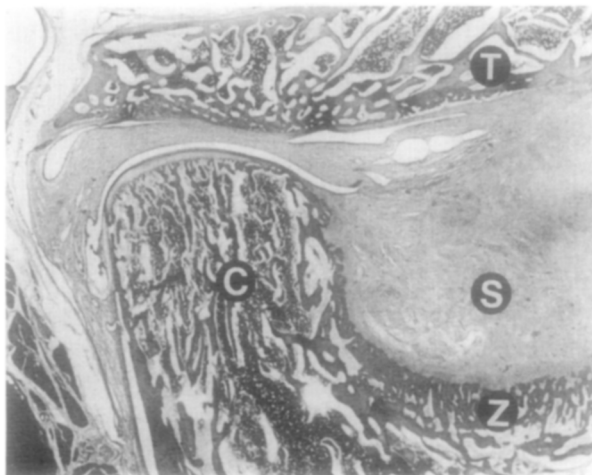


Fig. 3. Right TMJ from experimental animal with disk in normal position above condyle (*C*) and with well-defined joint spaces. Dense scar tissue (*S*) occupying area anterior to joint was attached to zygoid notch (*Z*) and temporal bone (*T*). (Hematoxylin-eosin stain.)

iodine (Betadine) solution bilaterally, and the animal was draped. Lidocaine (2%) was injected subcutaneously in the area of the TMJ.

An inverted hockey stick incision, approximately 1.5 cm horizontally just anterior to the auditory canal and 1 cm obliquely towards the tragus of the ear, was made through the skin and underlying subcutaneous tissues with a scalpel. The posterior portions of the zygomatic arch and deep masseter muscle were exposed by blunt dissection. An incision was then made along the exposed zygomatic arch through the periosteum. With a periosteal elevator, the periosteum and muscle were stripped off the arch and



Fig. 4. Detail from region anterior to condyle in Fig. 5. Dense scar tissues attach to condyle (*C*). Small vessels (*V*) are visible, but muscle fibers are absent. (Hematoxylin-eosin stain.)

reflected inferiorly to expose the infratemporal fossa and the posterior third of the lateral pterygoid muscle through the mandibular notch. An electrocautery knife was then used to sever the lateral pterygoid muscle at its junction with the capsule of the TMJ. The knife, set on coagulate, was then circulated throughout the rest of the infratemporal fossa to destroy the entire lateral pterygoid muscle. After direct observation to indicate that the muscle was no longer present and viable, the infratemporal fossa was packed with gauze sponges and the same procedure was repeated on the other side. After removal of the sponge, the masseter muscle and periosteum were sutured back to their original locations along the zygomatic arch with resorbable suture (4-0 gut), and the wound was closed in layers with 4-0 polyglycolic acid (Dexon) suture. Animals were returned to their cages after administration of antibiotics, which were continued on a daily basis for a 1-week period. Recovery from the surgery was uneventful.

Radiography

Lateral cephalometric head films were taken on each animal before any surgical procedures were performed and at postoperative intervals of 2, 4, 6, and 8 weeks and then every 12 weeks thereafter throughout the experimental period, which ranged from 2 weeks to 4½ years. The control animals were radiographically monitored over a period of from 6 months to 2½ years at corresponding intervals. At each session, cephalograms were taken at mouth closure and with the mouth wide open. Skeletal changes in the mandible were measured by tracing of the implants and of the outline of the mandible, including the mandibular condyle and coronoid process. Changes

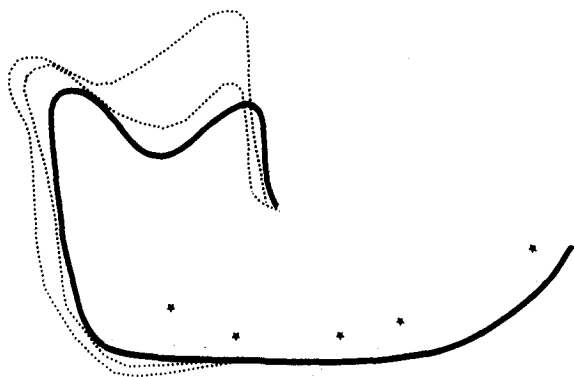


Fig. 5. Growth pattern of mandible in experimental animal with induced mandibular hypomobility due to dense scar tissue anterior to joint. Experimental period lasted 6 weeks. *Solid line* represents initial cephalometric tracing, and *dotted lines* successive tracings with metal implants superimposed. Note growth of coronoid process and bone deposition in gonial region.

were noted between successive cephalograms after superimposition of the implants. The heights of the mandible at the coronoid process and of the mandibular condyle were measured along a line perpendicular to the original mandibular plane. The location of the tip of the coronoid process in relation to the zygomatic arch was registered with the mouth closed and with the mouth wide open. Maximal mouth opening was determined during general anesthesia. Skeletal changes in the maxilla were determined by the same procedure.

Histology

The animals were put to death by cardiac perfusion with saline solution, the experimental animals 2 weeks to 4½ years postoperatively and the control animals at the end of the observation period, which ranged from 6 months to 2½ years. Neutral buffered formalin (5%) was used to fix the tissue for histologic analysis. Tissue blocks were taken from the right and left TMJ regions and kept in the fixation solution for a week before decalcification in 40% edetate at 37° C. After adequate demineralization, the specimens were routinely processed and embedded in paraffin with the cutting plane perpendicular to the long axis of the condyle. Parasagittal sections were collected from throughout the joint. The sections were stained with hematoxylin and eosin and were examined with a light microscope.

RESULTS

Experimental animals with TMJ ankylosis and mandibular hypomobility

Three of the experimental animals developed an impairment of mouth opening due to TMJ ankylosis.

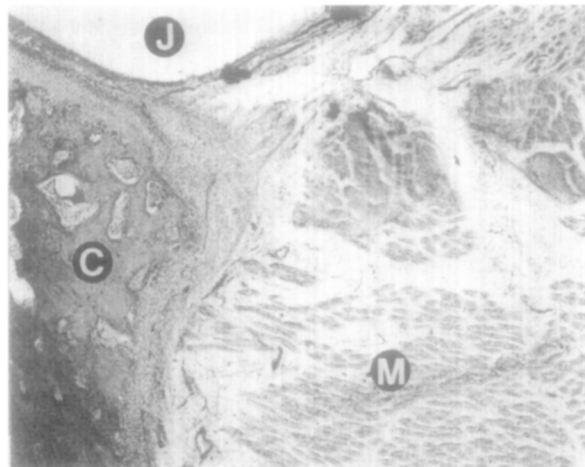


Fig. 6. Right TMJ from experimental animal with reattachment of lateral pterygoid muscle (*M*). Anterior lower joint space (*J*) and condyle (*C*). (Hematoxylin and eosin stain.)

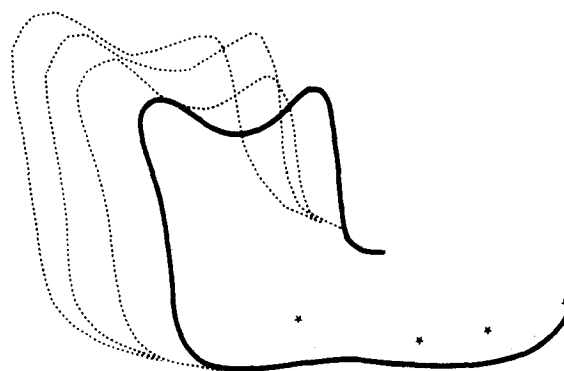


Fig. 7. Growth pattern of mandible in experimental animal with unaffected mandibular mobility postoperatively. Experimental period lasted 3 years. *Solid line* represents initial cephalometric tracing, and *dashed lines* successive tracings with metal implants superimposed. Height of coronoid process never exceeds height of condylar process. Note remodeling in gonial region.

One of these three animals was put to death 8 weeks postoperatively, at the time having a mouth opening of 15 mm. Histology of the joints revealed bilateral fibrous ankylosis (Fig. 1, *B* and *C*). (For comparison, a histologic section from a control animal is shown in Fig. 1, *A*). The disk tissue was absent in both joints. The cartilage on the articulating surfaces was eroded and had been replaced with connective tissue. Extensive bone resorption was present both on the condyle and in the temporal joint component. In the left joint, lymphoid follicles were seen and a generalized hypertrophy of cartilage into the joint space was present. The joint space was filled with connective tissue posterosuperiorly. Radiographically, no change in condy-

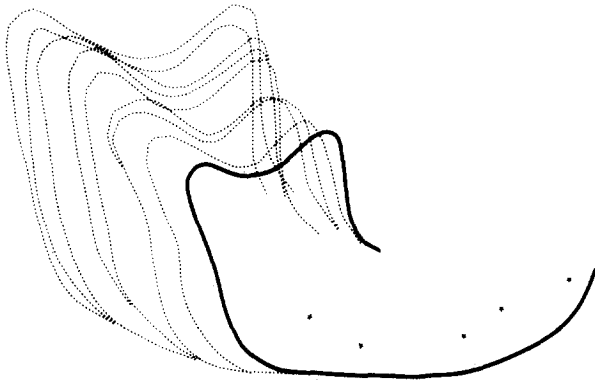


Fig. 8. Growth pattern in control animal. Observation period lasted 2½ years. *Solid line* represents initial cephalometric tracing and *dashed lines* successive tracings with metal implants superimposed. With exception of initial registration, height of coronoid process never exceeds height of condylar process.

lar height was present, but an increase in the height of the coronoid process was observed such that the tip of the coronoid process was located above the zygomatic arch at mouth closure. Bone deposition in the gonial region was also seen. The horizontal and vertical growth of the maxilla was within the same range as in the control animals.

In a second animal, put to death 3½ years postoperatively, a unilateral bony ankylosis had developed in the right TMJ. Maximal mouth opening in the animal was 5 mm. A definition of the condyle and distinct cartilage layers was not visible, and histopathologically the joint space had been replaced by bone. The growth of the mandible was grossly affected on the ipsilateral side, with a major reduction in ramus height and an elongation of the coronoid process observed when compared with the same dimensions in the control animals (Fig. 2). The tip of the coronoid process reached considerably above the zygomatic arch. At the end of the experiment this animal was a young adult with all third molars erupted. Growth of the coronoid process continued in the young adult after the growth of the rest of the mandible had essentially ceased. After 24 weeks, vertical maxillary growth was significantly greater in this animal than in the control animals, but after 48 weeks no further vertical growth was noted. At the end of the experimental period, the vertical dimension of the maxilla was impaired in comparison with that of the control animals. The occlusal contact was maintained throughout the experimental period.

A third animal, put to death after 4½ years, had TMJ bony ankylosis bilaterally. There was no opening ability at all. No joint structures were seen in the radiographs on either side. One joint was examined histologically, and no joint structures were visible.

The tip of the coronoid process was noted radiographically to be located considerably above the zygomatic arch. After 48 weeks the vertical growth of the maxilla was greater than that of the control animals, but the premaxilla had tilted upwards. At the end of the experimental period, the overall growth of the maxilla was significantly smaller than that of the control animals. Occlusal contact was maintained throughout the experimental period.

Experimental animals with dense scar tissue formation anterior to the joint and mandibular hypomobility

Three other experimental animals developed an impairment of mouth opening as a result of bilateral scar tissue formation anterior to the joint (Figs. 3 and 4). Normal condylar translation was thereby mechanically prevented. There was no involvement of their TMJs. These animals were put to death 2, 3, and 6 weeks postoperatively. Maximal opening was 17, 25, and 25 mm, respectively. The coronoid process reached the zygomatic arch, but the tip of the process was not seen above the arch. In the animals sacrificed after 2 and 3 weeks, the growth of the gonial region did not deviate from what was found in the control animals. In the animal observed during an experimental period of 6 weeks, bone deposition was noted (Fig. 5) after 4 weeks, but no further deposition was observed after another 2 weeks. The horizontal and the vertical growth of the maxilla was of the same magnitude as was observed in the control animals.

Experimental animals with unaffected mandibular mobility

The remaining three of the experimental animals were unaffected with regard to mandibular mobility. The experimental period lasted for 12, 163, and 156 weeks, respectively. Histologic evaluation of their TMJs showed a reattachment of the lateral pterygoid muscle but no involvement of the joint and no scar tissue anterior to it (Fig. 6). Bone remodeling was seen in the gonial region (Fig. 7) that was comparable to that registered in the control animals (Fig. 8). There was no difference in maxillary growth between the two groups.

The growth of the condylar and coronoid processes in the different animals is compared in Fig. 9. In all experimental animals with impaired mouth opening, the coronoid process was located superior to the condylar process. In neither the control animals nor the experimental animals with unaffected TMJs and unaffected mandibular function was the coronoid process located superior to the condylar process. The tip of the coronoid process in all of these animals at any phase of development did not reach the level of the zygomatic arch.

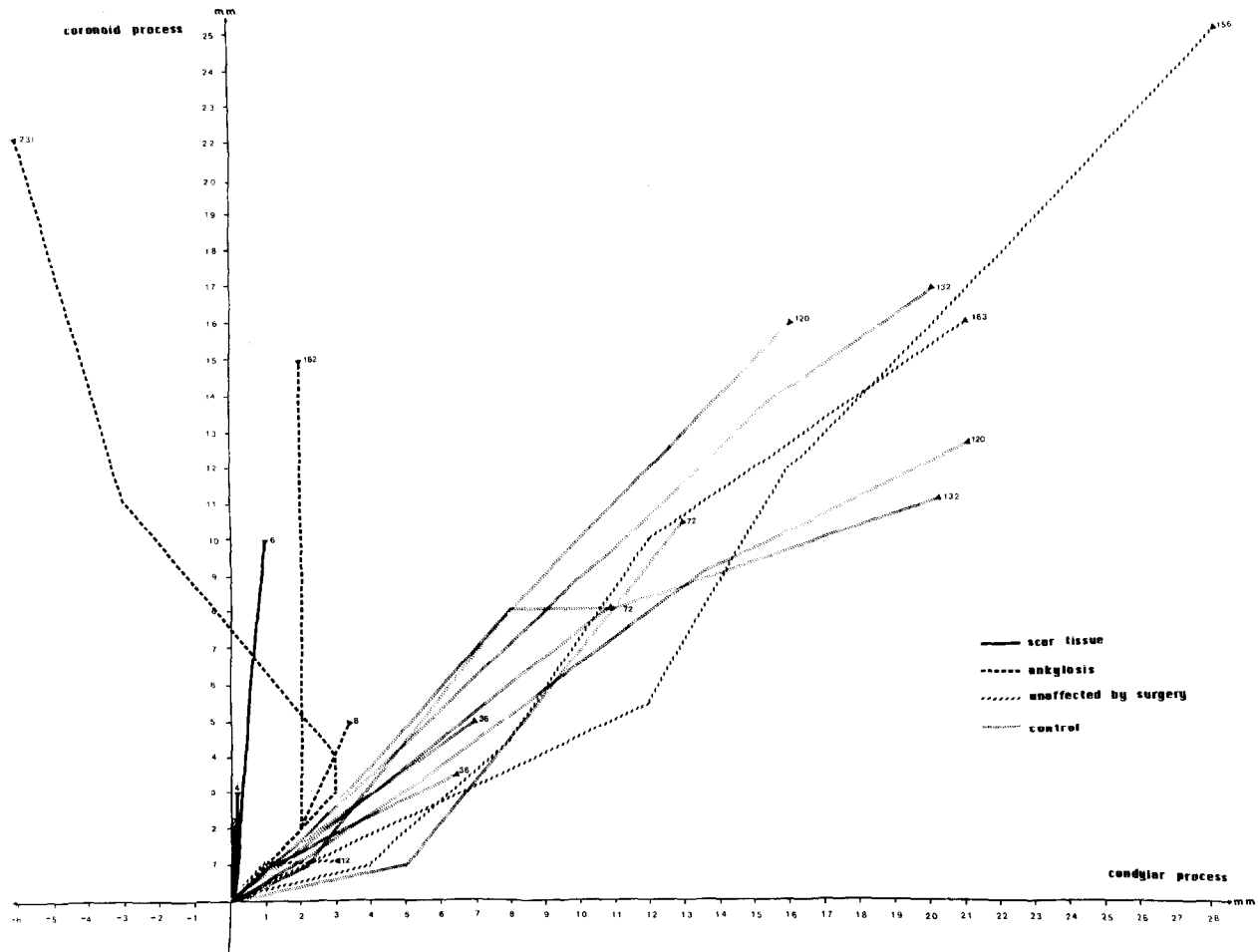


Fig. 9. Diagram showing amount of growth registered in experimental animals with induced mandibular hypomobility due to TMJ ankylosis and dense scar tissue formation anterior to joint, respectively, in experimental animals without any postsurgical influence on mandibular mobility and in control animals. Note excessive growth of coronoid process in experimental animals with hypomobility due to ankylosis or dense scar tissue formation anterior to TMJ. Length of experimental period is given at end of each curve.

DISCUSSION

The present experimental investigation demonstrated that mandibular hypomobility can result in an elongation of the coronoid process. In three experimental animals in this study, mandibular hypomobility was caused by induction of dense scar tissue anterior to the joint. To some extent, this condition resembles a TMJ with a disk permanently displaced anterior to its condyle. In contrast to joints with ankylosis, the articulating surfaces of the condylar and temporal joint components primarily remain unaffected in both these conditions. A displaced TMJ disk and dense scar tissue formation, respectively, develop a mechanical obstacle restricting condylar translation. It is therefore reasonable to assume that mandibular hypomobility of either origin may have the same effect of the craniofacial growth. The results of the present study lend support to the finding of an association between patients with longstanding mandibular hypomobility due to a permanently displaced

TMJ disk and those with mandibular locking due to coronoid process hyperplasia.⁵ In the experimental animals with scar tissue formation, the experimental period lasted only 2, 4, and 6 weeks, respectively. Despite the limited observation period, the growth of the coronoid process was found to exceed that of the condylar process and resulted in an elongation compared to the growth in the control animals. Although the growth was insufficient to cause any locking of the process against the zygomatic bone, locking is likely to have happened over a longer experimental period corresponding to the development of coronoid process locking in patients with a duration of permanent disk displacement of several years.⁵ Induction of growth of the coronoid process can take place also in adults, as demonstrated in a long-term experimental animal with mandibular hypomobility in this study.

There is a direct relationship between the development of the coronoid process and the function of the temporalis muscle.^{11, 12} Hyperactivity of the tempo-

ralis muscle has been registered experimentally in human subjects when they performed mouth opening without condylar translation.¹³ Muscular hyperactivity of the temporalis muscle is thus likely to have been triggered in the experimental animals with restricted condylar translation and is probably an etiologic factor in the reactive enlargement of the coronoid process.

The present study also showed that induction of coronoid process hyperplasia can occur in cases of TMJ ankylosis affecting condylar growth and joint mobility. When the restriction of mandibular movements in this study was caused by mechanical induction of TMJ ankylosis, it led to serious impairment of normal mandibular growth, both after a virtually complete bony ankylosis and as a result of partial ankylosis with considerable fibrosis. The deformation of the craniofacial skeleton was not confined to the condylar region but involved the mandibular body and also the maxilla. The changes in the maxilla are in part likely to be compensatory since occlusal contact was maintained throughout the experimental period in spite of the pronounced deviant mandibular growth. The results are in agreement with the findings by Hohl and coworkers,² who studied facial asymmetry after experimentally induced ankylosis in monkeys.

A cephalometric analysis was performed on patients with coronoid process elongation and locking.⁶ The study comprised both patients with coronoid process locking of congenital origin (i.e., their locking symptoms had developed during the period of pubertal growth) and patients with induced locking of adult onset. A markedly more prominent gonial region, expressed by a small gonial angle, was observed in the congenital group and was interpreted as bone deposition. Bone deposition in this region has previously been reported to occur in monkeys after removal of a protrusive appliance.¹⁴ The experimental monkeys with mandibular hypomobility in the present study also showed bone deposition in the gonial region in contrast to the gonial remodeling seen in both the experimental animals with normal mandibular mobility and in the control animals. Remodeling in the gonial region is in accordance with the expected normal mandibular growth pattern.⁹ An experimental locking of the mandible in human subjects has been found to result in an increased activity of the masseter and medial pterygoid muscles.¹³ Since the development of the gonial region is directly related to the functions of

the superficial masseter and the medial pterygoid muscles,¹⁵ such increased muscular activity is likely to have stimulated bone deposition in the areas of muscle insertion in the gonial region.

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