Brachycephalic Obstructive Airway Syndrome

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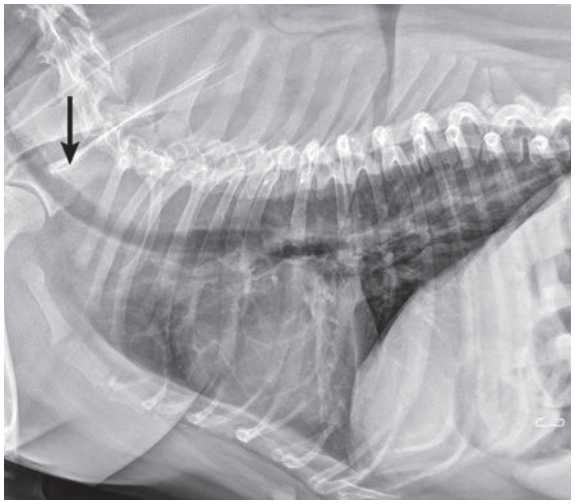
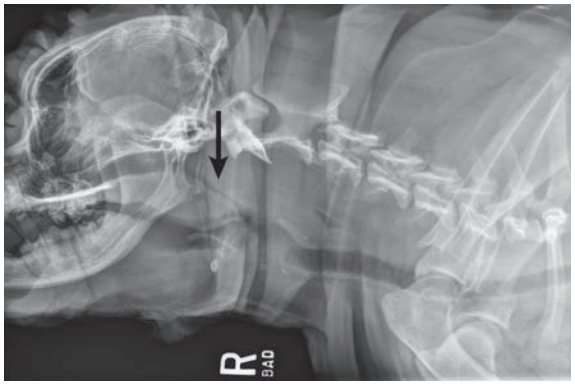
A 2-year-old spayed French bulldog is presented in respiratory distress. The patient was being exercised outside and experienced progressive stertor and dyspnea prior to presentation. She has a history of snoring, exercise intolerance with inspiratory effort, and occasional weekly regurgitation. Brief physical examination reveals moderately stenotic nares and a respiratory rate of 60 breaths per minute with loud upper airway stertor. Heart rate is 160 bpm, temperature is 103.5°F (39.7°C), and mucous membranes are cyanotic. The patient is immediately placed in a cooled oxygen cage for 10 minutes and monitored closely. Once the patient is stable, an intravenous catheter is placed and a sedative is administered. She is then placed back in the oxygen cage. Based on the patient's breed and history, thoracic radiography and a sedated airway examination are performed.

Thoracic radiographs reveal a mildly hypoplastic trachea and no evidence of aspiration pneumonia (***Figure 1***, next page). A lateral cervical radiograph shows an elongated, thickened soft palate partially obstructing the laryngopharynx and nasopharynx (***Figure 2***, next page). Airway examination confirmed the elongated soft palate obstruction and tonsillar eversion (***Figure 3***, next page). Everted laryngeal saccules are also present. Brachycephalic obstructive airway syndrome (BOAS) is diagnosed. Folded flap palatoplasty, laryngeal sacculectomy, and alarplasty are recommended after improvement in airway swelling associated with the acute episode.

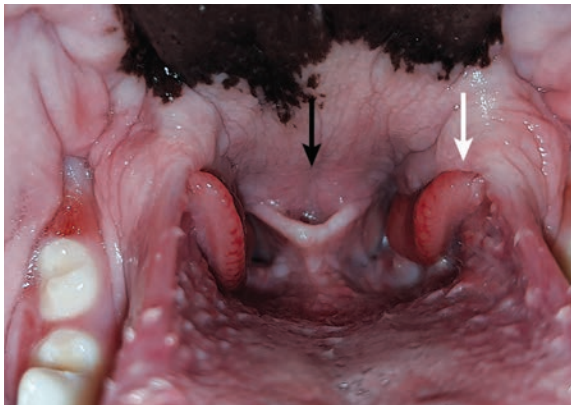
BOAS = brachycephalic obstructive airway syndrome



▲ **FIGURE 1** Left lateral thoracic radiograph demonstrating mildly hypoplastic trachea (***arrow***)



▲ **FIGURE 2** Right lateral cervical radiograph demonstrating an elongated, thickened soft palate partially obstructing the laryngopharynx and nasopharynx (***arrow***)



▲ **FIGURE 3** Elongated soft palate (***black arrow***) extending caudal to the everted palatine tonsils (***white arrow***)

Which of the following drugs would be appropriate for this patient?

Based on the information provided, how would you grade the following drugs and why?

RED = do not use YELLOW = proceed with caution GREEN = safe

Dexmedetomidine	RED	YELLOW	GREEN
Butorphanol	RED	YELLOW	GREEN
Acepromazine	RED	YELLOW	GREEN
Hydromorphone	RED	YELLOW	GREEN
Dexamethasone	RED	YELLOW	GREEN
Carprofen	RED	YELLOW	GREEN
Omeprazole	RED	YELLOW	GREEN
Cisapride	RED	YELLOW	GREEN
Metoclopramide	RED	YELLOW	GREEN
Sucralfate	RED	YELLOW	GREEN

Did you answer?

The following represents the best responses based on drug metabolism, pharmacokinetics, species, diagnostic differentials, clinical and laboratory data, and other pertinent findings.

DEXMEDETOMIDINE

CORRECT RESPONSE 

Dexmedetomidine is an α_2-adrenergic agonist commonly used for short periods of sedation or analgesia and can cause respiratory depression, resulting in decreased end tidal volume and respiratory rate.[1] Administration of low doses or CRIs may minimize these undesired effects. A recent study concluded that dexmedetomidine produces more sedation but has similar recovery quality as acepromazine when combined with methadone as premedication in dogs undergoing BOAS surgery.[2]

BUTORPHANOL

CORRECT RESPONSE 

Butorphanol is an antitussive, κ-agonist, μ-antagonist opioid that causes sedation and mild analgesia. It is an appropriate sedative to use in patients with BOAS because it causes minimal respiratory depression as compared with other sedatives. Butorphanol is commonly used in emergency settings to provide sedation to patients in respiratory distress. Careful monitoring (eg, measuring respiratory rate, oxygen saturation) is recommended when butorphanol is combined with other sedatives.

ACEPROMAZINE

CORRECT RESPONSE 

Acepromazine is a phenothiazine sedative/tranquilizer commonly used to relieve anxiety[3] and is extremely useful as a sedative/tranquilizer in BOAS patients with upper airway obstruction because it has minimal effects on respiratory function.[1] This drug has negligible analgesic effects; therefore, an opioid can be considered if pain management is also required. Acepromazine has hypotensive effects and use should be avoided in dogs with concurrent cardiac disease. Careful monitoring of respiratory rate and effort is recommended when acepromazine is combined with other sedatives.

HYDROMORPHONE

CORRECT RESPONSE 

Hydromorphone is a full μ-opioid agonist used for sedation and analgesia. In dogs, hydromorphone initially increases respiratory rates (panting), which is counterproductive in patients with BOAS and may worsen increased airway turbulence. Vomiting is also an adverse effect of hydromorphone. Hydromorphone is not recommended in dogs with BOAS because of the high prevalence of GI signs and dysmotility disorders. An alternative opioid medication (eg, methadone) with fewer respiratory and GI effects would be a safer choice.

BOAS = brachycephalic obstructive airway syndrome

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DEXAMETHASONE

CORRECT RESPONSE 

Patients with BOAS typically have compressed anatomy that can result in increased negative pressure on inspiration, inflammation and stretching of the pharyngeal tissues, and eventual obstruction, especially after excitement and activity.[4] Glucocorticoids (eg, dexamethasone) can be used to treat pharyngeal and laryngeal swelling in an emergency setting. This drug is also useful for controlling postoperative swelling after corrective surgery. Dexamethasone is a fast-acting corticosteroid. Most adverse effects are typically associated with long-term administration, which should not be necessary in patients with BOAS because use is limited to treating urgent distress episodes and perioperative inflammation.

CARPROFEN

CORRECT RESPONSE 

Dogs with BOAS that are in respiratory distress in an emergency setting are typically given a glucocorticoid to control airway inflammation. Concurrent administration of any NSAID (eg, carprofen, meloxicam) is contraindicated because of increased risk for GI ulceration and/or perforation. Analgesic alternatives can include an opioid or gabapentin if necessary.

OMEPRAZOLE

CORRECT RESPONSE 

GI signs have been reported in up to 77% of brachycephalic dogs.[5] Proton-pump inhibitors (eg, omeprazole) are indicated in dogs with BOAS because of the high prevalence of concurrent GI signs. Omeprazole and other substituted benzimidazole proton-pump inhibitors (eg, esomeprazole, pantoprazole) are the most potent antisecretory drugs and are reported to reduce gastric acid secretion by 80% to 95%.[6] These drugs are significantly more effective than H_2-receptor antagonists (eg, famotidine) in increasing gastric pH and enhancing healing of acid-related tissue injuries in humans.[7,8] Some dogs are sensitive to omeprazole and may have GI disturbance (eg, anorexia, nausea, diarrhea, flatulence). A histamine antagonist may be preferable in these cases.

CISAPRIDE

CORRECT RESPONSE 

Cisapride is a serotonin 5-HT_4-receptor agonist that is a prokinetic agent commonly used to promote GI motility. Cisapride has the broadest spectrum of action of the prokinetic agents and causes increased dose-dependent activity at all GI sites.[6] Brachycephaly has been significantly associated with esophageal dysmotility, prolonged esophageal transit time, gastroesophageal reflux (GER), and hiatal herniation.[9] Another study demonstrated that preanesthetic administration of cisapride with a proton-pump inhibitor decreases the number of reflux events in anesthetized dogs.[10] Addition of a prokinetic may help decrease the risk for aspiration pneumonia in dogs with chronic regurgitation. Hiatal hernia should be a differential in patients with regurgitation that is unresponsive to BOAS surgery and prokinetic medication.

BOAS = brachycephalic obstructive airway syndrome
GER = gastroesophageal reflux

METOCLOPRAMIDE

CORRECT RESPONSE 

Metoclopramide is a GI prokinetic and antiemetic agent that antagonizes dopamine receptors in the CNS and peripherally acts as both an antidopaminergic agent and a direct and indirect stimulator of cholinergic receptors.[6] Muscarinic receptor activity, dopamine receptor antagonist activity, and 5-HT_3 receptor activity collectively contribute to the prokinetic action of metoclopramide.[6] Although metoclopramide can act as an antiemetic agent, a study demonstrated that when administered perioperatively, metoclopramide does not influence the reduction of GER incidence.[11] Some studies have shown that typical doses of metoclopramide have no effect on GER incidence in anesthetized patients and on patient outcomes, but higher doses (eg, bolus injection of 1 mg/kg, followed by CRI of 1 mg/kg/hour) have been shown to reduce perioperative GER in anesthetized dogs.[12] A recent study included metoclopramide in a perioperative protocol for dogs with BOAS.[13] The protocol was found to decrease the incidence of postoperative regurgitation. Atropine and opioid analgesics have anticholinergic effects and thus can antagonize the action of metoclopramide.[6]

SUCRALFATE

CORRECT RESPONSE 

Sucralfate is an oral disaccharide aluminum hydroxide product that is a GI mucosal protectant commonly used in patients with severe gastritis, esophagitis, and gastric ulceration.[6] Sucralfate binds to and protects damaged epithelial cells and increases mucosal blood flow[7] and is most effective in acidic stomach environments. Therefore, sucralfate should be administered at least 30 minutes before administration of antacid medications and on an empty stomach at least one hour before feeding. Because of the significant association between brachycephaly and GER, sucralfate can be considered for the treatment of suspected or diagnosed esophageal erosions or ulcers.[9] Caution is warranted in dogs with BOAS that have a history of gagging or regurgitation because of the risk for aspiration.

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