Equine Respiratory Diseases

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About the Authors

Bonnie Rush earned her DVM degree from The Ohio State University in 1989. She completed her internship training at North Carolina State University in 1990, and her internal medicine residency training at The Ohio State University in 1993. She has been awarded the 1996 and 2003 Carl J. Norden Distinguished Teacher Award and the 2002 Pfizer Award for Research Excellence at Kansas State University. Dr. Rush’s research interest is equine respiratory disease, particularly respiratory physiology, immunology, and aerosol drug therapy. Her clinical interest is in equine respiratory, neurologic, and immune-mediated disease.

Tim Mair graduated from the University of Bristol in 1980. After working in general practice, he returned to the University of Bristol to undertake research into equine respiratory immunology. He earned his PhD in 1986, and then stayed on at Bristol as a lecturer in equine medicine. In 1990 he returned to practice, and is currently a partner at the Bell Equine Veterinary Clinic in Kent, England. He has been editor of Equine Veterinary Education since 1996. His particular interests are in respiratory and gastrointestinal medicine and surgery.
Diseases of the respiratory tract are common in horses of all ages and types. Respiration is a cellular activity, and the respiratory tract is the organ that permits respiration to take place. The requirements placed on the respiratory system by the body’s metabolism include the transfer of the precise quantity of oxygen from the inspired air to the arterial blood that the body tissues need, and the removal from venous blood the quantity of carbon dioxide they produce by metabolism. These requirements for respiration increase dramatically during exercise, and the respiratory system of the horse is designed to permit wide fluctuations in the amount of gas exchange taking place within the lungs.

The unique ability of the horse as an athlete is dependent on the integration of the respiratory system with a number of other body systems, including the musculoskeletal, nervous, and cardiovascular systems. Clinical or subclinical dysfunction of any of these systems can result in exercise intolerance, but in the healthy horse it appears to be the respiratory system that is the limiting factor that determines athletic performance.

The horse possesses several unique physiological responses to exercise that allow for an increased capacity for oxygen transport. The maximum oxygen consumption (VO₂ max) reached by the Thoroughbred racehorse can exceed 160 ml/kg/min, a value that is about double that of a human athlete. The horse experiences arterial hypoxemia, oxygen desaturation, and hypercapnia during exercise as a result of diffusion limitation and a relative hypoventilation. Any respiratory dysfunction can cause a further decline in ventilation and gas exchange, and therefore respiratory diseases are a major cause of exercise intolerance and poor performance. Respiratory tract diseases are second only to musculoskeletal diseases as the leading cause of wastage in racehorses.

Respiratory disease is rewarding to evaluate and treat for equine clinicians. The respiratory system is highly accessible for diagnostic testing, responds to
an extensive armamentarium of drugs, and has a relatively favorable capacity for healing. Techniques used to evaluate the equine respiratory tract include endoscopic examination, radiographic and ultrasonographic imaging, cytologic evaluation and bacterial culture of respiratory secretions, and histopathologic evaluation of respiratory mucosa and pulmonary parenchyma. Advanced imaging techniques, such as computed tomography and magnetic resonance imaging, are also increasingly used in certain diseases, especially diseases affecting the upper respiratory tract.

Unlike the central nervous system, the accessibility of the respiratory system allows clinicians to obtain a definitive diagnosis in most instances. Respiratory disorders typically respond favorably to appropriate medical therapy, and treatment options for bronchodilation, immunomodulation, antimicrobial activity, and reduction of pulmonary inflammation are well-characterized in horses. Surgical therapy for upper respiratory disease is common and results are often favorable. Surgical intervention for treatment of lower respiratory disease is less routine and is typically performed under grave or extreme circumstances.

Disorders of the respiratory system can be classified into 4 basic categories:

- Contagious upper respiratory tract (URT)
- Noncontagious URT
- Infectious lower respiratory tract (LRT)
- Noninfectious LRT disease

Chapters 3 through 22 represent each one of these categories of respiratory disease. Contagious URT pathogens include viral respiratory disease and strangles (Streptococcus equi), and are separated in this text from infectious, noncontagious conditions of the URT, such as guttural pouch mycosis and arytenoid chondritis. Noncontagious URT conditions include structural and functional abnormalities of the pharynx, larynx, and nasal passages. Pneumonia and pleuropneumonia are examples of infectious LRT conditions, whereas heaves, inflammatory airway disease, and exercise-induced pulmonary hemorrhage are common noninfectious LRT disease of horses.

Viral respiratory infections are common in horses, and equine herpesvirus type 4 (EHV-4, rhinopneumonitis), equine influenza, and equine viral arteritis are the most notable. Clinical signs of viral respiratory pathogens are often indistinguishable, and include pyrexia, serous nasal discharge, submandibular lymphadenopathy, anorexia, and cough. In addition to respiratory disease, equine herpesvirus type 1 (EHV-1) can cause abortion and neurologic disease. Equine viral arteritis produces respiratory disease, vasculitis, and abortion. Equine herpesvirus type 2, rhinovirus, and reovirus are ubiquitous viral respiratory pathogens, and infection results in minimal clinical disease. Hendra virus is a newly recognized, zoonotic disease of horses identified in Australia. The disease is rapidly fatal in horses, and close contact is necessary for disease transmission.
Secondary bacterial respiratory infections are primarily initiated by viral disease, because viral respiratory infections impair or destroy respiratory defense mechanisms. The most common bacterial organisms associated with pneumonia in horses are opportunistic bacteria originating from the resident microflora of the upper respiratory tract. These bacteria are not capable of primary invasion and require diminished pulmonary defense mechanisms to establish infection. Secondary bacterial disease may result in mucosal bacterial infections (rhinitis and tracheitis), or may produce more serious invasive disease, such as pneumonia and pleuropneumonia.

Clinical evidence of a secondary bacterial infection includes mucopurulent nasal discharge, depression, persistent fever, and abnormal lung sounds. *Streptococcus equi var zooepidemicus* is the most common opportunistic pathogen of the equine lung, although *Actinobacillus equuli*, *Bordetella bronchiseptica*, *Escherichia coli*, *Pasteurella* spp., and *Pseudomonas aeruginosa* are frequently isolated. *Strep. equi var equi*, the causative agent of strangles, is a contagious, primary bacterial pathogen of the URT, and is capable of mucosal invasion without predisposing factors. *Rhodococcus equi* is a primary pathogen of the LRT of foals less than 5 months of age, which produces pulmonary consolidation and abscessation.

Noninfectious LRT diseases are common and typically limit athletic performance. Inflammatory airway disease is characterized by excessive tracheal mucus, airway hyperreactivity, and poor exercise performance in young horses. Reactive airway disease (heaves) is triggered by exposure to organic dusts in older horses (>8 yrs) with a familial allergic predisposition. Small airways are obstructed by bronchoconstriction and excessive mucus production. The severity of clinical signs may range from exercise intolerance to dyspnea at rest. Exercise-induced pulmonary hemorrhage occurs during maximal exercise in short duration events (horseracing, barrel racing); the pathophysiology, impact on performance, and ideal treatment have been extensively studied but are poorly understood.

Diagnostic testing is usually rewarding in horses with respiratory disease. Endoscopic examination allows direct visualization of the upper respiratory tract, guttural pouches, trachea, and mainstem bronchi. Indications for endoscopic examination include URT noise, inspiratory difficulty, poor exercise performance, and nasal discharge. Radiographs of the skull are indicated to investigate facial deformity, abnormalities of the sinus (sinusitis, dental abnormalities, and sinus cyst), guttural pouch (empyema, tympany), and soft tissue structures (epiglottis, soft palate).

The two most important techniques for evaluation of lower respiratory tract secretions are transtracheal wash and bronchoalveolar lavage. Transtracheal wash is indicated to obtain secretions for bacterial and fungal culture of the lower respiratory tract. Bronchoalveolar lavage is indicated for cytologic evaluation of the lower respiratory tract in animals with diffuse, noninfectious pulmonary disease. Nasal/pharyngeal swab culture is inappropriate for investigation of pulmonary infectious disease, but is performed in horses with sus-
pected strangles infection. Thoracic radiography is most useful to identify abnormalities of the pulmonary parenchyma, mediastinum, and diaphragm. Pulmonary consolidation (pneumonia), peribronchial disease, pulmonary abscessation, interstitial disease, and mediastinal masses (neoplasia, abscess, granuloma) are most easily identified via thoracic radiography.

Thoracic ultrasound is the most appropriate technique to evaluate fluid in the pleural space, peripheral pulmonary consolidation, and peripheral pulmonary abscessation. Ultrasonographic examination can identify the volume, location, and character of pleural fluid (pleural effusion) or air (pneumothorax) within the pleural space. Additionally, ultrasound can identify fibrin tags, gas echoes (anaerobic infection), masses, and loculated fluid pockets. Ultrasound cannot penetrate air; therefore, deep pulmonary abscesses and consolidation cannot be detected via ultrasound examination and must be detected via thoracic radiography. Ultrasound examination allows the clinician to determine the most appropriate site for centesis and formulate a prognosis based on the presence of fibrin, gas echoes, and loculated pockets of fluid. Pleurocentesis is performed in animals with accumulation of fluid in the pleural space and should be performed with ultrasound guidance.

Lung biopsy and fine-needle aspirate are invasive procedures and are performed after other diagnostic procedures have been exhausted. Pulmonary neoplasia, pulmonary fibrosis, and interstitial diseases may require lung biopsy to obtain a definitive diagnosis.

Regardless of the type of respiratory disease, environmental factors and supportive care are important to aid the recovery of the horse. A dust- and ammonia-free stable environment prevents further damage to the mucociliary apparatus. Horses with respiratory disease have a variable to poor appetite; therefore, highly palatable feeds are indicated to prevent weight loss and debilitation during the treatment and recovery period. Adequate hydration will decrease the viscosity of respiratory secretions facilitating their removal from the lower respiratory tract. A comfortable and dry environment, maintained at an appropriate temperature will allow the horse to rest, and will minimize the role of the respiratory tract in thermoregulation.

This text is intended to provide diagnostic and therapeutic options for evaluation of horses with respiratory disease. The clinician’s primary goal during physical examination of the respiratory tract is to determine the origin (URT vs. LRT) and character (infectious vs. noninfectious) of the respiratory disease affecting the patient to direct diagnostic testing and therapeutic intervention.
The respiratory tract is relatively accessible to physical examination, and a thorough evaluation usually reveals constructive information that will direct subsequent diagnostic testing. Signalment is important to establish, because some respiratory conditions are age-dependent, (e.g., young—anomaly; aged—neoplasia), use-related (e.g., pleuropneumonia—long-distance transport; EIPH—short-burst, maximal exercise), or breed-predisposed (left laryngeal hemiplegia—draft, Thoroughbred). Next, it is essential to identify historical details relating to the primary respiratory complaint. The duration of clinical signs may differentiate infectious (acute to subacute) from noninfectious (chronic) respiratory disease. Traumatic injury to the respiratory tract (rib fracture, tracheal rupture) typically produces acute, severe clinical signs of respiratory distress or subcutaneous emphysema, and external signs of trauma may be obvious. Seasonal incidence of disease increases the suspicion of an allergic or environmental etiology. The rider/driver is carefully questioned regarding details of exercise performance and recovery, such as the presence and character of a respiratory noise (URT), nasal discharge (EIPH, URT infection), coughing (LRT, cardiac), and exercise tolerance. The horse’s vaccination status and history of transport will determine susceptibility and exposure to infectious disease; however, the clinician should recognize that a “current” vaccination status does not completely preclude the occurrence of the viral or bacterial respiratory pathogens in horses.

Examination of the equine respiratory tract is performed in a systematic manner, beginning with observation at rest. The rate (normal = 8 to 12 bpm) and effort of respiration is noted prior to physical examination of the horse. Tachypnea does not necessarily indicate a primary respiratory disorder; acidosis, fever, hyperthermia, anxiety, and pain are non-respiratory sources of increased respiratory rate. Conversely, many primary respiratory conditions may
present with a normal respiratory rate at rest. Normal breathing in resting horses is characterized by a unique, biphasic, “double effort” of respiration. In most species, inspiration is an active process using the diaphragm and intercostals muscles, and expiration is passively driven by elastic recoil. In horses, expiration is predominately passive, but the final phase is achieved by active abdominal effort. As a consequence, the initial phase of inspiration is passive due to recoil of the thoracic wall, and inspiration is predominately active. The additional abdominal effort of expiration in normal horses may be misinterpreted as low-grade expiratory difficulty.

Clinical signs of respiratory difficulty include flared nostrils, anxious appearance, and extended head and neck. The phase of respiration that is prolonged often indicates the portion and type (obstructive, restrictive) of the respiratory tract impairment. Inspiratory difficulty, particularly associated with inspiratory noise, indicates upper respiratory tract (URT) obstruction. The intra-airway pressures of the URT are subatmospheric during inspiration; therefore, negative pressure during inspiration exacerbates dynamic airway narrowing by drawing soft tissues into the airway. During expiration, intra-airway pressure is positive, which expands the diameter of the upper respiratory tract. A prolonged expiratory phase of respiration, accompanied by excessive abdominal effort, is consistent with obstruction of the lower respiratory tract (LRT). During expiration, intrathoracic pressure is positive, which narrows the diameter of small airways.

Bronchoconstriction and small airway inflammation exacerbate small airway narrowing during expiration, resulting in small airway collapse and air trapping during expiration. During inspiration, intrathoracic pressure is negative, and small airways are pulled open by parenchymal attachments, minimizing the effects of bronchoconstriction and inflammation. In contrast, restrictive pulmonary disease will produce rapid, shallow respiration with prolonged inspiratory and abbreviated expiratory phases of respiration. Pleural effusion, pneumothorax, and diaphragmatic hernia are examples of extrapulmonary restrictive disorders that prevent pulmonary expansion. Pulmonary fibrosis and granulomatous pneumonitis are intrapulmonary restrictive diseases producing difficult inspiration, exaggerated elastic recoil, and rapid, shallow respiration.
Most clinicians begin physical examination of the respiratory tract at the head of the horse:

- The character, frequency, and lateralization of nasal discharge may reveal the origin.
- Inspiratory noise is a hallmark sign of upper airway obstruction.
- Abnormalities of the maxillary sinus may present with unilateral nasal discharge, facial deformity, and/or epiphora.

The nares are evaluated for nostril flare, nasal discharge, and equivalent airflow. Nostril flare is observed in horses with respiratory distress originating from the upper or lower respiratory tract. As described above, a prolonged phase of inspiration, paired with an inspiratory noise is the hallmark of obstruction of the URT. Respiratory noise can be characterized as stridor, roaring, or snore. Respiratory stridor is a high-pitched inspiratory noise associated with pharyngeal obstruction due to retropharyngeal abscess, arytenoid chondritis, bilateral laryngeal paralysis, pharyngeal collapse (hyperkalemic periodic paralysis), or pharyngeal mass. Roaring is a specific term referring to the short, low-pitched respiratory noise observed during exercise in horses with left laryngeal hemiplegia (LLH). Horses with “roaring” are exercise-intolerant, but do not demonstrate respiratory noise at rest. Snoring is a low-pitched sound observed during inspiration originating from the nasal passages. Nasal septal dysplasia and nasal masses induce a snoring sound during inspiration. Dorsal displacement of the soft palate produces a characteristic guttural, low-pitched fluttering sound during exercise, and is associated with exercise intolerance. In most instances, the origin of noise from the URT can be identified by endoscopic examination.
Nasal discharge is characterized as serous, mucoid, purulent, hemorrhagic, or feed contaminated. Serous nasal discharge is observed in horses with viral respiratory infections and allergic rhinitis. Mucoid to purulent discharge indicates increasing evidence of a primary or secondary bacterial respiratory infection. Fresh blood may originate from the URT (guttural pouch mycosis, trauma) or LRT (EIPH). Brown (old hemorrhage), mucoid, malodorous discharge occurs in horses with necrotizing pneumonia and ruptured pulmonary abscess, both of which carry a poor prognosis. Copious nasal discharge (saliva) contaminated with feed indicates an inability to swallow due to mechanical (choke) or functional (neurogenic) obstruction. Low-volume mucoid nasal discharge that is discolored with feed material is consistent with low-grade dysphagia and chronic aspiration (pharyngeal dysfunction). Unilateral nasal discharge indicates that the origin of exudate is rostral to the caudal aspect of the nasal septum, and is most commonly observed in horses with unilateral sinusitis or a nasal/ethmoidal mass. The guttural pouch openings drain into the abaxial nasopharynx. Therefore, owners report nasal discharge to be predominately unilateral, with occasional observation of exudate from the contralateral nostril. Exudate originating from the guttural pouch is often observed during grazing or after exercise. Bilateral nasal discharge may originate from the LRT or bilateral URT infection. It is important to recognize that tracheal exudate is often coughed up and swallowed in horses; therefore, the presence of nasal discharge is inconsistent in horses with pneumonia. The origin of discharge can be definitively determined via endoscopic examination of the upper respiratory tract.

Equivalent airflow through the nasal passages is detected by placing the palms of your hands in front of the nares. Nasal masses (ethmoid hematoma) are the most common cause of unequal airflow. The visible portion of the nasal septum should be examined for fungal granulomas or amyloid plaques. Horner’s syndrome produces poor to absent airflow ipsilateral to the ophthalmologic signs, due to vascular dilation and mucosal edema associated with the loss of sympathetic tone to mucosal blood vessels. Airflow obstruction due to Horner’s syndrome may be performance-limiting for athletic horses.

The maxillary and frontal sinuses are evaluated by gentle percussion over the maxillary and frontal sinus areas. The maxillary is the largest sinus cavity of the horse and the most likely to be diseased. The maxillary sinus is a line drawn from the medial canthus of the eye to the nasoincissive notch, and the caudal limit is at the level of the temporomandibular joint. The lateral limit is a line drawn from the medial canthus to the nasoincissive notch. Dull, hyporesonant percussion, pain, and/or unilateral nasal discharge are
consistent with sinusitis, sinus cyst, or neoplasia. Characteristic malodorous discharge is observed in horses with sinusitis secondary to tooth root abscess. Sinus cyst and neoplasia may produce facial deformity, exophthalmia, and ipsilateral epiphora (obstruction of the nasolacrimal duct). Patency of the nasolacrimal duct can be determined by retrograde lavage of the nasolacrimal duct, or observation of fluorescein at the nasolacrimal opening after fluorescent stain is placed in the eye (Jones test).

Submandibular lymph nodes are readily palpable in horses less than 24 months of age, but are difficult to detect in adult horses. Lymphadenopathy, characterized by palpably discrete, firm, and moderately painful lymph nodes, is observed in horses with viral respiratory disease. Markedly enlarged, coalescing, painful submandibular lymph nodes should be considered suspect *Streptococcus equi* infection, until proven otherwise. The abscessed lymph nodes may have a palpable soft area, indicating impending rupture, or there may be evidence that some lymph nodes have ruptured already. Markedly enlarged, solid (homogenous via ultrasound, nonproductive aspiration) submandibular lymph nodes are observed in rare cases of multisystemic lymphoma and mycobacterial or fungal infection of the URT.

Palpation of the larynx, pharynx, and proximal trachea is a relatively insensitive measure (compared to endoscopic examination) for detection of abnormalities of these structures. Nonetheless, careful palpation of the cricoid cartilage and muscular process of the larynx may identify atrophy of the cricoarytenoideus dorsal muscle in horses with left laryngeal hemiplegia. The “slap test” may provide additional evidence of LLH. This test is performed by having the examiner palpate the left arytenoid cartilage, while an observer firmly “slaps” the withers on the right side of the horse. The anticipated response is adduction of the contralateral (left) arytenoid. In addition to LLH, fourth branchial arch defect can also be detected by laryngeal palpation. An abnormally wide gap can often be palpated between the caudal margin of the thyroid and the rostral edge of the cricoid; in the normal larynx the two structures overlap. Detection of this palpable abnormality indicates additional investigation via endoscopic and radiographic examination.

Digital compression of the proximal trachea will elicit a cough in horses with tracheal inflammation or irritation (inducible cough). Horses normally have a high cough threshold and will not respond to this procedure. Inducible or spontaneous coughing is a nonspecific response, and is observed in horses with infectious and noninfectious respiratory disease. Paroxysmal coughing is particularly common in horses with influenza. Influenza replicates within respiratory epithelial cells, denuding the mucociliary apparatus and exposing irritant receptors in the submucosa. Auscultation of the trachea will reveal a “tracheal rattle” in horses with exudate in the lumen of the trachea. Because horses expectorate exudate, it is important for the clinician to detect tracheal exudate via tracheal auscultation, rather than relying on the appearance of nasal discharge.
Physical examination of the lower respiratory tract provides diagnostic clues:

- Crackles are short, explosive sounds consistent with excessive exudate, whereas wheezes are long, musical sounds indicative of bronchoconstriction.
- Rebreathing procedure is performed to accentuate abnormal lung sounds and identify lung borders.
- Thoracic pain may manifest as rapid, shallow respiration, abducted elbows, or avoidance/objection to thoracic auscultation.

Physical examination of the lower respiratory tract relies primarily on thoracic auscultation. The caudal borders of the lung correspond to the following landmarks: 17th intercostal space (ICS) at tuber coxae, 16th ICS at the tuber ischi, 13th ICS at mid-thorax, 11th ICS at the point of the shoulder, and the 5th ICS at the point of the elbow. Horses should be examined at rest to detect abnormal lung sounds, expanded pulmonary fields, or diminished pulmonary fields. Abnormal lung sounds include crackles, wheezes, and pleural friction rubs. Crackles are short, explosive sounds produced by airflow bubbled through airway secretions (exudates), or sudden opening of a collapsed small airway. Wheezes are long, musical sounds generated by oscillation of bronchial and bronchiolar walls in patients with bronchoconstriction. Pleural friction rubs are less commonly observed in horses compared to cattle. The sound originates from rubbing of inflamed visceral and parietal pleura during the respiratory cycle. Horses typically produce effusion in response to inflammation; therefore, pleural friction rubs are observed prior to formation of significant effusion (peracute disease) or after drainage of the pleural cavity.

Expanded pulmonary fields are observed in horses with small airway ob-
structive disease (particularly heaves), due to their strategy to breathe at high lung volumes to maximize airway caliber. Diminished pulmonary borders are observed in horses with numerous conditions, including (but not limited to) pleural effusion, pneumothorax, pulmonary abscess, pulmonary consolidation, atelectasis, and diaphragmatic hernia. Detection of borboygmi during thoracic auscultation is common, and does not necessarily indicate the presence of a diaphragmatic hernia. Thoracic percussion and ultrasonographic examination is indicated in horses with diminished pulmonary borders to differentiate these conditions.

A rebreathing procedure is performed to increase the rate and depth of respiration, accentuate abnormal lung sounds, or detect altered pulmonary borders. The procedure is performed by placing a large (40 L) plastic bag over the entire muzzle of the horse, allowing the horse to rebreathe expired air, thereby increasing PCO2 and respiratory drive. In addition to auscultation during the procedure, the clinician should subjectively evaluate time necessary for recovery and induction of cough. Horses with normal respiratory function rapidly recover from a rebreathing procedure by taking 3 or 4 deep breaths. Rebreathing is contraindicated in horses with respiratory difficulty or distress.

Thoracic percussion is performed using a large spoon and a rubber reflex hammer. The clinician begins in the dorsal lung field, holding the convex surface of the spoon firmly against the surface of the horse in an intercostal space. The hammer is tapped against the concave surface of the spoon, and the clinician listens for a resonant sound generated by air-filled lung. Hyporesonance indicates an absence of air-filled lung. The examiner progresses ventrally along a single ICS, and the process is repeated at each ICS throughout all pulmonary fields. The goal is to identify a region of hyporesonance indicating a pulmonary or pleural abnormality. In horses with pleural effusion, thoracic percussion can often identify a fluid line. Thoracic percussion should not be performed in horses with pleural pain (pleurodynia). Horses with pleural pain may stand with abducted elbows and demonstrate rapid, shallow respiration. These horses may object to thoracic auscultation (bite, pin ears) or perform evasive maneuvers to attempted auscultation.

Horses with pleuropneumonia demonstrate many of the clinical signs presented above, including pleural pain, diminished pulmonary fields, and a fluid line on thoracic percussion. In addition, horses with pleural effusion (septic or nonseptic) may have a plaque of edema in the ventral pectoral region due to the weight of the fluid within the thoracic cavity. Cardiac sounds may be absent or radiating in horses with extensive pleural effusion.

Synchronous diaphragmatic flutter (“thumps”) is an uncommon respiratory pattern in which respiration is triggered by cardiac contraction. Thumps is often observed in horses with moderate to severe derangement of acid-base and serum electrolyte concentrations, specifically hypocalcemia, hypochloremia, and metabolic alkalosis. The phrenic nerve travels over the base of the right atrium. Electrolyte imbalance is hypothesized to produce irritability of