Respiratory system

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Respiration includes four processes:

1. Pulmonary ventilation (air in/out, gas exchanged in lungs)

2. External respiration (gas exchanged between blood and alveoli in lungs)

3. Transport of respiratory gases (transport of oxygen and carbon dioxide)

4. Internal respiration (gas exchange between blood and tissues).

Functional Anatomy of Respiratory System (Nose, pharynx, larynx, trachea, bronchi, lungs, and alveoli) Consists of two zones:

1. **Respiratory zone** - actual site of gas exchange (bronchioles, alveolar ducts, alveoli)
2. **Conducting zones** - all other respiratory passageways

A. **Nose**. Functions:

- Provides airways for respiration
- Moistens and warms entering air
- Filters inspired air and cleanses it
- Serves as resonating chamber for speech
- Houses the olfactory (smell) receptors
- Nose structure
- External nose composed of dense fibrous connective tissue and cartilage attached to nasal, frontal, and maxillary bone
- External nares (nostrils) lead into nasal cavity which is divided by the nasal septum (composed of hyaline cartilage and vomer bone)
- Nasal cavity entrance contains vestibule with vibrissae

- Nasal cavity lined with olfactory mucosa and respiratory mucosa (mucous and serous glands) and contains lateral projections (superior, middle, inferior nasal conchae)
- Internal nares in posterior nasa cavity lead into the nasopharynx
- B. **Pharynx** Connects nasal cavity and mouth to larynx and esophagus (throat) Three regions:
- **Nasopharynx** - lined with pseudostratified epithelium and houses the pharyngeal tonsil (adenoids)
- **Oropharynx** - lined with stratified squamous epithelium, located posterior to oral cavity, include archway (fauces) between uvula and epiglottis, and contain palantine and lingual tonsils
- **Laryngopharynx** - lined with stratified squamous epithelium, common passageway for food and air, posterior to epiglottis
- C. **Larynx** Voice box attached to hyoid superiorly and inferiorly with trachea Composed of corniculate, arytenoid, cricoid, and thyroid cartilage
- Functions: Provide open airway;Act as switching mechanism (epiglottis) to route air and food and Voice production (vestibular fold or false vocal cord and vocal fold or true vocal cord)
- D. **Trachea** Descends from larynx through neck into mediastinum and ends by dividing (at carina) into two primary bronchi ;Walls (mucosa, submucosa, hyaline cartilage and adventitia)and lined with pseudostratified epithelium
- E. **Bronchi and Subdivisions (bronchial tree)**
- **Conducting zone structures** - right and left primary (principle) bronchi branch into:
- Secondary bronchi (3 on right 2 on left) which branch into
- Tertiary (segmental) bronchi which branch into 4th, 5th, etc... 23 orders
- Until bronchioles then terminal bronchioles

- Tissue composition of walls of primary bronchi is same as trachea but decreases in size of conducting tubes, then increase in change in composition with each subsequent subdivision:
- Cartilage rings replaced by irregular plates of cartilage
- F. **Lungs and Plurae**
- i. Lungs Three lobes on right and two lobes on left ;Occupy the entire thoracic cavity, suspended in its own pleural cavity connected to the mediastinum by vascular and bronchial attachments (roots); Lung tissue also referred to as stroma
- ii. Pluera Thin, double-layered serosa Parietal pluera lines the thoracic cavity and visceral pleura covers external lung surface
- III. **Mechanisms of Breathing (Pulmonary ventilation - inspiration and expiration)**
- A. **Pressure relationships in the thoracic cavity**
- Respiratory pressures are always given relative to atmospheric pressure
- Thoracic cavity
- **Intrapulmonary pressure** - pressure within the alveoli of the lungs; always equal to atmospheric pressure
- I**ntrapleural pressure** - pressure within pleural cavity; always 4 mm Hg less than atmospheric pressure in alveoli; results from factors holding lungs to thorax wall and those acting to pull lungs away from wall
- Negative pressure in the intrapleural space results from the interaction between factors acting to hold the lungs to the thorax wall and factors acting to pull the lungs away
- factors holding lungs to thorax walls:
- Adhesive force (surface tension) created by pleural fluid in pleural cavity

- Positive pressure within lungs (interpleural pressure is always greater than intrapleural)
- Atmospheric pressure acting on thorax (atmospheric pressure pushing on chest is greater than intrapleural pressure, therefore thorax wall tends to be "squeezed" in)
- Factors forcing the lungs away from thorax wall
- Natural recoil tendency of lungs (due to elasticity)
- Surface tension of the fluid film in alveoli (draws the alveoli to smallest possible dimension)
- Question: what is the role of the diaphragm and the role of internal/external costal muscles in regulating pressure differentials?
- B. **Pulmonary Ventilation: Inspiration and Expiration**
- Volume changes lead to pressure changes, which lead to the flow of gases to equalize the pressure
- $P_1V_1=P_2V_2$ (Boyle's Law) pressure of gas is inversely proportional to volume at a constant temperature
- <u>Inspiration</u>
- Inspiratory muscles (external intercostal) contract; diaphragm descends; rib cage rises
- Thoracic cavity volume increases
- Lungs stretched; intrapulmonary volume increases
- Intrapulmonary pressure decreases
- Air (gases) flows into lungs down its pressure gradient until intrapulmonary pressure is zero (equal to atmospheric pressure)
- Ribs elevated and sternum flares as external intercostal contract; diaphragm moves inferiorly during contraction

- Expiration
- Inspiratory muscles relax (diaphragm rises, ribcage descends due to gravity)
- Thoracic cavity volume decreases
- Elastic lungs recoil passively; the intrapulmonary volume decrease
- Intrapulmonary pressure rises
- Air (gases) flows out of lungs down its pressure gradient until intrapulmonary pressure is zero
- Ribs and sternum depressed as external intercostal relax; diaphragm moves superiorly as it relaxes
- C. **Respiratory Status**
- i. Respiratory Volumes: (measured by spirometer)
- (TV) **tidal volume** - amount of air inhaled or exhaled with each breath under resting conditions
- (IRV) **inspiratory reserve volume** - amount of air that can be forcefully inhaled after a normal tidal volume inhalation
- (ERV) **expiratory reserve volume** - amount of air that can be forcefully exhaled after a normal tidal volume exhalation
- (RV) **residual volume** - amount of air remaining in the lungs after a forced exhalation
- ii. Respiratory Capacities:
- (TLC) **total lung capacity** - maximum amount of air contained in lungs after a maximum inspiratory effort (TLC = TV + IRV + ERV + RV)
- (VC) **vital capacity** - maximum amount of air that can be expired after a maximum inspiratory effort (VC = TV + IRV + ERV)
- (IC) **inspiratory capacity** - maximum amount of air that can be inspired after a normal expiration (IC = TV + IRV)

- (FRC) **functional residual capacity** - volume of air remaining in the lungs after a normal tidal volume expiration (FRC = ERV + RV)
- IV. **Transport of Respiratory Gases by Blood**
- A. **Oxygen**
- Molecular oxygen is carried in blood in two ways:
- Bound to hemoglobin within red blood cells (98.5%)
- Dissolved in plasma (1.5%)
- Oxyhemoglobin (HbO_2) / doexyhemoglon (Hhb)
- Hemoglobin - composed of four polypeptide chains each containing and iron (heme) group;
- Oxygen binding is result of "cooperation"; first oxygen bound to one heme causing change in shape of hemoglobin so that the other peptides will bind more oxygen
- affinity for oxygen increases with each oxygen bound until saturation; conversely, release of oxygen from hemoglobin increases with each oxygen released
- B. **Carbon Dioxide**
- Active body cells produce about 200ml of carbon dioxide/minute(released by lungs)
- Carbon dioxide is transported in blood in three forms:
- Dissolved in plasma (7% to 10%), remainder enter RBCs
- Chemically bound to hemoglobin in RBCs (20% to 30%); carried within RBCs as carboaminohemoglobin; carbon dioxide binds to amino acids not heme, therefore does not compete with the oxyhemoglobin transport
- As a bicarbonate ion in plasma (60% to 70%), carbon dioxide is converted to bicarbonate ions and transported in plasma

- C. **Respiratory Center**
- Medulla oblongata
- Inspiratory center - sets the respiratory rhythm
- Pons
- Contains centers that influence and modify the activity of the medullary neurons
- Pneumotaxic center continuously transmits inhibitory impulses to the inspiratory center of the medulla
- Apneustic center continuously transmits stimulatory impulses to the inspiratory center of the medulla
- Clinical Terms
- **Eupnea** - quiet breathing
- **Tachypnea** - rapid breathing
- **Costal breathing** - shallow
- **Diaphragmatic breathing** - deep
- **Atelectasis** - collapse or incomplete expansion of lungs
- **Cheyne-Stokes respiration** - irregular breathing (increase/decrease in depth and rapidity)
- **Laryngitis** - inflammation of the vocal cords
- **Pleurisy** - inflammation of the pleura
- **Infant respiratory distress syndrome (IRDS)** - insufficient surfactant produced, surface tension forces collapse of the alveoli
- **Hypoxia** - inadequate amount of oxygen circulated to no oxygen delivered to body cells
- Hypoxic - lack of oxyen getting from lungs to heart

- Anemic - to few RBCs, or RBCs with inadequate hemoglobin
- Ischemic (stagnant) - blood circulation is impaired or blocked
- Histotoxic - interference with gas exchange betweem capillaries and target tissue/organ
- **Hypercapnia** - increase in carbondioxide levels in cerebrospinal fluid, causing pH to decrease, exciting chemoreceptors to make synapses with respiratory centers; depth and rate of breath increases (hyperventilation)
- **Hypocapnia** - apnea (breathing cessation)
- **Chronic Obstructive Pulmonary Disease (COPD)**, common features:
- Patients with history of smoking
- Dyspnea - difficult or labored breathing
- Coughing and frequent pulmonary infection
- Will develop respiratory failure
- COPDs:
- **Obstructive emphysema** - permanent enlargement of the alveoli, deterioration of alveolar walls
- Chronic inflammation leads to lung fibrosis (lungs lose their elasticity)
- Victims sometimes called "pink puffers" - breathing is labored, but doesn't become cyanotic because gas exchange remains adequate until late in the disease
- **Chronic bronchitis** - inhaled irritants lead to chronic excessive mucus production by the mucosa of lower respiratory passageways and inflammation and fibrosis of that mucosa

Victims sometimes called "blue bloaters" - hypoxia and carbon dioxide retention occur Rhinology is an incision into the nasal cavity.

Tracheotomy is an incision through the tracheal wall.

Tracheostomy is the creation of a temporary or permanent opening into the trachea to facilitate airflow.
tracheostoma The permanent tracheostomy opening
Tracheal resection and anastomosis consists of removal of a segment of trachea and reapposition of the divided tracheal ends.
Ventriculocordectomy (debarking or devocalization) is resection of the vocal cords
Indications for Upper Respiratory Tract Surgery

• Brachycephalic syndrome –Devocalization- Laryngeal collapse Laryngotracheal trauma -Laryngeal paralysis-Tracheal collapse-Laryngeal masses -Tracheal masses- Nasal masses or infection- Nasal trauma and • Foreign bodies

• Congenital abnormalities

NASAL ASPERGILLOSIS(sinonasal aspergillosis) is characterized by infection of the nasal cavity and often the frontal sinus by large colonies of fungal hyphae. Masses formed by the fungal hyphae are sometimes called aspergillomas. Trephination is the removal of a circular piece of bone, especially of the skull. Nasal aspergillosis is a relatively common disease in dogs and clinically may mimic nasal neoplasia It is the second most common cause of chronic nasal discharge in dogs, following nasal neoplasia. Nasal aspergillosis should not be confused with systemic aspergillosis; the latter is almost uniformly fatal. The nasal form usually remains confned to the nasal cavity or the paranasal sinus, where it causes metastasis have shortened median survival times. The prognosis for carcinoma is better than that for sarcoma, and adenocarcinoma appears to have the best overall prognosis. It is unlikely that therapy will result in cure in most dogs, and more successful local control may lead to increased detection of metastasis. Conversely, the prognosis for cats with lymphoid neoplasia of the nasal cavity appears good.

Affection of the nostrils and nasal cavity

The need for diagnosis and surgical treatment of respiratory disease in cattle is not common. However, several disorders are well documented and are most expediently addressed with surgical therapy. Although some of these disorders are congenital malformations, the majorities are acquired through trauma or are infectious in origin. Thorough physical examination can often determine an accurate diagnosis, which improves the success rate of the treatment. Several ancillary diagnostic exercises can assist physical examination findings to further direct specific treatment selection.

Physical examination

Clinical signs can often dictate the specific target of a physical examination. However, any thorough examination should evaluate the respiratory and cardiovascular systems. The
focus and depth of the examination should be on both the morphology of the relevant anatomic structures and evidence of physiologic dysfunction of each of these systems. When the animal is approached, indications of respiratory system dysfunction can be appreciated by noting respiratory characteristics such as rapid or shallow breaths, coughing, and open-mouth breathing, which can all be signs of impaired ventilation. Determination of respiratory rate is not as important as noting the pattern and ease of respiratory effort. Upper respiratory sounds can increase or be abnormal in cases of upper airway obstruction. Grunts can be heard at the end of a forceful expiration in cattle suffering from severe pneumonia, pain, or pneumothorax. In addition,
other vital signs such as heart rate and body temperature are very important in assessing a patient. These can often help determine the likelihood of

involvement of a septic process.
Body condition and knowledge of the duration of the problem can help in determining potential success when considering surgical therapy.
The upper respiratory tract can be evaluated relatively readily via palpation, percussion, and auscultation. The nares of cattle should be moist and readily and regularly cleaned by the tongue; therefore the presence of even serous nasal discharge is abnormal. The openings of the nares do not flare or move as much as those of horses. Patency of the nares and nasopharynx can be readily accomplished by placing cupped hands in front of the nares and assessing the volume of air flow or, in cold climates, observing the condensed expired air. Inspiration is difficult to assess, but the relative volume of expired air can be readily determined. Symmetry of air flow may be the most important aspect of exhalation to be determined at the nares.
The nature of any fluid at the nares should be examined. Exhaled air should be evaluated for odors that may be indicative of an infectious and/or necrotic process. Any abnormal sounds associated with inspiration or expiration should also be noted. Audible whistles, gurgles, or other abnormal sounds can be indicative of upper airway compromise.
Facial symmetry should be assessed, and any distortion may indicate an underlying disease disrupting upper airway anatomy. Percussion of the nasal passages and paranasal sinuses can be performed with fingers or with a plexometer. Placing a stethoscope over the percussed region may help determine the presence of abnormal tissue or fluid presence in otherwise air-flled spaces. The sounds can be augmented by opening the mouth during percussion. The ventral aspect of the head should be visually examined and palpated. The intermandibular space should be examined for swelling and painful response to palpation. Congenital malformation, trauma associated with balling gun injury, or foreign body penetration may result in a perilaryngeal mass that can compromise the upper respiratory tract at the pharynx, larynx, and proximal trachea. These can be suspected by detection of proximal cervical swelling The cervical locations of palpable lymphatic tissues should be closely examined visually and by hand. The trachea should be auscultated with attention to airflow or abnormal sounds indicating the presence of fluid.
The trachea and tracheal region should be palpated. Subcutaneous crepitation should be noted. Tracheal sensitivity to pressure and the ease of eliciting a

cough should also be determined.

The thoracic cavity should be evaluated by observing the overall condition of the patient as well as the basic movements of the thoracic wall during respiratory efforts. A decline in body score and abnormal respiratory movements can indicate a primary disease process in the thorax. The dorsum is palpated for subcutaneous emphysema. Auscultation is important in evaluating the thorax. Careful attention and assessment of ventilation and lung sounds should be performed. This should include notation of the location or regionalization of abnormal findings. Abnormal lung sounds can indicate different pulmonary diseases, which may or may not need surgical therapy. Thoracic and cardiac surgical diseases are suspected by the absence of respiratory sounds or muffled normal sounds, which indicates a need for further diagnostic procedures. In conjunction with auscultation, percussion of the thoracic wall should be performed. A normal bovine thorax should have air-fled resonance throughout the thorax except for sites of closest cardiac association. Fingers or a plexometer used in a dorsal-to ventral direction in the intercostal spaces can help detect loss of resonance associated with accumulated fluid or solid tissue

SURGICAL ANATOMY

The nasal cavity extends from the nostrils to the nasopharyngeal meatus and is separated into two halves by the *nasal septum* .The septum is mostly cartilaginous but also has bony and membranous portions. The *nasal conchae* develop from the lateral and dorsal walls of the nasal cavity.

The air passages between the conchae are known as the *meatus.* The *paranasal sinuses* include a maxillary recess, a frontal sinus, and a sphenoidal sinus. The *frontal sinus* occupies the supraorbital process of the frontal bone . The two sides are separated by a median septum, and in dogs each side is divided into rostral, medial, and lateral compartments. The *thyroid cartilage* forms the ventral and lateral walls of the larynx .. It surrounds the lateral aspect of the cricoid cartilage and articulates with the dorsolateral aspect of the cricoid cartilage (caudal) and thyrohyoid bones(cranial). Ventrally the cricothyroid ligament joins the caudal border of the thyroid cartilage to the cricoid cartilage. The *cricoid cartilage* is a complete ring that is five times wider dorsally than it is ventrally

The glottis (laryngeal inlet) consists of the vocal folds, the vocal processes of the arytenoid cartilages, and the rima glottidis The vocal folds extend dorsally from the vocal processes of the arytenoids to the thyroid cartilage ventrally. Rostral and lateral to the vocal folds are the laryngeal ventricles, or saccules. The laryngeal saccules are mucosal diverticula bounded laterally by the thyroid cartilage and medially by the arytenoid cartilage. The vestibular fold (false vocal cord) forms the rostral border of the laryngeal saccule and attaches to the cuneiform process. The intrinsic muscles of the dog's larynx are innervated by somatic efferent axons from the vagus nerve. Some axons leave the vagus in the cranial laryngeal nerve to innervate the cricothyroid muscle; others provide sensory innervation to the mucosa. The recurrent laryngeal nerve, a branch of the vagus, terminates as the caudal laryngeal nerve, which innervates the remaining intrinsic muscles of the larynx. The caudal laryngeal nerve travels along the dorsolateral surface of the trachea and continues over the lateral surface of the cricoarytenoideus dorsalis before deviating to the medial surface of the thyroid cartilage lamina. The cranial laryngeal artery, a branch of the external carotid artery, travels with the cranial laryngeal nerve. It is the main blood supply to the larynx. The cranial laryngeal vein empties into the hyoid venous arch and then the external jugular vein. Lymphatics drain into the retropharyngeal lymph node.

The trachea is a semi-rigid, flexible tube that extends from the cricoid cartilage to the mainstem bronchi at about the fourth or ffth thoracic vertebra. Thirty-fve to 45 incomplete C-shaped hyaline cartilages, joined by annular ligaments ventrally and laterally and by the trachealis muscle (dorsal

tracheal membrane) dorsally, form the trachea. The tracheal vessels and nerves, which are found in the lateral pedicles, supply the trachea segmentally. Loose areolar connective tissue surrounds the trachea and forms the lateral pedicles. The cranial and caudal thyroid arteries and veins, the bronchoesophageal arteries and veins, and the internal jugular veins supply vascular branches to the trachea. Innervation is provided by the autonomic nervous system. Sympathetic fbers from the middle cervical ganglion and the sympathetic trunk inhibit tracheal muscle contraction and glandular secretions, whereas parasympathetic

fbers from the vagus and recurrent laryngeal nerves cause tracheal muscle contraction and glandular secretions.

Rhinotomy

The nasal cavity may be approached through dorsal, ventral, or lateral approaches. The dorsal approach is most commonly used for exploration and biopsy; however, the ventral approach can be used to explore the region caudal to the ethmoid turbinates and the ventral aspect of the turbinates. Lateral approaches are limited to lesions in the rostral aspect of the nasal cavity.

Tracheostomy ;Tracheostomy allows air to enter the trachea distal to the nose, mouth, nasopharynx, and larynx. A tracheotomy is performed to insert a tube (temporary tracheostomy) or create a stoma (permanent tracheostomy) to facilitate airflow. A nonreactive tube that is no larger than half the size of the trachea should be selected. Cuffed or cannulated autoclavable silicone, silver, or nylon tubes are recommended. In an emergency situation, a standard endotracheal tube can be used, but make sure the tube is not inserted too far into the respiratory tree, and that the cuff is not inflated. Polyvinyl chloride and red rubber tubes are irritating and should be avoided. If the animal is to be placed on a respirator, a cuffed tube is necessary.

Temporary tracheostomy. A temporary tracheostomy is most commonly performed to provide an alternate airflow route during surgery or as an emergency procedure in severely dyspneic patients. Tube tracheostomies usually aPermanent tracheostomy.

Permanent tracheostomy is the creation of a stoma in the ventral tracheal wall by suturing tracheal mucosa to skin. Tracheostomas are maintained for life, or until the stoma is surgically closed. Tracheostomy tubes are not needed to maintain lumen patency after this procedure. Permanent tracheostomies are recommended for animals with upper respiratory obstruction causing moderate to severe respiratory distress (e.g., laryngeal paralysis, laryngeal collapse, upper airway neoplasia) that cannot be successfully treated by other methods. Owners should be warned that these animals must be restricted fromre maintained for only a short time. swimming, and that vocalization is diminished or absent after

this procedure. Furthermore, ongoing care of the site will be necessary to keep it clean

Tracheal Resection and Anastomosis

Removal of a tracheal segment may be necessary to treat tracheal tumor, stenosis, avulsion, or trauma. Depending on the extent of injury, tears in the tracheal wall that occur as a consequence of bite wounds or endotracheal intubation may be allowed to close spontaneously, may be closed primarily, or may be resected and the tracheal ends anastomosed. Depending on the degree of tracheal elasticity and tension, approximately 20% to 50% of the trachea in an adult dog (pproximately 8 to 10 rings) may be resected and direct anastomosis achieved. The split-cartilage technique is preferred because it is easier to perform and results in more precise anatomic alignment with less luminal stenosis than many other techniques. Imprecise anastomosis and tension across the suture site are considerable risk factors in the development of tracheal stenosis. Accurate and meticulous surgical technique is crucial for reconstruction of the trachea. Diseased trachea that exceeds the limits of resection and anastomosis may be managed with permanent tracheostomy, intraluminal silicone tubes, grafts, or prostheses with .

Ventriculocordectomy is removal of the vocal cords to alter vocalization, remove masses, or enlarge the ventral glottis for dogs with laryngeal paralysis. The procedure may be performed through an oral or ventral (laryngotomy) approach. Anesthesia is maintained by using a tube tracheostomy, manipulating the endotracheal tube to the contralateral side of the larynx, or using injectable anesthetic agents. Ventriculocordectomy performed to widen the ventral glottis requires that more vocal fold be resected than is required for debarking.

Oral approach. Position the patient in ventral recumbency with the neck extended. Suspend the maxilla and pull the mandible ventrally to maximally open the mouth. Extend the tongue from the mouth to get maximum exposure of the glottis. Retract the cheeks laterally to improve visualization.

Avoid placing padding or hands in the region of the larynx because this may distort the nasopharynx. Remove the central margin of the vocal cord for debarking with a laryngeal or uterine cup biopsy forceps cartilages with simple

interrupted sutures beginning at the ventral midpoint of the trachea. Space additional sutures 2 to 3 mm apart. Place three or four retention sutures to help relieve tension on the anastomosis. Place and tie these sutures so that they encircle an intact cartilage cranial and caudal to the anastomosis, crossing external to the anastomotic site healing of the respiratory tract Laryngeal wounds heal by re-epithelialization if mucosal edges are in apposition. Epithelial cells at the wound margins extend and spread over the wound until it is covered. Constant motion associated with breathing and head movement inhibits primary healing. Laryngeal wounds with gaps heal by secondary intention, first fling with granulation tissue and then re-epithelializing. Secondary intention healing may cause scarring across the glottis. Restricting surgery to one side of the larynx and leaving epithelium at the dorsal and ventral commissures intact may prevent scarring. Tracheal epithelium responds immediately to irritation or disease by increased production of mucus. If the insult continues, cells desquamate and goblet cell hyperplasia occurs to increase the protective mucous layer. Superficial wounds heal by re-epithelialization. Healing begins within 2 hours after sloughing of superficial cells. Intact ciliated columnar cells surrounding the defect flatten, lose their cilia, and migrate over the wound. Mitosis begins about 48 hours after injury in the ciliated columnar and basal epithelial cells. Organization and differentiation begin after 4 days. Squamous cells replace ciliated and goblet cells if injury recurs without healing. Full-thickness tracheal mucosal wounds with a gap between mucosal edges fll with granulation tissue before re-epithelialization. Full-thickness wounds may heal with scar tissue protruding into the lumen. Scar tissue narrows the lumen and may interfere with transport of mucus. A 20% reduction in lumen diameter may reduce mucociliary clearance by more than 50%

COMPLICATIONS

Acute respiratory obstruction caused by mucosal swelling, edema, irritation, and increased mucus production and/or laryngeal or tracheal collapse may occur after upper respiratory surgery and must be relieved promptly Infection can be a problem because the nose, nasopharynx, larynx, and trachea have a resident bacterial flora. Using strict aseptic technique and lavaging contaminated tissues usually prevent infection. Injury to the recurrent laryngeal nerve may cause laryngeal spasms, paresis, or paralysis, leading to aspiration

pneumonia. Mucostasis may occur after nerve damage. Gentle tissue handling, appropriate dissection, and careful tissue retraction prevent nerve damage.

Complications associated with rhinotomy include excessive blood loss, subcutaneous emphysema, gagging, coughing, and/or vomiting associated with aspiration of blood and exudates. Bone flaps that have been replaced following rhinotomy may sequester or harbor infectious organisms or tumor cells, leading to recurrence of disease. Caudal choanal stenosis may occur following severe rhinitis associated withinfection or after extensive débridement of nasal epithelium. Signs include those of nasal obstruction with minimal nasal discharge and stridor. These stenotic lesions can be diffcult to resolve; however, scar tissue may be perforated and then dilated with a balloon catheter or excised and covered with a mucosal flap. Stents can be very helpful in patients that experience recurrence of the stenosis. Intensive monitoring of a patient with a temporary tracheostomy tube is required to avoid life-threatening complications, particularly in smaller animals. Complications associated with tube tracheostomy include gagging, vomiting, coughing, tube obstruction, tube dislodgment, emphysema, tracheal stenosis, tracheal malacia, and tracheocutaneous or tracheoesophageal fistula. Some animals occlude the tracheostomy tube when the neck is flexed, and when they sleep with bedding. Major complications (occlusion, dislodgment) were reported in 44% of cats with temporary tracheostomies . Cuffed tracheostomy tubes and endotracheal tubes may cause pressure necrosis of the tracheal mucosa or cartilages, which may result in tracheal strictures. Animals with permanent tracheostomy have high complication and mortality rates primarily caused by occlusion of the stoma from mucus, blood, or stricture. In particular, cats are at high risk for acute occlusion and sudden death The long-term main complication of permanent tracheostomy is stomal occlusion from accumulated mucus, skin folds, or stenosis. Mucus accumulation, coughing, and gagging may also occur because of tracheal irritation. Complications after tracheal resection and anastomosis may include hemorrhage, voice change, fistula formation, and cartilage malacia. Malacia is uncommon, and the other complications are manageable. Dehiscence occurs after tracheal anastomosis if excessive postoperative tension or neck movement is present. Subcutaneous emphysema, acute respiratory distress, hemoptysis, and subcutaneous swelling suggest

dehiscence. Excessive anastomotic tension and secondary intention healing may cause tracheal stenosis. Excessive dissection may cause ischemic necrosis of the remaining trachea. Traumatizing the recurrent laryngeal nerves may cause laryngospasm, laryngeal paresis, or laryngeal paralysis. After ventriculocordectomy, scar tissue may form within the larynx and trachea, causing obstruction weeks postoperatively. Clinical signs of obstruction are not usually apparent until luminal compromise approaches 50%. Scar tissue forms across the larynx as the result of mucosal damage or with second intention healing near the dorsal and ventral commissures. Other complications include edema, hemorrhage, cough, gag, stenosis, and altered vocalization. Mucosal

edema may partially obstruct the glottis and can be reduced by pretreatment with corticosteroids. Stenosis may occur at the dorsal or ventral commissures of the glottis after ventriculocordectomy if intact mucosa is not preserved in these areas, and healing occurs by secondary intention. Approximating mucosa over the ventriculocordectomy sites also minimizes stenosis. Ventriculocordectomy is expected to alter the normal bark, making it lower pitched and harsher.

Resumption of a near-normal bark may occur within months after removal of only the vocal fold margin and secondary intention healing

LARYNGEAL COLLAPSE Laryngeal collapse is a form of upper airway obstruction caused by loss of cartilage rigidity that allows medial deviation of the laryngeal cartilages. Collapse of the cuneiform process of the arytenoid cartilage is referred to as aryepiglottic collapse or stage 2 layngeal collapse. Collapse of the corniculate process of the arytenoid cartilage is referred to as corniculate collapse or stage 3 laryngeal collapse.

PATHOPHYSIOLOGY Laryngeal collapse occurs secondary to chronic upper airway obstruction or trauma. Trauma may fracture or disrupt the laryngeal cartilages and allow medial collapse. Laryngeal collapse most often is caused by chronic upper airway obstruction (e.g., brachycephalic syndrome, laryngeal paralysis) and cartilage fatigue or degeneration. The obstruction causes increased velocity. These forces displace laryngeal structures medially, airway resistance, increased negative intraglottic luminal pressure, and increased air

with permanent cartilage deformation, and also fatigue the cartilages. Increased inspiratory effort irritates the mucosa, causing inflammation and edema. This further obstructs the airway, causing greater airflow resistance and increasing the effort of breathing. Laryngeal collapse is described in three stages:

Stage 1 is commonly referred to as laryngeal saccule eversion

stage 2 collapse is medial deviation of the cuneiform cartilage and aryepiglottic fold, or aryepiglottic collapse;

stage 3 collapse is medial deviation of the corniculate process of the arytenoid cartilages, or corniculate collapse. Stages 2 and 3 are advanced stages of laryngeal collapse.

The diagnosis of laryngeal collapse that occurs concurrently with other upper respiratory abnormalities (i.e., elongated soft palate [see p. 924] and stenotic nares may be easily overlooked on oral and laryngeal examination. The incidence of laryngeal collapse in brachycephalic dogs varies widely (8% to 53%) and may be related to regional differences and the defnition of laryngeal collapse (Torrez asphyxiation. Postoperative coughing and gagging are common. Water, ice water, or ice chips may be offered when the animal is fully recovered from anesthesia; however, food should be withheld for 12 to 24 hours .Offering food soon after surgery may traumatize swollen tissues, causing swelling, airway obstruction, aspiration, or all of these.

COMPLICATIONS

If stenotic nares are the patient’s only abnormality, complications are minimal. Dehiscence may occur if the patient frequently licks or rubs its nose; healing then occurs by secondary intention and may cause a pink scar. Respiratory distress may persist if other areas of the airway are obstructed

(especially from postoperative edema), or if insufficient palate was resected. Acute respiratory distress and death following surgery are possible given the high anesthetic risk of these dogs. PROGNOSIS Surgical correction of brachycephalic syndrome will alleviate signs of respiratory distress and improve quality of life in most dogs. The outcome is dependent on the age of the animal at the time of surgery and how severely the dog is affected

preoperatively. Recent studies describe a good to excellent long-term outcome in 88% to 94%, with mortality rates less than 5% English Bulldogs have been found to have a worse response to surgery when compared with all other breeds combined and are more likely to develop aspiration pneumonia postoperatively.

An association with gastrointestinal disease has been investigated, and it is believed that brachycephalic dogs surgically treated for upper airway disease and concurrently medically managed for gastrointestinal disease have an overall better outcome. Without surgery, the prognosis for dogs with elongated soft palate and everted laryngeal saccules is guarded because respiratory signs and laryngeal collapse progress over time. If advanced laryngeal collapse has developed, the prognosis is often poor unless additional surgery is considered.

DIFFERENTIAL DIAGNOSIS

Differential diagnoses include laryngeal or tracheal obstruction caused by masses, paralysis, everted laryngeal saccules, elongated soft palate, and stenotic nares.

MEDICAL MANAGEMENT

Medical therapy is recommended to alleviate acute respiratory distress. A weight reduction program should be instituted for obese animals. Exercise restriction and elimination of precipitating causes may be beneficial when clinical signs are mild. A weight reduction program should be instituted if the animal is obese. Exercise restriction and elimination of precipitating causes may be beneficial when clinical signs are mild. Sedation ,corticosteroids, supplemental oxygen, and cooling may be necessary for moderate to severe respiratory distress. An anti-inflammatory dose of a corticosteroid (dexamethasone 0.5 to 2 mg/kg IV, IM, or SC; start with the lower dose and use the higher dose only if the low dose has failed to reduce swelling, and if the swelling is suffciently severe that the animal cannot breathe) may reduce nasopharyngeal and upper airway edema. Note that large doses or repeated administration of dexamethasone typically causes gastrointestinal irritation, erosion, or ulceration. Prolonged medical therapy for the respiratory signs may allow progression of degenerative laryngeal changes. If the response to

treatment is less than expected after appropriate surgery for these abnormalities, laryngeal collapse may be present

Laryngeal paralysis is complete or partial failure of the arytenoid cartilages and vocal folds to abduct during inspirationLaryngeal paralysis causes upper respiratory obstruction and mild to severe dyspnea. It occurs because of dysfunction of the laryngeal muscles, recurrent laryngeal or vagus nerves, or cricoarytenoid ankylosis; acquired or congenital neurologic causes are most common. The intrinsic laryngeal abductor and adductor muscles are innervated by the recurrent laryngeal nerves Atrophy ofthe cricoarytenoideus dorsalis muscle causes the cartilages to remain in a paramedian position during inspiration, preventing maximal air intake and increasing airflow resistance. The narrowed rima glottidis increases resistance to airflow and creates turbulence, which gives rise to laryngeal stridor.

Partial laryngectomy may be done by an oral approach or by a ventral laryngotomy approach. Bilateral vocal fold resection alone or in conjunction with unilateral resection of the corniculate, cuneiform, and vocal processes of the arytenoid cartilage may be performed. Partial laryngectomy by an oral approach is extremely diffcult in small dogs because of limited exposure. Oral approach. Grasp the corniculate process and retract it medially with biopsy forceps. Use a long-handled scalpel or scissors to excise the corniculate process and the proximal half and base of the cuneiform process

Do not excise the aryepiglottic fold or the distal half of the cuneiform process. Remove the vocal fold, vocal process, and vocal muscle with biopsy forceps or Metzenbaum scissors (or both) (Fig. 29-24, B). Leave the ventral aspect of the vocal cord intact. Control bleeding by applying pressure with gauze sponges. Limit resection to one side of the glottis. Laryngotomy approach. Make a ventral midline incision over the larynx. Separate the sternohyoid muscles and incise the cricothyroid membrane and thyroid cartilage on the midline. Retract the edges of the thyroid cartilage with small Gelpi forceps. Visualize the arytenoid cartilages and vocal folds. Have an assistant visualize the larynx per os to help determine how much should be removed. After incising the mucosa over the corniculate, cuneiform, and vocal processes of one arytenoid cartilage, excise them with scissors or a scalpel.

Tracheal collapse.

Grade 1 Relatively normal tracheal cartilage anatomy; redundant dorsal tracheal membrane decreases luminal diameter up to 25%

Grade 2 Mild to moderate flattening of tracheal cartilages; 50% loss of luminal diameter

Grade 3 Severe flattening of tracheal cartilages; 75% loss of luminal diameter

Grade 4Complete obstruction; tracheal lumen is obliterated940 paRT TWO Soft Tissue Surgery supplemental oxygen

LARYNGEAL AND TRACHEAL TUMORS Oncocytomas arise from epithelial cells called oncocytes, small numbers of which are found in various organs, such as the larynx, thyroid, pituitary, and trachea.

Tumors of the larynx are uncommon in the dog and cat. Numerous types of tumor have been reported in the dog, including rhabdomyosarcoma, squamous cell carcinoma, adenocarcinoma, and mast cell tumor. Squamous cell carcinoma and lymphoma are the most common tumors of the

Larynx in the cat . Rhabdomyomas and oncocytomas are laryngeal tumors that appear histologically similar

With light microscopy; electron microscopy and immunocytochemistry are necessary to distinguish them. Oncocytomas

have been reported in young dogs and warrant special consideration because long-term survival of patients without metastasis has been reported after surgical resection Tracheal tumors are even less common than laryngeal masses. Malignant and benign tracheal tumors have been reported. Tracheal osteochondromas may occur with collapse may require a permanent tracheostomy within the first 24 hours to relieve respiratory distress. Nasal insufflation of oxygen and an anti-inflammatory dose of corticosteroids may be benefcial in animals with edema and inflammation. Mucolytics and saline nebulization may be appropriate for those with severe inflammation. Antibiotics should be continued for 7 to 10 days if bacterial tracheitis is present. Antitussives, bronchodilators, analgesics, and sedatives may be given as necessary to control coughing and excitement . These animals should have strict exercise restriction (cage rest) for 3 to 7 days. Thereafter exercise may be increased gradually. A harness rather than a collar should be used for leash walking. Weight reduction is important in obese patients. Tracheoscopy is recommended 1 to 2 months after surgery and later if respiratory signs deteriorate. Immediate improvement in clinical signs may be seen with both extraluminal and endoluminal stents; however, coughing and lack of marked improvement in clinical signs should be expected for several weeks postoperatively because of tracheitis, peritracheal swelling, and suture irritation. However, significant clinical improvement (e.g., decreased respiratory noise, less respiratory effort, increased exercise tolerance, fewer tracheobronchial infections) should be noted within 2 to 3 weeks of surgery. Some animals have nearly complete remission of clinical signs after surgery whereas others continue to have episodes of coughing or other respiratory noises. The quality of life is improved for most patients, but neither surgery nor stents cure the condition.

COMPLICATIONS

Death may result if the trachea is obstructed by severe inflammation or is damaged by severe infection or necrosiswith extraluminal or intraluminal prostheses. Co ughing after surgery is expected until inflammation subsides. Infection is a possible problem because the trachea contains bacteria that may be harbored in implants. Bruising and mild cervical swelling are expected postoperatively after placement of extraluminal prostheses. Recurrent laryngeal

nerve damage may result in laryngospasm, laryngeal paresis, or paralysis. Tracheal necrosis may occur if too much dissection strips the blood supply away from the trachea, or if improperly aerated prostheses (gas sterilized) are implanted Incorrect placement or sizing of intraluminal implants may result in death. Failure to stent the entire involved trachea typically results in collapse proximal and/or distal to the stent. A stent that is too narrow may migrate, whereas one that is too wide may cause pressure necrosis. Stents placed too close to the larynx may cause intractable laryngospasm. Tracheal obstruction caused by granuloma formation, which may be steroid responsive, occurs in about 20% to 30% of cases. Other reported complications include cough, expectoration, tracheal hemorrhage, emphysema, pneumomediastinum, infection, mucous obstruction, tracheal rupture, squamous metaplasia and ulceration of the944 part two soft tissue Surgery tracheal cytology. Anthelmintic therapy and surgical resection have met with varying success. Aberrant Cuterebra larvae and associated tissue trauma may obstruct the laryngeal or tracheal lumen. Other non-neoplastic differentials for tracheal masses include inflammatory nodules, including lymphoplasmacytic inflammation, lymphoid hyperplasia, and granulomatous tracheitis. All of these lesions must be differentiated from neoplastic lesions. Granulomatous laryngitis is an uncommon nonneoplastic proliferation of the arytenoid cartilages of the larynx that has been reported in both dogs and cats. Severe in dogs younger than 1 year of age. These masses probably reflect a malfunction of osteogenesis and are benign. Their growth is expected to stop with skeletal maturity. In

Nasal tumors or sinonasal tumors are tumors that arise from Neoplasms of the nasal cavity and paranasal sinuses are rare in most domestic species; the reported incidence varies from0.3% to 2.4% of canine tumors. They occur more commonly in dogs than in cats. Sinonasal tumors may be classified histologically as epithelial, nonepithelial, or miscellaneous . Neoplasms of epithelial origin are most common, with adenocarcinoma being the single most frequent histologic diagnosis in dogs. Nonepithelial tumors of skeletal origin (i.e., chondrosarcoma and osteosarcoma) account for approximately one-ffth of canine nasal tumors. In cats, lymphoproliferative and epithelial tumors are most prevalent.

Lungs and Thoracic Wall Thoracotomy is surgical incision of the chest wall; it may be performed by incising between the ribs (intercostal or lateral thoracotomy) or by splitting the sternum (median sternotomy). Pulmonary lobectomy is removal of a lung lobe (complete) or a portion of a lung lobe (partial). Pneumonectomy is removal of all lung tissue on one side of the thoracic cavity.

preoperative management Animals with traumatic lesions that impair respiration (e.g., flail chest) or those with acute respiratory impairment (i.e ruptured bulla or ruptured pulmonary abscess) often require emergency stabilization (e.g., stabilization of rib segments thoracentesis, and oxygen therapy) before surgery. Equipment for thoracentesis and chest tube placement should be readily available, and clinicians should be familiar with these techniques. The thorax is one of the most common regions injured following blunt trauma; thoracic injuries were identifed in over 72% of patients in a recent study. Concurrent abdominal and chest injuries were found in 50% of injured animals. or evaluation with pulse oximetry is warranted preoperatively in patients undergoing thoracic surgery to detect and defne the severity of respiratory impairment. Unexplained abnormalities should be investigated because ventilatory impairment caused by nonsurgically correctable disease (i.e., diffuse micrometastasis) occasionally is identifed. If possible, signifcant anemia should be corrected before surgery

Partial lobectomy may be performed to remove a focal lesion involving the peripheral one half to two thirds of the lung lobe or for biopsy. Partial lobectomy may be performed through a lateral fourth or ffth space intercostal thoracotomy or median sternotomy Partial lobectomy may also be performed with stapling devices (e.g., TA stapler;). The stapling equipment comes in various sizes, which produce staple lines 30 mm, 55 mm, or 90 mm long. Select the staple size based on the width of the lung so that the staple line extends across the entire width of the lung to be removed but not beyond the edges. If air leaks or hemorrhage are noted, place a simplecontinuous pattern of absorbable suture along the lungmargin. The stapling devices compress tissue to a thickness of 1.0 mm (2.5-mm staples; 30-mm length only), 1.5 mm (3.5-mm staples), or 2 mm (4.8-mm staples). Avoid stapling excessively thick or fbrotic lung because this may result in

Guttural pouch

Guttural pouches are large, auditory-tube diverticula that contain between 300 and 600 ml of air. They are present in odd-toed mammals, some bats, hyraxes, and the American forest mouse. They are paired bilaterally just below the ears, behind the skull and connect to the nasopharynx.

Due to the general inaccessibility of the pouches in horses, they can be an area of infection by fungi and bacteria, and these infections can be extremely severe and hard to treat. The condition guttural pouch tympany affects several breeds, including the Arabian horse. The condition predisposes young horses to infection, often including severe swelling and often requires surgery to correct. The guttural pouch is also the site of infection in equine strangles.

Structure:-The guttural pouches are located behind the cranial cavity, caudally the skull and below the wings of the atlas (C1). They are enclosed by the parotid and mandibular salivary glands, and the pterygoid muscles. The ventral portion lays on the pharynx and beginning of the esophagus, with the retropharyngeal lymph nodes located between the ventral wall and pharynx. The left and right pouches are separated by the longus capitis and rectus capitis ventralis muscles dorsomedially. Below these muscles, the two pouches fuse to form a median septum.

The guttural pouches connect the middle ear to the pharynx. The opening into the pharynx is called the nasopharyngeal ostium, which is composed of the pharyngeal wall laterally and a fibrocartilaginous fold medially. This opening leads to a short soft tissue passageway into the respective guttural pouch. The openings are located rostrally to enable drainage of mucous when the head is lowered and prevent fluid build-up. The plica salpingopharyngea, a mucosal fold at the caudal portion of the Eustachian tube, forms an uninterrupted channel between the medial lamina of the Eustachian tube and the lateral wall of the pharynx. The plica salpingopharyngea can sometimes act as a one-way valve trapping air in the pouch, causing guttural pouch tympany. Each pouch is moulded around the stylohyoid bone which divides the medial and lateral compartments. The medial compartment is much larger, and protrudes more caudally and ventrally.

The epithelium is pseudostratified and ciliated containing mucous-secreting goblet cells; lymph nodules are also present.The compartments of each guttural pouch contain many important structures including several cranial nerves and arteries that lie directly against the pouch as they pass into and out of foramina in the caudal aspect of the skull. The glossopharyngeal, vagus, accessory and hypoglossal nerves. the sympathetic trunk leaving from the cranial cervical ganglion; and the internal carotid all cause a mucosal fold indent within the medial compartment, visible when viewed endoscopically. The facial nerve is in contact with the dorsal part of the pouch. The external carotid artery passes ventral to the medial compartment before crossing to the lateral wall of the lateral compartment. The pouch also covers the temporohyoid joint.

Function

For many years, the functionality of the guttural pouches remained an unsolved mystery. Recent studies have shown that they play a role in cooling the blood from the internal carotid destined for the brain during hyperthermia. Even-toed artiodactyls possess a carotid rete, responsible for heat exchange, to cool arterial blood before it enters the cranial cavity. This anatomical adaptation acts to protect friable brain tissue from injury due to overheating.Odd-toed perrisodactyls such as horses lack a carotid rete, but since the internal carotid artery passes through the guttural pouches, it has been discovered that the air within the pouches cools the blood during exercise. Brain cooling by convection in the cerebral blood also occurs.

Pathology

If the drainage tract becomes blocked for any reason, the mucous secretions can accumulate and cause the pouch to distend, producing a visible and palpable protrusion behind the mandible. The exudate may become contaminated with pathogens. The bacteria Streptococcus equi, the causative agent of strangles, is commonly involved. Mycotic infections can also occur. Some visible symptoms of guttural pouch disease include abnormal head and neck carriage, nasal discharge, painful swelling and occasional abnormal functioning of the structures associated with the pouch. Secondary problems may include inflammation of the middle ear due to migration of the infection along the

auditory tube; nasal bleeding caused by damage to the internal carotid artery; with vagus nerve involvement there may be laryngeal hemiplegia (roaring) or difficulty swallowing (also if glossopharyngeal nerve is involved); and Horner's syndrome from the involvement of sympathetic nerves. Involvement of the facial nerve is rare.

As the guttural pouches are covered by respiratory epithelium and mucosa, they have the potential to be affected by all respiratory pathogens. Most infections are self-limiting, requiring no or little medical intervention. Upon endoscopy, affected guttural pouches often house mucopurulent fluid that is in the process of draining.

Guttural Pouch Empyema.

Guttural pouch empyema is characterized by the accretion of purulent, bacteria infested exudate in the pouch. The bacteria is primarily Streptococcus equi, the infectious agent of strangles. Clinically apparent symptoms include painful swelling of the parotid area and recurrent infected nasal discharge, and in severe circumstances, difficulty breathing and abnormal head carriage may be observable. Fever, anorexia, difficulty swallowing and soft palate displacement may or may not be seen. Empyema is often secondary to guttural pouch tympany (distention of the pouches with air) in foals and weanlings. Arabians, in particular, are inclined to guttural pouch tympany, as many have a congenital defect in the pharyngeal orifices of the pouches.The infection can also be due to the rupture of the nearby retropharyngeal lymph nodes, usually caused by an abscess.

Diagnosis is established through and endoscopic examination. Radiographic examination of the area will show an opaque fluid line in the pouch and if a retropharyngeal lymph node is involved, it may reveal a mass. In mild, acute cases of empyema, a saline or polyionic solution lavage is often performed via an endoscope or catheter repeatedly until the exudate drains..Antimicrobial therapy without a lavage seldom remedies the infection. In more complex cases, where concretions have formed, surgical intervention may be necessary to ensure appropriate drainage and removal of the hardened material. The area can be accessed surgically through the Viborg's triangle.

Guttural Pouch Tympany is an uncommon ailment in which excessive amounts of air become trapped in the pouch, resulting in abnormal expansion. Tympany is usually unilateral, but in some cases can affect both pouches. It is seen most often in young foals and is more common in females than in males. Tympany results in non-painful, soft swelling beneath the ear and behind the jaw. Additional symptoms may include roaring, difficulty breathing, and difficulty swallowing and/or aspiration pneumonia. Diagnosis is achieved through radiography and endoscopic evaluation. The specific cause of guttural pouch tympany is not known, but it is suspected that it is more common when there are defects of the plica salpingopharyngeus, and/or the pharyngeal orifice where they act as a one-way valve that does not allow air to escape. Because of the risk of secondary infection, it is crucial that tympany be treated as soon as possible. Treatment protocols may include, but are not limited to, surgical intervention and in cases where surgery is not an option, insertion of a transnasal Foley balloon catheter in an attempt to remodel the pharyngeal orifice.

Guttural Pouch Mycosis(GPM) is a fungal disease that is rare but potentially life-threatening. GPM is of unknown pathogenesis currently and no predisposing factors have been identified. Fungal plaque is usually located in the medial guttural pouch, near the internal carotid artery. Clinical signs include unilateral or bilateral epistaxis due to erosion of the artery walls, nasal discharge and cranial nerve dysfunction. GPM is a dangerous condition as spontaneous fatal hemorrhaging can occur, usually within a few days to weeks after the first bout of epistaxis .he most common fungus associated with GPM is Aspergillosis.

Diagnosis is made based on the history of the animal, presenting clinical signs and endoscopic exploration. Pharmaceutical treatment is not suggested without coinciding surgery.Treatment typically consists of topical as well as systemic antifungal medication, paired with surgery to occlude or embolise affected arteries. Early intervention is necessary to ensure the best chance of survival. Horses that experience dysphagia or other forms of nerve dysfunction as a result of GPM have a poorer prognosis that those who have not exhibited those symptoms.