Growth of Maxillae in Dogs after Palatal Surgery: I

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This report presents findings from a study of effects of palatal surgery on maxillary bone growth in dogs. Three different surgical procedures were performed, one on each of three groups of $6\frac{1}{2}$ -week-old normal Beagle pups. The study is a replication and extension of research reported in 1954 and 1956 by Herfert (5, 7). Both this study and those studies of Herfert involved surgical manipulation of mucoperiosteum and vasculature on one side only of pups' hard palates; both were designed to yield basic information about effects of specific surgical variables on skeletal growth in the face. The present report (Part I) includes findings from maxillary symmetry analyses of the mature cleaned skulls of 21 experimental and six control dogs. Specifically, information is presented about skeletal symmetry in three dimensions: maxillary width, maxillary height, and maxillary length.

Part II (to be reported later) will contain findings from analyses of longitudinal growth records collected on the same animals and a full comparison of the findings from the study with those reported by Herfert.

Perspective

While it is now generally agreed that the primary objective of surgery for cleft palate is to provide for the establishment of good speech, allowance for the best possible facial development through minimization of growth interference is also recognized as an important consideration. Significant improvements in the management of patients with cleft lip and palate have been made since the facial growth reports of Graber and others, in the late 1940s and early 1950s (3, 4, 12). These reports served to indict the more traumatic variables in the repair techniques of Brophy and others. Reports appearing in recent years suggest that more favorable facial growth results are now being obtained through use of improved

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surgical techniques (1, 2). Horowitz and Hixon have recently presented an excellent historical summary of trends in the management of patients having clefts (8).

Surgeons continue to strive for improvement however; a 1962 poll by Lewin showed that many surgeons still believed that midfacial growth might be adversely affected by surgical trauma incident to currentlyused palatal repair techniques (9). The improvement of cleft palate surgical technology necessitates the identification of specific surgical variables which may be responsible for interference with growth. Although the most appropriate method of approaching this problem is through laboratory research, only a few pertinent animal studies have been reported. One, a frequently cited study by Sarnat, published in 1958, suggested that there was little to be concerned about (11). Sarnat performed experimental palatal surgical procedures on one side of hard palates of seven young monkeys, removing large areas of bone and/or mucoperiosteum and ligating the 'descending palatine artery' on the side of operation. Sarnat observed that: 'The findings in this experiment do not substantiate the thesis that decrease in vascularity to the palate or injury to the sutural growth sites will affect palatal or facial growth.'

Also appearing in 1958 was an account, in English, of Herfert's two earlier reports which had appeared in the German literature (6). Herfert's findings from experimental palatal surgery on one side of palates of seven young normal dogs were in distinct contrast to those reported by Sarnat. His surgery was less extensive than that of Sarnat, involving primarily the elevation and immediate repositioning of mucoperiosteum on one side of the palate. One procedure also involved interruption of the 'posterior palatine artery' and removal of a strip of palatal mucoperiosteum, 4 mm in width, adjacent to the posterior teeth. Herfert reported that the palatal shelves which had been operated were up to 23%narrower than the shelves on the opposite unoperated side, and stated that '... limitation of growth does in all cases appear to have taken place....'

The differences between the Sarnat and Herfert findings are of some importance and deserve comment here as perspective for the present report. The Sarnat and Herfert studies were similar in design and objective; both involved use of normal (noncleft) animals, both involved performance of surgical procedures on one side only, and in both, subsequent growth in operated and unoperated sides was compared. Each investigator hoped to gain basic information from his study; neither considered his experiment a direct test of any specific cleft palate surgical technique. Herfert's aim was '... to see whether by raising the periosteum from the palate the growth of bone would be restricted.' Sarnat noted that his findings were meant only as a '... contribution to the general subject of the effect of decreased circulation and trauma upon facial growth....'

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Although both the Herfert and the Sarnat studies have faults, the latter study seems most vulnerable to serious criticism. Sarnat's statement that his findings suggest that '... surgical trauma incident to the raising of mucoperiosteal palatal flaps is not the cause of lack of maxillary and facial growth ...' loses much authority when scrutiny of his Table 1 and Figure 2 reveals that the average postoperative survival time of his monkeys was less than 14 months (ranging from 1 to 34 months) and that only two of his seven animals had matured as far as the stage of eruption of maxillary permanent incisors at the time of death. A basic requirement of experimental growth research is that animals used in an experiment be allowed reasonable time in which to mature before concluding that treatments performed have no effect on growth. Sarnat does not report the cause of death of any of the monkeys. In addition, the conclusion that there were 'no significant gross differences' between facial growth on left and right sides in surgical group animals and between surgical and control group animals was apparently reached only by subjective judgment of the author; no measurements were reported.

Herfert's design did not provide for an adequate number of control animals, and, as in Sarnat's work, no tests of significance of the sample findings were performed. It is worthy of note that Herfert did not sacrifice his dogs until a time then it was reasonable to assume that most of the ultimate skeletal growth had already occurred (well after eruption of all permanent teeth). He then performed measurements on the cleaned skulls and stated conclusions based on those objective findings.

The decision of the present authors to repeat the Herfert experiments was prompted by the apparent contradictions between the Sarnat and Herfert findings. In an area where little experimental work has been done, findings from studies having serious inadequacies of design and method tend to take on unwarranted authority. It seemed prudent, therefore, in initiating a program of growth research, to begin by attending to some of the residue of unsettled issues remaining from previous efforts.

In repeating the Herfert work, it was decided to revise the original design and method in such a way as to obtain maximum yield of information while still providing findings comparable to those of the original study. To that end, the number of animals having palatal surgery was increased, a control group was added, a surgical group having a procedure not performed by Herfert was included, and longitudinal growth records (maxillary casts) were maintained. These additions to Herfert's original plan of study enabled the present authors to perform tests for significance of the sample growth findings, derive information on the effect of an individual variable within one of the original Herfert procedures, obtain data on the timing of growth effects



FIGURE 1. Palatal aspect of maxillae of puppies in their 6th postnatal week. Flap designs for animals in each of the three surgical groups are depicted. Striped areas denote elevation of mucoperiosteum; the darkened area in the Group I diagram indicates the strip of tissue removed from the flap edge. Depiction of the major palatine foramen in the Group I and III diagrams indicates that the neurovascular bundle was severed in those operations. Crowns of second and third molars are still partly covered by gingiva at this age. The diagrams are approximately actual size.

occurring during the period of study, and finally, identify the age by which most skeletal growth in the maxilla had occurred.

Methods

Five litters of 51/2-week-old purebred Beagle pups were obtained from a commercial supplier of laboratory animals¹ and maintained in the University of Iowa Animal House facilities. Surgical procedures were performed one week later on the pups' 46th postnatal day. Of the 27 dogs used, six were controls having no surgery, and 21 had one of three procedures performed on the right sides of their palates. (Comparison of serial maxillary casts for the control group shows that, at 61/2 weeks, maxillae were approximately 2/3 their adult width and 1/2 their adult length.) Surgery was performed by a maxillofacial surgeon (WCH); anesthesia for all dogs was provided by intravenous injection of a thiobarbiturate² and was supplemented in dogs having surgery by subperiosteal infiltration of a local anesthetic agent³ in the operative area.

Diagrams of the three surgical procedures appear in Figure 1. The procedures were as follows.

SURGICAL GROUP I. An incision of the palatal mucoperiosteum was made approximately 1 mm lingual to the teeth and extending from the third deciduous molar to the deciduous canine. The mucoperiosteum

¹ Animals for Research Inc., 1305 N. 20th St., Arlington, Va. ² Thiamylal Sodium 2.5%. Parke, Davis and Co., Detroit, Mich.

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

medial to the incision was elevated away from the underlying bone, the major palatine neurovascular bundle (10) was ligated and severed at its foramen, and a 4 mm wide strip of the elevated tissue was removed from the flap edge. The remaining elevated soft tissue was repositioned in its original location, leaving an area of exposed bone 4 mm wide by about 25 mm long. The operative design is that used by Herfert in one group of his animals; he reasoned that it was grossly analogous to the surgical technique employed in a cleft repair when a flap is elevated and swung medially to bridge the cleft.⁴

SURGICAL GROUP II. In this procedure, also after Herfert, three incisions were made: the first parallel to the teeth as in Group I, the second extending medially to the midline from the anterior terminus of the first, and the third extending back along the midline and terminating at the level of the distal margins of the third deciduous molars. This posteriorly based mucoperiosteal flap was elevated from the underlying bone, then immediately repositioned and sutured in place. The major palatine neurovascular bundle was *not* interrupted in this procedure nor was any bone left denuded by removal of a strip of mucoperiosteum. Herfert stated that the purpose of this design was to determine if there was a difference in growth effect when vasculature was interrupted and when it was not.

SURGICAL GROUP III. In this procedure, unique to the present study, only the major palatine neurovascular bundle was interrupted. A single short incision was made to facilitate exposure, ligation, and severance of the bundle. This procedure was performed to help determine whether growth effects which might be observed in the Group I animals were primarily the result of the flap elevation and bone denudation or of the neurovascular interruption.

Postoperative healing was rapid and uneventful for all 21 dogs in the three surgical groups. Subsequent environmental conditions were the same for all animals. The magnitude of successive palatal growth increments was determined by comparing measurements on the maxillary cast made each month with those of previous months. Increments after the 64th postnatal week were very small and the experiment was terminated after the 68th week. All soft tissue was removed from the heads, and measurements were performed on the cleaned dry skulls.⁵

Measurement Technique

Figures 2, 3 and 4 illustrate the three linear dimensions measured on each maxillary bone. *Maxillary width* was taken as the breadth of the

⁴ A communication from Professor Herfert in 1964 has confirmed that while his reports mention interruption of the artery only, he actually interrupted the entire neurovascular bundle. That procedure was thus followed in Group I of the present experiment.

experiment. ⁵ Two of the dogs were not sacrificed at termination of the experiment. On these animals, one each from Groups I and II, measurements corresponding to those made on the dried skulls were made by surgically exposing appropriate osseous landmarks.

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FIGURE 2. Palatal aspect of mature maxillae showing the dimensions measured for right and left maxillary width.

palatal process of each maxilla at the level of the second premolar tooth (PM2). It was measured as the shortest straight line distance between a) the palatal extremity of the medial wall of the alveolus of the distal root of the PM2 and b) the median palatine suture. Maxillary height was taken as the height of the facial surface of each maxilla. It was measured as the straight line distance between a) the crest of the interradicular septum between the PM2 alveoli and b) the point of junction of the nasomaxillary and incisivomaxillary sutures. Maxillary length was taken as the depth of the palatal process of the maxilla and was obtained on each maxilla as the arithmetic mean of the straight line distances between a) the most posterior point on the palatal process and b) points A, B, C, and D on the incisivomaxillary suture. Point A was defined as the incisivomaxillary suture as its junction with the medioposterior lip of the palatine fissure; point B as the incisivomax-



FIGURE 3. Arrows denote the four dimensions measured on each maxilla for maxillary length. The average of the four measures on each side was then obtained for use in the symmetry analyses.

illary suture at its junction with the lateral lip of the palatine fissure; point C as the most posterior point on the incisivomaxillary suture; and point D as the incisivomaxillary suture at its junction with the medial lip of the alveolus of the permanent canine tooth.

All measurements were made with sliding calipers having vernier scales and read to the nearest 0.1 mm. The caliper used for maxillary height and width measurements had both arms sharply pointed; the caliper for maxillary length measurements was modified to provide a point on the fixed arm and a face surface (for contact against the most posterior maxillary point) on the sliding arm.

A total of 162 linear measurements was obtained for use in the



 $\rm FIGURE$ 4. Facial aspect of a right maxilla showing the dimension measured for maxillary height.

symmetry analyses. In an effort to minimize introduction of error in the data collection, each of two investigators (working independently) first recorded a set of measurements for each skull. Next, each investigator, on a separate occasion, again independently made a second set of measurements on the same skulls. When a comparison of these two sets of measurements showed an investigator differing from his own previous reading by 0.3 mm or more, he obtained a third measurement; obvious scale misreadings were discarded and the two or three measurements were averaged. These average values of the two workers for each dimension were then compared; it was found that the greatest between-investigator difference in the 162 comparisons was 0.23 mm. No further remeasurements were considered necessary and the two means for each of the 162 measurements were averaged to yield the final data used in the symmetry analyses.⁶

Findings

Asymmetry findings for the three dimensions studied appear in Tables 1, 2, and 3. The first two data columns of each table contain group means for linear dimensions in millimeters; the last three columns display group means and ranges for right: left asymmetry expressed as percentage differences. For example, reading across the top line of Table 1 (Control Group) reveals that the mean width of left maxillae was 11.9 mm, mean

⁶The mean difference between the two sets of independently obtained measurements for maxillary width was 0.009 mm; S.E. of measurement was 0.063 mm. The corresponding reliability statistics for the height and length dimensions were 0.028, 0.066, and 0.028, 0.036 mm, respectively.

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TABLE 1. Data for left and right maxillary width in controls and in the three surgical groups and for asymmetry of maxillae. Columns 4–6 contain relative right:left asymmetries expressed as per cent R < L. The mean for asymmetry for Surgical Group I (which is asterisked) is significantly greater than the mean for the control group. Negative values for asymmetry indicate L < R. (Surgery was on right maxillae only.)

Group	N	Mean width of maxillae, in mm		Asymmetry, as $\%$ R < L		
		left	right	range	mean	range
Control Surgical I Surgical II Surgical III	6 7 6 8	$ \begin{array}{r} 11.9 \\ 13.0 \\ 11.8 \\ 12.1 \end{array} $	$ 11.8 \\ 9.6 \\ 11.3 \\ 11.7 $	$ \begin{array}{r} -6.6 \\ 21.3 \\ -3.4 \\ -3.1 \end{array} $	$0.5 \\ 25.8^* \\ 4.4 \\ 2.8$	5.735.815.710.5

width of right maxillae was 11.8 mm, right maxillae averaged 0.5% narrower than left, and the range within the group was from 6.6% narrower on left than right, to 5.7% narrower on right than left.⁷

In contrast to the negligible amount of mean width asymmetry in the control group, Surgical Group I maxillae averaged nearly 26% narrower on right sides than left. The group range was from 21% narrower on right than left to 36% narrower on right than left. The difference between width asymmetry in Group I and in the control group is highly significant; width asymmetries for the other two surgical groups are not significantly greater than in the control group.⁸

Inspection of the statistics for asymmetry in maxillary height in Table 2 reveals that, as was seen for width, only the Group I asymmetry is significantly greater than the asymmetry in the control group. Maxillary height in Group I averaged 6.4% less on right sides than left, range for the group was from 3.9 to 10.6% less high on right than left. While this asymmetry is significantly greater than that in the control group, it is markedly less than that found in the width dimension.

The findings for group asymmetries in the maxillary length dimension appear in Table 3. Asymmetries were smaller in this dimension than in either width or height, but in both Surgical Groups I and II they were significantly greater than the corresponding asymmetry in the control

⁷ Percentages were calculated by subtracting the measurement of the smaller side from that of the larger (that is, 11.8 mm from 11.9 mm) and dividing the difference by the minuend (that is, 0.1 mm divided by 11.9 mm). The quotient times 100 is the percentage by which the small dimension is smaller than the large dimension. In the present example, 11.8 is 0.5% smaller than 11.9.

⁸ Student's t was used as the test statistic; the hypothesis of no difference between the means of two normally distributed populations was tested for each of the three possible experimental:control asymmetry comparisons in each of the three dimensions. Nine comparisons were thus tested; each involved a surgical group asymmetry and the corresponding control group asymmetry. In testing for significance of the Group I width asymmetry, for example, the sample asymmetry means from which the difference factor was obtained were 0.05% and 25.8%. All tests were two-ended with α at 0.05. Surgical group asymmetries which were found to be significantly greater than control group asymmetries are designated in the tables.

TABLE 2. Data for left and right maxillary height in controls and in the three surgical groups and for asymmetry of maxillae. Columns 4-6 contain relative right:left asymmetries expressed as per cent R < L. The mean for asymmetry for Surgical Group I (which is asterisked) is significantly greater than the mean for the control group. Negative values for asymmetry indicate L < R. (Surgery was on right maxillae only.)

Group	N	Mean height maxillae, in mm		$\begin{array}{ c c } Asymmetry, as \% \\ R < L \end{array}$		
		left	right	range	mean	range
Control Surgical I Surgical II Surgical III	6 7 6 8	$25.2 \\ 26.0 \\ 25.3 \\ 25.9$	$25.0 \\ 24.3 \\ 24.6 \\ 25.7$	$ \begin{array}{r} -2.5 \\ 3.9 \\ 0.7 \\ -1.3 \end{array} $	$0.7 \\ 6.4^* \\ 2.8 \\ 0.8$	$ \begin{array}{c} 2.2 \\ 10.6 \\ 7.0 \\ 2.5 \end{array} $

TABLE 3. Data for left and right maxillary length maxillae in controls and in the three surgical groups and for asymmetry of maxillae. Columns 4–6 contain relative right:left asymmetries expressed as per cent R < L. The means for asymmetry for Surgical Groups I and II (which are asterisked) are significantly greater than the mean for the control group. Negative values for asymmetry indicate L < R. (Surgery was on right maxillae only.)

Group	N	Mean length of maxillae, in mm		Asymmetry, as $\%$ R < L		
		left	right	range	mean	range
Control	6 7 6 8	56.1 57.4 57.5 57.5	$56.1 \\ 56.1 \\ 55.8 \\ 57.5$	$ \begin{array}{r} -1.1 \\ 0.6 \\ 1.5 \\ -2.0 \end{array} $	0.0 2.2* 3.0* 0.0	$ \begin{array}{r} 1.3 \\ 3.2 \\ 4.0 \\ 2.2 \end{array} $

group. On the average, Group II right maxillae were 3.0% shorter than left; on the average Group I right maxillae were 2.2% shorter than left. This was the only dimension in which maxillae of Group II dogs were significantly more asymmetrical than were those in the controls. Asymmetry in Surgical Group III dogs was not significantly different from that in the control group in any of the three dimensions studied.

Discussion

The dogs in Groups I and II of this study correspond to those of the two Herfert experiments. Findings concerning the effects of surgery on growth of maxillae in *width* are similar to and support the findings of the Herfert study. Herfert did not study growth in dimensions corresponding to those of maxillary length or height in the present study.

Surgical Group III has no counterpart in the Herfert work; the surgery for that group (interruption of the neurovascular bundle only) was

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included here to aid in distinguishing between the effects of the three variables of the Group I procedure, that is, the flap elevation, mucoperiosteal strip removal, and neurovascular bundle interruption. Herfert was also interested in distinguishing between effects of the different surgical variables. In his 1956 report he explained that his purpose in performing surgery on a second group of pups (corresponding to Group II here) was to answer skeptics who suggested that the asymmetry of maxillae reported from his first experiment might have resulted from the 'posterior palatine artery'⁹ interruption alone, and might be causally unrelated to the other variables in the procedure.

Herfert then changed his surgical design for the second group of pups to exclude the artery interruption. He also excluded, however, removal of the strip of mucoperiosteum from the flap edge, and he changed from a medially based flap to a posteriorly based flap. The fact that the difference between his two experimental procedures involved more than the mere interruption or lack of interruption of vasculature received little attention from Herfert in the discussion of clinical implications of his findings. Noting that there was less 'restriction of growth' in his dogs following the flap elevation with preservation of the artery, he cautioned surgeons against use of cleft repair procedures which involve artery ligations.

While it may be true that interruption of the artery is detrimental to maxillary growth, Herfert's experiments did little to demonstrate such a fact. The purpose of inclusion of Group III in the present study was to throw some additional light on the problem and to help determine if Herfert's conclusions were supportable. The present Group III finding that interruption of the right major palatine neurovascular bundle resulted in no significant asymmetry in any of the three maxillary dimensions studied suggests that one should view Herfert's interpretation of his findings with caution.

The search for a single surgical variable responsible for all of Herfert's 'restriction of growth' is not very fruitful of course, since variables may interact. Findings from the present study do suggest, however, that vascular interruption is less disturbing to maxillary growth than is removal of mucoperiosteum from an area of hard palate. Since bone denudation occurs as an integral part of most palatal cleft repairs, substantiation of such a finding would be of practical interest.

Summary

Surgical procedures involving manipulation of mucoperiosteum and vasculature on the right side only of hard palates of 21 normal 6¹/₂-week-

⁹ Miller, Christensen, and Evans (11) designate this the major palatine artery in the dog and that nomenclature is adopted in this report. They note that the contents of the palatine canal include not only the *major palatine artery* but also the *major palatine vein* and *major palatine nerve*. In this report the complex of vessels and nerve emerging from the major palatine foramen has been called the *major palatine neurovascular bundle*.

old Beagle pups were performed in replication and extension of experiments by Herfert. Findings regarding subsequent growth of right and left maxillae in width, length, and height were derived from measurements on the mature cleaned skulls. Mean maxillary asymmetry in each dimension in each of three surgical groups was compared with mean asymmetry in the control group. Asymmetry in Surgical Group I (flap elevation, removal of mucoperiosteal strip, and neurovascular bundle interruption) was significantly greater than that in the control group in each of the three dimensions studied. The probability is very high that much of the asymmetry was the result of the surgery. Findings from Groups II and III suggest that the surgical variable most responsible was that involving denudation of palatal shelf bone just medial to the alveolar process.

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