Growth of Maxillae in Dogs After Palatal Surgery: II

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The purposes of this report are to (a) present information about timing of surgically induced inhibition of maxillary breadth increase in normal (non-cleft) beagle dogs, and (b) compare findings from the present investigation with those of similar studies reported previously by Herfert (*1–3*). Both this study and those by Herfert were designed to yield basic information about effects of specific surgical variables on skeletal growth in the maxillary complex.

The information in Part I (*7*) included data on the ultimate magnitude of maxillary growth changes in the dogs of three experimental groups (Figure 1). Pups in each of these groups had a different surgical procedure performed on the right side of their hard palate on their 46th postnatal day. The finding of primary interest was that right maxillae in Surgical Group I dogs, at maturity, were significantly smaller than control maxillae in linear dimensions of breadth (width), height, and depth (length). The skeletal growth inhibition seen in Group I was apparently the result of the portion of the surgical procedure which involved mucoperiosteal denudation of palatal shelf bone adjacent to primary teeth.[1]

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[1] This conclusion was based on indirect evidence and inference, and was at odds with the interpretation of similar findings by Herfert. Three surgical variables were present in the Group I procedure: flap elevation, bone denudation, and neurovascular bundle interruption. In Herfert's view, the vascular interruption was responsible for the growth inhibition which he saw. The Group III procedure was designed (by us) as an indirect test of that hypothesis; only neurovasculature was interrupted, and there was no flap elevation or bone denudation. Our Group III animals, at maturity, showed no significant growth inhibitions in the test dimensions of maxillary breadth, height, or depth. Considering also our Group II findings, we hypothesized that denudation of shelf bone adjacent to the primary teeth, not interference with blood supply, was the primary surgical variable responsible for the growth inhibition seen. Subsequent findings in later experiments have reinforced our assumption that that hypothesis is correct (*10, 11*).

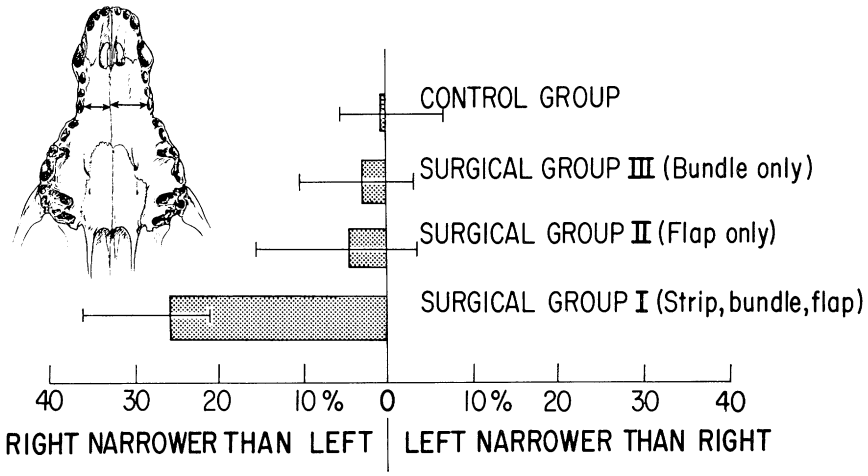

FIGURE 1. Asymmetry in maxillary palatal shelf breadth in four beagle groups as measured on cleaned dry adult skulls (7). Measurements were from distal root sockets of second premolars (PM2's) to the intermaxillary suture. Bars indicate magnitude of mean group asymmetries, error lines show the range of asymmetry within the groups. Surgery was performed on pups' 46th postnatal day and on the right side of their hard palates only. N's ranged from 6 to 8 animals per group.

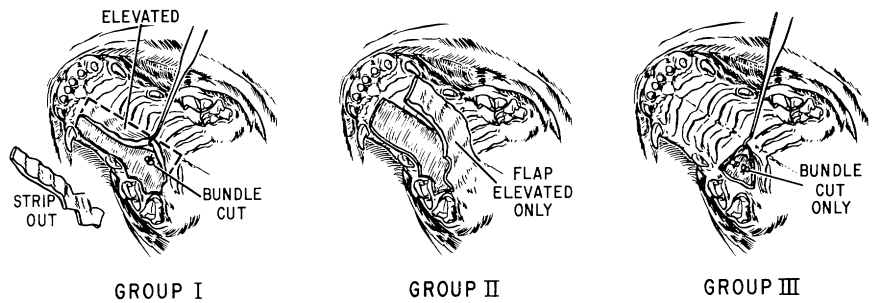

FIGURE 2. Palatal aspect of maxillae of beagle pups in their 6th postnatal week. Surgical designs for animals in each of the three groups (7) are shown; surgery was performed on right sides only. Mucoperiosteum in *Group I* was elevated to the midline, the major palatine neurovascular bundle was interrupted, and a 4 mm wide strip of the lateral flap margin was removed. Mucoperiosteum was elevated in *Group II* without removal of tissue or interruption of neurovasculature. There was no flap elevation in *Group III* except as necessary for access to and interruption of the major palatine bundle. The designs for Groups I and II were originated by Herfert (1–3).

The findings presented in this report (Part II) provide additional information about (a) the timing of the inhibition of shelf breadth increase in Group I, and (b) the degree of concordance of some findings from this study with those reported previously by Herfert. The data pertaining to timing were derived from measurements on the serial maxillary casts of the 7 Group I dogs and the 6 Control Group dogs. Data for the Herfert comparisons were from measurements on cleaned

dry skulls of the dogs in our three experimental and one control group.

Methods

Maxillary Casts: Preoperative maxillary casts were made on all pups when they were 6 weeks old. Surgery was performed on the pups' 46th postnatal day, and additional casts were made on the 49th and 56th days. Casts were then made every other week through the 22nd week, every third week through the 28th week, and every fourth week through the 68th week. All casts were made by the same person using customized acrylic impression trays and an alginate dental impression paste.[2] High density artificial stone[3] was poured into each impression immediately after its jellation. Pups became accustomed to the impression making routine rapidly, and sedation was never necessary.

Cast and Skeletal Measurements: Measurements on the maxillary casts and cleaned dry skulls were made using a single 10 cm sliding caliper having a vernier scale which was read to the nearest one tenth mm. Both the fixed and sliding arms were ground to sharp points. Care was taken to minimize systematic and random error in the measuring procedures. Casts were measured while clamped in a surveyor base having a ball and socket joint; a 1.5 × magnifier lamp[4] was used during measuring and caliper reading, and a small high intensity lamp[5] was focused on casts from the side to aid in indentification of the median raphe landmark. The magnifier lamp was also employed when making measurements on the dry skulls.

All measurements were made twice, independently, by each of two investigators. Each first compared his own two measurements of a given dimension. In cases of disagreement with himself exceeding 0.2 mm, he obtained at least one additional remeasurement before averaging his two or more determinations. Obvious scale misreadings were discarded and replaced. For *cast* measurements, the two investigators then compared their averaged determination for each dimension, and in cases of inter-investigator disagreements exceeding 0.2 mm, each obtained two additional remeasurements before calculating his final average for the dimension in question. Determinations of the two investigators were then averaged to yield the final datum to be used in analysis. The *skeletal* measurements of the two investigators were averaged without making final inter-investigator comparisons.

Cast Data: Data for longitudinal analysis of increase in palatal breadth were derived from the linear measurements made on each of the 22 maxillary casts for each animal. Palatal breadth was defined, for right and left sides, as the shortest straight line distance between (a)

[2] Heavy Body Jeltrate, L. D. Caulk Co., Milford, Del.
[3] Glastone, Ransom and Randolph Co., Toledo, Ohio.
[4] Luxo magnifier lamp, Luxo Lamp Corp., Port Chester, N.Y.
[5] Tensor Mod. 5975, Tensor Corp., Brooklyn, N.Y.

the most lateral point in the buccal gingival crevice of the deciduous first molar (DM1) or later its successor, the permanent second premolar (PM2), and (b) the center of the median raphe (Figure 3).

Skeletal Data: Data for comparison of findings with those of the Herfert studies (*1–3*) were obtained by making linear measurements on the cleaned dry skull of each animal. Right and left palatal breadths were measured as the shortest linear distance between the median suture and the medial lip of the various tooth sockets. The midline landmark at the 3rd incisor and canine levels was the suture between the two incisive (premaxillary) bones. Measure points for the two rooted premolars were (a) the medial lip of the *distal* root socket, and (b) the intermaxillary suture. Measure points for the three rooted 4th premolar and two molars were (a) the lip of the *lingual* socket and (b) the interpalatine suture. Maximum breadth of the palatal process of each palatine bone was measured as the shortest linear distance between the most lateral extremity of the palatine palatal process and the interpalatine suture (Figure 4).

Reliability: Cast Measurement Technique: A total of 44 final breadth determinations was obtained by each of the two investigators on the series of 22 casts for each of the 13 animals considered here. Since each investigator independently performed each measurement at least twice

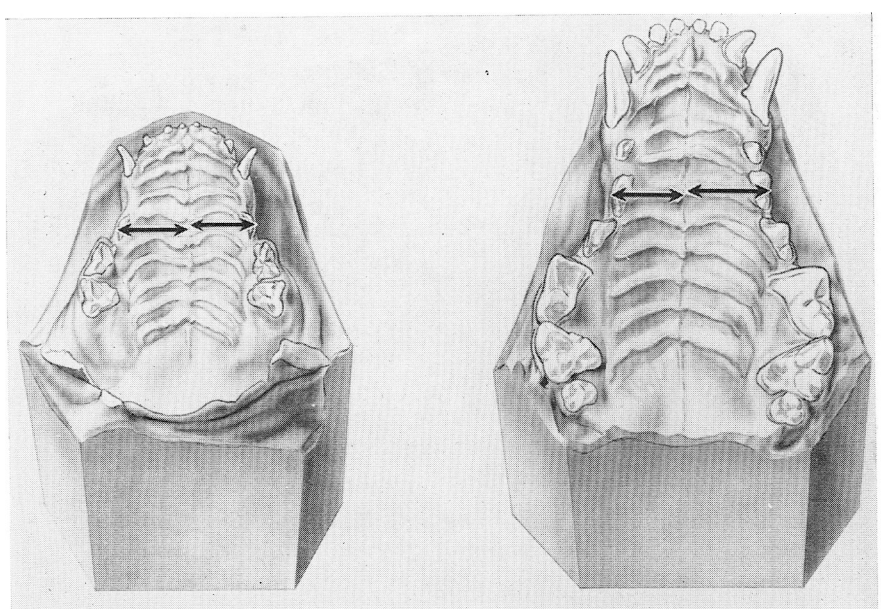

FIGURE 3. Palatal breadth as measured at the DM1/PM2 level on maxillary casts (Tables 1 and 2). DM1's are replaced by PM2's at 20–25 weeks. The cast on the left shows the 14 tooth deciduous dentition at 10 weeks, that on the right shows the 20 tooth permanent dentition at 68 weeks; the drawings are in actual proportion.

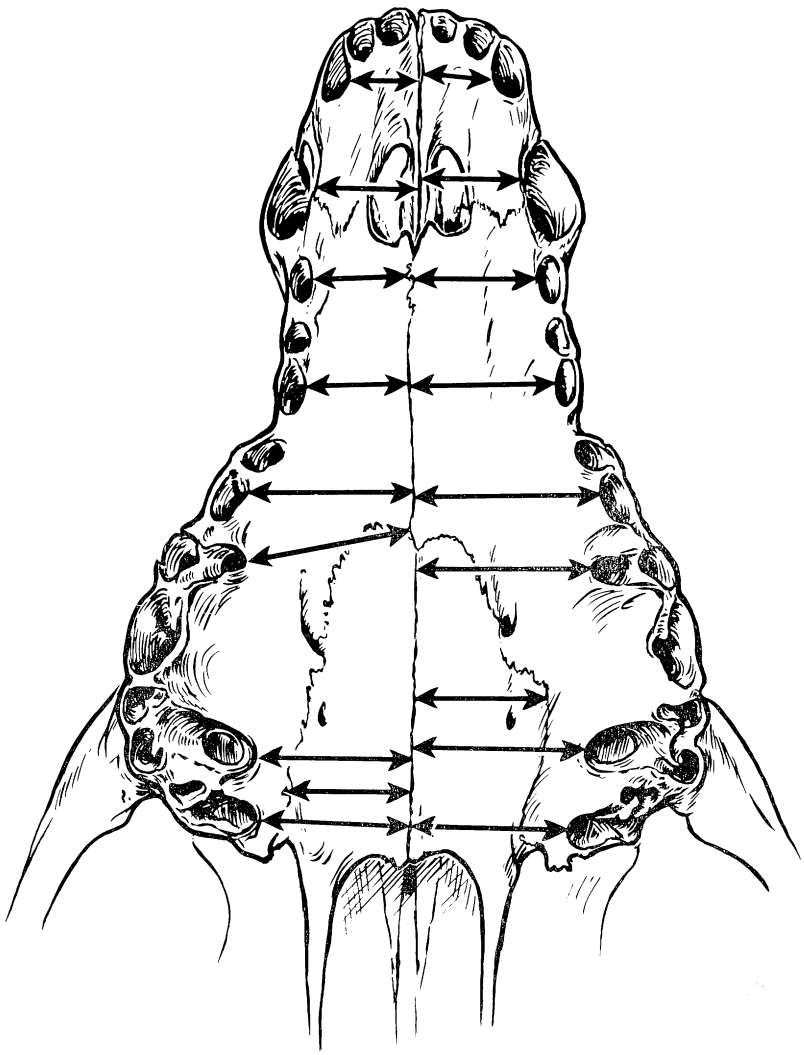

FIGURE 4. Palatal breadths as measured on the cleaned dry adult skulls (Tables 3–8). Landmarks are described in the text. The skull depicted is from a Group I beagle.

to obtain his final breadth determination, a total of approximately 2350 individual measurements was performed to produce the data in Table 1. When the 572 final determinations of one investigator were correlated with those of the other, the Pearson r coefficient of reliability was found to be 0.996. Analysis for systematic error revealed that the mean difference between final determinations of the two investigators was only 0.0001 mm. Analysis for random error revealed that the standard error of measurement was 0.118 mm (*4*).

Reliability: Skeletal Measurement Technique: A total of 18 final breadth determinations was obtained by each of the two investigators on the skulls of the 27 dogs of the three surgical and one control group.[6] Since each investigator independently performed each measurement at least twice to obtain his final breadth determination, a total of approximately 1000 individual measurements was performed to produce the data in Tables 4, 5, 7, and 8. When the 486 final determinations of one investigator were correlated with those of the other, the Pearson *r* coefficient of reliability was found to be 0.998. Analysis for systematic error revealed that the mean difference between final determinations of the two investigators was 0.145 mm. This relatively substantial level of systematic error in measurement technique precluded an analysis of random error by the difference method. Since, however, the coefficient of reliability was high, all final determinations by the two investigators were accepted for the purpose of the Herfert comparisons, and were averaged to yield the data appearing in Tables 4, 5, 7, and 8.

Data Transformations: Palatal breadth, as measured on the casts, included the buccolingual breadth of the DM1 tooth or its successor, the PM2 (*8*). Shelf breadth, as measured on the skulls, did not include the tooth breadth, and a problem arose in comparing relative asymmetries derived from the two forms of data. To make the two breadths comparable, the cast data were transformed by subtracting from them the breadths of the appropriate teeth. It was determined that the average difference between breadth as measured to the buccogingival crevice and measured to the medial lip of the root socket was 1.5 mm when the DM1 was in place and 3.6 mm when its successor, the PM2, was in place. Thus, 1.5 mm was subtracted from all cast data through the 22nd week (the last age at which DM1's were still in place), and 3.6 mm was subtracted from all cast data from the 25th week on, when all PM2's were in place. This difference in transformation constants reflects the fact that the DM1 is a small tooth, averaging only about 1.5 mm in effective buccolingual breadth,[7] while the PM2 is a relatively large tooth, averaging about 3.6 mm in effective breadth.

Findings

Timing of Inhibition of Palatal Shelf Breadth Increase: Longitudinal data from the cast measurements of palatal shelf breadth at the DM1/PM2 level are displayed in Table 1 and Figures 5–8. This, as transformed by the subtraction of tooth breadth, is the same dimension

[6] 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 the appropriate landmarks. Complete sets of measurements were not obtained on these animals, and this is reflected by the inconsistent N's in Tables 4 and 7.

[7] The term effective breadth, as used here, is not strictly the same as crown breadth, since the cast landmark was adjacent to the buccal crown surface, while the skull landmark was adjacent to the lingual root surface.

of maxillary breadth that was measured on the mature cleaned dry skulls and reported in Part I (7). Data on proportional asymmetry in shelf breadth at the same level were derived from the grouped absolute data at each age and are displayed in Table 2 and Figure 9.

Findings from analysis of the longitudinal data may be summarized as follows: (a) Left and right shelf breadths were approximately the same at 6 weeks for both the Control Group and Group I. (b) At 8 weeks, ten days after surgery, right (surgical side) shelves in Group I averaged nearly 16% narrower than left shelves. Absolute increase in left shelf breadth from 6 to 8 weeks was 1.4 mm, while right shelves in the same period showed an absolute decrease of 0.2 mm in breadth. (c) The rate of Group I breadth increase was only slightly less on right sides than on left from 8 through 20 weeks. Relative right-left breadth asymmetry, however, was nearly 18% in Group I at 20 weeks, compared with about 2% in the Control Group. (d) Exfoliation of DM1's and their replacement by PM2's in the 20–25 week interval resulted in an absolute narrowing of left and right palatal shelves in both groups of animals. This decrease in shelf breadth accompanying the change in dentitions was the natural consequence of the buccolingually narrow DM1's being

TABLE 1. Absolute breadths of maxillary left and right palatal shelves in mm for the 7 Beagles in Surgical Group I and 6 in the Control Group. Measurements from the deciduous first molar (after 25 wks. its replacement, the permanent second premolar), to the median raphe were made on maxillary casts obtained on exact weekly birth anniversaries.

age (wks)	*surgical group I (N = 7)*				*control group (N = 6)*			
	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	8.6 ± 0.3	7.6–9.6	8.5 ± 0.3	7.3–9.3
7	9.5 ± 0.2	8.9–10.2	9.4 ± 0.1	9.0–10.0	9.0 ± 0.2	8.1–9.7	9.0 ± 0.3	8.0–10.0
8	10.4 ± 0.2	9.7–11.2	8.8 ± 0.2	7.9–9.5	9.6 ± 0.2	8.8–10.5	9.5 ± 0.3	8.4–10.5
10	11.3 ± 0.2	10.5–11.7	9.1 ± 0.2	8.3–9.8	10.5 ± 0.3	9.3–11.4	10.2 ± 0.3	9.3–11.6
12	12.1 ± 0.1	11.4–12.5	9.7 ± 0.2	8.9–10.4	11.2 ± 0.4	10.0–12.5	10.9 ± 0.4	9.8–12.4
14	12.8 ± 0.1	12.3–13.4	10.4 ± 0.2	9.7–11.1	11.8 ± 0.3	10.6–12.8	11.6 ± 0.4	10.3–13.1
16	13.6 ± 0.1	13.2–14.0	11.0 ± 0.2	10.2–11.5	12.4 ± 0.3	11.0–13.4	12.3 ± 0.4	11.2–13.7
18	14.0 ± 0.1	13.7–14.3	11.4 ± 0.2	10.6–12.1	12.8 ± 0.4	11.3–13.7	12.6 ± 0.4	11.6–14.1
20	14.3 ± 0.1	14.0–14.5	11.7 ± 0.2	11.0–12.4	13.1 ± 0.4	11.4–14.2	12.9 ± 0.4	11.6–14.1
22	14.3 ± 0.1	12.8–14.6	11.5 ± 0.2	11.0–12.8	13.1 ± 0.4	11.7–14.0	13.0 ± 0.3	11.8–14.0
25	12.6 ± 0.1	12.2–13.1	9.6 ± 0.2	9.0–10.2	11.6 ± 0.3	10.2–12.3	11.5 ± 0.3	10.2–12.2
28	12.7 ± 0.2	12.2–13.5	9.6 ± 0.2	8.8–10.1	11.7 ± 0.4	10.2–12.8	11.6 ± 0.4	10.2–12.5
32	12.8 ± 0.2	12.2–13.4	9.5 ± 0.2	8.8–10.0	11.7 ± 0.4	10.2–12.9	11.7 ± 0.4	10.2–12.4
36	12.7 ± 0.1	12.1–13.3	9.5 ± 0.2	8.8–10.0	11.7 ± 0.4	10.2–12.8	11.8 ± 0.4	10.3–12.8
40	12.8 ± 0.1	12.2–13.4	9.5 ± 0.2	8.6–10.0	11.8 ± 0.4	10.4–13.0	11.8 ± 0.4	10.3–12.8
44	12.8 ± 0.1	12.2–13.2	9.4 ± 0.1	8.7–9.8	11.8 ± 0.4	10.4–13.0	11.8 ± 0.4	10.2–12.8
48	12.9 ± 0.2	12.4–13.6	9.5 ± 0.1	8.8–9.9	11.9 ± 0.4	10.4–13.1	11.8 ± 0.4	10.3–12.6
52	12.9 ± 0.2	12.3–13.4	9.5 ± 0.2	8.7–10.0	11.8 ± 0.4	10.4–13.1	11.8 ± 0.4	10.4–12.7
56	12.9 ± 0.2	12.3–13.4	9.6 ± 0.2	8.7–10.0	11.9 ± 0.4	10.2–13.1	11.8 ± 0.3	10.4–12.7
60	12.9 ± 0.1	12.4–13.4	9.7 ± 0.2	8.8–10.2	11.9 ± 0.4	10.5–13.0	11.8 ± 0.4	10.4–12.7
64	12.9 ± 0.2	12.4–13.6	9.8 ± 0.2	8.9–10.2	12.0 ± 0.4	10.6–13.0	12.0 ± 0.4	10.6–13.2
68	12.9 ± 0.2	12.4–13.4	9.7 ± 0.2	8.9–10.2	12.0 ± 0.4	10.6–13.1	12.0 ± 0.4	10.7–13.2

TABLE 2. Relative asymmetries in breadth of maxillary left and right palatal shelves calculated from group means in Table 1. 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.)	*surgical group I (N = 7)*	*control group (N = 6)*	*difference in asymmetry (s-c)*
6	−0.1%	1.8%	1.9%
7	1.9	0.8	1.1
8	15.5	1.6	13.9
10	19.4	2.3	17.1
12	19.9	2.2	17.7
14	19.4	1.0	18.4
16	19.5	1.2	18.3
18	18.5	1.6	16.9
20	17.9	1.9	16.0
22	19.5	0.9	18.6
25	23.9	1.1	22.8
28	24.4	0.8	23.6
32	25.8	0.5	25.3
36	25.2	−1.4	26.6
40	25.8	0.5	25.3
44	26.3	0.7	25.6
48	26.1	1.1	25.0
52	26.3	0.2	26.1
56	25.6	0.2	25.4
60	25.1	0.7	24.4
64	24.6	0.1	24.5
68	24.5	−0.6	25.1

replaced by the broader PM2's, the latter teeth actually encroaching on, and erupting into the lateral border of the maxillary palatal process. (e) Right-left asymmetry was slightly greater at the end of the dentition change in Group I than before; asymmetry in controls remained essentially unchanged. (f) Shelf breadths showed slight but inconstant increases from 25 through 67 weeks on both sides in the Control Group and on left sides in Group I. Breadth on the right (surgical) side in the latter group decreased slightly from the 25th to the 44th week, then increased slightly through the 63rd week. (g) Maximum asymmetry in Group I (26.3%) was at 52 weeks; maximum asymmetry in controls (2.3%) was at 10 weeks, and it varied only slightly in that group between −1.4% (at 36 weeks) and the maximum. (h) A relatively large proportion of the ultimate Group I breadth asymmetry developed in the early postsurgical weeks; 63% of ultimate asymmetry was already present at 8 weeks, 79% was present at 10 weeks, and 97% was present at 25 weeks.

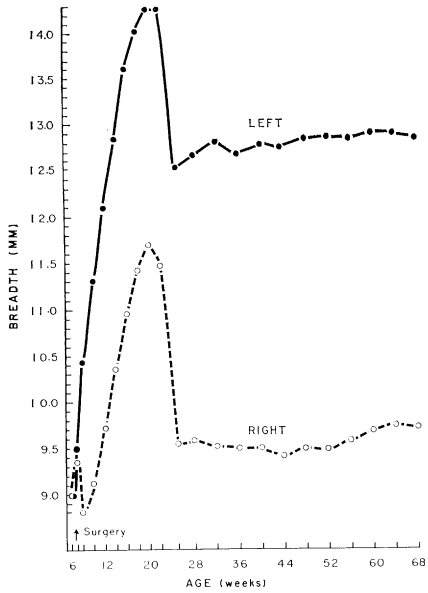

FIGURE 5. Plotted mean DM1/PM2 level palatal shelf breadths in the 7 *Group I* beagles. Measurements were made on serial maxillary casts (Table 1). Right (surgical side) breadths were less than left at all postsurgical ages. The reduction in shelf breadths after the 22nd week reflects exfoliation of DM1's and their replacement by the broader PM2's. Compare with Fig. 6.

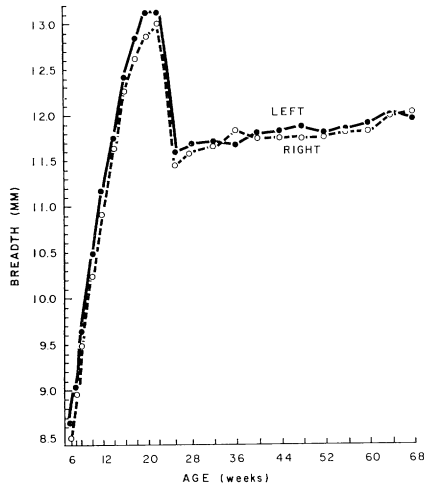

FIGURE 6. Plotted mean DM1/PM2 level palatal shelf breadths in the 6 *Control Group* beagles. Measurements were made on serial maxillary casts (Table 1). Right and left side breadths were approximately symmetrical at all ages. The reduction in shelf breadths after the 20th–22nd weeks reflects exfoliation of DM1's and their replacement by the broader PM2's. Compare with Fig. 5.

Comparison with Herfert Findings: The Group I surgical procedure of this study was essentially the same as that used by Herfert for his Group I (Figure 2).[8] Herfert reported individual measurements for 6 palatal breadth dimensions in each of his Group I animals (*1, 3*). To

[8] Several minor differences in the studies may be noted. (a) Herfert's three Group I dogs were six weeks old at the time of surgery; age of the 7 Group I dogs in this study was 46 days. (b) In Herfert's procedure the vessels were "ligated or intentionally torn," while in this study they were double ligated, then severed by removal of a 2–3 mm segment between the ligatures. (c) Age at sacrifice for Herfert's Group I was 9½ months; in this study it was 17 months.

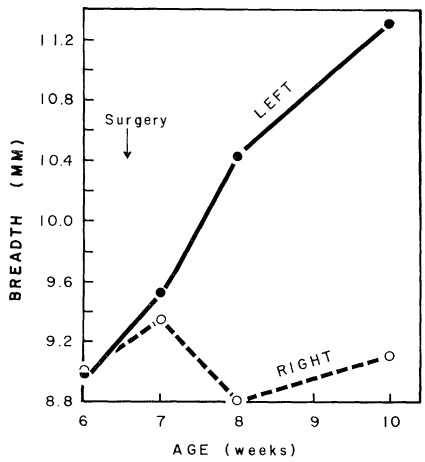

FIGURE 7. Plotted mean DM1 level palatal shelf breadths in the *Group I* beagles for postnatal weeks 6–10. This expansion of the first part of the Fig. 5 plot illustrates more clearly the nature of the early postsurgical breadth changes. Compare with Fig. 8.

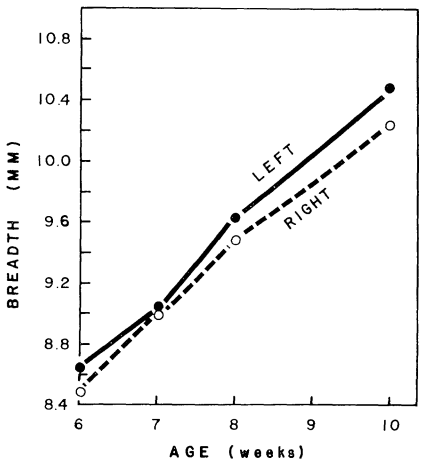

FIGURE 8. Plotted mean DM1 level palatal shelf breadths in the *Control Group* beagles for postnatal weeks 6–10. This expansion of the first part of the Fig. 6 plot clearly illustrates the early breadth trends in animals having no palatal surgery. Compare with Fig. 7.

facilitate group comparisons, those published data were collated and averaged, and appear here in Table 3. Analogous data for Group I of this study appear in Table 4, which also contains data for three shelf breadths not studied by Herfert.[9] Table 5 contains Control Group data from this study; data from Herfert's single control animal are not included here.

[9] The anatomic landmarks for measurement of the shelf breadth dimensions at each tooth level appear to have been slightly different in the Herfert study than those used in this study. Such differences are of little importance in intergroup comparisons of absolute right-left breadth differences. Intergroup comparisons of percent asymmetries should be performed with caution, however, since each of these data are dependent on both the absolute difference between a right and a left breadth and the magnitude of the absolute breadth of the wider side.

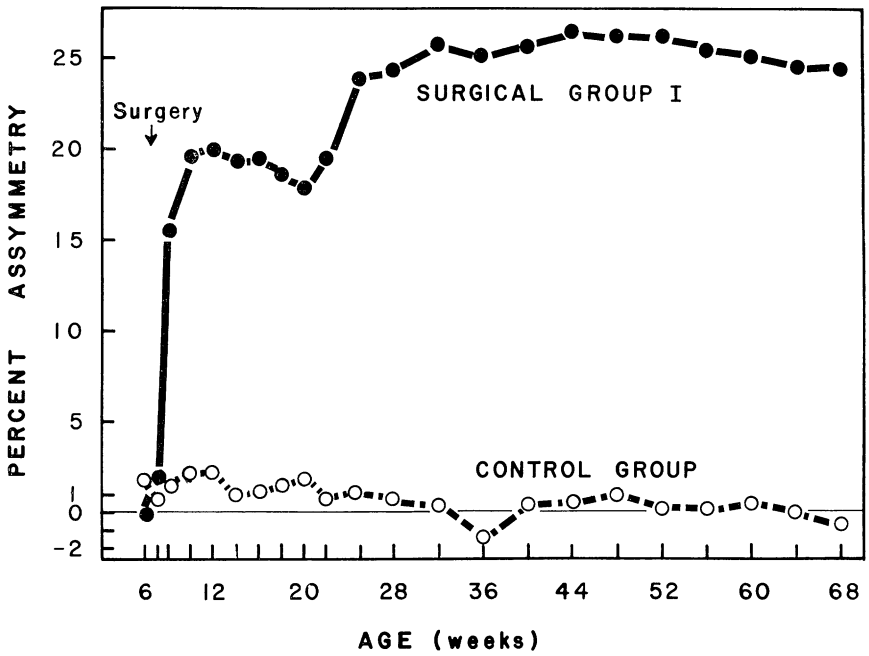

FIGURE 9. Plot of the DM1/PM2 level palatal shelf asymmetry data shown in Table 2. The zero line represents perfect bilateral symmetry; plotted points above zero are read as percentages by which right shelves were narrower than left, while points below zero are read as percentages by which left shelves were narrower than right.

TABLE 3. Herfert's Surgical Group I absolute means for six palatal shelf breadths (columns 3 and 4), and corresponding percent left-right asymmetries and ranges of asymmetry (columns 6–8). Asymmetries were calculated as in Table 2.

Meas. level	*N*	*Left* $\bar{x}$ *mm*	*Right* $\bar{x}$ *mm*	*L* $\bar{x}$ *Minus R* $\bar{x}$	*Percent R < L*		
					range	$\bar{x}$	*range*
PC	3	9.4	8.8	0.6	0.0	5.7	12.1
PM1	3	10.0	8.4	1.6	9.4	15.4	21.6
PM2	3	11.5	9.5	2.0	9.1	17.2	22.8
PM3	3	14.2	12.7	1.5	5.2	10.7	15.7
PM4	3	19.5	17.5	2.0	7.4	10.1	11.6
M1	3	12.7	11.3	1.4	8.1	11.0	13.9

A comparison of information in Tables 3 and 4 may be summarized as follows: (a) The asymmetry findings in both Group I's are similar. Right shelves were narrower than left in both groups for each of the 6 possible comparisons. (b) There were slight to moderate regional differences between the two groups in magnitude of asymmetry. The asymmetry was greater in the middle part of the palate in this

TABLE 4. Surgical Group I absolute means for nine palatal shelf breadths (columns 3 and 4), and the corresponding percent right-left asymmetries and ranges of asymmetry (columns 6–8). Asymmetries were calculated as in Table 2.

Meas. level	*N*	*left* $\bar{x}$ *mm*	*right* $\bar{x}$ *mm*	*l* $\bar{x}$ *minus r* $\bar{x}$	*percent r < l*		
					range	$\bar{x}$	*range*
PI3	6	6.7	6.4	0.3	−0.4	3.9	8.4
PC	6	9.4	9.0	0.4	0.5	4.0	7.2
PM1	6	11.2	8.7	2.5	17.2	22.2	27.0
PM2	7	13.0	9.6	3.4	21.3	25.8	35.8
PM3	7	17.0	14.7	2.3	9.6	13.4	17.4
PM4	7	16.9	15.8	1.1	2.1	6.1	12.0
M1	7	16.7	16.3	0.4	−1.7	2.8	6.8
M2	6	13.5	13.7	−0.2	−6.7	−1.4	2.5
PAL.	6	11.5	11.4	0.1	−4.9	0.9	9.2

TABLE 5. Control Group absolute means for nine palatal shelf breadths (columns 3 and 4), and the corresponding percent right-left asymmetries and ranges of asymmetry (columns 6–8). Asymmetries were calculated as in Table 2.

Meas. level	*N*	*left* $\bar{x}$ *mm*	*right* $\bar{x}$ *mm*	*l* $\bar{x}$ *minus r* $\bar{x}$	*percent r < l*		
					range	$\bar{x}$	*range*
PI3	6	6.2	6.2	0.0	−3.0	0.2	2.0
PC	6	10.3	10.1	0.2	−0.8	1.6	6.5
PM1	6	10.3	10.1	0.2	−1.4	1.9	4.4
PM2	6	11.9	11.9	0.0	−6.6	0.5	5.7
PM3	5	15.2	15.3	−0.1	−2.3	−0.8	1.0
PM4	6	14.3	14.2	0.1	−2.1	0.1	3.1
M1	6	14.3	14.5	−0.2	−4.0	−1.1	0.7
M2	6	13.1	13.5	−0.4	−4.6	−2.4	−0.7
PAL.	6	11.2	11.2	0.0	−5.1	−0.2	7.0

study than in Herfert's, but it was somewhat less in anterior and posterior regions of the palate in this study than in Herfert's.

The Group II surgical procedure of this study was the same as that reported by Herfert for his Group II animals. In his report on that group, Herfert again presented individual measurements for 6 palatal breadth dimensions in each of his animals (*2*, *3*).[10] Those data, collated and averaged, appear in Table 6. Analogous data for Group II of this study appear in Table 7. Comparison of the information in the two tables reveals that asymmetry was somewhat greater in magnitude and

[10] Herfert's four Group II animals were six weeks old at the time of surgery; age of the 6 Group II dogs in this study was 46 days. Age at sacrifice for Herfert's Group II was 10½ months, while in this study it was 17 months.

TABLE 6. Herfert's Surgical Group II absolute means for six palatal shelf breadths (columns 3 and 4), and corresponding percent right-left asymmetries and ranges of asymmetry (columns 6–8). Asymmetries were calculated as in Table 2.

Meas. level	*N*	*left* $\bar{x}$ *mm*	*right* $\bar{x}$ *mm*	*l* $\bar{x}$ *minus r* $\bar{x}$	*percent r < l*		
					range	$\bar{x}$	*range*
PM1	3	10.2	9.9	0.3	−2.0	3.4	6.9
PM2	2	11.0	10.4	0.6	0.0	5.4	10.8
PM3	4	14.4	13.2	1.2	0.0	7.8	13.0
PM4	4	19.3	18.5	0.8	0.0	4.0	9.0
M1	4	13.2	12.7	0.5	0.7	3.6	6.5
M2	4	12.9	12.7	0.2	0.0	1.5	4.2

TABLE 7. Surgical Group II absolute means for nine palatal shelf breadths (columns 3 and 4), and the corresponding percent right-left asymmetries and ranges of asymmetry (columns 6–8). Asymmetries were calculated as in Table 2.

Meas. level	*N*	*left* $\bar{x}$ *mm*	*right* $\bar{x}$ *mm*	*l* $\bar{x}$ *minus r* $\bar{x}$	*percent r < l*		
					range	$\bar{x}$	*range*
PI3	5	6.3	6.2	0.1	1.2	2.4	5.2
PC	5	8.9	8.5	0.4	−0.9	4.4	7.4
PM1	5	10.5	10.1	0.4	−0.3	3.4	10.6
PM2	6	11.8	11.3	0.5	−3.4	4.4	15.7
PM3	6	15.8	15.7	0.1	−3.6	0.8	9.5
PM4	6	15.7	15.9	−0.2	−5.6	−1.4	4.9
M1	6	15.6	15.9	−0.3	−3.7	−2.0	−0.4
M2	5	12.2	12.4	−0.2	−5.6	−1.4	2.0
PAL.	5	10.8	10.8	0.0	−5.0	0.5	5.8

more consistent in direction in Herfert's Group II than in that of this study. The greatest intergroup differences were in the posterior region of the palate, where right side breadths were actually greater than left in animals of this study. Comparison of the asymmetry data from the two Group II's with those of the Control Group (Table 5) shows that (a) asymmetry variability was considerably greater in both Group II's than in controls, and (b) mean asymmetries in Group II of this study were only slightly greater than in controls, while mean asymmetries in Herfert's Group II were somewhat more marked.

Individual asymmetry data for Group II animals of this study were examined in an attempt to determine the reason for the wide range of asymmetries seen in the grouped data. Noteworthy asymmetries were seen in only two of the 6 animals. Breadth at the PM2 level, for example, averaged approximately 14% less on right than left sides in the

two dogs, while in the other four, the analogous breadths averaged approximately 2% less on left than on right sides. Examination of the maxillary casts for these 6 animals provided the probable explanation for these findings. Nearly exact readaptation of the elevated mucoperiosteal flap to its original position against the shelf bone appeared to have occurred in the four dogs showing little asymmetry. In contrast, flap readaptation had been decidedly imperfect in the two animals showing the marked asymmetry. In both cases, the flap had shifted slightly medially, leaving a narrow area of denuded shelf bone adjacent to the teeth.

Asymmetry data for Surgical Group III of this study appear in Table 8. The Group III procedure was performed an an extension of the Herfert experimental design and involved interruption of the right major palatine neurovascular bundle only. Comparison of the information in Table 8 with that for the Control Group (Table 5) may be summarized as follows: (a) Mean asymmetries are less than in the Control Group in two of the 8 breadth dimensions, and exceed control asymmetries only slightly in the other 6 dimensions. (b) The range of breadth asymmetry in Group III is greater than in the Control Group in 7 of the 8 breadth dimensions studied.

Maximum breadth of the palatal processes of the right and left palatine bones was measured on the cleaned skulls of 25 of the dogs in this study. These data for each of the three surgical groups and the Control Group appear in the bottom line of Tables 4, 5, 7, and 8. The information about palatine palatal process asymmetry may be summarized as follows: (a) The central tendency and variability statistics for breadth asymmetry are very similar for both Surgical Groups I and II and for the Control Group. (b) Breadth asymmetry in Group III was considerably greater than in the Control Group both in terms of central tendency and variability.

TABLE 8. Surgical Group III absolute means for eight palatal breadths (columns 3 and 4), and corresponding percent right-left asymmetries and ranges of asymmetry (columns 6–8). Asymmetries were calculated as in Table 2.

Meas. level	*N*	*left* $\bar{x}$ *mm*	*right* $\bar{x}$ *mm*	*l* $\bar{x}$ *minus r* $\bar{x}$	*percent r < l*		
					range	$\bar{x}$	*range*
PI3	8	6.5	6.4	0.1	−4.7	0.9	10.6
PC	8	8.9	9.0	−0.1	−11.2	−1.4	5.4
PM1	8	10.7	10.3	0.4	−2.0	3.8	11.3
PM2	8	12.1	11.7	0.4	−3.1	2.8	10.5
PM3	8	15.4	15.2	0.2	−1.7	1.2	4.8
PM4	8	14.3	14.4	−0.1	−2.6	−0.6	2.2
M1	8	14.4	14.0	0.4	−7.6	2.4	14.4
M2	8	13.1	13.0	0.1	−2.3	0.8	5.5
PAL.	8	11.8	11.3	0.5	−12.6	4.5	22.0

Discussion

The finding that the onset of Group I palatal shelf asymmetry was a relatively early postsurgical phenomenon is of interest for at least two reasons: (a) it suggests that the skeletal growth interference may be directly related to some aspect of mucoperiosteal wound healing, and (b) it suggests that experimental attempts to alleviate or control the growth disturbance must be initiated soon after surgery.

Some observations regarding interpretation of the early postsurgical shelf asymmetries are appropriate. The decrease in Group I right shelf breadth between the 3rd and 10th postsurgical days probably reflects a moving together of the measurement landmarks only, and not a real decrease in *skeletal* breadth of the right palatal process. Inspection of the casts supports this view; the median raphe on the 7 week casts is a relatively straight line, but on the 8 week and subsequent casts it deviates slightly to the right in the molar areas. One hypothesized explanation for this is that soft tissue contraction associated with wound healing in the surgical area stretches the adjacent mucoperiosteum and pulls the raphe slightly to the right. While the typical position of the median raphe is directly over the intermaxillary suture, this postsurgical deviation probably results in its being temporarily pulled laterally and away from its original location. A test of that hypothesis could be performed by comparing measurements made to the *suture* on coronal sections through this area with measurements made to the *raphe* on maxillary casts made just before sacrifice on the same animals. While this test has not been conducted at 8 weeks (the low point in the right shelf breadth curve), it has been conducted, in a separate investigation (*5, 6*) at 10 weeks. It was found, as in this study, that the median raphe deviated to the right on 8 week and subsequent casts, but that at 10 weeks, when the pups were sacrificed, the suture was directly beneath the deviated raphe. Presumably sutural remodeling had occurred after the deviation of the raphe from the midline, so that by 10 weeks the suture again lay directly beneath the raphe. Our conclusion, then, based on the information thus far at hand, is that shelf breadth, as measured to the *raphe* on the 8 week casts, is slightly less on the right and more on the left than would have been seen if measurements could have been made to the *suture*. It is probable that the plotted postsurgical *decrease* in right shelf breadth (7–8 weeks, Figure 7) is mainly a soft tissue phenomenon, and it is probable that *skeletal* right shelf breadth increased slowly during the *entire* 6–10 week interval.

Another factor which could play a role in producing the 7–8 week apparent decrease in right shelf breadth is slight medial tipping of the DM1 tooth. Inspection of casts from this study and of casts and tissue sections from animals sacrificed at 10 weeks (*5, 6*) indicates that while some medial tipping did occur, its contribution to the initial

shelf "narrowing" is probably minimal compared with that of median raphe deviation.

There is still another factor which should be considered in the interpretation of the asymmetry data. Since the intermaxillary suture deviated slightly to the right after surgery (average shift away from the midline in the 7 Group I skulls was approximately 1 mm), it seems apparent that left shelf breadth, as measured to the suture, was somewhat greater than it would have been if the suture had remained in the midline. This had the effect of exaggerating the asymmetry, since even without any growth inhibition on the right sides, manipulation of the position of the suture would introduce asymmetry in shelf breadths. An indication of the magnitude of this exaggeration can be appreciated if the 1 mm sutural shift is arbitrarily subtracted from mean left shelf breadth and added to right shelf breadth. When this calculation is performed, right shelf breadth in Group I averages about 10% less than left shelf breadth. It is not known what the effect on growth in the suture would have been if surgery had been performed on both sides instead of only on the right. It seems probable, however, that total maxillary breadth at the DM2 level would have been at least 20% less than normal. To further demonstrate that the sutural deviation was an exaggerating influence on asymmetry only, Group I right side breadth may be compared with Control Group right or left shelf breadths.

Differences of the magnitude seen between the Herfert Group I and II findings and those of this study are not surprising in view of the small sample sizes, the difference in breeds of dogs used, the differences in ages at sacrifice, and the probable slight differences in surgical techniques and postoperative care. In general, the two sets of Group I and II findings for ultimate palatal shelf asymmetry are complementary. As noted in Part I (7), however, we do not agree with Herfert's assumption that his findings demonstrate the critical importance of uninterrupted palatal vasculature. Considering all findings from both studies, our tentative conclusions are that (a) Group I type surgery results in significant inhibition of maxillary growth which is most marked in the dimension of breadth in the mid-snout area, (b) the growth inhibition seen is probably attributable to mucoperiosteal denudation of shelf bone adjacent to teeth, and not to interference with blood supply, and (c) simple elevation and repositioning of a mucoperiosteal flap in its original site, as in Group II, is insufficient surgical insult to result in important or consistent maxillary growth disruptions.

Considering Herfert's studies alone, it is possible to infer that simple elevation and reapposition of palatal mucoperiosteum (as in Group II) may be a significant growth inhibiting variable. The finding, in this study, that Group II shelf asymmetries were marked only in the animals where flap readaptation was poor suggests that such an inference is

unwarranted. It is possible that poor flap readaptation occurred in some of Herfert's Group II animals as it did in two of those in this study. It seems probable that imperfect readaptation of mucoperiosteum and the resulting inadvertent denudation of a narrow area of palatal shelf bone adjacent to the teeth was responsible for the observed asymmetries in both Group II's. Again, our tentative conclusion, based on Group II findings from both studies, is that simple elevation and readaptation of palatal mucoperiosteum is insignificant as a growth affecting surgical variable in this model.

Some of the asymmetry variability data for Surgical Group III (Table 8) is puzzling and cannot be satisfactorily explained at this time. Mean asymmetry at the M1 level in Group III, for example, was only slightly different than in the Control Group or in Group I, but the range of asymmetry was much greater in Group III than in either of those two other groups. The wide range of palatine bone palatal process breadth asymmetry is also unexplained at this time.

No existing data are known pertaining to the age at which ultimate size of facial bones in dogs is achieved. All animals of this study were sacrificed after their 68th week, since it appeared that no important dimensional changes in maxillary breadth were likely to occur after that age. Examination of the trend lines for DM1/PM2 level maxillary breadth (Figures 5 and 6) shows that that assumption was probably correct, although the general slightly upward trend of all four lines prior to the 64th week leaves the matter open to some question.

Summary

The timing of surgically induced inhibition of growth in palatal shelf breadth in dogs was studied using data from maxillary casts. The study was an extension of previous work (Part I) which indicated that surgery involving removal of mucoperiosteum from palatal shelf bone adjacent to teeth resulted in significant maxillary growth inhibition. Investigation of the timing of that inhibition in this study showed that most of the surgically induced growth effect was expressed within the first few weeks after surgery.

Growth data from this study were compared with those previously reported by Herfert. Differences in findings of the two investigations were examined, and it was concluded that disagreements were mainly in the interpretation of essentially complementary findings. It remains highly probable that, in the model studied, denudation of palatal shelf bone adjacent to teeth is the surgical variable responsible for subsequent maxillary growth disturbances, and that temporary elevation of mucoperiosteum *per se,* or interruption of major palatine neurovasculature *per se,* are not important as maxillary growth inhibiting variables.

References

1. Herfert, O., Experimenteller Beitrag zur Frage der Schadigung des Oberkiefer-Wachstums durch vorzeitige Gaumenspaltoperation. *Deutsche Zahn-, Mund- und Kieferheilkunde, 20,* 369–381, 1954.
2. Herfert, O., Tierexperimentelle Untersuchungen uber die biologische Wertigkeit von Bruckenlappen (Axhausen) und Stiellappen (palatinalappen) bei der Gaumenplastik. *Deutsche Zahn-, Mund- und Keiferheilkunde, 24,* 112–120, 1956.
3. Herfert, O., Fundamental investigations into problems related to cleft palate surgery. *Brit. J. Plastic Surg., 11,* 97–105, 1958.
4. Horowitz, S. L., and E. H. Hixon, *The Nature of Orthodontic Diagnosis.* C. V. Mosby, St. Louis, 1966, p. 297.
5. Hugg, J. R., Growth of the maxillae in dogs after palatal surgery studied with the aid of vital staining. M.S. Thesis, University of Iowa, Iowa City, 1968.
6. Hugg, J. R., and C. R. Kremenak, Jr., Growth of maxillae in dogs after palatal surgery studied with the aid of vital staining. (Abstract) *Amer. J. Orthodont., 54,* 930, 1968.
7. Kremenak, Jr., C. R., W. C. Huffman, and W. H. Olin, Growth of maxillae in dogs after palatal surgery: I. *Cleft Palate J., 4,* 6–17, 1967.
8. Kremenak, Jr., C. R., Dental exfoliation and eruption chronology in beagles. *J. Dent. Res., 46,* 686–693, 1967.
9. Kremenak, Jr., C. R., and J. H. Hugg, Growth of maxillae in dogs after palatal surgery: vital staining and early sacrifice. (In preparation.)
10. Kremenak, Jr., C. R., W. H. Olin, and W. C. Huffman, Maxillary growth inhibition by mucoperiosteal denudation of palatal shelf bone in non-cleft beagles. *Cleft Palate J.* (In press.)
11. Kremenak, Jr., C. R., S. E. Demjen, and J. M. Grewe, Absence of maxillary growth inhibition following bilateral interruption of palatine vasculature in beagles. (In preparation.)