A Question of the Role of the Vomer in the Growth of the Premaxillary Segment

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Research on the problem of antero-posterior growth retardation of the maxillae following cleft lip and palate surgery has been relatively neglected compared to that on growth retardation in width (3, 5).

Most of the work on normal forward growth of the middle third of the face has centered on the role of the cartilaginous component of the nasal septum (4, 6, 9, 10, 17). However, with reference to treatment of cleft lip and palate, it is important that information be obtained about the role of the vomer.

An observation is reported on growth retardation in the upper jaw in dogs with artificial cleft palate and extirpation of the vomer which suggests that the dog might serve as a useful model for studying this problem in the human.

Material and methods

Observations were made on five dogs with surgically placed clefts of the hard palate, (Figure 1) and five which had congenital complete clefts of the secondary palate (Figure 2).

The artificial clefts were made in the palate of five puppies under anesthesia by removing a median strip of palatal mucosa and an 8 mm wide portion of palatal bone from the incisive canal to the posterior border of the hard palate. The greater part of the vomer was removed using a round dental bur, leaving the cartilaginous nasal septum intact. The nasal mucosa was incised and the edges sutured to the oral mucosa to cover the exposed bone (7). The periods of survival following cleft formation were from nine weeks to two years and four months (Table 1).

Of the congenitally cleft animals (Table 2), the Shetland Sheepdog received no surgery and the clefts of four Golden Retrievers were surgically closed at the age of about 10 weeks.

Findings

Anterior crossbite of the incisor teeth became conspicuous 8 weeks following surgery in all 5 dogs with artificial clefts (Figure 3). The third lateral incisor
FIGURE 1. Photograph of dog 1 palate showing surgically placed cleft.

FIGURE 2. Photograph of dog 6 palate showing congenital cleft. Shetland Sheepdog, 22 weeks.

TABLE 1. Dogs With Artificial Cleft Palate

<table>
<thead>
<tr>
<th>no.</th>
<th>breed</th>
<th>age when cleft was placed</th>
<th>post-operative survival</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>Mongrel</td>
<td>7 weeks</td>
<td>21 weeks</td>
</tr>
<tr>
<td>2</td>
<td>Mongrel</td>
<td>7 weeks</td>
<td>9 weeks</td>
</tr>
<tr>
<td>3</td>
<td>German Shepherd</td>
<td>8 weeks</td>
<td>10 weeks</td>
</tr>
<tr>
<td>4</td>
<td>Mongrel</td>
<td>8 weeks</td>
<td>106 weeks</td>
</tr>
<tr>
<td>5</td>
<td>Mongrel</td>
<td>8 weeks</td>
<td>18 weeks</td>
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</tbody>
</table>

TABLE 2. Dogs With Congenital Cleft Palate

<table>
<thead>
<tr>
<th>no.</th>
<th>breed</th>
<th>survival</th>
</tr>
</thead>
<tbody>
<tr>
<td>6</td>
<td>Shetland Sheepdog</td>
<td>22 weeks</td>
</tr>
<tr>
<td>7</td>
<td>Golden Retriever</td>
<td>surviving</td>
</tr>
<tr>
<td>8</td>
<td>Golden Retriever</td>
<td>surviving</td>
</tr>
<tr>
<td>9</td>
<td>Golden Retriever</td>
<td>surviving</td>
</tr>
<tr>
<td>10</td>
<td>Golden Retriever</td>
<td>surviving</td>
</tr>
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</table>

articulated with the mesial surface of the lower canine which prevented further deterioration of the antero-posterior incisor relationship (Figure 4). This resulted in a deep wear groove on the mesial surface of the lower canines. In three dogs the arch alignment of the incisor teeth was collapsed lingually.

The 5 dogs with congenital clefts maintained a normal jaw relationship throughout the period of observation (Figure 5).

Discussion

Atherton (7) observed that the position of the premaxillae in the human with bilateral cleft was similar to that of the normal premaxillae in animals. He found that cleft of the canine primary palate did not affect the position of the
premaxillae and that the protruded premaxillae of the human bilateral cleft merely conformed to the characteristic mammalian growth pattern. What the premaxillo-maxillary sutures allow in the dog in terms of forward premaxillary growth, the bilateral clefts allow in the case of the human premaxillae. Therefore, for experimental purposes, it is possible to regard the premaxillary condition in the newborn dog as a model of the bilateral cleft condition in the human.

In view of the bony continuity between the premaxillary and maxillary parts of the upper jaw in the normal human facial skeleton, it appears that the vomer functions only in a minor supporting role. In the long snouted animals, the supporting stresses of the premaxillary segment are shared amongst the premaxillary sutural articulations, namely the bilateral premaxillo-maxillary sutures and the premaxillo-vomeral suture. The premaxillo-vomeral suture, and hence the vomer itself, provides support of a similar order as that provided by the premaxillo-maxillary sutures. Thus, in the dog, it may be presumed that the vomer makes a more important contribution to the growth and support of the upper jaw than in the normal human. In bilateral cleft lip and palate in man, in

FIGURE 3. Profile view of dog 5, 18 weeks old, showing anterior crossbite indicative of retarded upper jaw growth.

FIGURE 4. Profile view of dog 4, aged 2 years 6 months, showing anterior crossbite indicative of retarded upper jaw growth and locking of upper lateral incisor.
In the artificially cleft dogs there was disturbance of growth in the premaxillary region similar to that in the repaired human bilateral cleft patient. Factors capable of influencing upper jaw growth and resulting in premaxillary retrusion in patients with repaired cleft lip and palate include: tightness of the upper lip, the presence of scar tissue (3, 5), vomerine resection for setting back the protruded premaxillary segment, and vomerine destruction subsequent to its denudation in raising extensive vomero-septal flaps (2).

In analyzing the nature of the antero-posterior growth retardation in these dogs, the effect of a tight lip does not appear applicable. The growth retardation may then be attributed to either the tethering effect of scar tissue at the anterior edge of the artificial cleft or to disruption of the vomer. With regard to the former, there was minimal exposure of raw surface with the technique utilized, and the cleft was not near to the incisors (i.e. 26-30 mm) and the premaxillo-maxillary sutures were not involved.

Lynch and Piel (8), using a technique similar to ours, formed clefts in dogs, removing not only part of the vomer, but also part of the nasal septum. They reported that surgical closure of these clefts caused retardation of subsequent growth in jaw width and attributed this to scar tissue tethering. They did not comment on upper jaw growth retardation in the antero-posterior dimension.

With regard to antero-posterior growth retardation, there is a case for presuming that the difference between our dogs with artificial clefts and those with congenital cleft palate may be the presence of a normal vomer in the latter. However, the determination of whether vomeral disruption or mucosal scar tissue is the actual causative factor may be determined by further research. It is likely that this may result in greater emphasis on the role of the vomer in overall facial growth following cleft palate repair.
ROLE OF VOMER

Summary

Antero-posterior retardation of upper jaw growth became evident in all five dogs with surgically placed clefts and extirpation of the vomer within 8 weeks following surgery. Five control dogs with congenital cleft palate maintained a normal jaw relationship. The evidence indicated that the difference in growth between the two groups may be explained in terms of the role played by the vomer in the growth and support of the upper jaw in the dog.

This observation suggests that the dog may be used as a model for the study of retardation of growth in the antero-posterior dimension and indicates a need for research concerned with the role of the vomer in the human bilateral cleft condition.

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