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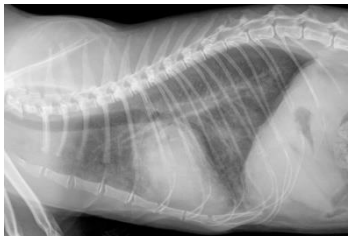
Thoracic Radiology

Mini Series

Session Two: The cardiovascular system – should I just do an echocardiogram?

Abby Caine
MA VetMB CertVDI DipECVDI MRCVS
European and RCVS-Recognised Specialist in
Veterinary Diagnostic Imaging



Session 2 - Monday 23rd July 2018 1pm-3pm

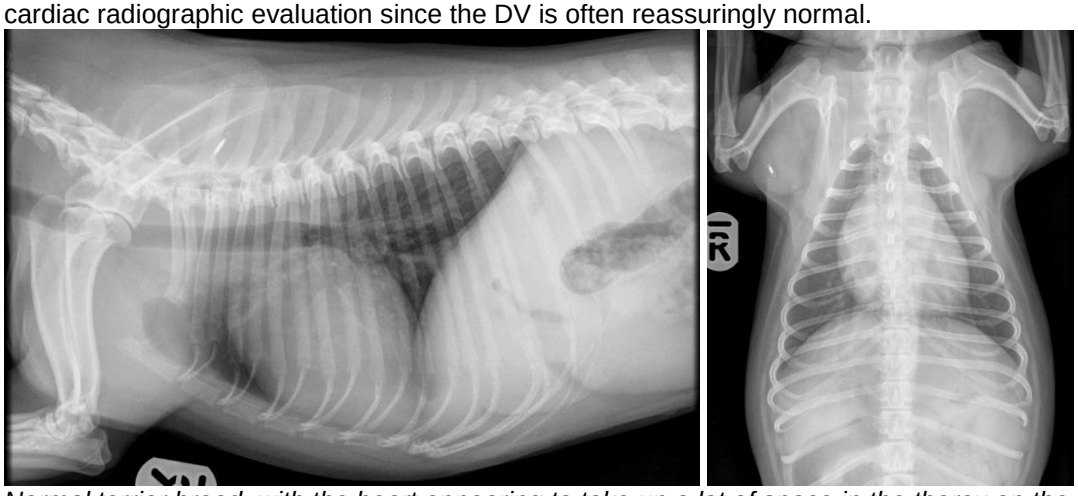
The cardiovascular system – should I just do an echocardiogram?

This session will focus on the role of radiography in the cardiac patient – assessing if the patient is in cardiac failure; and monitoring for disease progression. It will also discuss findings for assessing cardiac shape and how this is complemented by echocardiography.

- The normal heart shape of different breeds
- What happens to the cardiac shape with pathology?
- What can changes to the pulmonary vasculature tell us about the patient's disease?

So, echocardiography is so sensitive and specific for diagnosing cardiac disease, it must have replaced thoracic radiography by now surely? Certainly, it is a better way of diagnosing cardiac disease. However, there is still a valuable role of thoracic radiography in giving an overview of the cardiac patient, given it allows the pulmonary vasculature to be assessed for the presence of congestion, and allows for the presence of congestive heart failure to be identified. It also provides a good baseline for the monitoring of a cardiac patient over time, which is not as operator dependent as echocardiography. And of course, it is important to have an understanding of cardiac disease on the thoracic radiograph since cardiac pathology could be an unexpected finding on taking a thoracic screening radiograph for other purposes.

This webinar will run first through the huge variability of "normal" – there is a great variation in the cardiac shape and size **varying with dog breed**, that makes it very challenging to have a definitive rule of thumb as to when cardiomegaly is present, and care should be taken to avoid overdiagnosing this. There is extreme variation between a very deep/narrow chested dog such as a Doberman, which has a very tall upright heart (which might appear slightly narrow on the lateral and very round on the DV since it is seen end on) compared with a barrel chested breeds such as a Bulldog (which will have a heart that appears to take up a large amount of space on the lateral view, however appears normal on the DV. Athletic breeds such as Greyhounds and Springer Spaniels genuinely do have slightly large hearts; as do retriever breeds (with a parallelogram shape to the heart) and some terrier breeds that appear to have large rounded hearts on the lateral. Note, many of the breeds mentioned above which might have a questionable heart size on the lateral have a normal appearance on the DV, so it is essential to have at least two orthogonal views when performing a cardiac radiographic evaluation since the DV is often reassuringly normal.

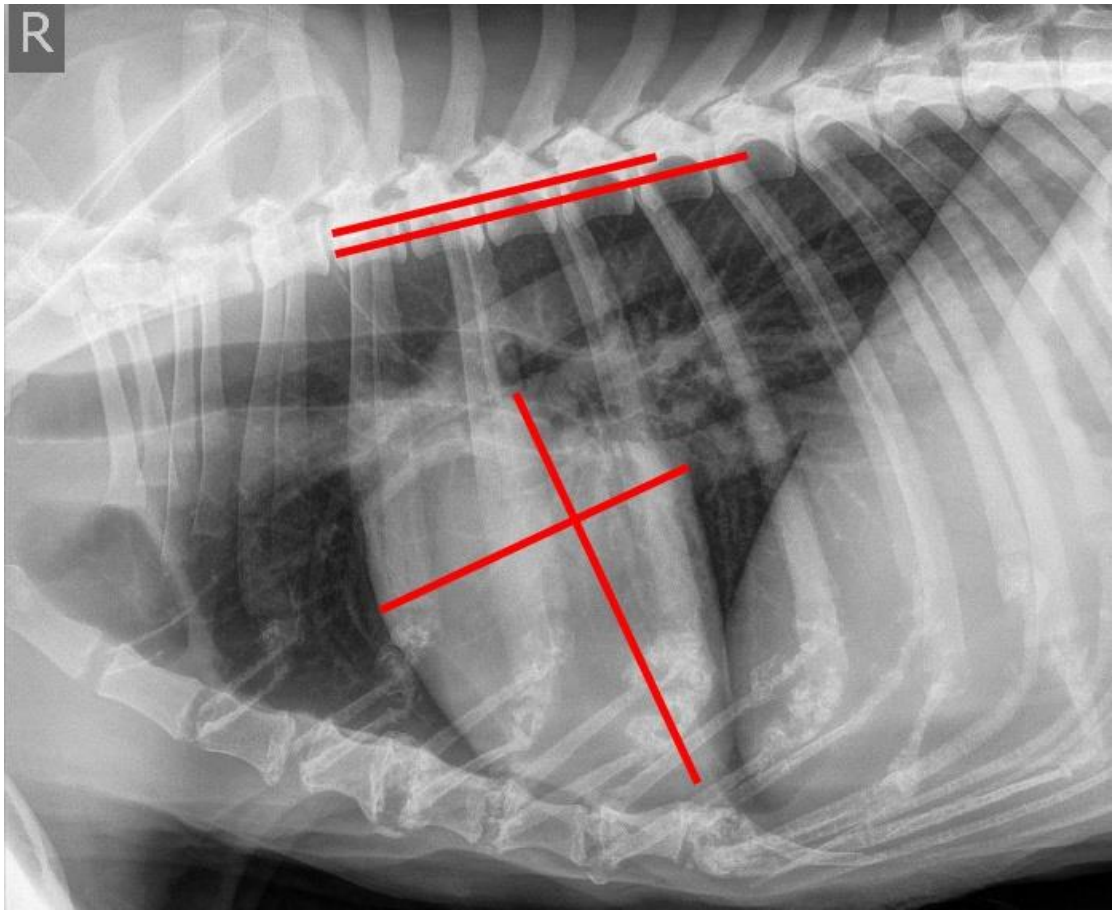


Normal terrier breed, with the heart appearing to take up a lot of space in the thorax on the lateral view, confirmed as normal on the DV.

Two methods are available to assess **cardiac size** – subjective and objective (namely, the vertebral heart score). It has been shown in experienced hands, either performs just as well, however the vertebral heart score is more reproducible for inexperienced film readers.

Subjective methods involve comparing the cardiac silhouette (comprising the heart and its surrounding pericardial sac, since these can not usually be distinguished radiographically) to the thoracic margins. On the lateral, the height of the heart would not be expected to exceed 2/3 of the height of the thoracic cavity; and the width of the heart would not be more than 2.5-3.5 intercostal spaces in width. Increased height would indicate left sided enlargement, increased width right-sided enlargement. On the DV radiograph, the width of the heart at its widest would not exceed 2/3 of the width of the thorax; if increased this might be due to left or right sided enlargement.

The **vertebral heart score (VHS)** is measured on a lateral radiograph, usually the right lateral. A line is taken from the ventral aspect of the carina to the apex of the heart. Another line is made perpendicular to this at the level of the caudal vena cava (CVC). Both these lines are transposed to the spine, starting at the cranial aspect of T4, counting the number of vertebrae that the line measures (to 0.1 of the vertebral body, with each vertebral body measurement including the disc space caudal to the vertebra). The two numbers of vertebrae are added together, to give a single number.



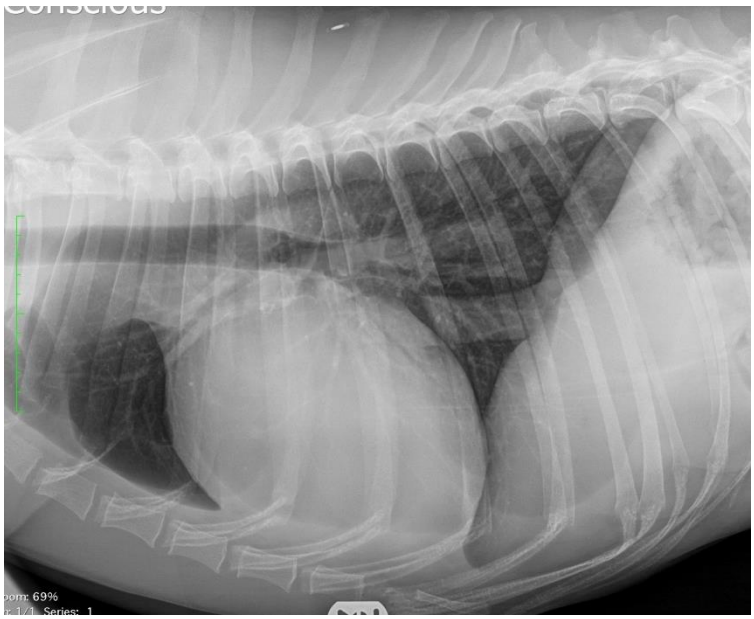
Performing a VHS – 9.7.

The original paper suggested that dogs should have a VHS of 9.7+/- 0.5. Several papers have followed confirming that many dog breeds lie outside of this range (and even outside the larger more generous range of 9.7 +/- 1). One study assessed 6 breeds, and concluded that only German Shepherd Dogs and Yorkshire terriers are within the original range; while breeds which frequently have significant cardiac disease lie outside the range - Boxers: 11.6 +/- 0.8; CKCS: 10.6 +/- 0.5; Labradors: 10.8 +/- 0.6 and Whippets: 11 +/- 0.5. This study also concluded that the VHS had a poor sensitivity and specificity for distinguishing between cardiac and pulmonary disease. Dogs that tended towards a dilating type heart disease (e.g. CKCS and Yorkshire terriers) allowed the VHS to perform better than other breeds such as Boxers, where the cardiac disease tends to be more associated with concentric thickening. Further studies have confirmed that some small breeds (Pugs, Pomeranians, Boston terriers and Bulldogs) also lie outside of the normal VHS range. So although the VHS might be easy and repeatable with accuracy for a novice to perform, is there any point given all the pitfalls…? One significant use is in monitoring of a single patient over time, where a measurement can confirm progression of cardiomegaly, without the need for comparing an individual patient to a published range.

It is worth noting that even with a single patient being monitored, the radiographic technique should be the same to allow comparison, since inflation reduces the vertebral heart score, and some authors indicate that there are differences in the VHS on a right lateral compared to a left lateral.

Not all causes of cardiac disease will have radiographic signs of cardiomegaly. If there is a rhythm disturbance, or a disease that is acute or causing purely concentric thickening, the radiographic appearance may be normal.

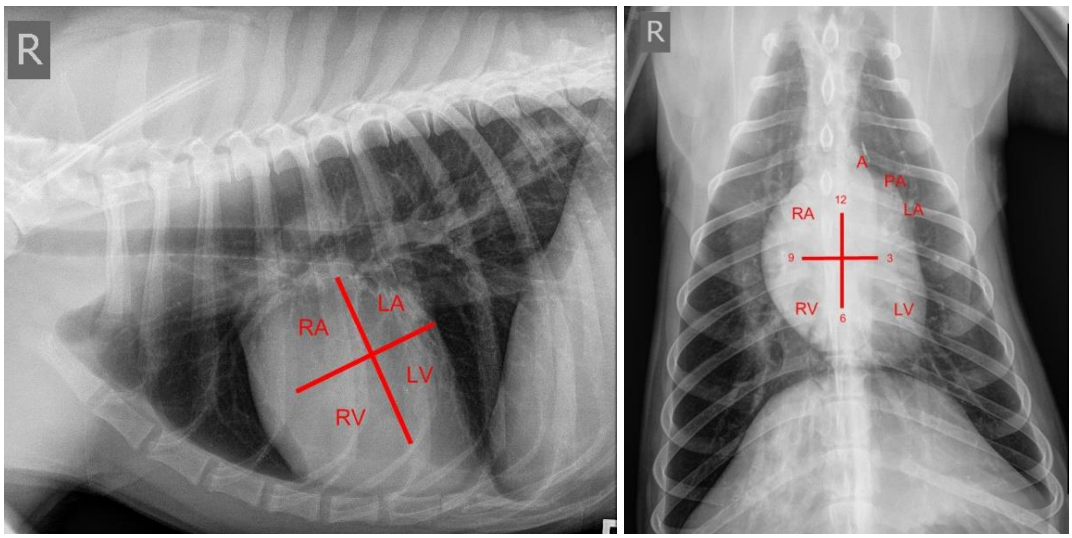
Global cardiomegaly: The entire heart can be enlarged due to systemic disease, for example anaemia in cats leads to a mild global cardiomegaly, as can fluid overload. Also, bilateral cardiac disease (e.g. bilateral atrioventricular valve insufficiency), global myocardial disease (DCM (dilated cardiomyopathy), acromegaly, metabolic disease, amyloidosis, carnitine deficiency, toxicity (e.g. Doxirubicin), and many other uncommon causes). The cardiac silhouette might enlarge with pericardial disease also, most commonly a pericardial effusion. Pericardial effusions will usually have a very crisp outline, since there is no cardiac motion in the time of the exposure to lead to blurring as most cardiac radiographs will have (DCM may also have sharp margins, given the poor motion of the ventricular walls). A pericardial effusion might be idiopathic, however it might be due to neoplasia and an echocardiogram is needed to establish if a heart base mass (haemangiosarcoma, chemodectoma, mesothelioma, lymphosarcoma, others less commonly) are present. Very rarely, pericardial effusions might be inflammatory such as septic (following penetrating wound or foreign body migration), FIP (feline infectious peritonitis), autoimmune; or traumatic. Cats will not infrequently have a small effusion secondary to congestive heart failure.



Crisp round silhouette with mild globoid cardiomegaly in a dog with pericardial effusion

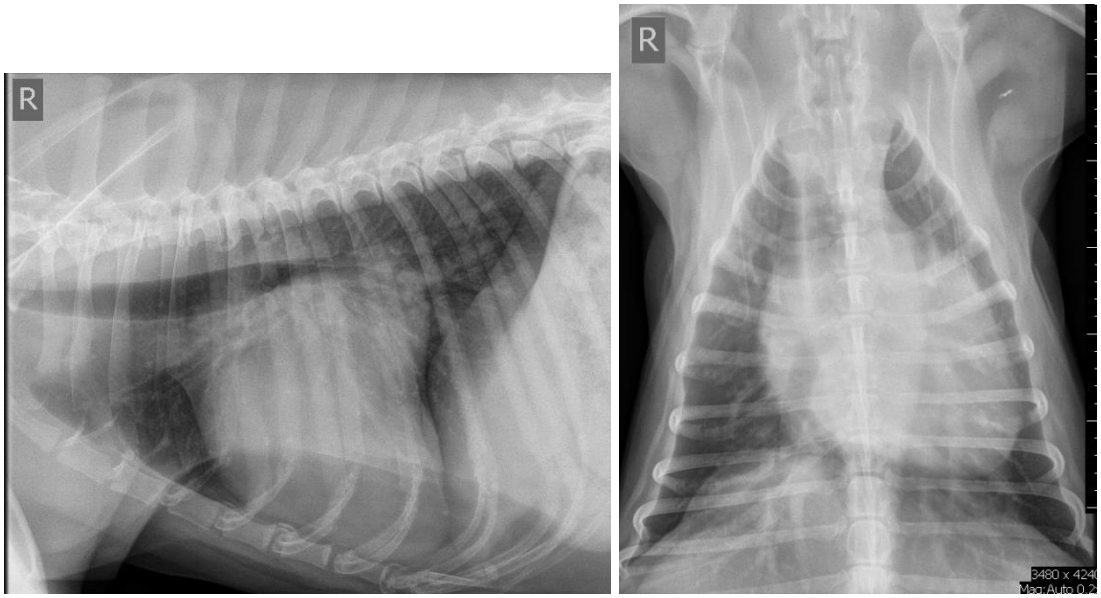
The pericardial effusion might lead to tamponade; where the less rigid right heart collapses under pressure during systole. This leads to right heart failure, so the radiographic appearance might be complicated with pleural effusion, CVC (caudal vena cava) distension and ascites.

For specific chamber enlargement, the heart can be thought of as a clock face.



Clock face analogy for identifying the cardiac chambers on the lateral (above left) and DV views.

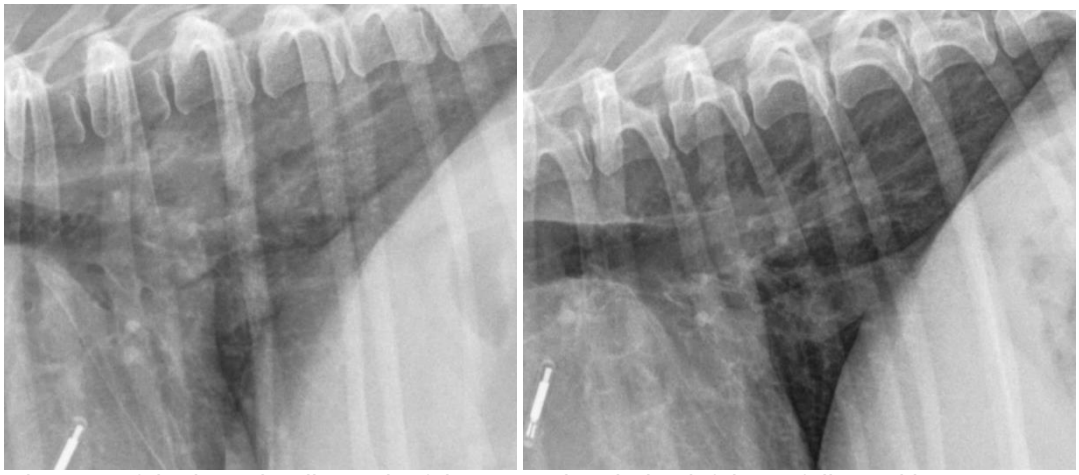
Left heart enlargement is characterised by tenting of the left atrium at 12-2 o'clock (this might lead to a straight caudal border of the heart on the lateral view), and enlargement of the left atrium on the DV separating the mainstem bronchi on the DV (the mainstem bronchi look like cowboy legs), with the left atrium sat on top of the cardiac silhouette like a cottage loaf. If the left ventricle is also increased in size, the heart often appears taller than the expected 2/3 of the height of the thorax, elevating the carina (the trachea becomes more parallel to the vertebral bodies).



Labrador with DCM demonstrating left heart failure – left atrial tent on the lateral, and a cottage loaf appearance on the DV with cowboy legged mainstem bronchi.

Enlargement of the chambers can be thought to be due to the need of the heart chamber to cope with an increased volume of blood, increased pressure within that chamber, or (in the case of the ventricles) myocardial failure.
For the left atrium, volume overload occurs due to mitral valve insufficiency (e.g. endocardiosis, endocarditis, dysplasia as seen in Great Danes, German Shepherd Dogs, Bull Terriers and cats); due to left to right shunting e.g. a patent ductus arteriosus (PDA), a ventricular septal defect (VSD) or an aorticopulmonary septal defect); with rare causes such as left atrial blood pooling due to diastolic dysfunction and endocardial fibroelastosis. Left atrial pressure overload occurs with left ventricular hypertrophy (aortic stenosis, hypertrophic cardiomyopathy (HCM), and restrictive cardiomyopathy (RCM)); and mitral valve stenosis.
For the left ventricle, volume overload occurs due to mitral or aortic insufficiency; left to right shunting (PDA, VSD), and endocardial cushion defects. Pressure overload occurs with aortic stenosis, systemic hypertension, HCM, and rarely coarctation (narrowing) or the aorta. Left ventricular myocardial failure occurs most commonly with DCM, by rarely myocarditis or myocardial neoplasia may be present.

Left heart failure is characterised by pulmonary oedema in the dog. First there will be sufficient congestion of the pulmonary veins that this might be visible on the radiograph. Pulmonary arteries and veins should be equal in size at the same level, and the caudal lobar vessels should be the same size as the 9th rib as they cross it on a DV view; and the cranial lobar vessels should be 0.5-1 times the width of the proximal third of the fourth rib at the point in which they cross rib 4. Both enlargement of the veins relative to the arteries, and enlargement of arteries and veins compared to the normal range, may be seen in pulmonary venous congestion. Following congestion, oedema begins to accumulate in the lungs; initially interstitial giving a hazy appearance to the vessels crossing the lung parenchyma; eventually becoming alveolar in nature. The perihilar region is a predilection site for this change, exacerbated by the left atrial tent also increasing opacity here.



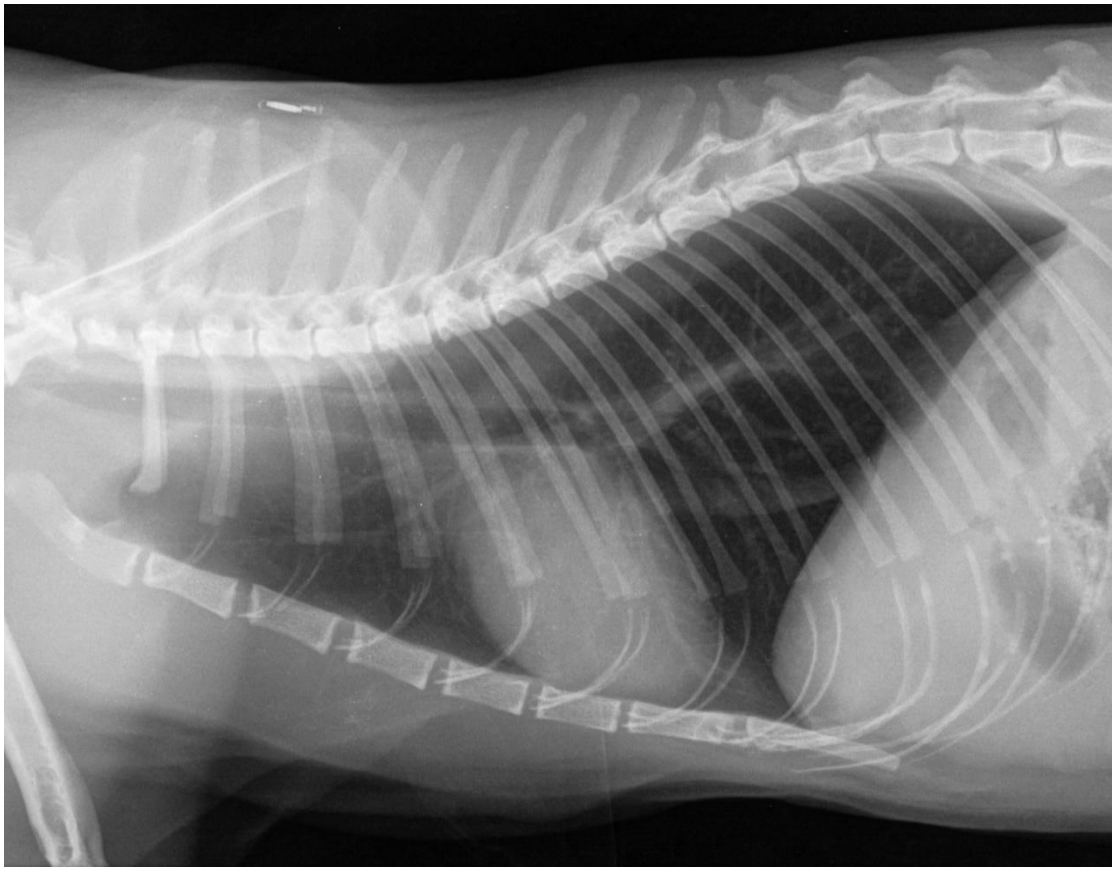
Close up of the lateral radiograph of the same dog during left heart failure with venous congestion and hazy pulmonary interstitial oedema (above left) and (above right) 3 days later following diuresis showing resolution of the heart failure (while maintaining evidence of left cardiomegaly with a left atrial tent.

Right heart enlargement is trickier to identify. Different cardiac shapes in different breeds lead to a variety of cranial cardiac silhouette appearances, and the cranial cardiac silhouette is made up from the right heart chambers. The right atrium is at 10-11 o'clock, and on the DV it is at 9-11 o'clock. Volume overload of the right atrium may occur with tricuspid insufficiency (endocardiosis, endocarditis, dysplasia, Ebstein's anomaly (valve leaflets are displaced congenitally into the right ventricle) and left to right shunting of an atrial septal defect (ASD). Pressure overload may be seen with right ventricular hypertrophy; for example due to pulmonic stenosis seen in certain breeds such as the Bulldog, reducing the flow through the right ventricular outflow tract with consequent increase right ventricular pressure; or due to Tetralogy of Fallot (the four components of this are right ventricular hypertrophy, pulmonic stenosis, an overriding aorta and a high VSD; so right and left sided blood mix prior to unoxygenated blood entering the systemic circulation via the overriding aorta (right to left shunting). Cor pulmonale is increased right-sided cardiac pressure due to primary or secondary (to lung disease such as pulmonary fibrosis) pulmonary hypertension. Very rarely, the right atrium might be enlarged due to right atrial neoplasia or a right atrial aneurysm/herniation of the right atrium though a pericardial defect.

Enlargement of the **right ventricle** leads to increased width of the heart on the lateral view, with increased sternal contact (the cardiac apex might start to tip up away from the sternum; note to assess this on the right lateral rather than on the more rounded cardiac silhouette seen on the left where this can be over interpreted). On the DV view, the heart might take on a reverse D appearance – note that all hearts have a flatter/straighter left side than right side on the DV, so to call a true reverse D, there should be bulging out of the 6-9 o'clock position. It is easy to mimic the appearance of right heart enlargement in some breeds of dogs (e.g. Retriever breeds and terrier breeds which have larger hearts on the lateral that mimics increased sternal contact); also if the DV radiograph if the thorax is rotated slightly so the sternum tips to the right, the right side of the heart might artifactually appear to bulge. For these reasons, diagnosis of right heart enlargement on the radiograph should be made with caution, supported by the clinical findings of the patient and backed up by echocardiography. Right ventricular enlargement might be due to volume overload (tricuspid insufficiency, pulmonary insufficiency, VSD, ASD, endocardial cushion defects). Pressure overload is due to pulmonic stenosis, pulmonary hypertension (cor pulmonale), or Eisenmenger's syndrome (right to left shunting of blood and cyanotic patient). Myocardial failure might be DCM, arrhythmogenic right ventricular cardiomyopathy, or uncommonly myocardial neoplasia or myocarditis.

Right-sided heart failure leads to pleural effusion, caudal vena cava distension, hepatomegaly and ascites; each of which can be seen radiographically.

What about cats though – how similar are the measurements taken? The indices used are similar; however there is actually less breed difference making fewer exceptions to the standard rules, with the cats heart typically ovoid on both views.

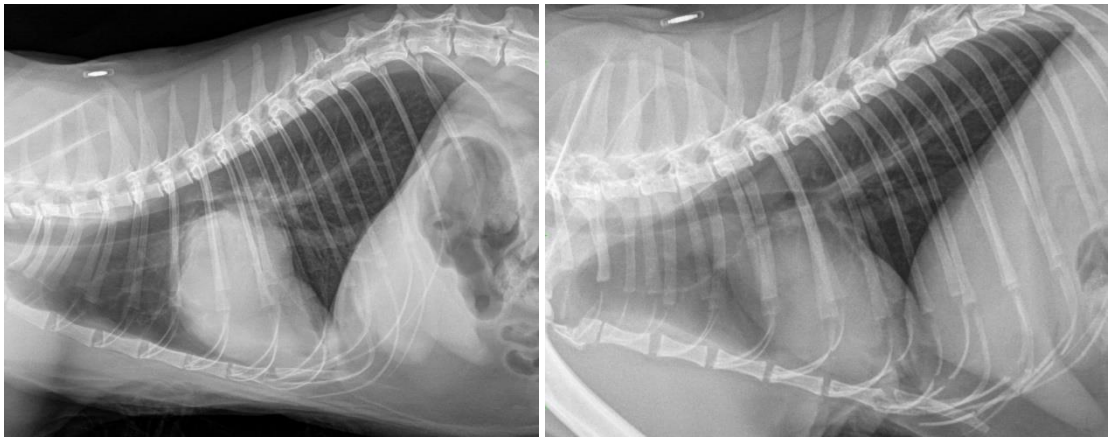


Normal cat thorax

The height of the cardiac silhouette on the lateral should not exceed 2/3 of the thoracic cavity height; while the width of the heart on the lateral should be roughly equal to the distance from the cranial aspect of rib 5 to the caudal aspect of rib 7. If this distance exceeds 4 vertebral body lengths, this is suggestive of left atrial enlargement. The width of the heart on the DV is much more consistent between cat breeds, and should be less than 2/3 width of the thorax. It is one of the most sensitive markers for cardiac enlargement, and can be compared to vertebral body lengths where it most cats are 3.4 lengths +/- 0.6.

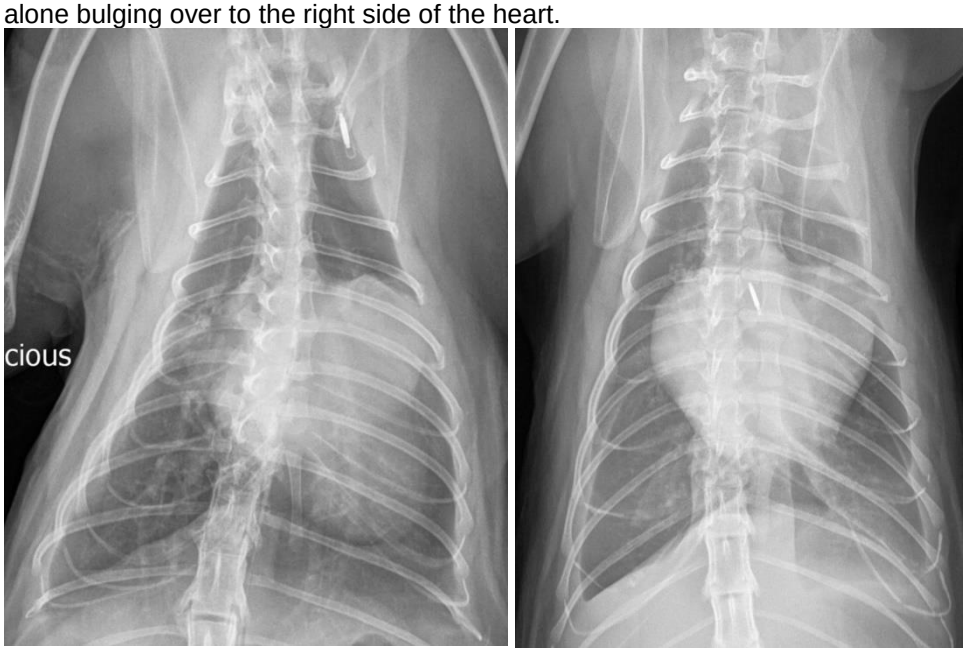
The vertebral heart score can be performed, with the range less dependent on breed – the measurement point of the base of the heart is the ventral border of the left apical vein rather than the carina (which is not always at the true heart base). The standard range is published as being 6.9-8.1; however stray DSH have a slightly tighter range (7.3+/-0.5), suggesting mild breed differences.

With **left heart enlargement**, which is the most commonly seen form of cardiomegaly given the predilection for cardiomyopathy, the atrium enlarges giving the heart a kidney bean shape on the lateral.



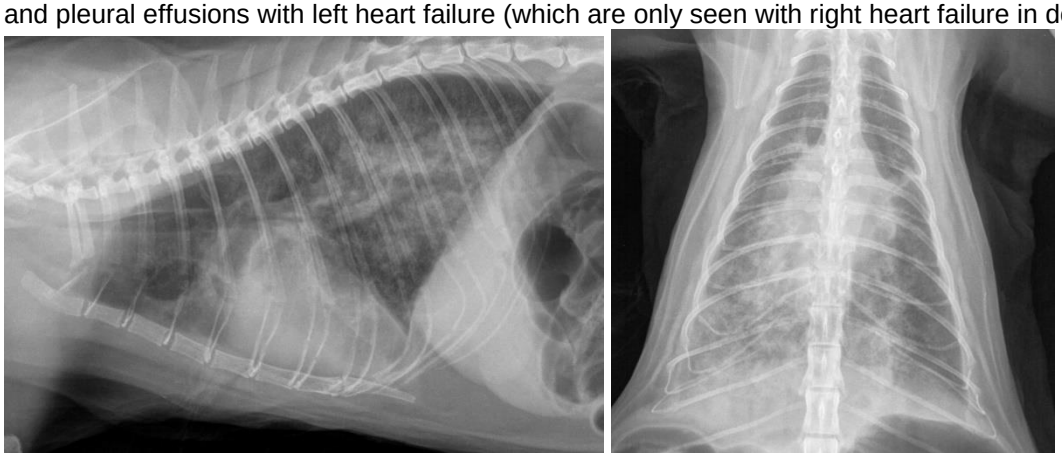
Two examples of left atrial enlargement: more marked above right giving a kidney bean shape to the heart.

On the DV, the cardiac silhouette develops a widened base (sometimes a Valentine heart shape) on the DV. Where there is a Valentine heart shape, with bulging of the base to the left and the right, this may be due to bilatrial enlargement, however might also be due to massive left atrial enlargement alone bulging over to the right side of the heart.



Cat with left atrial enlargement only (above left) and cat with biatrial enlargement (above right). Both have HCM.

Left sided heart failure in cats leads to cardiogenic pulmonary oedema in a similar to dogs, with initially pulmonary venous congestion that progresses to an interstitial or alveolar pattern. Cats have some quirks however, in that the cardiogenic oedema is less commonly bilaterally symmetric; and is often patchy and even asymmetrically distributed. Also, cats may develop both pericardial effusion and pleural effusions with left heart failure (which are only seen with right heart failure in dogs).

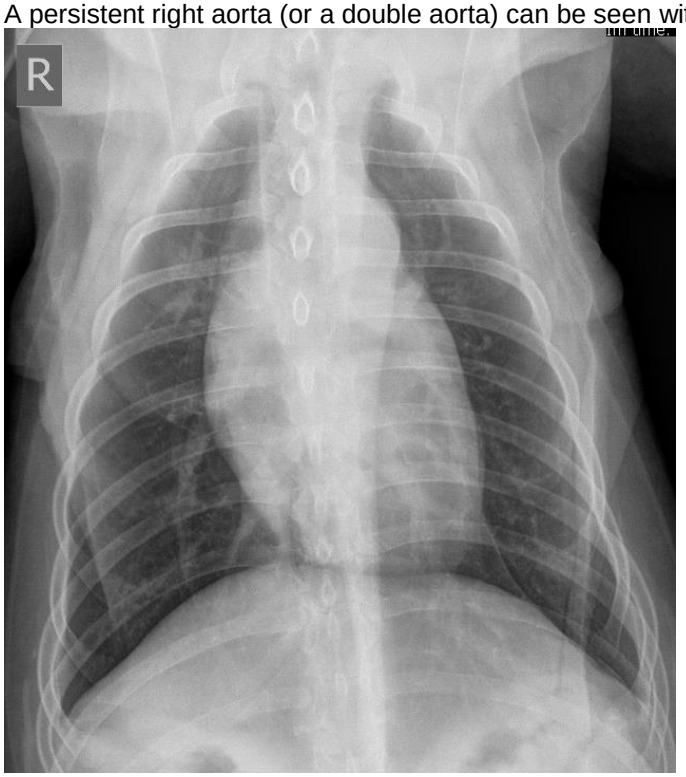


Patchy and asymmetrically distributed (left sided) cardiogenic pulmonary oedema

Right heart enlargement is less commonly seen. It may cause increased sternal contact (beware mistaking normal aging cats with hearts that have rotated so they lie against the sternum for genuine increased sternal contact); and also increased bowing of the right side of the heart on the DV.

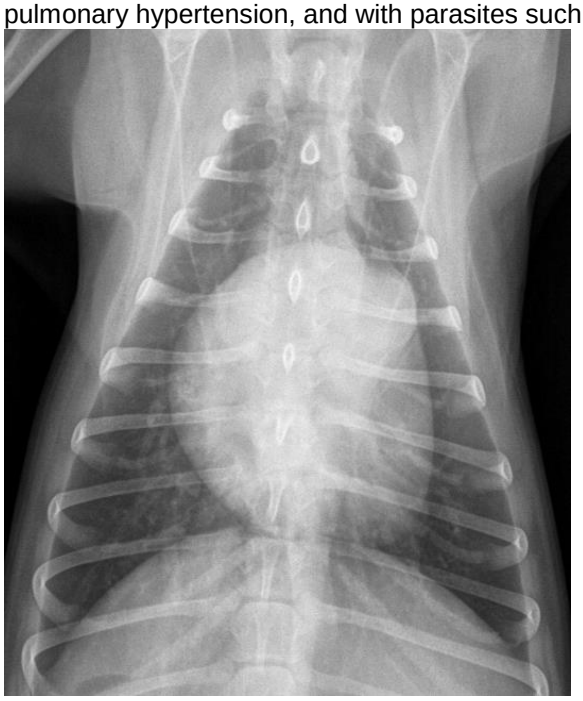
The **aorta** can be seen on the normal radiograph – the aortic arch curving craniodorsally from the heart base at 11-12 o'clock on the lateral, and bulging slightly to the left at 12-1 o'clock on the DV (the left wall of the aorta can be seen all the way along to the diaphragm on the DV as the left edge of the dorsal mediastinum). The aortic arch can be "saggier" and appear more prominent in some ageing cats and breeds such as Bulldogs. The aortic arch might enlarge with post-stenotic dilation in aortic stenosis (or coarctation, rare); in a PDA, or due to an aortic body tumour (chemodectoma).

Rarely, the aorta might be more visible since it is mineralized (this can occur in any kind of metastatic mineralizing condition such as lymphosarcoma, renal failure, hypervitaminosis D, hyperparathyroidism, hyperdrenocorticism). In other countries, Spirocerca Lupi can migrate via the aorta, leading to multiple local aneurysmal dilatations and mineralization. It is however a normal variation to have some linear/branching mineralisation of the aortic valves in older large breed dogs, without clinical consequence.
A persistent right aorta (or a double aorta) can be seen with vascular ring anomalies.

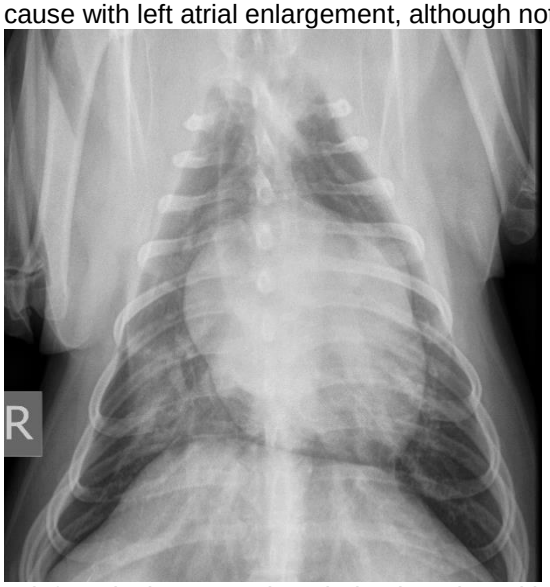


Aortic bulge at 12-1 o'clock in a patient with aortic stenosis.

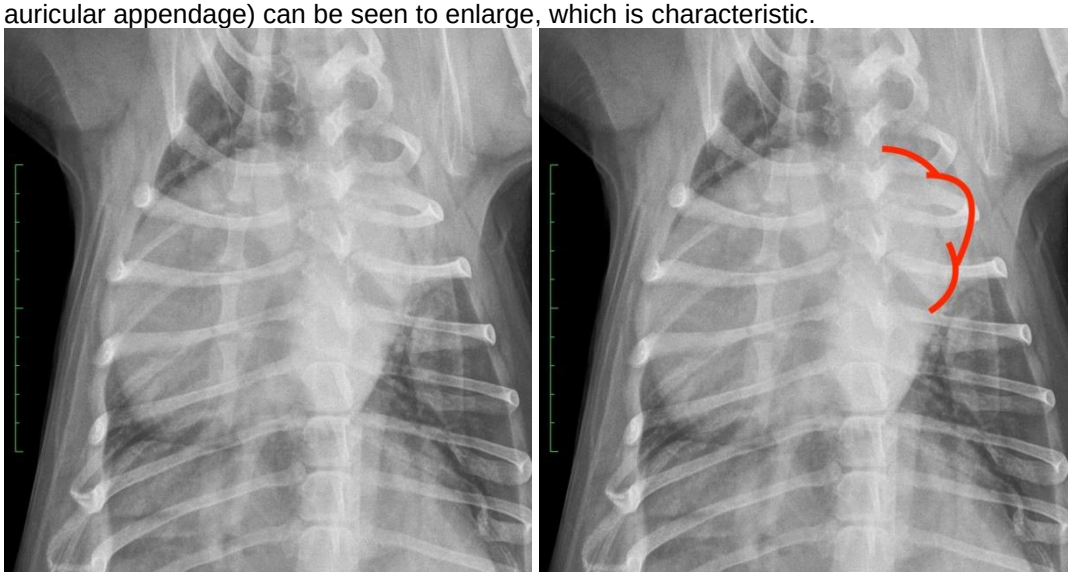
The **pulmonary artery** is identified at 1-2 o'clock on the DV radiograph. This may appear to bulge with artifact, since deep chested dogs, particularly with slight rotation of the DV view, can have a "normal" pulmonary artery skylined. If necessary, echocardiography can be used to distinguish normal from abnormal. The pulmonary artery enlarged with post stenotic dilation with pulmonic stenosis, with large left to right shunts (increased circulating blood volume e.g. PDA, VSD, very large ASD), with pulmonary hypertension, and with parasites such as Dirofilaria (not UK).



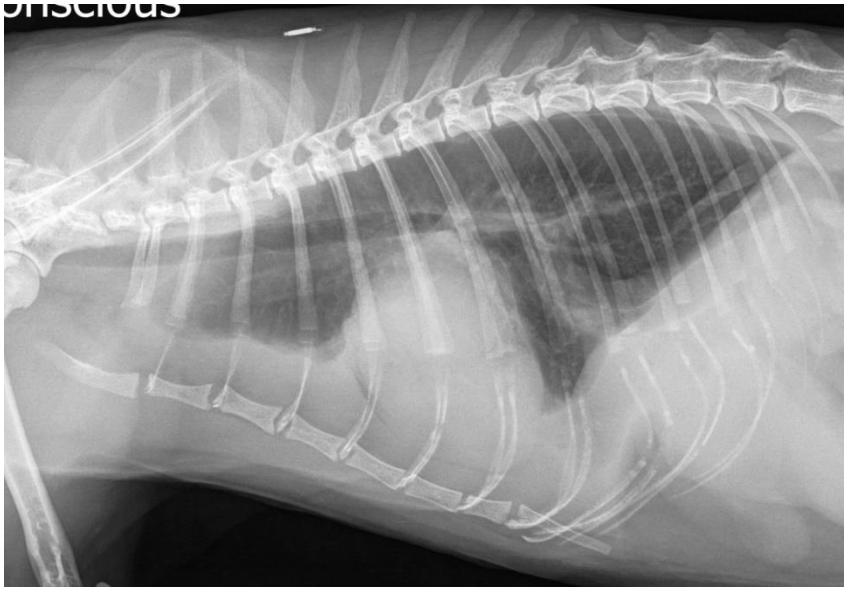
Pulmonary artery bulge at 1-2 o'clock in a dog with pulmonic stenosis
The left auricular appendage bulges at 2-3 o'clock on the DV, and can potentially be seen in any cause with left atrial enlargement, although not all.



A left auricular appendage bulge in a dog with left atrial enlargement due to mitral endocardiosis, note also the "cottage loaf" appearance as the left atrium sits on top of the cardiac silhouette, and the splitting of the mainstem bronchi.
In a proportion of cases of PDA, all three knuckles (12-1 o'clock aorta, 1-2 pulmonary artery, 2-3 left auricular appendage) can be seen to enlarge, which is characteristic.

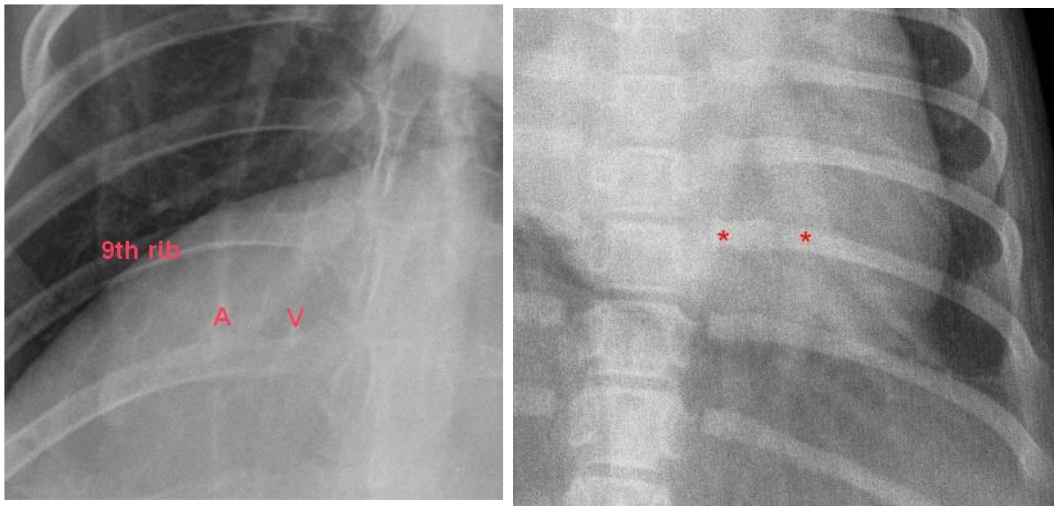


The **aorta and caudal vena cava** should be roughly equal in height, and are typically the height of the adjacent vertebral body. It is uncommon for the descending aorta to increase in size, except in rare cases such as aortic coarctation, aneurysm, neoplasia and in other countries Spirocerca Lupi, a parasite in non-UK countries that has an aortic migration. The caudal vena cava varies hugely with cardiac and respiratory cycle, so is only considered increased in size if it exceeds 1.5 times the height of the aorta.



Hugely enlarged caudal vena cava in cat with right heart failure and HCM.
It can increase in size with cardiac disease (right heart failure, right atrial anomalies or masses, tricuspid dysplasia), with caudal vena cava obstruction (CVC thrombus, or (rare) CVC stenosis), pericardial disease particularly if restrictive/causing tamponade, and in the correct geographic region Dirofilaria.

The normal **main pulmonary vessels** are visualized either side of the main lobar bronchus, and should be equal in size to each other at the same level. In dogs, the cranial lobar vessels are best seen in the lateral view, where the vein lies ventral to the bronchus and the artery dorsal; with the size as the cross the 4th rib measuring between 0.5 and 1 times the width of the proximal third of the 4th rib. The caudal lobar artery and vein are best seen on the DV, where the artery is lateral to the bronchus and the vein is medial; they are considered normal in size if they are the same width as the 9th rib where they cross it.
Enlargement of both arteries and veins is seen with overcirculation (large left to right shunts (PDA,VSD)), left heart failure, overhydration (iatrogenic). The arteries may be larger than the veins in cases of very large left to right shunts, some parasitic condition (most notably in Dirofilaria, which is not UK based), and focally in cases of downstream pulmonary thromboembolus. The veins may be larger than the arteries in left heart failure most commonly; however right to left shunting might have this appearance given the arteries are relatively small; and very occasionally with left to right shunts a the pressure effect of overcirculation is greater on the thin walled veins.
Finally, there is generalized reduction in pulmonary vascularization (both veins and arteries are reduced in size, termed a hypovascular pattern) with forced manual inflation/air trapping lung disease, hypoperfusion (shock), pulmonic stenosis, severe right to left shunts, and some right heart failures/pericardial effusions.



Example of reduced size of pulmonary artery and vein above left (undercirculation, hypovascular pattern) in dog with haemorrhagic shock, and of an enlarged artery and vein above right (overcirculation, hypervascular pattern) in a dog with a PDA.

The most common **congenital pericardial disease** is a peritoneal pericardial diaphragmatic hernia, a true hernia in the midline where there is a diaphragmatic defect allowing the abdominal contents to communicate freely with the pericardial contents. Since the heart is fixed in position, the abdominal contents may move into the pericardial sac, which gives a very enlarged cardiac silhouette on a radiograph. The exact opacity will be determined by the displaced organs – typically liver, spleen (soft tissue), falciform fat, and stomach or small intestines (which may contain some gas). Other pericardial anomalies include pericardial defects (through which the heart can herniate), and pericardial cysts.