Development
The formation of the face and oral cavity is complex with fusion of multiple tissue processes in a highly orchestrated fashion. The incisive bones result from the fusion of the paired medial nasal processes. Although one study reported that the maxillary processes in the dog fuse at the midline to produce the majority of the upper lip, it has recently been accepted that it is the nasal processes that fuse with the maxillary processes beginning at the palatine fissures to form the upper lip, alveolar process and primary palate, as in humans. The alveolar process forms through the fusion of the bilateral maxilloincisive suture lines, followed by closure of the rostral nose and the upper lip. Initially, a common oronasal cavity is bounded rostrally by the primary palate and is occupied mainly by the developing tongue. Only after the development of the secondary palate the distinction between the oral and nasal cavities is possible. The secondary palate, which makes up 90% of the hard and soft palates, is formed from the maxillary processes after fusion of the palatal processes of the maxillae with the incisive bone and then with each other and with the vomer. Initially, these shelves are oriented in a vertical position on each side of the developing tongue. As the mandibles grow, the tongue drops down, allowing the palatal shelves to rotate to a horizontal position and grow toward one another and start fusing at the rostral aspects. The palatal shelves also fuse with the primary palate and the nasal septum. The fusion progresses in the rostro-caudal direction, and it is completed by the 12th week in humans. The soft palate forms afterwards. For fusion of the palatine shelves to occur (as applies to fusion of any other processes), elimination of the epithelial covering of the shelves is necessary. As the two palatine shelves meet, adhesion of the epithelia occurs so that the epithelium of one shelf becomes indistinguishable from that of the other, and a midline epithelial seam that consist of two layers of basal epithelial cells forms. This midline seam must be removed to permit ectomesenchymal continuity between the fused processes. Even though the epithelial cells of the seam continue to divide, growth of the seam fails to keep pace with palatal growth so that the seam first thins to a single layer of cells and then breaks up into discrete islands of epithelial cells. The basal lamina surrounding these cells then is lost, and the epithelial cells lose their epithelial characteristics and assume fibroblast-like features. In other words, epithelial cells transform into mesenchymal cells (epitheliomesenchymal transformation). This is a fundamental embryonic process that also is implicated in the invasive behavior of epithelial neoplastic cells.

Defects
Disturbances in the growth of these tissue processes or their fusion may result in the formation of orofacial clefts. Clefting is one of the most common major congenital defects in humans and shows considerable racial and sex variation. In a colony of beagles bred as a pure strain the occurrence of cleft lip (CL) and cleft palate (CP) was 1.1 per 1000 beagles. Clefts are considered very rare in the cat. Although the exact location of the boundary between the primary and secondary palate is still controversial and definitions and classifications of oral clefts may vary in humans and dogs, a cleft palate
(CP) involves the structures of the secondary palate and may be an isolated defect, or be associated with a cleft lip (CL) which is a defect involving the structures of the primary palate. Cleft lip is a defective fusion of the medial nasal process with the maxillary process. Likewise, failure of the palatal shelves to fuse results in CP. Pedigree analysis in Pyrenees shepherd dogs showed that CP only is not genetically distinct from CL with or without CP but is inherited in this breed as a monogenic autosomal recessive trait. Most of the CLs in humans are unilateral (80%), with 70% occurring on the left side. About 70% of unilateral CLs are associated with CPs, and 85% of bilateral CLs are associated with CPs. Cleft lip can be complete (extending into the nostril) or incomplete (not involving nose). Incisor teeth in the cleft can be missing or may be supernumerary. Cleft palate can vary in its severity – from cleft uvula in humans only to complete hard and soft palate cleft. A submucous palatal cleft is also possible, where mucosa is intact, but the defect exists in the underlying musculature of the soft palate. Minor degrees of velar asymmetry are not uncommon in patients with CL, CP, or both. However, gross unilateral hypoplasia of the velum and pharynx is rare and probably a separate entity in humans. Unilateral congenital soft palate hypoplasia is also rare in dogs and its development unknown.

**Etiology**

The causes of CL and CP are still debated. Many facial clefts are isolated anomalies (non-syndromic clefts), however, more than 350 developmental syndromes have been identified in humans that may be associated with CL and CP. The cause of non-syndromic clefts does not follow any simple mendelian pattern of inheritance but appears to be heterogeneous. Thus the propensity for cleft development may be related to a number of major genes, minor genes, and environmental factors that can combine to surpass a developmental threshold. A number of candidate clefting genes and loci have been identified. It has recently been described in boxers, that there is evidence of monogenic autosomal recessive inheritance for a non-syndromic cleft lip and palate. Among some factors, maternal alcohol consumption, cigarette smoking, folic acid deficiency, parental age, corticosteroid use and anticonvulsant therapy have been associated with an increased risk for orofacial clefts in humans.

**Therapeutic decision-making**

Animals with congenital palatal defects are usually presented due to difficulties nursing/drinking; gagging, coughing, or sneezing while eating, and possibly signs of respiratory tract infection.

Surgical correction of palatal defects is preferably delayed until the animals is at least 3-4 months old, or, in selected cases, until permanent dentition has fully erupted allowing maximum amount of tissue available for repair.

Preoperatively, thoracic radiographs are recommended especially in animals with signs of aspiration pneumonia. Routine preanesthetic bloodwork and urinalysis are also recommended. Skull CT should be recommended for all dogs with congenital palatal defects to better assess likely associated craniofacial defects and to better plan the treatment. A tracheal wash and/or nasal bacterial and fungal culture are performed as clinically indicated.
The best chance of success is with the first surgical procedure, but sometimes closure needs to be staged. Extremely large defects will not permit successful closure, and while use of a palatal obturator may be considered as a salvage procedure, grave prognosis in these patients should be considered. Clients should also be counseled concerning the possible heritability factor and the animals should not be used for breeding. Euthanasia may be considered in newborn puppies.

References: