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Thoracic Radiology Mini Series

Session Three: The invisible margins of the thorax – not overlooking non cardiopulmonary thoracic pathology

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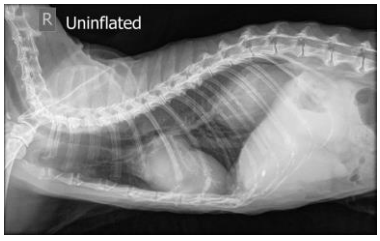


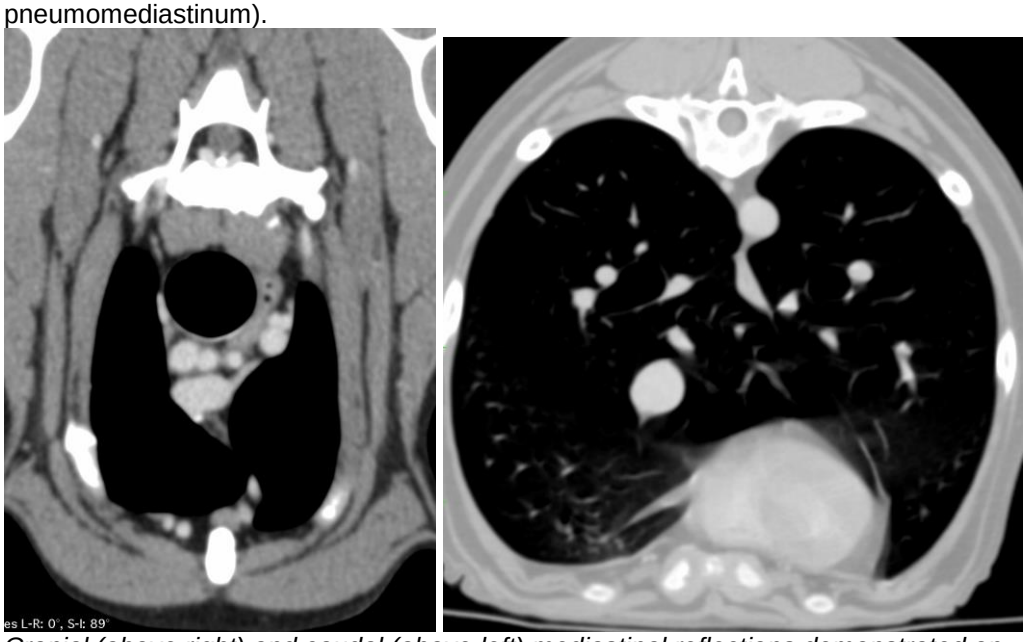
Session 3 - Monday 6th August 2018 1pm-3pm

The invisible margins of the thorax – not overlooking non-cardiopulmonary thoracic pathology

It is easy to forget about mediastinal and thoracic wall structures, since they can be overlooked if the heart and lungfields are focused on during radiographic review. When abnormal, they can be a cause of significant pathology, and must not be confused with pulmonary disease as they superimpose on the lungfields.

- What structures are in the mediastinum, and what pathology is seen
- Anatomy and pathology of the pleural space
- Pathology of the diaphragm and thoracic wall

The **mediastinum** is a space lying between the medial pleurae of the left and right lungs that contains all of the midline structures of the thorax – the heart, the trachea, the oesophagus, the great vessels, and the lymphoid structures. It is not an enclosed space, and is continuous with the fascial planes of the neck and with the retroperitoneal space caudally (via the aortic hiatus). The normal mediastinum is uniform soft tissue opacity, unless is becomes enlarged due to fat deposition or it becomes pathologically filled with gas (termed a pneumomediastinum).

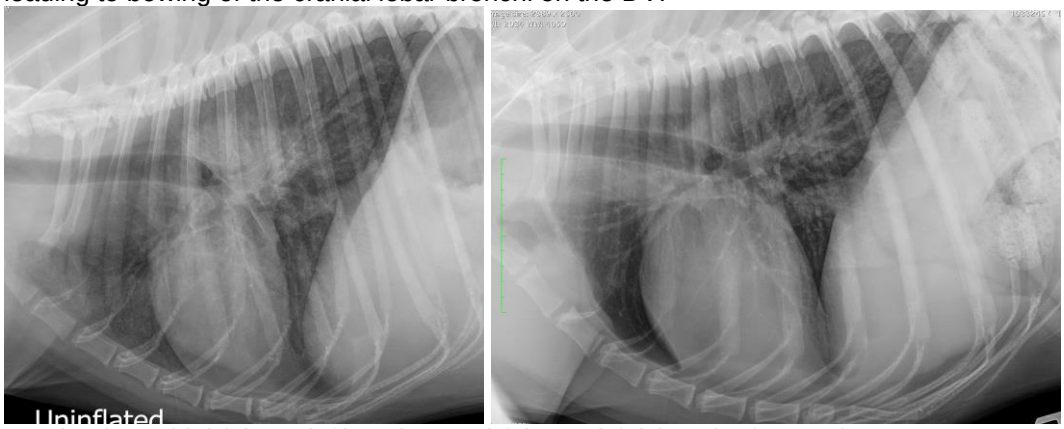


Cranial (above right) and caudal (above left) mediastinal reflections demonstrated on transverse CT images.

The normal mediastinum is not seen on the lateral unless there is a division between right and left cranial lobes, so a positive effort is needed to avoid overlooking the mediastinal structures. On the DV it is seen as a midline soft tissue band through the length of the thorax, bulging at the level of the heart, and in some dogs with a visible cranial and caudal mediastinal reflection located between (respectively) the right and left cranial lobes, and the accessory and left caudal lobes. The mediastinum should not be wider than the vertebral column in cats, and not more than twice the width of the vertebral column in dogs. The cranial mediastinum might be more visible in young dogs due to the presence of a thymus – in young dogs this is seen as the thymic "sail" sign in the cranial mediastinal reflection on the DV, reaching its maximal size at approximately 4 months, regressing until about 1 year of age. In cats, the thymus is seen best as a soft tissue opacity cranial to the heart on the lateral, and may persist for slightly longer. Obese animals deposit fat in their cranial mediastinum. It is rarely enough to cause the individual structures of the mediastinum to be visible, but can cause quite significant mediastinal widening (well above the normal range) and therefore be cautious not to overinterpret this widening as being due to a mediastinal mass.

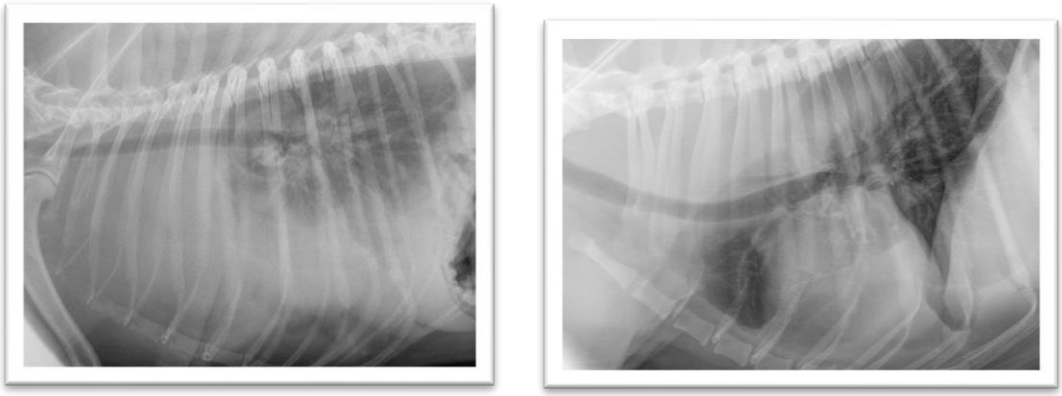
Many mediastinal structures are not visible radiographically unless there is pathology – large vessels such as the brachiocephalic trunk and left subclavian arteries, the azygous vein and main pulmonary artery, the thoracic duct, the vagus, recurrent laryngeal and phrenic nerves, and the tracheobronchial, mediastinal and sternal lymph nodes.

The sternal lymph nodes are usually paired (although the exact number varies), and lie dorsal to the second sternebra. They drain the thoracic wall but also the cranial abdominal wall, mammary glands and some of the cranial abdominal organs – therefore if there is enlargement of these nodes, the pathology is usually not intrathoracic, and abdominal pathology should be excluded (e.g. using ultrasound). Enlargement is noted by an extra pleural broad based mass at the level of the second sternebra.
The tracheobronchial lymph nodes lie around the bifurcation of the trachea – the middle lying in the V made by the bifurcation, with the left and right lying respectively cranial to the left and right cranial lobar bronchi. Enlargement of the middle lymph node is noted as a soft tissue mass on the DV splitting the mainstem bronchi (in a manner similar to an enlarged left atrium, however can be distinguished from this on the lateral since the increased hilar soft tissue opacity of lymphadenopathy depresses the carina and mainstem bronchi whereas left atrial enlargement will elevate the carina). Left and right tracheobronchial lymphadenopathy is commonly seen in association with middle lymphadenopathy; and is seen just cranial to the carina on the lateral (with soft tissue opacity dorsal and ventral to the terminal trachea) and leading to bowing of the cranial lobar bronchi on the DV.



Tracheobronchial (above left) and sternal (above right) lymphadenopathy.

Mediastinal structures are better visualized if there is free air present (a pneumomediastinum). The vessels of the cranial mediastinum become visible (brachycephalic trunk and left subclavian arteries, cranial vena cava); also the outside of the trachea is highlighted by gas. Causes include: leak of air from the trachea or oesophagus, leak of air from the lung (tracking up the bronchial tree), extension of retroperitoneal or cervical fascial plane gas. Underlying trauma, (severe) respiratory disease, rarely lung lobe torsion and bronchial rupture are described, although sometimes the cause is not identified.
Mediastinal masses may be identified by looking for evidence of mediastinal widening, or displacement of the mediastinal structures (in particular the trachea). Masses are defined by their location.
Cranioventral masses are the most common; they widen the cranial mediastinum, and may elevate the trachea (be sure to rule out obesity and fat deposition); differentials include neoplasia (e.g. lymphoma, thymoma, ectopic thyroid/parathyroid and carcinoma, sarcomas); cysts (thymic, branchial arch, pericardial etc.); mediastinal haemorrhage, granulomas (e.g. FIP), and sternal lymphadenopathy.
Craniodorsal masses are uncommon and will depress the trachea ventrally and to the right, including masses of the oesophagus (or distension of the oesophagus with fluid), neuroendocrine tumour, aortic aneurysm, paravertebral tumours (e.g. sarcoma).
Perhilar mediastinal widening is associated with widening of the caudal mainstem bronchi and can be seen with lymphadenopathy (will also depress the tracheal bifurcation ventrally), heart base masses, cardiac or vascular disease mimicking a mass, oesophageal foreign bodies, and (rarely) bronchogenic cyst.



The trachea is a marker for where a mediastinal mass is located – above left is a cranioventral mass, above right is a craniodorsal mass.

Caudodorsal mediastinal masses widen the caudal mediastinum, and may silhouette with the diaphragm; and might arise from or involve the oesophagus (oesophageal masses, hiatal masses and hernias, diaphragmatic lesions, neural lesions); also it is not an uncommon location for mediastinitis (relating to migrating foreign body or otherwise).

Masses are rarely seen caudoventrally other than associated with the diaphragm (including true hernias, PPDH and ruptures).

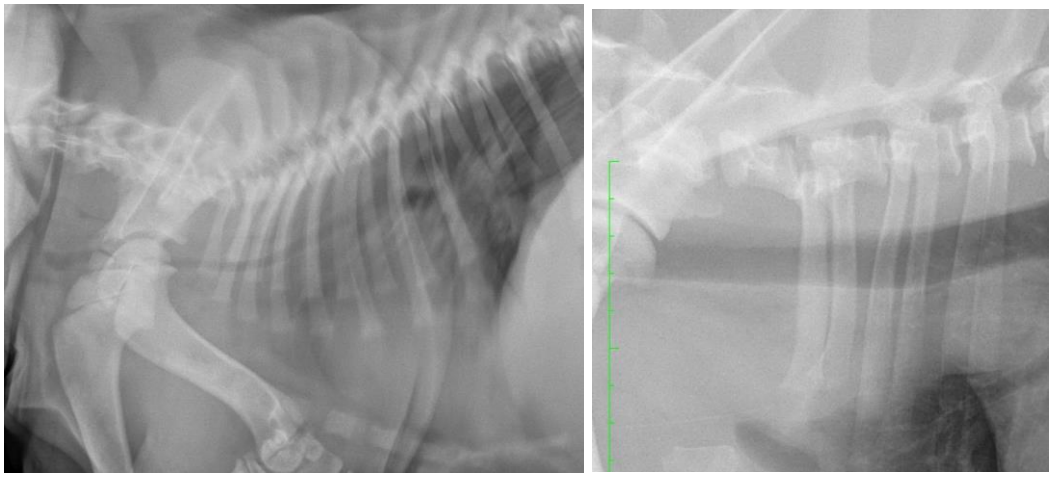
Mediastinal diffuse widening, occasionally with “reverse fissures” extending out from the mediastinum between the lung lobes, may be due to free mediastinal fluid. Haemorrhage is the most common if the fluid is isolated to the mediastinum (following trauma, rodenticide toxicity or coagulopathy) however mediastinitis may lead to pus free or encapsulated within the mediastinum (commonly due to a migrating foreign body, although oesophageal rupture can lead to this also). Chyle, transudate and exudate may accumulate in the mediastinum, however commonly these will extend to the pleural space, and the mediastinal involvement is overlooked.

Running through the mediastinum are two essential structures to assess – the oesophagus (lies slightly to the left) and the trachea (to the right).

The **trachea** is a rigid tube lined with C shaped cartilages; dorsally the C is completed with the dorsal trachealis muscle. The cartilages might mineralise with age and become visible. The trachea is usually straight although can start to form as S bend if the head is flexed. The inner margin is usually smooth (occasionally irregular in Dachshunds and other chondrodystrophic breeds). The trachea should be $1/5^{th}$ of the height of the thoracic inlet in most normal dog breeds; however is considerably smaller as an average in Bulldogs (ratio of 0.1) and other brachycephalic breeds (0.16).

Much of identification of tracheal pathology relies on noting narrowing of the luminal gas column. In the neck, the only gas filled structure is the trachea, therefore the true width of the trachea if it remains round is easy to identify, as it is demonstrated by the margins of the gas column. There should be little difference between inspiration and expiration unless there is softening of the rigid cartilage rings (malacia).

An overall narrowing of the entire length of the trachea is called tracheal hypoplasia; which is most commonly seen in English Bulldogs although it has been rarely described in larger dogs and the cat. In Bulldogs it is one component of the brachycephalic obstructive airway syndrome (BOAS), alongside stenotic nares, elongated/thick soft palate and everted laryngeal saccules; and is the component that cannot be surgically corrected. There is a uniform narrowing throughout, to a greater extent than the uniform narrowing expected with the breed.



Above left: Tracheal hypoplasia narrows the entire trachea in an English Bulldog puppy; above right is an incidentally invaginated dorsal trachealis muscle (without overall reduction in the height of the gas column.

Sometimes, especially in large breeds, the dorsal trachealis muscle may fold in, and be seen as a soft tissue band within the trachea however the overall height of the gas column is unchanged, since at the margin, the C shaped cartilages maintain their normal height and shape. The invaginated dorsal tracheal muscle might be dynamic between radiographs, and usually has no associated clinical signs.

Tracheal collapse is a degenerative pathology of the tracheal cartilage seen in middle-aged toy and small breed dogs. Tracheal collapse can be missed on a static radiograph, as it is a dynamic process, so fluoroscopy/bronchoscopy +/- CT are often more helpful in the diagnosis. In the case of tracheal collapse, the C shaped rings are not rigid, and flatten out so the dorsoventral height of the trachea is dynamically reduced for a segment. This varies between inspiration and expiration (the intra-thoracic trachea collapses with expiration, the extra-thoracic trachea with inspiration).

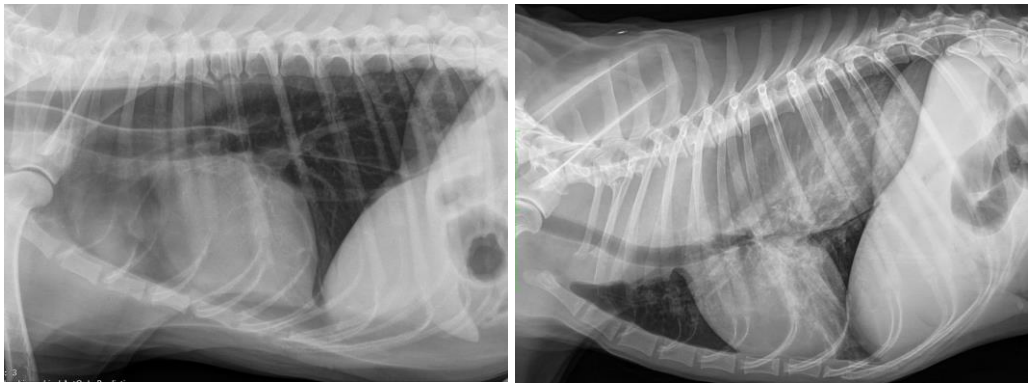
Tracheal mural thickening might be the result of trachea neoplasia, tracheitis, or mural haemorrhage (for example rodenticide toxicity).

Rarely, a tracheal stricture will form. Most commonly this is an acquired disease, and underlying aetiologies might include an over cuffed ET tube, or a traumatic event (the cranial thoracic trachea is susceptible to trauma, and might lead to the formation of a 'pseudotrachea' as air accumulates in a bubble around the focal tracheal interruption as the external fascia are preserved).

The **oesophagus** is a muscular tube running through the dorsal half of the mediastinum, with 4 histologic layers (mucosa, submucosa, muscularis and serosa (like bowel)). It transports a bolus of food from the pharynx to stomach using a primary peristaltic wave. If the primary wave is not sufficient to transmit the bolus (or if the bolus is too small) a secondary wave is generated; and oesophagus should completely empty.

If normal, the oesophagus is usually not seen on a radiograph since it is soft tissue opacity surrounded by the other soft tissue structures of the mediastinum. It is seen in animals with pneumomediastinum, and occasionally in very thin deep chested dogs. It can be identified in patients without oesophageal pathology if there is a small amount of gas present within the oesophagus (might occur due to sedation, aerophagia or sometimes in normal patients) or if there is fluid regurgitated into the oesophagus (seen as a soft tissue band in the caudal thorax between aorta and CVC).

However, it becomes increasingly visible as it enlarges due to pathology, and becomes distended with gas or fluid. With a large amount of gas distension, thin converging fine soft tissue stripes of the oesophageal wall are seen in the caudodorsal thorax on the lateral. In the cranial mediastinum there is a tracheal stripe sign and increased visibility of longus colli muscles due to oesophageal gas lying between these structures. On the DV view, the mediastinum is diffusely widened, and may bow to the right if it distends dramatically (bounded by the aorta on the left).



Two examples of megaoesophagus; filled with gas (above left) and fluid (above right).

Megaoesophagus occurs due to a functional disturbance of oesophageal motility. This can be seen congenitally (GSD, great Danes, Labrador retriever amongst others have a (likely) familial predisposition for these diseases). Acquired generalized megaoesophagus may be idiopathic, or secondary to neuromuscular disease (myasthenia gravis, polymyositis, Distemper, tetanus, systemic lupus erythematosis, glycogen storage disease) and toxicities (lead, organophosphate).

A more focal megaoesophagus might be seen with causes of oesophageal obstruction (foreign body, stricture, vascular ring anomaly, neoplasia). Oesophageal foreign bodies commonly lodge at the heart base, however might lodge at the thoracic inlet or the hiatus. Radiographs might show a mineral opacity at the location of the oesophagus, or a soft tissue mass with oesophageal dilation cranial to the obstruction. Post removal, it is prudent to repeat radiographs to ensure that no oesophageal perforation has occurred, which might be indicated by evidence of mediastinal fluid or gas.

Vascular ring anomalies occur secondary to abnormal embryonic development of the aorta (aortic arches 3, 4 or 6). The abnormal location of the aorta (most commonly, in 95%, a persistent right aortic arch) means the oesophagus is trapped by the ductus that runs between the right-sided aorta and (normally) left sided pulmonary artery, and the trachea ventrally. This leads to dilation of the oesophagus cranial to the external oesophageal narrowing at the heart base. These animals have typical onset of regurgitation once they start solid foods. Contrast oesophograms help to identify the narrowing at the heart base, and CT is helpful to characterize the vascular anomaly. Other vascular anomalies include a double aortic arch (these patients might also be dysphonic since they also have some compression on the trachea); aberrant subclavian (less likely intercostal) arteries, and rarely a normal left aortic arch but a right ligamentum arteriosusm.

Strictures are uncommon, and often occur a short time after some trauma (perhaps a foreign body) or caustic event, which might at the time have presented with oesophagitis (note, oesophagitis is often radiologically normal at the time and might be missed, although the abnormal motility can lead to a small amount of gas being present in the oesophagus.

Neoplasia is rare in the oesophagus. Types of oesophageal neoplasia include leiomyoma/osarcoma, carcinoma and chondrosarcoma in dogs, squamous cell carcinoma and lymphoma in the cat. Contrast studies are needed to identify a small mass, although large masses are recognized by the presence of a soft tissue (+/- some amorphous mineralization) mass in the mediastinum.

All causes of oesophageal dysfunction might have concurrent aspiration pneumonia.

Oesophageal redundancy is seen with a small U shaped fold in the oesophagus at the thoracic inlet. This is generally thought as “incidental” and is seen on young brachycephalic dogs, however these dogs have so much alteration in the respiratory pressures due to brachycephaly that they very frequently have concurrent oesophageal functional disturbance and hiatal hernias.

The oesophagus passes through the diaphragm at the central hiatus oesophageal hiatus. This hiatus allows for several types of (usually congenital) herniations:

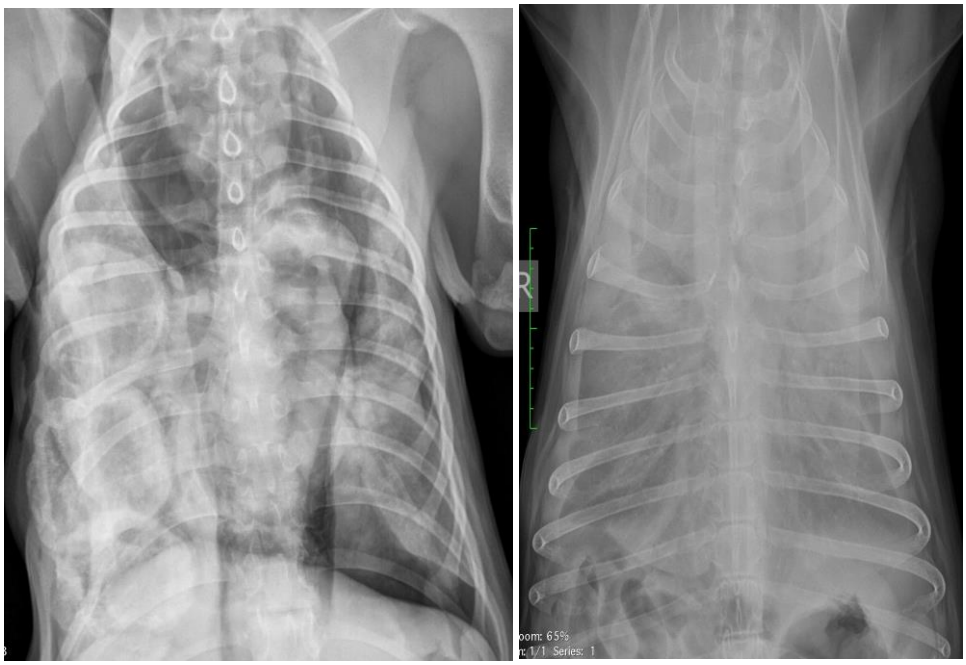
- The oesophagus might be short, with the caudal oesophageal sphincter lying permanently in the thorax;
- The caudal oesophageal sphincter might slide in and out of the thoracic cavity;

- There could be a paraoesophageal hernia, where the stomach (or less commonly liver and small intestine) slides through the hiatus next to the normally located oesophagus.

All have similar radiographic findings of a soft tissue mass in the caudal mediastinum, silhouetting caudally with the diaphragm. Occasionally gas may accumulate and highlight rugal folds, which is diagnostic. Barium studies can be helpful, as can fluoroscopy, which helps in cases of dynamic disease. If a large amount of soft tissue is seen in the caudal mediastinum with acute signs, a gastro-oesophageal intussusception should be considered.
True diaphragmatic hernias may be seen in the ventral midline due to a fusion failure of the primitive embryologic diaphragm; leading to abdominal content bounded by peritoneum lying in the caudoventral mediastinum, or the pericardial sac (a peritoneal pericardial diaphragmatic hernia).
The true hernias can be differentiated from a traumatic diaphragmatic rupture partly by their history and more acute clinical presentation. The diaphragm may rupture traumatically following increased abdominal pressure. It might rupture centrally (in cats, occasionally only a small tear is present ventrally and just the falciform fat slips through); or on the right or left; the tear occasionally propagates both sides. Radiographic signs rely on loosing the visibility of the diaphragm; pleural effusion, identification of abdominal organs in the thorax (lung lobe collapse, mediastinal shift) and a loss of organs in the cranial mediastinum. Ultrasound and contrast studies are complimentary techniques.

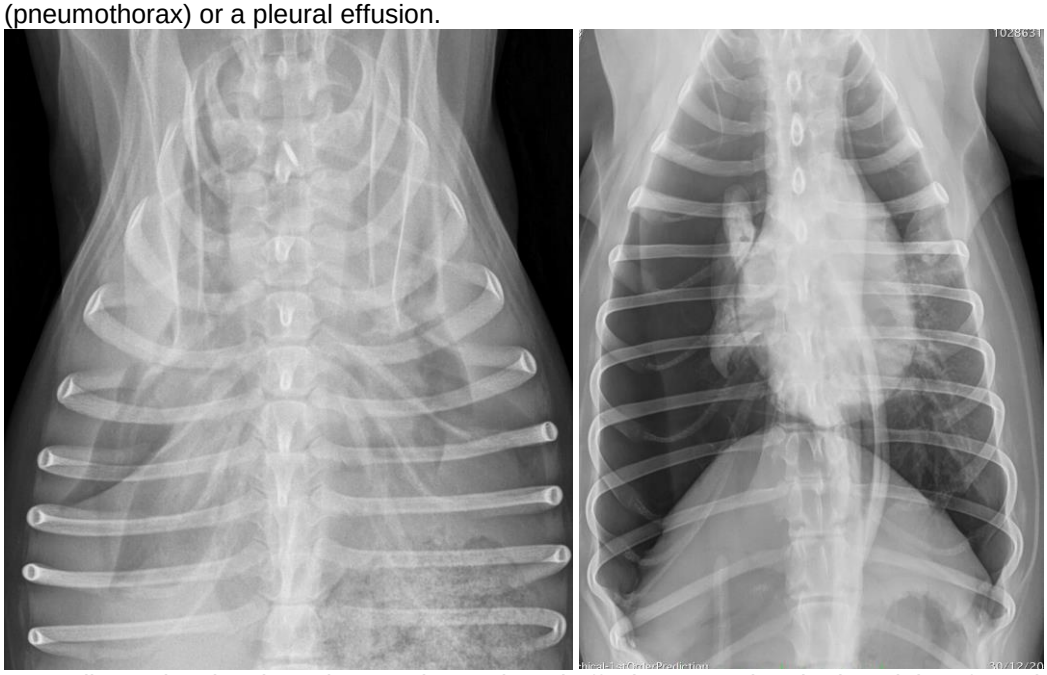
The **diaphragm** has a functional role in respiration, controlled by the phrenic nerve. Trauma to the phrenic nerve, which can occur during surgery of the cranial mediastinum or pericardial sac, can paralyse the diaphragm, leading to paradoxical movement (the diaphragm moves cranially on inspiration). This can be identified by diaphragmatic asymmetry on a DV radiograph. Other diseases, such as muscular dystrophy, are described as causing alterations to the diaphragm shape due to hypertrophy of its muscular portion.

The **thoracic skeletal structures** can have all the same pathologic conditions occur as elsewhere in the body, including developmental disease, degenerative conditions (including disc degeneration, although the thoracolumbar junction is the most common location), trauma, neoplasia and infection.
Trauma might be identified as vertebral fractures, which can be easy to overlook on a radiograph) and rib fractures. Although a rib fracture is a cause of clinical pain for an animal, it indicates that there has been a significant trauma and indicates a further examination of deeper structures is needed. It might have further clinical significance itself if a flail chest develops.
Rib neoplasia may be relatively subtle clinically, since the mass often preferentially expands inwards, leaving little to no palpable external mass. There is frequently bony lysis, with a predilection for the distal third of the rib, and these lesions often are associated with pleural effusion that can further confuse examination of the ribs for a subtle, often lytic lesion.
Primary neoplasia of the rib might be osteosarcoma, chondrosarcoma, less commonly haemangio- or fibrosarcomas. Metastatic lesions can affect the ribs, as can multiple location neoplasia such as multiple myeloma, which is frequently seen in the ribs and vertebrae as subtle lytic foci. The thoracic wall might be affected by fatty or soft tissue masses; with fibrosarcoma the most frequently seen cause of the latter. With soft tissue neoplasia, there should be an extra pleural sign (where there is a broad based soft tissue mass deviating the lung inwards from the pleural margin – the lung makes an angle of greater than 90 degrees where it meets the soft tissue; whereas a pulmonary origin mass that contacts the body wall should make an angle of less than 90 degrees). Benign masses of the ribs include osteochondromas and periosteal reactions associated with infection (often migrating foreign bodies).



Benign (above left, multiple cartilaginous exostoses on several ribs) rib masses may be easier to spot than the predominantly lytic aggressive rib neoplasia (above left, where the pleural effusion partly masks the predominantly lytic chondrosarcoma of right rib 6)

There are two layers of parietal **pleura** – the visceral lining the lungs, and parietal lining the thoracic cavity, with a small amount of fluid (2-3ml) between to allow movement. Surface tension in the pleural fluid allows the lung to be held against the thoracic wall. The pleura are not usually seen with a radiograph; although there may be some thickening of the pleura with ageing; if the X-ray beam hits the pleura tangentially this leads to a fine line being visible on the radiograph (not indicating a small pleural effusion).
The pleural space is only visualised on a radiograph if it is expanded with gas (pneumothorax) or a pleural effusion.



DV radiographs showing a large volume pleural effusion retracting the lung lobes from the thoracic wall (above left), and a large volume pneumothorax with some tension on the right leading to lung lobe collapse and mediastinal shift (above right).

A pneumothorax is recognized radiographically if there is gas displacing the lung margins inwards from the thoracic wall. In particular, there is often “elevation” of the heart from the sternum on a lateral radiograph (since the dependent lungfield is relatively collapsed, and the heart falls dependently so only pneumothorax air is visible along the sternum). Depending on the volume of the pneumothorax, there is reduced size of the lung lobes; if the pneumothorax is particularly large or there is tension (a one way valve allows air to enter the pleural space but not exit) the lungs might completely collapse to adjacent to the hilus. Care should be taken not to mistake a skin fold lying over the thoracic margin (leading to a line which could be mistaken for a linear margin of a lung lobe retracted by gas) for pneumothorax by looking very carefully to see that small pulmonary vessels continue to the margin of the thorax. The lateral view might be slightly more sensitive for a small volume of pneumothorax.
Pneumothorax is caused by rupture of the lung leaking into the pleural space – this is commonly traumatic, although sometimes might result from chronic pulmonary pathology. Pneumothorax might also be spontaneous (i.e. a cause is not known/identified – a ruptured bulla which is therefore no longer seen could have this appearance).
Pleural effusion leads to a soft tissue opacity retracting the lungs from the thoracic wall, scallops the ventral lung margins on the lateral view and variably widens the pleural fissures. The volume of effusion means there is less room for the lungs to expand, therefore lung opacity is increased.
It is easier to see a small volume effusion on a lateral radiograph, where signs might be limited to rounding of the ventral lung lobes, some obscuring of the heart and diaphragm, and fine linear interlobar fissures. If the effusion progresses, this might lead to wedge shaping of the interlobar fissures, rounding of the costodiaphragmatic recesses on the DV, and progressive obscuring of the cardiac silhouette and diaphragm. Effusion in the cranial thorax means the cranial lung lobes do not reach as far cranially as the first rib, and care must be taken to differentiate a pleural effusion from a cranial mediastinal mass since the trachea may elevate along its length due to the effusion (note, only a mass can distort or compress the trachea, or displace the cardiac silhouette caudally). Causes of pleural effusion include transudate, exudate (pyothorax is a septic exudate), chylothorax, and haemothorax. The “thicker” effusions might be less able to cross from one pleural space to the other, so if the effusion is predominantly unilateral, a pyothorax (or chylothorax/haemothorax) is more likely the cause. The effusion might be loculated in one location. Effusions are more likely to occur around the site of pathology (for example around a pathologic lung lobe such as lung lobe torsion, or adjacent to a thoracic wall mass). Septic effusions might also be encapsulated or loculated given the amount of fibrous tissue surrounding the inflamed pleura. Following a pyothorax, the pleura might have been so scarred that it persists in maintaining an abnormal, rounded, incompletely expanded lung lobe margin even when the patient has resolved the pleural infection, a process known as restrictive pleuritis or cortication.

Reading a thoracic radiograph
Reading a film well takes care and practice.
Suggestions include:

- Take time in good conditions
  - Don’t rush to make a diagnosis while the patient is waiting on the table. Review in good light conditions with an adequate quality screen.
- How was the radiograph taken?
  - Was the patient under GA? (could there be atelectasis?). Was the radiograph taken in inspiration or expiration?
- What about the breed and the age of the patient?
  - Breed can have a dramatic effect on conformation, in particular cardiac shape. Age can alter lung opacity, in particular many structures start to mineralise with age.
- Follow a set approach
  - Anatomic: examine each in turn… soft tissues surrounding the thorax, the neck, the abdomen and diaphragm, the bony structures, the pleural space, the mediastinum (including trachea, oesophagus), the bronchi and lungs, the heart and great vessels.
  - Topographical: assess areas of the radiograph step by step …. Front to back, top to bottom, outside inwards.

- Use roentgen signs
  - Look for change in opacity, size, shape, margin and number of each structure.
- Look for shift in the location of normal structures
  - In particular mediastinal shift, but also of the trachea, the heart, the diaphragm etc.
- Look for asymmetry
  - On the DV, is the left hemithorax the same size and opacity as the right hemithorax.

Further reading:
The BSAVA manual of Canine and Feline Thoracic Imaging (eds. Schwarz and Johnson), published 2008, is an excellent companion to this mini series course.