Maxillary Growth Inhibition by Mucoperiosteal Denudation of Palatal Shelf Bone in Non-Cleft Beagles

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This report contains information about growth of canine non-cleft maxillae after experimental palatal surgery. Findings supplement those previously reported from this laboratory (*1, 2*), and relate to problems of timing and design of surgery for repair of cleft palates in humans. The previous studies in this series have focused on identification of growth inhibiting surgical variables and have involved utilization of an experimental model in which surgery has been performed on one side only of hard palates in normal weanling Beagles. Inferences based on the data from several surgical experiments have led to formulation of the hypothesis that, in this model, mucoperiosteal denudation of palatal shelf bone adjacent to deciduous molars is the single surgical variable responsible for the maxillary growth inhibitions observed.

Specifically, the purpose of this report is to present findings from a study designed as the critical test of that hypothesis. A surgical procedure involving *only* the variable of mucoperiosteal denudation of palatal shelf bone adjacent to molars was performed on a group of 8 Beagle pups (Surgical Group IV). Findings pertaining to postsurgical growth in palatal shelf breadth were then compared with the analogous previous findings from Surgical Group I on which a procedure involving a combination of three surgical variables had been performed. (Figure 1)

Methods

Eight 5½ week-old Beagle pups, 4 from each of two litters, were obtained from a commercial supplier of laboratory animals[1] and maintained

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[1] Animals for Research, Inc., Arlington, Virginia 22203.

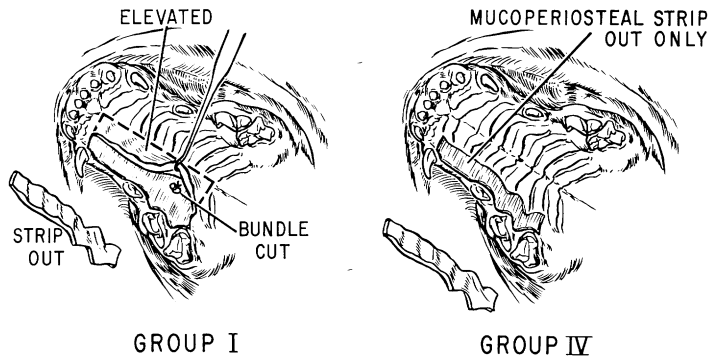

FIGURE 1. The surgical procedure for *Group I* involved three variables: elevation of mucoperiosteum to the midline, interruption of the major palatine neurovascular bundle, and removal of the strip of mucoperiosteum. The procedure for *Group IV* involved removal of the strip of mucoperiosteum only.

in the University of Iowa Animal Care Facility. Surgery on each pup's right hard palate was performed on the 46th postnatal day by a maxillofacial surgeon (WCH); anesthesia was provided by intravenous administration of a thiobarbiturate[2] and was supplemented by subperiosteal injection of a local anesthetic agent[3] in the operative area.

Design of the surgical procedures for both Surgical Groups I and IV is shown in Figure 1. Details of the Group I procedure have been described previously (*1*). In the Group IV procedure, an incision of the right palatal mucoperiosteum was made approximately 1 mm lingual to the teeth and extending from the third deciduous molar to the deciduous canine. A second parallel incision was made 4 mm medial to the first, and transverse incisions were made connecting the termini of the first two incisions. The mucoperiosteum within the incisions was elevated from the underlying bone and removed in one piece. The resulting surgical wound was identical to that produced in animals of Group I, except that there was no mobilization of mucoperiosteum medial to the area denuded, and there was no interruption of the right major palatine neurovasculature. Healing, as in Group I, was rapid and uneventful; care and maintenance procedures were as for the previous group.

Preoperative maxillary casts were made on all pups when they were 6 weeks old. After surgery on the 46th postnatal day additional casts were made on the 49th, 56th, and 63rd days. Casts were then made every other week from the 10th through the 22nd week. Details of the impression making and casting technique were the same as described previously for Group I (*2*).

Data Collection and Analysis: Attention in this report concentrates on

[2] Thiamylal Sodium 2.5%. Parke, Davis and Co., Detroit, Michigan.

[3] Lidocaine HCl 0.5% with epinephrine 1:100,000. Astra Pharmaceutical Products, Inc., Worcester, Massachusetts.

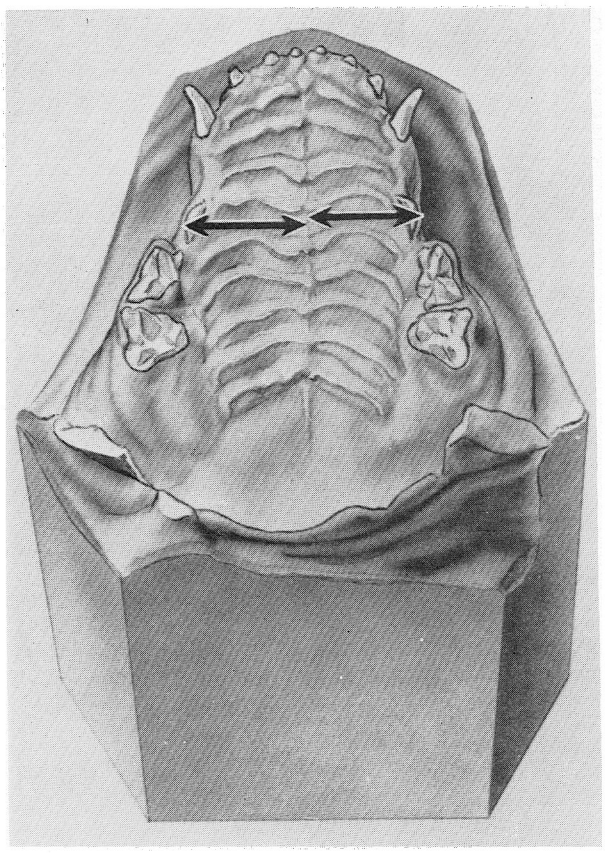

FIGURE 2. Maxillary cast from a 10 week old beagle. The dimension measured on left and right sides was the *length of the shortest straight line* between (a) the most lateral point in the buccal gingival crevice of the DM1 (deciduous first molar), and (b) the center of the median raphe. Note that, since the median raphe landmark was defined by the measurement method, it was not necessarily at the same point for adjacent left and right breadth determinations on a given cast.

palatal shelf breadth at the deciduous first molar (DM1) level only, since in the previous studies (*1*, *2*) this is the region of the maxillae where the most severe postsurgical growth inhibition has been observed. Thus, in testing the hypothesis in question, palatal shelf breadth at the DM1 level has been considered the critical test dimension.

All new data presented here were derived from measurements made on the 11 serial maxillary casts made on each of the 8 Group IV dogs (Figure 2). Analogous previously reported Control Group and Surgical Group I data are also shown for comparison (Tables 1 and 2). Data acquisition and manipulation procedures were identical with those described previously (*2*).

Testing of the hypothesis in question required only simple analytic

TABLE 1. Absolute breadths of maxillary left and right palatal shelves in mm for the 7 Beagles in Surgical Group I and 8 in Surgical Group IV. Measurements from the deciduous first molar to the median raphe were made on maxillary casts obtained on exact weekly birth anniversaries.

Age (wks)	*Surgical Group I (N = 7)*				*Surgical Group IV (N = 8)*			
	Left		*Right*		*Left*		*Right*	
	$\bar{x} \pm SE_{\bar{x}}$	*Range*	$\bar{x} \pm SE_{\bar{x}}$	*Range*	$\bar{x} \pm SE_{\bar{x}}$	*Range*	$\bar{x} \pm SE_{\bar{x}}$	*Range*
6	9.0 ± 0.1	8.3–9.4	9.0 ± 0.1	8.7–9.4	8.8 ± 0.3	7.9–10.3	8.7 ± 0.3	7.7–10.0
7	9.5 ± 0.2	8.9–10.2	9.4 ± 0.1	9.0–10.0	9.4 ± 0.3	8.4–11.2	8.9 ± 0.3	7.6–10.5
8	10.4 ± 0.2	9.7–11.2	8.8 ± 0.2	7.9–9.5	10.0 ± 0.3	9.2–11.5	8.2 ± 0.4	6.9–10.0
9	—	—	—	—	10.4 ± 0.2	9.7–11.9	7.8 ± 0.4	6.1–10.1
10	11.3 ± 0.2	10.5–11.7	9.1 ± 0.2	8.3–9.8	10.8 ± 0.2	9.9–12.2	7.9 ± 0.5	6.1–10.3
12	12.1 ± 0.1	11.4–12.5	9.7 ± 0.2	8.9–10.4	11.3 ± 0.3	10.4–12.8	8.4 ± 0.6	6.5–11.3
14	12.8 ± 0.1	12.3–13.4	10.4 ± 0.2	9.7–11.1	11.9 ± 0.2	11.2–13.3	8.8 ± 0.6	6.7–12.0
16	13.6 ± 0.1	13.2–14.0	11.0 ± 0.2	10.2–11.5	12.5 ± 0.2	11.8–13.7	9.5 ± 0.5	7.2–12.5
18	14.0 ± 0.1	13.7–14.3	11.4 ± 0.2	10.6–12.1	12.9 ± 0.3	12.1–14.1	10.0 ± 0.6	7.5–13.3
20	14.3 ± 0.1	14.0–14.5	11.7 ± 0.2	11.0–12.4	13.3 ± 0.3	12.2–14.6	10.5 ± 0.4	8.3–13.5
22	14.3 ± 0.1	12.8–14.6	11.5 ± 0.2	11.0–12.8	13.7 ± 0.3	12.5–14.9	10.7 ± 0.5	8.5–13.5

TABLE 2. Relative asymmetries in breadth of maxillary left and right palatal shelves calculated from group means in Table 1. [Absolute data for the Control Group appears elsewhere (2).] Percentage asymmetry at each age was calculated as 100 times the difference between breadth of wide side and narrow side divided by breadth of the wide side. When right breadth was less than left, the quotient was read as the percent right sides were narrower than left. When left breadth was less than right, the quotient was assigned a negative value, and was read as the percent left sides were narrower than right.

Age (Wks.)	*Control Group (N = 6)*	*Surgical Group I (N = 7)*	*Surgical Group IV (N = 8)*
6	1.8%	−0.1%	0.1%
7	0.8	1.9	4.5
8	1.6	15.5	17.7
9	—	—	24.8
10	2.3	19.4	26.8
12	2.2	19.9	26.1
14	1.0	19.4	25.8
16	1.2	19.5	24.0
18	1.6	18.5	22.5
20	1.9	17.9	21.0
22	0.9	19.5	21.9

procedures. Plotted absolute and relative asymmetry data were compared by inspection, and a t-test was perfomed to determine if there was a statistically significant intergroup difference in shelf breadth asymmetry at the DM1 level at 22 weeks.

All animals were sacrificed after the 22nd week since earlier findings

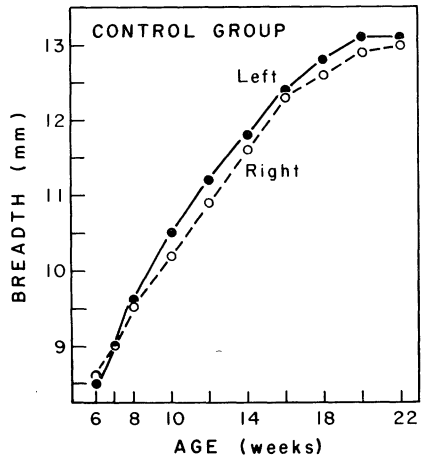

FIGURE 3. Growth curves for right and left palatal shelf breadth at the DM1 level in the 6 beagle pups of *Control Group I*. Mean left and right breadth was approximately symmetrical at each of the 10 ages studied.

had indicated that the probability of important changes in asymmetry after that age was minimal (*2*).

Findings

Longitudinal data from the Group IV cast measurements of DM1 level palatal shelf breadth are displayed in Table 1. Also appearing in that table, for comparison, are the analogous previously reported data for Group I. Plots of these data plus that of the corresponding Control Group data appear as Figures 3, 4 & 5. Data on proportional asymmetry in shelf breadth were derived from the grouped absolute data at each age and are shown in Table 2 and Figure 6.

The findings regarding Group IV shelf growth at the DM1 level may be summarized as follows: (a) Left and right shelf breadths were approximately symmetrical at 6 weeks. (b) At 8 weeks, 10 days after surgery, right (surgical side) shelves averaged 18% narrower than left. Average absolute increase in left shelf breadth from 6 to 8 weeks was 1.2 mm, while right shelves in the same period showed an average absolute decrease in breadth of 0.5 mm. (c) Right shelf breadth continued to decrease through the 9th postnatal week, then increased at roughly the same rate as left shelf breadth through the 20th week. (d) Maximum asymmetry (26.8%) was at 10 weeks.

Comparison of the Group IV findings with those from Group I revealed that (a) The postsurgical decreased in right shelf breadth which had been seen in Group I was also seen in Group IV, but to a greater degree. It should be noted, however, that casts were not made at 9 weeks for Group I as they were for Group IV, and that the low point in the Group I breadth curve may have gone unobserved. (b) The overall form of the left shelf

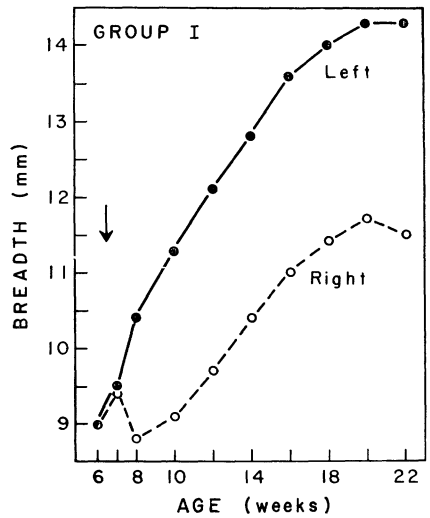

FIGURE 4. Growth curves for right and left palatal shelf breadth at the DM1 level in the 7 beagle pups of *Surgical Group I*. Unilateral (right side) surgery was performed on postnatal day 46 (arrow). Mean left and right shelf breadth was significantly asymmetrical at all ages after the 7th week. The initial postsurgical decrease in right shelf breadth may have been the result of wound healing contraction and related movement of the measurement landmarks.

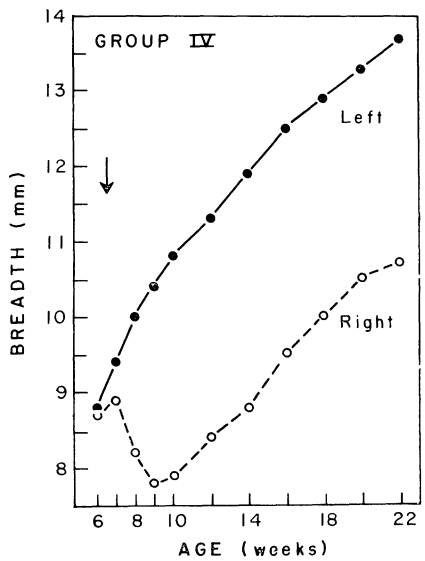

FIGURE 5. Growth curves for right and left palatal shelf breadth at the DM1 level in the 8 beagle pups of *Surgical Group IV*. Unilateral (right side) surgery was performed on postnatal day 46 (arrow). Mean left and right shelf breadth was significantly asymmetrical at all ages after the 7th week. The initial postsurgical decrease in right shelf breadth was more extreme than in *Group I*.

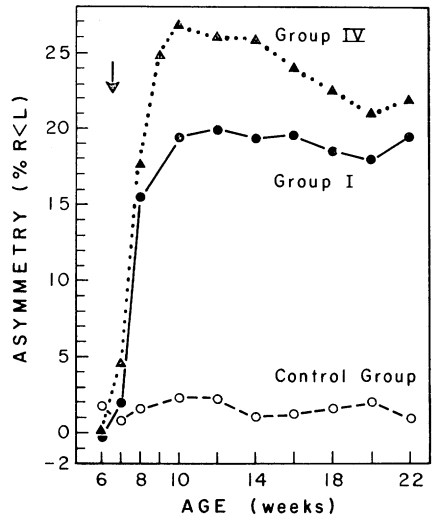

FIGURE 6. Right-left DM1 level palatal shelf asymmetry as plotted against postnatal age for *Control Group, Surgical Group I,* and *Surgical Group IV* pups. The arrow indicates age at surgery for *Groups I and IV*. See Table 2 for the percentage asymmetry calculation method.

breadth curves was similar in both Surgical Groups I and IV and in the Control Group; forms of right shelf breadth curves in Groups I and IV were similar, but were in distinct contrast to that for the Control Group. (c) Mean presurgical left shelf breadth was 0.2 mm less in Group IV than in Group I, and by 22 weeks the intergroup difference had increased to 0.6 mm. The corresponding statistics for right shelf breadth were 0.3 mm and 0.8 mm. (d) Mean asymmetry at 22 weeks was slightly, but insignificantly ($p < 0.01$) greater in Group IV than in Group I.

Conclusion: Accept the hypothesis that, in this model, mucoperiosteal denudation of palatal shelf bone adjacent to deciduous molars is the single surgical variable responsible for the maxillary growth inhibition seen. Acceptance of this hypothesis requires the rejection of alternate hypotheses regarding the growth influences of neurovascular interruption and/or mucoperiosteal mobilization.

Discussion

This series of experiments was begun as a replication and extension of work reported by Herfert (*3, 4, 5, 6*).[4] The surgical procedure used in our Group I was the same as that used by Herfert, and our growth findings were similar to those reported by him. Herfert interpreted his findings as a demonstration of the importance of uninterrupted blood supply for normal postsurgical maxillary growth after cleft palate repair. Findings

[4] We have recently learned that similar research was begun at about the same time in Japan by Moriya (6).

from our Surgical Group III (*1*), in which blood supply was interrupted only, suggested that Herfert's interpretation of his findings was erroneous, and that mucoperiosteal denudation of shelf bone next to teeth may have been the damaging surgical variable in his experiments as well as in ours. The present study was designed as a direct test of that hypothesis, and the results now indicate that there is high probability that the hypothesis is correct.

While this finding may have implications for the design of surgical repairs for cleft deformities in children, we are reluctant to overinterpret our findings at this time. While we appear to have established, within the bounds of the present experimental model, that shelf denudation adjacent to deciduous molars results in serious disturbances in the normal processes of maxillary growth remodeling, our understanding of the basic mechanisms of the interference is superficial. More must be known before firm recommendations regarding physical management in children can be made intelligently. Illustrative of the general state of present knowledge is the fact that there is still profound uncertainty about the nature of the control systems which regulate *normal* growth in the facial skeleton.

It may be noted, in this regard, that postsurgical growth disturbances in rapidly growing animals (such as dogs) may be exaggerations of those which might be seen in more slowly growing animals (such as humans). While we know of no data relating growth rates of organs (e.g., jaws), with wound healing rates, it is our assumption that one of the advantages of experimental systems such as that used here is that phenomena which may be too subtle to study economically in humans or slow growing primates may be studied with considerable economy in rapidly maturing species.

A comment is also in order on the use of non-cleft animals in studies related to problems of cleft palate rehabilitation. Our choice of non-cleft dogs in this series of experiments has been deliberate. The rationale again involves economy of time and other resources in the search for explicit answers to basic questions. There are important growth experiments which will require the use of animals having cleft deformities. We have reasoned that the nature of the question or problem at hand should be the prime determinant of the experimental animal to be used. The questions to which we have addressed ourselves thus far have not required the use of animals having clefts.

The results of this study confirm our impression that the onset of shelf asymmetry in this model is a relatively early postsurgical phenomenon. As was noted previously, however (*2*), the apparent decrease in right shelf breadth immediately after surgery is probably the result of a moving together of the measurement landmarks only (DM1 and the median raphe), and not a real decrease in skeletal breadth of the right palatal process[5]. Right skeletal breadth (i.e., as would be measured to the inter-

[5] See (2) for a more complete discussion.

maxillary suture instead of the soft tissue median raphe) probably stays relatively unchanged in the early postsurgical weeks.

Inspection of the growth curves (Figures 4, 5) suggests that the postsurgical processes operating in Group I also operated in Group IV but to an exaggerated degree. One possible explanation of the exaggerated immediate postsugical asymmetry seen in Group IV is that the Group IV pups may have been relatively less mature than the Group I pups at the time of surgery (even though they were the same postnatal age). This interpretation is consistent with the observation that mean shelf breadth in Group IV was slightly less, both at 6 and 22 weeks, than in Group I, and that the Group IV breadth curves (Figure 5) were still ascending at 22 weeks while those for Group I (Figure 4) were leveling off or declining preparatory to emergence of the permanent successors of the DM1s.

Summary

An animal study was performed to test a hypothesis about growth effects of some individual surgical variables which are often components of palatal cleft repair procedures for humans. Normal (noncleft) weanling Beagle pups were used in an experimental model involving performance of surgery on one side of hard palates only on the 46th postnatal day. Previous findings (*1, 2, 3*) had shown that, in this model, maxillary growth in breadth showed marked inhibition after unilateral surgery involving blood supply interruption, mucoperiosteal flap mobilization, and mucoperiosteal denudation of shelf bone adjacent to deciduous teeth was performed. Variants of this procedure suggested that the critical variable responsible for the growth inhibition was denudation of the shelf bone, and not vascular interference as had been previously claimed (*3, 4, 5*).

The present study was designed to test the hypothesis that, in this experimental model, mucoperiosteal denudation of palatal shelf bone adjacent to deciduous molars is the single surgical variable responsible for the maxillary growth inhibitions seen. The findings have dictated acceptance of the hypothesis. Understanding of the growth inhibitory mechanisms involved is incomplete.

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