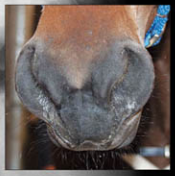
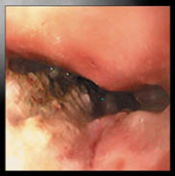
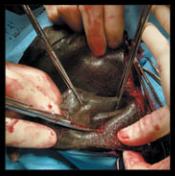
Respiratory Diseases of the Horse

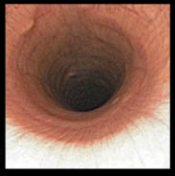
A problem-oriented approach to diagnosis & management

Laurent Couëtil
Jan Hawkins









MANSON PUBLISHING

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A problem-oriented approach to diagnosis & management

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MANSON PUBLISHING/THE VETERINARY PRESS

ISBN: 978-1-84076-186-3

A CIP catalogue record for this book is available from the British Library.

For full details of all Manson Publishing Ltd titles please write to:
Manson Publishing Ltd, 73 Corringham Road, London NW11 7DL, UK.
Tel: +44(0)20 8905 5150
Fax: +44(0)20 8201 9233
Email: manson@mansonpublishing.com
Website: www.mansonpublishing.com

Commissioning editor: Jill Northcott
Project manager and book design: Ayala Kingsley
Copy editor: Peter Beynon
Illustration: Cactus Design, Ayala Kingsley
Proof reader: Sarah Binns
Index: Jill Dormon
Colour reproduction: Tenon & Polert Colour Scanning Ltd, Hong Kong
Printed by: Grafos SA, Barcelona, Spain

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PREFACE

THIS BOOK is the result of the career-long interests of the two authors in diseases of the equine respiratory tract, from both medical (LC) and surgical (JH) perspectives. The equine respiratory tract presents unique questions and challenges that equine practitioners struggle with daily. For example: Where is the abnormal breathing sound coming from? Why does this horse have a nasal discharge? Why is this horse not performing up to expectations?

This book attempts to provide a framework to help equine practitioners systematically evaluate, diagnose, and treat the most common (and some not so common) disorders of the equine respiratory tract. It is organized in a way that emulates the approach that a clinician would use to assess a horse with a suspected respiratory abnormality. Hence, the book starts with a detailed description of anatomic features and pulmonary function, followed by an in-depth discussion of clinical examination methods and the use of appropriate diagnostic tests to help arrive at a clinical diagnosis. A novel *modus operandi* taken by the authors is the use of a problem-oriented approach to the diagnosis of equine respiratory diseases. With this in mind, there are chapters dedicated to the evaluation of a horse presenting with a complaint of coughing, nasal discharge, increased respiratory effort, abnormal respiratory noise, or congenital respiratory abnormalities. Each starts by discussing the pathophysiology of a particular respiratory disease manifestation and then suggests an approach to reaching a diagnosis. Once a presumptive diagnosis is obtained, the reader can refer to the comprehensive discussion of each particular respiratory disease, including medical and/or surgical therapy.

LAURENT COUËTIL
JAN HAWKINS

ABBREVIATIONS

6

$A–aD_{O_2}$	alveolar–arterial oxygen tension difference
ABG	arterial blood gas
ACTH	adrenocorticotropic hormone
AHR	airway hyperresponsiveness
BAL	bronchoalveolar lavage
CAD	cricoarytenoideus dorsalis (muscle)
CAJ	cricoarytenoid joint
C_{aO_2}	oxygen content of arterial blood
CCA	common carotid artery
C_{dyn}	dynamic lung compliance
CFC	chlorofluorocarbon
CMS	caudal maxillary sinus
CN	cranial nerve
CNS	central nervous system
CO_2	carbon dioxide
COPD	chronic obstructive pulmonary disease
CRI	constant rate infusion
CT	computed tomography
DDSP	dorsal displacement of the soft palate
DMSO	dimethylsulfoxide
2,3-DPG	2,3-diphosphoglycerate
DTPA	diethylene triamine pentaacetic acid
EE	epiglottic entrapment
EHV	equine herpesvirus
EIPH	exercise-induced pulmonary hemorrhage
EPM	equine protozoal encephalomyelitis
EVA	equine viral arteritis
F_A	fractional concentration of a gas in alveolar air
F_{ACO_2}	fractional concentration of carbon dioxide in alveolar air
F_{AO_2}	fractional concentration of oxygen in alveolar air
FE	forced expiration
FEF	forced expiratory flow
FEV_1	forced expiratory volume during 1 second
F_I	concentration of a gas in inspired air
F_{ICO_2}	fraction of inspired carbon dioxide
F_{IO_2}	fraction of inspired oxygen
FOM	forced oscillatory mechanics
FRC	functional residual capacity
FVC	forced vital capacity
GP	guttural pouch
GPE	guttural pouch empyema
GPM	guttural pouch mycosis
H	hemagglutinin
Hb	hemoglobin
HFA	hydrofluoroalkane
HYPP	hyperkalemic periodic paralysis
IAD	inflammatory airway disease
IFNα	interferon alpha
IM	intramuscular/intramuscularly
IV	intravenous/intravenously
LAP	left atrial pressure
LRT	lower respiratory tract
MMAD	mass median aerodynamic diameter
N	neuraminidase
NSAID	non-steroidal anti-inflammatory drug
O_2	oxygen

ΔP	pressure gradient
ΔP_{plmax}	maximal change in transpulmonary pressure
P_{aCO_2}	partial arterial pressure of carbon dioxide
P_{ACO_2}	partial alveolar pressure of carbon dioxide
PAM	pulmonary alveolar macrophage
P_{aO_2}	partial arterial pressure of oxygen
P_{AO_2}	partial alveolar pressure of oxygen
PAP	pulmonary artery pressure
Pb	barometric pressure
P_{CO_2}	partial pressure of carbon dioxide
PCR	polymerase chain reaction
PCV	packed cell volume
Pet_{CO_2}	end-tidal partial pressure of carbon dioxide
P_{ICO_2}	partial pressure of inspired carbon dioxide
P_{IO_2}	partial pressure of inspired oxygen
pMDI	pressurized metered-dose inhaler
PO	per os
P_{O_2}	partial pressure of oxygen
PRT	proximal respiratory tract
P_{tp}	transpulmonary pressure
P_{vCO_2}	partial venous pressure of carbon dioxide
$\dot{Q}$	perfusion
RAO	recurrent airway obstruction
R_{aw}	flow resistance through the airways
RBC	red blood cell
R_{cw}	chest wall resistance
RDPA	rostral displacement of the palato-pharyngeal arch
R_L	pulmonary resistance
RMS	rostral maxillary sinus
RR	respiratory rate
R_{rs}	resistive component of the respiratory system
R_{ti}	lung tissue resistance
RV	residual volume
S_{aO_2}	oxygen saturation of hemoglobin in arterial blood
SC	subcutaneous
SCC	squamous cell carcinoma
TLC	total lung capacity
TW	tracheal wash
URT	upper respiratory tract
$\dot{V}$	ventilation or airflow (volume of gas per minute)
V_A	alveolar volume
$\dot{V}_A$	alveolar ventilation (per minute)
VC	vital capacity
$\dot{V}_{CO_2}$	carbon dioxide produced per minute by metabolism
V_D	physiologic dead space
$\dot{V}_D$	physiologic dead space ventilation (per minute)
$\dot{V}_E$	expired minute ventilation (expired volume per minute)
$\dot{V}_{O_2}$	oxygen consumed per minute by metabolism
$\dot{V}_{O_2max}$	maximal oxygen consumption or maximal aerobic capacity
$\dot{V}/\dot{Q}$	ventilation–perfusion (ratio)
V_T	tidal volume
WBC	white blood cell
Zrs	impedance of the respiratory system

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CHAPTER 1

ANATOMY OF THE EQUINE RESPIRATORY TRACT

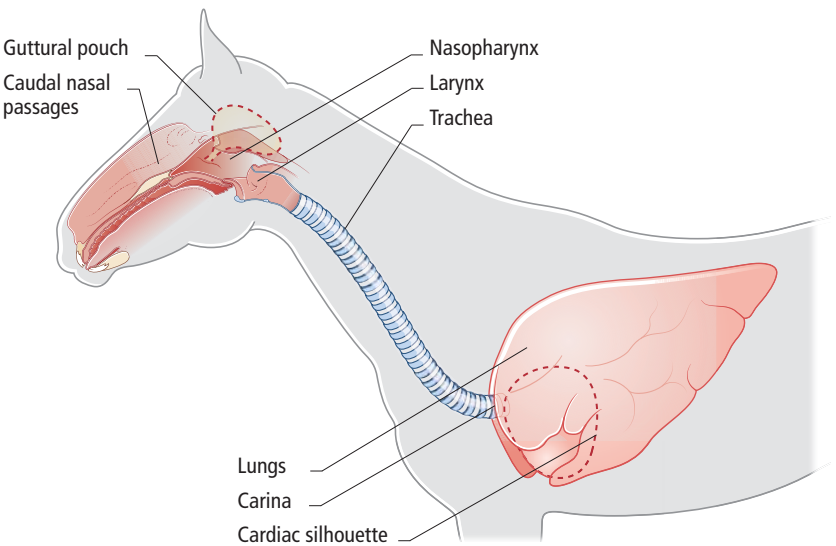
INTRODUCTION

The anatomy of the respiratory tract greatly influences its function (**1**). Additional factors, such as exercise, will result in further adaptation of the respiratory tract anatomy by increasing contraction of the accessory respiratory muscles to accommodate the increase in ventilation or exercise-induced hyperpnea. From a functional standpoint, it is helpful to separate the respiratory tract into the extrathoracic airways and the intrathoracic airways, with the former extending from the nose to the extrathoracic portion of the trachea.

EXTRATHORACIC AIRWAYS

Overview

The main role of the airways is to act as a conduit for air flow between the nasal opening and the gas-exchanging areas of the lung. To accommodate convection of air, the extrathoracic airways, and in particular the nasal passages, modify the air by adjusting its temperature nearer to normal body temperature, increasing the relative humidity closer to full saturation, and by filtering larger particles. The total cross-sectional area of the proximal respiratory tract is minimal compared with the vast surface area of the distal airways (bronchioles) and alveolar spaces. Any disease resulting in a decrease in airway patency will impair airflow and, ultimately, pulmonary function; however, a decrease in airway diameter will have maximum impact if located along the proximal airways, since all air travels through this bottleneck. In contrast, diseases involving the distal airways in the lung have to be severe and extensive to cause flow limitation. At rest, horses use only a small portion of their vital respiratory capacity (approximately 10%), so diseases resulting in airway obstruction have to be severe in order to impair pulmonary function. During strenuous exercise, horses utilize 100% of their respiratory capacity and mild airway obstruction that would not normally affect pulmonary function at rest may greatly interfere with gas exchanges during exercise.



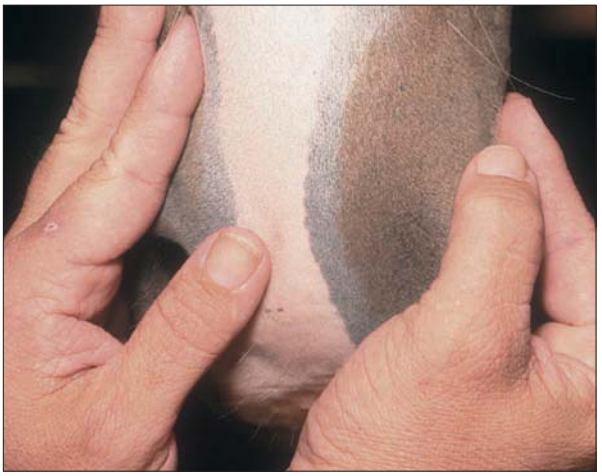
◀ **1 Anatomy of the equine respiratory tract, with anatomic regions corresponding to endoscopic views in the following figures: caudal nasal passages (6); guttural pouch (15); nasopharynx (19); larynx (20); trachea (37); carina (41).**

Assessment of diseases of the extrathoracic airways requires a full understanding of the unique anatomic features of the equine proximal respiratory tract (PRT). Knowledge of the gross and endoscopic anatomy of the PRT is crucial to arriving at a working diagnosis in a horse with clinical signs of respiratory noise and exercise intolerance. The normal anatomy of the structures (nares and rostral nasal passages, nasal septum, conchae and paranasal sinuses, guttural pouches (GPs), nasopharynx, hard and soft palate, epiglottis, larynx, and cervical portion of the trachea) that make up the equine PRT are described below.

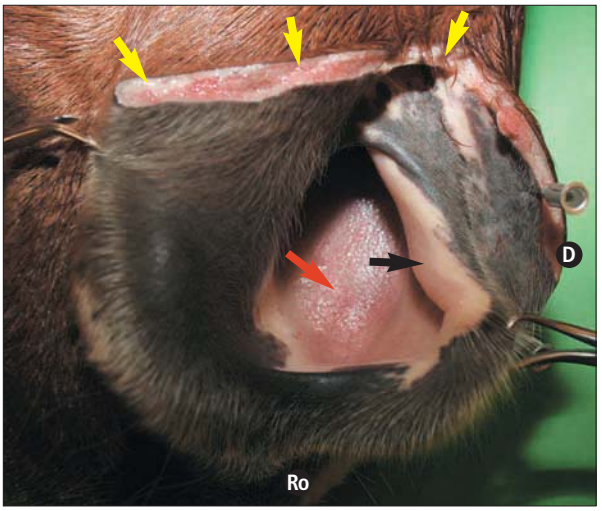
Nares and rostral nasal passages

Airflow from the left and right nares should be equal and easily palpable or visualized by movement of soft cotton or condensation on a mirror (**2**).

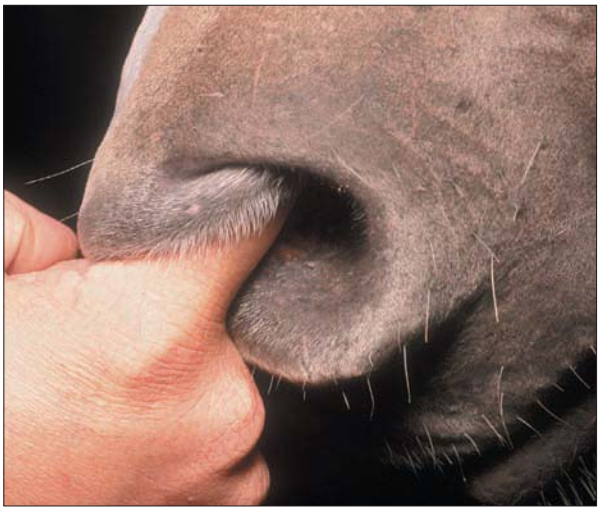
The nares consist of the nasal opening, alar folds, nasal diverticulum, and the rostral aspect of the nasal septum (**3**).



◀ 2 **Subjective evaluation of palpable airflow from each nasal passage. Clinical assessment of airflow can be enhanced by the use of a rebreathing bag.**



▲ 3 **Gross postmortem photograph illustrating the cut edge of the nasal diverticulum (yellow arrows), alar fold (black arrow), and rostral nasal septum (red arrow). (D) dorsal; (Ro) rostral.**



▲ 4 **Manual palpation of the rostral nasal septum.**

The false nostrils should be capable of full and symmetrical opening. The alar fold should be thin, pliable, and not edematous. The nasal diverticulum is located dorsal to the alar fold. The rostral aspect of the nasal septum is palpated manually with the index fingers (**4**). The normal rostral nasal septum is bilaterally symmetrical and the mucosa should be smooth and have no appreciable pitting edema.

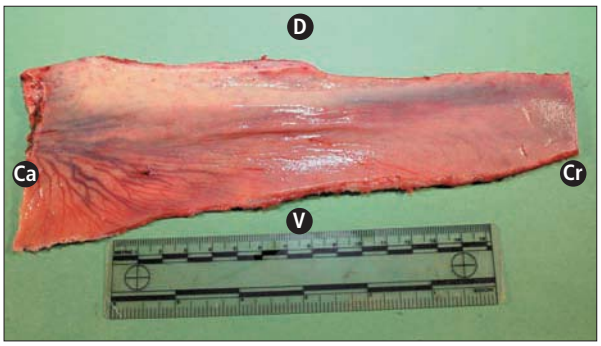
Nasal septum

The equine nasal septum consists predominantly of fibrocartilage (**5**). The ventral aspect of the nasal septum is attached to the vomer bone. A normal nasal septum should be straight and not deviated towards either nasal passage. It is covered by nasal mucosa.

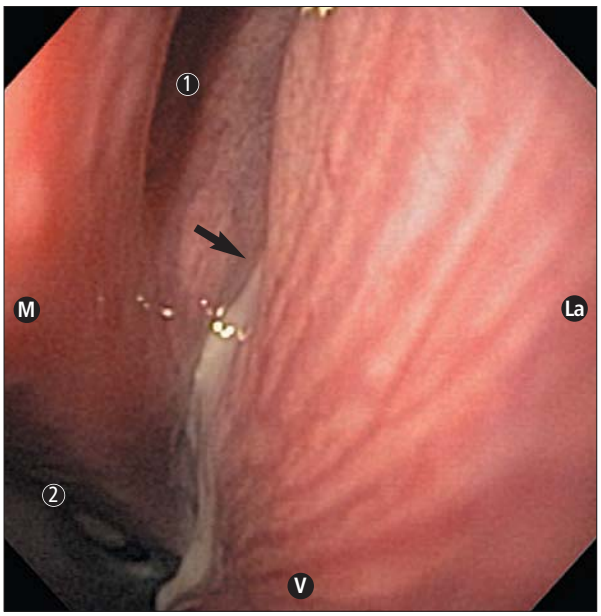
The nasal cavities are lined with a richly vascularized mucosa to allow warming and humidification of inhaled air. Vasomotor tone is under the control of the autonomic nervous system, with excitation of sympathetic and parasympathetic nerves leading to nasal vasoconstriction and vasodilation, respectively. Exercise is associated with sympathetic excitation and increased nasal volume. Despite these mechanisms, which are designed to increase airway patency during exercise, the nasal passages still represent one of the bottleneck regions of the equine respiratory tract.

Conchae and paranasal sinuses

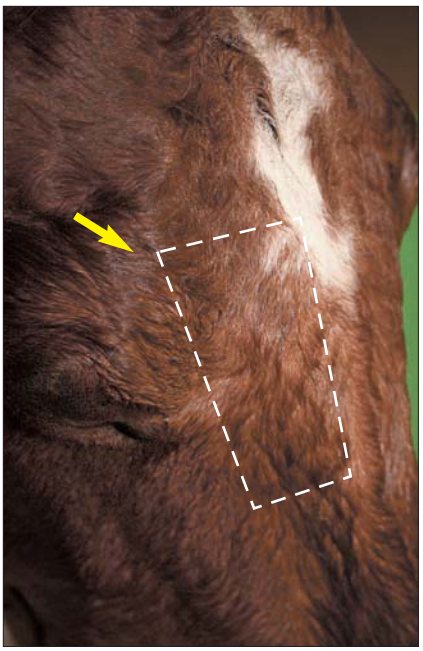
The horse has seven paranasal sinuses on each side of the skull. These are the dorsal, middle, and ventral conchal sinuses, the rostral and caudal maxillary sinuses, the frontal sinus, and the sphenopalatine sinus. The conchae and sinuses communicate through multiple natural openings. The dorsal and middle conchal, sphenopalatine, and frontal sinuses communicate with the caudal maxillary sinus. The ventral conchal sinus communicates with the rostral maxillary sinus. The nasomaxillary opening is the communication between the nasal passage and the maxillary sinus (**6**). It is located in the middle meatus in the area of the ethmoid turbinate and it can be visualized with an endoscope.



▲ **5** **Gross postmortem appearance of a normal nasal septum. Note the prominent vascular pattern on the caudal and ventral aspect of the septum. (Ca) caudal; (Cr) cranial; (D) dorsal; (V) ventral.**

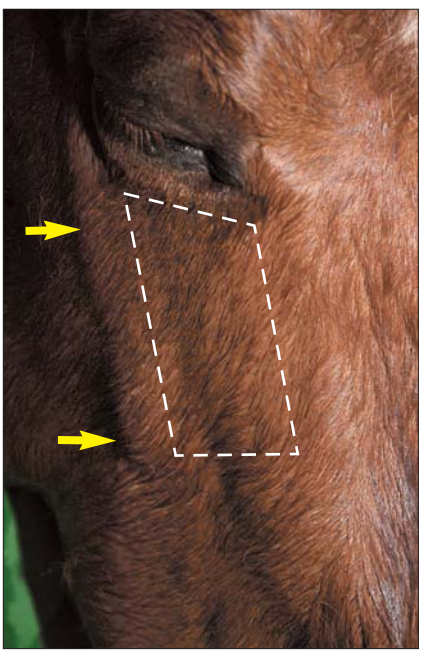


▲ **6** **Endoscopic view of the left nasomaxillary opening (arrow) with purulent exudate exiting the maxillary sinus. (1) ethmoid turbinates; (2) nasopharynx; (La) lateral; (M) medial; (V) ventral.**



▲ 7 **Gross postmortem appearance of the surgical limits of the frontal sinus (dotted line). Arrow, supraorbital foramen.**

▼ 8 **Gross postmortem photograph illustrating the boundaries of the maxillary sinus (dotted line). Arrow, facial crest.**

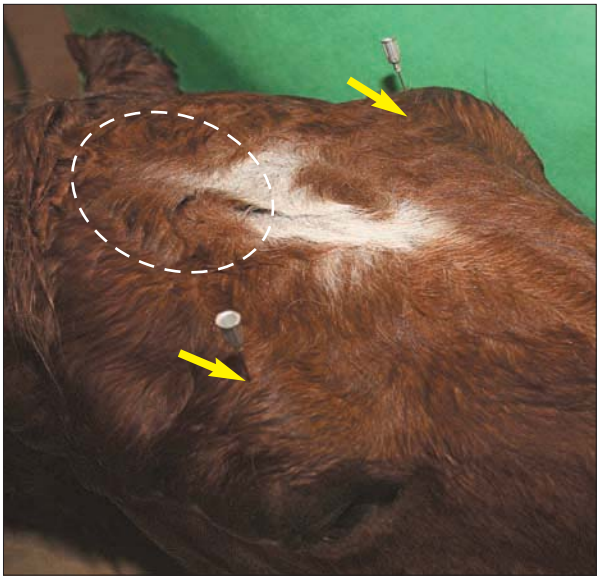


Along with an understanding of the natural openings between the sinuses, the surgical limits of the frontal and maxillary sinuses should also be known. The surgical limits of the frontal sinus are: medially, the midline of the forehead; caudally, a line joining the right and left supraorbital foramina; rostrally, a line approximately 8–10 cm rostral, or a line perpendicular to the facial crest, half-way between the medial canthus and the infraorbital foramen; and laterally, a line drawn from the medial canthus of the eye to the nasoincisive notch (**7**).

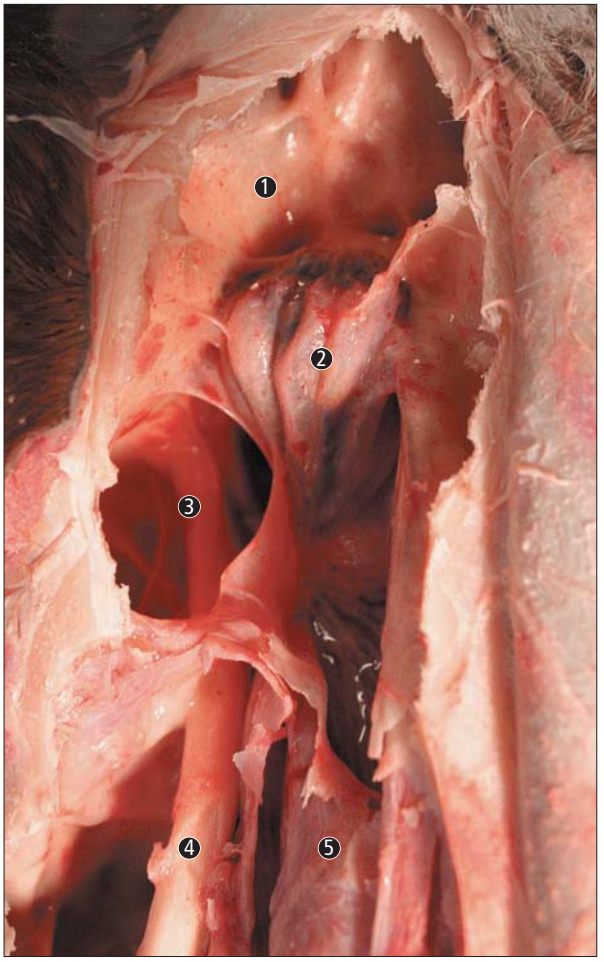
The surgical limits of the maxillary sinus are: dorsally, a line from the medial canthus of the eye to the infraorbital foramen; caudally, a line from the medial canthus to the facial crest; rostrally, a line from the infraorbital foramen to the facial crest; and ventrally, the facial crest (**8**).

Other important anatomic features of the frontal and maxillary sinuses include the following:

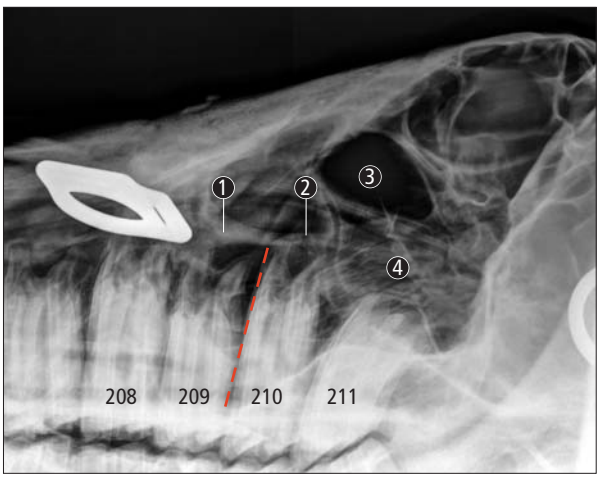
- The rostral limit of the frontal sinus is 2–3 cm from where the nasal bones diverge and the calvarium is close to the caudal limit of the sinus (**9**). Therefore, it is very important not to go beyond the supraorbital foraminae when surgically opening the frontal sinus.
- At the rostral end of the frontal sinus there is a large medial and ventral communication with the dorsal conchal sinus (**10**). At this point the common area between the two is called the conchofrontal sinus. The ethmoidal labyrinth is located on the floor of this sinus between the two orbits. Finally, the frontal sinus communicates with the maxillary sinus via the frontomaxillary opening.
- The rostral and caudal maxillary sinuses are separated by an incomplete bony septum. The floor of the rostral maxillary sinus contains the roots of the 3rd (4th premolar; 108, 208) and 4th (1st molar; 109, 209) cheek teeth (**11**). The floor of the caudal maxillary sinus contains the roots of the 5th (2nd molar; 110, 210) and 6th (3rd molar; 111, 211) cheek teeth.



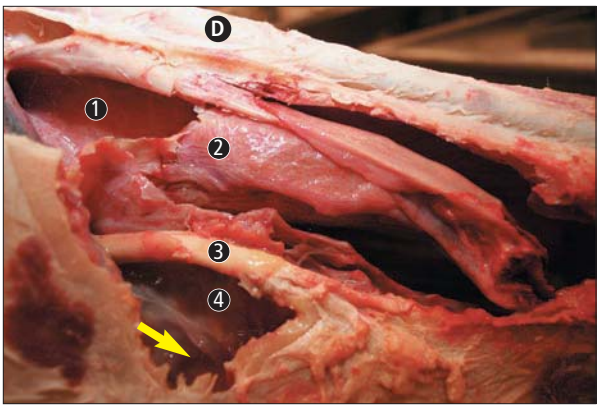
▲ 9 Gross postmortem photograph illustrating the relationship between the caudal aspect of the frontal sinus and the calvarium (circle). Arrows, supraorbital foramina.



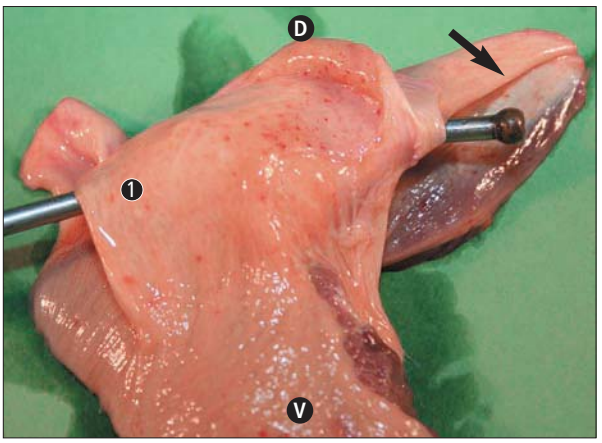
▶ 10 Gross postmortem photograph detailing the relationship between the right frontal sinus (1), the ethmoid turbinates (2), the frontomaxillary opening (3), the infraorbital canal (4), and the dorsal nasal conchae (5).



◀ 11 Digital oblique radiographic view of the left maxillary and frontal sinus of an 11-year-old Thoroughbred gelding. This view demonstrates the location of cheek teeth 208 and 209 in the rostral maxillary sinus (RMS [1]) and cheek teeth 210 and 211 in the caudal maxillary sinus (CMS [2]). The dotted red line represents the bony septum between the RMS and the CMS. (3) frontal sinus; (4) ethmoid turbinates.



▲ 12 **Gross postmortem photograph demonstrating the relationship between the right caudal nasal septum (1), the nasal conchae (2), the infraorbital canal (3), the rostral maxillary sinus (4), and the teeth roots (arrow) within the rostral maxillary sinus. (D) dorsal.**



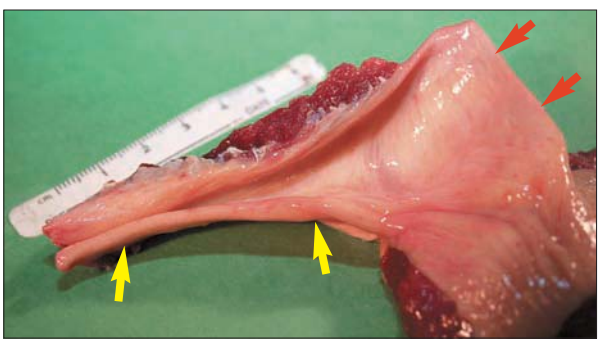
▲ 13 **This specimen portrays the right cartilaginous flap (1) of the guttural pouch and the plica salphingopharyngea (arrow) dissected away from the guttural pouch. A Chambers catheter is being used to elevate the flap. (D) dorsal; (V) ventral.**

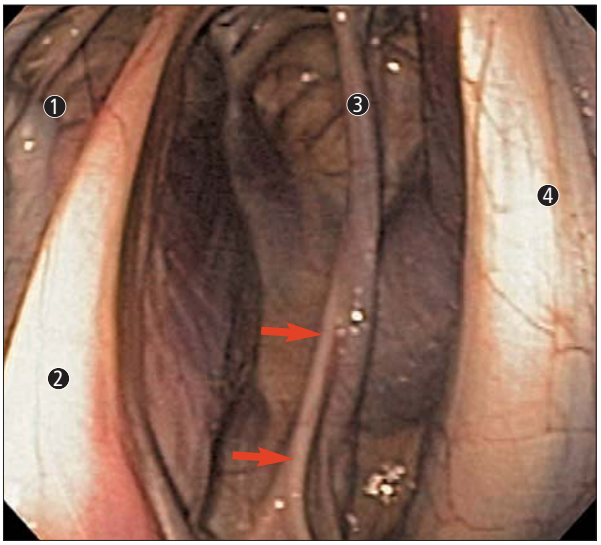
▶ 14 **Gross postmortem photograph detailing the right medial aspect of the guttural pouch cartilaginous flap (red arrows) and the plica salphingopharyngea (yellow arrows).**

- The infraorbital nerve within the infraorbital canal and the lacrimal canal is located along the medial wall of the maxillary sinus, with the lacrimal canal located dorsal to the infraorbital canal (**12**). The volume of the maxillary sinus increases with age as the reserve crowns of the cheek teeth become smaller with use. There is a large opening into the sphenopalatine sinus caudal and medial to the infraorbital canal and a small opening medially into the middle conchal sinus. The maxillary sinus communicates with the nasal passage via the nasomaxillary opening.

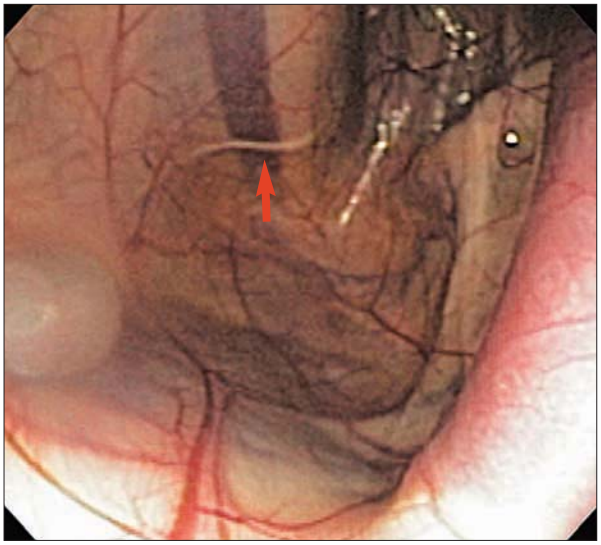
Guttural pouches

The GPs are anatomic structures unique to the horse among all domestic species. Each pouch is a diverticulum of the eustachian tube. The function of the GPs is unknown, though one report has suggested that it is to aid cooling of blood within the carotid and maxillary arteries during exercise. The GPs are paired structures divided by a medial septum. Each pouch has a capacity of approximately 300 ml, although it can accommodate up to 1 liter of fluid due to the distensible nature of the walls. The right and left pouches are separated caudally by the rectus capitus ventralis and longus capitus muscles and rostrally by a thin median septum, formed by the right and left pouch mucosal lining. Each GP communicates with the pharynx through the pharyngeal orifice. A fibrocartilaginous flap closes over the orifice (**13**). The free edge of this flap slopes caudally and ventrally along the wall of the pharynx. The pharyngeal orifice is shaped like a funnel with the mouth of the funnel located rostrally and the end of the funnel





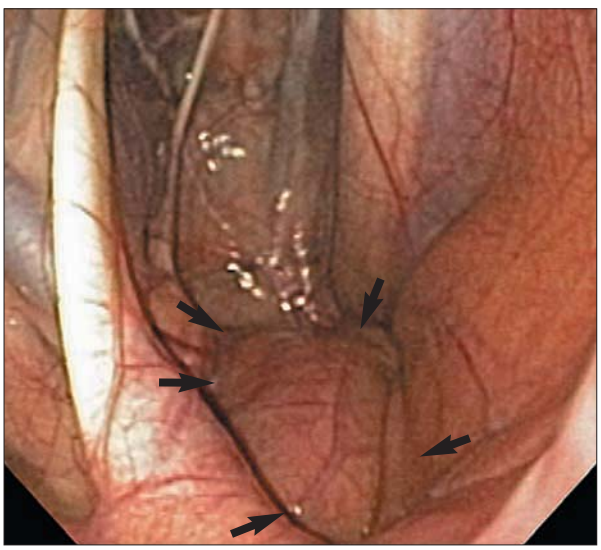
▲ 15 **Endoscopic view of the interior of the medial compartment of the right guttural pouch. (1) maxillary artery; (2) stylohyoid bone; (3) internal carotid artery; (4) longus capitus muscle; arrows, glossopharyngeal and hypoglossal nerves.**



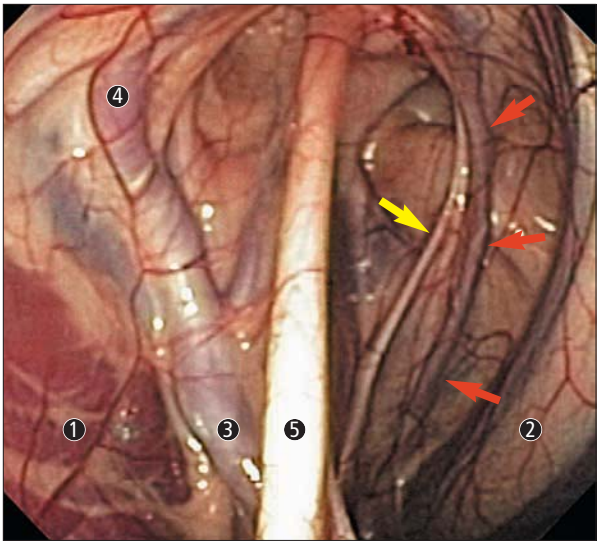
▲ 16 **Endoscopic view of the interior of the left medial compartment of the guttural pouch. The ventral branch of the vagus nerve is visible (arrow).**

located caudally. The caudal narrowing is due to a transverse fold of mucous membrane (plica salpingopharyngea) on the floor of the opening that connects the medial lamina (flap) with the lateral wall of the pharynx (**14**). The stylohyoid bone separates the interior of each pouch into medial and lateral compartments (**15**). The medial compartment is approximately twice the size of the lateral compartment. It contains the internal carotid artery, the cranial cervical ganglion, the cervical sympathetic trunk, the vagus nerve (CN X), the glossopharyngeal nerve (CN IX), the hypoglossal nerve (CN XII), and the spinal accessory nerve (CN XI) along the roof of the guttural pouch and traversing the caudal wall.

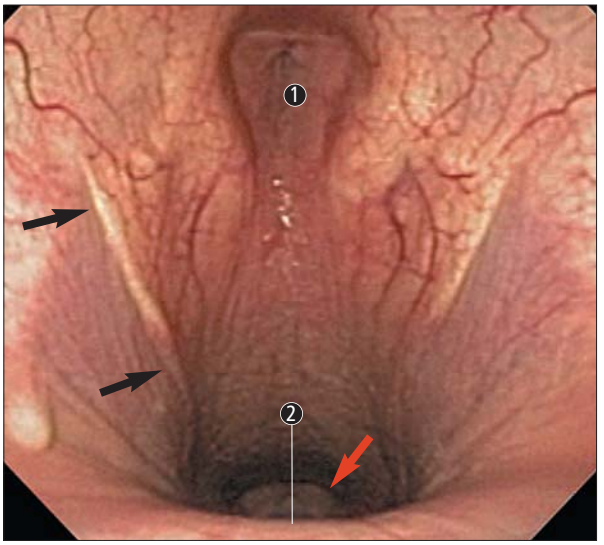
The pharyngeal branch of the vagus nerve (**16**), the cranial laryngeal nerve, and the medial retropharyngeal lymph nodes lie beneath the mucosa on the floor of the medial compartment (**17**).



▲ 17 **Endoscopic view of the right guttural pouch of a horse. Note the enlargement of the retropharyngeal lymph nodes (arrows) on the floor of the medial compartment of the pouch.**



▲ 18 **Endoscopic photograph illustrating the structures within the lateral (1) and medial compartments (2) of the right guttural pouch. (3) external carotid artery; (4) maxillary artery; (5) stylohyoid bone; red arrows, internal carotid artery; yellow arrow, glossopharyngeal and hypoglossal nerves.**



▲ 19 **Endoscopic view of the normal nasopharynx. (1) dorsal pharyngeal recess; (2) soft palate; black arrows, pharyngeal openings into the guttural pouch; red arrow, corniculate processes of the arytenoid cartilages.**

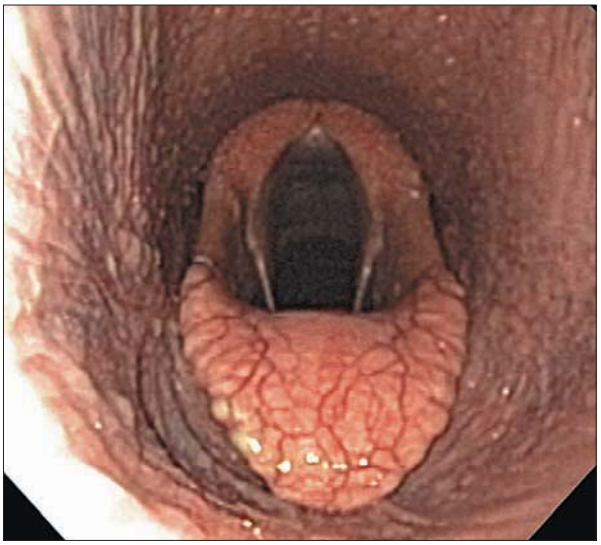
The lateral compartment contains the external carotid artery along the ventral surface, where it is in close association with the glossopharyngeal and hypoglossal nerves (**18**). Dorsally, the external carotid artery continues as the maxillary artery. Also within this compartment is the maxillary vein, the chorda tympani nerve, and branches of the mandibular nerve. The facial nerve (CN VII) courses over the caudal dorsal aspect.

Nasopharynx

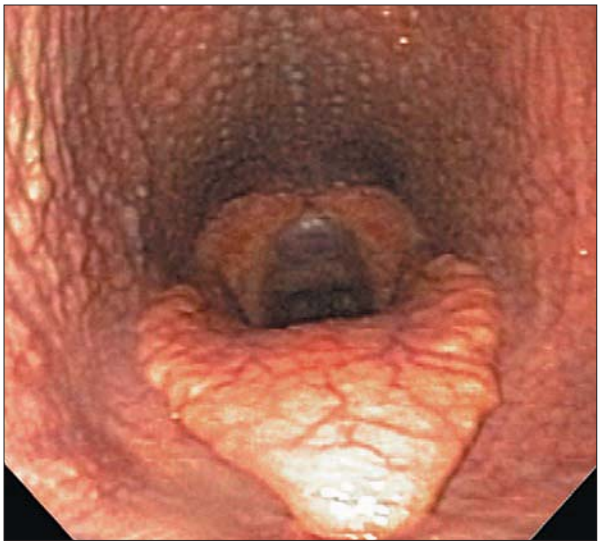
The nasopharynx contains the guttural pouch openings, dorsal pharyngeal recess, soft palate, palatopharyngeal arch, larynx, and esophageal opening (**19**).

The mucosa of the nasopharynx is comprised of a pseudostratified columnar epithelium sprinkled with goblet cells and lymphoid tissue, below which lie elastic connective tissue and muscle. In young horses the lymphoid tissue is active and this can lead to the visual appearance of pharyngeal lymphoid hyperplasia, which may be graded from 1 to 4:

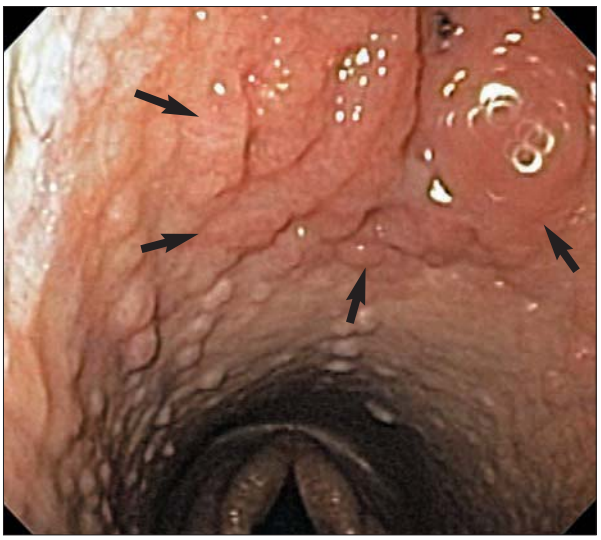
- **Grade 1** is present when there is a small number of white follicles in multiple positions over the dorsal pharyngeal wall (**20**).
- **Grade 2** is present when there are many small, white, lymphoid follicles located over the dorsal and lateral walls of the pharynx, to the level of the guttural pouch openings (**21**). Among the scattered white follicles are numerous follicles that are pink and glistening. Pink or red edematous follicles are considered to be immunologically active.
- **Grade 3** is present when there are many large, coalescing follicles covering the lateral and dorsal pharyngeal wall (**22**). Some of the follicles may spread across the surface of the soft palate.
- **Grade 4** is present when active, pink, glistening follicles cover the pharynx and include the dorsal surface of the soft palate and, occasionally, the surface of the epiglottis and the lining of the guttural pouches (**23**). Some of the follicles coalesce into larger masses.



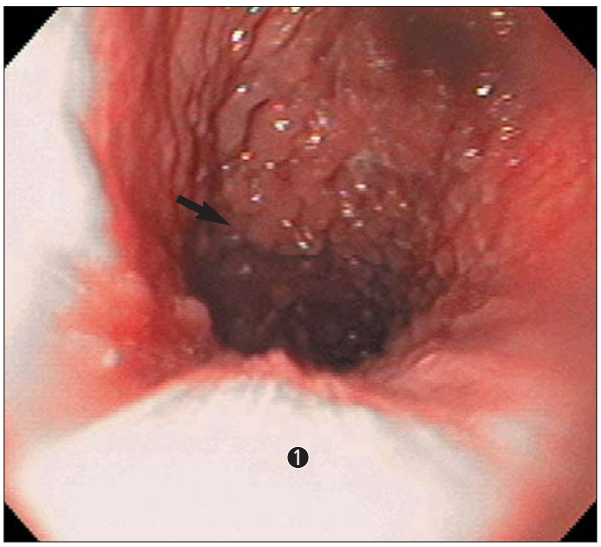
▲ 20 Endoscopic view of a horse with grade 1 pharyngeal lymphoid hyperplasia.



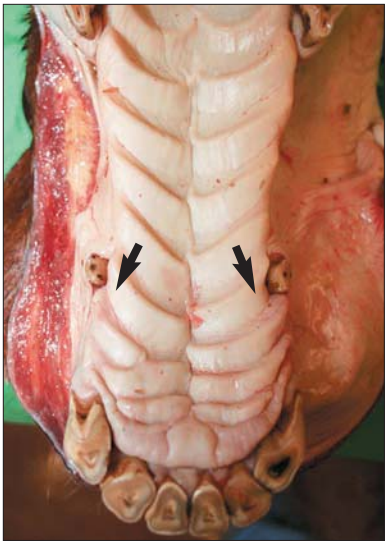
▲ 21 Endoscopic view of a horse with grade 2 pharyngeal lymphoid hyperplasia.



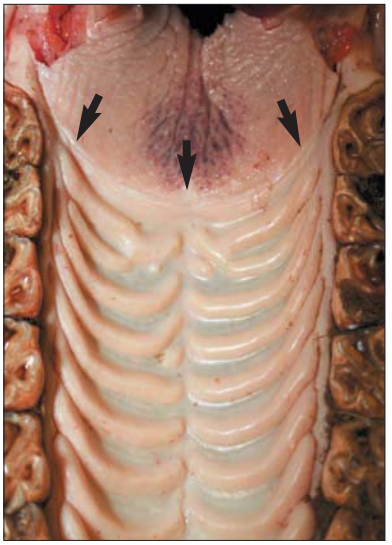
▲ 22 Endoscopic view of a horse with grade 3 pharyngeal lymphoid hyperplasia (arrows).



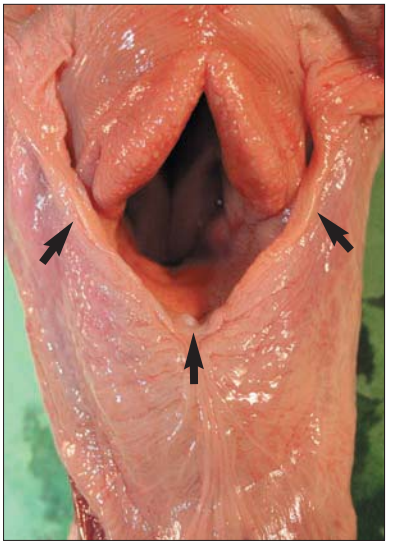
▲ 23 Endoscopic view of a horse with grade 4 pharyngeal lymphoid hyperplasia. The arrow indicates coalescing lymphoid follicles, extending onto the soft palate (1), characteristic of grade 4 pharyngeal lymphoid hyperplasia.



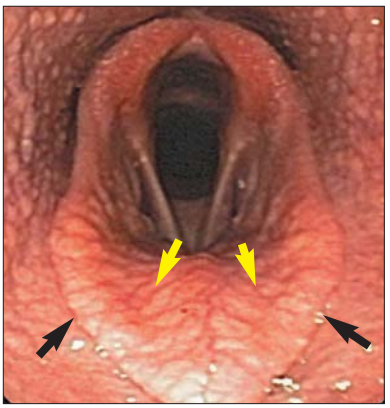
▲ 24 Gross postmortem photograph illustrating a normal hard palate. Note the prominent rugae of the hard palate and the location of the palatine arteries (arrowed) adjacent to the canine teeth.



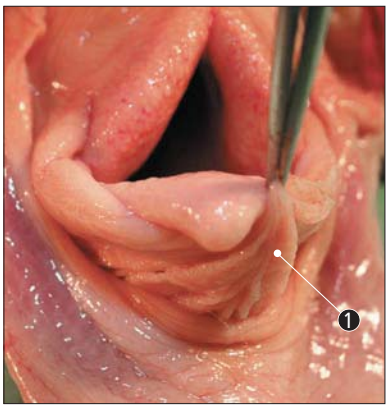
▲ 25 Gross postmortem photograph detailing the junction between the hard and the soft palate (arrows).



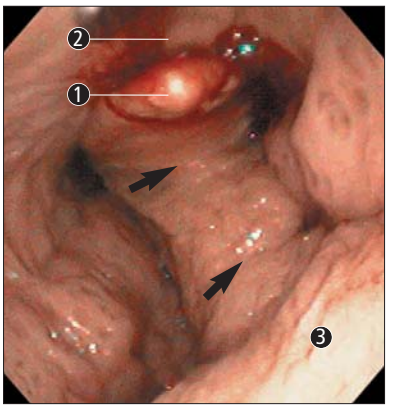
▲ 26 Gross postmortem photograph of normal soft palate. The epiglottis has been deflected ventral to the soft palate to allow visualization of the caudal edge. The arrows indicate the caudal aspect of the soft palate with the edges becoming part of the palatopharyngeal arch.



▲ 27 Endoscopic view of a normal epiglottis. The black arrows identify the serrated edge of the epiglottis and the yellow arrows identify the normal vascular pattern on its dorsal surface.



▲ 28 Gross postmortem photograph illustrating the redundant nature of the aryepiglottic fold (1).



▲ 29 Endoscopic view of the epiglottis (1) in the oral cavity. The soft palate (2) is dorsal to the epiglottis. (3) base of the tongue; arrows, location of the hyoepiglotticus muscle. An epiglottic hook is located dorsal to the epiglottis.

Hard and soft palate

The hard palate begins rostrally at the maxillary incisors and ends caudally approximately at the level of the last caudal cheek tooth (**24**). At this level it intersects with the soft palate. Within the hard palate is the incisive bone. The oral surface of the hard palate consists of mucosal ridges. The major palatine arteries encircle the hard palate and lie in close approximation to the lingual aspect of the canine and cheek teeth.

The soft palate begins rostrally at its intersection with the hard palate and ends caudally as palatopharyngeal arches, which encircle the larynx (**25**). The soft palate at this level has a 'button-hole' appearance with the larynx acting as the 'button' (**26**). The soft palate consists of a mucosal lining of pseudostratified columnar epithelium with underlying muscle and fat. Normally, the soft palate rests beneath the epiglottis. This anatomic feature makes the horse an obligate nose breather.

Epiglottis

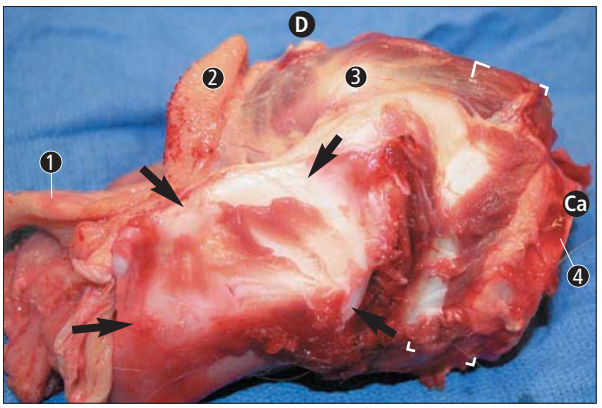
The epiglottis makes up a portion of the larynx. It has a leaf-like appearance (**27**) and several important anatomic features. The edges of the epiglottis appear serrated and there is a prominent vascular pattern on its surface. The epiglottis is attached to the arytenoid cartilages by the aryepiglottic fold (**28**), which originates from the lingual surface of the epiglottis.

The major muscle controlling dorsal and ventral positioning of the epiglottis is the hyoepiglotticus (**29**). Normally, the epiglottis rests dorsal to the soft palate except during deglutition, when it covers the rima glottitis.

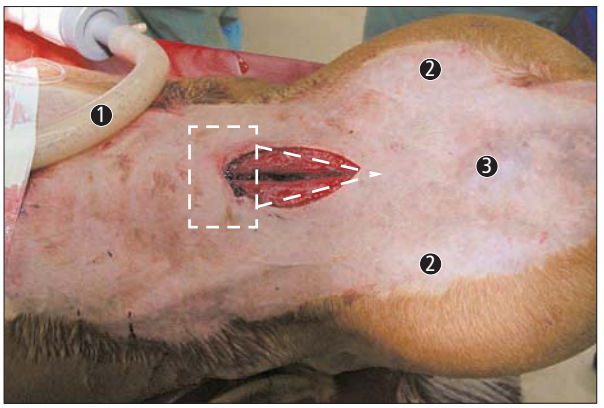
Larynx

The larynx consists of four cartilaginous structures: the epiglottic, thyroid, cricoid, and paired arytenoid cartilages (**30**).

The thyroid cartilage has a keel-like appearance and articulates with the cricoid cartilage via the cricothyroid joint. The cricoid cartilage has a signet ring appearance. The cricothyroid ligament spans the thyroid and cricoid cartilages on the ventral aspect of the larynx (**31**). The cricotracheal space is the junction between the cricoid cartilage and the trachea.



▲ **30** **Gross postmortem photograph of a normal larynx (lateral view). (1) epiglottis; (2) arytenoid cartilage and its muscular process (3); (4) trachea; arrows, thyroid cartilage; dotted line, cricoid cartilage; (D) dorsal; (Ca) caudal.**

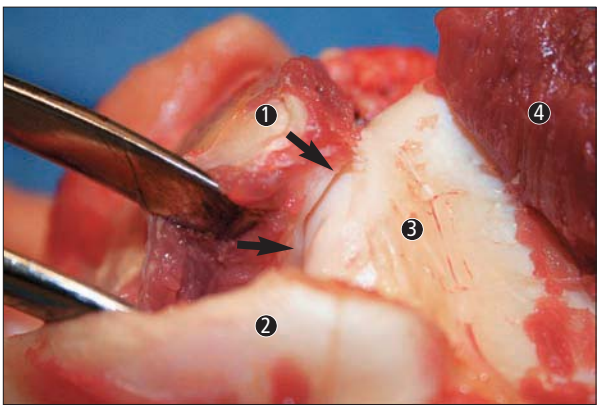


▲ **31** **Intraoperative photograph of a horse in dorsal recumbency. The rectangular box represents the cricoid cartilage. The triangle represents the thyroid cartilage. The surgical incision incises the cricothyroid ligament. (1) endotracheal tube; (2) mandibular rami; (3) basihyoid bone.**

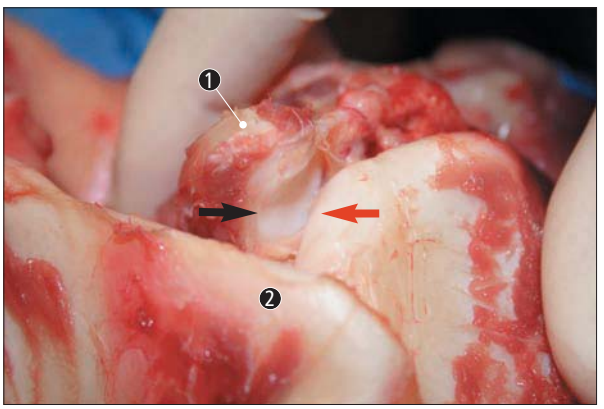
The cricoid cartilage articulates with the arytenoid cartilages via the right and left cricoarytenoid joints (**32**, **33**). This articulation is the major joint allowing movement of the arytenoid cartilages.

The arytenoid cartilages are paired structures. The arytenoid consists of the corniculate, vocal, and muscular processes (**34**). Between these processes is the body of the arytenoid. The vocal process is the insertion point for the vocal cord and the muscular process is the insertion point for the cricoarytenoideus dorsalis (CAD) muscle.

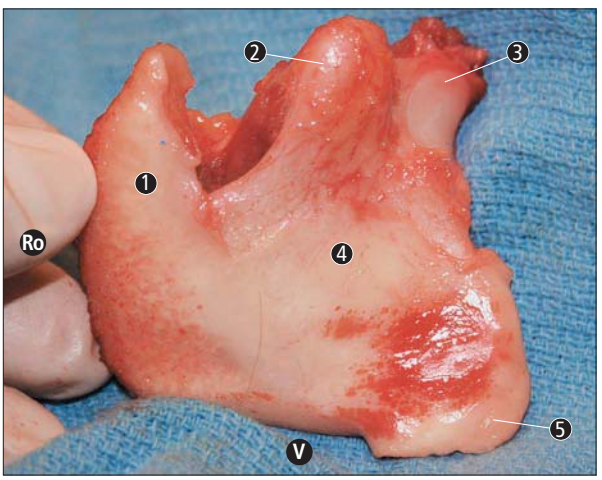
The CAD muscle is the major abductor of the arytenoid cartilage, and is innervated by the recurrent laryngeal nerve. The vocal cord is a fibroelastic



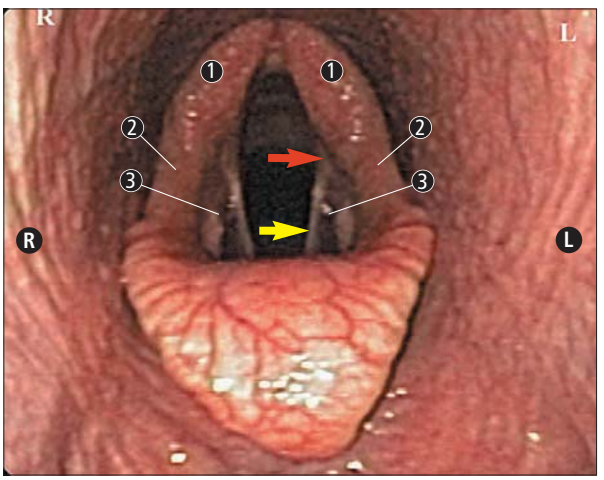
▲ **32** **Gross postmortem photograph showing the left cricoarytenoid joint (arrows), the muscular process (1), the thyroid (2) and cricoid (3) cartilages, and the reflected cricoarytenoideus dorsalis muscle (4).**



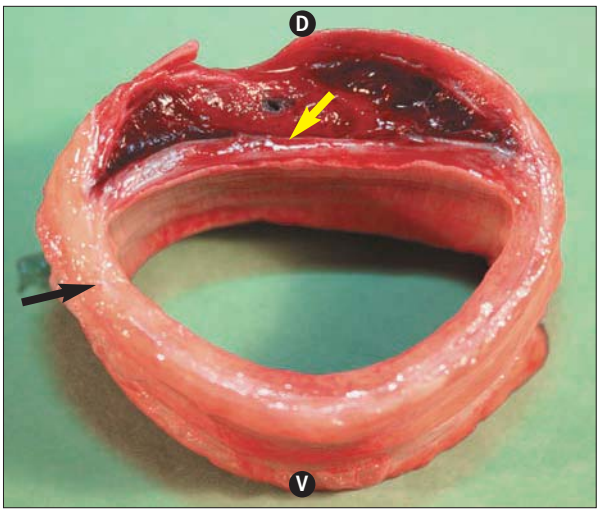
▲ **33** **Gross postmortem photograph showing an opened cricoarytenoid joint. (1) muscular process; (2) thyroid; black arrow, arytenoid facet; red arrrow, cricoid facet.**



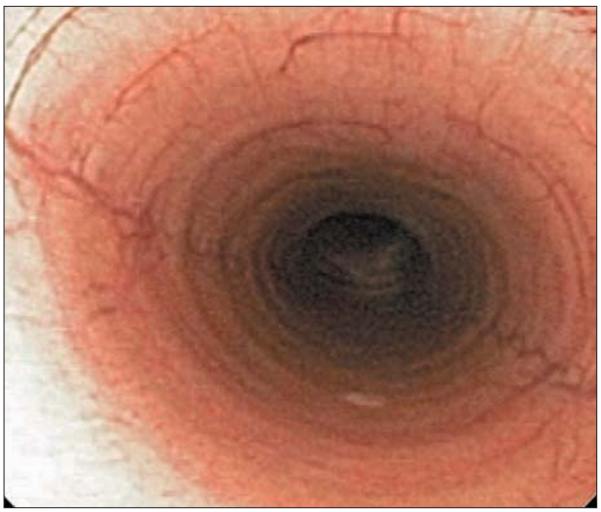
▲ **34** **Complete left arytenoid cartilage dissected free of the remainder of the larynx. (1) corniculate process; (2) muscular process; (3) articular facet of cricoarytenoid joint; (4) body of the arytenoid; (5) vocal process; (Ro) rostral; (V) ventral.**



▲ **35** **Endoscopic view of a normal larynx at rest. (1) corniculate processes of the arytenoid cartilages; (2) aryepiglottic fold; (3) laryngeal ventricle; red arrow, vocal process of the left arytenoid cartilage; yellow arrow, vocal cord; (R) right; (L) left.**



▲ 36 **Gross postmortem photograph of the divided cervical trachea. Black arrow, tracheal cartilage, which is incomplete dorsally; yellow arrow, dorsal tracheal ligament and trachealis muscle; (D) dorsal; (V) ventral.**



▲ 37 **Endoscopic view of a normal trachea.**

structure that originates on the ventral surface of the larynx and inserts on the vocal process of the arytenoid cartilage (**35**). Axial to the vocal cord is the laryngeal ventricle. The laryngeal ventricle is a blind-ended sac that extends caudally for approximately 3–4 cm.

Cervical trachea

The trachea is a flexible, non-collapsing tube that extends 70–80 cm (45–55 cm in ponies) from the cricoid cartilage of the larynx to the hilus of the lung. The trachea spans the distance from the first or second cervical vertebra to the sixth intercostal space. Distally, the trachea bifurcates into right and left principal bronchi. The cervical portion of the trachea extends from the larynx to the thoracic inlet and the thoracic portion extends from the thoracic inlet to the tracheal bifurcation. The thoracic portion of the trachea wall is subjected to pleural pressure, but the lumen is near atmospheric pressure. Proximally, the trachea is superficial and is covered ventrally by the cutaneous colli and sternothyrohyoideus muscles. Distally, towards the thoracic inlet, the trachea is covered ventrally by the sternocephalicus muscle.

The trachea of the horse has 48–60 concentric, incomplete hyaline cartilage rings. These rings are rigid and enclosed in a fibrous membrane, which prevents collapse. They also overlap dorsally to provide flexibility, and because of this the trachea can be collapsed laterally by manual pressure or pressure caused by enlarged external structures adjacent to the trachea (**36**). Dorsally, the trachealis muscle is attached to the inner surface of the tracheal rings and helps to maintain the patency of the tracheal lumen.

The tracheal lumen is lined by a mucous membrane consisting of ciliated pseudostratified columnar epithelial cells and mucus-producing goblet cells (**37**). A blanket of translucent mucus coats the respiratory mucosa, giving it a shiny, wet appearance. The mucus layer aids in removal of particulate foreign material from the respiratory tract. The submucosa, located below the mucous membrane lining, contains elastic tissue, which adds to the flexibility of the trachea.

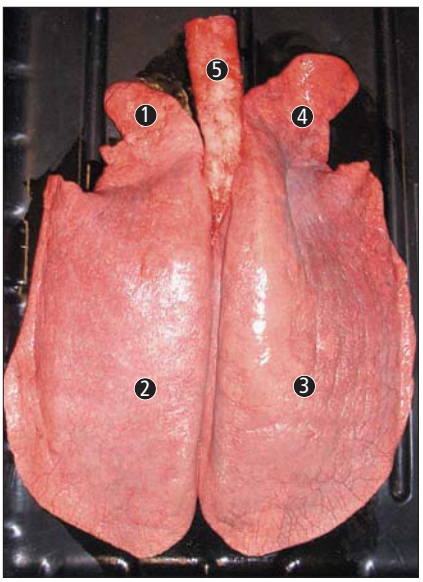
INTRATHORACIC AIRWAYS AND THE LUNGS

The external anatomy of the equine lung reveals no distinction between lobes except for the accessory lobe on the right side. The cardiac notch marks the separation between the cranial and caudal lobes (**38**).

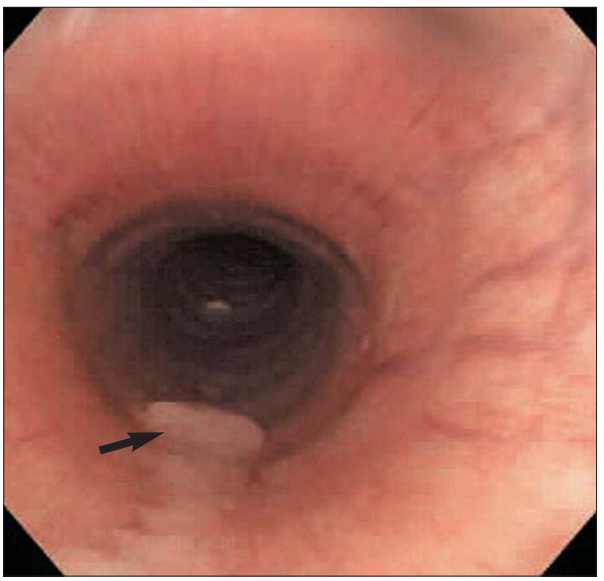
Tracheobronchial tree

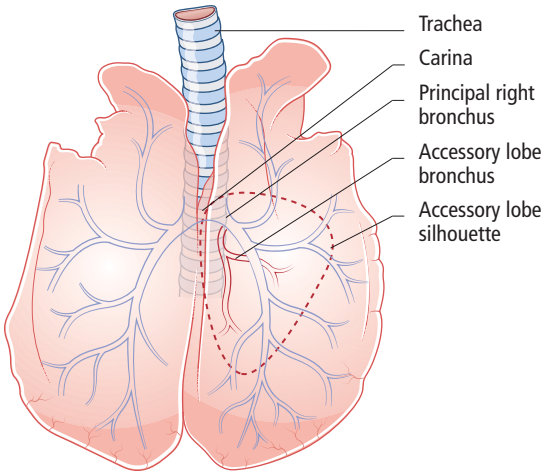
The ventral aspect of the trachea is lowest where it enters the thoracic inlet. As a result, excess mucus tends to pool at this level (**39**) and fluid infused during a tracheal wash will be easily aspirated by advancing the catheter tip in the tracheal puddle. The intrathoracic trachea is usually narrower proximal to the carina because the aorta pushes the left side of the tracheal wall inwards. The trachea ends above the heart base at the level of the left atrium, slightly right of midline. The trachea then gives way to the left and right principal bronchi at the carina (**40, 41**). After the carina, the airways are divided into lobar, segmental, and subsegmental bronchi, bronchioles, and, finally, terminal bronchioles. The right cranial lobar bronchus originates from the right principal bronchus at approximately the 10 o'clock position (**42**). The accessory lobar bronchus originates from the right principal bronchus at the 4 o'clock position, next to the first bronchus of the right caudal lung lobe at 7 o'clock. The right principal bronchus then becomes the right caudal lobar bronchus. The left principal bronchus becomes the left caudal lobar bronchus after supplying the left cranial lobar bronchus at the 2 o'clock position (**42**). Terminal bronchioles are the smallest conducting airways (i.e. without alveoli). Conducting airways do not participate in gas exchange, although diffusion may occur in terminal bronchioles since convective flow is near zero. Conducting airways constitute the anatomic dead space. The terminal bronchioles divide into respiratory bronchioles, then into alveolar ducts and end in alveolar sacs (**43**). Respiratory bronchioles are poorly developed in the horse but are the first airways with occasional alveoli protruding from their walls, whereas alveolar ducts and alveolar sacs are lined with alveoli. The distal airways containing alveoli constitute the respiratory zone.

▼ 38 Gross postmortem photograph of the costal surface of the lungs of a healthy horse showing the lack of interlobar fissures. (1) left cranial lobe; (2) left caudal lobe; (3) right caudal lobe; (4) right cranial lobe; (5) trachea.

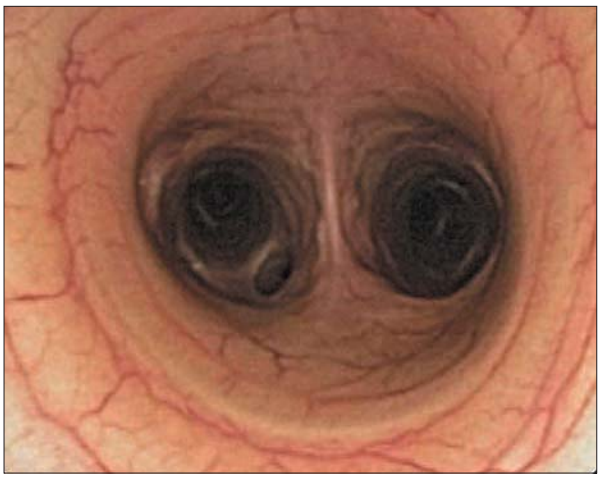


▼ 39 Endoscopic view of the trachea at the level of the thoracic inlet. White mucoid secretions may be seen accumulated on the floor of the trachea (arrow).



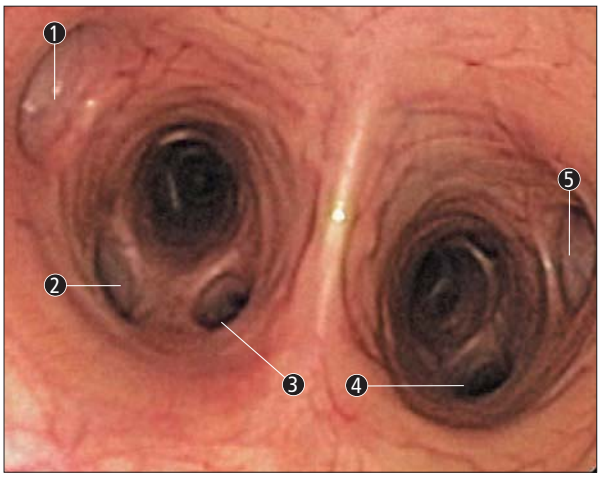


▲ 40 Tracheobronchial tree anatomy illustrated from a dorsal (costal) view of the lungs.



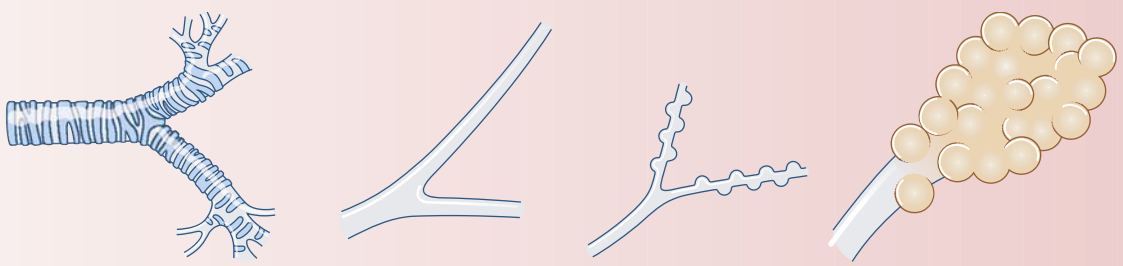
▲ 41 Endoscopic view of the carina (135 cm from nares) in a healthy adult horse.

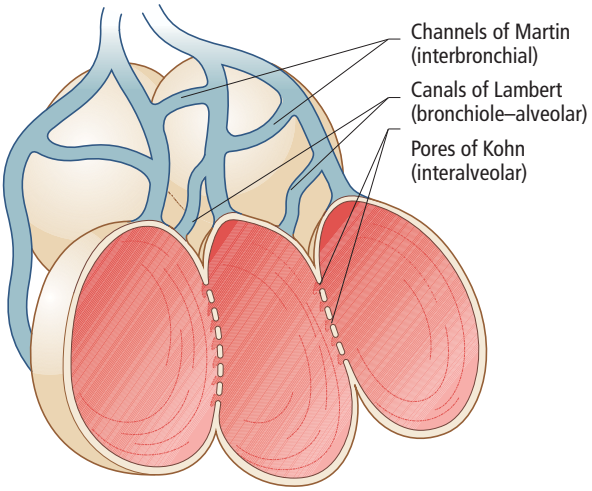
▶ 42 Endoscopic view of the carina (145 cm from nares). (1) right cranial lobar bronchus; (2) first right caudal lobar bronchus; (3) accessory lobe bronchus; (4) first left caudal lobar bronchus; (5) left cranial lobar bronchus.



▼ 43 Schematic illustrating the subdivisions of the tracheobronchial tree from the large cartilaginous airways (trachea and bronchi) to the smaller membranous bronchioles and, finally, the respiratory bronchioles and alveoli where gas exchange occurs.

CONDUCTING AIRWAYS						GAS EXCHANGE AIRWAYS				
Cartilaginous				Membranous		Respiratory zone				
Trachea	Bronchi			Bronchioles		Respiratory bronchioles	Alveolar ducts		Alveolar sacs	
Division 0	1	2	→10	11–13	→16	17–19	20	22	23	24





▲ **44 Schematic representation of the different types of collateral ventilation that can bypass airflow along the tracheobronchial tree.**

During inhalation, airflow through the conducting zone reaches maximal velocity in the proximal airways. The velocity of the gas decreases dramatically as air progresses beyond the terminal bronchioles because of the marked increase in total cross-sectional diameter of the respiratory zone. This rapid drop in gas velocity explains why dust particles frequently settle in terminal bronchioles. The lack of ventilation in areas distal to obstructed airways may be compensated partially by collateral ventilation through pathways other than the tracheobronchial tree. These pathways include interalveolar pores of Kohn, canals of Lambert, and communicating respiratory bronchioles and alveolar ducts (**44**). Collateral ventilation in the horse supplies a maximum of 16% of respiratory volume, which represents one-fifth of the amount reported for the dog.

Comparative respiratory physiology studies indicate that the size of the functional lung unit (i.e. alveolar size, thickness of the diffusion barrier) is relatively constant across mammals, with pulmonary volumes, total alveolar surface area, and pulmonary capillary volume increasing linearly with body weight. Consequently, the capacity of the lung for oxygen diffusion (i.e. pulmonary diffusion capacity) increases with body weight. However, the alveolar surface density of athletic animals, in particular the horse, is comparatively larger than that of non-athletic animals (100% higher than in ponies and 10–30% higher than in dogs). Horses also possess larger lung volumes per unit of body weight compared with ponies, which explains, in part, the relatively higher mass-specific oxygen consumption in horses.

Pulmonary circulation

The pulmonary artery receives the deoxygenated blood from the right side of the heart and then divides into branches that follow the conducting airways to form a dense network of capillaries in the alveolar walls. The diameter of pulmonary capillaries is just large enough for a red blood cell (5–6 µm in the horse) and the blood–gas barrier is as thin as 0.5 µm. Pulmonary artery branches adjacent to peripheral airways (bronchioles to alveolar ducts) contain a thick layer of smooth muscle that contracts in response to hypoxia. The hypoxic vasoconstriction of the small pulmonary arteries varies in intensity from species to species and the intensity appears correlated to the thickness of the smooth muscle layer of these arteries. Horses show an intermediate response compared with cattle (strongest) and dogs (minimal). This hypoxic vasoconstriction may be advantageous for improving ventilation/perfusion mismatch; however, it may result in excessive vasoconstriction and pulmonary hypertension in conditions such as heaves (recurrent airway obstruction, RAO). The oxygenated blood returns to the left side of the heart via the pulmonary veins.

Bronchial circulation

The bronchial circulation originates from the aorta and supplies oxygenated blood to the conducting airways, pleura, and associated structures. The right apical bronchial artery supplies blood to airways of the right apical lobe and the rest of the lung is supplied by the bronchoesophageal artery. The blood is carried away from the lungs via the azygos and pulmonary veins. The flow through the bronchial circulation represents a small fraction of that through the pulmonary circulation.

The respiratory tract is constantly exposed to a variety of microbes, particles, and potentially toxic gases present in the air. Protection of the tract is based on mechanical and immunologic defense mechanisms. The design of conducting airways, in particular the nasal passages, allows trapping of larger particles (>10 μm) onto the mucosa. Deposited particles are trapped on the surface of the mucus layer, which is being constantly produced by goblet (mucous) cells, serous cells, submucosal glands, and transepithelial fluid exchange along conducting airways. The mucociliary apparatus creates a constant flow of mucus from the periphery of the lung (respiratory bronchioles) towards the pharynx, where it is eventually swallowed. The alveoli have no cilia and particles deposited there are phagocytized by alveolar macrophages.

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CHAPTER 2

PULMONARY FUNCTION

VENTILATION

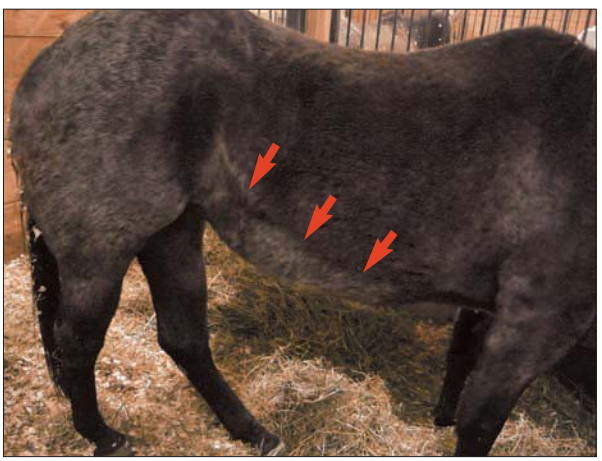
Ventilation is the process by which lung volume changes in response to rhythmic contraction of the respiratory muscles. The action of respiratory muscles on the chest is capable of producing a wide range of lung volumes and flows.

Respiratory muscles

Some muscles of the proximal airways, notably the nose, pharynx, and larynx, contribute to ventilation by controlling airway resistance. The activity of these muscles is particularly important during exercise where flaring of the nostrils, contraction of the pharyngeal dilator muscles, and abduction of the arytenoid cartilages result in an increased diameter of the airways, thereby reducing the effort (work of breathing [see Mechanics of breathing]) performed by the respiratory muscles acting on the chest wall.

Movements of the lung are entirely passive and result from the action of respiratory muscles on the chest wall and diaphragm. In humans, as in most other mammals breathing at rest, inspiration is primarily active and expiration is primarily passive. The horse constitutes an exception to this rule with a passive and active component to both inspiration and expiration.

The main inspiratory muscle is the diaphragm, which separates the abdomen from the thorax. The muscle attaches ventrally to the xyphoid process, laterally to the costochondral junctions of the eighth to fourteenth ribs, and dorsally to the ventral aspect of the first three lumbar vertebrae. The diaphragm is convex cranially and its apex extends to the level of the eighth intercostal space. The external intercostal muscles located between the ribs and the serratus ventralis also contribute to inspiration. The expiratory muscles are the abdominal muscles (rectus abdominis, external and internal obliques, and transversalis muscles) and internal intercostal muscles. Contraction of abdominal muscles results in an increase in abdominal pressure and displacement of the diaphragm in a cranial direction. Horses affected by chronic pulmonary airway obstruction resulting in marked increased respiratory efforts, such as heaves, will over time develop hypertrophy of the external abdominal oblique muscles. This is called a 'heave line' (**45**). Respiratory muscles



◀ **45 Hypertrophy of the external abdominal oblique muscle visible as a 'heave line' (arrows) in a horse with heaves or recurrent airway obstruction. The line is very prominent during exhalation.**

receive approximately 10% of the cardiac output during rest and 15% during maximal exercise. With strenuous exercise, blood flow to the diaphragm and other respiratory muscles increases by approximately 30 times the resting values. Of all the respiratory muscles, the diaphragm receives the greatest blood flow relative to tissue mass. However, a higher percentage of cardiac output is supplying the serratus ventralis because its mass is 80% greater than that of the diaphragm.

In the horse, the first part of expiration is passive, as in humans, but subsequent contraction of abdominal muscles generates the second, active phase of expiration. Consequently, the first part of inspiration is passive and is followed by an active phase involving contraction of the diaphragm and external intercostal muscles. Therefore, the horse breathes not from, but around, the relaxation volume of the respiratory tract, also called functional residual capacity (FRC).

Lung volumes and ventilation

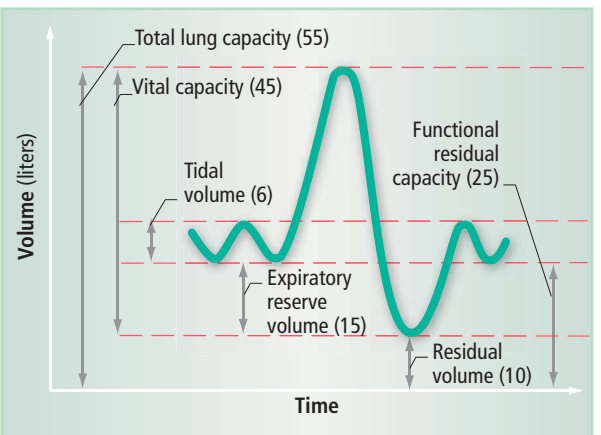
The subdivision of lung volumes is summarized in **46**. The volume of air inhaled and exhaled during each breathing cycle is called the tidal volume (V_T). Average resting V_T in horses and ponies ranges from 10–14 ml/kg. Minute ventilation is the product of V_T and breathing frequency (f). Expired and inspired V_T are not the same, therefore there is a difference between expired and inspired minute ventilation. Expired minute ventilation ($\dot{V}_E$) increases with exercise intensity from resting values around 60 l/min to reach values up to 1,600 l/min in horses exercising strenuously. The rise in minute ventilation required to ensure adequate gas exchange can be achieved by increasing V_T, breathing frequency, or both. In trotting horses, both breathing frequency and V_T increase as minute ventilation increases, with the contribution of the respiratory frequency being twice that of the V_T. In galloping horses, breathing frequency and V_T increase similarly when exercising at slow speed; however, further increase in minute ventilation is mainly the result of increased V_T. The difference in breathing strategy is probably due to the tight coupling between breathing and locomotion (one breath per stride) in gallopers. For example, a galloper running on a flat treadmill at a given speed will increase its minute ventilation by a rise in V_T when the treadmill is tilted up.

At the end of expiration in a patient at rest, when no air is flowing, the inward elastic recoil of the lung is exactly balanced by the outward recoil of the chest wall. The volume of air present in the relaxed respiratory tract is then the FRC. In horses, the end-expiratory lung volume is smaller than the FRC because of active muscle contraction during the last phase of expiration.

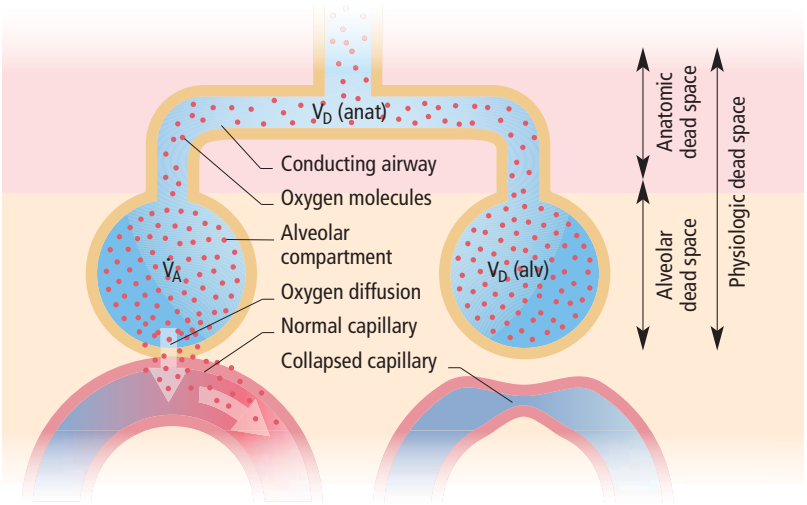
The volume of air contained in the respiratory tract at maximal inspiration is the total lung capacity (TLC) (**46**). The volume exhaled after a maximal expiration from TLC is the vital capacity (VC) and that remaining in the lung is the residual volume (RV).

The air contained in the conducting airways does not participate in gas exchange and this volume is referred to as anatomic dead space (**47**). The volume of air contained in the alveolar spaces of the lung, but not taking part in gas exchange, is the alveolar dead space. Gas in these alveoli does not come into contact with blood due to poor perfusion of the alveoli, resulting in wasted ventilation. The physiologic dead space (V_D) is the sum of anatomic and alveolar dead spaces. The physiologic dead space to tidal volume ratio (V_D/V_T) indicates the wasted part of a breath. In the horse, V_D/V_T is approximately twice as large as reported in other athletic species such as humans and dogs. The effective part of the ventilation participating in gas exchange is the alveolar ventilation ($\dot{V}_A$). The relationship between $\dot{V}_A$ and V_T is as follows:

$$\dot{V}_A = f(V_T - V_D)$$



▲ **46 Average lung volume and capacities (liters) in a healthy, 500 kg horse.**



◀ 47 **Conducting airways where no gas exchange takes place are known as 'anatomic dead space'. Physiologic dead space includes both anatomic dead space V_D (anat) and aveolar dead space V_D (alv) – alveoli that are well ventilated but poorly perfused due to lack of blood flow. V_A, alveolar gas volume.**

Minute ventilation may be expressed as the sum of the physiologic and alveolar dead space ventilation:

$$\dot{V}_E = \dot{V}_D + \dot{V}_A$$

In other words, optimal gas exchange at a given $\dot{V}_E$ occurs when $\dot{V}_A$ is the highest, which is to say when V_D/V_T is the lowest.

Exercise results in increased $\dot{V}_E$; however, the effect on $\dot{V}_A$ and V_D/V_T depends on exercise intensity and duration. During mild to moderate exercise, V_D remains constant and V_T increases, resulting in lower V_D/V_T and improved ventilation efficiency. However, if this type of exercise is sustained, breathing frequency and V_D/V_T will increase, resulting in a rise in $\dot{V}_D$. Presumably this breathing strategy favors thermoregulation, which becomes a significant issue during prolonged exercise. During intense exercise, V_D/V_T decreases from 60% to 20% with a 30% drop in V_D. A good example of exercise-induced drop in V_D/V_T occurs when the incline on the treadmill increases, causing the horse to breathe deeper and slower, in keeping with the slower stride frequency. The anatomic dead space is not expected to change with exercise. Consequently, the drop in V_D is likely attributable to a reduction in alveolar dead space due to recruitment of previously underperfused pulmonary capillary beds.

Regional differences in ventilation

In the horse, as in other mammals, there is a vertical gradient of pleural pressure due to gravitational force with the lowest pressure (negative relative to atmospheric pressure) being recorded dorsally. As a result, transpulmonary pressure at FRC is greater dorsally than ventrally and dorsal alveoli are more distended than ventral alveoli. The rate of inflation of an area of lung depends on inflation pressure, airway resistance, and compliance. The product of compliance and resistance is a time constant. Therefore, for a given inflation pressure, the lung areas that will fill faster are the areas with a short time constant (i.e. the dorsal lung areas because they are characterized by low resistance and low compliance). However, ventilation of the ventral lung is greater because for the same pressure change during inspiration, the more compliant alveoli (dependent lung) will experience a greater change in volume. Other factors that may influence regional ventilation are the shape of the thoracic cavity, the orientation of respiratory muscles, body position, and anesthesia that relaxes the respiratory muscles.

During low-frequency breathing, regional differences in ventilation have no significant effect on gas exchanges. However, at high frequencies of breathing, such as during exercise, both temporal and spatial inequalities of ventilation are highest. Horses with airway inflammation may experience various degrees of airway obstruction that contribute to inequalities of ventilation. These inequalities are magnified during exercise and potentially result in abnormal gas exchanges or lung injury.

DIFFUSION

Respiratory gases

For a given barometric pressure (e.g. Pb = 760 mmHg at sea level), the partial pressure of gases is equivalent to their concentration in the gas mixture or in plasma and tissues when physically dissolved, multiplied by Pb. The composition of ambient air is 78.08% nitrogen, 20.95% oxygen (O_2), and 0.03% carbon dioxide (CO_2), corresponding to partial pressures of approximately 159 mmHg (760 × 0.2095) for O_2 and 0.23 mmHg (760 × 0.0003) for CO_2. Air becomes saturated with water after passing through the nasal passages. At a body temperature of 37°C, water vapor exerts a partial pressure of 47 mmHg. Consequently, the partial pressure of inspired gases decreases as they travel down the conducting airways. For example, the partial pressure of inspired oxygen (P_{IO_2}) decreases from 159 mmHg to 149 mmHg ([760 – 47] × 0.2095).

The composition of alveolar gases is not constant. It lies between that of inspired air and gases in mixed venous blood depending on the rate of alveolar ventilation and diffusion of gases across the alveolar capillary membrane, according to the following alveolar gas equations:

$$F_{AO_2} = F_{IO_2} - \dot{V}_{O_2}/\dot{V}_A$$
$$F_{ACO_2} = F_{ICO_2} + \dot{V}_{CO_2}/\dot{V}_A$$

where F represents the concentration of the gas in alveoli (F_A) or in inspired air (F_I) and $\dot{V}$ is the rate of exchange of the relevant gases. The same equations apply to partial pressure of respiratory gases.

$$P_{AO_2} = P_{IO_2} - \dot{V}_{O_2}/\dot{V}_A$$
$$P_{ACO_2} = P_{ICO_2} + \dot{V}_{CO_2}/\dot{V}_A$$

As alveolar ventilation increases, alveolar gas composition becomes closer to inspired gases. When alveolar ventilation decreases, alveolar gas composition approaches that of mixed venous blood. On average, alveolar gas composition at rest is 13.6% O_2, 5.6% CO_2, 6.2% water, and 74.9% nitrogen, corresponding to partial pressures of approximately 100 mmHg for O_2 and 40 mmHg for CO_2.

Laws of diffusion

The process of diffusion corresponds to the transfer of gas across the blood–gas barrier at the level of the alveoli. Gas diffusion may be described by Fick's law, which states that the rate of transfer of a gas (v) across a membrane is inversely proportional to its thickness (T) and is proportional to the pressure gradient of the gas across the membrane ($P_A - P_{cap}$), a diffusion constant (D), and the surface area (A) available for transfer.

$$v = A \times D \times (P_A - P_{cap}) \times 1/T$$

The diffusion constant is proportional to the solubility of the gas and inversely proportional to the square root of its molecular weight. Because CO_2 and O_2 are of similar molecular weight (44 and 32, respectively), but CO_2 solubility is 23.7 times that of O_2, the rate of CO_2 diffusion is 20.3 times that of O_2.

Diffusion limitation

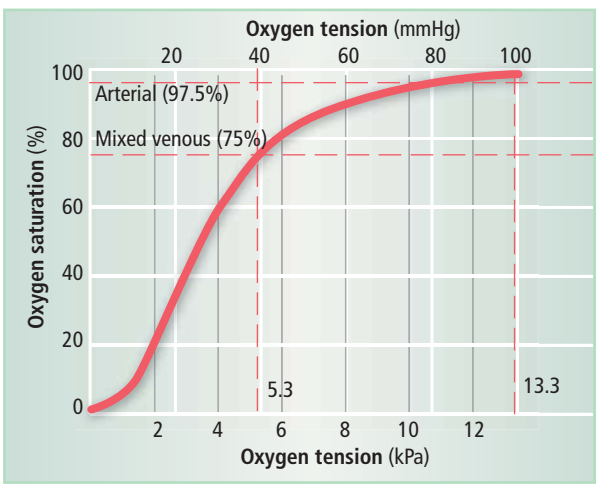
The partial pressure of oxygen (P_{O_2}) in a red blood cell (RBC) entering a pulmonary capillary is approximately 40 mmHg in a healthy animal. Across the alveolar capillary membrane is the alveolar gas with alveolar oxygen partial pressure (P_{AO_2}) of approximately 100 mmHg. Therefore, a large force of 60 mmHg is driving oxygen across the membrane into the blood. The difference between the ideal P_{AO_2} and the measured partial pressure of oxygen in arterial blood (P_{aO_2}) is called the alveolar–arterial difference for oxygen ($A–aD_{O_2}$). In the healthy horse at rest, the $A–aD_{O_2}$ is <10 mmHg. The $A–aD_{O_2}$ remains constant during mild to moderate exercise intensity (i.e. <50% $\dot{V}_{O_2max}$); however, at higher exercise intensity $A–aD_{O_2}$ increases to values up to 40 mmHg. No significant diffusion limitation is detected at rest or during moderate exercise intensity where $A–aD_{O_2}$ results mainly from $\dot{V}/\dot{Q}$ mismatch. As exercise intensity increases, diffusion limitation accounts for >75% of the widening in $A–aD_{O_2}$. Less than 25 % is due to $\dot{V}/\dot{Q}$ mismatch, while total right-to-left shunt remains negligible.

The transfer of oxygen across the alveolar capillary membrane is rapid and virtually complete by the time the RBC has been in the capillary for 0.25 seconds.

RBC transit time in capillaries at rest is estimated to be 0.7 seconds. In horses exercising at $\dot{V}_{O_2max}$, the transit time has been calculated at 0.4–0.5 seconds. Therefore, it should be expected that diffusion of oxygen across the alveolar capillary membrane is not limited in horses exercising strenuously. To the contrary, horses exercising strenuously exhibit marked exercise-induced hypoxemia as a result mainly of diffusion limitation. The reason for this apparent discrepancy is not known. Interestingly, dogs do not experience significant diffusion limitation during heavy exercise despite a shorter capillary transit time, estimated at around 0.3 seconds.

Transport of gases in blood

The concentration of a gas carried by the blood varies as a function of the type of gas transported and its partial pressure in the blood. O_2 and CO_2 are mainly transported in chemically combined forms and a smaller fraction is dissolved in plasma. As a result, the relationship between respiratory gas concentration and partial pressure follows a curvilinear relationship, also called a dissociation curve (**48**). Gases that are physically dissolved in blood, but do not combine chemically with it, have a linear relationship between concentration and pressure (e.g. nitrous oxide).



Oxygen

O_2 is carried in the blood in two forms: combined with hemoglobin (Hb) and dissolved in plasma and RBC fluid. The solubility of O_2 in plasma is 0.01 mmol/l/kPa (0.003 ml/dl/mmHg) at 37ºC, which corresponds to approximately 1.5% of the total blood O_2 content or 0.3 ml of O_2 per 100 ml of plasma when PaO_2 is 13.3 kPa (100 mmHg). Most of the oxygen transported is in combination with Hb or oxyhemoglobin. The Hb molecule is composed of four protein chains each capable of binding one molecule of oxygen. For some time the oxygen-carrying capacity of Hb was believed to be somewhere between 1.34 and 1.39 ml/g. However, direct measurements of the oxygen-carrying capacity of human Hb have shown values of 1.306 ml/g for adult and 1.312 ml/g for fetal blood. The oxygen content of arterial blood (C_{aO_2}) may be calculated according to the following equation:

$$C_{aO_2}\,(ml/dl) = 0.003 \times P_{aO_2}\,(mmHg) + [Hb]\,(g/dl) \times S_{aO_2} \times 1.3$$

In resting horses, Hb concentration ranges from 110–190 g/l (11–19 g/dl), with most values in non-excited horses ranging between 120 and 150 g/l (12 and 15 g/dl). Corresponding C_{aO_2} average values are about 16–20 ml/dl of blood. The Hb concentration increases moderately with training. Exercise is associated with a marked increase in Hb concentration (217 ± 9 g/l [21.7 ± 0.9 g/dl]) compared with resting values because of splenic contraction. As a result, C_{aO_2} increases with speed of exercise despite reductions in P_{aO_2} and S_{aO_2}. However, in fit racehorses, C_{aO_2} rises to a maximum during moderately intense exercise and then decreases as exercise continues to intensify.

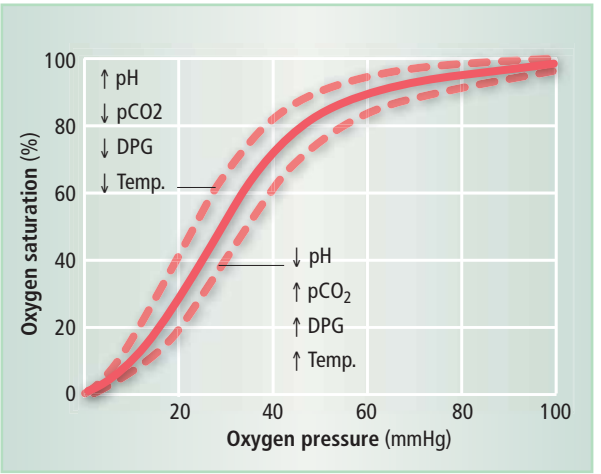
The shape of the oxygen dissociation curve is sigmoid, with the upper part of the curve flattening as P_{O_2} increases towards values obtained in the lung. The flat part of the curve tends to stabilize the quantity of oxygen in the arterial blood and maximizes saturation of hemoglobin (e.g. S_{aO_2} = 97.5% and PaO_2 = 13.3 kPa or 99.6 mmHg for normal blood at pH = 7.4 and

◀ 48 Dissociation curve of hemoglobin in standard conditions (pH = 7, temperature 37ºC, zero base excess).

temperature = 37°C). The steep middle part of the curve ensures that a large portion of the oxygen is delivered to tissues at a relatively high P_{O_2} (e.g. S_{aO_2} = 75% and P_{aO_2} = 5.4 kPa or 40.4 mmHg for normal blood at pH = 7.4 and temperature = 37°C). If a horse develops moderate hypoxemia (e.g. P_{aO_2} = 60 mmHg) or a 40 mmHg drop from normal (e.g. P_{aO_2} = 100 mmHg), then the corresponding decrease in S_{aO_2} would be only 8.5% (97.5 – 89 %). Therefore, P_{aO_2} is a more sensitive indicator of a mild degree of hypoxemia than S_{aO_2}.

The position of the oxyhemoglobin dissociation curve may be defined by the P_{O_2} at 50% oxygen saturation or P50. In horses, P50 is 24.8 ± 2.0 mmHg under standard conditions, which is lower than the human standard P50 (26.6 ± 1.2 mmHg). Breed has an effect on P50, with values being lowest in Thoroughbred horses (21.2 ± 1.1 mmHg) and intermediate in Hanoverian horses (23.8 ± 0.8 mmHg).

The strength of the bond between O_2 and Hb depends on factors such as temperature, pH, P_{CO_2}, and RBC concentration of 2,3-diphosphoglycerate (2,3-DPG) (**49**). CO_2 diffuses into alveoli when the blood passes through the lungs, resulting in a decrease in the blood P_{CO_2} and also a decrease in hydrogen ion concentration (due to the decrease in blood carbonic acid). This shifts the dissociation curve to the left. Therefore, the amount of O_2 that binds with Hb at any given P_{AO_2} increases and provides for greater O_2 transport to the tissues. When the blood reaches tissue capillaries, CO_2 enters the blood and shifts the curve to the right, thus displacing O_2 from Hb and allowing O_2 delivery to occur at a higher P_{O_2}. This phenomenon is also known as the Bohr effect. During exercise the oxygen dissociation curve is shifted to the right in relation to the increase in blood temperature and P_{CO_2} and the decrease in pH and RBC concentration of 2,3-DPG. The Bohr effect reported in horses is similar to the human Bohr effect; however, the effects of temperature and P_{CO_2} are smaller in the blood of horses compared with human blood. During peak exercise in horses, the oxyhemoglobin dissociation curve shifts to a P50 value, resulting in optimal oxygen transport.



▲ **49 Bohr effect. There is a shift of the oxyhemoglobin dissociation curve to the right (reduced affinity for oxygen) when pH decreases or CO_2 tension, 2,3-DPG concentration, and temperature increase. A shift to the left (increased affinity for oxygen) is produced by opposite blood changes.**

Carbon dioxide

CO_2 is the end-product of aerobic metabolism, most of it being produced in the mitochondria. From the tissue, CO_2 diffuses into the blood along P_{CO_2} gradients. In the lungs, it diffuses back into the alveoli. CO_2 is transported in the blood in three forms: dissolved in plasma, bound to hemoglobin (carboxyhemoglobin), and in the form of bicarbonate. The solubility of CO_2 in plasma is 0.231 mmol/l/kPa (0.0308 mmol/l/mmHg) under standard conditions or 23.1 times higher than O_2. Approximately 5% of CO_2 is transported in solution, where it exerts a tension of 5.3 kPa (P_{aCO_2} = 40 mmHg) in arterial blood and 6.1 kPa (P_{vCO_2} = 46 mmHg) in mixed venous blood. While in solution, some of the CO_2 combines with water to form carbonic

acid and the enzyme carbonic anhydrase accelerates the reaction in both directions. Carbonic acid can then dissociate into bicarbonate and hydrogen ions:

$$H_2O + CO_2 \leftrightarrow H_2CO_3 \leftrightarrow HCO_3^- + H^+$$

Under physiologic conditions, carbonic acid is about 96% dissociated. As a result, approximately 90% of CO_2 is transported in the blood as bicarbonate. The remaining 5% is combined to reduced hemoglobin to form a carbamino compound. The reaction is facilitated in tissue capillaries because the quantity of reduced hemoglobin increases as oxyhemoglobin releases its oxygen. The process is reversed in the lungs, thereby facilitating the uptake of CO_2 from the tissues and its release into the lungs (the Haldane effect). In addition, very small amounts of CO_2 are carried in carbamino compounds with plasma proteins.

During exercise, large quantities of CO_2 are released by locomotor muscles, a process that is matched by a parallel increase in ventilation. In humans exercising at moderate levels, blood gas values are maintained at resting values, but as exercise intensity increases beyond the lactic threshold, P_{aCO_2} falls while P_{aO_2} remains relatively constant. Horses are unique among mammals because they are unable to eliminate CO_2 during strenuous exercise and become progressively hypercapnic, resulting in O_2 consumption $\geq$90% $\dot{V}O_{2max}$. It has been suggested that the mechanical constraint on ventilation because of the coupling of stride with breathing in horses explains exercise-induced hypercapnia. However, horses exercising strenuously in hypoxic conditions become normocapnic by achieving higher ventilation compared with the level achieved under normoxic conditions. Therefore, ventilation is not limited during exercise at high intensities, but the horse does not adopt a breathing strategy to normalize CO_2 even if it can. This phenomenon may be a normal control mechanism with the aim of minimizing the mechanical cost of breathing. The shift in the oxyhemoglobin curve occurring during peak exercise also maximizes CO_2 output through optimization of the Haldane effect.

VENTILATION–PERFUSION RELATIONSHIP

Ideally, gas exchange would be optimum if alveolar gas and pulmonary capillary blood were delivered to the lung in the same amounts and each alveolus was equally ventilated and perfused ($\dot{V}/\dot{Q} = 1$). However, ventilation and perfusion are not evenly distributed in the normal lung, ranging from not ventilated but perfused alveoli with a $\dot{V}/\dot{Q} = 0$ to ventilated but not perfused alveoli with a $\dot{V}/\dot{Q}$ = infinity. Horses at rest are better at matching ventilation and perfusion, with $\dot{V}/\dot{Q}$ centered around 1 and little $\dot{V}/\dot{Q}$ inequality, in comparison with young, healthy humans. Furthermore, $\dot{V}/\dot{Q}$ is unaffected by exercise and accounts for <25% of the increase in $A–aD_{O_2}$.

Airway disease, such as recurrent airway obstruction (RAO), may result in significant $\dot{V}/\dot{Q}$ mismatching at rest and lead to hypoxemia and hypercapnia. Inhomogeneity in $\dot{V}/\dot{Q}$ is likely to be magnified during exercise and lead to more pronounced gas exchange abnormalities.

MECHANICS OF BREATHING

The work of breathing generated by the action of respiratory muscles on the thorax and the elastic recoil of the lungs and chest wall is responsible for ventilation. The movements of the lungs are entirely passive. It is useful to consider the respiratory system as a passive mechanical system composed of simple elements each with a particular elastance (1/compliance), resistance, and inertance. (*Note:* Pulmonary inertance is the ratio of transpulmonary pressure gradient due to inertia and the volume of air accelerated [i.e. the portion of differential pressure needed to accelerate and decelerate air during breathing]). Active and passive forces create a pressure gradient between the atmosphere and the alveoli, resulting in airflow. The pressure gradient (ΔP) is the sum of the pressures needed to overcome the elastic (P_{el}), flow-resistive (P_r), and inertial (P_{in}) forces of the lung and chest wall.

$$\Delta P = P_{el} + P_r + P_{in}$$

The forces necessary to overcome the elastic recoil (compliance) of the lung and chest wall may be measured when gas is not flowing within the lung. Flow-resistive forces resulting from frictional resistance to gas flow through the airways (airway resistance) and viscoelastic deformation of the thorax (tissue resistance), and inertia associated with acceleration of gas and tissue are measured while gas is flowing in the airways. The balance of forces acting on the respiratory system may be described by the equation of motion for a three-dimensional mechanical system analogue:

$$\Delta P = (1/C)V + R\dot{V} + I\ddot{V}$$

The pressure gradient is a function of lung compliance (C) and volume (V), frictional resistance (R) and flow ($\dot{V}$), and inertance (I) and acceleration ($\ddot{V}$) of the respiratory system. Inertial forces are negligible at rest, which allows for calculation of resistance and compliance based on the equation of motion if pressure and airflow are measured. During exercise or tachypnea (e.g. >42 breaths/min), inertial forces increase considerably and should be taken into account.

Airflow resistance

Components of total airflow resistance

During quiet breathing, the pressure gradient required to inflate the lungs can be divided into an elastic and a resistive component according to the simplified equation of motion:

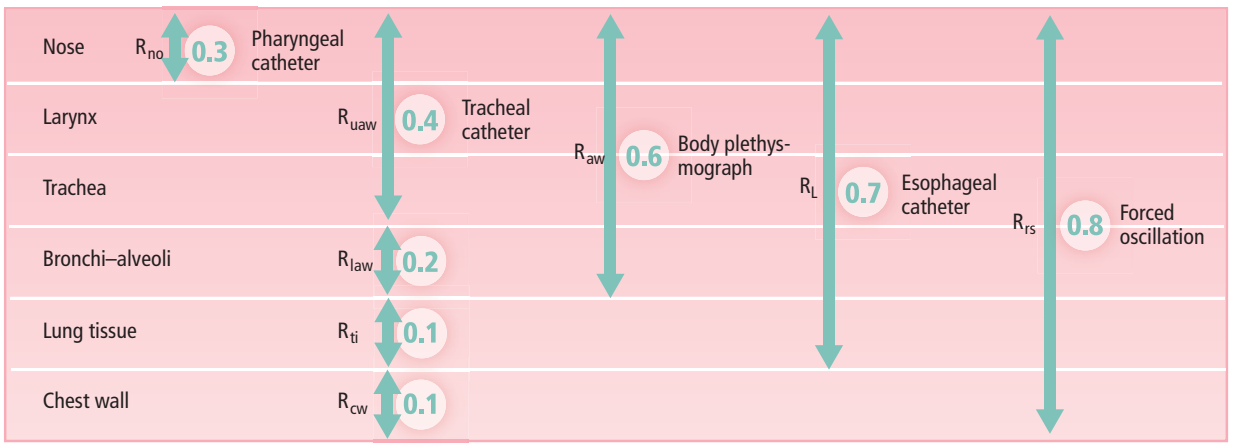
$$\Delta P = (1/C)V + R\dot{V}$$

The resistive component of the respiratory system (R_{rs}) can be further divided into the pressure gradient required through the tracheobronchial airways (airway resistance, R_{aw}), across the lung tissue (lung tissue resistance, R_{ti}) and through the chest wall (chest wall resistance, R_{cw}) in order to generate airflow. All the components of the respiratory system are arranged in series, therefore R_{rs} is the sum of each segment's resistance (**50**):

$$R_{rs} = R_{aw} + R_{ti} + R_{cw}$$

The drop in pressure through the airways is proportional to R_{aw} and airflow; however, the drop in pressure across lung tissue and the chest wall is not proportional to airflow, but depends on end-expiratory lung volume, V_T, and breathing frequency. For example, R_{ti} and R_{cw} will be higher if the animal is initiating V_T from a higher end-expiratory lung volume.

Various techniques may be used to measure the different components of R_{rs}. Body plethysmography allows direct measurement of R_{aw}. The esophageal balloon catheter technique provides a measurement of



▼ **50 Resistive components of the respiratory system and measurement methods. The total airflow resistance (R_{rs}) is the sum (in $cmH_2O/l/s$) of all the components ($R_{uaw} + R_{law} + R_{ti} + R_{cw}$). R_{no}, nasal resistance; R_{uaw}, upper airway resistance; R_{law}, lower airway resistance; R_{ti}, lung tissue resistance; R_{cw}, chest wall resistance; R_L, lung resistance; R_{rs}, respiratory system resistance.**

transpulmonary pressure (P_{tp}) and allows computation of total lung resistance (R_L), which is the sum of R_{aw} and R_{ti}. Finally, measurement of R_{rs} is only feasible using the forced oscillation technique. All three methods have been described in the horse. In healthy horses breathing quietly at rest, it is estimated that R_L represents approximately 80% of R_{rs}.

Laminar gas flow rate through a tube is directly proportional to the pressure gradient along the tube and the ratio is a constant defined as resistance to gas flow:

$$\Delta P/\dot{V} = R \text{ (kPa/l/s or cm}H_2O\text{/l/s)}$$

Resistance may be determined by the length (L) and radius (r) of the tube and by the viscosity of the gas (μ) according to the Hagen–Poiseuille equation:

$$R = (8\,L\mu) / \pi\, r^4$$

This equation explains the major importance of airway narrowing in breathing mechanics because a 50% decrease in airway caliber will result in a 16-fold increase in resistance to airflow.

Laminar flow becomes turbulent in conditions of high flow rates, in particular through irregular or branched tubes. The pressure–flow relationship in these conditions is not linear anymore because resistance increases in proportion to the flow rate. As flow increases, the simple relationship between pressure and flow may be extended by adding a term accounting for turbulent flow:

$$P = K_1\dot{V} + K_2 \times \dot{V}^2$$

in which K_1 and K_2 are constants. Alternatively, the pressure–flow relationship may be accurately estimated over a wide range of flow rates using an exponential function:

$$P = K\dot{V}^n$$

in which K and n are constants, with the latter ranging between 1 and 2 depending on whether the flow is laminar (n = 1) or fully turbulent (n = 2).

Partitioning of airflow resistance

Nasal airflow resistance in humans represents the largest portion of airway resistance (50%) during nasal breathing; however, after switching to mouth breathing, upper airway resistance decreases to one-third of total airway resistance. Unlike humans, horses are obligatory nasal breathers, therefore nasal resistance largely dictates the work and energy cost of breathing during exercise. Estimations of the contribution of the upper airways to the total respiratory resistance vary markedly between studies. Some authors have reported that upper airway resistance in horses represents 10–30% of total respiratory resistance, and that nasopharyngeal resistance accounts for 80–90% of upper airway resistance at rest. Others have measured the contribution of the upper airways to the total respiratory resistance to range from 50% to 85%, with 50% being the most common estimate. The different results between these studies may have a methodological basis.

During exercise, the increase in ventilation is facilitated by the action of specific respiratory muscles and sympathetic nerve activation that lead to increased cross-sectional areas of the upper airways, hence decreasing resistance to airflow. Nostril flaring, nasal passage mucosal vasoconstriction, and laryngeal abduction are well known representations of this phenomenon. Despite these adaptations, nasal resistance remains higher than oral resistance to airflow and most humans switch from nasal to oronasal breathing as soon as the ventilatory rate reaches approximately 50% of its maximum. The horse, being an obligatory nose-breather, cannot switch from nasal to oronasal breathing in order to decrease upper airway resistance to airflow. Furthermore, nasal resistance to airflow rises as the intensity of exercise increases, and thus remains the major contributor to total respiratory resistance.

In the tracheobronchial tree, airway diameters become smaller when advancing from the central airways towards the peripheral airways, but at the same time the number of branching airways increases exponentially. As a result, the total cross-sectional area of all the airways is larger in the lung periphery and most of the resistance to flow is in the central airways with internal diameters >2 mm, since flow rates are much higher in these segments.

Gas flow through the upper and central airway (e.g. the trachea) is turbulent, particularly during exercise. The pressure drop across the upper and central airways is therefore proportional to the airflow through the airway to the power of 2. Laminar flow conditions are present in peripheral airways and result in pressure losses that are directly proportional to airflow and inversely proportional to the square of the cross-sectional area.

Lung elasticity

When no air flows through the respiratory tract and the respiratory muscles are relaxed, the tendency of the lung to contract is balanced by the tendency of the rib cage to expand. This recoil of the lung is generated by elastic fibers in the parenchyma and the surface tension existing at the level of the air/water interface in the alveoli. The surface tension of the alveolar lining fluid is kept at a low level by the presence of surfactant. Surfactant is composed of 90% lipids (most of them phospholipids), 10% proteins, and small amounts of carbohydrates. Alveolar epithelial type II cells secrete surfactant, which is stored in lamellar bodies. Elastic forces and surface tension provide the lung with an intrinsic stiffness, also called compliance. Lung compliance is proportional to the change in lung volume per unit change in P_{tp} (i.e. pressure gradient between pleural cavity and alveoli).

$$C = \Delta V/\Delta P \quad (l/kPa \text{ or } l/cmH_2O)$$

Pleural pressure is often approximated by measuring esophageal pressure using the balloon catheter technique. Lung volume increases as P_{tp} rises and the relationship is almost linear over the range of normal tidal volume. Lung compliance is the slope of the near linear segment of the pressure–volume curve which occurs during deflation. However, as P_{tp} continues to rise, lung volume increases in smaller increments until TLC is reached. During deflation of the lung, the pressure–volume relationship will be shifted because of the viscoelastic properties of the lung, a phenomenon known as hysteresis. Consequently, the resting volume of the respiratory system is higher when reached after deflation from TLC than after inflation from RV.

Following inflation to a given lung volume in a patient under general anesthesia, P_{tp} falls from its initial value and stabilizes within 1 minute to a level approximately 20–30% lower. Static compliance is determined by the change in lung volume divided by the change in P_{tp} (ΔP_{tp}) after enough time has been allowed for stabilization of P_{tp}, whereas dynamic compliance is measured during tidal breathing as the change in lung volume divided by the initial ΔP_{tp}. Therefore, static compliance will be greater than dynamic compliance by an amount dependent on the elastic behavior of a particular lung. Elastance is the reciprocal of compliance. Stiff lungs have a high elastance and a low compliance.

Pulmonary inertance

The pressure gradient required to accelerate or decelerate respiratory gases is proportional to pulmonary inertance (I) and $\dot{V}$. As a result, inertance may be calculated as follows:

$$I = \Delta P/\dot{V} \quad (kPa/l/s^2 \text{ or } cmH_2O/l/s^2)$$

In humans, pulmonary inertance is negligible at rest, but should be taken into consideration during high frequency breathing or in patients with a large body weight. In horses, the pressure gradient required to accelerate respiratory gases during quiet breathing represents 1.5% of ΔP_{tp} (0.01 kPa versus 0.65 kPa) and, therefore, can be neglected. However, the pressure gradient needed to produce acceleration of gases during exercise accounts for approximately 50% of ΔP_{tp} (1.9 kPa versus 4 kPa).

CHAPTER 3

CLINICAL EXAMINATION

GENERAL INSPECTION

Historical and clinical findings will help the clinician determine the origin of the respiratory disease and generate a list of differential diagnoses. Assessment of body condition is useful in the evaluation of diseases where weight loss and poor body condition are indicative of chronic conditions (e.g. chronic pneumonia/pleuritis, neoplasia, RAO, chronic pain). The presence of ventral edema is often visible with pleural disease as a result of decreased lymphatic return (e.g. pleural effusion, pleuropneumonia, thoracic lymphoma).

It is important to evaluate behavior and posture. Lethargy may be associated with hypoxemia, sepsis, fever, or neoplasia. Severe hypoxemia, airway obstruction, or chest pain may result in agitated, aggressive, or dangerous behavior.

Signalment

Many respiratory diseases affect horses at a particular age. Coughing and nasal regurgitation of milk while nursing in a neonatal foal indicate a possible congenital physical abnormality, such as cleft palate, dorsal displacement of the soft palate (DDSP), subepiglottic cyst, or a functional abnormality secondary to diseases such as hypoxic ischemic encephalopathy, hydrocephalus, or pharyngeal–cricopharyngeal incoordination. Arabian foals developing repeated bouts of pneumonia between birth and 2 months of age should be suspected of combined immunodeficiency. Signs of pneumonia unresponsive to beta-lactam and aminoglycoside antimicrobials presenting in foals between 1 and 3 months of age strongly suggests *Rhodococcus equi* infection. An acute onset of cough, fever, and serous nasal discharge in young horses suggests a potential viral etiology (e.g. influenza or herpesvirus).

History

Obtaining a detailed vaccination history of the horse and other horses on the premises, or of the mare in the case of a foal, provides important information in cases of contagious disease. Horses sharing pasture with donkeys are at risk of acquiring lungworm infection. The type of anthelmintic used and frequency of administration may suggest the possibility of lungworm infection.

The geographic location and history of travel may help determine the likelihood of certain diseases. For example, *R. equi* can multiply in the intestines of foals and in soil under certain environmental conditions (e.g. high temperature). In addition, the main route of infection is via contaminated dust inhalation. As a result, *R. equi* infection is endemic on some farms with a high concentration of horses and a hot climate. Previous medical history will help establish possible chronicity or recurrence of the condition. Weight loss would indicate chronicity of the disease.

Seasonality of the clinical signs suggests an allergic condition. Horses suffering from summer pasture-associated RAO tend to develop clinical signs while at pasture between June and September (in the northern hemisphere). Similarly, horses with RAO often exhibit clinical signs during the cold season when they are housed indoors and fed hay.

Horses subjected to stressful situations, such as long travel, strenuous exercise, or general anesthesia, are more likely to be immunosuppressed due to reduced phagocytic clearance of inhaled pathogens and are at risk of developing diseases such as pneumonia and pleuropneumonia. A history of recent contact with other horses at a sale barn, show, racetrack, or breeding farm or the introduction of a new horse should raise the suspicion of contagious diseases such as strangles and viral respiratory diseases.

Assessing the housing conditions of the horse and the type of feed is important. Horses housed in poorly ventilated stalls, fed insufficiently cured hay, and bedded on straw are exposed to high levels of organic molds and endotoxins, which may result in lower airway inflammation and bronchial hyperresponsiveness. This type of environment is likely to induce or exacerbate signs of respiratory disease in horses with RAO or infectious respiratory disease. Horses with RAO kept outdoors but fed hay from round bales left in the field and exposed to the elements tend to exhibit worsening of the clinical signs.

Assessment of the timing of the cough may guide diagnostic tests. Cough associated with feeding may indicate allergy to inhaled molds (e.g. heaves), inflammatory diseases of the pharynx and larynx, esophageal obstruction, or dysphagia with passage of ingesta in the airways. Exercise often exacerbates coughing because of the higher mechanical stress placed on pulmonary cough receptors and the larger amount of particles inhaled with the increase in ventilation. Coughing following strenuous exercise is more suggestive of exercise-induced pulmonary hemorrhage (EIPH).

Breathing pattern

Movements of the lung are entirely passive and result from the action of respiratory muscles on the chest wall. In humans, as in most other mammals breathing at rest, inspiration is primarily active and expiration is primarily passive. The horse is an exception to this rule with a passive and active component to both inspiration and expiration.

In the horse, the first part of expiration is passive, but subsequent contraction of abdominal muscles generates the second, active phase of expiration. Consequently, the first part of inspiration is also passive, as abdominal muscles relax, and this is followed by an active phase involving contraction of the diaphragm and intercostal muscles. Therefore, the horse breathes not from but around the relaxation volume of the respiratory tract, also called FRC.

Several breathing patterns are recognized (*Table 1*). Tachypnea, hyperventilation, and hyperpnea are accompanied by varying degrees of nostril flaring, head and neck extension, and increased breathing effort. Hyperpnea is associated with normal blood gases, whereas hyperventilation and tachypnea are associated with abnormal gas exchanges.

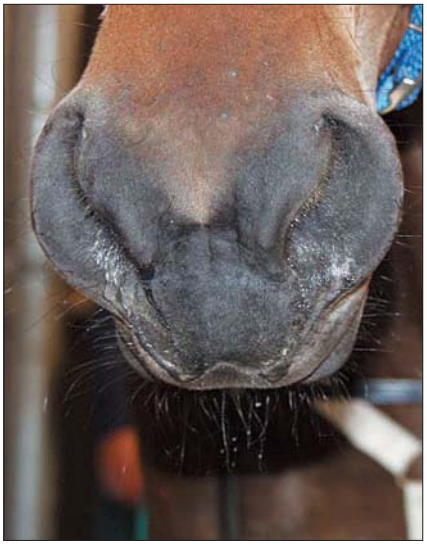
Table 1 **Breathing patterns in the horse**

BREATHING PATTERN	DEFINITION	CAUSES
Eupnea	Normal quiet breathing at rest	Metabolic demand
Hyperpnea	Increased breathing rate and depth to meet metabolic demand	Exercise; high altitude
Tachypnea	Abnormally rapid breathing	Lung and heart disease; anemia; thermoregulation
Hyperventilation	Increased alveolar ventilation causing decreased P_{aCO_2} (beyond demand)	Stress; lung and CNS disease
Hypoventilation	Decreased alveolar ventilation causing increased P_{aCO_2}	CNS and neuromuscular diseases, pleural effusion; sedation
Respiratory distress or dyspnea	Labored breathing	Respiratory and cardiac diseases; acute, severe anemia

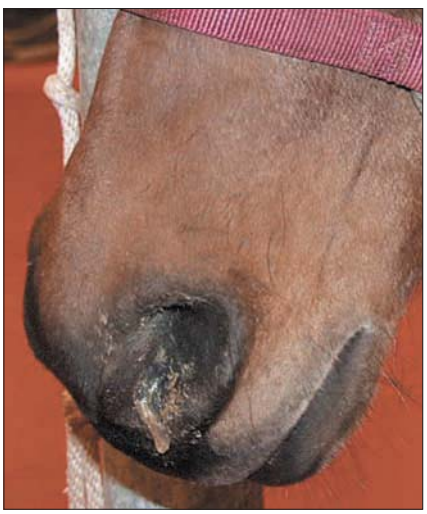
Respiratory distress is defined as labored breathing characterized by an exaggerated effort to breathe relative to the degree of physical activity. Dyspnea is defined as 'undue awareness of breathing or awareness of difficulty in breathing' and has been used interchangeably with respiratory distress in the veterinary literature. However, the term dyspnea is inappropriate in veterinary patients because they cannot verbalize breathing difficulty. Clinical signs associated with respiratory distress in the horse include inactivity, exercise intolerance, anxious or restless expression, flaring of the nostrils, increased respiratory effort, asynchrony between thoracic and abdominal movements, stridor, extended head and neck, cyanosis, and pumping of the anus synchronized with the respiratory cycle. Acute respiratory distress may be a manifestation of impaired gas exchanges resulting from obstruction of conducting airways, failure of the muscles and structures responsible for ventilation, pulmonary disease, or cardiac disease. Acute, severe anemia from blood loss or hemolysis (e.g. red maple leaf toxicosis, neonatal isoerythrolysis) can also cause respiratory distress. Labored breathing in response to pain, hyperthermia, and metabolic acidosis is not associated with abnormalities in gas exchange.

Nasal discharge

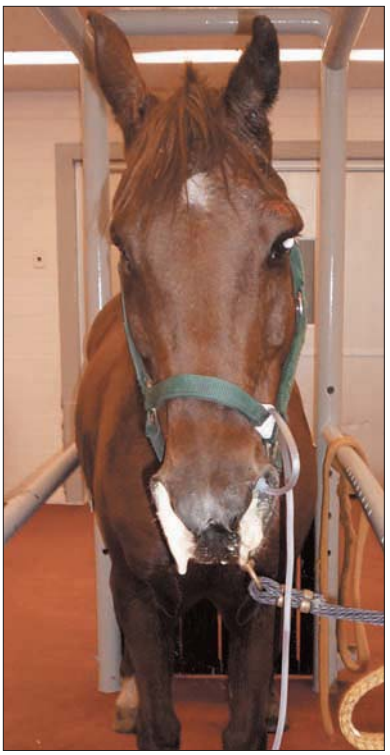
Nasal discharge can originate from diseases involving any part of the respiratory tract, from the nasal passages to the alveoli, as well as diseases involving structures adjacent to or communicating with the respiratory tract. Inflammation of the nasal passages results in increased glandular secretions. Secretions are initially serous, then mucoid (**51**) and they may become purulent (**52**) as a result of neutrophilic accumulation. It is not possible to predict the cause of a purulent discharge based solely on its appearance. Thick, white- to-yellow purulent discharges may be associated with infectious (e.g. strangles) and non-infectious diseases (e.g. heaves). Some discharges have a yellow–green color caused by enzymes released during neutrophil breakdown. Various ocular conditions can result in increased lacrimal secretions and nasal discharge on the affected side, due to drainage from the nasolacrimal duct. Secretions from the tracheobronchial tree are usually swallowed when they reach the pharynx without draining into the nose. However, nasal discharge may



▲ **51** **Bilateral seromucoid nasal discharge.**

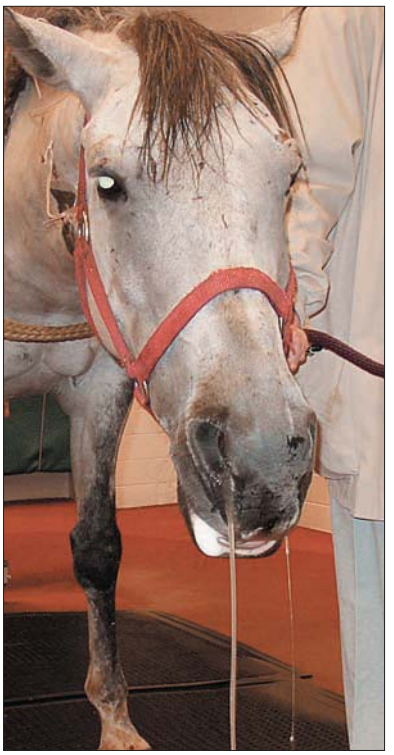


▲ **52** **Mucopurulent nasal discharge.**



▲ **53** **Bilateral foamy nasal discharge in a horse with acute pulmonary edema secondary to smoke inhalation during a barn fire. Note the clear tubing in the left nostril, used to deliver supplemental oxygen.**

▲ **54** **Epistaxis in a horse with severe thrombocytopenia.**



▲ **55** **Nasal discharge composed of clear saliva in a horse with esophageal obstruction. Foamy saliva is also visible on the commissure of the lips.**

develop in cases of increased volume of secretions, coughing, exercise, or maintaining the head and neck in a low position (e.g. grazing, sedation). Pulmonary edema may lead to a clear serous nasal discharge and in some severe cases to bilateral foamy exudate (**53**).

Bleeding from the nose (epistaxis; **54**) may range from a few drops of blood-tinged secretions to profuse, fatal hemorrhage. Depending on the cause of the bleeding, blood may be mixed with serous, mucoid, or purulent discharge. Nasal discharge containing a mixture of blood and purulent secretions is usually seen with infections, neoplasia, necrotic lesions, inhaled foreign bodies, and bronchoesophageal fistulas.

Malodorous or fetid nasal discharge usually indicates anaerobic infection and is often associated with tooth root abscess, tissue necrosis, or trauma. Presence of ingesta in the nasal discharge points to impaired deglutition and may be secondary to a variety of physical or functional abnormalities such as esophageal obstruction (**55**), neuromuscular disease involving the pharyngeal region (e.g. botulism, GP mycosis), or pharyngeal masses impairing deglutition (e.g. retropharyngeal abscess secondary to strangles). The discharge is usually mixed with saliva.

PHYSICAL EXAMINATION

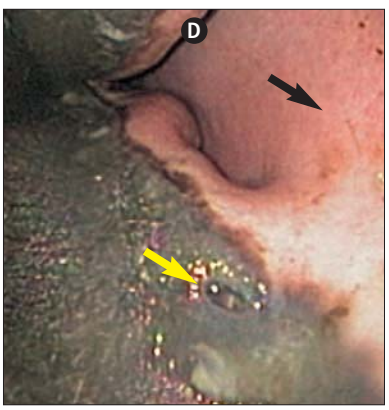
Extrathoracic airways: nose to extrathoracic trachea

Palpation

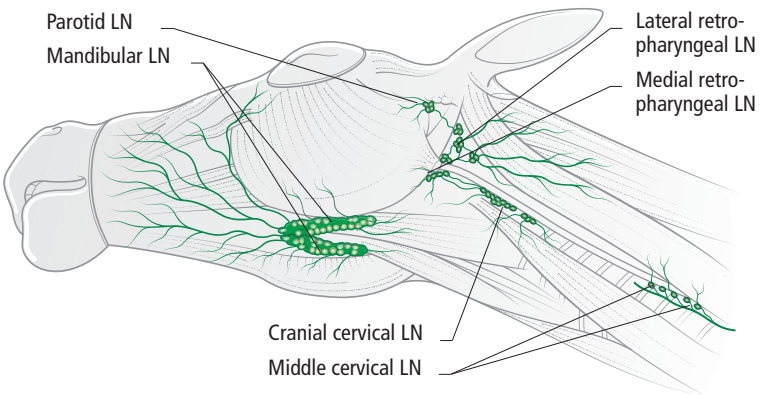
The nostrils in the horse are large and include a nasal diverticulum (false nostril) and alar fold dorsally and medial and lateral wings of the nostrils or alae nasi (see **3**, p. 10). Contraction of alae nasi muscles during inspiration results in closure of the diverticulum and nostril flaring. At rest, healthy horses do not flare their nostrils unless excited. Nostril flaring during quiet breathing at rest indicates serious respiratory disease. During exercise, nostril flaring increases as exercise becomes more intense, the nostrils becoming permanently flared during both inspiration and expiration as horses exercise strenuously.

Examination of the nose should start by placing both hands in front of the nostrils and feeling the warm airflow during expiration (see **2**). Appreciating symmetrical airflow out of both nostrils is subjective and may be difficult to gauge. Alternatively, one nostril may be occluded completely with the hand and respiratory effort observed in the following couple of minutes. A horse with normal nasal passages should breathe with no increased effort from one nostril. Horses with unilateral nasal obstruction will have increased respiratory effort and may exhibit a stridor when the opposite nostril is occluded. The nostril wings should be thin and pliable. The rostral part of the alar fold is supported by the dorsal portion of the alar cartilage and therefore feels more rigid to the touch. A palpable increased thickness and pitting of the nostrils as well as the nasal septum indicates edema. Congestion or thickening of the nasal septum may be evaluated by pinching the rostral nasal septum between the thumb and index finger (see **4**). The nasolacrimal duct opens via an orifice on the ventral aspect of the nasal vestibule, approximately 5 cm from the nostril opening (**56**). Manual dilation of the nostril and good lighting are often required to visualize the orifice. The rest of the nasal passage is not accessible to direct palpation. Thickened alar folds may cause nasal obstruction, abnormal respiratory noise, and exercise intolerance.

Palpation of the locoregional lymph nodes draining the upper respiratory tract (URT) (mandibular and retropharyngeal) allows evaluation of their size, texture, and potential painful response to touch. Also, knowing which anatomic structures are drained by the respective lymph nodes helps determine the location and cause of the disease (**57**). The mandibular



▲ **56** **Nasolacrimal duct orifice on the ventral aspect of the nasal vestibule (yellow arrow) in the right nasal passage. The nasal septum is covered by pink mucosa (black arrow). (D) dorsal.**

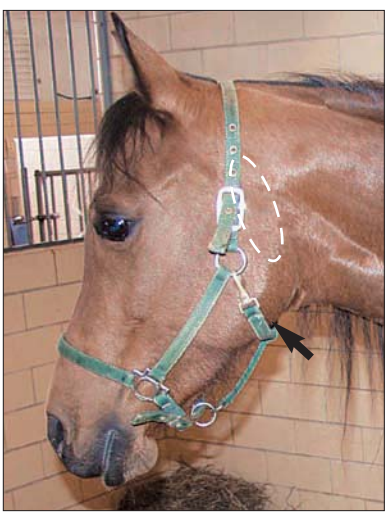


▲ **57** **Lymph nodes (LN) of the head and proximal cervical region. (Adapted from Barone R [1996] *Anatomie comparée des mammifères domestiques: Tome 5, Angiologie.* Vigot, Paris.)**

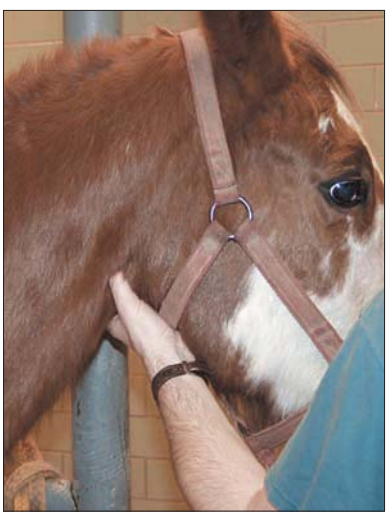
lymph nodes are palpated ventrally between the rami of the mandible (**58**). They collect lymphatic vessels originating from the face, oral and nasal mucosa and bones, teeth, tongue, masseter muscles, and salivary glands except the parotid gland. Retropharyngeal lymph nodes are located near the wing of the atlas (lateral group; **58**) and on the dorsolateral surface of the pharynx (medial group), ventral to the medial compartment of the GP. Retropharyngeal lymph nodes are normally not palpable unless they are enlarged, because they are covered for the most part by the parotid gland. They collect lymphatic vessels originating from the pharynx, the base of the tongue, the GPs, and the caudal aspect of the nasal passages. Lymph nodes may be slightly enlarged secondary to inflammatory or viral diseases, but bacterial infections, in particular *Streptococcus* spp., often result in marked enlargement and possibly abscess formation. In rare cases, lymph node enlargement is secondary to neoplastic infiltration (e.g. lymphoma).

Palpation of the larynx is conducted to assess symmetry of the cricoarytenoideus dorsalis muscles and conformation of laryngeal cartilages. Compression of the trachea should not elicit coughing in a normal horse. One way of attempting to trigger coughing is by squeezing the dorsal aspect of the tracheal rings just behind the larynx between the thumb and index finger (**59**). A normal horse will cough once at most. In horses with upper airway inflammation, coughing will be triggered easily and result in multiple coughs. Dry, short, loud, and harsh coughs with frequent bouts of coughing are common with URT inflammation, particularly inflammation secondary to viral infections. Deep, soft coughs usually result from lower respiratory tract (LRT) diseases such as pneumonia or heaves. Painful conditions involving the URT (e.g. retropharyngeal abscess) or LRT (e.g. pleuropneumonia) can decrease both the intensity of respiratory effort and the sounds produced during coughing.

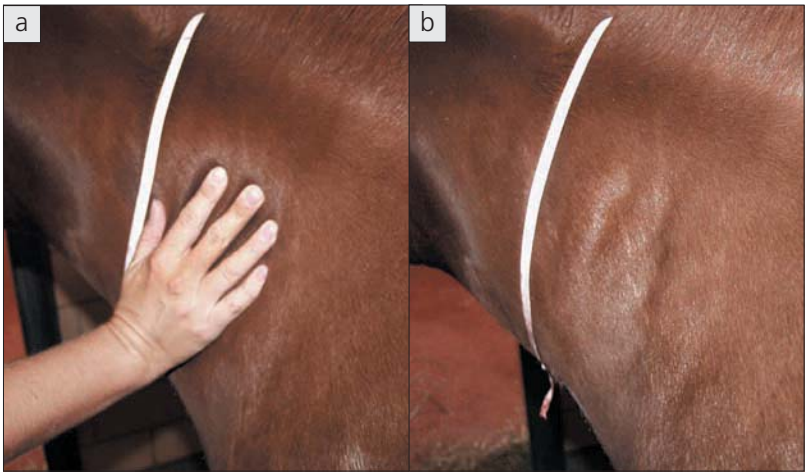
The skin over the cervical region is carefully palpated for pain or abnormal findings (e.g. subcutaneous emphysema, edema, or mass; **60**). Tracheal rings are easily palpated on the ventral aspect of the neck. Cartilage abnormalities or defects from previous tracheostomy, trauma, or tracheal collapse may be detected. The latter is often secondary to a congenital abnormality more commonly observed in ponies and miniature horses. In these cases, cartilage rings flattened dorsoventrally may be palpated when the cervical trachea is affected. Unfortunately, collapse occurs more frequently in the distal trachea, which cannot be palpated.



◀ **58 Location of the lymph nodes of the head. The retropharyngeal lymph nodes are located medially to the parotid gland (dotted line) and the mandibular lymph nodes are ventrally between the rami of the mandible (arrow).**



▶ **59 Coughing characteristics may be assessed by squeezing the dorsal aspect of the tracheal rings just behind the larynx between the thumb and index finger.**



◀ **60 Foal with subcutaneous emphysema secondary to tracheal laceration extending over the neck. The hand is pushed against the side of the neck and crackles can be felt as air is pushed away (a). Note how the hand leaves an impression mark on the skin (b).**

Auscultation

At rest, normal respiratory sounds generated by the upper airways are predominant during tracheal auscultation but are difficult to hear at the level of the nostril opening. Because there is a close relationship between airflow and sound intensity, increased ventilation (e.g. during exercise) results in more audible upper airway sounds. Studies in exercising horses demonstrated that the spectrum of respiratory sounds recorded over the trachea or in front of the nose is in the audible range, with most of the sound energy contained in frequencies up to 800 Hz. Expiratory sounds predominate in horses exercising strenuously. The nomenclature of upper airway sounds is variable. Stridor refers to high intensity and frequency sounds in general and stertor describes high intensity sounds occurring in coma or deep sleep. Descriptions of abnormal sounds occurring during exercise use colorful terms such as roaring, rattling, or gurgling.

During inspiration, subatmospheric pressures in the airway lumen tend to collapse extrathoracic airways not supported by rigid structures (e.g. nares, pharynx, larynx). Collapse is prevented by dynamic contraction of the upper airway muscles resulting in stabilization of these structures. The role of these muscles is particularly important during exercise because pressure changes across airway walls increase dramatically as ventilation increases. As soon as a horse starts exercising, muscle contraction dilates the nostrils, tenses the soft palate and pharyngeal walls, and abducts the arytenoid cartilages in order to increase airway diameter and accommodate for the increase in ventilation. Extension of the horse's head and neck also stretches the trachea, which becomes less compliant and less susceptible to dynamic collapse.

Perturbation of airflow may be secondary to fixed or variable obstructions. Abnormal respiratory sounds have different characteristics depending on the site of obstruction (i.e. extrathoracic or intrathoracic) and on the type of obstruction (i.e. fixed or variable). Fixed obstructions (e.g. deviated nasal septum) result in a similar degree of airflow obstruction during inhalation and exhalation. Variable obstructions (e.g. laryngeal hemiplegia, DDSP) generate different degrees of airway obstruction based on flow rate, site of obstruction, and phase of the respiratory cycle. In exercising horses with laryngeal hemiplegia, while the expiratory sounds are normal, the inspiratory sounds are markedly different ('roaring' or 'whistling') and are associated with a significant reduction in inspiratory flows. These sounds appear to have a unique frequency spectrum that may prove useful for diagnosing laryngeal

hemiplegia in the field using a portable microphone. Upper airway obstruction may occur during expiration, as seen in cases of DDSP in exercising horses. In these cases, abnormal respiratory sounds develop throughout exhalation and are often described as rattling or gurgling noises. It is noteworthy that some horses may experience upper airway obstruction without generating abnormal respiratory sounds. Therefore, upper airway obstruction is not necessarily accompanied by abnormal respiratory sounds. Alternatively, loud respiratory sounds are not necessarily associated with airway obstruction. For example, young horses (1–2 years old) in the early stages of training often produce loud, rattling expiratory sounds that appear to originate from nostril fluttering. These abnormal sounds eventually disappear as the horse matures.

Differentiating between expiratory and inspiratory noise may be difficult in the field when the horse is running at a distance from the observer. During cold weather, exhaled air is readily visible as condensed water vapor in front of the nose. Otherwise, the clinician has to pay close attention to the horse's breathing in relation to the gait because there is coupling between stride and breathing at many different gaits. Usually, horses breathe one breath per stride during galloping, but coupling may be less consistent at slower speeds. During galloping, inspiration begins with the suspension phase and expiration is initiated as the forelimbs contact the ground.

Percussion

In order to amplify sound intensity, percussion of the frontal and maxillary sinuses is best conducted while the mouth is kept slightly open. The boundaries of the sinuses are shown in **7** and **8** (p. 12). Affected sinuses are often painful and horses will resent percussion. Dullness over one side indicates space-occupying fluid or tissue. However, the technique is not sensitive and normal percussion does not rule out sinus disease.

Intrathoracic airways and chest wall: intrathoracic trachea to alveoli

Palpation

The skin over the thorax is carefully palpated for pain or abnormal findings (e.g. subcutaneous emphysema, edema, mass; **61**). Horses with pleuritis will often display a painful response (pleurodynia), such as grunting, guarding of the chest, or moving away from the pressure, when pressure is applied to the ribs. Some normal but sensitive horses may exhibit the same type of response. The rib cage of neonatal foals should be palpated carefully, as they commonly exhibit rib fracture as a complication of parturition. Most fractures are located at or within 3 cm of the costochondral junction and more often on the left side. Usually, the rib segment ventral to the fracture will be displaced outwards and the clinician will feel a 'bump' while palpating the rib from dorsal to ventral. Thoracic radiographs and ultrasonography are more sensitive than palpation for detecting rib fractures. Most rib fractures of neonates heal without adverse consequences.

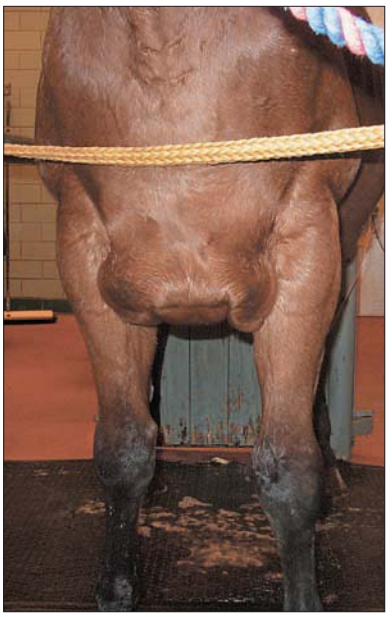
Auscultation

Lung sounds are divided into breath sounds (normal) and adventitious (abnormal) sounds. Breath sounds are produced by turbulent flow associated with air movement through the large airways (diameter >1–2 mm). Originally, Laënnec introduced the term 'vesicular' sounds to refer to sounds heard over the chest. This terminology assumed that breath sounds originated from the alveoli, but since air flow through the bronchioles and alveoli is silent to the human ear, this term should not be used.

The terminology for adventitious (abnormal) sounds has been described by the International Lung Sounds Association and is broadly accepted in veterinary medicine. Adventitious sounds should be classified as wheezes or crackles. Wheezes are continuous, musical sounds that are generated principally by the flutter of airway walls and potentially by movement of

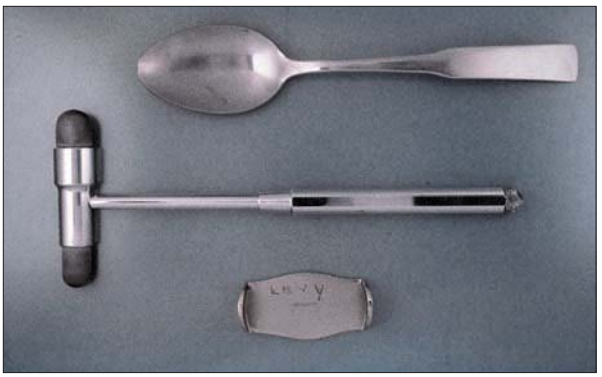
airway secretions. Experiments conducted *in vitro* and *in vivo* showed that flow limitation is necessary for wheezing to occur and that sufficient transpulmonary pressures were required. Factors influencing the sound spectrum of wheezing are airway wall thickness, bending stiffness (compliance), and longitudinal tension. Crackles are short, non-musical sounds similar to the noise made by paper being crumpled between someone's hands. Crackles appear to be produced by the explosive opening of small airways previously kept closed by surface forces or by air moving through secretions.

Chest auscultation during rebreathing maneuver will make breath sounds more audible and increase the likelihood of detecting abnormal sounds (i.e. wheezes, crackles, friction rubs) (**62**). This technique is contraindicated in horses that are already breathing with increased respiratory effort. Decreased intensity of breath sounds is associated with a thick chest wall, pleural effusion, pulmonary consolidation, atelectasis, decreased ventilation, emphysema, diaphragmatic hernia, and pneumothorax. Sound transmission is more effective through consolidated or atelectatic lung than through aerated lung. As a result, breath sounds over a consolidated or atelectatic lung region may be increased or decreased depending on the intensity of sound and degree of attenuation. Increased intensity of breath sounds may be due to a thin chest wall (e.g. foal), increased ventilation, a lung mass, or pulmonary consolidation. Normally, breath sounds are fairly evenly audible over areas of auscultation. Lung sounds that are grossly different when comparing the left with the right side of the thorax or various areas on the same hemithorax suggest pulmonary, pleural, or chest wall abnormalities. Symmetrical enlargement of the area of pulmonary auscultation caudally indicates hyperinflation of the lungs and when it is accompanied by abnormal breath sounds widespread over the chest it is strongly suggestive of diffuse peripheral airway obstruction (e.g. RAO).



▲ **61 Ventral edema in a mare with pleuropneumonia.**

▲ **62 Plastic bag placed over a horse's muzzle for rebreathing maneuver.**



◀ 63 **Tools that may be used for chest percussion. From top to bottom: a spoon, a plexor, and a pleximeter. (Photo courtesy Dr. Michel Levy)**

▲ 64 **Outline of the area of thoracic percussion.**

Percussion

Percussion can be performed using fingers or a plexor and pleximeter (or spoon) along the intercostal spaces (**63**). The boundaries of the pleural space are (**64**):

- Dorsally: vertebral transverse processes (tuber coxae level).
- Cranially: forelimb musculature.
- Caudally: 6th rib at the level of the elbow, 11th rib at the level of the shoulder, and 16th rib at the level of the tuber coxae.

Percussion of the chest in adult horses should be interpreted with caution because thickness of the chest wall has a major influence on the findings. Pain elicited on percussion is often associated with pleuritis and ventral dullness suggests pleural effusion. Increased resonance on percussion of the chest accompanied with decreased breath sounds is consistent with pneumothorax.

Percussion of the chest is of low sensitivity. Ultrasonography is the most sensitive technique, allowing detection of small amounts of pleural fluid and abnormalities of the chest wall or lung surface.

CHAPTER 4

DIAGNOSTIC TESTS AND THERAPEUTIC PROCEDURES

ENDOSCOPY

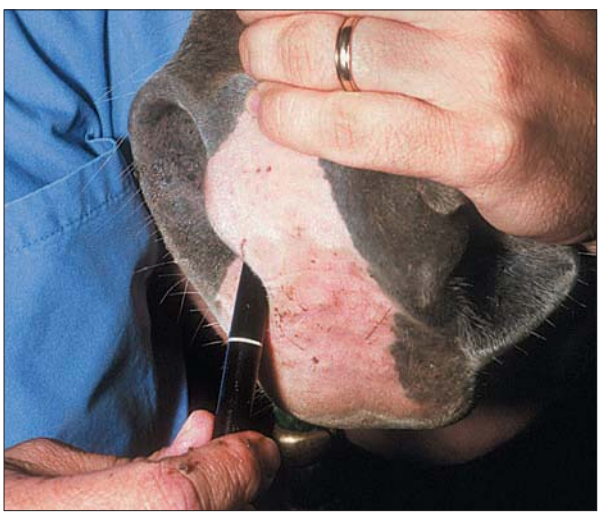
Endoscopy is the most important diagnostic tool for the evaluation of disorders involving the extrathoracic airways. Endoscopic examination starts with a good quality endoscope. Currently, two types of endoscope are available: video and fiberoptic.

Video endoscopes allow the image to be projected on a monitor for clinician and client viewing. The downside is cost, as video endoscopes are expensive. Video endoscopy is preferred because it allows the examiner to discuss the endoscopic findings with others and allows for a magnified and high-quality image of the URT. Video endoscopy has the advantage of being able to record images onto either video or digital storage. Images can be replayed in real time or in slow motion. The ability to review a video in slow motion is very helpful, especially when reviewing high-speed treadmill examinations.

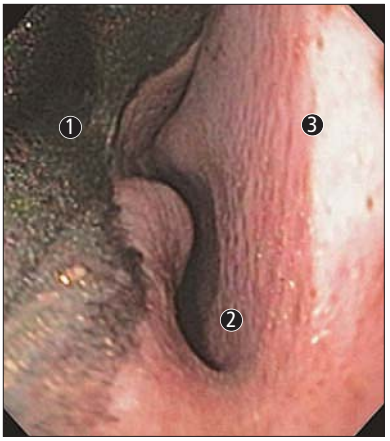
Fiberoptic endoscopes are less expensive and are readily available for purchase. However, only the examiner can view the image unless the endoscope is connected to an external video camera. When evaluating the URT the authors recommend performing the examination without sedation because sedation alters the function of the soft palate and larynx by decreasing muscle tone. Most horses can be examined with the aid of a nose twitch and without sedation. However, if the horse is intractable, sedation should be administered. Stocks are useful because they decrease the movement of the horse from front to back and side to side.

Endoscopic examination begins with insertion of the endoscope into the ventral nasal meatus (**65**). This is accomplished by lifting the alar fold with a thumb and directing the endoscope beneath the thumb. The examination should be performed in a systematic fashion. The following anatomic structures should be identified and examined: nasal septum; ventral, middle and dorsal nasal meati; nasomaxillary opening; ethmoid turbinates; dorsal pharyngeal recess; pharyngeal lymphoid tissue; pharyngeal openings of the GPs; soft palate; epiglottis; aryepiglottic fold; arytenoid cartilages; rima glottis; and trachea. The normal anatomy seen on endoscopy of the extrathoracic airways is shown in the endoscopic photographs (**66**–**79**) on the following two pages.

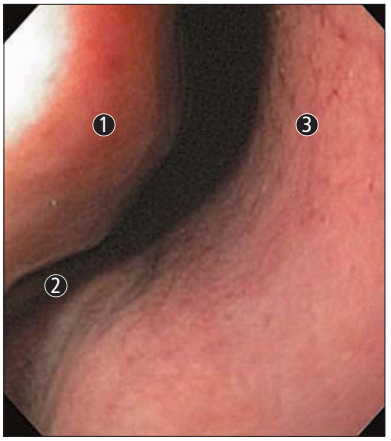
Resting endoscopy is sufficient to diagnose many abnormal conditions involving the URT. Examples include epiglottic entrapment (EE), left laryngeal hemiplegia, and some cases of DDSP.



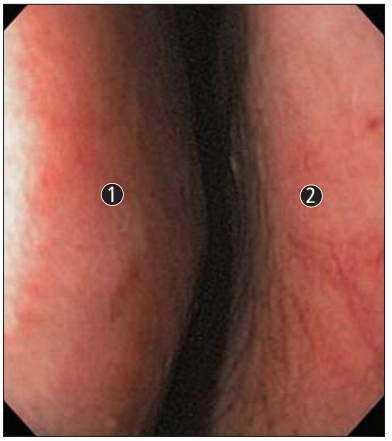
▶ **65 Proper positioning of the endoscope in the right ventral nasal meatus for endoscopic examination.**



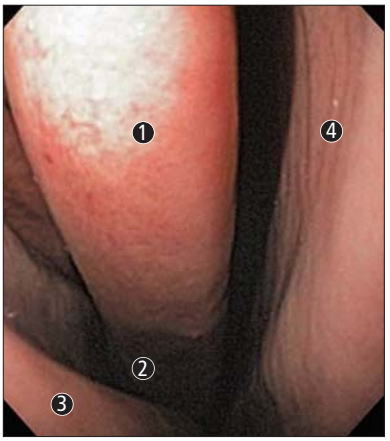
▲ 66 Endoscopic photograph 5 cm from the nares (1). (2) ventral nasal meatus; (3) rostral nasal septum.



▲ 67 Endoscopic photograph 10 cm from the nares. (1) ventral nasal conchae; (2), ventral nasal meatus; (3) nasal septum.

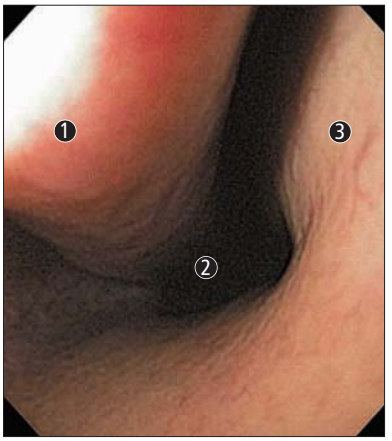


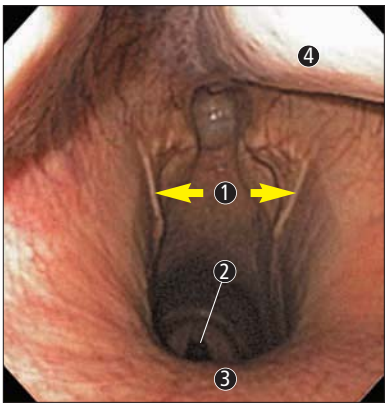
▲ 68 Endoscopic photograph 15 cm from the nares. (1) ventral nasal conchae; (2) nasal septum.



◀ 69 Endoscopic photograph 20 cm from the nares. (1) middle nasal conchae; (2) middle nasal meatus; (3) ventral nasal conchae; (4) nasal septum.

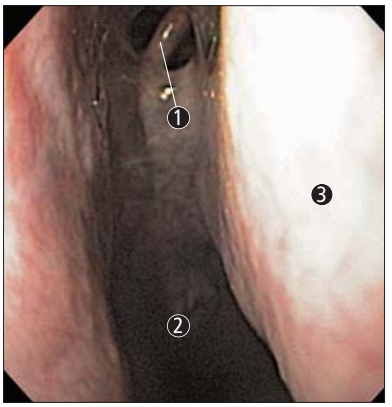
▶ 70 Endoscopic photograph 25 cm from the nares. The endoscope tip has been tilted ventrally. (1) ventral nasal conchae; (2) ventral nasal meatus; (3) nasal septum.

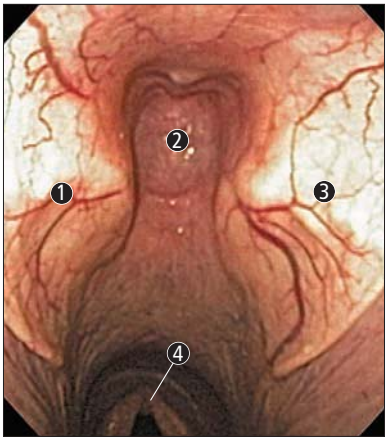




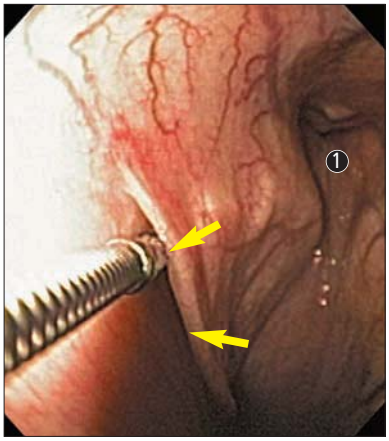
◀ 71 Endoscopic photograph 30 cm from the nares. (1) roof of nasopharynx; arrows, guttural pouch openings; (2) larynx; (3) soft palate; (4) nasal septum.

▶ 72 Endoscopic photograph 30 cm from the nares. The endoscope tip has been tilted dorsally. (1) ethmoid turbinates; (2) nasopharynx; (3) nasal septum.

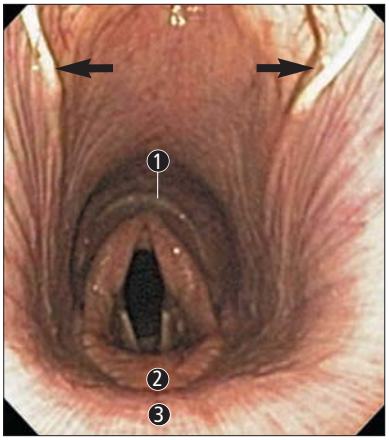




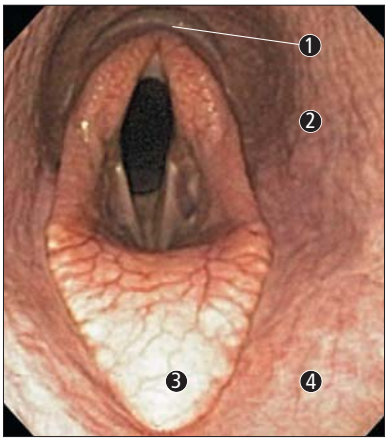
▲ **73** Endoscopic photograph 35 cm from the nares. (1) right guttural pouch; (2) dorsal pharyngeal recess; (3) left guttural pouch; (4) larynx.



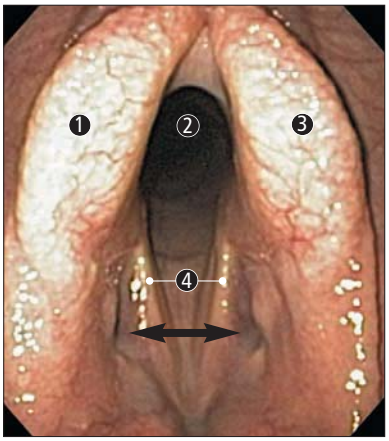
▲ **74** Endoscopic photograph detailing the use of an endoscopic biopsy forceps to aid entry into the right guttural pouch (arrows). (1) dorsal pharyngeal recess.



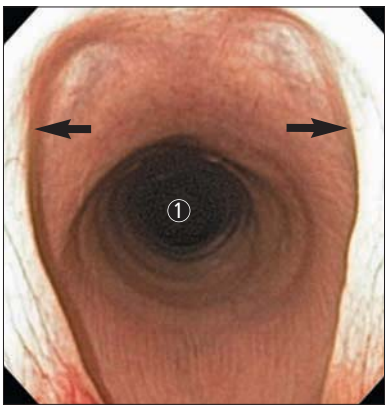
▲ **75** Endoscopic photograph 35 cm from the nares. Arrows, guttural pouch openings; (1) esophageal opening (hidden by palatopharyngeal arch); (2) epiglottis; (3) soft palate.



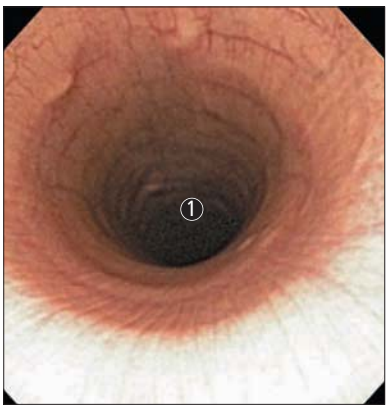
◀ **76** Endoscopic photograph 40 cm from the nares. (1) esophageal opening; (2) wall of the nasopharynx; (3) epiglottis; (4) soft palate.



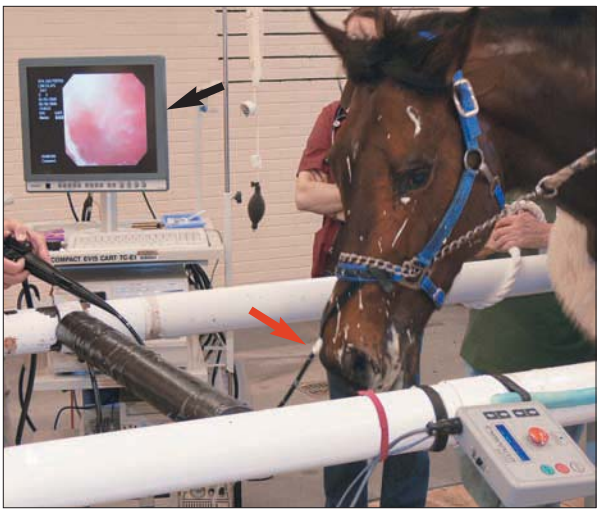
▶ **77** Endoscopic photograph 45 cm from the nares. (1) right arytenoid; (2) rima glottis; (3) left arytenoid; (4) vocal folds (cords); arrows, laryngeal ventricles.



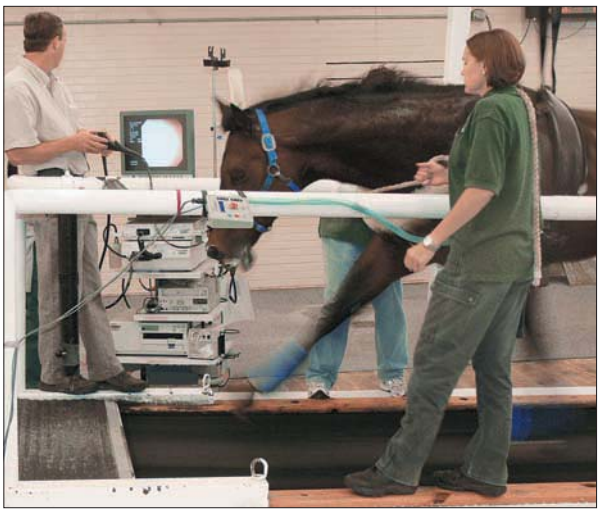
◀ **78** Endoscopic photograph 50 cm from the nares. Arrows, axial aspect of the arytenoid cartilages; (1) tracheal lumen.



▶ **79** Endoscopic photograph 55 cm from the nares. The scope is within the proximal trachea. (1) tracheal lumen.



▲ **80** **Video-endoscopy (black arrow pointing at the monitor) during high-speed treadmill evaluation. Note the positioning of the endoscope (red arrow) and its attachment to the halter.**



▲ **81** **A Thoroughbred racehorse being examined for dorsal displacement of the soft palate on a high-speed treadmill.**

Dynamic endoscopy

When standing endoscopic examination is non-diagnostic, high-speed treadmill examination provides the best means of making a diagnosis during exercise. Horses are conditioned to the treadmill prior to endoscopic examination at speed. Following adaptation to the treadmill, the endoscope is positioned in the ventral nasal meatus with its tip approximately 40 cm from the nares and secured to the nose band of the halter with Velcro® (**80**).

A standardized exercise protocol is used to evaluate the horse. Horses must be exercised maximally for the test to be of the most use to the examiner. Conditions that can only be diagnosed with high-speed treadmill examination include epiglottic retroversion, dynamic collapse of the aryepiglottic folds, and cases of intermittent EE. Horses with grade 3 left laryngeal hemiplegia are best examined on the treadmill because of the variable amount of arytenoid abduction present in these horses at rest (see Chapter 8, Abnormal respiratory sounds: Laryngeal hemiplegia). Horses with a history of DDSP can also be confirmed during treadmill exercise (**81**).

Recently, various types of 'over-ground' telemetric endoscopy systems have become commercially available. These systems are composed of a semi-rigid, flexible endoscopy piece that is inserted in the nasal passages, fixed to the horse's bridle, and connected to a recording device worn by the horse or carried by the rider. Over-ground endoscopy systems have the advantage of allowing dynamic endoscopy in the field. However, it is difficult to standardize exercise tests in field situations and reproduce the presenting complaint (e.g. abnormal respiratory noise or poor performance).

DIAGNOSTIC IMAGING

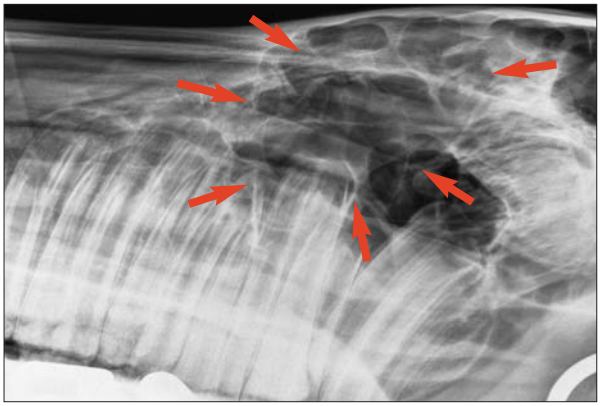
Radiography

Radiography is an excellent tool to use in conjunction with endoscopy when evaluating diseases of the respiratory tract. Skull radiographs can be readily obtained with portable equipment. The authors currently favor the use of digital radiography because of the improved image quality and the ability to adjust contrast and brightness. The other advantage of digital radiography is the ability to send data to owners and veterinarians digitally.

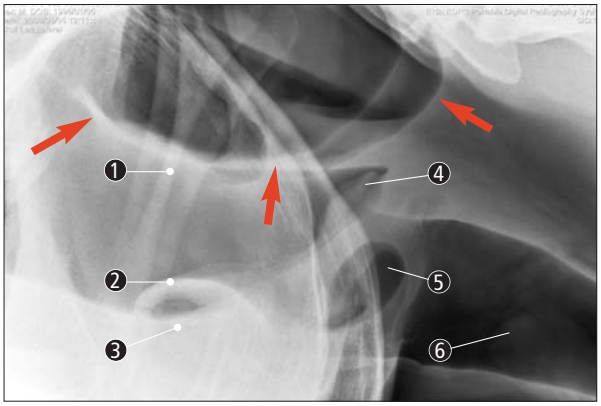
Except in extreme circumstances, radiographs can be obtained standing with intravenous (IV) sedation. The GPs, larynx, epiglottis, soft palate, hyoid apparatus, paranasal sinuses, and nasal passages can all be identified radiographically. Air contrast allows for visualization of many soft tissue structures even with portable radiographic equipment (**82, 83**).

The type and number of radiographic views will vary, depending on the region of interest, but should be at least two: dorsoventral and lateral–medial. For evaluation of the nasal passages and paranasal sinuses the minimum number of views is four: dorsoventral, lateral–medial, and right and left obliques.

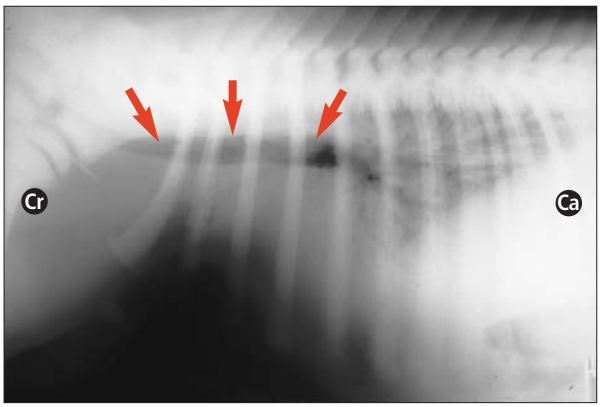
Lateral thoracic radiography in neonatal foals is readily performed with portable units, and the entire lung field can be seen on a single view using a large cassette (e.g. 30 × 40 cm or 36 × 43 cm; **84**). In older foals, two views with large cassettes are usually sufficient to cover the caudodorsal and cranioventral lung fields. In adults, four views are needed to image the entire lung field (caudodorsal, craniodorsal, caudoventral, and cranioventral). Only the caudodorsal lung area of adult horses can be imaged with a portable radiographic machine. The cranioventral lung area is often difficult to image because of overlapping shoulder musculature, even with high-output machines.



▲ **82** **Digital lateral radiograph of the paranasal sinuses in a horse with an abnormal radiopacity originating in the frontal sinus (arrows).**



▲ **83** **Digital lateral radiograph of the throatlatch region. Arrows, guttural pouch; (1) stylohyoid bone; (2) epiglottis; (3) soft palate; (4) arytenoid cartilages; (5) laryngeal ventricle; (6) trachea.**



▶ **84** **Lateral thoracic radiograph of a neonatal foal with acute respiratory distress syndrome. Most of the lung field shows diffuse opacity, with air bronchograms consistent with an alveolar pattern. The cardiac silhouette is not visible because of lung opacity. The trachea may be seen as a lucent tubular structure across the dorsal lung field (arrows). (Cr) cranial; (Ca) caudal.**

Computed tomography

Computed tomography (CT) is no longer limited to veterinary schools. CT scanners are readily available in many veterinary schools and private referral practices. Specially made tables are used to position the horse (**85**) and the table is attached to the power tray connected to the CT unit.

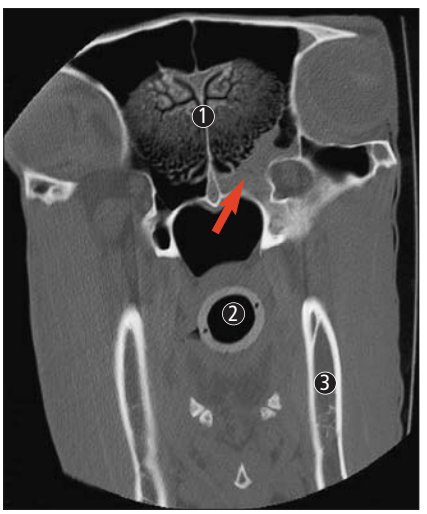
For CT scans of the skull, horses are positioned in either lateral or dorsal recumbency. The entire skull and the proximal cervical spine can be imaged in most horses. The authors recommend a minimum of 100, 5–10 mm slices to image a standard skull. CT can be performed with or without IV contrast. IV contrast is used to highlight enhanced vascularity to abnormal growths and/or chronic infection. Its primary usefulness is in evaluation of masses involving the paranasal sinuses (**86**).

Nuclear imaging

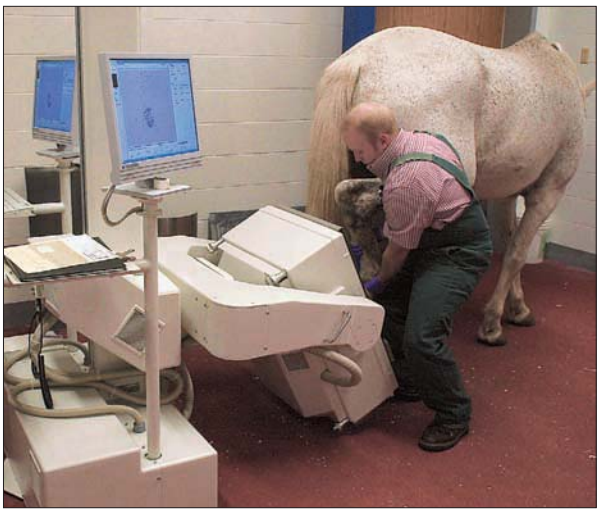
Nuclear scintigraphy uses a radioisotope that can be labeled for bone and white blood cells (WBCs) (**87**). Technetium-99m is the most commonly used radioisotope label. A technetium-99m–phosphorus label is used for both soft tissue and bone phase imaging of the musculoskeletal system. The bone phase is most useful for disorders involving the skull (**88**). A labeled WBC scan is useful for identification of sites of microbial infection (e.g. periapical tooth root abscesses).

Pulmonary scintigraphy allows assessment of regional lung ventilation and perfusion and $\dot{V}/\dot{Q}$ matching. Scintigraphic imaging of ventilation is usually done with technetium-99m bound to diethylene triamine pentaacetic acid (DTPA). The label is administered to the horse by nebulization and lateral images are obtained with the gamma camera positioned against the chest. Imaging of perfusion is conducted following IV administration of technetium-99m-labeled albumin aggregates in the jugular vein. Digital images of ventilation and perfusion can then be combined to create a computerized image of the regional $\dot{V}/\dot{Q}$ ratio. In healthy horses, the $\dot{V}/\dot{Q}$ ratio is close to 1 throughout most of the lung field (**89**). Regional abnormalities in the ratio may be detected in cases such as chronic EIPH where proliferation of bronchial circulation in the caudodorsal lung results in a deficit in pulmonary perfusion and a high $\dot{V}/\dot{Q}$ ratio (**90**).

▲ 85 CT setup at Purdue University. The horse is being positioned on a custom-designed table (1) which is set onto the dedicated CT table (2). The horse's head will be positioned on the headboard (3) prior to entering the CT unit (4).

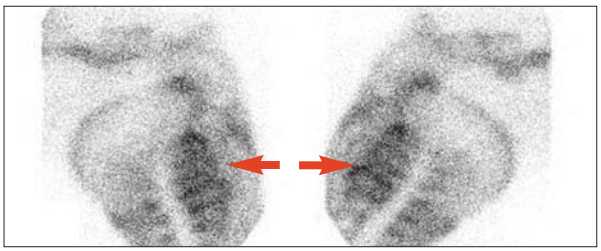


▲ 86 CT image of a horse with blood (arrow) on the floor of the frontal sinus. This horse was later diagnosed with a hemangiosarcoma. (1) ethmoid turbinates; (2) endotracheal tube; (3) ramus of the mandible. (Case material courtesy Dr. Stephen B Adams)

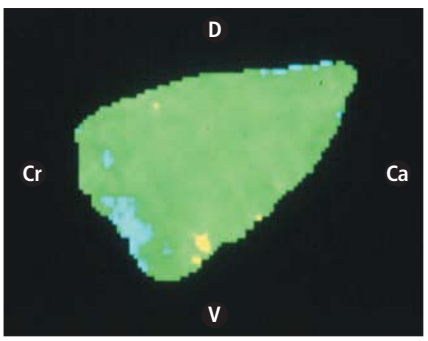


◀ **87** Gamma camera used for nuclear imaging of a horse's hindfoot.

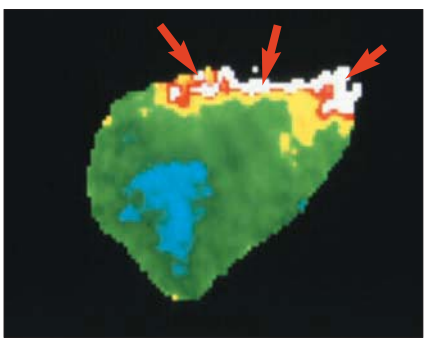
▼ **88** Right and left lateral bone phase scintigraphic views of the skull. Arrows represent the reserve crown of the maxillary cheek teeth. (Photo courtesy Dr. Michael Ross)



▶ **89** Ventilation to perfusion ratio in a healthy horse (lateral view of the left lung). The green color represents areas where $\dot{V}/\dot{Q} = 1$. (Ca) caudal; (Cr) cranial; (D) dorsal; (V) ventral. (Photo courtesy Dr. Michael O'Callaghan)



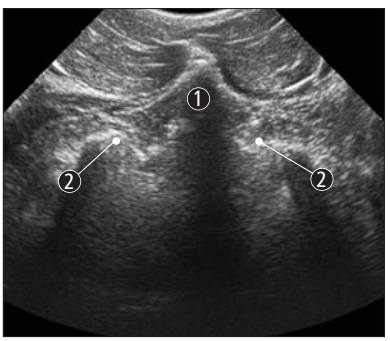
▶ **90** Abnormal ventilation to perfusion scan in a horse with chronic EIPH (lateral view of the left lung). Note the yellow to white colors in the caudodorsal lung region indicating high ventilation to perfusion ratios (arrows). Yellow, $\dot{V}/\dot{Q} = 1.5–2$; red, $\dot{V}/\dot{Q} = 2–4$; white, $\dot{V}/\dot{Q} > 4$. (Photo courtesy Dr. Michael O'Callaghan)



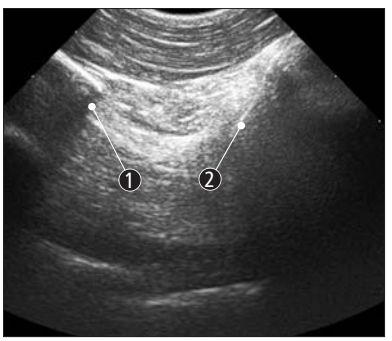
Ultrasonography

Ultrasonography has been described for evaluation of laryngeal function and inflammation involving the arytenoid cartilages and perilaryngeal tissues. Four acoustic windows have been developed to allow for examination of the larynx and perilaryngeal tissues (**91–94**). Ultrasonography has also been used to evaluate horses with arytenoid chondritis and laryngeal hemiplegia. Abnormal findings associated with arytenoid chondritis include thickening of the arytenoid cartilages, arytenoid abscessation, mineralization, and perilaryngeal inflammation.

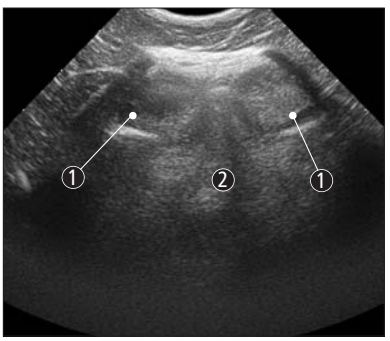
Ultrasonography is also the diagnostic tool of choice for evaluation of the equine thorax, particularly the chest wall and pleural space. This technique is more sensitive than radiography for detection of pleural effusion, peripheral lung disease, and diaphragmatic hernia. The major drawback is that ultrasonographic sound waves do not travel through air, therefore aerated lung and all structures underneath cannot be imaged. The hair coat should be clipped if it is thick, otherwise it should be cleaned of debris and wetted down profusely with water or alcohol. Application of gel onto the skin improves image quality. The ultrasound probe is held against the skin starting dorsally in the 17th intercostal space and sliding slowly ventrally while keeping the probe beam perpendicular to the skin surface. The normal appearance of the lung surface is a white hyperechoic line corresponding to reverberation of the beam at the pleural surface (**95**). Pleural effusion may be detected as hypoechoic fluid between the chest wall and the lung. Atelectasis and lung consolidation over the lung periphery are seen as hypoechoic areas next to hyperechoic aerated lung. 'Comet-tail' artifacts are sometimes visualized as hyperechoic, narrow-based reverberation fanning from the lung–chest wall interface (**95**). These artifacts are more easily seen when ultrasound probes with divergent beams are used (sector, convex, and annular-array). The phenomenon is believed to result from back-and-forth echoing of the ultrasound beams in areas where there is a marked difference in acoustic impedance. For example, a small amount of fluid accumulation (below resolution of the ultrasound beam, i.e. <1 mm) in a peripheral lung area and surrounded by



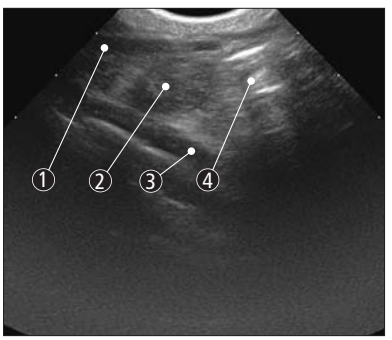
◀ 91 Ultrasonogram of the larynx via the rostroventral window. (1) basihyoid bone; (2) ceratohyoid bones.



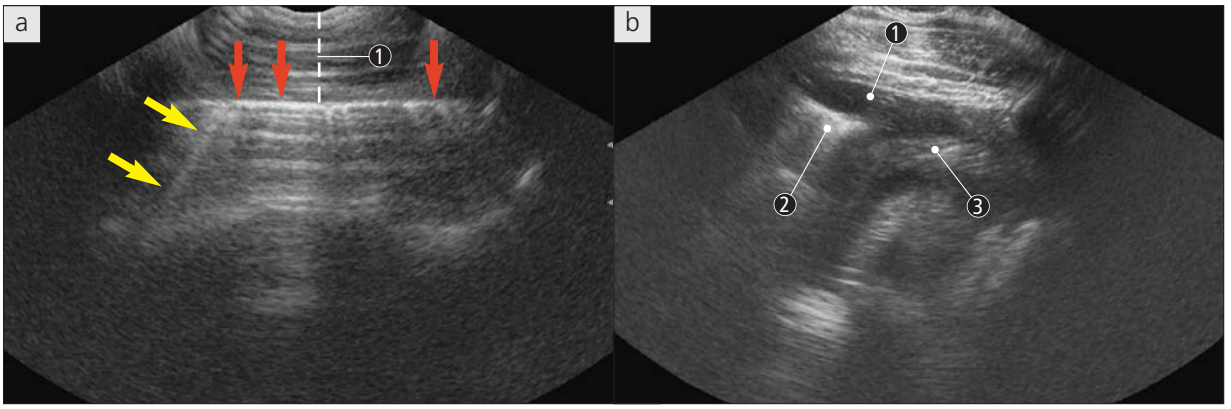
▶92 Ultrasonogram of the larynx via the midventral window. (1) basihyoid bone; (2) thyroid cartilage.



◀ 93 Ultrasonogram of the larynx via the caudoventral window. (1) vocal folds; (2) lumen.



▶ 94 Ultrasonogram of the larynx via the caudolateral window. (1) thyroid cartilage; (2) cricoarytenoideus lateralis; (3) arytenoid cartilage; (4) cricoid cartilage.



▲ 95 **Ultrasonographic image of the thorax (transversal view). (a) The air-filled lung interface is seen as a white hyperechoic line (red arrows). Additional hyperechoic lines parallel and below the lung surface are artifacts. 'Comet-tail' artifacts may be seen radiating from the lung surface (yellow arrows). (1) chest wall. (b) Horse with pleuropneumonia. (1) pleural effusion; (2) lung; (3) diaphragm.**

aerated lung will be associated with comet-tail artifacts. Identifying a small number of isolated comet-tail artifacts (<5) is not uncommon in healthy horses, in particular in the lung periphery and the most cranioventral aspect of the lung. However, large numbers of comet-tail artifacts, especially multiple artifacts fanning out from a single intercostal space, suggest lung pathology. In humans, comet-tail artifacts have been associated with pulmonary edema and interstitial lung disease (subpleural thickened alveolar septa). In horses, conditions associated with comet-tail artifacts are mild, early lung parenchyma consolidation, pulmonary edema, and interstitial lung disease.

SAMPLING OF RESPIRATORY SECRETIONS

Mucociliary clearance constantly moves mucus, cells, and potential contaminants from the peripheral lung regions towards the carina and up the trachea before they are expectorated. Secretions are usually collected by tracheal wash (TW) or bronchoalveolar lavage (BAL). Cytologic and microbiologic evaluation of respiratory samples provides invaluable information about the disease process. In cases of infectious lung disease (e.g. bacterial pneumonia), affected areas are often adjacent to unaffected areas. Secretions originating from affected lung segments will eventually collect in the trachea. In these cases, cytologic examination and microbiologic culture of TW fluid is likely to yield an etiologic diagnosis. In contrast, fluid collected by BAL is only representative of the lung region distal to the bronchus where the tube or endoscope was wedged. Infectious lung diseases such as pneumonia lead to focal or multifocal disease usually affecting cranioventral lung regions. Even with endoscopic guidance it is difficult to follow airways down to an affected lung segment. As a result, it is common to harvest cytologically normal BAL samples from horses with pulmonary infection. However, in cases of diffuse lung disease (e.g. RAO, inflammatory airway disease [IAD]) BAL fluid cytology correlates well with histology, whereas TW cytology does not. This is probably due to contamination of the trachea by inhaled materials or nasopharyngeal secretions.

What are the indications for TW or BAL? In general, TW is the preferred method for suspected localized infectious cases and BAL is preferred for characterizing diffuse lung disease. In cases of uncertain etiology, it is best to perform both tests in order to maximize diagnostic yield. If both diagnostic techniques are used, TW is performed first to avoid cross contamination between procedures.

Tracheal wash

Tracheal secretions may be collected by direct aspiration or tracheal lumen wash via transcutaneous catheterization (*Table 2*) or through an endoscope (*Table 3*). The transcutaneous route is the least expensive method, but care is required in cases of infectious disease because of the risk of subcutaneous abscessation at the puncture site. It is important to keep the needle in place until the catheter has been pulled out after sample collection, in order to decrease the chances of subcutaneous abscessation. Injection of antibiotics through the needle as it is being withdrawn may help prevent local neck infection. Commercially available kits are supplied with a trocar introducer inside a blunt-ended cannula. After tracheal puncture, the trocar is removed and the catheter introduced, thus reducing the risk of iatrogenic trauma to the trachea. Alternatively, tracheal secretions may be aspirated through the instrument channel of the endoscope. A sterile, multiple lumen catheter (double or triple lumen) is needed if microbiologic culture of the sample is desired (**96**). Large commensal bacterial populations inhabit proximal airways, including *Streptococcus equi* subsp. *zooepidemicus*. Therefore, direct aspiration of tracheal secretions via the instrument channel of the endoscope will result in contamination from proximal airways and will be inappropriate for microbiologic culture.

Cytology specimens demonstrating a predominance of neutrophils with various degrees of degenerative changes (karyolysis and cytoplasmic vacuolation) and the presence of intra- or extracellular bacteria are consistent with infection and should prompt microbiologic culture of the fluid. Coughing during the TW procedure may lead to oropharyngeal contamination, recognized by the presence of squamous epithelial cells laden with bacteria. Improper handling of TW fluid

Table 2 **Transtracheal wash procedure**

Equipment and supplies

- Sterile 10 gauge needle or trocar.
- Sterile No. 7 French polyethylene catheter.
- No. 15 surgical blade.
- 35 ml sterile syringe.
- Sterile isotonic saline solution .
- 2–3 ml of 2% lidocaine solution.

Technique

- The animal should be tranquilized (e.g. xylazine: 0.3–0.5 mg/kg IV).
- An area approximately 10 x 10 cm over the mid- to distal-third of the trachea is clipped and prepared aseptically.
- Lidocaine is injected subcutaneously over the site on the midline and a final scrub is performed.
- A small stab incision through the skin is made with a No. 15 blade.
- The trachea is stabilized with one hand and a 10-gauge needle introduced between two tracheal rings. After penetrating the trachea, the needle is directed downward and held tight against the neck.
- The catheter is inserted through the needle approximately 30–40 cm into the trachea. There should be no resistance when passing the catheter and air should be easily aspirated.
- A 20–30 ml bolus of sterile isotonic solution is then injected rapidly through the catheter and immediately aspirated while the catheter position is adjusted to maximize yield. Once an adequate sample has been obtained, first the catheter and then the needle is removed. A small volume (2–3 ml) of antibiotic (e.g. procaine penicillin, ceftiofur) may be injected as the needle is withdrawn, to avoid abscess formation.
- The sample should be cultured for aerobic/anaerobic bacteria if indicated. Another aliquot should be used for cytology (prepare air-dried slides directly or submit sample in syringe/EDTA tube to laboratory).
- The neck incision may be covered with an elastic adhesive bandage (no more than 24 hours).

Complications

- Pharyngeal contamination if the needle and catheter were directed upward or if the horse coughed during the procedure.
- Cutting off the catheter at the needle and loss into the airways. Usually, the catheter is rapidly coughed up.
- Local cellulitis, abscessation, or subcutaneous emphysema at the incision site.
- Trauma to the trachea resulting in hemorrhage or damaged tracheal ring with subsequent chondroma formation.

Table 3 **Tracheal wash procedure with an endoscope**

Equipment and supplies

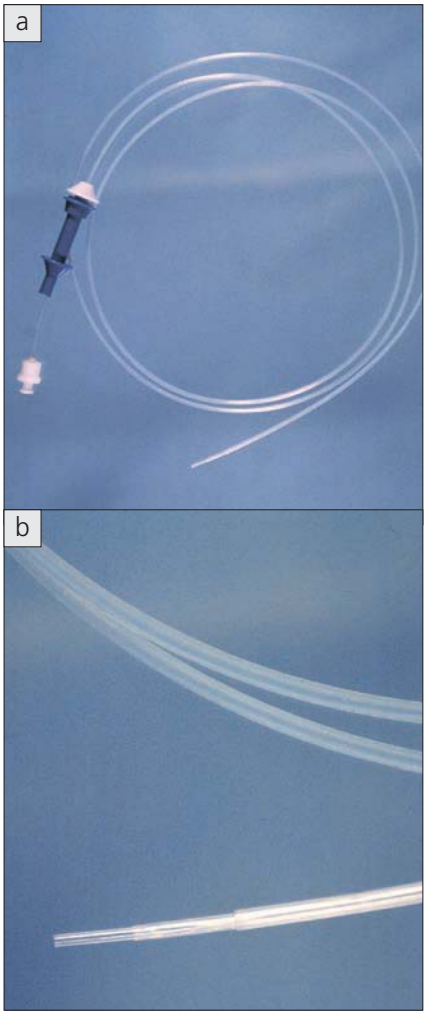
- Minimum 1 meter long endoscope.
- Double or triple lumen catheter.
- 35 ml sterile syringe.
- Sterile isotonic saline solution.

Technique

- The animal should be tranquilized (e.g. xylazine: 0.3–0.5 mg/kg IV) or a nose twitch applied.
- The catheter is advanced in the endoscope instrument channel until it reaches the tip.
- The endoscope is passed through the proximal airways until it reaches the mid-cervical trachea.
- The catheter is exteriorized and the sterile inner tubings deployed.
- A 20–30 ml bolus of sterile isotonic solution is then injected rapidly through the catheter and immediately aspirated while positioning the catheter tip in the tracheal puddle. A minimum of 2 ml of fluid is needed for cytology and bacteriology.
- The sample should be cultured for aerobic/anaerobic bacteria if indicated. Another aliquot should be used for cytology (prepare air-dried slides directly or submit sample in syringe/EDTA tube to laboratory).

Complications

- Pharyngeal contamination if the horse coughs during the procedure. It is important to keep the head elevated.
- Difficulty in aspirating fluid due to thick secretions and small catheter lumen. It is helpful to infuse additional sterile solution to unplug the catheter followed by gentle aspiration from the liquid–air interface.



▲ 96 **(a) Triple lumen catheter used to perform a tracheal wash. (b) Close-up view of the tip showing the two inner tubes protruding through the larger diameter outer tube.**

may also lead to degenerative changes of neutrophils in cases of non-infectious purulent inflammation. The absence of bacteria or degenerative neutrophils does not rule out the possibility of an infectious disease. Aerobic and anaerobic cultures are recommended in febrile horses or those with a history suggestive of infectious disease. A Gram stain may help guide antibiotic therapy while waiting for culture results. Culture of TW fluid from infectious cases resulting in no growth may be due to too few bacteria, prior antimicrobial therapy, inappropriate culture medium (e.g. mycoplasmal and fungal infections), or viral infection without secondary bacterial infection. Alternatively, a bacterial isolate should be considered significant if it is a known pathogen, fluid cytology is consistent with infection, and clinical examination is suggestive of an infectious process.

Normal TW fluid cytology should contain a scant amount of mucus, a predominance of pulmonary alveolar macrophages (PAMs), and epithelial cells, with <20% neutrophils, <10% lymphocytes, and <1% eosinophils.

Bronchoalveolar lavage

BAL can be easily performed in field conditions using either a flexible endoscope (≥1.8 m long) or an equine BAL catheter at least 2.5 m long and 10 mm diameter with an inflatable cuff at the end (**97**). The technique is described in *Table 4*. The horse should be sedated with xylazine hydrochloride (0.4–0.8 mg/kg IV) or detomidine hydrochloride (0.01–0.02 mg/kg IV) and restrained with a nose twitch. The flexible endoscope or BAL tube is then passed through the nasal passages and advanced gently until it is wedged into the distal airways. Coughing may be prevented by spraying the airways with a 0.2–0.5% lidocaine solution (5–10 ml at a time) as the instrument is advanced into the respiratory tract, focusing particularly on the glottis and carina. Horses with RAO (heaves) may cough excessively during the procedure and routine premedication with butorphanol tartrate (0.01–0.02 mg/kg IV) or inhaled albuterol sulfate (1–2 µg/kg) 5–10 minutes prior to the lavage are beneficial. A volume of 250–500 ml of warm sterile saline solution is infused (in one or two boluses) under pressure followed by immediate but gentle aspiration of the fluid using 60 ml syringes or a suction pump. It is important always to use the same technique because the volume of fluid used, as well as the number of boluses administered, has a significant effect on cell count and differential. Infusion of at least 250 ml is required to collect an appropriate sample. On average, 50–70% of the infused volume can be aspirated. Smaller volumes are retrieved from horses with collapsible airways because of diseases such as heaves. Fluid samples should be processed within 1 to 2 hours or stored on ice or at 4°C if transport to the laboratory is likely to be delayed. Normal BAL fluid should appear slightly turbid with a layer of white foam on the surface (**98**) and contain <400 cells/µl.

Normal BAL fluid cytology should show mainly lymphocytes (30–60%) and PAMs (40–70%), with <5% neutrophils, <2% mast cells, and <1% eosinophils.

Table 4 **Bronchoalveolar lavage procedure**

Instrumentation and supplies

- A 240–300 cm long, 10 mm diameter bronchoalveolar tube or a 180–200 cm long, 8–10 mm diameter endoscope.
- 60 ml sterile syringes or suction pump.
- 250–500 ml of warm sterile isotonic saline solution.
- 60 ml of warm lidocaine solution (1 volume of 2% lidocaine/6 volumes of sterile solution).
- Bucket of ice.

Technique

- The animal should be tranquilized (e.g. xylazine: 0.4–0.8 mg/kg IV) and a nose twitch applied. Butorphanol administration (0.01–0.02 mg/kg IV) helps decrease coughing.
- The bronchoalveolar lavage tube or endoscope is passed through the nose into the trachea until it becomes wedged in a distal airway. Infuse diluted lidocaine solution as needed (particularly around the carina). Inflate the tube cuff with 5–10 ml of air.
- Infuse sterile solution rapidly (e.g. using a pressurized bag) while keeping the tube/scope wedged. It is best to infuse large volumes (e.g. 500 ml) in two boluses.
- Then gently suction bronchoalveolar lavage fluid by hand with 60 ml syringes or via a suction pump (<300 mmHg).
- Place bronchoalveolar lavage fluid on ice or in a refrigerator (4°C) until processing (within 4 hours of collection).

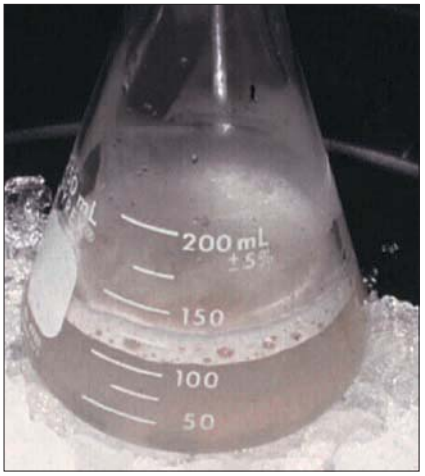
Complications

- Excessive coughing.
- Local neutrophilic inflammation that spontaneously resolves within 48 hours.
- Transient pyrexia (rare), which usually resolves within 24 hours, without treatment.

Airway brushing

A cytology brush may be used for collection of epithelial cells or tissue fragments. The brush is retracted into a protective sheath and inserted through the endoscope instrument channel. Cells are obtained by gently brushing the airway wall numerous times at each of a series of locations inside the airway of interest, to obtain a representative sample. Care should be taken to avoid mucosal bleeding. The brush is then retracted into its protective sheath and removed from the endoscope channel. Two or three smears may be prepared directly by rolling the brush over a slide or cells may be dislodged by shaking the brush into a tube filled with fixative solution. Airway brushing may be valuable when direct biopsy of tissue present some risks (e.g. hemorrhage).

▲ 97 **Bronchoalveolar lavage performed in the field using a flexible tube with cuff at the distal end. (See *Table 4* for a description of the procedure.)**



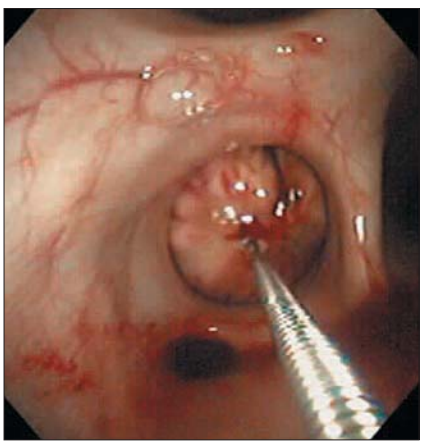
▲ 98 **Normal bronchoalveolar lavage fluid. The fluid is turbid in appearance and foam containing surfactant is floating on top.**

AIRWAY AND LUNG BIOPSY

Collection of tissue samples from the respiratory tract is a valuable diagnostic and prognostic tool. It allows for histopathology, but also for more advanced testing such as immunohistochemistry. The least invasive technique involves endobronchial sampling of airway tissue under bronchoscopic guidance. The technique is safe, but only small, superficial layers of airway mucosa are sampled. Parenchymal tissue may be obtained using closed or open lung biopsy techniques. Closed techniques include percutaneous and thoracoscopic lung biopsy.

Endobronchial biopsy

Transendoscopic biopsy of the airway may be done from the nasal passages to peripheral airways as small as 2 mm in diameter (**99**). The forceps used may be cupped, fenestrated, or alligator type. In general, larger forceps allow a larger amount of tissue to be collected and provide a better chance of obtaining diagnostic samples. Alligator forceps usually sample more tissue than cupped forceps. The horse should be sedated and restrained as for any endoscopic examination. Horses with increased breathing effort due to bronchoconstriction, such as in heaves, should be premedicated with a bronchodilator (e.g. 500 µg of inhaled albuterol).



▲ 99 **Biopsy of a mass protruding through a bronchus, using cupped forceps. Histologic findings were consistent with a granular cell tumor.**

Nasal oxygen supplementation (5–8 l/min) should be considered in horses with increased breathing effort associated with hypoxemia. Topical anesthesia is recommended when sampling upper airways or to decrease coughing when sampling lower airways. A solution of diluted lidocaine (1 volume 2% lidocaine/6 volumes sterile saline) is prepared in a 20 ml syringe and sprayed onto the airway surface either directly through the endoscope working channel or via a catheter.

In the lungs, samples are taken at the airway bifurcation from the main carina to the subsegmental airways. A minimum of five samples is recommended and up to 20 bronchial biopsies can be obtained safely from one adult horse.

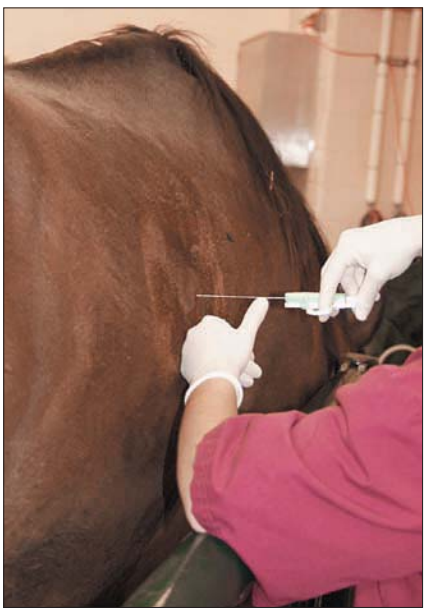
Percutaneous lung biopsy

This procedure is performed with the horse restrained in stocks and sedated. A 5 × 5 cm area of skin is clipped between the 7th and 10th intercostal spaces, approximately 8–10 cm above the shoulder joint, or over any lung area identified for sampling by ultrasonography or radiography. The skin is aseptically prepared and subcutaneous tissue and intercostal muscles are infiltrated with 20 ml of 2% lidocaine solution followed by a surgical scrub. Spring-loaded biopsy needles (14 g × 20 cm) are preferred to Tru-Cut biopsy needles because they result in fewer complications (airway bleeding, hematoma). The biopsy needle is carefully inserted through a small skin incision just cranial to the rib and advanced through the chest wall (**100**). The needle is then inserted rapidly into the lung periphery at the end of inspiration and retrieved immediately after triggering a sample collection. Multiple biopsies (2–4) should be taken to improve the diagnostic value since samples are small. Lung tissue should be carefully lifted from the biopsy needle using a sterile needle and placed in 10% neutral buffered formalin. A tissue sample may be saved for microbiologic culture if indicated.

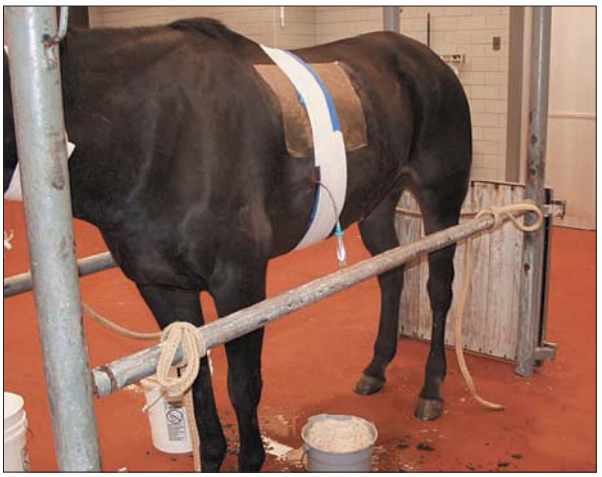
Percutaneous lung biopsy is a relatively safe diagnostic method. However, significant complications may occur, including the possibility of death from hemorrhage or pneumothorax in approximately 3% of cases, and owners should be warned of these complications. Other complications, such as coughing, airway bleeding, epistaxis, and hematoma at the biopsy site, are common but usually self-limiting. Some horses that receive insufficient local anesthesia may show signs related to pain from the biopsy site, such as depression, with intermittent signs of mild colic, collapse inside the stock or when walking out, or ataxia.

Thoracoscopic lung biopsy

A relatively large tissue specimen may be obtained by thoracoscopic guided lung resection using pre-tied ligating loops or a commercially available endoscopic stapler. An area of diseased lung or the edge of the lung is grasped with forceps and a wedge is resected after the base is secured by ligature or staples. The most common complications are pneumothorax and hemorrhage. Slippage of the ligature is common, therefore a stapling device should be readily available during the procedure. Nasal oxygen supplementation or positive pressure ventilation of horses undergoing thoracoscopic lung biopsy is recommended.



▲ **100 Percutaneous lung biopsy performed using a spring-loaded biopsy needle.**

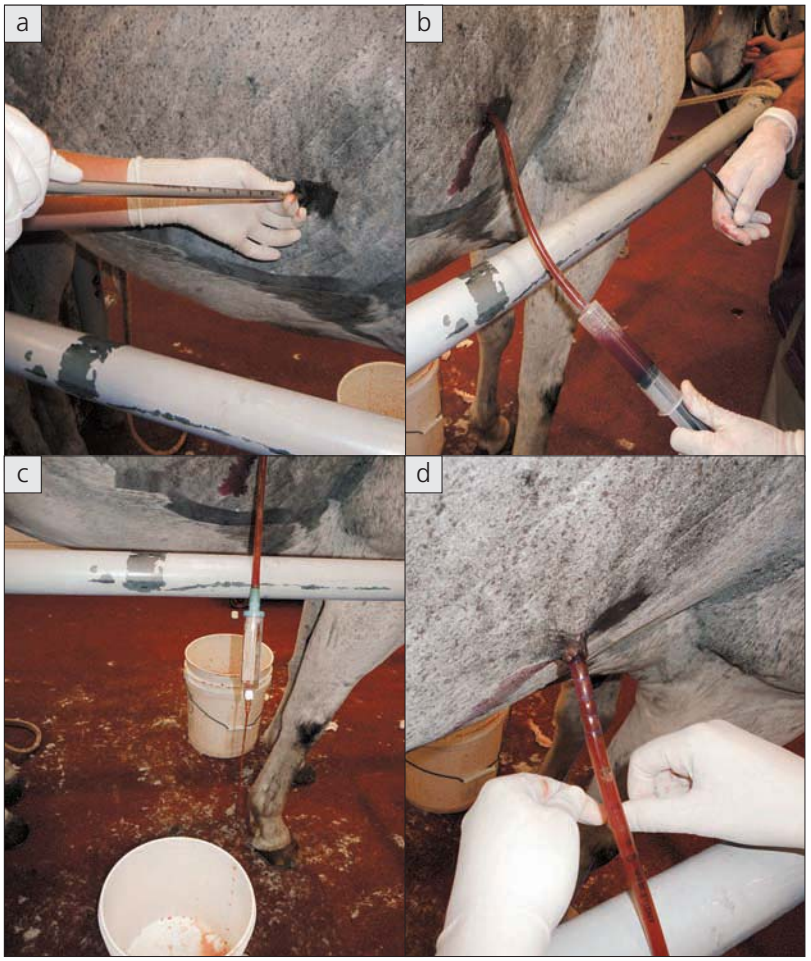


▲ **101** **Horse with pleuropneumonia secondary to chest trauma, with a chest tube and a Heimlich valve in place. Approximately 10 liters of pleural fluid has drained into the bucket and a proteinaceous foam is visible on top of the fluid.**

THORACOCENTESIS

Thoracocentesis is performed for diagnostic and therapeutic purposes in cases of pleural effusion. The procedure is performed on standing horses restrained in stocks and with sedation if appropriate. Ideally, thoracic ultrasonography is conducted to identify the extent of pleural effusion and important landmarks, such as the heart and lungs, in order to optimize positioning of the thoracocentesis needle. Alternatively, the extent of the fluid line may be determined by auscultation and percussion. The equine mediastinum is fenestrated cranioventrally allowing fluid to migrate from one side to another. However, communications between the hemithoraces are often sealed in inflammatory processes and collection of pleural fluid from both the left and right side is recommended to maximize diagnostic ability.

The puncture site on the right side is located in the 6th–7th intercostal spaces and approximately one hand width (10 cm) above the olecranon. The site on the left is in the 6th–9th intercostal spaces and 5 cm above the olecranon. The site is clipped and surgically prepared. The subcutaneous tissue and intercostal muscle are infiltrated with 5–10 ml of 2% lidocaine solution, while carefully avoiding damage to blood vessels and nerves running immediately caudal to each rib. Infiltration with lidocaine should also extend caudally over the adjacent rib for 2–3 cm. A stab incision is then made 2–3 cm caudal to the intercostal space. A sterile teat cannula is preferred for thoracocentesis because the blunt tip will avoid lung laceration. The cannula is first connected to extension tubing and a three-way stopcock in closed position. The cannula is carefully inserted through a small stab incision at a shallow angle while pointing the tip cranially and advancing it under the skin until it reaches the cranial aspect of the rib. This gap between skin incision and point of entry in the pleural space will prevent pneumothorax on removal of the cannula. The needle is then rotated perpendicularly to the chest wall and pushed through it. Passage into the pleural space will be felt as a sudden loss of resistance to advancement of the cannula. At this point, the three-way stopcock is opened to allow drainage of pleural fluid. If fluid does not drain spontaneously, gentle suction may be applied with a syringe. Aliquots of the fluid are placed in an EDTA tube (2 ml) for cytology and into a dry sterile tube for aerobic and anaerobic bacterial culture. Additional fluid may be drained from the pleural space if justified therapeutically. It is not uncommon to drain 10–15 liters from one side of the chest in horses with pleuropneumonia (**101**). In such cases, large indwelling chest tubes (e.g. 24–30 French or 8–10 mm diameter) are put in place because inflammatory debris often occludes small-bore tubes. Chest tube thoracostomy is performed with hollow tubes fenestrated at the tip and loaded onto a trocar for ease of insertion. Chest tube placement follows the same



◀ 102 (a) Thoracocentesis technique. Insertion of a chest tube in a horse with pleuropneumonia. (b) Aspiration of pleural fluid with a catheter tip syringe connected to the chest tube. (c) One-way valve (Heimlich) placed on the end of the chest tube to allow pleural fluid drainage while preventing aspiration of air into the pleural space. (d) A thoracostomy tube is secured to the chest by a purse-string suture followed by a 'Chinese finger trap' suture pattern.

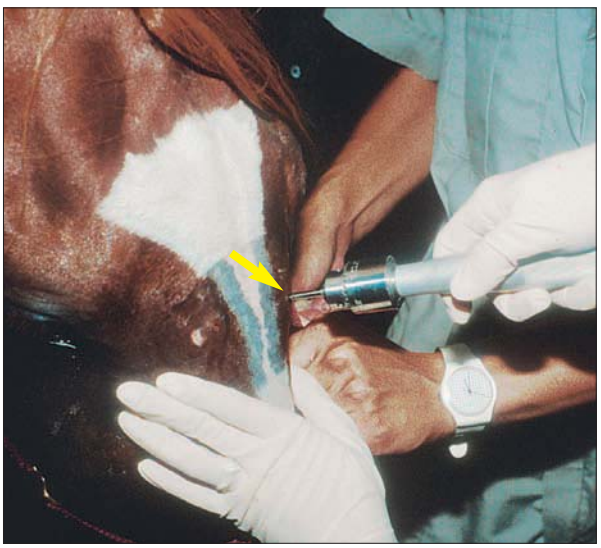
technique described for a teat cannula, but because penetration of the chest wall requires a strong push on the trocar, care must be taken not to penetrate the pleural space too rapidly and damage the lung surface. This may be accomplished by holding the chest tube tight with one hand as it enters the skin in order to control the depth of penetration, while the other hand pushes on the trocar (**102**a). As soon as the chest wall is punctured, the trocar is pulled out with one hand, while the other hand holds the tubing in place. Pleural fluid should be seen coming out from behind the tip of the trocar. If not, a catheter tip syringe may be inserted to aspirate fluid (**102**b) or the trocar is reinserted and the chest tube advanced further until fluid drainage is accomplished. Ultrasound guidance may be useful if fluid does not drain readily. A one-way valve (Heimlich valve; **102**c), penrose drain, or finger from a surgical glove with the tip cut off, is then secured at the end of the tubing to avoid air aspiration. Finally, the skin incision is sealed around the chest tube by a purse-string suture, and a 'Chinese finger trap' suture pattern is used to fasten the tube to the chest wall (**102**d).

SINUS TREPHINATION

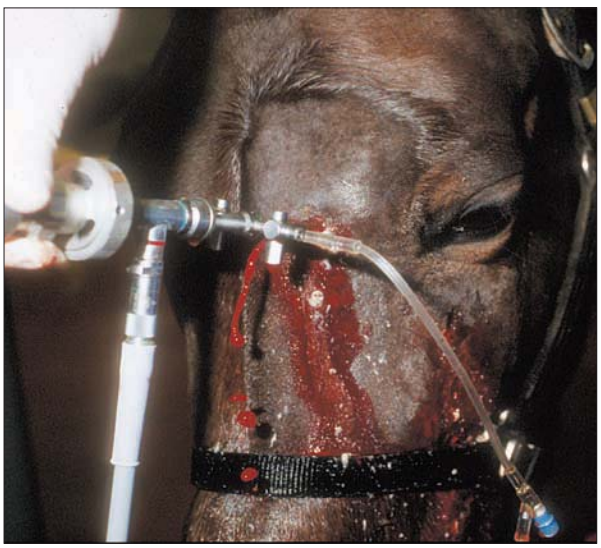
Trephination of the paranasal sinuses has been used extensively for sinus lavage and exploration, teeth removal, and to obtain biopsy samples. The technique involves local anesthesia and a small curvilinear or X-shaped incision over the sinus to be entered. The maxillary (rostral and caudal) and frontal sinuses are the most common sites for trephination. A galt trephine or Steinmann pin is used either manually or with motorized equipment to enter the sinus (**103**). A trephine creates a circular opening 5–10 mm in diameter. The opening can be enlarged with bone rongeurs or two holes can be created side by side. The main disadvantage of trephination is the limited access to the sinus. This limits the technique to minor procedures and tooth root removal. For tooth removal the trephine site needs to be located directly over the abnormal tooth. The trephine site should be confirmed with diagnostic imaging prior to beginning the procedure.

Sinoscopy

Sinoscopy is used in the evaluation of diseases involving the paranasal sinuses and is helpful in preoperative planning for osteoplastic bone flap placement. It involves the use of a flexible or rigid endoscope (arthroscope) positioned into the maxillary or frontal sinus via a small trephine hole (**104**). The authors prefer to use a 4 mm arthroscope with a fluid delivery system attached. The endoscope is positioned into the maxillary and frontal sinus and the interior of the sinus is examined. Sinoscopy can be used for the diagnosis of sinusitis, neoplasia, periapical tooth root infection, ethmoid hematoma, and epistaxis. More than one sinoscopy portal can be created to facilitate collection of biopsy samples or for sinus lavage.



▲ **103** **Trephination of the frontal sinus of a horse with a Steinmann pin (arrow).**



▲ **104** **Frontal sinus sinoscopy in a horse with chronic sinusitis. Note the purulent exudate mixed with blood on the skin, which is being flushed out during the sinoscopy procedure.**

THORACOSCOPY

Endoscopy of the thoracic cavity has been used in the evaluation of pleuropneumonia, thoracic neoplasia, and pleural disease, and to examine trauma induced by foreign body penetration. It has also been used for biopsy of the lung. Thoracoscopy is performed using standard laparoscopic techniques. The horse is placed in stocks and sedated IV or placed under general anesthesia (**105**). The laparoscopic portal is locally anesthetized and a small stab incision is made to allow for introduction of the laparoscope. If performed standing, it is very important to provide nasal oxygen and anticipate the potential need for ventilatory support during the procedure. The ability to examine the thoracic cavity completely will depend on the extent of pathology within the thorax. Pleural adhesions, neoplastic masses, thoracic effusion, or abscessation can make complete thoracic examination difficult or impossible. Nonetheless, this minimally invasive technique can be very useful in the diagnosis, treatment, and prognosis of thoracic disease.

LUNG FUNCTION TESTS

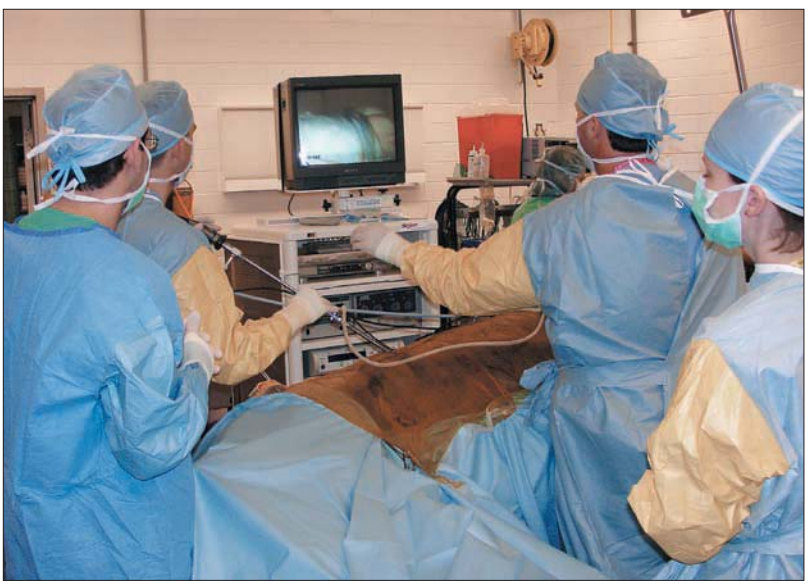
Arterial blood gases

Arterial blood gas (ABG) measurements are commonly used to assess the efficiency with which the lung exchanges gas. ABG provides information about three physiologic processes: alveolar ventilation, oxygenation, and acid–base balance. In order to understand these processes four equations are needed:

- P_{aCO_2} equation (alveolar ventilation).
- Alveolar gas equation (oxygenation).
- Oxygen content equation (oxygenation).
- Henderson–Hasselbalch equation (acid–base balance).

Other information needed to interpret ABG includes:

- Animal's environment (fraction of inspired O_2 [F_{IO_2}], barometric pressure).
- Additional lab data (previous ABG, electrolytes, chest X-ray).
- Clinical information (respiratory rate [RR], respiratory effort, mental status, tissue perfusion).



▲ 105 A horse in lateral recumbency for thoracoscopy. The laparoscope has been inserted in the paralumbar fossa, in order to visualize a diaphragmatic hernia of the large colon into the right hemithorax.

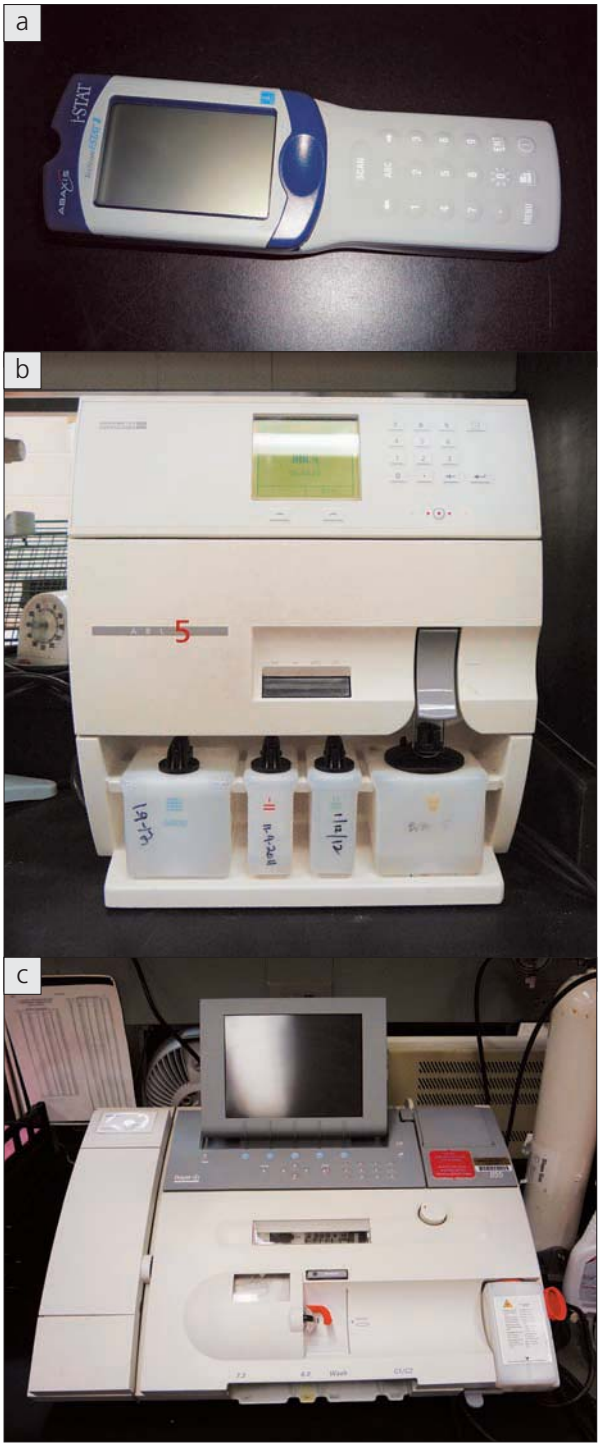
Blood gas analyzers

There are three main types of blood gas analyzers: a point-of-care ('portable') unit, a table-top machine, and a co-oximeter (**106**). The last is the only machine that actually measures hemoglobin content, S_{aO_2}, as well as carboxyhemoglobin and methemoglobin percentages. The other analyzers measure pH, P_{aO_2}, and P_{aCO_2}, and calculate other parameters such as bicarbonate concentration. Table-top analyzers are highly accurate; however, they are expensive and require demanding maintenance and calibration routines. Point-of-care analyzers are becoming widely used in practice because results are obtained rapidly (2–3 minutes) on the spot, calibration is automated, and maintenance is minimal.

Blood collection procedure

A volume of 2–4 ml of blood is adequate in most cases. A syringe (3–5 ml) and small-gauge needle (25–22 gauge) should be used, according to the blood volume needed and artery size. Plastic syringes are adequate if blood gas analysis is conducted within 10–15 minutes of collection. Blood collected in plastic syringes should be kept at room temperature until analyzed, because storing it in iced water will artificially raise P_{aO_2}. Otherwise, blood should be collected in glass syringes, stored on ice, and analyzed within 1 hour. Heparin solution (1,000 units/ml) is aspirated into the syringe and expelled until no visible accumulation of heparin is detected (i.e. heparin only fills dead space). Excess heparin dilutes blood and results in changes in P_{aO_2} and P_{aCO_2}. Alternatively, blood gas syringes preloaded with lyophilized lithium heparin may be used to mitigate the potential effect of liquid heparin on blood gas values.

Blood may be collected from any accessible artery, as long as puncture is performed through intact skin and tissues and the horse does not suffer from a coagulopathy. Arterial puncture is usually done without local anesthesia. However, some horses and most foals may resent skin puncture, in which case a small volume (0.5 ml) of 2% lidocaine solution should be injected subcutaneously over the site using a 25 gauge needle. Sedation is not recommended, in particular with alpha-2 agonists (e.g. xylazine, detomidine, and romifidine) because of their marked effect on blood gases. Alpha-2 agonists affect blood gases by decreasing



▲ **106 Three types of blood gas analyzer. (a) Point-of-care unit (i-STAT®). (b) Table-top blood gas analyzer (ABL-5). (c) Co-oximeter (Bayer 800).**

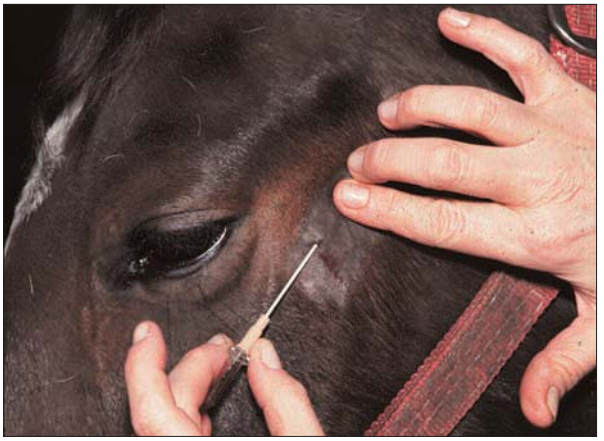
minute ventilation. Reduction in ventilation results in hypoxemia and hypercapnia. Similarly, excitement or stress may lead to hyperventilation and artificially alter blood gases. The puncture site is clipped or shaved and disinfected with an alcohol swab. The blood must be collected anaerobically, and any air bubble expelled and the syringe capped immediately. After sample collection, the puncture site should be compressed for at least 3 minutes and then observed for any signs of active bleeding. The animal's body temperature, position (standing, recumbent sternal or lateral), sample site, and supplemental oxygen flow rate should be recorded with the results of blood gas analysis.

Collection sites and technique

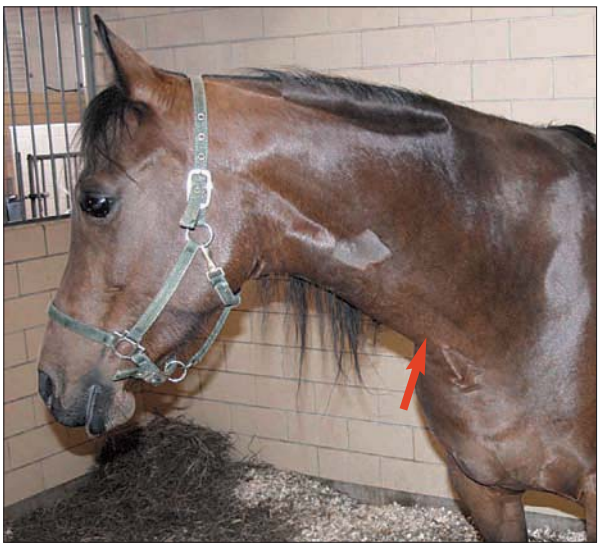
Blood can be readily collected from the transverse facial and carotid arteries in standing and non-sedated adult horses. In foals it is recommended that arterial puncture is only conducted in recumbent animals and with proper restraint to avoid damage to surrounding tissue if they move. The dorsal metatarsal artery is the site of choice in foals, because it does not roll when punctured and there is no adjacent vein. It is important to keep the foal in sternal recumbency while collecting arterial blood, in order to avoid blood gas changes due to atelectasis from lateral recumbency, in particular in foals with significant lung pathology. In anesthetized recumbent horses several other arteries may be sampled, such as the auricular, brachial, facial, and digital arteries.

Transverse facial artery puncture is easily performed and well tolerated by most horses. The pulse may be felt caudally to the eye and approximately 3 cm below the zygomatic arch (**107**). The syringe and needle are held parallel to the zygomatic arch, pointing caudally with a shallow angle towards the artery. A slight vacuum is created with the plunger as the needle is advanced until blood readily fills the syringe.

Carotid artery puncture is well tolerated by adult horses standing, but is not recommended in foals. The needle should be at least 5 cm long and larger than



▲ 107 Placement of an indwelling catheter in the transverse facial artery to allow serial blood sampling. The right hand fingers are placed over the artery to feel the pulse and guide the needle during puncture. The left hand positions the catheter parallel to the artery. The same guidelines are used to collect blood with a syringe and needle.



▲ 108 Location and direction for carotid puncture (arrow).

needles used for other puncture sites (e.g. 19–21 gauge). The needle and heparinized syringe are held horizontally at a 45° angle from the sagittal plane, while aiming for the carotid in the lower third of the neck between the trachea and the jugular vein (**108**). While it may take several attempts to hit the carotid artery, significant damage to adjacent structures is rare. After a sample is collected, strong pressure with a closed fist should be kept on the puncture site for 5 minutes.

The dorsal metatarsal artery is located in a groove between the cannon bone and the lateral splint bone of the hindlimb. The arterial pulse is easily palpated if the limb is abducted while the foal is maintained in sternal recumbency. A small needle (24–25 gauge) connected to the heparinized syringe is placed over the groove in the proximal third of the splint bone, aiming towards the hock. The needle should enter the skin at a shallow angle.

Blood gas interpretation

P_{aCO_2} is proportional to CO_2 produced by metabolism ($\dot{V}_{CO_2}$) and inversely proportional to alveolar ventilation ($\dot{V}_A$) according to the equation:

$$P_{aCO_2} = 0.863 \times \dot{V}_{CO_2}/\dot{V}_A,$$

where $\dot{V}_A = f \times (V_T - V_D)$

[f = respiratory rate; VT = tidal volume; VD = dead space volume]

From this equation, it is clear that it is impossible to predict P_{aCO_2} from clinical examination alone because many different combinations of respiratory rate, depth or breathing effort may correspond to a given P_{aCO_2}. Therefore, P_{aCO_2} measurement is the best gauge to determine efficacy of ventilation. Normal P_{aCO_2} in adult horses breathing ambient air is between 36 and 46 mmHg. Hypoventilation decreases effective alveolar ventilation and causes an increase in P_{aCO_2} or hypercapnia. Deep sedation, anesthesia, severe neurologic disease (e.g. 'dummy' foal syndrome), respiratory muscle weakness (e.g. botulism), or severe lung disease may cause hypercapnia. Hyperventilation decreases P_{aCO_2} (hypocapnia) and may be observed with recovery from exercise, excitement, respiratory compensation for metabolic acidosis, or chronic lung disease. Noninvasive measurement of end-tidal P_{CO_2} (Pet_{CO_2}) by capnography is convenient and can provide an estimate of P_{aCO_2} as long as there is even distribution of ventilation and no major perfusion abnormality. In cases of pulmonary disease, there could be a significant different between Pet_{CO_2} and P_{aCO_2}. However, because both measurements will follow the same trend over time, determination of Pet_{CO_2} and P_{aCO_2} at baseline would enable subsequent continuous monitoring of the patient by capnography, thereby reducing the number of arterial blood gas measurements needed.

The difference between the P_{AO_2} and the P_{aO_2} is called the alveolar–arterial oxygen tension difference ($A–aD_{O_2}$). In a healthy horse at rest, the $A–aD_{O_2}$ is <10 mmHg. Since P_{AO_2} is difficult to measure, the ideal alveolar P_{O_2} or the alveolar O_2 that would exist in the lung if there were no $\dot{V}/\dot{Q}$ abnormality is calculated using the ideal gas equation:

$$P_{AO_2} = P_{IO_2} - 1.2(P_{aCO_2}),$$

where P_{IO_2} = % inspired O_2 × (barometric pressure – 47 mmHg) and P_{AO_2} = average P_{O_2} for all the alveoli

Therefore, in a normal horse with P_{aCO_2} = 40 mmHg, breathing room air (% inspired O_2 = 21%) with barometric pressure 760 mmHg, and $P_{AO_2} \approx 100$ mmHg, we would expect P_{aO_2} to be ≈ 90–95 mmHg.

Calculation of $A–aD_{O_2}$ allows identification of mechanisms responsible for hypoxemia, which helps with therapy. $A–aD_{O_2}$ is very likely to be elevated in horses with significant $\dot{V}/\dot{Q}$ mismatch or physiologic shunt, whereas it will be unchanged in horses with hypoventilation. When shunting is a problem, the calculated $A–aD_{O_2}$ will increase progressively as the concentration of inspired O_2 increases, since none of the shunted blood contacts alveoli with increased P_{AO_2}, but P_{aO_2} will increase very little in comparison.

However, when $\dot{V}/\dot{Q}$ mismatch is a problem, the calculated $A–aD_{O_2}$ will increase less as the concentration of inspired O_2 increases and P_{aO_2} will increase significantly. A more rapid but less accurate estimation of what the expected P_{aO_2} should be for any given inspired percent O_2 is given by multiplying the % inspired O_2 by 5:

- At 20% O_2 (room air) expect P_{aO_2} of 100 mmHg.
- At 50% O_2 expect P_{aO_2} of 250 mmHg.
- At 100% O_2 expect P_{aO_2} of 500 mmHg.

In adult horses, normal P_{aO_2} ranges between 90 and 100 mmHg, and hypoxemia is when P_{aO_2} is <80 mmHg. It is important to remember that barometric pressure is lower at higher altitude, resulting in a drop in P_{aO_2} of ≈18 mmHg for every 1,000 m of elevation if P_{aCO_2} remains constant. Because $A–aD_{O_2}$ changes little with elevation, every 1,000 m elevation will result in a decrease in P_{aO_2} of approximately 18 mmHg (e.g. P_{aO_2} = 95 mmHg at sea level and 77 mmHg at 1,000 m). The horse's response to rapid elevation is hyperventilation and splenic contraction. Over time, adaptation to high altitude environment will result in higher RBC numbers that are detectable as early as 1 week following transport to a high altitude environment. Note also that in neonatal foals during the first week of life, P_{aO_2} ranges between 65 and 80 mmHg. Therefore, hypoxemia occurs in foals if P_{aO_2} is <60 mmHg.

Increased respiratory effort most commonly results from the attempt made by the respiratory tract to meet metabolic demand, while maintaining P_{aO_2} and P_{aCO_2} within physiologic ranges. Various types of cardiopulmonary disease may result in abnormal gas exchanges. In general, five causes of hypoxemia are recognized: hypoventilation, $\dot{V}/\dot{Q}$ inequality, shunt, diffusion impairment, and reduction of inspired P_{aO_2}. The last cause is encountered in situations such as high altitude environment. Hypoventilation is a reduction in the delivery of fresh gas to the alveoli and is always accompanied by hypercapnia. The magnitude of the decrease in P_{aO_2} is directly related to the increase in P_{aCO_2} according to the alveolar gas equation. In first approximation, every increase in P_{aCO_2} of 1 mmHg results in a decrease in P_{aO_2} of 1 mmHg. Causes of hypoventilation include depression of the respiratory center by pharmacologic agents (e.g. anesthetic drugs) or diseases of the nervous system (e.g. botulism, hypoxic–ischemic encephalopathy), restrictive diseases (e.g. pleural effusion), and upper airway obstruction.

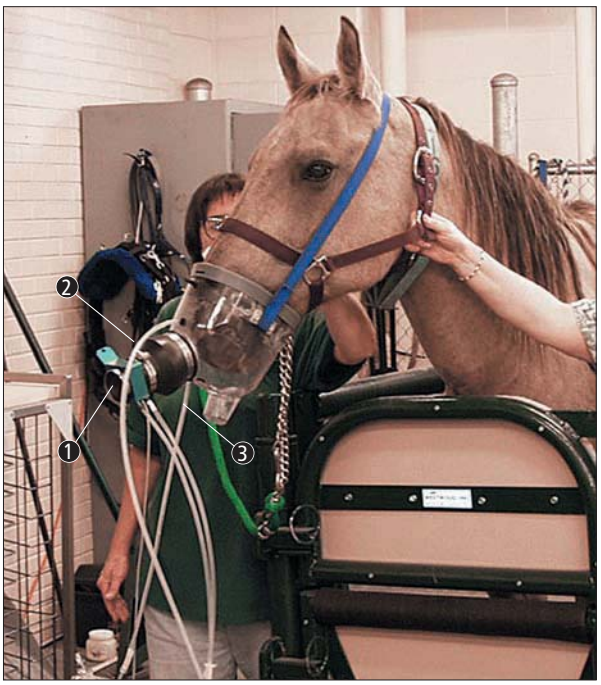
$\dot{V}/\dot{Q}$ inequality is characterized by a mismatch of alveolar blood flow and ventilation. This mechanism is the most common cause of hypoxemia in obstructive and restrictive diseases. Respiratory diseases with high $\dot{V}/\dot{Q}$ ratios are usually caused by decreased lung perfusion (e.g. shock, pulmonary thromboembolism). Diseases with low $\dot{V}/\dot{Q}$ ratios are commonly associated with decreased ventilation, such as obstructive lung diseases (e.g. heaves) and pulmonary atelectasis and consolidation.

Shunt is one extreme of the $\dot{V}/\dot{Q}$ inequality spectrum ($\dot{V}/\dot{Q}$ = 0) where some blood reaches the pulmonary veins without passing through ventilated areas of the lung. Intrapulmonary causes of shunt are pulmonary consolidation, atelectasis, and arterial–venous fistulas. Extrapulmonary shunts are commonly associated with congenital cardiac diseases (e.g. patent ductus arteriosus, atrial and ventricular septal defects).

Diffusion limitation means that P_{aO_2} in the alveoli and capillaries does not reach equilibrium. Diseases can result in diffusion limition according to two main mechanisms: (1) thickening of the blood–gas barrier causing diffusion of oxygen to be slowed down (e.g. pulmonary interstitial fibrosis, pulmonary edema), (2) a decreased surface area available for gas exchange (e.g. emphysema). In both cases, exercise will result in worsening of the hypoxemia. Hypoxemia may be readily reversed by administration of 100% oxygen in cases of hypoventilation, $\dot{V}/\dot{Q}$ inequality, and diffusion impairment. In patients with shunt, the increase in P_{aO_2} in response to 100% oxygen is marginal.

Lung mechanics

The principal function of the respiratory tract is to generate sufficient ventilation to maintain blood gas values in the physiologic range. Adequate pulmonary function can therefore be assessed by measuring factors affecting ventilation and gas exchanges. The former is influenced by the mechanical properties of the lung such as lung volume, airway patency, and lung elasticity. The latter is ultimately determined by the measurement of arterial blood gases (i.e. P_{aO_2} and P_{aCO_2}) and depends on adequate ventilation, appropriate matching of ventilation with lung perfusion, and sufficient diffusion capacity.



▲ **109** **Measurement of lung mechanics during tidal breathing from a pneumotachograph (1) and pressure recordings. (2) mask pressure catheter; (3) esophageal pressure catheter.**

Dynamic lung mechanics

Three methods have been used clinically in unsedated horses to measure mechanical behavior of the respiratory system during spontaneous breathing: esophageal catheter pressure technique (standard lung mechanics), forced oscillation, and plethysmography. These tests may be conducted either during tidal breathing at rest, during increased ventilation (e.g. exercise, lobeline-induced hyperventilation), or during inhalation challenge (bronchoprovocation). They are performed only at specialized referral centers or in research laboratories, except for one plethysmographic technique called 'open plethysmography', which can be performed in the field.

Standard lung mechanics

The principle of the technique is to use a balloon catheter positioned in the thoracic portion of the esophagus to estimate pleural pressure and a face mask with a pneumotachograph to measure airflow at the airway opening (**109**). The catheter inserted into the esophagus via the nasal passages must be sufficiently stiff to remain unchanged in diameter during breathing or swallowing. The measuring tip of the catheter has to be positioned inside the distal third of the thoracic portion of the esophagus in order to minimize cardiac artifacts and maximize the signal to noise ratio. With the appropriate technique, measurements are reproducible and equivalent to lung mechanics data collected using direct pleural pressure measurements. Selection of the pneumotachograph (e.g. Fleisch, ultrasonic, Pitot tubes) is aimed at minimizing the inherent resistance of the instrument compared to pulmonary resistance (R_L), which is especially important during exercise.

According to the simplified equation of motion (below), the difference between airway opening (mask) and esophageal pressure generated by respiratory muscles during tidal breathing is balanced by total R_L and dynamic lung compliance (C_{dyn}):

$$\Delta P_L = (1/C_{dyn})\, V + R_L \dot{V}$$

The graphical method of Amdur and Mead may be used to measure R_L and C_{dyn}. This method separates the elastic and resistive components by measuring C_{dyn} at points of zero flow and R_L at points of equal volume (usually 50% V_T). The difference between end-inspiratory and end-expiratory volume divided by the difference in P_L at the same points is equal to C_{dyn}. The difference between P_L measured at 50% V_T during inspiration and expiration divided by the corresponding airflow ($\dot{V}$) is equal to R_L. Typically, a minimum of 10 breaths without artifacts (e.g. swallowing) are measured and the results averaged. More commonly, derived parameters such as R_L and C_{dyn} are automatically calculated by dedicated software (e.g. Buxco).

The R_L is the sum of flow resistance through the airways (R_{aw}) and lung tissue resistance (R_{ti}); however, the latter represents only a small portion (≈10%). Pulmonary fibrosis and other conditions where the quantity of interstitial lung tissue is increased are usually associated with increased R_{ti}. Since the bulk flow rate diminishes in smaller airways and flow becomes progressively less turbulent, R_L largely represents resistive work in the larger airways. As a result, R_L is relatively insensitive to airflow obstruction in small airways and horses with RAO will exhibit abnormal R_L only when the degree of airway obstruction is marked and they are already showing clinical signs of the disease, such as increased breathing effort.

Forced oscillatory mechanics

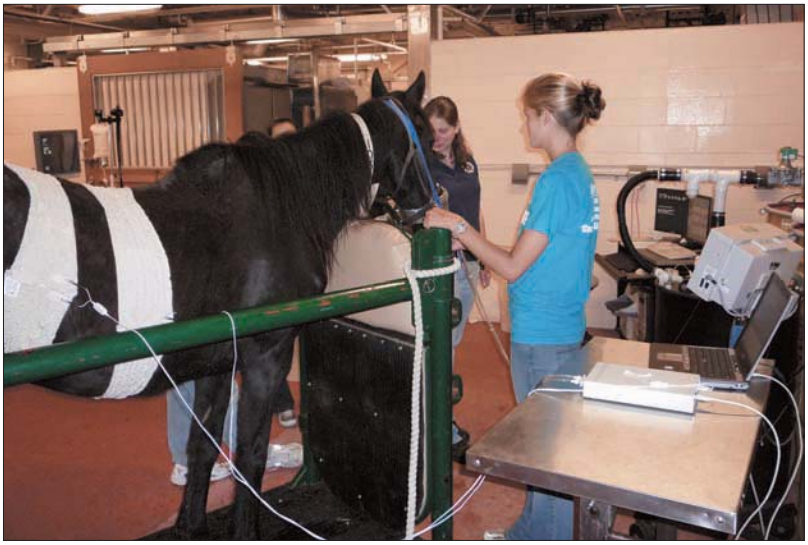
Various non-invasive methods for evaluating the dynamic behavior of the lung have been developed in horses, including forced oscillatory mechanics (FOM). The advantage of FOM is the measurement of the frequency dependence of respiratory system resistance; this is relevant to IAD and RAO, diseases where ventilation is uneven throughout the lung and is detected by frequency dependence of resistance. FOM uses an oscillating pressure or flow signal – superimposed on tidal breathing – that is generated by a pump or loudspeaker, connected to a face mask. The resulting pressure, volume, and flow perturbations are measured and impedance of the system (Zrs) calculated. The frequency of oscillation is set to exceed the breathing frequency and the oscillating pressure changes are small (usually <1–2 cmH_2O). A single frequency may be used for measurement of Zrs. However, using a spectrum of frequencies may better characterize lung mechanical properties, in particular differentiating properties of central airways from peripheral airways. In the horse, two types of multiple-frequency FOM methods have been used: monosinusoidal oscillations and impulse oscillometry.

Plethysmography

In humans, plethysmography is mainly performed with the patient seated in a whole-body box, called a plethysmograph, while breathing through a pneumotachograph back into the box. The advantage of this method is direct measurement of R_{aw} without including the lung tissue component that is measured during standard lung mechanics. This method is impractical in the horse, although it has been performed experimentally. Other approaches have been developed using less bulky and less expensive equipment. The main principle of these methods is to measure the difference in phase or magnitude between signals obtained from changes in thoracic volume, and signals from airflow at the mouth during tidal breathing. With increasing resistance to flow, there is an increasing difference between the two signals due to thoracic gas compression. Two techniques have been used in horses: impedance plethysmography and inductance plethysmography.

Impedance plethysmography is based on the fact that variation in electrical impedance between electrodes placed on opposite sides of the chest is proportional to changes in thoracic volume. It has not been used clinically to detect airway obstruction in horses.

Inductance plethysmography quantifies external movements of the chest wall by placing elastic belts around the thorax and abdomen. The belts contain inductance coils, which generate an oscillatory current when stretched. The output signal of each belt is proportional to the volume change of each compartment.



◀ 110 Inductance plethysmography using the commercially available Open Pleth™.

Tidal motion of the lungs and chest wall against the diaphragmatic and abdominal muscles can be measured by adding the volume displacements of the rib cage and abdomen to obtain V_T. In the presence of airway obstruction, gas compression within the airways may result in asynchrony between thoracic and abdominal contractions. In adult horses, differences between flow measured by pneumotachograph and inductance plethysmography are significantly correlated with R_L or C_{dyn} both during histamine challenge and in horses with RAO.

The distinct advantage of inductance plethysmography is its non-invasive nature, satisfactory correlation with standard lung mechanics, and the fact that it is commercially available for field testing (**110**).

Airway responsiveness

Airway hyperresponsiveness (AHR) is used to describe an increased tendency of airways to constrict in response to irritant stimuli. The stimulus may be specific (e.g. an allergen) or non-specific (e.g. histamine or methacholine). It is known that human asthmatics demonstrate AHR when exposed to aerosolized chemical mediators such as histamine. Similarly, AHR is detected in ponies and horses with RAO during exposure to moldy hay and in most horses with IAD. Most RAO horses will lose their AHR during periods of disease remission when they are asymptomatic. A few RAO-affected horses in remission may still have some increased response to irritant challenge, although of a lesser degree than during disease exacerbation.

The mechanism of AHR is still uncertain; however, several factors appear to play a determining role: airway wall thickening, airway smooth muscle contractile properties, and airway inflammation. Increased thickness of the airway wall, in particular the basement membrane, subepithelial layer, and smooth muscle, has been reported in human asthmatics. Evidence of airway wall and smooth muscle thickening is also observed in horses with RAO and some with IAD. Because airway resistance is inversely proportional to the internal radius of the airway to the fourth power, the same degree of smooth muscle contraction in a thickened airway wall will result in an enhanced narrowing in response to bronchoconstrictor stimuli compared with normal airways.

Forced expiration

Parameters measured during a maximal expiratory effort, or forced expiration (FE), constitute a special case of assessing dynamic lung mechanics that allows detection of airway obstruction with greater sensitivity and descriptive value than measurements during tidal breathing. It is the most commonly used test of lung function in humans, mainly because patients are easily trained to perform FE and it provides an early indication of lung disease. It is also more reproducible than lung function tests during tidal breathing. The maneuver requires the patient to inhale to TLC and immediately exhale as hard and completely as possible to RV. The relationships between flow, volume, and time are measured during FE. The most commonly measured parameters in humans are forced expiratory volume in 1 second (FEV_1) and forced vital capacity (FVC). The patient's cooperation and proper coaching by trained personnel are essential to the accuracy of the test.

A minimally invasive forced expiration method has been developed in the horse. The method induces FE in a standing, sedated horse by connecting the animal's airways to a vacuum reservoir (**111**). Forced expiratory flow (FEF) and volume are measured indirectly by computing changes in vacuum pressure. Prior to performing the FVC maneuver, the lungs are inflated progressively until the pressure–volume curve reaches a plateau, which correspond to P_L values around 25–30 cmH_2O. The volume of the negative pressure reservoir should be sufficient to avoid substantial change in driving pressure during FE. The advantage of this method is the ability to detect a small amount of peripheral airway obstruction in horses with mild RAO or IAD. Residual airway obstruction may even be detected by FE in horses with RAO that have been in clinical remission (i.e. asymptomatic) for months to years when other methods indicate normal measurement of lung mechanics.

▲ **111** **Forced expiration maneuver being performed in a horse. (1) vacuum tanks; (2) nasotracheal tube; (3) esophageal pressure catheter; (4) positive pressure tank; (5) mechanical ventilator.**

ALLERGY TESTS

Two types of allergy test are currently available: intradermal skin testing and in-vitro allergy tests. Serum based allergy tests (ELISA) measure allergen-specific IgE or sulfidoleukotrienes released from peripheral blood leukocytes. These tests have proved useful for skin allergy (insect bite hypersensitivity), but not for respiratory diseases. Immunotherapy based on allergy testing has not been shown scientifically to be effective for the treatment of respiratory diseases. Anecdotal reports of efficacy have always been accompanied by concomitant environmental changes.

AEROSOL THERAPY

Infectious and inflammatory respiratory diseases are common causes of morbidity and decreased performance in horses. Antimicrobials, corticosteroids, and bronchodilator drugs, administered systemically, are effective treatment options for these respiratory diseases. However, adverse effects represent potential complications of systemic therapy (e.g. diarrhea and renal toxicity in the case of antimicrobials; laminitis and immunosuppression in the case of corticosteroids; and excitement, sweating, and tachycardia in the case of bronchodilators). Treatment of pulmonary disease by administration of therapeutic substances directly into the respiratory tract allows a reduction in the total dose used while delivering a high concentration of the drug directly into the lungs and decreasing the amount absorbed systemically. Lower systemic drug levels reduce the risk of side-effects and shorten drug elimination times, thereby optimizing the drug benefit to risk ratio.

Principles of aerosol therapy

The clinical response to aerosol medication is a function of the dose deposited in the airways, which is dependent on the type of delivery device, the particle size characteristics of the inhaled aerosol, the pattern of breathing, and the type of airway disease. Deposition of therapeutic aerosols within the respiratory tract occurs mainly by inertial impaction and gravitational sedimentation. Inertial impaction is largely responsible for particle deposition in proximal airways such as the nasal passages, nasopharynx, and central airways. Impaction of aerosolized particles on airway walls can occur if their size is sufficiently large ($\geq$1 µm) or the air stream is rapidly changing direction because of branching airways or turbulent flow, for example. Smaller particles ($\geq$0.5 µm) that are able to reach the peripheral airways and alveoli may deposit on the airway surface by gravitational sedimentation when the air stream is sufficiently slow.

Therapeutic aerosols are composed of particles of different sizes. Particle size distribution of an aerosol is usually described by its mass median aerodynamic diameter (MMAD). By definition, 50% of the aerosol mass is composed of particles smaller than the MMAD and half is composed of particles larger than the MMAD. Particles >5 µm are mainly deposited in the upper airways, while the majority of particles <1 µm are exhaled. The deposition of particles in small conducting airways and alveoli is maximal when the aerosol MMAD is between 1 and 5 µm.

Another important factor for aerosol deposition is the speed of inhalation. As inspiratory flow rate or breathing frequency increases, more particles get deposited in the upper airways. Penetration of aerosol into peripheral airways is improved when the inhaled volume increases. In humans, slow, steady inhalation followed by a period of breath holding is recommended to maximize lung deposition. Obviously, manipulation of the breathing pattern is limited in the horse. Placement of the face mask used to deliver the aerosol, or the noise made by administration of a puff of aerosol, may make horses anxious. Nervous horses may hold their breath or adopt a shallow breathing strategy during aerosol therapy. Therefore, it is important to get horses used to the treatment protocol by using positive reinforcement (e.g. food reward) and potential masking noises (e.g. turning on a radio).

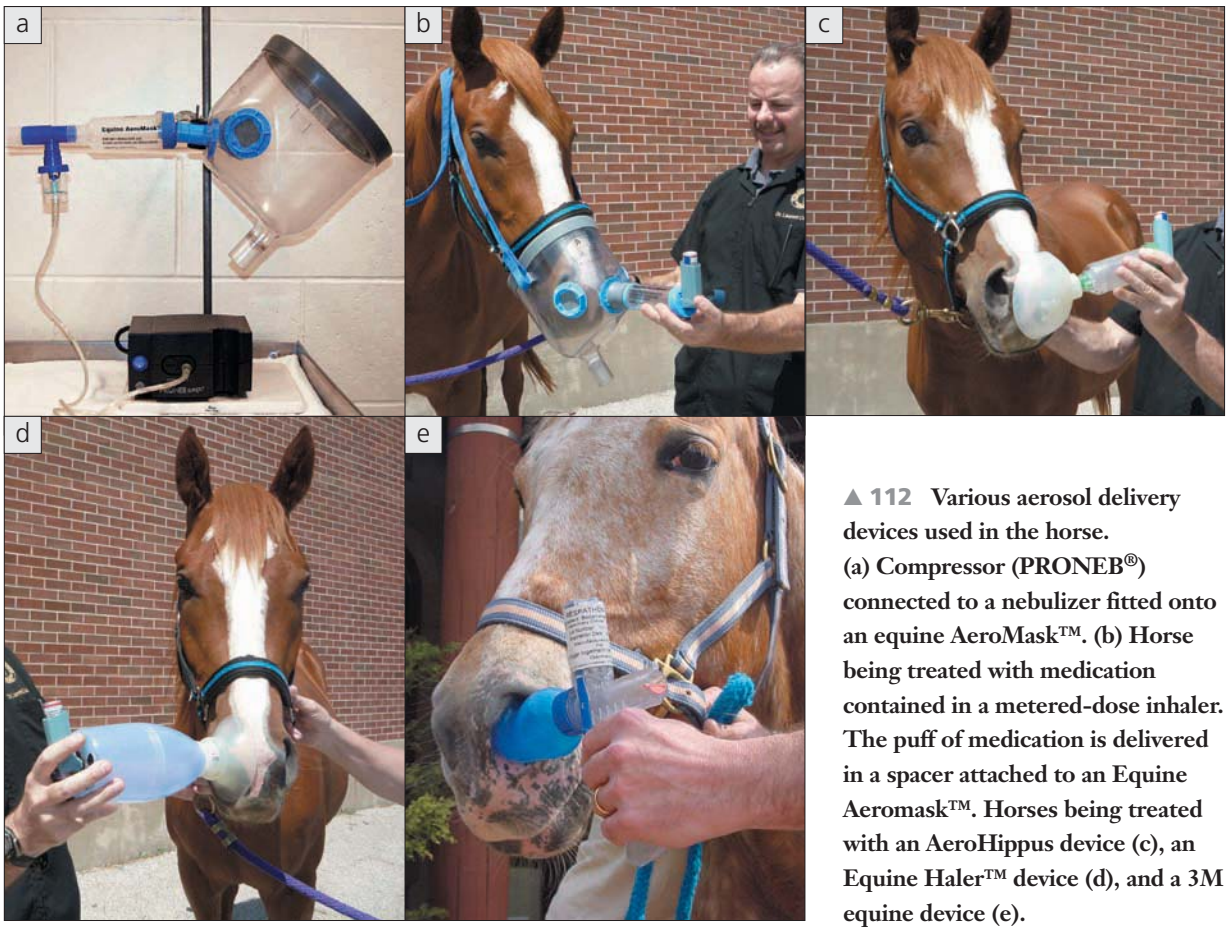
Airway narrowing increases aerosol deposition in the central airways and results in poor deposition in the peripheral airways. Administration of a bronchodilator results in rapid improvement in peripheral airway deposition in horses with bronchoconstriction associated with diseases such as RAO. Therefore, corticosteroid therapy for the treatment of RAO should be preceded by bronchodilation in order to maximize its anti-inflammatory effectiveness.

Aerosol delivery devices

Therapeutic aerosols may be produced by nebulizing a solution or administrating aerosols prepackaged in pressurized metered-dose inhalers (pMDI). Drugs available to treat respiratory diseases using pMDI are limited mainly to corticosteroids and bronchodilators, whereas any aqueous solution may be delivered with a nebulizer (e.g. water soluble antimicrobials).

Masks, spacers, and holding chambers

Several types of devices, such as a face mask, nosepiece, and extension tubing (spacer or holding chamber), are used to improve delivery of aerosol to the horse's lung. Data are available concerning lung deposition of drugs administered using commercially available masks in horses. The amount of drug delivered by a pMDI that reaches the lung varies depending on the mask system used (6.1%, AeroMask™; 8.2%, Equine Haler™; 18.2%, AeroHippus™; 23.3%, 3M equine device) (**112**). These data indicate that the drug dose required to treat a horse with the 3M equine device is approximately a fourth of the dose required with the AeroMask™ and a third of the dose needed with the Equine Haler™. However, the 3M equine device is no longer commercially available. Another important factor affecting deposition of aerosol in the lung is the type of propellant used in the pMDI.



▲ 112 Various aerosol delivery devices used in the horse. (a) Compressor (PRONEB®) connected to a nebulizer fitted onto an equine AeroMask™. (b) Horse being treated with medication contained in a metered-dose inhaler. The puff of medication is delivered in a spacer attached to an Equine Aeromask™. Horses being treated with an AeroHippus device (c), an Equine Haler™ device (d), and a 3M equine device (e).

Spacers and holding chambers are designed to alter the size distribution of particles originating from the pMDI or nebulizer, resulting in a reduction in upper airway deposition and an increase in the mass of drug contained in respirable particles. A valve is usually present between the spacer and the horse's nostril, and so precise synchronization between pMDI actuation and the onset of inhalation is not required. However, the presence of a one-way valve between the nebulizer and the horse may result in as much as a 50% reduction in aerosol delivery. Therefore, it is recommended that if a one-way valve is present, it is removed when nebulizing a drug.

▲ **113** **Face mask with built-in ultrasonic nebulizer (SaHoMa™) being used to deliver aerosol therapy.**

Nebulizers

Two main types of nebulizer are commercially available to generate therapeutic aerosols. A solution may be aerosolized by atomization of liquid (jet nebulizer; **112**a) or vibration of liquid (ultrasonic nebulizer; **113**). The rate of nebulization is usually higher with ultrasonic than with jet nebulizers. Lung deposition depends on particle size distribution, which is mainly influenced by the nebulizer type and design. In general, jet nebulizer particle size is inversely related to gas flow rate. With ultrasonic nebulizers, particle size is a function of the wavelength produced by the device. As a result, the fraction of drug deposited into the lungs varies significantly between different types of nebulizers, with a range of 0.3–7.4%.

Pressurized metered-dose inhalers

The type of propellant used in a pMDI is an important factor affecting lung deposition. Until recently, most propellants used were chlorofluorocarbons (CFCs); however, their use was progressively phased out because of CFCs' contribution to the depletion of the atmospheric ozone layer and they were eventually prohibited worldwide by the end of 2008. CFCs have since been replaced by another type of propellant called hydrofluoroalkanes (HFA). Some of the drugs have been reformulated as a solution with HFA instead of a suspension (e.g. flunisolide, beclomethasone), resulting in a significantly reduced MMAD and improved lung deposition. Consequently, the dose recommendations for beclomethasone HFA in people with asthma are approximately half of the dose with beclomethasone CFC. This improvement in lung deposition has not been observed for drugs that remained as suspension in HFA, such as fluticasone or triamcinolone. The problem is that most equine studies published used pMDI with CFC, except for one study with albuterol HFA. The particle size characteristics of HFA-pMDI suggest that improvement in lung deposition would be expected in horses as it was demonstrated in humans and, therefore, lower drug doses would be required to achieve a similar therapeutic effect. In this context, lung deposition reported for AeroHippus™ (18.2%) was achieved using beclomethasone HFA, while percentages of lung deposition using AeroMask™ (6.1%) and Equine Haler™ (8.2%) were obtained with fenoterol CFC and fluticasone HFA, respectively.

Aerosolization of antimicrobials

Effective treatment of respiratory infections depends on achieving a therapeutic concentration of antimicrobial in the lung tissue and airways. Many antimicrobials reach relatively low concentrations in epithelial lining fluid when administered systemically, and often high doses of IV drugs are required for therapeutic efficacy. Antimicrobial administration by nebulization results in high drug levels in the airways and is particularly effective in humans with pulmonary infections associated with chronic respiratory diseases such as cystic fibrosis and bronchiectasis. However, many factors, such as intracellular pathogens and thick respiratory secretions, can potentially impair antimicrobial efficacy.

Factors affecting antimicrobial efficacy

The physicochemical properties of antimicrobials and drug concentration may affect nebulizer output and MMAD. Solutions with higher drug concentrations are associated with increased viscosity and decreased nebulizer output. In general, maximum output is achieved with an antimicrobial concentration at 50 mg/ml with ultrasonic nebulizers and 100 mg/ml with jet nebulizers. In particular, the optimal concentration of antimicrobial with an ultrasonic nebulizer is 50 mg/ml for gentamicin diluted with sterile water or 0.9% saline and 25 mg/ml for ceftiofur sodium diluted with sterile water. Nebulization of a solution with an extreme pH (<3 or >9) or osmolality (<100 mOsm/kg or >1,100 mOsm/kg) can cause side-effects such as coughing and bronchoconstriction. The physicochemical properties of such a solution should be adjusted prior to nebulization, although this may cause precipitation. Otherwise, prior administration of a bronchodilator would help prevent excessive coughing and bronchoconstriction.

Pulmonary infections are associated with excess airway mucus, bronchoconstriction, and bronchial edema, which result in airway obstruction. Obstructed airways receive decreased to no ventilation and, therefore, little to no aerosolized medication will reach the affected airways. Once the drug is deposited into the airways, it may be antagonized by mucus components that impair antimicrobial penetration into bacteria or bind certain drugs such as aminoglycosides. Ultimately, dissolved drug molecules can diffuse through the mucus layer and into the respiratory epithelium where their antimicrobial properties are needed to help clear infection. Some of the drug will reach the pulmonary circulation and be metabolized as if administered IV and some will be transported to the pharynx by mucociliary clearance, swallowed, absorbed, and metabolized as if administered orally.

Aerosolized antimicrobials in animals

In calves with experimentally induced *Pasteurella haemolytica* bronchopneumonia, once a day treatment with sodium ceftiofur (1 mg/kg) for 3 days has been proven to be more effective when administered by nebulization rather than by intramuscular (IM) injection. Currently, there is no published study on the efficacy of aerosolized antimicrobials in horses with infectious respiratory disease. However, nebulization of injectable gentamicin solution (2.2 mg/kg diluted with sterile water to obtain a concentration of 50 mg/ml) in healthy horses results in epithelial lining fluid levels 12 times higher than those recorded after IV administration of the drug (6.6 mg/kg). No side-effects have been reported despite the presence of preservatives and the acidic pH of the solution. Similarly, nebulization of marbofloxacin (300 mg as a 25 mg/ml solution) is not associated with adverse effects or decrement in lung function in healthy horses.

Horses with clinical bronchopneumonia or pleuropneumonia present with variable degrees of lung consolidation, airway obstruction, and parenchymal pathology, which would impair lung deposition of aerosolized antimicrobials and distribution in affected areas. Therefore, nebulization of antimicrobials in clinical cases should not be used alone, but in addition to systemic antimicrobial therapy, particularly in cases of aspiration pneumonia.

CHAPTER 5

THE COUGHING HORSE

INTRODUCTION

Definition

Coughing is a defense mechanism that helps prevent or limit inhalation of foreign materials into the tracheobronchial tree and contributes to clearance of the airways. Coughing is a reflex elicited by stimulation of receptors located from larynx to bronchi.

Characteristics

The cough is composed of four consecutive phases: inspiration, compression, expiration, and cessation. Coughing is preceded by inspiration of a variable volume, followed by glottis closure and forceful expiratory effort, which raise thoracoabdominal pressures >100 cmH_2O above ambient pressure. Inhalation of foreign material through the larynx may trigger an immediate cough without prior inspiration to prevent further entry into the tracheobronchial tree. Clearance of secretions may be achieved by forced expirations without glottis closure. However, thoracic gas compression against a closed glottis is thought to augment cough effectiveness.

High linear velocities achieved by air flow cause shearing forces at the level of the airway walls and result in the suspension and clearing of secretions adherent to the airways. Higher flow velocities are achieved in large airways such as the trachea and major bronchi. Therefore, coughing is likely to help clear materials from central airways (trachea and large bronchi), but not from peripheral or small airways. Because the extrathoracic trachea does not collapse during coughing, it is not cleared effectively during coughing. In the horse, maintaining the head in a low position, such as when eating from the ground, appears to be an important clearance mechanism for secretions. For example, horses confined with their heads elevated for up to 12 hours experience bacterial colonization of the tracheobronchial tree, despite development of a cough. Clinical signs resolve within 12 hours if horses are allowed to lower their heads.

Elimination of normal respiratory secretions appears to depend mainly on mucociliary clearance mechanisms and not cough.

Pathophysiology

Stimulation of irritant receptors located in the nasal passages produces sneezing. Coughing is produced by stimulation of receptors located in the larynx and airways distal to it. The cough reflex is initiated by activation of nerve endings called irritant receptors that send impulses through myelinated vagal fibers to the cough center in the medulla of the brain and in return cause coughing, contraction of the trachea and bronchi, and mucus secretion. The cough reflex is not blocked with bronchodilators. Although bronchoconstriction accompanies coughing, it can be produced individually and separated pharmacologically, indicating that each response is a different reflex. Irritant receptors are located between airway epithelial cells from the larynx to the respiratory bronchioles. They are particularly abundant in the trachea, carina, and large bronchi, which explains the cough response during bronchoscopic examination.

Irritant receptors are stimulated by both mechanical and chemical stimuli. Mechanical stimulation of the respiratory epithelium may result from accumulation of secretions, inhalation of particulate matter, or bronchoconstriction. In the horse, irritant receptors seem to be less sensitive to mechanical stimuli than in other species because the presence of large amounts of mucopurulent exudate in central airways, as seen in

horses with RAO, is not necessarily accompanied by a cough. Chemical stimulation with various endogenous (histamine, prostaglandins) and exogenous (dusts, noxious gases) substances can also stimulate irritant receptors and trigger a cough.

C-fiber receptors ('J receptors'), another type of receptor, are located in the alveolar walls in close association with the pulmonary capillaries and may cause coughing indirectly by releasing neurotransmitters such as tachykinins which, in turn, stimulate irritant receptors in the airways. These receptors may play a role in the mechanism causing increased cough sensitivity in inflamed airways.

Because coughing may result from a combination of pathways, it is important to understand those mechanisms in order to design an appropriate treatment plan. For example, administration of bronchodilators is expected to help reduce coughing in respiratory diseases characterized by significant bronchoconstriction, such as RAO. On the other hand, coughing secondary to equine viral respiratory infections is due to both airway inflammation and decreased mucus clearance. The loss of epithelial integrity seen after respiratory infections may lead to increased sensitivity of irritant receptors and result in increased bronchial responsiveness and coughing. Left-sided heart failure is rare in the horse; however, when it does occur, clinical signs of respiratory disease such as coughing are common. In this case, coughing results from mechanical stimulation of receptors secondary to pulmonary edema.

Coughing is an important defense mechanism of the tracheobronchial tree; however, in some instances it does not have a useful purpose and may even be deleterious to the horse (e.g. rib fracture, pneumothorax). Chronic cough can contribute to fatigue and decrease food intake.

ACUTE COUGH

Viral respiratory infections

Definition/overview

Viral respiratory infection is considered the most common illness of horses >1 year of age in North America and Europe. The most important viruses that cause respiratory disease are equine influenza A/H3N8 (also known as equine 2) and equine herpesviruses type 1 (EHV-1) and type 4 (EHV-4). Equine influenza virus type A/H7N7 can cause disease in equids, but it has not been isolated since the late 1970s. Viruses that may cause or have been associated with respiratory disease on rare occasion are adenovirus, equine viral arteritis (EVA), and rhinovirus.

Affected horses are typically young (weanlings to 5 years of age) with a recent history of mixing with new horses (e.g. racetrack, show, sale barn) or exposure to various stressors (e.g. overcrowding, extreme climate conditions, concurrent disease). Clinical signs of infection include acute onset of lethargy, fever, serous to mucopurulent nasal discharge, submandibular lymphadenopathy, cough, and partial anorexia.

Etiology/pathophysiology

All equine influenza viruses are type A RNA viruses (Orthomyxovirus family), which are classified into different subtypes based on hemagglutinin (H) and neuraminidase (N) surface glycoproteins. Recent outbreaks of equine influenza have all been from type A/H3N8 virus. Infection with type A/H7N7 has not been identified in horses since 1979. Marked antigenic drift has been detected in H3N8 viruses isolated around the world. Australia, Iceland, and New Zealand are considered free of equine influenza. A worldwide surveillance system is in place to monitor equine influenza virus drift in order to recommend appropriate vaccine updates. Aquatic birds are believed to be a natural reservoir of influenza A viruses.

EHV-1 and EHV-4 are DNA viruses. Compared with equine influenza, EHV-1 and EHV-4 display little antigenic variability. The majority of respiratory diseases caused by EHVs are associated with EHV-4. EHV-1 is capable of inducing respiratory disease, but the most severe clinical manifestations of infection are abortion, neonatal foal death, and neurologic disease.

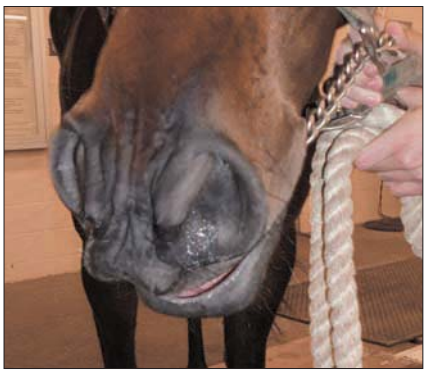
Severity of disease is influenced by the immune status of the population and environmental conditions. The incubation period is short in cases of experimental infection with equine influenza virus (1–3 days) and EHVs (2–5 days). The incubation period in cases of field infections is usually longer. Viruses gain access to the respiratory tract either directly via aerosolized infective droplets or indirectly through fomites (e.g. clothing, buckets, twitch, endoscope). Once in the URT, viruses replicate in epithelial cells, resulting in destruction of respiratory epithelium from the nasal passages to the tracheobronchial tree. Pulmonary defenses are compromised as a result of impaired mucociliary clearance (up to 1 month with influenza), alveolar macrophage dysfunction, and destruction of surfactant-producing alveolar type 2 pneumocytes. Viral replication in the URT with EHV-1 leads to infection of mononuclear leukocytes in draining lymph nodes, allowing their migration in the circulation and thus causing viremia. The latter results in widespread vasculitis, in particular in endometrial and central nervous system (CNS) blood vessels, ultimately leading to abortion or myeloencephalopathy. Mares infected with EHV-1 during late gestation may deliver a live infected foal that is weak at birth or shortly thereafter and will usually succumb to respiratory distress. A few cases of abortion and neurologic disease have been linked to EHV-4 infection.

Infection with these EHVs occurs early in life, with almost all horses >2 years of age having seroconverted for EHV-4, but fewer seroconverted to EHV-1. Up to 60% of infected horses will become latently infected within lymphoid or neural tissues. These horses are asymptomatic carriers of the virus, but can become reactivated on immunosuppression resulting from stress or glucocorticoid administration.

Clinical presentation

Clinical signs are usually more pronounced in naive animals and may be subtle in horses with partial immunity. Poor performance and a serous nasal discharge (**114**) may be noticed first. Clinical signs of respiratory viral infection include repeated and biphasic fever (39.2–41.7°C [102.5–107.0°F]), harsh dry cough (especially with influenza), mucopurulent nasal discharge due to secondary bacterial infection, submandibular lymph node enlargement, anorexia, and lethargy. Dependent edema may develop in the legs of horses with influenza virus infection. In young foals before weaning, EHV-4 may spread to the LRT resulting in bronchopneumonia. Abortion results from infection with EHV-1 via the respiratory tract during the last trimester of gestation. Neurologic disease associated with EHV-1 infection is rare (**115**), but affects horses of all ages and usually follows signs of respiratory infection or abortion.

The long-term sequelae of viral respiratory disease can be reduced performance, secondary bacterial bronchitis, rhinitis, sinusitis, GP infections, or pneumonia.



▲ **114 Serous nasal discharge in a horse with acute respiratory viral infection.**

▲ **115 Horse with the neurologic form of EHV-1 infection 'dog sitting', because of weakness and incoordination of the hindlimbs.**

Differential diagnosis

The following infectious agents should be considered in outbreaks of upper respiratory disease: influenza virus; herpesviruses 1 and 4; other viruses (adenovirus, rhinovirus, equine arteritis virus); *Streptococcus equi* subsp. *equi* (*S. equi*); *Streptococcus equi* subsp. *zooepidemicus* (*S. zooepidemicus*); *Streptococcus pneumoniae*.

Adenovirus seroconversion is widely distributed, but infection is not commonly associated with disease. Infection is usually asymptomatic in adults, whereas in foals there may be severe signs of respiratory disease. Adenovirus infection in foals with combined immunodeficiency syndrome results in 100% mortality.

Rhinovirus infection is localized to the nasal passages and nasopharynx. Clinical signs include pharyngitis, nasal discharge, fever, and lethargy; however, signs are minimal compared with influenza and herpesvirus infections.

EVA causes peripheral edema, ocular and nasal discharge, anorexia, depression, abortion, and, less frequently, coughing. Transmission occurs via the respiratory tract as well as via venereal spread. Infected stallions can carry and shed the virus for an indefinite period.

S. equi results in severe lymphadenitis with purulent exudate draining from the nose or ruptured lymph nodes. Coughing is not a prominent feature of the disease (see Chapter 6, p. 104).

S. zooepidemicus is most important as a secondary invader after viral infection or stress (e.g. transportation). Proximal airway infection (sinusitis, lymphadenitis, GP infection) may spread to distal airways, resulting in bronchopneumonia or pleuropneumonia.

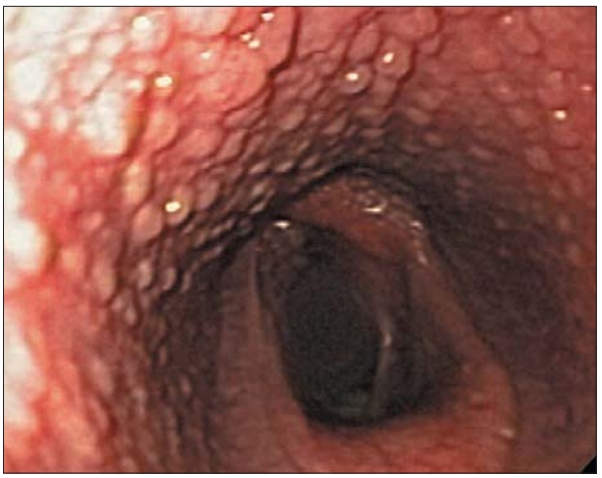
Diagnosis

Influenza infection spreads rapidly among unvaccinated animals. In these cases coughing is prominent. In immunized horses, the disease is clinically more difficult to differentiate from other contagious respiratory diseases. Because of the similarities between clinical diseases with the various respiratory viruses, isolation of virus from nasal secretions or a significant rise in specific antibody titer is required to confirm the diagnosis.

Viral isolation should be performed from nasal swabbing or from tracheal wash fluid collected within 48 hours of initial illness, when the horse is febrile, in order to improve the chances of success. However, the procedure is typically low yield, with virus isolated in <10% of affected horses. The nasal swab should be at least 15 cm long, be inserted into the ventral meatus, and advanced into the nasopharynx. The swab should be placed in a special transport medium containing antimicrobials and shipped on ice. If herpesvirus infection is suspected, blood should be collected (20–30 ml) within 4–10 days of respiratory illness and shipped on ice for virus isolation. Direct detection of influenza A antigen can be obtained rapidly using various commercially available test kits. Polymerase chain reaction (PCR) tests are more sensitive than direct antigen detection or virus isolation and are available at some commercial laboratories.

Serologic testing is more sensitive than virus isolation for diagnosing viral respiratory disease, but it requires collection of an acute sample and a convalescent sample 2–3 weeks later. An increase in virus-specific antibody titer of at least four-fold between the acute and convalescent samples is consistent with acute viral infection. Antibodies elicited from vaccination cannot be differentiated from natural infection. Therefore, detailed vaccination information is required to interpret the test results. Serologic diagnosis is retrospective in nature and is often not useful in the management of an outbreak. Detection of influenza antibodies with a single-radial hemolysis test is more sensitive and reliable than with a hemagglutination inhibition test.

Endoscopy of the respiratory tract may reveal hyperemic respiratory epithelium limited to the upper airways including the nasopharynx (**116**), trachea, and primary bronchi.



▲ 116 **Endoscopic view of the nasopharynx in a horse with severe pharyngitis secondary to respiratory viral infection. This horse shows a grade 3 out of 4 pharyngeal lymphoid hyperplasia.**

Management/treatment

The main goals of therapy are to provide stall rest, supportive care, and treatment of secondary complications. Strict stall rest for 1 week for each day the horse exhibited an elevated temperature is recommended. Exercise should not be resumed until 1 week after most of the signs have abated, except coughing, which may last as long as 1 month after disease onset.

Non-steroidal anti-inflammatory drugs (NSAIDs) are indicated to control high fevers and decrease excessive respiratory inflammation (flunixin meglumine, 1.1 mg/kg IV or IM q12h, or phenylbutazone, 2.2 mg/kg PO q12h). Monitoring water intake and hydration status is important.

Antimicrobials effective against *Streptococcus* spp. should be used in case of secondary bacterial infection (procaine penicillin G, 22,000 IU/kg IM q12h, or trimethoprim–sulfonamide, 15–30 mg/kg PO q12h). Foals or adult horses with bacterial pneumonia should be treated aggressively with IV antimicrobials (see Chapter 7, p. 144).

The antiviral drugs amantadine and rimantadine have been investigated for the treatment of influenza in horses. Both drugs are poorly bioavailable after oral administration and IV injection can be associated with neurotoxicity signs such as seizures and sometimes death. Oral rimantadine (30 mg/kg, q12h) reduced clinical signs of equine influenza in an experimental exposure study, with no evidence of toxicity. However, therapy with amantadine or rimantadine is limited by cost and resistance of influenza A virus to the drugs that may develop as quickly as 1 day after treatment initiation. Acyclic nucleoside analogues are another category of antiviral drugs that have been investigated for the treatment of EHV infection. The oral bioavailability of acyclovir is extremely poor (~3%), but frequent administration of high doses (20 mg/kg q8h) may result in effective serum concentration. Slow IV administration of acyclovir (10 mg/kg over 1 hour q12h) or as a constant rate infusion (CRI) may prove effective; however, cost will limit its use. A pro-drug for acyclovir, valacyclovir, is significantly more bioavailable in horses (26–60%). Pharmacokinetic studies suggest that a dose of 40 mg/kg q8h would be needed to provide effective plasma concentration. However, clinical studies showed a clinical response with lower dosages (27 mg/kg q8h for 2 days, then 18 mg/kg q12h). Therapy is likely to be more effective when started early in the infection process. Adverse side-effects have not been reported with acyclovir or valacyclovir oral administration. Therapy with ganciclovir (2.5 mg/kg q8h for 1 day, then 2.5 mg/kg q12h) may be considered in cases with herpes myeloencephalopathy.

Animals that are sick or exposed to infecton should be quarantined for 2–3 weeks. Environmental ventilation and general hygiene should be optimized.

Vaccination may be helpful in the face of an outbreak of viral respiratory disease, but it is important to remember that at least 7–10 days is required for a booster vaccine to be protective.

Prevention

Newly arrived horses should be isolated for 10–14 days then vaccinated. Vaccines so far developed to prevent viral respiratory diseases have shown limited efficacy and can only be expected to lessen disease severity and shorten virus shedding.

Influenza virus

A primary vaccination series should be given with three doses of the killed-virus vaccine administered 3–6 weeks apart with the third booster given 2–3 months after the second. Subsequent vaccinations should be given at 3–12 month intervals depending on age and risk level. Vaccinal immunity lasts no more than 4–6 months. For high-risk horses (e.g. racehorses, show and competition animals) adequate protection requires vaccination as often as every 3–4 months. If a dam was vaccinated within 1 month of foaling, vaccination of the foal should not be carried out before 8 months of age. A modified-live vaccine for intranasal administration is available in non-pregnant animals over 11 months of age and it has shown efficacy in challenge studies.

Herpesviruses

Inactivated or modified-live vaccines containing either EHV-1 or EHV-4, or both, are commercially available. Post-vaccinal immunity is short lived, therefore frequent booster administration is recommended for high-risk horses. A primary series should be given with two doses of the killed-virus vaccine administered 3–6 weeks apart and subsequent vaccinations at 2-month intervals depending on age and risk. Some clinicians recommend boosters as often as monthly; however, a minimum of 60 days between injections should be allowed to avoid local Arthus reaction. The anti-abortion vaccine performs better because it contains 4–5 times more antigens. Vaccination protocols have been developed specifically for pregnant mares and vary depending on the manufacturer. Foals should be vaccinated starting at 5 months of age.

Pneumonia

Definition/overview

Pneumonia is an inflammation of the lung parenchyma that may result from infection with bacteria, viruses, or fungi. The disease may affect any age group from neonatal foals to older horses. Various stress factors, such as transportation or competition, predispose horses to pneumonia. Respiratory viral infections are not the most common predisposing factors for bacterial pneumonia. Horses typically present with fever, cough, nasal discharge, anorexia, and lethargy.

Etiology/pathophysiology

Infectious agents include *S. zooepidemicus*, *S. pneumoniae*, *Rhodococcus equi* (in foals), *Bordetella bronchiseptica*, *Klebsiella* spp., *Pasteurella* spp., *Actinobacillus* spp., α-hemolytic streptococci, *Staphylococcus* spp., and *Escherichia coli*. The most common bacterial species identified is *S. zooepidemicus*; however, multiple species are frequently isolated in an affected horse. In rare cases, *Mycoplasma* spp. infection has been implicated. Bacterial pneumonia may also develop secondary to esophageal obstruction (**117**) or dysphagia (aspiration pneumonia), viral respiratory infection, or parasite migration.

Apart from *R. equi*, bacteria isolated from horses with pneumonia are opportunistic pathogens that are part of the normal flora of the proximal respiratory tract, or the upper gastrointestinal tract in cases of aspiration pneumonia. Mucociliary clearance and the immune system normally prevent pathogenic colonization of the distal respiratory tract. These protective mechanisms either become impaired or overwhelmed by factors such as stress of anesthesia, transportation, competition, racing, or comingling with large numbers of horses from different origins (e.g. at sales, shows, and breeding farms). In foals, overcrowding and insufficient immune response are additional risk factors for pneumonia. Tying a horse's head up during long-distance transportation is by itself sufficient to impair mucociliary clearance and allow bacterial colonization of the tracheobronchial tree by pharyngeal opportunistic pathogens. Providing transported horses with prolonged stops (e.g. 2 hours) on a frequent basis (e.g. every 4 hours) and allowing horses to drop their heads to ground level is advisable to reduce the risk of 'shipping fever' or pneumonia.

Clinical presentation

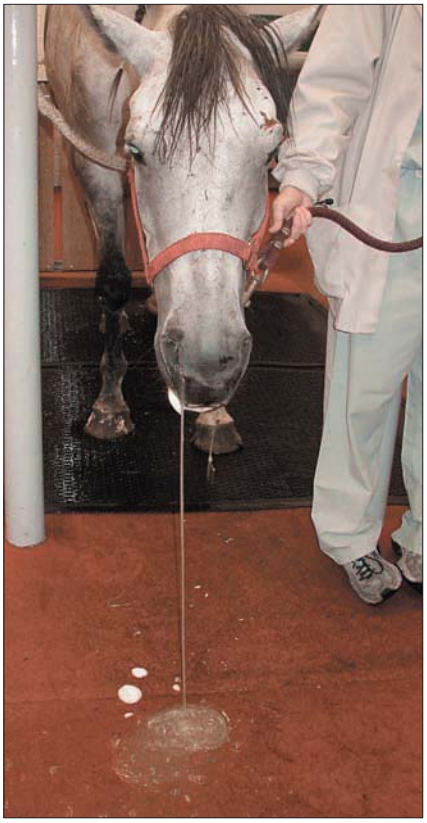
Fever, lethargy, anorexia, and a mucopurulent nasal discharge (**118**) are commonly observed in cases of pneumonia. Foals between 1 and 6 months of age are commonly affected by pneumonia. Foals with *R. equi* pneumonia usually present with intermittent fever; however, foals with other types of pneumonia may not present with a fever. Spontaneous coughing is often not reported in cases of pneumonia, but most foals cough following rebreathing maneuver or manual compression of the trachea. Wheezes and crackles may be heard in the cranioventral thorax during tidal breathing, but are more likely to be detected after a rebreathing test. Respiratory rate and pattern may be normal in early and mild cases, but increased effort or respiratory distress is evident in severe cases. Adult horses have a thicker chest wall, which makes breath sounds more difficult to hear. Thoracic auscultation after rebreathing maneuver is indicated in these cases, except in horses with labored breathing. Decreased breath sounds over the ventral thoracic region are suggestive of pleural effusion or lung consolidation. Auscultation of abnormal breath sounds confirms the presence of pulmonary disease; however, absence of abnormal breath sounds does not rule out pneumonia.

Differential diagnosis

Viral respiratory infections; interstitial pneumonia; parasitic pneumonitis; RAO; thoracic neoplasia.

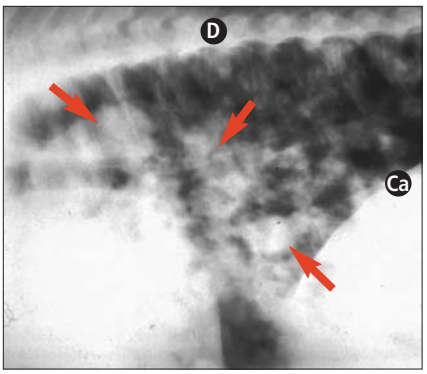
Diagnosis

A clinical presentation including fever, lethargy, anorexia, cough, tachypnea, or labored breathing is strongly suggestive of pneumonia. Detection of crackles or wheezes on thoracic auscultation confirms pulmonary disease and should be more evident during rebreathing examination. Hematology often indicates neutrophilic leukocytosis with elevated blood fibrinogen concentration in adult horses with pneumonia or in foals with *R. equi* infection. Tracheal wash cytology reveals neutrophilic inflammation with intra- or extracellular bacteria. Gram staining is helpful to characterize the type of infectious agent present and guide initial antimicrobial therapy. Bacterial culture and sensitivity should be performed, especially in foals in order to differentiate *R. equi* from other etiologic agents, and in adults with severe clinical signs to help adjust antimicrobial choice

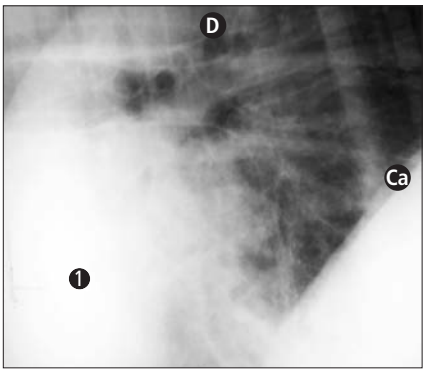


▲ **117** **Horse with esophageal obstruction and pneumonia displaying a characteristic nasal discharge mixed with saliva and feed material.**

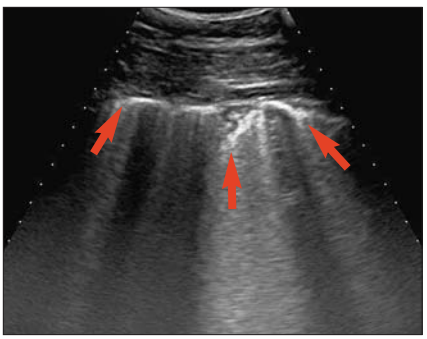
▲ **118** **Mucopurulent nasal discharge in a horse with bacterial pneumonia.**



▲ 119 **Nodular opacities (arrows) located throughout the lung parenchyma, consistent with pulmonary abscesses, in a foal with *R. equi* pneumonia.**



▲ 120 **Thoracic radiograph in a horse with aspiration pneumonia. The typical alveolar pattern in the cranioventral lung region obscuring the cardiac silhouette (1) is consistent with lung consolidation.**



▲ 121 **Lung consolidation detected by thoracic ultrasound of a horse with pneumonia. Note the irregular hyperechoic lung interface (arrows).**

in case of therapeutic failure. Thoracic radiography is helpful to differentiate pulmonary abscesses (**119**) or lung consolidation (**120**) from interstitial pneumonia; however, the technique is limited to foals when using portable equipment. Alternatively, thoracic ultrasonography is useful both in foals and adults to detect pleural effusion or peripheral lung consolidation or abscesses (**121**). The main disadvantage of ultrasonography is that lesions separated from the thoracic wall by aerated lung will not be detected.

Management/treatment

Affected foals or horses should be rested in a stall and therapy initiated with β-lactam (e.g. penicillin G, ceftiofur) or trimethoprim–sulfonamide antimicrobials (*Table 5*) targeted at *Streptococcus* spp. If tracheal cytology staining indicates the presence of gram-negative organisms, antimicrobial therapy should be complemented with an aminoglycoside (e.g. gentamicin) or fluoroquinolone (e.g. enrofloxacin). Alternatively, oxytetracycline has a broad spectrum of action against gram-negative and gram-positive aerobic and anaerobic bacteria and some *Mycoplasma* spp., though activity against Enterobacteriaceae (e.g. *E. coli*, *Enterobacter* spp., *Klebsiella* spp.) is variable. In cases of severe pneumonia, it is better to administer antimicrobials IV or IM to achieve the highest possible lung tissue and epithelial lining fluid drug levels. Therapy is continued until clinical signs resolve and at that point, antimicrobial therapy may be switched to an orally administered drug to provide coverage for at least 1–2 additional weeks depending on the initial disease severity.

The choice of antimicrobials for *R. equi* infection should not rely on susceptibility testing, because drugs effective *in vitro* may not be effective *in vivo* due to the intracellular nature of the bacterium. Macrolides are particularly successful for the treatment of *R. equi* pneumonia in foals, with the clarithromycin/rifampin combination being more effective than azithromycin/rifampin or erythromycin/rifampin (*Table 5*). Potential complications of macrolide therapy include colitis and hyperthermia. Mares of treated foals are at risk of colitis if they ingest antibiotic residues. Cleaning the foal's mouth or any contaminated area after treatment is therefore recommended in order to minimize the chances of exposure.

Administration of antimicrobials by nebulization results in high drug levels in the airways; however, studies demonstrating their efficacy in horses with pneumonia are lacking. Nebulization of injectable gentamicin solution in healthy horses (2.2 mg/kg diluted with sterile water to obtain a concentration of 50 mg/ml) results in epithelial lining fluid levels 12 times higher than those recorded after IV administration of the drug at 6.6 mg/kg. Similarly, nebulization of marbofloxacin (300 mg as a 25 mg/ml solution) achieves high drug concentration in airways and is not associated with adverse side-effects. In general, maximum aerosol output is achieved with an antimicrobial concentration of about 50 mg/ml for ultrasonic nebulizers and 100 mg/ml for jet nebulizers. Nebulization of solutions with extreme pH (<3 or >9) or osmolality (<100 mOsm/kg or >1,100 mOsm/kg) causes side-effects such as coughing and bronchoconstriction. The physicochemical properties of such solutions should be adjusted prior to nebulization, although this may cause precipitation. Otherwise, prior administration of a bronchodilator would help prevent excessive coughing and bronchoconstriction.

Adjunctive therapy includes oxygen supplementation if there is marked hypoxemia (P_{aO_2} <60 mmHg) or if labored breathing is noted, and administration of NSAIDs to control fever and inflammation. Keeping affected animals in a cool environment is helpful to maintain normal body temperature, but water and alcohol baths may be needed to control fever spikes.

Table 5 **Antimicrobials commonly used to treat horses with pneumonia**

DRUG	ROUTE	DOSE	INTERVAL
Azithromycin	PO	10 mg/kg	q24h first 5–7 days, then q48h
Ceftiofur	IV, IM	2.2–5.0 mg/kg	q12h
Chloramphenicol	PO	25–50 mg/kg	q6h
Clarithromycin	PO	7.5 mg/kg	q12h
Enrofloxacin	IV PO	5 mg/kg 7.5 mg/kg	q24h q24h
Erythromycin	PO	25 mg/kg	q6–8h
Gentamicin	IV	6.6 mg/kg	q24h
Metronidazole	PO Per rectum	15–25 mg/kg 25–35 mg	q8–12h q8–12h
Oxytetracycline	IV	5 mg/kg	q12h
Penicillin G, Na/K	IV	22,000 IU/kg	q6h
Penicillin G procaine	IM	22,000 IU/kg	q12h
Rifampin	PO	5–10 mg/kg	q12–24h
Trimethoprim–sulfonamide	PO	30 mg/kg	q12h

CHRONIC COUGH

Heaves (recurrent airway obstruction)

Definition/overview

RAO, or heaves, is an inflammatory lung disease characterized by chronic coughing, increased airway secretions, abnormal breath sounds, increased respiratory effort, and exercise intolerance. Horses affected with RAO experience exacerbation of clinical signs when exposed to organic dust originating from hay and bedding, in particular thermophilic molds present in moldy hay. As a result, clinical signs tend to be worse during the winter when horses are housed indoors for extended periods of time. Affected horses improve within 1 or 2 weeks after being placed on pasture with no access to hay or following reduction in airborne dusts and increased ventilation in the stall. Some horses are affected by summer RAO; they exhibit disease flare-ups while at pasture during summer months. These horses improve clinically during winter or after they are housed indoors. Finally, a small percentage of horses appear to suffer from both classic RAO and summer RAO. Horses with RAO tend to be mature (>7 years) to old animals and mares. In addition, some breeds (Thoroughbred, American Trotter, Morgan, Arab) tend to be predisposed to the disease. A genetic predisposition has also been identified in some families of horses. After horses first show clinical signs they are considered affected for life, although signs tend to wax and wane over time.

RAO used to be called chronic obstructive pulmonary disease (COPD); however, use of the term RAO, or heaves, is preferred since human COPD is fundamentally different in etiology, clinical presentation, and pathophysiology.

Etiology/pathophysiology

RAO is associated with exposure to high levels of organic molds, particularly abundant in moldy hay and poorly ventilated stables. Clinical signs may develop within hours to days of exposure. Even good quality hay contains molds that may trigger clinical signs in susceptible horses. Hay from round bales has been associated with a worse clinical presentation presumably because they are usually kept on the field where they are exposed to rain. However, small bales of hay harvested insufficiently dry and stored indoors will also lead to mold growth, which reaches a peak for hay baled at 35–50% moisture. Approximately 70 species of fungi and actinomycetes have been identified in hay dust and among them thermophilic molds such as *Aspergillus fumigatus*, *Faenia rectivirgula*, and *Thermoactinomyces vulgaris* are particularly abundant in moldy hay. These spores are small in diameter (<5 μm) allowing them to be inhaled deep into peripheral airways (respirable particles) where they may trigger an inflammatory reaction. The severity of the inflammatory response is dose dependent.

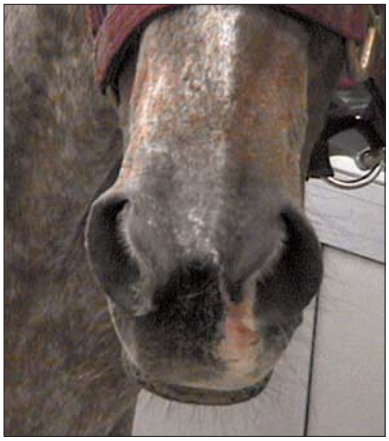
Endotoxins are present in large quantities in the environment horses inhabit and they potentiate the inflammatory response to inhaled molds. Outdoors, airborne pollen and molds appear to also play a role in summer RAO and classic RAO.

Clinical signs such as cough, increased respiratory effort, and mucopurulent secretions are the result of chronic neutrophilic airway inflammation, bronchoconstriction, and airway remodeling. In addition, peripheral airways of RAO-affected horses have an increased tendency to constrict excessively when exposed to both specific antigens (e.g. molds) and nonspecific irritants (e.g. histamine, endotoxin), a condition known as hyperresponsiveness. In many respects, RAO in horses is similar to severe asthma in people but not COPD.

The cascade of events leading to pulmonary dysfunction starts shortly after susceptible horses are exposed to allergens. Circulating neutrophils are recruited to the lungs within 4 hours of an allergen challenge and are detectable in airway mucus in 5 hours. Inflammatory cells release numerous inflammatory mediators; however, the complex relationship between effector cells, inflammatory mediators, and clinical signs is unclear. Airway remodeling is characterized by peribronchiolar lymphoplasmacytic, neutrophilic, and sometime eosinophilic cell infiltration, airway smooth muscle hyperplasia, bronchiolar goblet cell metaplasia, epithelial hyperplasia, and accumulation of mucopurulent exudate in the lumen of the bronchioles. Other lesions reported in severely affected horses are peribronchiolar fibrosis and epithelial metaplasia. Horses with summer RAO display histopathologic changes that are indistinguishable from RAO.

Emphysema is sometimes present in horses with RAO, but lesions are usually focal and do not appear to contribute significantly to lung dysfunction. Both centrilobular and panlobular forms have been reported. Lungs of affected horses tend to remain overinflated at postmortem examination because of air trapping secondary to airway obstruction, but not emphysema.

Airway smooth muscle tone is controlled by the autonomic nervous system and circulating levels of receptor agonists and antagonists. Healthy horses do not exhibit resting bronchomotor tone. Horses with RAO present with a degree of bronchoconstriction that is correlated with clinical severity. Bronchoconstriction is readily reversed by administration of cholinergic antagonists (e.g. atropine, glycopyrrolate, ipratropium bromide), beta-2 adrenergic agonists (albuterol, clenbuterol), and phosphodiesterase inhibitors (aminophylline).



▲ **122** **Nostril flaring in a horse in RAO crisis.**

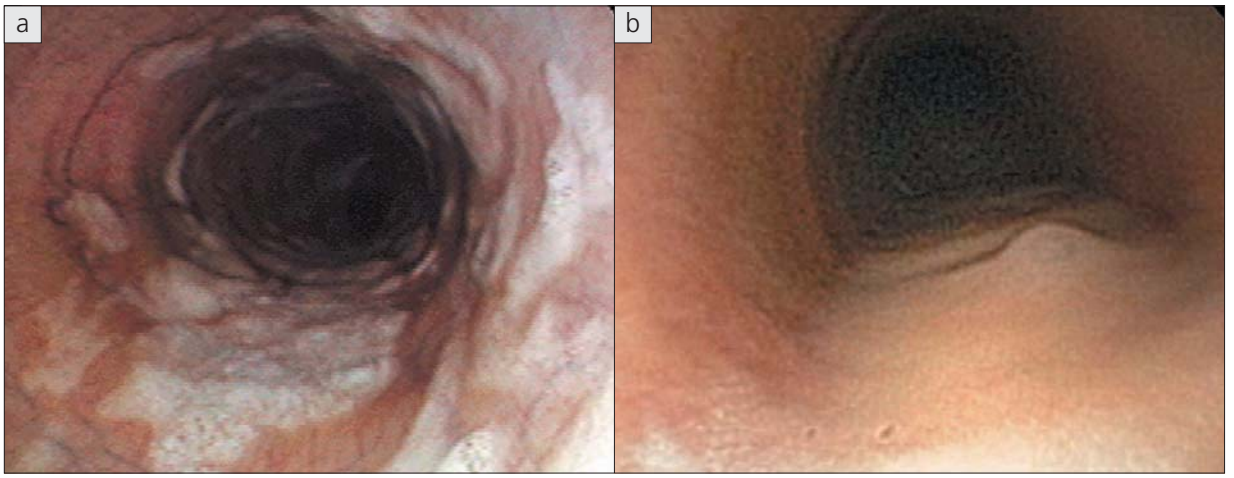
Clinical presentation

Horses with RAO present with a wide spectrum of clinical signs depending on disease severity. Horses with mild RAO may exhibit few or no clinical signs of respiratory disease except for exercise intolerance and may be difficult to differentiate from horses with IAD. At the other end of the spectrum, horses with severe RAO show lethargy and markedly increased respiratory effort manifested by nostril flaring (**122**), head and neck extension, pronounced abdominal muscle contraction during expiration (heaving), and exaggerated rib excursion during inspiration. Severe RAO commonly results in decreased appetite and weight loss. Rectal temperature is usually normal, but hyperthermia may occur in horses with severe RAO during warm weather because of increased energy expenditure or it may indicate fever due to infectious complications (e.g. secondary pneumonia). As disease progresses, hypertrophy of the external abdominal oblique muscles develops, resulting in the characteristic 'heave line' (see **45**). The respiratory rate is quite variable but is usually elevated. Clinical signs result from small airway obstruction secondary to airway inflammation, bronchospasm, mucus plugging of airways, and thickening of the airway wall.

Bilateral serous discharge is common; however, mucopurulent nasal discharge is rare despite large accumulations of mucopus in the trachea. A deep cough may be heard intermittently or in bouts of paroxysmal coughing. Thoracic auscultation may reveal increased breath sounds bilaterally, extended area of auscultation, and abnormal breath sounds (i.e. crackles, wheezes). Detection of breath sounds beyond the usual boundaries of thoracic auscultation indicates lung overinflation and is almost pathognomonic for RAO. However, the thick chest wall of horses makes auscultation an insensitive indicator of pulmonary disease, with abnormal findings obtained in less than 50% of horses with RAO.

Differential diagnosis

IAD; parasitic pneumonitis (lungworm); upper airway obstruction; bronchopneumonia; pleuropneumonia; pneumothorax; interstitial pneumonia; granulomatous pneumonia; thoracic neoplasia, synchronous diaphragmatic flutter; severe anemia.



▲ **123 Marked accumulation of mucopurulent discharge in the trachea of horses with RAO. (a) Mucus covering the tracheal surface circumferentially. (b) Mucus forming a large stream in the ventral trachea.**

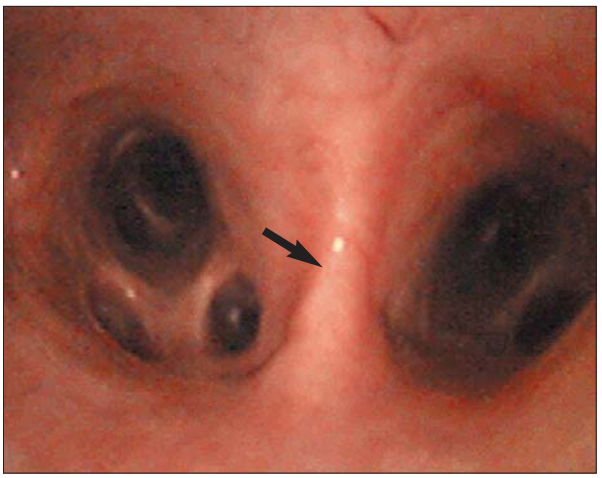
Diagnosis

A detailed history and thorough physical examination should indicate if RAO is a likely diagnosis in most cases. However, confirmation of a presumptive diagnosis often requires additional diagnostic tests. Hematology and serum biochemistry are usually within the normal range. Endoscopy of the respiratory tract is a valuable diagnostic tool. Horses with a normal respiratory tract have either no mucus or just a few flecks visible in the airways. The presence of increased respiratory secretions in the tracheobronchial tree is found in the majority of RAO horses (**123**); however it is also common in horses with IAD and infectious pulmonary diseases. Horses with severe pulmonary disease, including RAO, often exhibit marked airway erythema and bronchial edema illustrated by blunting of the carina (**124**) and 'bumpiness' of the airway surface. Nevertheless, many horses with severe RAO and large amounts of tracheal exudate do not have visible signs of tracheobronchial inflammation.

Collection of respiratory secretions using TW or BAL is important for diagnostic purposes and monitoring response to therapy. A description of these techniques and their indications are discussed in detail in Chapter 4. Administration of alpha-2 agonist sedatives (e.g. xylazine, detomidine) to horses with RAO prior to diagnostic procedures is considered safe. The more severe the airway obstruction the smaller the volume of fluid retrieved during BAL because of small airway collapse as fluid is aspirated back. However, as long as the volume of fluid infused is sufficient (250–500 ml) there is no significant difference in absolute or differential cell counts between aliquots retrieved sequentially. Therefore, interpretation of BAL fluid cytology in horses with RAO has diagnostic value even if the procedure only yields a small amount of fluid (e.g. 50–80 ml). Increased amounts of mucus containing casts of inspissated mucus originating from the bronchioles (Curschmann's spirals) are commonly present in long-standing cases (**125**). A marked absolute and relative neutrophilia (>20%) is usually observed in BAL fluid from horses with RAO and summer RAO (**126**). The range of neutrophil percentages is wide (10–98%), but no significant differences exist between samples collected from different regions of the lung.

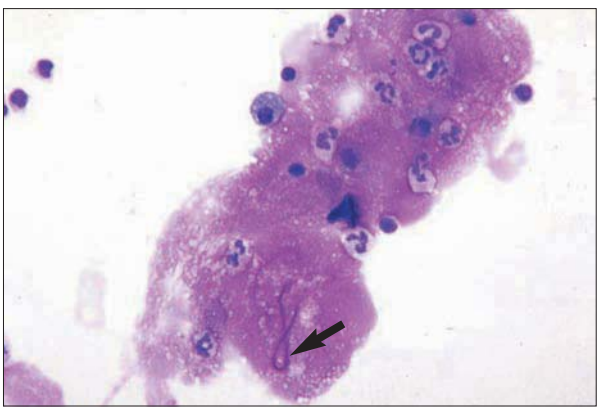
TW fluid obtained from horses with RAO and summer RAO usually reveals a marked increase in neutrophil percentages (>50%; range, 7–96%). Neutrophils are non-degenerate and even if bacteria may be isolated in some cases, they do not play a role in the pathogenesis of the disease.

Thoracic radiographs may rule out pulmonary diseases other than RAO. However, interpretation of radiographs is considered insensitive and non-specific for the diagnosis of RAO.

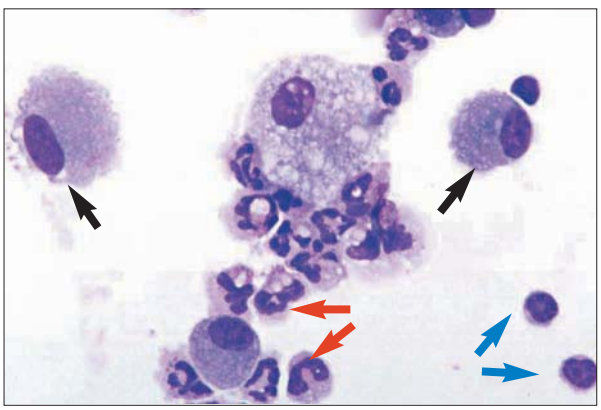


▲ 124 **Bronchoscopy in a horse with severe RAO, showing bronchial erythema and edema resulting in blunted carina (arrow).**

Pulmonary function tests allow quantification of lung dysfunction using various techniques (see Chapter 4). During episodes of disease exacerbation, bronchoconstriction, edema of the airway wall, and accumulation of secretions result in airflow obstruction, $\dot{V}/\dot{Q}$ mismatch, and stiffening of the lungs. These structural changes translate into functional changes such as arterial hypoxemia, increased maximal alterations in transpulmonary pressure (ΔP_{plmax}), R_L (pulmonary resstance), and decreased C_{dyn} (dynamic lung compliance). Arterial hypoxemia is usually pronounced during disease exacerbation (P_{aO_2} <80 mmHg). Values return to within the normal range when horses are in disease remission. Measurement of lung mechanics during tidal breathing is not sensitive and test results usually become abnormal when horses display obvious clinical signs of RAO. This test is still useful in clinical practice to evaluate reversibility of airway obstruction after administration of a bronchodilator or to assess response to therapy, because clinical signs alone are poor predictors of lung function (i.e. a significant degree of airway obstruction may still be present after the course of therapy even though clinical signs have resolved). Unfortunately, lung mechanics can only be measured at a few referral centers. Another noninvasive method that is well suited for field testing combines the use of respiratory inductanceplethysmography and pneumotachography during normal tidal breathing at rest (see Chapter 4, Plethysmography, p.70. Airflow measured at the nostril opening by a pneumotachograph is compared with flow signals from bands placed around the chest and abdomen, specifically the sum of these two signals. Several indices derived from this lung function test correlate with



▲ 125 **BAL fluid cytology from a horse with RAO prepared with Wright's stain. Note the large amount of mucus containing a Curschmann's spiral (arrow).**



▲ 126 **BAL fluid cytology from a horse with RAO prepared with Wright's stain. A large number of nondegenerate neutrophils (red arrows), alveolar macrophages (black arrows) and a few small lymphocytes (blue arrows) can be seen.**

conventional lung mechanics (ΔP_{plmax}, R_L) and allow quantification of airway obstruction in horses with RAO, as well as response to bronchodilators or histamine challenge. This lung function test is currently commercially available (Open pleth™).

A unique feature of horses with RAO, summer RAO, and IAD is the increased tendency for airways to constrict in response to a challenge with irritants such as histamine or methacholine (hyperresponsiveness). Response to bronchoprovocation challenge may be assessed using different types of lung function test such as standard lung mechanics or respiratory inductance plethysmography. In general, the greater the airway obstruction, the more pronounced the bronchoconstriction for a given concentration of irritant. Bronchoprovocation may prove to be a useful method for the detection of mild to moderate RAO in horses that do not display overt clinical signs.

Management/treatment

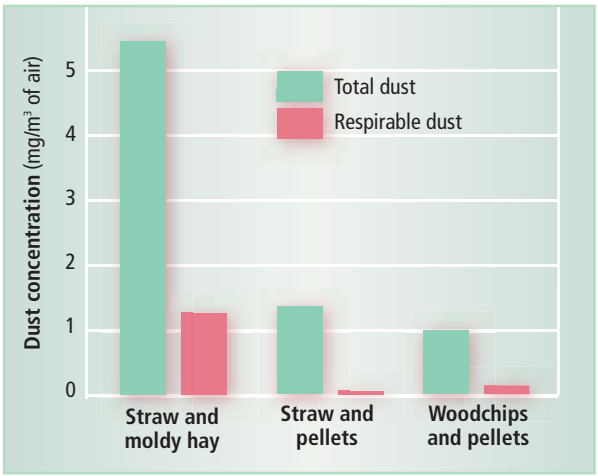
The goals of therapy are to control airway inflammation and relieve airflow obstruction. Treatment of RAO should place emphasis on reducing exposure to environmental triggers. Medical therapy is useful to control clinical signs in severely affected animals and when improvement in environmental management is insufficient. The main classes of drug recommended for treatment of RAO are corticosteroids to treat airway inflammation and bronchodilators to relax airway smooth muscle. Improvement of mucociliary clearance may also help reduce airway obstruction.

Therapeutic goals depend on the types of activity expected, disease severity, and prior therapy. A show jumper with decreased performance and coughing who is maintained in a low-dust environment should benefit from medical therapy. However, a broodmare with an acute RAO crisis 2 weeks after being housed in a barn for the winter may only need simple environmental control measures to be implemented, such as removing hay and feeding pellets, in order to improve clinical signs and allow her to carry a normal pregnancy.

Environmental modifications

Two main approaches help reduce exposure of the horse's airways to respirable particles. The first approach is to use feedstuff and bedding that generate low dust levels. The second approach is to increase removal of airborne particles by improving ventilation in the building. The ideal environment for horses with RAO is pasture because exposure to dust is significantly less than in stalls, regardless of feed and bedding quality. If for practical reasons the horse cannot be kept on pasture at all times, ventilation in the barn and stall, the type of bedding, feedstuff, and general management should be scrutinized in order to minimize allergen exposure. Most horses become free of clinical signs 1–2 weeks after being turned outside onto pasture. Most RAO-affected horses are allergic to dust and particular molds contained in hay. Even good quality hay contains molds, but moldy hay and hay from round bales left outdoors, unprotected from the rain, expose horses to the largest number of allergens. In order to decrease dust exposure in the stall, horses should be bedded on low-dust bedding, such as wood chips or shredded paper, and fed low-dust feed, such as pellets or haylage (**127**). Placing an RAO horse with disease exacerbation into a low-dust environment results in marked improvement in lung function within a few days, even if environmental changes only take place in the horse's stall and not in the adjacent ones. Horses become free of clinical signs within 2–4 weeks in optimal indoor housing or outside on pasture. Once the horse becomes free of clinical disease (remission), medication can be discontinued. Because of the nature of the disease, susceptible horses may suffer another bout of the disease when exposed to allergens.

Horses with summer RAO are generally affected between June and September (northern hemisphere). The recommended environment for these horses during the summer is low-dust indoor housing. The management of affected horses may be complicated by the fact that some horses suffer both from RAO and summer RAO. It is important to realize that every horse responds differently based on whether the allergenic particulates originate from indoor versus outdoor (or both) environments and depending on the immune response (genetics, prior exposures).



◀ 127 Dust levels measured around the nose of horses kept in a stall are reduced dramatically when moldy hay is replaced by complete pelleted feed. Good quality straw and woodchip beddings result in a similar dust exposure level. Total dust concentration (mg/cubic meter of air) represents the number of particulates of all sizes; respirable dust represents the number of particulates less than 5 micrometers in MMAD. The smaller size respirable particles can penetrate deep within the lung and cause inflammation.

Medical therapy

Most horses are easy to treat by the oral or parenteral route and the cost of systemic therapy is usually less than that of aerosol therapy. However, systemic therapy with corticosteroids or bronchodilators may result in adverse effects. Also, appropriate reduction of exposure to RAO triggering factors is as effective as corticosteroid therapy in ameliorating clinical signs and lung function. The added benefits of corticosteroids are a further reduction in airway inflammation and faster clinical improvement.

Corticosteroids

Corticosteroids are potent inflammation inhibitors proven to be effective for the treatment of RAO. Recommended dosages are summarized in *Table 6* (next page). Triamcinolone acetonide is a long-acting corticosteroid that may improve lung function for 2–4 weeks after administration of a single dose. Triamcinolone acetonide administration should not be repeated at less than 3 months' interval because of the risk of complications such as laminitis.

Dexamethasone induces a marked improvement in clinical signs within hours of treatment, but maximal benefit may take a week. A reduction in BAL fluid neutrophilia is evident within 3 days of therapy. Dexamethasone has good oral bioavailability; however, it may be impaired by feeding. Oral administration of the drug may require high dosages (0.16 mg/kg) and it should be given after fasting to achieve consistent results. Treatment of horses with RAO with dexamethasone 21-isonicotinate reduces airway obstruction within 3 days after treatment initiation, with a maximum effect obtained after 7 days. Isoflupredone acetate has a similar efficacy to dexamethasone.

Prednisone is poorly absorbed after oral administration of tablets or liquid forms. Conversely, both liquid and tablet forms of prednisolone are well absorbed in the horse with a bioavailability >50%. The recommended dose is 0.5–1 mg/kg q24h.

Deleterious side-effects associated with corticosteroid therapy depend on drug potency, dose used, and treatment duration. Adrenal suppression may be minimized by giving medication in the morning, particularly when using alternate day therapy. Long-acting and potent corticosteroids (e.g. triamcinolone, dexamethasone) are more likely to cause adverse effects such as immune suppression, iatrogenic Cushing's disease, adrenal cortex suppression, and laminitis. Discontinuation of dexamethasone after an extended treatment period (>3 weeks) should be done carefully to avoid acute adrenocortical insufficiency. Dexamethasone results in adrenal suppression for up to 3 days,

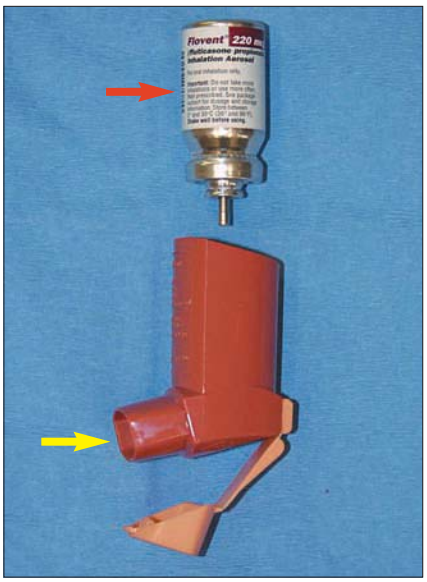
compared to <24 hours for prednisolone. Therefore, discontinuation of prolonged dexamethasone therapy should be performed by slowly and gradually decreasing the dose until the least suppressive amount (0.01 mg/kg) is given every 4th day for a minimum of 2 weeks. Alternatively, dexamethasone therapy may be replaced by an equipotent dose of prednisolone (1 mg dexamethasone ≈8 mg prednisolone), which will be tapered down to alternate day treatment. Before treatment is discontinued, an adrenocorticotropic hormone (ACTH) stimulation test should be performed to assess the adrenocortical reserve necessary for the horse to cope with stress.

Administration of therapeutic substances via inhalation has the advantage of delivering a high concentration of the drug directly into the lungs while minimizing the amount absorbed systemically and, therefore, reducing the risk of adverse effects. In addition, systemic side-effects and drug residue are decreased. At least five different inhaled corticosteroids are available to treat inflammatory lung diseases in humans: beclomethasone dipropionate, budesonide, flunisolide, fluticasone proprionate, and triamcinolone acetonide. A common test of potency for inhaled corticosteroids, called the McKenzie skin blanching test, allows relative ranking of the compounds from least to most potent (relative to dexamethasone potency = 1): flunisolide = triamcinolone acetonide (330) < beclomethasone dipropionate (600) < budesonide (980) < fluticasone propionate (1200). At the time of writing, only clinical trials with beclomethasone and fluticasone have been reported in the horse.

Table 6 **Medications used for systemic therapy of RAO**

MEDICATION	DOSAGE	FREQUENCY OF ADMINISTRATION
Corticosteroids		
Dexamethasone	0.04–0.1 mg/kg IV or IM 0.08–0.165 mg/kg PO	Once per day or every 2 days
Dexamethasone 21-isonicotinate	0.04 mg/kg IM	Every third day
Isoflupredone acetate	0.03 mg/kg IM	Once per day
Prednisolone	1.1–2.2 mg/kg PO	Once per day
Triamcinolone acetonide	0.04–0.09 mg/kg IM	No less than 3-month interval
Bronchodilators		
Aminophylline	5–12 mg/kg IV 6 mg/kg PO	Every 12 hours
Atropine	0.01–0.02 mg/kg IV	Once
Clenbuterol	0.8–3.2 µg/kg PO	Every 12 hours
Glycopyrrolate	0.0022–0.007 mg/kg IV	Once
Isoproterenol	0.1–0.2 mg/kg IV	Once
Pentoxifylline	35 mg/kg PO	Every 12 hours
Theophylline	5–10 mg/kg PO	Every 12 hours

Clinical trials in horses with RAO indicate that beclomethasone dipropionate at a dose of 1,000 µg q12h administered with the 3M Equine device (see **112e**) results in a similar clinical and lung function improvement as 3,750 µg q12h administered with the AeroMask™. An almost four-fold reduction in dose with the 3M Equine device is consistent with the fact that lung deposition is approximately four times higher (23.3/6.1 = 3.8) with it than with the AeroMask™ (see **112b**). Therapeutic effects are measurable within 24 hours of administration. Low-dose beclomethasone dipropionate (500 µg q12h) delivered with the 3M Equine device to horses with RAO results in similar efficacy to a high-dose (>1,500 µg q12h), but with less adrenal suppression. However, currently the 3M Equine device is no longer on the market. Also, these studies were conducted using CFC as propellant for beclomethasone dipropionate. Only HFA propelled beclomethasone is currently available as pMDI. Because aerosolized particles of beclomethasone generated with HFA propellant have a lower MMAD, it can be estimated that the dose needed to achieve the same effect in RAO-affected horses would be approximately half the dose needed with CFC propelled beclomethasone. Therefore, treatment of RAO with a dose equipotent to 3,750 µg q12h administered with the AeroMask™ would require ~1,875 µg of beclomethasone HFA (3,750/2 = 1,875) q12h using the same mask system or ~1,250 µg of beclomethasone HFA (3,750/3 = 1,250) q12h using an AeroHippus™ (see **112c**). Similarly, lung deposition of beclomethasone HFA administered with the Equine Haler (see **112d**) would be expected to double in comparison with a CFC formulation (8.2 × 2 = 16.4%). Consequently, a dose equipotent to 3,750 µg q12h administered with the AeroMask™ would be ~1,400 µg of beclomethasone HFA (3,750 × 6.1/16.4 ~ 1,400) q12h. Future clinical trials are required to determine if these estimated dosages are appropriate.



▲ **128** **Pressurized metered dose inhaler (pMDI) containing fluticasone propionate at a concentration of 220 µg per puff. The canister (red arrow) has been disconnected from the plastic actuator. (Yellow arrow) mouthpiece.**

Fluticasone propionate has been used successfully for the treatment of RAO in horses using 2,000 µg q12h (AeroMask) for 3–4 weeks. A higher dose of fluticasone (6,000 µg) is required to obtain a significant improvement in clinical signs and lung function in a shorter period of time (3rd day of therapy) using an Equine Haler. Administration of fluticasone (3,000 µg) prior to exposure to moldy hay can effectively prevent clinical disease and deterioration in lung function. Treatment of horses with inhaled Fluticasone (AeroMask) using 3,000 µg q12h results in adrenal suppression; however, no adrenal suppression is detectable with 2,000 µg q12h. Fluticasone propionate is formulated as a suspension regardless of the propellant used in pMDI (**128**). Therefore, lung deposition in horses would be expected to be similar with CFC or HFA as demonstrated in human studies.

Comparison between drug efficacies in the treatment of RAO can be extrapolated from studies that used the same aerosol delivery device to treat horses kept in a similar environment. For example, one study treated RAO-affected horses fed moldy hay using an AeroMask™ and beclomethasone dipropionate (3,750 µg q12h for 2 weeks) and reported a 56% improvement in lung function on average. In another study, RAO-affected horses fed moldy hay were treated using the same mask but with fluticasone propionate (2,000 µg q12h for 3 weeks) and reported a similar degree of improvement in lung function (44%). These results suggest that fluticasone is approximately 1.9 (3,750/2,000 ~ 1.9) times as potent as beclomethasone, which is consistent with skin blanching potency test results. The caveat is that currently available pMDIs contain beclomethasone HFA that requires approximately half the dose to achieve the same effect. Therefore, beclomethasone and fluticasone pMDI should be considered equipotent with currently available formulations.

Bronchodilators

Bronchodilators are indicated for relaxing airway smooth muscle and relieving airflow obstruction, but they should not be used alone for extended periods of time because they have no anti-inflammatory properties and do not reduce airway hyperresponsiveness. In addition, prolonged use of certain types of bronchodilators (e.g. beta-2 agonists) as solo medication induces airway receptor downregulation and renders the drug less effective. This phenomenon is prevented by combined use of beta-2 agonists with corticosteroids.

Three classes of substances are available as systemic bronchodilators: anticholinergics, beta-2 agonists, and methylxanthine drugs (*Table 6*). Atropine is an anticholinergic drug that provides rapid and marked improvement in lung function (mean reduction in ΔP_{plmax} of 70–80%) and clinical signs. Effects occur within 10 minutes of administration, peak around 30 minutes, and last a maximum of 1–2 hours. Potentially serious side-effects, such as ileus and abdominal pain, usually develop when higher dosages are used (22–88 mg), but are rare with low dose atropine (≤0.02 mg/kg) unless administration is repeated. Another anticholinergic drug, glycopyrrolate, has similar efficacy to atropine but without deleterious effects on gut motility. These drugs may be used as a single dose for rapid relief of severe airway obstruction and for diagnostic purposes.

Clenbuterol hydrochloride is approved for the treatment of RAO in horses in Australia, Canada, the European Union, South America, and the US. Injectable and oral formulations are available in several countries. Treatment should be initiated at a low dose rate and increased progressively if no clinical response is noted. Some horses (25%) may not respond to clenbuterol even at a high dose rate. Clenbuterol also has anti-inflammatory properties and may help airway mucociliary clearance. Mild side-effects, such as sweating, muscle tremors, and excitement, occur in <10% of horses treated with oral clenbuterol. More concerning is the side-effect of cardiovascular remodeling detected by echocardiography, suggesting a deleterious effect of the drug with medium doses of clenbuterol (2.4 µg/kg PO q12h) for 8 weeks. However, the clinical relevance of these findings is unknown.

Administration of the beta-adrenergic drug isoproterenol to horses with RAO results in clinical and lung function improvement within 15 minutes; however, results are variable between animals and the heart rate more than doubles because of stimulation of cardiac beta-1 receptors. Terbutaline, a beta-2 receptor agonist, is poorly absorbed orally; however, nebulization (0.02 mg/kg) may improve lung function of horses with RAO for up to 6 hours.

Methylxanthine and its derivatives may be beneficial in horses with RAO; however, the plasma levels necessary for bronchodilation vary widely between horses and the range between an effective and a toxic concentration is narrow. Aminophylline and theophylline administered every 12 hours improve lung function and clinical signs in up to 50% of affected horses. Common side-effects are hyperesthesia, hyperexcitability, and muscle tremors. Pentoxifylline administered to RAO-affected horses results in significant improvement in lung function and is not associated with adverse effects.

Two main classes of inhaled bronchodilators have been used in the horse: beta-2 agonists and anticholinergic drugs (*Table 7*). Bronchodilators should not be used as the only therapy for RAO because they do not suppress airway inflammation and do not reduce airway hyperresponsiveness. In addition, prolonged

use of beta-2 agonists without corticosteroids induces receptor downregulation, which renders the drug less effective. In horses with significant airway obstruction, bronchodilators should be administered prior to corticosteroids in order to optimize lung deposition.

Beta-2 agonists induce airway smooth muscle relaxation regardless of bronchoconstriction mechanism and inhibit mast cell degranulation. Albuterol, pirbuterol, and fenoterol are short-acting bronchodilators (1 hour) with a rapid onset of action (5 minutes). Some horses may benefit from the effects of albuterol for up to 7 hours. Salmeterol and formoterol are long-acting beta-2 agonists (6–8 hours) suitable for twice daily dosing, but with a slow onset of action (15 minutes). Salmeterol is currently not available as a stand-alone aerosol medication because of its association with an increased risk of severe asthma exacerbations and asthma-related death in people. However, it is available in combination with steroids. On average, maximum bronchodilation is achieved with 540 µg of inhaled albuterol (~1 µg/kg, metered-dose inhaler) using an AeroHippus™ or Equine Haler™ delivery device. Some horses may reach maximum bronchodilation after as little as 180 µg, while others may require 900 µg. The more severe the airway obstruction, the higher the dose required. Response to inhaled albuterol may be achieved with 24% less drug using an AeroHippus rather than an Equine Haler, suggesting higher lung deposition with the AeroHippus delivery device.

Ipratropium bromide is an anticholinergic drug chemically derived from atropine but devoid of side-effects when administered by inhalation. Nebulization of 2 µg/kg causes bronchodilation for approximately 6 hours with a maximum effect obtained 1 hour after administration. The effects of anticholinergic drugs on airway smooth muscle are additive to beta-2 agonists.

Table 7 **Medications used for aerosol therapy of RAO**

DRUG	DOSE DELIVERED PER ACTUATION	PROPELLANT	DEVICE	DOSE	DURATION OF ACTION
Corticosteroids					
Beclomethasone	80 µg	HFA	EADDS	1–3 µg/kg q12h	
Fluticasone	220 µg	CFC	AeroMask	2–4 µg/kg q12h	
Bronchodilators					
Albuterol	120 µg	HFA	EADDS AeroHippus/Equine Haler	360–720 µg 1–2 µg/kg	1–3 hours
Ipratropium	20 µg 200 µg/capsule 0.02% solution for nebulization	CFC DPI 2.5 ml vial	Aeromask EquiPoudre Ultrasonic nebulizer	0.2–0.4 µg/kg 2–3 µg/kg 2–3 µg/kg	4–6 hours
Fenoterol	200 µg	CFC	AeroMask	1–2 mg	4–6 hours
Pirbuterol	200 µg	CFC	EADDS	600 µg	1 hour
Salmeterol	50 µg	CFC	AeroMask	210 µg	6–8 hours
Cromones					
Cromolyn sodium	0.02% solution for nebulization	2 ml vials	Jet nebulizer Ultrasonic nebulizer	200 mg q12h 80 mg q24h	

CFC, chlorofluorocarbon; HFA, hydrofluoroalkane; DPI, dry powder inhaler; EADDS, 3M equine aerosol drug delivery system

Inflammatory airway disease

Definition/overview

IAD is the most common chronic airway disease of athletic horses, with an incidence ranging between 10 and 60% worldwide. The disease is characterized by cough, poor performance, and excess mucus in the airways on endoscopic examination. Affected horses do not show systemic signs of illness such as fever, lethargy, or decreased appetite. Clinical signs are usually subtle and non-specific and may be difficult to differentiate from other causes of poor performance. IAD is also characterized by non-septic inflammation, detected by cytologic examination of BAL fluid, and pulmonary dysfunction. The duration of IAD typically is in the range of 4–22 weeks, with an average of 8 weeks. In contrast to RAO, horses with IAD typically do not exhibit increased respiratory effort at rest. Some horses with IAD exhibit airway hyperresponsiveness and airway obstruction.

Several other terms, such as small airway inflammatory disease, small airway disease, bronchiolitis, and COPD, have been used in the literature to describe horses with this syndrome.

Etiology/pathophysiology

Proposed causes of IAD include inhaled environmental particles, noxious gases, pollutants, and infectious agents, with a modulatory role played by factors such as the horse's immune response and genetic make-up.

The role of bacterial infection is controversial. Tracheal inflammation and the presence of bacteria are common in horses, in particular racehorses, and the likelihood of isolating bacteria from TW samples collected from racehorses in training is strongly associated with inflammation severity. However, bacteria may in fact be present in the trachea because of contamination during sampling or they may represent transient colonization of the proximal airways. No bacteria are cultured from TW samples in up to 54% of horses with IAD. Also, the trachea is not a sterile environment and potentially pathogenic bacteria may be isolated by tracheal wash in as many as 25% of healthy horses, with isolation of non-pathogenic organisms in up to 75% of those horses. In racehorses, tracheal inflammation is associated with coughing; however, it is not associated with decreased performance.

The role of respiratory viruses in IAD is unclear. Several reports have shown no evidence of viral infections in horses with IAD based on serology or virus isolation aimed at detecting EHV, influenza, adenovirus, and rhinoviruses. These findings are consistent with the fact that no relationship has been found between the presence of fever and IAD. However, one study used PCR for the detection of herpesviruses in TWs and BAL fluids and found that horses with IAD were three times more likely to have EHV-2 DNA in a TW than healthy controls.

There is strong evidence to support the role of exposure to dust and molds in the pathogenesis of IAD. Healthy yearlings fed hay demonstrate an increased BAL fluid neutrophil count and percentage and more severe airway inflammation when housed in a stable than when kept on pasture. Exposure of healthy horses to moldy hay or endotoxins results in BAL fluid neutrophilia and airway hyperresponsiveness. Also, horses in training kept on straw bedding experience episodes of IAD that last longer than in horses bedded on shredded paper. These findings are consistent with data showing markedly higher dust exposure levels in conventional indoor housing with straw bedding and hay feeding compared with wood shavings and pelleted feed or grass pasture. Some horses with IAD demonstrate increased eosinophil or metachromatic cell counts in BAL fluid, suggesting a type I hypersensitivity mechanism in response to inhaled allergens.

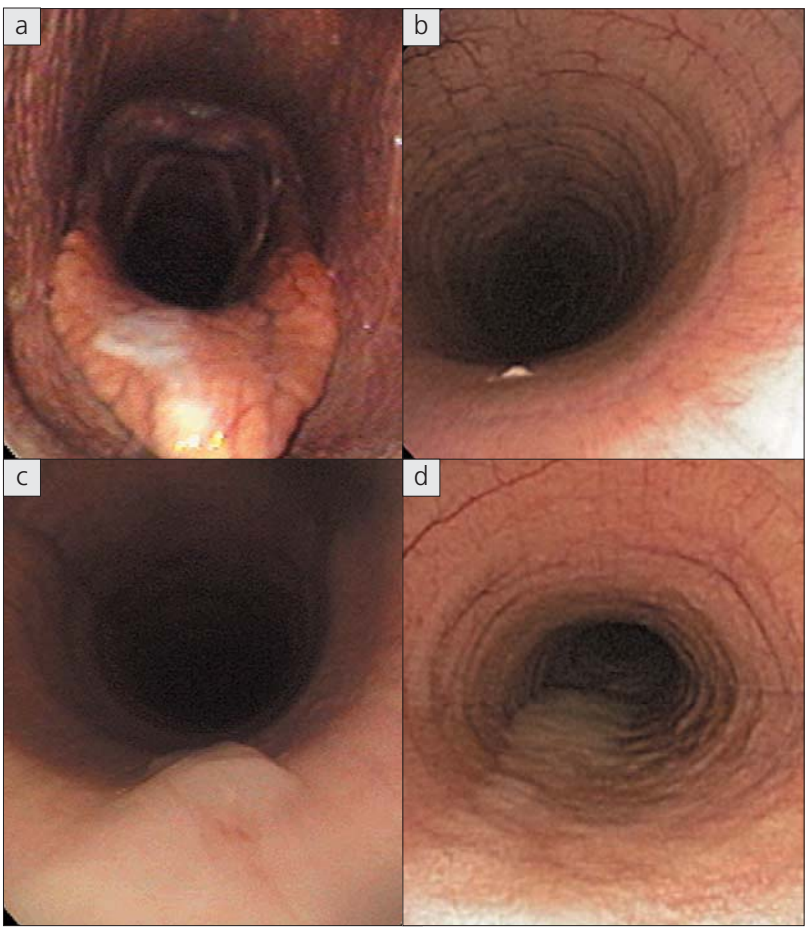
Exposure to atmospheric pollutants, such as ozone, nitrogen dioxide, carbon monoxide, and sulfur dioxide and noxious gases present in stables, such as ammonia, hydrogen sulfide and methane, have the potential to cause IAD in horses. Many of these compounds have been shown to cause airway disease in humans and animals, but data in horses are scarce.

An association between IAD and exercise-induced pulmonary hemorrhage (EIPH) has been found in some studies but not in others.

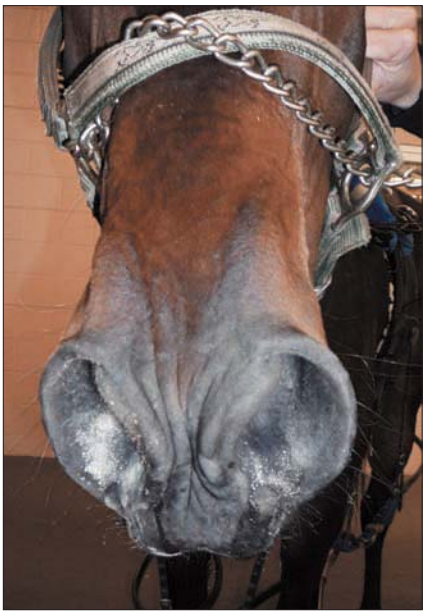
Clinical presentation

The most common clinical signs associated with IAD are increased respiratory secretions, cough, and decreased performance. Horses free of respiratory disease should have either no mucus or a few isolated flecks visible on endoscopy of the URT. Horses with IAD display various amounts from a few blobs of mucus to a continuous stream of variable width from the nasopharynx to the tracheobronchial tree (**129**). The incidence of tracheal exudate has been found by some investigators to increase after strenuous exercise, but not by others. In healthy horses, the amount of tracheal mucus is not affected by age.

Coughing can occur at rest or during exercise, but the absence of cough does not rule out IAD. Hence, coughing is reported in 38% of racehorses with IAD, but 85% of coughing horses have IAD. Epidemiologic studies of Thoroughbred racehorses in training demonstrate a strong association between coughing, the amount of mucus present in the upper airways, and pharyngeal lymphoid hyperplasia.



◀ **129 Endoscopy of the upper respiratory tract in horses with IAD. (a) Mucus accumulation in the nasopharynx. (b–d) Degrees of mucus accumulation in the trachea: mild (b); moderate (c); severe (d).**



▲ **130 Bilateral seromucoid nasal discharge in a horse with IAD, shortly after exercise.**

Fever is not associated with IAD. Serous to mucopurulent nasal discharge is commonly observed in young racehorses, but infrequently seen in older horses. An increased amount of seromucoid nasal discharge post exercise is commonly observed in racehorses with IAD (**130**), but this may also be observed in healthy horses. Thoracic auscultation is usually normal; however, some horses may exhibit increased breath sounds or wheezes, particularly during rebreathing maneuvers. Horses with severe IAD may have a slightly increased respiratory rate and abdominal contraction on expiration, but breathing efforts are not increased (maximum transpulmonary pressure within normal limits [i.e. <10 cmH_2O]). For the most part, IAD is subclinical and may go undetected unless coughing is present or tracheal exudate is detected by endoscopy.

The degree of BAL fluid neutrophilia is linked to poor racing performance and abnormal gas exchanges during exercise. The effect of IAD on performance is dependent on the level of exercise and the severity of the disease. Pulmonary gas exchanges are the limiting factor to performance in fit horses as illustrated by the marked exercise-induced arterial hypoxemia and hypercapnia developed by healthy racehorses exercising strenuously. During a race, horses exercise at or above maximum aerobic capacity (V_{O_2max}). In this situation, a relatively mild degree of IAD may significantly impair gas exchanges and result in decreased performance. IAD is not likely to cause exercise intolerance in a dressage horse exercising at less than 50% of V_{O_2max} until the disease causes marked airflow obstruction or frequent coughing. Therefore, the clinician needs to select diagnostic tools and interpret test results based on the horse's fitness level and the type of activity.

Differential diagnoses

The clinical findings associated with IAD are nonspecific and are shared with a diversity of other respiratory conditions of horses such as: RAO and summer RAO; upper airway diseases; bronchopneumonia, pleuropneumonia, and pulmonary abscess; viral diseases (equine influenza, EHVs, rhinoviruses); EIPH; parasitic pneumonitis (lungworm); neoplasia.

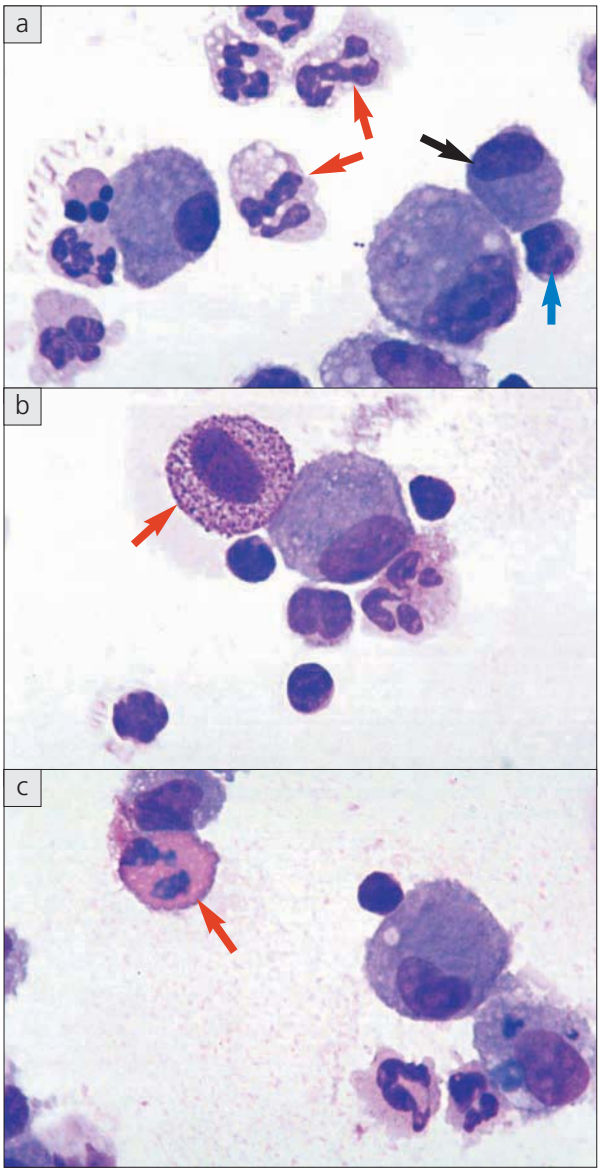
Diagnosis

A detailed history, physical examination, and diagnostic tests of horses presenting with clinical signs including cough, increased respiratory secretions, and poor performance should help eliminate differential diagnoses. The presence of a fever suggests infectious respiratory diseases such as viral diseases, bronchopneumonia, pleuropneumonia, and pulmonary abscess. Hematology findings may be helpful to rule out bacterial infections. Leukocytosis with neutrophilia is commonly found with bacterial respiratory infections and during the acute phase of a bacterial infection, increased numbers of immature neutrophils ('left shift') may be seen. Neutrophilia may also accompany non-infectious inflammatory diseases (e.g. toxins),

neoplasia, and mycotic and parasitic infections. Hematologic changes during the early phase of a viral respiratory infection (e.g. influenza) are often characterized by normocytic, normochromic anemia, lymphopenia or lymphocytosis, and sometimes neutropenia. Neutrophilia may follow within a week of initial clinical signs, particularly in cases of secondary bacterial infection. Monocytosis may develop during the recovery phase of a viral infection.

Chronic, intermittent cough (>3 weeks) is common in horses with IAD. Main differential diagnoses are mild cases of RAO and parasitic pneumonitis. Horses with RAO typically display severe exercise intolerance and increased respiratory effort during periods of disease exacerbation; however, these signs may be subtle during periods of disease remission. In these cases, pulmonary function testing or moldy hay challenge will help reach a definitive diagnosis. Coughing is associated with BAL fluid neutrophilia and mastocytosis with poor performance.

Cytologic analysis of BAL fluid allows recognition of three types of inflammatory profiles in IAD (**131**). The most commonly encountered profile is characterized by an increased total nucleated cell count with mild neutrophilia (5–20% cells), lymphocytosis, and monocytosis. The two other cytologic profiles are characterized by an increased percentage of mast cells (>2%), and/or an increased percentage of eosinophils (>0.1%) is also observed in some horses with IAD. The severity of BAL fluid neutrophilia is usually more pronounced with RAO and summer RAO than with IAD; however, there is significant overlap between these diseases. Eosinophilic inflammation may be associated with IAD, parasitic pneumonitis (*Parascaris equorum*, *Dictyocaulus arnfieldi*), hypersensitivity pneumonitis, fungal pneumonia, and cutaneous habronemiasis. A presumptive diagnosis of parasitic pneumonitis may be reached when respiratory secretions reveal eosinophilic inflammation with evidence of parasite eggs or larvae, exposure to donkeys exists, or anthelmintic therapy results in clinical improvement. Increased metachromatic cells (mast cells, basophils) have been described in horses with IAD, but have not been associated with other types of respiratory disease.



▲ **131 BAL fluid cytology prepared with Wright's stain from horses with IAD. (a) A large number of non-degenerate neutrophils (red arrows), alveolar macrophages (black arrow), and a few lymphocytes (blue arrow). (b) A mast cell (arrow), characterized by its small metachromatic granules, next to an alveolar macrophage and a small lymphocyte. (c) An eosinophil (arrow), characterized by its large eosinophilic granules, next to an alveolar macrophage.**

Direct quantification of airway obstruction requires sophisticated equipment and expertise only available at specialized referral centers. Another means of detecting airway obstruction is by testing airway reactivity in response to an inhaled irritant such as histamine. Exaggerated airway narrowing in response to an irritant is called airway hyperresponsiveness. Airway reactivity may be quantified using portable equipment such as Open Pleth™ (see **110**) that allows field testing. Airway hyperresponsiveness is a prominent feature of IAD in horses with increased BAL fluid eosinophil and mast cell counts. This increased bronchoconstriction in response to inhaled irritants plays an important role in the pathogenesis of the cough and, presumably, exercise intolerance.

Management/treatment

There are limited evidence-based data regarding therapy for IAD, in particular concerning aerosol therapy. Considering the putative pathophysiology of IAD, treatment should combine environmental changes and medical therapy. The goals of medical therapy are to control airway inflammation and relieve airflow obstruction using mainly corticosteroids and bronchodilators. Neutrophilic airway inflammation is a common element in both IAD and RAO, therefore most of the drugs and dosages recommended are based on studies performed on horses with RAO; however, satisfactory clinical response has been observed after treatment of horses with IAD following those guidelines (see *Tables 6* and 7). Both systemic and aerosolized drugs are effective; however, the potential for adverse effects and prolonged elimination times is greater with systemic administration. The advantages of aerosol therapy are ease of administration, high efficacy, and safety. The disadvantages are cost and paucity of data from clinical trials. NSAIDs and antihistamine drugs are ineffective for the treatment of IAD.

Environmental modification

Inhaled dust particles play an important role in the pathophysiology of IAD and treatment of IAD should always include a recommendation to decrease environmental irritants to the airways. Several measures may help reduce exposure of the horse's airways to respirable particles and these are discussed in detail in the section on RAO.

Systemic medical therapy

The same corticosteroids and bronchodilators used for RAO may be used to treat IAD (see RAO section for complete discussion). However, the low end of the dose range is usually sufficient to achieve clinical response in horses with IAD. Short-acting corticosteroids such as prednisolone are preferred in cases where infection has not been ruled out. Corticosteroids are effective in controlling all three cytologic profiles of IAD.

Oral administration of low-dose interferon alpha (IFNα; 50–150 U q24h) has been shown to reduce neutrophil, macrophage, lymphocyte, and total nucleated cell counts as well as immunoglobulin and inflammatory mediator concentrations in the BAL fluids of racehorses with IAD followed over 2 weeks. Higher doses of IFNα (450 U) appeared to be less effective. Endoscopic scores based on respiratory exudate, cough, and pharyngeal lymphoid hyperplasia were significantly reduced after 1 week of therapy, but were no different from placebo at 2 weeks. Mast cell and eosinophil counts did not change after IFNα therapy. Until the pathophysiology of IAD is established, the pulmonary anti-inflammatory effects of IFNα may be attributed to antiviral activity or immunomodulatory properties.

Aerosol therapy

This topic is discussed in detail in the RAO section (see *Table* 7).

Corticosteroids

No clinical trials have been reported in the peer-reviewed literature concerning the use of inhaled corticosteroids for IAD. However, the same drugs used to treat RAO are beneficial for IAD and as a general rule the low end of the dose range recommended for RAO is appropriate for IAD cases. Improved clinical signs and lung function, decreased airway hyperresponsiveness, and reduced pulmonary inflammation are detectable within 2 weeks of therapy.

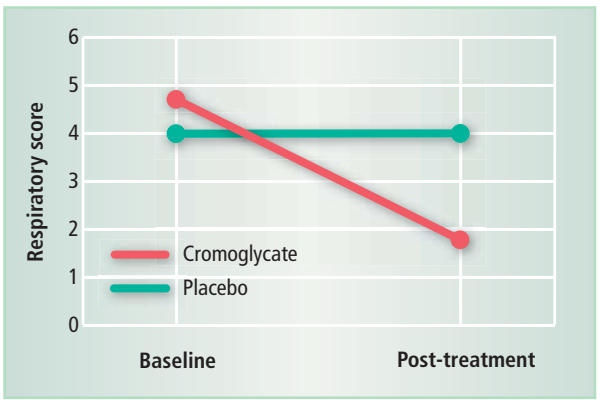
Bronchodilators

Bronchodilators are indicated to relax airway smooth muscle and relieve airflow obstruction. Bronchodilators should not be used as the only therapy for IAD because they do not suppress airway inflammation and do not reduce airway hyperresponsiveness. In addition, prolonged use of beta-2 agonists without corticosteroids induces receptor downregulation, which renders the drug less effective. In horses with significant airway obstruction, bronchodilators should be administered prior to corticosteroids in order to optimize lung deposition. The choice of inhaled bronchodilator and the dosages recommended to treat IAD are the same as for RAO.

Cromones

Sodium cromoglycate (cromolyn sodium; 200 mg q24h or q12h) has been shown to improve clinical signs and to decrease bronchial hyperresponsiveness when administered to horses with IAD characterized by a high mast cell count in BAL fluid (**132**). However, it is ineffective for the treatment of IAD with other inflammatory profiles.

▼ **132 Respiratory score (possible range 0–15) in racehorses with an elevated mast cell count in response to sodium cromoglycate or placebo inhaled therapy (200 mg q12h for 7 days). (Adapted from Hare *et al.*, *J. Vet. Pharmacol. Therap.* 17:237–244, 1994.)**



Parasitic pneumonitis

Definition/overview

Parascaris equorum affects young foals (average age 2–4 months; range 2 weeks to 10 months) and results in coughing and mucopurulent nasal discharge. *Dictyocaulus arnfieldi* infection in adult horses results in coughing and increased expiratory effort in advanced cases. The disease is usually chronic and endemic on the farm.

Etiology/pathophysiology

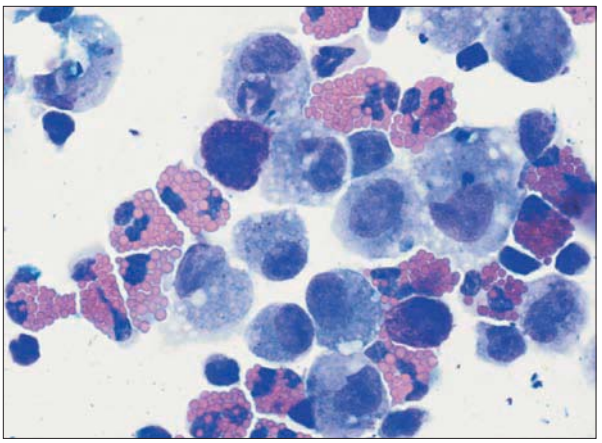
P. equorum larvae migrate from the small intestine to the liver within 7 days of egg ingestion. At 7–14 days larvae are found in the lungs, causing first an eosinophilic then a lymphocytic inflammatory response. Larvae are then coughed and swallowed resulting in attachment to small intestine 33–100 days after ingestion. The minimum pre-patent period is 12–14 weeks. Parasites are shed at 9–12 months and horses are generally immune to reinfection. Eggs survive for up to 5 years in the environment. Fecal flotation using the Baermann technique is often negative for *P. equorum* because migration through the lungs occurs before eggs are released in the feces (pre-patent period).

Infection with *D. arnfieldi* develops when horses come in contact with infected donkeys, mules, or asses. Respiratory secretions show eosinophilic inflammation with sometimes evidence of parasite eggs or larvae. Fecal flotation is useful for detecting *D. arnfieldi* because the infection is usually not patent in horses. Foals may develop patent lungworm infection, but they are usually asymptomatic.

Clinical presentation

Foals with *P. equorum* infection often exhibit chronic mucoid to thick mucopurulent nasal discharge and intermittent coughing. Wheezes and crackles may be heard on rebreathing examination. In cases of severe infection, foals may present for colic due to small intestinal obstruction and poor body condition.

Horses infected by the lungworm *D. arnfieldi* present with prominent coughing, increased respiratory secretions, and exaggerated expiratory effort. Clinical disease is usually observed in late summer or early fall in temperate and continental climates. Cases may occur on a farm as an 'outbreak' of RAO.



◀ **133** **BAL fluid cytology from a horse with parasitic pneumonitis, showing the large percentage of eosinophils present (38%; normal <1%).**

Differential diagnosis

IAD; summer pasture-associated RAO; bronchopneumonia; hypersensitivity pneumonitis; fungal pneumonia; hydatid cyst.

Diagnosis

Lungworm infection should be suspected in horses in contact with donkeys or mules presenting with respiratory signs such as coughing or increased expiratory effort. Similarly, disease in foals with mucopurulent nasal discharge and intermittent coughing may be secondary to *P. equorum* lung migration. Fecal flotation (Baermann technique) is often not diagnostic for *P. equorum* infection in foals because migration through the lungs occurs during the pre-patent period. *D. arnfieldi* follows a complete cycle in donkeys, mules, and asses; however, the infection is usually not patent in horses, so the Baermann fecal flotation is not useful here either. Clinical resolution after anthelmintic therapy confirms the diagnosis.

Respiratory secretions collected by TW or BAL are characterized by eosinophilic inflammation (**133**) and may reveal presence of parasite larvae. Peripheral blood eosinophilia may be present as well as hyperglobulinemia and mild hypoalbuminemia.

Management/treatment

Macrocyclic lactones (ivermectin, moxidectin) are effective against migrating parasite larvae. Fenbendazole may be given to foals with a heavy *P. equorum* infection using a half dose (5 mg/kg PO) initially and giving the second half 24–48 hours later to avoid intestinal obstruction. Migrating larval stages may not be killed by this regimen.

CHAPTER 6

THE HORSE WITH NASAL DISCHARGE

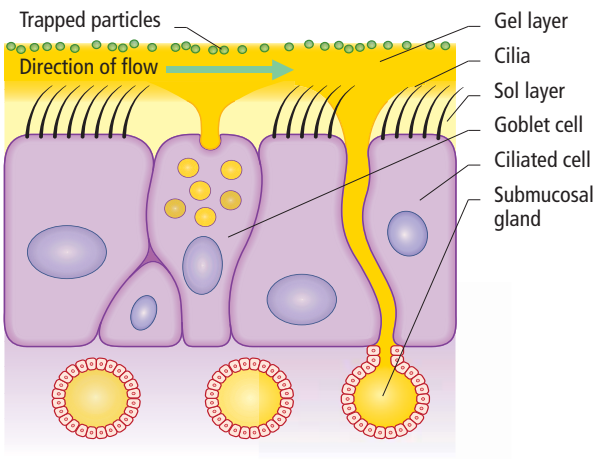
INTRODUCTION

The respiratory tract is protected by a thin layer of mucus that is constantly produced by goblet (mucous) cells, serous cells, submucosal glands, and transepithelial fluid exchange along conducting airways (**134**). The mucus layer forms a mechanical and immune barrier. The design of the upper airways, in particular the nose, allows trapping of larger particles onto the mucosa. The mucociliary apparatus creates a constant flow of mucus directed caudally from the nose to the pharynx, where it is eventually swallowed. As a result, horses do not normally show evidence of a nasal discharge. Mucus secretion is influenced by environmental factors and the submucosal vascular network under the control of the autonomic nervous system. For example, when breathing dry cold air, the nose adds significantly more water to the air, which results in more condensation and dripping of clear secretions from the nose.

Nasal discharge can originate from any part of the respiratory tract, from the nasal passages and paranasal sinuses to the alveoli. Serous discharge is a clear, water-like secretion. Mucoid secretion may vary in appearance from clear and gel-like to thick and turbid. Serous to mucoid nasal discharge is triggered by inflammation of the respiratory epithelium. Thick, white to yellow purulent discharges may be associated with infectious (e.g. strangles, sinusitis, pneumonia) and non-infectious diseases (e.g. RAO). Some discharges have a yellow–green color caused by enzymes released during neutrophil breakdown. Eye diseases resulting in increased lacrimal secretions are accompanied by nasal discharge on the affected side via drainage from the nasolacrimal duct. Secretions from the tracheobronchial tree are usually swallowed when they reach the pharynx without draining into the nose. However, nasal discharge may develop secondary to lower airway disease in cases with a marked increase in secretion volume, coughing, or when the head is held down and gravity facilitates mucus drainage. Pulmonary edema may lead to a clear serous nasal discharge and, in some severe cases, to bilateral foamy exudate.

Bleeding from the nose (epistaxis) may range from a few drops of blood-tinged secretions to profuse fatal hemorrhage. Depending on the cause of the bleeding, blood may be mixed with serous, mucoid, or purulent discharge. Nasal discharge containing a mixture of blood and purulent secretions is usually seen with infections, neoplasia, and necrotic lesions.

A malodorous purulent discharge usually indicates anaerobic infection and is often associated with tooth root abscess, tissue necrosis, or trauma. The presence of feed material in the nasal discharge points to impaired deglutition and may be secondary to a variety of physical or functional abnormalities.



▲ **134 Mucus produced by goblet cells and submucosal glands within the airway surface epithelium forms both a mechanical barrier and a transport mechanism.**

SEROMUCOID NASAL DISCHARGE

Seromucoid nasal discharge may develop as a result of rhinitis, but more commonly is due to generalized airway inflammation secondary to infectious (e.g. viruses) or non-infectious causes (e.g. RAO, IAD).

Rhinitis

Definition/overview

Rhinitis is an inflammation of the nasal epithelium resulting from allergic, vasomotor, or infectious causes. Viral infections are usually associated with seromucoid secretions, whereas bacterial and fungal infections typically result in mucopurulent nasal discharge.

Etiology/pathophysiology

Vasomotor rhinitis is an uncommon cause of serous nasal discharge that may be secondary to abnormal autonomic control of mucosal vasculature. Allergic rhinitis is also rare and is presumably triggered by a variety of inhaled allergens.

Clinical presentation

Clinical signs associated with allergic and vasomotor rhinitis include serous nasal discharge (see **51**, **114**), headshaking, nasal pruritus, and sneezing. Palpation of the nasal septum or nostril wall may reveal pitting edema.

Differential diagnosis

Headshaking related to causes other than rhinitis (e.g. dental problems, ear mites, sinusitis, photic headshaking, infraorbital or trigeminal neuralgia); ocular disease; RAO; IAD; viral infection (rhinovirus, EHV-1, EHV-2, equine influenza virus); irritant exposure (e.g. dust, ammonia); ethmoid hematoma.

Diagnosis

Endoscopy of the upper airways is usually unremarkable with allergic or vasomotor rhinitis; however, it is useful to rule out other causes of nasal discharge. Depending on the origin of the disease, a diagnosis may be confirmed by cytological examination of respiratory secretions (nasal wash or TW, BAL) or biopsy.

Management/treatment

Clinical signs of allergic rhinitis may respond favorably to administration of aerosolized corticosteroids by nebulizer or pMDI (e.g. beclomethasone, fluticasone). Systemic administration of corticosteroids (e.g. dexamethasone, prednisolone), antihistamine drugs (e.g. tripelennamine), or mast cell stabilizer (sodium cromoglycate) may also be tried; however, side-effects are more likely to occur than with aerosolized medication.

Viral infections

Respiratory viral infection typically results in clinical signs including fever, coughing, and bilateral serous nasal discharge. The etiologic agents most commonly implicated are equine influenza viruses, equine herpesviruses, and equine rhinoviruses. Rhinoviruses usually result in subclinical infections, whereas influenza and herpesviruses are often accompanied by outbreaks of respiratory disease and are discussed in Chapter 5 (The coughing horse).

MUCOPURULENT NASAL DISCHARGE

Sinus diseases

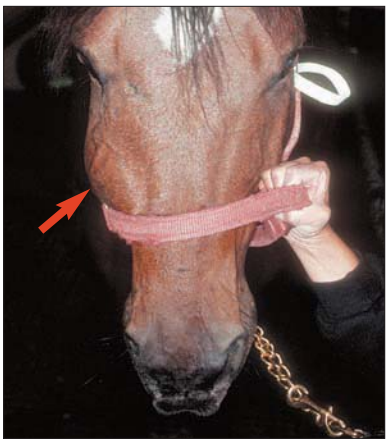
Definition/overview

Nasal discharge originating from the paranasal sinuses can have primary or secondary causes. Primary sinusitis occurs following bacterial or fungal infection of the sinus. Secondary sinusitis occurs secondary to paranasal sinus cysts, neoplastic masses, or dental disease.

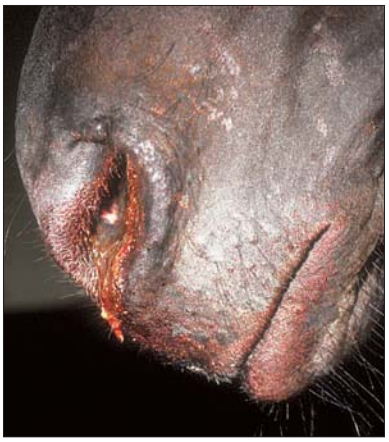
Etiology/pathophysiology

Primary sinusitis affects a wide age range of horses and there is no apparent breed predisposition. Horses with Cushing's disease may be at risk because of high endogenous levels of circulating corticosteroids and resulting immune suppression. Both bacterial and fungal species can cause primary sinusitis. Bacteria can gain entrance into the sinuses from systemic or direct inoculation. *Streptococcus equi* subsp. *zooepidemicus* and *Streptococcus equi* subsp. *equi* are the two most common bacteria causing primary sinusitis, with staphylococcal granuloma (botryomycosis) also occurring. In addition to aerobic organisms, anaerobic bacteria are often

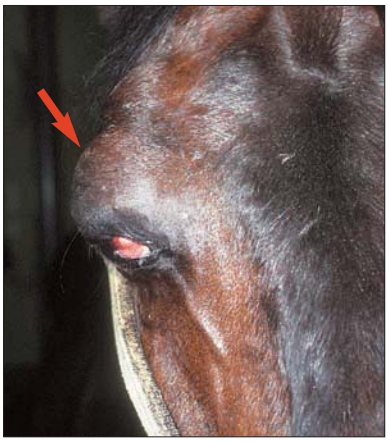
◀ **135** **Primary sinusitis and mucopurulent nasal discharge.**



▶ **136** **Conchofrontal sinusitis. Note the facial distortion (arrow).**



◀ **137** **Horse with mucopurulent and hemorrhagic nasal discharge.**



▶ **138** **Right-sided exophthalmos (arrow) secondary to an ethmoidal carcinoma.**

involved. The hallmark of anaerobic sinusitis is a fetid nasal discharge. Anaerobic bacteria include *Peptostreptococcus* spp. and *Bacteroides fragilis*.

Fungal infection of the paranasal sinuses is rare. There is no known age, breed, or sex predilection. Numerous fungal species causing infection have been reported and include fungi in the group Phycomycetes (from the southwest and southeast portions of the US), *Cryptococcus neoformans*, *Coccidioides immitis*, *Aspergillus fumigatus*, *Rhinosporidium*, and fungi causing eumycotic mycetoma (e.g. *Pseudallescheria boydii*).

Secondary sinusitis is usually related to the presence of paranasal sinus cysts, neoplastic masses, or dental disease.

Clinical presentation

Clinical signs of primary and secondary sinusitis include unilateral or bilateral mucopurulent nasal discharge (**135**), respiratory noise, facial distortion (**136**), epiphora from obstruction of the nasolacrimal duct(s), enlarged submandibular lymph nodes, epistaxis (**137**), exophthalmos (**138**), and a dull sound on percussion of the sinuses.

Differential diagnosis

Primary and secondary sinusitis; dental disease: perialveolar periostitis, dental tumors; neoplastic masses: squamous cell carcinoma, adenocarcinoma, lymphosarcoma, and fibrosarcoma; paranasal sinus cysts; ethmoid hematoma; conchofrontal sinusitis.

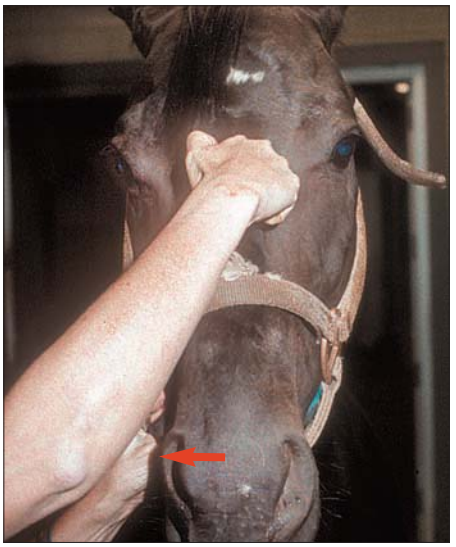
Diagnosis

Physical examination findings are an important diagnostic aid for sinusitis. The primary physical examination abnormalities associated with sinusitis include dull percussion (**139**) of the paranasal sinuses and purulent to mucopurulent nasal discharge.

An oral examination is required to evaluate the teeth for evidence of periodontal pockets, fractured teeth, and abnormal odor. Endoscopic examination of the nasal passage, nasomaxillary opening, ethmoid turbinates, and nasal septum can reveal the source of abnormal drainage (**140**, **141**) and identify neoplastic masses and cystic structures.

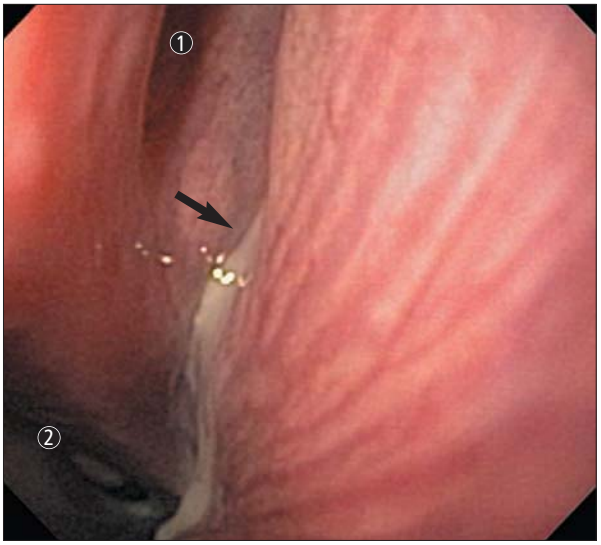
Radiography is very helpful in revealing fluid lines within the sinuses and in the identification of neoplastic masses, deviation of the nasal septum (**142**), and evaluating tooth roots for signs of perialveolar periosteitis. Nuclear scintigraphy can be useful in the evaluation of perialveolar periosteitis. CT and MRI are readily available in many referral centers and play an important role in the evaluation of paranasal sinus disease (**143**).

Sinocentesis is used to obtain fluid and tissue samples from the paranasal sinuses. The procedure involves inserting a 3.0–6.4 mm Steinman pin with a Jacob's pin chuck into the affected sinus. A sterile teat cannula or surgical instrument (e.g. Ferris Smith rongeur) can be inserted into the sinocentesis site to obtain fluid or tissue for biopsy from the sinus. For the caudal maxillary sinus, the pin is inserted 2.5–3 cm rostral to the orbit and 2.5–3 cm dorsal to the facial crest (**144**). For the rostral maxillary sinus, the pin is inserted 3 cm caudal to the infraorbital foramen and 3 cm dorsal to the facial crest (**144**). For the frontal sinus, the pin can be inserted anywhere within the following limits: dorsally, the midline of the head; caudally, a line joining the right and left supraorbital foramina; rostrally, a line approximately 8–10 cm rostral or a line perpendicular to the facial crest, half way between the medial canthus and the infraorbital foramen; laterally, a line drawn from the medial canthus of the eye to the nasoincisive notch.

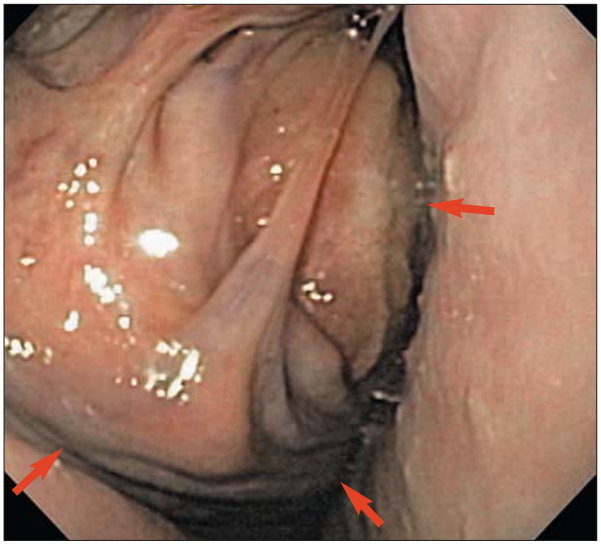


▲ **139 Manual percussion of the left frontal sinus. Note thumb of the left hand inserted into the mouth to increase resonance (arrow).**

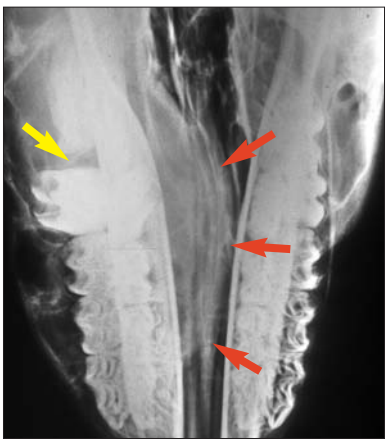
The final diagnostic test to consider for evaluation of sinusitis is sinoscopy. Sinoscopy is accomplished with the insertion of a rigid 4 mm arthroscope or flexible endoscope into the sinus portals previously described. Sinoscopy can be very helpful in confirming a preliminary diagnosis, obtaining biopsy and microbial culture samples, and preoperative planning for definitive surgical treatment.



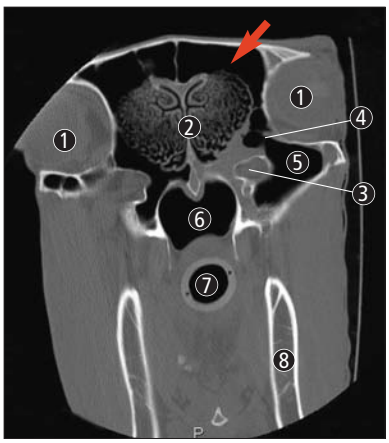
▲ 140 Endoscopic photograph of a purulent discharge exiting the left nasomaxillary opening. (1) ethmoid turbinates; (2) nasopharynx.



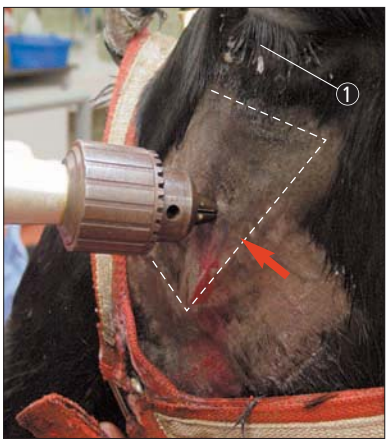
▲ 141 Endoscopic photograph of a horse with chronic, right-sided nasal discharge secondary to a nasal and paranasal sinus cyst (arrows).



▲ 142 Dorsoventral radiographic view of the skull of a horse with a large paranasal sinus cyst resulting in deviation of the nasal septum (red arrows). Note the abnormally shaped tooth (yellow arrow), which is sometimes seen in horses with congenital sinus cysts.



▲ 143 CT image of a horse with a hemangiosarcoma in the left frontal sinus (arrow). Note the following anatomical structures: globe (1), ethmoid turbinates (2), infraorbital canal (3), frontomaxillary opening (4), maxillary sinus (5), nasopharynx (6), endotracheal tube (7), and mandibular ramus (8). (Photo courtesy Stephen B Adams)



▲ 144 Sinocentesis of the left maxillary sinus (dotted line) using a Jacob's pin chuck. Note the sinocentesis site just dorsal to the facial crest (arrow); (1) orbit.

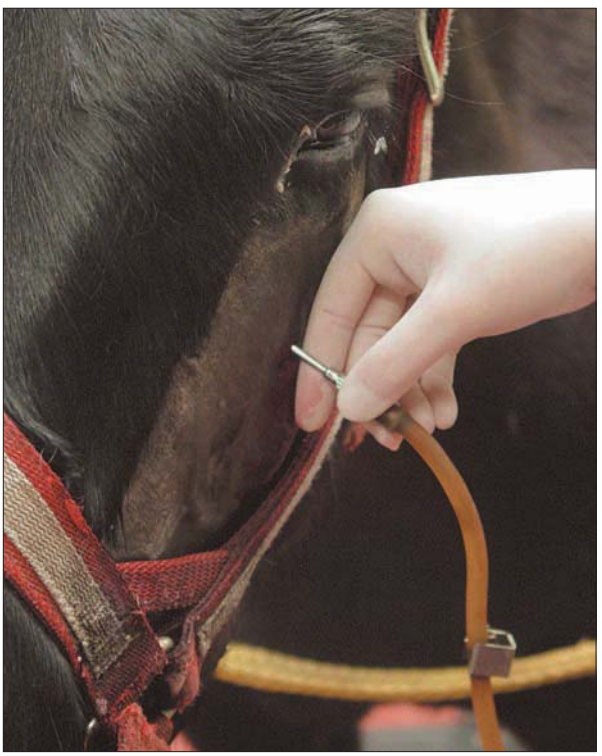
Management/treatment

Primary bacterial sinusitis

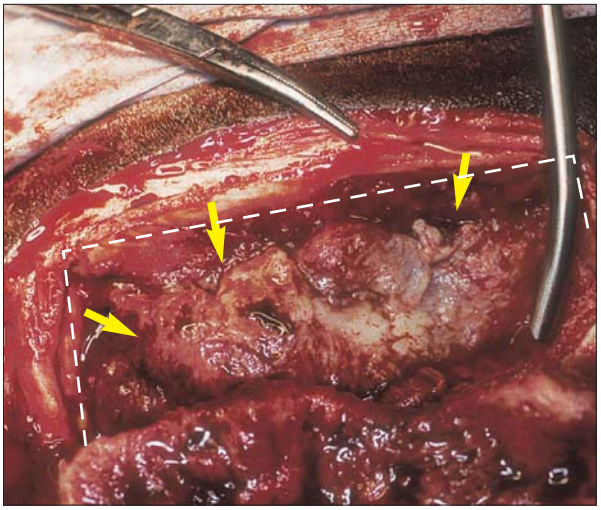
A combination of medical and surgical management is frequently needed to resolve clinical signs. Penicillin G is the drug of choice for primary sinusitis. It is highly effective against *Streptococcus* spp. and has a good spectrum against anaerobes with the exception of *Bacteroides fragilis*. Metronidazole can be administered in combination or used alone for treatment of anaerobic bacterial infections. If needed, NSAIDs can be administered to aid patient well being and comfort. Most, if not all, cases of primary sinusitis should be treated with sinus lavage (**145**). Sinus lavage is useful in removing exudate from the sinuses and maintaining patency of the nasomaxillary opening. To perform sinus lavage the sinocentesis portals previously described can be used. Following IV sedation with xylazine or detomidine hydrochloride combined with butorphanol tartrate, a sterile teat cannula or fluid administration set is inserted into the sinocentesis portal and 1–2 liters of sterile balanced polyionic fluids are instilled into the sinus cavity.

In cases non-responsive to antimicrobial therapy and sinus lavage, surgery may be indicated to remove inspissated purulent exudate and to enlarge the nasomaxillary opening to allow for unimpeded drainage. This is best accomplished with an osteoplastic bone flap of the affected sinus or nasal passage. In chronic cases, surgery generally results in the best chance for a favorable outcome (**146**).

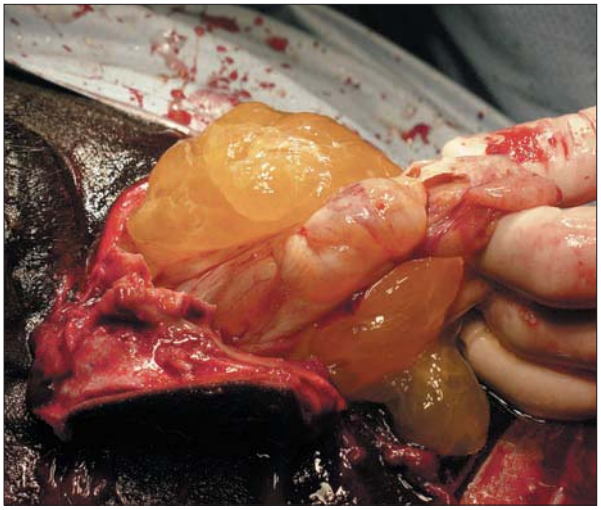
The medical management of fungal sinusitis is expensive and is often not very effective. Numerous antifungal medications, such as itraconazole, ketaconazole, fluconazole, amphoterin B, and sodium iodide, have been used to treat fungal infections conservatively. In most situations surgery provides the best chance of a successful outcome. Nasal and sinus osteoplastic bone flaps provide the best access to these lesions. Complete resection is necessary to prevent reoccurrence. Surgeons may also combine surgical resection with an Nd:YAG or diode laser to cauterize the origin of the fungal growth. The prognosis for fungal sinusitis is guarded to poor unless the fungal mass is completely resected.



◀ **145 A horse with acute sinusitis being treated with maxillary sinus lavage with a teat cannula placed in the sinocentesis site.**



▲ **146** **Intraoperative photograph of a right maxillary sinus (dotted line) osteoplastic flap. Note the accumulation of inspissated purulent exudate within the maxillary sinus (arrows).**



▲ **147** **Large paranasal sinus cyst being removed from an osteoplastic flap.**

Secondary sinusitis is best treated with surgical management. However, if the presence of a neoplastic mass has been confirmed, the appropriateness of surgical management should be discussed with the owner. The prognosis for surgical management of neoplastic masses is guarded, mainly because complete surgical removal cannot always be performed. Surgical management of paranasal sinus cysts is readily accomplished utilizing an osteoplastic bone flap (**147**). The lining of the cyst is manually 'stripped' from the interior of the sinus and removed. Sinusitis secondary to dental disease is generally more complicated because in most cases extraction of an infected tooth is required. Sinusotomy is always combined with sinus lavage and surgical enlargement of the nasomaxillary opening or a new opening is created between the sinus and the nasal passage. The enlargement of a normal opening or creation of a new one greatly improves drainage from the sinus and allows for postoperative evaluation of the affected sinus with endoscopy via the nasal passage. The prognosis for surgical management of secondary sinusitis secondary to sinus cysts and dental disease is good.

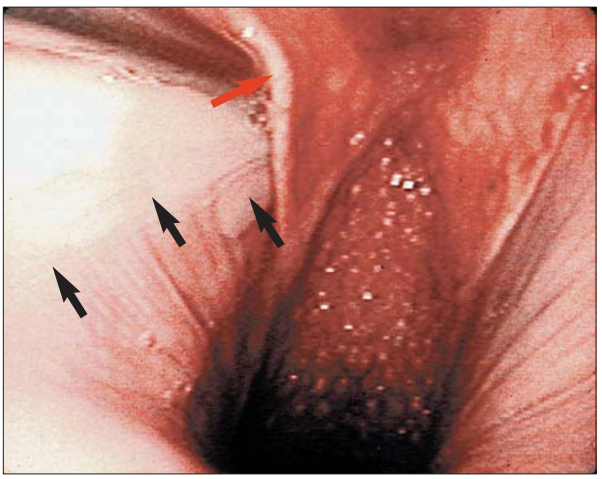
Key points

- Mucopurulent nasal discharge is frequently associated with primary and secondary sinusitis.
- A combination of medical and surgical management usually carries the best prognosis.
- Prior to surgery, neoplastic masses within the sinuses should be confirmed.

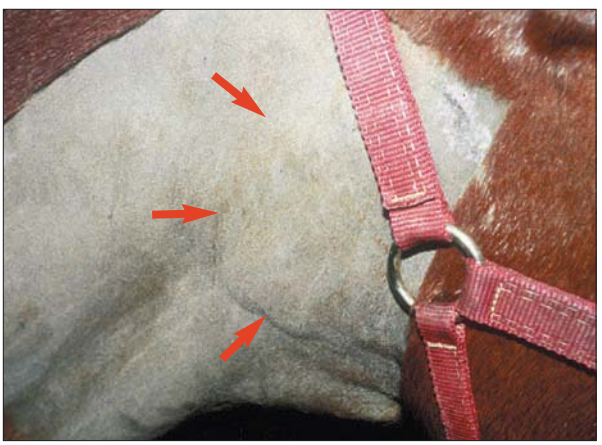
Guttural pouch empyema

Definition/overview

Guttural pouch empyema (GPE) is a common cause of mucopurulent nasal discharge and is defined as an accumulation of exudate within the guttural pouch.



▲ 148 **Endoscopic photograph of the classical white exudate frequently found in horses with guttural pouch empyema (black arrows). Note the dorsal positioning of the Chambers catheter (red arrow) being used to lavage the guttural pouch.**



▲ 149 **Horse with external distension of the guttural pouch (arrows). The guttural pouch of this horse was filled with multiple chondroids.**

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Etiology/pathophysiology

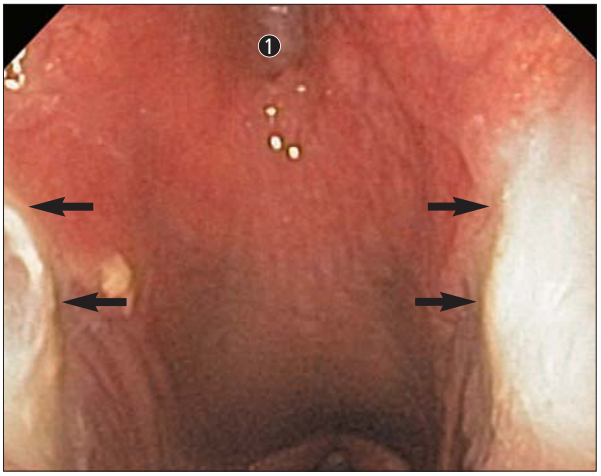
GPE occurs secondary to upper respiratory tract infections in horses of any age, but typically younger horses are more commonly affected. The most common historical finding is nasal discharge of large quantities of exudate when the horse lowers its head. The primary causes of GPE include URT infections, rupture of a retropharyngeal lymph node abscess into the pouch, local irrigation of the GP with irritating drugs (e.g. betadine, vinegar, dimethyl sulfoxide [DMSO]), complication from GP tympany, and abnormal function of the pharyngeal orifice of the GP. The most common bacterial organisms causing GPE are *S. zooepidemicus* and or *S. equi*.

Clinical presentation

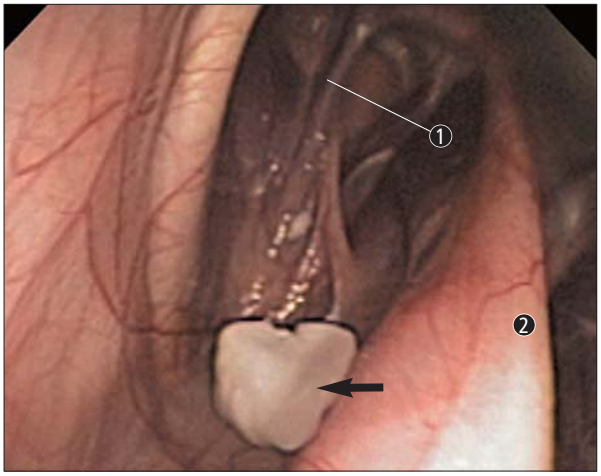
The primary clinical sign of GPE is unilateral nasal discharge. The nasal discharge can be malodorous, mucoid, or even milky in appearance and may be intermittent (**148**). The nasal discharge is most noticeable when the horse is eating with its head down. This occurs because the pharyngeal orifice of the GP opens when the horse swallows. Other clinical signs associated with GPE include swelling in the parotid region (**149**), stiff head carriage, retropharyngeal or submandibular lymphadenopathy, dysphagia, and respiratory distress and upper respiratory noise. Dysphagia results when the nerves controlling swallowing are irritated or damaged by accumulation of exudate within the GP.

Differential diagnosis

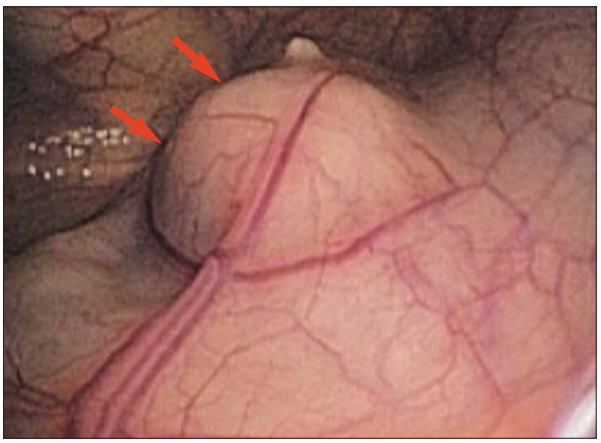
Primary or secondary sinusitis; rhinitis; pneumonia; dysphagia.



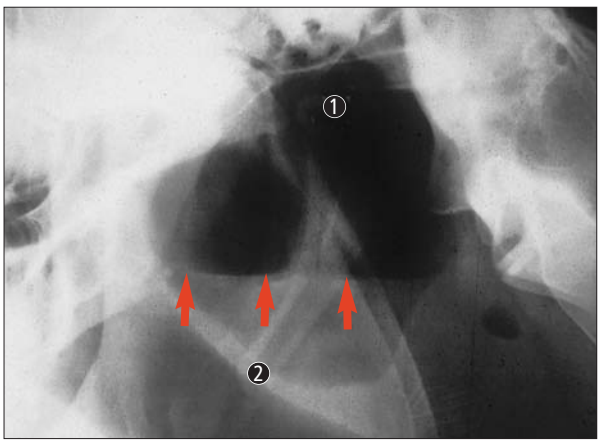
▲ **150** **Endoscopic photograph of bilateral guttural pouch empyema (arrows). (1) dorsal pharyngeal recess.**



▲ **151** **Endoscopic photograph of a chondroid (arrow) in the medial compartment of the left guttural pouch. (1) internal carotid artery; (2) stylohyoid bone.**



▲ **152** **Endoscopic photograph of an enlarged retropharyngeal lymph node (arrows) discharging into the guttural pouch.**

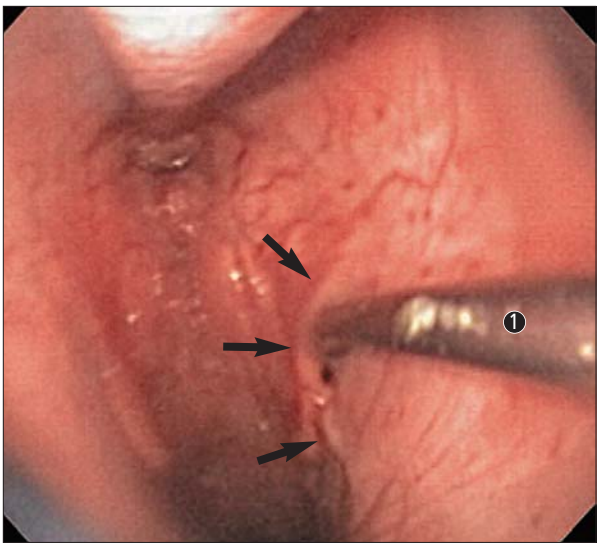


▲ **153** **Lateral view radiograph of a fluid line (arrows) within the guttural pouch (1). (2) stylohyoid bones. (Photo courtesy JF Fessler)**

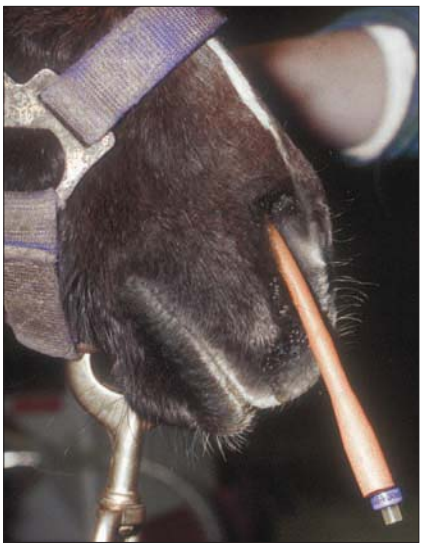
Diagnosis

A diagnosis of GPE is made based on clinical signs, endoscopy, and radiography. Endoscopy is necessary to confirm a diagnosis of GPE (**150**). Endoscopic findings compatible with GPE include exudate exiting the involved pouch, discharge from the pouch opening when external pressure is applied to the pouch, visualization of exudate and/or chondroids within the pouch (**151**), and an enlarged retropharyngeal lymph node bulging into the pouch (**152**). Chondroids are accumulations of inspissated pus.

If endoscopy is not available, plain view or digital radiographs can be used to identify fluid lines within the pouch (**153**) and increased radiopacity compatible with fluid accumulation.



▲ **154** **Endoscopic photograph of successful Chambers catheter (1) insertion into the left guttural pouch under the cartilage flap (arrows) and plica salpingopharyngea.**

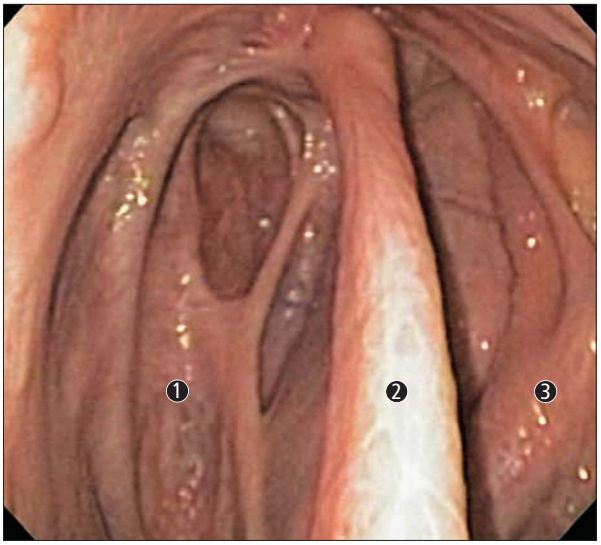


▲ **155** **An indwelling Foley catheter being used for lavage of the guttural pouch.**

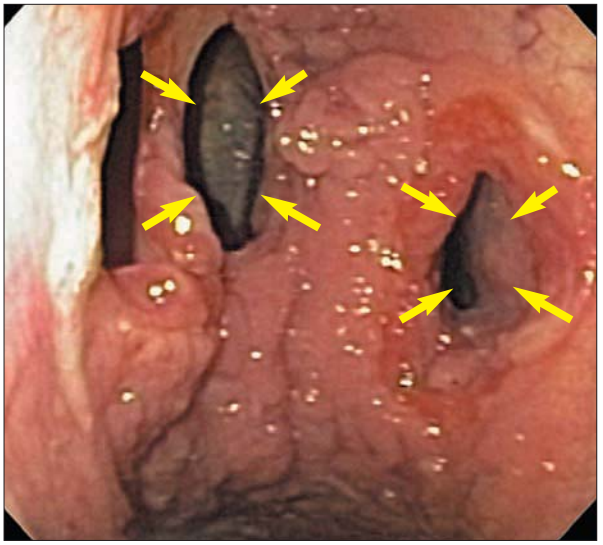
Management/treatment

The majority of cases of GPE can be treated with medical management (see Strangles management/treatment, p. 117). The use of antimicrobial therapy is controversial and is not recommended by all practitioners. Penicillin G is very effective against hemolytic *Streptococcus* species. NSAIDs should be administered to improve patient comfort. In addition to antimicrobial therapy, the GP should be lavaged to remove accumulated exudate. Lavage is accomplished following IV sedation. An endoscope is inserted on the contralateral side of the affected GP and a mare Chamber's urinary catheter is inserted into the ipsilateral side of the affected pouch. Under endoscopic guidance the catheter is inserted into the pouch (**154**). One to two liters of sterile balanced polyionic solution is instilled into the pouch. It is important to keep the horse's head low during this procedure to prevent aspiration of fluid into the trachea. If GP lavage is to be performed more than once, an indwelling Foley catheter should be placed into the affected pouch (**155**). The balloon of the Foley catheter is inflated to maintain the position of the catheter inside the pouch. The authors do not recommend suturing the catheter to the nares because it would reduce the ability of the veterinarian to recognize if the catheter was being maintained in its desired location. In some instances, small numbers of chondroids can be successfully removed by use of a Dormia basket inserted via the biopsy channel of the endoscope.

Chronic cases of GPE non-responsive to medical therapy or horses with many chondroids can be treated by surgical drainage of the affected GP (**156**). Several surgical approaches (e.g. modified Whitehouse or hyovertebrotomy) to the GP have been used to resolve chronic GPE. The authors' preference is to use a diode laser to create a salpingopharyngeal fistula into the affected pouch (**157**). A catheter or stomach tube can



▲ 156 **Endoscopic photograph of chronic guttural pouch empyema which was non-responsive to conservative management. Note the hypertrophy of the mucosal lining of the guttural pouch and the difficulty in the identification of the vital structures within the pouch. (1) medial compartment; (2) stylohyoid bone; (3) lateral compartment. This horse was treated with bilateral plica salpingopharyngeal fistulation with a diode laser.**



▲ 157 **Endoscopic photograph of bilateral guttural pouch fistulation (arrows) for management of chronic guttural pouch empyema.**

then be inserted to remove exudate and/or lavage chondroids from the pouch. To treat GPE associated with malfunction of the pharyngeal orifice of the pouch, a Foley catheter can be inserted through the fistula to form a permanent fistula between the pharynx and the pouch. If a diode or Nd:YAG laser is not available, the most suitable surgical approach is a modified Whitehouse. This provides excellent ventral drainage to the GP. A drain can be inserted or the incision can be left open to heal via second intention. The primary disadvantage to the modified Whitehouse approach is that GPE can reoccur if the pharyngeal orifice of the pouch is malfunctioning. A plica salpingopharyngeal fistula would provide a better alternative in this situation.

The prognosis for medical and surgical management is good provided the horse does not experience neurogenic dysphagia. The prognosis for GPE with dysphagia is guarded to poor.

Key points

- GPE is a common cause of mucopurulent nasal discharge in the horse.
- GPE is more common in young horses with a history of upper respiratory infection or retropharyngeal or submandibular lymphadenopathy.
- The best antimicrobial to use for medical treatment of GPE is penicillin G.
- Most cases of GPE can be managed successfully without surgery.
- Chronic cases of GPE should be strongly considered for surgery.
- The prognosis for GPE is favorable provided dysphagia is not a complication.

Strangles

Definition/overview

Strangles is caused by the gram-positive bacterium *S. equi*. Young horses (1–3 years of age) are more susceptible, but horses of any age may be affected depending on their immune status. No breed or sex predisposition has been reported. Clinical signs are characterized by an acute onset of fever, depression, swollen retropharyngeal and mandibular lymph nodes, painful swallowing, anorexia, and a purulent discharge from the nose or other locations depending on where abscesses rupture. The term 'strangles' refers to the fact that severely affected horses may develop respiratory obstruction from obstructed upper airways secondary to enlarged retropharyngeal lymph nodes. Coughing is detected in a minority of strangles cases; however, it may be triggered by compressing the proximal tracheal rings.

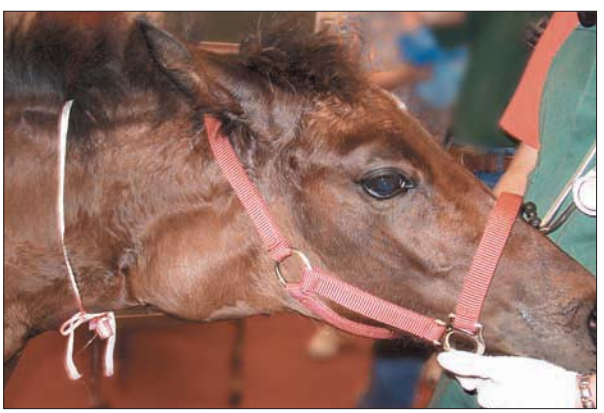
Etiology/pathophysiology

S. equi is highly virulent and contagious. Infection occurs via the mouth or nose following contact with an infected horse, aerosol, or fomites. *S. equi* rapidly colonizes the pharynx and associated lymphoid tissue where it can be isolated from lymph nodes within hours of infection. Abscess formation in lymph nodes takes at least 3–5 days before it can be detected clinically. Maturation of the abscess and rupture occur 1–2 weeks following infection. Typically, the mandibular and retropharyngeal lymph nodes are affected. An atypical form of the disease may occur in older horses with waning immunity against *S. equi*. Affected horses develop a milder form of strangles that tends to resolve rapidly. In some cases, *S. equi* spreads via blood circulation or lymphatic vessels to distant locations in the body, such as the brain, lungs, and mesentery, to form abscesses. These complications of *S. equi* infection are called 'bastard strangles'. Rarely, hematogenous spread of bacteria may trigger an immune-mediated vasculitis called purpura hemorrhagica.

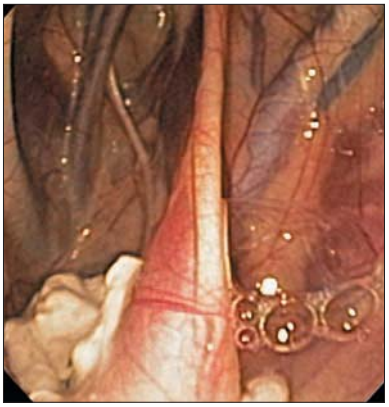
Clinical presentation

Clinical signs develop within 7–12 days of exposure to an infected horse, but the incubation period can range between 1 and 14 days. Fever is the first manifestation of disease followed by lymph node swelling and nasal discharge. Initially, the nasal discharge is serous, but it rapidly becomes purulent (**158**). An important aspect of the clinical progression is that nasal shedding of the organism follows the onset of fever by 2–9 days, but subsequent nasal shedding can last for 3–8 weeks even after resolution of clinical signs. Lymph node abscessation and rupture usually occur between 1 and 2 weeks following onset of clinical signs. GPE may develop secondary to rupture of a retropharyngeal lymph node through the floor of the medial compartment or by ascending infection from the nasopharynx. Drainage of

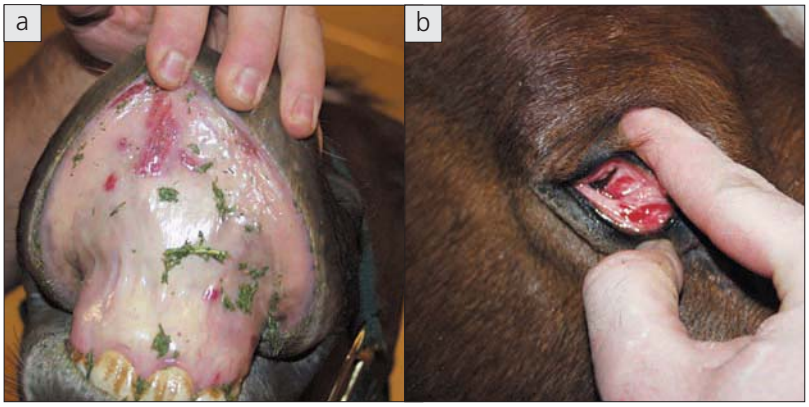
▲ **158 Purulent nasal discharge.**



▶ **159 Foal with severe swelling of retropharyngeal lymph nodes causing airway obstruction that necessitated placement of a tracheostomy tube.**



▲ **160** **Chondroids accumulated in the guttural pouch.**



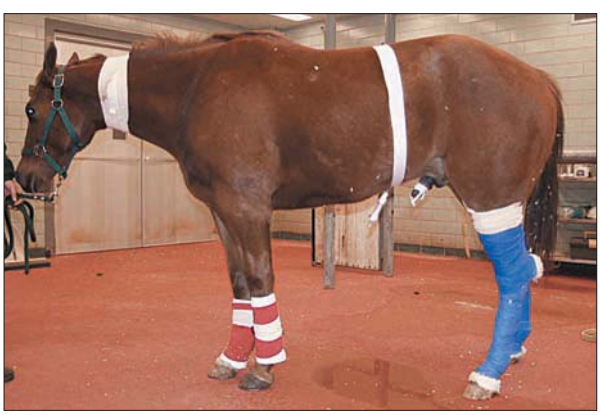
▲ **161** **Petechiae involving the oral mucosa (a) and conjunctiva (b) in a horse with purpura hemorrhagica.**

pus from the GPs results in a mucopurulent nasal discharge. Swollen lymph nodes are usually painful to the touch and can lead to dysphagia. Excessive distension of retropharyngeal lymph nodes may cause life-threatening obstruction of the nasopharynx (**159**), resulting in abnormal respiratory noise (stridor).

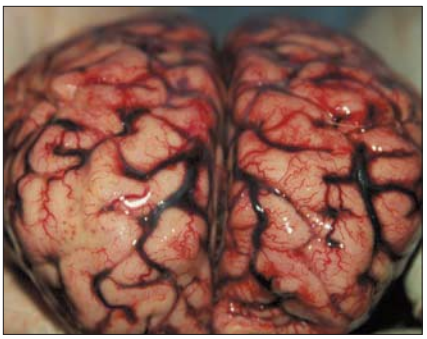
Chronic GPE may lead to accumulation of inspissated pus, forming chondroids (**160**). Chondroids are often tightly adherent to the pouch mucosa, allowing them to persist for months to years. Horses by then are not exhibiting clinical signs of disease, but carry chondroids that harbor live bacteria. Therefore, affected animals are 'silent carriers' and shed *S. equi* intermittently in nasal secretions. It is estimated that at least one silent carrier persists in 50% of outbreaks and these horses are commonly responsible for future outbreaks.

Spread of *S. equi* to organs beyond the head (bastard strangles) may result in abscess formation almost anywhere in the body (brain, thorax, abdomen, and musculoskeletal system). Clinical signs will depend on abscess location and severity and usually will be accompanied by systemic signs of chronic infection such as fever, depression, anorexia, and weight loss.

Purpura hemorrhagica usually develops acutely 2–4 weeks following the onset of respiratory disease or vaccination. Clinical signs of vasculitis are petechiae and ecchymotic hemorrhages (**161**), skin swelling resembling urticaria, and subcutaneous edema in various locations (**162**) such as limbs, chest, abdomen, and head. Edema can lead to exudation, crusting, and eventually sloughing of the skin. Vasculitis may affect other organs such as the kidneys, heart, and skeletal muscles and, as a result, cause a variety of clinical signs (e.g. reluctance to move, anuria, hemorrhage, anemia, thrombocytopenia, colic, epistaxis, fever, anorexia). The disease may prove fatal unless aggressive therapy is rapidly instituted.



▶ **162** **Edema extending over the limbs, ventral chest and abdomen in a horse with purpura hemorrhagica.**



▲ **163** **Brain from a horse with bastard strangles, showing gross lesions of meningitis.**

▲ **164** **Turbid fluid (purulent exudate) obtained by abdominocentesis in a horse with a mesenteric lymph node abscess caused by bastard strangles.**

Other uncommon complications are agalactia in mares, laryngeal hemiplegia, myocarditis, cutaneous abscesses and ulcers, septicemia, meningitis (**163**), and spinal cord compression.

Differential diagnoses

Acute viral infection (equine influenza, equine herpesvirus and equine viral arteritis; *S. zooepidemicus* infection; GP infection secondary to other pathogens (*S. zooepidemicus*, *Staphylococcus aureus*, or fungi); vasculitis caused by infectious agents (equine viral arteritis, equine infectious anemia, *Anaplasma phagocytophilum*, *Staphylococcus* spp.), toxins, and neoplasia.

Diagnosis

A strong suspicion of strangles should arise for any horse with acute onset of fever, purulent nasal discharge, and lymph node swelling, particularly in an outbreak situation. Hematology often shows leukocytosis with neutrophilia. Horses with purpura may display more profound changes, such as anemia, neutrophilia, hyperfibrinoginemia, hyperglobulinemia, and elevated muscle enzymes (creatinine kinase and aspartate transaminase).

Diagnostic confirmation requires bacteriologic culture of nasal secretions or pus from a lymph node or GP. Testing for *S. equi* via PCR is available and increases the likelihood of detection, particularly in chronic carriers. Endoscopy of the upper airways allows access to the GPs and evaluation of airway obstruction. In cases of bastard strangles, sampling of fluid from the thorax and abdomen may reveal a purulent exudate (**164**). Biopsy of skin lesions associated with purpura may confirm a diagnosis of vasculitis, but it is not specific for the disease. Very high titers for *S. equi* M-protein-specific antibodies are found either 1–3 months post infection or in cases of bastard strangles and purpura hemorrhagica. Serology does not distinguish between a vaccine and a post-infection response, but comparison of paired titers collected 2–3 weeks apart may help in making this distinction. Furthermore, serology may prove to be useful in screening for carriers, since a seronegative horse is very unlikely to be a carrier of *S. equi* in its GPs. Ultrasonography and rectal examination may help locate abdominal abscesses. Horses with colic may require exploratory laparotomy and/or laparoscopy in order to identify and treat internal abscesses.

Management/treatment

Strict quarantine of affected and exposed horses should be implemented to prevent further spread of the disease. Protective clothing should be worn when dealing with clinically affected animals. Care should be taken that tack, buckets, or equipment is not shared between sick and unaffected horses. Horses showing no clinical signs should be separated from affected animals and monitored daily for fever and other signs. Each new case (e.g. fever, purulent nasal discharge, lymph node swelling) should be moved in with other affected horses. Contaminated surfaces and reusable equipment should be disinfected daily with bleach (1:10 to 1:256 dilution with water) or other appropriate disinfectant. Pastures and stalls where affected horses have had access should be kept unused for 4 weeks and 7 days, respectively, before allowing access to unaffected horses.

Antimicrobial therapy in classic cases of strangles is controversial. Horses presenting with already formed abscesses should not be treated with antibiotics. Abscess maturation may be promoted by application of hot compresses and mature abscesses may be lanced. Supportive therapy and administration of NSAIDs (flunixin meglumine, 1.1 mg/kg IV q12–24h; phenylbutazone, 2.2 mg/kg PO q12h) will help control pain, fever, and inflammation. The majority of affected horses recover from strangles without sequelae and will benefit from long-lasting immunity (>5 years). Therefore, letting the disease run its natural course is an option. Antimicrobials (drug of choice: procaine penicillin, 22,000 IU/kg IM q12h; alternatives: potentiated sulfonamides and ceftiofur) are recommended in severe cases and in horses with complications (e.g. airway obstruction, bastard strangles, purpura hemorrhagica). Alternatively, antimicrobial treatment may be implemented in early disease (fever) before lymph node enlargement occurs in order to stop disease progression. Unfortunately, determining the appropriate length of treatment is difficult and recurrence of disease may occur if therapy is discontinued too soon or the horse becomes reinfected before a strong immunity has had a chance to develop.

Treatment of GPE depends on the type of purulent material present. Local therapy should be accompanied by systemic administration of an antimicrobial for at least 2–3 weeks. In cases where there is pus, repeated lavage by infusion of warm isotonic solution via the biopsy channel of an endoscope or indwelling catheter is usually successful. The horse should be sedated to allow the head to drop down in order to facilitate drainage. Lavage should be continued as long as it is productive, which may require 1–3 liters of solution. Lavage sessions may be repeated 1–3 times per week until resolution of the empyema. Instillation of penicillin (5 million IU/GP) after lavage is completed improves the chances of a cure. The horse's head should be kept elevated for at least 15 minutes after instillation to maximize contact time. Mixing penicillin with gelatin acts as a slow release formulation. Gelatin (2 g) is mixed with 40 ml of sterile water and heated until dissolved. The solution is cooled to 45–50°C and mixed with 10 ml of penicillin solution containing 10 million IU to make a total of 50 ml. The solution is drawn into two syringes (25 ml with 5 million IU each) and cooled in a refrigerator (4°C) until used. The solution is stable for 7 days at 2–8°C. Topical therapy with acetylcysteine solution (40 ml of 20% w/v solution) may help loosen up purulent material in cases where pus or chondroids are tightly adhered to the wall. Similarly, the horse's head should be kept elevated after instillation to maximize contact time. Treatment should not be repeated too frequently (once or twice per week) because acetylcysteine is irritant to the GP mucosa. Alternatively, chondroids may be removed using a retrieval basket or biopsy forceps through the biopsy channel of an endoscope. Finally, refractory cases may be treated surgically (see Guttural pouch empyema, management/treatment, p. 112).

Treatment of purpura hemorrhagica should be aggressive with antimicrobials and immunosuppressive doses of dexamethasone (0.04–0.15 mg/kg IV q24h) until clinical signs improve. The dose of dexamethasone can be decreased 2 days after clinical signs have resolved and it should be continued for at least 7 days after. If the horse has recovered clinically, dexamethasone should be switched to an equipotent dose of prednisolone (1 mg dexamethasone ≈20 mg prednisolone) for another 2–3 weeks before being tapered down. Supportive therapy may include IV fluids to ensure diuresis while avoiding worsening of subcutaneous edema, and hydrotherapy on the legs alternating with pressure wraps and gentle controlled exercise.

Treatment of bastard strangles depends on the location of the abscess. The main goal of therapy is to allow drainage of the pus and control associated side-effects. For example, an abdominal abscess causing peritonitis may require exploratory laparotomy to allow drainage of the abscess and peritoneal lavage. In all cases of metastatic strangles, prolonged therapy with penicillin (22,000 IU/kg IM q12h or IV q6h) is recommended until clinical signs and hematology indicate resolution of infection and inflammation.

After an outbreak of strangles, follow-up management should focus on the detection of asymptomatic carriers. Nasal shedding of *S. equi* lasts up to 2 months after infection in most cases except in horses that become chronic carriers secondary to persistent GPE. Therefore, culture of a nasal swab or GP lavage fluid should be initiated 8 weeks following the last case of infection.

Key points

- Strangles is a highly contagious infection caused by *S. equi* and characterized by fever, swollen upper respiratory lymph nodes, and a purulent nasal discharge.
- In a few cases, the infection spreads to other sites such as the abdominal cavity, brain, and lungs ('bastard strangles').
- Nasal shedding of *S. equi* can last 3–8 weeks after clinical resolution and horses with GPE can shed for months.
- Antibiotic therapy is not recommended for horses with formed abscesses unless secondary complications have developed (e.g. respiratory obstruction, bastard strangles, or purpura hemorrhagica).

EPISTAXIS

Exercise-induced pulmonary hemorrhage

Definition/overview

EIPH is defined by evidence of blood in the airways during or shortly after exercise and where no other cause can be found. The blood may be either visualized directly at the nose (epistaxis; **165**) or within the tracheobronchial airways by endoscopy (**166**). Alternatively, pulmonary hemorrhage may be inferred from the presence of hemosiderophages or excess RBCs in TW or BAL fluid.

Etiology/pathophysiology

The most accepted mechanism of EIPH is pulmonary capillary rupture secondary to excessive transmural pressure in the pulmonary capillary circulation during strenuous exercise, resulting in extravasation of RBCs into alveoli. Strenuous exercise is also associated with large swings in alveolar pressure. Subatmospheric pressure in alveoli during each inspiration adds to capillary blood pressure and generates high transmural pressures. Capillaries rupture when wall stress exceeds wall strength, resulting in blood cells and plasma flowing into the pulmonary interstitium and eventually into alveoli. Exercising horses are known to achieve an extremely high pulmonary artery pressure (PAP) and left atrial pressure (LAP) compared with other mammals (mean PAP >120 mmHg and mean LAP >70 mmHg, respectively) resulting in an average pulmonary capillary pressure in excess of 100 mmHg. For example, pulmonary capillary stress failure occurs in isolated rabbit and dog lungs when transmural pressure exceeds 40 and 70 mmHg, respectively. Upper airway obstruction also results in increased transmural pressure and risk for capillary failure. Similar experiments conducted in isolated horse lungs indicate that pulmonary capillary failure occurs at significantly higher pressure (>75–100 mmHg). Experimental evidence *in vivo* indicates that significant EIPH occurs in horses exercising strenuously on a treadmill when pulmonary artery pressure exceeds 100 mmHg.

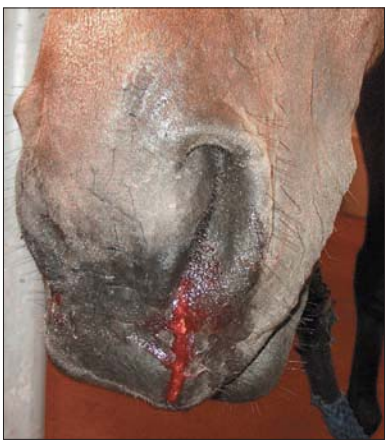
The reason why EIPH occurs preferentially in the caudodorsal lung region is still unclear. There is evidence that more blood flow is directed towards the caudodorsal lung region because of anatomic structure and differential vascular tone between the dorsal and ventral pulmonary vessels.

Pulmonary lesions associated with EIPH are hemosiderin accumulation, bronchiolitis, fibrosis, and bronchial arterial neovascularization. Recently, investigators have shown that vascular remodeling characterized by thickening of pulmonary veins occurs in regions where chronic bleeding is evident. They reasoned that repeated bouts of hypertension in the caudodorsal pulmonary artery vessels, associated with exercise, result in vein wall remodeling and subsequent pressure increase in pulmonary capillaries, thereby contributing to pulmonary capillary rupture. In turn, blood extravasation results in EIPH, hemosiderosis, and pulmonary inflammation. Chronic bleeding would lead to bronchial artery proliferation in response to the normal lung repair process as well as pulmonary fibrosis. Therefore over time, lesions would tend to progress, in particular in a cranioventral direction.

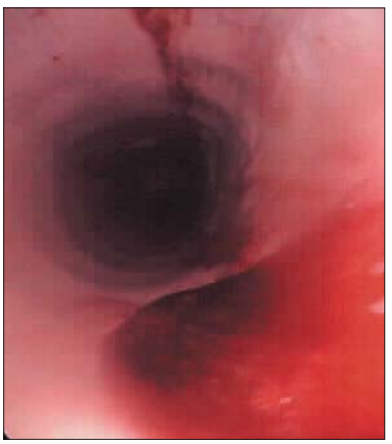
Some workers have proposed that EIPH results from a defect of hemostasis. Strenuous exercise in horses leads to activation of both coagulation and fibrinolytic pathways. However, abnormal pathway regulation in horses with EIPH has not been documented. Nevertheless, several racing jurisdictions have allowed the use of adjunct bleeder medications in combination with furosemide to treat horses with refractory EIPH. Some of the drugs reduce fibrinolysis (e.g. aminocaproic acid, tranexamic acid), while others act via an unknown mechanism (e.g. conjugated estrogens, carbazochrome). Few safety and efficacy studies have been conducted to establish the value of these drugs in treating EIPH in racehorses and so far none of the adjunct bleeder medications has demonstrated efficacy.

Others have hypothesized that airway inflammation may predispose horses to EIPH. Some studies suggest that experimentally induced neutrophilic pulmonary inflammation may be associated with pulmonary hemorrhage. Pathologic studies of EIPH have reported evidence of bronchiolitis and macrophagic inflammation in affected areas. However, field studies are controversial, with some epidemiologic studies reporting an association between mucus score and detection of blood and hemosiderophages in tracheal samples in racehorses, while other studies have not.

In some cases, atrial fibrillation is the cause of severe EIPH and epistaxis. The proposed mechanism is that atrial fluttering leads to incomplete emptying of the left atrium, resulting in increased pulmonary venous pressure and, in turn, elevated pulmonary capillary pressure. Cardiac auscultation and an electrocardiogram are essential to detect and treat those cases appropriately. Typically, EIPH resolves promptly after successful treatment of atrial fibrillation.



◀ **165 Horse with bilateral epistaxis secondary to exercise-induced pulmonary hemorrhage associated with atrial fibrillation.**



▶ **166 Exercise-induced pulmonary hemorrhage as evidenced by blood in the trachea visualized by endoscopy of the trachea (grade 3).**

Clinical presentation

The prevalence of epistaxis has been reported as between 0.15 and 6.8% worldwide, depending on the study. A large scale study conducted in Thoroughbreds racing in Japan reported a prevalence of 0.15% in a population of over 251,000 horses. The prevalence is slightly higher in steeplechasers and in older horses.

The prevalence of EIPH based on endoscopic evidence of blood in the trachea (**167**) after a race is 42–75% in Thoroughbreds, 25–77% in Standardbreds, and 62% in Quarter Horses. Thoroughbred and Standardbred horses subjected to endoscopy following two or more races exhibit EIPH more than 80% of the time. The prevalence of EIPH is lower in other disciplines such as barrel racing (30%), polo (11%), and cross-country (10%), and EIPH was not detected in one study involving endurance horses. The following grading system is commonly accepted because it shows excellent agreement between observers:

- **Grade 0:** no visible blood by endoscopy post exercise.
- **Grade 1:** one or more flecks of blood or 1–2 short (<¼ tracheal length), narrow (<10% tracheal surface area) streaks of blood.
- **Grade 2:** one long stream of blood (>½ tracheal length) or >2 streaks of blood (<⅓ tracheal surface area).
- **Grade 3:** multiple streams of blood (>⅓ tracheal surface area), with no blood pooling at the thoracic inlet.
- **Grade 4:** multiple, coalescing streams of blood (>90% tracheal surface area), with blood pooling at the thoracic inlet.

Cytologic evidence of hemorrhage in BAL fluid (e.g. erythrophagocytosis, hemosiderophages) is present in virtually all horses actively racing or in race training.

The most common complaint in horses with EIPH is poor performance. There is no doubt that severe (grade 4) EIPH negatively affects performance. However, the effect of mild or moderate EIPH (which is more common) on performance has been controversial for many years. Recently, a study conducted in 744 Thoroughbreds racing in Australia provided strong evidence that EIPH is associated with impaired performance. Investigators used an endoscopic EIPH scoring system based on the scale 0–4 (see above) with 'grade 4 indicating multiple coalescing streams of blood covering >90% of the tracheal surface with pooling of blood at the thoracic inlet'. They demonstrated that the distance finished behind the winner was associated with the grade of EIPH (i.e. the higher the EIPH grade, the farther the horse finished behind the winner).

Other studies have shown that horses exhibiting EIPH experience a more severe exercise-induced arterial hypoxemia than horses without EIPH. This mechanism may explain the negative impact of EIPH on performance.

Horses with EIPH may swallow repeatedly or cough after a race, although those signs are nonspecific. In such cases, red discoloration of the saliva coating the horse's tongue may be seen, presumably due to coughed-up blood present in the larynx and pharynx being mixed with saliva as the horse swallows. Often, trainers will say that the horse 'bit its tongue', but in fact it is likely due to EIPH. Severe EIPH may result in respiratory distress and, in rare cases, death.

Differential diagnosis

Epistaxis: GP mycosis; ethmoid hematoma; trauma/foreign body; infection (nasal passage, sinuses); thrombocytopenia/vasculitis; neoplasia. Bleeding from the lower airways: infection (pneumonia, abscess); neoplasia (hemangiosarcoma, lung tumor); trauma/foreign body.

Diagnosis

There are two main ways to confirm the presence of EIPH: endoscopy of the upper airways and examination of respiratory secretions collected by TW or BAL.

Endoscopy of the airways

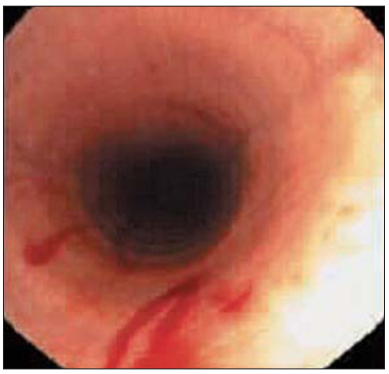
A 1-m flexible endoscope allows visualization down to the lower cervical region of the trachea (**167**). A 1.5-m endoscope is required in an average sized horse to be able to see the carina and entrance to the mainstem bronchi (**168**). Endoscopy of the respiratory tract is extremely valuable to confirm the origin of the bleeding and its cause, but timing of the examination is important. Visualization of blood in the trachea within 30 minutes to 2 hours post exercise is diagnostic for EIPH. Blood may still be visible 12 hours after a race and in some cases up to 1 week; however, if confirmation of the diagnosis is required beyond that time, it is preferable to collect respiratory secretions.

Estimation of the severity of EIPH is important since it has been linked to decreased performance.

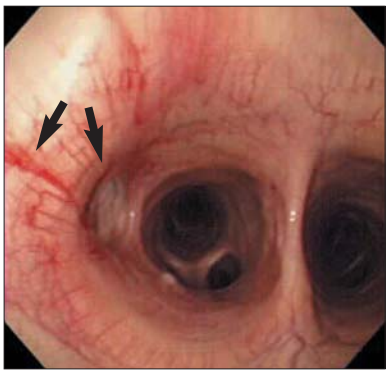
Examination of respiratory secretions

Sometimes, no blood is visible by endoscopy but BAL fluid will show obvious red discoloration (**169**).

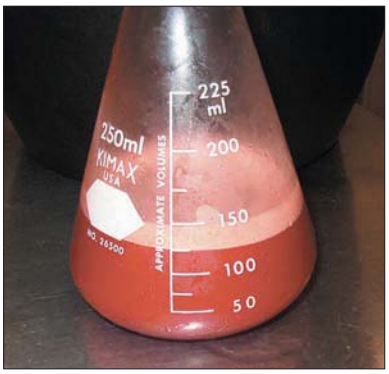
Detection of RBCs or macrophages containing phagocytized RBCs or breakdown product (hemosiderin) in TW or BAL fluid confirms EIPH (**170**). This technique is more sensitive than endoscopy since hemosiderophages may be detected for weeks following a single EIPH episode. The severity of EIPH is correlated with RBC numbers and the percentage of



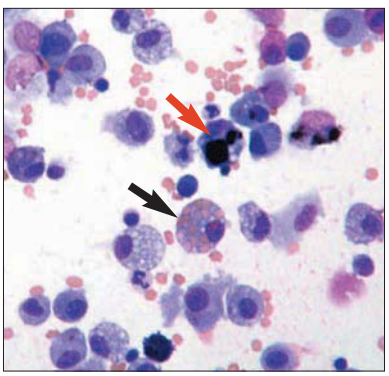
▲ **167** **Endoscopy of a horse with exercise-induced pulmonary hemorrhage 1 hour post treadmill exercise, showing bleeding of grade 2 severity.**



▲ **168** **Thin streaks of blood (arrows) are visualized at the entrance of the right mainstem bronchus.**



▲ **169** **Hemorrhagic bronchoalveolar lavage fluid collected from a horse that had raced 4 days previously, but had no blood visible by endoscopy.**



◀ **170** **Bronchoalveolar lavage fluid cytology from a horse with chronic exercise-induced pulmonary hemorrhage, showing free red blood cells, hemosiderophages with dark brown hemosiderin (red arrow), and many red blood cells phagocytized by an alveolar macrophage (black arrow). Wright's stain.**

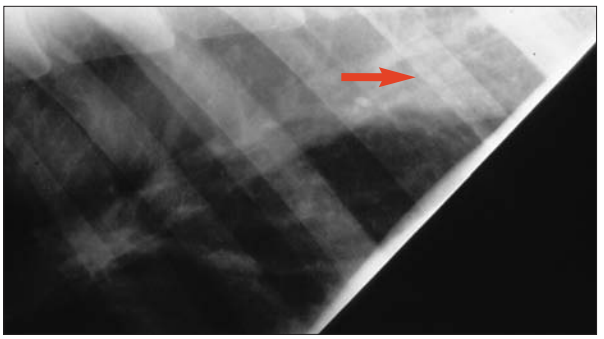
hemosiderophages in BAL fluid. In horses with poor performance associated with EIPH, hemosiderophages represent on average more than 30% of the BAL fluid alveolar macrophage population. However, the technique of quantification of EIPH in respiratory secretions has not been validated against tracheoendoscopy. EIPH is a regional disease affecting more commonly the caudodorsal lung area. Therefore, it is important to sample this region when performing a BAL; however, it is unclear how representative this sample is compared with the total amount of lung hemorrhage. The EIPH endoscopy score is at its maximum 30 minutes after strenuous exercise, but blood may be visible for several days after a severe bout of EIPH. Microscopic evidence of RBCs and hemosiderophages in BAL may be detected up to 14 days and 4 weeks after an episode of EIPH, respectively.

Diagnostic imaging

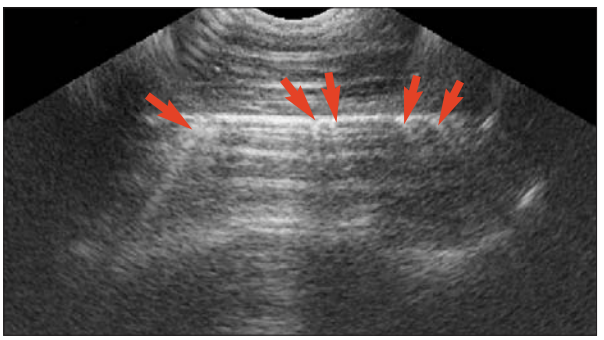
Chronic EIPH results in lung fibrosis, which may be detected as increased opacity in the caudodorsal lung region on lateral thoracic radiographs (**171**). Radiography is poorly sensitive, but it may help rule out other causes of pulmonary hemorrhage. Ultrasonography of the chest dorsally between the 10th and 17th intercostal spaces may reveal comet tail artifacts with high sensitivity but low specificity for EIPH (**172**). Other conditions resulting in accumulation of fluid in airspaces (e.g. infection, inflammation) can also be associated with comet-tail artifacts (see Chapter 4, Thoracic ultrasonography).

Other diagnostic tools

Horses with EIPH experience a more severe exercise-induced hypoxemia than healthy horses. However, the subtlety of the difference requires a well standardized exercise protocol and collection of arterial blood samples during exercise, which can only be done conveniently on a high-speed treadmill. Horses with other pulmonary diseases, such as IAD, experience a similar degree of exercise-induced hypoxemia. Therefore, detection of decreased Pa_{O_2} in arterial blood during exercise is not specific for EIPH, but it is a sensitive test.



▲ **171** **Lateral thoracic radiograph of a horse with chronic exercise-induced pulmonary hemorrhage showing increased lung opacities in the caudodorsal lung field (arrow).**



▲ **172** **Ultrasonography of the thoracic wall showing comet tail artifacts originating from the lung surface (arrows).**

Management/treatment

Possible approaches to reduce EIPH severity based on the various proposed mechanisms include decreasing transmural pressure in pulmonary capillaries, improving hemostasis, or treating airway inflammation.

Furosemide administration prior to exercise has been shown to decrease pulmonary capillary pressure by approximately 15–20%. Treadmill exercise tests have documented that this effect on blood pressure translates to a 60–70% decline in BAL fluid RBC concentration collected 1 hour post exercise. A randomized, crossover field trial reported that furosemide

administration (500 mg IV) 4 hours prior to racing was associated with an average decrease of the EIPH endoscopy severity score of 0.6 based on a scale of 0–4.

Nasal strips placed on the brim of the horse's nose are supposed to help prevent nasal valve collapse during high-speed exercise (**173**). Several treadmill studies have shown that application of a nasal strip results in a significant decrease in BAL fluid RBC concentration post exercise. The magnitude of EIPH reduction with nasal strips was less than that obtained with furosemide administration (1 mg/kg) and concurrent use of a nasal strip with furosemide did not result in an additional reduction in BAL fluid RBCs compared with furosemide alone.

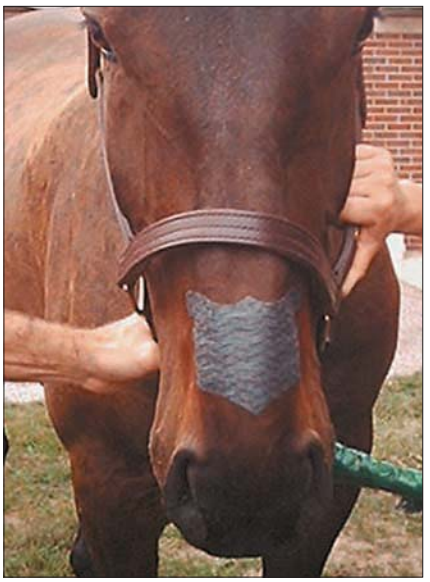
Carbazochrome subsalicylate, also known as 'Kentucky red', is used as an adjunct bleeder medication in a few racing jurisdictions in the US. In a randomized clinical trial, administration of furosemide (250 mg IV) in combination with carbazochrome (100 mg) 4 hours prior to a treadmill test did not significantly decrease BAL fluid RBCs after the exercise test compared with furosemide alone.

A randomized, crossover field trial was conducted in racing Thoroughbreds to compare furosemide administration (250 mg IV) with a furosemide (250 mg) –aminocaproic acid (5 g IV) combination. The investigators reported no significant different in BAL fluid RBC concentration collected post racing.

Several Chinese herbs are supposedly capable of mitigating EIPH, but available data do not support their efficacy. The effect of a mixture of Yunnan Paiyao (4 g q12h for 3 days) and Single Immortal (another herb) (50 g q12h for 3 days) was tested against a placebo in a randomized crossover trial design. No significant difference in RBC concentration was observed in BAL fluid collected 1 hour after treadmill testing.

A clinical trial including 10 Thoroughbred racehorses trained on a treadmill compared weekly administration of concentrated equine serum (Seramune®) with placebo. A significant decrease in BAL fluid RBC concentration was reported after 4 weeks of serum therapy; however, the effect was modest.

Pre-race administration of furosemide is the only therapy providing strong evidence that it decreases EIPH severity. Administration of furosemide is also associated with improved performance, which is likely due to the significant body weight loss resulting from urination (≈77 kg). The data reported above on other compounds should be considered preliminary and further research is needed to confirm or refute the effect of these drugs on EIPH.



▲ **173 Nasal strip applied over a horse's nose.**

Key points

- More than 80% of racehorses experience EIPH, but less than 1% show epistaxis.
- Performance is impaired in racehorses with an EIPH score of at least 2 out of 4, but minimal EIPH (score of 1) does not affect performance.
- The most sensitive diagnostic tools are upper airway endoscopy and BAL cytology following exercise.
- Administration of furosemide 4 hours prior to a race is the only treatment proven to decrease EIPH severity.

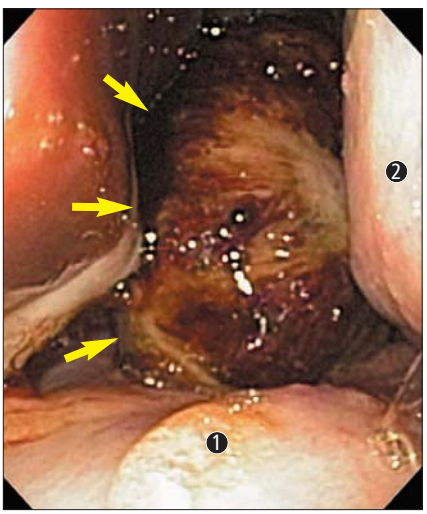
Ethmoid hematoma

Definition/overview

Ethmoid hematoma is a benign neoplastic growth (**174**), originating from the ethmoid turbinates, which causes epistaxis.

Etiology/pathophysiology

The etiology of ethmoid hematoma is idiopathic. Possible etiologies include chronic submucosal hemorrhage originating from the ethmoid turbinates or abnormal blood supply to the ethmoidal labyrinth. The majority of horses with ethmoid hematoma are affected unilaterally. However, bilateral cases of ethmoid hematoma occur in 16–35% of affected horses. Ethmoid hematoma typically affects middle-aged (>10 years old) geldings, with Arabians, Thoroughbreds, Quarter Horses, and Warmbloods being the most commonly affected breeds. No Standardbreds have ever been reported with this condition. Ethmoid hematomas continue to enlarge unless removed, although there have been anecdotal reports of spontaneous regression. Ethmoid hematomas typically originate from the ethmoid turbinates, but other anatomic locations include the sphenopalatine, frontal, or maxillary sinuses.



▲ **174 Endoscopic photograph of a large ethmoid hematoma (arrows) in the right nasal passage. (1) soft palate; (2) nasal septum.**

Clinical presentation

The most common presenting clinical sign is intermittent, but not severe, unilateral epistaxis. Other clinical signs of ethmoid hematoma include chronic nasal discharge, decreased airflow through the nasal passage, upper respiratory noise, exercise intolerance, yellow–green to brown mass present at the nares (**175**), and, less commonly, difficulty in passage of a nasogastric tube.

Differential diagnosis

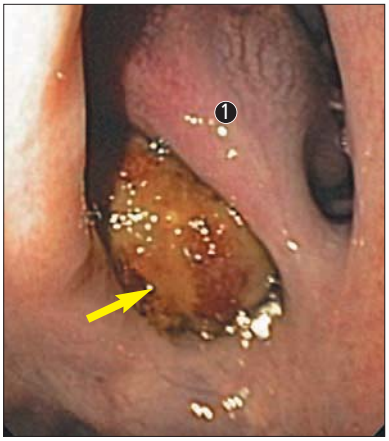
GP mycosis; EIPH; skull trauma; dacryohemorrhea (hemorrhage from the nasolacrimal duct); neoplasia involving the nasal passage, paranasal sinuses, GP, and trachea; nasal passage, pharyngeal, oral, or tracheal foreign bodies.

Diagnosis

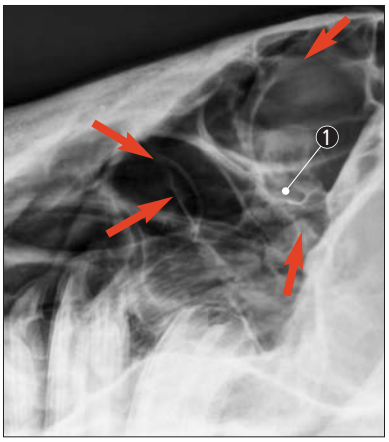
Clinical signs are important in the diagnosis of ethmoid hematoma, with the primary clinical sign being unilateral or bilateral epistaxis. The best diagnostic tool for confirmation of ethmoid hematoma is endoscopy (**176**). Endoscopic findings compatible with ethmoid hematoma include the presence of a pink to yellow–green mass originating from the ethmoid turbinates, mass obstruction of the nasal passage or a portion of the nasopharynx, and hemorrhage seen exiting the nasomaxillary opening.

Both nasal passages should be examined endoscopically because of the high incidence of bilateral, occult ethmoid hematoma. In addition to endoscopy, plain film or digital radiography is indicated to determine bilateral involvement or a hematoma originating within the frontal or maxillary sinuses (**177**). At a minimum, lateral, oblique, and dorsoventral radiographic views are recommended. CT and MRI can also be used to determine the location and extent of the hematoma within the paranasal sinsues prior to surgical management. Biopsy confirmation of ethmoid hematoma is not recommended because of the potential for profuse hemorrhage following the biopsy.

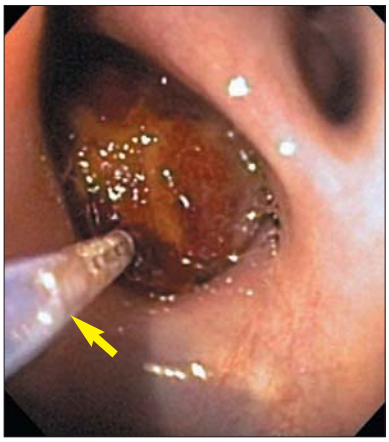
◀ **175** An unusually large ethmoid hematoma exiting the right nostril.



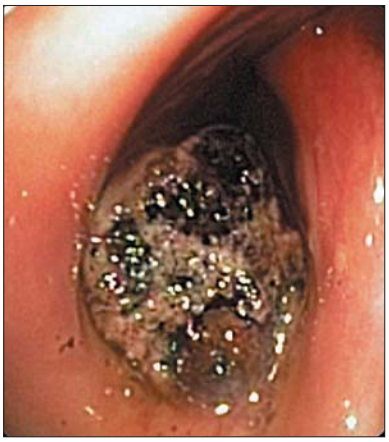
▶ **176** Endoscopic photograph of an ethmoid hematoma (arrow) ventral to the greater ethmoid turbinate bone (1).



▲ **177** Digital oblique radiograph of the left paranasal sinuses with an ethmoid hematoma (arrows) adjacent to the ethmoid turbinates (1).



▲ **178** Ethmoid hematoma being photovaporized with a non-contact diode laser (arrow).



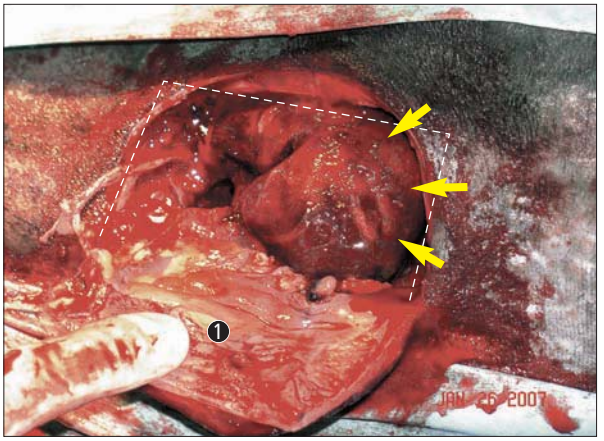
▲ **179** Ethmoid hematoma post-laser photovaporization. This will require multiple treatments to obtain a successful resolution.

Management/treatment

Medical therapy with multiple repeated intralesional injections of formalin has been used to cause regression of hematomas. However, the authors do not recommend the use of formalin because of the risk of brain necrosis following intralesional injection. Cases of meningitis have been reported following intralesional formalin injection because of the close approximation of the ethmoid hematoma to the cribriform plate. Surgery is the treatment of choice. Surgical therapies that have been used include Nd:YAG or diode laser photovaporization, frontonasal sinusotomy combined with laser excision, frontonasal sinusotomy combined with cryosurgery, and surgical resection alone. Laser photovaporization can be performed with the horse in a standing position (**178**). Large hematomas require multiple laser treatments for successful resolution (**179**). Laser treatments are typically performed on alternate days to allow for latent thermal necrosis to occur and are repeated as needed until the hematoma has resolved. Because of the multiple treatments

required and the prolonged hospitalization associated with this method, the authors only use standing laser photovaporization for small hematomas.

Large hematomas are best treated with the combination of laser excision of the hematoma and frontonasal sinusotomy. Before surgery, a cross-match should be obtained to find a suitable blood donor. This is necessary because of the risk of severe hemorrhage following surgical extirpation of the hematoma. In addition to the cross-match, IV fluids should be administered prior to and during anesthesia to increase the circulating blood volume. Hypertonic saline can be administered if necessary to boost circulating blood volume. A well prepared surgical team is also necessary to perform this procedure. A frontonasal osteoplastic bone flap is centered over the hematoma (**180**). Following elevation of the bone flap the hematoma is visualized. The base of the hematoma is identified and the diode or Nd:YAG laser is used to excise the mass in a contact fashion. The use of the laser in this fashion has been shown to lower the recurrence rate following surgical removal. Handheld laser scalpels are available to hold the flexible laser fiber during the procedure. If a laser is not available, the mass is removed with a combination of manual and mechanical extraction. Following removal of the mass the sinus is packed with sterile gauze packing and the sinusotomy is closed in a routine fashion. If the hematoma is located bilaterally, the horse is rotated to the opposite lateral recumbency and the procedure is repeated. A tracheotomy is necessary for bilateral lesions, because packing occludes both nasal passages. The aftercare for surgical removal of ethmoid hematomas includes administration of antimicrobials and anti-inflammatory drugs. The gauze nasal packing is removed on the third day postoperatively. By leaving the nasal packing for 3 days, problems with hemorrhage following removal of the packing are rare. Sinus lavage is sometimes necessary, depending on the degree of exudation from the nasal passage post surgery. Horses should be examined with endoscopy at 6-month intervals after surgery to evaluate for hematoma recurrence. The recurrence rate for bilateral lesions is between 30% and 50% while that for unilateral lesions using the Nd:YAG laser is 8%. The authors' experience supports the use of surgical lasers to lessen the recurrence rate following surgery.



▲ **180** **Intraoperative photograph of an ethmoid hematoma (arrows) following the creation of a frontal sinus osteoplastic flap (1). Note the position of the flap (dotted line) adjacent to the orbit.**

The prognosis for ethmoid hematoma is favorable, but recurrence is a problem. However, despite this impression, elimination of recurrence in all surgically treated horses is not always possible. Owners should be informed about the potential for recurrence and that it is very important to perform regular endoscopic examinations. If small areas of hematoma regrowth occur, they can be managed successfully with standing laser photovaporization. If regrowth is allowed to occur unchecked, repeat frontonasal sinusotomy may be required.

In conclusion, ethmoid hematoma is a benign form of neoplasia that originates from the ethmoid labyrinth. The primary clinical sign of ethmoid hematoma is mild epistaxis. The best method of management for large ethmoid hematoma is frontonasal sinusotomy combined with excision with a diode laser. Small ethmoid hematomas can be managed successfully with standing diode laser photovaporization. The prognosis for ethmoid hematoma is favorable but recurrence is a problem. Frequent monitoring after surgery is crucial to recognizing early signs of recurrence.

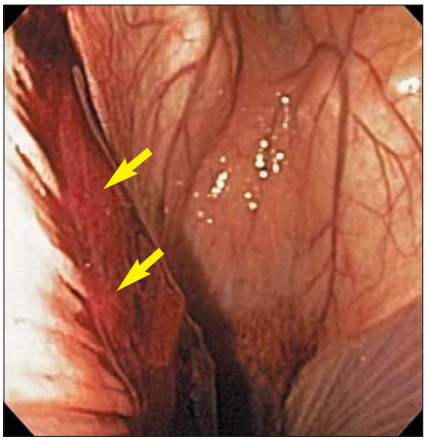
Key points

- The primary clinical sign of ethmoid hematoma is epistaxis.
- Endoscopy is crucial to a successful diagnosis.
- Surgical management provides the best method of achieving a successful outcome.
- Frequent monitoring following surgery is important in order to recognize recurrence.

Guttural pouch mycosis

Definition/overview

Guttural pouch mycosis (GPM) is the direct result of fungal invasion of the lining of the GP. Multiple clinical signs can be associated with GPM, but the most serious and life-threatening complication is epistaxis secondary to erosion of the vascular wall of the arteries located within the GP.



▲ **181** **Endoscopic photograph of blood exiting (arrows) the right guttural pouch in a horse with guttural pouch mycosis.**

Etiology/pathophysiology

GPM has no age, sex, or breed predisposition. The disease seems to occur more frequently in stabled horses during the warmer months of the year. GPM is more common in the UK than in the US and is more common in the northern hemisphere than in the southern hemisphere. The cause of the fungal invasion of the GP is unknown. A number of fungi have been isolated, but *Aspergillus nidulans* and *Aspergillus fumigatus* are the most commonly implicated. However, these organisms may not be the primary pathogens. They are ubiquitous and can be recovered from the GPs of normal horses. Once the fungus invades the lining of the pouch a diphtheritic membrane is formed, which is attached to the underlying tissue. It is usually composed of necrotic tissue and debris,with an irregular suface bearing a variety of bacteria. It may appear brown, yellow, black, or white and can be a discrete nodule or can cover the roof of the medial and lateral compartments. Histopathologic findings include invasion of fungal mycelia in underlying arteries, nerves, and other tissue within the pouch. Erosion of the wall of the internal carotid artery or external carotid artery (maxillary artery) leads to an aneurysmal dilatation and eventual rupture. Once rupture occurs hemorrhage ensues, which can be fatal or intermittent. The artery may become partially or totally occluded by thrombosis. Active inflammation may extend into adjacent bone (stylohyoid bone) and muscles, causing rupture of the ventral straight muscles of the head and fistulas between pouches or betweenthe pouch and the pharynx. The other potentially life-threatening complication of GPM is dysphagia secondary to nerve damage to the 9th, 10th, and 12th cranial nerves within the guttural pouch.

Clinical presentation

Epistaxis is the most common presenting clinical sign (**181**). Epistaxis can be severe and there are cases where hemorrhage has occurred intermittently over several weeks before the fatal hemorrhage occurred. In addition to epistaxis, mucoid to mucopurulent, unilateral nasal discharge can occur. Dysphagia is the second most common clinical sign. Other clinical signs associated with GPM include parotid pain, abnormal head posture, head shyness, respiratory noise, sweating, and shivering, Horner's syndrome, corneal ulcers, colic, facial nerve paralysis, tongue paralysis, and otic discharge. These signs will vary with the structure involved within the GP. For example, one of the authors has seen a horse with GPM where the only clinical sign at presentation was otic discharge.

Differential diagnosis

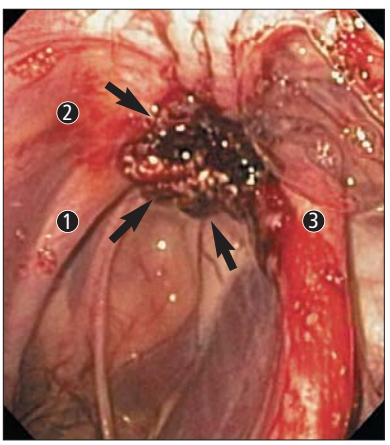
EIPH; ethmoid hematoma; rupture of the ventral straight muscles of the head; skull trauma; dacryohemorrhea (hemorrhage from the nasolacrimal duct); neoplasia involving the nasal passage, paranasal sinuses, GP, and trachea; nasal passage, pharyngeal, oral, or tracheal foreign bodies.

Diagnosis

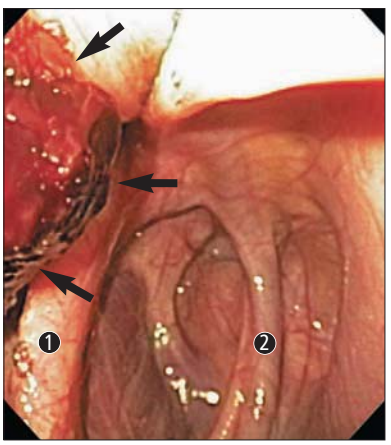
Diagnosis of GPM is based on history and clinical signs on presentation. An endoscopic examination is required to confirm the diagnosis (**182**, **183**, **184**). Endoscopic findings compatible with GPM include blood draining from the pharyngeal opening of the GP, food material in the nasopharynx or trachea, collapse of the roof of the pharynx, DDSP, and laryngeal hemiplegia. In some cases the GP can be entered, but the involved structures causing hemorrhage cannot be visualized because of the large hematoma located within the pouch. Gentle endoscopic lavage can be performed to aid evacuation of the hematoma from the pouch. Obviously, care must be taken with this procedure to avoid disruption of the thrombus from the bleeding artery. If possible, the interior of the GP should be searched for the location of the fungal mycelia. This is necessary to aid surgical planning. It is very important to identify the source of hemorrhage. The surgical technique varies with the location of the plaque. Surgical occlusion of the internal carotid artery is very different to occlusion of the external carotid or maxillary artery. If the source of the hemorrhage cannot be determined endoscopically, the internal and external carotid and maxillary arteries should be occluded to eliminate the risk of fatal hemorrhage following surgical treatment.

Management/treatment

Medical and surgical management can be performed. The authors recommend that all horses with GPM are treated surgically. This is the only way to prevent fatal hemorrhage from the affected artery. Medical management can be effective, but by the time antifungal treatment has resolved the infection, the horse may have suffered a fatal hemorrhage.



▲ **182** **Endoscopic photograph of guttural pouch mycosis (arrows) involving the internal carotid artery (1) in the left guttural pouch. (2) medial compartment; (3) stylohyoid bone.**

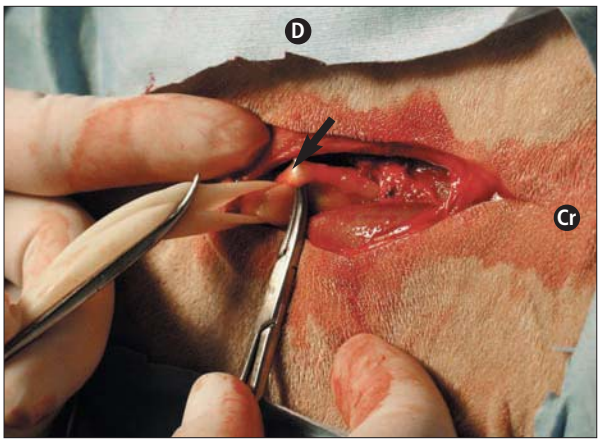


▲ **183** **Endoscopic photograph of a fungal plaque on the maxillary artery (arrows). (1) stylohyoid bone; (2) internal carotid artery.**

▲ **184** **Endoscopic photograph of a large blood clot and fungal plaque secondary to guttural pouch mycosis. The presence of such a large clot makes determination of vessel involvement challenging without angiography.**

Medical management involves the systemic or topical delivery of antifungal drugs. Daily endoscopic infusions are performed for 4–6 weeks, but the response is slow. Solutions of betadine, gentian violet, formaldehyde, thiabendazole, amphotericin B, natamycin, nystatin, and miconazole have all been used. Other medical therapies include systemic antifungal drugs, corticosteroids, NSAIDs, and antimicrobials. The response to medical therapy is slow and does not prevent fatal hemorrhage.

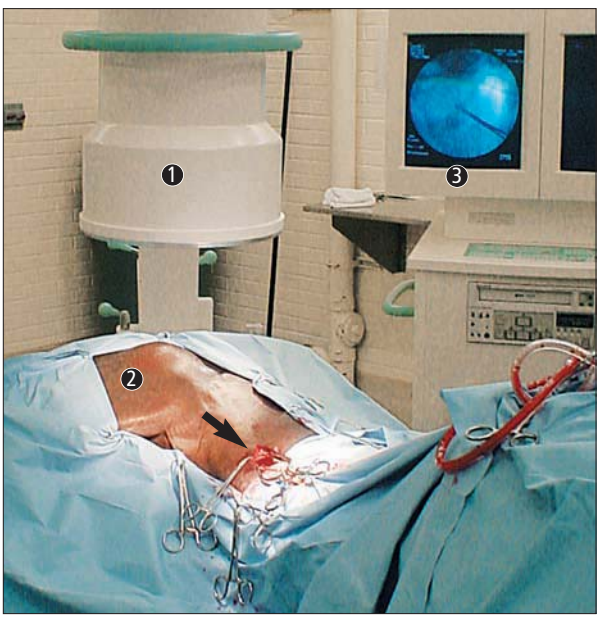
Surgical therapy provides the best option for a successful outcome. The primary goal of surgical management of GPM is to prevent fatal hemorrhage. This is best achieved by occluding the offending vessel on the cardiac and non-cardiac sides of the vessel. This is necessary because of retrograde blood flow from the cerebral arterial circle. Occlusion of the affected artery on the cardiac side only will not prevent fatal hemorrhage. It has been shown experimentally that occlusion on the cardiac side only does not significantly lower the arterial blood pressure from the artery. That said, successful management of GPM with occlusion of the artery on the cardiac side only has been reported. If this approach is taken, the surgeon must be willing to accept the risk of arterial hemorrhage still occurring following simple arterial ligation. This approach may be successful if the horse is fortunate enough to form a thrombus, which is maintained following cardiac side ligation. The other benefit of surgical management is the recognition of anomalous arterial vessels, which is important because the wrong arterial vessel may be occluded by the surgeon. The best way to evaluate the arterial circulation is to perform an angiogram prior to surgery, but this is not always available.



▲ **185** **Intraoperative photograph of exposure of the common carotid artery (Penrose drain) and recurrent laryngeal nerve (arrow). (D) dorsal; (Cr) cranial.**

Surgical correction can be performed via open or minimally invasive approaches. Open surgical approaches have been described in detail by other authors. The authors' preferred surgical method of treating GPM is fluoroscopic placement of nitinol vascular plugs on each side of the vascular defect secondary to fungal invasion of the vascular wall. To perform vascular plug placement the horse is anesthetized and positioned in lateral recumbency with the affected side up. A fluoroscope is necessary to perform this procedure as the entire technique is carried out with angiographic techniques. Following induction an endoscope is positioned in the affected GP. Placement of the endoscope into the affected GP allows direct visualization of the affected artery and helps confirm successful catheter placement. The common carotid artery is then exposed with cut-down just dorsal to the jugular vein in the proximal aspect of the neck. The common carotid artery (CCA) is isolated from the carotid sheath. It is important not to damage the recurrent laryngeal nerve adjacent to the CCA iatrogenically. The CCA is isolated with a ½-inch Penrose drain (**185**).

An introducer catheter is inserted into the CCA. Contrast media is then injected through the catheter to identify the bleeding vessel (**186**). Angiography is very important because it allows for precise placement of the vascular plug in the affected artery. This avoids the complications that have been associated with open techniques in which anomalous vessels have been catheterized. Following the injection of contrast media, the dye can be seen exiting the defect in the affected vessel. Once the affected artery is identified, angiographic catheters are inserted to deploy the vascular plug. First, a vascular plug is inserted on the brain side (non-cardiac side) of the affected artery. Once this plug has been deployed a second plug is deployed on the cardiac side of the lesion. Once both plugs have been positioned, contrast media is again injected to make sure the affected segment of vessel has no blood flow. In one horse treated by the authors a vascular plug could not be deployed on the non-cardiac side of the affected vessel. This horse was treated by implantation of a Fogarty venous thrombectomy catheter on the non-cardiac side of the vessel. Therefore, surgeons should be comfortable with and knowledgeable of traditional surgical techniques. Once successful plug placement has been confirmed, the CCA arteriotomy site is occluded with a purse-string suture and the cut-down is closed routinely.



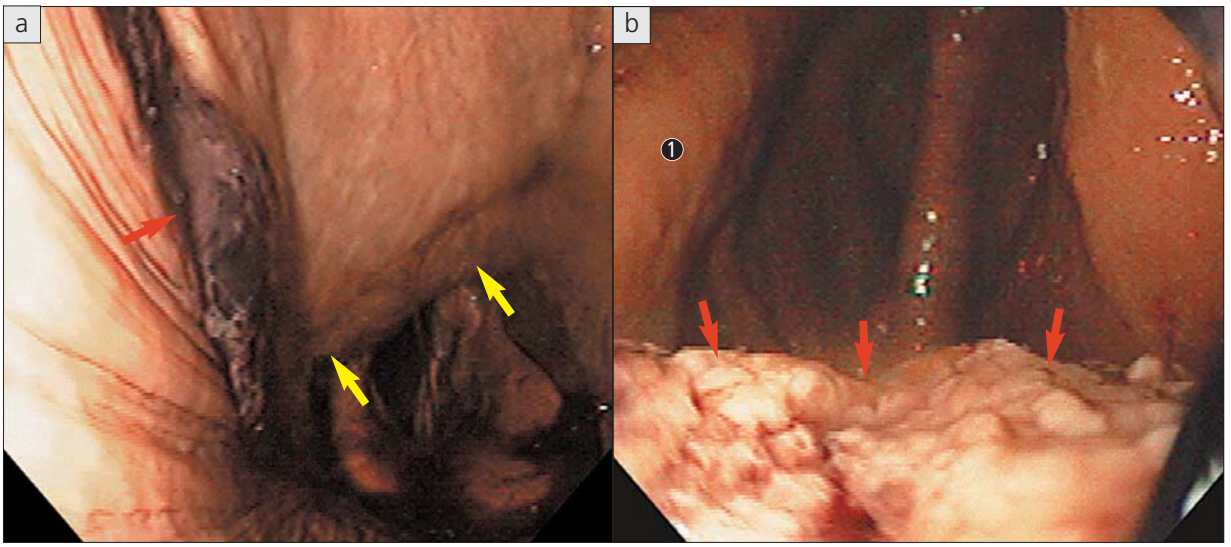
◀ **186 Set-up for angiography and vascular plug placement in a horse with guttural pouch mycosis. Note the fluoroscope (1) on the dorsal side of the head (2), the video monitor for angiographic imaging (3), and the exteriorization site for the common carotid artery (arrow).**

It is the authors' impression that horses treated with the described minimally invasive technique recover faster and with fewer complications than have been reported with open surgical techniques. Once blood flow to the affected artery has been occluded, the fungal infection resolves without specific therapy. It appears that with the elimination of blood flow to the site of fungal infection the fungus is 'starved' and infection resolves. Subjectively, fungal hyphae have resolved at a faster rate with vascular plug placement as opposed to transarterial catheter placement. The prognosis for GPM treated with surgery is good to excellent. The one complication that does worsen the prognosis is the presence of dysphagia. Horses with GPM and dysphagia have a guarded prognosis.

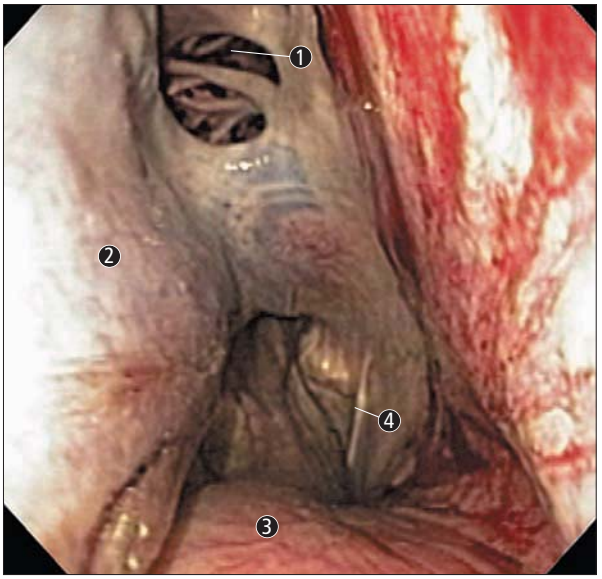
Complications of GPM following surgery include fatal hemorrhage with single ligation of the internal carotid artery, incisional infection associated with the surgical site, catheterization of the wrong vessel leading to hemorrhage, laryngeal hemiplegia (which may be permanent), and dysphagia. Horses can recover from Horner's syndrome and facial nerve paralysis.

Key points

- The most common presenting clinical sign of GPM is mild to severe epistaxis; this can be life threatening.
- In addition to epistaxis, a variety of other clinical signs have been associated with GPM. These include dysphagia, nasal discharge, laryngeal hemiplegia, and otic discharge.
- The best method for treating GPM is surgery. The risk for fatal hemorrhage while being treating conservatively is high as medical therapy does not work quickly enough to lower the risk of fatal hemorrhage.
- The prognosis for GPM treated with prompt, surgical treatment is good to excellent. The prognosis of GPM combined with dysphagia is guarded.



▲ **187** **(a) Endoscopic photograph of epistaxis originating from the guttural pouch in a horse with squamous cell carcinoma within the pouch. Note the blood clot (red arrow) exiting the right guttural pouch and the ventral enlargement of the guttural pouch into the nasopharynx (yellow arrows). (b) Note the presence of the squamous cell carcinoma on the floor of the right guttural pouch (arrows). (1) stylohyoid bone.**



◀ **188** **Endoscopic photograph of a horse with hemorrhage from the paranasal sinuses secondary to skull trauma. (1) ethmoid turbinates; (2) nasal septum; (3) soft palate; (4) pharyngeal opening of left guttural pouch.**

Miscellaneous causes of epistaxis

Other causes of epistaxis include neoplasia involving the paranasal sinuses, nasal passage and GPs (**187**), and trauma (**188**).

MILK

Cleft palate

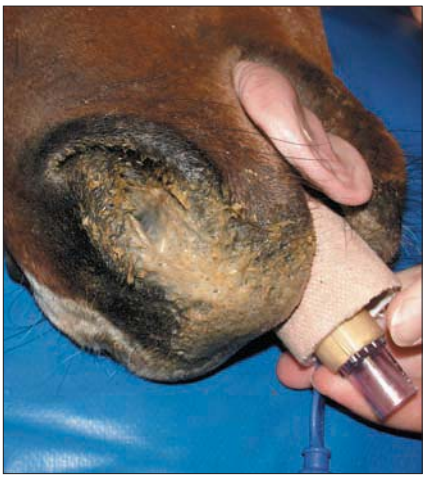
Definition/overview

Cleft palate, or palatoschisis, is a congenital defect that can involve the hard palate and/or the soft palate. The most common presenting clinical sign is nasal discharge of milk.

Etiology/pathophysiology

Cleft palate is typically seen in very young foals. Affected foals have a history of nasal discharge of milk soon after birth. There is no known breed or sex predilection. Cleft palate results from abnormal embryologic fusion of the palatal folds. This process takes place on the midline in a rostral to caudal direction and should be complete by the 47th day of gestation. The cleft may involve the hard and soft palates. The majority of affected animals have soft palate defects only, but it is not uncommon for the cleft to extend into the caudal aspect of the hard palate. The heritability of this condition is not known.

▼ **189 Foal with cleft palate with the classic clinical signs of nasal discharge of milk and feed material.**



Clinical presentation

The most common clinical sign of cleft palate is dysphagia with nasal discharge of milk (**189**).

Foals with dysphagia frequently have clinical signs of aspiration pneumonia, including coughing, tachypnea, pyrexia, and abnormal lung sounds (e.g. crackles and wheezes). Foals with deviation of the premaxilla (wry nose) should always be evaluated for the presence of a cleft palate.

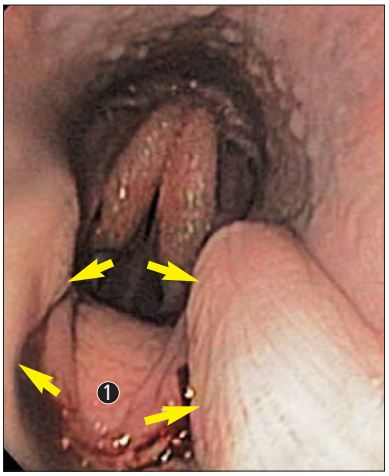
Differential diagnoses

Hyperkalemic periodic paralysis (HYPP); dysphagia unrelated to the cleft palate; GPE; DDSP; idiopathic dysphagia.

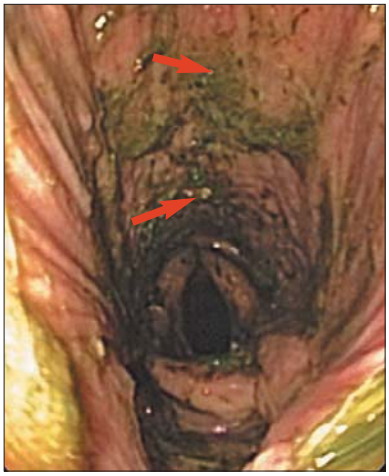
Diagnosis

Clinical signs should always be taken into account when evaluating a foal with nasal discharge of milk. Oral examination with a mouth speculum and a light source can allow for visualization of most hard palate and some soft palate defects. Endoscopy is required to fully visualize the soft and hard palates and to determine the severity of the defect. Endoscopic findings can include clefts of the soft palate only (**190**) or clefts of the soft and hard palates together. Endoscopic findings compatible with cleft palate include food located within the nasal passage (**191**) and visualization of the oropharynx through the cleft. Some horses with cleft palate have concurrent aryepiglottic fold entrapment (**192**).

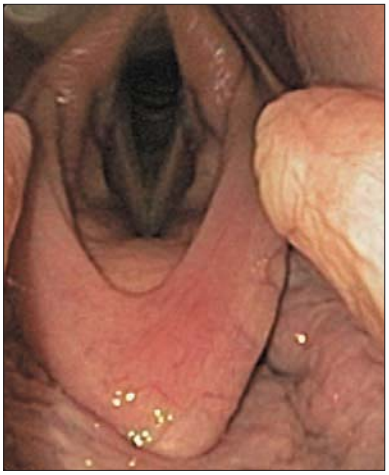
To determine whether or not surgical correction is possible an evaluation must be made on whether or not there is adequate tissue present to allow for a tension-free closure of the defect. In addition to endoscopy, all foals with a cleft palate should be evaluated for aspiration pneumonia. The work-up for aspiration pneumonia should include a complete blood count, a biochemistry profile, auscultation of the heart and lungs, and lateral thoracic radiographs. A transtracheal wash is recommended to determine which pathogens are the cause of the pneumonia in cases refractory to therapy.



▲ 190 **Foal with a cleft soft palate (arrows). (1) epiglottis.**



▲ 191 **Endoscopic photograph of feed material (arrows) within the trachea of a foal with a cleft soft palate.**



▲192 **Endoscopic photograph of a foal with a combination of cleft soft palate and epiglottic entrapment. Note the oral mucosa visualized ventral to the epiglottic tip and the cleft soft palate.**

Management/treatment

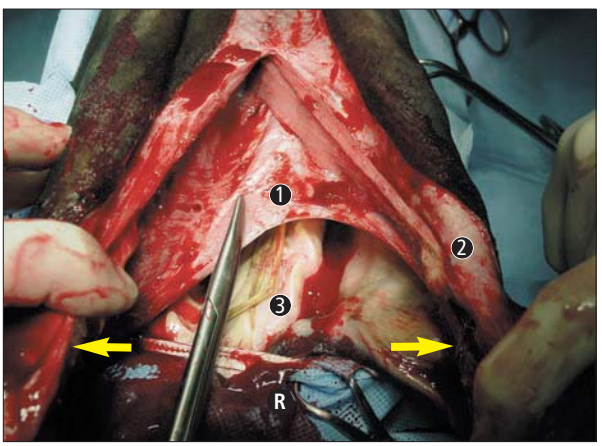
If the palate defect is excessively wide or if it appears there is not enough palatal tissue to perform surgical repair, euthanasia should be considered. The primary reason for this recommendation is that the risk of fatal aspiration pneumonia is very high in these cases. Conservative management of a cleft soft palate is frequently unsuccessful. However, the authors have seen three horses that have grown to adulthood before the diagnosis of cleft palate was made. All of these horses were somewhat small for their age and had a chronic nasal discharge containing feed. Interestingly, none had clinical signs of aspiration pneumonia.

Surgical management provides the best chance for a favorable outcome. Surgical correction can be attempted in valuable foals, foals that have emotional value, or in cases where the goal is to improve the quality of life. Surgery should be considered only as a salvage procedure as there is little likelihood that the horse will be athletically useful, because the laryngopalatal junction is likely to be unstable. The success rate of surgery is no better than 50%. Owners should be counseled about a realistic prognosis for surgical treatment and should be well informed of the potential postoperative complications, including failure of the surgical repair. Once the owner has decided to proceed with surgery, it should be performed as soon as possible after birth. This is because surgical correction is less complicated in the younger foal, mainly because the head is not particularly large. The larger and older the foal, the greater the disadvantage the surgeon operates under. A larger head makes surgical access to the caudal aspect of the soft palate difficult.

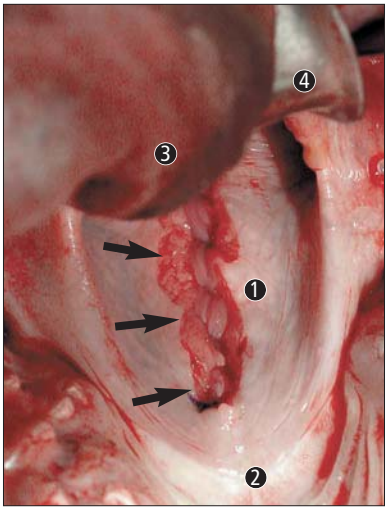
The surgical approach of choice is a mandibular symphysiotomy (**193**). Together with splitting of the lip on the midline, this provides the best surgical access to the hard and soft palates. In addition to the mandibular symphysiotomy, a laryngotomy is required to aid in elevation of the tongue and to position stay sutures at the caudal aspect of the soft palate. To provide the best chance for a successful outcome, the following four goals should be achieved by the surgeon:

- First, the soft palate must be reconstructed with minimal tension. Excessive tension on the soft palate repair ultimately results in partial or complete failure. The key to resolving this problem is that there must be adequate soft palate tissue to appose. The lack of pre-existing soft palate usually results in failure.
- Second, the soft palate should be reconstructed so that after it has been sutured it contacts the epiglottis. The further the soft palate can be sutured under the epiglottis the better the outcome will be. Unfortunately, this is not easy for the surgeon to accomplish because of the limited access to the caudal portion of the soft palate. Even with long handled surgical instrumentation this is the most difficult part of the surgical procedure.
- Third, a three-layer closure is required to repair the soft palate without tension. If a hard palate defect is present, mucoperiosteal flaps are created to bridge the defect.
- The final key to a successful outcome is surgical reconstruction of the symphysiotomy site. Alignment of the mucous membrane of the mouth and the muscle layers is straightforward. However, mandibular symphysis reconstruction is crucial. The authors recommend that the mandibular symphysis is repaired with two 6.5-mm fully-threaded cancellous screws in lag fashion. Washers should be placed on the screws to lessen the chance for fracture of the bone around the screw head. Finally, an 18-gauge wire should be placed around the central incisors to complete the repair. In addition to repair of the symphysis lip reconstruction should be performed very carefully. To reconstruct the lip the oral mucous membrane is closed first in a simple continuous pattern (**194**). Next, the fibrous connective tissues of the lip are sutured in multiple layers of simple interrupted sutures. Finally, the skin of the lip is closed in an interrupted vertical mattress pattern (**195**). The authors have found that if the mandibular symphysis repair is stable, the lip will generally heal without incident as long as t he lip is sutured as described (**196**).

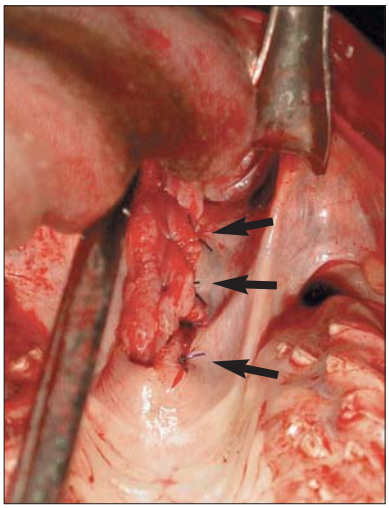
Complications of cleft soft palate repair include dehiscence of the soft/hard palate surgical repair, dehiscence of the lower lip, instability of the mandibular symphysis, mandibular osteomyelitis, incisional infection, hypoglossal nerve damage, and chronic dysphagia.



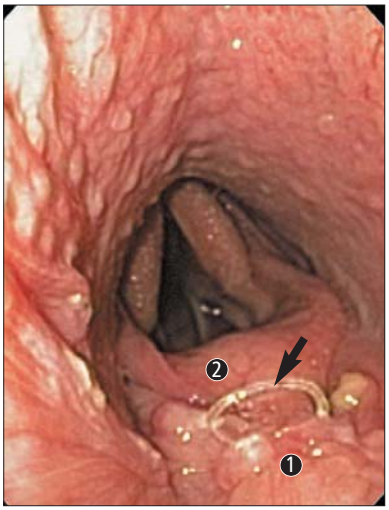
◀ **193** **Intraoperative photograph of mandibular symphysiotomy (arrows) and incision into the mucous membrane of the mouth (1). (2) right mandible; (3) hard palate; (R) rostral.**



▲ 194 **First layer closure (arrows) of a cleft soft palate (1). (2) hard palate; (3) tongue; (4) Kelly retractor for tongue.**



▲ 195 **Second layer closure of a cleft soft palate with vertical mattress sutures (arrows).**



▲ 196 **Postoperative endoscopic view of sutured cleft palate 60 days following surgical repair. Note the loop of suture material (arrow) above the dorsal surface of the soft palate (1) and the epiglottis (2) contacting the border of the soft palate repair.**

Dysphagia is observed if a portion of the palate repair fails, a palatal fistula develops, or if the soft palate is not sutured so that it contacts the epiglottis. It is also possible for the palate to be successfully repaired, but aspiration pneumonia leads to an unsuccessful outcome. Owners should be warned that some horses do not grow to the same size as their peers following surgical repair. If the palate repair is not successful, repeat surgery is not recommended because it is not very likely that a successful outcome will be achieved. Therefore, owners should be informed prior to surgery that only one attempt at surgical repair will be performed. Owners should also be counseled against breeding of horses affected with cleft palate.

The prognosis for symmetrical, midline soft palate defects ranges from 50 to 60%. Repairs of the hard and soft palates have no more than a 20% success rate.

Key points

- Cleft palate is most commonly diagnosed and treated in the young foal.
- Aspiration pneumonia is common and should be evaluated prior to deciding on surgical correction.
- Conservative management is frequently unsuccessful and not recommended.
- Surgical repair of the palate provides the best chance of improving the quality of life, but is not likely to result in an athletically useful animal.
- Mandibular symphysiotomy provides the best access to the palate and is the recommended surgical approach.
- Surgical experience with the technique is the most important factor leading to a successful outcome.
- The complication rate even with surgery is high and owners should be given a realistic prognosis prior to surgical correction.

Dorsal displacement of the soft palate

Definition/overview

DDSP occurs when the soft palate becomes oriented dorsal to the epiglottis. DDSP has been well described in the older (>2 years) horse. DDSP in foals is not common and instead of having the primary clinical signs of upper respiratory noise and exercise intolerance, foals typically present with signs of dysphagia and nasal discharge of milk or feed.

Etiology/pathophysiology

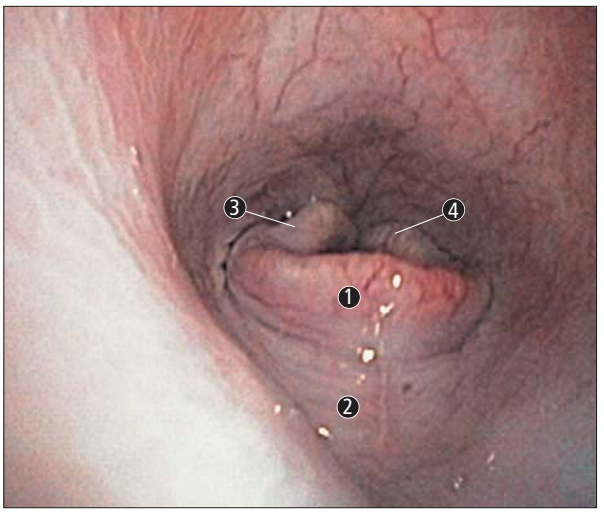
Nasal discharge of milk in foals with DDSP affects predominantly neonatal foals, with the oldest reported cases being 14 days old. The cause of DDSP in foals is idiopathic. Some have speculated that it occurs either because of primary pharyngeal or palatal muscular activity or is secondary to redundancy of the soft palate. Regardless of the cause, DDSP in foals, especially if persistent, results in nasal discharge of milk and, potentially, aspiration pneumonia.

Clinical presentation

Foals with DDSP typically have nasal discharge of milk or other feed material. Affected foals may also be dyspneic and tachypneic, depending on whether they are suffering from upper respiratory obstruction and/or aspiration pneumonia. Some foals may be observed coughing. All foals should be evaluated for clinical signs compatible with septicemia, including tachycardia, delayed capillary refill time, congested or dark mucous membranes, and ecchymotic or petechial hemorrhages on the conjunctiva or the inner surface of the pinna.

Differential diagnosis

Persistent epiglottic frenulum; subepiglottic cyst; cleft palate; idiopathic dysphagia; failure of passive transfer with septicemia; GPE or tympany; bronchopneumonia; HYPP; vitamin E and selenium deficiency.



▲ **197** **Foal with dysphagia secondary to dorsal displacement of the soft palate. (1) epiglottis; (2) soft palate; (3, 4) left and right arytenoid cartilages.**

Diagnosis

Foals should have a complete blood count, biochemical profile, and IgG level performed to evaluate for signs of infection or failure of passive transfer. Any foal with nasal discharge of milk should have an endoscopic examination performed (**197**). Because of the potential for a cleft palate in this age of foal, the entire length of the hard and soft palates should be examined. Foals with DDSP typically have persistent (soft palate always displaced) or intermittent (soft palate occasionally returns to normal position) displacement. Some foals may experience respiratory difficulty and stridor during the examination if the palate is persistently displaced. The trachea should be evaluated for the presence of aspirated milk. Lateral thoracic radiographs should be obtained to evaluate for signs of aspiration pneumonia. Lateral radiographs of the skull are useful in subjectively evaluating the thickness of the soft palate and abnormal fluid accumulation in the paranasal sinuses or GPs. Finally, a transtracheal wash is recommended for cytology and microbial culture to guide future antimicrobial therapy.

Management/treatment

All foals with DDSP should be treated with IV antimicrobials to treat aspiration pneumonia or bronchopneumonia. The following antimicrobials should be considered: penicillin G, gentamicin sulfate, amikacin sulfate, trimethoprim sulfamethoxazole, and metronidazole. Anti-inflammatory treatment with flunixin meglumine is also recommended. Prophylaxis against gastric ulceration is also recommended. IV fluid therapy, total or partial parenteral nutrition, and treatment for failure of passive transfer may be administered if necessary. If severe problems with milk or feed aspiration are observed, total parenteral nutrition or feeding via a nasogastric tube is recommended to lessen the risk of aspiration pneumonia.

It has been the experience of the authors and others that DDSP in foals should be handled initially with aggressive medical management. Most foals will respond to medical management, and clinical signs of DDSP resolve without surgical management. A temporary tracheostomy may be required for foals experiencing marked increased respiratory effort and stridor. Surgical management of DDSP in foals has been reported, but the authors feel that this should be reserved for cases of DDSP that do not respond to medical management. They recommend a minimum of 7 days of treatment before considering surgery. Of the two cases of DDSP in foals treated with surgery, both had long-term issues with either respiratory noise or nasal discharge of feed from the nostrils. Many options for surgical treatment of DDSP exist, but both of the two reported cases were treated with staphylectomy. In the largest published case series of foals with DDSP, all cases resolved with medical management within 4 days of the onset of clinical signs. Based on the limited case numbers available, the prognosis for foals with DDSP appears good with medical management.

Key points

- DDSP in foals can cause nasal discharge of milk.
- The cause of DDSP in foals is idiopathic, but it may be due to neuromuscular dysfunction of the soft palate or be secondary to redundant soft palate tissue.
- Most cases of DDSP should be treated medically, with surgical management only chosen for cases non-responsive to medical management.
- The prognosis is favorable with medical management. Reported cases treated with surgical management have had long-term complications.

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CHAPTER 7

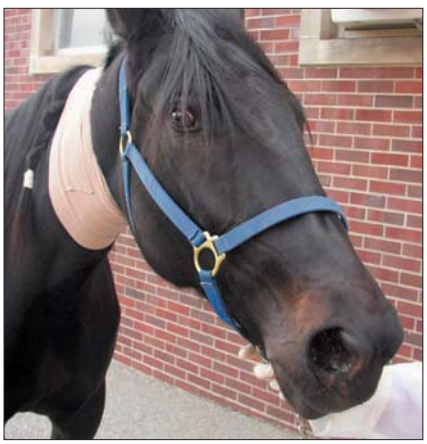
THE HORSE WITH INCREASED RESPIRATORY EFFORT

INTRODUCTION

Respiratory distress or labored breathing is characterized by exaggerated breathing effort relative to the degree of physical activity or physiologic demand. Dyspnea is defined as 'undue awareness of breathing or awareness of difficulty in breathing'. This term is therefore inappropriate for describing veterinary patients and the terms labored breathing, increased respiratory effort, or respiratory distress are preferred. Labored breathing can result from impaired gas exchanges secondary to obstruction of conducting airways, failure of the muscles and/or structures responsible for ventilation, pulmonary disease, or cardiac disease. Increased respiratory effort in response to pain, hyperthermia, and metabolic acidosis is not associated with gas exchange abnormalities. Diseases causing a decline in blood oxygen content (e.g. anemia) do not usually stimulate breathing except in cases of severe anemia when tissue hypoxia results in increased anaerobic metabolism and lactic acidosis.

Breathing is controlled by several mechanisms that regulate delivery of air to the alveoli (i.e. ventilation) to match metabolic demand. The respiratory center located in the brainstem responds to information originating from central and peripheral receptors and in turn modulates the activity of the respiratory muscles. As a result, P_{aO_2} and P_{aCO_2} are maintained within tight limits. An understanding of the basic control mechanisms and the mechanics of breathing is helpful to diagnose and treat the causes of respiratory distress.

Clinical signs associated with labored breathing in the horse may include inactivity, exercise intolerance, restless movements, anxious expression, flaring of the nostrils, increased thoracic and abdominal movements, asynchrony between thoracic and abdominal movements, extended head and neck, cyanosis, and pumping of the anus synchronized with the respiratory cycle (**198**). In addition, affected horses are anorectic and over time experience weight loss. Careful thoracic auscultation may reveal some important clues and allow differentiation between various causes of respiratory distress. For example, auscultation of increased breath sounds or abnormal breath sounds beyond the normal boundaries of lung auscultation is consistent with lung overinflation and is almost pathognomonic for RAO. Decreased breath sound intensity over the ventral thoracic area is suggestive of pleural effusion. A diffuse increase in breath sound intensity over the usual area of lung auscultation in a horse with marked increased respiratory effort is suggestive of interstitial pneumonia.



▶ **198 A horse with severe pleuropneumonia exhibiting respiratory distress characterized by nostril flaring, neck and head extension, and an anxious expression.**

Pleuropneumonia

Definition/overview

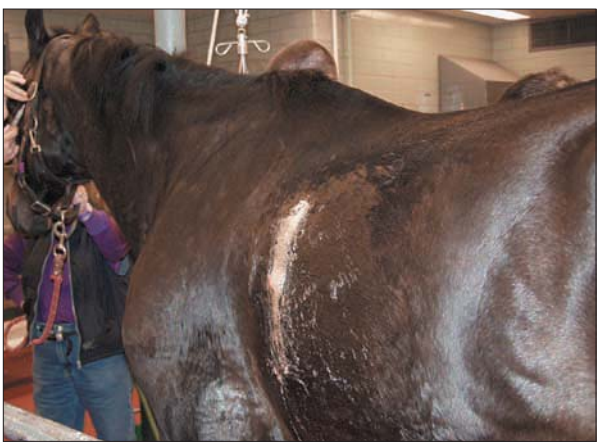
Bacterial pleuropneumonia, also known as pleuritis or pleurisy, is due to bacterial colonization of the pleural space and is accompanied by various degree of pleural fluid accumulation. Affected horses usually have a history of recent travel over long distances ('shipping fever'), have been subjected to stressful events, or have been exposed to a large number of new horses. Therefore, the condition is commonly seen in young athletic horses (racehorses or show horses), but foals to older horses can be affected.

Etiology/pathophysiology

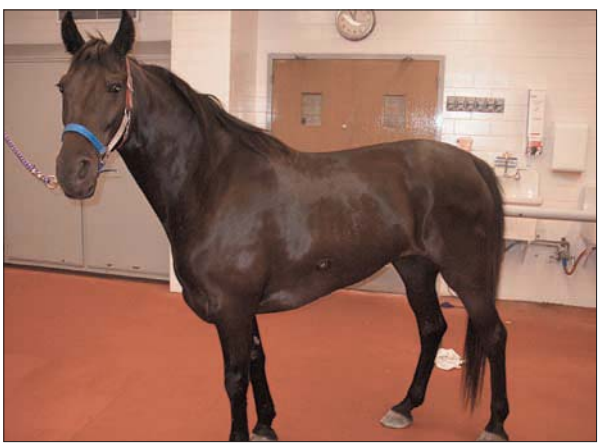
Bacteria most often gain access to the pleural surface from lung parenchyma infection (pneumonia or lung abscess) or, on rare occasions, from direct trauma to the chest wall or esophageal rupture and subsequent inoculation of the pleural space. Pleuritis may also be associated with fungal infection (coccidioidomycosis) or neoplasia (lymphosarcoma). In the majority of cases of pleuropneumonia multiple organisms are isolated, with a combination of aerobic and anaerobic bacteria. The most frequent aerobic isolates are *Streptococcus equi* subsp. *zooepidemicus* (*S. zooepidemicus*), *Pasteurella* spp., *Actinobacillus* spp., *Klebsiella* spp., *Escherichia coli*, and *Enterobacter* spp. About 25–46% of isolates are anaerobic bacteria such as *Bacteroides*, *Clostridium*, *Peptostreptococcus*, and *Fusobacterium* spp. Anaerobic cultures are important to identify *Bacteroides fragilis* because the organism is often resistant to penicillin. A putrid odor is commonly detected from pleural fluid or from the breath of horses with anaerobic infection; however, the absence of such odor does not rule out the possibility of anaerobic infection.

Pleuropneumonia is commonly seen 1–10 days after transportation over long distances. The stress of transportation depresses the phagocytic and killing capacity of alveolar macrophages as well as mucociliary clearance, thereby permitting opportunistic infection to develop. Another important contributing factor is head elevation during transport because keeping horses with their heads tied up slows down mucociliary clearance, thus favoring bacterial colonization of the LRT. Other predisposing factors are general anesthesia, exposure to cold environment, strenuous exercise, viral infections, noxious gases, and trauma. The route of infection in primary pleuritis is unknown, but may be via hematogenous spread of infectious agents to the parietal pleura or from airways to pleural lymphatics. Alternatively, inoculation of the pleural space may result from external puncture of the thorax (**199**). The disease is usually

▼ 199 Purulent exudate draining from a chest perforation site in a horse injured in a trailer accident.



▼ 200 Ventral edema extending from the pectoral region to the sternum in a horse with pleuropneumonia.



bilateral, even in cases of unilateral chest trauma, because of the normal communication that exists between the left and right pleural spaces via the fenestrated cranioventral mediastinum. The ventral location of these fenestrations explains why in cases of unilateral chest trauma with ipsilateral pneumothorax and lung collapse, collapse of the contralateral lung is rare.

Pleural effusion results from a combination of increased fluid leakage secondary to lung and pleural inflammation, and reduced lymphatic drainage due to fibrin deposition. In later stages, the pleural effusion may become organized in pockets of various sizes surrounded by fibrous tissue.

Clinical presentation

Bacterial pleuropneumonia usually presents as an acute or subacute onset of fever, anorexia, depression, mild colic, reluctance to move, ventral edema, nasal discharge, cough, and tachypnea. Progression over time may result in weight loss and respiratory distress.

Chest pain may be evidenced by a grunting noise during respiratory movement, defecation, and urination, or by the response elicited during manual pressure over the rib cage. As a result, horses are often reluctant to cough and when they do cough, the sound is soft and the cough non-productive.

Thoracic auscultation usually reveals normal to increased breath sounds dorsally and diminished or absent sounds ventrally. The transition between the two zones of auscultation is a sharp horizontal line of demarcation corresponding to the dorsal aspect of the fluid line. Heart sounds can be muffled or radiate over the chest. Percussion may reveal ventral dullness; however, the intensity of sounds elicited is dependent on chest wall thickness and body condition. As a result, percussion is insensitive compared with auscultation and, especially, with thoracic ultrasonography.

Ventral edema is a common finding in horses with pleuropneumonia, but is not a specific finding. It may start as a small amount of pitting edema in the ventral pectoral region and progress to a large plaque of ventral edema extending from the forelimbs to the inguinal region (**200**).

Complications from bacterial pleuropneumonia include laminitis, thrombophlebitis, pleural adhesions, sepsis, pleural or pulmonary abscesses, bronchopleural fistulae, pneumothorax, and pericarditis.

Differential diagnosis

A history of recent transportation or competition suggests a strong possibility of pleuropneumonia in a horse with fever and reluctance to move. Laminitis secondary to colitis, peritonitis, or other severe infectious process could present with similar signs. Since laminitis is a common sequela of bacterial pleuropneumonia, the absence of elevated digital pulses or the ability to pick up feet without difficulty would help rule out laminitis. Ventral edema is common in cases of pleuropneumonia, but may also develop secondary to other causes of pleural effusion such as congestive heart failure, neoplasia (e.g. thoracic lymphosarcoma), or hypoproteinemia.

Diagnosis

Hematology changes are non-specific and may reveal leukocytosis with neutrophilia and mild anemia in chronic cases. Serum biochemistry often shows hyperfibrinogenemia with hypoalbuminemia and hypergammaglobulinemia.

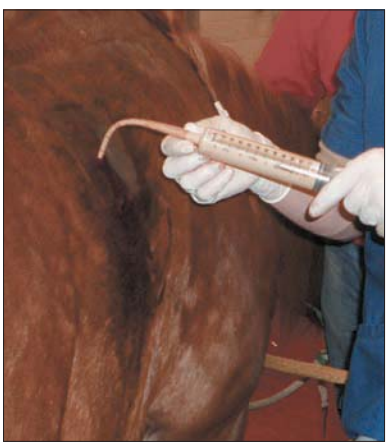
Collection of tracheal secretions for cytology and aerobic and anaerobic culture is important to confirm the presence of LRT infection, particularly because pleural fluid and tracheal aspirate isolates are commonly different. Culture for *Mycoplasma* spp. is indicated in cases unresponsive to usual antibiotic therapy or in stables where the infectious agent has been implicated. Endoscopic-guided sample collection via a guarded catheter is preferred to blind transtracheal aspiration in order to avoid contamination of the ventral cervical region and potential infection of the puncture site. BAL should not be used to collect respiratory secretions because the disease is multifocal and it is difficult to know if the sampled area is affected by the disease process. Consequently, there is a high likelihood of obtaining normal epithelial lining fluid even if the horse is suffering from bacterial pleuropneumonia.

Thoracocentesis is an essential diagnostic tool for collecting a fluid sample for cytology and bacteriology in horses suspected of having pleuropneumonia. In horses with bilateral pleural effusion, a sample from both sides should be submitted for microbiologic culture because the bacteriologic flora may differ between sides (**201**). Samples may be pooled for culture to limit expense. Thoracocentesis may be performed with a sterile cannula with a blunt tip (e.g. teat cannula, bitch urinary catheter) to avoid lung laceration. The cannula length should be at least 5–8 cm for an adult horse, although draft and obese horses may require longer instruments. Ultrasonography prior to thoracocentesis is ideal for selecting an appropriate cannula length and puncture site and for avoiding damage to underlying structures. Otherwise, the most commonly used site is the sixth or seventh intercostal space just dorsal to the palpable bulge of the costochondral junction. The cannula should be connected to an extension tube and a three-way stopcock to avoid unwarranted movement of the cannula, which could damage adjacent structures, and to facilitate fluid aspiration while preventing air from entering the pleural space. Pleural fluid should be seen coming down the extension tubing as soon as the pleural space is entered; however, gentle aspiration with a large sterile syringe (35–60 ml) may be required for highly viscous purulent exudate.

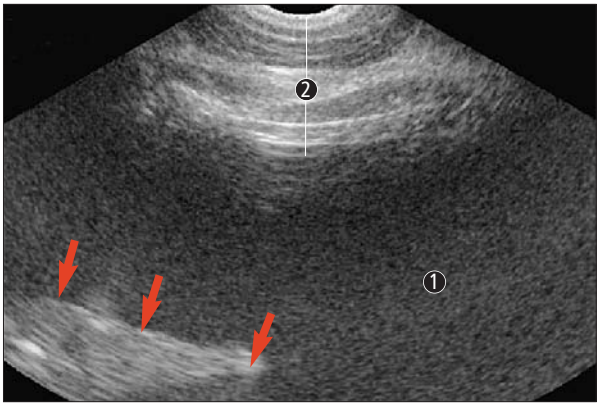
In cases where large accumulations of pleural fluid are causing respiratory distress, thoracocentesis should be performed without delay to restore suitable lung function. In such situations, pleural drainage is performed by placing a sufficiently large indwelling chest tube (24–32 French; 8–11 mm outer diameter) in the affected hemithorax (**202**). Smaller chest tubes tend to become obstructed by cellular debris, necrotic tissue, or fibrin clots. Pleural fluid analysis should include pH, glucose, and lactate measurement. Septic effusions are characterized by increased lactate and decreased pH and glucose concentration compared with venous blood. A complete biochemical profile and blood gas analysis should also be performed.

Thoracic ultrasound is the most effective and sensitive imaging tool to confirm a diagnosis of pleural disease. Pleural effusion is evident as homogeneous anechoic or hypoechoic fluid between the thoracic wall and lung (**203**). Exudate is usually characterized by hypoechoic fluid containing small echogenic particles representing fibrin tags, gas bubbles, blood, or cellular debris. Atelectatic lung usually appears as a wedge of hypoechoic, liver-like tissue floating in hypoechoic fluid (**203**). Consolidated lung appears as hypoechoic tissue with irregular margins and containing occasional island of aerated lung (**204**). Necrotic or abscessed lung appears as a homogeneous hypoechoic mass lacking

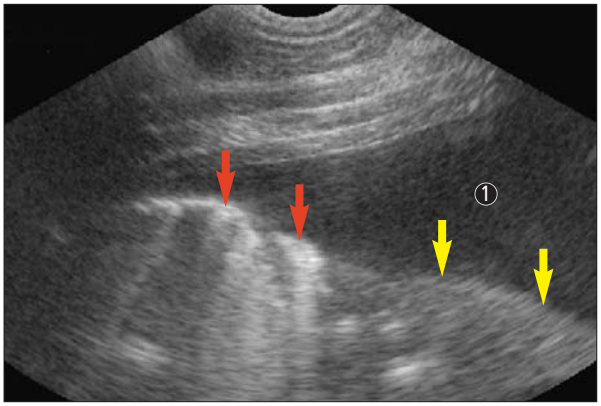
◀ **201** **Pleural fluid from the left and right hemithoraces often differ from a cytology, bacteriology, and gross appearance standpoint.**



▶ **202** **Chocolate-milk-colored purulent exudate aspirated from the chest of a horse with pleural empyema.**

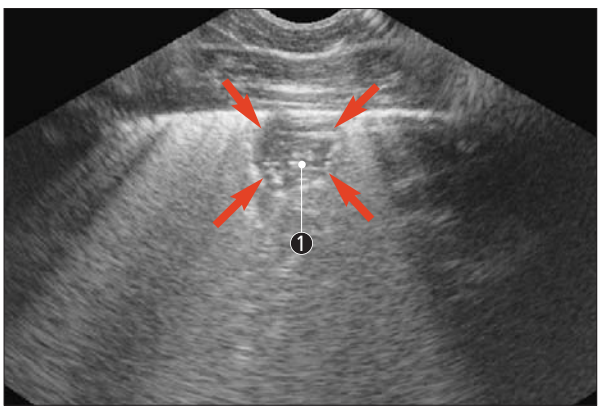


▲ **203** **Ultrasonographic image recorded from the right 11th intercostal space revealing a large accumulation of pleural fluid (1) in a horse with pleuropneumonia. The chest wall is at the top of the picture (2) and a section of atelectatic lung can be seen in the lower left corner (arrows).**



▲ **204** **Ultrasonographic image of consolidated lung in a horse with pleuropneumonia. The irregular, hyperechoic line represents aerated lung (red arrows) contiguous with hypoechoic consolidated lung (yellow arrows). Pleural fluid (1) accumulation is seen between the chest wall and lung.**

▶ **205** **Pulmonary abscess revealed by thoracic ultrasonography. The abscess appears as a round, hypoechoic area (1) adjacent to the pleura. It contains some hyperechoic specks consistent with purulent material and is delineated by a hyperechoic ring (arrows) suggestive of a fibrous capsule.**



normal lung architecture (blood vessels, airways) and surrounded by a thick echogenic capsule (abscess) or aerated lung (**205**). Ultrasound may be a very sensitive tool to detect superficial lung disease, but aerated lung below the pleural surface may mask deeper lung lesions. The latter may be detected by radiography. In some rare cases of pleuropneumonia (e.g. *Mycoplasma* spp.) pleuritis is the main feature, with few or no abnormalities in lung architecture on ultrasound.

Thoracic radiography using portable equipment is limited to foals. Referral hospitals usually have equipment that will image the adult thorax, although multiple views are needed to visualize the entire lung field. Radiography should be done after drainage of pleural effusion otherwise the fluid will obscure most of the structures in the ventral thorax. Thoracic radiography may help identify deep lesions such as lung abscesses or mediastinal lymphadenopathy.

Pleuroscopy, using a rigid or flexible endoscope, will help assess the extent and nature of any lung lesions. The technique is performed standing with the horse sedated in order to assist with careful debridement of the pleural space and pneumonic lung in cases where thoracocentesis is unable to achieve proper drainage.

Management/treatment

Aggressive systemic antimicrobial therapy is essential for successful management of pleuropneumonia cases. Initial therapy should be broad spectrum, targeting gram-positive and gram-negative aerobes and anaerobes. The IV route is preferred because it achieves a higher peak serum concentration, which will result in higher lung tissue and pleural fluid drug levels. Broad-spectrum activity can be obtained with synergistic drug combinations such as beta-lactam (e.g. penicillins, cephalosporins) and aminoglycoside (e.g. amikacin, gentamicin), beta-lactam and fluoroquinolone (e.g. enrofloxacin), or trimethroprim–sulfonamide (*Table 8*). A large number of anaerobic bacteria are susceptible to penicillin except for some isolates that produce beta-lactamases (e.g. *B. fragilis*). In these cases, metronidazole is used in combination with other antimicrobials effective against aerobic organisms. In some selected cases that are resistant to other antimicrobials, chloramphenicol may be considered because it has a broad spectrum of activity against aerobic and anaerobic bacteria; however, humans exposed to the drug may suffer from bone marrow suppression and, in some rare cases, irreversible aplastic anemia. Fluoroquinolones can reach high lung tissue and intracellular drug concentrations and are effective against *Mycoplasma* spp., but they have poor efficacy against *S. zooepidemicus* and anaerobic organisms, which are commonly isolated in horses with pleuropneumonia. Therefore, they should not be used as the sole antimicrobial therapy unless indicated by susceptibility testing.

IV antimicrobial therapy should be continued until the horse improves clinically and pleural fluid stops accumulating after removal of the indwelling chest tubes (minimum 7–10 days). In such a situation, therapy may be switched to the oral route based on culture and susceptibility testing. Prolonged antimicrobial therapy is required for at least 2–4 weeks with some cases requiring months of treatment to avoid sequelae such as lung abscesses, pleural empyema, or bronchopulmonary fistulae.

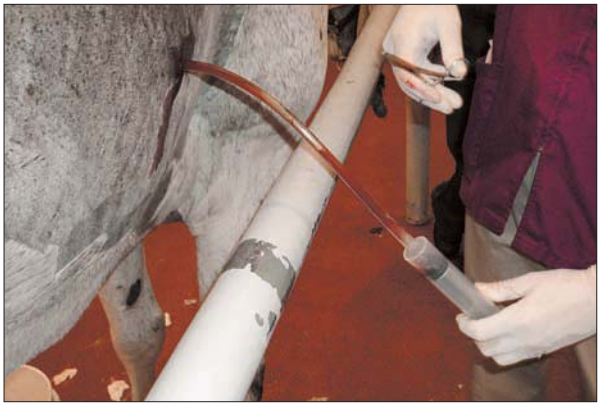
NSAIDs are indicated to reduce pain and inflammation. The most commonly used medications are phenylbutazone (2.2–4.4 mg/kg IV or PO q12h), flunixin (0.25–1.1 mg/kg IV q6–12h), ketoprofen (2.2 mg/kg IV q24h), and firocoxib (0.09 mg/kg IV q24h; 0.1 mg/kg PO q24h). IV fluid therapy is recommended until the horse has an adequate urine output and is able to maintain proper hydration. Preventive therapy for laminitis should be implemented, such as removing shoes, icing feet, and bedding the stall heavily.

Horses with respiratory distress secondary to voluminous pleural effusion (fluid line at the level of the shoulder joint or above) should have an emergency pleural drainage performed. A sufficiently large indwelling chest tube (24–32 French; 8–11 mm outer diameter) should be placed in the affected hemithorax following an aseptic technique. Local anesthesia of the thoracostomy site is performed by injecting 5–10 ml of 2% lidocaine as the needle (20 G/0.9 mm diameter; 4–6 cm long) is advanced towards the parietal pleura perpendicularly to the chest surface. Care should be taken to avoid vascular and nerve structures running

Table 8 **Antimicrobials used in systemic treatment of pleuropneumonia**

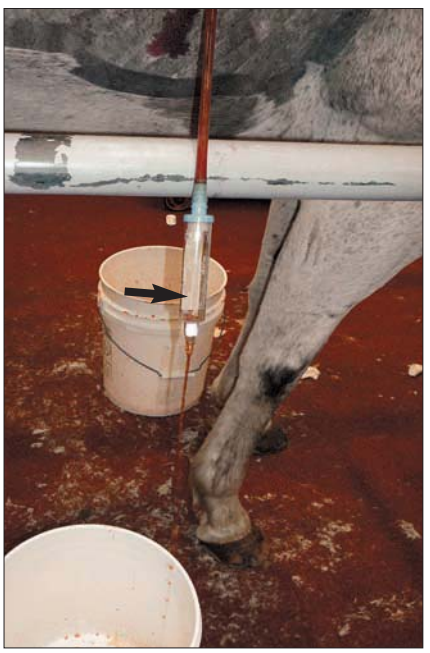
DRUG	ROUTE	DOSE	INTERVAL
Amikacin	IV	21 mg/kg (foals)	q24h
Ceftiofur	IV, IM	2.2 mg/kg	q12h
Chloramphenicol	PO	25–50 mg/kg	q6h
Enrofloxacin	IV PO	5 mg/kg 7.5 mg/kg	q24h q24h
Gentamicin	IV	6.6 mg/kg	q24h
Metronidazole	PO Per rectum	15–25 mg/kg 25–35 mg/kg	q8–12h q8–12h
Penicillin G, Na/K	IV	22,000 IU/kg	q6h
Penicillin G procaine	IM	22,000 IU/kg	q12h
Trimethoprim/ sulfonamide	PO	30 mg/kg	q12h

▲ 206 **Technique for insertion of a chest tube in the seventh intercostal space.**



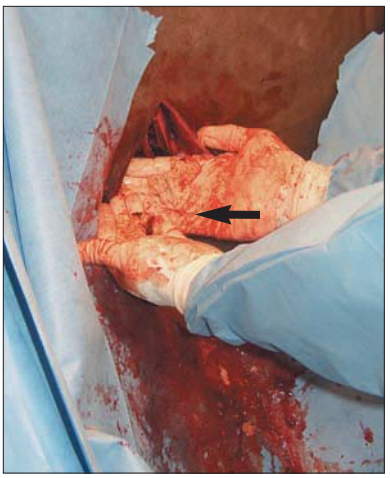
▲ 207 **Gentle aspiration with a catheter tip syringe may help remove debris that could obstruct outflow through an indwelling chest tube.**

parallel to the caudal aspect of each rib. A chest tube with trocar is inserted into the intercostal space with the tip pointing downward and forward (**206**). Pleural fluid should drain spontaneously through the tube; however, gentle aspiration with a catheter tip syringe may help drainage if debris obstructs the outflow (**207**). The tube is secured to the skin with a purse-string suture and the distal end is fitted with a Heimlich valve or Penrose drain to allow continuous fluid drainage out and avoid aspiration of air (**208**). Multiple chest tubes may be placed in one hemithorax if fluid loculation prevents appropriate drainage. Indwelling chest tubes should be removed when fluid is not dripping anymore (typically after 1–4 days) and pleural effusion is reduced to a minimum amount as assessed by thoracic ultrasonography. Fibrin clots or debris commonly occlude tube outflow; they can be removed by shaking the Heimlich valve vigorously, but taking care not to pull on the chest tube.



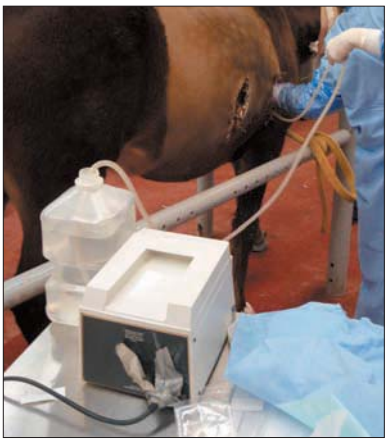
▲ 208 **An indwelling chest tube secured to the chest wall with a Heimlich valve (arrow) inserted at the tip.**

Thoracotomy with or without rib resection should be considered in horses with chronic pleuropneumonia that fail to respond to systemic antimicrobial therapy and pleural drainage. Horses exhibiting fever spikes despite aggressive therapy, and with ultrasonographic findings such as persistent pleural effusion with fluid loculation, lung necrosis, or abscess formation, are candidates for thoracotomy. Thoracotomy is preferably performed standing because these horses present a significant general anesthetic risk and drainage by gravity is best done in the standing position. Thoracotomy with

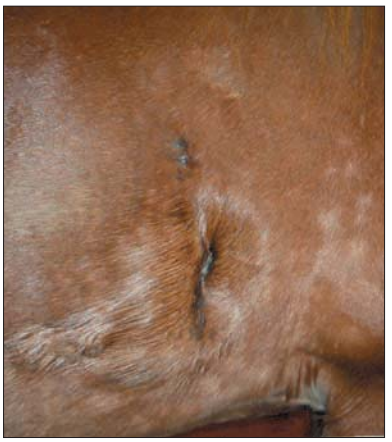


◀ **209** **Manual debridement following thoracotomy with rib resection in a horse with chronic pleuropneumonia. Large clumps of debris composed of fibrin clots and necrotic tissue (arrow) are removed by gentle debridement of the chest cavity.**

▶ **210** **Sterile packing secured over the thoracotomy site to prevent contamination of the chest cavity.**



◀ **211** **Flushing of the chest cavity with sterile isotonic solution using a peristaltic pump.**



▶ **212** **Thoracostomy site 6 months after rib resection.**

rib resection allows optimal access for manual, gentle debridement of necrotic lung tissue and fibrin tags and drainage of loculated fluid (**209**). Most procedures are performed between the fifth and eighth intercostal spaces; however, the appropriate intercostal space should be determined with ultrasonography to optimize access to affected structures and minimize damage to vital structures (e.g. heart, diaphragm). Bilateral pneumothorax should be considered if the horse develops increased respiratory effort during or shortly after the procedure has been completed. Air from pneumothorax may be aspirated manually with a large-sized syringe connected to a thoracic cannula via extension tubing and a three-way stopcock valve or by continuous aspiration using a pressure regulated system (e.g. Pleurovac). The thoracotomy incision is then packed with a sterile towel held in place with mattress sutures to avoid contamination with bedding materials (**210**). The packing is removed daily and the chest cavity flushed with warm, sterile, isotonic solution (**211**) until adequate granulation tissue is visible on the incision. The incision is left to heal by second intention, which takes 2–3 months on average, but may take up to 1 year in some cases (**212**).

The survival of horses with pleuropneumonia varies between 44% and 96%, largely based on how rapidly and aggressively they are treated. Horses with anaerobic infection have a poorer prognosis (33% survival). In Thoroughbred racehorses, up to 61% return to racing. Placement of an indwelling chest tube does not affect the prognosis for return to racing adversely. The prognosis after thoracotomy is fair to good (up to 88% survival) and for horses that survive, return to prior level of performance is achieved in 46% of cases.

Key points

- Pleuropneumonia is an infection of the lung parenchyma and pleural space caused by a variety of aerobic and anaerobic bacteria.
- Most cases develop in young horses within a few days of long distance travel.
- Clinical signs are acute onset of fever, anorexia, depression, cough, and tachypnea. Signs may progress to respiratory distress, ventral edema, and weight loss.
- Diagnosis is based on detection of pleural effusion by thoracic auscultation and ultrasonography and cytology of the fluid.
- Therapy includes broad-spectrum antimicrobial and anti-inflammatory drugs and supportive care. Pleural drainage is indicated in horses with copious effusion, particularly when it is associated with respiratory distress.
- Prognosis is less favorable in horses with anaerobic infection or in chronic cases.

Heaves (recurrent airway obstruction)

Heaves, or RAO, is discussed in detail in Chapter 5 (The coughing horse). Horses with severe RAO show lethargy and markedly increased respiratory effort manifested by nostril flaring, head and neck extension, and exaggerated rib cage and abdominal movements. Severe RAO often results in decreased appetite and weight loss. As the disease progresses, hypertrophy of the external abdominal oblique muscles develops and results in the characteristic 'heave line'. The absence of fever, history (e.g. exposure to moldy hay, potential prior episodes, chronicity), and rapid improvement after administration of bronchodilators differentiate RAO from septic lung diseases such as bacterial and viral pneumonia and interstitial pneumonia.

Interstitial lung diseases

Definition/overview

Interstitial lung diseases comprise pulmonary disorders characterized by infiltration of alveolar spaces or interstitial structures. These diseases represent an uncommon cause of pulmonary disease in horses, with both acute and chronic presentations having been observed. Affected animals may vary in age from foals as young as 1 month of age to horses over 20 years. Horses typically present with acute or chronic onset of tachypnea, exercise intolerance, increased respiratory effort, fever, and cough. Clinical presentation may vary from horses developing acute respiratory distress in less than 24 hours to horses exhibiting progressive weight loss and exercise intolerance over a period of months.

Etiology/pathophysiology

A cause is only found or suspected in a small number of affected horses. Infectious agents associated with interstitial lung diseases include viruses (e.g. EHV-5, *Morbillivirus*), bacteria (*S. zooepidemicus*, *Rhodococcus equi*, *E. coli*), parasites (*Parascaris equorum*, *Dictyocaulus arnfieldi*), protozoa (*Pneumocystis carinii*), and fungi (*Aspergillus* spp., *Cryptococcus* spp., *Histoplasma* spp.). Pneumotoxins released after ingestion of certain plants (e.g. *Perilla frutescens* and *Eupatorium adenophorum*) or inhalation of chemicals (e.g. smoke, silicon dioxide crystals) or organic antigens (fungi, endotoxin) may directly injure the lungs and result in interstitial lung disease. Alternatively, inhalation of an aerosol containing organic dust (e.g. fungi, chicken antigens) or systemic administration of immunostimulant drugs may result on rare occasion in hypersensitivity pneumonitis. Finally, interstitial lung disease may be a manifestation of pulmonary involvement from a generalized disease such as multisystemic eosinophilic epitheliotropic disease.

The initial insult may result from direct injury to the alveolar epithelium (pneumocytes I and II) from inhaled toxins or from hematogenous injury to pulmonary capillaries or the alveolar basement membrane. Acute lung injury results in plasma exudation from a disrupted alveolar–capillary barrier. Exudate may lead to hyaline membrane formation partially attached to alveolar and airway walls. Inflammatory cells, in particular neutrophils, migrate to alveolar walls and may damage tissue by releasing proteases and

reactive oxygen species. A proliferative phase characterized by type II pneumocyte hyperplasia follows the exudative phase within a few days. Epithelial hyperplasia results in thickened alveolar walls. If the horse survives the initial pulmonary insult, lesions may progress towards alveolar fibrosis characteristic of the chronic phase of the disease. The structural lung alterations observed with interstitial pneumonia are accompanied by functional changes such as decreased lung compliance and vital capacity and impaired gas diffusion, which translate clinically into increased respiratory effort, exercise intolerance, and hypoxemia.

Clinical presentation

Horses presenting with acute forms of the disease usually exhibit respiratory distress characterized by nostril flaring, tachypnea, increased respiratory effort, and cyanotic mucous membranes. Unlike RAO, horses with interstitial lung disease are systemically ill. Fever, tachycardia, and abnormal breath sounds (e.g. wheezes, crackles) are frequently detected on thoracic auscultation during physical examination. Some cases may display decreased breath sound intensity over the entire lung field despite obvious breathing difficulty. Chronic cases often exhibit progressive weight loss and increasing breathing difficulties. Horses may be asymptomatic in the early stages of chronic interstitial lung disease (e.g. silicosis). The acute inflammation observed in inflammatory interstitial lung disease may be an exacerbation of chronic, low-grade lung inflammation (i.e. 'acute on chronic') or an acute inflammatory process.

Differential diagnosis

The main differentials include bronchopneumonia, pleuropneumonia, and RAO. Horses with RAO are not febrile, have normal hematology, and improve substantially and rapidly after administration of corticosteroids and bronchodilators. Most horses with bronchopneumonia or pleuropneumonia respond to systemic antimicrobials, anti-inflammatory drugs, and pleural drainage if indicated. Thoracic radiography and ultrasonography are useful in these cases to confirm or rule out lung parenchymal or pleural involvement, respectively.

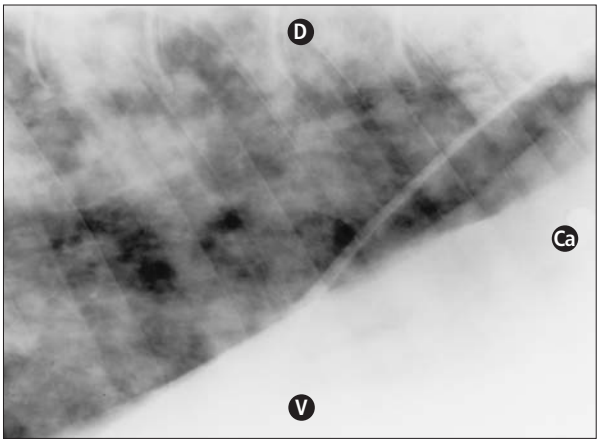
Diagnosis

Hematology typically reveals leukocytosis, neutrophilia, and hyperfibrinogenemia. Occasionally, thrombocytopenia and abnormal clotting times may be detected in severely affected animals with bleeding diathesis (e.g. epistaxis, petechiae, ecchymosis). Common abnormalities detected on arterial blood gas analysis include hypoxemia and hyper- or hypocapnia.

Various bacteria (e.g. *S. zooepidemicus*, *R. equi*, *E. coli*) may be isolated from respiratory secretions collected from young horses; however, bacteria, viruses, or fungi are usually not cultured from adult horses except for EHV-5. Identification of EHV-5 from BAL fluid or lung biopsy using PCR is diagnostic in horses showing clinical signs consistent with interstitial lung disease. Cytologic analysis often shows inflammatory changes characterized by an increased number of non-degenerative neutrophils and no visible pathogens. Accumulation of intracellular crystalline materials in pulmonary alveolar macrophages is commonly detected in horses with silicosis using polarized contrast microscopy. Thoracic radiographs often reveal a severe, diffuse interstitial pattern, occasionally forming a miliary to nodular pattern (**213**). Areas of alveolar opacities with air bronchogram may be seen in addition to areas of bronchointerstitial lung patterns. Silica can also be detected in giant cells in BAL fluid. Repeat radiography may be useful for following disease progression, but it is a poor predictor of lung function since radiographic findings may be normal in a horse with marked exercise intolerance.

Thoracic ultrasonography may reveal an irregular pleural surface or multiple superficial nodular hypoechoic lesions of various diameters.

Transcutaneous lung biopsy, preferably ultrasound guided, often provides a definitive diagnosis. Complications such as pulmonary hemorrhage and pneumothorax are common, but death is a rare occurrence. Histological findings in acute severe cases reveal diffuse, necrotizing bronchiolitis and alveolitis, hyaline membrane formation, interstitial edema and fibrosis, and type II pneumocyte hyperplasia. Horses with multinodular pulmonary fibrosis may be distinguished from horses with other lung diseases because of the nodular pattern of the collagen deposition, the



▲ 213 **Thoracic radiograph of a horse with interstitial pneumonia revealing a severe, diffuse interstitial and nodular pattern. The diaphragm can be seen running cranioventrally to caudodorsally. Ribs and vertebrae are faintly visible. (Ca) caudal; (D) dorsal; (V) ventral.**

presence of inflammatory cells within an alveolar-like architecture, and intranuclear viral inclusion bodies within enlarged macrophages. Lesions secondary to silicosis are characterized by areas of fibrosis with multiple granulomatous lesions containing macrophages with intracytoplasmic, eosinophilic, birefringent crystalline material. Gross pathologic findings include diffusely enlarged and abnormally heavy lungs that fail to collapse on opening of the thorax, variable amounts of pink foamy liquid in airways, a mottled, lobulated appearance of the lungs, and in some cases, interstitial emphysema. Diffuse coalescent nodules of fibrosis (up to 5 cm in diameter) or multiple discrete nodules (up to 10 cm in diameter) separated by normal lung are suggestive of equine multinodular pulmonary fibrosis. The nomenclature of interstitial lung disease in the horse is in its infancy, and may change in the future.

Lung function testing is used to characterize the type of functional impairment (restrictive lung pattern of interstitial lung disease versus obstructive lung pattern of RAO), the severity of pulmonary disease, and the lack of response to bronchodilators. Interstitial pneumonia results in decreased lung elasticity (decreased compliance) and, as a result, greater distending pressure is required from inspiratory muscles in order to achieve any volume change (restrictive lung disease). Arterial blood gas analysis usually reveals hypoxemia with normocapnia or hypocapnia that is responsive to intranasal oxygen supplementation.

Management/treatment

The main goals of therapy are to improve tissue oxygenation, decrease pulmonary inflammation, treat underlying infections and potential complications, and avoid additional stressors. The latter includes strict stall rest in a well ventilated, cool, dust free environment. Severe hypoxemia (P_{aO_2} <60 mmHg) should be treated with oxygen supplementation through nasal or transtracheal insufflation and bronchodilator administration. In order to improve arterial blood oxygen tension, minimum oxygen flow rates of 5 l/min in foals and 12 l/min in adults may be required and should be adjusted based on clinical response or, preferably, repeated arterial blood gas analysis. Long-acting inhaled bronchodilators may help decrease the work of breathing (see Chapter 5, The coughing horse, Heaves [RAO], Management/treatment). NSAIDs (e.g. flunixin meglumine) may be beneficial; however, corticosteroids appear to be more effective at decreasing pulmonary inflammation and preventing fibrosis, and their use is associated with a positive outcome. Both inhaled and systemic corticosteroids may be administered using similar dosages as for RAO horses in crisis. Antimicrobial therapy is recommended to treat primary or opportunistic infections. Broad-spectrum treatment should be initiated while waiting for TW cytology and culture results, and therapy should last at least 3–6 weeks. There are some anecdotal reports of successful therapy of multinodular pulmonary fibrosis associated with EHV-5 infection using oral acyclovir (20 mg/kg q8h). However, the drug's poor absorption after oral administration suggests that other antiviral

drugs should be investigated. For example, valacyclovir is well absorbed in horses after oral administration, but its effect on EHV-5 is unknown at the present time.

IV fluid therapy should be used with caution because cases of severe interstitial pneumonia often exhibit pulmonary hypertension and additional fluids may lead to, or aggravate, pulmonary edema. Furosemide therapy may be useful in such cases.

Horses with interstitial pneumonia have a poor prognosis; however, some cases have been successfully treated and returned to athletic activities, especially foals. In humans, adipose derived and other mesenchymal stem cells may show therapeutic benefits, but the effects of this approach have not been reported in horses with interstitial lung disease.

Key points

- Interstitial lung diseases are uncommon disorders usually caused by viral or bacterial pathogens.
- Affected horses may be of any age and typically exhibit fever, tachypnea, and respiratory distress.
- Thoracic radiography and ultrasonography are helpful to differentiate interstitial pneumonia from pleuropneumonia or bronchopneumonia.
- Treatments include supportive therapy (e.g. supplemental oxygen, bronchodilators), corticosteroids and antimicrobials. Antiviral therapy should be considered in cases where EHV-5 infection is suspected.

Proximal airway obstruction

Labored breathing associated with upper airway obstruction is usually associated with loud abnormal respiratory sounds or stridor. Such diseases are discussed in detail in Chapter 8 (Abnormal respiratory sounds).

Lesions such as nasal cysts or ethmoid hematomas cause fixed airway obstructions resulting in respiratory effort and sound intensity that are comparable during inspiration and expiration. Variable (dynamic) airway obstructions such as pharyngeal or tracheal collapse are characterized by a marked difference between inspiratory and expiratory sounds. Increased inspiratory effort accompanied by stridor indicates proximal airway obstruction from collapse of non-rigid structures such as the nostrils, alar folds, pharynx, larynx, and extrathoracic trachea. Depending on the obstruction severity, labored breathing may be evident at rest and amplified by light exercise (e.g. tracheal collapse) or it may only occur during exercise (e.g. laryngeal hemiplegia). Diagnostic work-up of affected horses should be done while minimizing stress levels because hypoxemia may worsen rapidly, resulting in uncontrollable excitement, collapse, and death. Horses that are not accustomed to being restrained (e.g. foals) should be started on nasal oxygen supplementation and lightly sedated. Emergency tracheostomy is recommended in horses exhibiting severe proximal respiratory tract obstruction accompanied by inspiratory stridor.

CHAPTER 8

ABNORMAL RESPIRATORY SOUNDS

INTRODUCTION

Respiratory sounds are generated by airway wall vibration during breathing. Vibrating airways produce pressure waves that travel through the body and along conducting airways and may be heard at the nostril opening or by auscultation if they are in the audible range (20–20,000 Hz). Abnormal respiratory sounds are superimposed on normal sounds and reflect a change in the relationship between flow rate through the airways, their size, and wall compliance.

Clinically, respiratory sounds are classified based on their origin into extrathoracic (proximal airways including the extrathoracic trachea) or intrathoracic sounds (pulmonary and intrathoracic trachea). The former may be heard directly at the nostril opening or by auscultation over the cervical trachea. The latter are best heard by thoracic auscultation using a stethoscope. Increased ventilation (e.g. post exercise, rebreathing bag) and auscultation of animals with a thin chest wall (e.g. foals) will result in an increase in sound intensity.

At rest, normal respiratory sounds originating from the proximal airways are predominant during tracheal auscultation, but they are difficult to hear at the level of the nostril opening. Expiratory sounds predominate in horses exercising strenuously. Stridor refers to high intensity and frequency sounds generated by proximal airway obstruction. Other terms used to describe abnormal sounds occurring during exercise are roaring, rattling, or gurgling.

Lung sounds are divided into breath sounds (normal) and adventitious (abnormal) lung sounds. Breath sounds are produced by turbulent flow associated with air movement through large airways. Therefore, the term 'vesicular' sounds should not be used because it assumes that sounds originate from the alveoli. Adventitious sounds are classified as wheezes, crackles, or friction rubs. Wheezes are continuous, musical sounds originating mainly from vibrating airway walls and potentially from movement of airway secretions. Crackles are short, 'crepitating' sounds possibly produced by the sudden opening or closure of small airways.

Abnormal respiratory sounds may have different characteristics based on the site of obstruction and on the type of obstruction. Fixed obstructions (e.g. sinus cyst) result in a similar degree of airflow obstruction during inhalation and exhalation. Variable obstructions (e.g. laryngeal hemiplegia, DDSP) generate different degrees of airway obstruction based on flow rate, site of obstruction, and phase of the respiratory cycle. In general, loud abnormal respiratory sounds or stridor, audible without a stethoscope at rest or during exercise, indicate proximal airway obstructive disease. Abnormal inspiratory sounds (inspiratory stridor) indicate airway obstruction by collapsible structures such as the external nares, pharynx, larynx, or extrathoracic trachea.

Increased lung sounds heard on thoracic auscultation diffusely over both sides of the chest suggest increased ventilation due to a variety of physiologic or pathologic conditions. Increased lung sounds over a limited area imply the presence of lung consolidation or a mass. Lung consolidation refers to the texture of pneumonic lung due to atelectasis and flooding of airspaces with secretions. Decreased breath sounds may result from pleural effusion (ventrally) or pneumothorax (dorsally) or increased wall thickness (e.g. obesity, draft horses).

STRIDOR

Nasal diseases

Atheroma (epidermal inclusion cyst)

Definition/overview

An atheroma (epidermal inclusion cyst) is a benign, cystic structure that is typically located in the caudal, innermost aspect of the nasal diverticulum.

Etiology/pathophysiology

Atheromas are sebaceous gland cysts within the false nostril. It is not known why this cystic structure is located in such a consistent location. The cysts are not painful and rarely result in clinical signs of upper respiratory noise. The primary concern to the owner is usually cosmetic only.

Clinical presentation

Atheromas are smooth, non-painful, enlargements within the nasal diverticulum (**214**); most are approximately 5 × 5 cm (e.g. golf ball) in size. When palpated the cyst is soft and compressible. If aspirated, it contains yellow, mucinous fluid. It is unusual for these types of cysts to cause nasal obstruction.

Differential diagnosis

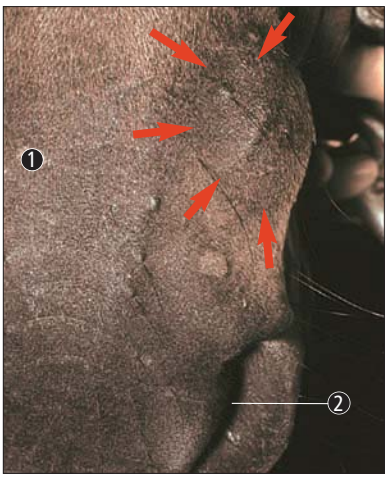
Abscess; neoplastic growth; foreign body; granulation tissue mass.

Diagnosis

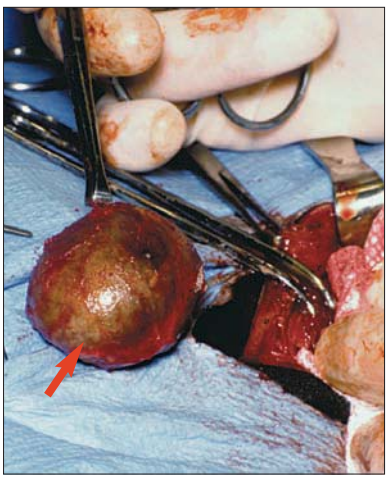
Physical examination and palpation of the cyst within the nasal diverticulum is usually diagnostic. If a biopsy or aspirate is elected, then excisional biopsy is recommended. Histopathologic findings are compatible with a sebaceous cyst.

Management/treatment

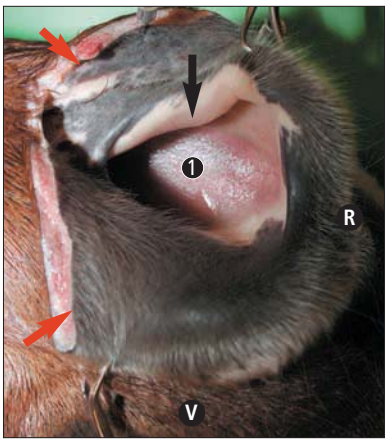
Since atheromas are generally just a cosmetic issue, it is up to the owner to pursue surgical removal. The easiest way to resolve the cyst is to destroy or excise the cystic lining, either by mechanical disruption or by injection of irritating compounds. The best method of mechanical disruption is to make a small incision into the cyst from the nasal diverticulum and then insert a curette or roaring burr and physically remove the cystic lining. If this is not completely removed, the cyst will reform. Alternatively, the cyst can be injected with 2.0–4.5 ml of neutral buffered 10% formalin, which causes necrosis and cauterization of the lining. This has been reported to be a simple and effective way to resolve atheromas. The final method of surgical removal is to make an incision directly over the cyst, followed by careful dissection of the entire cyst, preferably without rupture. Once dissected free, the cyst is removed and the skin incision closed in a routine fashion (**215**). However, because this method has some risk of scarring, most surgeons currently recommend mechanical disruption or chemical ablation.



◀ **214** **Horse with an atheroma (arrows) within the left nasal diverticulum. (1) dorsal midline; (2) nostril. (Photo courtesy Stephen B Adams)**

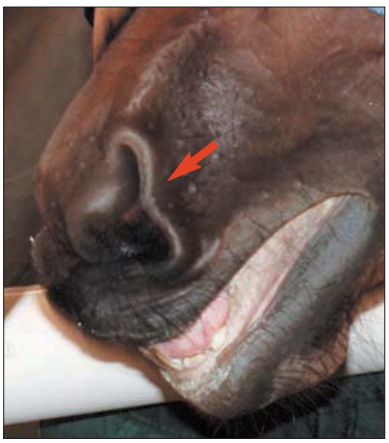


▶ **215** **Excised atheroma (arrow) from the horse pictured in 214. (Photo courtesy Stephen B Adams)**



◀ **216** **Gross postmortem image of the normal alar fold (black arrow) and nasal diverticulum (red arrows). (R) rostral; (V) ventral; (1) rostral nasal septum. The nasal diverticulum was transected to allow visualization of underlying structures.**

▶ **217** **Standardbred racehorse with collapse of the left nasal diverticulum (arrow) while exercising on a high-speed treadmill.**



Key points

- Atheromas (epidermal inclusion cysts) are benign, cystic masses within the nasal diverticulum.
- They are of cosmetic significance only.
- Mechanical or chemical disruption of the cystic lining typically results in successful resolution and a good prognosis for a cosmetic outcome.

Alar fold obstruction

Definition/overview

Alar fold obstruction is the direct result of redundant or enlarged alar folds. The alar folds are located within the rostral aspect of the nares.

Etiology/pathophysiology

The alar fold is a soft-tissue structure located within the nasal diverticulum. It is supported by the C-shaped alar cartilage. In some breeds of horses the alar folds are subjectively thickened and edematous. The most commonly affected breeds of horse include Standardbred, Saddlebred, and Thoroughbred. The thickened and edematous alar folds undergo dynamic collapse during exercise. Dynamic collapse of the folds leads to clinical signs of inspiratory noise and exercise intolerance. Alar fold obstruction is not usually recognized until the horse is placed into athletic training. An additional cause of alar fold obstruction is damage to the neuromuscular structures that control dilation of the nasal diverticulum. This can include damage to the levator nasolabialis muscle and facial nerve paralysis.

Clinical presentation

The primary clinical signs of alar fold obstruction include URT noise and exercise intolerance. The respiratory noise can be expiratory or inspiratory, with the noise loudest on expiration. Collapse of the folds during inspiration results in partial airway obstruction during exercise. The clinical signs of alar fold collapse are worse in horses with small nares and small nasal passages. Palpation of the alar folds should be performed during physical examination. Palpation is subjectively abnormal when the folds feel thickened and redundant. The best way to gain an appreciation of this is to palpate the folds of normal horses (**216**).

Differential diagnosis

Nasal septum disease; nasal passage masses or neoplasia; laryngeal hemiplegia; epiglottic disorders.

Diagnosis

Physical examination and history (e.g. collapse of the nostrils during exercise) provides an initial impression of alar fold obstruction. It is helpful to observe the horse during exercise. The best way to do this is with the horse on a high-speed treadmill (**217**). A diagnostic test is available to confirm a diagnosis of alar fold collapse. There are two methods of performing this test. The first is to place size 2 suture material through each alar fold (avoiding the cartilage). A loop of suture material is secured. The loop of suture in each alar fold is tied over the bridge of the nose with umbilical tape.

The second method is to place a stainless steel ring in the fold (hog ring; **218**, **219**) rather than suture material. The horse is then exercised with the alar folds under tension. If the signs of respiratory noise are eliminated, the diagnosis is confirmed.

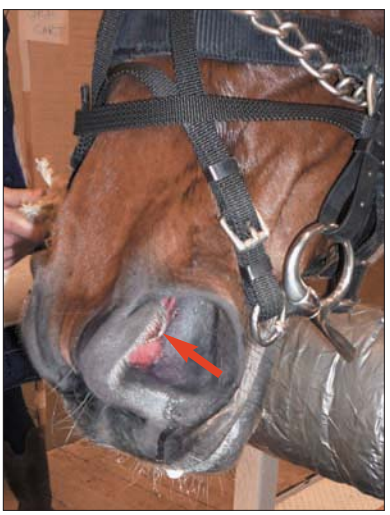
The final method of confirmation of this disorder is to use plastic clips inserted into the nasal diverticulum (**220**, **221**). The plastic clip is then attached to the nose band of the halter.

Endoscopy should also be performed. Approximately 33% of horses with alar fold obstruction will have narrow nasal passages. Endoscopy is useful to rule out other causes of respiratory tract obstruction including DDSP, EE, and laryngeal hemiplegia.

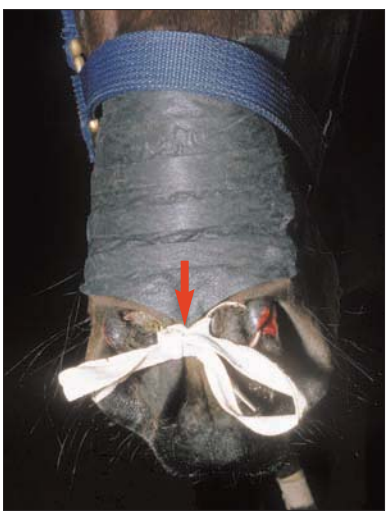
Narrow nasal passages may be suspected based on the subjective external appearance of the skull and endoscopic examination. Objective determination of narrow nasal passages can be measured with acoustic rhinometry. Comparison of left-to-right nasal volume is helpful in unilaterally affected horses (normal L/R nasal volume ratio = 1.0 ± 0.1). The case shown in **217** and **221** had a L/R nasal volume ratio of 1.3 and the 30% decrease in volume was located between 7 and 15 cm from the nasal opening. Endoscopy and skull radiographs were unremarkable, therefore congenital narrowing was suspected in this 3-year-old Standardbred that had never raced.

Management/treatment

Alar fold obstruction can be managed conservatively or with surgical resection of the folds. Conservative management involves the use of rings placed in the alar folds and then tied with umbilical tape over the bridge of the nose. Plastic clips can also be used in some racing jurisdictions to maintain dilation of the nasal diverticulum. Conservative management is best used in mild cases of alar fold obstruction. More severe cases should be managed surgically. For surgical correction the horse is anesthetized and positioned in lateral or dorsal recumbency depending on surgeon preference. The lateral aspect of the nasal diverticulum is completely incised to expose the alar fold (**222**). Once exposed the fold is excised using Mayo scissors. The alar cartilage should be avoided even though it will be exposed following removal of the alar fold. The most rostral tip of the ventral nasal concha is incised as well. After excision of the fold, the mucous membrane is sutured to the skin of the nasal diverticulum to obtain a primary closure using a simple continuous pattern of absorbable suture material (**223**, **224**). The lateral aspect of the nasal diverticulum is closed in two layers using absorbable suture material.

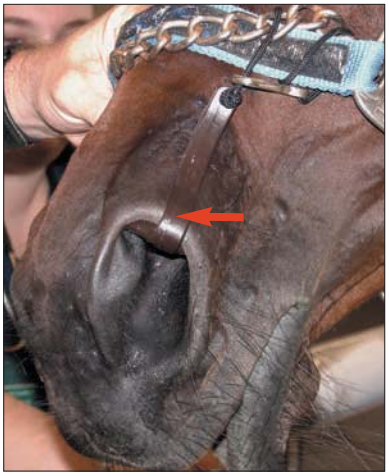


◀ **218** **Stainless steel ring in the left alar fold (arrow).**

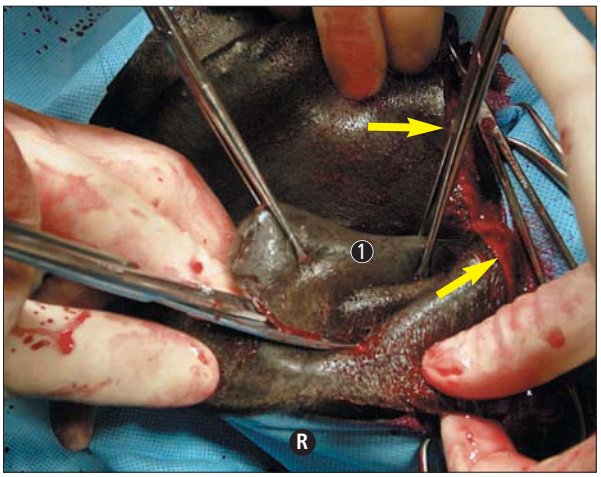


▶ **219** **Horse with stainless steel rings in both alar folds. The rings are tied together with tape to retract the alar folds dorsally (arrow). Note the nasal strips applied over both nasal diverticula to stabilize the nares.**

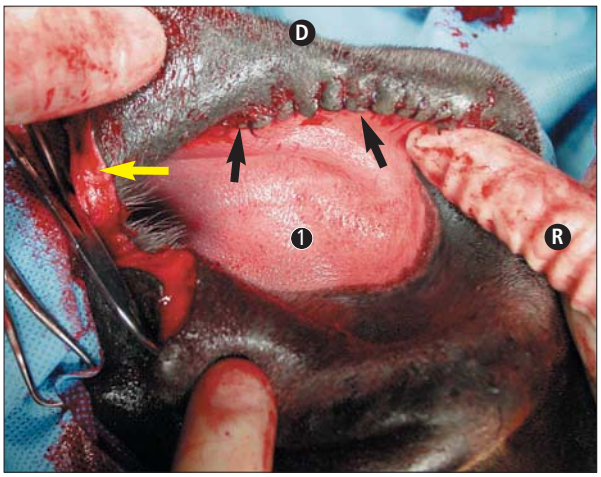
◀ 220 Plastic nasal clip used to dilate the nasal diverticulum.



▶ 221 Breathe E-Z™ nasal clip (arrow) in position in the nasal diverticulum. The clip is secured to the nose band of the halter.

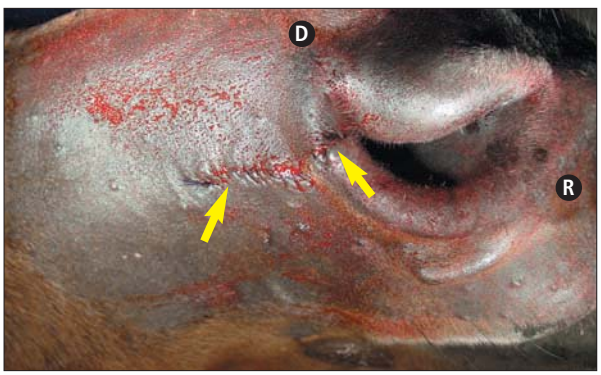


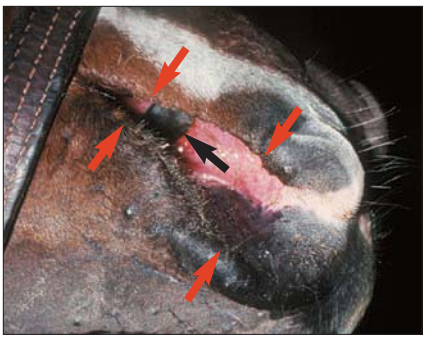
▲ 222 Surgical resection of the alar fold (1) following incision into the wall of the nasal diverticulum (arrows). Scissors are being used to excise the fold adjacent to the alar cartilage. (R) rostral.



▲ 223 Suturing of the mucous membrane of the nostril to the skin of the nasal diverticulum (black arrows) following removal of the alar fold. The yellow arrow indicates the cut edge of the nasal diverticulum. (R) rostral; (D) dorsal; (1) rostral nasal septum.

▶ 224 Surgical closure of the incision into the nasal diverticulum (arrows). (R) rostral; (D) dorsal.





▲ **225 Postoperative photograph of a horse with collapse of the nasal diverticulum and alar fold, treated with resection of the lateral wall of the nasal diverticulum (red arrows), alar fold, and a portion of the rostral nasal septum (black arrow).**

Aftercare involves administration of antimicrobials and NSAIDs for 3–5 days. The skin sutures do not need to be removed if absorbable suture material is used. Horses with dynamic collapse of the alar folds and nasal diverticulum are best treated with a combination of surgical procedures. Even though it is not cosmetically pleasing, the best surgical method of correction is to completely resect the alar fold and the nasal diverticulum (**225**). This is done because alar fold resection alone is not likely to resolve clinical signs.

The prognosis following alar fold obstruction is generally favorable. Cosmetic healing is excellent following surgery. In a series of cases treated for alar fold obstruction, 71% of horses had decreased respiratory noise after surgery. However, horses with narrow nasal passages have a 50% chance of being successful racehorses after surgery.

Key points

- Alar fold obstruction is more common in Standardbred, Saddlebred, and Thoroughbred horses.
- Alar fold obstruction results in clinical signs of upper respiratory noise and exercise intolerance.
- Surgical removal of the alar fold generally results in the best outcome.
- The prognosis is guarded for horses with a combination of alar fold and nasal diverticulum collapse.

Nasal septum obstruction

Definition/overview

Nasal septum thickening is caused by multiple etiologies and results in URT obstruction and noise.

Etiology/pathophysiology

The primary external causes of nasal septum thickening are skull or nasal passage trauma and the presence of masses within the nasal passage or the paranasal sinuses resulting in nasal septum deviation towards the contralateral side. Skull fractures or foreign body penetration of the nasal septum from the nasal passage can damage the septum. Trauma results in edema, hematoma formation, or fracture of the cartilaginous portion of the septum or the vomer bone. Chronic hematoma formation can lead to abscessation of the nasal septum. The presence of masses within the nasal passage or paranasal sinuses can result in deviation of the nasal septum. Deviation towards the contralateral side can result in nasal passage obstruction.

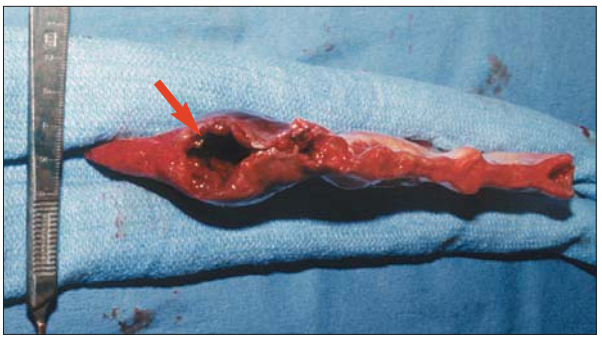
Internal factors on the contralateral side leading to thickening of the nasal septum include abscessation secondary to *Streptococcus equi* subsp. *equi* or *zooepidemicus* infection (**226**) and cysts located within the nasal passage. Other causes include chondritis, cryptococcal granuloma, and wry nose. Nasal septum abscessation is generally secondary to sepsis; this generally affects horses <4 years of age.

Cystic structures within the nasal septum are believed to be of congenital origin and can result in nasal passage obstruction and URT noise (**227**).

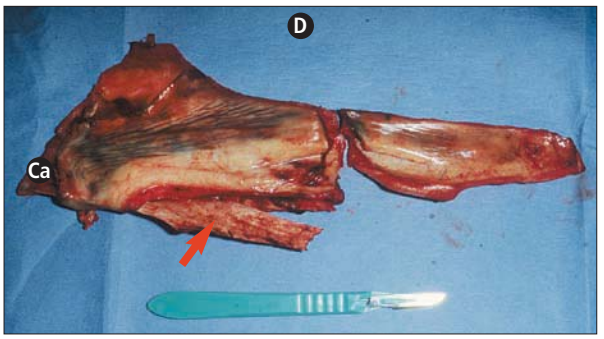
Neoplasia, including amyloidosis, squamous cell carcinoma (SCC), adenocarcinoma, lymphosarcoma, and hemangiosarcoma, can also affect the nasal septum.

Clinical presentation

The primary clinical signs of nasal obstruction related to the nasal septum include upper respiratory noise and exercise intolerance. A subjective evaluation of airflow should be performed by placing a hand over each of the nares and assessing airflow through each nostril. There will be decreased airflow on the side of the obstruction. Some horses will also have a purulent to mucopurulent nasal discharge if infection is present within the nasal passage or sinuses. In addition to airflow evaluation, the rostral aspect of the nasal septum should be palpated manually. In some instances, palpation of the septum



▲ 226 **Excised nasal septum with an abscess (arrow) at the rostral end.**



▲ 227 **Excised nasal septum with cystic enlargement. Note the portion of the vomer bone (arrow) remaining following nasal septum excision. (D) dorsal; (Ca) caudal.**

will reveal cartilaginous abnormalities and palpable enlargement of the septum. Fractures of the septum can also be palpated. If strangles is a potential diagnosis, palpation of the submandibular or retropharyngeal lymph nodes may reveal enlargement or focal areas of lymph node drainage. If paranasal sinus disease is suspected as a cause of nasal septum deviation, the sinuses may be dull following digital percussion.

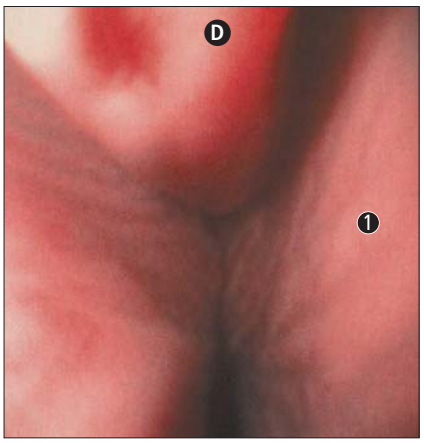
Differential diagnosis

Neoplasia within the nasal passages or paranasal sinuses; primary or secondary sinusitis; pharyngeal disease; laryngeal disease; epiglottic abnormalities.

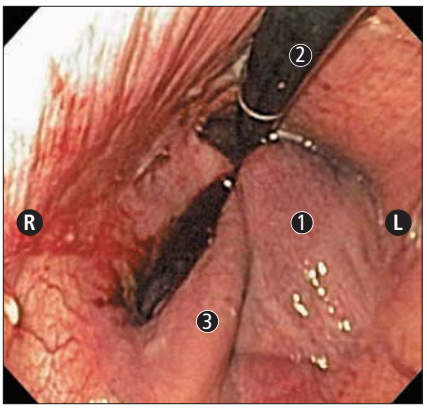
Diagnosis

Diagnosis is based on physical examination findings, subjective evaluation of airflow from each nostril, and palpation of the rostral aspect of the nasal septum. Endoscopy should be performed in horses suspected of nasal obstruction secondary to disease of the nasal septum (**228**). This may reveal visible enlargement or areas of trauma. Purulent drainage from the nasal septum may also be observed.

Deviation of the nasal septum may preclude the passage of the endoscope on the deviated side because of severe restriction of the opening. When this occurs, it may be feasible in some instances to insert the endoscope into the normal side and retrovert the scope to examine the contralateral nasal passage and septum from its caudal aspect. Retroversion is performed by



▲ 228 **Endoscopic photograph of nasal passage narrowing secondary to deviation of the nasal septum towards the right side. (D) dorsal; (1) nasal septum.**



◀ **229** **Endoscopic photograph using retroversion of the endoscope to visualize the caudal aspect of the nasal septum in a horse with a paranasal sinus cyst (1) in the left nasal passage. Note that the endoscope (2) is ventrally located, not dorsal as the 'upside-down' appearance of the image seems to indicate. (R) right; (L) left; (3) dorsal aspect of the nasal septum.**

inserting the end of the endoscope completely into the nasopharynx and then turning the large wheel on the control towards the person performing the endoscopy. This will turn the end of the endoscope rostrally towards the nares. Endoscope retroversion can be confusing because the resulting image is upside down (**229**). Nevertheless, in some instances this is the only way an image can be obtained of the nasal septum when the nasal passage is too narrow to allow passage of the endoscope on the affected side.

In addition to endoscopy, diagnostic imaging can be useful for evaluating the contour of the nasal septum. The minimum radiographic views required are dorsoventral, lateral–medial, and right and left oblique views of the septum and sinuses. Radiographic abnormalities associated with the nasal septum include deviation of the nasal septum, fracture of the vomer bone, and focal thickening of the septum. If available, CT and MRI can be used to confirm the radiographic findings. Acoustic rhinometry can also be performed. This provides an objective method of measuring nasal passage diameter. It is especially helpful when evaluating whether or not one nasal passage is smaller or larger than the contralateral passage.

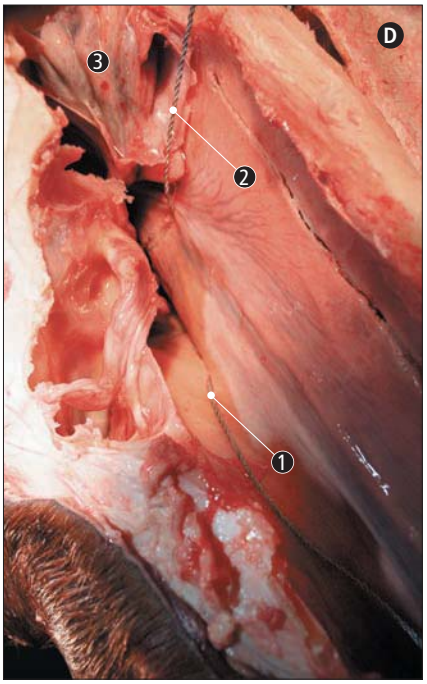
Management/treatment

Medical management of abscessation or trauma to the nasal septum can be attempted. This should include administration of antimicrobials, NSAIDS, and corticosteroids. Neoplasia involving the nasal septum is rarely treated surgically because it is very difficult to remove the neoplastic growth completely, although it would be possible to treat a focal neoplasm within the nasal septum successfully with surgical resection.

If affected horses are to be considered for an athletic career, surgery is indicated to resolve airway obstruction. Surgical resection of the septum can be incomplete or complete, depending on the degree of septal involvement. Small, focal areas of septum pathology can be resected without removal of the entire septum. Diffuse disease involving the septum or severe enlargement or deviation of the septum will require its complete removal. Preoperative planning should include a cross-match for a whole blood transfusion. A horse can lose 8 liters of blood quickly during this procedure, so it is advisable to administer IV fluids prior to surgery to increase the circulating blood volume.

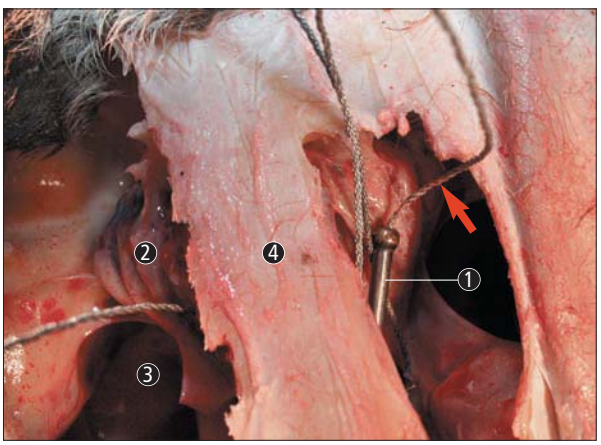
Briefly, the surgical procedure is performed under general anesthesia with the horse positioned in lateral recumbency. A tracheotomy is required because both nasal passages will have to be packed with sterile gauze to control hemorrhage following removal of the septum. The tracheotomy is performed just before anesthesia or just after anesthetic induction. An endotracheal tube is then inserted through the tracheotomy.

The preferred method of nasal septum resection is to use the three-wire technique. The first wire is positioned along the ventral aspect of the nasal septum.

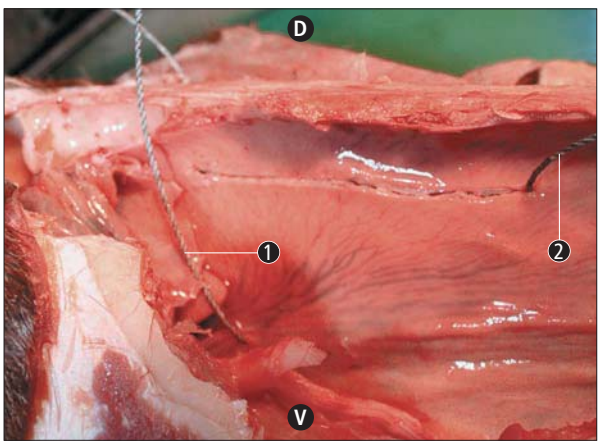


▲ **230** **Gross postmortem photograph with the right aspect of the nasal bones and turbinates removed. Note the position of the ventral wire (1), caudal wire (2), and the ethmoid turbinates (3). (D) dorsal.**

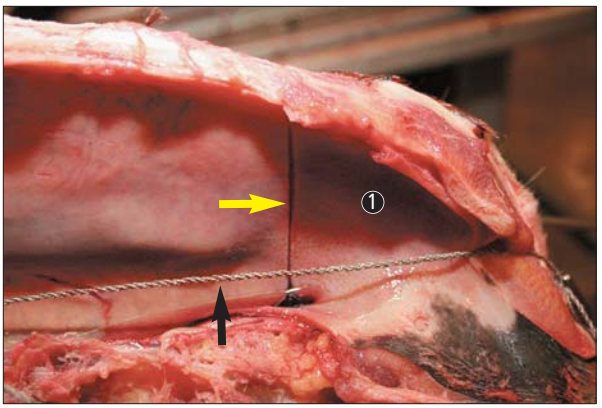
A 100-cm length of obstetrical wire is inserted on the left side of the nasal septum and passed towards the nasopharynx. A hand is inserted into the oral cavity to grasp this wire. This is repeated for the right side of the septum. The two ends of the wire are retracted from the mouth and spliced together. Once spliced, the wire is pulled from one side until the spliced section is out of the contralateral nostril, thus leaving one length of wire to perform the ventral septal cut (**230**). The second wire is inserted along the dorsal aspect of the septum. To access the most dorsal aspect, a trephine hole is made on the midline at the junction of the nasal bones. A Chambers catheter is inserted up the left nasal passage until it can be visualized at the trephine hole. A wire is inserted into the catheter and retracted out of the left nasal passage. This is repeated on the right side (**231**). Finally, the third wire is positioned along the caudal aspect of the septum. It is very important to position this wire to make sure the septum is cut at an angle, with the longest end caudally and the shortest end emerging at the trephine site. To place this wire, a Chambers catheter is inserted from the trephine hole into the nasopharynx. The wire is inserted through the catheter and grasped manually via a hand located in the oral cavity (**232**). This is repeated for the other side and spliced together as described for wire 1.



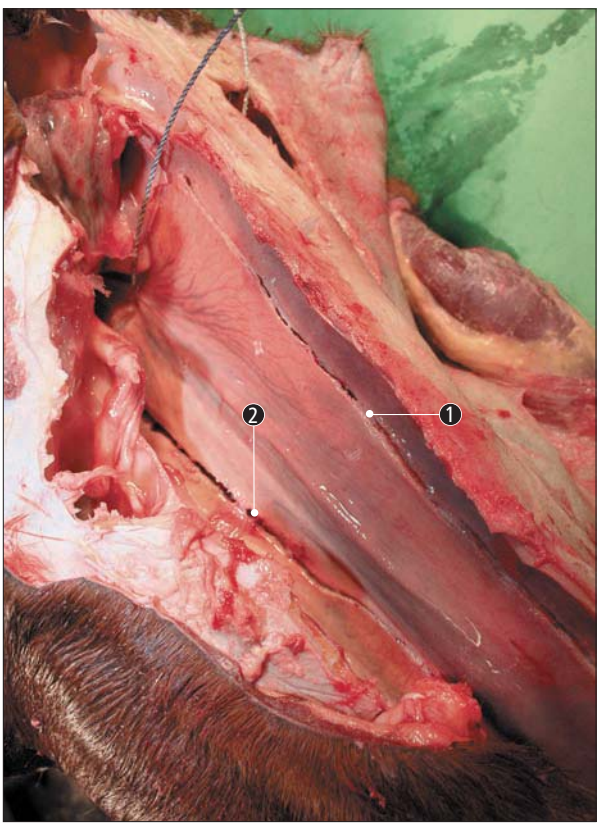
▲ **231** **Gross postmortem photograph illustrating the positioning of the dorsal wire (arrow) with the aid of a Chambers catheter (1) inserted into the dorsal nasal meatus. (2) ethmoid turbinates; (3) right frontomaxillary opening; (4) dorsal midline of the skull.**



▲ **232** **Gross postmortem photograph showing successful positioning of the caudal wire (1). Note the partial incision of the dorsal aspect of the nasal septum with the dorsal wire (2). (D) dorsal; (V) ventral.**



▲ 233 **Gross postmortem photograph detailing the positioning of the incision (yellow arrow) into the rostral aspect of the nasal septum (1). The black arrow denotes the positioning of the ventral wire.**



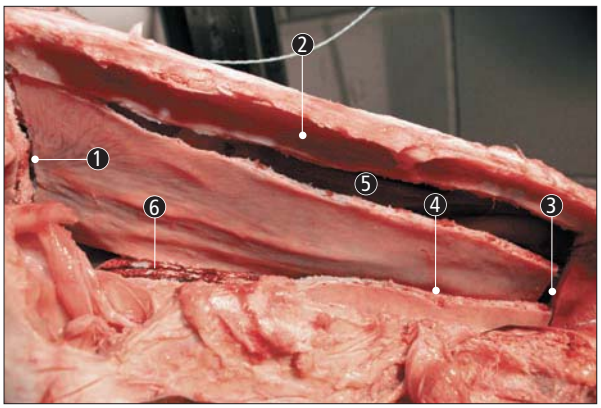
▲ 234 **Gross postmortem photograph following dorsal (1) and ventral (2) nasal septum cuts. A caudal cut has not been made.**

When all three wires have been positioned, the first stage of the procedure is to incise the rostral aspect of the nasal septum with a hard-back scalpel, leaving a 5-cm rim of rostral nasal septum in place (**233**). This is followed by transection of the septum with wire 3 (caudal aspect) and simultaneous transection with the dorsal and ventral wires (**234**). Once all four cuts have been made, the septum is grasped with a Vulsellum forceps and retracted out of the nares (**235**–**237**).

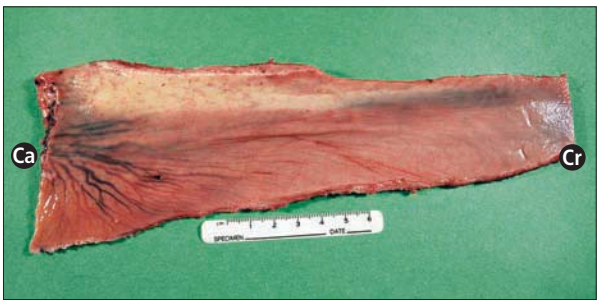
The ventral aspect of the transected septum is palpated for loosening of the palatine process of the incisive bone. If this process is loose, it is removed. The surgical site is immediately packed with sterile gauze to control hemorrhage. The packing is secured by suturing the nares closed by incorporating the packing in with the nostril sutures. This will prevent inadvertent removal of the packing by the horse. Following surgery horses are treated with antimicrobials and anti-inflammatory drugs. If required, based on postoperative packed cell volume (PCV) and total protein concentration, a whole-blood transfusion can be administered. The nasal packing is removed after 3 days. At this point the risk of hemorrhage is low. The nasal passages can be flushed with sterile polyionic fluids via the trephine hole for 3–5 days. The tracheotomy tube can usually be removed following packing removal. The tracheotomy and trephine sites are cleaned daily until closed by granulation tissue. A 60-day period of rest from exercise is recommended following septum removal. Complications of septum removal include swallowing of the nasal packing, postoperative hemorrhage, and incomplete caudal resection of the nasal septum. The prognosis following septum removal is fair to good. Younger horses may develop flattening of the nose because too much of the rostral septum is removed, thus not leaving enough septum to support the nares and alar cartilages.

Key points

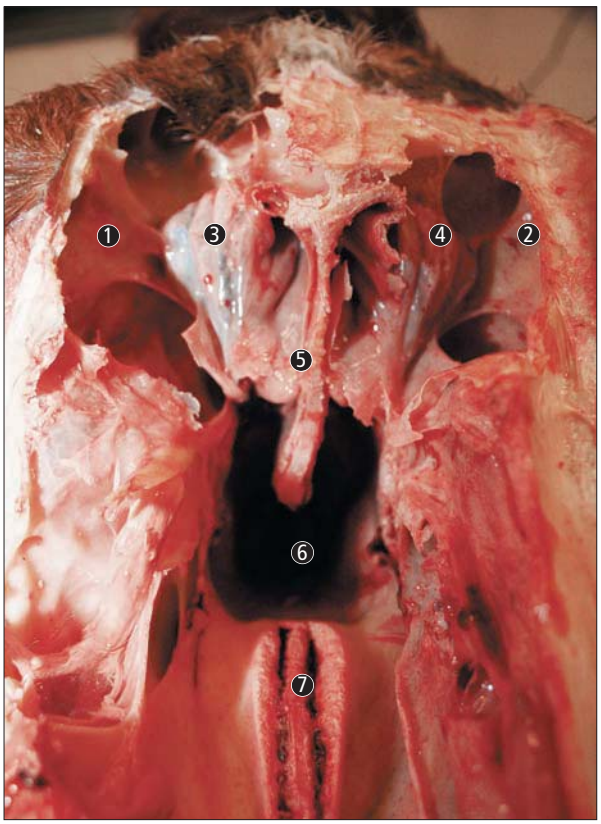
- Nasal septum pathology can lead to nasal passage obstruction and clinical signs of respiratory noise and exercise intolerance.
- Focal pathology of the nasal septum can be removed without complete resection of the nasal septum.
- Diffuse disease of the nasal septum requires complete nasal septum resection.
- The best method of nasal septum resection is the three-wire technique.
- The prognosis following nasal septum resection (in the absence of neoplasia) is favorable for athletic use.



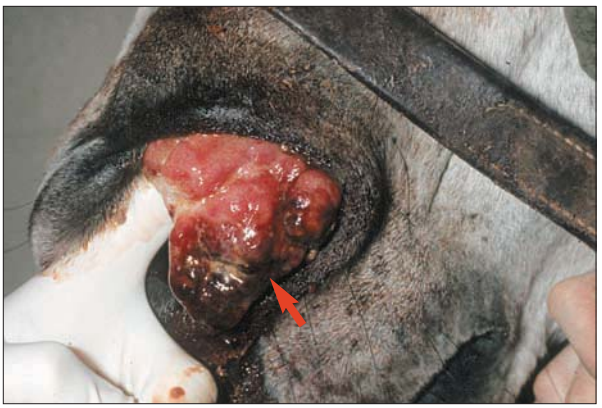
▲ 235 **Gross postmortem photograph following all four cuts: caudal cut (1), dorsal cut (2), rostral cut (3), and ventral cut (4). Note the contralateral nasal passage (5) and the base of the incisive bone (6).**



▲ 236 **Surgically resected nasal septum. (Cr) cranial; (Ca) caudal.**



▶ 237 **Gross postmortem photograph with the dorsal aspect of the nasal bones removed. Note the right (1) and left (2) frontal sinuses, the right (3) and left (4) ethmoid turbinates, the caudal nasal septum (5), the nasopharynx (6), and the vomer bone (7).**



◀ **238** **Horse with a large nasal polyp (arrow) exiting the left nostril.**

Nasal polyps

Definition/overview

Nasal polyps are benign, expansive masses that originate in the caudal nasal passage.

Etiology/pathophysiology

Nasal polyps are of unknown etiology. They are usually attached to the dorsal caudal aspect of the nasal passage or the alveolus of the third cheek tooth (108 or 208). Histopathology of nasal polyps reveals fibromatous tissue or bone, with respiratory epithelium covering the polyp. They are benign masses.

Clinical presentation

Nasal polyps are generally diagnosed in older horses (>10 years of age). In most cases the polyp can be visualized in the nares are just within the rostral aspect of the nasal passage (**238**).

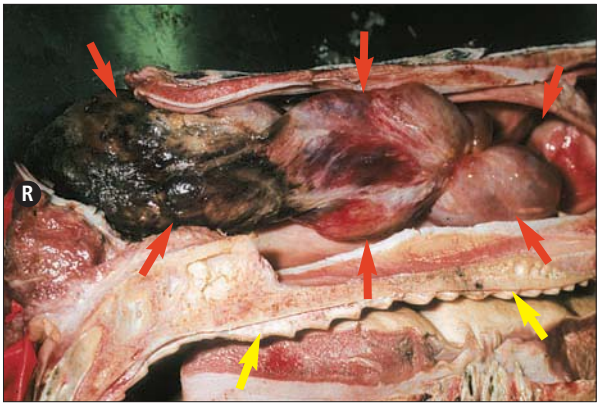
Nasal polyps are frequently referred to as a neoplastic mass because they appear aggressive externally. Other clinical signs include unilateral nasal discharge, fetid odor from the nares, and respiratory noise. Polyps should not be confused with ethmoid hematoma. Polyps are usually yellow to reddish and can be covered with granulation tissue. They sometimes appear gelatinous.

Differential diagnosis

Nasal passage neoplasia; ethmoid hematoma; conchofrontal or maxillary sinus sinusitis; sinus cysts; foreign body.

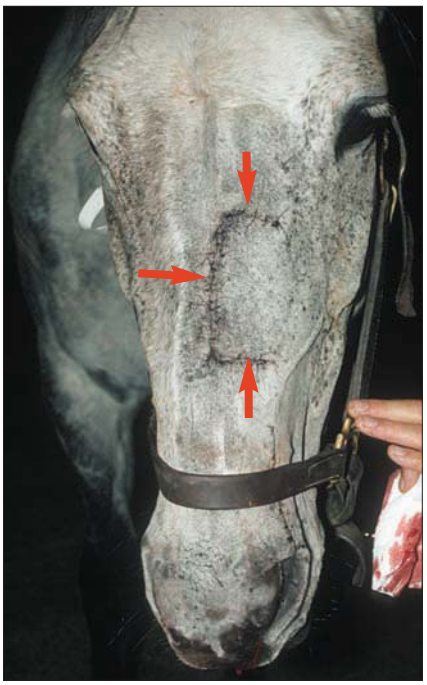
Diagnosis

Physical examination findings can be useful in making a preliminary diagnosis. Endoscopy should always be performed when evaluating nasal polyps. Endoscopic findings may include unilateral nasal obstruction, making it difficult to pass the endoscope around the mass. If the major bulbous portion of the mass can be bypassed with the endoscope, the operator may be able to visualize a stalk-like attachment to the nasal passage. Typically, the polyp appears yellow with a viscous mucous covering. Radiography should be performed to help determine the origin and attachment of the polyp. The minimum radiographic views required to evaluate for a nasal polyp include dorsoventral, lateral, and right and left oblique views of the nasal passage and paranasal sinuses. Radiographic findings compatible with a nasal polyp include a large, smooth mass located within the nasal passage. In some views a stalk of attachment may be observed. There should be no radiographic signs of bone lysis or destruction. If the diagnosis cannot be confirmed on physical examination and endoscopic and radiographic findings, a biopsy of the mass should be carried out. Samples for histopathology can be obtained by using finger fracture, uterine biopsy forceps, or following surgical removal. The authors generally recommend an excisional biopsy because small biopsies of the polyp are usually non-diagnostic. The biggest concern is that the histopathologic diagnosis may be compatible with a neoplastic growth, when in actuality nasal polyps are benign and not neoplastic.



▲ 239 **Nasal polyp (red arrows) *in situ* within the nasal passage. The yellow arrows identify the hard palate. (R) rostral.**

▼ 240 **Postoperative photograph of a horse following the creation of a nasal osteoplastic flap (arrows) to remove a nasal polyp from the caudal nasal passage. Note the orientation of the nasal flap with the base towards the lateral side. Most osteoplastic flaps are hinged on the midline. This type of flap affords better access to the attachment point of the polyp.**



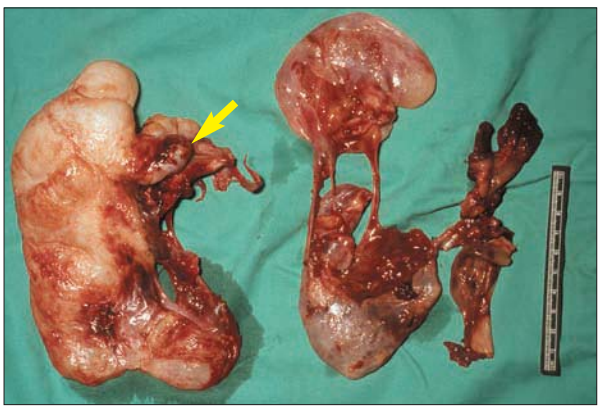
Management/treatment

There are no conservative treatments for nasal polyps. The best approach is surgical removal. There have been reports of minimally invasive polyp removal and there is one case report of using obstetrical wire to snare the cyst and remove it by sawing its point of attachment. The authors' preference is to approach the cyst via an osteoplastic nasal bone flap (**239**, **240**). However, it is important to center the flap over the origin of the polyp. The polyp generally only originates from a small attachment site, making surgical removal straightforward (**241**). The stalk is transected with sharp excision. If desired, a diode or Nd:YAG laser can be used to cauterize the stalk at the time of surgical removal. Once the attachment point is transected, the mass is removed *en bloc* and the origin is curetted. The prognosis for surgical removal is good and polyps do not recur if the origin of the mass is completely excised.

Key points

- Nasal polyps are benign masses originating from the nasal passage.
- They are locally expansive, but only originate from a narrow 'stalk' of attachment.
- Surgical management is the preferred method of treatment and results in the best chance for a successful outcome.

▼ 241 **Excised sections of a nasal polyp. Note the relatively small attachment point (arrow) to the nasal passage.**



Pharyngeal and laryngeal diseases

Dorsal displacement of the soft palate

Definition/overview

DDSP occurs when the soft palate displaces dorsally in relation to the epiglottis. It can be intermittent or persistent. Intermittent DDSP is diagnosed when soft palate displacement occurs at uncertain intervals and persistent DDSP occurs when the soft palate is displaced continuously.

Etiology/pathophysiology

Subluxation of the soft palate results in displacement of the free edge of the soft palate dorsal to the epiglottis. DDSP causes a narrowing of the airway and creates air turbulence during inspiration and expiration. During expiration, air is directed into the mouth and nasopharynx. This is a very inefficient means of respiration. DDSP can be intermittent (during exercise) or persistent as previously stated.

Causes of DDSP include:

- **Paralysis or paresis of the soft palate.** Causes of soft palate paralysis or paresis include cranial nerve dysfunction associated with GP disease and botulism.
- **Elongated soft palate.** There is no scientific evidence that the soft palate is too long. It is much more likely that the soft palate is not too long, but rather that the epiglottis is too short (epiglottic hypoplasia).
- **Epiglottic hypoplasia.** The length of a normal epiglottis in Thoroughbred and Standardbred racehorses is 8.0–8.5 cm. Most horses with epiglottic hypoplasia have an epiglottic length of 7.0–7.5 cm. Epiglottic hypoplasia, both in length and thickness (flaccidity), does occur. Either change can contribute to the development of DDSP.
- **Pharyngeal hyperplasia** or inflammation of the URT.
- **Caudal retraction of the larynx.** This occurs secondary to excessive pull of the ventral strap muscles of the neck (sternothyroideus and sternohyoideus muscles) or increased negative pressure in the pharynx.
- **Excessive head and neck flexion** can predispose to soft palate displacement because of narrowing of the nasopharynx.
- **Root of the tongue pushing up on the palate** during swallowing. Opening the mouth or swallowing during exercise.
- **Dysfunction of the soft palate musculature.** Of all the potential causes of DDSP, neuromuscular dysfunction of the soft palate is the most likely cause of soft palate displacement.
- **Fatigue** from intense exercise can trigger DDSP in predisposed horses.

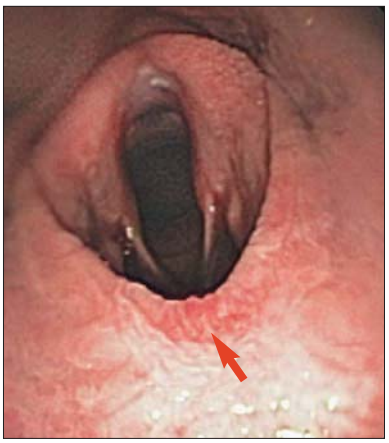
Obviously, when there are numerous potential causes of DDSP, the disease process is most likely multifactorial and the definitive cause is unknown.

Clinical presentation

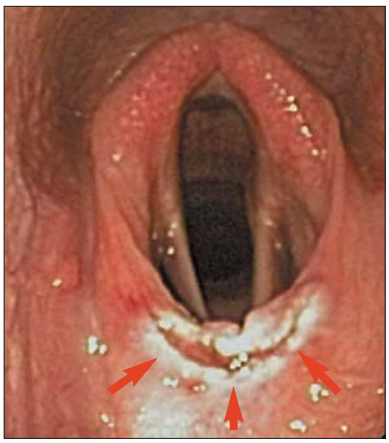
The most common age group affected with DDSP is young (2–3 year old) racehorses. Thoroughbreds and Standardbreds are the most common breeds represented, although the disease does occur in other breeds and horses of all ages. Breeds that emphasize excessive poll flexion or collection are at risk of DDSP because of narrowing of the nasopharynx with poll flexion. Horses with GP disease may also develop DDSP. Clinical signs associated with intermittent DDSP include upper respiratory tract noise and exercise intolerance. The most common type of respiratory noise is expiratory, and some owners report a gurgling or choking type of noise. Horses with exercise intolerance have a history of 'stopping' during racing or choking down. Once the soft palate displaces, horses almost immediately have problems maintaining racing speed because of the inefficiency associated with mouth breathing following displacement of the palate. Standardbred racehorses typically show clinical signs at the ¾-mile point of their 1-mile race and Thoroughbred racehorses show clinical signs near the ½-mile point of the race.

Differential diagnosis

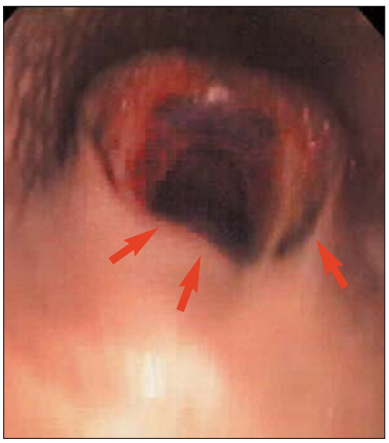
EE; epiglottitis; epiglottic retroversion; laryngeal hemiplegia; subepiglottic cysts; tracheal disorders.



▲ 242 **Dorsal displacement of the soft palate identified during standing endoscopy. Note area of hyperemia (arrow) along the caudal free edge of the soft palate**



▲ 243 **Endoscopic photograph of a Standardbred racehorse with ulceration of the soft palate (arrows) secondary to repeated episodes of dorsal displacement of the soft palate.**



▲ 244 **Endoscopic photograph of dorsal displacement of the soft palate (arrows) during high-speed treadmill exercise.**

Diagnosis

History is very important in the diagnosis of DDSP. DDSP is frequently confused with laryngeal hemiplegia. The predominant difference between the two disorders is the type of respiratory noise made. Horses with DDSP typically make an expiratory noise and horses with laryngeal hemiplegia make an inspiratory noise. A complete physical examination including external palpation of the head and neck should be performed. Some horses with DDSP have palpable hypertrophy of the sternohyoideus or sternothyroideus muscles.

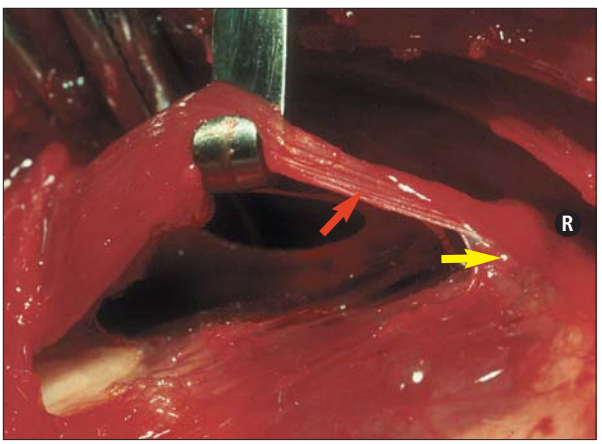
The best diagnostic tool for DDSP is endoscopy. All horses with a history compatible with DDSP should have a standing endoscopic examination performed (**242**). Horses with a normal standing endoscopic examination should be considered for a dynamic examination, either on a treadmill or over ground using portable units. Endoscopic findings compatible with or contributing to DDSP include easily inducible palate displacement by swallowing, hyperemic pharyngeal ring in acute cases, epiglottic hypoplasia, narrow pharyngeal vault, EE, epiglottitis, and ulceration on the caudal free edge of the soft palate (**243**). It is possible for endoscopy at rest to be normal even in horses with a strong history of DDSP. If the standing endoscopic examination is unremarkable or not diagnostic for DDSP, a dynamic endoscopic examination is indicated. High-speed treadmill examination allows for dynamic assessment of soft palate and epiglottic function (**244**). Recently, portable endoscopy systems have allowed dynamic endoscopy to be conducted in the field. However, diagnosis of DDSP is more likely to be obtained with treadmill endoscopy. Visual observation of DDSP is confirmatory of the disease, but not observing DDSP does not rule it out. The predominant reason to perform dynamic endoscopy in horses suspected of DDSP is to eliminate other causes of respiratory noise and exercise intolerance. Therefore, if no other disorders are diagnosed and the horse does not develop DDSP during exercise, but the historical findings are compatible with DDSP, then the most likely cause for the clinical signs is DDSP.

Management/treatment

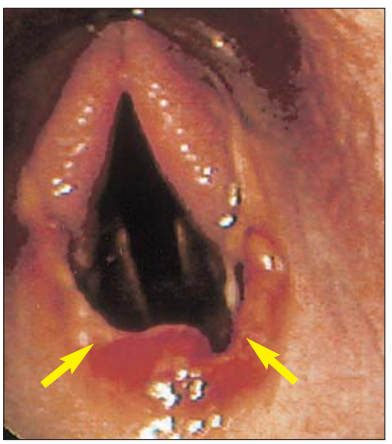
Management of DDSP can be conservative or surgical. Conservative management should always be pursued before proceeding with surgical treatment. Some horses with documented inflammation of the URT, including grade 3 or higher pharyngeal lymphoid hyperplasia, can be treated with antimicrobial and anti-inflammatory therapy. Horses with inflammatory conditions should be treated by withdrawal from exercise. Unfortunately, racehorse trainers are reluctant to completely rest horses with this problem. Additional conservative treatments for DDSP include a tongue tie to prevent caudal retraction of the tongue and limit swallowing, a figure-of-eight or dropped nose band to prevent opening of the mouth during exercise, straightening of the head position to prevent excessive flexion of the head and neck, and changing the type of bit used. Finally, a Cornell Collar® can be used. This is an external device that supports the larynx during exercise. The collar has a thermoplastic lifting mechanism, which moves the larynx into a more rostral and dorsal position by mimicking the action of the thyrohyoid muscle. This collar is worth considering when managing horses initially diagnosed with DDSP.

Horses who do not respond to conservative management should be considered for surgery. Many procedures have been described to treat DDSP, but there is no single procedure that provides the best chance for a successful outcome. Procedures include sternohyoideus, sternothyroideus myectomy, sternothyroideus tenectomy (**245**), staphylectomy (**246**), epiglottic augmentation, combinations of these procedures, laser palatoplasty, and laryngohyoid shortening (laryngeal tie forward). The three procedures currently used the most are sternothyroideus tenectomy alone or combined with staphylectomy, laser palatoplasty, and laryngeal tie forward. Typically, a decision is agreed between the trainer/owner and the surgeon as to which should be performed. Sternothyroideus myotenectomy in combination with staphylectomy can be performed on an outpatient basis and in general most horses can return to exercise in 2–3 weeks following surgery.

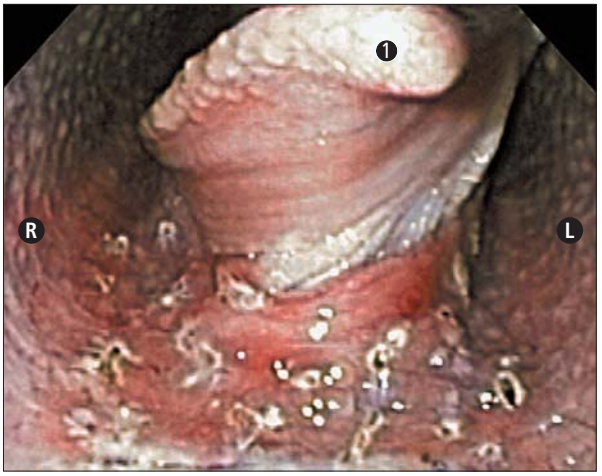
Laser palatoplasty is performed with the horse standing and can be done on an outpatient basis (**247–249**). Its advantage is that horses can return to exercise promptly and an external surgical incision is not required. Laser palatoplasty can also be combined with the laryngeal tie forward.



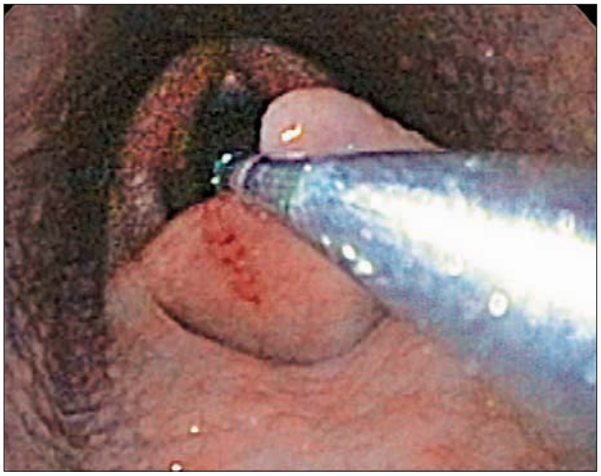
▲ **245** **Intraoperative photograph of the sternothyroideus muscle and tendon of insertion on the thyroid cartilage (yellow arrow). The musculotendinous junction is being elevated with an ovariohysterectomy hook. The red arrow indicaes the tendon of this muscle prior to insertion on the thyroid cartilage. (R) rostral.**



▲ **246** **Endoscopic photograph of a horse following staphylectomy (arrows).**

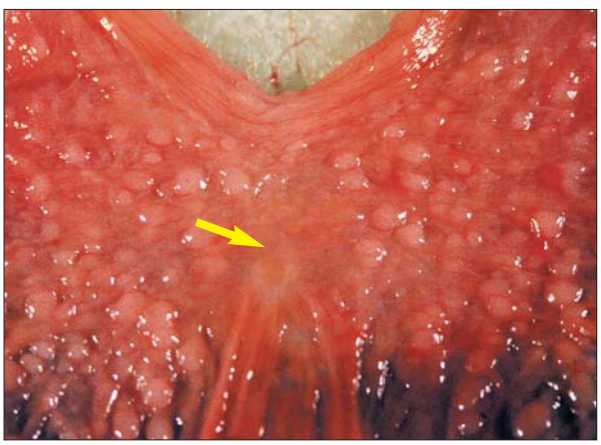


▲ 247 **Endoscopic photograph following diode laser palatoplasty. (1) epiglottis; (R) right aspect of the nasopharynx; (L) left aspect of the nasopharynx.**

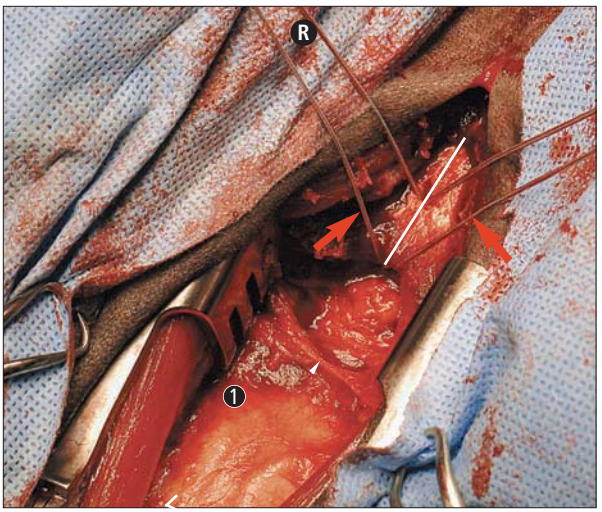


▲ 248 **Endoscopic photograph of the horse in 247 45 days following laser palatoplasty.**

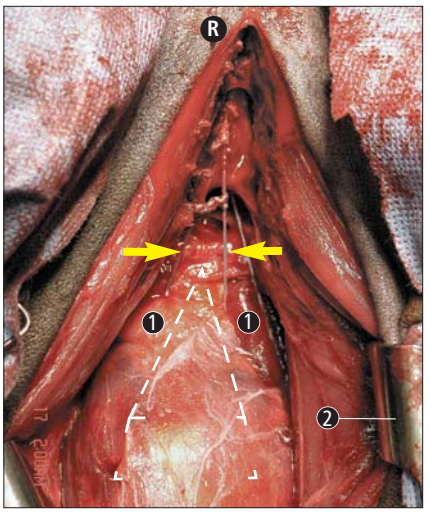
Because of a higher published success rate, the authors generally recommend that laryngeal tie forward, which was developed at Cornell University, is presented as the surgical treatment of choice. The basis for this technique is to surgically shorten the distance between the larynx and the hyoid apparatus. Researchers at Cornell University determined that transection of the thyrohyoid muscle on the ventral aspect of the larynx resulted in DDSP in clinically normal horses. They then developed a surgical technique to re-establish the function of the muscle and move the larynx forward towards the hyoid apparatus. Laryngeal tie forward involves a ventral approach to the larynx. The ventral aspect of the larynx is exposed via a ventral midline incision. Two, separate, non-absorbable sutures are used for the procedure. One suture is looped around the basihyoid bone adjacent to



▲ 249 **Postmortem photograph of the soft palate 45 days post diode laser palatoplasty. Note the focal areas of soft palate fibrosis (arrow) following the procedure.**



▲ **250** **Intraoperative photograph detailing the successful positioning of the right and left tie forward sutures (arrows) prior to their insertion through the lamina of the thyroid cartilage (1). The triangle indicates the cricothyroid space; the solid line identifies the basihyoid bone. (R) rostral.**



▲ **251** **Intraoperative photograph showing the final positioning of the larynx following tying of the sutures (arrows). Note the thyroid cartilage (1), cricothyroid space (triangle), cricoid cartilage (rectangular box), and the Beckman Adson forceps (2) used to retract the sternohyoideus muscles. (R) rostral.**

the lingual process of this bone. This is repeated for the opposite side (**250**, **251**).

Next, one suture is passed twice through the thyroid cartilage to minimize disruption of the cartilage when the suture is tightened. Alternatively, a suture button can be used to lessen the likelihood of suture pullout, which has been observed with the 'two-pass' suture technique. This is repeated for the opposite side. The final step of the procedure before tying the sutures is to flex the head. With the head flexed each suture is tightened sequentially so that the rostral aspect of the thyroid cartilage is within 2 cm of the basihyoid bone. The reported success rate of laryngeal tie forward is approximately 80%. This is higher than has been reported for any of the other surgical procedures used for treating DDSP. The success rate for all other procedures ranges from 60–65%. However, a consistent, well established postoperative protocol for evaluation of the horse following surgical management of DDSP has not been accomplished.

Key points

- DDSP is a common cause of respiratory noise and exercise intolerance in horses.
- DDSP is typically diagnosed on the basis of history and clinical signs and a resting endoscopic examination. High-speed treadmill examination should be considered for cases in which the resting endoscopic examination is not confirmatory of DDSP.
- Conservative management should be pursued initially for all cases of DDSP.
- Horses who do not respond to conservative management should be considered for surgical management.
- The preferred method of surgical treatment is the laryngeal tie forward.

Epiglottic entrapment

Definition/overview

EE occurs when the aryepiglottic fold becomes edematous and/or hypertrophied and envelops the epiglottis. EE can be diagnosed when the serrated edge of the epiglottis and the prominent vascular pattern on the dorsal surface of the epiglottis cannot be seen. EE should not be confused with DDSP. With DDSP the epiglottic cartilage is not visible because it is below the displaced soft palate.

Etiology/pathophysiology

The etiology of EE is multifactorial. It can be congenital and found as an incidental finding. This seems to be more common in horses with epiglottic hypoplasia. EE has also been reported in young horses with cleft palate or soft palate hypoplasia. Most horses acquire EE or are diagnosed with the condition after they start race training. Standardbred and Thoroughbred racehorses are at highest risk of developing DDSP. Risk factors for EE include epiglottic hypoplasia, subepiglottic cysts, epiglottitis, and DDSP. Some investigators believe that frequent DDSP irritates the lingual aspect of the epiglottis, resulting in edema and hypertrophy of the aryepiglottic fold. A hypertrophic aryepiglottic fold is more likely to entrap the epiglottis; this can contribute to DDSP and respiratory tract obstruction. Respiratory tract obstruction is more pronounced during exhalation; when air is trapped under the aryepiglottic fold, it billows the epiglottis dorsally.

Clinical presentation

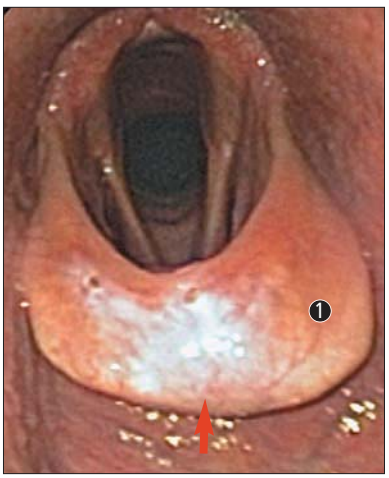
The most common clinical feature of EE is URT noise and exercise intolerance. Sometimes the only clinical sign may be poor racing performance. EE is then discovered on a post-racing endoscopic examination. In some horses, EE is intermittent and only occurs during high-speed exercise, but repeated swallowing as the horse slows down may resolve the EE, making standing endoscopy immediately after exercise inconclusive.

Differential diagnosis

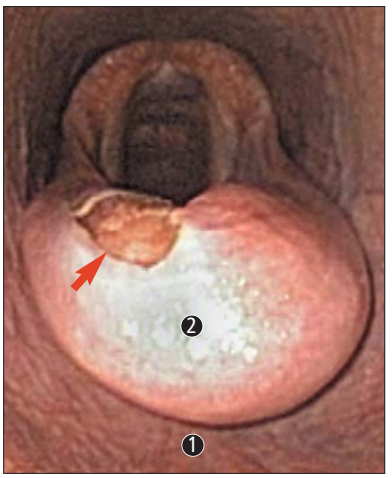
DDSP; epiglottitis; epiglottic retroversion; collapse of the aryepiglottic folds; laryngeal hemiplegia; subepiglottic cysts; tracheal disorders.

Diagnosis

The best diagnostic test for EE is endoscopy. Endoscopic findings compatible with EE include the presence of the aryepiglottic fold enveloping the epiglottis, inability to visualize the serrated edge of the epiglottis, and inability to visualize the normal vascular pattern on the dorsal surface of the epiglottis. It is not uncommon for the entrapping membrane to be partially ulcerated. EE has been classified as acute or chronic, thick or thin, ulcerated or non-ulcerated, wide or narrow, and complete or incomplete (**252**, **253**). The majority of horses with EE can be diagnosed with standing endoscopic examination. However, in cases of intermittent EE it may be necessary to perform a high-speed treadmill examination to confirm the diagnosis. Some horses



◀ 252 Chronic, thin, wide, complete, epiglottic entrapment. Note the blunted epiglottic tip (arrow) indicating chronic deformation of the epiglottis by the entrapping aryepiglottic fold (1).

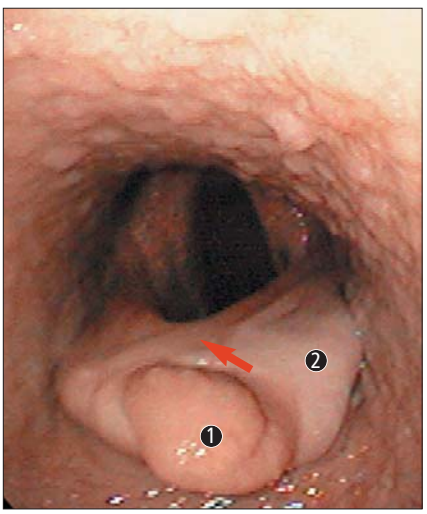


▶ 253 Chronic, thick, wide, complete epiglottic entrapment with ulceration (arrow). There is a normal orientation between the epiglottis and the soft palate (1). (2) aryepiglottic fold.

will only develop EE during high intensity exercise. Another diagnostic test that can be used is a lateral view radiograph of the throat latch area. Horses with EE may have radiographic evidence of epiglottic hypoplasia and/or thickening of the epiglottis, with a soft-tissue mass effect on its dorsal surface.

Management/treatment

EE can be managed conservatively or surgically. Conservative management is chosen for incomplete or intermittent cases of EE and is sometimes chosen if the horse still needs to compete in a race prior to definitive treatment. Conservative management should include complete removal from exercise, topical and systemic anti-inflammatory drugs (e.g. corticosteroids, phenylbutazone, and/or flunixin meglumine), and in some instances, where infection is of concern, antimicrobial therapy. The most important treatment is rest, which is sometime difficult to get trainers and owners to agree to. Rest helps minimize continued inflammation of the epiglottis while medical management is attempted.

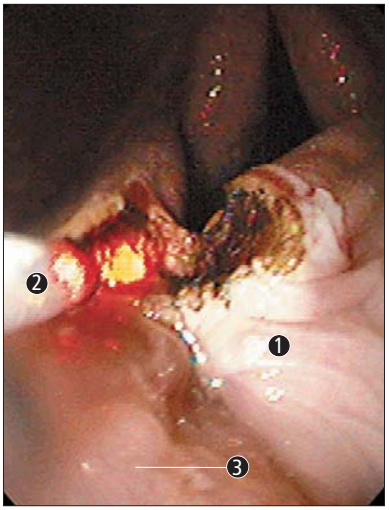


▲ **254 An unusual case of epiglottic entrapment with the epiglottic tip (1) protruding through a defect in the aryepiglottic fold (2). The arrow denotes the 'bridge' of the fold between the right and left aspects of the aryepiglottic fold. This is an ideal candidate for standing diode laser correction.**

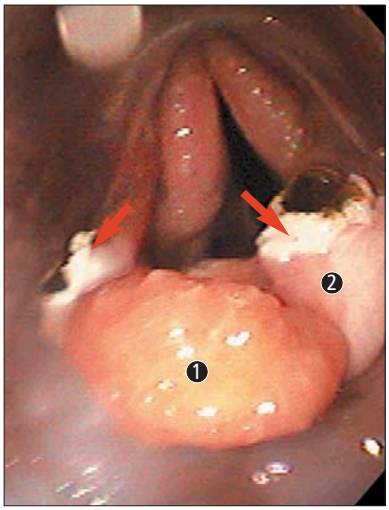
Nevertheless, the majority of horses with EE should be treated by surgical intervention. The accepted surgical techniques for management of EE include axial division of the aryepiglottic fold with standing laser correction, bistoury, or via laryngotomy. Horses with thin, wide, mildly ulcerated, and complete EE can be treated successfully either with a diode laser or with a bistoury. Horses with thick, wide, ulcerated, and complete EE should be strongly considered for resection of the aryepiglottic fold via laryngotomy.

Important points to consider when performing standing diode laser correction of EE include: minimizing iatrogenic trauma to the epiglottis, especially the epiglottic tip (**254**); using contact techniques rather than non-contact technique; precise, axial division of the aryepiglottic fold with the laser fiber (**255**, **256**); and adequate incision of the fold. The goal of standing correction is to minimize the amount of laser energy to the epiglottis and to avoid damage to the epiglottic tip cartilage (**257**). Surgeon experience with this technique is paramount to achieving a successful outcome.

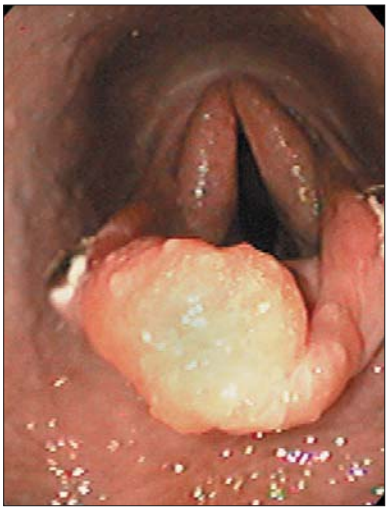
If a laser is not available, the second best option is axial division of the aryepiglottic fold with a bistoury (**258**, **259**). A hooked, curved bistoury is used to perform transaction of the fold. The bistoury can be inserted through the nasal passage with the horse standing (**260**) or through the oral cavity with the horse standing or recumbent. The primary disadvantage of standing nasal correction of the EE is iatrogenic damage to the soft palate, which may result in a cleft palate. This occurs when the horse moves during transection of the fold and the bistoury cuts the soft palate instead of incising the aryepiglottic fold. This seriously limits the chances of the horse resuming an athletic career. Therefore, this method is not recommended by the authors. Oral correction while standing has been recently described and is an acceptable method of treatment. However, the authors prefer to use a bistoury with the horse anesthetized in either lateral or dorsal recumbency. The primary disadvantage of this method is the anesthetic risk. However, the advantages are ease of bistoury placement and direct endoscopic observation of the transection, which outweigh the general anesthesia risks. The prognosis following laser or bistoury correction of EE is the same.



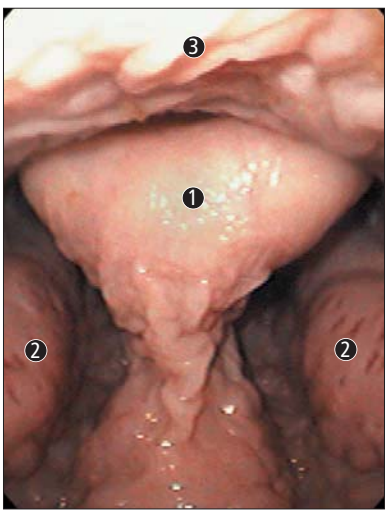
▲ 255 Axial division of the aryepiglottic fold (1) with a diode laser (2). Note the epiglottic tip (3) showing through a defect in the aryepiglottic fold.



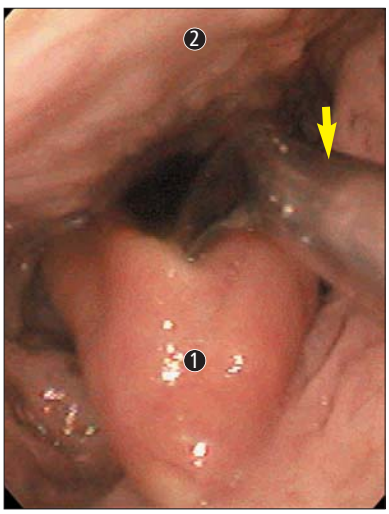
▲ 256 Completed laser correction of an epiglottic entrapment. Note the cut edges of the aryepiglottic fold (arrows), with a freed epiglottic tip (1). (2), left aryepiglottic fold.



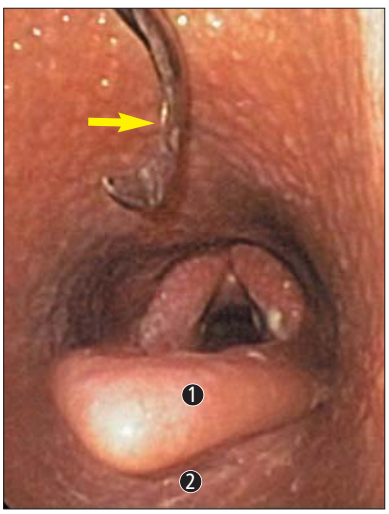
▲ 257 Retracted edges of the right and left aryepiglottic folds. Note the bulbous appearance of the epiglottic tip.



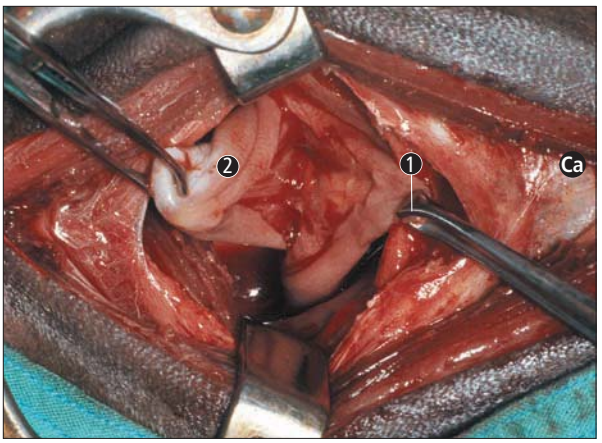
▲ 258 Intraoral view of epiglottic entrapment by the aryepiglottic fold (1). The right and left oral tonsils (2) are to the sides of the epiglottis. (3) soft palate.



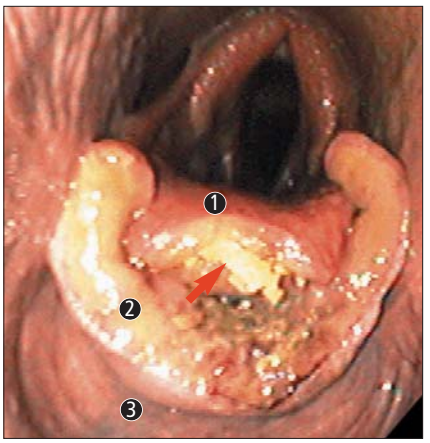
▲ 259 Oral endoscopic photograph of the bistoury knife (arrow) engaging the aryepiglottic fold (1). (2) soft palate.



▲ 260 Standing transnasal correction of an epiglottic entrapment (1) with a bistoury knife (arrow). Note the normal relationship between the epiglottis and the soft palate (2).



▲ **261** **Intraoperative photograph of correction of an epiglottic entrapment via laryngotomy. An Allis tissue forceps is grasping the epiglottic tip (1) and scissors have incised a portion of the aryepiglottic fold (2). (Ca) caudal; the horse's right side is at the bottom of the image. (Photo courtesy JF Fessler)**



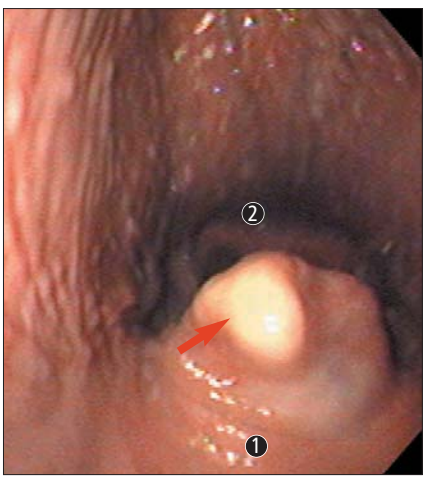
▲ **262** **Endoscopic photograph of a horse with complications following laser correction of epiglottic entrapment. Because of the chronicity of the entrapment, necrosis of the epiglottic tip (1) occurred, allowing for exposure of epiglottic tip cartilage (arrow). Note the granulation tissue on the margins of the aryepiglottic fold (2). The prognosis for a successful return to athletic function is guarded because of the increased risk of epiglottic deformity following the resolution of severe epiglottic inflammation. (3) soft palate.**

The final method of correction is via laryngotomy (**261**). The authors reserve correction via laryngotomy for thick, wide, ulcerated, and complete EE. In their experience, standing laser correction of this type of EE typically results in prolonged healing, inflammation, and a delay in return to training. Laryngotomy correction has the advantage of excision of the majority of the aryepiglottic fold via sharp incision. This tends to limit the amount of inflammation the horse experiences following surgery. The authors prefer to use the endoscope, positioned transorally, to visualize the epiglottis before grasping the aryepiglottic fold via the laryngotomy. A sponge forceps is used to grasp the fold, which is retracted towards the laryngotomy incision. The central third of the aryepiglottic fold is then resected with scissors. The epiglottis is returned to the oropharynx and examined endoscopically to ensure that an adequate amount of aryepiglottic fold has been removed. The fold should also be manually palpated via the mouth to make sure the epiglottis cannot be re-entrapped. If re-entrapment can be induced, the epiglottis is again retroverted into the laryngotomy incision and more of the aryepiglottic fold removed. The prognosis following laryngotomy correction of EE is poorer than that observed with other methods of surgical correction.

The overall prognosis for treatment of EE is approximately 65–75%. Regardless of surgeon experience and postoperative management, not all horses treated surgically have a successful outcome. The primary reasons for this include prolonged epiglottic inflammation (**262**), epiglottic deformity, DDSP, and re-entrapment. It is crucial that trainers and owners understand that it is the rest following the surgical procedure that leads to a better chance at a successful outcome.

Key points

- EE is a common cause of URT noise and exercise intolerance.
- EE is diagnosed with endoscopy.
- Epiglottic hypoplasia and DDSP are the major risk factors for development of EE.
- Thin, wide, mildly ulcerated, and complete EE can be managed successfully with either contact diode laser or bistoury axial division of the aryepiglottic fold.
- Chronic, thick, wide, ulcerated, and complete EE should be considered for surgical correction via laryngotomy.
- The prognosis following surgical correction is favorable, but problems with prolonged inflammation and edema can lead to a delayed return to racing.



▲ **263** **Endoscopic photograph of a horse with epiglottic retroversion during high-speed treadmill examination. Note the elevation of the epiglottis and epiglottic tip (arrow). There is normal orientation between the epiglottis and the soft palate (1). (2) arytenoid cartilages.**

Epiglottic retroversion

Definition/overview

Epiglottic retroversion is a disease of unknown etiology that results in elevation of the epiglottis towards the rima glottis during high-speed exercise.

Etiology/pathophysiology

The etiology of epiglottic retroversion is unknown. Possible etiologies that have been suggested include trauma to the hyoid apparatus secondary to respiratory disease and neuromuscular dysfunction of the geniohyoid muscle and hyoglossal nerve Epiglottic retroversion has been created experimentally by local anesthesia of the geniohyoid muscle and hyoglossal nerve within the GP.

Clinical presentation

The primary clinical feature of epiglottic retroversion is a gurgling or honking inspiratory noise during high-speed exercise. Horses do not usually make the abnormal noise at rest. Affected horses also exhibit exercise intolerance in addition to the URT noise.

Differential diagnosis

DDSP; epiglottitis; collapse of the aryepiglottic folds; laryngeal hemiplegia; subepiglottic cysts/abscess; tracheal disorders.

Diagnosis

Evaluation of a horse suspected of epiglottic retroversion should include a complete physical examination, auscultation of the heart and lungs, and a standing endoscopic examination. Endoscopic examination at rest may reveal no abnormalities involving the epiglottis. Because of the potential for an inflammatory etiology, the interior of both GPs should be examined. In the absence of any abnormalities during a standing endoscopic examination, horses should be exercised on a high-speed treadmill. During treadmill exercise horses affected with epiglottic retroversion exhibit dorsal elevation of the epiglottis towards the rima glottis during inspiration (**263**). A loud gurgling or honking noise may also be heard. In addition to elevation, the epiglottis may appear to undulate or vibrate with inspiration.

Management/treatment

As only a few cases of epiglottic retroversion have been reported, it is difficult to determine what is the best method of treatment for this condition. In one case report detailing the treatment of two horses with epiglottic retroversion, both horses were treated with Teflon augmentation of the epiglottis. One horse was able to return to racing at a lower level and one horse was retired from racing and became a broodmare. The authors currently do not recommend a specific therapy and offer owners a poor prognosis for return to athletic performance. However, new treatments for this condition, which may be useful in the surgical management of this disease in the future, are being developed at Cornell University in the US.

Key points

- Epiglottic retroversion is a rare condition of unknown etiology, although neuromuscular dysfunction of the geniohyoid muscle or hypoglossal nerve is suspected.
- Confirmation of epiglottic retroversion must be made during high-speed endoscopy.
- Treatment options for epiglottic retrovesion are limited, although some horses may respond to epiglottic augmentation.

Axial deviation of the aryepiglottic folds

Definition/overview

Dynamic collapse of the aryepiglottic fold(s) occurs during high-speed exercise; the folds collapse axially causing URT noise and exercise intolerance.

Etiology/pathophysiology

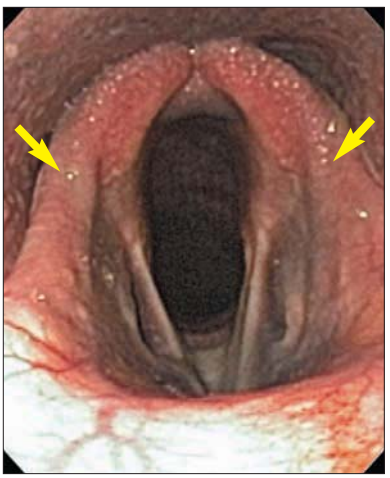
The etiology of axial deviation of the aryepiglottic fold is idiopathic. Investigators have speculated that it could be a neurologic dysfunction or of a similar pathogenesis to DDSP.

Clinical presentation

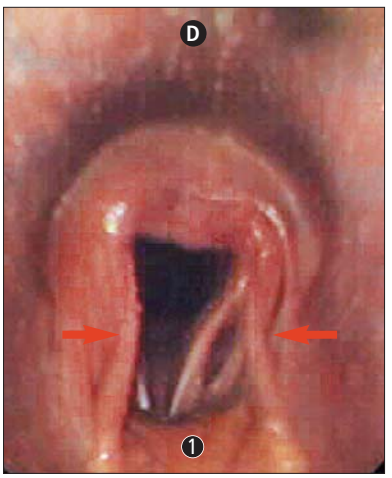
Horses present with clinical signs of URT noise and exercise intolerance. The disorder can be confused with DDSP and laryngeal hemiplegia. Axial deviation of the aryepiglottic fold can only be recognized during high-speed exercise.

Differential diagnosis

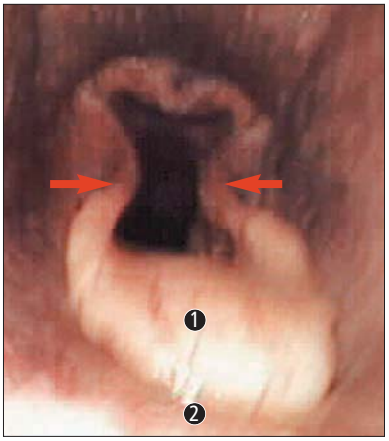
DDSP; epiglottitis; epiglottic retroversion; laryngeal hemiplegia; subepiglottic cysts; tracheal disorders.



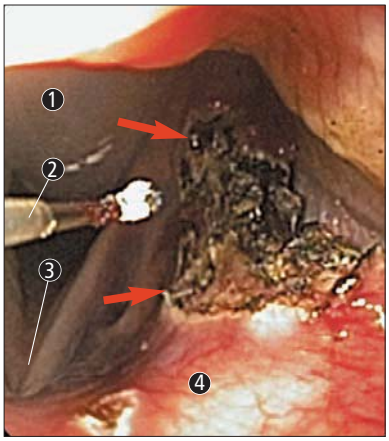
◀ 264 **Standing endoscopic examination at rest of a horse with axial deviation of the aryepiglottic folds during high-speed treadmill exercise. Note the normal appearance of the larynx and no evidence of abnormalities involving the aryepiglottic folds (arrows).**



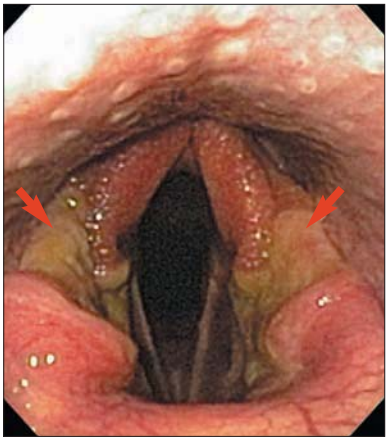
▶ 265 **Endoscopic photograph of a horse with mild aryepiglottic fold collapse (arrows) on the high-speed treadmill. (1) epiglottis; (D) dorsal.**



▲ **266** **Endoscopic photograph of a horse with moderate aryepiglottic fold deviation (arrows) while exercising on a high-speed treadmill. (1) epiglottis; (2) soft palate.**



▲ **267** **Endoscopic photograph of the horse in 266 being treated with laser excision of the left aryepiglottic fold (arrows) while under general anesthesia. (1) endotracheal tube; (2) laser fiber; (3) vocal cords; (4) epiglottis.**



▲ **268** **Endoscopic photograph of the horse in 266 7 days post laser surgical excision of the aryepiglottic fold (arrows).**

Diagnosis

Standing endoscopy should be performed in all cases of suspected aryepiglottic fold collapse. This is mainly to rule out other disorders causing URT noise and exercise intolerance. In most horses suspected of having this disorder the standing endoscopic examination is normal (**264**); to confirm the diagnosis they must be examined by dynamic endoscopy at high speed. Horses with axial deviation of the aryepiglottic fold on the treadmill can be categorized into mild, moderate, or severe. Mild cases have both folds deviated towards the midline approximately 2 cm (**265**). Moderate cases have both folds almost touching the midline of the larynx (**266**). Severe cases have both folds touching the midline of the larynx. Affected horses will make a respiratory noise while exercising on the treadmill.

Management/treatment

Horses with axial deviation of the aryepiglottic folds (**266**) are best treated with surgical excision of the folds either via laryngotomy or with standing or recumbent diode laser excision. The advantage of surgical removal with a diode laser is that the procedure can be done with the horse standing under chemical restraint. The authors' preference is to perform the procedure with the horse standing. A bronchoesophageal forceps is passed through the nasal passage contralateral to the endoscope. The forceps are then used to grasp the fold. A triangular portion of the fold is then excised using a contact diode laser fiber (**267**, **268**). The prognosis following surgical resection of the aryepiglottic folds is good and complications associated with the laser procedure are rare.

Key points

- Axial deviation of the aryepiglottic folds results in exercise intolerance and URT noise in racehorses.
- Diagnosis of axial deviation of the aryepiglottic folds can only be made during high-speed treadmill examination.
- Surgical resection of the aryepiglottic folds provides the best chance for a return to athletic performance.
- The prognosis following surgical resection of the aryepiglottic folds is good.

Epiglottitis

Definition/overview

Epiglottitis is an inflammatory process of the epiglottis that results in edema, reddening, and thickening of the epiglottis and aryepiglottic fold.

Etiology/pathophysiology

The cause of epiglottitis in horses is not known. Possible predisposing factors include: pharyngeal inflammation from respiratory tract infection or inhaled dust, dirt, or allergens; mucosal irritation initiated by intermittent DDSP, EE, trauma from ingestion of foreign bodies, or coarse roughage; or non-specific irritation caused by the stress of race training. Approximately 90% of affected horses are racehorses. Therefore, the disease is most common in Thoroughbred and Standardbred horses. In one study the majority of horses referred for treatment had a preliminary diagnosis of EE. Careful endoscopic examination may be necessary to differentiate epiglottitis from EE and subepiglottic cysts.

Clinical presentation

The primary clinical signs of epiglottitis include exercise intolerance, respiratory noise, and coughing. Some horses also have problems with swallowing and may cough or experience pain during deep palpation of the larynx.

Differential diagnoses

DDSP; epiglottic retroversion; laryngeal hemiplegia; subepiglottic cysts; tracheal disorders.

Diagnosis

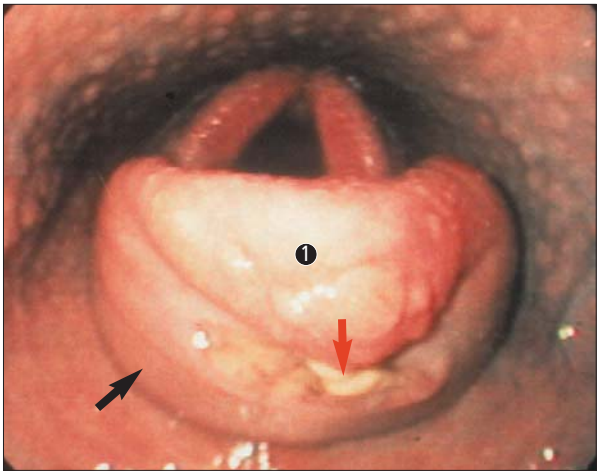
A physical examination should be performed in all horses suspected of epiglottitis. Affected horses experience pain during palpation of the larynx and may cough during manipulation of the larynx and trachea. Rectal temperature may be elevated (>38.3°C [101°F]) in some cases. Endoscopic examination is the preferred method of making a diagnosis of epiglottitis. Epiglottitis is diagnosed if any or all of the following abnormalities are seen endoscopically (**269**–**271**): ulceration and reddening of the epiglottic mucosa; epiglottic thickening; edema and discoloration of the epiglottis and aryepiglottic folds without EE; exposed cartilage at the tip of the epiglottis; granulation tissue involving the lingual aspect of the epiglottis; and dorsal elevation of the epiglottic axis. In addition to endoscopy, lateral view radiographic views of the throat latch should be obtained to evaluate the larynx. Epiglottic thickening and soft-tissue swelling ventral to the epiglottis are the most common abnormal radiographic findings.

Management/treatment

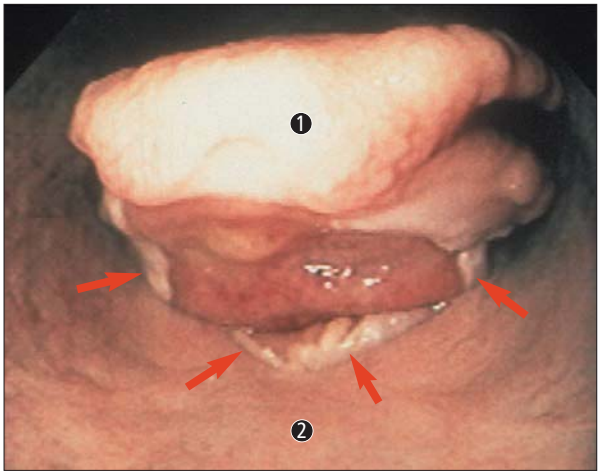
Horses affected with epiglottitis should be treated with a combination of anti-inflammatory and antimicrobial therapy. Anti-inflammatory therapy should be administered systemically and topically. Dexamethasone and phenylbutazone are administered as systemic anti-inflammatory agents. A topical pharyngeal spray (DMSO, glycerin, and prednisolone) should be administered twice daily via a transnasal catheter. Effective antimicrobial agents include penicillin G (22,000 IU/kg IV q6h or IM q12h), gentamicin sulfate (6.6–8.8 mg/kg IV q24h), and trimethoprim–sulfamethoxazole (30 mg/kg PO q12h). Rest is an important part of the management of epiglottitis. Affected horses should be rested until all subepiglottic inflammation has resolved. Surgery is not recommended during the early phases of medical management. However, surgical correction of EE secondary to epiglottitis is sometimes necessary. Long-term sequelae of epiglottitis include epiglottic deformity, intermittent or persistent DDSP, and EE. The prognosis following treatment is guarded, with 50% of racehorses developing performance-limiting complications.

Key points

- Epiglottitis is an inflammatory condition of the epiglottis resulting in exercise intolerance, respiratory noise, and coughing.
- The cause of epiglottitis is unknown, but is most likely of an inflammatory nature.
- Endoscopy is the best way to confirm a diagnosis of epiglottitis.
- The primary treatment method is antimicrobial and anti-inflammatory therapy with surgery only being elected for EE following resolution of epiglottic inflammation.
- The prognosis for epiglottitis is guarded with sequelae including epiglottic deformity, intermittent or persistent DDSP, and EE.

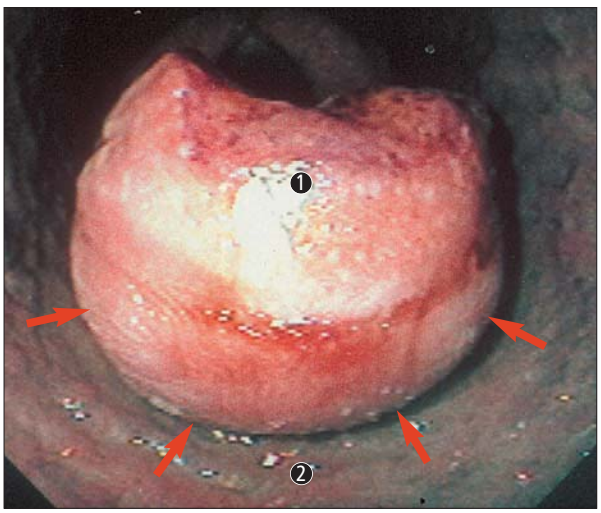


▲ 269 Endoscopic photograph of a Standardbred racehorse with epiglottitis. Note the elevated dorsal epiglottic axis secondary to edema and inflammation associated with the subepiglottic surface (black arrow) of the epiglottis (1). This horse also has an exposed epiglottic tip cartilage (red arrow), a predisposition to epiglottic cartilage deformity.



▲ 270 Endoscopic photograph of a horse with epiglottitis. Note the blunted, deformed tip of the epiglottis (1) and the granulation tissue involving the aryepiglottic fold (arrows). The soft palate (2) is normal.

▶ 271 Endoscopic photograph of a combination of epiglottic entrapment and epiglottitis. Note the thickening of the aryepiglottic fold (1) and ulceration and the severe edema of the subepiglottic tissues of the epiglottis (arrows). (2) soft palate.



Rostral displacement of the palatopharyngeal arch

Definition/overview

Rostral displacement of the palatopharyngeal arch (RDPA) occurs when the dorsal aspect of the arch displaces over the corniculate processes of the arytenoid cartilages.

Etiology/pathophysiology

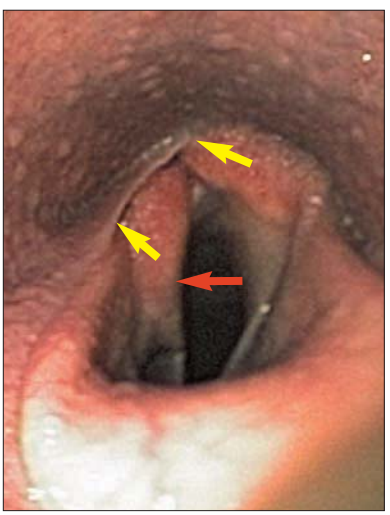
RDPA is not typically an isolated, standalone disease. It may be associated with other developmental abnormalities of the larynx (**272**). The proposed mechanism is a developmental abnormality of the fourth branchial arch (**273**), which causes anatomic changes in the thyroid cartilage that lead to displacement of the arch. The thyroid cartilage is shaped abnormally with shortened lateral and posterior laminae that are tilted dorsally and do not articulate with the cricoid cartilage. The abnormal thyroid cartilage conformation limits normal movement of the arytenoid cartilages. The cricopharyngeus muscles may also be absent. RDPA results in respiratory obstruction by overlying the corniculate processes of the arytenoid cartilages.

Clinical presentation

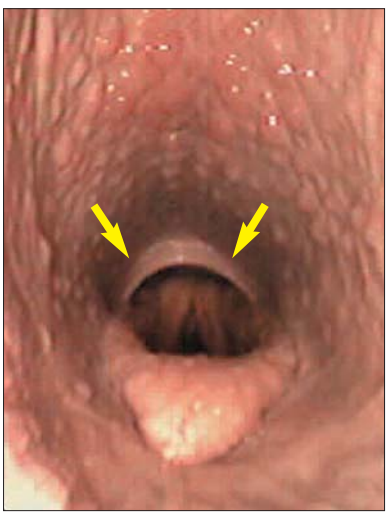
RDPA is usually diagnosed in horses <3 years of age. Congenital malformation of the larynx is usually the underlying cause. Thoroughbreds appear to be the most commonly affected breed. RDPA can present with a variety of clinical abnormalities. These abnormalities include dysphagia, nasal discharge of food, inspiratory noise, and exercise intolerance. The absence of the cricopharyngeus muscle and a loss of normal coordination between the pharynx, larynx, and upper esophageal sphincter during swallowing can result in dysphagia. Inspiratory noise occurs when the palatopharyngeal tissue causes physical obstruction to outflow and the arytenoid cartilages do not abduct normally because of the thyroid cartilage malformation.

Differential diagnosis

EE; epiglottitis; DDSP; epiglottic retroversion; laryngeal hemiplegia; subepiglottic cysts; tracheal disorders.



◀ **272** **A horse with rostral displacement of the palatopharyngeal arch on the right side (yellow arrows) and right laryngeal hemiplegia (red arrow). This horse had a right-sided branchial arch deformity.**



▶ **273** **Rostral displacement of the palatopharyngeal arch (arrows) in a horse with a fourth branchial arch defect.**

Diagnosis

The best diagnostic procedure for RDPA is endoscopy. Endoscopic findings in affected horses include the palatopharyngeal arch located rostral to the corniculate processes of the arytenoid and inadequate abduction of the arytenoid cartilages. Digital palpation of the larynx may reveal an abnormally shaped thyroid cartilage. Concurrent laryngeal abnormalities may be identified during a high-speed treadmill examination. Other diagnostic tests used to evaluate the extent of laryngeal malformations include laryngeal ultrasonography and MRI.

Management/treatment

If an athletic career for the horse is desired, a high-speed treadmill examination would help determine the optimal surgical approach to treatment. High-speed treadmill findings will allow the surgeon to prepare a specific surgical plan and recommend the appropriate surgical treatment to the owner. A poor prognosis for athletic use should be given if RDPA is present in conjunction with laryngeal cartilage abnormalities, such as laryngeal hemiplegia or DDSP. Euthanasia should be considered in horses with dysphagia. If the palatopharyngeal arch is causing airway obstruction alone, surgical resection of the arch is performed. The palatopharyngeal arch can be surgically removed via laryngotomy or it can be removed standing with a diode laser. Diode laser removal can be accomplished using contact or non-contact techniques. The prognosis following surgery is guarded to poor depending on concurrent laryngeal abnormalities.

Key points

- RDPA is a congenital deformity of the larynx.
- A variety of clinical signs is possible, including dysphagia, coughing, inspiratory noise, and exercise intolerance.
- Horses with RDPA should be evaluated on a high-speed treadmill in order to make treatment recommendations to the owner.
- Horses with arch displacement without other laryngeal abnormalities are candidates for surgical removal of the arch.
- The prognosis for RDPA is guarded to poor depending on the extent of other laryngeal abnormalities.

Laryngeal hemiplegia

Definition/overview

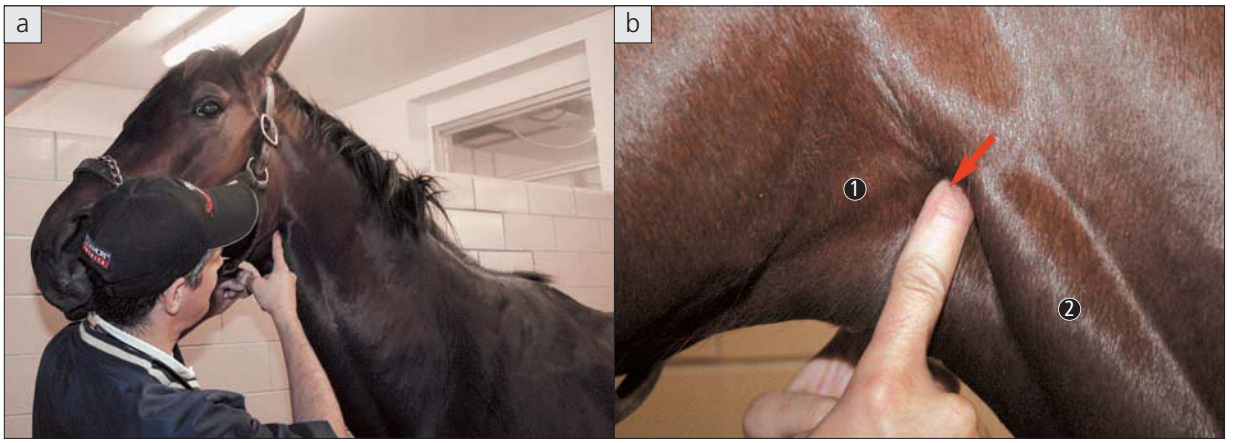
Laryngeal hemiplegia is an idiopathic condition that results in partial or complete paralysis of the arytenoid cartilage and vocal cord. The condition affects 95% of horses on the left side and 5% on the right side.

Etiology/pathophysiology

The primary pathologic finding of idiopathic laryngeal hemiplegia is a distal axonopathy of the left recurrent laryngeal nerve. Histopathologic findings include distal, progressive loss of large myelinated nerve fibers. Demyelination of the recurrent laryngeal nerve results in neurogenic atrophy of the cricoarytenoideus dorsalis (CAD) muscle. This is the major abductor muscle of the larynx. However, adductor muscles are affected, with the cricoarytenoideus lateralis being more severely affected than the CAD. Anatomic positioning of the recurrent laryngeal nerve may be a factor, as the left recurrent laryngeal nerve loops around the aorta in the thorax. The recurrent laryngeal nerve is one of the longest nerves in the body, therefore long-necked horses may place excessive tensile forces on the nerve when the horse moves its head and neck or bends it to the right. Compression from enlarged thoracic lymph nodes has also been suggested as a potential cause of laryngeal hemiplegia. Heredity may also play a role. The mode of inheritance has not been elucidated. Trauma to the recurrent laryngeal nerve secondary to blunt force, or jugular thrombophlebitis combined with cellulitis, can also result in laryngeal hemiplegia. At present, a definitive etiology for the idiopathic form of laryngeal hemiplegia has not been determined.

Clinical presentation

The most commonly affected breeds include Thoroughbred, Standardbred, Warmblood, and Draft. The most commonly affected age group is horses <3 years old. Historical findings compatible with laryngeal hemiplegia include inspiratory respiratory noise and exercise intolerance. Of affected horses, approximately 95% involve the left arytenoid cartilage and 5% the right arytenoid cartilage. However, rare cases of bilateral laryngeal hemiplegia do occur. It has been reported in association with equine protozoal encephalomyelitis (EPM), toxic insult, and following general anesthesia. Other historical findings associated with



▲ 274 **(a) Palpation of the muscular processes of the arytenoid cartilages in the standing horse. (b) Close-up image of manual palpation, indicating the relative position of the linguofacial vein (1) and the tendon of the sterno-mandibularis muscle (2) to the index finger (arrow).**

laryngeal hemiplegia include jugular vein thrombophlebitis, URT or LRT infection, and trauma to the neck. The primary clinical signs of laryngeal hemiplegia include respiratory noise, exercise intolerance, and occasional coughing. Trainers or owners may describe the noise as 'roaring'. Dynamic collapse of the left arytenoid cartilage into the airway during strenuous exercise produces the classical noise. Exercise intolerance results secondary to exercise-induced hypoxemia.

Differential diagnosis

EE; epiglottitis; DDSP; epiglottic retroversion; RDPA; arytenoid chondritis; subepiglottic cysts; tracheal disorders.

Diagnosis

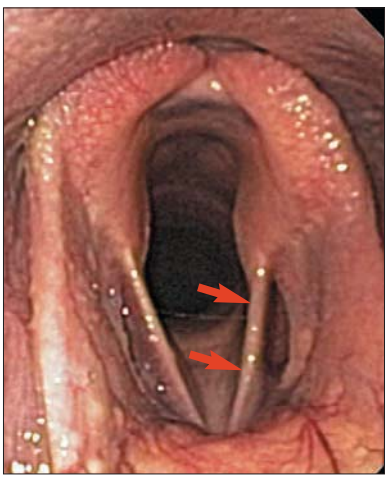
Diagnosis of laryngeal hemiplegia starts with a physical examination, which should include digital palpation of the larynx. Laryngeal palpation begins by facing the horse and resting the rostral mandible on the shoulder of the examiner. The index fingers are curled over the dorsal surface of the larynx. With practice, the left and right muscular processes can be palpated (**274**). It is best to compare the two sides. Affected horses typically have a palpable prominence of the muscular process of the arytenoid cartilage on the affected side. A prominent left muscular process indicates atrophy of the left crico-arytenoideus dorsalis muscle.

The jugular vein should be palpated for evidence of thrombophlebitis and cervical cellulitis. A final physical examination test that can be performed is a slap test. This is a reflex examination. When the right saddle pad area is slapped with the flat of the hand, the left arytenoid should experience adduction. The test is repeated on the contralateral side. Horses with laryngeal hemiplegia have a negative slap test. The slap test can also be performed during standing endoscopic examination.

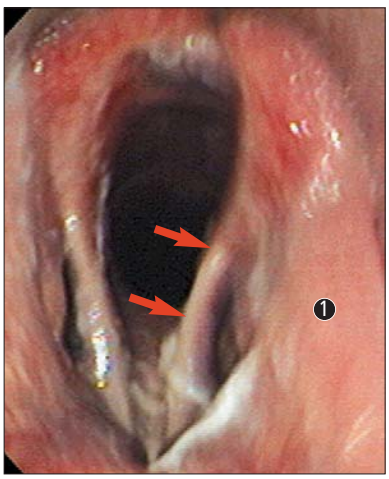
The best diagnostic tool to confirm a diagnosis of laryngeal hemiplegia is endoscopy. An endoscopic grading system has been developed to grade laryngeal function while standing. Laryngeal function has been classified into four endoscopic grades:

- **Grade 1.** Normal arytenoid function; both arytenoids abduct equally and symmetrically.
- **Grade 2.** Both arytenoids achieve full abduction, but the left arytenoid abducts asynchronously with the right arytenoid.
- **Grade 3.** The left arytenoid cannot achieve maximal abduction (e.g. partially paralyzed or hemiparesis). However, some horses with grade 3 laryngeal hemiplegia can achieve normal abduction during high-speed exercise. Therefore, if there is any doubt about laryngeal function, a high-speed treadmill examination should be performed.
- **Grade 4.** Complete paralysis of the left arytenoid; no purposeful movement.

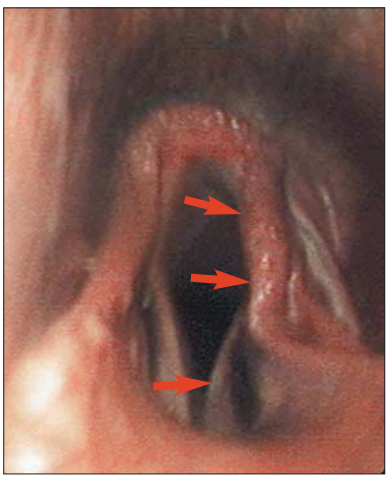
Horses with grade 1 laryngeal hemiplegia are normal and do not require further diagnostics. Horses with grade 2 laryngeal hemiplegia are usually normal, but horses with a history of respiratory noise and exercise intolerance should be examined during high-speed exercise. Horses with grade 3 laryngeal hemiplegia are the most challenging group to evaluate. The major reason for this is that incomplete abduction of the affected arytenoid cartilage varies with the degree of atrophy of the CAD muscle. Therefore, most horses with grade 3 laryngeal hemiplegia should be evaluated at high speed. Horses with grade 3 laryngeal hemiplegia at rest have been subcategorized into A, B, and C grades while exercising on a treadmill (**275–277**). Horses with grade 3A laryngeal hemiplegia are normal during high-speed treadmill examination and do not require treatment. Horses with grade 3B laryngeal hemiplegia are able to maintain abduction of the arytenoid during exercise, although not fully, but experience dynamic collapse of the vocal cord. Horses with grade 3C laryngeal hemiplegia experience dynamic collapse of the arytenoid and vocal cord during exercise and are treated the same as a horse with a laryngeal grade of 4.



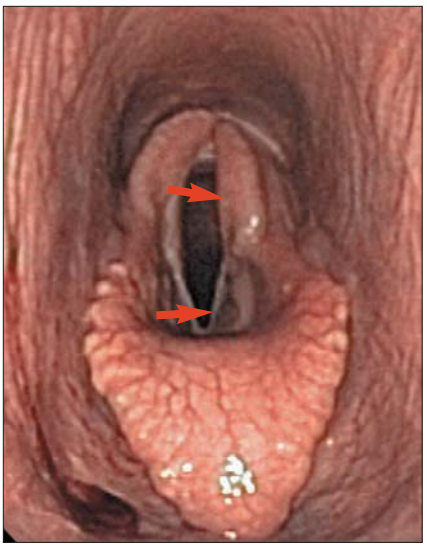
▲ **275** **Standing endoscopic examination of a horse with grade 3 left laryngeal hemiplegia. Note the axial positioning of the left vocal cord and incomplete abduction of the left arytenoid (arrows).**



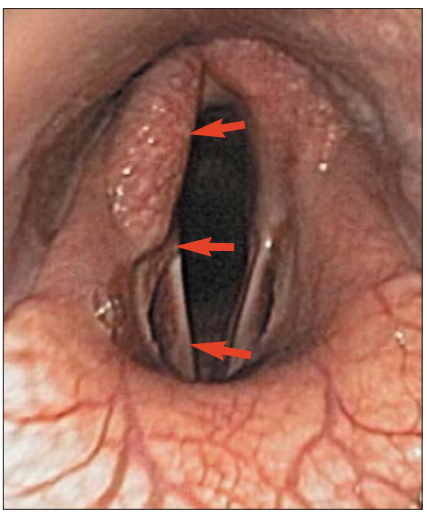
▲ **276** **Dynamic treadmill endoscopy of a horse with grade 3B left laryngeal hemiplegia. Note the axial deviation of the left vocal cord (arrows) and incomplete abduction of the left arytenoid. (1) aryepiglottic fold.**



▲ **277** **Dynamic treadmill endoscopy of a horse with baseline grade 3 left laryngeal hemiplegia at rest. Based on the dynamic collapse of the left arytenoid (arrows), this horse has a grade 3C left laryngeal hemiplegia.**



▲ **278** **Standing endoscopic examination of a horse with grade 4 left laryngeal hemiplegia. Note the axial positioning of the vocal cord and left arytenoid cartilage (arrows). Although the right arytenoid does not appear to be abducted normally in this photograph, this arytenoid was capable of full abduction during the dynamic portion of the endoscopic examination.**



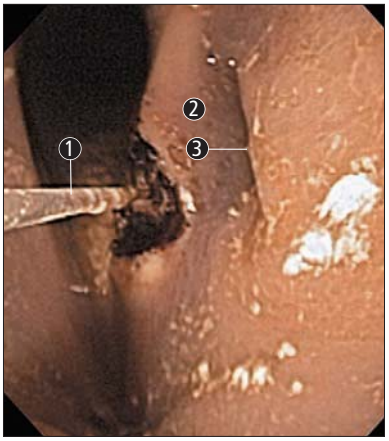
▲ **279** **Standing endoscopic examination of a horse with grade 4 right laryngeal hemiplegia secondary to jugular vein thrombophlebitis. Note the axial positioning of the right vocal cord and arytenoid cartilage (arrows).**

Horses with laryngeal function grade 4 experience dynamic collapse of the arytenoid cartilage and require surgical correction if an athletic career is required (**278**, **279**).

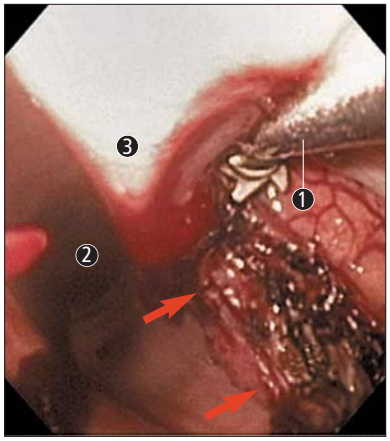
Management/treatment

The form of treatment for laryngeal hemiplegia will depend on the athletic use of the horse. Horses that are used for exercise of low intensity do not require surgical correction. Horses that exercise at a high-intensity level generally require surgical correction. In addition, consideration should be given as to whether or not the amount of respiratory noise is hampering the horse while showing.

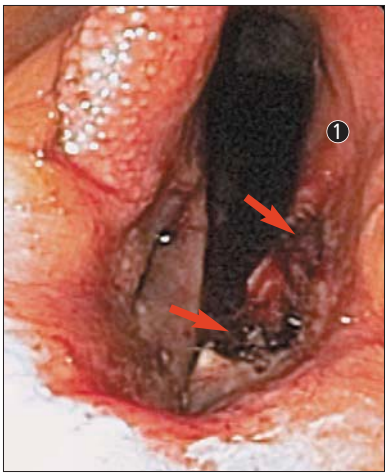
Horses with mild to moderate clinical signs of laryngeal hemiplegia, and/or in which the primary owner complaint is excessive noise, can be treated with standing laser ventriculocordectomy. This is performed with the horse sedated and the larynx anesthetized topically. Under endoscopic guidance, a custom-designed roaring burr is inserted into the left nasal passage (for the left ventricle and vocal cord). The dorsal and ventral aspects of the vocal cord are incised with a diode laser in a contact fashion (**280**, **281**). The burr is then inserted into the laryngeal ventricle and twisted at least 360 degrees. Traction is then placed on the ventricle to evert it into the laryngeal lumen. The laser is then used to excise the ventricle. Typically, as the ventricle is excised the vocal cord is removed with it. If not, the vocal cord can be grasped with bronchoesophageal grasping forceps and excised with the laser (**282**, **283**). An alternative to this technique is to photoablate the vocal cord and laryngeal ventricle with a combination of contact and non-contact techniques. When performed in this fashion, higher amounts of laser energy are required (>30 watts) and this will result in increased collateral injury to adjacent tissues. Laser ventriculocordectomy has been shown to be effective in eliminating the noise associated with laryngeal hemiplegia and mildly to moderately effective in resolving signs of exercise intolerance in affected horses.



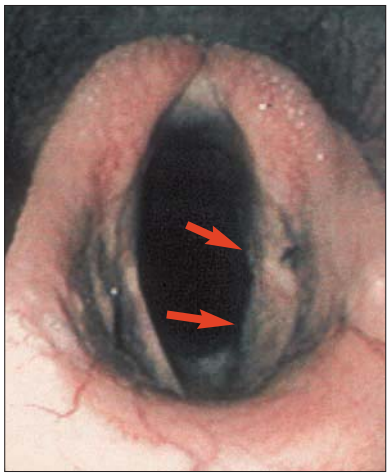
◀ **280** **Intraoperative view of the initial cut of a laser ventriculocordectomy. Note the contact laser fiber (1), the left vocal fold (2), and the laryngeal ventricle (3).**



▶ **281** **Intraoperative photograph of a left laser ventriculocordectomy using the transorally positioned roaring burr (1). Note the location of the rima glottidis (2), the left vocal process (3), and the excised vocal cord (arrows).**

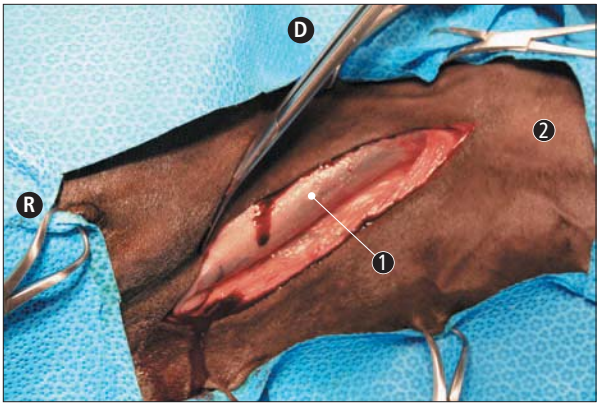


◀ **282** **Immediate postoperative photograph of a laser ventriculocordectomy (arrows). Note the left vocal process (1). It is important not to cause iatrogenic damage to the vocal process during this procedure.**

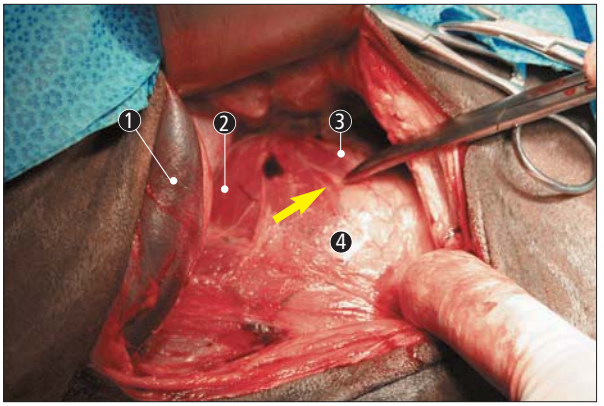


▶ **283** **Endoscopic photograph of a healed left laser ventriculocordectomy site (arrows) in a horse with a repeat laryngoplasty. (Photo courtesy EP Tulleners)**

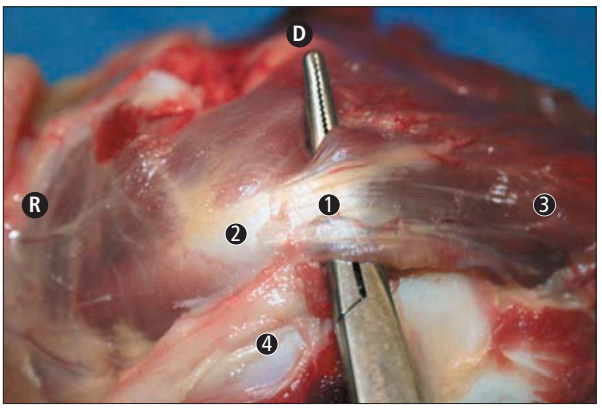
Horses that exercise at a high intensity, such as racehorses and 3-day eventers, are best managed with prosthetic laryngoplasty combined with laser ventriculocordectomy. Prosthetic laryngoplasty involves the placement of one or two non-absorbable sutures to permanently retract the arytenoid cartilage. Retraction of the arytenoid prevents dynamic collapse and alleviates the clinical signs of respiratory noise and exercise intolerance. The surgical technique of prosthetic laryngoplasty has been described previously (see Further reading). Differences in this technique that have not been previously emphasized will be discussed here. The authors always recommend that laser ventriculocordectomy is performed prior to prosthetic laryngoplasty. Following induction of general anesthesia the horse is intubated nasotracheally so that the tube is out of the way of the surgeon. The procedure is performed as before, but instead of being done via the nasal passages, the surgery is done through the oral cavity. Because the laser is discharged adjacent to the nasotracheal tube, inhalation gases and oxygen are disconnected during the procedure and the horse is maintained under anesthesia with injectable anesthetic agents. Following ventriculocordectomy the horse is reconnected to gaseous anesthesia and the prosthetic laryngoplasty is performed.



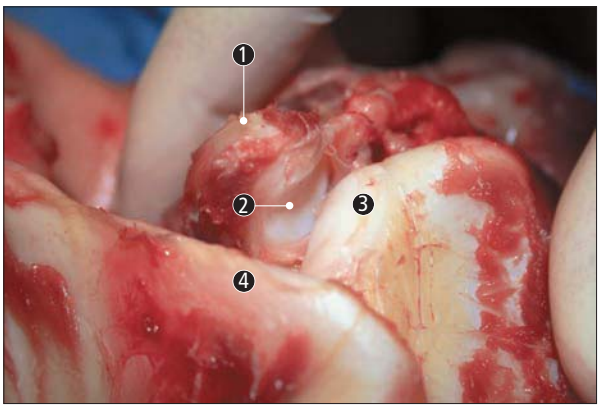
▲ 284 Intraoperative photograph detailing the approach to the larynx for laryngoplasty. Note the incision made just ventral to the linguofacial vein (1) and ending at the level of the tendon of insertion of the sternomandibularis muscle (2). (R) rostral; (D) dorsal.



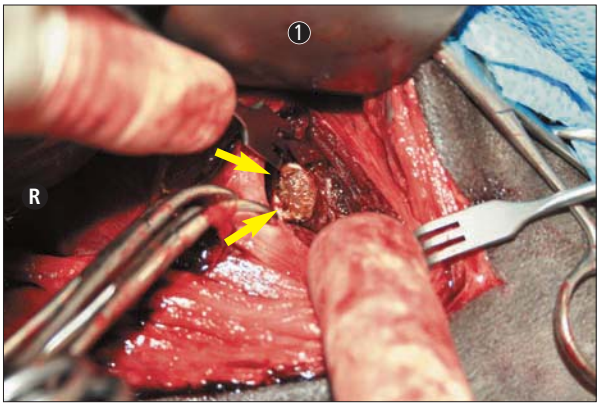
▲ 285 Intraoperative photograph of the lateral aspect of the left hemilarynx following retraction of the linguofacial vein (1) dorsally with a retractor. Note the septum (arrow) between the thyropharyngeus muscle (2) and the cricopharyngeus muscle (3). The muscular process of the arytenoid cartilage lies beneath this septum; the thyroid lamina (4) is at its ventral aspect.



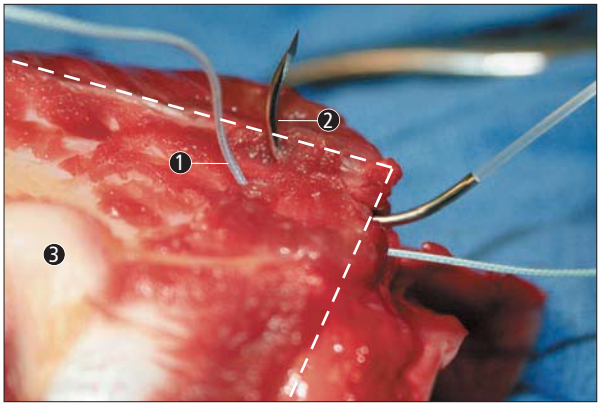
▲ 286 Gross dissection of the larynx demonstrating the relationship between the tendon of insertion of the cricoarytenoideus muscle (1), elevated by hemostat onto the muscular process of the arytenoid cartilage (2), and the muscle belly of the cricoarytenoideus dorsalis muscle (3). (4) thyroid lamina; (R) rostral; (D) dorsal.



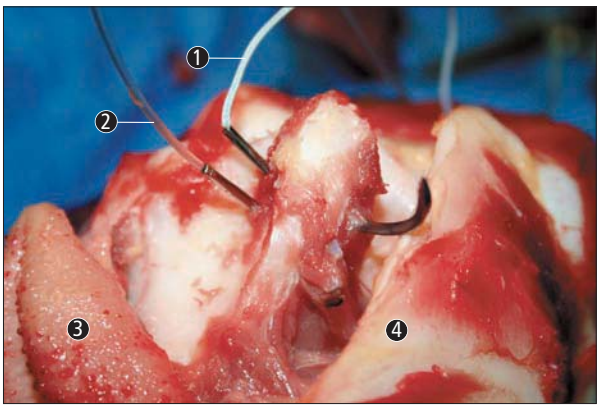
▲ 287 Gross postmortem photograph of the retracted cricoarytenoid joint following transection of the cricoarytenoideus tendon of insertion on the muscle belly and removal of the cricoarytenoideus dorsalis muscle. Note the muscular process (1) of the arytenoid cartilage, the arytenoid facet (2) of the cricoarytenoid joint, the cricoid facet (3) of the cricoid cartilage, and the ventrally located thyroid cartilage (4).



▲ **288** **Intraoperative photograph of a cricoid facet following CO_2 laser ablation of the cartilage (arrows). The retractor (1) is elevating the linguofacial vein and is located dorsal to the larynx. (R) rostral.**

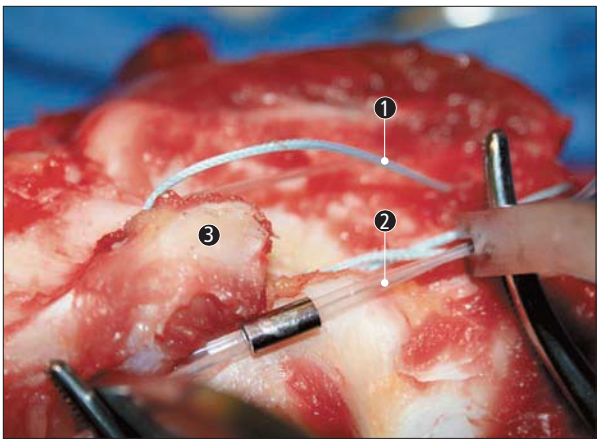


▲ **289** **Gross postmortem photograph detailing a two-suture technique for prosthetic laryngoplasty. The dotted lines represent the dorsal midline and caudal aspect of the cricoid cartilage. Suture 1 (1) is positioned lateral to suture 2 (2), which is positioned as close as possible to the midline of the cricoid cartilage. Note the caudal aspect of the thyroid cartilage (3).**



◀ **290** **Gross postmortem photograph detailing the positioning of the two laryngoplasty sutures in the muscular process of the left arytenoid cartilage. Suture 1 (1) is positioned towards the apex of the muscular process and suture 2 (2) is positioned rostral to suture 1. Note the corniculate processes of the arytenoid cartilages (3) and the thyroid cartilage (4).**

A standard approach is used to access the larynx (**284**). The most recent ancillary procedure performed in conjunction with laryngoplasty is ablation of the cartilaginous surfaces of the cricoarytenoid joint (CAJ). The CAJ is the major articulation between the arytenoid and cricoid cartilages. An arthrosis of this joint is created to minimize the risk of arytenoid abduction following laryngoplasty. The authors use a CO_2 laser to ablate the cartilaginous surfaces. The CAJ is accessed by transecting the cricoarytenoideus dorsalis tendon and incising the lateral aspect of the joint just ventral to the CAD tendon. The joint is retracted open with Senn retractors (**285**–**287**). The CO_2 laser is used in a non-contact fashion to ablate all visible portions of the articular cartilage from the arytenoid and cricoid facets (**288**). Once the cartilage has been laser ablated, a laryngoplasty is performed (**289**, **290**) (see Further reading). The authors recommend that the degree of arytenoid abduction is assessed with intraoperative endoscopy.



◀ **291** **Gross postmortem photograph detailing the tightening of a monofilament nylon suture with a commercially available tension device. Note how suture 1 (1) loosens when suture 2 (2) is tensioned with the tension device. The final positioning of the left arytenoid abduction is performed with the aid of intraoperative endoscopy. (3) muscular process.**

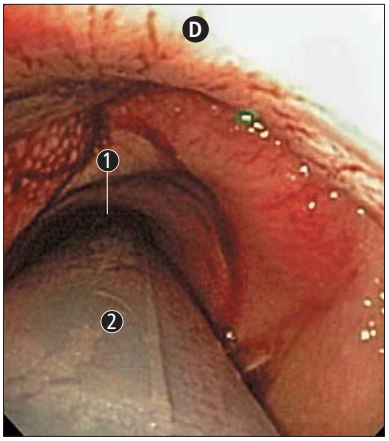
This is important because it ensures that the arytenoid cartilage can be abducted and it allows the surgeon to determine how much abduction should be obtained with tightening of the laryngoplasty sutures (**291**). Ideally, the arytenoid cartilage should be positioned between moderate and full abduction (**292**, **293**). This should allow for an adequate airway diameter postoperatively and minimize the possibility of coughing following surgery. Incisional closure is performed in a routine manner.

Postoperative complications following prosthetic laryngoplasty include failure of the laryngoplasty, coughing, dysphagia, incisional infection, and aspiration pneumonia. Coughing is the most common complication, occurring in 25% of cases. Dysphagia occurs less frequently with 3–5% of horses being affected. Most horses with postoperative dysphagia will require removal of the prosthetic suture. Typically, clinical signs of dysphagia will resolve with suture removal. Incisional infection is managed with drainage and antimicrobial therapy (**294**). Most horses do not require removal of the prosthesis. Horses in which the laryngoplasty has failed have two options if an athletic career is desired. The first is to repeat the laryngoplasty. Repeat laryngoplasty is successful in 60–65% of cases. If a repeat laryngoplasty is not possible, the remaining option is a partial arytenoidectomy.

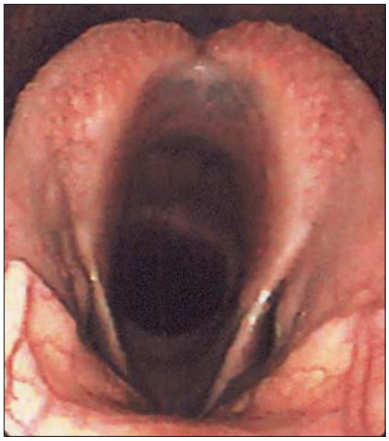
The prognosis following surgical treatment of laryngeal hemiplegia depends on the intended use of the horse. Surgery improves performance in 55% of racehorses and approximately 70–80% of horses used for athletic pursuits other than racing.

Key points

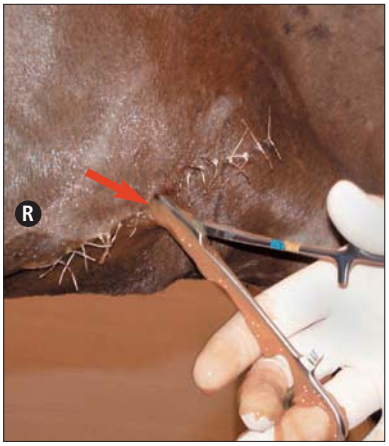
- Laryngeal hemiplegia is one of the most common diseases causing exercise intolerance and URT noise.
- Diagnosis of laryngeal hemiplegia is confirmed with an endoscopic examination and, in some cases, high-speed treadmill examination to determine the most appropriate method of treatment.
- Horses with a primary complaint of upper respiratory noise and no exercise intolerance may be candidates for laser ventriculocordectomy only.
- Horses with a primary complaint of upper respiratory noise and exercise intolerance are best treated with a combination of laser ventriculocordectomy and prosthetic laryngoplasty.
- Laser anklyosis/arthrosis of the cricoarytenoid joint improves postoperative stability of the arytenoid cartilage and decreases the likelihood of loss of abduction postoperatively.
- Surgeon experience and compentency is paramount to a successful outcome.
- The prognosis following surgical correction is good, but complications associated with the surgical procedure do occur and may require specific treatment to ensure a favorable outcome.



▲ 292 **Intraoperative endoscopy following ideal positioning of the left arytenoid. Note the widening of the rima glottis (1) following successful abduction of the left arytenoid and the location of the endotracheal tube (2). (D) dorsal.**



▲ 293 **Endoscopic photograph of a horse following left laryngoplasty. Note the excellent abduction of the left arytenoid cartilage.**



▲ 294 **Incisional infection and purulent drainage (arrow) post laryngoplasty. (R) rostral.**

Arytenoid chondritis

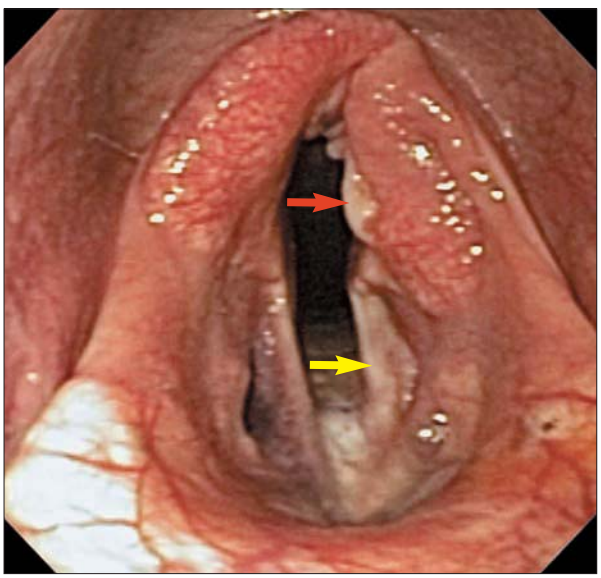
Definition/overview

Arytenoid chondritis is an inflammatory condition of the arytenoid cartilages. The disease typically involves the corniculate processes of the cartilages. Bacteria invade the mucosa and cartilage of the arytenoid secondary to disruption of the mucosal surfaces of the corniculate process(es). This results in edema and abscessation of the cartilage. Enlargement of the affected arytenoid results in respiratory noise and exercise intolerance.

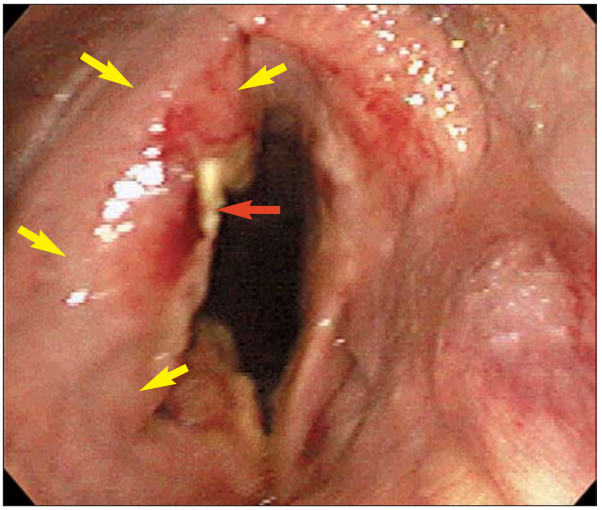
Etiology/pathophysiology

Arytenoid chondritis develops in two distinct clinical syndromes:

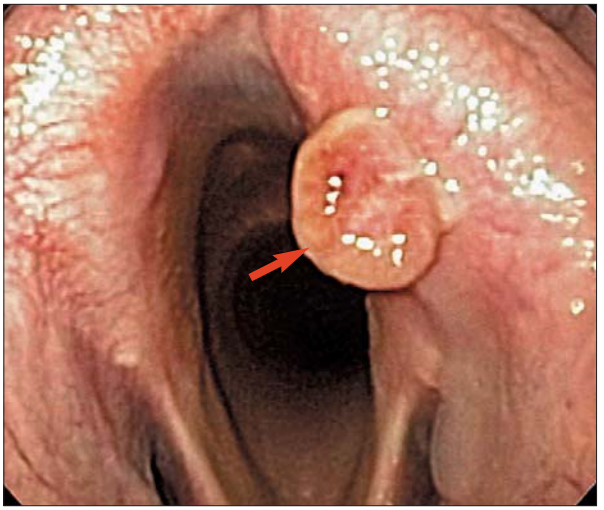
- The first syndrome occurs in racehorses, who most likely develop the condition because of the abrasive and concussive forces that develop between the two arytenoids during high-speed exercise. In most cases the areas of infection are on the axial aspects of the corniculate cartilages (**295**).



▲ 295 **Left-sided arytenoid chondritis in a Standardbred racehorse. Note the thickening of the left arytenoid cartilage, an axial lesion on the corniculate process (red arrow), and edema and thickening of the vocal cord (yellow arrow).**



▲ **296** **Quarter Horse broodmare with right-sided arytenoid chondritis. Note the generalized edema and enlargement of the right corniculate process (yellow arrows) and an axial mass with purulent drainage (red arrow). This horse is a candidate for partial arytenoidectomy because of the diffuse thickening of the right arytenoid cartilage and the purulent axial drainage.**



▲ **297** **Axial granulation tissue mass (arrow) in a Standardbred racehorse with mild arytenoid chondritis.**

- The second syndrome occurs in horses who exercise at low athletic intensity. The cause of arytenoid chondritis in these horses is not known, but trauma from coarse feed or procedures such as nasogastric intubation is suspected. Once the arytenoid cartilage is infected the chondritis can be diffuse throughout the cartilage or involve small granulation tissue masses on the axial aspect of the corniculate cartilage (**296**). Infection can also invade the adjacent musculature and mucous membrane of the larynx.

The overall effect of arytenoid chondritis is a reduction in the cross-sectional area of the rima glottidis as a result of the space-occupying effect of the enlarged cartilage and decreased abduction. The loss in the range of motion is attributed to three factors: thickening of the arytenoid cartilage, inflammation of the musculature surrounding the arytenoid, and involvement of the cricoarytenoid articulation.

Clinical presentation

Arytenoid chondritis occurs in two age groups: young racehorses (<3 years old) and older broodmares (>10 years old). The most common breed of racehorse affected is the Thoroughbred, with the Standardbred being less commonly affected. Broodmares of all breeds can be affected with arytenoid chondritis, but the condition is more common in Quarter Horses and Thoroughbreds. Affected horses have a history of exercise intolerance, respiratory noise, or respiratory distress. Horses that are not as closely monitored as broodmares may not present until signs of respiratory distress are evident. Clinical signs of arytenoid chondritis include exercise intolerance, respiratory noise, respiratory distress, pyrexia, coughing, and pain on palpation of the larynx.

Differential diagnoses

EE; epiglottitis; DDSP; epiglottic retroversion; RDPA; laryngeal hemiplegia; subepiglottic cysts.

Diagnosis

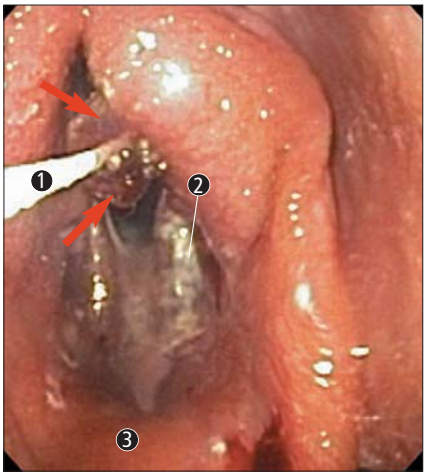
Endoscopy is required to confirm a suspicion of arytenoid chondritis. Endoscopic findings compatible with arytenoid chondritis include thickening of the right or left arytenoid, granulation tissue masses (**297**), ulceration on the axial aspect of the other arytenoid, and laryngeal hemiplegia. (*Note:* Care must be taken when making a diagnosis of laryngeal hemiplegia because closer examination may reveal mild thickening or ulceration of the affected arytenoid.)

Other abnormal findings that may be identified in horses with arytenoid chondritis include epiglottic entrapment, DDSP, and epiglottic hypoplasia. Lateral view laryngeal radiographs may reveal mineralization, distortion of the arytenoids, and obliteration of the laryngeal ventricle.

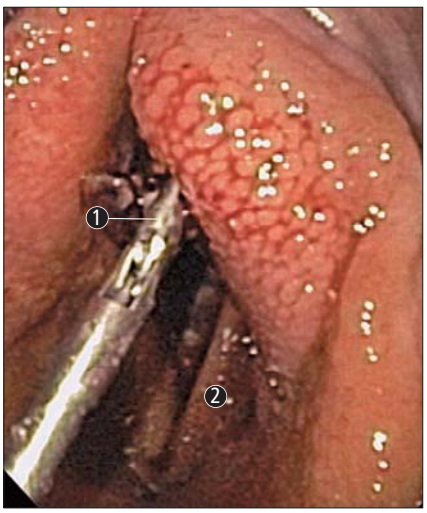
Management/treatment

Treatment for arytenoid chondritis can be medical or surgical depending on the degree of severity. Mild cases of arytenoid chondritis can be treated with antimicrobials, anti-inflammatory drugs, including corticosteroids and topical anti-inflammatory products, and enforced rest. Small granulation tissue masses on the axial aspect of the arytenoid can be removed with a diode or Nd:YAG laser in a contact or non-contact fashion without removal of the arytenoid cartilage (**298**, **299**). In some cases this allows for excellent results. However, this technique works best in horses in which the affected arytenoid cartilage is capable of abduction.

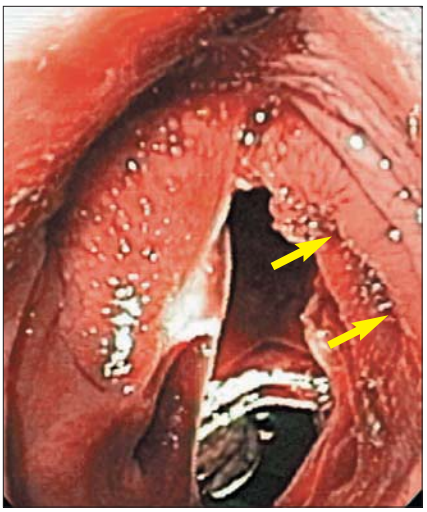
Horses with a granulation tissue mass combined with thickening of the body of the arytenoid and no purposeful movement of the arytenoid cartilage are candidates for arytenoidectomy (**296**). Two techniques are used for arytenoidectomy: subtotal and partial.



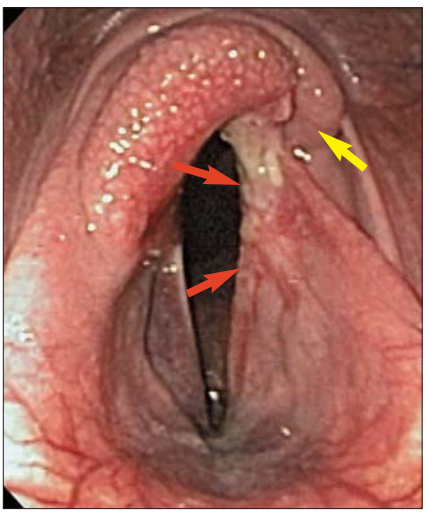
▲ **298** **Intraoperative endoscopy to remove an axial granulation tissue mass (arrows) with a contact diode laser fiber (1). (2) left vocal cord; (3) epiglottis.**



▲ **299** **Bronchoesophageal grasping forceps (1) are being used in the horse in 298 to remove the granulation tissue mass following diode laser incision. (2) left vocal cord.**



▲ **300** **Intraoperative endoscopic photograph of a left partial arytenoidectomy. The endoscope has been positioned in the oral cavity and directed towards the laryngotomy incision and the arytenoidectomy site. The arrows indicate the remaining rim of the corniculate mucosa. The arytenoidectomy was performed without mucosal closure.**



▲ **301** **The horse in 300, 30 days after partial arytenoidectomy. Note the remaining portion of left corniculate mucosa (yellow arrow) and the remaining area of granulation tissue and epithelization at the arytenoidectomy site (red arrows).**

Subtotal arytenoidectomy involves resection of the main body of the arytenoid, but leaving a portion of the corniculate process and the muscular process. A partial arytenoidectomy involves resection of the corniculate process and the body of the arytenoid, leaving behind a small portion of the muscular process. Partial arytenoidectomy is preferred for athletic animals because it leaves behind less tissue, which could lead to airway obstruction once the horse returns to athletic use. The technique has been detailed elsewhere. The most debate amongst surgeons concerns whether or not to perform arytenoidectomy with or without mucosal closure. The authors favor arytenoidectomy without mucosal closure. The advantage of this technique is that no loose tissue is left behind and it does not distort the remaining corniculate mucosa, thus minimizing the chances of dysphagia following arytenoid removal. If mucosal closure is performed, dehiscence is not uncommon. Dehiscence leads to granulation tissue formation and loose tissue, which can later cause laryngeal obstruction to airflow. Resection of the mucosa without closure eliminates this. Not tensing the corniculate mucosa, as occurs with mucosal closure, can distort the piriform recess and lead to dysphagia. Again, this is minimized when the mucosa is not sutured. A 3 mm rim of mucosa should remain to minimize the risk of postoperative aspiration and dysphagia (**300**, **301**).

Complications following partial arytenoidectomy include aspiration pneumonia, dysphagia, coughing, nasal discharge of food and water, and RDPA. The prognosis for racing after arytenoidectomy is 50–60%, with aspiration pneumonia and dysphagia being the main detriments to racing success. Small focal lesions removed with a diode or Nd:YAG laser have a good prognosis. The prognosis for broodmares or sedentary horses treated with partial arytenoidectomy is good. The biggest advantage of treating these types of horses is that the surgeon does not have to be as aggressive in the amount of corniculate mucosa removed compared with the same procedure in racehorses. Therefore, the risk of postoperative coughing and dysphagia is less.

Key points

- Arytenoid chondritis is a not uncommon cause of exercise intolerance in racehorses and occasionally occurs in broodmares.
- Endoscopy is the best method of determining the diagnosis and developing a treatment plan.
- Axial granulation tissue masses without evidence of laryngeal hemiplegia can be treated with standing laser removal of the masses.
- Horses with diffuse thickening and edema of the arytenoid cartilage in combination with laryngeal hemiplegia are best managed with partial arytenoidectomy.
- The prognosis for arytenoid chondritis with surgical treatment is favorable, although owners should be warned of the risk of postoperative aspiration of food following partial arytenoidectomy.

Tracheal diseases

Collapsing trachea

Definition/overview

Tracheal collapse can develop secondary to a congenital abnormality in cartilage development, degeneration of cartilage, or inflammatory diseases of the LRT.

Etiology/pathophysiology

Tracheal stenosis in horses can be divided into two categories depending on whether the defect is intrinsic to the cartilaginous supporting structure (primary) or due to external structures impinging on the trachea (secondary). Primary tracheal stenosis results from trauma to the tracheal rings by external foreign body penetration, iatrogenic trauma to the tracheal rings secondary to tracheotomy, and damage to the tracheal mucosa and cartilage following endotracheal intubation. Causes of secondary tracheal stenosis include abscessation of regional lymph nodes, peritracheal hematomas, and neoplasia.

Clinical presentation

Regardless of the cause of the tracheal stenosis or collapse, the severity of clinical signs will depend on the severity and location of the narrowing. The presence of external wounds or palpation of the trachea may provide preliminary evidence of abnormality. Wheezing or respiratory stridor may be heard on auscultation of the trachea during inspiration and expiration in the area of the lesion. In mild cases clinical signs consist of upper respiratory noise without exercise intolerance. With mild stenosis the horse may exhibit signs only while at exercise, with exercise intolerance, increased inspiratory and expiratory noise, and increased respiratory rate being the most common ones.

In horses with marked tracheal narrowing, nostril flaring may be evident as minute ventilation is increased. Affected horses have increased inspiratory and expiratory stridor generated by turbulent air flow through the stenotic area and tachypnea, respiratory distress, and cyanosis may be present even at rest. Although coughing occurs commonly in dogs with tracheal collapse, this is not a common feature in horses. Additional clinical signs associated with tracheal stenosis or collapse include pyrexia secondary to peritracheal abscesses or bronchopneumonia and laryngeal hemiplegia.

Differential diagnosis

EE; epiglottitis; DDSP; epiglottic retroversion; RDPA; laryngeal hemiplegia; arytenoid chondritis; subepiglottic cysts.

Diagnosis

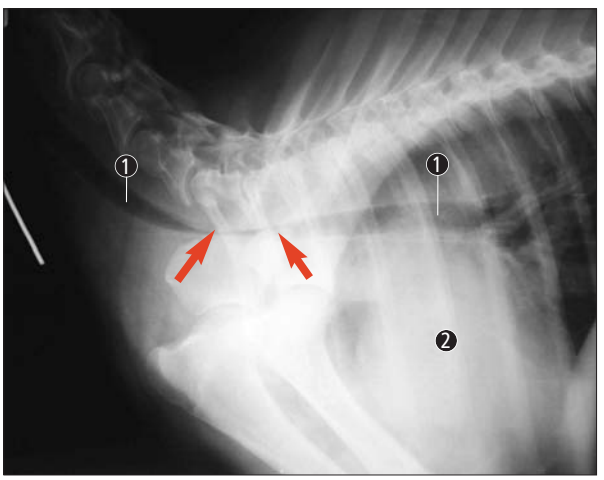
Diagnosis of tracheal stenosis and collapse is based on history, signalment, clinical signs, and endoscopic and radiographic findings. A complete history is fundamental to the detection of tracheal stenosis in the horse. Horses from farms with endemic strangles may develop secondary tracheal stenosis from lymphadenopathy or lymph node abscessation. Foals or adult horses with a history of tracheotomy or external cervical trauma may develop tracheal stenosis secondary to tracheal ring and mucosa trauma. Ponies and miniature horses are included in the most common breeds associated with tracheal collapse syndrome (**302**). Primary and secondary tracheal stenosis can occur in any breed, age, or sex of horse.

The physical examination of horses suspected of tracheal disease should include recording of body temperature and heart and respiratory rates, palpation of the trachea, and auscultation of the trachea and thorax. Increases in body temperature could be related to peritracheal abscesses or bronchopneumonia. Heart and respiratory rates may be increased due to respiratory distress. Deformities, abscesses, or hematomas in the cervical trachea may be palpated. Manual compression of the trachea can usually increase respiratory noise and effort. Auscultation of the trachea and lungs may reveal increased breath sounds or crackles and wheezes associated with underlying lung disease.

Additional diagnostic tests that should be performed include diagnostic imaging and endoscopic examination. Radiographs are beneficial in localizing areas of stenosis or collapse in the cervical and thoracic trachea (**303**).

Although not commonly performed in horses, fluoroscopic evaluation of the trachea during respiration may be helpful in confirming a diagnosis of tracheal collapse. Ultrasonographic examination of the cervical region can be of assistance in differentiating abscesses and hematomas from neoplastic masses. Endoscopic examination of the trachea is invaluable in identifying the location of stenotic lesions and eliminating the URT as a source of noise, exercise intolerance, and respiratory distress. This modality provides visualization of anatomic abnormalities associated with the stenotic lesion, such as tracheal ring and mucosal abnormalities (**304**). When tracheal rings have been damaged the affected portion of the trachea can have an abnormal ('keyhole') shape. Hyperemia and thickening of the tracheal mucosa and increased mucus are often visualized in horses with lower airway disease.

▲ **302** **Miniature horse stallion affected by tracheal collapse.**



▲ **303** **Lateral view thoracic radiograph of the horse shown in 302. Note the collapsed portion (arrows) of the trachea (1) and the position of the tracheal collapse in relation to the heart (2).**

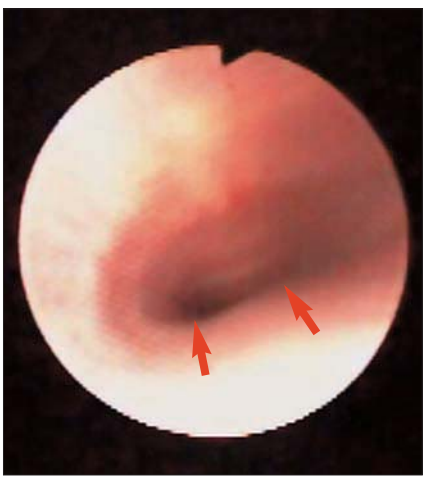
Management/treatment

The primary goal of treating horses with tracheal stenosis and collapse is to restore the normal oval to round lumen of the trachea and normalize airflow to the lungs. The method of treatment depends on the cause of the stenosis and the length and location of the tracheal segment involved. Treating the underlying condition is necessary in all instances. Extratracheal abscesses or hematomas can be drained percutaneously with or without ultrasonographic guidance. A sample of purulent debris should be submitted for microbial culture and antimicrobial sensitivity. Broad-spectrum antimicrobials should be administered until the results of the microbial culture are available. In most instances the abscess rapidly resolves with adequate drainage and appropriate antimicrobial therapy. Enlarged lymph nodes without abscessation may resolve with antimicrobial therapy, NSAIDs, topical anti-inflammatory drugs such as dimethylsulfoxide (DHSO), and hot compresses. In horses with severe respiratory distress secondary to proximal tracheal obstruction a temporary tracheotomy may be needed until the peritracheal abscess or hematoma resolves. Bronchodilators may be of benefit in horses with lower airway disease and in ponies and miniature horses with tracheal collapse if the primary disease is of pulmonary origin. Environmental management should be included in horses with peripheral airway disease.

In general, the prognosis for extraluminal tracheal collapse is favorable if the primary lesion can be treated successfully. Resolution of the inflammatory process surrounding the trachea will allow the lumen size to gradually return to normal. If the tracheal lumen size does not return to normal, surgical reconstruction of the trachea may be required, especially if athletic use is needed. Horses with tracheal collapse secondary to pneumonia can have a successful outcome with appropriate medical therapy.

Surgical intervention is necessary to restore normal trachea contour and function when structural abnormalities of the trachea or neoplasia result in tracheal stenosis or collapse. Focal lesions of the cervical trachea that involve fewer than five tracheal rings can be surgically resected. Before proceeding with tracheal resection and anastomosis, the horse should be acclimated to wearing a martingale apparatus. A martingale is necessary to maintain the horse's head position in flexion and to lessen tension forces on the tracheal anastomosis after surgery.

The technique of tracheal resection and anastomosis has been described (see Further reading). Recovery from anesthesia may be complicated with the martingale and manual assistance is needed to help the horse stand. The martingale is left in place for 30 days after surgery. Complications that can develop following tracheal resection and anastomosis include wound infection, disruption of the anastomosis, especially if a martingale is not used, suture-tract granulomas, and decreased tracheal lumen size secondary to excessive fibrosis. The prognosis for focal lesions involving fewer than five tracheal rings and treated with resection and anastomosis is good.



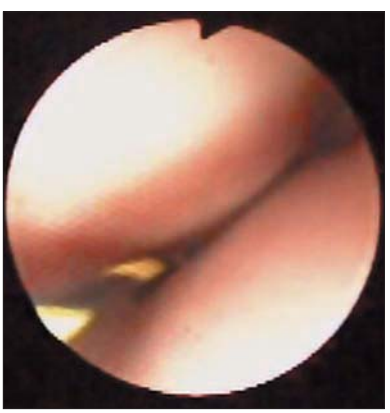
▲ **304 Endoscopic photograph of the horse shown in 302 and 303. Note the collapsed area of the trachea (arrows).**

In horses with tracheal collapse or lesions involving more than five tracheal rings, extraluminal support using polypropylene prosthetic rings is the technique of choice. The most common source for the prosthesis is a 60 ml syringe case. The case is cut longitudinally and small holes are placed along its entire length, using a 14 gauge needle, every 4 mm in rows approximately 5 mm apart. A longitudinal segment 5 mm wide can also be removed to allow for expansion. The length of the prosthesis ranges from 5–10 cm for a single section and from 2–5 cm if multiple rings are used. Multiple smaller prosthetic rings may allow for more normal tracheal flexibility and movement. Reported complications of tracheal prostheses in horses include wound infection, soft-tissue damage adjacent to the prosthesis, and nerve trauma. In calves, tracheal prosthetic devices resulted in tracheal stenosis secondary to continued tracheal growth within the confines of the prosthetic ring(s). The end result was an infolding of the tracheal rings. Therefore, in calves it is recommended that the rings are removed 2–3 months after surgery. In general, the prognosis for tracheal reconstruction with prosthetic rings is fair, depending on the length of trachea involved and the underlying disease process. Horses with collapse involving the majority of the cervical or thoracic tracheal rings are not good candidates for extraluminal support and the prognosis is poor.

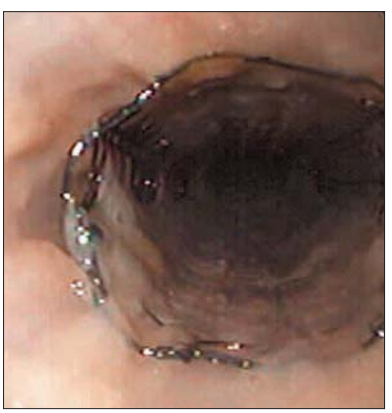
Horses with proximal tracheal obstructions that are unsuitable for tracheal resection and anastomosis or extraluminal support can be treated with permanent tracheostomy; this can be performed either standing or under general anesthesia. Both techniques have been described (see Further reading). The prognosis for horses treated with permanent tracheostomy is good and it should be considered when the other surgical techniques previously described cannot be performed.

Focal lesions within the tracheal lumen that have resulted in stenosis can be removed with a diode or Nd:YAG laser in contact or non-contact fashion. The laser fiber is placed in the biopsy channel of the endoscope. Under direct observation the lesion is identified and excised or ablated with direct thermal injury. Masses within 60 cm of the nares can be removed with a 600-mm-long bronchoesophageal grasping forceps after excision. For masses beyond 60 cm a tracheotomy can be performed proximal to the lesion to allow for tissue retrieval.

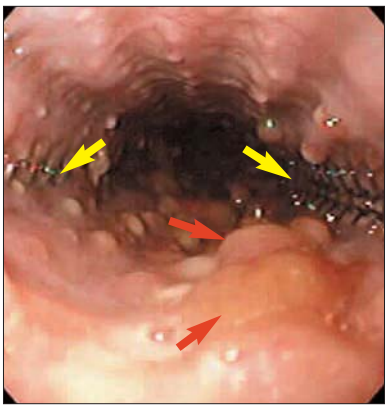
The final method of treatment for tracheal collapse in miniature horses and ponies is implantation of an intraluminal tracheal stent (**305**, **306**). Two main types of stents are available: tubular stents made of silicone or expandable metallic stents such as nitinol stents. Metallic stents can be covered or uncovered. Silicone stents are prone to migration and also disturb mucociliary clearance, leading to accumulation of secretions within the airway lumen. Metallic stents are easier to postion, with better clearance of secretions. Their major disadvantages include growth of granulation tissue through the stent wall in areas of the trachea that were in contact with non-covered regions of a stent or at the ends of covered stents. In addition, tracheal collapse is usually extensive (67% of collapsed tracheas extend from the cervical region to the carina), thus requiring placement of multiple stents in series.



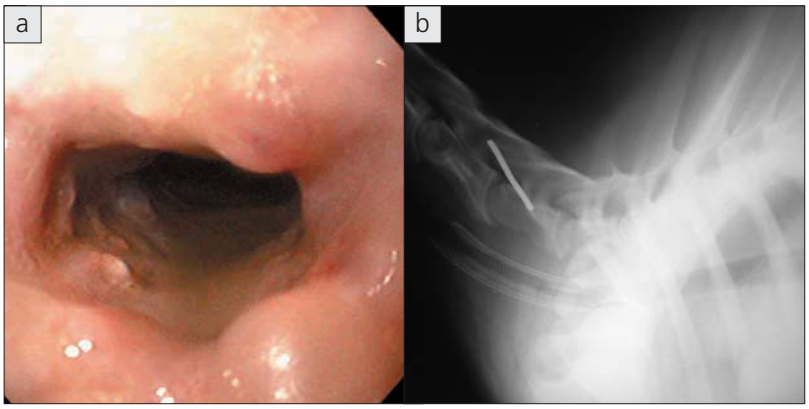
◀ **305** **A guide wire (black and yellow striped) is advanced under endoscopic guidance through the stenotic trachea prior to deployment of the endotracheal stent. Note the extreme narrowing of the tracheal lumen.**



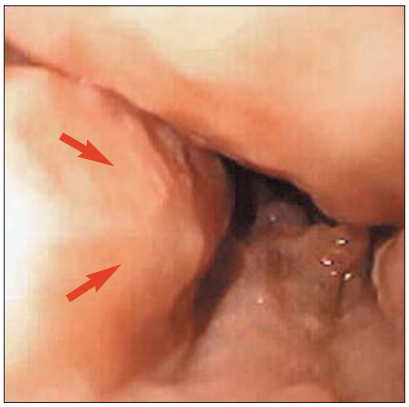
▶ **306** **Non-covered nitinol tracheal stent 6 weeks after placement in a miniature horse with a collapsed trachea.**



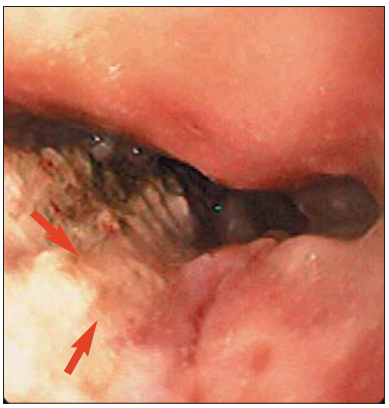
▲ **307** **Endoscopic photograph of the horse in 306, 6 months post stenting. Note the position of the stent (yellow arrows) and the granulation tissue forming in the gaps in the stent (red arrows).**



▲ **308** **Endoscopic photograph (a) and a lateral thoracic radiograph (b) of the horse in 306, 11 months post stenting.**



▶ **309** **Endoscopic photograph of the horse in 306, 2 years post stenting. Note granulation tissue (arrows).**



◀ **310** **Endoscopic photograph of the horse in 306 the day following electrocautery of the granulation tissue shown in 309 (arrows).**

Selection of stent size should be based on estimations of normal tracheal diameter and length of the collapsed area recorded from radiographs. The stent should extend 10–15 mm proximal and distal to the site of pathology in cases of localized stenosis and should be at least 2 mm greater than the lumen diameter at the proximal end of the normal airway.

Nitinol tracheal stents can be deployed under endoscopic guidance (**306**–**309**). The major disadvantage of nitinol stents is granulation tissue formation around the stent. Excessive granulation tissue formation leads to tracheal obstruction and, eventually, difficulty breathing. In one case treated with a tracheal stent, chronic problems with granulation tissue formation occurred over a 3-year period and eventually resulted in euthanasia. Various methods may be used to reduce granulation tissue formation, including inhaled corticosteroids (e.g. fluticasone proprionate), intralesional injection of corticosteroids (e.g. triamcinolone), topical application of mitomycin, and resection of granulation tissue via laser or electrocautery (**310**).

Key points

- Tracheal collapse occurs most frequently in miniature horses and ponies, athough extraluminal tracheal collapse secondary to lymph node abscess and external trauma can occur in horses.
- Diagnosis of tracheal collapse can be confirmed with a combination of endoscopy and cervical/thoracic radiography.
- Focal areas of tracheal collapse involving fewer than five tracheal rings can be managed with tracheal resection and anastomosis.
- Tracheal collapse involving more than five tracheal rings in foals or yearlings may be surgically managed with extraluminal tracheal stenting.
- Miniature horses with tracheal collapse can be treated with intraluminal stents, but granulation tissue formation around the stent may prevent a successful long-term outcome.
- The prognosis for tracheal collapse is better for secondary causes than for primary causes of tracheal stenosis and is also dependent on the success of attempted treatment.

Tracheal trauma

Definition/overview

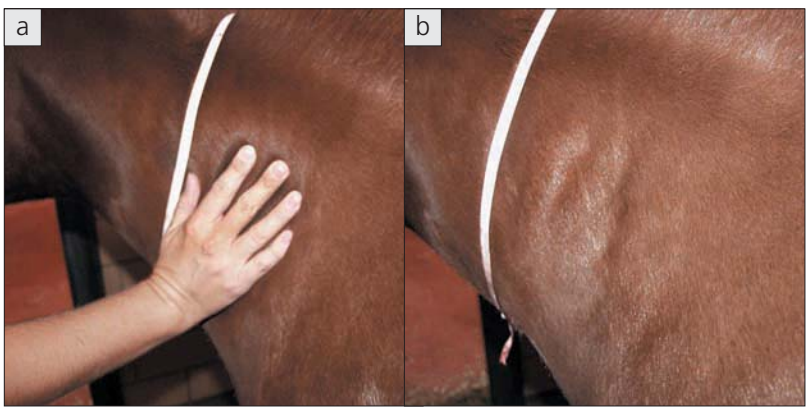
Tracheal trauma occurs because of blunt or sharp injury to the cervical portion of the trachea.

Etiology/pathophysiology

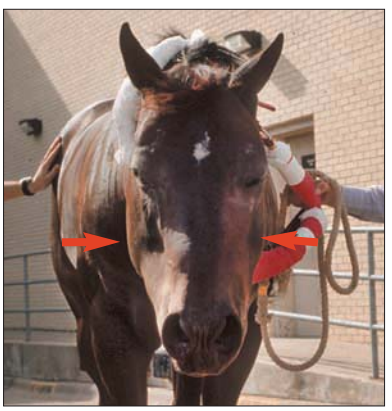
Horses are prone to self-inflicted trauma. External trauma to the cervical portion of the trachea can be blunt or sharp. Sharp trauma to the cervical region injures the trachea from the outside in and is easy to determine because of the air movement in and out of the wound. Blunt-force trauma to the trachea is difficult to detect because the trachea can be ruptured without a skin wound. Affected horses with blunt-force trauma present with clinical signs of subcutaneous emphysema.

Clinical presentation

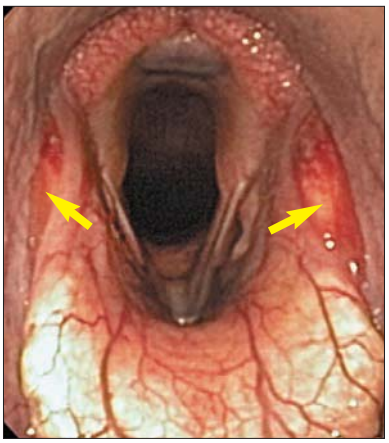
The most common clinical feature of tracheal laceration is subcutaneous emphysema surrounding the wound and trunk and an obvious movement of air in and out of the wound synchronized with respiration. Horses with tracheal rupture without an external wound present with subcutaneous emphysema only. Subcutaneous emphysema can be found along the neck (**311**), skull (**312**), and trunk.



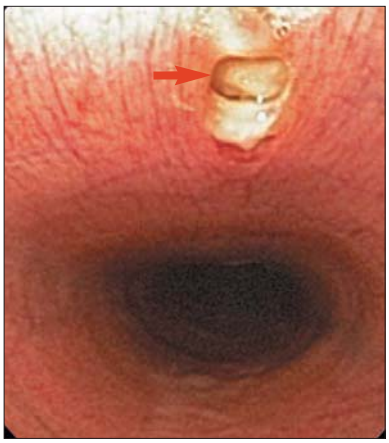
▲ 311 Foal with tracheal laceration from blunt trauma. (a) Note how the hand sinks into the skin, which has become distended by subcutaneous emphysema. (b) An imprint can be seen on the side of the neck after the hand was removed.



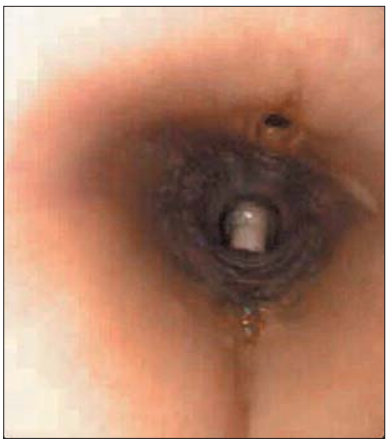
▲ 312 Horse with subcutaneous emphysema of the skull (arrows).



▲ **313** **Endoscopic photograph of a horse with tracheal rupture in the absence of an external wound. Note the edema of the pillars of the soft palate (arrows) under the aryepiglottic folds.**



▲ **314** **Endoscopic photograph of the horse in 313. Note the rupture site involving the dorsal trachea (arrow).**



▲ **315** **Endoscopy of the trachea of the foal with tracheal rupture secondary to blunt trauma seen in 311. A tracheotomy tube can be seen protruding from the trachea on the opposite side to the tracheal laceration.**

Differential diagnosis

External trauma to the cervical region not associated with the trachea, with esophageal rupture being most common; axillary lacerations causing subcutaneous emphysema formation; ruptured lung bullae, penetrating thoracic wounds; clostridial myositis.

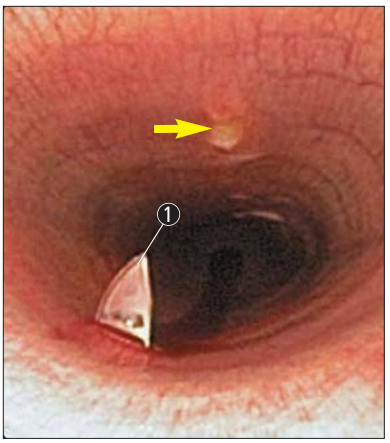
Diagnosis

Examination of a horse with tracheal laceration or rupture begins with a complete physical examination. Horses with severe subcutaneous emphysema are at risk of pneumothorax, therefore the horse should be evaluated closely for clinical signs of pneumothorax. Pneumothorax can be diagnosed on auscultation of the lungs, thoracic radiographs, and thoracic ultrasound. Endoscopy is necessary to confirm a diagnosis of tracheal rupture in the absence of an external skin wound (**313**, **314**). Horses with tracheal rupture typically have a small rupture site on the dorsal surface of the trachea.

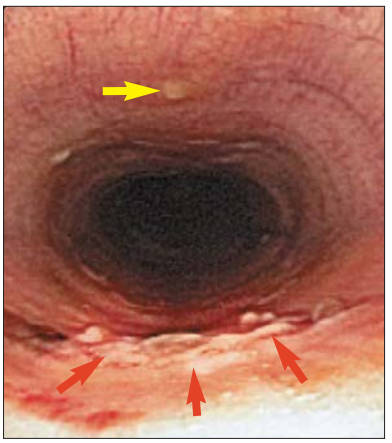
Management/treatment

Horses with a tracheal laceration can be managed conservatively or surgically depending on the extent of the laceration. Tracheal lacerations <3–5 cm in length are best left to heal via second intention. Tracheal lacerations involving >50% of the diameter of the trachea should be reconstructed as described for tracheal resection and anastomosis (see Further reading). A martingale apparatus is required post surgical repair to lessen the chance of disruption of the surgical site. The prognosis for small tracheal defects managed conservatively is good. The prognosis for large tracheal defects managed surgically is guarded because of the risk of tracheal stricture and deformity.

Tracheal rupture (without a skin laceration) is best managed with a temporary tracheotomy (**315**). A tracheotomy is performed as close as possible to the tracheal rupture site. Location of the appropriate



◀ 316 A tracheotomy being performed (1) opposite to the tracheal rupture site (arrow).



▶ 317 Healed tracheal rupture site (yellow arrow) 7 days post tracheotomy, and granulation tissue at the tracheotomy site (red arrows).

tracheotomy site is aided by concurrent tracheal endoscopy (**316**). The advantage of tracheotomy is that it prevents further accumulation of subcutaneous air. The tracheotomy diverts airflow through the tube instead of through the rupture site. The tracheotomy tube is removed once the rupture site has granulated. This usually occurs in 5–7 days (**317**). The prognosis for tracheal rupture managed with tracheotomy is good.

Key points

- Tracheal rupture can result secondary to external trauma to the cervical trauma with or without an external skin wound.
- Diagnosis of tracheal rupture without an external wound is best confirmed with endoscopy.
- Open tracheal lacerations can be managed conservatively or surgically depending on the extent and length of the tracheal involvement.
- Closed tracheal rupture is best managed with a temporary tracheotomy.

Tracheal neoplasia

Definition/overview

The most common neoplasia of the trachea is squamous cell carcinoma (SCC). Other neoplasms affecting the trachea include lymphoma and primary lung tumors.

Etiology/pathophysiology

SCC is the most common neoplasm involving the respiratory tract of horses. The etiology is unknown, although some studies have incriminated papilloma virus as an inciting factor. Breeds of horse with white coat patterns seem to be more at risk of SCC. This suggests that the lack of melanin has an influence on the absorption of UV radiation. Tracheal neoplasia causes clinical signs of upper respiratory noise and exercise intolerance secondary to local invasion of the trachea and obstruction of the tracheal lumen.

Clinical presentation

Affected horses are usually >15 years old. There is no known sex predilection. Breeds with non-pigmented skin seem to be more at risk of SCC. Horses affected with tracheal neoplasia typically present for evaluation of tracheal obstruction, which results in respiratory distress in severe cases and respiratory noise and exercise intolerance in mild to moderate cases. Other clinical signs in affected horses include coughing, especially following manipulation of the trachea, and epistaxis, and some horses may have a palpable mass in the cervical region involving the trachea.

Differential diagnosis

EE; epiglottitis; DDSP; epiglottic retroversion; RDPA; laryngeal hemiplegia; arytenoid chondritis; subepiglottic cysts; tracheal collapse.

Diagnosis

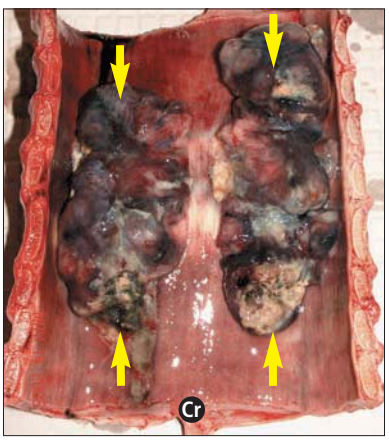
Signalment and clinical signs are usually suggestive of tracheal neoplasia. Physical examination should include auscultation of the heart and lungs. Careful auscultation of the trachea itself can reveal evidence of mucus or accumulation of exudate. Endoscopy is required to confirm a presumptive diagnosis of tracheal neoplasia. Endoscopic findings compatible with tracheal neoplasia include exudate or hemorrhage in the nasal passage or laryngeal lumen, masses within the nasopharynx, and tracheal obstruction with a mass or masses. SCC involving the trachea appears as a granulating, white to red mass intimately attached to the tracheal wall. Tracheal lymphoma has a pink or red, granulating appearance (**318**).

The masses are usually sessile and not pedunculated. They can almost completely obstruct the trachea in severe cases. Before deciding on treatment, a biopsy of the mass is recommended. Small samples can be obtained with endoscopic biopsy forceps placed down the biopsy channel of the endoscope. Unfortunately, since these biopsy samples are small, a diagnostic sample might not be obtained. It is not unusual to have granulation tissue surrounding a neoplastic growth, therefore if biopsy results indicate granulation tissue, this does not rule out neoplasia. To avoid this problem the authors recommend that a tracheotomy is performed as close as possible to the tracheal mass (**319**). Bronchoesophageal or Ferris-Smith rongeurs can then be used to obtain a representative biopsy of the entire mass. The authors have found that these larger biopsy samples are more reliable as a diagnostic tool.

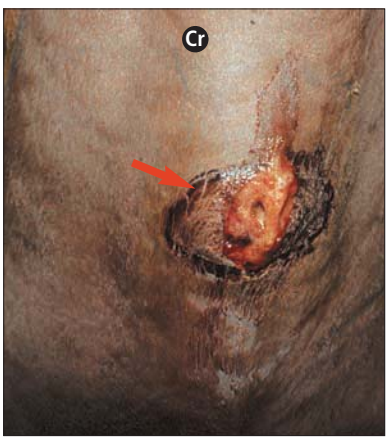
Finally, all horses with a presumptive diagnosis of tracheal neoplasia should have thoracic radiographs obtained and ultrasound performed. Some affected horses may also have neoplastic masses involving the lungs, mediastinum, or thoracic wall. Right and left lateral view thoracic radiographs should be obtained. Ultrasound is helpful in identifying masses on the surface of the lung or attached to the thoracic wall.

Management/treatment

Treatment options should be considered after a biopsy has been obtained and histopathologic findings are available. The management of tracheal neoplasia is limited. Small focal masses involving one portion of the trachea can be managed surgically with endoscopic laser surgery. A contact diode laser can be used to excise these types of masses. In some cases a tracheotomy is required to allow access to the tumor and permit removal of the mass following laser resection. Unfortunately, the experience of the authors is that most horses with tracheal neoplasia cannot be managed successfully with surgery because it is unlikely to result in complete remission of the neoplasia. However, masses can be palliated temporarily with a laser in horses with emotional or financial value.



◀ **318** **Gross postmortem photograph of tracheal lymphoma (arrows). (Cr) cranial.**



▶ **319** **Mid-cervical tracheotomy used to obtain a biopsy from a tracheal neoplastic mass (arrow). (Cr) cranial.**

Key points

- The most common type of tracheal neoplasia is SCC.
- Affected horses present with the primary clinical sign of respiratory distress, respiratory noise, or exercise intolerance.
- Endoscopy is the best diagnostic tool to identify tracheal neoplasia.
- Biopsy samples can be obtained with the aid of a tracheotomy.
- Small focal masses may be treated successfully with contact diode laser surgery.
- Most horses with tracheal neoplasia cannot be managed successfully with surgery.
- Palliative therapy is effective in some horses.

ABNORMAL LUNG SOUNDS

Overview

For a description and interpretation of the abnormal lung sounds that may be heard on auscultation of the chest see Chapter 3 (Clinical examination).

Increased lung sounds heard diffusely over both sides of the chest may occur with a physiologicl increase in ventilation such as secondary to exercise, hyperthermia, or excitement. The pathologic causes of increased lung sounds are fever, shock, sepsis, pain, non-cardiogenic pulmonary edema, anaphylaxis, obstructive lung disease (RAO, severe IAD; see Chapter 5, The coughing horse), cardiac disease, acidemia, and severe anemia. Anemia itself has no major effect on pulmonary function because peripheral chemoreceptors detect a decrease in P_{aO_2} and not arterial oxygen content. Increased ventilation in cases of severe anemia occurs when tissue hypoxia results in increased anaerobic metabolism and lactic acidosis.

A focal increase in lung sounds suggests the presence of lung consolidation or a mass. Lung consolidation refers to the firmer texture of pneumonic lung due to atelectasis and filling of airspaces with exudate (see Chapter 5, The coughing horse).

Decreased breath sounds may result from pleural effusion (see Chapter 7, The horse with increased respiratory effort) due to pleuropneumonia, pleuritis, pericarditis, peritonitis, viral respiratory diseases, mycoplasmal infection, congestive heart failure, liver disease, diaphragmatic hernia, hypoproteinemia, neoplasia (e.g. lymphoma, gastric SCC), equine infectious anemia, pulmonary granulomas, chylothorax, and fungal pneumonia. Pulmonary edema often presents with decreased lung sounds or silent areas. Lung sounds are absent ventrally with pleural effusion and dorsally with pneumothorax. Percussion of the thorax also allows differentiation between pleural effusion, resulting in ventral dullness, and pneumothorax, associated with increased resonance dorsally, but normal percussion ventrally. Emphysema can also result in decreased or absent lung sounds because of decreased airflow velocity in peripheral airways.

Wheezing heard over multiple areas on both sides of the chest indicates diffuse airflow obstruction and is often detected in horses with RAO and severe IAD. Expiratory wheezes over focal areas are also commonly heard in bronchopneumonia (see Chapter 5, The coughing horse). Inspiratory wheezes are common in horses with atelectasis or consolidation. Atelectasis may be congenital, as in foals with acute respiratory distress syndrome, or acquired. Acquired atelectasis may develop after prolonged recumbency, from airway obstruction, or from compression of the lung secondary to pleural effusion, masses, pneumothorax, and excessive abdominal distension.

CONGENITAL ABNORMALITIES

Wry nose (deviation of the premaxilla)

Definition/overview

Wry nose, or deviation of the premaxilla, is a congenital abnormality. It typically involves the premaxilla, nares, rostral nasal passages, and the nasal septum. The deviation is in a medial to lateral direction.

Etiology/pathophysiology

The etiology of wry nose is unknown. The most commonly incriminated cause is an abnormal intrauterine position. There is no known breed or sex predilection. The heredity of wry nose is unknown. The breeding of affected horses is discouraged.

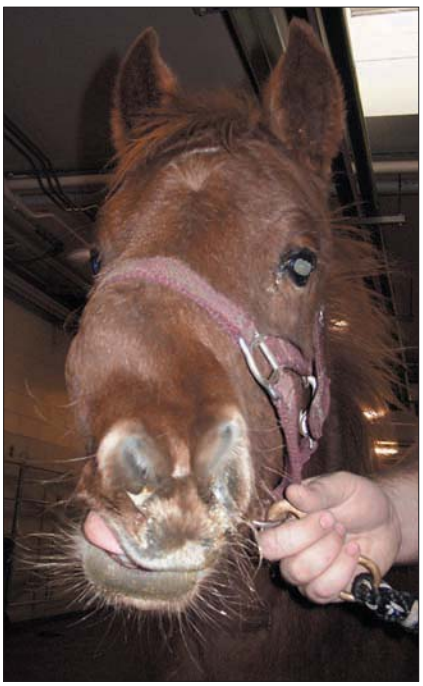
Differential diagnosis

The type of physical deformity is pathognomonic for this disease, so it is difficult to confuse wry nose with any other congenital deformity.

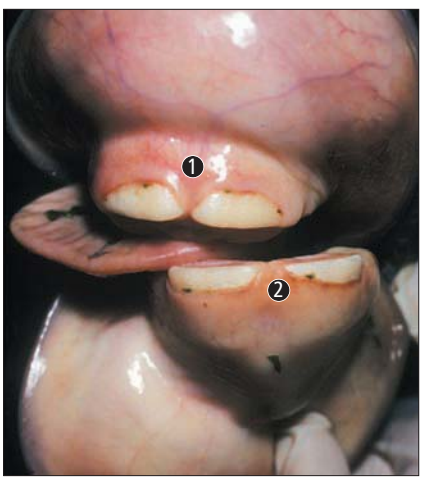
Diagnosis

Because the clinical signs are severe, diagnosis of wry nose is readily made on physical examination (**320**). An oral examination allows for evaluation of the degree of upper incisor and cheek teeth misalignment with the lower incisors and cheek teeth (**321**). Affected foals should also be evaluated for concurrent cleft palate, which is not unusual and can complicate treatment.

Endoscopy and radiography are recommended. Endoscopy allows for evaluation of the hard and soft palate and the relative size of the nasal passages. Lateral and dorsoventral radiographic views of the skull are recommended to evaluate the incisive bone and the extent of the bony deformity of the maxilla.



▲ **320** **Severe deviation of the premaxilla towards the left side.**



▶ **321** **Incisor malocclusion associated with the deviation of the premaxilla (1). The mandible (2) is in normal alignment.**

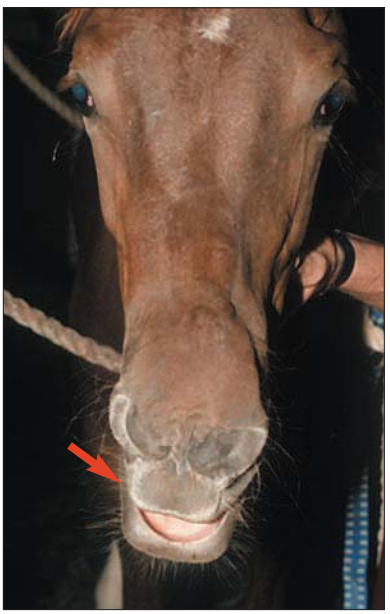
Management/treatment

Horses with mild wry nose can be managed conservatively. Affected horses can grow to adulthood with a mild degree of deviation. Horses with severe wry nose in combination with cleft palate should be considered for euthanasia. In situations where the premaxilla deformity is mild to moderate and the palate is normal, surgical correction can be considered. However, in some cases owners may elect for surgical correction despite a guarded to poor prognosis for success.

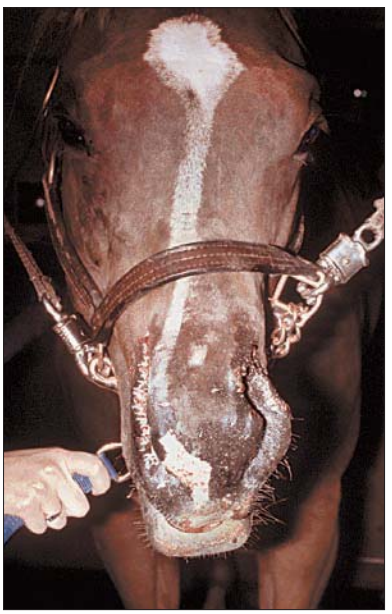
Surgical correction of wry nose is achievable and has been described (see Further reading). The main problem is maintaining normal alignment of the incisors. Straightening of the premaxilla can be readily accomplished, but aligning the maxillary incisors with the mandibular incisors is difficult. The procedure involves an osteotomy of the maxilla and incisive bone and, sometimes, the nasal septum. The maxilla is realigned so that it is in line with with the mandible (**322**, **323**). The maxilla is then stabilized with either intramedullary pins or with an external fixator or an Ilizorov-type device. If cleft palate is also present, it is repaired as previously described (Chapter 6, pp. 133–135). The main postoperative complication is maintaining normal alignment between the maxillary and mandibular incisors and improving the postoperative appearance of the nares and rostral nasal passage. Most foals following surgery appear to have collapse of the nares despite a normal alignment. For some foals this will improve with age; in others, where an athletic career is desired, resection of the alar folds and/or nasal diverticulum may be required. Overall, the prognosis for life following corrective surgery is fair to good, but the prognosis for athletic use is poor to guarded. If there is concurrent cleft palate, the prognosis is even worse.

Key points

- Wry nose, or deviation of the premaxilla, is a common cause of facial deformity in the foal.
- Mildly affected foals can be managed without surgery.
- Moderately affected foals can be treated with osteotomy of the maxilla and incisive bone, with a fair to good prognosis for life.
- Severely affected foals should be considered for euthanasia.
- The prognosis for athletic use following surgical correction is poor to guarded.



◀ **322** **Postoperative photograph of a yearling Paso Fino following osteotomy and realignment of the premaxilla. Note that the maxillary deviation has been corrected, but prognathism is present along with deformation of the nares and rostral nasal passage (arrow).**



▶ **323** **Postoperative appearance of a horse following surgical correction of deviation of the premaxilla and resection of the nasal diverticulum bilaterally to increase airflow during exercise.**

Choanal atresia

Definition/overview

Choanal atresia is a congenital abnormality resulting in unilateral or bilateral obstruction of the choanae. Choanal atresia is caused by failure of the bucconasal membrane to rupture. The obstruction can be membranous or fibrocartilaginous.

Etiology/pathophysiology

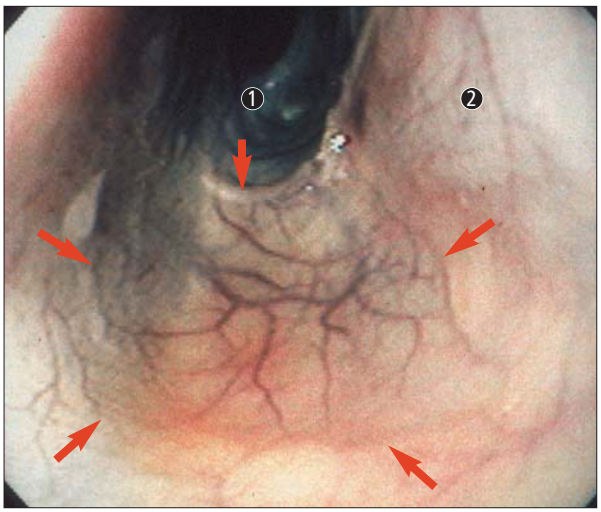
Choanal atresia results from failure of the bucconasal membrane to separate from the primitive buccal or oral cavity and the nasal pits during embryologic development. This membrane can be complete, consisting of bone or fibrocartilage, or be simply membranous. In horses, the majority of affected cases are membranous. Bilateral choanal atresia results in respiratory distress at birth and can lead to death if orotracheal intubation or emergency tracheotomy is not performed. Unilateral choanal atresia is usually not diagnosed until the horse is placed into training, and it is not a life-threatening condition.

Clinical presentation

There is no known breed or sex predilection, although most of the reported cases have been in Standardbreds. Bilateral choanal atresia is readily diagnosed because affected foals are dyspneic at birth and will die unless an emergency intervention is performed. In unilateral cases, placement of hands over the nares reveals no air movement in the affected side. In addition, it may prove impossible to pass a nasogastric tube on the affected side(s). Unilateral choanal atresia may not be clinically evident until the horse is placed into training. Affected horses make a respiratory noise and develop exercise intolerance.

Differential diagnosis

EE; epiglottitis; DDSP; epiglottic retroversion; RDPA; laryngeal hemiplegia; arytenoid chondritis; tracheal collapse.

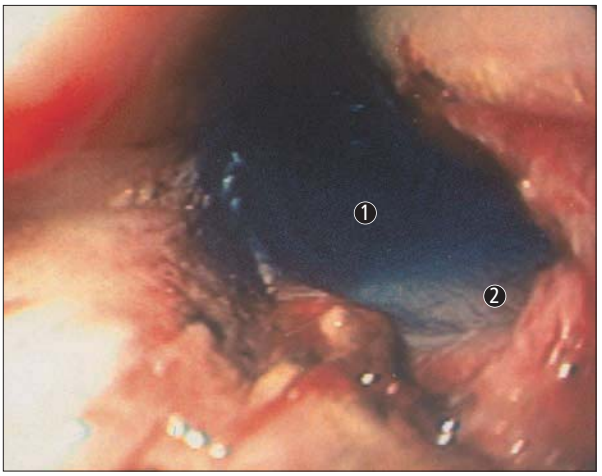


▲ **324 Endoscopic photograph of right-sided, unilateral choanal atresia in a Standardbred racehorse. Note the inability to view the nasopharynx because of the obstructing membrane (arrows). (1) ethmoid turbinates; (2) caudal nasal septum.**

Diagnosis

Diagnosis of choanal atresia is suspected based on clinical signs of nasal obstruction or respiratory distress in foals at birth. Evaluation of airflow from the nares reveals no air movement from the affected side(s). Endoscopy is the best method to confirm choanal atresia. Endoscopic findings compatible with choanal atresia include failure to pass the endoscope into the nasopharynx and the presence of a sheet of tissue at the caudal aspect of the nasopharynx (**324**).

Radiography can also be performed to confirm a diagnosis of choanal atresia. Contrast medium is placed into the nasal passage and lateral, dorsoventral, and oblique skull radiographic views are obtained. The contrast medium will outline the obstructing membrane. The final diagnostic test that can be used to determine nasal obstruction is acoustic rhinometry.



◀ 325 Postoperative endoscopic photograph of the horse in 324. The obstructing membrane was incised with a contact Nd:YAG laser fiber. The nasopharynx (1) and soft palate (2) can be visualized after the membrane has been removed.

Management/treatment

Foals with bilateral choanal atresia must have an emergency tracheotomy or an orotracheal tube placed as soon as possible after birth. Following tracheotomy, treatment options can be discussed with the owner. Few cases of choanal atresia have been reported in the veterinary literature. This makes treatment recommendations difficult because of lack of experience with the condition. Nonetheless, there are basically two methods of surgical management. The first technique is to resect the membrane through unilateral or bilateral nasal osteoplastic flaps. The second technique is surgical ablation using a non-contact or contact diode or Nd:YAG laser (**325**). The problem with both these surgical techniques is that following resection or ablation of the obstructing membrane, stricture of the new opening is not uncommon and should be expected to occur to some degree. To help minimize the chances for stricture, the choanae should be stented for 2–3 weeks. The authors currently recommend ablation of the obstructing membrane with non-contact diode laser surgery. Once the choana has been opened, the new opening is stented with a shortened nasotracheal tube for at least 2–3 weeks. The prognosis for choanal atresia is poor unless the new choanal opening remains patent. Horses with unilateral choanal atresia may be successfully treated and returned to athletic activity. However, the majority of horses with choanal atresia have a guarded prognosis for athletic activity, especially racing. Horses with small nasal passages and stricture of the choanae following surgery have a guarded prognosis. Some horses experiencing problems with stricture following laser treatment can be treated with partial nasal septum resection.

Key points

- Choanal atresia is a congenital obstruction of the nasal choanae caused by failure of the bucconasal membrane to rupture.
- Horses with bilateral choanal atresia will die unless an emergency procedure is performed to provide an airway at birth (tracheotomy or oral intubation).
- Horses with unilateral choanal atresia may not show clinical signs until the horse enters athletic training.
- Diagnosis of choanal atresia can be confirmed readily with endoscopy.
- Surgery can be used to resect or ablate the obstructing membrane, but the prognosis for athletic activity following surgery is guarded to fair.

Cleft palate

See Chapter 6, pp.132–135.

Subepiglottic and pharyngeal cysts

Definition/overview

Subepiglottic and pharyngeal cysts are congenital malformations of the pharyngeal pouches.

Etiology/pathophysiology

The pharyngeal pouches originate from the pharyngeal endoderm and project between the branchial arches. Malformations of the pharyngeal pouches lead to the formation of subepiglottic, pharyngeal, and other cystic structures involving the nasopharynx, soft palate, larynx, or perilaryngeal structures. Subepiglottic cysts are remnants of the thyroglossal duct. Dorsal pharyngeal cysts are remnants of Rathke's pouch (craniopharyngeal duct). Other locations include the caudal free border of the soft palate, the laryngeal ventricle, the arytenoid cartilage, and perilaryngeal structures. Cystic structures here are lined by stratified squamous, pseudostratified, columnar, or cuboidal epithelium or a combination of epithelial types. It has also been suggested that subepiglottic inflammation can block the mucus-secreting glands and contribute to the development of a subepiglottic cyst.

Clinical presentation

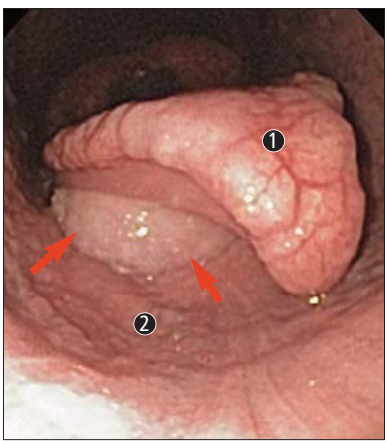
Congenital cystic structures are usually diagnosed in young (<3 years old) racehorses presented with a complaint of respiratory noise and exercise intolerance. Commonly affected breeds include Thoroughbred and Standardbred racehorses and Quarter Horses. Clinical signs of congenital cystic structures include respiratory noise, airway obstruction in foals, dysphagia, chronic cough with bilateral mucopurulent nasal discharge and pneumonia, and a history of DDSP in racehorses.

Differential diagnosis

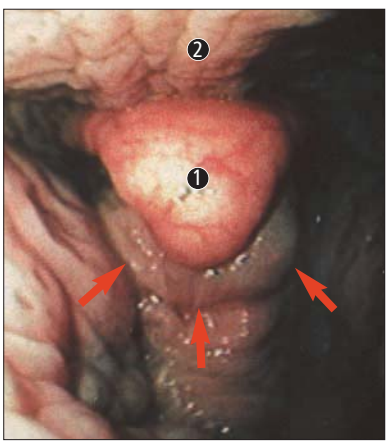
EE; epiglottitis; DDSP; epiglottic retroversion; RDPA; laryngeal hemiplegia; arytenoid chondritis; tracheal collapse.

Diagnosis

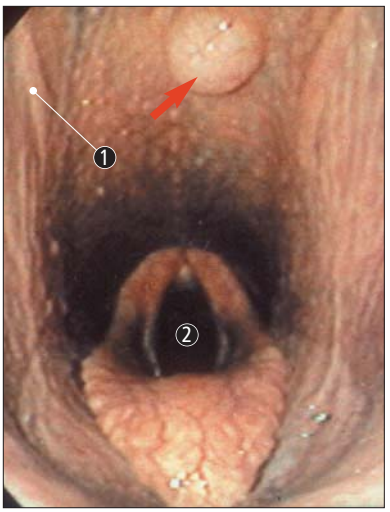
Endoscopy is the best method of diagnosis for congenital cysts. Subepiglottic cysts are typically visualized on the lingual surface of the epiglottis (**326**). However, in some horses the cyst is located beneath the edge of the palate and cannot be visualized until the horse swallows; in some cases it cannot be seen at all. In cases where doubt exists, these horses need to be examined under general anesthesia. The endoscope is positioned in the oral cavity and the lingual surface of the epiglottis examined for presence of a cyst (**327**). In addition to endoscopy, a hand can be placed in the mouth and the epiglottis manually palpated. A lateral view skull radiograph, which will reveal a soft tissue mass beneath the epiglottis, will help confirm the diagnosis.



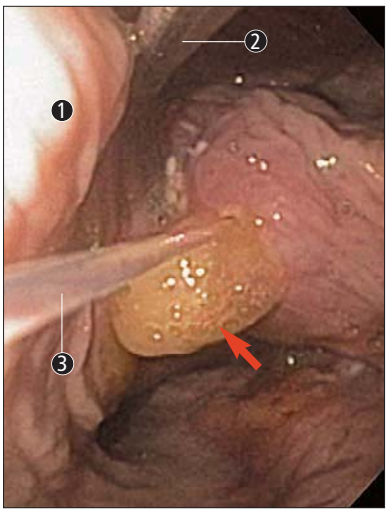
◀ **326** **Subepiglottic cyst (arrows) in a Standardbred racehorse. (1) epiglottis; (2) soft palate.**



▶ **327** **Intraoral endoscopic view of a subepiglottic cyst (arrows). Note the positioning of the epiglottis (1) and soft palate (2).**



◀ **328** **Endoscopic photograph of a horse with a dorsal pharyngeal cyst (arrow). (1) guttural pouch opening; (2) larynx.**



▶ **329** **Subepiglottic cyst (arrow) being treated with intraoral diode laser ablation. The epiglottis (1) is being retracted with a broncho-esophageal grasping forceps (2) to improve visualization of the cyst. The diode laser fiber (3) is being inserted into the cyst to cauterize the cyst lining.**

Dorsal pharyngeal cysts are easily visualized with endoscopy. They are usually close to the dorsal pharyngeal recess (**328**).

Management/treatment

The majority of congenital cysts that are causing respiratory noise or exercise intolerance will require surgery. Some cysts, particularly dorsal pharyngeal cysts, can be incidental findings and do not cause clinical signs. There are three methods of surgically removing subepiglottic cysts: surgical resection via laryngotomy; ablation or removal with a diode (**329**) or Nd:YAG laser; and oral excision of the cyst using obstetrical wire positioned around the cyst via the oral cavity under general anesthesia. The authors' preference is to treat subepiglottic cysts with laser ablation or resection. The laser is applied to the exterior of the cyst in order to heat the cystic fluid, and the resultant thermal injury cauterizes the cyst lining. The cyst is then punctured to allow the fluid to escape. The laser fiber is inserted into the cyst and the lining heated with the fiber. Small pedunculated cysts, which are retracted with a grasping forceps positioned via the oral cavity, can be surgically excised using a contact laser fiber. Finally, large, thickened subepiglottic cysts, which are sometimes diagnosed in older horses, may be best treated with surgical resection via a laryngotomy. Laser surgery does not work as well in these cases because the subepiglottic tissues may be fibrotic and not easily fenestrated with a contact laser fiber.

Dorsal pharyngeal cysts causing clinical signs can be treated using the laser technique described above for subepiglottic cysts. Cystic structures involving the soft palate or larynx may require surgical removal using either the laser while standing or with traditional surgical techniques. The prognosis for surgical treatment of congenital cystic structures is good, although some horses can develop DDSP following treatment of subepiglottic cysts.

Key points

- Pharyngeal, laryngeal, soft palate, and subepiglottic cysts are congenital defects secondary to malformations of the pharyngeal pouches.
- Congenital cystic structures can result in respiratory noise and exercise intolerance.
- Dorsal pharyngeal cysts may be incidental findings and cause no clinical signs.
- Most congenital cystic structures are amenable to laser resection or ablation. Subepiglottic cysts in older horses should be considered for removal via laryngotomy.
- The prognosis following surgical treatment is favorable.

Branchial arch defects

Definition/overview

There are six branchial arches in the embryo of vertebrates. The branchial arches form the skeletal, muscular, neurovascular, and cartilaginous structures of the head and neck. In horses, defects of the fourth branchial arch are the most clinically important. Fourth branchial arch defects lead to anatomic abnormalities involving the cricothyroid cartilages, cricothyroid muscle, upper esophageal sphincter, and the thyro- and cricopharyngeus muscles.

Etiology/pathophysiology

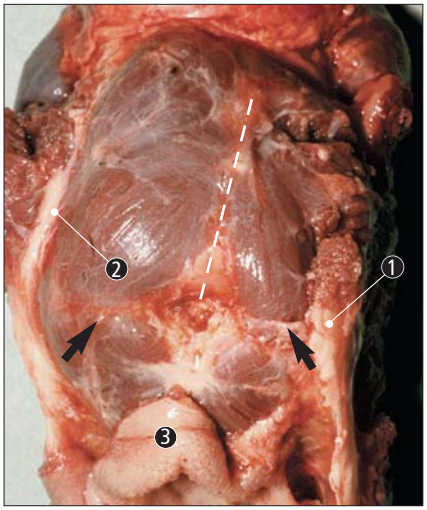
The laryngeal muscles and cartilages originate from the fourth and sixth branchial arches. The extrinsic structures of the larynx include the wing of the thyroid, the cricothyroid articulation, the cricothyroid muscle, the upper esophageal sphincter, and the thyro- and cricopharyngeus muscles originating from the fourth branchial arch. The intrinsic structures of the larynx originate from the sixth branchial arch and include the intrinsic laryngeal muscles and the cricoid and arytenoid cartilages. Fourth branchial arch defects result in bilateral or unilateral deformities, leading to a variety of clinical signs such as RDPA, right and left laryngeal hemiplegia, dysphagia, respiratory noise, exercise intolerance, and signs of abdominal pain. These signs can occur alone or in combination.

Clinical presentation

Thoroughbreds appear to be the most common breed affected by defects involving the fourth branchial arch. There is no apparent sex predilection and horses ranging in age from 3 months to 6 years have been diagnosed with the condition. Clinical signs of this disorder include respiratory noise, exercise intolerance, eructation secondary to aerophagia, dysphagia, and signs of abdominal pain.

Differential diagnosis

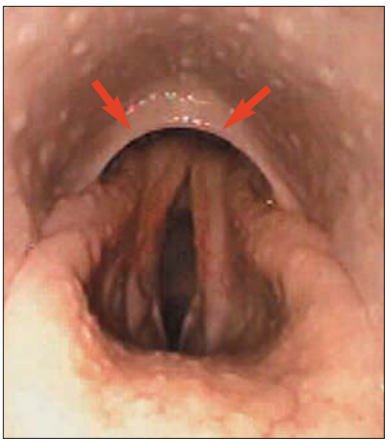
EE; epiglottitis; DDSP; epiglottic retroversion; RDPA; laryngeal hemiplegia; arytenoid chondritis; tracheal collapse.



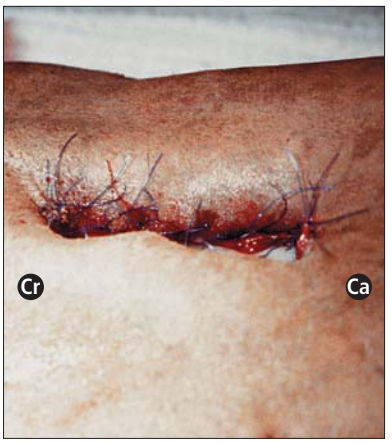
▲ **330 Gross postmortem photograph of a horse with a left-sided branchial arch defect. Note the abnormal shape of the left thyroid cartilage (1) compared with the right thyroid cartilage (2), the abnormally shaped cricoarytenoideus dorsalis muscle belly, and the abnormally shaped muscular process of the arytenoid cartilage (arrows). The dotted line indicates the midline of the cricoid cartilage. (3) corniculate processes of the arytenoid cartilages. (Photo courtesy JF Fessler)**

Diagnosis

Diagnosis of fourth branchial arch defects starts with a complete physical examination including careful palpation of the larynx. Manual palpation of the larynx should include an evaluation of the thyroid cartilage and the space between the cricoid and thyroid cartilages. Affected horses typically have an abnormally shaped or absent thyroid cartilage and an increased space between the thyroid and cricoid cartilages on one or both sides depending on whether the defect is unilateral or bilateral (**330**).



◀ **331** **Branchial arch defect. Note the rostral displacement of the palatopharyngeal arch (arrows) over the corniculate processes of the arytenoid cartilages, the opening of the esophageal sphincter, and the bilateral laryngeal hemiplegia.**



▶ **332** **Horse with a branchial arch defect being treated with a permanent tracheotomy while in dorsal recumbency under general anesthesia. (Cr) cranial; (Ca) caudal.**

Endoscopy is the most useful diagnostic test for evaluation of laryngeal function. Endoscopic abnormalities include RDPA, right, left, or bilateral laryngeal hemiplegia, opening of the rostral esophageal sphincter, and vocal cord prolapse (**331**).

Lateral view radiographs of the larynx may show continuous air opacity between the nasopharynx and the esophagus. Magnetic resonance imaging and laryngeal ultrasound have also been used to further characterize the anatomic abnormalities associated with a fourth branchial arch defect. Treadmill endoscopy can be useful in evaluating horses in which the resting endoscopic findings are equivocal. Once a diagnosis has been made using dynamic endoscopy, the surgical options can be considered. The final surgical treatment plan can only be determined once all the structures involved in the defect have been identified.

Management/treatment

Management of fourth branchial arch defects includes conservative treatment, surgery, and euthanasia. Mildly affected horses or horses in which an athletic career is not desired can be managed conservatively. Surgical management should be considered in horses for which an athletic career is desired. The authors recommend performing a high-speed treadmill examination so that a surgical plan can be tailored to the horse's need. Deformities that can be considered for surgical correction include RDPA and vocal fold prolapse without laryngeal hemiplegia. Management of laryngeal hemiplegia with prosthetic laryngoplasty is not likely to be successful because of the abnormal laryngeal anatomy. Unfortunately, this may not be determined until the larynx is surgically explored.

For severely affected horses, one surgical option is a permanent tracheotomy (**332**). Horses managed in this way can be used for light exercise or breeding. The final option for severely affected horses is euthanasia.

The prognosis is poor to guarded even with surgical management.

Key points

- Branchial arch defects in the horse most commonly involve the fourth branchial arch.
- Fourth branchial arch defects involve the thyroid cartilage and the cricothyroid articulation.
- Manual palpation of the larynx is important for diagnosis of fourth branchial arch defects.
- Horses with RDPA and right laryngeal hemiplegia should be strongly suspected of a branchial arch defect.
- Surgical management of branchial arch defects should be guided by high-speed treadmill evaluation.
- The prognosis for athletic use, even with surgery, is poor to guarded.

Guttural pouch tympany

Definition/overview

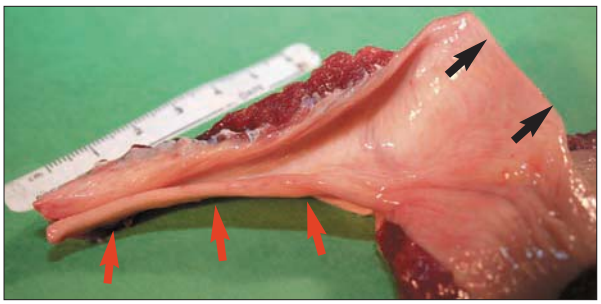
GP tympany is a congenital abnormality causing air accumulation in the GP secondary to an abnormality involving the plica salpingopharyngea or salpingopharyngeal fold.

Etiology/pathophysiology

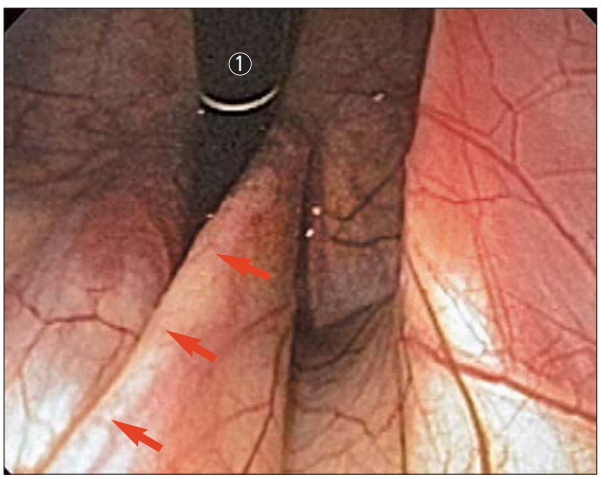
Tympany of the GP is the direct result of entrapment of air within one or both GPs. In the normal horse the GP openings open when the horse swallows, thus allowing passage of air in and out of the pouch. In GP tympany, air is allowed into the pouch via the plica salpingopharyngea, but is not allowed to exit the pouch. The etiology of GP tympany is most likely secondary to redundancy of the plica salpingopharyngea. The salpingopharyngeal fold is a soft tissue structure that lies just beneath the cartilaginous opening of the GP (**333**, **334**). A redundant plica salpingopharyngea can be congenital or can occur secondary to chronic GP inflammation.

Clinical presentation

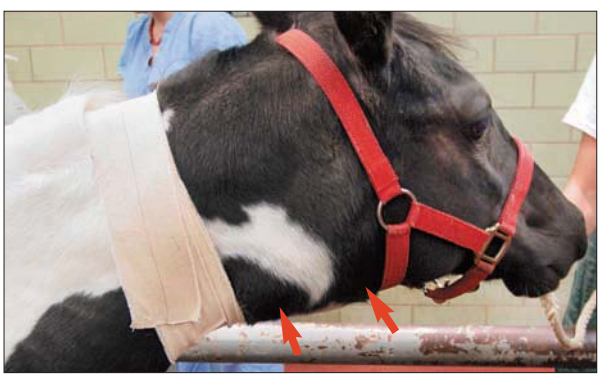
The most commonly affected horses are 1 year of age or less. There does appear to be a sex and breed predilection with fillies and Arabians being commonly affected. Other affected breeds include Thoroughbreds and Quarter Horses. In affected foals, one or both GPs become distended with air and form a characteristic non-painful and elastic swelling in the parotid region (**335**).



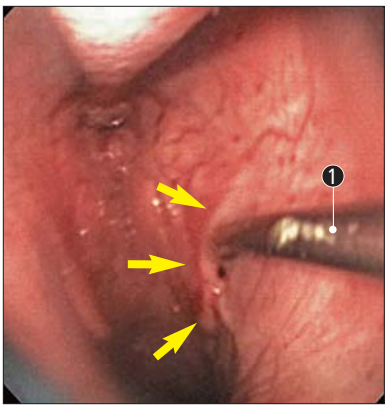
▲ **333** **Gross postmortem photograph of the medial aspect of the cartilaginous flap (black arrows) and plica salpingopharyngea (red arrows) of the right guttural pouch.**



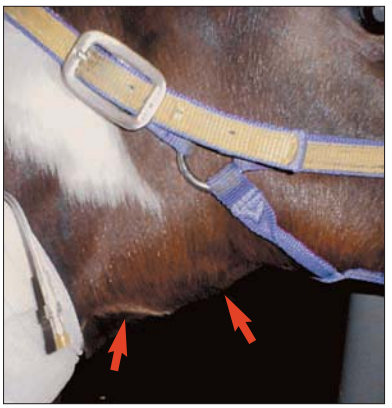
▲ **334** **Endoscopic photograph of a retroflexed endoscope visualizing the plica salpingopharyngea (arrows) from the interior of the guttural pouch. A portion of the endoscope is visible (1) and the image appears 'upside down'.**



▶ **335** **A Paint Horse filly affected with unilateral guttural pouch tympany. Note the characteristic swelling of the throat latch (arrows).**



◀ **336** **Chambers catheter (1) decompression of the left guttural pouch (arrows).**



▶ **337** **The horse in 335 following decompression (arrows) of the affected guttural pouch.**

Air distension of the GP can result in respiratory noise, respiratory distress, dysphagia, aspiration pneumonia, and secondary GPE. It is important to determine whether the condition is unilateral or bilateral because the treatment options vary depending on this determination.

Differential diagnosis

GPE; retropharyngeal lymph node enlargement; parotid salivary gland swelling; bronchopneumonia.

Diagnosis

Diagnosis is readily made based on clinical signs alone. The characteristic non-painful, soft swelling in the parotid region is easily differentiated from other conditions. The most challenging diagnostic dilemma is determining whether or not the condition is unilateral or bilateral.

There are several methods of determining whether the condition is bilateral or unilateral. These include: passage of an endoscope into one or both GPs, aspiration of air from the pouch with the most distension, and a dorsoventral radiographic view of the skull. Passage of an endoscope into the affected GP results in complete decompression of the pouch in cases of unilateral GP tympany. If one side is decompressed with the endoscope but the contralateral side is not, the condition is bilateral. If there is no change in the external appearance of the throat latch area after the endoscope is advanced in one pouch, the condition is most likely unilateral on the contralateral side.

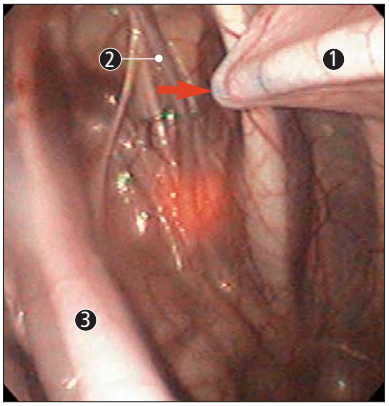
The second method of determination is to pass a Chambers catheter into the most distended pouch under endoscopic guidance (**336**, **337**). The findings described above hold true for this technique. Some reports have mentioned external needle decompression of the GP, but the authors do not recommend this technique because of the possibility of damage to the neurovascular structures within the pouch.

The final method of determination of unilateral or bilateral disease is a dorsoventral radiographic view of the skull. Unilateral GP tympany causes displacement of the affected medial septum of the pouch towards the contralateral side, whereas bilateral GP tympany results in bilateral air distension of each pouch.

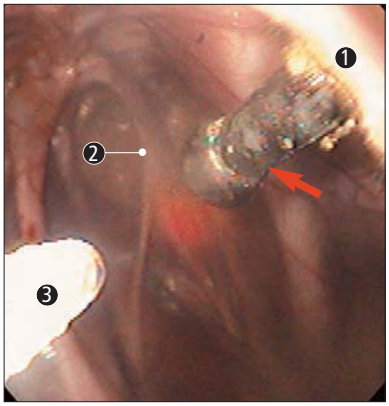
Endoscopic findings compatible with GP tympany include collapse of the dorsal pharyngeal wall and, if concurrent GPE is present, purulent exudate can be visualized exiting the GP opening.

Management/treatment

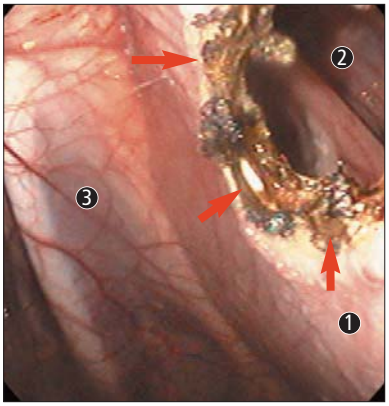
Surgical management is the only viable technique to resolve the clinical signs of GP tympany. The most important surgical consideration is the determination of unilateral versus bilateral disease. The surgical techniques for GP tympany have been well described (see Further reading). Acceptable surgical techniques for unilateral disease include fenestration of the medial septum of the GP (allowing the abnormal pouch to communicate with the normal pouch) with laser surgery or traditional surgery, surgical resection of the plica salpingopharyngea on the affected side, and laser plica salpingopharyngea fistulation.



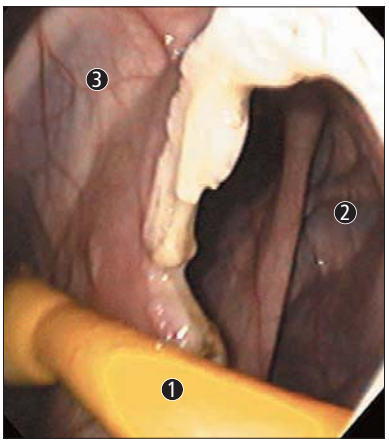
▲ **338** **Final positioning of a Chambers catheter (arrow) in the left guttural pouch to facilitate laser fenestration of the median septum (1) from the right guttural pouch. Note the internal carotid artery (2) and stylohyoid bone (3) within the right guttural pouch.**



▲ **339** **Chambers catheter (arrow) exiting a laser fenestration in the median septum (1). (2) internal carotid artery; (3) laser fiber.**

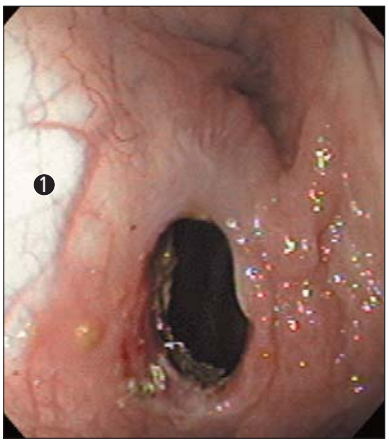


▲ **340** **Completed laser fenestration (arrows) of the median septum (1). The interior of the left guttural pouch (2) can now be visualized from the right side. (3) right guttural pouch.**



◀ **341** **Endoscopic photograph 7 days post laser fenestration of the median septum. Note the presence of the Foley catheter (1) through the fenestration to minimize the risk of postoperative stricture formation. (2) left guttural pouch; (3) right guttural pouch.**

▶ **342** **Healed laser plica salpingopharyngea fistula of the right guttural pouch. Note the relationship between the cartilage flap of the guttural pouch (1) and the fistula.**



Bilateral GP tympany can be managed with a combination of medial septum fenestration with resection of the plica salpingopharyngea on one side, with laser fenestration of the medial septum and unilateral laser plica salpingopharyngea fistula, or with bilateral laser plica salpingopharyngea fistulation (**338**–**342**). Fenestration of the median septum between the normal and the abnormal pouch allows air to escape through the normal GP opening. The principle behind resection of the plica salpingopharyngea is to enlarge the normal opening into the GP. Fistulation through the plica salpingopharyngea allows for continuous decompression of the pouch.

The most significant complication of any of these techniques is stricture of the newly created opening. Frequent monitoring following surgery is important to determine whether or not stricture is occurring. The prognosis following surgical treatment of GP tympany is good, although in the authors' experience more than one surgical procedure may be required. Foals suffering from dysphagia or aspiration pneumonia in combination with GP tympany have a guarded to fair prognosis.

Key points

- GP tympany is a congenital abnormality involving the plica salpingopharyngea resulting in air accumulation within the GP.
- Diagnosis of GP tympany is straightforward and readily accomplished based on clinical signs alone.
- The most challenging diagnostic problem is differentiating unilateral from bilateral disease.
- Surgical management will depend on whether or not the condition is unilateral or bilateral. Minimally invasive techniques using endoscopy in combination with laser surgery can be performed standing without general anesthesia.
- The prognosis following surgical management is good, but recurrence of clinical signs may develop secondary to stricture of the newly created surgical opening in some horses.

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Chapter 5: The coughing horse

Acute cough

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Chapter 6: The horse with nasal discharge

Mucopurulent nasal discharge

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