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Thoracic Radiology Mini Series

Session One: Assessing the lungs – moving beyond lung pattern recognition

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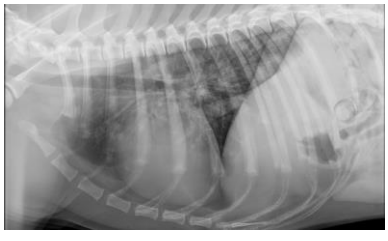


MS180 - Thoracic radiology Mini Series

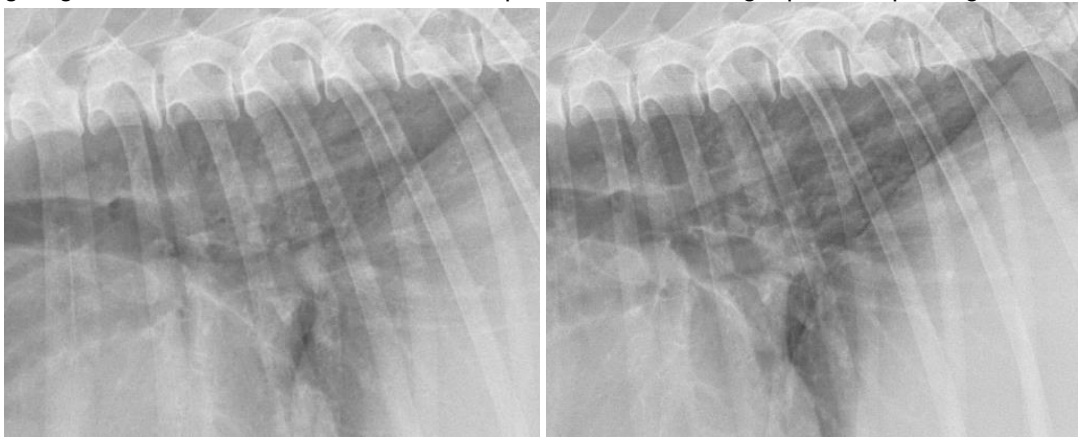
Session 1 - Monday 16th July 2018 1pm-3pm

Assessing the lungs – moving beyond lung pattern recognition

This session compares the traditional schemes of lung pattern assessment (alveolar, bronchial, interstitial and vascular) with alternative approaches to best focus differential lists.

- What is the normal radiographic appearance of the lungfields?
- What are the hallmarks of a bronchial, interstitial and alveolar lung pattern?
- What other features can we use to better distinguish what lung pathology is present?

We need to **appreciate normal first**, and understand what effect our settings and patient factors might have on normal, before we can start to understand abnormal.
Achieving **technically good radiographs** is a challenge in practice.
The first issue is that many patients preventing for thoracic radiographs may be clinically unstable due to cardiac or respiratory disease. The most technically beautiful radiographs can be obtained with the patient under general anaesthesia with manual inflation, however care must be taken to ensure that the lungs do not become atelectatic (ensure a DV view is taken first, with the patient not in lateral recumbency for intubation, then quickly take any lateral radiographs required, ventilating the patient as needed – the procedure must be managed carefully as atelectasis precludes assessment of the lung and renders the radiograph non diagnostic). Also, many patients cannot be anaesthetised due to their clinical status. In those more unstable patients, care should be taken to establish if they are safe to radiograph at all, since the stress of positioning can destablise a barely stable patient; and sometimes delaying the diagnostic procedure is safer.
The other issue with a dyspnoeic patient is the difficulty of obtaining radiograph without movement blur. The goal should be to obtain radiographs at peak inspiration, which is challenging if the patient is tachypnoeic. This may be counteracted by ensuring that the radiograph is taken with the shortest exposure time as possible (if time of exposure can not be manipulated alone, making the mAs as low as possible); and in some circumstances giving sedation to reduce the stress of the patient, as stress might promote panting.

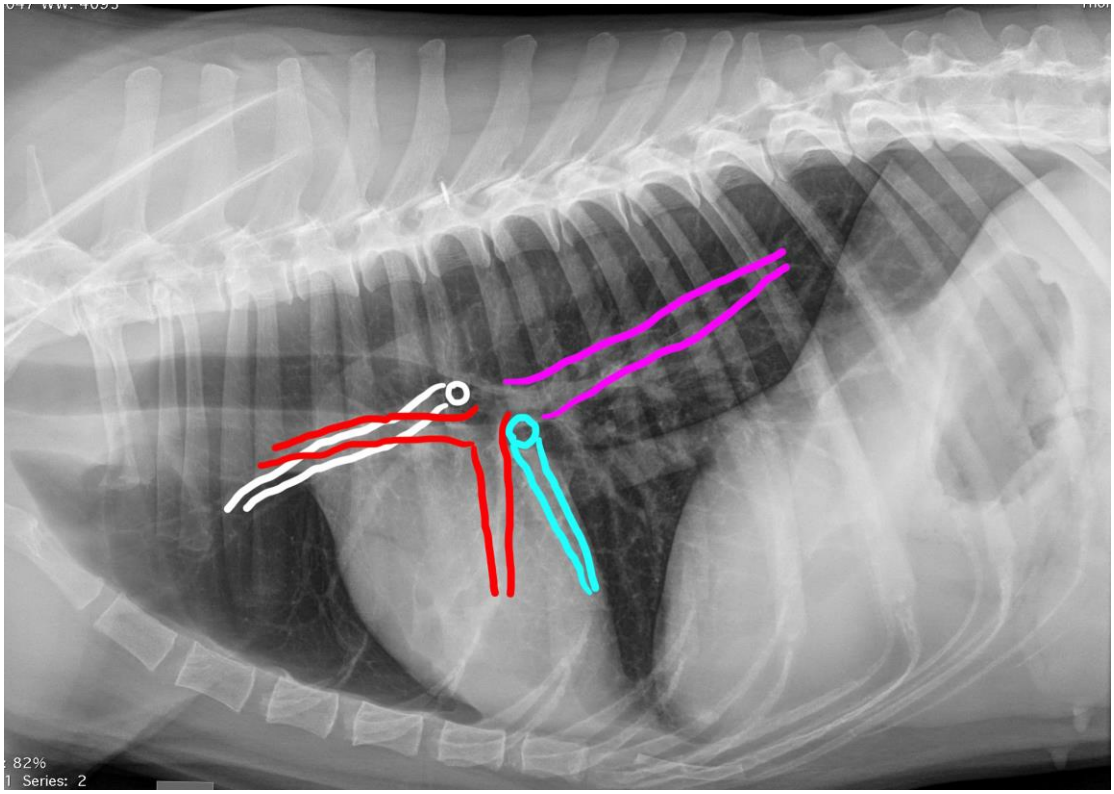


The difference between the appearance of the lungs in a radiograph taken at the expiratory (above left) and inspiratory (above right) pauses. Better lung detail is seen at inspiration (or with manual inflation).

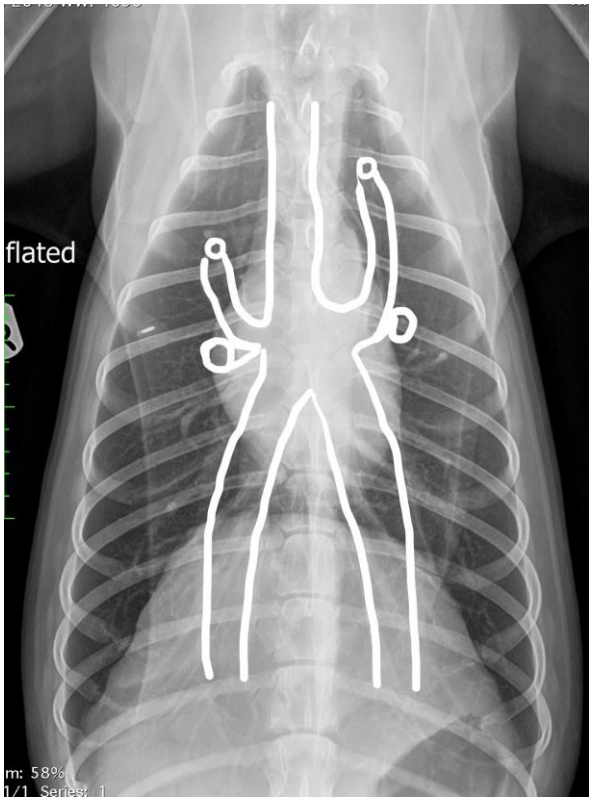
The goal for a **good radiographic exposure** of the thorax is to allow a wide range of grey scale, since the thorax contains a greater variation of densities (ranging from lucent gas to opaque bone); and a high kV, low mAs combination will facilitate this. Now most practices use digital radiography, there is a greater inherent ability to display a range of greyscales that reduces the need for this to some extent; however adds the additional need to ensure that the correct processing algorithm is selected to optimize the image. This is preset into the digital processing workstation (so one can select “thorax” which should be optimized); if the presets do not give a pleasing image it is best to contact the digital imaging system support.

Finally, and perhaps most importantly, an **adequate number of views** must be obtained to ensure that lesions are not overlooked. Given the dependent lung relatively collapses, smaller pulmonary lesions can be masked if they lie in the dependent side. Therefore, for screening for pulmonary disease, both lateral recumbency radiographs must be obtained. Studies have shown that a third (orthogonal) view (either the DV (dorsoventral) or VD (ventrodorsal)) increases ability to identify pulmonary nodules by a small fraction; also the orthogonal view is essential for lesion lateralization and triangulation of other pathologies; so for pulmonary disease three views is advised. For other screening procedures (for example, cardiac disease), a lateral and DV might suffice.

On a gross level, both cats and dogs have **7 divisions of the lung** – on the right there is right cranial, middle, right caudal and the accessory lobe; on the left the left cranial (with a cranial and caudal subsegment) and the left caudal. The bronchial tree can be seen dividing into these structures:



On the right lateral view (noted since the diaphragmatic crura are parallel to each other and the CVC goes into the most cranial crus); the bronchi are as follows: right cranial (white), left cranial (red), right middle (blue) and the caudal lobes (pink, superimposed upon each other in this dog, although they might be slightly separated in others).



On the DV, the bronchi can be delineated as above.

The lungs should be assessed on a sublobar level, by looking for abnormalities of opacity. The lungs may be increased in opacity (which can divided into a diffuse increase, classically divided into bronchial, interstitial and alveolar patterns; and a more focal/nodular increase) or decreased in opacity (more lucent than usual). Less commonly, the lung parenchyma might mineralise. These changes will be discussed in turn.

Diffuse increase in opacity

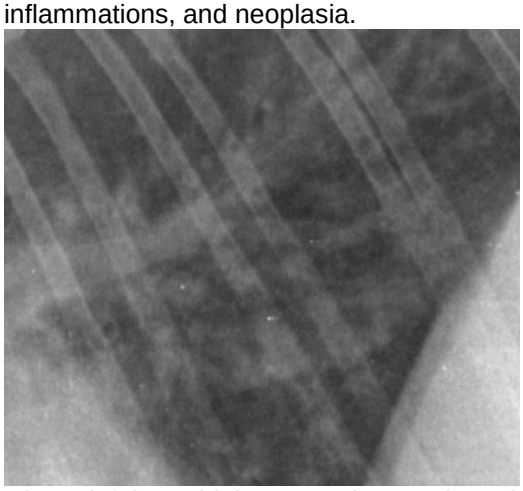
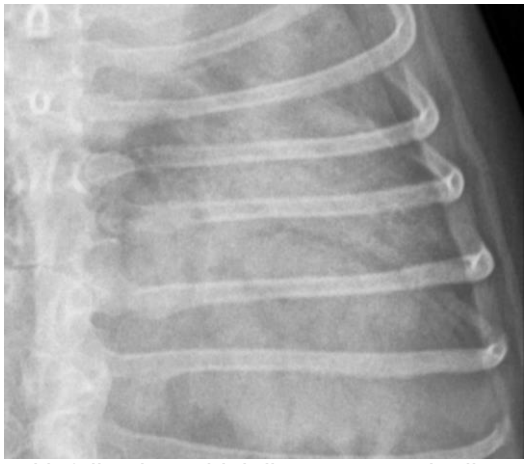
The traditional way to discuss increased opacity is to divide the lesions into a lung pattern – bronchial, interstitial, alveolar.

A bronchial lung pattern is where the bronchi are seen more distinctly (i.e. either there is increased opacity of the normal thickness bronchi by mineralization, or the bronchial wall becomes thickened). The main lobar bronchus lies between the lobar artery and vein (with the vein in the caudal lobes lying medial to the bronchus, and those in the cranial lobes lying ventral to the bronchus), however the bronchial walls are usually not seen (other than close to the hilus). If the bronchial walls become thickened, in long axis the walls are an additional pair of thin linear structures inside the artery and vein (tapering to the periphery) and if seen end on, they form a ring or doughnut. If the wall is visible due to mineralization and remains thin, this may be normal due to ageing and in certain breeds (e.g. chondrodystrophic and larger breeds). The mineralization does not extend out into the periphery and remains hilar. A few diseases that cause soft tissue mineralisation, such as hyperadrenocorticism, will also lead to mineralised bronchial walls.

Diseases that cause true bronchial wall thickening include eosinophilic bronchopneumopathy, feline bronchial disease, bronchitis (allergic, irritant, due to underlying infection), parasites (angiostrongylus, aleurostrongylus, crenosoma); with peribronchial cuffing in particular seen with bronchial oedema and inflammation. Many of these diseases will have more than one lung pattern present; for example eosinophilic bronchopneumopathy always has a bronchial pattern but commonly has concurrent interstitial, alveolar and even nodular patterns. Bronchopneumonia is a disease that is always seen as a mixed pattern, usually involving some bronchial pattern and some alveolar pattern.

The alveolar lung pattern has the hallmark of effacement of the outer aspects of the pulmonary vessels with soft tissue opacity due to flooding of the alveoli with cells or fluids.

This leads to an “air bronchogram” where the only air filled structures are the air filled bronchi passing through otherwise soft tissue opacity filling the usually air-filled lung. The types of fluid that might fill the alveoli are oedema and haemorrhage, and the types of cells are inflammatory or neoplastic, therefore alveolar lung patterns are seen with cardiogenic and non cardiogenic oedemas, haemorrhage /contusions, pneumonia and other pulmonary inflammations, and neoplasia.

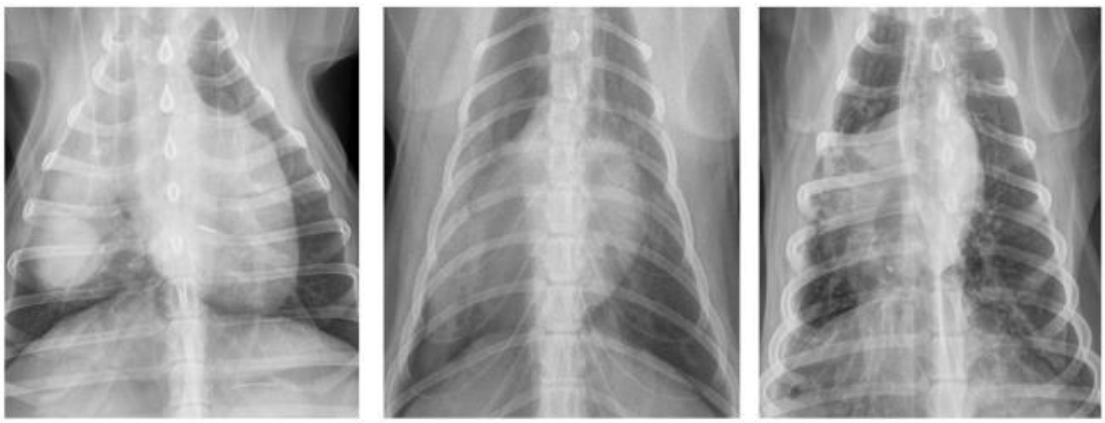



Above left bronchial pattern close up in patient with feline bronchial disease, note the lines and doughnuts; above right is an airbronchogram indicating an alveolar lung pattern in a patient with non cardiogenic oedema.

The **interstitial lung pattern** is the hardest to identify, partly because it is present in so many cases due to poor technique and patient factors, since it leads to a diffuse sense of increased opacity of the lungs without effacement of the edges of the vessels, which can be seen with obesity, ageing, and a poorly inflated lung. It may be structured (often called linear or reticular) where the pattern appears as lines across the lung field, or a more ill-defined, hazy increase in opacity without structure. It is rarely seen on its own, often seen in combination with bronchial and/or alveolar patterns.
Oedema will lead to an interstitial pattern before the alveoli are completely flooded, many infectious diseases will have an interstitial component (for example, mycobacterial disease, pneumocystis carinii), and some neoplasms might have metastasis that are diffuse throughout the lungfields without truly becoming nodular (although a micronodular pattern is really a subset of interstitial) for example lymphoma and carcinoma metastasis. The most characteristic disease associated with an interstitial lung pattern is pulmonary fibrosis, most associated with the WHWT although other breeds and cats may develop this.
The majority of lung patterns are mixed, and differentials should be given first for the predominant lung pattern, finessed into a more manageable prioritized differential list using assessment of where the pattern is distributed on the radiograph, and integration of signalment and clinical presentation.

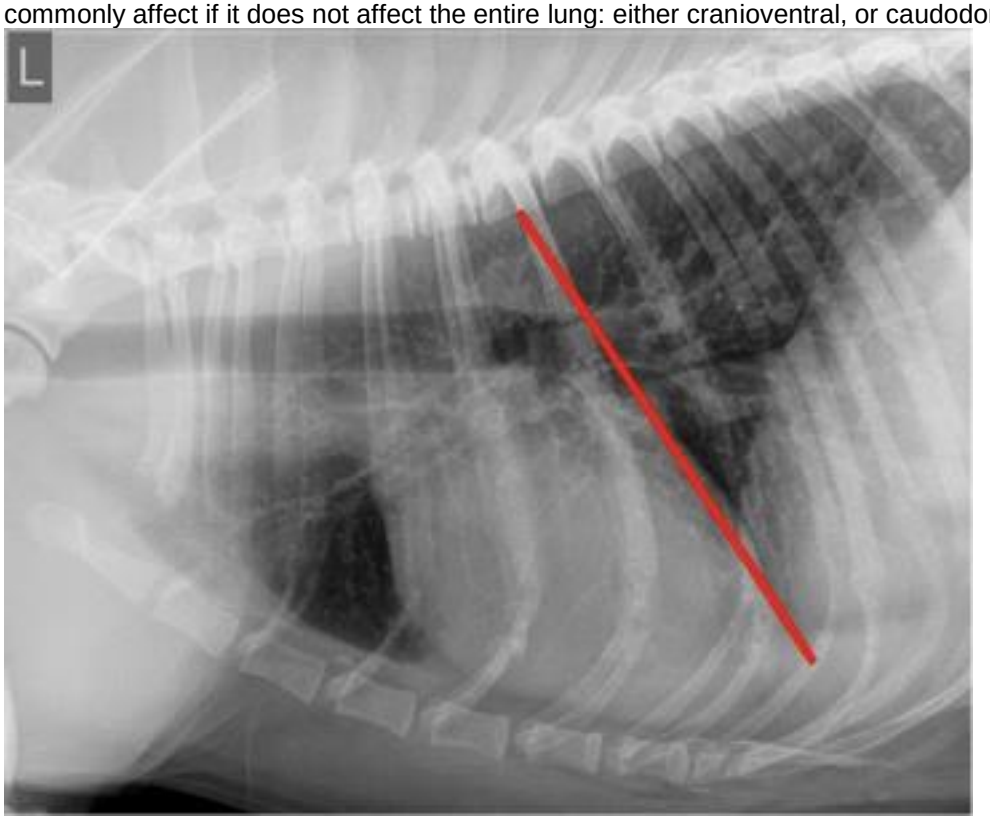
The alternate approach to assessing increase in pulmonary opacity encourages the reader to ask a series of questions of each radiograph. It is summarized in: Nykamp et al. (2002) Radiographic signs of pulmonary disease: An Alternate approach. Compendium Cont. Education 24:1:25-34.

The first step is to assess whether the radiograph is normal or not. This is mostly based on the opacity of the lungs, taking into account the degree of inflation (less inflated will be more opaque relatively). Normal ageing changes can be also excluded at this point. The lungs should be categorized according to size: there is a different set of differentials for lungs that have reduced in size (demonstrating atelectasis) than those that are either a normal size or enlarged (due to a mass, being hyperinflated or trapping gas). One of the easiest markers for size if the change affects one side is to look for mediastinal shift on the DV. Most causes of atelectasis are iatrogenic, such as GA, however some diseases that cause bronchial obstruction will lead to reduced size of the lobe.



Using mediastinal shift to assess for changes in lung volume in lung lobes with soft tissue opacity: Above left shows mediastinal shift away from the lesion in a patient with an expanding lung (neoplasia); above central shows consolidation of the lung however a normal size is maintained (no mediastinal shirt, seen here with contusion); and finally above right with collapse of the right middle lobe due to GA related atelectasis with mediastinal shift toward the lesion.

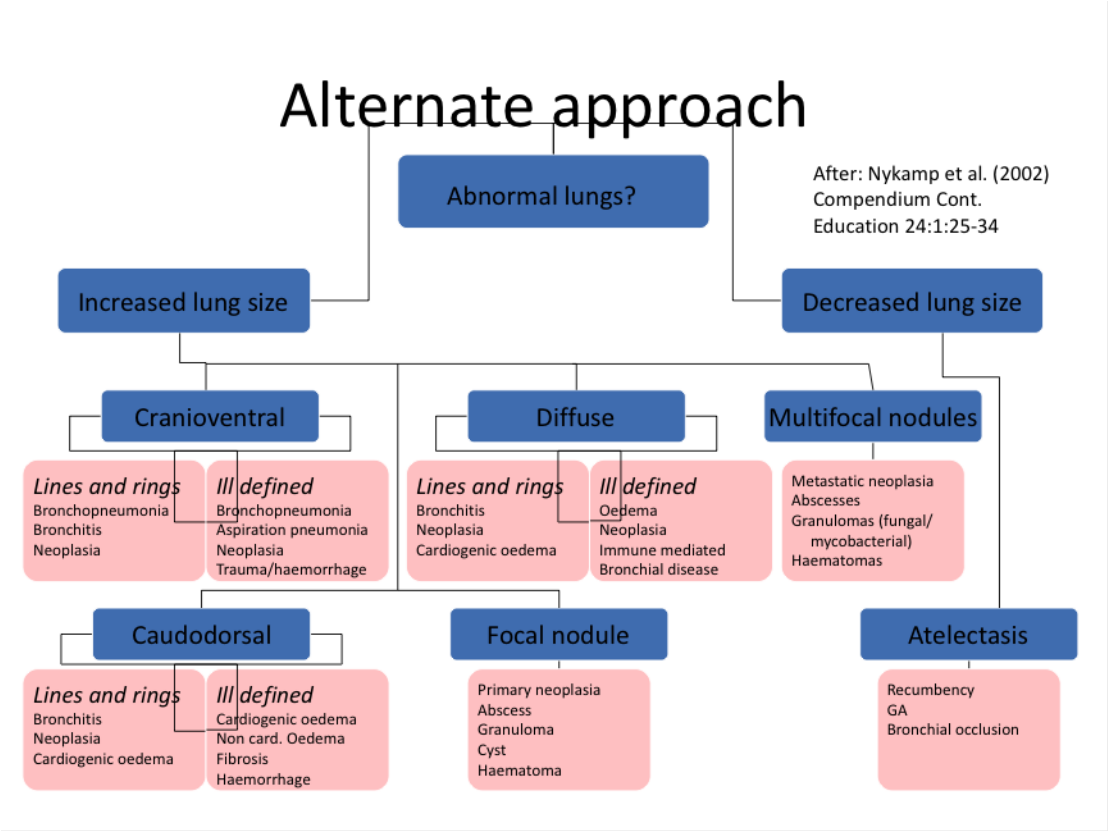
The next question for lungs that are not reduced in size is: where is the pathology? Location is very helpful to narrow the differential list. There are two locations that the pathology could commonly affect if it does not affect the entire lung: either cranioventral, or caudodorsal.



Dividing the lungs into cranioventral (left and right cranial lobes, and right middle lobe) and caudodorsal (caudal lobes and accessory lobe).

Certain diseases affect certain locations more frequently, for example aspiration (and other) pneumonias more commonly affect the cranioventral lungfields, however non-cardiogenic oedemas typically affect the caudodorsal regions. Pathology may be diffuse, such as many cardiogenic oedemas, which affect all lung lobes. A final category for location is a nodular distribution – this could be a single nodule, or multiple nodules, with appropriate differentials for both.

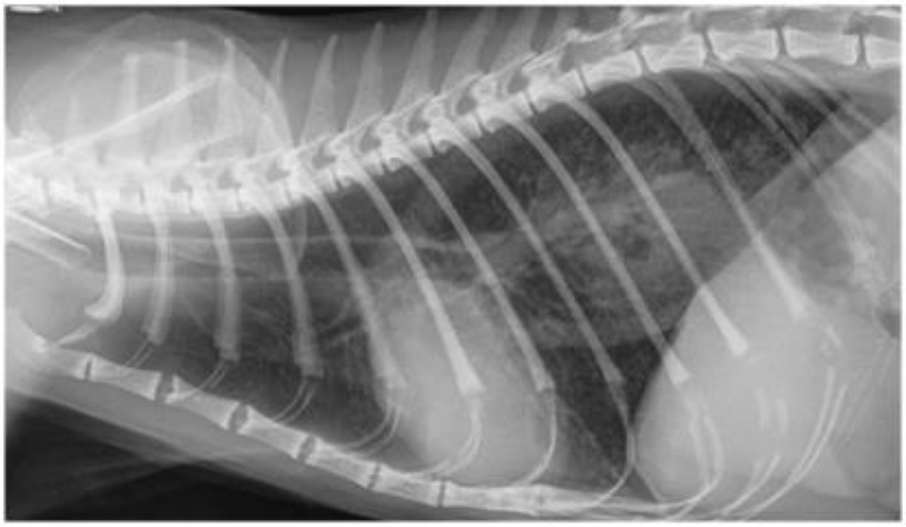
Finally, there is assessment of the type and severity of increase in opacity. This is divided into ill-defined increase in opacity, where features similar to alveolar pattern are assessed (such as air bronchograms, lobar signs and effacement); and lines and rings (which are the features assessed as a bronchial pattern in the traditional approach). Severity is defined as the degree of increased opacity (i.e. the degree of “whiteness of the lungfield”).



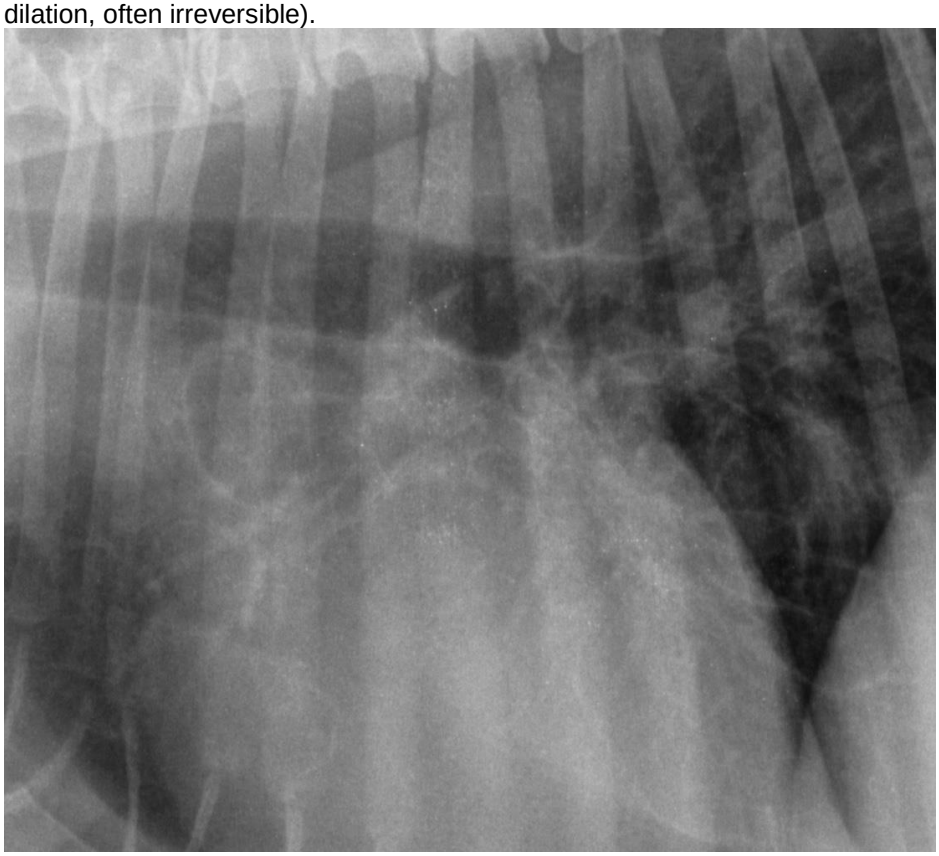
The above algorithm is a cut down version of the much fuller version that is included in the compendium paper, and the reader is encouraged to access the full paper.

Both approaches assess the same things but from a top down or bottom up approach, and the experienced clinician/radiologist will come to the same conclusions and differential lists with both approaches. It is best to find which method suits your way of working, which may be to use one or other of the systems, or even a combination of both; which ever way, the most important thing is to have a system which you use for each radiograph assessed since this will avoid oversight, and also lead to better interpretation with time.

Not all pulmonary disease cause increased opacity, some lead to reduced opacity or lucency of the lung. Given the lung is usually predominantly lucent (black) on a radiograph, this usually means regions of lung that have fewer or no lung markings present. Focally this may be due to the presence of a pulmonary bulla (a thin wall round structure that is filled with gas) or pleural bleb (similar air-filled thin walled structure seen marginally. Both of these may be congenital or acquired, and are often clinically silent; however may rupture and lead to a pneumothorax. Gas may also be seen focally in cases of cavitation of a pulmonary nodule. Cavitation occurs when a nodule has a necrotic centre, which communicates with an air-filled bronchus, allowing the necrotic centre to be replaced with gas. This appears as an irregular gas filled centre with a thick soft tissue rim. Lesions that can have central necrosis are predominantly neoplastic, however abscess and (rarely) granuloma could also be considered.

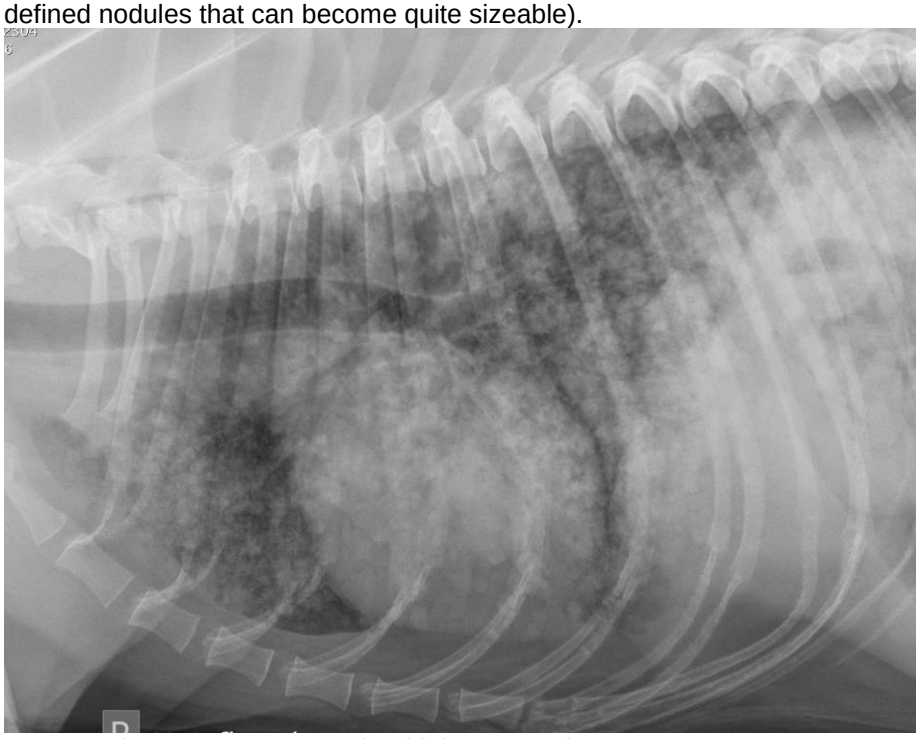


Cavitary mass lesion due to central necrosis in a cat with a primary pulmonary carcinoma.
More diffuse reductions in pulmonary lucency can be seen with areas of emphysema (defined as the permanent enlargement of airspaces distal to the terminal bronchioles and the destruction of the alveolar walls) that is secondary to severe chronic pulmonary disease – bronchitis, or pulmonary fibrosis for example. It may appear as rounded regions of emphysema (bullous emphysema), and may be associated with bronchiectasis (bronchial dilation, often irreversible).

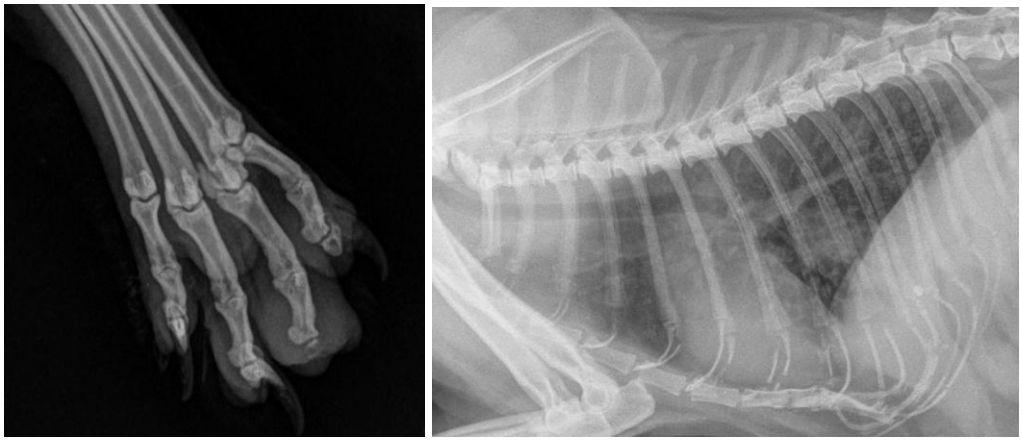


Area (arrow) of bullous emphysema and bronchiectasis in a dog with chronic bronchitis
Increased pulmonary lucency might be seen with pulmonary air trapping, where bronchial disease allows the bronchus to act as a one way valve (gas is allowed in but not out, for example in cats with mucous plugs in the bronchus with feline bronchial disease). In this way, one lobe or part lobe will be relatively more lucent. Lung lobes also relatively overexpand if there is a portion of lung missing (surgical resection) or collapsed, so the lungfield expands to fill the thoracic cavity.

Pulmonary nodules form a separate group of diseases, with nodules varying in size from very tiny (miliary or micronodular patterns, really a variety of interstitial lung pattern) to very large (cannon ball). It is important to identify that a nodule is truly a nodule, and not an artifact (for example, a superimposed nipple/cutaneous mass (nipples have characteristic locations, and cutaneous masses are not surrounded by air, therefore do not stand out as clearly as an air surrounded pulmonary nodule). End on vessels can be distinguished from nodules since they are relatively more opaque than a nodule would be of that diameter (since the X-ray beam is being attenuated by a greater depth of tissue along the length of the vessel than it would be for a round nodule). Finally, pulmonary osteomata or pulmonary heterotopic bone formation are small very opaque (mineral opacity) nodules with sharp angular margins identified often ventrally and round the margin of the lungfield as an incidental ageing finding in older dogs, and should not be mistaken for multiple nodules.
Differentials of a **solitary soft tissue pulmonary nodule** on a radiograph are: cyst, haematoma, abscess, neoplasia, granuloma (acronym CHANG). Multiple nodules can be the same, however multiple neoplasias (metastasis), abscesses or granulomata are more common than multiple cysts or haematomata. It is not possible to determine the cause of nodules from a radiograph alone; and while neoplasia is the most common cause of pulmonary nodules it is worth considering that multiple nodules are seen with non-neoplastic conditions (e.g. eosinophilic bronchopneumopathy, infectious diseases such as mycobacterial, fungal disease, leptospirosis and toxoplasma, and DIC). It is not possible to determine the cause of nodules from a radiograph, however clues might be discerned – presence or not of lymphadenopathy, appearance of the nodules (for example, carcinomas have a variable appearance including miliary nodules, haemangiosarcomas may have hazy margins to the nodules (local reaction or haemorrhage), sarcomas tend to have sharply defined nodules that can become quite sizeable).



Haemangiosarcoma metastasis with hazy margins.
A quirk of cats is the **lung - digit syndrome**, in which cats have primary masses in their lungs which metastasize to the digits – in a dog, an aggressive lesion in the digit and the lung would be presumed to be a digital neoplasia with pulmonary metastasis, however this can be the other way around in the cat.



Digital metastasis from a primary pulmonary carcinoma in a cat.

Marie's disease is a curious phenomenon by which space occupying lesions in the lungs lead to bilaterally symmetric palisading periosteal reactions seen on the axial and abaxial surface of the diaphysis of the limb bones starting distally (metacarpi/metatarsi) and progressing proximally. The space occupying mass in the thorax is commonly neoplastic, although may not be neoplastic in origin (and rarely, the mass is abdominal rather than thoracic). Uncommonly, there is mineralisation of pulmonary tissue. This may be diffuse (metastatic mineralisation due to systemic disease such as hyperparathyroidism, hyperadrenocorticism, uraemia), or focal. The majority of small foci of mineralisation are benign and incidental, for example pulmonary heterotopic bone formation in older dogs, and broncholithiasis/mineralised peribroncial glands in cats. Some non-neoplastic pathologies will mineralise for example some granulomas (e.g. Histoplasmosis, not in the UK); and aspirated foci of barium following from a prior barium swallow may mimic mineralized nodules. Primary lung neoplasias will occasionally mineralise, and in cats this is a marker for malignancy. Very rarely, osteosarcoma metastasis will be mineralised.