The use of an islandized angularis oris axial pattern buccal flap for the reconstruction of a recurrent cleft palate in a cat

*Het gebruik van een buccale-eilandflaptechniek van de angularis oris voor de reconstructie van een terugkerend gehemeltedefect bij een kat*

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**ABSTRACT**

A 7-month-old male castrated European shorthair cat was presented because of a recurrent cleft of the soft palate. Surgical correction had already been performed twice by a local veterinary practitioner. For the third correction, a pharyngeal overlapping flap was used but again, dehiscence occurred. Finally, the angularis oris axial pattern buccal flap technique, as described in dogs, was applied. The flap covered the defect without any tension, and good vascularization was appreciated during the whole procedure. Because of severe swelling of the intraoral tissues at the end of the procedure, dexamethasone was administered intravenously. Almost instantly after the injection, the cat went into cardiorespiratory arrest, and died shortly after.

**SAMENVATTING**

Een zeven maanden oude, mannelijke, gecastreerde Europese korthaar werd aangeboden omwille van een terugkerend gespleten gehemelte, dat eerder door de eigen dierenarts reeds tweemaal chirurgisch gecorrigeerd werd. Voor de derde correctie werd gebruik gemaakt van een faryngeale overlappende flap maar opnieuw trad dehiscentie op. Uiteindelijk werd de buccaleflaptechniek van de angularis oris, zoals die beschreven is bij de hond, gebruikt. De flap kon zonder spanning over het defect gebracht worden en een goede vascularisatie was zichtbaar gedurende de hele procedure. Omwille van de opgetreden zwelling van de intraorale weefsels op het einde van de ingreep werd intraveneus dexamethasone toegediend. Bijna onmiddellijk na deze injectie kreeg de kat een cardiorespiratoire stilstand en overleed kort daarna.

**INTRODUCTION**

A palatalal cleft creates an open communication between the oral and nasal cavities, making it impossible for a severely affected animal to adequately suck and swallow (Headrick et al., 2004; Kelly and Bardach, 2012; Reiter and Holt, 2012). In the majority of cases, alimentary nasal discharge, coughing, sneezing and malnutrition are the first clinical symptoms (Nelson, 2003; Kelly and Bardach, 2012; Reiter and Holt, 2012). Food particles that enter the nasal cavity initiate an inflammatory reaction, which causes chronic serous to purulent nasal discharge (Reiter and Holt, 2012). Swallowing difficulties predispose the animal to regurgitation and aspiration pneumonia (Griffiths et al., 2001; Nelson, 2003; Reiter and Holt, 2012; Kelly and Bardach, 2012).

Most palatal clefts are congenital, and the condition has been reported in all mammals, humans included. There are different pathomechanisms involved in the disruption of palatogenesis (Van den Berghe et al., 2010). In cats, the reported prevalence of congenital palatoschisis is 0.06% (Noden and de Lahunta, 1985), but presumably, this is an underestimation. The Abyssinian and Siamese cat are clearly overrepresented (Saperstein et al., 1976; Noden and de Lahunta,
1985; Headrick et al., 2004). This might indicate some form of heredity, but until today, it has not been demonstrated. Palatal defects can also occur after tumor resection, trauma, extensive periodontal disease, tooth extractions, chronic severe infection or inflammation (Luskin et al., 2000; Sivacolundhu, 2007).

In severely affected animals, surgical intervention is indicated for the animal’s survival as well as for its wellbeing (Griffiths et al., 2001). There are different surgical options and depending on the location, extent, previous interventions and the surgeon’s experience, some techniques will be preferred. The oral cavity is far from an ideal site for reconstructive surgery, and dehiscence often occurs. Excessive tension on the wound edges in both hard and soft palate reconstructions is the main reason for failure, regardless of which technique is used (Bryant et al., 2003).

The present case report describes the use of the angularis oris axial pattern buccal flap in a young cat with a recurrent palatal cleft, on which previously other surgical techniques had been performed.

**CASE REPORT**

A 7-month-old male castrated European shorthair cat was admitted with a recurrent soft palatal cleft. The owners first noticed nasal discharge shortly after weaning, and presented the kitten to a local veterinary practitioner. A respiratory infection was suspected, and the kitten was treated twice with antibiotics at both occasions without any clinical improvement. Two months later, a cleft soft palate was diagnosed on pharyngeal inspection. Surgical closure was performed twice by the local veterinary practitioner with a one-month interval, but at both occasions, dehiscence occurred within one week’s time.

At admission, the physical examination yielded no abnormalities other than mucopurulent nasal discharge and bilaterally enlarged submandibular lymph nodes. The inspection of the oral cavity was performed under general anesthesia. The cat was premedicated with dexmedetomidine (3 μg/kg IV; Dextromitor®; Orion Corporation), and anesthesia was induced with alfaxalone (2.3 mg/kg IV; Alfaxan®; Jurox). For the maintenance of the anesthesia, a total intravenous anesthesia technique (TIVA) was used, with alfaxalone bolus on effect. A total dose of 7 mg/kg was administered over the entire anesthetic period. The inspection of the oral cavity revealed suture material and scar tissue formation in the midline of the soft palate with a remaining caudal palatoschisis of about 1 cm in length (Figure 1). Rostral of the palatoschisis site, there was a 2-mm diameter oronasal fistula in the scar tissue. The edges of the soft palate showed a distinct inflammatory reaction. The remaining suture material was removed. The cat was treated with pradofloxacin (25 mg/kg SID per os; Veraflox®; Bayer) for ten days, and fed canned food. The cat did generally well and was scheduled for reconstructive surgery.

For the third correction (the first correction after referral), a pharyngeal overlapping flap was used. The cat was premedicated with dexmedetomidine (10 μg/kg IV) and methadone (0.2 mg/kg IV; Comfortan®; Eurovet). Anesthesia was induced with midazolam (0.2 mg/kg IV; Dormicum®; Roche) and ketamine (10 mg/kg IV; Anesketin®; Eurovet). After endotracheal intubation, anesthesia was maintained with isoflurane on effect (Forene®; Abbott) evaporated in 100% oxygen by means of a rebreathing system (Narcose Spiromat 656; Dräger). Ringer’s lactate solution (10 ml/kg/h IV; Hartmann®; B. Braun) was infused during the surgery. The patient was positioned in

![Figure 1](image1.png)

**Figure 1.** Intraoral view of a 7-month-old male castrated European shorthair with a recurrent palatal cleft. A midline scar from the previous surgery is visible, containing a rostral oronasal fistula (probed with an instrument). The most caudal aspect of the soft palate is cleft.

![Figure 2](image2.png)

**Figure 2.** Schematic representation of the pharyngeal overlapping flap. After tonsillectomy, a triangular flap was created and hinged into the defect.
dorsal recumbency, and a left tonsillectomy was performed. The crypt wall was retracted and incised in a lateroventral direction (Figure 2). Next, an incision was made reaching from the crypt wall to the oronasal fistula, incorporating the small bridge of healed tissue. The medial free border of the right palatal shelf was incised on its border so the oral and nasal mucosa could be parted. The obtained triangular flap could then be medially repositioned over the defect with its oral mucosa facing the nasal cavity. The flap was sutured to the nasal mucosa of the right palatal shelf by means of continuous suutures in 5/0 polyglecaprone 25 (Monocryl®; Ethicon, Johnson & Johnson Intl). Finally, the oral mucosa of the right palatal shelf was sutured to the non-epithelialized border of the flap, again by using continuous suutures in 5/0 polyglecaprone 25. The recovery was quick and uneventful, and the cat went home two days after surgery. Liquid food (Convalescence support®; Royal Canin), meloxicam (0.05 mg/kg SID per os; Metacam® 0.5 mg/ml suspension cat; Boehringer Ingelheim Vetmedica), and pradofloxacin (25 mg/kg SID) were continued for one week, after which canned food was introduced. A revisit was planned 14 days postoperatively.

However, ten days after surgery, the cat was readmitted because of recurrent mucous nasal discharge and nasal regurgitation of food. Inspection of the oral cavity revealed partial dehiscence of the flap. Subsequently, an oesophageal feeding tube (Rush®; Teleflex) was placed to avoid further nasal regurgitation and subsequent inflammation. The patient was hospitalized till the next correction of the palatal defect, three days later. This time, the angularis oris axial pattern buccal flap, as described in dogs (Bryant et al., 2003), was used. The premedication consisted of methadone (0.2 mg/kg IV). Anesthesia was induced with midazolam (0.2 mg/kg IV) and ketamine (10 mg/kg IV). The trachea was intubated with a 3-mm internal diameter cuffed endotracheal tube. Because of the expected need for repeated intrasurgical extubation and intubation, it was decided to administer only oxygen through the endotracheal tube (fresh gas flow of 1.5 L/minute) and to maintain anesthesia with non-volatile anesthetics on a patient-based need (three injections of ketamine (10 mg/kg IV) with an interval of approximately 40 minutes between the administrations). The cat was ventilated with intermitted positive pressure ventilation during anesthesia, and the respiratory rate, heart rate, arterial oxygen saturation, the inspired oxygen percentage and end-tidal partial pressure of CO2 were measured continuously and recorded every ten minutes (Cicero; Dräger, Lübeck, Germany). The measurement of the systolic blood pressure was attempted by means of Doppler oscillometry, but was unsuccessful. A three-lead electrocardiogram was attached to screen for arrhythmias (Datex Cardiopac; Datex-Ohmeda, Helsinki, Finland). The body temperature was controlled with a circulating hot water mattress (Gaymar TP500; Gaymar Industries Inc., New York, USA). A Ringer’s lactate solution (10 ml/kg/h IV) was infused during the surgery. Furthermore, meloxicam (0.1 mg/kg SC; Metacam® 2 mg/ml injection suspension cat; Boehringer Ingelheim Vetmedica) was administered. In addition, a bilateral infraorbital and maxillary block...
was performed with bupivacaïne (1.5 mg/kg; Marcaïne® 0.5%; AstraZeneca). The patient was placed in right lateral recumbency, and the hair was clipped starting at the midline of the upper lip along the zygomatic arch as far as the caudal border of the temporomandibular joint and below the mandibula to the ventral midline. The skin was aseptically prepared, and the mouth was rinsed with a chlorhexidine solution (1/40; Ecutan® 5%; Ecuphar). A 5-cm-long skin incision was made starting at the lip commissure in a caudal direction (Figure 3). The subcutis was bluntly dissected dorsally as well as ventrally. Two parallel incisions, 1.5 cm apart, were made through the subcutis, the muscles and the buccal mucosa in a rostrocaudal direction starting from the lip commissure and ending at the cranial border of the m. masseter. Next, a dorsoventral cut was made through the buccal mucosa, connecting the most caudal aspect of the previous incisions. This resulted in an island flap. Subsequently, the ramus of the left mandible was undermined ventrolaterally to lead the flap to the oral cavity. The flap was transposed to the palatal defect with its buccal mucosa facing the nasal cavity. It was fixed with single interrupted sutures in 5/0 polyglecaprone 25. Next, the buccal mucosa, muscles, subcutis and cutis of the donor place were apposed with continuous sutures in 5/0 polyglecaprone 25. The flap covered the defect without any tension (Figure 4). A good vascularization of the flap was appreciated during the whole procedure. Because of severe swelling of the intraoral tissues at the end of the anesthetic period, dexamethasone (0.1 mg/kg IV; Rapidexon®; Eurovet) was administered. Almost instantly after the injection, the cat suffered a cardiorespiratory arrest. External cardiac massage (cardiac pump technique) and manual intermittent positive pressure ventilation were started immediately. Adrenalin (0.01 mg/kg IV; Adrenaline®; Sterop) was administered three times with an interval of two to three minutes in combination with naloxone (0.02 mg/kg IV; Naloxon®; B. Braun). Since crackled lung sounds were heard on lung auscultation, furosemide (2 mg/kg IV; Dizam®; Intervet) was administered twice at an interval of 15 minutes but all these efforts had only a temporarily effect. The cat eventually died.

DISCUSSION

There still is a lot of controversy on the optimal management of a cleft palate (Nguyen et al., 1993). In human medicine, the maintenance of the normal function of the palatine muscles is essential because of their role in speech development. Extensive research into surgical approaches has been performed, because every human patient with a cleft palate will eventually be scheduled for surgery. This is definitely not the case in veterinary medicine, where most patients are euthanized rather than treated (Griffiths et al., 2001). The decision to correct a cleft palate in a pup or kitten has to be well considered. There are technical, financial, ethical as well as practical aspects that should be taken into account. In the case described, the owners were very motivated to proceed.

In animals, the function of the palatine muscles is not of major concern, and subsequently the goals for reconstructive surgery differ greatly (Sylvestre et al., 1997; Griffiths et al., 2001). In the authors’ opinion, veterinary surgeons have to remain critical when considering extrapolations from human literature.

It is better to wait until the age of three to four months when there is a higher availability and quality of tissue and likewise a greater chance of success (Harvey and Emily, 1993; Kelly and Bardach, 2012; Reiter and Holt, 2012). This recommendation originates from experience with dogs but the same principles apply to cats.

The nasal discharge in this cat was noticed shortly after weaning. Probably, the kitten had already shown nasal discharge in the litter, but this information was not available. Despite its non-responsiveness to antibiotics, a cleft palate was only diagnosed at the age of four months. This is not uncommon, since it is very difficult to adequately inspect the oral cavity without sedation, and especially, defects on the caudal aspect of the palate can easily be missed (Nelson, 2003). Therefore, a lot of the affected animals are first treated for assumed respiratory infections while in fact they have a cleft palate. Inadequate diagnosis may be one of the reasons why cats are clearly underreported in comparison with dogs.
degree from rhinitis and possibly from a secondary (aspiration) pneumonia (Luskin et al., 2000; Griffiths et al., 2001; Nelson, 2003; Kelly and Bardach, 2012; Reiter and Holt, 2012). Prior to any surgical intervention, the secondary consequences of the palatal defect should be treated accordingly. It may therefore be necessary to place an esophageal or gastric feeding tube in combination with an appropriate antibiotic treatment (Kelly and Bardach, 2012). In this case, prior to the third surgical intervention, the patient was treated with suture removal and antibiotics to reduce the inflammatory process at the soft palate. An esophageal feeding tube was placed prior to the fourth correction to keep the oral cavity free from food during the pre- and postoperative period.

The oral cavity is far from an ideal site for reconstructive surgery. Firstly, there is only a limited availability of tissue for the reconstruction of palatal clefts, and the tissues of the oral cavity do not tolerate a lot of manipulation (Pavletic, 1999). Secondly, there are several predisposing factors that may lead to dehiscence. Excessive tension on the wound edges in both hard and soft palate reconstructions is the main reason for failure, regardless of which technique is used (Bryant et al., 2003). After the correction of a cleft soft palate, the palatine muscles tend to draw the free border of the soft palate in a rostromedial direction, which causes a lot of tension on the suture line (Sager et al., 1998). Other factors which may delay healing and predispose to dehiscence include inadequate debridement, insufficient blood supply, mechanical stress during mastication and swallowing, tissue damage by previous surgical intervention(s) or radiation therapy, infection or inflammation and persistent or recurrent neoplasia (Bryant et al., 2003). Repeated failure leads to scar formation and concomitant wound contraction that makes the defect even bigger (Ninkovic et al., 1997). Although in the present case, the flap was sutured over the defect without tension, many of these factors were present, and could be responsible for the dehiscence.

A temporary tracheostomy can be useful to maintain a patent airway during the surgical manipulation of the oral cavity. However, this approach is not common in cats, and a high risk for complications has been reported in this species (Guenther-Yenke and Rozanski, 2007). A classical endotracheal intubation was performed in the cat in the current case report, with the intention of repeated ex- and intubation. Due to the shallow aspect of the feline oral cavity however, the presence of the endotracheal tube was never found to be troublesome during the surgical reconstruction of the palate.

At the moment of writing, there are no publications on the use of the angularis oris axial pattern buccal flap for palate reconstruction in cats, although two studies exist on the feline angularis oris cutaneous flap (Milgram et al., 2011; Bradford et al., 2011). In dogs, a thick, robust buccal flap with a good vascularization and mobility can be created (Bryant et al., 2003). However, harvesting the angularis oris flap implies a rather complicated dissection. The technique described in dogs (Bryant et al., 2003) was slightly modified in this cat. Instead of making a bridging incision, the vascular pedicle was placed in a submucosal tunnel behind the mandibular molar, while undermining the mandible. There was obviously less tension on the vascular strand of the island flap, and it seemed to be more protected from trauma during mastication. This pathway appeared to be a more convenient way to reach caudal palatal clefts by avoiding the palatoglossal arch and thus by reducing interference with swallowing.

In dogs, the ability to open the mouth postoperatively is reported to be limited (Bryant et al., 2003). Since in this case, the patient died in the immediate postoperative period, this could not be evaluated. There was indeed less buccal tissue left to open the mouth, but it is not known whether this would have been clinically relevant. In contrast to dogs, the precise vascularization of the buccal tissue has not yet been investigated in cats. As it is of major concern to preserve adequate vascularization, further anatomical studies are needed to correctly dissect the flap and thus to optimize the technique in cats. Additionally, the maximum dimensions of the flap in cats should be investigated. For more extensive defects, this flap will probably need to be combined with other techniques as cats, in comparison with dogs, have less buccal tissue.

The cat in this case report unfortunately did not survive the perioperative period. After disconnection from the anesthetic machine, but still intubated and positioned on the surgery table, the patient suffered a cardiorespiratory arrest immediately following the IV dexamethasone injection. In the authors’ opinion, cardiac failure due to an unidentified pre-existing cardiac pathology, intraoperative cardiac or cerebral ischemia (possibly due to the cumulative effects of several anesthetic episodes in a short time interval) eventually resulting in cardiac arrest at the time of disconnection of the oxygen supply and anaphylaxis following the corticosteroid injection are possible causative factors for the sudden postoperative death of this patient. Although an anaphylactic reaction cannot be absolutely confirmed in the present case, several factors contribute to the likeliness of this diagnosis. First, the timing of the fatal event was very indicative for a relation to the injection of the corticosteroid. Secondly, the anesthesia had been uneventful until the end of the procedure, and thirdly crackled lung sounds were observed after the cardiorespiratory arrest, suggesting the presence of pulmonary edema. Although pulmonary edema might have been present already before the injection of the corticosteroid, the peak airway pressures during IPPV, necessary to achieve a normal tidal volume, were acceptable (never more than 9 cm of H_2O), whereas they would be expected to be increased in edematous lungs. Allergic anaphylaxis is a generalized type I hypersensitivity
reaction characterized by the excessive release of inflammatory mediators from mast cells, eosinophils and basophils. The overwhelming release of histamine results in fulminant vasodilation and consequently in hypotension (Estelle et al., 2009). The clinical signs of anaphylaxis differ among species (Tizard, 2009), but mainly the lungs are affected, resulting in severe dyspnea from pulmonary edema. Anaphylaxis has mostly been reported in combination with antibiotics or vaccines (Keller and Bataller, 2000). There is only one report on a similar reaction after the administration of dexamethasone in a dog (Schaer et al., 2005).

It is a life-threatening condition that invariably leads to death when not treated instantly. Although in the current case, the cardiopulmonary arrest was treated according to standard resuscitation protocols, the event was fatal.

CONCLUSION

The islandized angularis oris axial pattern buccal flap seems an appropriate and feasible technique for the correction of palatal clefts in cats. Further anatomical studies in cats are needed to define the vascularization pattern and the maximal extent of the flap.

REFERENCES


