Radiology of the Equine Lungs and Thorax   (2-Mar-2004)

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Summary
Interpretation of radiographs of the lungs and thorax of the horse require fundamental knowledge of the anatomy of the equine lung. Interpretation is often made based on criteria described for other species (e.g., canine, feline and human). Although some of the diseases have similar etiologies the distribution of pathologic changes is different because of differences in gross anatomy and histology of the equine lung.

Radiographic findings can be made using gross and subgross anatomic description, pattern recognition or probability of occurrence. The most frequently recognized pulmonary pattern is an "interstitial pattern" because it is an expected normal pattern that is the result of a large mass of tissue with geometric blurring and variable aeration. Unfortunately, inappropriate significance is given to generic interstitial changes and structured changes are often missed. Other patterns and distributions of disease correlate well with the disease process and expected distribution of disease. A simple but systematic interpretation will give more accurate and more consistent diagnosis of pulmonary and thoracic disease.

Introduction
Radiography of the thorax of the horse and interpretation of changes seen on the radiographs of equine pulmonary disease is a challenge for any radiologist or clinician. Most radiologists lack confidence in their interpretation of benign or unimportant findings and diagnoses are often made to fulfill a perceived obligation to diagnose a disease process. A diagnosis of "normal" thorax and lung is, perhaps, the most difficult diagnosis to make. Clinicians are trained to find disease and one presumes that only those patients with disease are examined. Therefore, finding no abnormalities is difficult to resolve and many interpretations find "interstitial changes" in the lungs which are probably normal or expected findings based on the radiographic technique or respiratory phase of the horse.

Those cases that have obvious changes seen on radiographs may have had a clinical diagnosis prior to the radiographic examination and radiography is performed to evaluate the tissues of interest with respect to the severity of the disease, progress of recovery or deterioration, location of the disease and some information on the metabolic state of the patient. The radiographic interpretation should give the clinician more information than is available on routine clinical examination and provide a preview of what might be seen if necropsy had been performed. Correlation of the anatomy of the lungs and thorax of the horse with the disease patterns that are seen on radiographs will be presented in this manuscript.

Anatomy
Knowledge of gross and radiographic anatomy of the equine lung is essential to the discussion of diagnostic radiography and the interpretation of equine thoracic radiographs. Each of the gross lungs of the horse has two major parts, a small apical part and the large caudal part or body of the lung. The right lung also has a small intermediate lobe separated from the body of the lung by the posterior vena cava and the right phrenic nerve. The lungs are conjoined by a common area of adhesion at the root of the lungs, in the mediastinum [1]. The lungs have clefts but no fissures. A small cardiac notch is present in the cranial ventral right lung where the heart comes in contact with the thoracic wall at the right third intercostal space (Fig. 1) and a larger cardiac notch is present in the cranial ventral left lung, which extends from the third to the sixth rib (Fig. 2). These notches provide "windows" for echocardiography of the horse. In contrast to most mammals, the lungs of the horse are not separated by inter lobar fissures although small pleural clefts are present. The radiographic significance of this anatomic feature is seen in the presence of hydrothorax where, in most mammals, free fluid within the pleural space will
accumulate in inter lobar fissures causing retraction and delineation of multiple pulmonary margins. In the horse, there must be a significant volume of pleural fluid present to displace the ventral margin of the lung.

Figure 1. The right lung of a horse. There is a small cardiac notch. The cranial lung is small and there are incomplete fissures indenting the ventral margin of the lung. - To view this image in full size go to the IVIS website at www.ivis.org.

Figure 2. The left lung of a horse. The cardiac notch is much larger and the cranial lung smaller than the right. No fissures are present but some segmental septa are indented. - To view this image in full size go to the IVIS website at www.ivis.org.

Morphologic variations are important to lung function and pulmonary disease. The fundamental organization of the lung is similar among mammals but each species may have its variation of anatomical and physiological form. Subgross anatomy of the lung is similar in man and horse and both have incomplete separation of the lobules and extensive inter lobular connective tissue. In contrast, bovine, ovine and porcine lung lobules are completely separated with extensive interlobular connective tissue and dogs, cats and monkeys have poorly defined lobules and very little interlobular connective tissue. Terminal bronchioles, respiratory bronchioles and pleural thickness differ among these species [2]. The gross and subgross anatomy of the human lung and the horse lung has central and peripheral lung morphology of a type-III category. Categorization is based on the development of interlobular septa, the relationship of vessels to airways and the thickness of visceral pleura [3,4]. The impracticality of using the horse as a model for human lung disease has limited study of horses in preference to other animals [5].

Bronchovascular pulmonary anatomy results in compartmentalization of the lung. The most peripheral compartments are lobules of lung tissue that contain the end-airway units. Integrity of the interlobular septa may significantly affect the distribution of disease and the horse has well-defined lobules peripherally but incomplete septa more centrally [5].

Diseases of the lungs may occur spontaneously within pulmonary tissue or arrive there by pulmonary arteries or through the airways and the absence of inter lobar pulmonary fissures is of little significance. In most mammals the pulmonary arteries remain in close proximity to their respective airway and the location of the pulmonary vein may vary. Pulmonary arteries, airways and veins are in close proximity in the peripheral lung parenchyma of the horse and more centrally the pulmonary veins course separately from the bronchi and pulmonary artery [5].

Intermediate compartmentalization of the lung occurs where the lobar bronchi branch and become the segmental bronchi. The human lung has 18 - 20 pulmonary segments [6] and it is reasonable to expect that the horse has similar segmentation. Endoscopic mapping of the bronchi of horses has documented 18 segmental bronchi in the right caudal lung and 17 in the left caudal lung. Cranial lobes were not accessible for endoscopic examination [7]. These lung segments also serve to confine a disease and limit its spread, which may result in a coarse mosaic pattern (Fig. 3) compared to the more diffuse appearance of lobular or interstitial disease (Fig. 4, Fig. 5 and Fig. 6).

Figure 3. A left lateral radiograph of the thorax of a young horse with acute viral pneumonia. Some lung lobules are consolidated and adjacent lobules are hyperaerated. Changes are more severe around the hilus and in dependent (ventral) lung segments. This pattern could be confused with chronic cavity lung disease. - To view this image in full size go to the IVIS website at www.ivis.org.

Figure 4. A right lateral radiograph of the dorsal caudal thorax of a horse with acute viral pneumonitis. Interstitial infiltration of inflammatory cells is beginning to define small airway walls and is unstructured in most of the interstices, which serves to camouflage detail of large vessels and airways even though the area of the inset is hyper aerated and over exposed (the adjacent diaphragm is flattened and concave) making lung look more normal in that segment. - To view this image in full size go to the IVIS website at www.ivis.org.
The peripheral lung parenchyma is composed of the alveoli and end airway units which combine to form lobules; lobules combine to form segments and segments to form lobes. Separation of the lobes (fissures) may not be seen but the lobes are isolated by their separate bronchovascular components. Bronchi in the horse branch to cranial, middle and larger caudal lobes in both lungs and the right lung has a relatively small but a significant intermediate lobe [1]. Vessels and the respective airway of the intermediate lobe are superimposed on the posterior vena cava and are easy to identify on radiographs of the normal lung (Fig. 7). This lobe is frequently the first to be consolidated with systemic disease and the ventral margins are the first to be recognized in the presence of hydrothorax.

Figure 7. A right lateral radiograph of the ventral caudal thorax of a 750-kg horse (Cassette position C, Fig. 8). The Pulmonary arteries (PA), Pulmonary veins (PV), caudal vena cava (CVC) are seen in relation to the caudal margin of the heart (black arrows). Lung margins at the pleural reflection are seen ventrally (white arrows). - To view this image in full size go to the IVIS website at www.ivis.org.

It should be obvious from the discussion of gross and subgross anatomy of the equine lung that the "anatomy" of diseases and patterns seen in the equine lung may be quite different than that seen in other species. Interpretation of radiographic findings is often made using experience and perception acquired studying pulmonary radiography of dogs and cats, which results in errors and misleading diagnoses in horses. Correlation of radiography of lung disease and the gross and histopathologic appearance lung disease in the horse is not available and one must be careful to avoid presuming that the radiology of lung disease in all species has the same findings. Interstitial and bronchointerstitial pulmonary patterns are frequently described in horses. A study, which correlated collagen content in clinically normal adult thoroughbreds in race training strongly correlated the volume density of small airway wall thickness and morphometric estimates of parenchymal tissue and parenchymal collagen with the "perceived" prominence of the interstitial pulmonary patterns observed radiographically [8]. Another study of pulmonary radiology in horses found excellent inter-rater reliability between radiologists regarding the diagnostic quality of the film but poor reliability in assessing severity of the primary pulmonary patterns within the radiographic area of interest [9]. In other words, radiologists agree on film quality but are inconsistent in interpreting radiographic findings in equine lungs. Small airway disease probably occurs far more often than is recognized radiographically or clinically as has been shown in at least two studies [10,11]. Comprehensive knowledge of respiratory anatomy, physiology and pathology is desirable for a radiologist or a clinician. An excellent symposium on the diagnosis and treatment of equine respiratory disease contains much fundamental information that will not change with time [12].

Radiography
Radiography of the thorax of the horse may require only two (35 X 43 cm) projections for a foal's thorax or as many as eight projections of an average horse's (450 kg) thorax. Orthogonal projections of a foal's thorax are difficult and virtually impossible in larger horses. A single lateral projection of the thorax of a foal may provide radiographic findings of diffuse pulmonary disease [13] but right and left lateral projections are needed to predict the sagittal location of lesions. The large
The thorax of the adult horse requires overlapping projections to construct a composite view of the entire thorax. Four, 35 X 43 cm, right lateral projections have been proposed as a minimum study [13-15]. Due to magnification factors, bilateral images are essential for defining the sagittal location of lesions. Ideally, this would require a total of eight projections for the complete thorax; however, the cranial ventral thorax is superimposed by the shoulders and front limbs. Radiographs of the cranial thorax are not often diagnostic even when using the most sophisticated radiographic equipment and techniques. Radiographic technology commonly available to practitioners can produce diagnostic images of the dorsal thorax including the base of the heart and the caudal dorsal and the caudal ventral thorax; a total of six projections are usually sufficient for a bilateral study. Eight projections using large cassettes (35 x 43 cm) are required to image the entire thorax and lungs of an average horse (750 kg). The ventral cranial projection (D) usually results in suboptimal film quality due to the superimposed muscle mass and the small lung volume (Fig. 8).

Figure 8. A silhouette of a horse showing placement of cassettes (35 x 43 cm) required to image the entire thorax and lungs of an average horse (750 kg). The dorsal caudal projection (A) is most representative of the systemic lung and combined with the ventral caudal projection (C) the majority of pulmonary and thoracic disease is seen. The dorsal cranial projection is best for evaluation of the conductive airways and hilar lymph nodes. The ventral cranial projection (D) usually results in suboptimal film quality due to the superimposed muscle mass and the small lung volume. - To view this image in full size go to the IVIS website at www.ivis.org.

Radiographic technique will vary according to the screen film combination chosen. Some radiographic detail may be sacrificed for the advantage of reducing exposure. However, the rare earth screen and film combinations are excellent for this purpose. Using a 400-speed system*, a technique of 90 kVp and 10 mAs will produce diagnostic film quality of the thorax of a 400 - 450 kg horse. If fluid or pulmonary infiltrates are present, the technique may require up to 30 mAs. Regardless of the film/screen combination or the technique used, it is important to assess the technical quality of each exposure individually. Phase of respiration, respiratory tidal volume, and displacement of the diaphragm, beam centering, over exposed or under exposed films may result in different scales of contrast that may obscure lesions or be misinterpreted as disease. If available, a Bucky and grid can be used to improve radiographic detail by reducing scatter.

**Radiographic Anatomy**

The lungs of the horse are not equal size, the right being larger than the left, but both are large volume structures that fill space about 30 - 40 cm from the basal margins (diaphragmatic reflection) to the midline not including the body wall. Increasing distance from the film increases magnification and decreases resolution, which results in blurred margins, loss of detail and opacification and results in confusing summation opacities. A rib on the side away from the film is magnified approximately two and one-half times the size of a rib near the film. Geometric distortion resulting from the divergent X-ray beam results in nine ribs from the side of the thorax nearest the film being imaged with crisp margins, while only 3 - 4 magnified and blurred ribs from the X-ray tube side of the thorax are projected on a 35 x 43-cm film. These factors combine to cause summation opacity of a magnified rib and the aorta in the caudal dorsal thorax, which fills an entire intercostal space on the film side of the thorax. This opacification may be misinterpreted as pulmonary disease such as edema or hemorrhage. Systematic interpretation and identification of skeletal structures before embarking on evaluation of pulmonary changes will help avoid this type of interpretation error.

Magnification and geometric distortion errors are diminished when using a focus-film-distance of 150 centimeters but a minimum technique needed to expose film because of the law of inverse squares would require an X-ray generator capable of producing 30 mAs and 90 - 100 kVp when using a 400 film-speed system.

Radiographic anatomy of the equine thorax is best recognized with all available projections present on the illuminator at the same time. Films should be positioned on the illuminator as they were spatially positioned during the radiographic examination. This will result in the vertebral dorsal and the head to the left on right lateral projections and the head to the right on left lateral projections. Comparison of detail of structures and the magnification of similar structures will provide the clues needed to determine the sagittal or parasagittal location of the respective structures.

A right lateral projection of the dorsal cranial thorax of a 750 kg horse is presented (Fig. 9). The thoracic trachea (arrow) is located in the middle of the projection and the tracheal bifurcation is located between the aorta (A) and the pulmonary arteries (PA). Pulmonary veins (PV) are superimposed on the caudal vena cava (CVC) in the lower left corner of the figure.
Figure 9. A right lateral radiograph of the dorsal cranial thorax of a normal 750 kg horse (Cassette position B, Fig. 8). The thoracic trachea (arrows) is located in the middle of the projection and the tracheal bifurcation is located between the aorta (A) and the pulmonary arteries (PA). Pulmonary veins (PV) are superimposed on the CVC in the lower right corner of the figure. - To view this image in full size go to the IVIS website at www.ivis.org.

A right lateral projection of the dorsal caudal thorax of a 750 kg horse has the majority of lung volume available for evaluation (Fig. 10). The diaphragm forms the caudal margin and is flat or convex. Pulmonary arteries (PA) are seen ventral to the tracheal bifurcation (arrow) and distribute to the lung margins. Pulmonary veins (PV) are ventral to the PA at the heart base. The most dorsal caudal reflection of the lung margin is often excluded on routine radiography and it is that area where the lesions of exercise induced pulmonary hemorrhage (EIPH) may be found. The dorsal limit of this projection is delineated by the vertebra and the cranial limits by the base and caudal margin of the heart. This projection provides the best information regarding the normal airways, vasculature, interstitium and alveolar structures of the lung. Systemic lung disease, when present, will usually be recognized on this projection.

Figure 10. A right lateral radiograph of the dorsal caudal thorax of a 750 kg horse (Cassette position A, Fig. 8). The diaphragm forms the caudal margin and is flat or convex. Pulmonary arteries (PA) are seen ventral to the tracheal bifurcation (arrow) and distribute to the lung margins. Pulmonary veins (PV) are ventral to the PA at the heart base. - To view this image in full size go to the IVIS website at www.ivis.org.

The lateral projection of the ventral caudal thorax of a 750 kg horse (Fig. 7) contains information in the area of the caudal vena cava (CVC) which is in the top one-third of the image. The caudal margin of the heart (white arrows) and the diaphragm converge at the bottom of the image. Pulmonary arteries (PA) emerge from the heart base dorsal and cranial to the pulmonary veins (PV) and the CVC. Pulmonary veins return to the cardiac margin dorsal to the CVC and the vasculature and airways of the pulmonary parenchyma are seen as summation densities superimposed between ribs and over large vascular structures.

The lateral projection of the ventral cranial thorax (Fig. 11) has the aortic arch (white arrows) and cranial margin of the heart (black arrows) outlined by air in the cranial lung. The thoracic trachea passes through the projection. Triceps muscles superimpose this location and essentially no pulmonary detail is available since the high radiographic technique required to obtain this projection results in overexposure of the lungs. A request for this projection should be made as a result of well defined clinical signs of disease in the ventral cranial thorax. Often it is the absence of detail of the heart and aorta, displacement of the trachea or visualization of unexpected structure (e.g., cavitary lesions, esophageal dilatation) that confirms the clinical diagnosis.

Figure 11. A right lateral radiograph of the ventrocranial quadrant of the thorax of a 750-kg horse (Cassette position D, Fig 8). The cranial margin of the heart (white arrows) and the aorta (black arrowheads) can be seen. The pulmonary vasculature is obscure. - To view this image in full size go to the IVIS website at www.ivis.org.

Radiographic Interpretation
Radiographic evaluation and arriving at a diagnosis may be made by recognition and comparison. Excellent textbook references are available [14-17]. Interpretation of the radiographic findings should follow a systematic process if one is to correlate findings and clinical signs. Radiographs should be placed on the illuminator in a consistent position, as previously described, and a systematic method of interpretation used that insures all tissues and systems have been evaluated. Objective findings should be recorded and compared to location of projections, radiographic technique, patient positioning, and phases of respiration.

Systematic and thoughtful evaluation of radiographic findings presented in the ideas and methods that follow are the core of building personal confidence in one’s radiographic impression of equine pulmonary disease. Radiographic interpretation and selection of a list of differential diagnoses may be separated into at least four categories that are mutually reinforcing and should evolve findings, which are consistent with the patient’s clinical signs. 1) Anatomic patterns which include, a) Gross
anatomic patterns, b) Subgross histological patterns; 2) Etiologic or disease patterns; 3) Process of elimination, and 4) Probability. The latter method may be the most frequently used; it is usual to make a tentative diagnosis at the time the patient is presented to the clinician and a presumptive diagnosis is made, based on probabilities derived from the history. A diagnosis based on probability is often accurate but it does not provide quantitative or qualitative information about the disease process.

The gross anatomic pattern approach uses a systematic description of radiographic findings relative to organs, viscera, tissues, and other gross anatomic structures, (e.g., heart, lungs, aorta, CVC, lymph nodes, etc.). The subgross or histological pattern approach relies on recognition of the organization of normal tissues in the lung and the patterns that evolve as a result of disease. The etiologic or disease pattern approach is best applied when one has had previous diagnostic experience with the disease but it is important to realize one may not recognize all of the radiographic and clinical permutations of that disease.

The subgross pattern approach to radiographic diagnosis of pulmonary disease was effectively introduced in human radiology [18,19] and modified to veterinary, small animal diagnostic radiology [20]. The pattern approach has been modified for the horse lung [21-23] and is expanded in this manuscript.

Combining the gross anatomic pattern approach and the subgross histological approach, one can derive a suitable list of findings that will describe each disease. More valuable, however, is the information that one derives from these findings relative to the tissue changes and metabolic changes necessary to produce those findings.

In addition to being helpful as a diagnostic aid, the process of disease elimination or "damnit" approach can be used to organize a discussion of pulmonary diseases using the mnemonic "DAMNNIITT", (consisting of the first letter of each of the major etiologic categories). The most common condition encountered in veterinary medicine is NORMAL. The majority of primary, pulmonary diseases will fall into the subdivisions, neoplasia, inflammatory, traumatic or toxic (damNIiTT) but in the horse neoplasm in the lung tissue is infrequent. Degenerative changes, anomalies, metabolic and iatrogenic factors are often identified by subgross findings but are just a part of the greater disease process. This approach is valuable in reminding one of disease processes that might not have been considered and it leads one to recognize disease patterns according an etiologic basis and their biologic personality.

Combining all of the approaches, one is able to cross-check their list of differential considerations and those which are common to each diagnostic approach are likely to define the final radiographic diagnoses.

**Radiographic Terminology**

The patterns and signs referred to in describing radiographic findings are usually subgross descriptions. A normal pattern requires that one be able to visualize the pattern of vascular branching that has previously been described. The "effect" of vessel detail in the lung is similar to that in a leaf; there is a pattern created by the veins although individual definition is difficult to resolve. One should be able to define detail of the bronchovascular anatomy to the third order branches and intermittent detail of smaller vessels may be seen (Fig. 7, Fig. 9 and Fig. 10). It is the absence of this normal pattern that indicates another pattern may be present.

An alveolar pattern is an area on the radiograph of a lung field where one can find air bronchogram signs. The background is more radio-opaque (white or opacified) and vessel detail has been lost. The presence of blood, serum, pus or any fluid medium can fill the air spaces create a radio-opaque background. Within this background one can see those airways that have not filled with fluid but still contain air so that the internal margin is defined but the wall thickness and external margin is not seen (Fig. 12). These "air bronchogram signs" may be so small as to be called "air alveolograms or bronchiolograms" or so large as to represent segmental bronchi. They are a definitive sign of some type of fluid entering and flooding the air spaces.

![Figure 12. A left lateral radiograph of the dorsal caudal quadrant of the thorax of a foal with an unusual cardiac anomaly and pulmonary edema. Numerous air bronchogram signs (white arrows) are seen in this example of an "alveolar pattern". - To view this image in full size go to the IVIS website at www.ivis.org. -](image-url)
An interstitial pattern refers to a lung field or portion of the field that lacks definition of either "air bronchogram sign" as occurs with the alveolar pattern or normal vascular detail. The infiltration of cells or fluid into the interstitial spaces accentuates attenuation of X-ray, increases opacification of the lung, causing reduced detail and soon the combined opacification "camouflages" the detail of larger, more significant, tissue structures. Consider the magnitude of an infiltrate needed to camouflage the heart. Accumulation of infiltrates or cellular proliferation may produce a structured interstitial pattern as with metastatic tumors and diffuse granulomatosis (Fig. 5 and Fig. 13), where nodular opacities may be seen and are sometimes described as being a reticulonodular pattern. An unstructured interstitial pattern as occurs with both very acute or chronic inflammation and fibrosis has a webbed or random interlacing appearance (Fig. 4 and Fig. 6).

Structured interstitial pattern is most often described as nodular or reticulated nodular accumulations of cells in the pulmonary parenchyma but other forms of interstitial structure can be recognized. Infiltrating inflammatory cells that invade the interstitium of normal structures (e.g., interstitium of the walls of the trachea, bronchi or bronchioles) and cause increased attenuation of X-ray, which results in enhanced visibility (thickening) of the wall structure. The radiographic image has increased opacity of the walls of airways that may be erroneously perceived as calcification, which if it occurred, would be dystrophic and would not form continuous and predictable lines always perfectly tangent to the primary X-ray beam (Fig. 4 and Fig. 6). Therefore these interstitial infiltrates have "structure" and document either acute or chronic inflammatory change in those tissues. Structured interstitial changes might be described as bronchointerstitial, small airway or bronchiolar interstitial and differentiated from nodular structure that might be caused by small tumors or granulomas (Fig. 5).

Mass lesions in the thorax are described according to their gross shape and location. Larger structures may cause a "mass effect" if the margins are not well defined but there is displacement of adjacent tissues. Masses are often referred to as spherical opacities although orthogonal projections are needed to confirm the volumetric shape of a mass. "Nodule" is usually used to describe a smaller mass of tissue. The term "coin lesion" has been used to describe a small solitary nodule (Fig. 14).

Ring opacities or ring lesions are often associated with normal bronchi in the lung of the horse. These margins may become linear lesions in an orthogonal plane. Bronchietasis may result from chronic inflammation and the airway walls become thick and camouflage adjacent vessels (Fig. 15). Ultimately bullae may develop and cavitary lung disease may be diagnosed with accentuated ring opacities [24].

A cavitary lung lesion is an area of lung that may have had some contents displaced by air resulting in definition of the internal surface of the wall of an excavated mass, a fluid-air interface seen on horizontal beam projections or a bulla caused by expansion of lung. Cavitary lesions are categorized by location, size, wall thickness, wall contour, contents, and the effect on the surrounding lung [24]. In horses, unlike dogs and cats, cavitary lesions are usually the result of chronic pneumonia and abscesses [25].

The presence of any disease in the lung or thorax will result in alteration of detail of the structures described in the previous
checklist. The manner in which the detail is changed, the location of the change, the aggregation or the diffusion of change, the intensity and the permutations of patterns should culminate in a probable diagnosis or several differential diagnoses.

**Radiographic Diagnoses**

**Normal**

Clear margins of the aorta and structures at the base of the heart including the termination of the trachea and the origin of major bronchi should be seen in the dorsal cranial projection. The pulmonary arteries to the right and left dorsal caudal lung segments are seen as they emerge from the base of the heart, ventral and slightly caudal to the tracheal bifurcation. In the ventral caudal projection, smaller branches are seen originating from the pulmonary arteries at the caudal margin of the heart and entering the darker background of pulmonary parenchyma or as summation opacities over the cranial part of the CVC and the caudal part of the cardiac margin (Fig. 9). These branches continue ventral, caudal and lateral to their respective lung segments and to the intermediate lobe. The pulmonary veins are seen as they return to the caudal margin of the heart, converging into the cardiac opacity in the angle formed by the CVC ventrally and the pulmonary arteries dorsally. The summation opacification of branches of the pulmonary veins is seen as vascular structures caudal to the heart and superimposed on the CVC (Fig. 7).

Airways can be seen as they branch from the bronchi on both the dorsal cranial and ventral caudal images, and although they have entered pulmonary tissue and are surrounded by air filled lobules, they have not yet joined their respective vascular partners. This creates a complicated image, especially in the ventral caudal projection, but the detail of the edges of vessels and airways should be sharp (Fig. 7). Unlike the caudal margin of the heart, the CVC and the thoracic margin of the diaphragm have well-defined detail.

In the normal lung parenchyma the pulmonary arteries are parallel with the airway walls and can be seen with good margin detail into their second order branches. Third order branches of vessels are recognized but do not have clear margin detail and airway walls no longer have thickness sufficient to produce linear opacities. In the normal pattern of the lung parenchyma, one should identify vascular structure to the third order branches and airway wall detail to an equivalent branching (Fig. 10). Bronchial walls of the horse are seen as distinct linear opacification penetrating the lung field to their third order branches. They are best seen over areas of summations of pulmonary vessels and ribs. Airway walls are seen as ring opacities near the hilus where they occur in transverse section as they branch laterally to lobes and segments and as parallel linear opacities in the caudal dorsal field as they distribute in a cranial caudal direction toward the periphery of the lung (Fig. 10).

Pulmonary detail can be deceivingly good if air is entrapped (increased residual volume), as occurs in obstructive pulmonary disease, or as a result of hyperaeration (increased tidal volume), as may occur as a result of anemia or other causes of tissue hypoxia. Pulmonary detail can be poor in the normal lung during expiration or if there is any other cause of reduced thoracic volume (e.g., bowel distension, organomegaly, pleural fluid, etc.). The mass of magnified blurred pulmonary detail contributes to the normal, expected interstitial pattern that is so often diagnosed and given false significance. If there is possible clinical significance to this finding, inspiratory and expiratory radiographic images should be compared.

**Pleural Disease**

Diseases of the pleura and the pleural space cause confusing opacities and changes in pulmonary tissue that may be difficult to differentiate from actual pulmonary disease. Equine lung and thorax is limited to right and left lateral projection where pleural disease is superimposed on the lung. Ventral dorsal or dorsal ventral projections are preferred for evaluating the pleural space and mediastinum.

Pneumothorax is probably the least confusing. Margins of the lung may be seen and structures like aorta and vena cava may have better detail and resolution than expected (Fig. 16). The entire aorta is seen (black arrows) and the pulmonary arteries are seen without superimposed lung tissue and vasculature (white arrows). The dorsal border of the lung is visible (white arrow heads).

![Figure 16. A right lateral projection of the thorax of an adult horse with pneumothorax resulting in excellent definition of the aorta (black arrows), pulmonary vasculature (white arrows) and lung margin (white arrow heads). - To view this image in full size go to the IVIS website at www.ivis.org . -](image-url)
Pleural fluid, either transudate or exudate, causes similar increase in total opacity of the thorax (Fig. 17A) in which there is generalized increased opacity of the thorax and lungs that progresses ventrally. Despite an initial impression of abnormal lung one can see normal lung vasculature. Lung volume is collapsed and interstitial pattern is enhanced, which may be "over interpreted" as lung disease. Unrecognized or diagnosed as lung disease the problem may exacerbate as in Fig. 17B, which is a right lateral radiograph of the same horse in November with progressive chronic cavitary pleuritis. Although pulmonary tissue must be compromised normal lung vasculature can be seen superimposed on large cavitary spaces in the pleura that contain fluid and air. However, it is not unusual to have pulmonary disease and pleural disease simultaneously as in Fig. 18, which is a right lateral projection of the caudal dorsal thorax of an adult horse with a cavitary abscess in the pulmonary parenchyma. (white arrow heads). The remaining aerated lung has an interstitial pattern and increased visualization of the walls of smaller airways. There is increased opacity and loss of detail in the ventral thorax caused by fluid in the pleural space.

Figure 17a. A left lateral radiograph made in July of the thorax of a horse with pleuritis. Diffuse increased opacity and loss of detail was misinterpreted as lung disease and dependent loss of detail due to fluid, which was causing reduced lung volume, was not recognized. - To view this image in full size go to the IVIS website at www.ivis.org. -

Figure 17b. A right lateral radiograph of the same horse in November with progressive chronic cavitary pleuritis. Remaining lung has good aeration and vascular detail despite the serious pleural disease. - To view this image in full size go to the IVIS website at www.ivis.org. -

Figure 18. A right lateral radiograph of the thorax of a horse that has a large pulmonary abscess (white arrows) with an air filled cavity. The ventral thorax has increased opacity and effacement of the diaphragmand vena cava by fluid. - To view this image in full size go to the IVIS website at www.ivis.org. -

**Pulmonary Diseases**

**Degenerative Diseases**

Diseases, which fit this category, have not been clearly identified in animals. Cystic fibrosis and Kartagener's syndrome are examples of diseases that predispose the lung tissue to inflammation and subsequent deterioration. Osseous metaplasia of the lung is a frequent finding that is recognized as multiple small osseous opacities throughout the pulmonary parenchyma of dogs. This disease has no clinical significance but is often mistaken for metastatic pulmonary disease.

Recurrent or chronic inflammatory diseases, such as recurrent airway obstruction (RAO), pulmonary fibrosis and EIPH are lung diseases of horses that might be classified as degenerative but are best deferred to a more specific etiologic category.

Silicosis has been reported endemically in areas of California and could potentially occur in any part of the world [26]. Silicosis occurs as a granulomatous foreign body response to silica and definitive diagnosis requires energy-dispersive X-ray analysis of pulmonary tissue. Radiographic signs include diffuse, structured, reticulonodular, miliary interstitial opacification of varying severity. These signs are essentially the same as those expected with chronic fibrosis and RAO.

**Anomalies**

Primary lung anomalies are probably not recognized in horses. Those anomalies that affect the lung are usually found in the heart. Obviously, all of the cardiac anomalies affect the blood supply to the lungs or away from the lungs; therefore, one must expect significant pulmonary change to occur. Usually the changes are related to over perfusion or under perfusion of the pulmonary vasculature. Pulmonary congestion and edema are the most frequent sequels to cardiac disease.
**Metabolic Diseases**

Bronchopulmonary Dysplasia, in the neonatal horse, is a multifactorial metabolic insult causing chronic pulmonary disease. The disease begins with dysmature lungs at birth, perhaps as a result of surfactant errors and is compounded by oxygen toxicity, baro-trauma and an imbalance of enzymes, enzyme inhibitors and nutrition. The radiographic signs are diffuse interstitial opacification with distinct air bronchogram signs indicating pulmonary edema and atelectasis. The diaphragm is cranially displaced and has a greater convexity than normal and the thorax is small as a result of incomplete aeration of the lungs [27].

Metabolic diseases with an effect on the cardiopulmonary system are best discussed in their respective system of primary significance (e.g., cardiac disease). Roentgen Signs (Roentgen Signs are descriptive terms used to convey what is observed) associated with these diseases are the result of alterations in capillary permeability, other forms of vasculopathy or mineralization of the pulmonary tissue. Therefore, one is likely to see an interstitial infiltrate or signs of pulmonary edema. Discussion of these diseases is deferred to the appropriate system, except for pulmonary edema and hyperaeration.

**Pulmonary Edema** may be used as a diagnosis or a finding. The finding refers to end airway flooding with serum or fluid in response to focal vasculopathy and increased capillary permeability. The Roentgen Sign is characterized by an area of increased opacity within the lung field containing small, air bronchiolograms or air bronchograms as is seen in the thorax of a foal with an unusual cardiac anomaly (Fig. 12). There is a generalized alveolar pattern with opacification of the pulmonary parenchyma and multiple air bronchogram signs (white arrowheads).

The diagnosis, pulmonary edema, describes a systemic condition of the lungs which involves a large volume of the lung tissue. Lobes, segments or entire lungs may be opacified with air bronchogram signs easily identified. Severe pulmonary edema may be caused by cardiovascular disease, toxins, electrocution, fluid volume overload, and primary metabolic conditions. Other causes may be discovered with careful investigation.

**Hyperaeration** of the lung may occur as a result of obstructive pulmonary disease or as a part of any metabolic condition that causes tissue hypoxia. The body recognizes a need for oxygen and respiration rate and volume may be increased despite the absence of primary, pulmonary disease. This results in an increased air volume in the thorax, a larger thorax volume, the heart appears small and, due to an increased air:tissue ratio, the background density may be blacker than expected and can be confused with pneumothorax. This results in caudal displacement of the diaphragm which becomes flattened or concave rather than convex as is seen in (Fig. 19), which is a right lateral projection of the dorsal caudal thorax of a horse with chronic regenerative anemia. Increased tidal and residual air volume in the lungs causes better than expected detail in the pulmonary parenchyma. The clue to this metabolic condition is the ability to identify pulmonary vasculature with enhanced detail despite an obvious hypovascular lung field with diminutive vessels.

![Figure 19. A right lateral radiograph of the dorsal caudal thorax of an adult horse with hyper aeration of the lung in response to severe anemia.](www.ivis.org)

**Nutritional Disease**

Nutritional diseases have little primary impact on the lung. Bronchopulmonary dysplasia in neonatal horses may have a nutritional imbalance as one part of the multifactorial insult thought to cause incomplete development of the lung [27].

**Neoplastic Diseases**

There are two primary, pulmonary neoplasms found in domestic animals. One is rare (an alveolar cell carcinoma) and the other is infrequent (a papillary adenocarcinoma or bronchogenic carcinoma). With the exception of lymphosarcoma, most thoracic neoplasms in the horse are metastatic and usually not clinically recognized. Neoplasm of the lung probably occurs less frequently than in the pleura and metastases are more frequent than primary neoplasms [28,29]. Although a bronchoalveolar carcinoma has been reported, an equine granular cell tumor is more frequently encountered [29]. Surveys of equine neoplasia have been published [29-33].

The practical importance of pulmonary neoplasms of the horse has been considered negligible [34]. Signs of respiratory disease in a horse should lead the clinician to eliminate all causes of infectious or allergic lung disease before considering
neoplasia in the differential diagnosis. Metastatic pulmonary disease is not likely to be misdiagnosed as another disease since the patient is usually examined because of a primary tumor at another site. Radiographs of the lungs are performed in order to detect evidence of metastases prior to surgical excision or treatment of the primary site.

Radiographic findings of pulmonary neoplasia are similar to those of granulomatous pulmonary inflammation. The large size of the thorax and magnification may make even larger, better circumscribed neoplastic masses appear to have irregular margins. This pattern is similar to pulmonary abscesses or granulomas. The rare case of an unidentified primary tumor with significant metastatic pulmonary disease may occur and be difficult to differentiate from the granulomatous pulmonary diseases based on Roentgen Signs. If this occurs one must rely on geographic history, tracheal wash and cytology, serology or transthoracic fine needle aspiration of the lung or lung lesion.

Metastatic pulmonary neoplasia is an interstitial disease and most often causes an interstitial pattern. The distribution may be diffuse and miliary (adenocarcinoma), proportionate nodules (hemangiosarcoma, melanosarcoma) or disproportionate nodules (some carcinomas). Nodules may be discrete or indiscrete and there may be pulmonary, fluid infiltrates or a pleural effusion. Each tumor may demonstrate a different biologic behavior pattern. Opacities may be superimposed, resulting in summation densities or merge into larger more irregular masses as seen in (Fig. 13). There are advanced interstitial infiltration and small nodules present (inset) and at least one large nodule (white arrows). This is an example of a mixed, structured and unstructured interstitial pattern.

**Inflammatory Diseases**

Common to inflammatory diseases of the lung is the potential for lymphadenopathy and enlargement of the peribronchial lymph nodes. Lymphosarcoma may cause lymphadenopathy in the absence of pulmonary disease. Inflammatory disease of lung is reactive in lymph nodes early in the course of the disease and pulmonary changes are expected. Metastatic neoplasms of lung become significant in the pulmonary tissue before moving to regional lymph nodes. Enlarged hilar lymph nodes are seen in (Fig. 20) (white arrows) as an increased opacity at the hilus, which should remain the same size and resolution on left and right lateral projections. The location between the bronchi is likely evidence of an enlarged lymph node and when combined with pulmonary changes it is probable indication of inflammatory disease. Enlarged hilar lymph node with no evidence of pulmonary disease might be due to lymphosarcoma. Evidence of pulmonary disease and no lymphadenopathy could be metastatic neoplasm to lung or chronic structured inflammatory disease.

**Figure 20.** A right lateral projection of the dorsal thorax at the hilus. There defined are of increased opacity at the hilus which remained the same size and resolution on left and right lateral projections, which indicates the tissue mass is located near the middle of the thorax. This location between the bronchi is likely evidence of an enlarged lymph node. - To view this image in full size go to the IVIS website at www.ivis.org. -

Exercise induced pulmonary hemorrhage (EIPH) in the horse lung is a difficult disease to classify in a list of disease etiologies as it is considered to be a complex, multifactorial disease with unknown etiology. Extensive studies of the disease have been reported [35-42]. Multifocal, small airway lesions are thought to result from a low grade viral bronchiolitis. Chronic inflammation, recurrent bleeding, neovascular proliferation of the bronchial circulation in affected areas resulted in left to right shunt of vascular supply of air exchange structures [42]. The function of bleeding may be induced by mechanical stresses occurring in altered lung tissues [43].

The incidence of EIPH has been estimated at 75% in Thoroughbreds and 66% of Standardbreds following exercise [44]. The occurrence and severity of EIPH may be related to exercise intensity; evidence of EIPH, either acute with 73% of samples containing free erythrocytes or chronic with 90% of samples containing hemosiderophages, was found in raced Thoroughbreds in Australia [45]. Sudden death in racehorses has been attributed to EIPH [46].

Distribution and severity of gross lesions are variable in the dorsal caudal lungs [36-38,41]. Radiographic examination is made following clinical signs of epistaxis (Fig. 21). A right lateral radiograph of the dorsal caudal thorax of a horse has a well defined segment of pulmonary opacity, which extends cranial and dorsal from the lung margin superimposed on the diaphragm. Correlation of radiographic changes with pathologic findings has been disappointing but a satisfactory prediction may be possible with carefully selected criteria [40].
Roentgen Signs: 1) Focal increased opacification may be found in the dorsal caudal lung and extending to the margins of the lung; 2) Radio-opacities have indistinct margins, circular or ovoid surface contour; 3) Opacities may have a positive silhouette sign with the diaphragm (effacement of the diaphragmatic border) and the opacification extends toward the hilus of the lung; 4) The radiographic lesions have variable opacity, shape and size and may become cavitary [47].

Interstitial Pneumonia is seldom diagnosed accurately in veterinary radiology. The etiology is usually one of several viruses. Viral pneumonitis in the horse results in some pulmonary infiltrates but the changes are difficult to recognize due to the size and thickness of the lung. Figure 4 is a right lateral radiograph of the dorsal caudal thorax of a horse with acute viral pneumonitis. Interstitial infiltration of inflammatory cells is beginning to define small airway walls and is unstructured in most of the interstices, which serves to camouflage detail of large vessels and airways. Magnification of the inset emphasizes the loss of detail created by diffuse pulmonary inflammatory disease. These changes are easier to see where they are superimposed over less exposed areas at the pulmonary veins and caudal vena cava. The area of the inset is hyperaerated and over exposed (the adjacent diaphragm is flattened and concave) making the lung look more normal in that segment. Similar to viral diseases of the human, the prodromal signs are not dramatic. Therefore, the patient's owner or trainer is not likely to seek the services of a veterinarian until the signs of secondary infection are present. The acute phase of the disease will have passed, the subacute signs will prevail and the criteria for a diagnosis of bronchopneumonia are fulfilled. Despite this, an interstitial pattern prevails and a diagnosis of interstitial pneumonia or disease will be made.

Roentgen Signs: 1) An interstitial pattern of the pulmonary parenchyma causes loss of detail of vessels and airways. More severe infiltration with inflammatory cells results in the progressive loss of detail of mediastinal structures. 2) Peribronchial or peribronchiolar thickening may be seen as circular structures ("ring lesions") or as linear structures on the lateral projections. During the acute phase these airway "cuffs" will have well defined interior and exterior margins. Subacute changes result in emergence of an alveolar pattern wherein the internal margin of the airway can be defined but the external margin is silhouetted with the fluid accumulating in the surrounding air spaces. 3) Increased opacity and loss of detail at the hilus indicate lymphadenopathy which should prevail in any inflammatory disease and become more significant as the subacute stage of the disease develop.

The Roentgen Signs described for acute interstitial pneumonia may be confused with those described for chronic fibrosis. Both conditions have peribronchial and interstitial thickening because both are stages of inflammatory disease. The former has infiltrates consisting of the white blood cells consistent with the disease (i.e., eosinophils in allergic manifestation, mononuclear infiltrates with viral disease, etc.). The latter have fibrosis and replacement of some respiratory epithelium with goblet cells and fibroblasts. The difference between acute and chronic disease will be the presence of lymphadenopathy as judged by detail and opacity at the hilus of the lung. Chronic recurrent airway disease is characterized by cavitatory changes in the airspaces including cylindrical or bullous bronchiectasis, and pulmonary cavitations and bullous emphysema.

Eosinophilic Interstitial Pulmonary Disease has been reported in horses [48]. The disease is responsive to corticosteroid treatment and is considered to be a form of immune-mediated reaction. Parasites are apparently not a factor in the etiology. Radiographic signs are severe infiltrative disease of the interstitium of lung tissue. Definitive diagnosis requires bronchoalveolar lavage and cytology. Chronic granulomatous eosinophilic pneumonia (Fig. 5) has generalized loss of detail caused by a diffuse miliary opacification of the lungs usually called a structured interstitial pattern. The majority of the structure is in the form of miliary nodules but on close inspection some small airway wall structure is seen (inset). The diaphragm is concave, which is evidence of increased residual air volume in the lungs and despite hyperaeration, the lungs are generally radio-opaque.

Recurrent Airway Obstruction (RAO) has been called Chronic Obstructive Pulmonary Disease and is an interstitial disease of horse lungs (see the chapter by Dr. Robinson Recurrent Airway Obstruction (Heaves)). Heaves [49] and chronic restrictive pulmonary disease [50] are included in this syndrome. Allergies have been incriminated [51] and allergic manifestation has been described stopping short of classifying the disease as RAO [52]. Despite allergic implications,
eosinophils are seldom found in tracheal or bronchial aspirates [53].

The most recent publication indicates this disease is three times more likely in Thoroughbreds than other horses, which supports other studies linking its occurrence to horses with a family history of RAO. Seasonal and environmental factors and traditional horse management factors that expose horses to high dust concentrations are also incriminated [54]. Some form of small airway disease is generally associated with RAO and chronic bronchiolitis with alveolar emphysema or hyperinflation of the lung is a common denominator [50,55-57]. Figure 6 is a right lateral radiograph of the dorsal caudal thorax of a horse with recurrent airway obstruction. There is diffuse loss of detail due to an unstructured interstitial pattern, which is the result of summation and geometric distortion of changes in the respiratory level of the lung. The diaphragm is concave. Some small airway wall thickening (structured bronchiolar interstitial change) is present. Radiographic findings are much like those in other interstitial diseases (review (Fig. 4). Radiology provides generic findings of interstitial disease but ventilation/perfusion scintigraphy provides valuable information on lung function that is not obtained from radiographs [57].

Roentgen Signs: 1) A diffuse interstitial pattern with equal distribution in the entire lung, 2) Absence of hilar opacity as one expects with lymphadenopathy and acute pneumonia, 3) Peribronchial cuffing or other interstitial infiltrates may be seen. 4) Loss of thoracic detail and the degree of opacification of the lung field is considered an indication of the severity of the response. 5) Hyperinflation of the lung may be present causing flattening or concavity of the diaphragm.

Tracheobronchitis generically refers to any inflammation of the trachea or upper pulmonary airways that include the "mucociliary ladder". Used as a radiographic diagnosis it may be one of the most misused terms in veterinary medicine [22]. Diagnosis of tracheitis or bronchitis is best made on clinical signs, tracheobronchoscopy, and cytology. Classic signs of bronchitis should be considered an indication of greater involvement of other pulmonary tissues.

Roentgen Signs of bronchitis (Fig. 15) are similar regardless of the species or the etiology. 1) Increased opacity and visibility of the walls of the upper pulmonary airways and some bronchi are dilated and cylindrical bronchiectasis (white arrowheads) is progressing to nonparallel or bullous dilatation of airways (white arrows), which 2) camouflage the vessels adjacent to the airways and result in 3) patchy loss of detail of the major vascular structures within the mediastinum (heart margins, pulmonary arteries, veins and vena cava). 4) Preservation of a normal alveolar-interstitial pattern of the lung may indicate more severe involvement of the upper pulmonary airways.

Chronic bronchitis results in repetitive insult, inflammation, and necrosis of the columnar epithelium of the airway walls. Ciliated columnar epithelial cells become desquamated and are replaced with fibroblasts and goblet cells. Mucus plugs form in the airways causing obstruction of air passages. Air is entrapped in these airways during excessive inspiratory efforts and dilation of the airway results. These changes are irreversible and result in destruction of the integrity of the airway tissue. Cylindrical bronchiectasis is the intermediate stage of airway degeneration and is indicated by increased opacity of airway walls, dilated airways, and "pruning" of the airways (review (Fig. 15). Bullous bronchiectasis is the end stage of airway degeneration and is characterized by cavitary spaces in the airway created by displacement of the airway wall. Bullae are variable in size, shape and wall thickness. These stages of chronic upper pulmonary airway disease are unusual and occur as a part of severe diseases of the pleura and pulmonary tissue that result in cavitary disease patterns.

Pneumonia may be the result of infection by virus, bacteria, saprophytic or pathogenic fungi or parasites. Aspiration of food or other foreign material results in a unique lobar distribution of necrosis and infection.

Viral pneumonia begins as an interstitial disease and, as previously discussed, is not often recognized until there are concurrent bacterial infections [22]. However, viral pneumonia may occur as a lobular or segmental lung disease with collapse and consolidation of some pulmonary lobules while adjacent lobules over inflate to fill the space left by consolidated lobules. This presents an unusual mosaic of contrast that is easily confused with cavitary lung disease (Fig. 3). Radiographic differentiation may be based on the acute onset and absence of fluid-air interfaces (fluid lines) on horizontal beam projections (review Fig. 17B and Fig. 18).

Bacterial pneumonia is the leading cause of morbidity of the horse lung. The disease may begin as a diffuse interstitial disease and, depending on the organism, develop pulmonary abscesses as occurs with Rhodococcus sp. (Fig. 22). Some air bronchogram signs are present. There are diffuse nodular opacities throughout the lung. Opacification and loss of detail are greatest around the hilus, which may be evidence of lymphadenopathy. Abscesses may be expected in 10 - 15 percent of the
lungs of infected horses [22]. Chronic abscess may remain as a fibrotic nodule (arrow heads) called a "coin" lesion as seen in Figure 14. Lingering abscesses (white arrows) may rupture and drain forming cavitary changes (white arrow heads) as seen in Figure 23. Sequelae to bacterial infection of the lungs may include pleural or mediastinal disease [58-61] (review Fig. 17B and Fig. 18). Disseminated intravascular coagulation has also been documented [62]. Treatment of some pulmonary infections may result in uneventful recovery [63]. Treatment and drug efficacy may vary significantly; isolation and identification of the infectious agents are essential [64].

Roentgen Signs: Radiographic findings vary according to the infectious agent and the stage of the disease, however the signs are generic and do not represent definitive evidence of a specific etiologic agent [22,64]. 1) Increased opacity in the region of the base of the heart and hilus of the lung is partly due to lymphadenopathy. 2) Loss of detail is more significant in the region of the base of the heart and the hilus of the lung. This probably is a function of the cellulitis and edema of connective tissues since the detail of the lymph nodes is camouflaged. 3) Patchy areas of pulmonary opacity and consolidation occur and have segmental air bronchogram signs. These are focal areas of inflammatory change in the pulmonary parenchyma that is associated with vasculopathy, edema and inflammatory infiltrates. 4) There is inconsistent or patchy loss of detail of the major bronchovascular structures in the mediastinum and the respective branches to the pulmonary lobes. This is a direct result of superimposition of the changes described in "3" above. 5) Both alveolar and interstitial patterns occur in the pulmonary parenchyma. This is referred to as a "mixed alveolar interstitial" pattern. 6) Nodules, coin lesions and cavitary changes can be caused by pulmonary abscesses (compare Fig. 14, Fig. 17B, Fig. 18 and Fig. 23).

Mycotic pneumonia is unusual in horses and when it occurs aspergillosis is most frequently diagnosed [22,65]. Pulmonary aspergillosis may be predisposed by corticosteroid therapy, other concomitant diseases, and immune compromised conditions [65]. Radiographic findings are consistent with other causes of pneumonia may have structured interstitial change with small to large nodules similar other granulomatous disease or metastatic neoplasm (review Fig. 5 and Fig. 13).

Lobar Manifestation is an anatomic description of a disease affecting one or more lobes of the lung. Neoplasia, trauma and thrombo-embolic diseases are differential considerations in most animals but not recognized as causing lobar disease in the horse. Lobar (or segmental) disease is usually seen with pulmonary abscesses or aspiration pneumonia. Regardless of the etiology, differentiation is not based on radiographic signs. A radiograph of the thorax of an orphaned foal is presented in (Fig. 24). The accessory lung lobe and the dependent segments of the middle lung lobes are opacified and contain air bronchogram signs. Cranial ventral lung segments are not affected.

Roentgen Signs: 1) Opacification of lobules, segments or lobes of a lung is present but there is no concomitant change in adjacent lobes. 2) Lobar, segmental or a lobular alveolar pattern is present, with or without air bronchogram signs. 3) Organ and tissue displacements occur, consistent with collapse and consolidation of lung lobes. 4) The accessory and right middle lung lobes are most frequently involved resulting in a silhouette sign of the caudal vena cava, heart and diaphragm in the ventral caudal projection.
Suppurative or chronic purulent pneumonia may be diagnosed in any horse at any age; however when it occurs in very young and very old patients, immune deficiency is probably a predisposing factor. Foals are susceptible especially in cases of failure of passive transfer of antibodies.

Roentgen Signs: 1) Increased opacity and loss of detail at the hilus are due to lymphadenopathy. 2) Dependent pulmonary consolidation is present and may have some air bronchogram sign. 3) Multiple, indiscrète nodular densities (abscesses), may be found throughout the lungs or concentrated in dependent lung segments. 4) Bronchiecstasy, (cylindrical or bullous) may occur. 5) There is loss of normal bronchovascular detail in the hilus of the lung. 6) Cavitary lung disease may be present within the pulmonary parenchyma.

Parasitic Pneumonia: Each species has host specific parasites that may reside in the lungs or occur as transient larval migrations. Occasionally, one species' parasite may invade another's tissues and cause severe pneumonia as occurs with *Ascaris lumbricoides*, the pig Ascaris when it invades foreign host tissue. The pulmonary lesions are the result of an allergic reaction, followed by infarction, necrosis and secondary bacterial infection. Granulomas, abscesses or cavitary changes may be present depending on the parasite and the biological response. This type of disease is expected to occur in horses but no reports were found.

*Dictyocaulus arnfieldi* occurs in the bronchi of equidae and is found in most areas of the world. Usually the parasite is not very pathogenic and favors donkeys.

*Iatrogenic*

Usually these circumstances are the result of drug or chemical sensitivity or perhaps due to oral administration of a substance with subsequent aspiration. The most frequent Roentgen Sign is associated with pulmonary edema and, of course, aspiration pneumonia in the case of improper nasogastric intubation.

*Trauma*

The permutations of Roentgen Signs that may result from trauma to the thorax are too numerous to discuss. The typical sign is pulmonary contusion which may be indistinguishable from edema or hemorrhage since it is composed of both. Usually one expects to find other clues indicating trauma. Broken ribs, pneumothorax, pneumomediastinum or subcutaneous emphysema may be detected [66]. The pattern of consolidation is usually lobar or segmental but may not be dependent since the lung change occurs in the vicinity of the concussion. Pulmonary edema and hemorrhage due to contusion should mobilize and resolve and the lung should aerate within 48 - 72 hours of trauma.

*Toxic*

Toxic diseases of the lung may be the result of ingested plant toxins [67] or inhalation of smoke or volatile chemicals resulting in pulmonary hemorrhage or edema.

Roentgen Signs include 1) lobar, segmental or lobular pulmonary consolidation, often located in the caudal, dorsal lung; 2) consolidated lung has alveolar pattern prevalent with air bronchogram signs.

*Conclusion*

The radiographic interpretation of the normal lungs and pulmonary disease in horses may follow the example of methods used in human beings and smaller mammals but should be made with a much different perspective. The size of the lungs of a horse is larger than most "small" animals and human lungs. Radiography of this volume of tissue will result in special image analysis problems. The gross, subgross and histological anatomy of equine lungs should be studied and correlated with necropsy and histopathology.
References


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