ORIGINAL ARTICLE

Condyle-fossa modifications and muscle interactions during Herbst treatment, Part 2. Results and conclusions

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Herbst appliances were activated progressively in growing nonhuman primates, and the results were compared with primate and human controls. The methods and materials of this research are explained in Part 1 of this study. The results are discussed here in Part 2. All experimental subjects developed large super Class I malocclusions, the result of many factors including posterior movement of the maxilla and the maxillary teeth, an increased horizontal component of condylar growth, and anterior displacement of the mandible and the mandibular teeth. The growth modification measured in the glenoid fossa was in an inferior and anterior direction. Restriction of the downward and backward growth of the fossa observed in the control subjects might additionally contribute to the overall super Class I malocclusion. Clinically, these combined effects could be significant at the fossa. The restriction of local temporal bone (fossa) growth cannot be observed clinically; thus, these results might also clarify some Class II correction effects that cannot be explained with functional appliances. Differences in the area and maximum thickness of new bone formation in the glenoid fossa and in condylar growth were statistically significant. The bony changes in the condyle and the glenoid fossa were correlated with decreased postural electromyographic activity during the experimental period. Results from permanently implanted electromyographic sensors demonstrated that lateral pterygoid muscle hyperactivity was not associated with condyle-glenoid fossa growth modification with functional appliances, and that other factors, such as reciprocal stretch forces and subsequent transduction along the fibrocartilage between the displaced condyle and fossa, might play a more significant role in new bone formation. These results support the growth relativity concept. (Am J Orthod Dentofacial Orthop 2003;124:13-29)

A review of the literature[1] indicates that the glenoid fossa has the potential to remodel during functional appliance therapy. For decades, the prevailing notion was that condylar growth modification was caused by lateral pterygoid muscle hyperactivity.[2] The growth relativity theory,[3] on the other hand, describes a specific set of reciprocal soft tissue forces that operate during Herbst treatment, when muscle hyperactivity does not appear to be the primary cause of skeletal change. The displaced condyle modifies in a radiating manner relative to the fossa, and the fossa grows in a radiating fashion relative to the condyle. The retrodiskal tissues stretched between the displaced condyle and the fossa contribute to the formation of new bone in each region. Furthermore, force referral or transduction from the posterior retrodiskal attachment to the condyle radiating along condylar fibrocartilage has been implicated in growth modification. The purpose of this research was to test the growth relativity theory to improve our understanding of how orthopedic appliances work.

We used cephalometric analysis plus intravenous tetracycline vital staining, histological assessment, and electromyographic analysis to study the glenoid fossa remodeling response associated with continuous mandibular protrusion in juvenile nonhuman primates.

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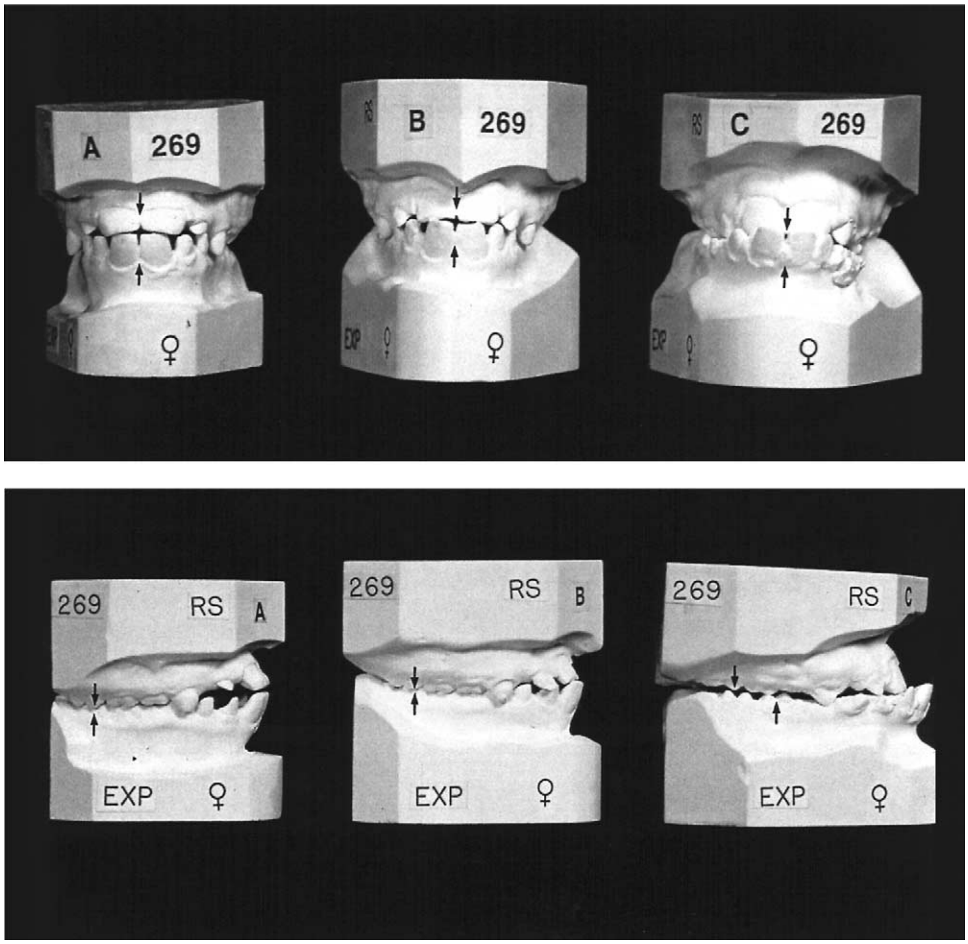

Fig 1. Juvenile animal #269. Note super Class I (or severity of Class III) malocclusion in both posterior and anterior segments. **A**, Start of control period; **B**, end of control period; **C**, end of 18-week experimental period.

These techniques are discussed in Part 1 of our study.[1] Here, in Part 2, we expand the discussion of our findings. By improving our understanding of how muscles and soft tissues function in relation to condyle-fossa bone modification, clinical finishing and retention of severe Class II malocclusions characterized by mandibular retrognathism and treated with Herbst appliances with occlusal coverage can be improved.

MATERIAL AND METHODS

The sample for this study included 56 nonhuman primate and human subjects. Fifteen cynomologus monkeys (*Macaca fascicularis*) were divided into juvenile, adolescent, and adult groups; the 8 juveniles underwent Herbst treatment or served as actual and sham controls; they are the focus of this study.[1] The rest of the sample comprised 17 human patients treated with Herbst appliances (future publication) and 24 human controls from the Burlington Growth Center. We used cephalometric, histomorphometric, and electromyographic (EMG) techniques to study muscle-bone interactions during Herbst appliance therapy. The material and methods are described in Part 1 of this report.[1]

RESULTS

In the 5 juvenile primates treated with Herbst appliances, the normal occlusion was altered to a super Class I malocclusion by the end of the 6-, 12-, and 18-week treatment period (Fig 1). At the end of the various experimental periods, the mandibles could not be manipulated posteriorly under general anesthesia. The dental changes in the juvenile animals[4] were similar to those observed in adolescent and adult nonhuman primate subjects.[5] This finding was generally consistent in both animal and human subjects. The 12-week sham control animal wearing an inactive

Table I. Changes in position of maxillary and mandibular first molars and incisors from superimposition on maxillary and mandibular implants

	Maxilla				Mandible			
	First molar (mm)		Incisor (mm)		First molar (mm)		Incisor (mm)	
Number	Horizontal	Vertical	Horizontal	Vertical	Horizontal	Vertical	Horizontal	Vertical
299	−1.2	−1.5	−0.6	−1.8	−0.4	−0.6	−1.1	−1.0
285	−0.3	−0.4	−1.3	−0.5	−0.5	−0.5	−2.1	−2.3
271	0.8	−0.7	−0.6	−1.2	−1.4	−0.7	−0.8	−1.6
270	−0.4	−0.7	−1.0	−1.2	−1.1	−0.5	−1.5	−1.5
269	−2.0	−1.4	−0.6	−2.0	−0.9	−0.8	−2.5	−0.6
288 (sham)	−1.1	−0.8	−1.8	−3.4	−1.1	−0.7	−2.3	−0.7
273 (control)	0.0	0.0	−0.1	−0.3				
272 (control)	0.0	0.0	0.0	−0.1				

(+) posterior or superior. (−) anterior or inferior.

Herbst appliance maintained a normal occlusion with mild intrusion of the buccal segments.

Cephalometric evaluation and coordinate analysis

Table I shows the data for the cephalometric changes in the experimental and control subjects. The horizontal and vertical measurements of the anterior and posterior mandibular metallic implants indicate that the mandible was displaced anteriorly in the experimental subjects (Fig 2, *A*).

Superimposition of the tracings on the maxillary implants (Fig 2, *B*) showed that, in the experimental animals, the first permanent molars moved distally and were intruded while the incisors moved palatally and were extruded (Table I). The mandibular first molars moved mesially and slightly inferiorly while the incisors moved labially and inferiorly (Fig 2, *D*, and Table II). Minimal incisor movement occurred in the sham control animal, although the first permanent molars underwent a small mesial and inferior movement.

All experimental subjects had additional condylar extension, as shown by the mandibular superimpositions (Fig 2, *D*). The measurements from condylion to condylion (Table II) also confirmed this finding. In addition, the condylion to anterior mandibular implant (mandibular length) showed increased condylar growth compared with the controls (Fig 2, *C* and *D*; Table II). The direction of condylar growth varied among the subjects during the experimental period but was generally in a superior and posterior direction (Fig 2, *D*). These changes were statistically significant. Some minor flattening of the gonial angle associated with decreased EMG masseter muscle activity appeared to shorten the ramus as the condyle grew posteriorly and superiorly, measured from the metallic implants. This was not, however, considered "bone bending" but, rather, further growth modification of the ramus including the gonial angle due to the orthopedic appliance. These observations in juvenile, nonhuman primates in the early permanent dentition were consistent with results in humans with Herbst treatment.

Histologic evaluations

Histological examination of control condyles at relatively low (8X), high (100X), and very high (225X) magnifications showed little proliferation of the prechondroblastic or chondroblastic zones (Fig 3). In addition, the underlying endochondral bone formation was relatively flat. Decalcified sections from the experimental subjects in comparison showed greater proliferation of the condylar prechondroblastic and chondroblastic regions (Fig 4, *A*). The new endochondral bone was clearly observed as multidirectional, finger-like processes in a starburst pattern. This growth modification was in a superior and posterior direction. The 6-week experimental animal showed the most extensive cartilaginous tissue changes (Fig 4, *A*). At the 12-week and 18-week experimental periods, the prechondroblastic and chondroblastic layers that were remodeled were not as thick (Fig 4, *B* and *C*). This remodeling was due to rapid tissue replacement by new endochondral bone formation. The rapid endochondral bone formation and the removal of tetracycline in the experimental animals did not permit quantitative histomorphometric analysis of bone formation for the condyle. Thus, cephalometry was used to study the condylar parameters. Cephalometric analyses confirmed greater condylar extensions in both the horizontal and vertical dimensions in the experimental subjects compared with the controls. These findings were supported by qualitative examination of endochondral bone formation in undecalcified sections.

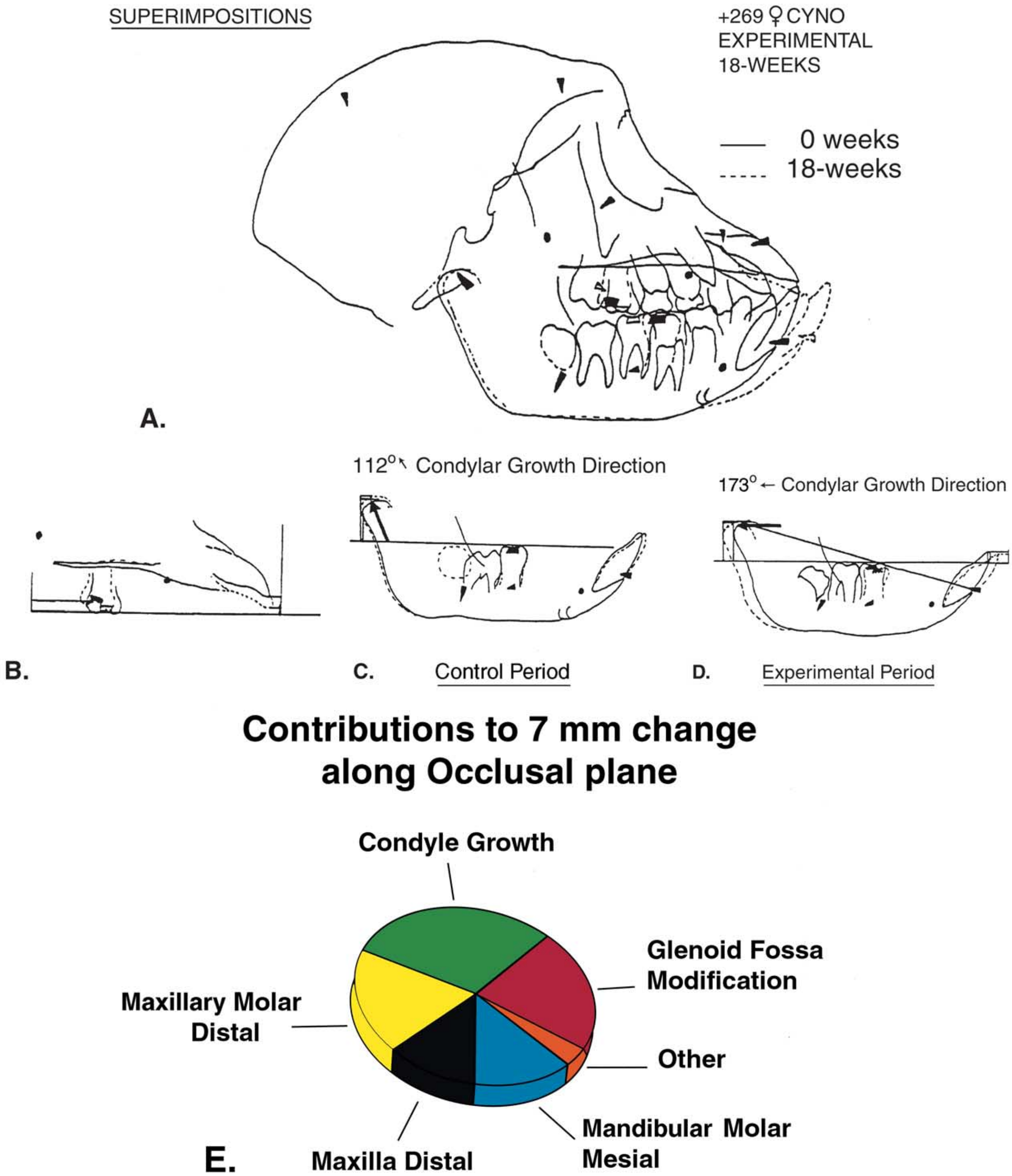

Fig 2. A, Horizontal and vertical distances measured in coordinate analysis of overall superimposition on anterior cranial base. **B**, Maxillary implants. **C**, Mandibular implants in control and, **D**, experimental periods. **E**, Contributions to 7-mm difference along occlusal plane between amalgam implants include condylar growth, condylar displacement, changes in maxilla, maxillary dental changes, mandibular dental changes, and miscellaneous changes that could be related to growth restriction of natural fossa growth. Condylar growth and condylar displacement or glenoid fossa remodeling contribute approximately 50% of changes parallel to occlusal plane. When restriction of maxillary growth is added, overall dentofacial orthopedic contribution is approximately 70% and orthodontic (dental) contribution is approximately 30% to changes parallel to occlusal plane.

Table II. Measured changed in mandibular morphology from overall superimposition on mandibular metallic implant during experimental period

			Condyle extension (mm)			
Number	*Duration (weeks)*	*Activation (mm)*	*Horizontal**	*Vertical***	*Co to Co (mm)*	*Co to AMI (mm)*
299	6	4	1.1	3.1	3.2	2.4
285	12	7	1.3	1.0	1.4	2.2
271	12	7	3.2	0.5	3.1	3.8
270	12	7	2.9	2.3	3.8	3.6
269	18	8	2.7	0.3	3.2	3.0
288 (sham)	12	0	2.5	1.1	2.5	2.1
273 (control)	6	0				
272 (control)	12	0				

(+) posterior or superior. (−) anterior or inferior.

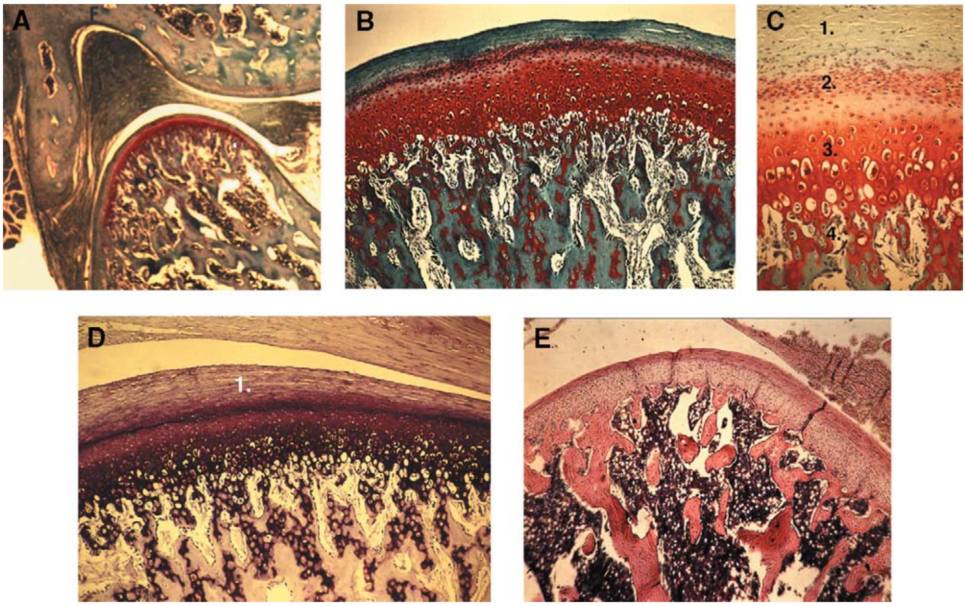

Fig 3. Photomicrographs of condyles from 2 control animals in pilot study. **A** and **B**, Central, decalcified section of condyle of juvenile control animal #273 stained with haematoxylin and eosin, originally photographed at 8× and 100× magnifications. **C**, Magnification (originally photographed at 225× shows *1*, fibrocartilage, *2*, prechondroblastic, and *3*, chondroblastic layers, and *4*, relatively flat region of endochondral bone formation under chondroblastic layer. **D**, Stained with toluidine blue at 100× magnification. **E**, Adolescent control comparison animal section stained with haematoxylin and eosin at 50× magnification.

Fig 4. Photomicrographic montages of central decalcified sections of temporomandibular joint, stained with haematoxylin and eosin and photographed under polarized light at 10× magnification. **A**, In 6-week experimental animal #299, *arrows* show forward (reverse) and downward growth of fossa during treatment. Below condylar chondroblastic layer (*B*), note increased multidirectional finger-like process of new endochondral bone formation compared with control. Experimental fossa shows significant bone formation. **B**, In 12-week experimental animal #271, *arrows* show forward (reverse) and downward growth of fossa during treatment. Note thickness of condylar chondroblastic layer is similar to that of controls due to rapid remodeling at 12 weeks, but glenoid fossa is distinctly different. **C**, In 12-week experimental animal #285, *arrows* show forward (reverse) and downward growth of fossa during treatment. Thickness of condylar chondroblastic layer is similar to controls due to remodeling, but glenoid fossa shows large increase in bone formation.

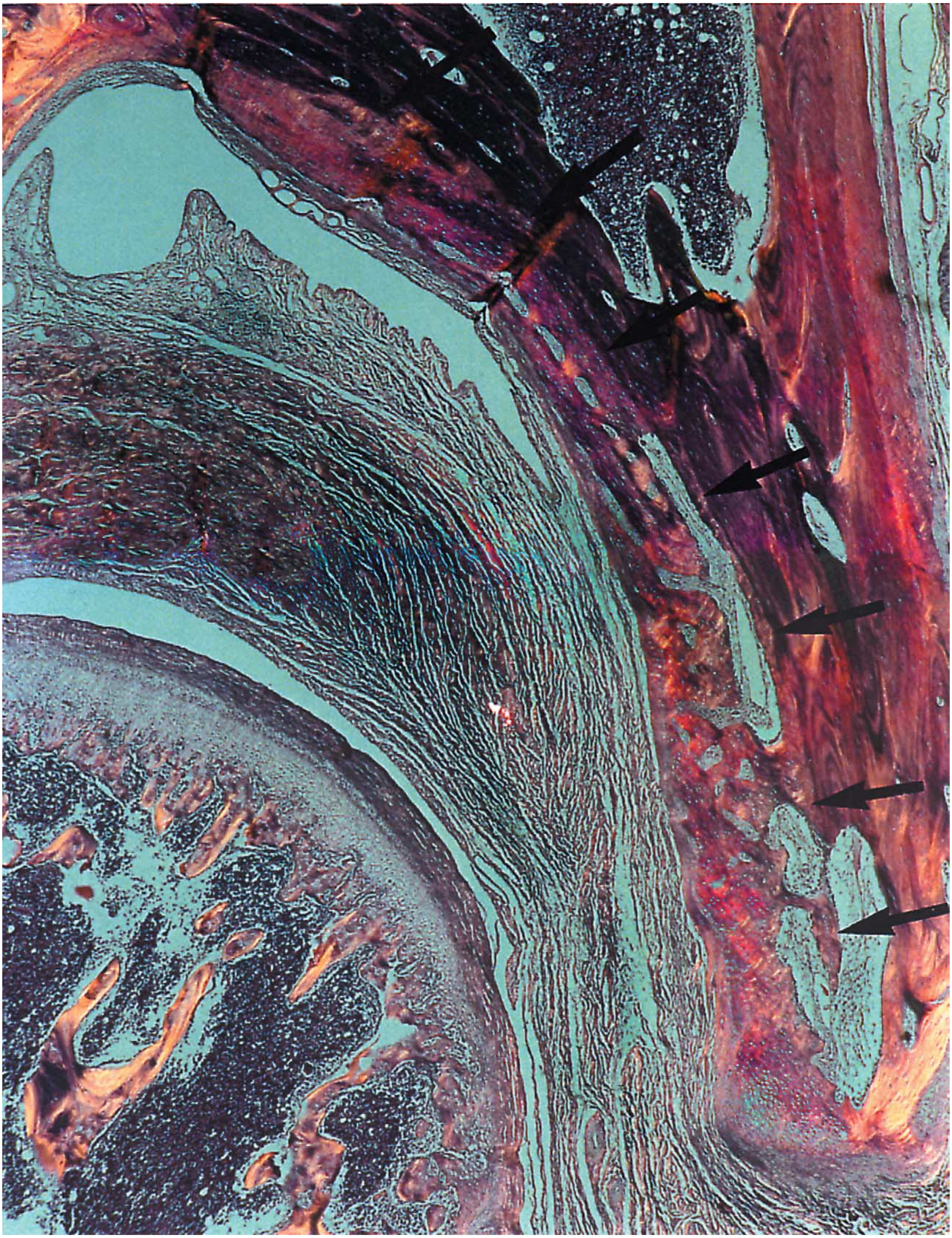

Fig 4, A.

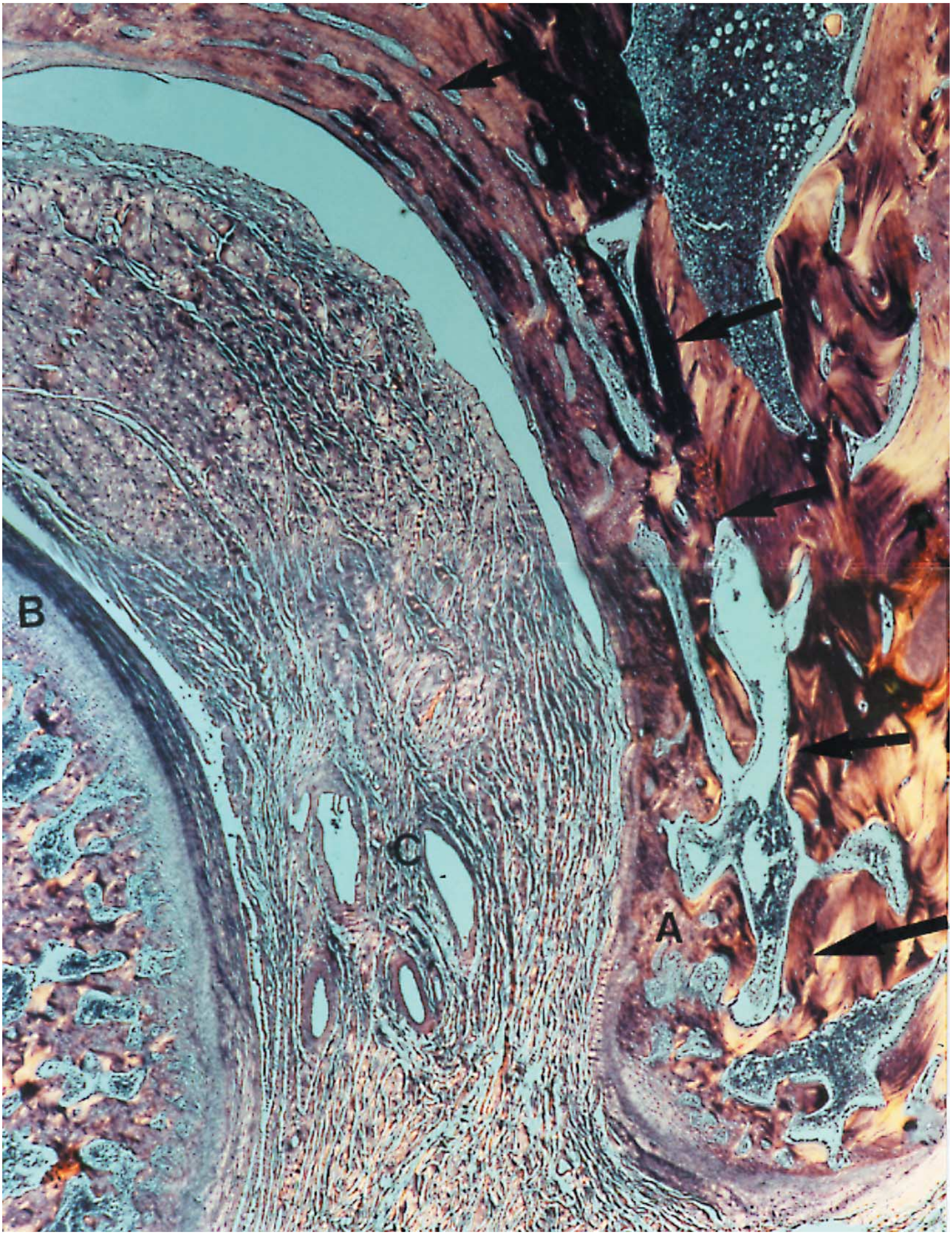

Fig 4, B.

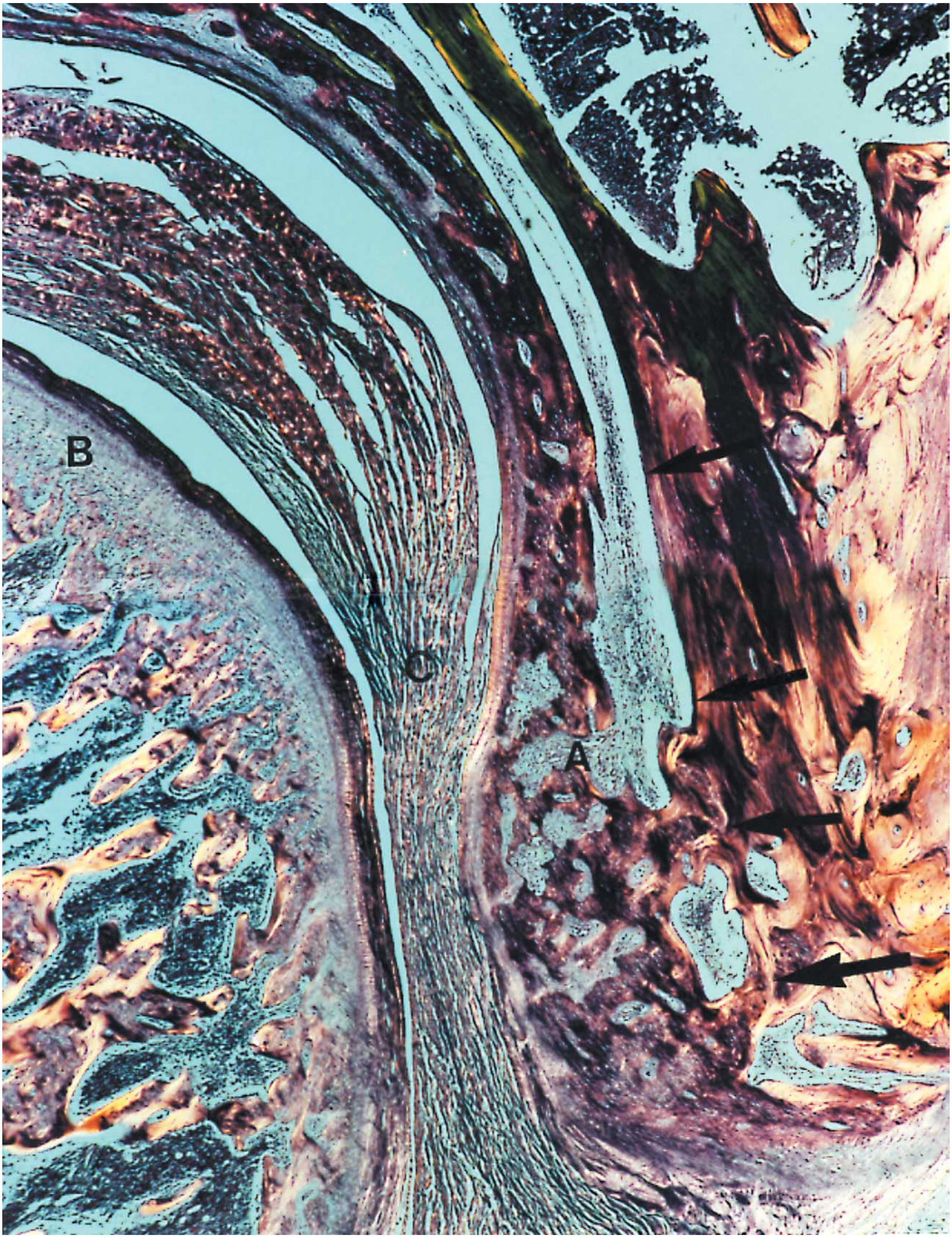

Fig 4, C.

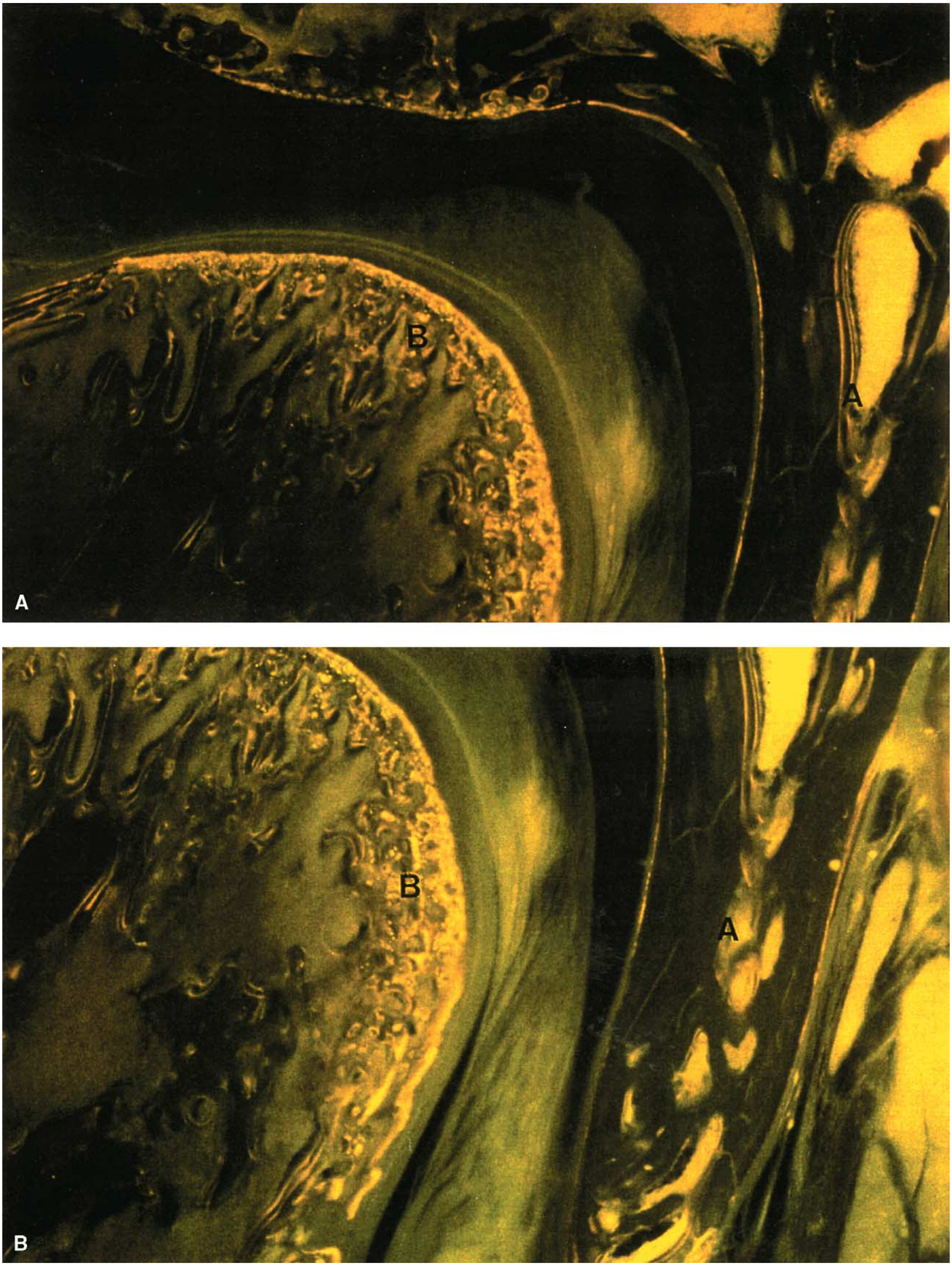

Fig 5, A-B.

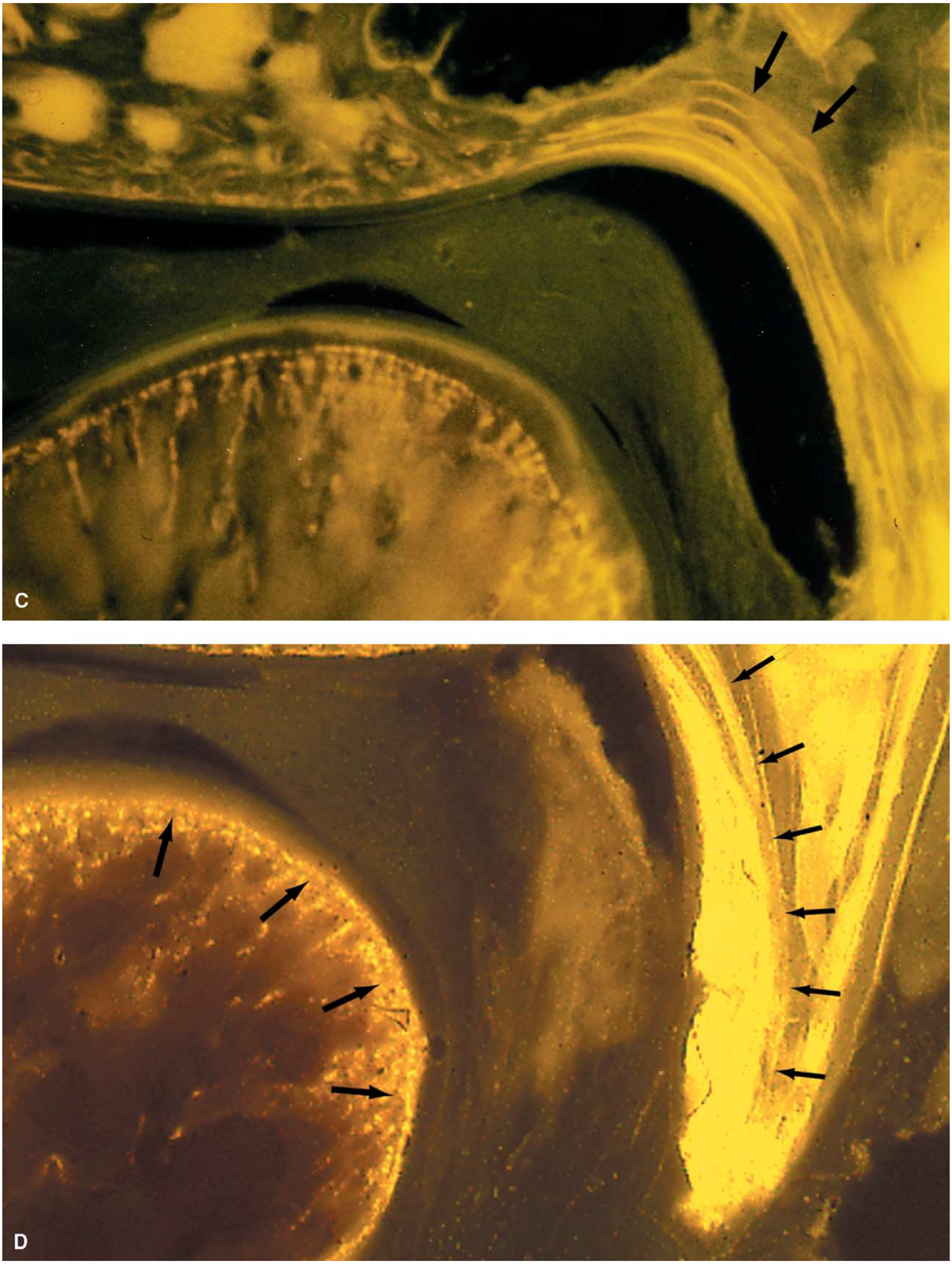

Fig 5, C-D.

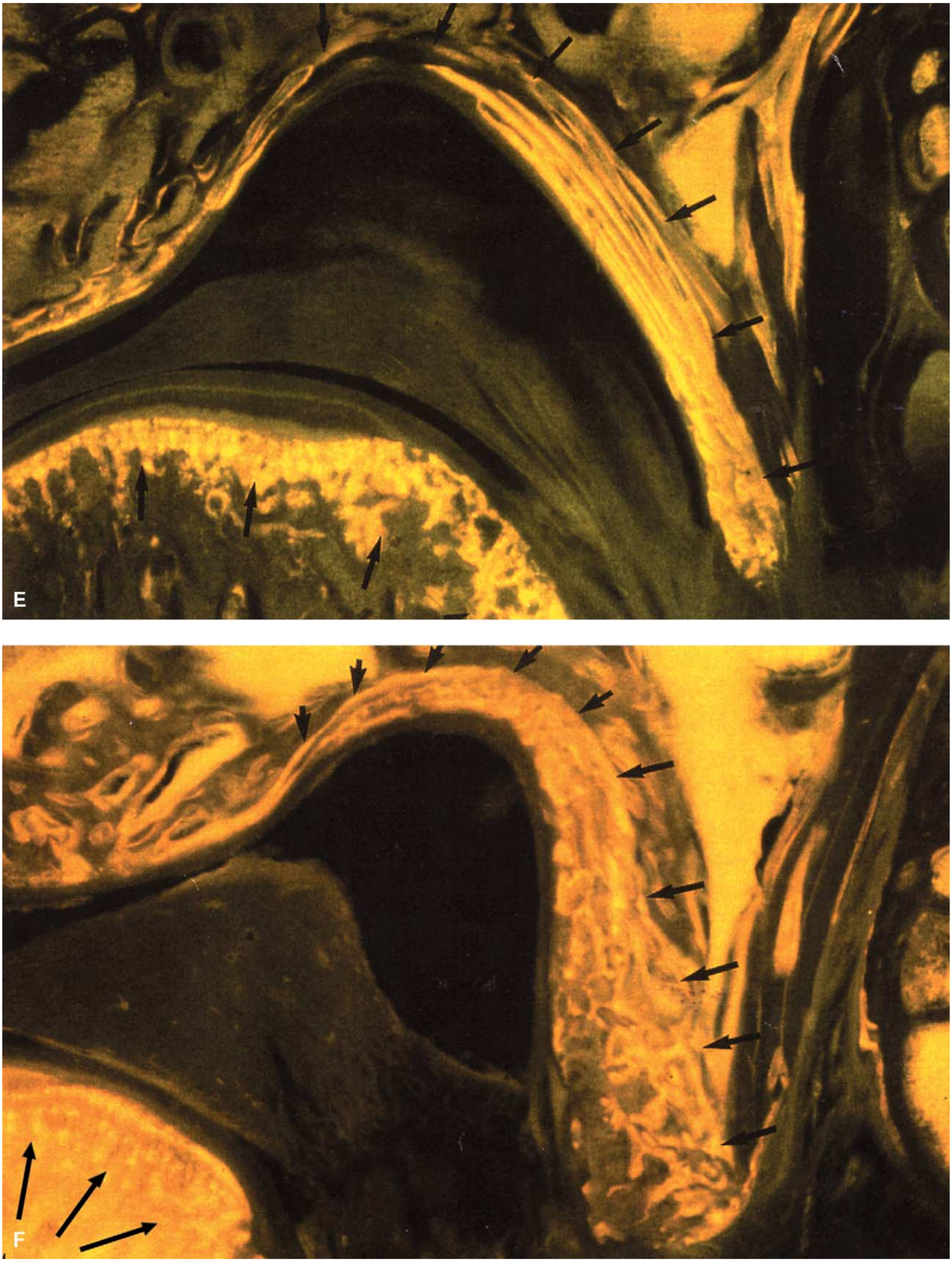

Fig 5, E-F

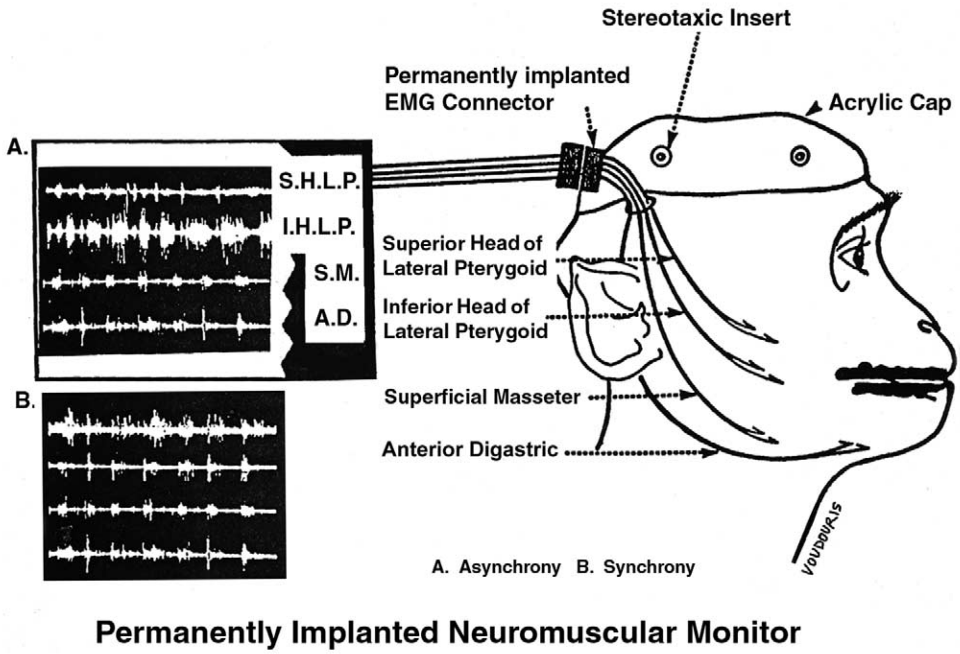

Fig 6. Permanently implanted EMG connector (black relay box) containing 12 electrodes, 3 for each of 4 muscles of mastication, connected to EMG laboratory equipment, including EMG monitor. Independent asynchronous and synchronous EMG muscle activities of superior and inferior heads of lateral pterygoid (*SHLP* and *IHLP*) muscles were recorded. Locations of surgically implanted superficial masseter (*SM*) and anterior digastric (*AD*) electrodes are also illustrated.

The control animals showed normal patterns of growth and remodeling of the glenoid fossa, with bone resorption along the anterior border of the postglenoid spine and deposition along the posterior border (Fig 5, *A* and *B*). This pattern was restricted and reversed in the experimental and sham control animals (Figs 4, *A*, and 5, *C*, *D*, *E*, *F*). There, both the decalcified and the undecalcified sections showed new bone formation on the anterior border of the postglenoid spine and bone resorption along the posterior border. In addition, the entire roof of the glenoid fossa showed new bone formation past the physical attachment of the retrodiskal tissues up to the height of the articular eminence (Figs 4, *B*, and 5, *C* and *E*). This was particularly evident with fluorescence microscopy with ultraviolet light to examine the undecalcified sections stained with tetracycline (Fig 5, *F*). This new bone was coarse, nonlamellar, and woven and appeared to remodel rapidly to form a more mature bone. The posterior part of the articular disk proliferated to fill the space created by the condylar displacement. This fibrous tissue contained numerous enlarged active fibroblasts and engorged blood vessels. The proliferated retrodiskal tissues

Fig 5. Photomicrographic montages of midsagittal and central undecalcified sections of temporomandibular joint with tetracycline vital staining and fluorescence microscopy, photographed with ultraviolet light at 3× magnification. **A** and **B**, Control animal #273 shows no increase in bone formation at anterior aspect of glenoid fossa. Letter *A* marks area where absence of yellow tetracycline staining means no new bone formed; letter *B* marks area of relatively low level of control endochondral bone formation at condyle. **C**, Sham control animal #288 (Herbst appliance bonded but not activated in forward direction for 12 weeks). Note new bone formation in glenoid fossa by simply opening vertical dimension with occlusal overlays. Condyle also shows signs of new bone formation. **D**, 6-week experimental animal #299. **E**, 12-week experimental animal #271 and, **F**, 18-week experimental animal. Note thickness of postglenoid spine area (doubled in thickness, multiple arrows in *F*) and bone formation in condyle (*yellow area*, marked by *3 arrows* in lower left). This was termed "lighting up" of condyle.

later appeared remodeled toward a pre-experimental morphology with experimental time (Fig 4, *A-C*).

The area of glenoid fossa bone formation increased (up to 3.6 mm^2) with longer experimental time (Fig 5, *E*). In the 18-week experimental animal, the post glenoid spine approximately doubled in thickness parallel to occlusal plane from the start to the end of the experiment. There was a statistically significant difference in the amount of new bone formed in the experimental subjects compared with controls ($P < .02$), even though there was considerable variation between subjects. There was a high correlation ($r = 0.95$) between the experimental time and both the area and maximum thickness of new bone formation.

It was evident from the morphometric analysis that the maximum thickness of new bone formed in the glenoid fossa contributed directly to the approximately 7-mm horizontal component of the super Class I malocclusion that was maintained (Fig 2, *E*). This new bone, when measured parallel to the occlusal plane, contributed between 6% and 32% (Fig 2, *E*) of the super Class I malocclusion (Fig 1). In addition, the horizontal condylar extension contributed from 1.10 to 3.23 mm, or 22% to 46% (Fig 2, *E*), to the super Class I malocclusion.

DISCUSSION

One of the most important findings of this experiment was that the statistically significant bone formation in the glenoid fossa and the increases in mandibular length were associated with decreased postural EMG activity in the masticatory muscles and not to lateral pterygoid muscle hyperactivity. This indicated that at least 2 other etiological factors might be responsible for the growth modification mechanism: the force of viscoelastic tissues and force transduction. The viscoelastic properties are associated with the stretched retrodiskal tissues, fibrous capsule, and sticky, hydrophylic synovial fluids communicating with the condyle and the glenoid fossa, described in the growth relativity hypothesis.[3] Second, bone formation found in the experimental animals up to the articular eminence, where there is no retrodiskal attachment, has also been linked to force transduction.[3] This transduction appears to be produced from the attachment of the retrodiskal tissues at the anterior aspect of the fossa. Electrophysiological signals generated over relatively long distances through osseous canaliculi might produce transduction.

The relocation of the glenoid fossa in an anterior and vertical direction with continuous functional appliance therapy was rapid and extensive in the 12-week experimental animals. The interim 6- and 18-week animals were selected to evaluate the progress of bone formation from the tetracycline stained sections, although individual responses were recognized.

The present study differs from previous studies of glenoid fossa remodeling[6-8] in nonhuman primates in 4 ways: First, the mandible was advanced continuously and progressively during the 6- to 18-week experimental periods. This design contrasted with other investigations that used intermittent mandibular protrusion and shorter experimental periods.[9-12] Second, we used computerized histomorphometric analysis to provide quantitative linear and area measurements of the amount of new bone formation in the glenoid fossa. Third, we statistically evaluated the histomorphometric findings. Fourth, the postural EMG activity[13,14] in the masticatory muscles studied was monitored serially by using permanently implanted EMG electrodes. Thus, quantitative assessment and statistical analysis of the changes in resting and functional EMG activity[14] associated with mandibular advancement were achieved (Fig 6).

It might be clinically significant that the anterior and inferior growth modification of the glenoid fossa occurred when growth of the fossa was in an inferior and posterior direction in the controls. This relative restriction of normal, backward fossa growth has been found in growing humans[15] and might be additive toward obtaining the super Class I malocclusion. This possible restrictive effect on fossa growth might not have been considered previously in orthopedic treatment. It is also possible that the forward growth modification of the glenoid fossa might continue with a further increase in treatment time due to the correlation between length of treatment and the degree of new bone growth ($r = 0.95$). Inspection of the decalcified sections showed large numbers of osteoblasts that covered the outer layer of new bone formation[1] providing further support for additional growth with longer treatment time. In the large number of serial sections in the controls, this anterior layer was covered with osteoclasts. We have also determined that without an adequate retention period to permit mineralization and the adaptation of muscle attachments, the positive condylar and glenoid fossa response is subject to some relapse. Muscle reattachment to new bony attachments on the inferior aspect of the chin has been demonstrated radiographically by Huang and Ross[16] during the retention of orthognathic surgery in children.

The cephalometric results showed that many factors, such as restriction of the maxilla, distal maxillary tooth movement, mesial mandibular tooth movement, and, in some animals, a more horizontal condylar growth direction, contributed to the conversion of a normal occlusion to a super Class I malocclusion. Overall, the combined Herbst dentofacial orthopedic contributions were consistently larger than the orthodontic contributions to the super Class I malocclusion. The 12-week sham control animal showed glenoid fossa remodeling (Fig 5, *C*), but the control animals did not (Fig 5, *A* and *B*). This observation suggests that the appliance thickness acted as a posterior occlusal bite block that distracted the condyle-disk complex vertically from the articular eminence and stretched the retrodiskal tissues. The bite-block additionally intruded the buccal segments, producing a small counterclockwise mandibular rotation over the 12-week period. The anterior mandibular autorotation in turn might have induced glenoid fossa growth modification by stretching the viscoelastic tissues.

Condylar response

Radiographic investigations superimposing on the metallic implants showed increases in condylar length in all juvenile experimental animals. Histological studies using undecalcified sections and tetracycline vital staining with fluorescence microscopy also confirmed the increased condylar response. In a previous study with adolescent animals (aged 36-48 months), there was no increase in the thickness of the prechondroblastic or chondroblastic zones at either 6 or 12 weeks after the start of the experiment.[14] In our study, which used younger animals, cartilage proliferation was not completely remodeled into bone at 12 weeks, but, at 18 weeks, there was calcification of the newly forming cartilage. The difference in condylar response in these 2 experiments confirmed the findings of others[17,18] that the condylar response appears to be age determined.[12,19,20]

Experiments in growing animals with intermittent forward positioning of the mandible have also demonstrated increased cellular activity at the prechondroblastic and chondroblastic zones of the condylar head,[21-26] although others showed little growth activity. They suggested that lateral pterygoid muscle activity was a necessary prerequisite for increased condylar growth.[2]

Longitudinal monitoring of the postural EMG activity of masticatory muscles with permanently implanted EMG electrodes was undertaken to determine whether the progressively activated Herbst appliances produced a change in EMG muscle activity. We found that appliance insertion and activation was associated with a decrease in postural EMG activity of the superior and inferior heads of the lateral pterygoid, superficial masseter, and anterior digastric muscles; the decrease in all but the anterior digastric muscle was statistically significant. This decreased postural EMG activity persisted for approximately 6 weeks, with a gradual return toward preappliance levels during a subsequent 6-week observation period (Fig 7). This return of EMG muscle activity did not reach the preappliance levels. Progressive mandibular advancement of 1.5 to 2 mm every 10 to 15 days did not prevent this decreased postural EMG activity. Similar results have also been found for functional activity such as swallowing in these muscles.[27] Ingervall and Bitsanis[28] obtained similar decreased muscle activity results to Auf der Maur[29] in humans. This was remarkable because condylar growth and the glenoid fossa growth response were related to the absence of increased postural activity in the masticatory muscles investigated[4] (Fig 7). This pointed to specific, nonmuscular soft tissues around the temporomandibular joint playing a direct or indirect role in the new bone formation described in the growth of the condyle relative to the glenoid fossa.[3]

This study also showed significant interanimal variability in condylar growth direction. This variability was important because it showed that changes in condylar growth direction are individual in monkeys, as in humans.

Clinical implications

Harvold et al[30] and Woodside et al[31] in human studies showed that intermittent condylar displacement achieved with activator use at night only did not produce clinically significant condylar growth. If human and nonhuman primate bone growth mechanisms are similar, our study suggests that the achievement of clinically useful posterior glenoid fossa restriction, anterior fossa remodeling, and increased mandibular growth might require a continuous anterior repositioning of the condyle-glenoid fossa relationship at least during the initial stages of treatment.

Comparison of our results in juvenile animals in the mixed dentition can be made with adolescent (and 1 adult) animals.[14] It suggests that the adaptive capacity of adolescent and adult monkeys, and possibly that of mature humans, might be limited chiefly to the glenoid fossa and the viscoelastic properties of the fibroelastic retrodiskal tissues with little potential for increased condylar length. This study has shown that juvenile animals in the mixed dentition appear to have the capacity to adapt significantly at both the glenoid fossa and the condyle. The work of Weislander[32] and Harvold et al[30] and Baume's[33] histological investigations of the temporomandibular joint support the view that these changes might indeed be possible in humans. The glenoid fossa results of headgear-Herbst treatment by Weislander[34] in treatment and retention might be larger when the restriction of the backward growth of the fossa in controls is considered. This restricted growth

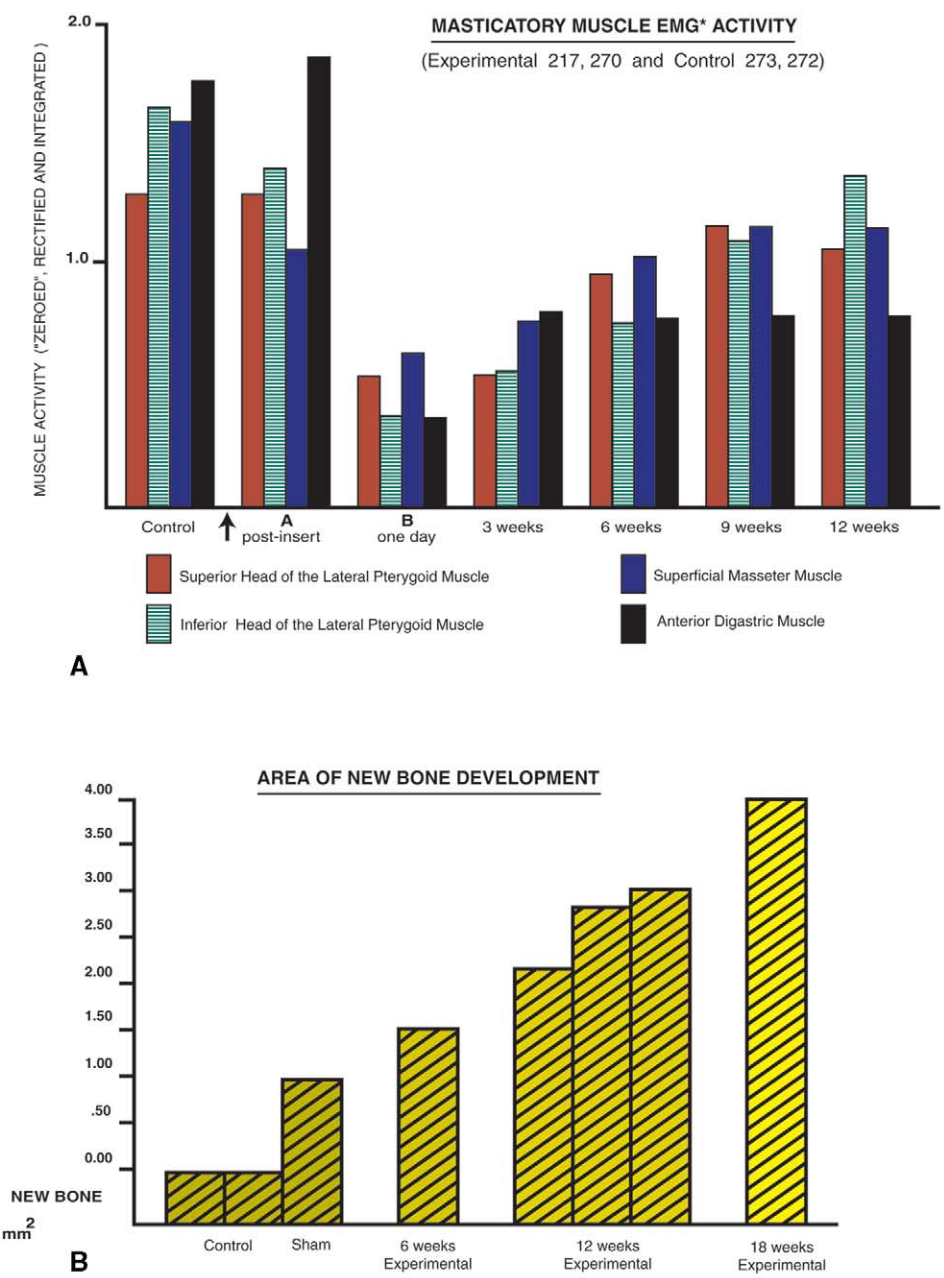

Fig 7. A, Postural EMG muscle activity; **B**, new bone formation. As muscle activity decreased, bone formation increased.

of the glenoid fossa is additive to the condylar changes and to maxillary growth restriction.

McNamara et al[35] further suggested that because of the approximately 3 times greater skeletal size differential between human and nonhuman primate subjects, 1.0 to 3.0 mm of mandibular growth in the animals might translate into even greater increases in humans. Similarly, the changes in the horizontal displacement of the glenoid fossa must be equally considered. Mills and McCulloch,[36] for example, have shown that up to 3 mm of condylar bone formation was maintained in retention in humans treated with the Twin-block appliance.

The findings of this study and several others in the literature review (see Part 1) suggest that functional appliances are not generally "functional" in reference to EMG muscle activity because lateral pterygoid muscle hyperactivity was not found as suggested in the past studies.[27-29] This decreased EMG muscle activity occurred during condylar bone formation while using a sophisticated, implanted EMG technique. It is suggested that the term *functional appliance* be associated with the function of viscoelastic tissue forces and the process of transduction of these forces for new bone formation. Functional appliances might be more appropriately considered to be *dentofacial orthopedic appliances*.

Herbst appliances appear to achieve skeletal results by stretching the bilaminar retrodiskal elastic band between the condyle and the glenoid fossa. In contrast, chronic compression of the condyle produces severe condylar resorption that can be prevented to some extent with occlusal coverage by distracting the condyle vertically. The condyle is negatively affected by the return of anterior digastric muscle function and by perimandibular connection tissue pull in retention, seating the condyle posteriorly into the fossa.[16] The occlusal bite-block addition to the Herbst appliance appeared to prevent severe condylar resorption or disk displacement in the experimental animals. To prevent mandibular incisor proclination with Herbst appliances, a contacting acrylic lip support (not a lip bumper) is recommended to provide a distal dental force. This is a topic for future study. This acrylic lip support uses lip pressure for anterior anchorage. It can be adapted closely to the mandibular bracket mechanisms and inserted into mandibular first molar tubes attached to a tied-back mandibular dental archwire.

The mechanism of growth modification is critical because a specific soft tissue mechanism might guide clinicians to plan appropriate future treatments. Treatment could some day include genetic therapies for condylar growth modification. These technologies could be directed to the soft or hard tissues. Consequently, this might lead to stable condyle-fossa growth modification in the long term that has so far been elusive. The general functional matrix theory was vague and largely unproven. These results have indicated a more specific mechanism from the connective tissues and the fluids in the growth relativity concept that uses more than muscle function alone to explain and achieve the clinical results of Herbst therapy.

CONCLUSIONS

1. Class I occlusions were converted to super Class I malocclusions in juvenile primates through a combination of factors, including anterior condylar growth modification and displacement, maxillary restriction, posterior maxillary dental changes, and anterior mandibular dental changes (increased condylar growth). These changes were combined with anterior and inferior remodeling of the glenoid fossa that was in a reverse direction to the normal posterior and inferior direction to the S-N plane.
2. In addition to the visible glenoid fossa change in an anterior direction, a second restrictive contribution was given serious consideration. The fossa has been shown in control humans to grow in a posterior direction, and eliminating this posterior growth might make a separate and cumulative contribution to the super Class I malocclusion. Consideration is needed for this relative, restrictive component of normal backward growth of the fossa.
3. Histomorphometric analysis showed that the increased amount and area of new bone in the glenoid fossa were statistically significant compared with the controls. This formation appeared to increase with time.
4. Increased condylar growth was demonstrated cephalometrically with the Björk implant method and confirmed histologically by both decalcified sections and undecalcified tetracycline vital staining with fluoresence microscopy in the experimental subjects.
5. The potential for condylar growth in juvenile nonhuman primates in the mixed dentition to induce increased mandibular length appears to be great. This is age related when the combined dentofacial orthopedic contributions of the Herbst were larger than the orthodontic (dental) contributions to the super Class I malocclusion in treated versus control subjects. This also supports the concept of functional appliance therapy in early treatment.
6. New bone formation at the condyle and the glenoid fossa was associated with decreased postural EMG activity in the superior and inferior heads of the lateral pterygoid, the masseter, and the anterior digastric muscles. There was a gradual return toward control levels without reaching them. These results support a nonmuscular etiology for condylar and glenoid fossa growth modification described in the growth relativity theory.
7. Fixed functional appliances (Herbst) produce consistent and reproducible condyle-fossa changes compared with the inconsistent results reported in the literature for removable functional appliances.

These results must be considered relative to treatment. A follow-up retention study[37] comparing these results has shown some maintenance of the condyle and glenoid fossa bone formation. The partial relapse is considered to be due to the return of muscle function—particularly of the anterior digastric muscles and the initial stretch of the perimandibular connective tissues that have a tendency to seat the condyle back toward the fossa; this is the topic of a future study.

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