**RESPIRATORY SYSTEM EXAMINATION AND APPROACH TO EMERGENCIES**

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## NORMAL BREATHING

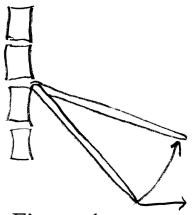
The first couple of pages of these notes describe the mechanical process of normal breathing. If you are not interested in this just skip ahead, but an understanding of how the bones and muscles of the torso work together to draw a breath and exhale it really helps your ability to figure out what's happening when things go wrong! Breathing requires an integrated system of control (CNS), movement of a bellows (ribs and associated muscle) to draw air into the lungs, exchange of gas within the lung, and a feedback system (chemoreceptors, stretch receptors) to close the loop. This portion of the lecture will present some basic elements of the bellows part of this system, providing a basis for evaluation of respiratory patterns in the dog.

### Lungs collapse, ribs spring out

Lungs are a viscoelastic tissue suspended within the pleural cavity. If the chest is opened to the atmosphere the lungs collapse until small airway occlusion prevents further loss of gas. The collapse won't stop until the lung volume is quite a bit smaller than the normal functional residual capacity (FRC) in the intact animal. The lung volume in a normal animal is maintained by support from the thoracic wall. Because there is an airtight seal within the pleural space and the lungs are coupled to the chest wall with a viscous fluid, the tendency of the lungs to collapse is countered by the tendency of the ribs to spring out. Between breaths, these opposing forces yield a pleural pressure of -5 cm $H_2O$ at the $8^{th}$-$9^{th}$ interspace in an average size dog. Breathing takes place around this equilibrium, with most of the energy of breathing expended to increase the thoracic volume above the equilibrium point during inspiration.

### Bucket handles

The ribs in the dog (and cat) are arranged in such a manner that most movement, and the inspiratory effect of that movement, is related to cranial displacement of the ribs during inspiration (Fig 1). Because of the 'bucket handle' relationship between the ribs and the spine and the caudal sweep of the ribs when the lungs are at functional residual capacity (FRC), cranial displacement of the ribs results in an increase in thoracic diameter and intrathoracic volume, a reduction in pleural pressure, and inspiration.[1] Therefore, any muscles that move the ribs forward and outward will contribute to inspiration, and any that move the ribs caudally and/or inward will contribute to exhalation. Until recently, the usual explanation for intercostal muscle function was based on the theory of Hamberger (1697-1755). He proposed that the external intercostals, because they were anchored hi( Figure 1 anial rib (closer to its axis of rotation) and lower on the caudal rib (further away) at each intercostal space, must have a net effect of raising the caudal rib, producing inspiration. Similarly, the internal intercostals, with their origins high on the caudal rib and insertions lower on the cranial rib at each space, must have a net effect of pulling the cranial rib more caudally, producing exhalation. This served, without experimental verification, as the leading explanation for intercostal function until the 1980's. Since then major advances in understanding have been obtained by the efforts of a small number of physiologist using the dog as a model. From this work comes an appreciation that the act of breathing is very complex, and the effects of muscle activation depend on many factors including location (dorsoventral and craniocaudal), muscle mass, patterns of innervation, and locomotion.[2]



### Respiratory muscle and 'abdominal breathing'

The primary muscles of inspiration include the diaphragm, levator costae, internal intercostals (ventro-cranial), and external intercostals (dorsocranial). The primary muscles of exhalation include the triangularis sterni and dorsocaudal internal intercostals. Accessory muscles of respiration are those that do not normally participate in breathing unless called upon during periods of high demand. Accessory muscles of inspiration include the scalenes and the sternomastoids. Accessory muscles of exhalation include the four abdominal muscle groups: rectus abdominis, external oblique, internal oblique, and transverses abdominis. Accessory muscles of respiration have other primary roles such as movement of the head and neck

(sternomastoids), stabilization of the spine (scalene), flexors and rotators of the trunk (rectus and obliques), and their function is synchronized with breathing when conditions demand. Contraction of the abdominal muscles increases intra-abdominal pressure and forces the diaphragm to a more cranial position within the rib cage. They can ONLY assist with exhalation, and the term “abdominal breathing” is therefore properly used to indicate vigorous exhalation.

**Location, location, location**

As depicted in figures 2 and 3, the external and internal intercostals in the cranial intercostal spaces have an inspiratory bias, and the same muscles in the caudal intercostal spaces have an expiratory bias.

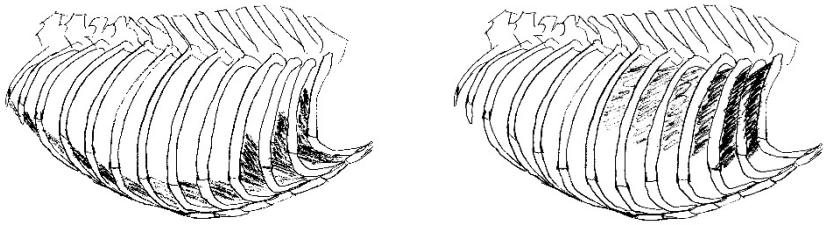


Figure 2. Inspiratory actions of the internal intercostals (left) and external intercostals (right). The shaded areas represent locations where the mechanical advantage and net respiratory effects favor inspiration at FRC. The portion of the internal intercostals between the bony rib elements of the first few spaces remain electrically silent during breathing, and the external intercostals in this region, when active, are active only during inspiration.

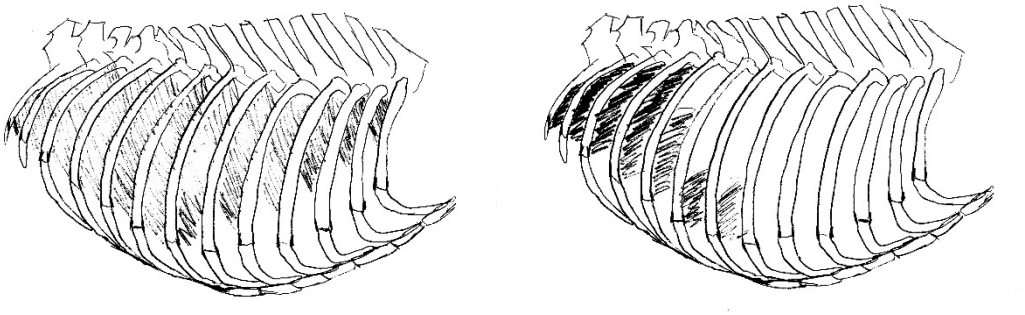
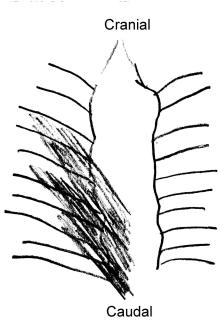


Figure 3. Expiratory actions of the internal intercostals (left) and external intercostal (right). The shaded areas represent locations where the mechanical advantage and net respiratory effects favor exhalation at FRC. This region of the internal intercostals, when active, is active only during expiration. This region of external intercostals is electrically silent during tidal breathing.

The muscle mass of the external intercostals is greatest in the dorsocranial region where they possess the greatest mechanical advantage for inspiration. The parasternal internal intercostals are a special group of internal intercostal muscles that play a greater role than the externals in inspiration during rest. These muscles also have a large mass and they possess a mechanical advantage for inspiration in every intercostal space. Their orientation is such that contraction produces outward movement of the distal rib relative to the sternum. They have a comparatively large effect on inspiration because they move the chondral aspect of the ribs outward, away from the midline. In the dog any given movement in the outward direction is roughly 4 times more effective at increasing lung volume than comparable movement in the cranial direction.[3]

The levator costae and parasternal and external intercostals are active during inspiration. Levator costae originate on the transverse process of the thoracic vertebrae and extend caudally to insert on the dorsal-cranial aspect of the rib so when they contract they rotate the ribs cranially. This muscle works in concert with the parasternal intercostals to provide most of the rostral rib displacement during inspiration. The levator costae and parasternal intercostals appear to function exclusively for breathing. In contrast, the function of the interosseous portions of the intercostal muscles may be given over to stabilization of the torso

during activity. For example, in trotting dogs the activity of the intercostal muscles becomes synchronized with locomotion and drifts relative to the phase of respiration.[4] Therefore, in active dogs the interosseous portions of the intercostal muscles are more important for stabilizing the trunk for locomotion than for any direct role in breathing. However, their action stabilizes the rib cage, which is essential for optimal function of the other respiratory muscles.




**Exhalation: never passive in dogs**

The triangularis sterni and internal intercostals are active during expiration. In particular, the triangularis sterni muscle is active even during quiet breathing, and is independent of both body position and intercostal activity.[5] Figure 4 illustrates the orientation of this muscle from a view inside the thorax. The muscle extends from the caudal half of the deep aspect of the sternum to the chondral (parasternal) portion of ribs 2-7. When it contracts, the triangularis sterni displaces the ribs caudally and the sternum cranially, positioning the ribs for a greater mechanical advantage and lengthening the parasternal intercostal muscles to enhance their function during the next breath.

**Diaphragm**

The diaphragm is now considered to be a combination of two functionally discrete muscles, the costal and crural diaphragm. The muscles separate the abdominal cavity from the thoracic, and diaphragm function is essential for effective ventilation. Contraction of the diaphragm in inspiration causes it to flatten and move the liver and abdominal viscera caudally into the abdominal cavity. However, because it is anchored to the chodral arch and caudal ribs, contraction of the diaphragm pulls this region of the rib cage rostrally. When working in concert with the parasternal intercostals and levator muscles the diaphragm assists in the craniolateral rib displacement of inspiration to increase thoracic girth and volume. The effect on airway pressure of this combined effort is significantly greater than the sum of the individual contributions from diaphragm and rib muscles.[6]

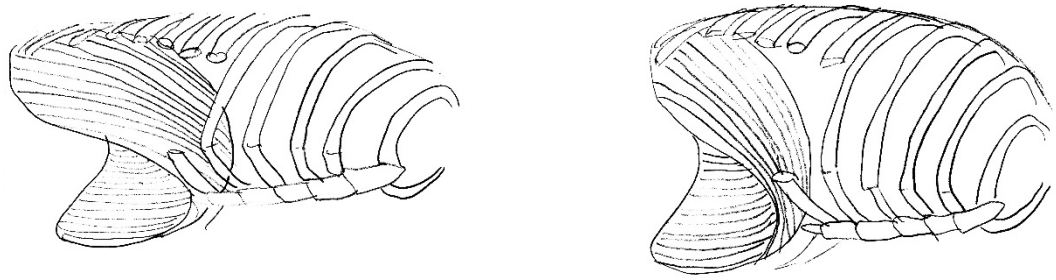


Figure 5. Relationship between the diaphragm and ribs during exhalation (left) and inspiration (right). Essential aspects include the spherical shape of the dome region (which greatly enhances the muscle's mechanical advantage) and the anchor to the chondral arch.

**Don't confuse fast breathing with panting**

Panting is a thermoregulatory maneuver and is the most important method of evaporative cooling in dogs exposed to heat or exercise. *It has minimal impact on gas exchange.* When the skin (environmental trigger) or core temperature (exercise) temperature rise, respiration shifts to a pattern optimized to remove heat from the body. The diaphragm and other muscles of respiration generate a rhythmic motion that cycles at a frequency of 3.5-5 Hz. The precise frequency is closely related to the resonant frequency for the individual, thus body conformation and the unique characteristics of that individual's respiratory system determine the rate. The oropharynx and larynx move in synchrony to 'valve' the system and provide an efficient unidirectional flow of air over the evaporative surface of the mouth.[7] When the need is minimal, dogs

will pant through the nose. As need for heat loss increases, dogs oscillate between two patterns: a) inhalation through the nose, exhalation through the nose and mouth and b) inhalation through the nose and mouth, exhalation through the nose and mouth. Lingual blood flow increases 6 fold, and the tongue, wet from increased salivation, hangs out of the mouth as it lengthens to increase its surface area.[8]

Panting requires a dramatic increase in minute ventilation, but blood gases do not change appreciably (although in severe heat stress panting will cause a significant respiratory alkalosis for a short period). This is possible because most of the increase in ventilation that occurs during panting is dead space ventilation, and the inspiratory volume is only slightly larger than the anatomical dead space of the airways. Ventilation is maintained owing to the principles of jet ventilation: individual molecules of gas enter the trachea at very high speeds. Mixing of gas in distal airways is enhanced by asynchronous contractions of the crural and costal segments of the diaphragm, and there is net displacement of carbon dioxide out and oxygen in.[9]

## ABNORMAL BREATHING

It is suspected that mammals generally adopt a respiratory pattern that meets their metabolic needs with the least metabolic energy cost. Because of the phenotypic diversity in healthy dogs and cats, how this is achieved varies dramatically between patients - what works best for a Borzoi may not work at all for a Pug. In addition to these species and breed differences, respiratory diseases cause characteristic changes in respiratory frequency, tidal volume, airflow velocity, and patterns of muscle activation that can provide clues for the clinician to help localize the cause of abnormal respiratory function. This part of the lecture will present the basic elements of how respiratory patterns change with disease states and emphasize important physical examination findings.

**Summary of signs to look for:**

1. **Working hard to breath, behavioral and facial features of distress:** This represent ***dyspnea*** – a true medical emergency ***that must be relieved within minutes!!***
2. **Working hard to breath, no behavioral signs of distress, interested in environment:** This usually represents a successful adaptation to respiratory disease, not as severe of an emergency. Note that puppies may die of respiratory failure without showing evidence of dyspnea, but that would be rare in any alert cat or adult dog.
3. **Cyanosis (very blue + normal CRT):** Hypoxemia from airway obstruction or pleural space disease; cardiovascular function usually normal.
4. **Pale grey, slow CRT:** Hypoxemia from heart failure
5. **Audible long-duration noise at mouth:** Large airway obstruction
6. **Long inspirations and exhalation:** Constant large airway obstructions
7. **Long inspiration, normal exhalation:** Dynamic obstructions such as laryngeal paralysis
8. **Long/forceful exhalation +/- cough:** Intrathoracic airway collapse or obstruction
9. **Contraction of abdomen:** Occurs only in exhalation, represents recruitment of those accessory muscles of exhalation, and is called abdominal breathing. Seen when there is resistance to exhalation (e.g., small airway collapse), especially when the frequency (RR) is high.
10. **Vigorous, large excursions of chest and abdomen:** Rule out pleural space disease (especially if cyanosis is present!)
11. **Fast, shallow breathing:** Think stiff lungs: edema, fibrosis. Inflammation, disseminated neoplasia.
12. **Refusal to lie lateral, head extended, forelimbs abducted, head bobbing:** Seen with stiff lungs and large effort.

***Dyspnea* vs increased respiratory effort**

The term dyspnea refers to the *experience* of distress secondary to respiratory disease. In man it is most often associated with increased resistance to airflow but may also be caused by anything leading to clinically significant hypoxemia or hypercapnia (especially if acute). Like pain, its presence is inferred from facial expressions and behavioral signs of distress. Dyspnea is a true medical emergency; our goal is to relieve it within minutes of presentation. In contrast, the terms *labored breathing* or *increased respiratory effort* refer to the physical manifestations of increased work of breathing. This may be objectively measured,

but most of the time is inferred from subjective appraisal of physical signs. Whereas an animal with dyspnea is having a crisis, an animal with labored breathing may be very well adapted to its condition and be free from distress. Where a given patient lies on this spectrum must be inferred from their behavior. In general, as an animal approaches dyspnea it must focus more and more conscious effort on the act of breathing. Animals that are interactive and engaged with their environment usually still have significant physiological reserves; those that appear withdrawn and focused on the act of breathing may be close to the edge of respiratory arrest. A notable exception to this generalization is the puppy. Juvenile dogs will often remain active, alert, and hungry even with advanced respiratory failure and may go straight to the brink of death before showing compelling behavioral signs of distress.

**Fixed airway obstruction**

*Neoplasia, constrictions, compression from abscesses or trauma:* A 'fixed' obstruction refers to one that does not change appreciably with the phase of respiration. As the airway lumen narrows with advancing disease, the animal will adopt a prolonged inspiration and exhalation that is characteristic of fixed obstructions. Respiratory frequency falls as the inspiratory phase becomes noticeably prolonged. As the problem becomes more severe, more energy is expended on both inspiration and exhalation. The intercostal muscles 'suck in' from markedly negative pleural pressures created by vigorous diaphragm contraction, and the animal may actively exhale, first by contracting the intercostal muscles alone, later by a combination of intercostals and abdominal muscles. The term 'abdominal breathing', referring to active contraction of the abdominal wall, can ONLY assist with exhalation and is a response to air trapping that slows exhalation to unacceptable rates.

Neoplasia and constrictions from scarring are typically insidious in onset, initially causing clinical signs only with exercise and usually allowing plenty of time for adaptive strengthening of the respiratory muscles. If acute, as for example from cervical swelling from trauma, the animal may have the same physical features as aspiration of a foreign body, including dyspnea and cyanosis.

*Foreign body:* This disorder is characterized by an acute onset of respiratory difficulty, often accompanied by coughing/retching and behavioral signs of dyspnea. Marked cyanosis may be present as hypoxemia occurs before there is any adaptive shift in hemoglobin oxygen affinity and cardiovascular responses are vigorous, with good perfusion of skin and mucus membranes. In the author's experience, inspiration is more severely affected than expiration but this would be expected to vary with location of the obstruction. Most obstructions are at the level of the larynx or tracheal bifurcation.

*Brachycephalic syndrome:* This syndrome is classically characterized as restriction of the upper airway secondary to excessive or extreme phenotypic expression of the abnormalities selectively bred for in some chondrodysplastic breeds. Although some anatomic features of the syndrome produce a dynamic obstruction, most individuals have at least some component of fixed obstruction. Any combination of stenotic nares, stenotic nasal cavity, elongated soft palate, myopathy of the pharyngeal opening musculature, laryngeal dysplasia, everted laryngeal saccules, and trachea hypoplasia are possible.[10-12] Collapse of the cervical and/or intrathoracic portion of the trachea is sometimes seen. Some dogs have concurrent abnormalities of respiratory drive[13], and some acquire rib fractures and/or hiatal hernia.[14] The breathing pattern is characterized by prolonged inspiration, and (usually) comparatively easy exhalation.[15] Affected dogs routinely develop stertorous sounds with the slightest provocation. In spite of evidently high resistance to air flow, affected animals are generally not focused on breathing and relatively free from distress, even if their arterial $pO_2$ is very low. This finding fits with the observation in many species that chronic hypercapnia and hypoxemia invoke adaptive responses that resets chemoreceptor response thresholds to relatively high values of $pCO_2$ and low values of $pO_2$.

**Dynamic airway obstruction**

*Laryngeal paralysis:* Although some dogs with idiopathic laryngeal paralysis have features of a fixed airway obstruction, this is probably the best example of a dynamic extrathoracic obstruction.[16] In this disorder, deterioration of the recurrent laryngeal nerves results in a loss of innervation of the abductor muscles of the larynx. It may occur as an isolated neuromuscular abnormality or be part of a polyneuropathy that may include swallowing disorders and/or megaesophagus. Most often, it is insidious in onset and many owners may recognize that clinical signs have been present for months only in retrospect after a crisis. During inspiration, the larynx is prone to collapsing into the lumen secondary to the subatmospheric pressure that creates a Venturi effect as described in Bernoulli's principle. Vigorous inspiration becomes prolonged and either stertorous (if the laryngeal structures vibrate) or attended by a high pitched squeak (if obstruction

is nearly complete). Many dogs seem to be able to prevent this for long periods by adopting an inspiratory pattern that is slow in frequency with a relatively prolonged inspiration and large tidal volume. They can thus avoid symptomatic obstruction unless forced to increase ventilation by the demands of activity or environment (by inducing panting from heat exposure). When signs occur in these dogs they often come on dramatically as the dog worsens the obstruction with rapidly escalating inspiratory efforts that in turn worsen the obstruction. Many present with cyanosis (from acute worsening of the obstruction) and hyperthermia (from the heavy muscular effort).

*Tracheal collapse, bronchial collapse*: These terms describe the obstruction of a large airway due to chondromalacia, compression, or in the case of the trachea, a redundant dorsal ligament. Clinical signs will depend upon which portion of the airway system is affected. Extra-thoracic tracheal collapse will tend to occur during inspiration when intraluminal pressure is subatmospheric and the dorsal ligament is sucked into the tracheal lumen. Intrathoracic collapse is more likely to occur during exhalation when pleural pressures approach (or exceed) intraluminal airway pressure. Either event may stimulate coughing via stimulation of submucosal nociceptors, but intrathoracic collapse usually causes more severe problems than cervical tracheal collapse. Animals with cervical tracheal collapse are often asymptomatic except with activity; once they begin collapsing the airway they may quickly transition to a pattern characterized by vigorous attempts at inspiration with loud stertor or sounds of 'choking off'. Animals with intrathoracic airway collapse may have no signs other than persistent cough. If severe, they may begin trapping air at relatively high lung volumes and be forced to recruit accessory abdominal muscles of expiration in an effort to forcibly 'burp' air past obstructed airways.

*Feline asthma*: If bronchospasm is severe, this syndrome has all the features of a fixed airway obstruction. In most patients, however, it is likely that restriction to airflow is worse on exhalation as small airway diameter falls with declining lung volume. Wheezes are commonly auscultated, but auscultatory evidence of large airway collapse is typically lacking.

**Restrictive lung disease**

Restrictive diseases are characterized by pulmonary changes that reduce lung compliance and make the lungs more difficult to inflate. At the same time, the underlying disorder typically compromises the oxygenating capacity of the lung. Arterial hypoxemia and lung inflammation or edema are direct respiratory stimulants and provoke an increase in desired minute alveolar ventilation. Thus, when severe, the animal is faced with the need to increase ventilation with lungs that are increasingly stiff and difficult to inflate. The archetypical pattern that results from this combination is breathing at a relatively fast rate and small tidal volume.

Clinically, the animal develops an increased frequency with rapid shallow inspirations. As inspiration becomes more difficult (or fatigue develops with acute illness), one of the first behavioral changes is a reluctance to lie in lateral recumbency. As difficulties progress, the dog may have trouble remaining in sternal recumbency for prolonged periods and position changes become more frequent. Finally it may be difficult to lie down for any significant period and the dog will prefer to sit or stand, even in the face of exhaustion. During this progression, the dog begins to extend the head and neck and recruit accessory muscles of inspiration, including the scalenes and the sternomastoids[17;18]. The forelimbs are abducted as breathing difficulty becomes greater. The head may bob up and down with respiratory effort. If the elastic recoil of the lung is sufficient, exhalation may be completely passive. If there is excessive air trapping from airway collapse, or if passive recoil of the lung does not provide sufficient emptying the dog may exhale actively (contraction of intercostal muscles) and may recruit accessory muscles of expiration (abdominal muscles).

In contrast to what is seen with airway obstruction or pleural space disease, hypoxemia from lung disease typically causes more pallor than cyanosis, yielding a pale-to-grey color of the mucus membranes. If the underlying cause is rapidly progressing the animal may develop fatigue of the respiratory muscles quickly and go on to develop hypoventilation (respiratory failure).

*Pulmonary edema/pneumonia:* Any increase in the water content of the lung tissue will reduce its compliance by increasing the opening pressure required to inflate pulmonary exchange units. As edema develops, regions of the lung will lose ventilation and venous admixture increases. Respiratory frequency increases, in some cases associated with stimulation of pulmonary C fibers (juxtacapillary receptors) from the increased fluid pressure of the interstitium, and in some from hypoxic stimulation of chemoreceptors. Because the lungs are relatively stiff, the prototypical pattern will be relatively shallow breaths at a relatively high frequency. As these disorders tend to develop rapidly, respiratory fatigue may develop quickly and hasten death in the absence of ventilatory support.

*Idiopathic pulmonary fibrosis:* This progressive disorder of cats and dogs results in loss of function (hypoxemia) and compliance, and is may be accompanied by cough. Compared to pulmonary edema, this disorder progresses relatively slowly and there is ample time for training of the respiratory muscles and development of fatigue resistance. Affected animals are often not in respiratory distress until functional loss is severe.

**Pleural space disease**

The thoracic girth is normally maintained by the opposing forces of lung (which wants to collapse) and rib cage (which wants to spring outward). Separation of the two with a layer of air or fluid in the pleural space produces a reduction in lung volume and an increase in thoracic girth. The effects of pneumothorax on breathing has been studied more than the effects of pleural effusion, but it appears that dynamic lung compliance, inspiratory duration and tidal volume decrease significantly with either disorder.[19;20] As lung volume falls exchange units are lost as the reduction in volume becomes sufficient to favor small airway collapse, leading to hypoxemia due to venous admixture and if severe, hypoventilation. At the same time this is occurring the total intrathoracic volume is growing, flattening the diaphragm (placing it at a mechanical disadvantage) and shifting external intercostal muscle function towards exhalation.

As the disorder progresses, inspiratory efforts increase and accessory muscles of inspiration may be recruited. The diaphragm will contract maximally, maintaining good excursion in spite of its flattened profile in exhalation. Pneumothorax directly stimulates phrenic nerve activity in a manner independent of the effects on gas exchange. [19]

**Diaphragmatic weakness or paralysis**

Diaphragm paralysis may be caused by injury to the phrenic nerve(s), degeneration of the phrenic nerves, or severe motor unit diseases such as botulism. If the condition is isolated to phrenic nerve involvement, dogs compensate by increasing the activity of the intercostal, levator costae and parasternal muscles. Although this may be a conscious effort in response to chemical stimulation, contraction of the normal diaphragm stimulates receptors whose output inhibits efferent activity to the intercostal and levator costae muscles. Clinically, the dog has an increased frequency, marked inspiratory excursion of the anterior ½ of the thorax, and sometimes paradoxical movement of the abdomen. The latter is characterized by a failure of the abdomen to maintain its girth during inspiration or a frank reduction of girth if the diaphragm and abdominal viscera are sucked into the thorax by the vigorous inspiratory efforts happening up front.

**Intercostal paralysis**

Weakness or paralysis of the intercostal muscles may be seen as part of a generalized myopathy, motor unit disease, and spinal cord injury due to trauma or neoplasia. In this patient, the thoracic wall may fail to maintain its normal position during tidal breathing and move paradoxically during diaphragm contraction. Because these dogs are typically paralyzed, if their lungs are otherwise normal they usually have adequate respiratory function with diaphragm movement alone.

**Disorders of the bony structures forming the thoracic bellows**

Several disorders affect the bellows mechanism of respiration, including rib fractures and breakdown of the chondral arch. Rib fractures are painful and may limit tidal volume, prompting a compensatory increase in respiratory frequency. In addition, fractured ribs disrupt the action of the intercostal muscles and may produce paradoxical motions of regions of chest wall that further compromise ventilation.

Some animals with stiff lungs or chronic airway disease will eventually suffer collapse of the costachondral arch, possibly secondary to excessive forces generated by a hypertrophied diaphragm. When this happens, the diaphragm is shortened, losing its dome shape and most of its mechanical advantage. When viewed with fluoroscopy, contraction of the diaphragm results in pulling the caudal aspect of the sternum closer to the spine, instead of forcing the caudal ribs to move in a cranial direction and increasing thoracic volume. Because these animals typically have chronic lung or airway disease and already suffer significant functional loss, their condition is often made acutely worse when the contribution of the diaphragm to inspiration is compromised.

## AUSCULTATION

**Using a stethoscope**

A comparison of 6 different stethoscope models in 1992 examined the transfer function of the

instruments for sound frequencies between 37.5 and 1000 HZ and demonstrated that, in most cases, low frequency sound (37.5-112.5 Hz) was amplified by bells and attenuated by diaphragms.[21] However, there were only small differences in sound transmission between stethoscopes, and at least one model had good similarity between bell and diaphragm. More recently, another group examined the performance of the bell and diaphragm of a Littmann Classic II SE stethoscope and found that sound transmission in the frequency range of 20 – 400 Hz (where most lung and cardiac sounds are found) was superior for the diaphragm.[22] They propose that it is the capacity for *selective hearing* that accounts for persistent recommendations to use the bell of the stethoscope to listen to breath sounds, in spite of this evidence that the diaphragm provides superior transmission of low frequency sounds. Selective hearing is the ability to mentally tune out extraneous noise and to actively enhance one's sensitivity ("*training the ear"*) to the frequency range of interest. This enhanced sensitivity is likely a function of one's ability to actively tune the cochlea to maximize it's response to a certain range of frequencies.[23] It has often been said that the skill of selective hearing through a stethoscope takes 3-5 years to master. Other factors that are likely important for optimal performance of a particular stethoscope are its physical condition and how well the ear pieces fit the listener's ear canals.

**Normal sounds**

All normal lung sounds are generated by turbulent airflow in the trachea and larger airways, as flow in small airways is slow and laminar and generates no audible sound. The origin of canine breath sounds has been investigated using a hollow airway cast instrumented with an intraluminal microphone introduced into different-sized airways ranging from 2 – 19 mm diameter and subjected to a range of airflows.[24] This work supports an intrapulmonic source of breath sounds: most inspiratory sound is produced within lobar and subsegmental airways (down to diameters of 5 – 8 mm), and most expiratory sound originates in the trachea and large bronchi.

The sound heard at the chest wall is the final product of its generation within the airways and subsequent modification as it passes through the structures of the chest. As sound travels from its source in the larger airways, it passes through lung parenchyma, the pulmonary pleura, the pleural space, the chest wall pleura, the muscle, bone and skin of the chest wall. The sound must then bridge the gap between skin and stethoscope – a gap usually filled with hair in veterinary patients. The sound is further modified acoustically by the stethoscope and how it fits the wearer's ear canals.

Normal lung and chest wall act as a low-pass filter. As sound travels across the parenchyma, it must traverse hundreds-to-thousands of air:tissue interfaces as it crosses pulmonary exchange units. Whenever energy waves (such as sound or light) pass through an interface between different media, a portion of the energy will be reflected back. In the case of sound, the higher-frequency components are reflected more efficiently. Thus, while sound recorded directly over the trachea remain intense over a broad frequency range (75-1000 Hz), breath sounds over the chest wall are lower in amplitude (quieter) and lower in pitch, with most sound energy contained in frequencies < 400 Hz. The amplitude and frequency range of normal lung sounds varies with respiratory pattern (airflow volume and velocity) and age.[25] Large breaths, high velocity airflow from panting, and increasing age all favor increased lung sound amplitude and frequency, and these must be taken into account when interpreting auscultation of a patient.

**Abnormal sounds**

Lung sounds may be increased or decreased in disease states. Decreased breath sounds may also be normal in small animals breathing quietly (as in the cat!). Pathological causes of reduced breath sounds include respiratory weakness (small breaths) and pleural space disease. The presence of air or fluid within the pleural space uncouples the chest wall from the lung acoustically and can greatly attenuate sound. Pathological causes of increased lung sounds include any disorder that prompts compensatory increases in airflow velocity (increased tidal volume, panting), causes loss of exchange units (reducing the number of air:tissue interfaces), or involves infiltration of the alveolar septa by edema or cells (pneumonia, neoplasia). Loss of exchange units or thickening of the water-dense medium of the lung allows more of high frequency sound to pass through to the surface and lung sounds become both louder (larger amplitude) and 'harsher' (more high frequency components) in quality. The extreme example is seen with complete consolidation of lung tissue around a patent lobar bronchus. In this case, the lung sounds heard over this nonventilated region of lung are loud and may approximate those heard directly over the trachea, and this has sometimes been called "bronchial breathing". If the bronchus is obliterated, the lobe loses its acoustical connection with the airways and lung sounds are diminished over that region. When measured electronically, increased lung sounds are a sensitive indicator of increased pulmonary water content in dogs with experimentally induced pulmonary edema[26]. This change occurs long before the development of adventitial sounds such as

crackles, and skillful clinicians can detect this change before radiographic changes are evident.

Adventitial sounds may be discontinuous (crackles) or continuous (wheezes and rhonchi). Crackles may be further divided into *coarse crackles* and *fine crackles.* Coarse crackles are related to mobilization of secretions in the large upper airways and are audible at the mouth. This sound is easily heard without a stethoscope, and referred sounds heard during auscultation may overwhelm other lung sounds. Fine crackles have nothing to do with the presence of intraluminal fluid. They are caused by the explosive opening of small airways and tend to be most intense late in inspiration and it the dependent region of lung; they are difficult to hear at the mouth. Their presence often indicates small airway closure due to the accumulation of cells and/or fluid in the pulmonary interstitium. This closure is particularly prominent during exhalation, when the vacuum applied by the chest wall and diaphragm is minimal and lung volume is small. During inspiration, as the lung expands, the pleural and interstitial pressure become more negative, and when sufficient to overcome the interstitial fluid pressure the airway snaps back open. This produces a short (< 20 msec), high-frequency sound, and when hundreds of airways in a single region of lung open simultaneously, they produce a sound similar to that produced by pulling Velcro$^{R}$ apart. The distribution and symmetry of fine crackles provides information about the underlying disease process. For example, most animals with cardiogenic pulmonary edema may have bilateral late-inspiratory fine crackles.

*Wheezes* are longer duration (>80 msec) continuous sounds with a musical (tonal) quality in the range of 100-1000 Hz, and in most instances are related to fluttering of the wall of an airway. Generation of a wheeze commonly indicates the presence of a flow-restricted zone (partial obstruction, bronchospasm) within an airway with downstream flattening of the tube, although in some instances a partial obstruction with vortex creation and vibration of a round airway may be the cause. Wheezes often indicate small airway disease with airway exudates or secretion. Polyphonic wheezing indicates multiple airway involvement. Wheezes may be heard at the mouth.

*Rhonchi* are repetitions of complex sound waves that have a tonal, snore-like quality typically < 300 Hz and lasting at least 100 msec and are produced by rupture of fluid films formed by airway secretions, and vibration/collapse of large airways. They are audible at the mouth.

Properly conducted, lung auscultation is a valuable component of the physical examination in critically ill patients. While I believe that it is a relatively low-yield test in apparently healthy dogs and cats, the real value of routine lung auscultation of asymptomatic animals is the ongoing training of the listener's ear. When the skilled auscultator turns his or her attention to the emergency or critically ill patient, the value of this diagnostic test is manifest.

**Thoracic ultrasound**

With a modest amount of training, thoracic ultrasound examination may be used to screen for pleural space disease, pericardial effusion, and pulmonary infiltrates. During the presentation I will demonstrate relevant findings with video; a comprehensive review of the technique by Lisciandro was published in 2011.[27]

## APPROACH TO RESPIRATORY EMERGENCIES

Compromised ventilation is an emergency that may require rapid intervention to keep the animal alive, sometimes in circumstances where just a little extra stress from handling and manipulation will push the patient "over the edge" and result in death. Regardless of the cause of trouble the critical first step in patient management is to evaluate the severity of respiratory compromise and decide how much "respiratory reserve" the animal has before it decompensates into an immediately life-threatening crisis. Localization of the problem (upper airway obstruction, intrathoracic large airway obstruction, small airway disease, parenchymal lung disease, pleural space disease, fractures/displacement of ribs, CNS disorders, neuromuscular disease, and increased ventilation requirements driven by non-respiratory disorders) is the subject of another talk, and this should be accomplished rapidly and based solely on evaluation of the respiratory pattern, auscultation, and possibly thoracic focused ultrasound evaluation (TFAST). Concurrent with this evaluation is the more difficult interpretation of muscular strength and behavioral evidence of distress. We constantly misuse the term *dyspnea* to indicate "difficulty breathing", when in reality the term means an adverse, distressing emotional response to difficulty breathing (for a veterinary review on the topic, see Mellema 2008[28]). This distinction is critically important, because by the time most dogs and cats show compelling behavioral evidence of dyspnea they may be very near death and treatment has to be prompt and aggressive. In contrast, an alert animal that is laboring to breathe but not showing any behavioral signs of dyspnea may have much more reserve and tolerate a

less aggressive approach to diagnosis and treatment. Things to look for in animals with labored breathing to decide if dyspnea is present include:

- Level of consciousness: If the animal is alert and interested in its surroundings it is less likely to be experiencing dyspnea. Obtunded, stuporous, or comatose animals may be in a crisis without compelling behavioral evidence of distress. Note that marked hypercapnea seen in animals with OK oxygenation (usually victims of neuromuscular disease) causes sedation and blunts behavioral features of dyspnea. Hypercapnea in animals that can't oxygenate well is usually mild because the animal dies of the hypoxemia before ventilation (exchange of $CO_2$) becomes very compromised.
- Interest in the environment: Animals developing respiratory failure have to focus more and more effort and attention on the act of breathing; they "pull within" and remove themselves from interactions with the environment.
- Facial expressions: The subjective appearance of widened palpebral fissures, failure to make eye contact/focus, facial grimace, and other features may indicate dyspnea.
- Specific features of labored breathing: Signs of maximal respiratory effort should bias you to suspect dyspnea is present. In cases where muscular fatigue prevents exaggerated respiratory movement, things to look for include flaring of the nasal alae during inspiration, determination to sit or stand despite appearing exhausted, recruitment of accessory muscle of inspiration (extension of head and neck to preload the strap muscles of the ventral neck, abduction of the forelimbs to preload the pectorals) and recruitment of accessory muscles of exhalation (abdominal breathing = contraction of the abdomen to exhale more rapidly).

If there is significant evidence of dyspnea that is not quickly and definitively corrected with administration of oxygen or thoracocentesis (if there is pleural space disease) should be addressed by crash induction and intubation to take over the work of breathing. Useful induction agents for hypoxic and/or hypercapnic animals include:

Dog:

- No heart disease: ketamine 2-5 mg/kg and midazolam (or diazepam) .25 - .5 mg/kg IV
- Heart failure suspected: morphine 1-2 mg/kg +/- midazolam (or diazepam) .25 - .5 mg/kg IV

Cat:

- No heart disease: ketamine 2-5 mg/kg and midazolam (or diazepam) .25 - .5 mg/kg IV
- Heart failure suspected: buprenorphine 10-20 mcg/kg +/- midazolam (or diazepam) .25 - .5 mg/kg IV

For any of these combinations the more depressed the patient, the less drug you may need. If the animal is very vigorous I routinely add the paralytic agent atracurium 0.1 mg/kg IV. Newer paralytics work even better but atracurium is comparatively cheap and has a long shelf life when refrigerated.

Be prepared to intubate and provide 100% oxygen *immediately!* If the animal has an upper airway obstruction, this will be your opportunity to remove it (if due to foreign body) or bypass it with an endotracheal tube (if due to an anatomical issue). If the animal has pulmonary edema, frothy fluid (perhaps blood tinged) will pour from the airway upon cessation of spontaneous breathing. Don't waste time suctioning this – just tip the patient for a moment to let the worst of it drain out, then connect the breathing system and begin ventilation immediately. Despite appearances, airway froth will not severely compromise your ability to ventilate the patient, and you may do so with only occasional interruptions to briefly disconnect the circuit and pour out accumulating fluid.

If the animal has pleural space disease you can now proceed to remove the air or fluid without concern that handling the patient for the procedure will kill it. If the animal has cardiogenic pulmonary edema, just an hour or two of manual or mechanical ventilation buys you some precious time to treat with diuretics (e.g., furosemide 2 mg/kg IV followed by continuous infusion of 0.25 - 0.5 mg/kg/hour) and (if possible) oral pimobendan (1-2 mg/kg) as a slurry via a stomach tube and (if available) intravenous afterload

reducers like nitroprusside (1-10 mcg/kg/min). This strategy can quickly salvage a patient that would have otherwise died; these treatments are far less likely to help an animal that has already developed respiratory arrest. Even if the animal has an underlying cause is not easily reversed (for example, aspiration injury), taking control of its airway and breathing allows you to be much more aggressive with diagnostics (airway wash, radiographs, etc.) and arrive at a diagnosis much quicker than possible in the awake, distressed patient.

If marked dyspnea is not present and the animal appears to have good 'respiratory reserve', intubation may not be necessary and you have time to do some more directed supportive measures that will be the subject of this talk (and the instructions mentioned above). These include:

- Oxygen administration: by the least stressful means possible; this includes blow-by, face mask, oxygen hood/tent, and oxygen cage (a commercial one, not one of those Plexiglas doors on a regular cage).
- Nasal oxygen administration: If done correctly, this will be accomplished with a catheter tip located over the soft palate, not within the nasal cavity. On occasion, the tip of the catheter is positioned within the lumen of the trachea.
- Therapeutic thoracocentesis: By a method that does not risk lacerating the lung
- Relief of obstruction: By temporary tracheostomy
- Improve the strength of the muscles of breathing: by fixing hypokalemia and other electrolyte disorders; sometimes by administration of aminophylline or other agents
- Reduce demand: by sedation, nursing care, and treatment of underlying non-respiratory disorders.

Specific techniques of nasal oxygen administration and thoracocentesis are described in detail in pictorial handouts available at http://www.ncstatevets.org/ECCresources/.

**References:**


1 Margulies SS, Rodarte JR, Hoffman EA. Geometry and kinematics of dog ribs. J Appl Physiol 1989;67:707-12.
2 DeTroyer A., Kirkwood PA, Wilson TA. Respiratory action of the intercostal muscles. Physiol Rev 2005 Apr;85(2):717-56.
3 DeTroyer A., Wilson TA. The canine parasternal and external intercostal muscles drive the ribs differently. J Physiol 2000 Mar 15;523 Pt 3:799-806.
4 Carrier DR. Function of the intercostal muscles in trotting dogs: ventilation or locomotion? J Exp Biol 1996 Jul;199(Pt 7):1455-65.
5 DeTroyer A, Ninane V. Triangularis sterni: a primary muscle of breathing in the dog. J Appl Physiol 1986 Jan 1;60(1):14-21.
6 DiMarco AF, Supinski GS, Budzinska K. Inspiratory muscle interaction in the generation of changes in airway pressure. J Appl Physiol 1989 Jun;66(6):2573-8.
7 Goldberg MB, Langman VA, Taylor CR. Panting in dogs: paths of air flow in response to heat and exercise. Respir Physiol 1981 Mar;43(3):327-38.
8 Ronert H, Pleschka K. Lingual blood flow and its hypothalamic control in the dog during panting. Pflugers Arch 1976 Nov 30;367(1):25-31.
9 Easton PA, Abe T, Young RN, Smith J, Guerraty A, Grassino AE. Costal and crural diaphragm function during panting in awake canine. J Appl Physiol 1994 Oct;77(4):1983-90.
10 Schotland HM, Insko EK, Panckeri KA, Leigh JS, Pack AI, Hendricks JC. Quantitative magnetic resonance imaging of upper airways musculature in an animal model of sleep apnea. J Appl Physiol 1996 Sep;81(3):1339-46.
11 Petrof BJ, Pack AI, Kelly AM, Eby J, Hendricks JC. Pharyngeal myopathy of loaded upper airway in dogs with sleep apnea. J Appl Physiol 1994 Apr;76(4):1746-52.
12 Hendricks JC, Kovalski RJ, Kline LR. Phasic respiratory muscle patterns and sleep-disordered breathing during rapid eye movement sleep in the English bulldog. Am Rev Respir Dis 1991 Nov;144(5):1112-20.
13 Hendricks JC, Kline LR, Kovalski RJ, O'Brien JA, Morrison AR, Pack AI. The English bulldog: a

natural model of sleep-disordered breathing. J Appl Physiol 1987 Oct;63(4):1344-50.
14 Hardie EM, Ramirez O, III, Clary EM, Kornegay JN, Correa MT, Feimster RA, et al. Abnormalities of the thoracic bellows: stress fractures of the ribs and hiatal hernia. J Vet Intern Med 1998 Jul;12(4):279-87.
15 Amis TC, Kurpershoek C. Pattern of breathing in brachycephalic dogs. Am J Vet Res 1986 Oct;47(10):2200-4.
16 Amis TC, Smith MM, Gaber CE, Kurpershoek C. Upper airway obstruction in canine laryngeal paralysis. Am J Vet Res 1986 May;47(5):1007-10.
17 Farkas GA, Rochester DF. Contractile characteristics and operating lengths of canine neck inspiratory muscles. J Appl Physiol 1986 Jul;61(1):220-6.
18 Muza SR, Criner GJ, Kelsen SG. Effect of lung volume on the respiratory action of the canine sternomastoid. J Appl Physiol 1996 Mar;80(3):852-6.
19 Lee BP, Chiang ST, Hwang JC. Effects of intrapleural pressure on phrenic nerve activity. Chin J Physiol 1986;29(2):79-90.
20 Krell WS, Rodarte JR. Effects of acute pleural effusion on respiratory system mechanics in dogs. J Appl Physiol 1985 Nov;59(5):1458-63.
21 Abella M, Formolo J, Penney DG. Comparison of the acoustic properties of six popular stethoscopes. J Acoust Soc Am 1992 Apr;91(4 Pt 1):2224-8.
22 Welsby PD, Parry G, Smith D. The stethoscope: some preliminary investigations. Postgrad Med J 2003 Dec;79(938):695-8.
23 Duke T. The power of hearing. Physics World Online 2002Available from: URL: http://physicsweb.org/articles/world/15/5/8
24 Kraman SS, Wang PM. Airflow-generated sound in a hollow canine airway cast. Chest 1990;97:461-6.
25 Gross V, Dittmar A, Penzel T, Schuttler F, von WP. The relationship between normal lung sounds, age, and gender. Am J Respir Crit Care Med 2000 Sep;162(3 Pt 1):905-9.
26 Ploysongsang Y, Michel RP, Rossi A, Zocchi L, Milic-Emili J, Staub NC. Early detection of pulmonary congestion and edema in dogs by using lung sounds. J Appl Physiol 1989;66:2061-70.
27 Lisciandro GR. Abdominal and thoracic focused assessment with sonography for trauma, triage, and monitoring in small animals. J Vet Emerg Crit Care (San Antonio ) 2011 Apr;21(2):104-22.
28 Mellema MS. The neurophysiology of dyspnea. J Vet Emerg Crit Care 2008;18(6):561-71.