7

The heart and major vessels

Victoria Johnson, Kerstin Hansson, Wilfred Mai, Joanna Dukes-McEwan, Nola Lester, Tobias Schwarz, Peter Chapman and Frederica Morandi

Radiographic anatomy

A thorough understanding of anatomy is an essential part of diagnostic imaging of the heart and cardiovascular system. This section covers the normal physiological and radiographic anatomy of the heart and the major vessels. An introduction to performing radiographic cardiac mensuration is also provided. The following section on interpretive principles covers the radiographic assessment of cardiac chamber enlargement, major vessel enlargement and differential diagnoses.

A full discussion of the ultrasonographic anatomy of the heart is beyond the scope of this manual. Chapter 2 and the Appendices provide detailed information on performing an echocardiographic examination.

Basic cardiac anatomy

The heart is the largest mediastinal organ and its anatomy is complex. The heart develops from paired endocardial tubes that arise from splanchnic mesoderm. These gradually fuse and undergo elongation, various dilations and constrictions, and finally partition into chambers to form the heart. A full description of the embryological origin of the heart is beyond the scope of this chapter, but a good understanding of cardiac formation will improve understanding of congenital heart disease.

Partitioning of the heart results in four chambers (right and left atria and right and left ventricles) and two great arteries (the aorta and main pulmonary artery):

- Blood enters the right atrium (RA) from the cranial and caudal vena cavae and the coronary sinus
- Blood leaves the right ventricle (RV) via the main pulmonary artery
- Blood enters the left atrium (LA) from the pulmonary veins
- Blood leaves the left ventricle (LV) via the aorta.

Valves divide the chambers. Each valve has a fibrous annulus and a number of cusps or leaflets. The atria are separated by atrioventricular valves; the mitral valve on the left and the tricuspid valve on the right. Both atrioventricular valves have two cusps and the term 'tricuspid' is a misnomer derived from human terminology. The ventricles are separated from the major arteries by semilunar valves (both are tricuspid); the aortic on the left and the pulmonic on the right.

The four chambers of the heart are encased within the pericardium or pericardial sac. There are two layers forming the pericardium:

- The *fibrous* pericardium – a strong external layer
- The *serous* pericardium – a thin lining that covers the heart. This is divided into parietal and visceral layers and there is a small amount of lubricating fluid between these layers.

The heart itself is supplied with blood by the coronary arteries. The left and right main coronary arteries arise from the root of the aorta. Venous drainage is via the coronary veins and the coronary sinus.

Normal cardiac radiographic anatomy

The heart dominates a thoracic radiograph as it is the largest single soft tissue opacity in the thorax. It sits within the mediastinum from approximately the third to the sixth intercostal space. The larger, more dorsal part of the heart is known as the *base* and the smaller, more ventral part is known as the *apex*.

In the lateral view the apex is formed by the interventricular septum (IVS). The heart lies at an angle within the thorax (easily seen on the lateral view) with the apex positioned more caudally than the base. The apex usually points towards the *left* on a dorsoventral/ventrodorsal (DV/VD) view. Variation in the location of the apex on a DV/VD view may be extrinsic (e.g. due to alteration in lung volume) or intrinsic (a congenital malpositioning of the heart). The latter may be an incidental finding or associated with other pathological abnormalities (e.g. situs inversus in Kartagener's syndrome) (Figures 7.1 and 7.2).

<<Figures 7.1, 7.2 near here>>

The actual outline of the heart is not seen on a radiograph. Instead, the term *cardiac silhouette* is used to include the heart, pericardium, pericardial contents (such as fat and a small amount of fluid) and the origin of the aorta and main pulmonary artery. This is in contrast to an echocardiographic examination where the heart, origin of the major vessels, pericardium and pericardial contents are seen as

separate structures. The outline of the cardiac silhouette is smooth and details such as the coronary grooves and the separation of atria from the ventricles are *not seen*. The coronary arteries and veins are also not seen radiographically. However, it is possible to infer the location of the major chambers on a radiograph and to identify pathological changes in their size (see below).

The central and ventral margins of the cardiac silhouette are easily outlined on a radiograph as these areas are surrounded by contrasting air in the lungs. It is not possible to outline the margins of the heart in the area of the heart base as the surrounding soft tissues, i.e. the major blood vessels (aorta, main pulmonary artery and pulmonary veins) and lymph nodes, have the same radiographic opacity. It is often difficult radiographically to appreciate changes and pathology in this region and ultrasonography and computed tomography (CT) are useful as alternative imaging modalities.

Many factors alter the appearance of a normal cardiac silhouette on a radiograph and this is a major source of misdiagnosis and confusion in thoracic radiography. These include tremendous variation with *breed*, as well as variation with respiratory phase, age and body condition and radiographic view (see below).

Identifying the cardiac chambers

The internal chambers of the heart and the lumens of the great vessels can only be visualized on a radiograph with the administration of positive contrast media. This is known as angiocardiography (see Chapter 1). However, it is possible to use the location of the borders of the various chambers to assess cardiac or great vessel enlargement on a standard radiographic series.

Normal angiocardiograms are very useful in understanding the location of the chambers within the shadow of the cardiac silhouette (Figures 7.3 and 7.4). These have been used to formulate useful diagrams showing the radiographic location of the cardiac chambers (Figures 7.5 and 7.6).

<<Figures 7.3, 7.4, 7.5, 7.6 near here>>

The presence of pericardial effusion usually precludes the evaluation of changes in specific cardiac chambers.

Some basic rules help in the assessment of cardiac chambers:

- In the dog the LV and LA are located on the left and caudal aspect of the heart; the RV and RA are located on the right and cranial aspect of the heart
- The position of the cardiac apex can be used to separate the right and left sides of the heart
- On a lateral view the RV is cranial to the apex and on a VD/DV view it is to the right. The reverse is true for the LV
- The exact apical point can be difficult to determine due to the variation in cardiac position. This can sometimes make evaluation of left- *versus* right-sided enlargement difficult
- The two atria are located dorsal to the level of the caudal vena cava (CdVC) on a lateral view
- The dorsal and caudodorsal borders of the LA do not have a distinct outline as the pulmonary veins enter in this area and the right pulmonary artery also overlaps this region.

Clock face analogy

A clock face analogy has been used to identify specific chamber locations on a radiograph. The clock numbers are used to indicate the approximate position of the chambers and great vessels (Figure 7.7).

<<Figure 7.7 near here>>

<<BOX>>

Lateral view

12.00–02.00 o'clock:	left atrium
02.00–05.00 o'clock:	left ventricle
05.00–09.00 o'clock:	right ventricle
09.00–10.00 o'clock:	main pulmonary artery and right auricular appendage
10.00–11.00 o'clock:	aortic arch

Dorsoventral view

11.00–01.00 o'clock:	aortic arch
01.00–02.00 o'clock:	main pulmonary artery
02.30–03.00 o'clock:	left auricular appendage
02.00–05.00 o'clock:	left ventricle
05.00–09.00 o'clock:	right ventricle
09.00–11.00 o'clock:	right atrium

Species differences

- On a VD/DV view the cardiac apex points more to the left in the dog. In the cat the apex is more variable, but usually closer to the midline.
- In the dog the left auricular appendage (LAu) is located at 02.30–03.00 o'clock on the DV/VD view. In the cat the LA and LAu are located at 01.00–02.00 o'clock on this view and the main pulmonary artery may be cranial to this or not seen at all.
- In the cat the more cranial location of the LA may make it difficult to see on the lateral view.
- When the LA is enlarged in the dog it is superimposed over the cardiac silhouette in the 05.00–07.00 o'clock position (between the caudal mainstem bronchi), whereas in the cat, it is located more cranially at the 01.00–02.00 o'clock position. This explains the so-called 'valentine heart shape' seen only in the cat and created by left and right atrial enlargement.

<<END BOX>>

Factors affecting cardiac size and appearance

Many factors can alter the appearance of the normal heart on a radiograph. It is extremely important to be aware of the influence of these factors to avoid the misdiagnosis of normal anatomical variation as disease.

<<BOX>>
Species difference

- The normal canine heart shows substantial breed-associated variations in size and shape. The normal feline heart is generally unaffected by breed (Figure 7.8).
- Variation in cardiac shape and size with body position is an important consideration in the dog. Generally size and shape alteration with body position are negligible in the cat.

<<END BOX>>

<<Figure 7.8 near here>>

Breed
The canine heart varies tremendously between different dog breeds; breed-associated conformational variation is the single most important cause of variation in the normal canine cardiac silhouette. A basic outline of these differences is shown in Figures 7.9 and 7.10; however, there are many more variations that cannot be listed here. A good understanding of these differences is paramount to allow accurate cardiac assessment in thoracic radiography. It may be useful to build up a collection of normal thoracic radiographs of different breeds for easy reference.

<<figures 7.9 (table 7.2), 7.10 near here>>

Pericardial fat
An incorrect diagnosis of cardiomegaly is often made in animals with a large amount of *pericardial fat* (see also Figure 1.24, **p. xx**). Pericardial fat contributes to the overall size of the cardiac silhouette but it is often possible to identify the presence of fat on careful inspection. Pericardial fat has a lower opacity compared with the heart and often the cardiac margin is not as sharp. The latter is due to the gradual change in opacity from soft tissue (heart) to fat to air-filled lung, rather than the sharp soft tissue–air interface seen in thinner animals. Altering radiographic technique may also assist in identification of pericardial fat.

In the cat, pericardial fat is seen better in lateral rather than in VD radiographs. Pericardial fat should be suspected if a large amount of falciform fat is present, and may have a characteristic square corner on the right cranial margin of the cardiac silhouette on the DV/VD view (Figure 7.11).

<<Figure 7.11 near here>>

Age
Young dogs appear to have large hearts. This is, in part, due to the relatively small size of the thoracic cavity. Thus, it is particularly important to take into account shape changes as well as overall cardiac size in younger animals.

About 40% of cats over 10 years old have a cranially sloping cardiac silhouette with increased sternal contact (Figure 7.12). The reason for this is poorly understood and no association with increased cardiac size has been found. It is considered likely to be associated with age-related changes in thoracic conformation.

<<Figure 7.12 near here>>

Body position
Variation in body position will cause variations in the appearance of the cardiac silhouette due to the effects of gravity. Consistency in the radiographic technique is important in assessment of sequential radiographs and helps to avoid misinterpretation. Generally right lateral and DV radiographs are acquired for radiographic assessment of cardiac disease (see Chapter 1).

Regardless of view, the cardiac silhouette is always slightly larger than the heart itself due to the effects of magnification. Specific changes in the appearance of the canine cardiac silhouette on different radiographic views are given in Figure 7.13 (see also Figures 1.28 and 1.29, **p. xx and p. xx**).

<<Figure 7.13 (table) near here>>

Respiratory phase
Expiratory radiographs create a false impression of cardiomegaly as the overall thoracic size is decreased, whilst the cardiac size stays the same (for examples of inspiratory and expiratory radiographs see Figure 12.9, **p. xx**). Sternal contact increases at expiration, which exacerbates the impression of cardiomegaly; there is also a small but real increase in cardiac size during expiration.

At expiration the cardiac silhouette can be more difficult to outline cranially due to adjacent mediastinal fat and caudally due to overlapping of the diaphragm. In addition, loss of air content in the lung tissue contacting the cardiac silhouette creates a surrounding hazy indistinct zone.

The effects of all of these changes are exaggerated in older, obese and shallow-breathing dogs.

Cardiac cycle
Variations due to cardiac cycle seldom cause major changes or radiographic misinterpretation. They are best seen in large-breed dogs and dogs with a slow heart rate.

Most radiographs are exposed during ventricular diastole simply because it is longer than systole. Near the end of ventricular systole the atrial borders can be rounded and bulging and the ventricles may appear as slightly smaller with a narrow 'V' instead of 'U' shape (Figure 7.14). On VD/DV views the main pulmonary artery can be more prominent in systole. One report suggested that there was less alteration in shape due to cardiac cycle on the VD view than the DV view in cats.

<<Figure 7.14 near here>>

Measuring the cardiac silhouette

It is important to be able to assess cardiac size radiographically and many techniques have evolved in order to do this. Note that radiographs are often

inaccurate in the assessment of heart size and identification of specific chamber enlargement. Ultrasonography remains the gold standard for chamber size assessment (see Chapter 2).

Guidelines for cardiac size
Numerous papers have been published on the subject of quantifying the normal heart size and various methods are in use. These techniques have many limitations and should only be used in combination with a good understanding of the normal sources of variation in the cardiac silhouette. A good general principle is that the heart should be considered radiographically normal unless there is an obvious change in size or shape. Note also that a radiographically normal heart by no means excludes cardiac disease.

<<BOX>>
Canine cardiac size: rules of thumb

- On a lateral view the cardiac length (apex to base) should be approximately 70% of the dorsal to ventral distance of the thoracic cavity.
- The width (craniocaudal dimension) has been defined to be between 2.5 (deep-chested breeds) and 3.5 (round-chested breeds) intercostal spaces.
- Cardiac width on a DV view is usually 60–65% of the thoracic width and no more than two thirds of the thoracic width at the widest point of the cardiac silhouette on a VD view.

<<END BOX>>

<<BOX>>
Feline cardiac size: rules of thumb

- In cats the width of the cardiac silhouette should be no more than 2–2.5 intercostal spaces in width on the lateral view.
- On the lateral view the maximal width should be approximately the same as the distance between the cranial border of the fifth rib and the caudal border of the seventh rib.

<<END BOX>>

Vertebral heart score/vertebral heart size
A novel system of cardiac measurement was proposed in 1995, which aimed to circumvent the limitations in other methods attributable to inherent breed variation in cardiac size. It is called the vertebral (or Buchanan) heart score (VHS). In this technique the long and short axes of the cardiac silhouette are measured on a lateral view and totalled. They are then scaled against the thoracic vertebral column (Figures 7.15 and 7.16).

<<Figures 7.15 and 7.16 near here>>

The VHS is useful for those new to evaluating cardiac size on radiographs and also for the sequential assessment of cardiac size on repeat radiographs of the same patient. It has not been proven to be superior to subjective assessment. The suggested mean value in the original multibreed study was 9.7 ± 0.5v (8.5–10.5v) (v: vertebrae). However, considerable breed variation exists even in the VHS, and later studies have established breed-specific normals (see Figure 7.16b).

Trachea and carina position
The location of the trachea and carina can be useful in assessing cardiac size and chamber enlargement (Figure 7.17). On a lateral radiograph the trachea diverges from the thoracic spine. The angle of divergence is fairly standard amongst cat breeds but shows variation between dog breeds. The greatest angle is seen in deep-chested dogs, whereas in shallow-chested breeds the trachea may be almost parallel to the spine. This should not be erroneously interpreted as representing cardiac enlargement.

<<Figure 7.17 near here>>

Just cranial to the tracheal bifurcation there is a normal *ventral bend* in the trachea (Figure 7.18). This is often lost in animals with left-sided cardiac enlargement; however, it may not be present in normal, shallow, round-chested dog breeds.

<<figure 7.18 near here>>

The carina should be located at the fourth or fifth intercostal space. The round radiolucent ring-like structures extending out from the trachea are cross-sectional views of either the left or the right cranial mainstem bronchus (see Chapters 10 and 11). On a VD/DV view the angle between the left and right mainstem bronchi is around 60 degrees at the bifurcation. The mainstem bronchi should create a upsidedown V shape on this view (Figure 7.19).

<<figure 7.19 near here>>

Pericardial fat stripe
It can be difficult to assess cardiac size when a pleural effusion is present. Fat present between the fibrous pericardium and the pericardial mediastinal pleura may remain visible on the lateral view in animals with pleural effusion. This is known as the *pericardial fat stripe* and may aid radiographic assessment of the heart in animals with pleural fluid (Figure 7.20).

<<Figure 7.20 near here>>

Normal major vessel physiological and radiographic anatomy

Aorta
The aorta is divided into three sections:

- The ascending aorta: short and arises from the cranial aspect of the heart. Has the same orientation as the LV
- The aortic arch (or transverse arch): short and curves caudally. Gives rise to the brachiocephalic trunk and left subclavian artery

- The descending aorta: long and can be divided into thoracic and abdominal portions.

The aortic isthmus is the junction of the ascending aorta and the aortic arch. The ascending aorta contributes to the cranial heart base area and cannot be seen clearly due to superimposition (see Figure 7.3). The aortic arch and descending aorta are identified on both lateral and DV/VD views. (Figure 7.21). In the normal animal the left border of the descending aorta is identified on the VD/DV view.

<<Figure 7.21 near here>>

The aortic diameter is similar to the height of adjacent vertebral bodies; aortic size does not alter in association with hypovolaemia or volume overload (unlike the CdVC).

A focal bulge in the aorta (sometimes also referred to as an elongated, redundant or tortuous aorta) at the aortic isthmus may be seen in a proportion of older cats (Figure 7.22). One study found that this was present in 28% of older (>10 years) cats. The change does not appear to be associated with systemic hypertension or hyperthyroidism.

<<Figure 7.22 near here>>

Caudal vena cava

The CdVC receives blood from the abdomen, pelvis and hindlimbs. The final abdominal tributaries are the hepatic veins. The CdVC enters the thorax by crossing the diaphragm on the right side within the plica vena cava. It then traverses between the accessory and right caudal lung lobes to enter the RA dorsal to the inlet of the coronary sinus. It lies in close association with the right phrenic nerve.

The majority of the intrathoracic CdVC is easily seen on radiographs (Figure 7.23). On the lateral view the CdVC crosses the caudal half of the right ventricular border and can often be seen overlapping the cardiac silhouette for a short distance. On VD/DV views it is seen to the right of the median plane between the caudal right border of the heart and the diaphragm.

<<Figure 7.23 near here>>

The diameter of the CdVC is highly variable due to variations in intrathoracic pressure during respiration and stage in the cardiac cycle. It may also vary in pathological conditions such as right-sided heart disease, hypovolaemia or CdVC obstructive conditions (caval syndrome, masses, etc.; see Acquired vascular diseases). Enlargement of the CdVC is more likely to suggest right-sided heart disease in the dog when:

- CVC:Ao >1.5 (strongly suggestive of right-sided heart abnormality)
- CVC:VL >1.3
- CVC:R4 >3.5.

Where Ao = aorta; CVC = the greatest diameter of the CdVC not overlapping the heart or diaphragm; R4 = diameter of the right fourth thoracic rib just ventral to the spine; and VL = the length of the thoracic vertebra over the tracheal bifurcation. A word of caution: there was shown to be considerable overlap in the CVC:Ao ratio between normal patients and those with right-sided heart disease.

Cranial vena cava

The cranial vena cava (CrVC) receives blood from the head, neck, thoracic wall and forelimbs. The axillary veins (from the forelimbs), together with the internal and external jugular veins, converge to form the right and left brachiocephalic veins. These then unite to create the CrVC. It travels in the cranial mediastinum and receives the costocervical and internal thoracic veins, as well as the azygos vein just cranial to the RA; it finally empties into the RA.

The CrVC is seen as an individual structure on a radiograph unless a pneumomediastinum is present. It forms the ventral border of the cranial mediastinum on the lateral view.

Azygos vein

The azygos vein forms from the first lumbar veins and passes through the aortic hiatus into the thorax. It then receives intercostal, subcostal, oesophageal and broncho-oesophageal veins and terminates in the CrVC. It is not seen on a radiograph unless a severe pneumomediastinum is present or it is markedly enlarged. It then appears as a wavy vessel immediately ventral to the thoracic spine, receiving tributaries from every intervertebral space (Figure 7.24). It may rarely be seen in very deep-chested, narrow breeds, such as the Greyhound, in the absence of a pneumomediastinum.

<<Figure 7.24 near here>>

Thoracic duct

The thoracic duct originates between the diaphragmatic crura. It has a variable course within the thorax but usually courses cranially along the right dorsal border of the aorta. Its termination is variable but it usually enters the CrVC or left jugular vein.

The thoracic duct is not seen on radiographs. It can be identified using radiographic or CT lymphangiography or on heavily T2-weighted magnetic resonance (MR) images.

Main pulmonary artery

The main pulmonary artery arises from the pulmonic valve and arches dorsally and caudally. It splits into right and left pulmonary arteries immediately caudal to the level of the aortic root. These then form further branches within the left and right lung. The ligamentum arteriosum (the ductus arteriosus in fetal life) joins the proximal part of the left pulmonary artery.

The main pulmonary artery is usually not seen on a lateral radiograph as it is superimposed on the cardiac silhouette. On occasions it may be identified as a round soft tissue opacity located immediately ventral to the carina and should not be confused with

a nodule in this location (see Figure 7.43b). The left and right branches may be radiographically identified on the lateral view. The left branch runs slightly more cranial and also dorsal to the right branch. On VD/DV views the main pulmonary artery contributes to the cranial left cardiac silhouette (Figure 7.25). It appears more prominent during systole and also on a VD view compared with a DV view. This should not be interpreted as pathology. The left and right main branches are superimposed on the heart, but more distally the caudal lobar right and left pulmonary arteries are seen as tubular soft tissue structures coursing caudolaterally in association with the lateral aspects of the caudal mainstem bronchi.

<<Figure 7.25 near here>>

Cranial lobar pulmonary arteries and veins

<<BOX>>
Useful tip
In order to remember the location of the pulmonary veins compared with the arteries use the rhyme:

"Veins are ventral and central"

(i.e. veins are ventral to arteries on the lateral view and central to the arteries on the VD view).
<<BOX END>>

The arteries and veins to the cranial lung lobes can be seen on lateral views with the arteries dorsal to the veins (Figure 7.26). They are best separated on the left lateral view. Arteries follow the bronchial tree closely, but the veins may not be as closely associated with the bronchi. Arteries are often slightly curved and often better defined compared with the veins. Normally, the artery and vein of each pair is approximately the same size.

<<Figure 7.26 near here>>

Various rules exist for cranial lobar vessel size (see below) but a good general rule of thumb is that *no cranial lobar vessel should be greater than the narrowest part of the third or fourth rib where it crosses the rib*.

<<BOX>>
Rules for cranial pulmonary vascular size in the dog and cat

Dog
In the dog the cranial lobar pulmonary vessels should be approximately the same in size. The ratio of the diameter of the artery or vein to the proximal third of the fourth rib, at the level of the fourth intercostal space is 0.73 (± 0.24) with a 95% confidence interval of 0.26–1.2.

These vessels should be considered enlarged when greater than 1.2 times the proximal third of the fourth rib at the fourth intercostal space.

OR

The cranial lobar pulmonary arteries should not be larger than the diameter of the proximal third of the third rib on the lateral radiograph.

Cat
In the cat the right cranial lobar artery should be 0.5–1.0 times the proximal third of the fourth rib (mean artery:rib ratio of 0.70) when measured at the level of the fourth rib. The cranial lobe veins should be 0.2 cm (± 0.03 cm) in diameter at the same point.
<<END BOX>>

Caudal lobar pulmonary arteries and veins
On VD/DV views the caudal pulmonary arteries are located lateral to the veins and can be traced cranially towards the main pulmonary artery segment (Figure 7.27). Caudal lobar pulmonary arteries and veins are more easily identified on DV views due to the presence of surrounding aerated lung and the effects of magnification. The veins are medially located and run towards the LA that is located centrally in the cardiac silhouette between the mainstem bronchi.

<<Figure 7.27 near here>>

<<BOX>>
Rules for caudal pulmonary vascular size in the dog and cat
The pulmonary artery and vein of each caudal lobe should be similar in size.

The diameter of the artery or vein should be no greater than the width of the ninth rib where they cross it (anecdotal).

In the cat, a cut-off for pulmonary arterial enlargement of 1.6 times the ninth rib has been suggested in assessment of heartworm disease.
<<END BOX>>

Interpretive principles

Thoracic radiographs are extremely useful in the evaluation of the patient with cardiovascular disease. However, it should be noted that radiography and echocardiography are complementary techniques and it is important to understand the limitations and benefits of these two imaging modalities in the assessment of cardiac disease.

In simple terms, echocardiography is far more accurate in identifying chamber enlargement and other structural abnormalities than radiographs. Echocardiography also provides vital functional information that cannot be obtained radiographically. However, radiographs remain indispensable in the evaluation of pulmonary vascular and parenchymal changes secondary to heart disease. For example, a radiograph will quickly reveal evidence of pulmonary venous congestion and/or cardiogenic pulmonary oedema in left-sided failure, or pleural effusion in right-sided failure. Thoracic radiographs are also extremely valuable in monitoring the progress of

therapy in heart failure.

Chapter 2 and the appendices provide further information on echocardiographic technique and normal echocardiographic measurements.

This section covers the radiographic features of specific cardiac chamber and major vessel enlargement. The evaluation of congestive heart failure is also covered here. It is important to have a good knowledge of cardiac anatomy, physiology and pathophysiology when approaching a thoracic radiograph for the evaluation of cardiac disease. Remember that when radiographic changes are present they do not show the disease itself, but rather the haemodynamic consequences of the condition.

Microcardia

Radiographic features

- Cardiac silhouette is narrow and pointed on the lateral view and narrow on the DV/VD view (Figure 7.28).
- Apex may lose contact with the sternum.
- There may be small pulmonary arteries and veins.
- Lung field may be hyperlucent (without hyperinflation).
- CdVC may be narrow.

<<Figure 7.28 near here>>

Differential diagnoses

- Hypovolaemia: shock, dehydration. Lung fields are also underperfused and CdVC may be small (Figure 7.29).
- Addison's disease (hypoadrenocorticism). Heart is actually physically smaller due to chronic electrolyte abnormalities with or without hypovolaemia and shock in an acute Addisonian crisis.
- Emaciation.
- Atrophic myopathies.
- Artefactual, such as pneumothorax, deep-chested dogs, deep inspiration or pulmonary underinflation.

<<Figure 7.29 near here>>

Normal radiographic cardiac size

Some cardiac diseases may not produce any apparent radiographic changes. Examples are:

- Endocarditis
- Concentric ventricular hypertrophy:
  - Aortic stenosis
  - Pulmonic stenosis
  - Hypertrophic cardiomyopathy (HCM).
- Other myocardial disease:
  - Acute myocardial failure
  - Early or mild myocarditis
  - Myocardial neoplasia.
- Acute ruptured chordae tendinae
- Small atrial septal defect (ASD), ventricular septal defect (VSD) or patent ductus arteriosus (PDA)
- Some types of pericardial disease:
  - Constrictive pericarditis
  - Acute traumatic haemopericardium.
- Arrhythmias
- Overzealous use of diuretic therapy in heart disease.

Cardiac chamber enlargement

The limitation of radiographs for the evaluation of specific cardiac chamber size have already been discussed. Often more than one chamber will be enlarged and this will complicate interpretation of changes in the cardiac silhouette. It should be emphasized that artefactual chamber and major vessel enlargement will be seen on poorly positioned radiographs and that the utmost care should be taken in obtaining perfectly straight views.

In the following section the DV view is described rather than the VD view, as the former is more commonly obtained in cardiac patients.

<<BOX>>

Concentric and eccentric hypertrophy: what is the difference?

Ventricular hypertrophy may occur in response to:

- Increased systolic pressure (a pressure overload) leading to *concentric* hypertrophy: the ventricular wall gets thicker and the lumen size remains normal (or reduced)
- Increased diastolic pressure and volume (a volume overload) leading to *eccentric* hypertrophy (also known as *dilation*): the ventricular wall remains a normal size and the lumen size increases.

The different types of hypertrophy have implication for radiographic interpretation. Concentric hypertrophy can be extremely difficult to recognize on a radiograph, whereas eccentric hypertrophy is more easily seen. Note that in contrast to the ventricles, *atria* usually only show *dilation* in cardiac disease.

<<END BOX>>

Right atrium

Right atrial enlargement is very uncommon in isolation and is also difficult to see radiographically unless it is severe (Figure 7.30).

<<Figure 7.30 near here>>

Lateral view:

- Focal bulge on cranial aspect of cardiac silhouette just ventral to the terminal trachea (cranial to the carina).
- May even push the trachea dorsally at this point if large enough or a right atrial mass is present.
- May merge with an enlarged RV.

Dorsoventral view:

- Bulge in cardiac silhouette at 09.00 – 11.00 o'clock.
- May merge with an enlarged RV.

Right ventricle

Right ventricular enlargement is commonly overdiagnosed on thoracic radiographs. This is partly because the shallow-chested conformation of many dog breeds can mimic certain features of right-sided cardiomegaly (such as increased sternal contact). Great care must be taken to differentiate breed variation from true right-sided cardiac enlargement. When truly present, right ventricular enlargement is often accompanied by left-sided changes and right atrial enlargement and may be hard to identify as a single entity (Figure 7.31).

<<Figure 7.31 near here>>

Lateral view: To evaluate the size of the RV, a line can be drawn from the carina to the cardiac apex; approximately two thirds of the cardiac silhouette should lie cranial to this line and one third caudal to it. An increased cranial component (e.g. four fifths cranial and one fifth caudal to the line) suggests right-sided enlargement. Rotation of the cardiac apex caudodorsally away from the sternum (i.e. pointing more towards the liver than the sternum) is a sensitive sign of right ventricular enlargement but is not always present. Right ventricular (or atrial) enlargement may displace the trachea dorsally over the heart base and cranial to the carina, but the trachea will still maintain its normal terminal ventral bend (unlike in left ventricular enlargement).

Other findings with RV enlargement include widening of the cardiac silhouette (non-specific) and increased cardiosternal contact (non-specific and not very useful: d*o not* rely on this in isolation).

Dorsoventral view: This is often more reliable than the lateral view for evaluation of right-sided enlargement. Findings include:

- Increased size of the cardiac silhouette on the right side of the thorax
- Reduced distance between the right cardiac border and the right thoracic wall
- A typical 'reversed D' shape
- On occasion, the apex may be pushed towards the left, creating the false impression of left-sided cardiomegaly.

Left atrium

Radiographs are very useful in identifying left atrial enlargement, and generally sensitive for moderate to severe dilation (Figure 7.32).

<<Figure 7.32 near here>>

Lateral view:

- Elevation (with or without compression) of the left caudal mainstem bronchus (seen as a separation of the two caudal mainstem bronchi) (see also Figure 8.10a**, p. xx**).
- Loss of the normal gentle cranial curvature of the caudal margin of the cardiac silhouette. Instead, the caudodorsal margin of the cardiac silhouette becomes straight and then eventually triangular, forming a left atrial 'tent' or 'wedge'.
- Increased height of caudodorsal border of the heart.
- Enlarged pulmonary veins may be seen entering the LA as indistinct nodular opacities in this region.
- In the cat, the LA is situated more cranially than in dogs and it is harder to identify on the lateral view.

Dorsoventral view:

- The enlarged LA is projected over the cardiac silhouette at the 05.00–07.00 o'clock position (between the caudal mainstem bronchi) on the DV/VD view. This results in divergence (or splaying) of the caudal mainstem bronchi (also known as the 'cowboy legs' sign). (This must be differentiated from enlargement of the middle tracheobronchial lymph node which will produce divergence of the caudal mainstem bronchi on a DV/VD view, but will push them *ventrally* on a lateral view; see Figure 8.10a, **p. xx**.)
- A large LA may also create a 'double density' or more correctly, 'double opacity' sign on this view. This is the presence of two differing soft tissue opacities between the caudal mainstem bronchi, one more opaque than the other. This results from superimposition of the enlarged atrium summating with the ventricle and producing more attenuation of the X-ray beam.
- An enlarged LAu may be seen at 02.30–03.00 o'clock position.
- The more cranial location means that an enlarged LA and LAu are seen at the left cranial border of the cardiac silhouette (even as cranial as the 01.00–02.00 o'clock position).
- A severely enlarged LA will bulge towards the left and can cause a shift of the cardiac apex to the right. When this is combined with severe right atrial enlargement this can create the appearance of a valentine-shaped heart.

Left ventricle

Left ventricular enlargement due to eccentric hypertrophy is usually easily recognized on a radiograph, whereas enlargement due to concentric hypertrophy is difficult to identify. This is important as animals with severe left-sided concentric hypertrophy (e.g. aortic stenosis) may have radiographically normal cardiac silhouettes.

Left ventricular enlargement is often accompanied by left atrial enlargement and also right-sided

changes.

Lateral view:

- Tracheal elevation (of the *entire* trachea and with loss of the normal terminal ventral bend) (Figure 7.33).
- Caudal border becomes straighter (or less commonly rounder) than usual.

<<Figure 7.33 near here>>

Dorsoventral view:

- Increased length of cardiac silhouette.
- Other signs are not as reliable.
- Cardiac apex may be more displaced into left hemithorax.

Generalized cardiomegaly

The entire cardiac silhouette may appear enlarged in many diseases and this is called generalized cardiomegaly. It is difficult (and usually inaccurate) to detect mild generalized cardiomegaly unless previous radiographs are available from the same patient. A moderate to severe generalized cardiomegaly is seen as an increase in width and height on the lateral view, and width and length on the DV view (Figure 7.34). The heart may also appear more generally rounded than usual. Care should be taken to distinguish generalized cardiomegaly from pericardial effusion (see below).

<<Figure 7.34 near here>>

Pericardial enlargement

Any increase in pericardial content or thickness can create the appearance of generalized, rounded (or globoid) enlargement of the cardiac silhouette. The commonest cause is the presence of pericardial effusion (for further information see Pericardial diseases, below).

Pericardial effusion can be distinguished from generalized cardiomegaly using:

- Ultrasonography (quick, accurate and extremely easy to perform for this distinction). Anechoic effusion peripheral to heart
- Radiographs (not nearly as reliable and not recommended):
  - Sharp border of cardiac silhouette in pericardial effusion compared with blurry border in generalized cardiomegaly (the normal movement blur of the beating heart is not recognized when it is encased in a fluid-filled pericardium) (Figure 7.35)
  - No individual chamber enlargement recognized in pericardial effusion (but often the case for generalized cardiomegaly as well by definition)
  - Lung fields are generally underperfused due to the effects of tamponade (may see pulmonary venous distension in generalized cardiomegaly)
  - Evidence of right-sided heart failure in pericardial effusion (but may also be present with generalized heart disease).

<<Figure 7.35 near here>>

Changes in the major vessels

Aorta

Entire aortic arch enlarged:

- Seen as a focal bulge on the cranial aspect of the heart on the lateral view.
- Seen as widening of the caudal part of the cranial mediastinum on the DV view.
- Possible causes:
  - Subaortic stenosis (or less commonly valvular), resulting in turbulence and post-stenotic dilation (Figure 7.36; see also Figure 7.64)
  - Age-related change in the cat (see Figure 7.22).

<<Figure 7.36 near here>>

Descending part of the arch enlarged:

- Usually only seen on the DV view as a focal bulge when the left lateral border of the aorta is traced from caudal to cranial.
- May be seen as a bulge at the classic 11.00–01.00 o'clock location on the DV view and an apparent elongation of the cardiac silhouette.
- Possible causes: PDA (see Figure 7.46a).

Other aortic enlargements or abnormalities:

- Aortic aneurysm:
  - Secondary to *Spirocerca lupi* infection (see Figure 9.20**, p. xx**)
  - Dissecting aortic aneurysms have been reported in dogs and cats
  - Ductus aneurysm or ductus diverticulum.
- Coarctation of the aorta with post-stenotic dilation (see Figure 7.90, **p. xx**).
- Redundant aorta:
  - Aged cats
  - Brachycephalic dogs
  - Congenital hypothyroidism.
- Abnormal location:
  - Situs inversus
  - Vascular ring anomalies (persistent right aortic arch most commonly).
- Calcification or mineralization of the aorta:
  - Incidental non-significant aortic mineralization in dogs (more common in older dogs and Rottweilers) (see Figure 7.37 and Appendix)
  - Primary or secondary hyperparathryoidism
  - Hypervitaminosis D
  - Lymphoma
  - Hyperadrenocorticism

  - *Spirocerca lupi* infection
  - Arteriosclerosis.
- Aortic body tumour (see Cardiac neoplasia, below).

<<Figure 7.37 near here>>

Caudal vena cava
The normal size of the CdVC is described above. Caution should be used when diagnosing changes in CdVC size on a radiograph. Caval size varies markedly with respiratory and cardiac cycle and thoracic and abdominal pressures. Changes in caval size should not be diagnosed from a single film. A genuine alteration in size will be seen on *repeated* radiographs.

Wide caudal vena cava:

- Right-sided heart failure.
- Cardiac tamponade.
- Constrictive pericarditis.
- Obstruction of the CdVC from the level of the hepatic veins to the level of the RA (Budd–Chiari-like syndrome):
  - Thrombosis
  - Caval syndrome associated with heartworm disease
  - Compression or invasion of the cava by tumours or other masses (see Figure 7.162)
  - Trauma-induced stricture
  - Fibrosis
  - Diaphragmatic hernia
  - Congenital cardiac (e.g. cor triatriatum dexter) or caval (e.g. membranous obstruction) anomalies.

Narrow caudal vena cava:

- Shock.
- Hypovolaemia (see Figure 7.29).
- Addison's disease (hypoadrenocorticism).
- Artefactual (pulmonary hyperinflation).

Mineralized caudal vena cava: This is rare. Causes include:

- Mineralized masses (see Figure 7.162)
- Other dystrophic mineralization
- Metastatic mineralization (hyperadrenocorticism, secondary hyperparathyroidism, etc.).

Segmental aplasia of the caudal vena cava: This is a rare congenital anomaly where part of the CdVC is missing and blood returns to the heart via the azygous vein. A markedly enlarged azgous vein will be identified.

Persistent left cranial vena cava
This is relatively common and only really of significance when performing cardiac catheterization or thoracic surgery. The left CrVC persists from fetal life and drains into the coronary sinus.

Main pulmonary artery
Dilation of the main pulmonary artery is difficult to detect on lateral views due to superimposition over the cardiac silhouette. The DV view will show an enlarged main pulmonary artery as a bulge at the 01.00–02.00 o'clock position.

Enlarged main pulmonary artery segment:

- Pulmonic stenosis: post-stenotic dilation (Figure 7.38).
- Increased circulating volume due to PDA, ASD or VSD.
- Pulmonary hypertension.
- Severe heartworm disease or angiostrongylosis.
- Artefactual:
  - VD position
  - Systole
  - Positional rotation.

<<Figure 7.38 near here>>

Pulmonary arteries and veins
Methods of assessing pulmonary artery and vein size are described above. Examples of pulmonary arterial and venous enlargement are shown in Figure 7.39. Figure 7.40 gives differential diagnoses for variations in pulmonary vascular size.

<<Figures 7.39, 7.40 near here>>

Heart failure

Heart failure can be defined in many ways. One definition is that heart failure is the end result of severe heart disease and is a clinical syndrome, resulting in systolic and/or diastolic dysfunction severe enough to overwhelm the cardiovascular system's compensatory mechanisms. Congestion, oedema, poor peripheral perfusion and/or systemic hypertension result. Radiographic examination provides an invaluable insight into the presence and severity of heart failure.

Radiographic features of left-sided heart failure in the dog
Cardiogenic pulmonary oedema is seen (see also Chapter 12) (Figure 7.41):

- Varies from faint interstitial infiltrate to severe alveolar pattern (see Chapter 12)
- Usually first seen in the perihilar region on the lateral view, though the right caudal lobe is also often a predilection site (seen on the DV view)
- Usually extends from a central to more peripheral location.

<<Figure 7.41 near here>>

Note that severe left-sided heart disease *must also be present* to make the diagnosis of left-sided heart failure. Non-cardiogenic pulmonary oedema may mimic the radiographic appearance (see Chapter 12). One complicating situation is that of an acute

onset of heart failure, such as secondary to ruptured chordae tendinae. In this situation substantial left-sided cardiomegaly may not be present. Echocardiographic examination will provide an accurate diagnosis.

Radiographic features of left-sided heart failure in the cat

- Cardiogenic pulmonary oedema (see also Chapter 12) (Figure 7.42). May appear similar to that in the dog or may appear as patchy unevenly distributed opacities throughout the lung fields. The variable appearance of cardiogenic oedema in the cat makes the diagnosis harder.
- Pleural effusion. In the cat, pleural effusion may be seen in left-sided heart failure. This is poorly understood but is thought to be due to an anatomical variation in visceral pleural drainage.

<<Figure 7.42 near here>>

Radiographic features of right-sided heart failure

- Pleural effusion (Figure 7.43):
  - Note that right heart failure is a *rare* cause of pleural effusion in the cat
  - Pleural effusion in both dogs and cats is usually a combination of both left- and right-sided failure
  - It is important to identify severe right-sided cardiac disease in order to attribute a pleural effusion to right-sided heart failure.
- Wide CdVC; see previous comments on CdVC size.
- Hepatomegaly.
- Ascites (peritoneal effusion).
- Pericardial effusion may also result from right-sided heart failure.

<<Figure 7.43 near here>>

Congenital cardiovascular diseases

Congenital cardiovascular diseases are anomalies of the heart and great vessels that have been present since birth. This definition applies even if the condition is not identified until later in life. A wide variety of congenital diseases exists and it is beyond the scope of this manual to explore all of these conditions. Figure 7.44 lists the congenital abnormalities that have been reported in the dog and cat and gives an indication as to which are the more common conditions.

<<Figure 7.44 (Table B7.1) near here>>

Patent ductus arteriosus

PDA is the most common congenital cardiac disease in dogs. Small-breed dogs (including, but not limited to, Chihuahua, Maltese, Poodle, Pomeranian, Bichon Frise, Shetland Sheepdog) and German Shepherd Dogs are predisposed. Bitches are more affected than dogs. Cats are also affected but the condition is less common than in dogs.

The ductus arteriosus is an important part of normal fetal circulation. It extends from the main pulmonary artery to the descending aorta and in the fetus functions to divert blood away from the lungs back into the systemic circulation. After birth, pulmonary vascular resistance falls and flow in the ductus reverses. The ductus then closes by constriction of smooth muscle within its wall, brought about by increased arterial oxygen tension that inhibits local prostaglandin release. The ductus is usually closed by 7–10 days after birth. It remains in the adult as the ligamentum arteriosum.

PDA results from failure of normal closure of the ductus arteriosus, resulting in a shunting vessel between the descending aorta and the main pulmonary artery.

There are three main types of PDA:

- The mildest form of the condition results in closure of the ductus at the pulmonary arterial end only. This produces a clinically insignificant blind-ended pouch called a 'ductus diverticulum'. *No shunting* of blood occurs and this anomaly may only be identified as an incidental finding on postmortem
- The commonest clinically presenting patent ductus is a complete tapering funnel-shaped tube with blood shunting from *left-to-right*. Volume overloading of the *left side* of the heart causes left atrial dilatation and left ventricular dilatation and hypertrophy
- The third and least common form for the ductus to take is a non-tapering cylindrical tunnel. This is associated with persistent postnatal pulmonary hypertension and *bidirectional* or *right-to-left shunting* (Eisenmenger's syndrome). These are sometimes known as 'reversed PDAs' and are discussed further, below. The condition results in decreased pulmonary blood flow, a normal to small LV and right ventricular concentric hypertrophy.

Left-to-right shunting

Clinically, a left-to-right shunting PDA is characterized by a continuous machinery-type heart murmur (continuous systolo-diastolic murmur) heard best over the main pulmonary artery. A bounding or 'waterhammer' arterial pulse may also be present.

Most animals are usually asymptomatic when the murmur is discovered. Surgical ligation is recommended in almost all cases.

Radiography: Radiographic findings include:

- Lateral view (Figure 7.45):
  - Classically left-sided changes are present, but generalized cardiomegaly may be seen
  - Prominent aortic arch
  - Prominent LA
  - Left ventricular enlargement, leading to straightening of the caudal margin of the heart

  - Increased vascular lung pattern, with increased size of both the pulmonary arteries and veins; very distally located vessels are visible due to enlargement (the vascular pattern is due to increased blood flow to the lung, caused by the left-to-right shunt)
  - Eventually signs of left-sided cardiac failure, with pulmonary oedema.
- DV view (Figure 7.46):
  - Elongated cardiac silhouette
  - In cats the left apex may be displaced into the right hemithorax
  - Prominence of the aortic arch, pulmonary trunk and LAu at 11.00–01.00, 01.00–02.00 and 02.00–03.00 o'clock, respectively: this triad has been reported to be pathognomonic for PDA but is not present in all patients (only around 20% of cases)
  - Occasionally an aortic bulge ('ductus bump') may be seen near the level of the ductus. This is caused by the abrupt narrowing of the descending aorta beyond the level of the ductus origin
  - Enlarged LA with splayed caudal mainstem bronchi with or without double opacity sign
  - Increased vascular lung pattern (Figure 7.46b)
  - Eventually signs of left-sided cardiac failure.

<<Figures 7.45, 7.46 near here>>

Angiocardiography: This is usually unnecessary, but should be performed if echocardiography is inconclusive or additional congenital complications are suspected. It is performed by selective catheterization of the aorta, via either a carotid or a femoral approach: contrast medium is injected when the tip of the catheter is just distal to the aortic valve.

- With left-to-right shunting, contrast medium is seen both in the aorta and the main pulmonary artery and its branches immediately after injection.
- The ductus arteriosus can sometimes be identified: the aortic orifice is located on the left ventral aspect of the aorta distal to the left subclavian artery, then extending cranioventrally and to the left to its pulmonic orifice (Figure 7.47).
- Often the ductus itself is not identified on angiocardiograms, due to partial superimposition of the aorta and main pulmonary artery. Dilation of the aorta at the usual location of the shunt is a hint that the left-to-right shunting is due to a PDA.

<<Figure 7.47 near here>>

Echocardiography: Ultrasonography is useful both for confirmation of diagnosis and to evaluate the consequences of the PDA on cardiac function. The ductus itself can be seen from either the right parasternal (RPS) short-axis or left parasternal (LPS) cranial window (Figure 7.48). In some cases the ductus is not seen on trans-thoracic echocardiograms but will be visualized with trans-oesophageal echocardiography.

<<Figure 7.48 near here>>

Very early in the disease, no changes in chamber size or cardiac function are identified. Later on, the following can be observed:

- Left atrial dilation
- Aortic dilation
- Main pulmonary artery dilation
- Left ventricular hypertrophy and dilation
- Decreased myocardial contractility (decreased fractional shortening, increased E point to septal separation (EPSS), increased left ventricular end-systolic dimensions).

Doppler studies: If the ductus is visualized, then high-velocity turbulent flow from the aorta, through the ductus and into the main pulmonary artery can be observed (Figure 7.49). Otherwise diagnosis is based on other characteristic Doppler findings:

- Main pulmonary artery: continuous high velocity ductal flow towards the pulmonic valve (up to 4.5–5 m/s) (Figure 7.50)
- Left ventricular outflow tract (LVOT): mild increase in outflow velocity (but usually <2.5 m/s)
- Mild aortic and pulmonic insufficiency.

Care should be taken to distinguish PDA from similar congenital abnormalities, such as an aortopulmonary window (but usually results in Eisenmenger's physiology) or an anomalous systemic to main pulmonary artery shunt. These conditions are very rare and are beyond the scope of this manual.

<<Figures 7.49, 7.50 near here>>

Scintigraphy: Observations with first pass radionuclide angiocardiography include:

- Prolongation of the radioactivity within the lungs with incomplete clearance of the lungs during the levophase
- Lack of lung clearance in the levophase, causing partial or complete obliteration of the borders of the aorta (Figure 7.51)
- QP:QS ratio >1.2.

<<Figure 7.51 near here>>

Right-to-left shunting

Occasionally, *right-to-left* shunting occurs through a PDA due to high pulmonary vascular resistance. This is thought to occur because the non-tapering tubular shape of the ductus in these animals permits aortic pressures to be transmitted to the pulmonary arterial system. The resulting extremely increased pulmonary perfusion eventually leads to intimal arteriolar damage and muscular proliferation, causing marked pulmonary hypertension and reversed flow through the ductus

arteriosus (Eisenmenger's syndrome). This change has usually been shown to occur in the first few weeks of life. The pulmonary vascular changes are poorly understood, but are known to be irreversible, thereby precluding surgical treatment for this condition.

The anomaly is quite different in presentation to a left-to-right shunting PDA. Clinical signs include fatigue, shortness of breath and weakness. No murmur or only a soft systolic murmur is heard on auscultation and a split second heart sound may be detected. Differential cyanosis may be seen with normal pink cranial mucous membranes but cyanotic caudal mucous membranes, due to the fact that the PDA originates distal to the brachiocephalic trunk and left subclavian artery. The animal may be polycythaemic.

Radiography: Radiographic findings include:

- Right heart enlargement on both lateral and DV views
- Dilation of the main pulmonary artery (seen best on the DV view)
- A 'ductus bump' may be seen
- Underperfusion of the lung fields may be evident (hypovascular pattern, hyperlucent lung fields).

Angiocardiography:

- Right-to-left shunting can be demonstrated by an injection of contrast medium in the RV or main pulmonary artery.
- The contrast medium then shunts from the main pulmonary artery to the descending aorta through the wide ductus (Figure 7.52).
- Pulmonary arteries may be normal or appear tortuous.
- Left-sided injections may show an extensive broncho-oesophageal collateral circulation.

<<Figure 7.52 near here>>

Echocardiography: Echocardiographic findings include:

- Enlarged main pulmonary artery
- Concentric hypertrophy of the RV
- May identify a wide tube-like ductus.

Doppler studies: Evidence of pulmonary hypertension is seen (tricuspid or pulmonic insufficiency jets, with increased pressure gradients (PGs) suggesting pulmonary hypertension).

Contrast echocardiography: This is extremely useful in confirming the right-to-left shunt and easy to perform. Agitated sterile saline is injected into a cephalic or saphenous vein. The descending aorta is observed for bubbles confirming shunting; this is best seen in the caudal abdomen, ventral to the spine.

Scintigraphy:

- First pass radionuclide angiocardiography:
  - In cases of reverse PDA, there is simultaneous appearance of the aorta and pulmonary arteries after the dextrophase as the radionuclide passes into the lungs (Figure 7.53)
  - It is not possible to distinguish a reverse PDA from tetralogy of Fallot on the basis of a first pass study alone.
- Scintigraphy with ^{99m}Tc-MAA. In cases of reverse PDA, activity is seen outside the lungs with clear visualization of the renal cortices; however, there is no activity in the neck, head and cerebral cortex due to the location of the shunt caudal to the brachiocephalic trunk and left subclavian artery, thereby allowing differentiation from tetralogy of Fallot (Figure 7.54).

<<Figures 7.53, 7.54 near here>>

Pulmonic stenosis

Pulmonic stenosis encompasses any obstruction of blood flow from the RV to the main pulmonary artery. The prevalence is higher in English Bulldogs, but also Beagles, Boxers, Chihuahuas, Schnauzers and all terriers. Pulmonic stenosis is a rare condition in cats. The condition usually occurs in isolation but on occasion other cardiac anomalies, such as tricuspid dysplasia, ASD, patent foramen ovale or VSD, may be present.

The stenosis may occur at different levels:

- *Valvular* stenosis (most common form): a variety of anomalies may occur including hypoplasia of the valve, thickened valve leaflets, asymmetric valve leaflets and incomplete commissural separation. A fibrous ring may also be present below the valve
- *Subvalvular* stenosis (less common): a specific form of this occurs in English Bulldogs and Boxers due to an anomalous origin of the left main coronary artery (Figure 7.55). Many variations are possible, but most commonly the left and right coronary arteries branch from a single large coronary artery that arises from the right aortic sinus of Valsalva and wraps around the right ventricular outflow tract (RVOT). These cases cannot be treated with balloon valvuloplasty
- *Supravalvular* stenosis (rare): may be seen more often in Giant Schnauzers.

<<Figure 7.55 near here>>

The stenosis leads to a pressure overload and concentric hypertrophy of the RV. This, in turn leads to decreased right ventricular diastolic compliance, ventricular filling impairment and increased right atrial pressure. Tricuspid regurgitation may result and further raises right atrial pressure with eventual right-sided heart failure.

The condition is usually asymptomatic at the time of diagnosis. However, it eventually leads to exercise

intolerance, syncope and possible right-sided heart failure if functional tricuspid insufficiency is present. On clinical examination pulmonic stenosis is characterized by a left-sided systolic heart murmur, most audible at the heart base (ejection type of murmur).

Therapy for the condition includes both medical and surgical options and the appropriate choice depends on the severity of the stenosis and the clinical status of the patient. The condition should be monitored over time as some dogs can gradually develop more severe obstructions.

Radiography
Radiographic findings include:

- Right-sided cardiomegaly:
  - Increased sternal contact on the lateral view (moderate) (Figure 7.56a)
  - Rounding of the heart with reverse D shape on the DV view (moderate) (Figure 7.56b).
- Prominence of the main pulmonary artery (post-stenotic dilation), best seen on the DV view (between 01.00 and 02.00 o'clock) (Figure 7.56b)
- In a minority of dogs, the dilated post-stenotic main pulmonary artery segment is seen superimposed over the caudal trachea on the lateral view. This has been termed the 'hat sign' (Figure 7.56a)
- In very severe cases, pulmonary hyperlucency and small pulmonary vessels (due to decreased pulmonary arterial outflow) may be seen
- There may be signs of right-sided cardiac failure: right ventricular and atrial enlargement, enlarged CdVC, hepatomegaly, ascites and hydrothorax.

<<Figure 7.56 near here>>

Angiocardiography: This is not usually used for diagnosis but is often used prior to surgical intervention. Diagnosis can be established after selective catheterization of the RVOT using a jugular approach. Right atrial injections of contrast medium are not suitable for the diagnosis of pulmonic stenosis, since the pulmonic valve area is usually superimposed upon the opacified RA.

- Stenosis is visible at the level of the valve, the infundibulum or at the subvalvular level (muscular hypertrophy, creating a filling defect in the outflow tract) (Figure 7.57a).
- Post-stenotic dilation of the main pulmonary artery can be identified (Figures 7.57ab).
- In cases of functional tricuspid insufficiency, regurgitation of contrast medium is seen from the RV to the RA.
- In English Bulldogs and Boxers, it is vital to search for an abnormal origin of the left coronary artery as an underlying cause of the stenosis (see Figure 7.55). Ideally, coronary arteriography can be performed. Alternatively, the aortic root and coronary arteries should be examined in the late levophase after a right ventricular injection of contrast medium, or just after a left ventricular injection of contrast medium. In affected animals a single coronary artery is seen wrapping around the RVOT and a right coronary artery branches from this single artery a few millimetres distal to its aortic origin (Figure 7.58).

<<Figures 7.57, 7.58 near here>>

Echocardiography
Echocardiography is useful to confirm the diagnosis and to grade the severity of the stenosis. Findings include:

- Concentric hypertrophy of the RV (Figure 7.59), secondary to pressure overload on the RV. Seen as a thickening of the right ventricular free wall that can approach or exceed the thickness of the left ventricular free wall
- Prominent right ventricular papillary muscles are sometimes visible
- Flattening of the IVS due to the elevated right ventricular pressure. On M-mode images, paradoxical motion of the septum may be seen (Figure 7.60)
- Abnormal pulmonic valve (Figure 7.61):
  - Thickened, with a limited systolic excursion
  - Hyperechoic
  - If the valve leaflets are fused, occasionally ballooning can be seen during systole.
- Abnormal subvalvular region: discrete subvalvular fibrous ring may also be seen as well as generalized infundibular hypertrophy
- Post-stenotic dilation of the main pulmonary artery distal to the valve
- Possible right atrial dilation (and occasionally right ventricular dilation).

<<Figures 7.59, 7.60, 7.61 near here>>

Doppler studies:

- *Accelerated ejection velocity* through the pulmonic valve (Figure 7.62). Care should be taken to achieve parallel alignment and to use continuous wave (CW) Doppler to achieve accurate velocity measurements.
- *Turbulent* ejection flow in the main pulmonary artery (Figure 7.63). Possible mild to moderate *pulmonary insufficiency* (diastolic back-flow from the main pulmonary artery to the RV) (Figure 7.62).
- The severity of the obstruction can be estimated by accurate measurement of the *peak flow velocity* through the main pulmonary artery. This is then used to calculate the instantaneous PG across the obstruction using the modified Bernouilli equation. The PG may be used to grade the severity of the stenosis, though note that the reliability of this measurement may vary with myocardial contractility and sedation/ anaesthesia:
  - Mild: <50 mmHg

  - Moderate: 50–80 mmHg
  - Severe: >80 mmHg. Such animals are likely to develop right-sided cardiac failure early and are candidates for balloon valvuloplasty.
- *Tricuspid insufficiency*. Measurement of this may also be useful to assess the degree of stenosis when accurate measurement of main pulmonary artery velocity cannot be obtained.

<<Figures 7.62, 7.63 near here>>

Aortic stenosis

The vast majority of aortic stenosis occurs in the *subvalvular* region (subvalvular aortic stenosis). This is the most common congenital cardiac malformation in large-breed dogs. *Valvular* stenosis (thickened valves and hypoplastic annulus) is rare, but Bull Terriers are predisposed. A bicuspid valve may also rarely cause a mild valvular stenosis. *Supravalvular* stenosis is also very rare. The remainder of this section relates to subvalvular aortic stenosis.

Large-breed dogs are more commonly affected by subvalvular aortic stenosis. Breed predisposition includes Golden Retrievers, Newfoundlands, German Shepherd Dogs, Boxers, Rottweilers and English Bull Terriers. It occurs occasionally in cats. The condition may be accompanied by other congenital cardiac disorders, such as mitral valve dysplasia and PDA.

The lesion is usually a fixed ridge or ring of fibrous tissue in the LVOT just below the aortic valve. The outflow obstruction leads to increased left ventricular systolic pressure and concentric left ventricular hypertrophy results. A post-stenotic dilation of the aorta and sometimes brachiocephalic trunk occurs. Mild aortic regurgitation is often present. Animals with subvalvular aortic stenosis are predisposed to aortic endocarditis, abnormal coronary arterial flow and myocardial infarctions.

The functional and clinical consequences vary with the severity of the obstruction. This ranges from minor obstructions with minimal hypertrophy and no clinical signs, to significant obstructions with concentric hypertrophy of the LV, left-sided heart failure, syncope or sudden death.

Radiography

Radiography is often unremarkable in mild cases. Changes may be seen in more severe cases:

- Lateral view (Figure 7.64a):
  - Elongated cardiac silhouette, with dorsally displaced trachea and carina and straightened caudal margin of the heart due to left ventricular enlargement
  - Prominent ascending aorta and aortic arch (post-stenotic dilation)
  - Sometimes mild left atrial enlargement (if severe, suggests concurrent mitral regurgitation)
  - In cases with left-sided heart failure: left atrial dilation, pulmonary venous congestion and pulmonary oedema.
- DV view (Figure 7.64b):
  - Rounding of the left contour of the heart
  - Aortic bulge at the 11.00–01.00 o'clock position
  - There may be left atrial enlargement (see above)
  - In cases with left-sided heart failure: left atrial dilation, pulmonary venous congestion and pulmonary oedema.

<<Figure 7.64 near here>>

Angiocardiography:

- Subaortic stenosis is demonstrated by a left ventricular injection of contrast medium.
- Narrowing of the outflow tract is usually obvious on lateral views (Figure 7.65).
- Varying degrees of post-stenotic dilation of the ascending aorta can be identified: in a normal dog, the maximum diameter of the ascending aorta distal to the sinus of Valsalva is always less than the diameter of the sinus of Valsalva.

<<figure 7.65 near here>>

Echocardiography

It can be extremely difficult to detect mild aortic stenosis with echocardiography. Subtle or no changes may be seen.

- Moderate to severe cases:
  - Left ventricular concentric hypertrophy is seen, with thickening of the left ventricular free wall and IVS (Figure 7.66a)
  - Hyperechoic ridge or circumferential ring of hyperechoic tissue in the region of the LVOT (Figure 7.66b and 7.67)
  - Post-stenotic dilation of the ascending aorta.
- Severe cases as above plus:
  - Hyperchoic papillary muscles and endocardial surface due to areas of myocardial ischaemia and subsequent fibrosis
  - If there is mitral regurgitation, left atrial enlargement may be observed
  - Systolic anterior motion of the mitral valve may be seen in animals with concurrent mitral dysplasia.

<<Figures 7.66, 7.67 near here>>

Doppler studies: These are characterized by high-velocity turbulent aortic flow (see Figure 7.67). The maximum normal aortic velocity in most dogs is 1.7 m/s. However, higher velocities can be seen in normal dogs and also breeds with slightly reduced LVOT size (e.g. Boxers, Golden Retrievers, Bull Terriers). Note also that conditions of excitement and stress will increase the velocity, and myocardial failure will reduce it.

Subcostal (retroxiphoid) views are ideal for good alignment and accurate estimation of aortic velocities, although they are difficult to achieve in larger-breed dogs due to the distance between the transducer and

the LVOT; CW Doppler should be used.

- The diagnosis is reinforced when increased velocities show rapid focal acceleration in the LVOT and/or are accompanied by turbulent flow and anatomical lesions.
- Calculation of the PG across the stenosis (using the maximal aortic velocity) may be used for grading severity and estimating prognosis:
  - Dogs with maximal PG of <50 mmHg and minimal ventricular hypertrophy are more likely to lead normal lives
  - Dogs with maximal PG of >125 mmHg are very likely to develop serious complications or die suddenly.
- Mitral inflow studies may show an increased A wave during diastole (decreased E:A ratio) due to loss of compliance in the hypertrophied LV.
- Functional mitral regurgitation might be present in severe cases and those with concurrent mitral dysplasia. When present, it usually has a higher velocity than normal due to increased left ventricular pressure (and hence increased PG between the LV and the LA).

Ventricular septal defect

VSD is a common congenital disorder in both dogs and cats. Breed predispositions include English Springer Spaniels, West Highland White Terriers and Lakeland Terriers, amongst others. VSD is known to have a genetic basis in the Keeshond with malformations of the conotruncal septum.

Anomalous development of any part of the ventricular septal components may lead to a VSD (see above). However, most commonly the defect occurs at the point of fusion of the membranous and muscular parts of the septum, resulting in a perimembranous lesion. Thus, the defect is most often located 'high' or dorsally on the septum. Occasionally, the defect occurs lower down in the muscular part of the septum.

On the *left* side the defect is usually located in the subaortic septum, just below the aortic valve and typically between the right coronary and non-coronary cusps. On the *right* side the location is more variable. Typically, the VSD opens under the septal cusp of the tricuspid valve (also known as infracristal or under the crista supraventricularis). Less commonly, the opening on the right is in a more cranial location and opens directly into the RVOT above the tricuspid valve (also known as supracristal or above the crista supraventricularis). The full classification of VSDs is complex and is beyond the scope of this manual.

The septal defect allows *left-to-right* shunting of blood unless other significant abnormalities are present, which result in shunt reversal (such as pulmonic stenosis, tricuspid dysplasia or severe pulmonary arterial hypertension). Pathophysiological and clinical effects of the VSD depend upon the size and location of the defect, and the presence or absence of associated malformations. VSDs may be classed as resistive or non-resistive:

- *Resistive* VSDs are small and provide resistance to flow between the right and left sides
- *Non-resistive* VSDs are large (approximately the same size as the aortic orifice), and there is no resistance to flow between the two sides.

The most common type of VSD in the dog is a *small resistive* VSD. However, these are often large enough to have clinical significance. With resistive lesions the flow across the defect depends both on its size and the pressure difference between the left and right sides. Blood is shunted from the high pressure left side to the right side across the defect (in the absence of other abnormalities).

In most cases it has been shown that the shunted volume moves directly into the RVOT, hence right-sided enlargement is not usually a feature of the condition. The shunted blood then enters the pulmonary circulation and finally returns to the left side of the heart. This results in *overcirculation of the pulmonary system and left-sided heart enlargement* (eccentric hypertrophy) if the shunt is significant. If left ventricular end-diastolic pressure increases sufficiently as a result, then left-sided heart failure will ensue.

Less commonly, the defect is lower in the septum and a significant volume of shunted blood enters the RV, rather than exiting immediately via the outflow tract. In this case, right-sided heart enlargement may occur.

Non-resistive VSDs result in the two ventricles functioning as a single chamber. The volume of blood that is shunted, and the direction that it is shunted in, depends purely on the difference between the systemic and pulmonary resistance. In most cases the systemic resistance is much higher and a huge left-to-right shunt occurs. However, this large shunt to the pulmonary circulation will result in increased pulmonary pressure. The RV hypertrophies concentrically in response to this. Both ventricles also hypertrophy eccentrically.

If pulmonary hypertension worsens (pulmonary vascular pathology, reactive pulmonary hypertension) then there will be increased resistance to the right ventricular outflow. The shunt fraction from left-to-right will decrease and eventually shunt reversal may occur should right ventricular systolic pressures exceed left ventricular systolic pressures. The latter is known as *Eisenmenger's complex*; and right-to-left shunting and cyanosis are observed.

The clinical presentation, prognosis and recommended treatment vary enormously with the type of VSD. In general, small, uncomplicated VSDs have a good prognosis and clinical signs may not be seen. Animals with larger shunts and cardiomegaly have a poorer prognosis. Those with Eisenmenger's complex have a very guarded long-term prognosis.

Radiography

Radiographic findings include:

- Small defects: thoracic radiographs can be totally normal

- Larger defects: left atrial and left ventricular enlargement with or without increased vascular pattern in the lungs (Figure 7.68)
- In cases with left-sided congestive heart failure: pulmonary oedema
- In cases with biventricular heart failure: pulmonary oedema, pleural and peritoneal effusion
- Varying degrees of right ventricular and main pulmonary artery enlargement are also possible, depending on the level and size of the defect (as explained above)
- Main pulmonary arterial enlargement and an underperfused lung periphery suggests pulmonary hypertension or pulmonic stenosis and shunt reversal.

<<Figure 7.68 near here>>

Angiocardiography: VSDs with left-to-right shunts are best demonstrated by injection of contrast medium in the LV (Figure 7.69):

- The left ventricular opening is usually high in the ventricular septum, in the outflow tract just below the aortic valve
- The right ventricular opening is usually just under the cranial part of the septal leaflet of the tricuspid valve
- Shunting usually occurs only in systole as in diastole the PG between both ventricles almost equals zero.

<<Figure 7.69 near here>>

VSDs with right-to-left shunts secondary to pulmonary hypertension (Eisenmenger's complex) are best demonstrated by injection of contrast medium in the RV; there is evidence of passage of contrast medium into the LV immediately after the injection into the RV, and simultaneous opacification of the aorta and main pulmonary artery. This condition can be difficult to differentiate from a tetralogy of Fallot.

Echocardiography
Echocardiographic findings include:

- Visualization of the septal defect may be possible, usually on a RPS long-axis five-chamber view, just below the root of the aorta (Figure 7.70a)
- Signs of eccentric hypertrophy of the left heart may be visible: left ventricular and left atrial dilation (Figure 7.70ab)
- Right ventricular hypertrophy and/or dilation can also be seen, depending on the nature of the VSD (Figure 7.70b).

<<Figure 7.70 near here>>

Doppler studies: Colour Doppler investigation is useful to identify VSDs (see Figures 7.70b and 7.71). Shunting through the defect can be demonstrated using Doppler studies, revealing high velocity and turbulent systolic flow through the defect; the direction of shunting can also be assessed.

<<Figure 7.71 near here>>

The usual velocity of the flow through an uncomplicated small resistive defect (the most common type in dogs) with normal left and right ventricular pressures is ≥4.5 m/s. A lower velocity should raise suspicion for increased right ventricular systolic pressure (pulmonary hypertension, pulmonic stenosis, etc.). Occasionally, there is secondary functional aortic regurgitation (due to the proximity of the defect to the insertion of the septal aortic leaflet) and this can be documented on Doppler examination.

Contrast echocardiography: Right-to-left shunts can be assessed using an ultrasound bubble study: an intravenous injection of vigorously shaken saline is performed. If a right-to-left shunt is present, echogenic gas bubbles appear simultaneously in the right and left cardiac cavities after injection.

Scintigraphy
The scintigraphic findings using first pass radionuclide angiocardiography include:

- Prolongation of the radioactivity within the lungs with incomplete clearance of the lungs during the levophase
- Lack of lung clearance in the levophase, causing partial or complete obliteration of the borders of the aorta
- In a medium to large VSD, reappearance of the RV during the levophase due to shunting of the radiopharmaceutical through the defect, allowing differentiation from a PDA (Figure 7.72)
- QP:QS ratio >1.2.

<<Figure 7.72 near here>>

Atrial septal defect

ASDs are uncommon congenital abnormalities in the dog. Breeds at risk include the Samoyed, Boxer, Newfoundland and Old English Sheepdog. They are also infrequently seen in cats (mostly endocardial cushion defects).

ASDs can occur at three main locations:

- High in the atrial septum, near the entrance of the pulmonary veins. These are *sinus venosus* ASDs and are very rare
- In the middle of the septum. These are *ostium secundum* ASDs and are the most common type. They vary in size. Some authors consider a patent foramen ovale to be a small ostium secundum defect. Conversely, other authors do not consider a patent foramen ovale to be a true ASD, as the atrial septum forms normally but the foramen walls are pushed apart, usually by conditions that increase right atrial pressure

- At the base of the septum. These are either *ostium primum* defects or *endocardial cushion* defects. Various types of endocardial cushion defects exist and they are often accompanied by abnormal atrioventricular valve development. These defects are usually big and are more commonly reported in cats. A complete endocardial cushion defect is a large ASD in the lower atrial septum, a high VSD and fusion of the septal leaflets of the two atrioventricular valves. This results in a communication between all four cardiac chambers and is also known as an *atrioventricular valve canal defect*.

Most ASDs are associated with left-to-right shunting unless there is a reason for reversal of the flow with increased right atrial pressure (pulmonic or tricuspid valve malformation or pulmonary hypertension). Just as with VSDs, the defect may be classed as resistive or non-resistive.

The shunting occurs during diastole. Large defects result in significant left-to-right shunting and eccentric hypertrophy of the RV, pulmonary overcirculation and sometimes right atrial dilation. Right-sided heart failure may result.

If left atrial enlargement is identified then the examiner should look for an additional defect, such as an endocardial cushion defect with mitral regurgitation. These animals may develop left-sided or bilateral heart failure.

Shunt reversal may occur in certain conditions, such as pulmonic stenosis or pulmonary hypertension (Eisenmenger's syndrome/physiology).

Most ASDs are not clinically significant and treatment is usually not necessary.

Radiography
Radiographic findings include:

- Often normal radiographs
- Right-sided cardiomegaly: dilation of the RV with or without dilation of the RA (Figure 7.73)
- There may be enlarged pulmonary arteries
- Left atrial changes are seen with some endocardial cushion defects.

<<Figure 7.73 near here>>

Angiocardiography: Uncomplicated ASDs are demonstrated using left atrial injections of contrast medium. The study reveals abnormal shunting of the contrast material from the LA to the RA, through the septal defect. In cyanotic animals, where right-to-left shunting is suspected, the septal defect can be demonstrated using a right atrial injection of contrast medium.

Echocardiography
Septal defects can be identified at various locations. They are best seen using a RPS long-axis four-chamber view (Figure 7.74). However, it is important to be aware that the thin nature of the septum and ultrasound beam orientation may result in the false appearance of a defect. Doppler studies must be used for confirmation (Figure 7.75). There may also be:

- Right atrial dilation
- Right ventricular eccentric hypertrophy
- Atrioventricular valve abnormalities, possibly with high VSD ± left atrial dilation in endocardial cushion defects (Figure 7.76).

<<Figures 7.74, 7.75, 7.76 near here>>

Doppler studies: Doppler should always be used to confirm the abnormal flow through the septal defect, usually from left-to-right (best demonstrated on the RPS long-axis four-chamber view).

Occasionally, there is increased velocity of the blood flow through the pulmonic valve, due to an increased volume of flow through a normal valve, associated with left-to-right shunting. On clinical examination this can be identified as a murmur of 'relative pulmonic stenosis'. There may also be evidence of additional defects (e.g. mitral regurgitation in some endocardial cushion defects).

Scintigraphy
Scintigraphic findings with first pass radionuclide angiocardiography include:

- Prolongation of the radioactivity within the lungs with incomplete clearance of the lungs during the levophase
- Lack of lung clearance in the levophase, causing partial or complete obliteration of the borders of the aorta
- In the case of a large defect, potential reappearance of the RA and/or RV; this has not yet been documented
- QP:QS ratio >1.2.

Mitral valve dysplasia

This condition is frequently encountered in dogs, especially Great Danes, English Bull Terriers, German Shepherd Dogs, Mastiffs, Golden Retrievers and Newfoundlands. Cats are also affected and mitral dysplasia may be the most common congenital cardiac defect in this species.

The disease encompasses any combination of malformed valve leaflet or cusps, abnormal chordae tendinae (short, absent or too long) and abnormal papillary muscles (fused, abnormally positioned). Concurrent cardiac abnormalities may be noted in dogs, such as subvalvular aortic stenosis. Most commonly the abnormality is associated with systolic mitral regurgitation. Valvular insufficiency leads to volume overloading, and atrial and ventricular dilation. Left-sided congestive heart failure may eventually result. Mitral stenosis can also occur and in dogs is seen in the Bull Terrier.

Animals may be asymptomatic or can present with exercise intolerance or even left-sided heart failure. A holosystolic murmur will be heard over the left apex (or left sternal border in cats). The prognosis is

variable and influenced by the presence of heart failure.

Radiography
Radiographic changes are very similar to those of acquired mitral insufficiency secondary to mitral endocardiosis:

- Left atrial and left ventricular enlargement (Figure 7.77)
- Enlargement of the pulmonary veins
- At the stage of cardiac failure: cardiogenic pulmonary oedema (± pleural effusion in cats)
- Mitral stenosis should be considered when the atrium is markedly enlarged but not accompanied by changes in the ventricle.

<<Figure 7.77 near here>>

Angiocardiography: This is generally not useful for diagnosis of mitral valve dysplasia. Mitral regurgitation is demonstrated by left ventricular injection of contrast medium. Prominent left atrial dilation accompanied by left ventricular dilation will usually be identified. Mitral stenosis is best demonstrated using a left atrial injection of contrast medium (performed by transeptal catheterization) but is certainly difficult to recognize.

Echocardiography
Echocardiographic findings include:

- Left atrial and ventricular enlargement (Figure 7.78)
- Abnormal mitral apparatus:
  - Malformed valve leaflet or cusps (thickened, hyperechoic)
  - Hyperechoic chordae tendinae, which can be short, absent or too long
  - Abnormal papillary muscles (fused or abnormally positioned)
  - Abnormal motion of the mitral leaflets: may appear rigid, lack of diastolic opening (potentially leading to mitral stenosis) (Figure 7.78a).
- If there is mitral regurgitation (most cases), in the early stage of the disease M-mode examination (at the transventricular level) shows a hyperkinetic ventricle with an increased fractional shortening as the regurgitation volume into the low-pressure LA assists rapid emptying of the LV.
- Left ventricular function may deteriorate over time.

<<Figure 7.78 near here>>

Doppler studies:

- High-velocity systolic regurgitant flow from the LV to the LA, best demonstrated using a left apical window (see Figure 7.78b).
- When mitral stenosis is present, an abnormally prolonged high-velocity jet will be noted from the atrium into the ventricle during diastole. There will be acceleration of the A wave (atrial systole) of ventricular filling and a decrease in the E:A ratio.

Tricuspid valve dysplasia

This condition is mostly observed in dogs, with larger breeds such as Labrador Retrievers most commonly affected (especially males). The condition is also seen in cats.

Tricuspid valve dysplasia is characterized by malformed tricuspid leaflets, chordae tendinae and/or papillary muscles. Occasionally, valvular stenoses may occur. Additional congenital cardiac anomalies may be present, such as pulmonic stenosis or ASD.

Ebstein's anomaly is a rare form of tricuspid valve dysplasia. In this condition the base of the cusps of the abnormal tricuspid valve are displaced distally into the ventricle and part of the RV is therefore 'atrialized'.

Clinical signs are usually not apparent early on in the condition. A holosystolic murmur will be identified over the tricuspid valve region. Eventually right-sided cardiac failure may result (e.g. hepatomegaly, ascites and distension of the jugular veins).

Radiography
Radiographic findings include:

- Right ventricular and right atrial dilation (Figure 7.79)
- Often a marked apex shift to the left is noted
- The cardiac enlargement can be impressive and almost appear globoid
- Valvular stenosis should be considered when the atrium is markedly enlarged but not accompanied by changed in the ventricle
- There may be enlargement of the CdVC
- Hepatomegaly and ascites, resulting in increased opacity and decreased serosal detail in the cranial abdomen, may be noted.

<<Figure 7.79 near here>>

Angiocardiography: Contrast studies are not very useful in the diagnosis and/or management of tricuspid valve malformations. They can demonstrate right atrial and right ventricular enlargement. Tricuspid regurgitation can be demonstrated by right ventricular injection of contrast medium.

Echocardiography
Echocardiographic findings include:

- Right ventricular dilation (unless the valve is stenotic) (Figure 7.80)
- Right atrial dilation
- Abnormal papillary muscles (may find large fused papillary muscle instead of the small discrete muscles)
- Abnormal conformation of the tricuspid valve:
  - Hypoechoic leaflets and chordae tendinae
  - Decreased motion of the tricuspid leaflets
  - Decreased separation from the right ventricular free wall and/or IVS

  - Apical implantation of the tricuspid valves with a seemingly reduced right ventricular cavity and a seemingly enlarged RA (right ventricular 'atrialization' – Ebstein's anomaly).
- Right ventricular dilation is evident on two-dimensional (2D) or trans-ventricular M-mode images – it is usually accompanied by a flattening or paradoxical motion of the IVS due to the increase pressure in the RV (Figure 7.80).

<<Figure 7.80 near here>>

Doppler studies:

- A high-velocity regurgitant flow from the RV to the RA, can best be demonstrated using a left apical window.
- The severity of the disease correlates to the extent of regurgitation into the RA.
- The PG is usually in the order of 30 mmHg (see Figures 7.80 and 7.81).
- When pulmonary hypertension is present the regurgitation flow is usually of very high velocity (>3 m/s).
- When stenosis is present, an abnormally prolonged high-velocity jet will be noted from the atrium into the ventricle during diastole.

<<Figure 7.81 near here>>

Tetralogy of Fallot

Tetralogy of Fallot is a complex congenital disorder, resulting from a failure of the conotruncal septum to align properly at the embryonic stage. The Keeshond and English Bulldog are predisposed and the condition is also seen in cats. Tetralogy of Fallot is the most common cause of *cyanotic* cardiac disease in young animals.

The disease is characterized by four features:

- Pulmonic stenosis leading to right ventricular outflow obstruction (main pulmonary artery atresia may also be present)
- Secondary right ventricular hypertrophy
- A subaortic VSD
- Dextroposition of the aorta (rightward positioning or overriding aorta).

The right ventricular outflow obstruction and increased right ventricular pressure lead to shunting of blood from the right side to the left via the septal defect. Deoxygenated blood mixes with left-sided oxygenated blood and hypoxaemia results. The LA and LV are small and the pulmonary arteries and veins are underperfused. The bronchial arteries increase the systemic collateral circulation that they provide to the lungs.

Animals present with exercise intolerance, failure to grow, shortness of breath, syncope, cyanosis and secondary polycythaemia. Note that since shunting occurs at the level of the ventricles the cyanosis is generalized and not differential as in reverse PDA. Medical and surgical treatment options exist and the prognosis is variable.

Radiography

Radiographic findings include:

- The overall size of the heart is usually small to normal
- Right ventricular enlargement may be apparent (Figure 7.82)
- The overriding of the aorta may produce a loss of the cranial waist on the cardiac silhouette on the lateral view (Figure 7.82)
- The main pulmonary artery is not enlarged (unlike the situation in pulmonic stenosis without a septal defect)
- Hypovascularization of the lungs may be identified: hyperlucent lung fields, decreased size of the pulmonary lobar vessels (Figure 7.82).

<<Figure 7.82 near here>>

Angiocardiography: Angiocardiography can be useful to confirm the diagnosis but is usually unnecessary if a careful echocardiographic examination is performed. It can be difficult to differentiate a tetralogy of Fallot from a VSD with reversed shunt direction (right-to-left) secondary to pulmonary hypertension (Eisenmenger's complex):

- In both cases, after a right ventricular injection of contrast medium, there will be simultaneous opacification of the main pulmonary artery and ascending aorta
- In tetralogy of Fallot, narrowing of the main pulmonary artery with post-stenotic dilation or hypoplasia of the main pulmonary artery should be looked for (Figure 7.83). This will assist in differentiation from a reversed VSD (in which the RVOT, main pulmonary artery and its branches will appear normal in size and will fill up with contrast medium)
- Note that pulmonic stenosis in combination with an isolated right-to-left shunting VSD can be extremely difficult to differentiate from a tetralogy of Fallot in all imaging studies.

<<Figure 7.83 near here>>

Echocardiography

Echocardiographic findings include:

- Right ventricular hypertrophy
- High and often large VSD
- Aorta 'overriding' the IVS (Figures 7.84 and 7.85)
- Pulmonic stenosis (Figure 7.85b)
- Reduced dimensions of the LA and LV.

<<Figures 7.84 and 7.85 near here>>

Doppler studies:

- A low-velocity systolic flow from the RV to the LV through the septal defect (Figure 7.86).

- A high-velocity turbulent flow across the pulmonic stenosis.

<<Figure 7.86 near here>>

Scintigraphy
Scintigraphic findings in first pass radionuclide angiocardiography include simultaneous appearance of the aorta and pulmonary arteries after the dextrophase. It is not possible to distinguish tetralogy of Fallot from a reverse PDA on the basis of a first pass study alone.

Scintigraphy with ^{99m}Tc-MAA shows activity outside the lungs with prominent uptake in the renal and cerebral cortices, the latter finding allowing differentiation from a reverse PDA (Figure 7.87).

<<Figure 7.87 near here>>

Vascular ring anomaly

A vascular ring anomaly results from an abnormal embryonic development of the primordial aortic arches (aortic arches III, IV or VI) around the embryonic pharynx. This leads to postnatal constriction of the thoracic oesophagus and development of a secondary megaoesophagus (see also Chapter 8). Dogs and cats are affected; in dogs, German Shepherd Dogs and Irish Setters are predisposed to this condition.

A spectrum of abnormalities may be encountered, the most common ones being:

- Persistance of the right fourth aortic arch (PRAA) with a left-sided ligamentum arteriosum (most common anomaly) and/or a retro-oesophageal left subclavian artery
- Double aortic arches
- Normal fourth aortic arch with retro-oesophageal (aberrant) right subclavian artery (usually not a major problem from a clinical stand-point).

Rarely, a PDA is associated with a vascular ring anomaly.

Radiography
Radiographic findings include:

- If the vascular ring anomaly has entrapped the oesophagus, then oesophageal dilation may be seen cranial to the heart base. Oesophagography may be required to identify this
- A soft tissue or heterogenous granular opacity, cranial to the heart base with general ventral displacement of the trachea may be seen on survey radiographs (Figure 7.88)
- There may be a ventral alveolar pattern if aspiration pneumonia has occurred
- Moderate or marked focal leftward curvature of trachea near the cranial border of the heart in DV or VD radiographs (see Figure 9.18**, p. xx**) is reliable sign of PRAA in young dogs with consistent clinical signs (as opposed to the normal right-sided tracheal curvature in normal animals)
- In cases of PRAA the leftward margin of the descending aorta is not visible
- If a well defined, normal left descending aortic margin is clearly identified on the VD or DV view, a less common vascular ring anomaly may be suspected: angiography is warranted in such cases, to confirm the diagnosis and plan the best surgical approach.

<<Figure 7.88 near here>>

Oesophagography: Oesophageal contrast studies show accumulation of the contrast medium in a distended oesophagus cranial to the heart base and abrupt tapering at the level of the sixth pair of ribs (Figure 7.89). Occasionally, the oesophageal dilation is generalized and these cases carry the worst prognosis (see Chapters 1 and 9 for more information).

<<Figure 7.89 near here>>

Angiocardiography: This is not necessary in all cases, as an adequate presurgical diagnosis of vascular ring constriction of the oesophagus can be made with barium studies of the oesophagus. It may be valuable to identify animals that have a normal left fourth aortic arch but abnormal formation of the right fourth and/or right sixth aortic arch: in these cases a right thoracotomy is the best surgical approach as opposed to the more common left approach.

A left-ventricular injection of contrast medium is usually performed:

- Persistent right fourth aortic arch is more consistently demonstrated on DV views, where the opacified aortic arch can be seen coursing to the right of the trachea
- A retro-oesophageal left subclavian artery can be demonstrated in lateral views as it courses cranially from its origin dorsally and to the left of the aorta at about the level where the thoracic aorta begins to parallel the vertebral column. The persistent left subclavian artery compresses the oesophagus dorsally when it begins running ventrally to the thoracic inlet.

Aortic coarctation

Aortic coarctation is a very rare condition in dogs and even more rare in cats. The disease consists of a narrowing of the aortic lumen that usually occurs at the aortic isthmus, the segment of the aorta between the origin of the left subclavian artery and the insertion of the ductus arteriosus.

It is believed to be due to spreading of the specialized contractile ductal tissue into the aorta to form a sling around it, which after birth becomes part of an obstructive curtain of tissue. The narrowing is responsible for obstruction to the flow into the descending aorta and aneurysmal dilation of the aorta distal to the obstruction. The narrowing process takes weeks to develop, which leaves time for the LV to

adjust to the increased pressure load and for collateral circulation to develop; this is why there are often no clinical signs associated with the disease.

Radiography
Radiographic findings are non-specific:

- An exaggerated and enlarged aortic arch is visible, creating a soft tissue opacity bulging out from the mediastinum into the left cranial thorax
- The trachea can be displaced ventrally (lateral view) and to the right (VD or DV view) by the soft tissue opacity
- Notching of the ribs (small indentation surrounded by fine rim of sclerotic bone) is highly suggestive of aortic coarctation in humans and has also been reported in dogs. It occurs due to enlargement of the intercostal arteries. These carry collateral circulation in a retrograde direction from both the costocervical trunk and internal thoracic arteries to supply the distal aorta.

Angiocardiography: Cardiac catheterization and aortography are indicated to localize accurately the site of obstruction, determine the length of the coarctation and identify associated malformations (Figure 7.90).

<<Figure 7.90 near here>>

Echocardiography
Echocardiography does not allow the diagnosis of aortic coarctation, though endo-oesophageal echocardiography might be helpful. Echocardiography can rule out the presence of associated cardiac anomalies, such as a VSD.

Cor triatriatum sinister and dexter

Cor triatriatum is a rare congenital defect that manifests as a partitioned RA (cor triatriatum dexter, CTD) or even less commonly, a partitioned LA (cor triatriatum sinister, CTS):

- CTD is caused by failure of the right sinus venosus valve to regress during embryogenesis
- CTS is caused by an abnormal connection of the LA with the pulmonary veins.

The condition is rare but well documented in dogs. It is extremely rare in cats.

In both CTD and CTS, there is a membrane dividing the atrium into two compartments, resulting in abnormal venous return to the affected atrium (CdVC in the case of CTD and pulmonary veins in the case of CTS). The degree of venous pressure elevation depends on the degree to which the dividing membrane is perforated. If the degree of obstruction is significant then subsequent cardiac failure will occur. This will be left-sided failure in CTS and right-sided failure in CTD. In the latter there may only be congestion of the caudal half of the body (i.e. ascites and hepatomegaly, but no jugular distension) and in this way the condition may mimic a Budd–Chiari-like syndrome.

Note that supravalvular mitral stenosis (SMS) closely resembles CTS. The only difference is the level of the obstructing membrane. In SMS the obstruction is distal to the foramen ovale and LAu enlargement will occur; in CTS the obstruction is proximal to the foramen ovale and the LAu is therefore downstream and does not enlarge.

Radiography
Radiographic findings include:

- CTD:
  - Marked dilation of the RA, creating a soft tissue opacity bulge between 09.00 and 11.00 o'clock on the VD and lateral views
  - Dilation of the CdVC
  - There may be hepatomegaly and peritoneal effusion (right-sided cardiac failure).
- CTS:
  - Left atrial dilation
  - Pulmonary venous enlargement
  - There may be cardiogenic pulmonary oedema (left-sided cardiac failure).

Angiocardiography:

- CTD: injection of contrast medium in the CdVC might reveal an obstruction to venous return at the level of the RA, with saccular dilation of the caudal RA (Figure 7.91).
- CTS: selective angiography is difficult as the dividing membrane in the LA makes it difficult to opacify the entire LA. Non-selective angiocardiography at the late phase (levophase) can provide more information, revealing that the large soft tissue opacity corresponds to a saccular dilation of the LA, which occasionally will appear bilobed (Figure 7.92).

<<Figures 7.91, 7.92 near here>>

Echocardiography
Echocardiographic findings include:

- CTD:
  - Echocardiography can provide the diagnosis of CTD
  - Right atrial dilation without other obvious changes is seen
  - A thin membrane can be seen in the caudal RA, dividing the atrium into a larger cranial compartment and a smaller caudal compartment
  - The caudal compartment includes the entrance of the CdVC, which is distended
  - Turbulent venous inflow can be seen through the membrane using Doppler studies.
- CTS:
  - Echocardiography can provide the diagnosis of CTS (Figure 7.93)
  - A visible partition of the LA is seen

  - The perforations in the dividing membrane are occasionally identified
  - Doppler studies reveal a turbulent venous flow through the dividing membrane.

<<Figure 7.93 near here>>

Myocardial diseases

Cardiomyopathies are defined as diseases of the myocardium associated with cardiac dysfunction. They may be primary or secondary to another insult. In making a diagnosis of idiopathic cardiomyopathy, active exclusion of inflammatory, infiltrative, metabolic, endocrine, nutritional, pulmonary, systemic hypertensive, toxic and other conditions is required. Specific secondary cardiomyopathies may be subdivided according to their aetiology (e.g. hyperthyroid cardiomyopathy).

Idiopathic cardiomyopathies are further subdivided according to their morphological (echocardiographic) appearance and dysfunction, according to the World Health Organization, and include:

- Dilated cardiomyopathy (DCM)
- Hypertrophic cardiomyopathy (HCM)
- Restrictive cardiomyopathy (RCM)
- Arrhythmogenic right ventricular cardiomyopathy (ARVC).

Canine myocardial disease

Dilated cardiomyopathy

- DCM is characterized by left-sided or four-chamber dilation and impaired left ventricular systolic function.
- DCM is a major cause of morbidity and mortality in various large and giant breeds of dogs, including Deerhounds, Dobermanns, Great Danes, Irish Wolfhounds, Newfoundlands, St Bernards and spaniel breeds (Cocker and American Cocker, English Springer).
- Presentation is usually associated with the onset of congestive heart failure, with coughing or dyspnoea. Exercise intolerance may be marked. It may be associated with episodic tachyarrhythmias, such as ventricular tachycardia, resulting in syncopal episodes.
- There is a long, presymptomatic phase of this disease and the onset of congestive heart failure is merely the 'tip of the iceberg'.
- Clinical findings may be subtle. Arrhythmias, such as atrial fibrillation, may be present. A soft murmur may be detected, due to mitral regurgitation, secondary to dilation of the mitral annulus. Diastolic gallops may be detected in the decompensated animal.
- Imaging is essential to making the diagnosis of DCM. It demonstrates the chamber dilation and impaired systolic function. However, it also has an important role in excluding other cardiac conditions, which may secondarily result in congestive heart failure or poor output signs. In the presymptomatic dog, from a breed or family with DCM prevalence, echocardiographic screening may be requested prior to breeding.

Radiography: Thoracic radiographs should be obtained in all dogs where left-sided congestive heart failure is suspected clinically, or is imminent. Radiographs are exquisitely sensitive at documenting the volume load associated with left-sided congestive heart failure (pulmonary oedema). A pulmonary infiltrate associated with left atrial enlargement and pulmonary venous congestion is consistent with cardiogenic pulmonary oedema (Figures 7.94 and 7.95).

<<Figures 7.94, 7.95 near here>>

In clinical symptomatic DCM, the cardiac silhouette is almost always abnormal. Possible findings include:

- Left atrial and left ventricular enlargement
- Right atrial and right ventricular enlargement
- The cardiac silhouette may have a sharp 'static' outline with severe systolic function (loss of normal systolic–diastolic movement blur with each cardiac cycle). The concomitant presence of left atrial enlargement helps distinguish this from a pericardial effusion
- In left-sided congestive failure:
  - Triad of signs: LA enlargement, pulmonary venous distension and pulmonary infiltrate
  - The pulmonary infiltrate due to pulmonary oedema is normally predominantly perihilar. It may be interstitial, alveolar or mixed.
- In right-sided congestive failure:
  - Evidence of abdominal effusion (ascites) in the cranial abdomen
  - Distended CdVC
  - Pleural effusion
  - Note: in a dog with cardiomegaly presenting with predominantly right-sided congestive heart failure, pericardial effusion *must* be excluded. DCM normally presents with left-sided congestive failure.

In presymptomatic ('occult') DCM, the lung field and pulmonary vasculature are usually unremarkable. The cardiac silhouette may or may not show signs of generalized cardiomegaly or specific chamber enlargement. Radiographs are not useful in screening for presymptomatic DCM. They are useful in determining whether or not the dog is likely to show clinical signs in the near future.

Echocardiography: 2D and M-mode echocardiography are normally sufficient to make the diagnosis of DCM. However, since the stringent diagnosis of DCM requires the active exclusion of other congenital or acquired cardiac disease, colour flow and Doppler echocardiography are indicated. Furthermore, Doppler studies are required for

identifying and classifying abnormalities in diastolic function, and for supporting the presence of systolic function. Guidelines for the robust diagnosis of DCM have recently been proposed.

2D echocardiographic abnormalities in DCM typically include:

- Subjective findings include LV dilation with relatively thin walls and poor contractility
- A rounded LV chamber (increased sphericity) – LV diastolic length:LV diastolic width at chordae tendinae level (= M-mode left ventricular internal dimension in diastole, LVIDd) <1.65 (Figure 7.96)
- End-systolic volume index >30 ml/m^2 (confirms systolic dysfunction and chamber dilation) (Figure 7.97b)
- Ejection fraction <40% (Figure 7.97)
- The LA is dilated in symptomatic dogs
- The right sided chambers may or may not be dilated.

<<Figures 7.96, 7.97 near here>>

M-mode echocardiographic abnormalities in DCM include:

- Increased LVIDd and left ventricular internal dimension in systole (LVIDs) for size or breed-based reference ranges (Figure 7.98)
- Relative wall thickness (left ventricular free wall in diastole, LVFWd:LVIDd) is decreased
- Fractional shortening <20%
- Increased mitral valve M-mode EPSS (Figure 7.99)
- Reduced aortic root systolic excursion on aortic M-modes, and possibly premature closure of aortic valve
- The LA may be enlarged in symptomatic dogs (increased M-mode LA:Ao ratio)
- Systolic time intervals: M-mode aortic pre-ejection period (PEP):ejection time (ET) >0.4.

<<Figures 7.98, 7.99 near here>>

Findings with Doppler studies in DCM include:

- Colour flow Doppler may identify mitral and/or tricuspid regurgitation, without grossly abnormal valve apparatus, due to stretch of the atrioventricular annuli
- Mitral regurgitant velocity may be lower than normal, due to impaired LV systolic function and elevated LA pressures, giving a reduced systolic PG between the LV and LA. Mitral regurgitant velocity <4 m/s is associated with poor prognosis
- Aortic velocity and velocity time integrals may be lower than normal, due to systolic dysfunction
- Aortic PEP:ET ratio may be increased (>0.4)
- The assessment of diastolic function is important to provide prognostic information. Dogs with a restrictive physiology have reduced survival.

If dogs are screened for presymptomatic DCM, various echocardiographic abnormalities may be identified, such as impaired systolic function prior to unequivocal evidence of LV dilation. Other cases may have LV enlargement with apparently preserved systolic function. The systolic time interval, PEP:ET ratio, appears to offer the best discrimination between normal and affected dogs. Serial evaluation is required to confirm that these echocardiographic findings precede the development of DCM, and a scoring system has been proposed to monitor these cases.

Boxer cardiomyopathy or arrhythmogenic right ventricular cardiomyopathy

Boxer dogs with cardiomyopathy may present with the classical findings of DCM, as described above. Until recently, this was the most common presentation in UK and European Boxers (see Figures 7.96 and 7.97). In contrast, North American Boxers are reported to present with malignant ventricular arrhythmias, of right ventricular origin, with minimal changes initially on echocardiography, although systolic dysfunction and LV dilation can occur later in the course. This form is now being increasingly recognized in the UK and Europe. In some affected Boxers, the RV may appear dilated (Figure 7.100) with dysplastic RV apical papillary muscles (Figure 7.100c). However, in many Boxers, there may be no echocardiographic evidence of structural abnormalities despite severe ventricular arrhythmias.

<<Figure 7.100 near here>>

Hypertrophic cardiomyopathy

HCM is rare in dogs, although it has been documented. Echocardiographic features are similar to those described in cats (see below). One unusual condition, which may represent a form of HCM, is *dynamic LVOT obstruction*, due to *systolic anterior motion* of the anterior mitral valve leaflet (Figure 7.101). This has been described in young, growing dogs. They may outgrow this lesion. It must be distinguished from subaortic stenosis with associated dynamic obstruction.

<<Figure 7.101 near here>>

Feline myocardial disease

Cats as a species display the plethora of primary myocardial diseases, classified by the World Health Organization. Imaging, particularly Doppler echocardiography, plays a vital role in determining the definitive diagnosis. Ancillary diagnostic techniques are required to exclude conditions, which may secondarily affect the myocardium (blood pressure measurement, clinical pathology, etc.).

Hypertrophic cardiomyopathy

HCM is the most common form of myocardial disease affecting cats. It is genetically transmitted as an acquired, autosomal dominant trait (proven in Maine Coons). It is characterized by concentric left ventricular hypertrophy, which may be regional or asymmetrical. The relative wall thickness:LV chamber ratio is greatly

increased. Evidence of diastolic dysfunction is documented by Doppler studies. This is manifested by progressive left atrial enlargement.

Symptomatic cats present with signs of left-sided congestive heart failure (dyspnoea due to pulmonary oedema). This is often of sudden onset, and may be preceded by a stressful incident or fluid loading. There is a long presymptomatic phase. Affected cats may fortuitously have a heart murmur detected, which leads to an early diagnosis. Other cats may be detected by echocardiographic screening schemes in breeds, such as Maine Coons and Persians.

Radiography: This is indicated to identify the haemodynamic consequences of heart disease, with evidence of increased left-sided filling pressures:

- Left atrial enlargement
- Pulmonary venous distension
- Pulmonary infiltrate, which may affect any region of the lung field, and be patchy, interstitial, alveolar or mixed. This is consistent with pulmonary oedema (Figure 7.102).

Radiography does not differentiate between the various myocardial diseases. In asymptomatic HCM, there may be no gross radiographic evidence of cardiomegaly or specific chamber enlargement. In symptomatic HCM, left atrial enlargement is evident. Radiographically, apparent bi-atrial enlargement may be documented, but in most cases, echocardiography shows that the marked left atrial enlargement pushes the interatrial septum and the right atrial wall more to the right and cranially, giving this impression on the cardiac silhouette. The apparent bi-atrial enlargement gives the classical 'valentine' shaped heart (Figure 7.102b). A pleural effusion may be present, associated with biventricular failure.

<<Figure 7.102 near here>>

Doppler echocardiography: This is the imaging modality of choice to distinguish between the various forms of myocardial disease. The following 2D and M-mode findings (Figure 7.103) are typical in HCM:

- Generalized or focal, symmetrical or asymmetric hypertrophy, with diastolic wall thickness ≥6 mm (Figure 7.104)
- The LV chamber diameter may be normal or small
- Subjective impression that the papillary muscles are hypertrophied
- LA size may be normal (Figure 7.105) or dilated (Figure 7.106)
- Systolic function is normally preserved; fractional shortening can often be >45%, indicating a hyperkinetic LV, due to the low wall stress (Figure 7.107).

<<Figures 7.103, 7.104, 7.105, 7.106, 7.107 near here>>

Hypertrophic (obstructive) cardiomyopathy: This term is reserved for forms of HCM where dynamic obstruction of the LVOT is documented by echocardiography. A simplified series of events leading to this finding include:

- Turbulence of flow in the LVOT as blood travels around a basal septal bulge associated with the hypertrophy. This may be recognized as colour variance in the LVOT
- This may result in a Venturi effect on the anterior mitral valve leaflet, which is 'sucked' into the LVOT during systole. This further narrows the LVOT
- The LVOT and aortic velocities may then be increased, with abnormal biphasic acceleration on spectral Doppler, giving a scimitar shape (Figure 7.108a)
- The mitral valve is therefore incompetent, and typically, an eccentric jet of mitral regurgitation, coursing towards the posteriolateral LA wall, is recorded by colour flow Doppler (Figure 7.108b)
- Other factors are almost certainly involved in *s*ystolic anterior motion of the mitral valve, such as altered papillary muscle alignment
- Systolic anterior motion can be confirmed by mitral M-mode (Figure 7.108c). Normally, the temporal resolution of 2D is not sufficient to confirm this in real time.

<<Figure 7.108 near here>>

Hypertrophic (obstructive) cardiomyopathy (HOCM) is frequently diagnosed in asymptomatic cats, due to the identification of a heart murmur. The murmur is due to mitral regurgitation or LVOT obstruction, and it may be variable depending upon how relaxed or stressed the cat is. It may not be possible to document HOCM in a sedated or very relaxed cat during echocardiography.

Pulsed wave Doppler assessment of diastolic function: Diastolic function can be classified by studies of:

- Mitral inflow
- Isovolumic relaxation time (IVRT)
- Pulmonary venous flow (PVF)
- Mitral inflow propagation (Vp)
- Tissue Doppler imaging.

The typical findings in feline HCM patients at various stages of the disease are documented in Figure 7.109. The mitral inflow patterns are illustrated in Figures 7.110 and 7.111. Corresponding abnormalities in diastolic dysfunction are illustrated for IVRT (Figure 7.112) and PVF (Figure 7.113).

<<Figures 7.109, 7.110, 7.111, 7.112, 7.113 near here>>

Restrictive cardiomyopathy

RCM is much more poorly understood than other

forms of cardiomyopathy in the cat. It is characterized by relatively normal wall thickness, LV chamber size and systolic function, but marked left atrial enlargement is apparent. The pathophysiology is associated with reduced LV compliance and diastolic dysfunction. With elevated filling pressures, there is a restrictive filling pattern on mitral inflow (however, note that this diastolic abnormality is not specific for RCM).

There are two main forms:

- An endomyocardial form: believed to be post-inflammatory (feline endomyocarditis). There is irregular endocardial thickening
- A myocardial form: the initiating factor is unknown and the endocardium appears normal.

A consequence of the marked left atrial enlargement may be thromboembolism. RCM is usually not detected until the cat presents with left-sided congestive heart failure or thromboembolic complications.

Radiography: This does not differentiate between the various myocardial diseases. In RCM, there is usually dramatic left atrial enlargement (which may appear radiographically to reflect bi-atrial enlargement). Signs of left-sided congestive heart failure include pulmonary venous distension and a pulmonary infiltrate.

Doppler echocardiography: Echocardiographic findings include:

- There is a marked left atrial enlargement (Figure 7.114)
- An organized thrombus may be apparent in the LA or LAu
- Spontaneous echocontrast of blood within the LA may be seen, giving the impression of swirling 'smoke'. This represents a prothrombotic state (Figure 7.114c)
- The LV has relatively preserved systolic function, and relatively normal LV dimensions and wall thickness
- This is a disease of diastolic dysfunction. Detailed Doppler assessment of myocardial function is consistent with a restrictive physiology (see Figures 7.103 and 7.109)
- In the endomyocardial form of RCM, adhesions may be seen crossing the LV chamber and the endocardium may appear irregularly thickened (Figure 7.114d)
- In the myocardial form of RCM, the myocardium and endocardium echotexture appear unremarkable.

<<Figure 7.114 near here>>

Dilated cardiomyopathy

DCM is now rarely diagnosed in the cat; it used to be one of the most common forms of feline myocardial disease prior to taurine deficiency being implicated in this disease in the late 1980s. The nutritional history of any cat diagnosed with this condition should be ascertained. DCM may be familial in certain breeds of cat, such as the Abyssinian.

Presymptomatic disease is not normally recognized. Cats normally present with severe, biventricular failure with cardiogenic shock. They, therefore, must be stabilized prior to carrying out diagnostic tests. It must be appreciated that myocardial dysfunction and LV dilation may be an end-stage consequence of a variety of primary and secondary cardiomyopathies in the cat (e.g. HCM, thyrotoxic cardiomyopathy).

Radiography: Radiographic findings include:

- Affected cats often have a pleural effusion, so assessment of the lung field and cardiac silhouette is compromised (Figure 7.115).
- There is generalized cardiomegaly and the LV apex may appear to be rounded.

Radiographic findings do not distinguish between the various forms of myocardial disease.

<<Figure 7.115 near here>>

Doppler echocardiography: Similar changes are described as listed for canine DCM:

- The LV is dilated in both diastole and systole (see Figures 7.103 and 7.116) and systolic function is impaired. Wall thickness is normal or reduced
- There may be mitral and/or tricuspid regurgitation, secondary to stretch of the atrioventricular annulus.

<<Figure 7.116 near here>>

An example of a cat with secondary myocardial dysfunction, secondary to untreated hyperthyroidism and possibly associated with myocardial infarction, is shown in Figure 7.117.

<<Figure 7.117 near here>>

Arrythmogenic right ventricular cardiomyopathy

Feline ARVC has been recently described. The disease is histopathologically characterized by fibrofatty replacement and infiltration in the myocardium, most pronounced in the RV. Affected cats may or may not show malignant ventricular arrhythmias. They may show ascites as a manifestation of right-sided congestive heart failure.

Radiography: Radiographic findings include:

- Changes may show evidence of right-sided congestive heart failure or biventricular failure
- Ascites in association with a dilated CdVC indicate that the abdominal effusion is associated with right-sided congestive failure (Figure 7.118). A pleural effusion may also be present
- There may or may not be radiographic evidence

of left-sided congestive heart failure.

<<Figure 7.118 near here>>

Doppler echocardiography: Echocardiographic findings include:

- There is marked right-sided RA and RV dilation (see Figure 7.118b)
- Tricuspid regurgitation may be evident due to stretch of the tricuspid annulus (Figure 7.119a). ARVC must be distinguished from tricuspid dysplasia (which will usually show an immature age of onset compared with the middle- or older-age onset in ARVC).
- A hallmark of this condition is evidence of dysplastic papillary muscles within the RV apex (the transducer needs to be moved far enough caudally and sternally to appreciate this feature from the RPS long-axis view)
- Variable changes affect the LV. In advanced disease, the LV also becomes dilated and hypokinetic (Figure 7.119b).

<<Figure 7.119 near here>>

Unclassified cardiomyopathy
The findings with feline myocardial disease can be diverse. In some cats, the echocardiographic findings do not neatly fall into one of the above categories. For example, the wall thickness may be normal, but systolic function is impaired and there is evidence of abnormal relaxation (Figure 7.120). It is perfectly acceptable to categorize these cats as unclassified. It should be borne in mind that this may represent a heterogenous group.

<<Figure 7.120 near here>>

Radiographic findings reflect the presence of congestive heart failure and echocardiographic findings will have features from more than one of the above groups.

Secondary myocardial disease

The myocardium is influenced by other systemic factors. These include:

- Systemic hypertension
- Hyperthyroidism
- Hypothyroidism
- Chronic renal failure
- Acromegaly
- Respiratory disease.

These may result in significant cardiac disease or congestive heart failure. Diagnostic tests other than imaging are required to make the diagnosis of a secondary myocardial disease.

Systemic causes of myocardial depression
In the presence of impaired systolic function, it should not be merely presumed that this represents, for example, a preclinical phase of DCM. Conditions which should be excluded are:

- Tachyarrhythmia. An animal which is tachycardic (e.g. supraventricular tachycardia) may develop a phenotype similar to DCM (called a tachycardiomyopathy). Control of rate (and/or rhythm) should result in improved systolic function
- Assessment of systolic function or chamber dimensions should not be carried out in the presence of a dysrhythmia, if possible. Altered electrical activation of the ventricles or abnormal rate can give misleading results
- Hypothyroidism. There have been several case reports of dogs with hypothyroidism and DCM, with improvement in systolic function associated with thyroid supplementation. It is not clear that the association is cause and effect; many breeds show prevalence of both conditions
- Systemically ill animals may show myocardial depression possibly associated with elevated cytokine levels.

Systemic hypertension
Systemic hypertension results in increased afterload (arterial resistance) on the LV; LV pressure then increases to maintain cardiac output. To normalize wall stress, concentric LV hypertrophy occurs. Therefore, the walls are thick and the LV chamber may be proportionately small.

Systemic hypertension is normally believed to be secondary to a primary problem in animals. Essential hypertension is considered to be rare. Conditions which may result in hypertension include:

- Chronic renal failure
- Hyperthyroidism
- Hyperadrenocorticism
- Acromegaly
- Phaeochromocytoma
- Hyperaldosteronism (Conn's syndrome).

Note that some of these conditions are associated with factors that have a direct trophic effect on the myocardium (see previously), so not all changes are related to the hypertension.

Radiography: Thoracic radiographs may be unremarkable where there is just concentric LV hypertrophy in the absence of left atrial enlargement. If the underlying disease and hypertension leads to left-sided congestive heart failure, there will be left atrial enlargement, pulmonary venous distension and a pulmonary infiltrate consistent with pulmonary oedema.

Doppler echocardiography: 2D and M-mode echocardiographic findings will be similar to those described for HCM.

- Abnormalities of relaxation may be apparent with gross hypertrophy.

- In older dogs, systemic hypertension may result in accelerated progression of degenerative valvular disease.
- Mitral regurgitation is often of high velocity (increased PG between the LV and LA).

Pulmonary hypertension (cor pulmonale)
Pulmonary hypertension is a consequence of primary respiratory disease or abnormalities of the pulmonary vasculature. Mild degrees of pulmonary hypertension are recognized in brachycephalic breeds of dogs and in obesity (Pickwickian syndrome).

Severe pulmonary parenchymal disease, such as idiopathic pulmonary fibrosis in West Highland White Terriers, can result in more significant pulmonary hypertension, although the cardiac manifestations are not usually clinically relevant to the presentation. Pulmonary vascular disease may be secondary to parasitism, such as *Dirofilaria immitis* or *Angiostrongylus vasorum* infestation.

Pulmonary vascular disease may be a consequence of an initially left-to-right shunting congenital heart disease, leading to Eisenmenger's physiology (e.g. reverse-shunting PDA or VSD). It is the pulmonary hypertension which results in reversal of the shunting from the pulmonary into the systemic circulation, for those phases of the cardiac cycle where pulmonary pressures exceed systemic pressures, resulting in cyanosis.

Sometimes, the cause of the pulmonary vascular disease is unknown (essential or primary pulmonary hypertension).

Radiography: Radiographic findings include:

- Evidence of pulmonary hypertension, including right heart enlargement (Figure 7.121).
- If pulmonary hypertension is associated with respiratory disease, abnormalities of the lung field should be apparent (Figure 7.122).
- Evidence of pulmonary vascular disease may include dilated, tortuous or 'pruned' appearance to the pulmonary arteries (particularly in heartworm disease) (Figure 7.123).

<<Figures 7.121, 7.122, 7.123 near here>>

Doppler echocardiography: Echocardiographic findings include:

- Pulmonary hypertension is associated with right ventricular enlargement. The RV is both dilated and concentrically hypertrophied (Figure 7.124a)
- As RV pressure is increased, the IVS appears flattened, which may be most apparent on the RPS short-axis view (Figure 7.124ab)
- Commonly, there is also right atrial enlargement (Figure 7.124a)
- The main pulmonary artery in frequently dilated (wider than the aortic diameter) (Figure 7.124c)
- A detailed Doppler echocardiographic examination, including intravenously administered echocontrast to demonstrate any right-to-left shunts, is indicated to actively exclude structural heart disease, which may result in pulmonary hypertension
- Tricuspid regurgitation (Figure 7.124d) and pulmonic insufficiency (Figure 7.124c) are commonly both present, whatever the cause of pulmonary hypertension
- In the absence of pulmonic stenosis, the tricuspid regurgitant velocity can indicate, by the modified Bernouilli equation, the systolic PG between the RV and RA, and therefore the systolic main pulmonary artery pressure. Thus, a non-invasive determination of systolic pulmonary arterial pressure is possible (Figure 7.124e)
- In the presence of pulmonic regurgitation, the velocity of this jet indicates the diastolic PG between the main pulmonary artery and RV. Thus, non-invasive estimation of diastolic pulmonary arterial pressure is possible (Figure 7.124f).

<<Figure 7.124 near here>>

Myocardial infiltrative disease
Unusually, the myocardium can be affected by infiltration. These infiltrations are normally neoplastic, such as lymphoma. Other, non-cardiac signs usually result in the presentation of the patient. Cardiac dysrhythmias, such as ventricular tachycardia, may be clinically important.

Radiography: Thoracic radiographs may or may not indicate any abnormalities in the cardiac silhouette. Radiographs normally reflect the presence of the primary lesion.

Doppler echocardiography: Echocardiographic findings include:

- Infiltration into the myocardium is usually generalized and results in the impression of thickened myocardial walls (Figures 7.125 and 7.126)
- The normal myocardial echotexture is disrupted and it may appear to be hyperechoic, or show focal hypoechoic regions, or a general 'moth-eaten' appearance
- Both systolic and diastolic dysfunction may be evident as a consequence of the infiltrate.

<<Figures 7.125, 7.126 near here>>

Acquired valvular disease

A list of the differential diagnoses of acquired valvular regurgitation is presented in Figure 7.127.

<<Figure 7.127 near here>>

Myxomatous atrioventricular valvular degeneration

Myxomatous atrioventricular valvular degeneration (also known as endocardiosis, chronic degenerative

valvular disease) is the most common acquired cardiac disease in small-breed dogs. Commonly affected breeds include the Papillon, Cavalier King Charles Spaniel, Poodle and Chihuahua. Myxomatous atrioventricular valvular degeneration is uncommon in dogs under the age of 5 years old. The condition is also seen in large-breed dogs and is rarely seen in cats. The aetiology is unknown but the condition is likely to be hereditary in small dog breeds.

The condition may affect either the mitral or the tricuspid valve. Each of these valves consists of two large leaflets (one mural and one septal) and sometimes smaller commissural cusps between the leaflets. Tough fibrous cords called chordae tendinae attach the components of the valve to the papillary muscles. The leaflets have chordae tendinae from both papillary muscles attached to them, the commissural cusps have chordae tendinae from only one papillary muscle.

The pathological changes in myxomatous atrioventricular valvular degeneration can involve the leaflets and the chordae tendinae. The valve leaflets become thickened (most marked at the free edge) and redundant. The thickening may extend to nodular change. The chordae tendinae may also become thickened where they attach to the valve. Eventually they may rupture and result in flail of the valve leaflet.

The clinical presentation of the disease varies from a soft heart murmur to end-stage heart disease and failure. Large dogs may be less tolerant and have a more drastic progression of the condition.

Radiography
There is marked variation in radiographic features, depending on the progression of the disease (Figure 7.128). The major features include:

- Left atrial enlargement (one of earliest and most consistent signs)
- Left ventricular enlargement
- Right atrial and ventricular enlargement, depending on the degree of tricuspid involvement.

There may also be:

- Pulmonary venous congestion (enlarged pulmonary veins)
- Evidence of left-sided heart failure: interstitial or alveolar pattern representing oedema. Supporting evidence of pulmonary venous congestion and left-sided enlargement is useful (see comments in Interpretive principles, above)
- Evidence of right-sided heart failure.

<<Figure 7.128 near here>>

Contrast studies: Angiocardiography has been replaced by echocardiography.

Echocardiography
Echocardiography is useful to confirm the diagnosis and assess systolic function. It cannot be used to identify heart failure, hence radiography and ultrasonography are complementary techniques in the evaluation of this condition. It is important to examine the entire valve as the lesions may be unevenly spread (see Chapter 2 for details about echocardiographic measurements).

- Early signs include a systolic bulging of the valve leaflet(s) with or without regurgitation.
- This progresses to thickening of the leaflet, most pronounced at the tip (Figure 7.129). The leaflet may appear thickened, nodular or club-like. Changes are often more pronounced on the septal leaflet. Note that the valvular changes in large-breed dogs may not appear obvious, yet severe disease may still be present.
- Thickening of the chordae tendinae.
- Flail leaflet (Figure 7.130):
  - Mild to moderate: the tip of the leaflet moves into the LA during systole
  - Severe: the entire leaflet moves into the LA during systole.
- Enlarged LA (Figure 7.131). It is very important to assess left atrial size as this parameter reflects severity (except in cases of acute chordae tendinae rupture).
- LV and M-mode parameters:
  - Mild mitral myxomatous valvular degeneration does not lead to abnormal left ventricular size
  - However, with time the left ventricular end-diastolic short-axis dimension increases but the end-systolic dimension remains the same. This is the result of *eccentric hypertrophy* seen in moderate to severe disease
  - It is important to note that with moderate to severe mitral regurgitation the values of ejection phase indices (e.g. fractional shortening, ejection fraction, velocity of circumferential fibre shortening) are often *increased*. This is due to the rapid regurgitation of left ventricular blood into the low pressure LA during systole
  - Hyperdynamic wall motion will be seen (Figure 7.132)
  - Small-breed dogs often have a fractional shortening percentage of greater than 50% with severe mitral regurgitation. Hence, if a normal fractional shortening (or other ejection phase index) is identified in a small-breed dog with severe mitral valvular disease, then *reduced myocardial contractility is present*
  - End-systolic volume indices more accurately assess myocardial contractility
  - Breed differences: note that myocardial failure is more severe and develops earlier in large-breed dogs with myxomatous mitral valvular degeneration. Also note that large dogs usually have a fractional shortening percentage within the 25–40% range with severe mitral regurgitation.
- Right ventricular and right atrial enlargement, depending on the degree of tricuspid disease.

<<Figures 7.129, 7.130, 7.131, 7.132 near here>>

Doppler studies:

- Valvular insufficiency (mitral, tricuspid) (Figure 7.133). Note that a small jet near the mitral valve should not be over interpreted as this can be seen in normal dogs.
- Typically the mitral regurgitant jet is 5–6 m/s in dogs without myocardial failure. The speed of the jet depends on the pressure differences between the LA and LV:
  - Systemic hypertension *increases* jet velocity
  - Systemic hypotension, a large orifice, dramatic increases in left atrial pressure and systolic failure *decrease* jet velocity.
- An increased tricuspid regurgitant jet velocity (≥2.8 m/s) indicates pulmonary hypertension. Pulmonary oedema, secondary to severe mitral disease or other causes of pulmonary hypertension should be considered (see Appendix). Pulmonic stenosis will also increase the velocity of a tricuspid regurgitant jet, but other changes will be present (see Congenital heart disease, below).
- The size of the mitral regurgitant jet can be compared with the size of the LA and is useful in assessing the severity of the disease.
- Additional Doppler techniques have been described, but left atrial size remains a more reliable parameter for the evaluation of the disease severity.
- Mitral diastolic filling velocity is usually increased when significant mitral regurgitation is present.
- Doppler parameters of systolic function can be assessed (see Chapter 2).

<<Figure 7.133 near here>>

Infective endocarditis

Infective endocarditis involves bacterial infection and inflammation of the cardiac valves in almost all cases, though strictly speaking the term refers to any infection of the endocardial surface of the heart. Transient or persistant bacteraemia is the most common aetiology. The condition is seen mainly in medium to large pure-breed dogs and rarely in cats. German Shepherd Dogs may be over-represented. The mitral valve and aortic valve are the most commonly affected in both species.

Bacteria colonize the valve and create vegetative lesions or destroy the valve, both of which lead to valvular regurgitation. Less commonly the valve may narrow and become stenotic. Organisms reported in canine endocarditis include *Staphylococcus aureus*, haemolytic and non-haemolytic streptococci and *Escherichia coli*. Rarer isolates include *Corynebacterium*, *Pseudomonas*, *Erysipelothrix* and infection with the proteobacteria *Bartonella vinsonii.*

Clinical presentation of the condition varies. Clinical signs include lethargy, pyrexia, weakness, anorexia, gastrointestinal disease and lameness. A heart murmur is usually, but not always, present. A recent onset of a murmur, pyrexia and the presence of lameness should increase the index of suspicion for infective endocarditis. Blood cultures are used to confirm the diagnosis of bacteraemia, but are not always sensitive.

Radiography
Radiography may show left-sided cardiac enlargement in chronic cases. Contrast studies have been superceded by echocardiography.

Echocardiography
This is extremely useful in diagnosis:

- Visualization of vegetations. Vary from small nodules to large florid cauliflower-like masses (Figure 7.134). The valve lesions can look identical to those in myxomatous valvular degeneration
- Infective endocarditis lesions are usually solitary and more echogenic. History and signalment are helpful in distinguishing the two:
  - Myxomatous mitral valve degeneration: small breed, older, chronic history of murmur, pyrexic, systemically well
  - Infective endocarditis: large breed, recent onset of murmur, pyrexic, systemic illness
- Valvular destruction in the absence of vegetations may also be present. Seen as a defect in the valve and consequent regurgitation
- Abnormal valvular motion.

<<Figure 7.134 near here>>

Doppler studies: Depending upon which valve is involved, aortic or mitral valve regurgitation may be seen.

Aortic and pulmonic insufficiency

There are many congenital diseases that result in clinically significant pulmonic or aortic insufficiency (see Congenital heart disease). However, both pulmonic and aortic insufficiency may also be seen as non-clinically significant findings in acquired conditions. This is especially the case for pulmonic insufficiency, which is an incidental finding in many dogs. Myxomatous degeneration of the valve leaflets has also been described in both locations, but is very unlikely to result in clinical signs.

Other acquired diseases that may result in aortic and pulmonic insufficiency are:

- Aortic – infectious endocarditis (see above)
- Pulmonic – pulmonary hypertension (various causes, see Appendix), main pulmonary artery dilation (e.g. heartworm).

Radiography
There are no significant radiographic changes. Contrast studies are not necessary.

Echocardiography

Pulmonic insufficiency:

- A small flame-like jet of pulmonic insufficiency is commonly seen in clinically normal dogs (Figure 7.135). The maximal velocity is usually 1 m/s.
- Pulmonary hypertension should be considered when the jet velocity is ≥2.2 m/s.
- Pulmonic stenosis will also give a high-velocity jet (see Congenital heart disease).

<<Figure 7.135 near here>>

Aortic insufficiency:

- A small flame-like jet of aortic insufficiency is occasionally seen in clinically normal dogs (Figure 7.136).
- A large jet should alert suspicion of subaortic or aortic stenosis (see Congenital heart disease) and requires further investigation.

<<Figure 7.136 near here>>

Traumatic valvular disease

Traumatic rupture of the chordae tendinae or papillary muscles may occur and has been reported in dogs and cats secondary to falling from a height or other blunt thoracic trauma. The result is acute onset mitral or tricuspid regurgitation.

Radiography

Varying degrees of chamber enlargement may be seen, depending on the time elapsed since the injury occurred, the extent of injury and whether the mitral or tricuspid valve was involved. Animals with left-sided injury develop rapid onset pulmonary oedema.

Echocardiography

Features seen include:

- Structural abnormalities depending on the extent of the injury:
  - Flail leaflet
  - Partial separation of the leaflet(s) of the valve from the valve annulus (Figure 7.137)
  - Abnormal papillary muscles.
- Extensive regurgitant jet from either the mitral or tricuspid valve
- Enlarged left atrial or right atrial dimensions (depending upon which valve is involved).

<<Figure 7.137 near here>>

Pericardial diseases

Pericardial effusion and cardiac tamponade

Pericardial effusion is the most common pericardial disease seen in small animals. Fluid can accumulate in the pericardial sac due to a variety of disorders that can be systemic, cardiac or pericardial in origin. All generally produce the same end result of cardiac tamponade and congestive heart failure.

Cardiac tamponade can be defined as compression of the heart due to collection of blood or fluid in the pericardial sac.

The effusion can be classed as a pure transudate, modified transudate, exudate or haemorrhagic (just as in other areas of the body). Chronic effusions can reach large volumes before clinical signs occur as the pericardium is able to slowly enlarge. More acute pericardial effusions (such as those which occur with left atrial rupture) can produce severe tamponade at small volumes (florid tamponade) and even result in death.

Pericardial effusion may be seen in any dog or cat but older large-breed dogs are predisposed. Clinical signs include lethargy, respiratory distress, anorexia, collapse and abdominal distension. Evidence of right-sided heart failure is usually identified on clinical examination. Heart sounds are muffled on auscultation, pulses are weak and pulsus paradoxus may be present.

Causes of pericardial effusion

The causes of pericardial effusion include (see also Cardiac neoplasia):

- Cardiac haemangiosarcoma
- Idiopathic pericardial effusion
- Heart base neoplasia
- Mesothelioma
- Other.

Cardiac haemangiosarcoma:

- Most common cause of pericardial effusion in retrospective studies.
- Older dogs, average weight 32 kg.
- Golden Retrievers and German Shepherd Dogs are over-represented.
- By the time of diagnosis, cardiac haemangiosarcoma has usually metastasized and should be considered a systemic disease.
- Effusion is haemorrhagic but does not clot.

Idiopathic pericardial effusion: By definition this is an effusion where no underlying cause is identified. It must be a cautious diagnosis as small mass lesions and mesothelioma may be missed on echocardiographic examination and fluid analysis. The features include:

- Older dogs
- Golden Retrievers are over-represented
- The aetiology is poorly understood
- Fluid appears haemorrhagic usually but does not clot.

Heart base neoplasia:

- Usually aortic body tumours.
- Older dogs.
- Brachycephalic breeds are predisposed. It may

be associated with chronic hypoxia inducing hyperplasia and neoplasia of the chemoreceptors in these breeds.
- Rarely reported in cats.
- May metastasize to lung, LA, pericardium and kidney.
- The effusion varies but is often haemorrhagic.

Mesothelioma:

- May be a more common cause of pericardial effusion than originally thought.
- There is no breed predisposition; occasionally seen in the cat.
- This is a difficult diagnosis to make even with histopathology.
- Mesothelioma is a diffuse neoplasm of the pericardium and cannot be diagnosed on echocardiography.
- Fluid analysis cannot distinguish between idiopathic pericardial effusion and mesothelioma.
- One paper reports that accumulation of significant amounts of fluid within 120 days of pericardectomy increases suspicion of mesothelioma.

Other:

- Congestive heart failure. Often present in right-sided heart failure but rarely of clinical significance.
- Feline infectious peritonitis (FIP). Common cause in the cat.
- Coagulopathy:
  - Rarely results in significant tamponade
  - Reported in both dogs and cats.
- Left atrial rupture (uncommon cause):
  - Small-breed dogs with chronic myxomatous mitral valve degeneration
  - Acute tamponade and life-threatening condition
  - Effusion will be haemorrhagic and will clot
  - Clots and fluid seen on echocardiography.
- Trauma. Rare, fluid will be haemorrhagic and will clot.
- Uraemia. Has been reported in both dogs and cats.
- Septic pericarditis (rare):
  - Migrating foreign bodies, such as grass awns
  - Bite wounds
  - Fungal disease – coccidiomycosis, one case of aspergillosis.
- Constrictive pericarditis (see below).
- Cardiac lymphoma. Rare, reported in dogs and cats, pericardial fluid will give diagnosis.
- Cardiac rhabdomyosarcoma. Rare, reported in dogs and cats.

Pericardial effusion in the cat: This is most commonly part of a more generalized disease:

- FIP
- Congestive heart failure
- Lymphoma.

Symptomatic pericardial effusion in the cat is uncommon.

Radiography
Findings vary with the amount of fluid and the rate at which it developed. Rapid accumulation of fluid may cause tamponade without radiographic signs of cardiomegaly. The classic appearance is a generally enlarged and globoid cardiac silhouette (Figure 7.138):

- Sharply defined margins
- No specific chamber enlargement.

<<Figure 7.138 near here>>

A careful examination for heart base masses and right atrial mass effect should be performed; however, often no radiographic evidence is seen. Pulmonary metastases and thoarcic lymphadenopathy should also be looked for on examination. There may be hypoperfused lung fields due to tamponade and radiographic features of right-sided heart failure.

Contrast studies have been replaced by echocardiography.

Echocardiography
This is the diagnostic technique of choice; it is easy, accurate and non-invasive.

<<BOX>>
Important tip
The ultrasound examination should be performed *before* draining the pericardial effusion to improve the chance of visualizing a mass lesion.
<<END BOX>>

- Pericardial effusion is seen as an *anechoic* (to slightly echoic, depending on type) *circular* region surrounding the heart. The pericardium is seen as a thin hyperchoic line surrounding the fluid (Figure 7.139).
- Pleural fluid can occasionally mimic pericardial effusion:
  - Pleural effusion is more diffuse and the edges of lung lobes and the mediastinal structures will be outlined by fluid (see Chapter 13)
  - Pericardial effusion is more abundant at the apex and is scant or absent behind the LA. Pleural effusion will surround the LA.
- The heart may swing in the pericardial effusion.
- Cardiac tamponade (Figure 7.140):
  - This affects the lower pressure right side more than the left
  - There is late diastolic to systolic inversion of the RA and diastolic collapse of the RV.
- Identification of mass lesions:
  - An extremely careful search should be performed from both LPS and RPS approaches. The tip of the right auricular appendage (RAu) should be visualized

(Figure 7.141) and the search should extend dorsally until the aorta and main pulmonary artery are no longer seen (Figure 7.142)
- A negative examination does not rule out neoplasia
- Haemangiosarcomas are often found in the RAu or RA (see Figure 7.149)
- Mass lesions surrounding the aorta and main pulmonary artery are commonly chemodectomas (see Figure 7.150c)
- Mesothelioma will not be seen
- Care should be taken not to diagnose intrapericardial thrombi as mass lesions, but it can be impossible to differentiate between them.

<<Figures 7.139, 7.140, 7.141, 7.142 near here>>

Constrictive and effusive–constrictive pericarditis

Constrictive pericarditis is characterized by a thickened, fibrotic and non-distensible pericardial sac. Two forms have been described: *constrictive* (fusion of the visceral and parietal serous pericardial layers) and *effusive–constrictive* (constriction by the visceral pericardium plus a small volume pericardial effusion). The latter is more common in dogs.

The condition can develop secondary to chronic idiopathic effusion, mycotic pericarditis, chronic septic pericarditis, foreign body reactions, traumatic pericardial haemorrhage and intrapericardial neoplasia. The stiff pericardium will limit ventricular diastolic filling abruptly in mid diastole. Animals present in right-sided heart failure with ascites and jugular venous distension.

Radiography

Radiography may or may not be helpful:

- Cardiac silhouette can be normal or enlarged, depending on the presence and amount of pericardial fluid
- Enlarged CdVC
- Pleural fluid is often seen
- Hepatomegaly and ascites.

Echocardiography

Echocardiographic findings include:

- Cardiac tamponade with only a *small* (millilitres) pericardial effusion
- Diagnosis can be extremely difficult in the absence of a pericardial effusion (constrictive form)
- Occasionally, the pericardium may appear thickened if effusion is present
- Signs of right-sided heart failure (pleural effusion, enlarged CdVC).

Doppler studies: Mitral and tricuspid inflow (E and A waves) may show a restrictive pattern (see Myocardial diseases and Figure 7.143).

<<Figure 7.143 near here>>

Peritoneopericardial diaphragmatic hernia

Peritoneopericardial diaphragmatic hernia (PPDH) is the most common congenital pericardial anomaly in both dogs and cats. This condition is extensively covered in Chapter 14 in the section on diaphragmatic hernias and so will not be discussed here.

Pericardial cysts

Pericardial cysts are rare in dogs and cats. There have been reports of unilocular or multilocular pericardial cyst-like masses in young dogs. These have most often been found to represent organized haematomas. Cysts may also be composed of encapsulated adipose tissue from the omentum or falciform ligament when examined histologically. In many cases the cyst is connected to a fatty pedicle, which enters the diaphragm through a small PPDH. Incarceration of hepatic cysts within the pericardium have been reported in a cat with PPDH.

True endothelium-/epithelium- or mesothelium-lined cysts occur in humans but are extremely rare in the dog and have not been reported in the cat.

Clinical signs of pericardial cysts include abdominal distension, dyspnoea and anorexia. Most cases have concomitant pericardial effusion (see above).

Radiography

Radiographic findings depend on the size and position of the cyst:

- Small cysts are not seen radiographically
- Large cysts can cause bulging of the cardiac silhouette in the location of the cyst
- If the pericardial sac also contains fluid the cardiac silhouette will have a globoid shape
- Other features of PPDH may also be present if this is co-existing.

Contrast studies: Pneumopericardiography has been described. Cysts were seen as mass-like lesions within the pericardial sac. This technique has been superceded by ultrasound examination.

Echocardiography

This is extremely useful in evaluating pericardial content:

- Single or multiple anechoic thin walled cyst-like structures within the pericardium
- There may be pericardial effusion
- There may be co-existing PPDH.

Pericardial defects

Pericardial defects are rare in dogs. Defects may be as extensive as a congenital absence of the pericardial sac (extremely rare) or smaller partial defects in varying locations. The aetiology is unknown; congenital, inflammatory, traumatic and necrotic causes have all been considered. Defects ventral to the level of the phrenic nerve are thought unlikely to

be congenital. Parts of the heart may herniate through the defect, including part or all of an atrium or myocardial tissue (Figure 7.144). If myocardial tissue becomes incarcerated then ischaemia, necrosis and shock can develop without specific clinical signs. Pericardial defects are most often incidental postmortem diagnoses.

<<Figure 7.144 near here>>

Radiography
The defect itself is *not* seen. Radiographic signs depend on the size of the rupture and the presence of herniated parts of the heart; a protruding soft tissue opacity mass(es) is seen bulging out from the contour of the RA, the RV or possibly left atrial area (Figure 7.145a). A pericardial defect should be considered as a differential for a *mediastinal mass* with a border that effaces with the cardiac silhouette. Fluctuations in size may be seen on repeated thoracic radiographs and provide a clue that this is not a typical solid mediastinal mass.

<<figure 7.145 near here>>

Contrast studies: A non-selective venogram is a fairly simple technique to assist in the diagnosis. This should be performed via the right jugular vein and an immediate lateral radiograph obtained (Figure 7.145b).

Echocardiography
This may not be very helpful in diagnosis. In the case of RAu herniation, there is marked right atrial dilation but with an inability to define the margins due to lung interference. Changes related to the anomaly may not be seen. In the case of cardiac apex incarceration in a cardiac defect, left ventricular dilation and poor myocardial contractility will be seen.

Computed tomography
CT angiography has been used successfully to confirm herniation of cardiac structures (RAu) through the pericardial defect.

Pneumopericardium
This is a very rare condition that has been diagnosed secondary to trauma and in association with pneumothorax. It can also be seen in combination with bronchial or pulmonary parenchymal tear and pneumomediastinum. Theoretically, an infection with a gas-producing organism may produce a pneumopericardium.

Radiography
Air must be seen surrounding the entire heart and outline the atria and the intrapericardial parts of the aorta and main pulmonary artery in order to diagnose a pneumopericardium (Figure 7.146). Pneumothorax with air surrounding the pericardium may look like pneumopericardium and is much more common. A horizontal beam (decubitus) view centred on the heart may help to differentiate between air in the pericardial sac and pneumothorax.

<<Figure 7.146 near here>>

Computed tomography
CT may be used to confirm the diagnosis, but is probably unnecessary. It may be useful when trying to identify the primary cause.

Cardiac neoplasia

Cardiac neoplasia is rare in the dog and is seen even less in the cat. The types of neoplasia seen vary between the two species (Figure 7.147). The most common type of cardiac neoplasia in dogs is haemangiosarcoma and in cats is lymphoma. Clinical signs vary with the type of tumour, location and presence of a pericardial effusion. Cardiac neoplasia generally has a poor prognosis.

<<Figure 7.147 near here>>

Radiographs are insensitive for the detection of cardiac neoplasia. Intrapericardial masses need to reach a substantial size before they produce any radiographic size and shape changes in the cardiac silhouette. Ultrasonography is much more valuable in evaluating the presence of cardiac masses but, as described in the section on pericardial diseases, small masses may be missed, particularly in the absence of pericardial effusion. An extremely careful examination is required from both LPS and RPS windows and in long and short axis. Every attempt should be made to visualize the entire RA and the tip of the auricular appendage. CT is also extremely useful, particularly in the evaluation of heart base masses.

Right atrial or auricular neoplasia
The most common right atrial neoplasm is haemangiosarcoma. This may be primary or metastatic and older German Shepherd Dogs and Golden Retrievers are predisposed. Canine haemangiosarcoma has usually metastasized by the time of diagnosis, and the lungs, spleen and liver in particular should be examined for evidence of metastatic disease. Animals usually present with a haemorrhagic non-clotting pericardial effusion (see Pericardial diseases).

Other right atrial or auricular masses have been reported and include benign lesions such as thrombi.

Radiography
There are usually radiographic features of pericardial effusion, with or without focal right atrial enlargement (Figure 7.148). The lungs should be evaluated for evidence of metastasis (most commonly these are poorly defined small coalescing nodules (see Figure 12.56**, p.xx**), less commonly they are well circumscribed nodules or an alveolar infiltrate secondary to haemorrhage).

Hepatosplenomegaly may be seen: in this case right-sided heart disease or metastatic neoplasia should be considered.

<<Figure 7.148 near here>>

Echocardiography
Echocardiographic findings include:

- Pericardial effusion may be seen
- Mass lesion (Figure 7.149):
  - Can arise from the RAu, the right atrial lateral wall or the junction between the RA and RV
  - Projects into the pericardium or less commonly into the atrium or auricular appendage
  - Haemangiosarcoma lesions usually appear hypoechoic and mottled due to cavitations.

<<Figure 7.149 near here>>

Computed tomography
This is useful to evaluate for the presence of a mass when ultrasonography is not conclusive or a pericardial effusion has been drained. It can also be useful for evaluating metastatic lung disease.

Heart base neoplasia

Heart base masses are usually chemodectomas, but ectopic thyroid and parathyroid tumours may be found less commonly in this location. Chemodectomas (also known as non-chromaffin paragangliomas, members of the APUDoma (amine precursor uptake and decarboxylation)) group of neoplasms) are tumours of the aortic bodies. These are congregations of neuroendocrine cells responsible for detecting blood pressure changes. Brachycephalic breeds are predisposed to chemodectomas and this is thought to have an association with chronic hypoxia.

Occasionally, chemodectomas are seen in other locations, such as the cranial mediastinum. In the heart, they wrap around the aortic root and main pulmonary artery. They are locally invasive and expansile. Metastasis is seen in dogs and cats but is not common (occurs in approximately 10–20% dogs) and is slow to occur. Metastatic sites include the lung, LA, pericardium, liver and kidney.

Radiography
Radiographic findings include:

- There is often minimal or no change in the appearance of the cardiac silhouette
- Mass border effacing or silhouetting with cranial aspect of the cardiac silhouette
- Focal elevation of the terminal (caudal) part of the trachea on the lateral view (Figure 7.150)
- Focal deviation of the terminal trachea on the DV/VD view in association with a soft tissue mass
- There may be radiographic features of pericardial effusion.

<<Figure 7.150 near here>>

Echocardiography
Echocardiographic findings include:

- Pericardial effusion may be seen
- Mass lesion (Figure 7.150c):
  - Extends around ascending aorta, though may be invasive and involve other areas of the heart
  - Usually homogenous and moderately echoic.

Computed tomography
A contrast-enhancing mass is seen around the ascending aorta (Figure 7.151). The mass is usually well defined and easily visualized using this imaging modality.

<<Figure 7.151 near here>>

Scintigraphy
Octreotide can be used to identify chemodectomas due to the presence of somatostatin receptors in this type of tumour. This was reported as useful in postoperative assessment of an incompletely resected left atrial chemodectoma.

Other cardiac neoplasia

Mesothelioma
Imaging techniques will identify a pericardial effusion (significant amounts of fluid within 120 days of pericardectomy increases suspicion of mesothelioma). Mesothelioma is likely to be underdiagnosed as it is a difficult diagnosis to establish antemortem. It is a diffuse neoplasm so there is no specific mass lesion identified on imaging (see also Pericardial effusions, above).

Myxoma
This is a rare benign neoplasm which has been reported in the dog and cat. It is usually gelatinous in nature but ossifying myxomas may also occur. Myxomas are often right-sided but they have also been reported on the left (Figure 7.152). A right atrial malignant *myxosarcoma* has also been reported.

<<Figure 7.152 near here>>

Lymphoma
This is the most common cardiac neoplasm in cats, and it is also seen in dogs. Unlike other cardiac neoplasms, examination of the pericardial effusion (if present) usually provides the diagnosis. It can be confused with myocardial disease (see Myocardial diseases and Figure 7.125).

Lymphoma appears as myocardial thickening, with or without hypoechoic mottled areas. This appearance is also seen in other myocardial infiltrative diseases (e.g. haemangiosarcoma).

Acquired vascular diseases

Dirofilariasis

Heartworm disease, caused by the nematode *Dirofilaria immitis,* affects both dogs and cats in endemic regions. The parasite has a worldwide

distribution, primarily in tropical and subtropical regions but with an increasing prevalence in temperate regions, particularly where there is a high urban (and pet) density and appropriate mosquito vectors. The life cycle of the parasite is similar in dogs and cats, but the parasite takes longer to reach patency in cats. Cats are also inherently more resistant to infection.

Adult heartworms live in the pulmonary arteries and in severe infections may also be present in the RV. Microfilariae produced by the adult female heartworm are released into the circulation. These are then ingested by feeding female mosquitos in which they undergo several moults to form an infective stage transmitted by the mosquito into another canine or feline host. About 3 months later the parasite reaches an immature adult stage and enters the vascular system. It then migrates to the heart and lungs to form the adult worm. The major effects of the disease relate more to changes induced in the walls of the pulmonary arteries rather than to actual obstruction. The pulmonary arterial response to heartworms in the cat is more severe than that in the dog, and in general the clinical manifestations of the disease tend to be more severe in this species.

The manifestations of heartworm disease depend on the number of worms, the chronicity of infection and on interactions between the parasite and host. Most dogs with heartworm infection are asymptomatic. Presenting signs in symptomatic dogs may include lethargy, coughing, poor condition, ascites and syncope. More serious consequences of heartworm disease, such as parasitic eosinophilic pneumonitis, may occur. Rare complications include eosinophilic granulomatosis, thromboembolic disease, pulmonary hypertension, glomerulonephritis, caval syndrome and disseminated intravascular coagulation (DIC).

Clinical signs in cats are often chronic and non-specific. These include anorexia, weight loss, vomiting, dyspnoea and coughing. More acute signs are usually due to aberrant worm migration (more common in cats than dogs) or worm embolization, and include shock, salivation, haemoptysis, neurological signs, vomiting, syncope and even death.

Radiography
Radiographic findings include:

- Dogs (Figure 7.153). Changes primarily reflect the pulmonary hypertension that results from physical obstruction to outflow by adult worms living in the RV and pulmonary outflow tract, as well as changes to the walls of the pulmonary arteries:
  - Enlargement of the right side of the heart ('reverse D' appearance on VD/DV view)
  - Enlargement of the main pulmonary artery segment
  - Enlargement and tortuosity of the (particularly caudal) lobar pulmonary arteries
  - Right heart failure may be evident in advanced disease
  - There may be diffuse bronchointerstitial pulmonary pattern due to eosinophilic infiltrates associated with an allergic response
  - Patchy dense interstitial to alveolar infiltrates may be seen, associated with pulmonary thromboembolism.
- Cats (Figure 7.154):
  - Most commonly *enlargement* of the caudal lobar pulmonary arteries (>1.6 times the ninth rib on the VD view)
  - Tortuosity of these vessels and enlargement of the right side of the heart is much less common than in dogs
  - Bronchointerstitial or even alveolar pulmonary infiltrates and pulmonary hyperinflation, mimicking feline bronchial disease have also been reported
  - Changes may also be seen in cats that ultimately resist heartworm infection and are eventually negative for the disease.

<<Figures 7.153, 7.154 near here>>

Contrast studies: Angiography is rarely necessary, but may demonstrate the cardiac chamber and pulmonary vascular changes typical of heartworm disease, and differentiate it from other cardiovascular diseases (Figure 7.155). Adult worms may be seen as linear filling defects within the contrast medium-filled peripheral pulmonary arteries.

<<Figure 7.155 near here>>

Changes that may be better demonstrated with contrast studies include main pulmonary artery segment dilation (not usually demonstrated on survey films of cats due to the medial location of the vessel) as well as pulmonary arterial enlargement (hilar region), tortuosity and loss of the normal tapering toward the periphery.

Ultrasonography
Ultrasonography can be a useful adjunctive test in dogs, and can provide definitive evidence of infection in cats when other tests are equivocal. Adult worms in dogs are usually present in the pulmonary arteries and, thus, may not be seen on ultrasound examination. However, they may be found in the main pulmonary artery, the proximal right and left caudal lobar arteries (Figure 7.156a), and occasionally the RA, RV or across the tricuspid valve (Figure 7.156bc). The latter has particularly been described in dogs with caval syndrome. On 2D echocardiography adult worms appear as strongly echogenic short parallel lines.

[au: should this heading be changed to echocardiography?]

<<Figures 7.156 (3 pics) near here>>

Supportive echocardiographic findings of the diagnosis in the absence of visible adult worms include (Figure 7.157):

- Right ventricular eccentric hypertrophy

- Right atrial and main pulmonary artery dilation
- Septal flattening and paradoxical motion
- Tricuspid regurgitation can be identified in dogs with advanced pulmonary hypertension or if adult worms are present across the valves.

<<Figure 7.157 near here>>

Angiostrongylosis

Angiostrongylus vasorum is a metastrongylid nematode that infects domestic dogs. *A. vasorum* has a worldwide distribution and within Europe is endemic in southern England, France, Ireland and Denmark. Foxes are presumed to act as a wildlife reservoir for the parasite. The adult worm is oviparous and lives in the right side of the heart or the pulmonary arteries from where the eggs are carried to the pulmonary capillaries. The eggs hatch and first-stage (L1) larvae migrate through the alveolar epithelium, are coughed up, swallowed and passed in the faeces. The life cycle is indirect and larvae undergo subsequent moults within a molluscan intermediate host, to become third-stage (L3) larvae. When a dog ingests an intermediate host, infective L3 larvae are liberated into the dog's small intestine. The L3 larvae undergo two further moults in the mesenteric lymph nodes and the L5 larvae migrate via the liver to the right side of the heart, completing the life cycle.

The most common clinical signs associated with angiostrongylosis are coughing and dyspnoea caused by the inflammatory response to the eggs and migrating larvae at the level of the alveolar membrane. The second common manifestation of angiostrongylosis is a coagulopathy. Subcutaneous, mucosal and internal haemorrhages may all be seen. Neurological signs associated with cerebral haemorrhage may also occur. Bleeding may occur with or without concurrent respiratory disease and the pathogenesis of the coagulopathy is undetermined. A minority of dogs suffer collapse episodes or sudden death, possibly due to aberrant larval migration into the myocardium. The majority of affected dogs are young and Cavalier King Charles Spaniels appear particularly predisposed to clinical disease.

Angiostrongylosis should be suspected in any young dog presenting with compatible clinical signs and a typical radiographic pattern (see below). The diagnosis is confirmed by demonstrating the presence of L1 larvae in the faeces or airway cytology samples. Other diagnostic findings are variable. Eosinophilic inflammation in airway cytology samples is less common than might be expected. The results of coagulation function testing are inconsistent and in some cases may be normal.

Radiography

Significant thoracic radiographic abnormalities are present in almost all clinically affected dogs (Figure 7.158):

- Most common findings are alveolar infiltrate and bronchial thickening (seen in approximately 80% and 70% of dogs, respectively). Typically, the alveolar infiltrate has a multifocal or peripheral distribution. Less commonly a mild, generalized interstitial pattern may be seen
- Note that the same thoracic radiographic changes are also seen in most dogs with *A. vasorum*-associated coagulopathy, even in the absence of clinical signs of respiratory disease
- Less common findings include a small volume pleural effusion and right ventricular enlargement
- Although *A. vasorum* is a pulmonary vascular parasite, significant radiographic changes affecting the pulmonary arteries are not usually seen
- Radiographic improvement may lag behind clinical resolution
- Residual alveolar infiltrate may still be present 1 month after successful treatment and an interstitial pattern may persist for up to 3 months.

<<Figure 7.158 near here>>

Ultrasonography

The ultrasonographic findings with *A.vasorum* have not been specifically described:

[au: should this heading be changed to echocardiography?]

- Evidence of right-sided cardiac enlargement and pulmonary hypertension may be seen
- *A.vasorum* larvae may be identified in ultrasound-guided fine needle aspirates of the lung (Figure 7.158c)
- In one case report, reversible pulmonary hypertension was reported in a dog with a right-to-left shunting ASD and angiostrongylosis. Pulmonary hypertension and shunt direction reversed after treatment.

Idiopathic pulmonary hypertension

Idiopathic pulmonary hypertension is a syndrome characterized by increased pulmonary arterial pressure due to hypertrophy of the tunica muscularis and tunica intima with no primary or discernible cause. The condition may be suspected in patients presenting with respiratory and right-sided heart disease in which no underlying cardiac or pulmonary disease can be demonstrated. In advanced disease, cor pulmonale and signs of right-sided heart failure (hepatomegaly, ascites, jugular pulse) may result due to prolonged pressure overload.

Radiography

Radiographic findings include:

- Features common to pulmonary hypertensive patterns and include enlargement of the right heart and main pulmonary artery segment, and enlarged lobar pulmonary arteries in the hilar region that taper rapidly toward the periphery
- The peripheral lung parenchyma appears hyperlucent due to poor perfusion
- Features such as variable diameter and saccular dilations of the peripheral vasculature

and pulmonary parenchymal infiltrates associated with hypertension due to other causes (chronic heartworm disease, pulmonary thromboembolism) are *absent* with idiopathic disease.

Ultrasonography
Ultrasonographic findings include:

[au: should this heading be changed to echocardiography?]

- Doppler ultrasound studies can be used to document increased velocity of right-sided regurgitant fractions. In the absence of pulmonic stenosis, pulmonic insufficiency velocity ≥2.2 m/s and/or tricuspid regurgitant velocity ≥2.8 m/s is reported to be indicative of pulmonary hypertension
- Additional echocardiographic findings include eccentric right ventricular hypertrophy, paradoxical septal motion and main pulmonary artery enlargement.

Pulmonary thromboembolism

Pulmonary thromboembolism (PTE) results in interruption of the blood supply to a portion of the lung due to obstruction of a pulmonary artery(ies) by embolic material (blood clots, parasites, tumour fragments, bacterial aggregates, bone marrow fat, etc.) delivered by the blood stream. Primary thrombosis (formation *in situ*) may result from local stasis, turbulent blood flow or vascular injury. Hypercoagulable states, such as those that exist with glomerulonephritis, Cushing's disease, pancreatitis or DIC, also predispose to thrombus formation in both the pulmonary and systemic circulation.

The consequences of PTE depend on the magnitude of the obstruction and the pre-existing condition; they may range from insignificant to severe ventilation–perfusion mismatching, development of pulmonary hypertension and right heart failure.

Radiography
Most commonly there are no survey radiographic abnormalities.

- Enlargement of the hilar portion of pulmonary arteries may be identified; there may be abrupt reduction in size or termination.
- Poor perfusion to part or all of a lung lobe may result in a hyperlucent appearance in the absence of hyperinflation.
- There may be atelectasis of a lung lobe, resulting in a mediastinal shift and displacement of the diaphragm.
- Focal pleural effusion may be seen around the affected lobe.
- Occasionally, there are peripheral triangular areas of consolidated lung extending to the pleural margin, particularly in the caudal lung lobes.

Contrast studies: Pulmonary angiography may delineate abrupt termination or filling defects in large pulmonary arterial segments, or areas of decreased or absent perfusion. Interpretation may be difficult in lungs with abnormal findings on plain films; abnormal perfusion to areas of lung that appear radiographically normal is much less equivocal.

Computed tomography
Helical CT angiography is a standard examination for suspected thromboembolism in humans. In dogs it is a useful and efficient technique to detect arterial thrombi in first- and second-order pulmonary arteries. Thrombi in subsegmental vessels may be too small to image reliably. An automatic power injector is required for a post-contrast scan.

Technique:

- Respiratory control is essential (see Chapter 3) as patients with PTE are often hyperpnoeic. If panting cannot be stopped for about 30 seconds, do not proceed with scanning. Image blur will render the examination non-diagnostic otherwise.
- Pre-contrast CT scan: thin-slice high-resolution axial lung CT scan with 5 mm slice gap.
- Post-contrast imaging:
  - A large volume of low concentration non-ionic contrast medium should be used to extend arterial phase (e.g. 4 ml/kg of 200 mg iodine/ml contrast medium)
  - Fast rate (e.g. 4 ml/second) injection via cephalic venous catheter
  - Scanning should be started approximately 5 seconds after the start of the injection
  - Time scan length according to duration of injection to ensure arterial filling
  - Helical scan settings with 2 or 3 mm slice width and interval, mediastinal window, soft tissue algorithm.

Computed tomography findings:

- Pre-contrast lung CT (Figure 7.159a):
  - Abnormally large (thrombosed) and/or small (hypovolaemic) pulmonary arteries, small pulmonary veins
  - Wedge-shaped peripheral consolidated lung tissue due to infarction.
- Post-contrast CT angiography (Figure 7.159b)
  - Arterial filling defects
  - Note that subsegmental embolism cannot be ruled out in case of normal CT scan.

<<Figure 7.159 near here>>

Magnetic resonance imaging
Contrast-enhanced magnetic resonance angiography (MRA) is a minimally invasive development in human medicine for evaluation of the pulmonary vascular system.

Scintigraphy
The classic appearance of PTE is photopenic, wedge-

shaped, pleural based defects with a lobar or subsegmental distribution (Figure 7.160). Sometimes there is lack of distribution in an entire lobe, indicative of the occlusion of a major lobar artery (Figure 7.161). Multiple small emboli, resulting in a mottled perfusion, pattern is common in dogs with heartworm disease. Normal or hyperlucent corresponding radiographs confirm a diagnosis of high probability of PTE. A corresponding normal ventilation scan confirms the diagnosis of PTE but this is very rarely obtained in small animals.

<<Figures 7.160, 7.161 near here>>

Budd–Chiari-like syndrome

In animals, this term describes post-sinusoidal hypertension and the development of a high-protein peritoneal effusion due to the obstruction of hepatic venous return to the heart. The obstruction may involve the:

- Junction of the hepatic veins with the CdVC
- CdVC itself between the entry of the hepatic veins and the heart
- RA.

Causes include thrombosis, caval syndrome associated with heartworm disease, compression or invasion of the cava by tumours or other masses, trauma-induced stricture, fibrosis, diaphragmatic hernia and congenital cardiac (e.g. CTD) or caval (e.g. membranous obstruction) anomalies.

Radiography

Other than ascites, radiographic features are not specific. Absence (congenital) of the CdVC, masses, hernias or other abnormalities in the region of the caudal mediastinum, diaphragm or liver may be seen.

Contrast studies: A caudal vena cavagram (lateral saphenous injection) may demonstrate invasion, compression or obstruction of the CdVC.

Ultrasonography

Abdominal ultrasonography may identify abnormalities of the liver or the abdominal portion of the CdVC. Doppler interrogation may identify abnormal or retrograde flow. Colour Doppler evaluation of the hepatic veins and CdVC provides the most information regarding the presence and direction of blood flow within the hepatic veins and CdVC.

Computed tomography

CT may demonstrate structural abnormalities or masses (Figure 7.162). CT angiography may be necessary to demonstrate vascular abnormalities.

<<Figure 7.162 near here>>

Cranial vena cava syndrome

CrVC syndrome results from obstruction of the CrVC with resulting impairment of the venous return from the cranial parts of the body to the heart. Reported causes include great vessel invasion/compression (tumour, e.g. thymoma), granuloma (e.g. blastomycosis) and thromboembolism. Predisposing factors include conditions that contribute to a hypercoagulable state (immune-mediated disease, sepsis, glomerulonephritis, cardiac disease, neoplasia, corticosteroid administration, central venous catheters with these pre-existing conditions).

The clinical signs include symmetrical, simultaneous swelling of the head, neck and forelimbs.

Radiography

Radiographs may be normal or may demonstrate the cause of the obstruction (e.g. cranial mediastinal mass).

Contrast studies: Angiography may demonstrate intraluminal filling defects, extraluminal compressive masses or other causes of CrVC obstruction. Non-selective angiography via the jugular vein is preferred but may be difficult in the face of swelling of the soft tissues of the neck; cephalic vein administration of contrast medium should also provide excellent opacification (Figure 7.163).

<<Figure 7.163 near here>>

Ultrasonography

In the presence of radiographic abnormalities of the cranial mediastinum, ultrasonography may provide additional information with regard to masses or other abnormalities. If thoracic radiographs are normal, ultrasonography is unlikely to provide additional information and angiography is recommended.

Computed tomography

CT angiography may provide an alternative means of evaluating the CrVC for filling defects or compressive lesions.

Coronary artery diseases

The right and left coronary arteries arise from the aortic bulb immediately distal to the aortic valve and supply the muscle of the heart (Figure 7.164). Coronary artery disease is extremely important in humans but rare in dogs and cats. A brief discussion of coronary disease in the dog and cat is given here.

<<Figure 7.164 near here>>

Congenital anomalies

There is variation in the anatomical pattern of the right and left coronary arteries. Five types of coronary arterial patterns have been identified in the dog (see Figure 7.55). Anomalous origin of the left coronary artery can lead to subvalvular pulmonic stenosis in English Bulldogs and Boxers. For surgical or interventional radiological correction of subvalvular pulmonic stenosis, exact knowledge of the anatomical pattern is required.

Angiography: Selective coronary angiography is

required to identify the coronary artery pattern (Figure 7.165).

<<Figure 7.165 near here>>

Coronary arterial and ischaemic heart disease
Ischaemic heart disease is rare in dogs and cats. Myocardial infarction can occur secondary to atherosclerosis or other forms of *arteriosclerosis* in both the dog and cat. *Arteriosclerosis* is arterial wall hardening, loss of elasticity and thickening, leading to luminal narrowing. Specific forms of arteriosclerosis include:

- Lipid deposition and thickening of the intimal cell layers within arteries (*atherosclerosis*)
- Calcification of the tunica media of muscular arteries
- Thickening of the walls of small arteries or arterioles due to cell proliferation or hyaline deposition (*arteriolosclerosis*).

Atherosclerosis has been strongly associated with diabetes mellitus and hypothyroidism (but not with hyperadrenocorticism). Mineralization of the walls of the coronary arteries may occur in chronic cases of arteriosclerosis. The gradual occlusion of the coronary arteries leads to myocardial ischaemia, myocardial infarction and fibrosis. The outcome may be sudden death, acute or chronic heart failure.

Ischaemic heart disease has been postulated as a cause of sudden death during anaesthesia or sedation. Myocardial infarction (generally left ventricular free wall) has also been reported secondary to HCM in the cat. Ischaemic heart disease can also rarely occur as a result of incarceration of the LV in a pericardial defect (congenital) or tear (traumatic) with strangulation of one or both coronary arteries (see Figure 7.144).

Imaging findings: Survey radiographs may reveal coronary artery mineralization in dogs (Figure 7.166). It can be difficult radiographically to distinguish coronary mineralization from aortic mineralization without the use of angiography. Coronary angiography is the diagnostic technique of choice, but is seldom used in animals.

<<Figure 7.166 near here>>

With echocardiography, ischaemic myocardium can show thinning, regional hypokinesis or akinesis and reduced or absent contractility.

References and further reading

Allema AR (2003) Abdominal, thoracic, and pericardial effusions. *Veterinary Clinics of North America: Small Animal Practice* **33**, 89–118

Basso C, Fox PR, Meurs KM *et al.* (2004) Arrhythmogenic right ventricular cardiomyopathy causing sudden cardiac death in boxer dogs: a new animal model of human disease. *Circulation* **109**, 1180–1185

Baty CJ (2004) Feline hypertrophic cardiomyopathy: an update. *Veterinary Clinics of North America: Small Animal Practice* **34**, 1227–1234

Beardow AW and Buchanan JW (1993) Chronic mitral valve disease in cavalier King Charles spaniels: 95 cases (1987–1991). *Journal of American Veterinary Medical Association* **7**, 1023–1029

Berg RJ, Wingfield WE and Hoopes PJ (1984) Idiopathic hemorrhagic pericardial effusion in eight dogs. *Journal of American Veterinary Medical Association* **185**, 988–992

Boag AK, Lamb CR, Chapman PS and Boswood A (2004) Radiographic findings in 16 dogs infected with *Angiostrongylus vasorum. Veterinary Record* **154**, 426–430

Bright JM, Herrtage ME and Schneider JF (1999). Pulsed Doppler assessment of left ventricular diastolic function in normal and cardiomyopathic cats. *Journal of the American Animal Hospital Association* **35,** 285–291

Bright JM and Mears E (1997) Chronic heart disease and its management. *Veterinary Clinics of North America: Small Animal Practice* **6**, 1305–1329 Erratum in *Veterinary Clinics of North America: Small Animal Practice* (1998) **1** x–xi

Bright JM, Toal RL and Blackford LA (1990) Right ventricular outflow obstruction caused by primary cardiac neoplasia. Clinical features in two dogs. *Journal of Veterinary Internal Medicine* **4**, 12–16

Buchanan JW (1990) Pulmonic stenosis caused by single coronary artery in dogs: four cases (1965–1984). *Journal of the American Veterinary Medical Association* **196,** 115–120

Buchanan JW (1999) Prevalence of cardiovascular disorders. In: *Textbook of Canine and Feline Cardiology: Prinicples and Clinical Practice, 2nd edn*, ed. PR Fox, D Sisson and NS Moïse, pp. 457–470. WB Saunders, Philadelphia

Buchanan JW and Bücheler J (1995) Vertebral scale system to measure canine heart size in radiographs. *Journal of American Veterinary Medical Association* **206(2)**, 194–199

Chapman PS, Boag AK, Guitian J and Boswood A (2004) *Angiostrongylus vasorum* infection in 23 dogs (1999–2002). *Journal of Small Animal Practice* **45**, 435–440

Chetboul V, Carlos C, Blot S *et al.* (2004) Tissue Doppler assessment of diastolic and systolic alterations of radial and longitudinal left ventricular motions in Golden Retrievers during the preclinical phase of cardiomyopathy associated with muscular dystrophy. *American Journal of Veterinary Research* **65,** 1335–1341

Connolly DJ and Boswood A (2003) Dynamic obstruction of the left ventricular outflow tract in four young dogs. *Journal of Small Animal Practice* **44**, 319–325

Dukes-McEwan J, Borgarelli M, Tidholm A, Vollmar AC and Häggström J (2003) Guidelines for the diagnosis of canine idiopathic dilated cardiomyopathy. The ESVC Taskforce for canine dilated cardiomyopathy. *Journal of Veterinary Cardiology* **5(2)**, 7–19

Dunning D, Monnet E, Orton EC and Salman MD (1998) Analysis of prognostic indicators for dogs with pericardial effusion: 46 cases (1985–1996). *Journal of American Veterinary Medical Association* **212**, 1276–1280

Ferasin L, Sturgess CP, Cannon MJ *et al.* (2003) Feline idiopathic cardiomyopathy: a retrospective study of 106 cats (1994–2001). *Journal of Feline Medicine and Surgery* **5,** 151–159

Foale RD, White RA, Harley R and Herrtage ME (2003) Left ventricular myxosarcoma in a dog. *Journal of Small Animal Practice* **44**, 503–507

Fox PR, Liu SK and Maron BJ (1995) Echocardiographic assessment of spontaneously occurring feline hypertrophic cardiomyopathy. An animal model of human disease. *Circulation* **92,** 2645–2651

Fox PR, Maron BJ, Basso C, Liu SK and Thiene G (2000) Spontaneously occurring arrhythmogenic right ventricular cardiomyopathy in the domestic cat: a new animal model similar to the human disease. *Circulation* **102,** 1863–1870

Fox PR, Moïse NS, Evans HE and Bishop SP (1999) Cardiovascular anatomy. In: *Textbook of Canine and Feline Cardiology: Principles and Clinical Practice, 2nd edn*, ed. PR Fox, D Sisson and NS Moïse, pp.13–24. WB Saunders, Philadelphia

Gavaghan BJ, Kittleson MD, Fisher KJ, Kass PH and Gavaghan MA (1999) Quantification of left ventricular diastolic wall motion by Doppler tissue imaging in healthy cats and cats with cardiomyopathy. *American Journal of Veterinary Research* **60,** 1478–1486

Gidlewski J and Petrie JP (2003) Pericardiocentesis and principles of echocardiographic imaging in the patient with cardiac neoplasia. *Clinical Techniques in Small Animal Practice* **18**, 131–134

Haggstrom J, Kvart C and Pedersen HD (2005) Acquired valvular heart disease. In: *Textbook of Veterinary Internal Medicine, 6th edn*, ed. SJ Ettinger and EC Feldman, pp. 1022–1039. Elsevier, St. Louis

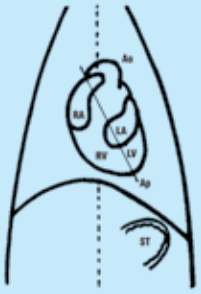
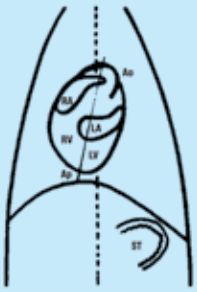
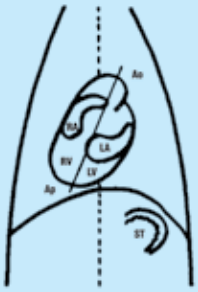
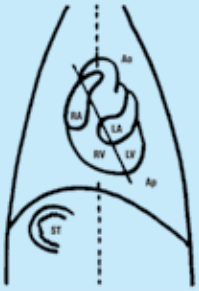
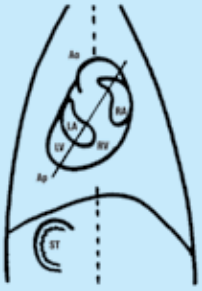
Hansson K, Haggstrom J, Kvart C and Lord P (2002) Left atrial root indices using two-dimensional and M-mode echocardiography in cavalier King Charles spaniels with and without left atrial enlargement. *Veterinary Radiology and Ultrasound* **6**, 568–575

Hansson K, Haggstrom J, Kvart C and Lord P (2005) Interobserver variability of vertebral heart size measurements in dogs with normal and enlarged hearts. *Veterinary Radiology and Ultrasound* **46**, 122–130

Hayward NJ, Baines SJ, Baines EA and Herrtage ME (2004) The radiographic appearance of the pulmonary vasculature in the cat. *Veterinary Radiology and Ultrasound* **45**, 501–504

Johnson L, Boon J and Orton EC (1999) Clinical characteristics of 53 dogs with Doppler-derived evidence of pulmonary hypertension:

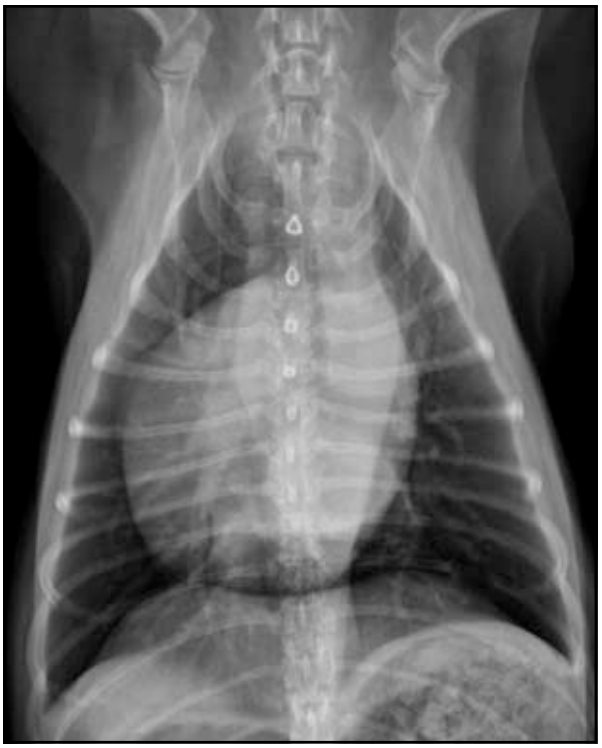
1992–1996. *Journal of Veterinary Internal Medicine* **13**, 440–447
Kittleson MD (1998) Radiography of the cardiovascular system. In: *Small Animal Cardiovascular Medicine.* ed. MD Kittleson and RD Kienle, pp. 47–71. Mosby, St. Louis
Koffas H, Dukes-McEwan J, Corcoran BM et al. (2006). Pulsed tissue Doppler imaging in normal cats and cats with hypertrophic cardiomyopathy. *Journal of Veterinary Internal Medicine* **20,** 65–77
Lamb CR (2000) Ability to visualise the cardiac silhouette in animals with pleural fluid: the pericardial fat stripe. *Veterinary Radiology and Ultrasound* **41**, 519–520
Lamb CR, Wikeley H, Boswood A and Pfeiffer DU (2001) Use of breed-specific ranges for the vertebral heart scale as an aid to the radiographic diagnosis of cardiac disease in dogs. *Veterinary Record* **148**, 707–711
Less RD, Bright JM and Orton EC (2000) Intrapericardial cyst causing cardiac tamponade in a cat. *Journal of American Animal Hospital Association* **36**, 115–119
Litster AL and Buchanan JW (2000) Vertebral scale system to measure heart size in radiographs of the cats. *Journal of American Veterinary Medical Association* **216**, 210–214
Lord PF and Suter P (1999) Radiology. In: *Textbook of Canine and Feline Cardiology: Principles and Clinical Practice, 2nd edn*, ed. PR Fox, D Sisson and NS Moïse, pp. 107–129. WB Saunders, Philadelphia
MacGregor JM, Faria ML, Moore AS *et al.* (2005) Cardiac lymphoma and pericardial effusion in dogs: 12 cases (1994–2004). *Journal of American Veterinary Medical Association* **227**, 1449–1453
Machida N, Hoshi K, Kobyashi M, Katsuda S and Yamane Y (2003) Cardiac myxoma of the tricuspid valve in a dog. *Journal of Comparative Pathology* **129**, 320–324
Machida N, Kobayashi M, Tanaka R, Katsuda S and Mitsumori K (2003) Primary malignant mixed mesenchymal tumour of the heart in a dog. *Journal of Comparative Pathology* **128**, 71–74
Mellanby RJ, Villiers E and Herrtage ME (2002) Canine pleural and mediastinal effusions: a retrospective study of 81 cases. *Journal of Small Animal Practice* **43**, 447–451
Merlo M, Bo S and Ratto A (2002) Primary right atrium haemangiosarcoma in a cat. *Journal of Feline Medicine and Surgery* **4**, 123–125
Olsen LH, Martinussen T and Pedersen HD (2003) Early echocardiographic predictors of myxomatous mitral valve disese in dachshunds. *Veterinary Record* **10**, 293–297
Owens JM (1977) Pericardial effusion in the cat. *Veterinary Clinics of North America* **7**, 373–383
Peterson PB, Miller MW, Hansen EK and Henry GA (2003) Septic pericarditis, aortic endarteritis and osteomyelitis in a dog. *Journal of American Animal Hospital Association* **39**, 528–532
Reed JR, Thomas WP and Suter PF (1983) Pneumopericardiography in the normal dog. *Veterinary Radiology* **24**, 112–119
Richardson P, McKenna W, Bristow M et al. (1996) Report of the 1995 World Health Organization/International Society and Federation of Cardiology Task Force on the Definition and Classification of cardiomyopathies. *Circulation* **93,** 841–842
Ruehl WW and Thrall DE (1981) The effect of dorsal *versus* ventral recumbency on the radiographic appearance of the canine thorax. *Veterinary Radiology* **22**, 10–16
Santilli RA and Bussadori C (1998) Doppler echocardiographic study of left ventricular diastole in non-anaesthetized healthy cats. *The Veterinary Journal* **156,** 203–215
Schober KE, Luis Fuentes V and Bonagura JD (2003) Comparison between invasive haemodynamic measurements and non-invasive assessment of left ventricular diastolic function by use of Doppler echocardiography in healthy anaesthetized cats. *American Journal of Veterinary Research* **64**, 93–103
Schwarz T, Willis R, Summerfield NJ and Doust R (2005) Aneurysmal dilatation of the right auricle in two dogs. *Journal of the American Veterinary Medical Association* **226**, 1512–1515
Simpson DJ, Hunt GB, Church DB and Beck JA (1999) Benign masses in the pericardium of two dogs. *Australian Veterinary Journal* **77**, 225–229
Sisson D, Kvart C and Darke P (1999) Acquired valvular heart disease in dogs and cats. In: *Textbook of Canine and Feline Cardiology: Principles and Clinical Practice, 2nd edn*, ed. PR Fox, D Sisson and NS Moïse, pp. 536–565. WB Saunders, Philadelphia
Sisson D and Thomas WP (1999) Pericardial disease and cardiac tumors. In: *Textbook of Canine and Feline Cardiology: Principles and Clinical Practice, 2nd edn*, ed. PR Fox, D Sisson and NS Moïse, pp. 679–701. WB Saunders, Philadelphia
Suter PF (1981) The radiographic diagnosis of canine and feline heart disease. *Continuing Education* **3**, 441–454
Thomas WP, Reed JR, Bauer TG and Breznock EM (1984) Constrictive pericardial disease in the dog. *Journal of American Veterinary Medical Association* **184**, 546–553
van den Broek AHM and Darke PGG (1987) Cardiac measurements on thoracic radiographs of cats. *Journal of Small Animal Practice* **28**, 125–135
Van der Gaag I and Van der Luer RJT (1977) Eight cases of pericardial defects in the dog. *Veterinary Pathology* **14**, 14–18
Venco L, Kramer L, Sola LB and Moccia A (2001) Primary cardiac rhabdomyosarcoma in a cat. *Journal of American Animal Hospital Association* **37**, 159–163
Ware WA and Hopper DL (1999) Cardiac tumours in dogs: 1982–1995. *Journal of Veterinary Internal Medicine* **13**, 95–103
Zoia A, Hughes D and Connolly DJ (2004) Pericardial effusion and cardiac tamponade in a cat with extranodal lymphoma. *Journal of Small Animal Practice* **45**, 467–471

	Levoposition	Dextroposition	Dextroversion	Levocardia	Dextrocardia
Cause	Normal	Displacement or congenital malposition	Congenital malposition	Congenital malposition	Congenital malposition
Location of viscera	Situs solitus	Situs solitus	Situs solitus	Partial situs inversus	Total situs inversus
Image					
Description	Normal situation in dogs and cats The heart is located slightly to the left and the apex points towards the left	Normal variant The apex is positioned more towards the right than normal. May be extrinsic (e.g. due to mediastinal shift) or intrinsic (actual embryological abnormality)	Congenital abnormality where the heart is positioned in the right side of the thorax Dextroversion also means that the heart is 'twisted' with the LV lying in the correct left-sided position but lying anterior to the RV	Heart is positioned on the left side of the thorax Mirror image arrangement of the abdominal viscera	Heart is positioned on the right side of the thorax Mirror image arrangement of the thoracic and abdominal viscera The most important of the positional abnormalities. Other congenital cardiac abnormalities may be present. May be seen as part of Kartagener's syndrome along with bronchiectasis and recurrent sinusitis

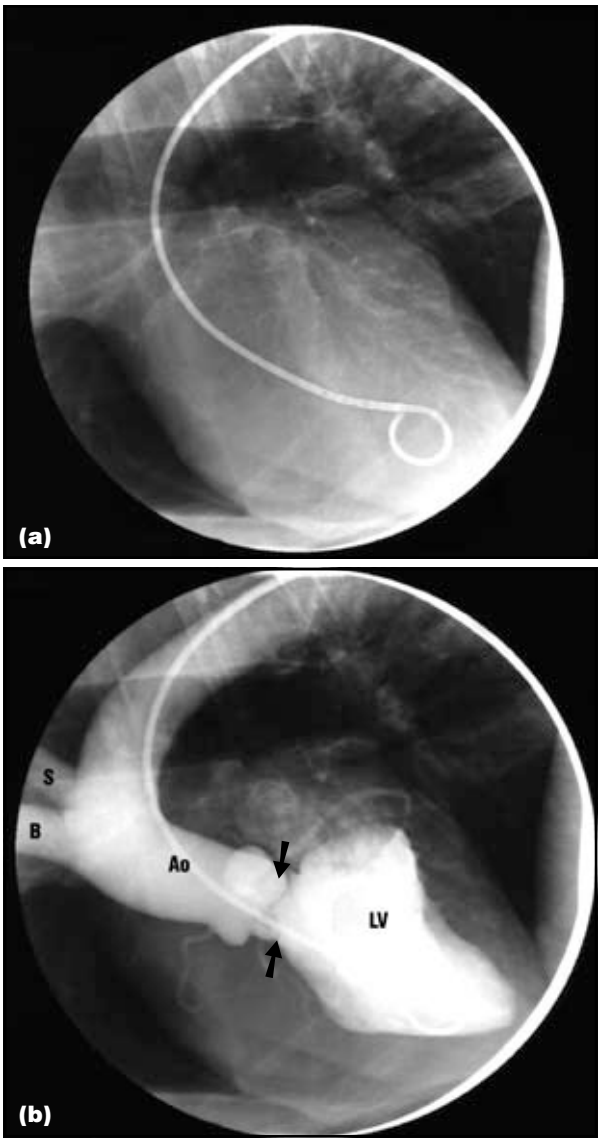
Definitions:

Dextrocardia – location of the heart in the right side of the thorax, the apex pointing towards the right
Dextroposition – displacement towards the right
Dextroversion – version (turning) to the right. In terms of the heart, dextroversion means the location of the heart in the right thorax, the LV lying in the correct position on the left but lying anterior to the RV
Levocardia – location of the heart in the left side of the thorax, the apex pointing towards the left
Levoposition – displacement towards the left
Situs inversus – the thoracic and abdominal viscera are reversed (i.e. a mirror image of the normal arrangement)
Situs solitus – the thoracic and abdominal viscera are in a normal location

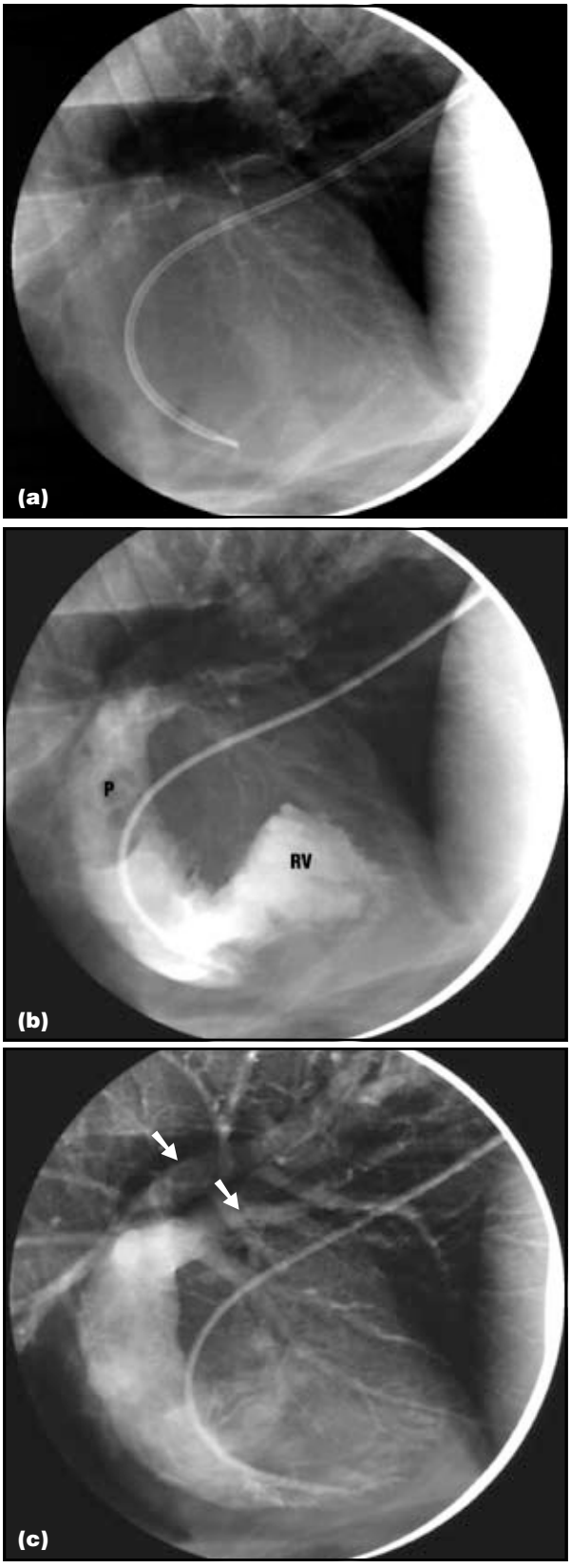
7.1 Cardiac position as seen on a DV radiograph. Variation in position may be a normal variant or a congenital abnormality. Some of the reported congenital abnormalities are included; many other variants are possible. Ao = Aorta; Ap = Apex; LA = Left atrium; LV = Left ventricle; RA = Right atrium; Right ventricle; ST = Stomach. (Line diagrams adapted from Suter (1984) with permission)



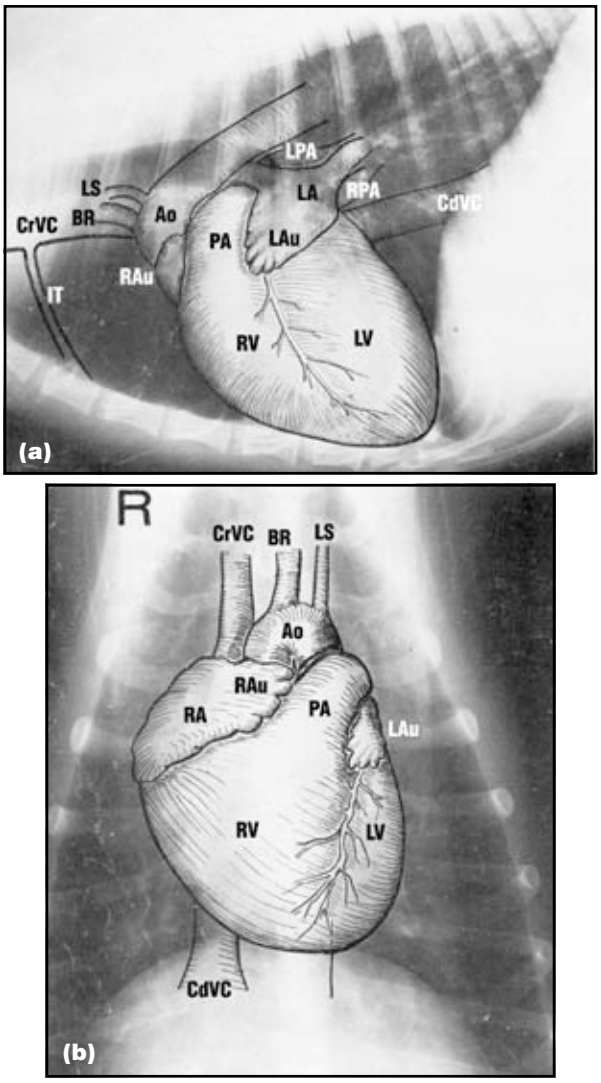
7.2 Dextroposition identified as an incidental finding in a dog. The cardiac silhouette and apex are located more in the right hemithorax than in the left and there is no evidence to suggest that this is associated with a mediastinal shift. Note the normal aorta on the left and the gastric fundus also seen on the left; there is no suggestion of a situs inversus in this dog.
[au: would it be helpful for the reader to add arrows to show the location of the aorta and gastric fundus?]



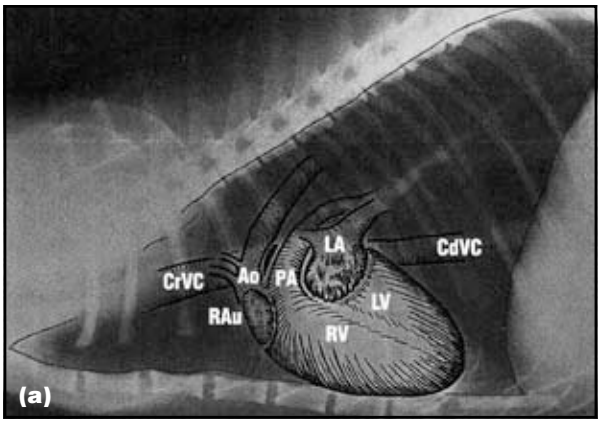
7.3 Normal left-sided angiocardiogram of a 5-year-old Golden Retriever. **(a)** The catheter has been placed into the LV via the femoral artery and aorta ready for the contrast medium injection. **(b)** Positive contrast medium outlines the LV, aortic valve (arrowed), ascending aorta (Ao), brachiocephalic trunk (B) and left subclavian artery (S). (Courtesy of Dr J. Buchanan)

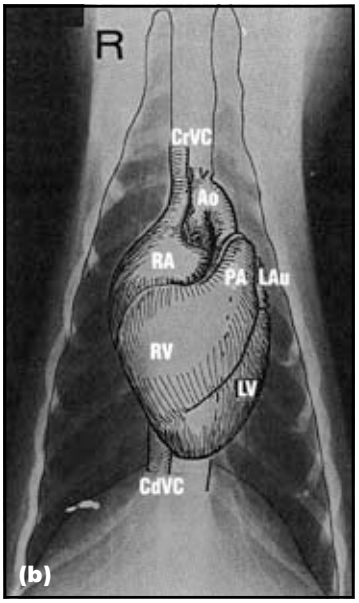


7.4 Normal right-sided angiocardiogram of the dog in Figure 7.3. **(a)** The catheter has been placed into the RV via the CdVC ready for the contrast medium injection. **(b)** Positive contrast medium is present within the RV and has started to enter the RVOT and main pulmonary artery (P). **(c)** The contrast medium has now reached the left and right pulmonary artery branches (arrowed) and the smaller pulmonary arterial branches in the lungs. (Courtesy of Dr J. Buchanan)

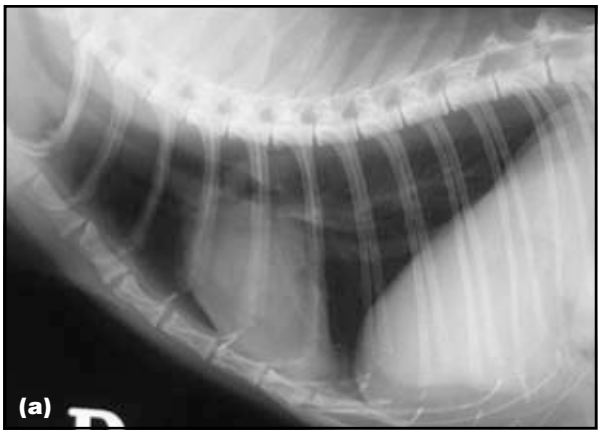


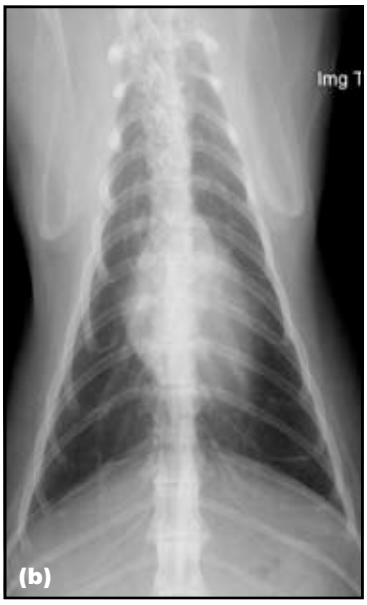
7.5 Location of the cardiac chambers on thoracic radiographs of a dog. **(a)** Lateral view. **(b)** Ventral view. Ao = Aortic arch; BR = Brachiocephalic trunk; CdVC = Caudal vena cava; CrVC = Cranial vena cava; IT = Internal thoracic arteries and veins; LA = Left atrium; LAu = Left auricular appendage; LPA = Left pulmonary artery; LS = Left subclavian artery; LV = Left ventricle; PA = Main pulmonary artery; RAu = Right auricular appendage; RPA = Right pulmonary artery; RV = Right ventricle. (Reproduced from Suter (1984) with permission)



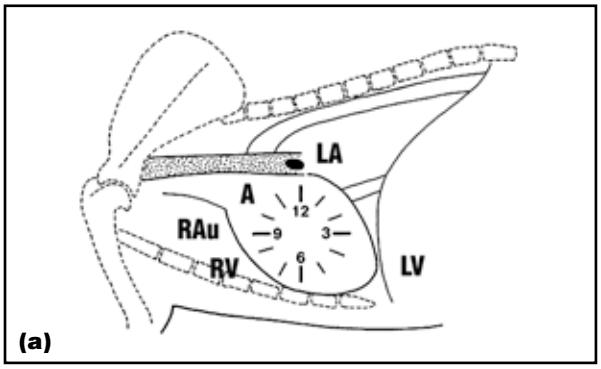


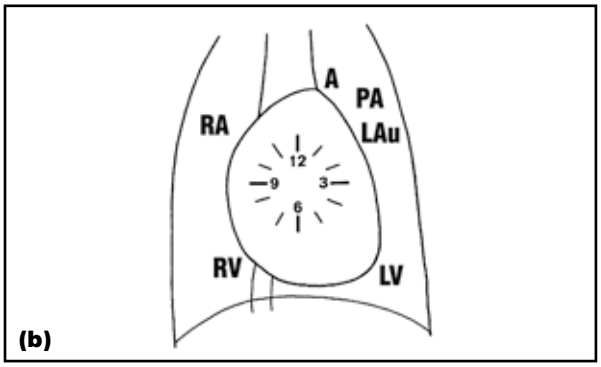
7.6 Location of the cardiac chambers on thoracic radiographs of a cat. **(a)** Lateral view. **(b)** Ventral view. Ao = Aortic arch; CdVC = Caudal vena cava; CrVC = Cranial vena cava; LA = Left atrium; LAu = Left auricular appendage; LV = Left ventricle; PA = Main pulmonary artery; RAu = Right auricular appendage; RV = Right ventricle. (Reproduced from Suter (1984) with permission)





7.8 The normal cardiac silhouette in a cat is fairly constant between different breeds. It is a similar neat ovoid or egg shape on both **(a)** right lateral and **(b)** DV views. Alteration in cardiac shape or size usually signifies cardiac disease in the cat.

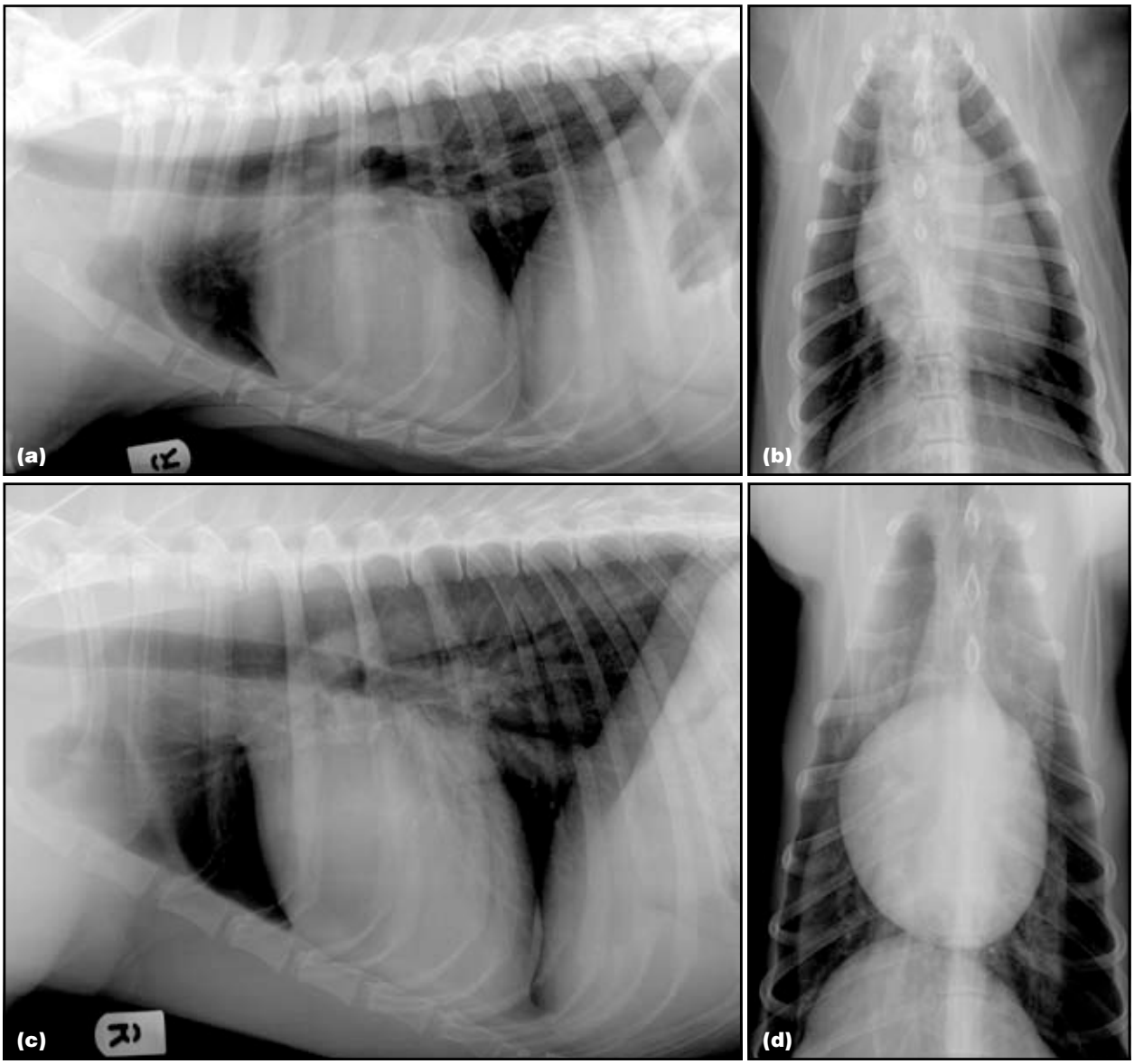




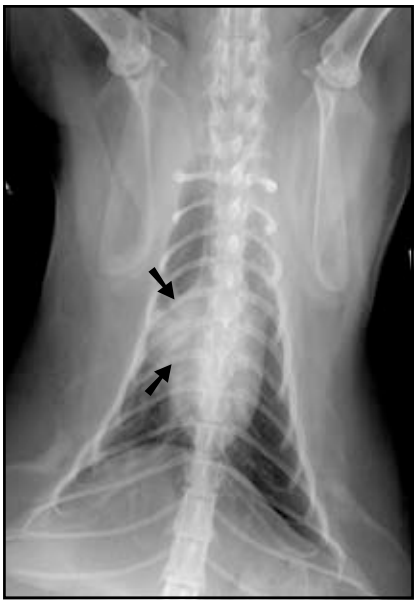
7.7 Clock face analogy identifying the location of the cardiac chambers. **(a)** Lateral view. **(b)** DV view. A = Aorta; LA = Left atrium; LAu = Left auricular appendage; LV = Left ventricle; PA = Main pulmonary artery; RA = Right atrium; RAu = Right auricular appendage; RV = Right ventricle. (Reproduced from Dennis *et al.* (2001) with permission from the publisher)

Thorax type	Lateral view	Dorsoventral view
Wide shallow (e.g. Dachshund, Shi-Tzu, Boston Terrier, Bulldog)	Shorter rounder cardiac silhouette at a large inclination to the spine Cardiac silhouette has a long contact area with the sternum (mimicking right-sided cardiomegaly)	Rounded right and left ventricular borders Apex is usually well to the left of the spine
Deep narrow (e.g. Greyhound, Afghan Hound, Whippet)	Long oval heart with a vertical position in the thorax (almost perpendicular to the spine)	Almost circular cardiac silhouette due to upright position of heart in thorax Apex is close to median plane
Intermediate (e.g. German Shepherd Dog, Labrador Retriever)	Heart appears ovoid or lop-sided egg-shaped	Heart appears similar to lateral view Apex is usually slightly to the left of the spine

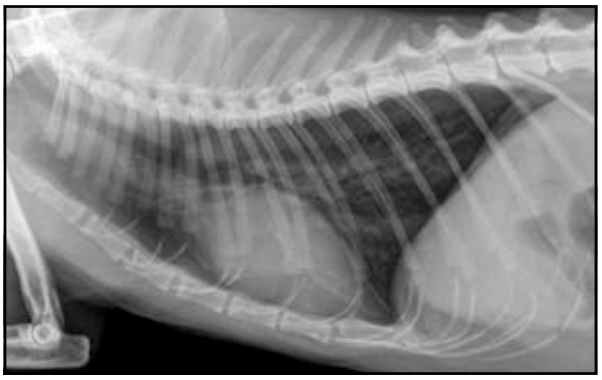
7.9 Variation in the appearance of the cardiac silhouette with thoracic shape.



7.10 The cardiac silhouette varies markedly between different dog breeds. **(a)** Lateral and **(b)** DV radiographs from a normal Golden Retriever. This breed often appears to have right-sided cardiomegaly on the lateral view due to the large amount of sternal contact (similar to wide shallow-chested dog breeds) and the rectangular shape of the cardiac silhouette. On the DV view the cardiac size appears normal and the apex is moderately displaced into the left hemithorax. **(c)** Lateral and **(d)** DV radiographs from a normal Greyhound. The cardiac silhouette is extremely upright on the lateral view due to the deep, narrow-chested nature of this breed. On the DV view the cardiac silhouette is very rounded in shape (compare with b) and the apex lies in the midline.



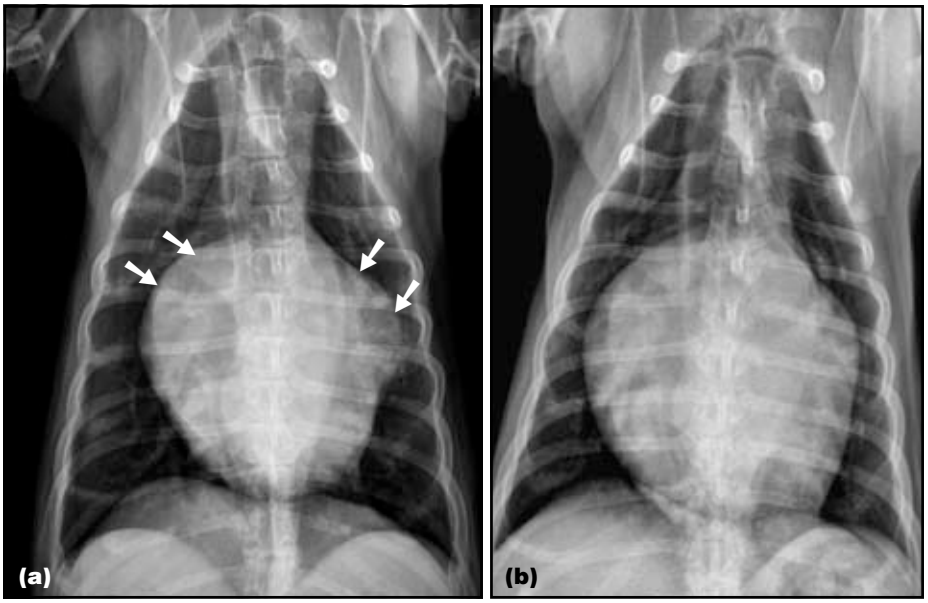
7.11 DV radiograph of an obese Domestic Shorthair cat. Note the triangular soft tissue opacity border effacing the right side of the cardiac silhouette (arrowed). This is due to a large amount of pericardial fat (even though it appears as a soft tissue opacity and should not be confused with pathology). A CT examination confirmed that no lung changes were present in this cat.



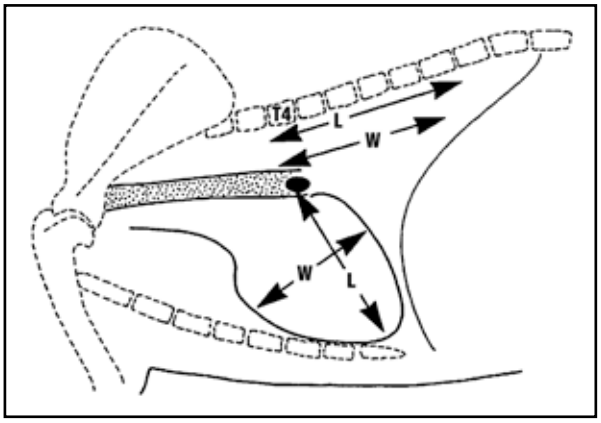
7.12 Left lateral view of a 12-year-old cat. In about 40% of older cats the heart has a more horizontal, cranially sloping position.

Right lateral
Cardiac silhouette shape and position of apex are more consistent May have a longer sternal contact area than on left lateral view
Left lateral
Cardiac silhouette is more rounded Apex may be displaced slightly dorsally from the sternum
Dorsoventral
Cardiac silhouette shape and position of apex are more consistent Cardiac silhouette is more oval The apex is more to the left on DV views
Ventrodorsal
Cardiac silhouette is more elongated Apex may be more in the midline May see bulge in position of main pulmonary artery

7.13 Variation in the appearance of the cardiac silhouette with body position.



7.14 VD views of the thorax of a Cavalier King Charles Spaniel with cardiomegaly obtained at different phases of the cardiac cycle. Note the difference in the appearance of the heart. **(a)** Systole: the ventricles are smaller and the atria are dilated (arrows show the RA and LAu). **(b)** Diastole: the entire heart is more rounded and the atrial and auricular appendage bulges are not as prominent. The changes in this dog are also exacerbated by differences in respiratory phase.



7.15 Technique to perform a VHS on a lateral radiograph. L = Length of the cardiac silhouette; T4 = Fourth thoracic vertebra; W = Width of the cardiac silhouette. (Reproduced from Dennis *et al.* (2001) with permission from the publisher)

(a) How to perform a vertebral heart score.

1. Take a well positioned lateral recumbent thoracic radiograph.
2. Measure the distance between the ventral aspect of the carina and cardiac apex.
3. Mark this on a piece of paper, with your starting point as a corner and mark along the edge to record the length.
4. Take a second measurement, perpendicular to the first at the widest point of the cardiac silhouette.
5. Mark this on the same piece of paper with the same starting point and on the same edge of the paper.
6. Hold the paper adjacent to the thoracic spine on the same radiograph with the starting point level with the cranial endplate of T4.
7. Count vertebral bodies along the spine (to the nearest 0.1 of a vertebra) until you reach your first mark. Note this figure.
8. Repeat step 7 counting to your second mark this time, being careful to start from T4 again. Note the second figure.
9. Add the two figures to get the VHS.

(b) Normal vertebral heart score. v = Vertebrae.

Dog breed-specific values

Boxer: 11.6 ± 0.8 v
Cavalier King Charles Spaniel: 10.6 ± 0.5 v
Dobermann: 10.0 ± 0.6 v
German Shepherd Dog: 9.7 ± 0.7 v
Labrador Retriever: 10.8 ± 0.6 v
Whippet: 11.0 ± 0.5 v
Yorkshire Terrier: 9.7 ± 0.5 v

Puppies

Have VHS values within the same 8.5–10.5 v range

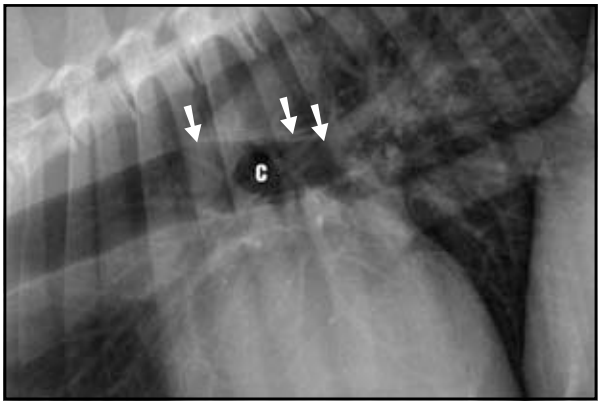
Cats

7.5 ± 0.3 v on a lateral view
The cardiac width on a VD view is 3.4 ± 0.25 v if measured perpendicular to the long axis

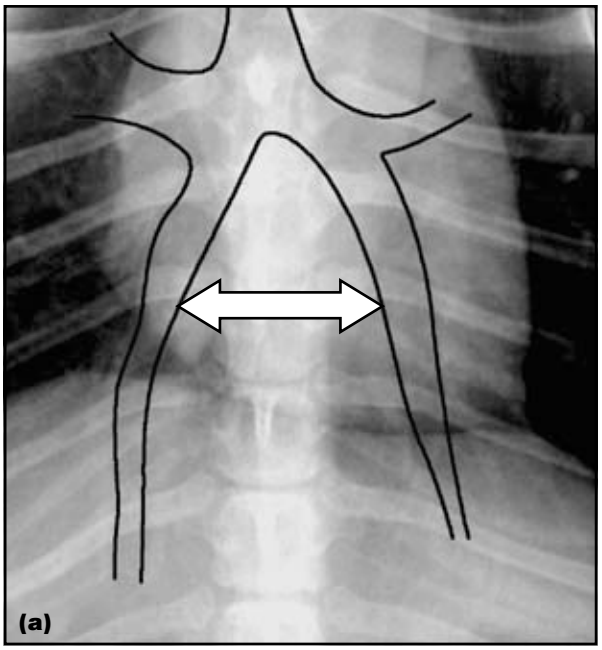
7.16 **(a)** How to perform a VHS. **(b)** Normal VHS. v = Vertebrae.

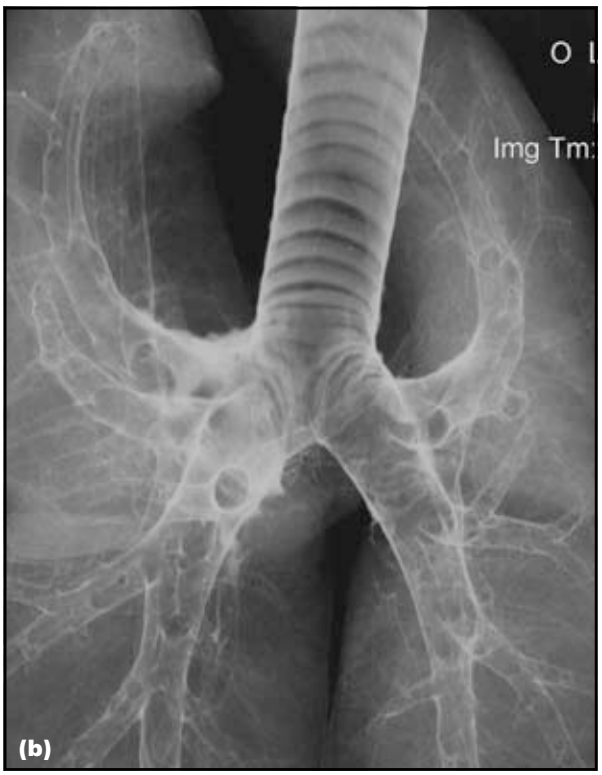
FIGURE 7.17 TO FOLLOW

7.17 Basic principles of tracheal and bronchial displacement on the lateral radiograph. The trachea, carina and caudal mainstem bronchi can be a very useful means to assess cardiac chamber enlargement. In this series of figures the changes in tracheal and bronchial position on a lateral radiograph are illustrated for various conditions. Note that there is also variation in tracheal position between different breeds of dogs. **(a)** Normal. The trachea and caudal mainstem bronchi have a gentle ventral divergence from the thoracic spine. Note the normal ventral bend (arrowed) in the trachea just cranial to the carina. **(b)** Left ventricular enlargement in isolation. The LV elevates the entire trachea when it is enlarged and there is loss of the normal ventral bend cranial to the carina. **(c)** Left atrial enlargement in isolation. The LA creates a triangular or wedge-shaped soft tissue opacity at the caudal border of the cardiac silhouette and pushes the left caudal mainstem bronchus (shown in white) dorsally. **(d)** The effect of a cranial mediastinal mass is shown for comparison. A cranial mediastinal mass (arrowheads) can elevate the trachea cranial to the carina anywhere along its length. Depending on the size and location of the mass, this may be focal elevation or more commonly elevation of the entire pre-cardiac trachea. Often the normal ventral bend in the trachea remains. The cardiac size will be normal. **(e)** Right atrial enlargement or right atrial mass in isolation. It is very rare to see severe focal right atrial enlargement. An enlarged RA can focally elevate the trachea cranial to the carina. (Note that a heart base mass can also elevate the trachea in this position but usually has a different appearance on the DV/VD view; see Cardiac neoplasia.)

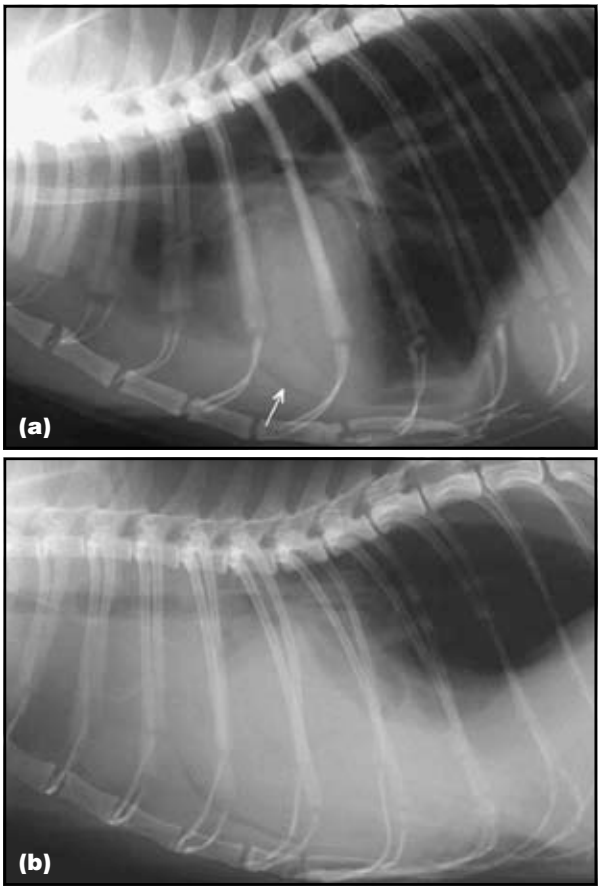


7.18 Close-up of a lateral thoracic radiograph of a dog demonstrating the normal appearance of the caudal trachea. Note the normal ventral bend (black arrow) cranial to the carina (C), and the two caudal mainstem bronchi almost level with one another (white arrows point to their dorsal margins).

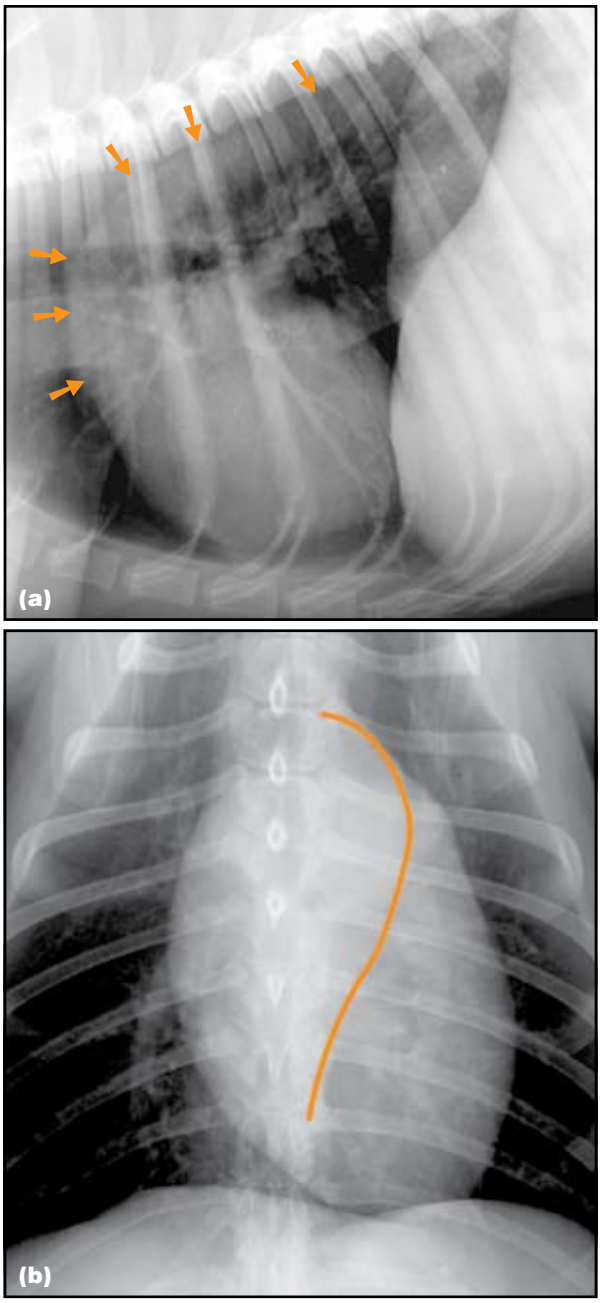




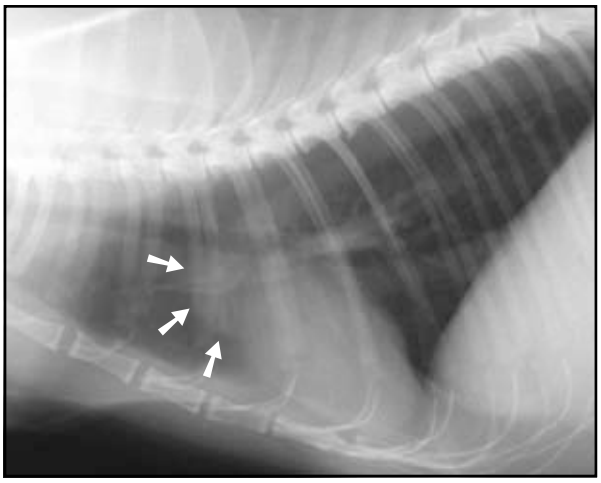
7.19 **(a)** Close-up of a DV radiograph of a normal dog at the level of the heart. The bronchial tree is shown with black lines. The angle between the left and right caudal mainstem bronchi (shown by the arrow) should be about 60 degrees and form an upside-down V shape. This does vary somewhat with breed, but an increased angle or splayed appearance ('cowboy legs' sign) suggests left atrial or tracheobronchial lymph node enlargement. **(b)** Postmortem bronchography image showing the normal angle between the caudal mainstem bronchi.



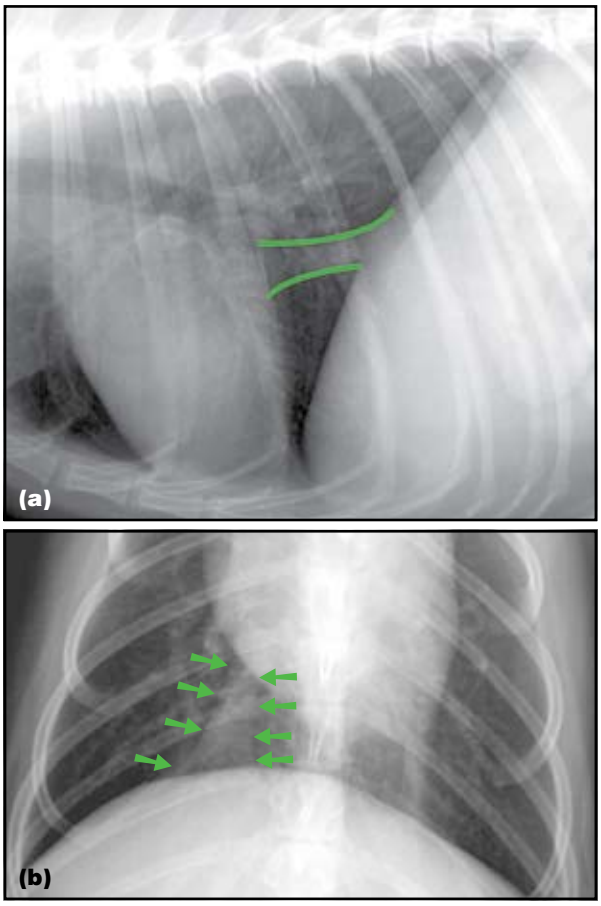
7.20 **(a)** Lateral thoracic radiograph of a cat with a small volume of pleural fluid associated with feline leukaemia virus infection. A narrow lucent line is visible in a position compatible with the cranial aspect of the heart (arrowed). Together with the position of the trachea and the caudal aspect of the cardiac silhouette, this line has the effect of completing a normal appearing cardiac silhouette. **(b)** Lateral thoracic radiograph of a different cat with a larger volume of pleural fluid. In this instance, the position and size of the pericardial fat stripe, relative to the position of the trachea, suggests enlargement of the cardiac silhouette. Ultrasonography subsequently confirmed marked HCM (and ruled out a mediastinal mass). (Reproduced from Lamb (2000) with permission from *Veterinary Radiology and Ultrasound*)



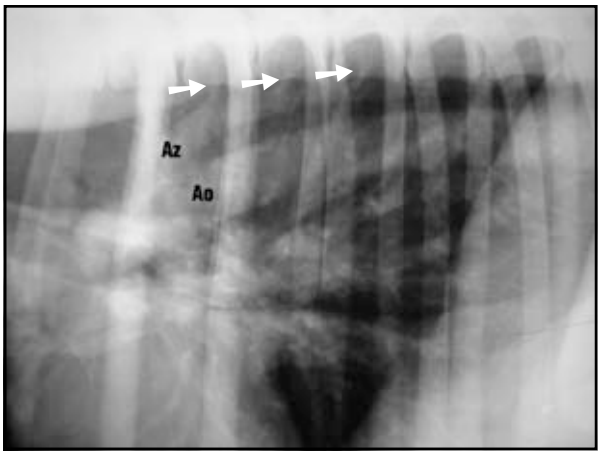
7.21 **(a)** Lateral and **(b)** DV radiographs of a dog showing the normal location and appearance of the aorta (orange arrows in (a); orange line in (b)).



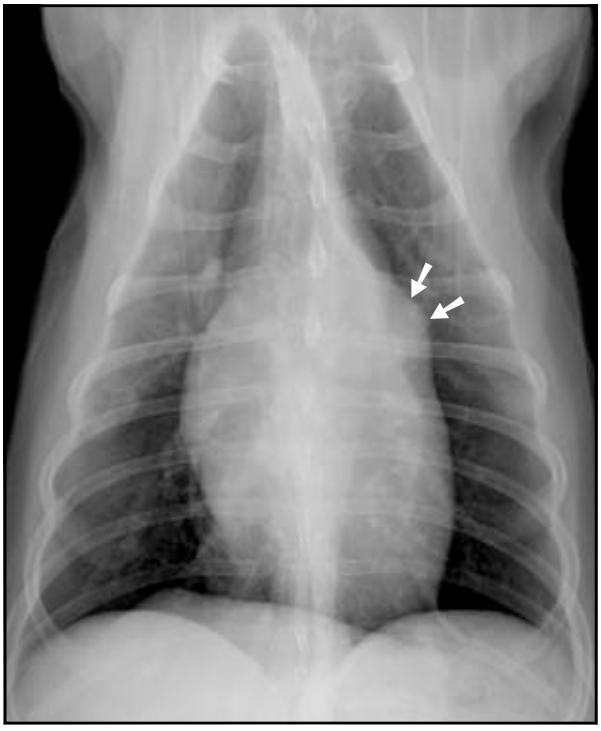
7.22 Lateral thoracic radiograph of an aged cat. Note the focal bulge in the aorta (arrowed).



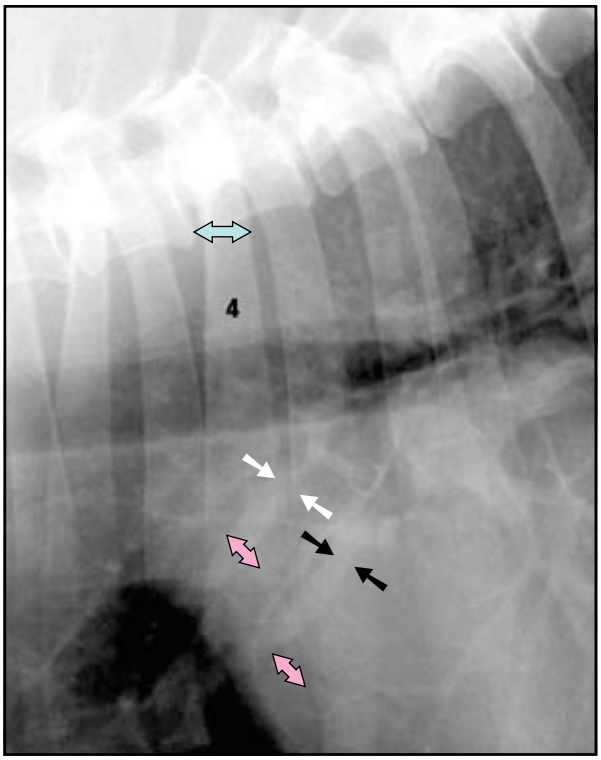
7.23 Close-ups of **(a)** lateral and **(b)** DV radiographs of a dog showing the normal size and location of the CdVC (green lines in (a); arrows in (b)).



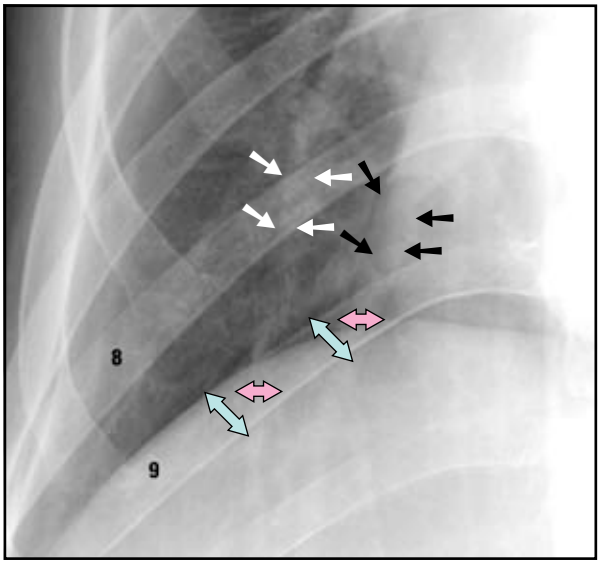
7.24 Close-up of a lateral thoracic radiograph of a dog with a severe pneumomediastinum after trauma. The azygos vein (Az) is visible as a narrow soft tissue opacity tube ventral to the spine and dorsal to the aorta (Ao), receiving tributary vessels from each intervertebral space (small white arrows).



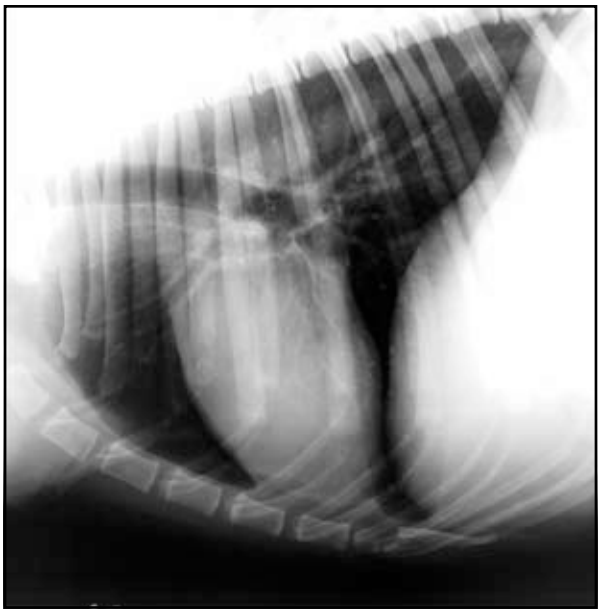
7.25 VD thoracic radiograph of a normal dog. The main pulmonary artery appears as a small focal bulge at the 01.00–02.00 o'clock location. This can be a normal finding on a VD view, or when an exposure is made during systole, and should not be interpreted as disease.



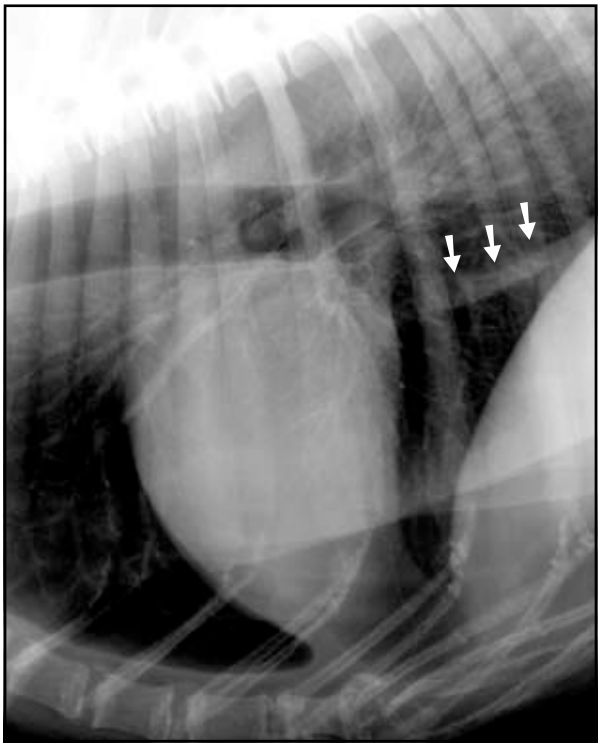
7.26 Close-up of the cranial lobar pulmonary vessels, the artery is shown with small white arrows and the vein with small black arrows. These vessels should be no greater than the narrowest part of the third or fourth rib (turquoise arrow) where they cross the rib (pink arrow).



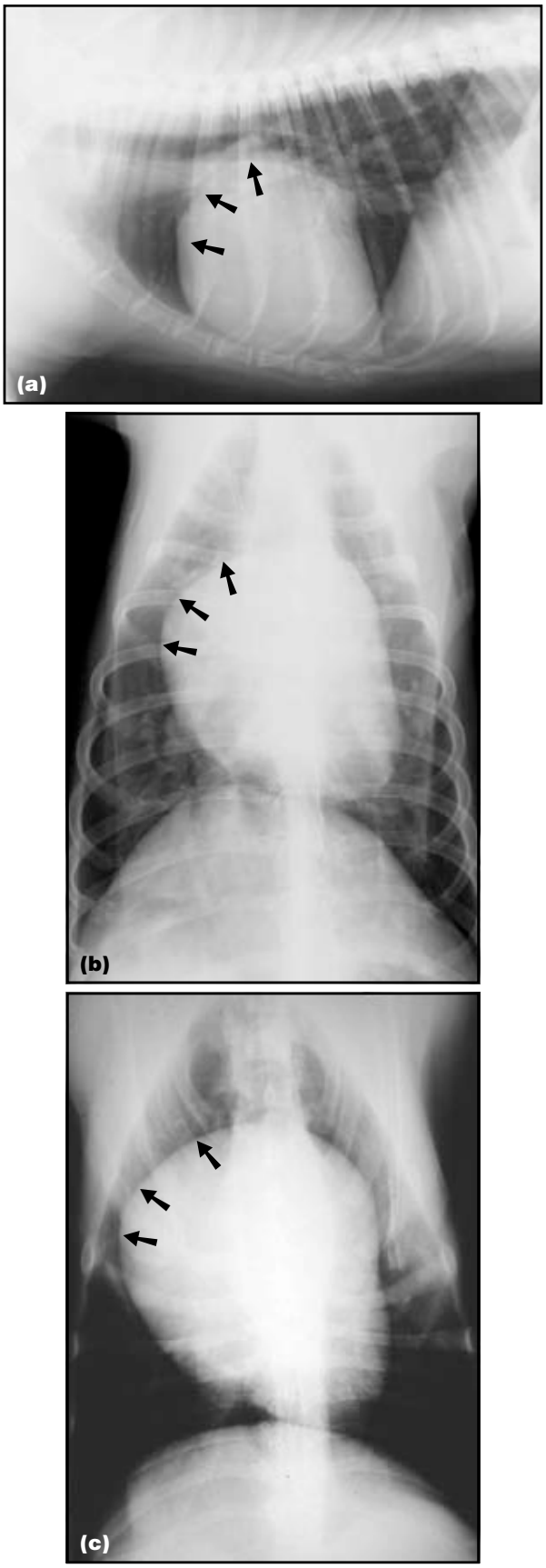
7.27 Close-up of the right caudal lobar pulmonary vessels as they cross the eighth and ninth ribs. The artery is shown with small white arrows and the vein with small black arrows. The vessels are measured where they cross the ninth rib (rib measurements shown by turquoise arrows and the vessel measurements are shown by pink arrows).



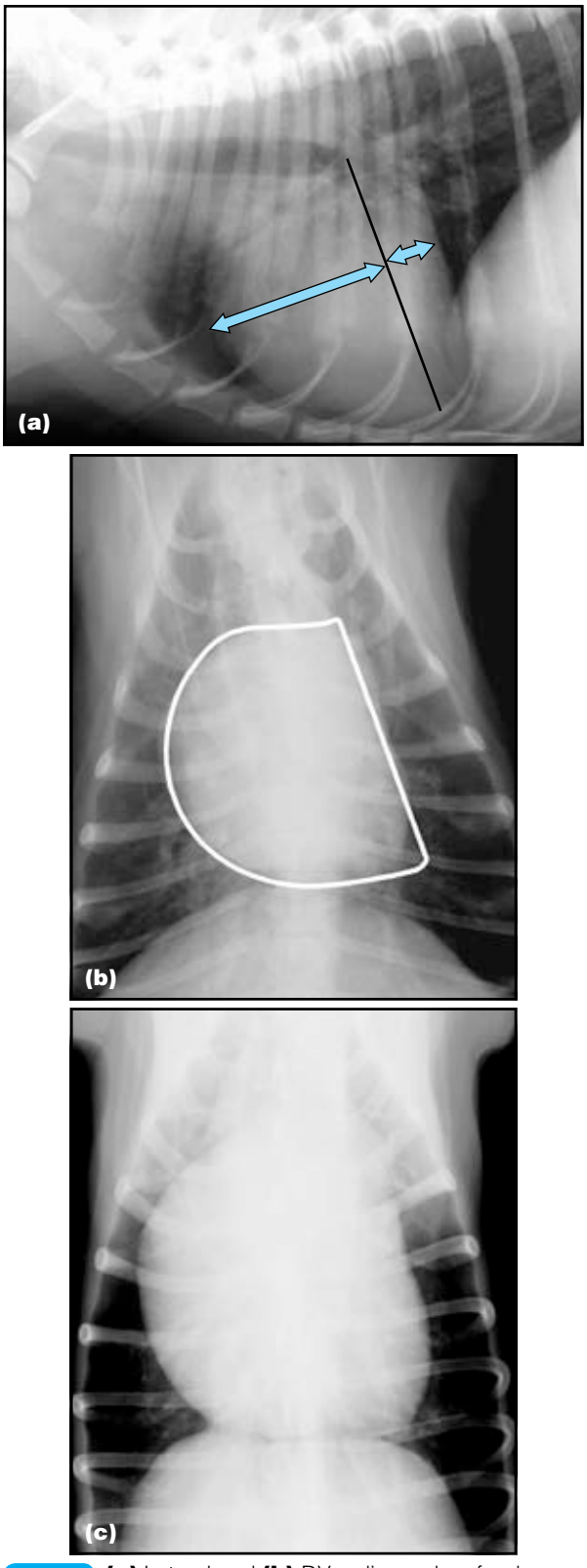
7.28 Microcardia in a dog with Addison's disease (hypoadrenocorticism). Note the narrow pointed appearance of the cardiac silhouette.



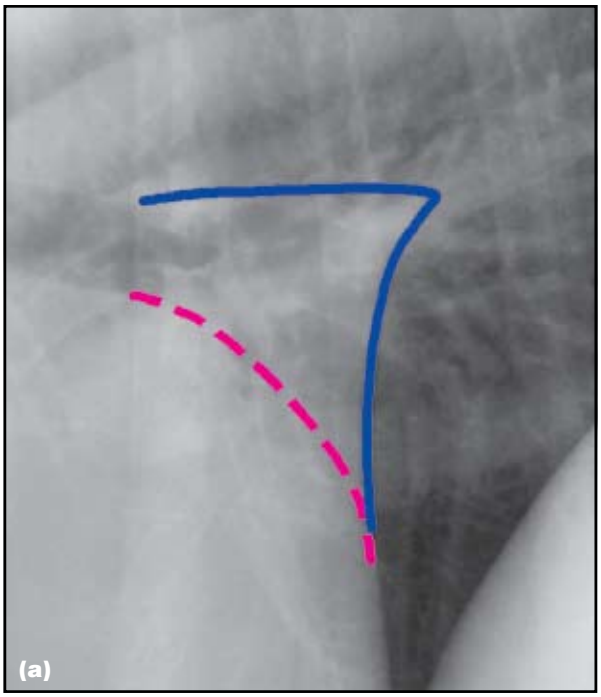
7.29 Lateral radiograph of an extremely dehydrated dog. The CdVC is narrow (arrowed), the lung fields are hyperlucent, and the cardiac silhouette is small.

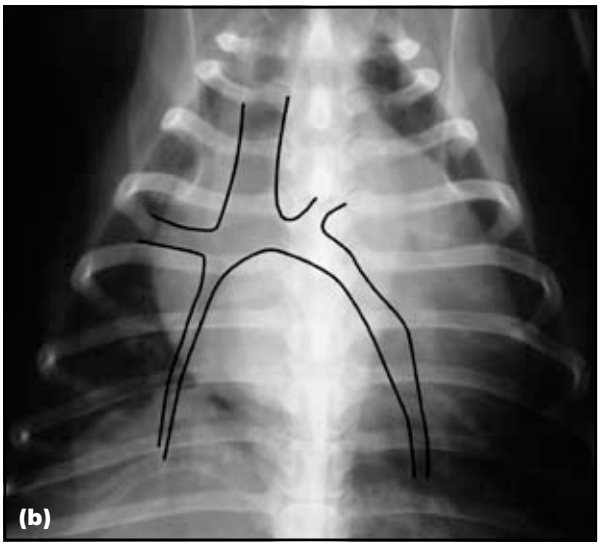


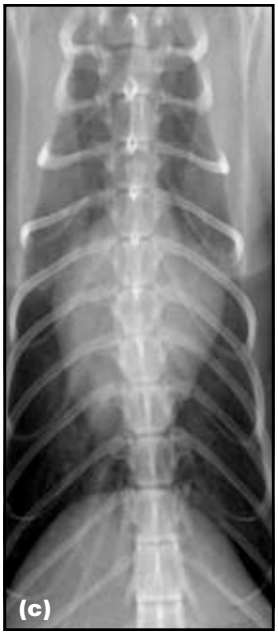
7.30 **(a)** Lateral and **(b)** DV radiographs of a 1-year-old Kelpie cross dog and **(c)** DV view of an 8-month-old Golden Retriever, both with tricuspid dysplasia. The RA is enlarged on all views (arrowed). Some right ventricular enlargement is also contributing to the appearance of the cardiac silhouette.



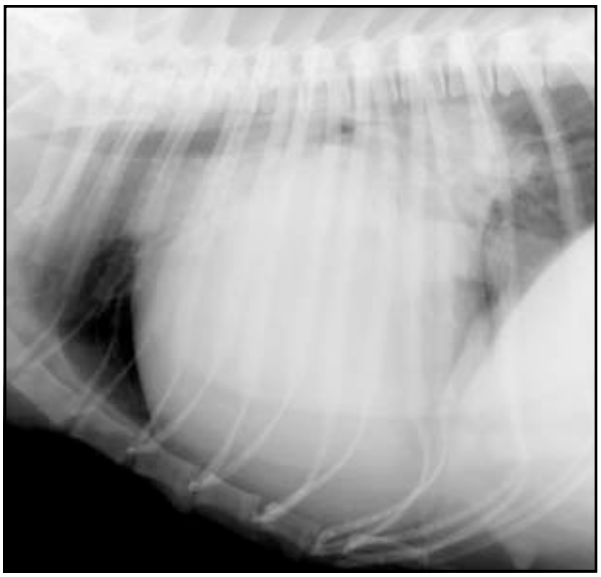
7.31 **(a)** Lateral and **(b)** DV radiographs of a dog with a moderately enlarged RV. In (a) when a line is dropped from the carina to the apex, approximately three quarters of the cardiac silhouette lies in front of the line and one quarter lies caudal to it. In (b) the cardiac silhouette has a reverse D-shaped appearance. Note that an enlarged RA is also contributing to the shape changes. **(c)** DV thoracic radiograph from a different dog with severe right-sided enlargement due to heartworm disease. There is an obvious reverse D-shaped silhouette seen in this patient. The main pulmonary artery is also enlarged.



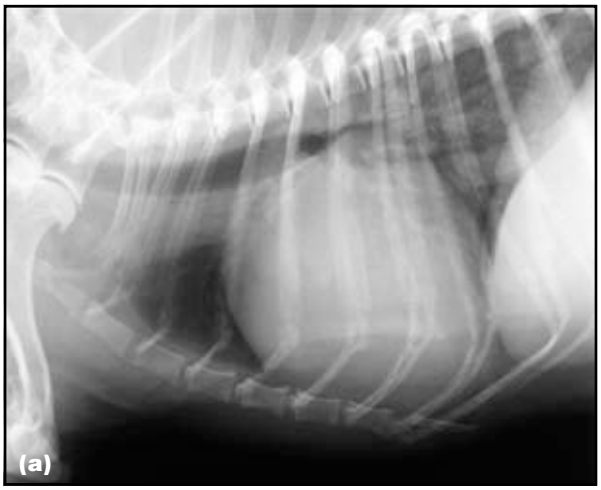


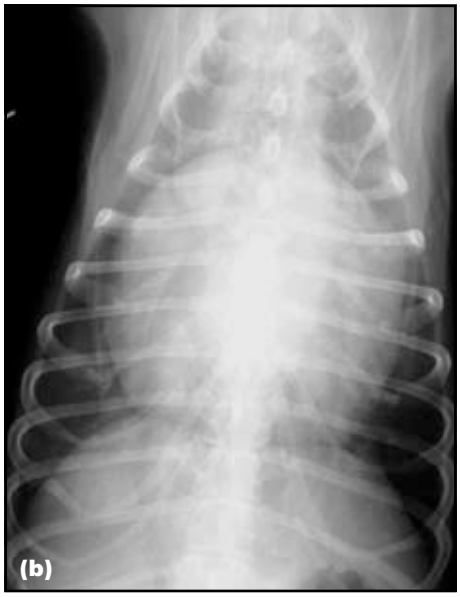


7.32 **(a)** Close-up of a lateral thoracic radiograph showing moderate left atrial enlargement in a dog. The left atrial outline is forming a wedge or tent shape (shown by the blue line). The shape of the normal LA is shown for comparison (pink dashed line). **(b)** DV radiograph of a dog with marked left atrial enlargement. The increased size of the LA has separated the caudal mainstem bronchi (black lines show the outline of the bronchial tree). **(c)** Left atrial enlargement in the cat produces a bulge at the cranial left aspect of the cardiac silhouette on the DV/VD view. When the RA is also enlarged a valentine heart shape results. This is evident in this DV view of a cat with HCM.

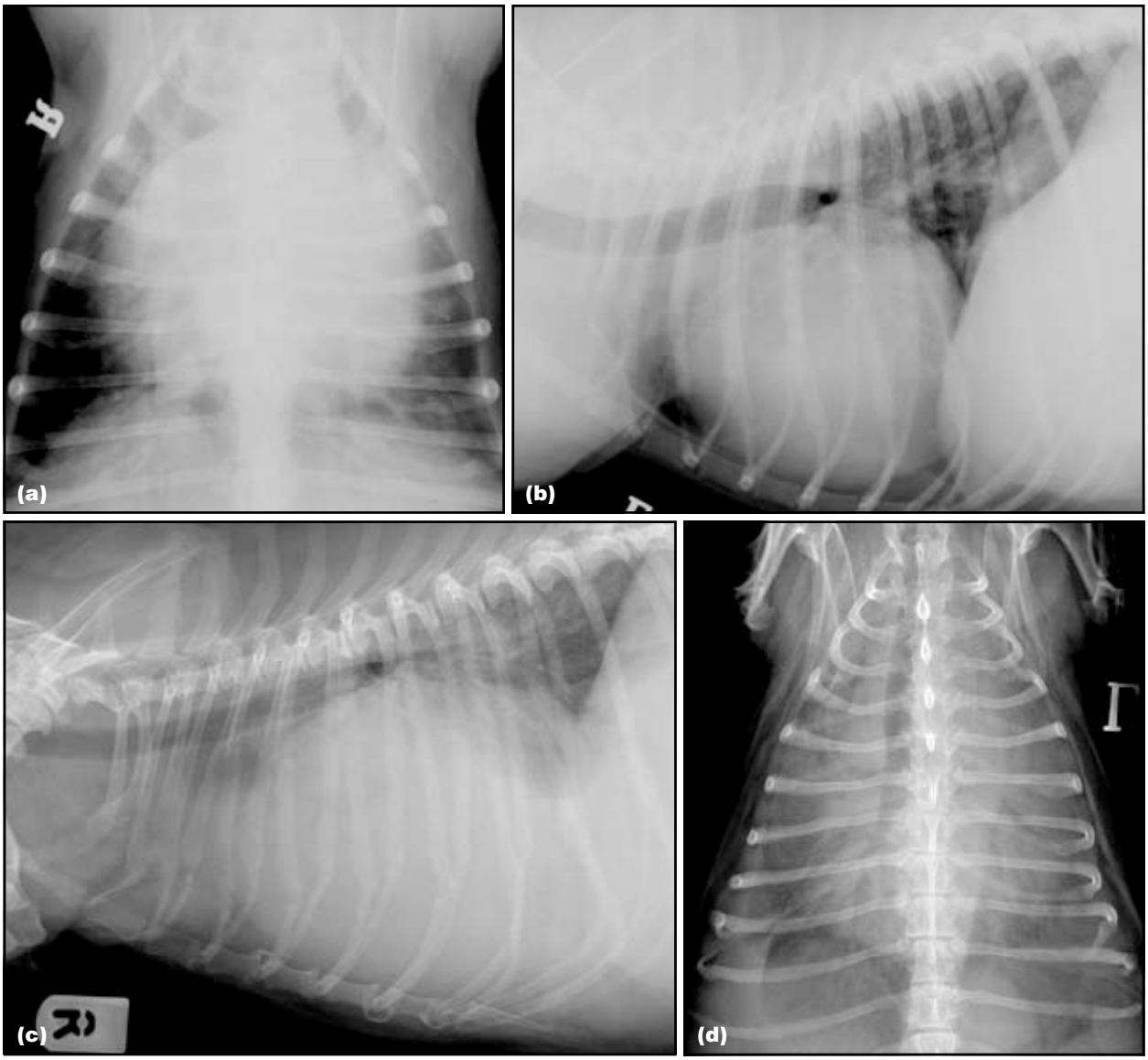


7.33 Lateral radiograph of a dog with severe left ventricular and left atrial enlargement due to DCM. Note the tracheal position and the left atrial tent (or wedge). (Courtesy of Dr B. Hopper)

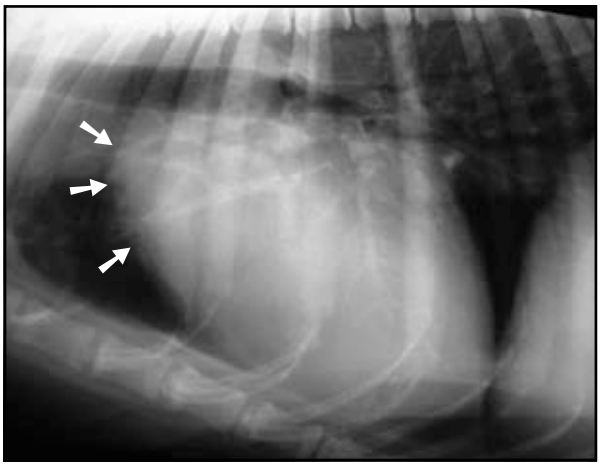




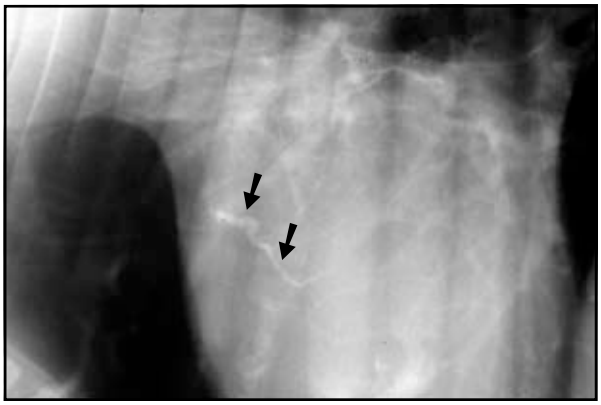
7.34 **(a)** Lateral and **(b)** DV radiographs of a middle-aged crossbreed dog with generalized cardiomegaly, secondary to endocardiosis.



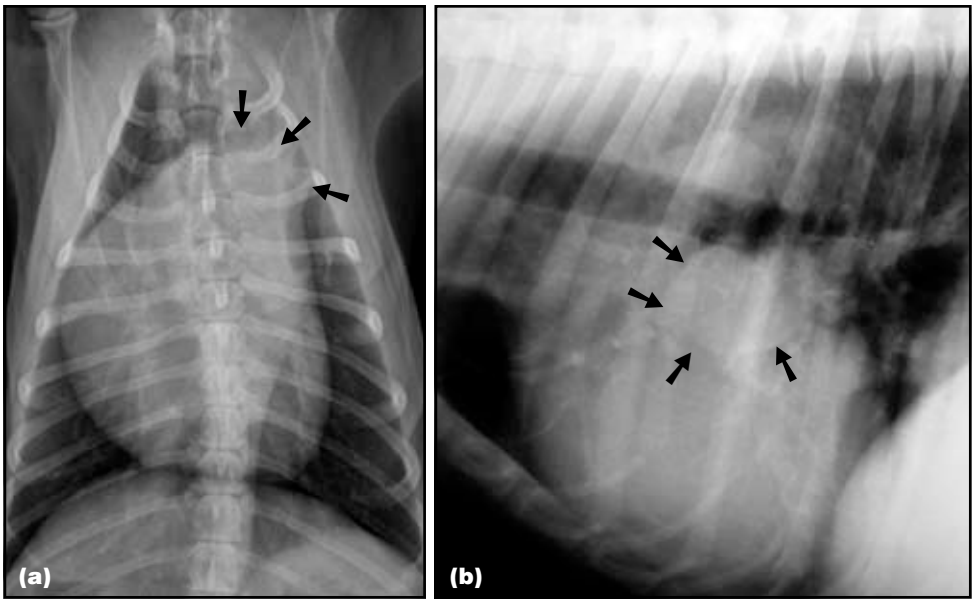
7.35 **(a)** Lateral and **(b)** DV radiographs of a dog with a pericardial effusion. The cardiac silhouette is globoid on both views and careful observation reveals sharp margins. This makes pericardial effusion more likely than generalized cardiomegaly. **(c)** Lateral and **(d)** DV radiographs of a dog with a pericardial effusion and right-sided heart failure. The cardiac silhouette is massively enlarged and globoid on both views. A pleural effusion is present due to the cardiac tamponade and right-sided heart failure.



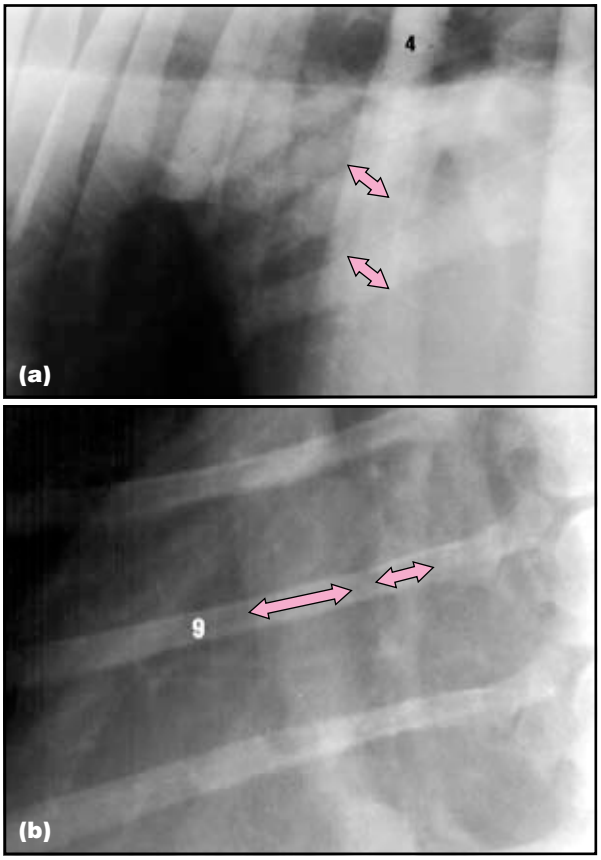
7.36 Lateral radiograph of a dog with subaortic stenosis. The entire aortic arch is enlarged (arrowed). This was confirmed on the DV view.



7.37 Incidental aortic calcification identified on the lateral thoracic radiograph of a 10-year-old Weimaranar. The calcification is seen as a wavy opaque line over the cranial border of the cardiac silhouette (arrowed).



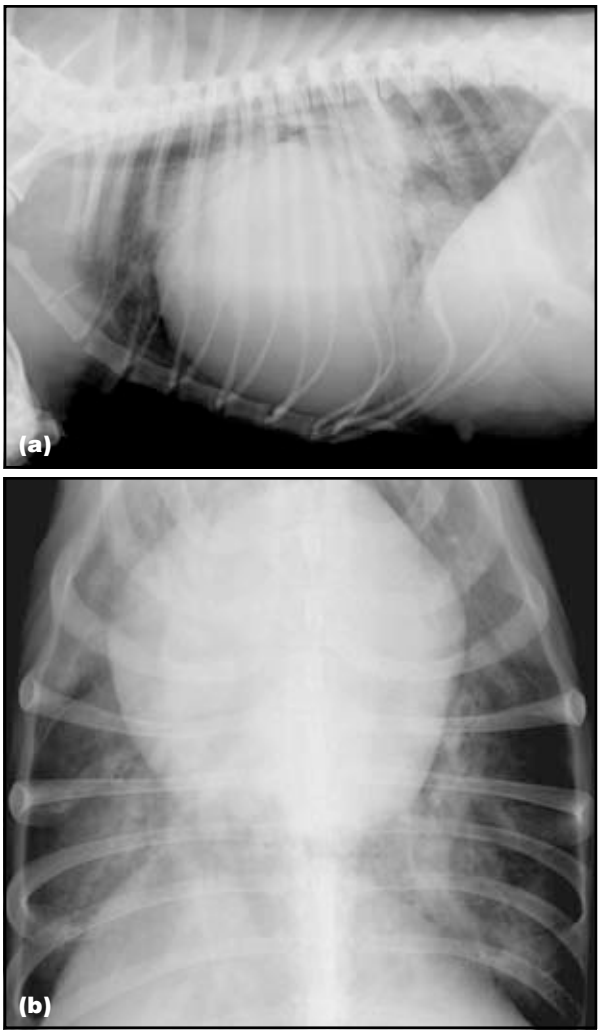
7.38 **(a)** Enlarged main pulmonary artery seen as a bulge at the 01.00–02.00 o'clock position in a dog with pulmonic stenosis (arrowed). **(b)** The main pulmonary artery may mimic a nodule on the lateral radiograph when an end-on view is obtained. This may occur, on occasion, in normal dogs, or as in this case, when the main pulmonary artery is enlarged. This is a dog with heartworm disease. The end-on main pulmonary artery is shown with arrows. The radiograph is slightly rotated.



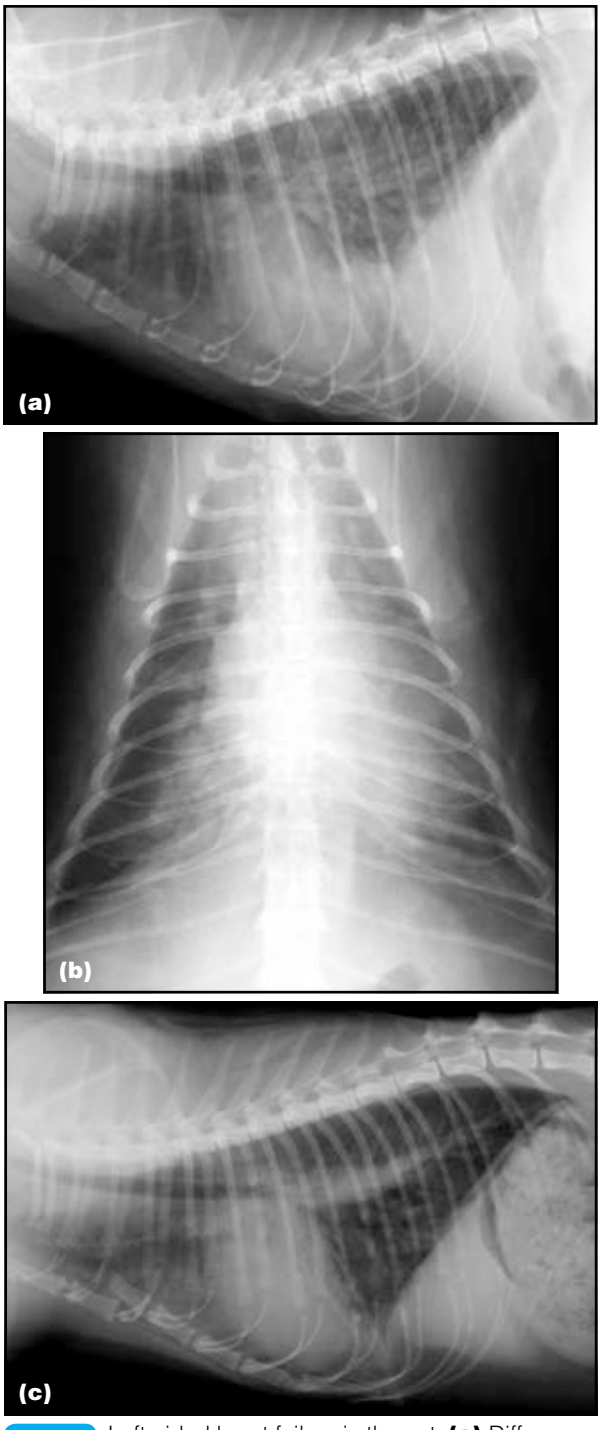
7.39 Pulmonary arterial and venous enlargement. The main pulmonary artery and vein measurements are shown by pink arrows and the rib number is also shown. **(a)** Moderately enlarged cranial lobar pulmonary arteries and veins on a close-up of a lateral radiograph of a dog with heart failure. **(b)** Massively enlarged right caudal lobar main pulmonary artery on a DV view of a dog with heartworm disease.

Small pulmonary arteries and veins
Dehydration
Shock
Hypoadrenocorticism
Positive pressure ventilation (and other causes of pulmonary hyperinflation)
Pericardial effusion with tamponade
Constrictive pericarditis
Severe pulmonic stenosis
Right-to-left shunts (tetralogy of Fallot, reverse PDA)
Focal due to pulmonary thromboembolism
Large pulmonary arteries
Pulmonary hypertension
Pulmonary thromboembolism
Heartworm disease
Left-to-right shunts (PDA, VSD, ASD)
Angiostrongylosis (but often not a feature)
Peripheral arteriovenous fistula
Large pulmonary veins
Left heart failure
(In right-to-left shunts the veins may appear larger as the arteries are small)
Large pulmonary arteries and veins
Left heart failure
Left-to-right shunts (PDA, VSD, ASD)
Excessive intravenous fluid administration

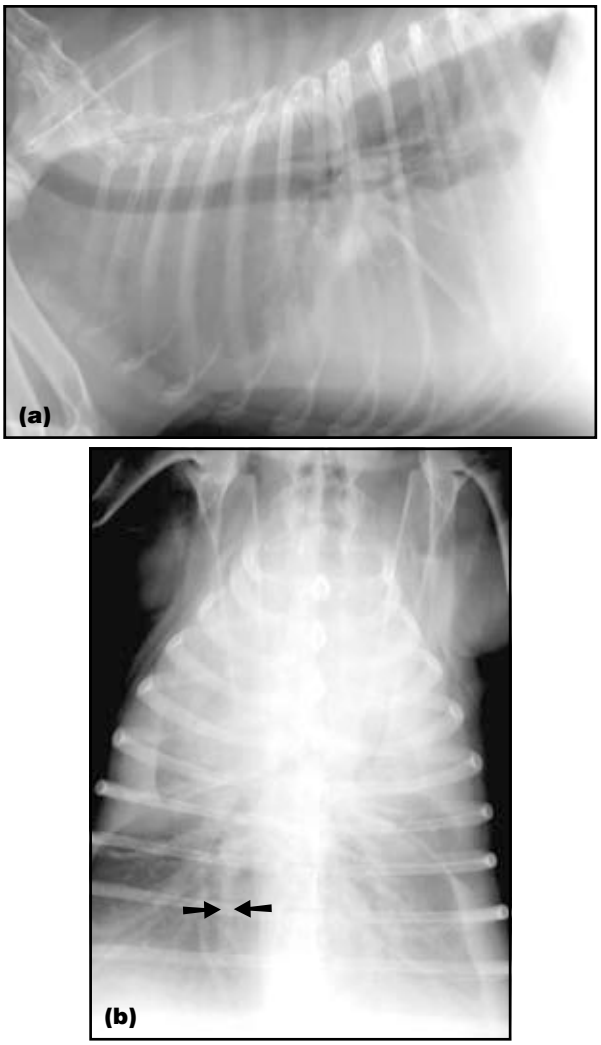
7.40 Differential diagnoses for variations in pulmonary vascular size in the dog and cat.



7.41 Left-sided heart failure in the dog. **(a)** Lateral radiograph of the same dog as in Figure 7.33 whose marked left atrial and ventricular enlargement is now accompanied by left-sided heart failure. An increase in opacity is evident in the perihilar and caudodorsal regions, due to the presence of cardiogenic alveolar oedema. The CdVC is also wide, due to accompanying right-sided heart disease. **(b)** DV view. An alveolar infiltrate is present in both left and right caudal lobes. The right caudal lobe is often an early location for oedema in left heart failure. A pleural line is seen between the right middle and caudal lobes (arrowed) and represents a small amount of pleural effusion also present in this patient.
[au: please can you add the arrows to part b – thanks]

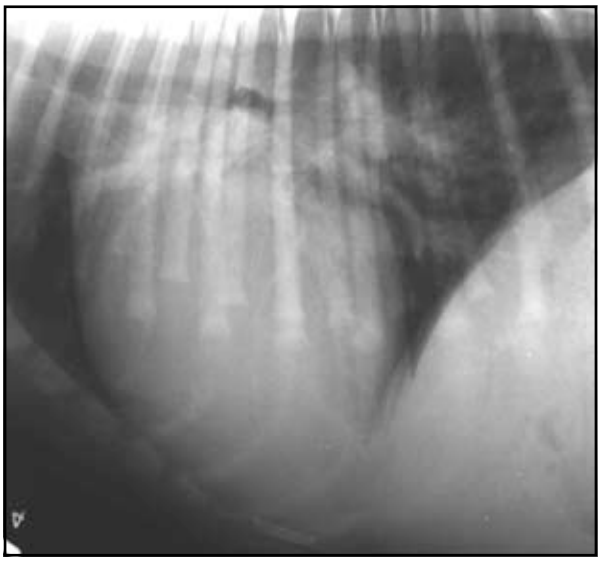


7.42 Left-sided heart failure in the cat. **(a)** Diffuse, patchy ill defined opacities (almost appear nodular) are scattered throughout the lung fields on this lateral radiograph. In some regions there is a granular interstitial infiltrate. There is a small volume of pleural effusion, recognized by the rounding and retraction of the dorsocaudal tips of the lung lobes. **(b)** DV radiograph. There is an ill defined increase in opacity in the central parts of both caudal lung lobes due to cardiogenic pulmonary oedema. The small pleural effusion is also evident. **(c)** Lateral radiograph of another cat with left-sided heart failure. The pulmonary vessels are enlarged. In this cat a more diffuse interstitial pattern is present, more marked just cranioventral to the cardiac silhouette. The cardiac silhouette is tall. A pleural fissure line suggests the presence of a small pleural effusion. An incidental old sternal injury is present between the third and fourth sternabrae.

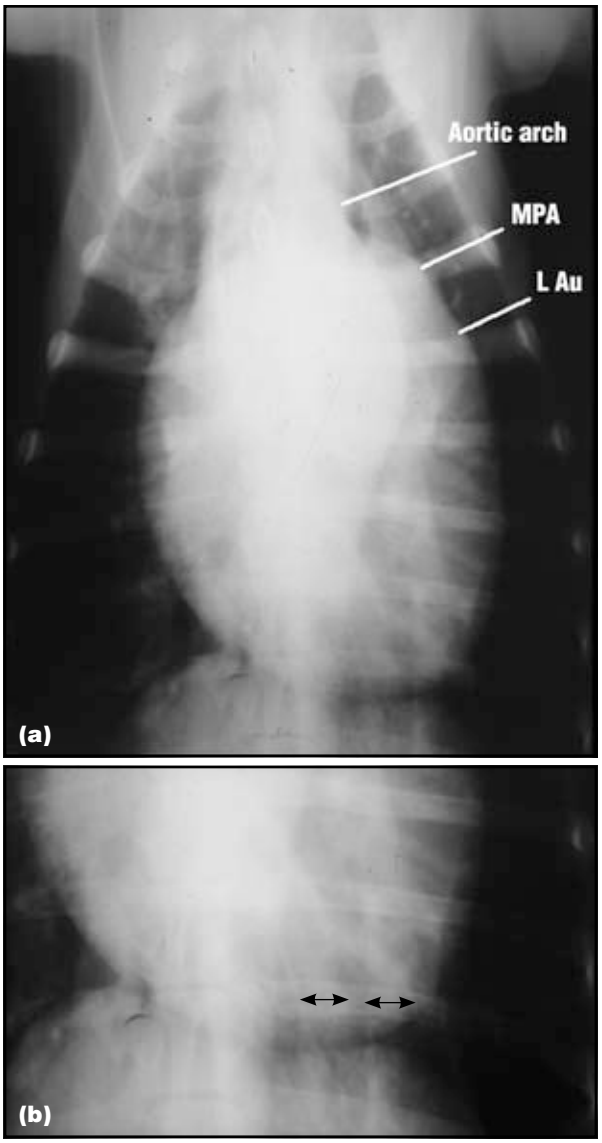


7.43 **(a)** Lateral and **(b)** DV radiographs of a dog with right-sided heart failure. A large volume of pleural effusion was present, obscuring the cardiac silhouette. The dog also had an enlarged CdVC, ascites and an enlarged liver (not seen on these radiographs). An enlarged right pulmonary caudal lobar vein (arrowed) suggests that pulmonary venous congestion may also be present due to left-sided heart disease. It is not possible to identify the cause of the effusion from these radiographs and further work-up would be required to confirm right-sided heart failure.

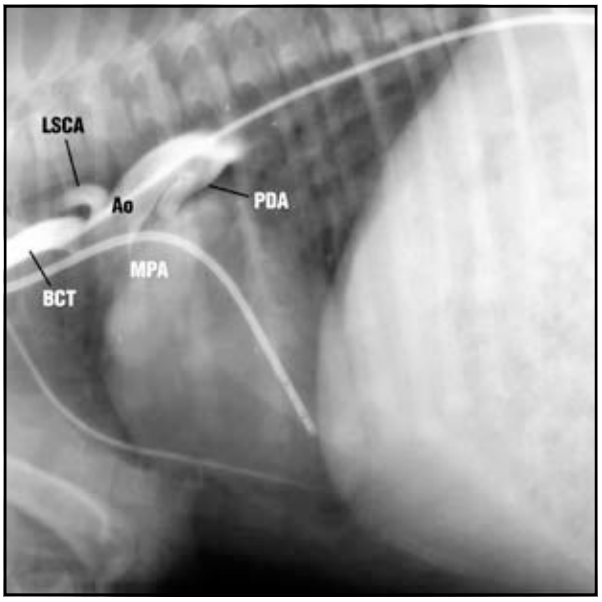
7.44 **<<To follow>>**



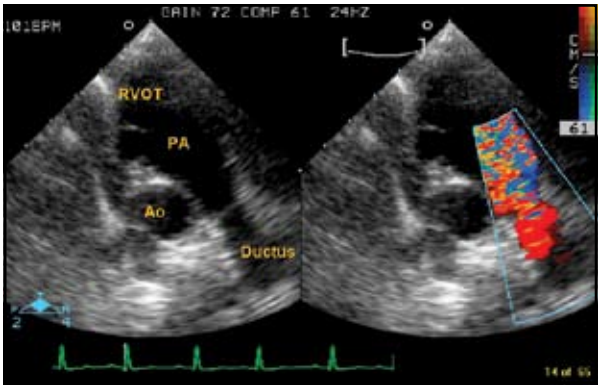
7.45 Lateral view of the thorax of a 3-month-old male puppy with PDA. There is mainly left-sided cardiomegaly with left atrial tent formation. Both the arteries and veins to the cranial lung lobes are enlarged (difficult to see on this image), which is a sign of a left-to-right shunt.



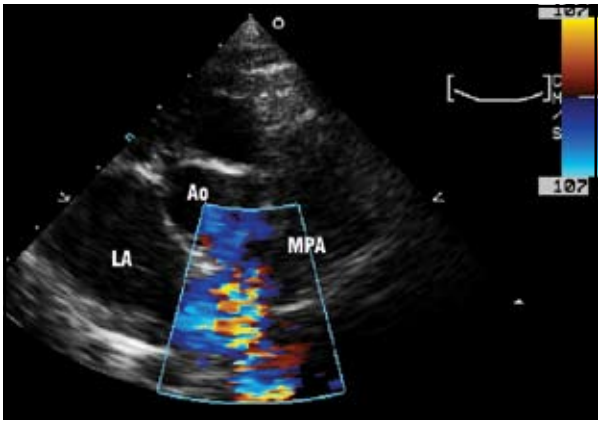
7.46 **(a)** DV view of the thorax of a dog with PDA. The cardiac silhouette is very long in a craniocaudal direction and there is bulging of the aortic arch, main pulmonary artery (MPA) and left auricular appendage (LAu). **(b)** Close-up view of the left caudal thorax. The artery and vein to the left caudal lung lobe (arrowed) have a diameter wider than the width of the ninth rib where they cross it. This is consistent with left-to-right shunting as seen with PDA.



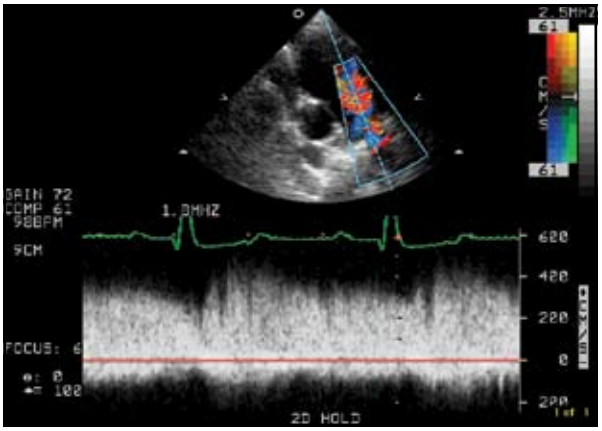
7.47 Angiocardiogram demonstrating a left-to-right shunt through a PDA in a dog. The contrast medium was injected in the aorta and the opacified ductus is seen (PDA). The contrast medium is opacifying the main pulmonary artery (MPA) at the same time as the aorta (Ao). BCT = Brachiocephalic trunk; LSCA = Left subclavian artery. (Courtesy of Dr J. Buchanan)



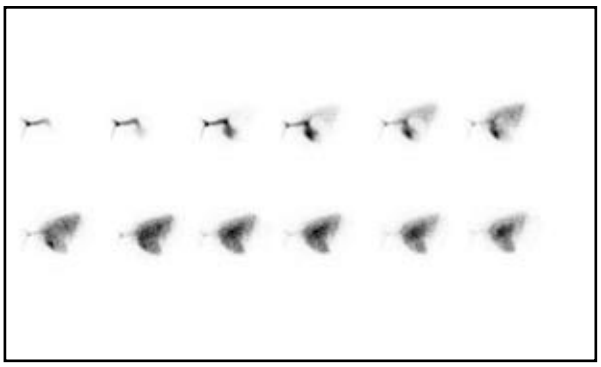
7.48 LPS echocardiogram in a dog with a PDA, optimized for visualization of the ductus. The 2D image on the left demonstrates the appearance of the ductus itself at its connection with the main pulmonary artery (PA). The aorta (Ao) and right ventricular outflow tract (RVOT) are also shown. The colour Doppler image on the right was acquired simultaneously and turbulent flow is evident within the ductus. (© Dr J. Dukes-McEwan)



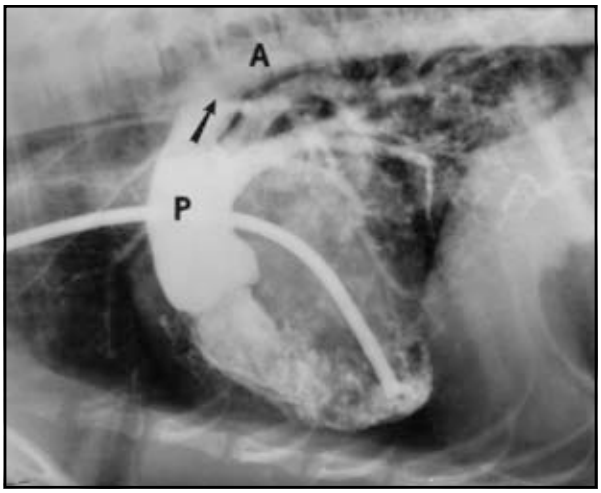
7.49 RPS short-axis echocardiogram of the heart base in a dog with a PDA. Colour Doppler shows turbulent flow in the main pulmonary artery (MPA), corresponding to the shunting of blood from the aorta (Ao). LA = Left atrium. (Courtesy of Dr M. Sleeper)



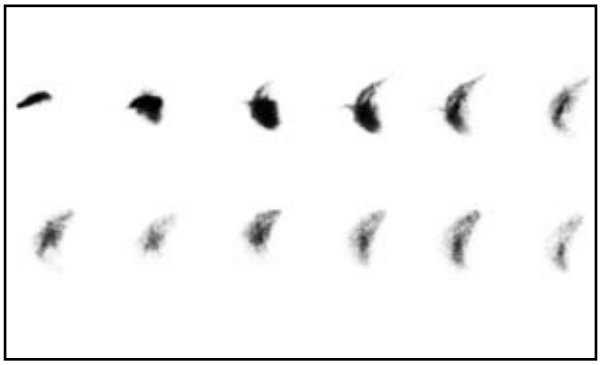
7.50 CW Doppler trace acquired via a LPS window in a dog with a PDA. The cursor is aligned with the ductus and continuous flow is evident throughout systole and diastole. (© Dr J. Dukes-McEwan)



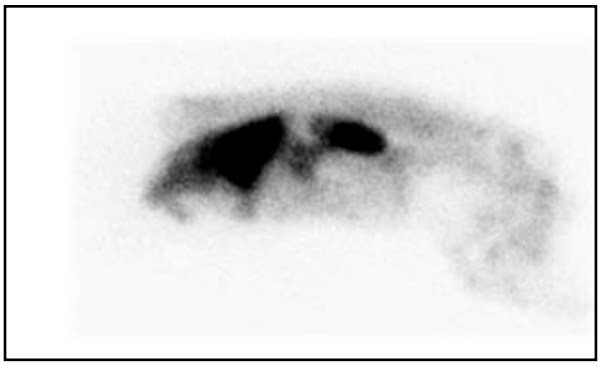
7.51 Left lateral images obtained at four frames per second during a first pass radionuclide angiogram in an 8-month-old Poodle with a left-to-right PDA. Notice the lack of clearance of the lungs in the levophase consistent with pulmonary recirculation; the aorta is never visible. The LA and LV are enlarged, consistent with volume overload.
[au: would it be helpful to the reader to label the images?]



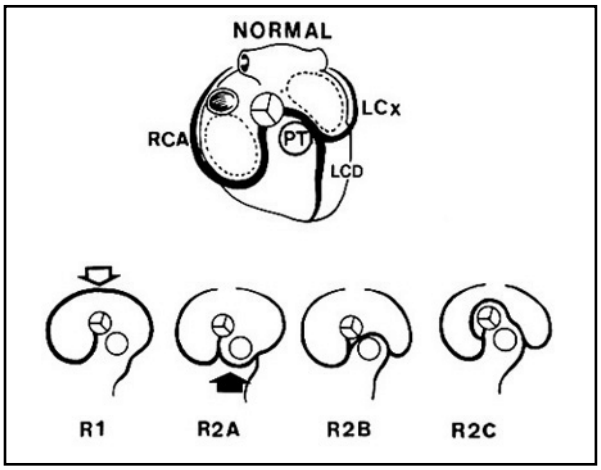
7.52 Angiocardiogram demonstrating a right-to-left shunt through a PDA in a case where significant pulmonary hypertension was present. The contrast medium was injected in the RV and the aorta (A) is opacified at the same time as the main pulmonary artery (P). The region of the PDA is indicated by the arrow. (Courtesy of Dr J. Buchanan)



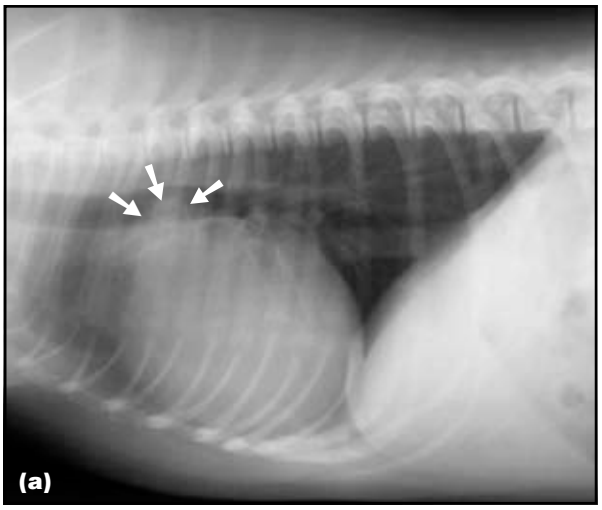
7.53 Left lateral images obtained at four frames per second during first pass radionuclide angiogram in a 6-month-old Collie with a right-to-left shunt. Notice the simultaneous visualization of the main pulmonary artery and aorta (top fourth and fifth images). A final diagnosis was not reached in this case as the owner elected euthanasia without necropsy.
[au: would it be helpful to the reader to label the images?]

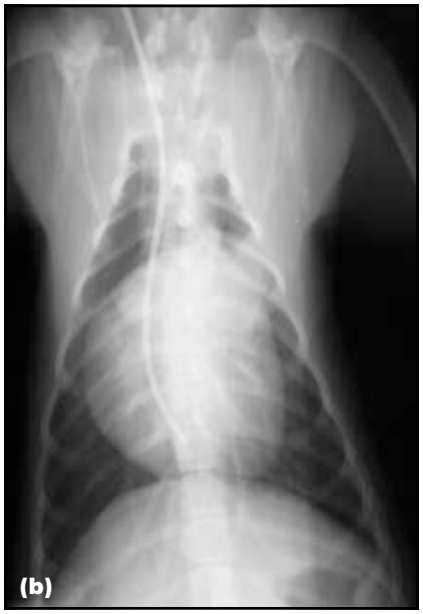


7.54 Whole body composite left lateral static image of a 1-year-old Toy Poodle with polycythaemia, obtained 5 minutes after intravenous injection of ^{99m}Tc-MAA. Note the extrapulmonary distribution of the radiopharmaceutical, with marked uptake at the level of the kidneys. Notice also the sharp cut-off at the level of the front limbs, with no radiopharmaceutical distribution to the neck, head and brain. This is consistent with a reverse PDA, in which the radioactive particles are shunted from the pulmonary to the systemic circulation caudal to the brachiocephalic trunk and left subclavian arteries.

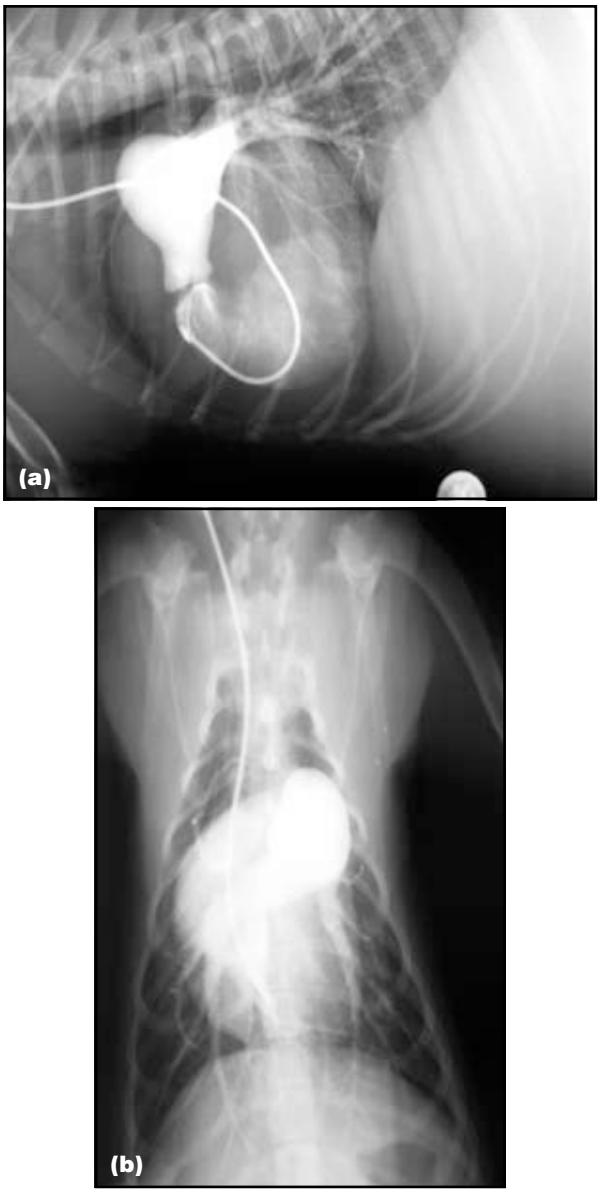


7.55 Normal coronary artery distribution in dogs and humans, and common patterns of single right coronary artery in the latter. In the type R1 pattern, the right coronary artery (RCA) continues as a single vessel and crosses the caudal crux of the atrioventricular sulcus (open arrow), then continues as the left circumflex (LCx) and left caudal descending (LCD) arteries. In type 2 patterns, the single vessel branches shortly after leaving the aorta. Sub-classifications are made depending on whether the crossing vessel (solid arrow) passes cranial to the pulmonary trunk (PT) (R2A), between the aorta and pulmonary trunk (R2B) or caudal to the aorta (R2C). (Reproduced from Buchanan (1990) with permission from the *Journal of the American Veterinary Medical Association*)

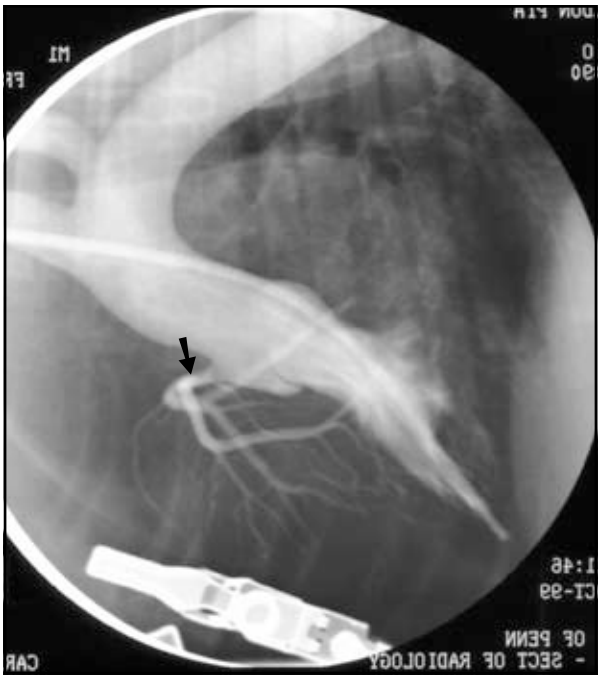




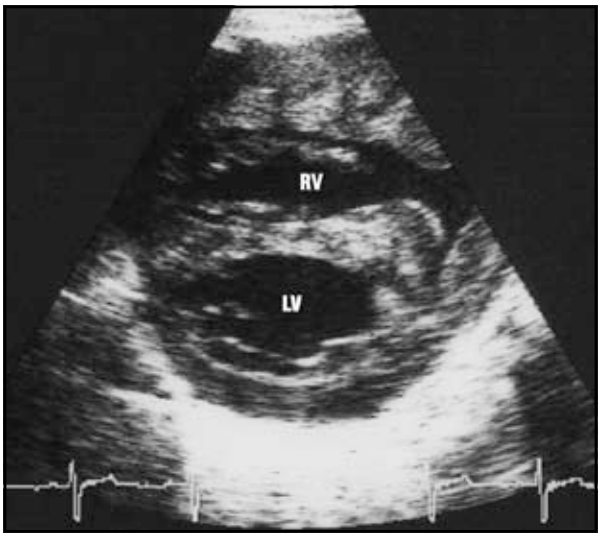
7.56 West Highland White Terrier with pulmonic stenosis. **(a)** Lateral view of the thorax. There is rounding of the cranioventral border of the heart and increased sternal contact, consistent with right-sided cardiomegaly. The dilated post-stenotic main pulmonary artery segment is seen protruding dorsally from the heart base (arrowed) and is superimposed over the ventral aspect of the caudal trachea. This feature has been termed the 'hat sign'. **(b)** VD view of the thorax. The cardiac silhouette has a reversed D shape, consistent with right-sided cardiomegaly. There is also a soft tissue opacity bulging out of the cardiac silhouette at the left fourth intercostal space, between 01.00 and 02.00 o'clock, consistent with dilation of the main pulmonary artery. A catheter is present in preparation for an angiogram. (Courtesy of Dr J. Buchanan)



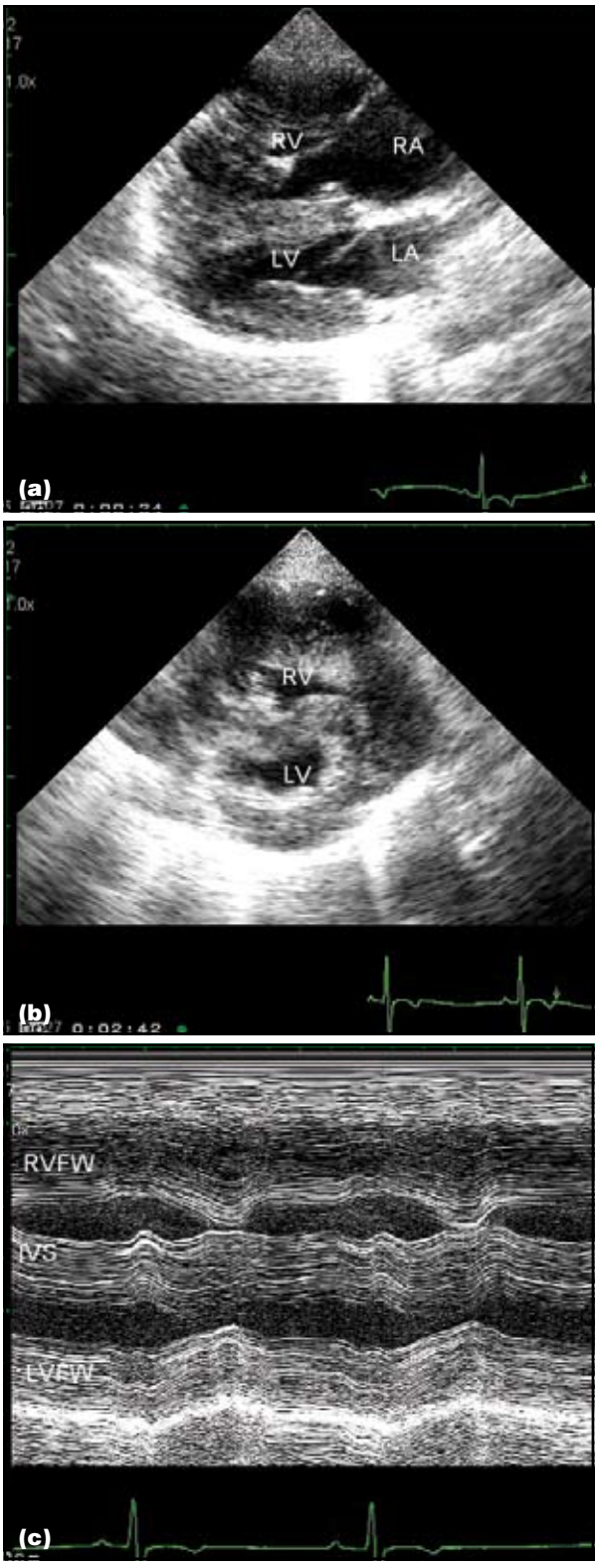
7.57 Angiocardiogram after injection of contrast medium in the RV. Same dog as Figure 7.56. **(a)** On the lateral view the main pulmonary artery is opacified and there is a clear narrowing at the level of the infundibulum as well as post-stenotic dilation of the main pulmonary artery. **(b)** On the VD view the main pulmonary artery is opacified and there is a clear dilation. (Courtesy of Dr J. Buchanan)



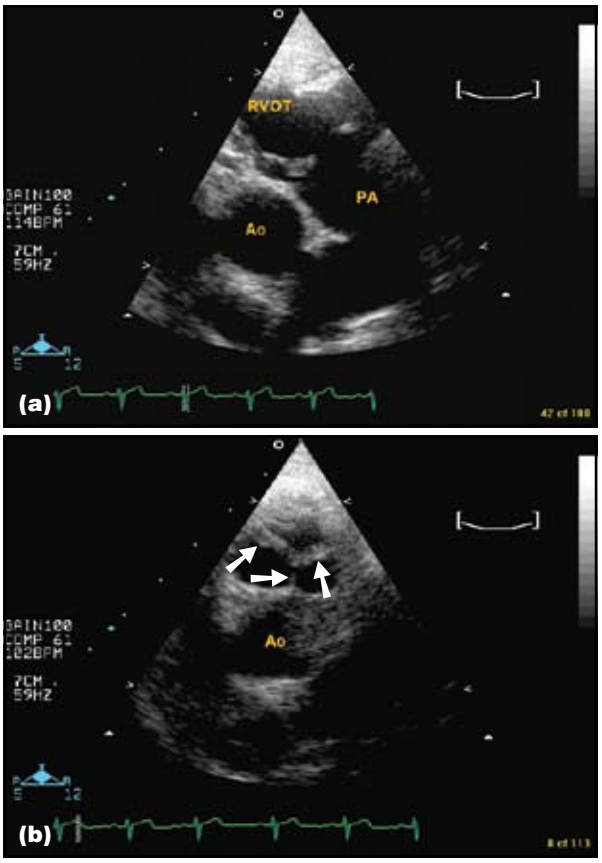
7.58 Angiocardiogram in a Bulldog Terrier with a pulmonic stenosis. The contrast medium injection was made in the LVOT in order to opacify the aorta and the coronary arteries. A single coronary artery (arrowed) is seen, which then branches into a right and left coronary artery. (Courtesy of Dr J. Buchanan)
[au: please confirm that breed is correct – thanks]



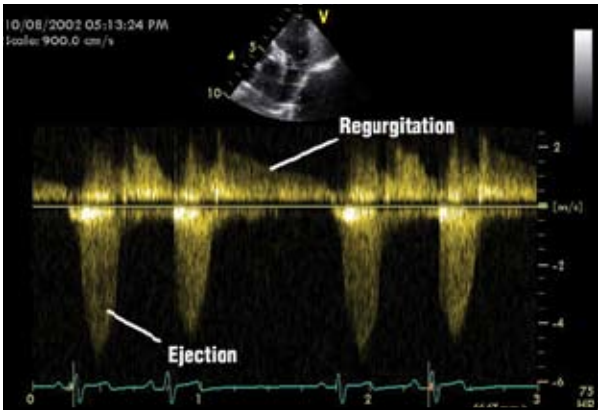
7.59 RPS short-axis echocardiogram at the level of the ventricles in a dog with pulmonic stenosis. There is a clear concentric hypertrophy of the right ventricle (RV) and flattening of the IVS, secondary to the pressure overload on the RV. LV = Left ventricle.



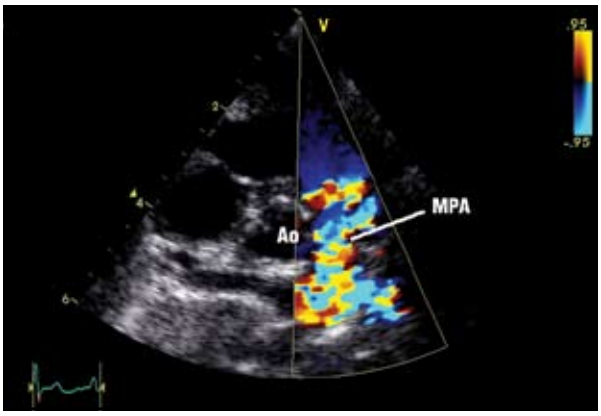
7.60 **(a)** RPS long-axis four-chamber view from a Lhaso Apso with pulmonic stenosis. Note the right ventricular hypertrophy and the right atrial enlargement. **(b)** RPS short-axis view at the level of the papillary muscles. The right ventricular free wall is markedly thickened and the IVS is flattened. **(c)** An M-mode echocardiogram acquired from the level shown in (b). Paradoxical motion of the IVS is present. IVS = Interventricular septum; LA = Left atrium; LV = Left ventricle; LVFW = Left ventricular free wall; RA = Right atrium; RV = Right ventricle; RVFW = Right ventricular free wall. (© Dr J. Dukes-McEwan)



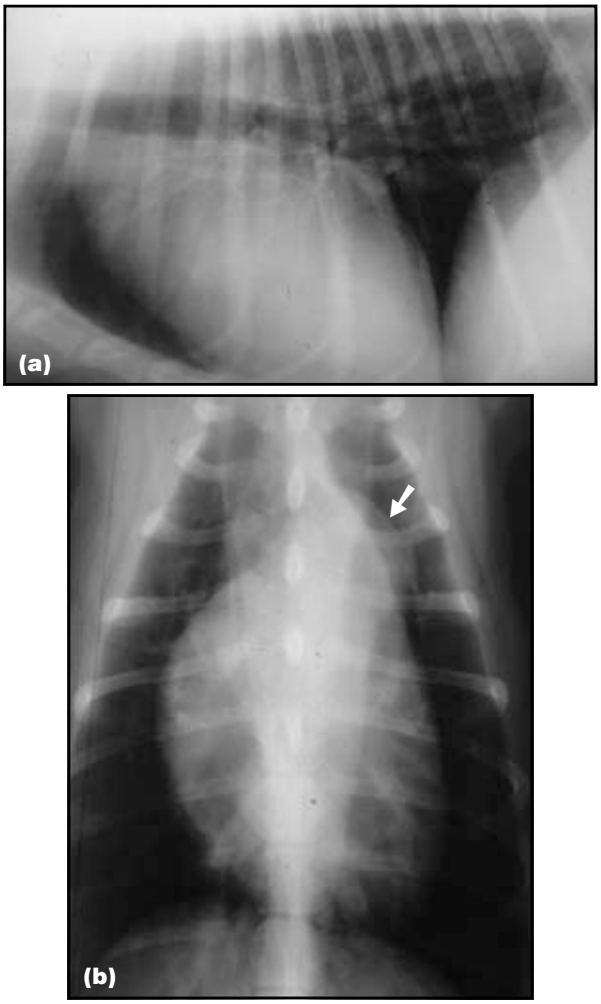
7.61 **(a)** LPS view optimized for the right ventricular outflow tract (RVOT) in a crossbred Labrador Retriever with pulmonic stenosis. Markedly thickened pulmonic valve leaflets are seen and there is a prominent post-stenotic dilation of the main pulmonary artery (PA). The aorta (Ao) is also seen in short axis. **(b)** LPS short-axis view. The pulmonic valve is seen in short axis during diastole. The valve leaflets (arrowed) are thickened and echogenic. The aorta is also seen on this image. (© Dr J. Dukes-McEwan)



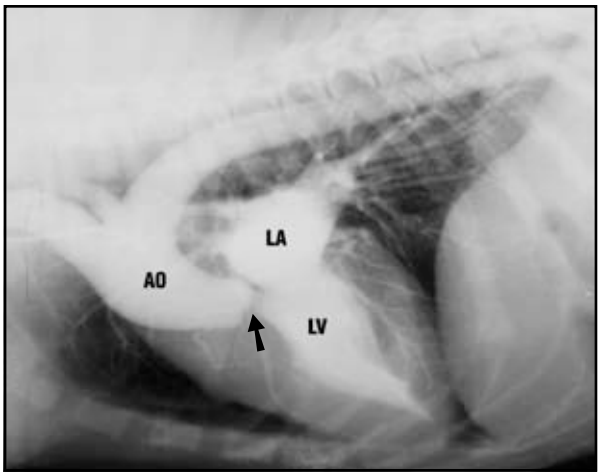
7.62 Spectral Doppler study of the main pulmonary artery in a dog with pulmonic stenosis. There is an increased velocity of the ejection flow, which is higher than 4 m/s. There is also a diastolic regurgitation from the main pulmonary artery to the RV due to some pulmonic insufficiency. (Courtesy of Dr M. Oyama)



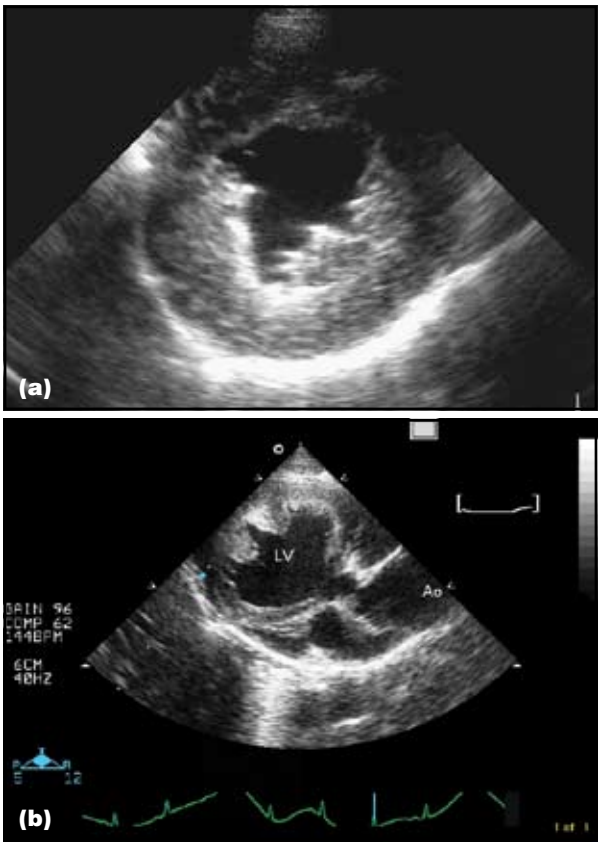
7.63 Colour Doppler study of the main pulmonary artery in a dog with pulmonic stenosis. There is a turbulent flow in the main pulmonary artery during systole as indicated by a mosaic-like colour display of the flow. Ao = Aorta; MPA = Main pulmonary artery. (Courtesy of Dr M. Oyama)



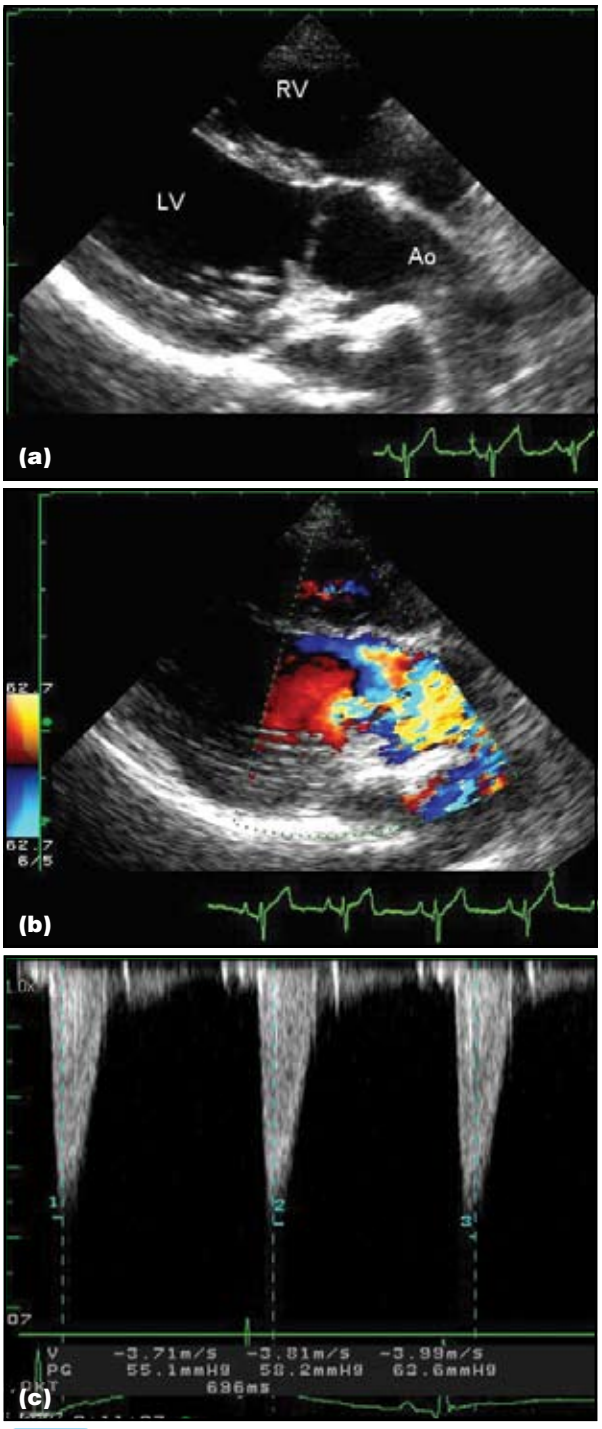
7.64 **(a)** Lateral view of the thorax in a dog with aortic stenosis. There is a marked rounding and bulging of the cardiac silhouette cranially in the region of the aortic arch. **(b)** VD view of the thorax. There is a marked bulge of the aortic arch visible between 11.00 and 01.00 o'clock (arrowed).



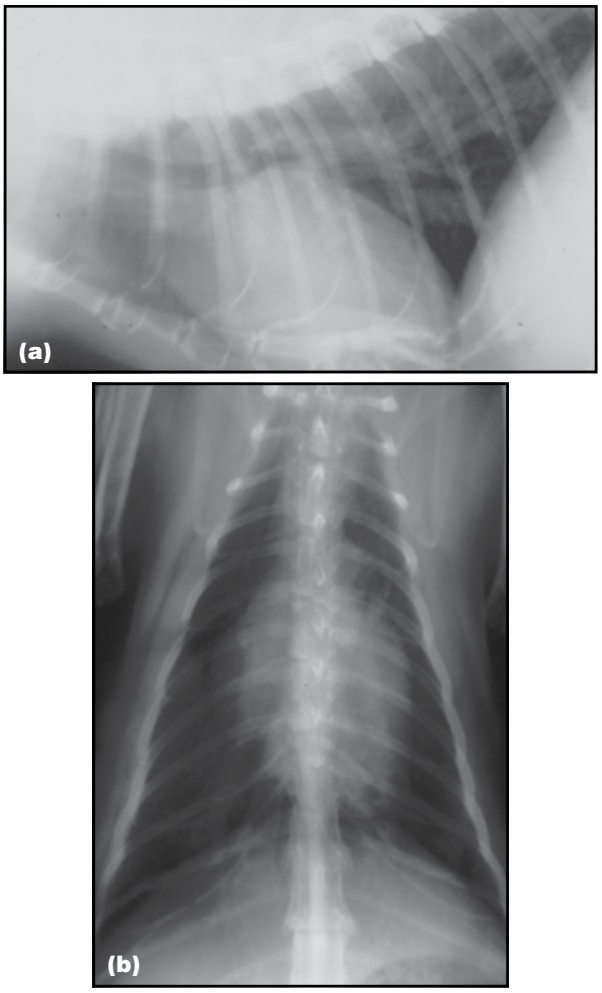
7.65 Angiocardiogram in a dog with subaortic stenosis. The contrast medium was injected in the RA and this is the levophase of the study, 7 seconds after injection of the contrast medium. There is clear narrowing of the subaortic region (arrowed) and mild post-stenotic dilation of the aorta. Some degree of concentric hypertrophy of the LV can also be appreciated on this view. AO = Aorta; LA = Left atrium; LV = Left ventricle. (Courtesy of Dr J. Buchanan)



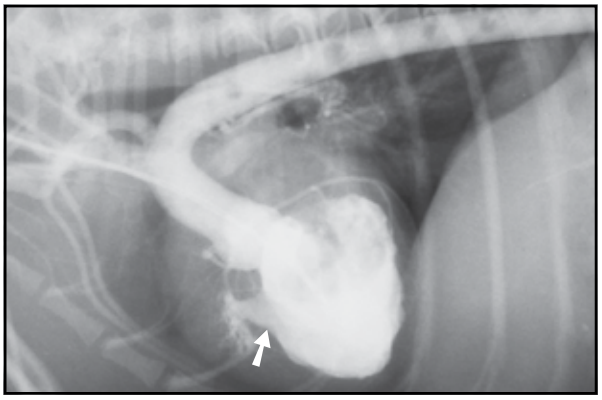
7.66 **(a)** RPS short-axis echocardiogram at the level of the papillary muscles in a dog with subaortic stenosis. There is concentric hypertrophy of the left ventricle (LV). The papillary muscles are more prominent than normal. The left ventricular cavity is reduced in this image acquired in diastole. **(b)** RPS five-chamber view of a Maine Coon cat with valvular and supravalvular aortic stenosis. Two focal regions of narrowing are identified in the LVOT and there is a dilation of the post-stenotic aorta (Ao). (© Dr J. Dukes-McEwan)



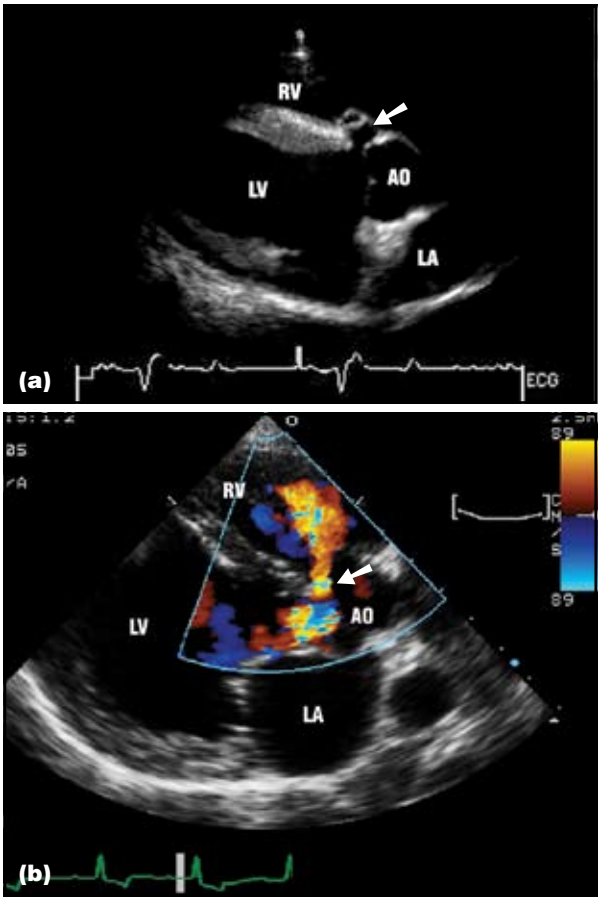
7.67 **(a)** RPS five-chamber view from a German Shepherd Dog with subvalvular aortic stenosis. Note the prominent ridge in close proximity to and just below the aortic valve. **(b)** RPS five-chamber view, showing a colour Doppler map of the aortic outflow during systole. Note the turbulent flow within the aorta due to the stenosis. **(c)** CW Doppler trace from the aorta obtained from a subcostal position. A maximal aortic velocity of 3.99 m/s was recorded. The maximal aortic velocity in most dogs is 1.7 m/s. Ao = Aorta; LV = Left ventricle; RV = Right ventricle. (© Dr J. Dukes-McEwan)



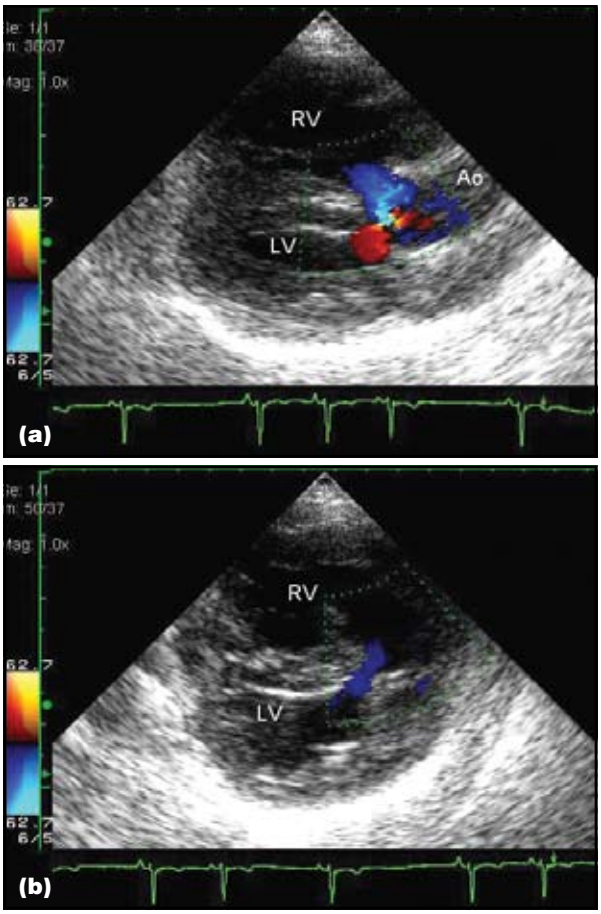
7.68 Thorax of a cat with a VSD. **(a)** Lateral view. There is generalized cardiomegaly and a marked increase in the vascular pattern of the lungs, which is consistent with left-to-right shunting. The cat had a pectus excavatum, making accurate positioning difficult. **(b)** VD view. There is moderate generalized cardiomegaly and enlargement of the caudal pulmonary lobar arteries and veins.



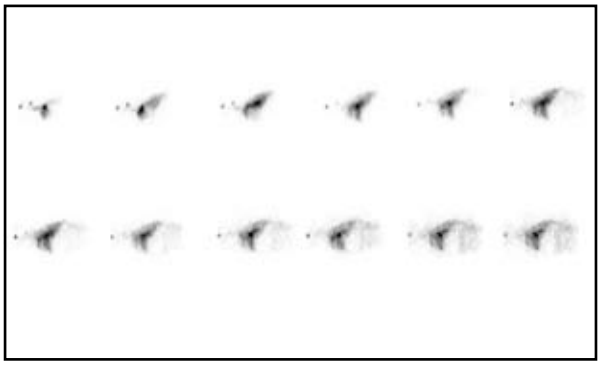
7.69 Angiocardiogram in a dog with a VSD. The contrast medium was injected in the LV and shunting can be seen from the LV to the RV through a membranous VSD (arrowed). There is also eccentric hypertrophy of the LV. The LA also appears dilated. (Courtesy of Dr J. Buchanan)



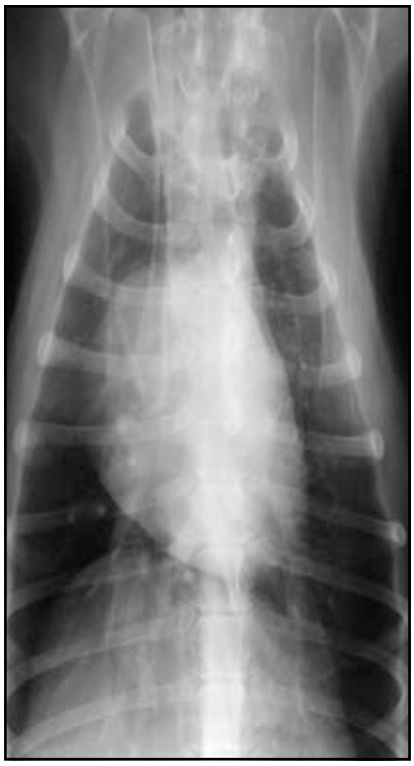
7.70 **(a)** RPS long-axis echocardiogram in a dog with a VSD. The defect is clearly visible (arrowed). There is mild left ventricular and atrial dilation. (Courtesy of Dr M. Oyama) **(b)** Colour Doppler study in a dog with a VSD. On this RPS long-axis echocardiogram, shunting is clearly seen as a turbulent flow through the defect from the LV to the RV (arrowed). There is left ventricular and atrial dilation as well as right ventricular dilation. AO = Aorta; LA = Left atrium; LV = Left ventricle; RV = Right ventricle. (Courtesy of Dr M. Sleeper)



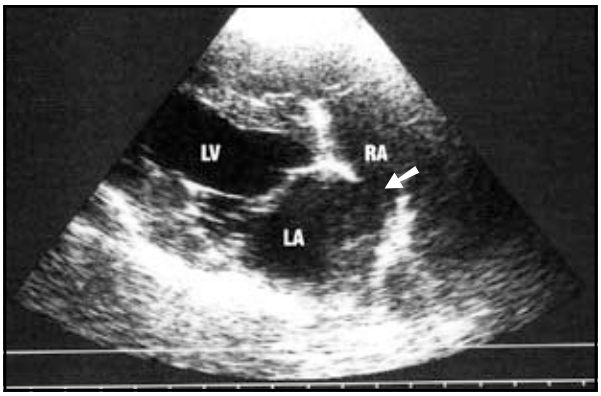
7.71 **(a)** RPS long-axis five-chamber view from a Cavalier King Charles Spaniel with a VSD and Eisenmenger's complex. The blood flows from right-to-left across the defect in this case (seen as blue on the colour Doppler map). **(b)** RPS short-axis view at the level of the ventricles. The small right-to-left shunting VSD is identified by colour Doppler as flow from the RV to the LV. This can be a subtle finding and is easily missed unless a careful and thorough examination is undertaken. Ao = Aorta; LV = Left ventricle; RV = Right ventricle. (© Dr J. Dukes-McEwan)



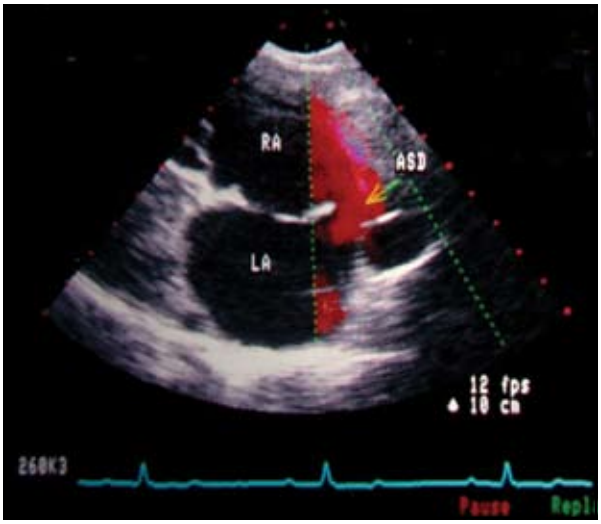
7.72 Left lateral images obtained at four frames per second during first pass radionuclide angiogram in a 1-year-old cat with a VSD. Notice the lack of clearance of the lungs in the levophase and the lack of distinction of the margins of the aorta, consistent with pulmonary recirculation in a left-to-right shunt. In this case, reappearance of the RV in the levophase (best seen in the two top right images) indicates a VSD.
[au: would it be helpful to the reader to label the images?]



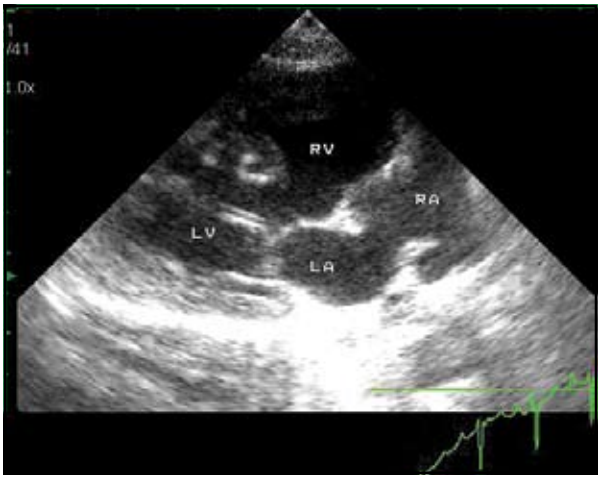
7.73 DV view of the thorax in a dog with an ASD. There is mild rounding of the cardiac silhouette in the region of the RA, between 09.00 and 11.00 o'clock.



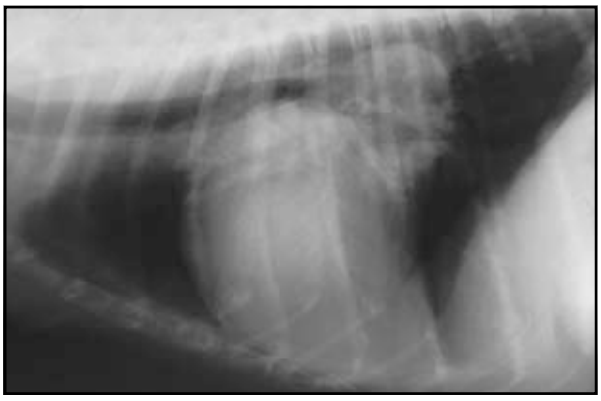
7.74 RPS long-axis echocardiogram in a dog with an ASD. The defect is clearly seen as an interruption of the echogenic line of the interatrial septum (arrowed). LA = Left atrium; LV = Left ventricle; RA = Right atrium.



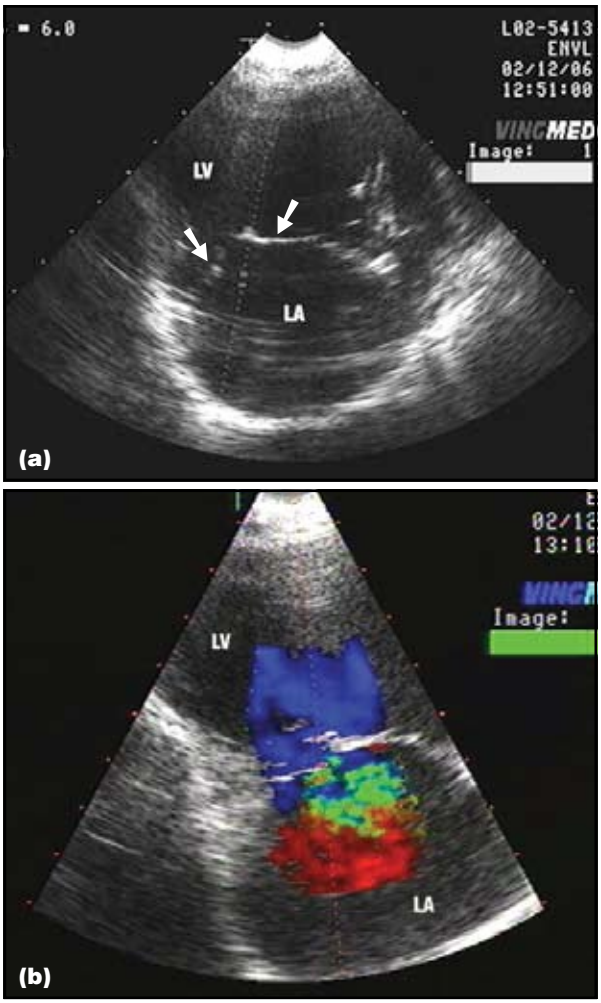
7.75 RPS long-axis four-chamber view of a Boxer with an ostium secundum type ASD. Flow across the defect is identified. ASD = Atrial septal defect; LA = Left atrium; RA = Right atrium. (© Dr J. Dukes-McEwan)



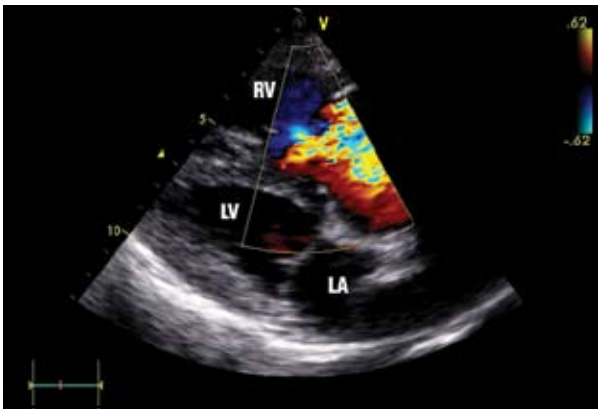
7.76 RPS four-chamber echocardiogram of a cat with an endocardial cushion defect. A large VSD and ASD are present. LA = Left atrium; LV = Left ventricle; RA = Right atrium; RV = Right ventricle. (© Dr J. Dukes-McEwan)



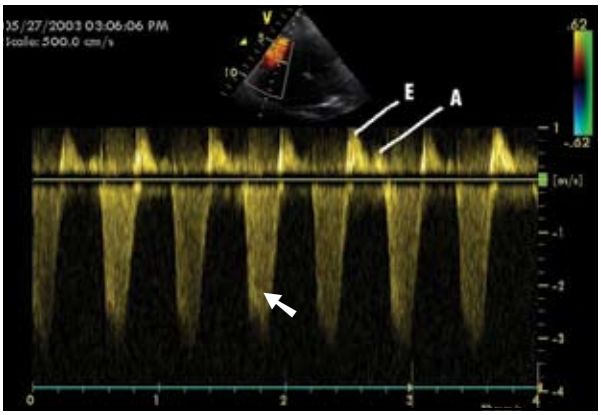
7.77 Lateral view of the thorax in a dog with mitral valve dysplasia. There is a marked dilation of the LA, creating a prominent bulge between 12.00 and 03.00 o'clock.



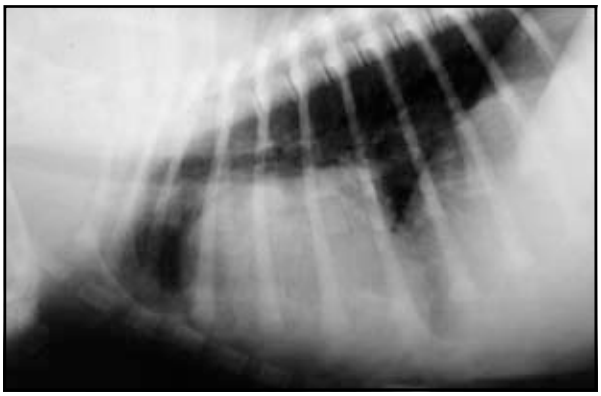
7.78 **(a)** Left apical echocardiogram of a dog with mitral valve dysplasia, presenting both mitral stenosis and regurgitation as a functional consequence of the valvular malformation. There is marked dilation of the LA. Note the lack of opening of the mitral valve leaflets, leading to a mitral stenosis (arrowed) in this diastolic image. **(b)** Left apical echocardiogram colour Doppler study in systole. There is a mitral regurgitation with a high-velocity, turbulent flow. LA = Left atrium; LV = Left ventricle.



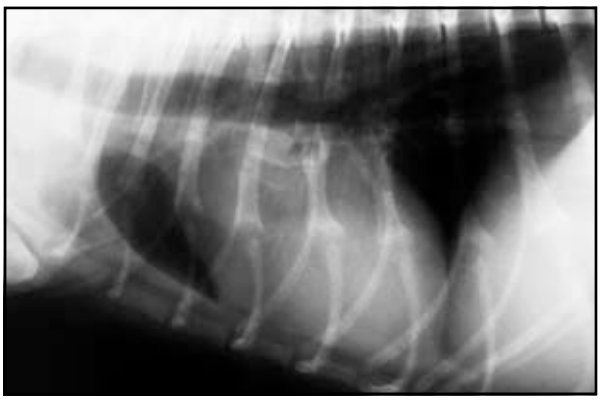
7.80 RPS long-axis echocardiogram in a dog with tricuspid valve dysplasia. Marked dilation of the right cardiac chambers is evident. The RV is almost larger than the LV and the IVS is flat and pushed to the left side of the heart. Colour Doppler shows a turbulent high-velocity tricuspid regurgitation. LA = Left atrium; LV = Left ventricle; RV = Right ventricle. (Courtesy of Dr M. Oyama)



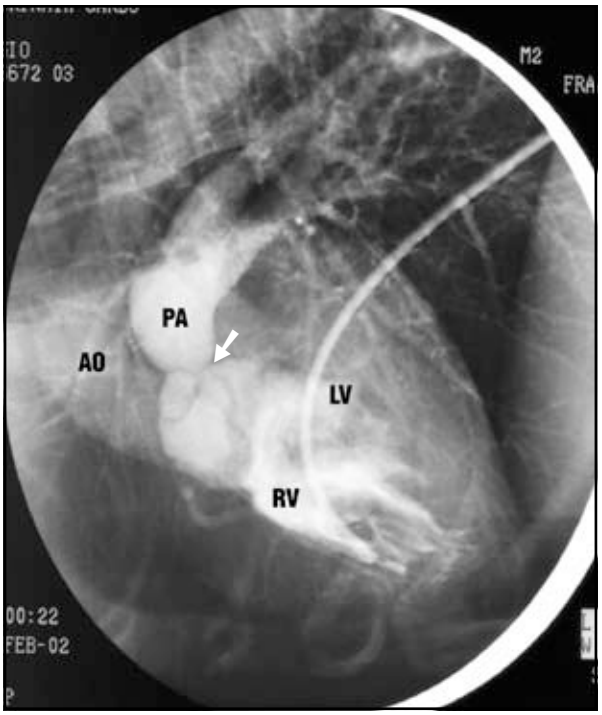
7.81 Spectral Doppler study of the tricuspid valve in a dog with tricuspid dysplasia. There is a systolic high-velocity tricuspid regurgitation reaching 3 m/s (arrowed). A = Normal diastolic A wave; E = Normal diastolic E wave. (Courtesy of Dr M. Oyama)



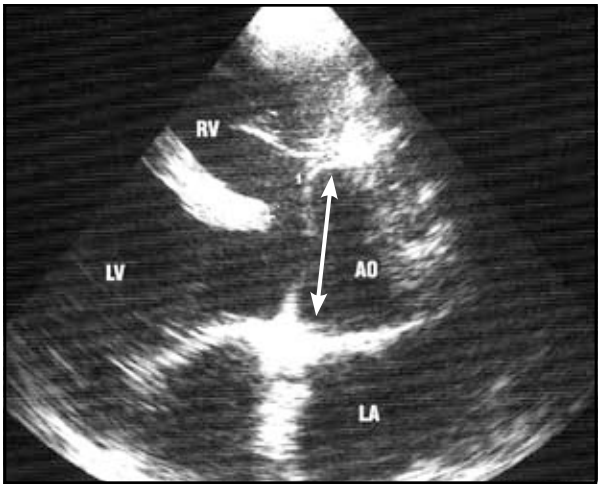
7.79 Lateral view of the thorax in a puppy with tricuspid valve dysplasia. Right-sided cardiomegaly is evident.



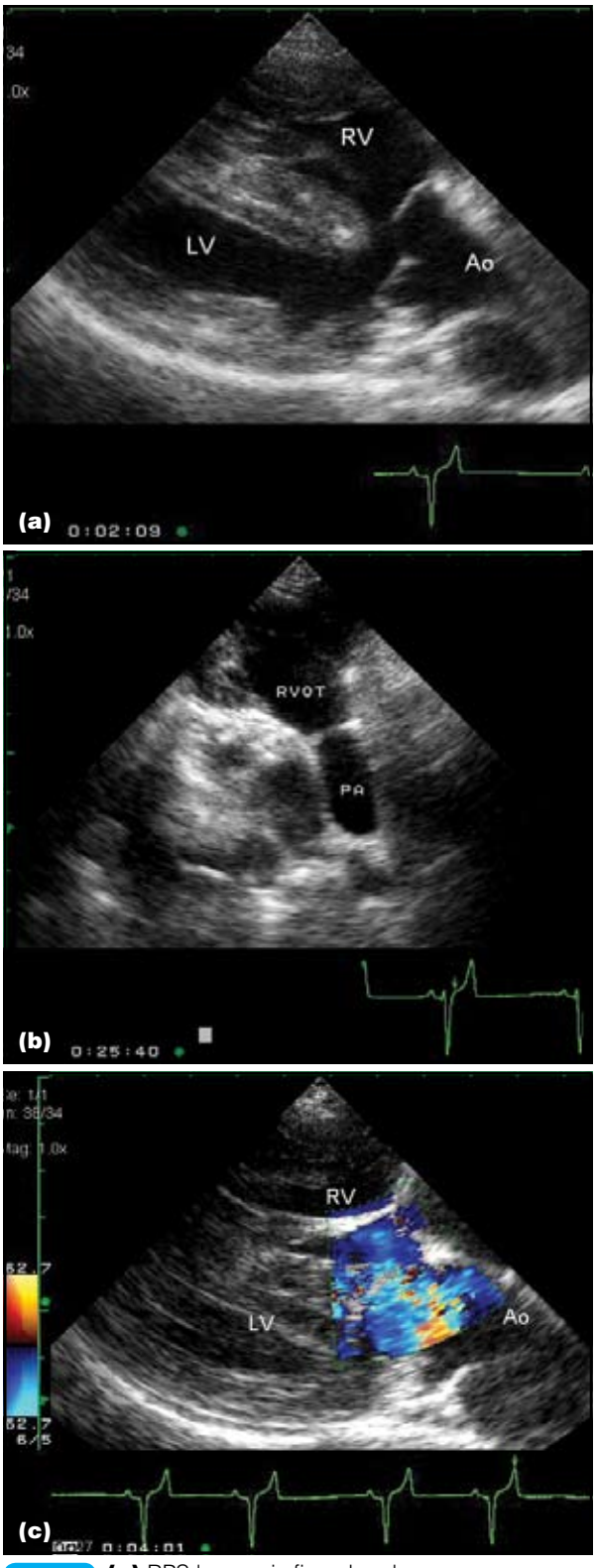
7.82 Lateral view of the thorax in a dog with tetralogy of Fallot. There is mild right-sided cardiomegaly. Note also the loss of the cranial waist of the cardiac silhouette, which is probably secondary to the overriding of the aorta. Diffuse hyperlucency of the lung fields is identified due to hypovascularization.



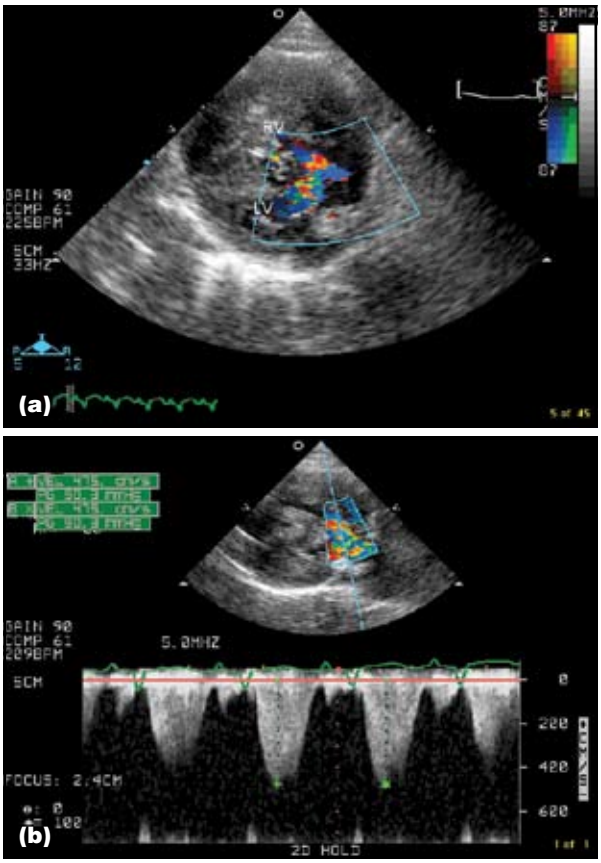
7.83 Angiocardiogram in a dog with tetralogy of Fallot. The contrast medium was injected in the RV. There is narrowing of the main pulmonary artery at the infundibular level (arrowed) and there is simultaneous opacification of the main pulmonary artery and the aorta, which means that there is a right-to-left shunt. AO = Aorta; LV = Left ventricle; PA = Main pulmonary artery; RV = Right ventricle. (Courtesy of Dr J. Buchanan)



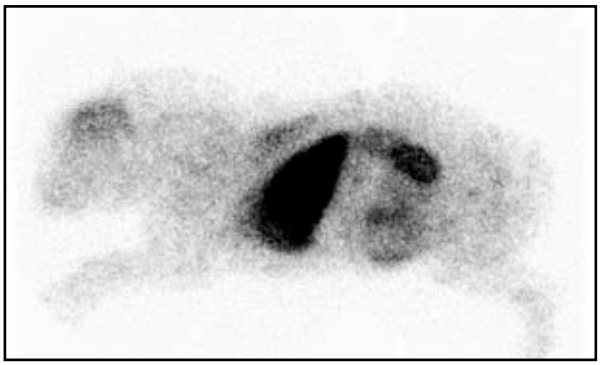
7.84 RPS long-axis echocardiogram in a dog with tetralogy of Fallot. There is overriding of the aorta (double headed arrow) over the IVS. A VSD is created by this abnormal position of the aorta. AO = Aorta; LA = Left atrium; LV = Left ventricle; RV = Right ventricle.



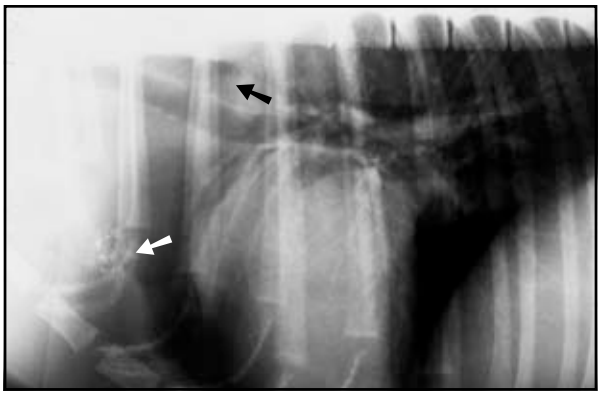
7.85 **(a)** RPS long-axis five-chamber echocardiogram of a Border Collie with tetralogy of Fallot. The aorta is overriding or dextraposed and a high VSD is present. **(b)** RPS short-axis view obtained at the level of the heart base and optimized for the RVOT. The pulmonic valve is stenotic and the valves are thickened and echogenic. The main pulmonary artery is hypoplastic. **(c)** RPS long-axis five-chamber view with colour Doppler. Blood is shunting from right-to-left across the VSD. Ao = Aorta; LV = Left ventricle; PA = Main pulmonary artery; RV = Right ventricle; RVOT = Right ventricular outflow tract. (© Dr J. Dukes-McEwan)



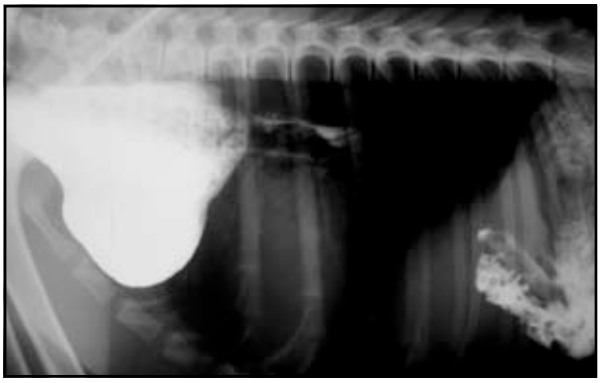
7.86 **(a)** RPS short-axis view of a Domestic Shorthair cat with a tetralogy of Fallot. A high VSD is present and the colour flow map documents the right-to-left flow. **(b)** CW Doppler shows the right-to-left flow across the septal defect. LV = Left ventricle; RV = Right ventricle. (© Dr J. Dukes-McEwan)



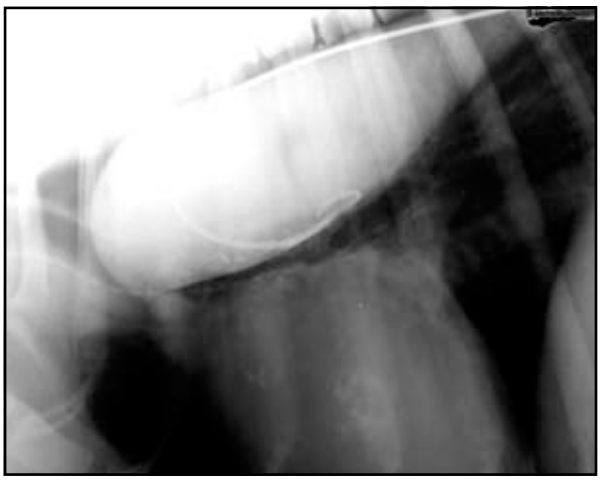
7.87 Whole body composite left lateral static image of a 4-month-old English Bulldog obtained 5 minutes after intravenous injection of ^{99m}Tc-MAA. Notice the extrapulmonary distribution of the radiopharmaceutical, with marked uptake at the level of the renal cortices, as well as the cerebral cortex; compare with Figure 7.54. The diagnosis was tetralogy of Fallot.



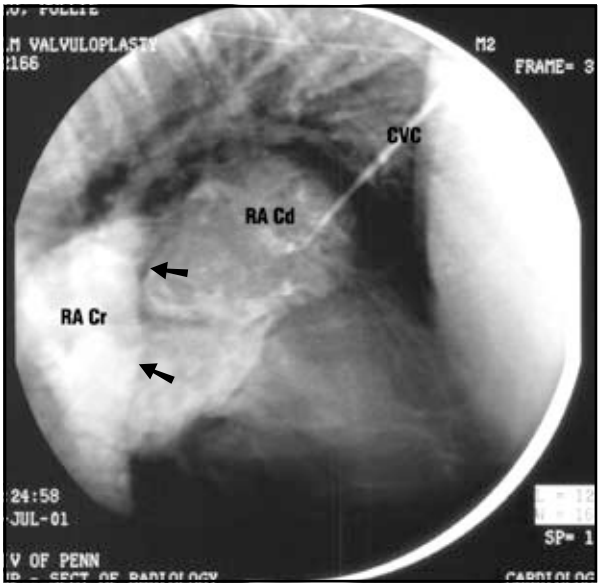
7.88 Lateral view of the thorax of a dog with a PRAA. Note the gas accumulation in the cranial portion of the thoracic oesophagus (black arrow) on this survey radiograph. There is mineralized material in the cranioventral thorax (white arrow), which represents material accumulating in the distended oesophagus cranial to the vascular anomaly. (Courtesy of Dr J. Buchanan)



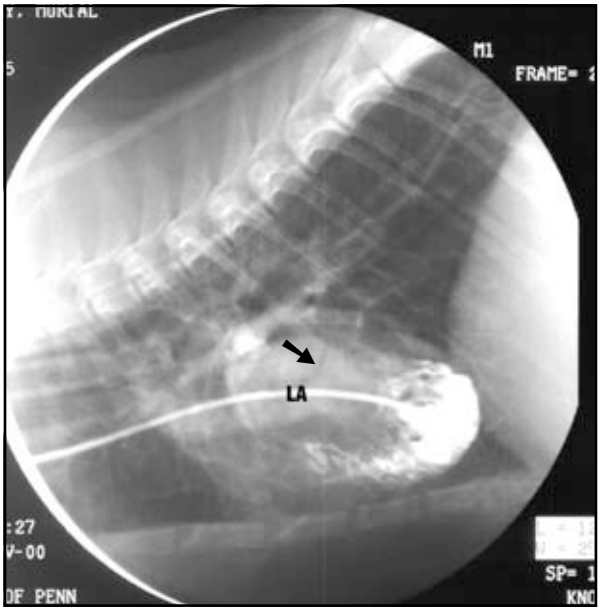
7.89 Lateral view of the thorax of a dog with a PRAA, after oral administration of barium sulphate. There is accumulation of the contrast medium in a distended oesophagus cranial to the heart base and abrupt tapering at the level of the heart base. (Courtesy of Dr J. Buchanan)



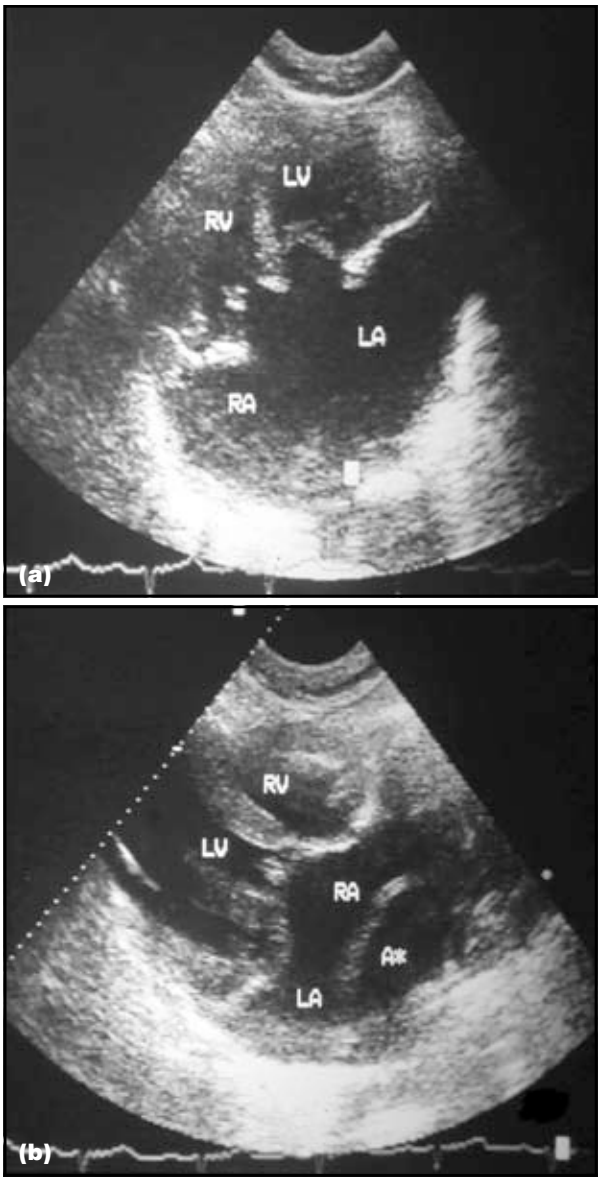
7.90 Lateral view of the thorax in a dog with aortic coarctation. Contrast medium was injected in the aortic arch after catheterization of the femoral artery. There is narrowing of the aorta distal to the origin of the left subclavian artery, followed by marked dilation of the aorta after the stenosis. The catheter is seen curving around in the dilated portion of the aorta. (Courtesy of Dr M. Herrtage)



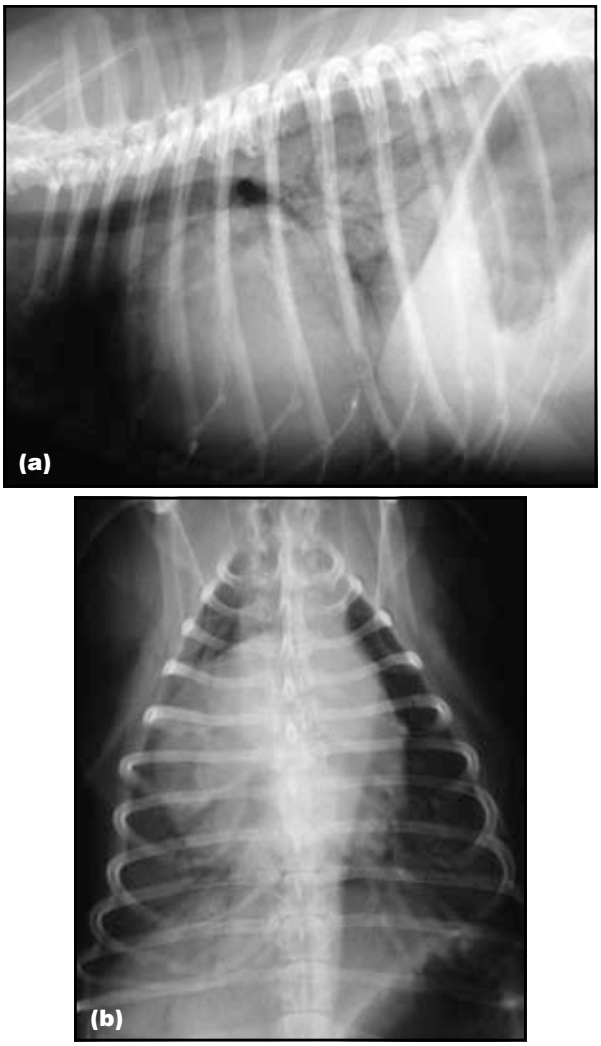
7.91 Angiocardiogram in a dog with CTD. The contrast medium was injected in the CdVC. The RA is markedly dilated and there is a linear filling defect, corresponding to the dividing membrane (arrowed). CVC = Caudal vena cava; RA Cd = Caudal section of the right atrium; RA Cr = Cranial section of the right atrium. (Courtesy of Dr J. Buchanan)



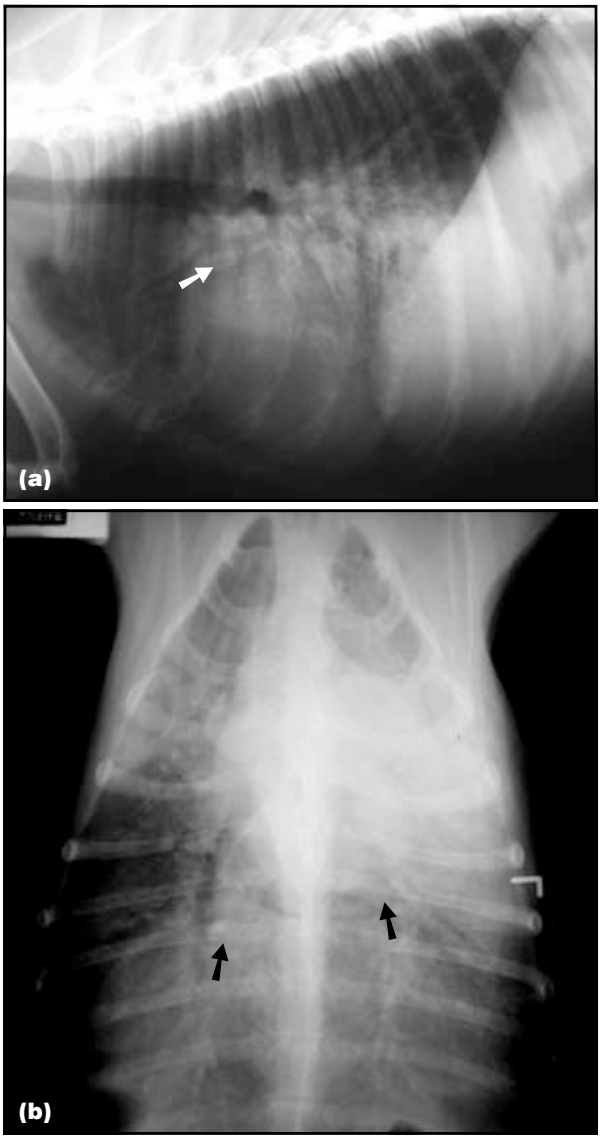
7.92 Angiocardiogram in a dog with CTS. The contrast medium was injected in the RV after catheterization of the jugular vein. A levophase image is presented, where the contrast medium has reached the left atrium (LA). The LA is enlarged and a linear filling defect is visible in its lumen, corresponding to the dividing membrane (arrowed). (Courtesy of Dr J. Buchanan)



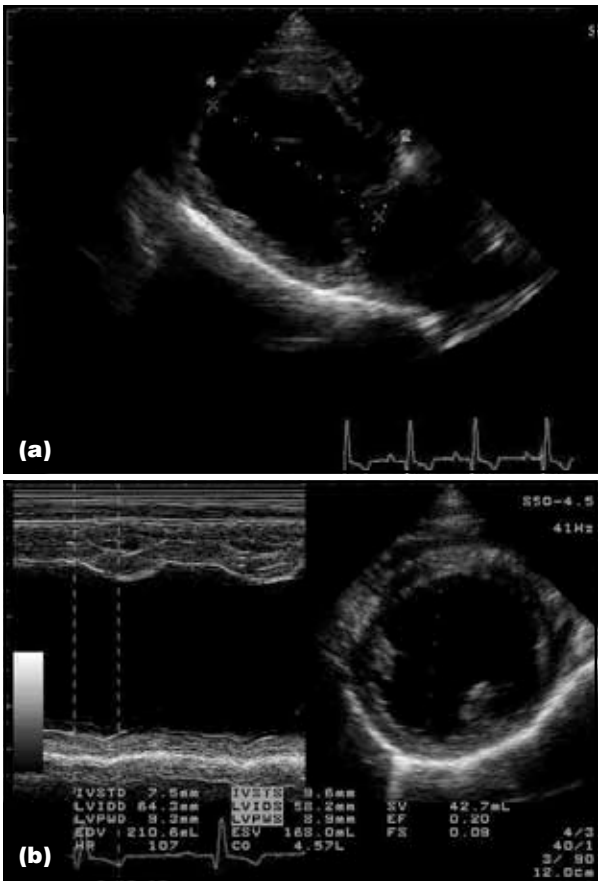
7.93 **(a)** Left apical four-chamber echocardiogram of a cat with an endocardial cushion defect and an unusual cor triatriatum. On this view the large confluent ASDs and VSDs are evident. **(b)** RPS long-axis view. The ASD can be seen and within the common atrium an additional thin dividing membrane is evident, creating an extra chamber (A*). This was considered to be a CTS; however, the decision as to whether the lesion is left- or right-sided is a difficult one when a common atrium is present. LA = Left atrium; LV = Left ventricle; RA = Right atrium; RV = Right ventricle. (© Dr J. Dukes-McEwan)



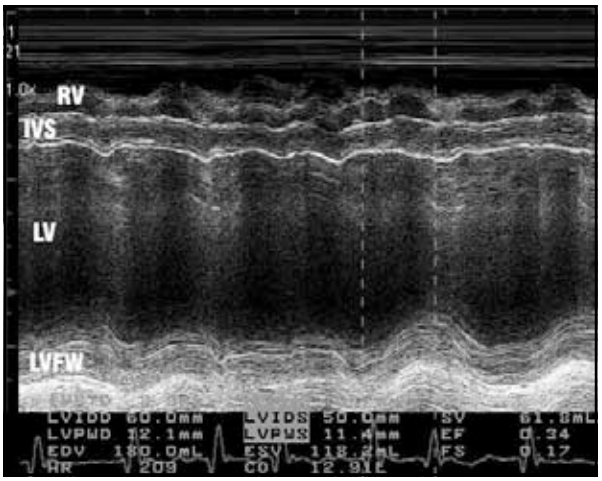
7.94 **(a)** Right lateral and **(b)** and DV thoracic radiographs from a Cocker Spaniel, which presented with coughing and dyspnoea. There is generalized cardiomegaly with left atrial enlargement. Pulmonary venous distension is apparent. There is a mixed interstitial–alveolar infiltrate, consistent with pulmonary oedema. Echocardiography confirmed that this dog had DCM, resulting in the left-sided congestive heart failure.



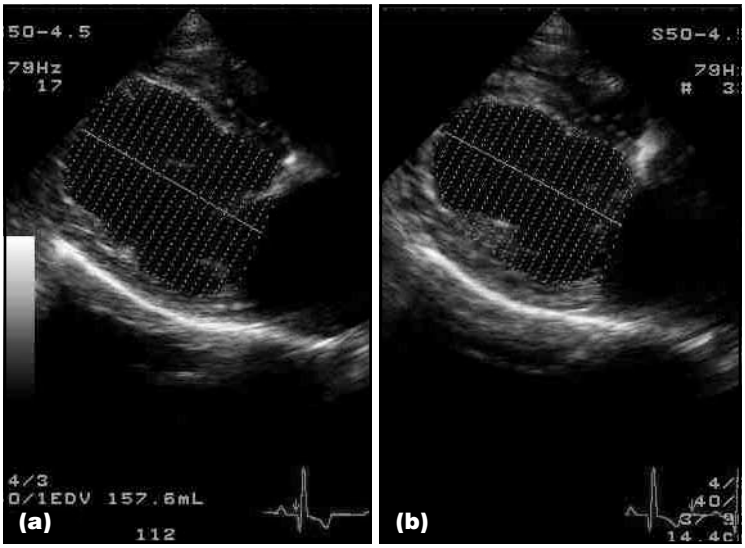
7.95 **(a)** Right lateral and **(b)** DV thoracic radiographs from a Dobermann, which presented with a history of several weeks coughing, recent syncopal episodes and then dyspnoea. DCM in the Dobermann is not associated with radiographic evidence of massive cardiomegaly, but there is left atrial and left ventricular enlargement. Note the pulmonary venous distension (arrowed) and the predominantly perihilar mixed interstitial and alveolar infiltrate.



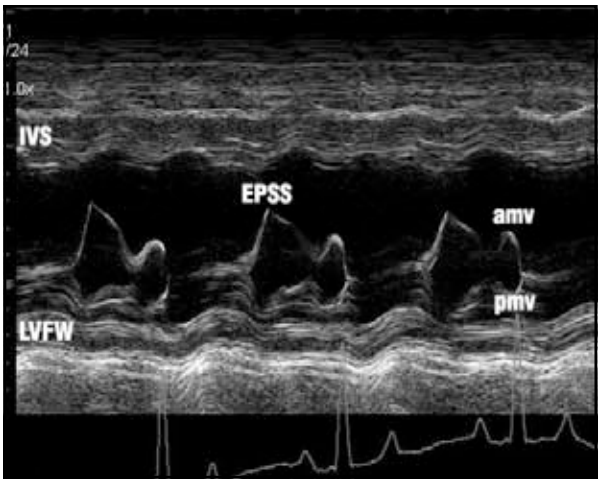
7.96 Calculation of the index of sphericity. **(a)** On a RPS long-axis view, every attempt is made to optimize the LV length. From a frozen image (diastolic frame: start of QRS complex), a measurement is taken from the mitral annulus to the apex of the LV. The diastolic LV length in this example is 77.8 mm. **(b)** The diastolic 'width' of the LV, at chordal level, is the M-mode diastolic chamber dimension (here 64.3 mm). In this example, the index of sphericity is the LV length (diastole)/LV width (diastole) = 77.8/64.3 = 1.21 (normal >1.7). The ventricle is confirmed as being abnormally rounded.



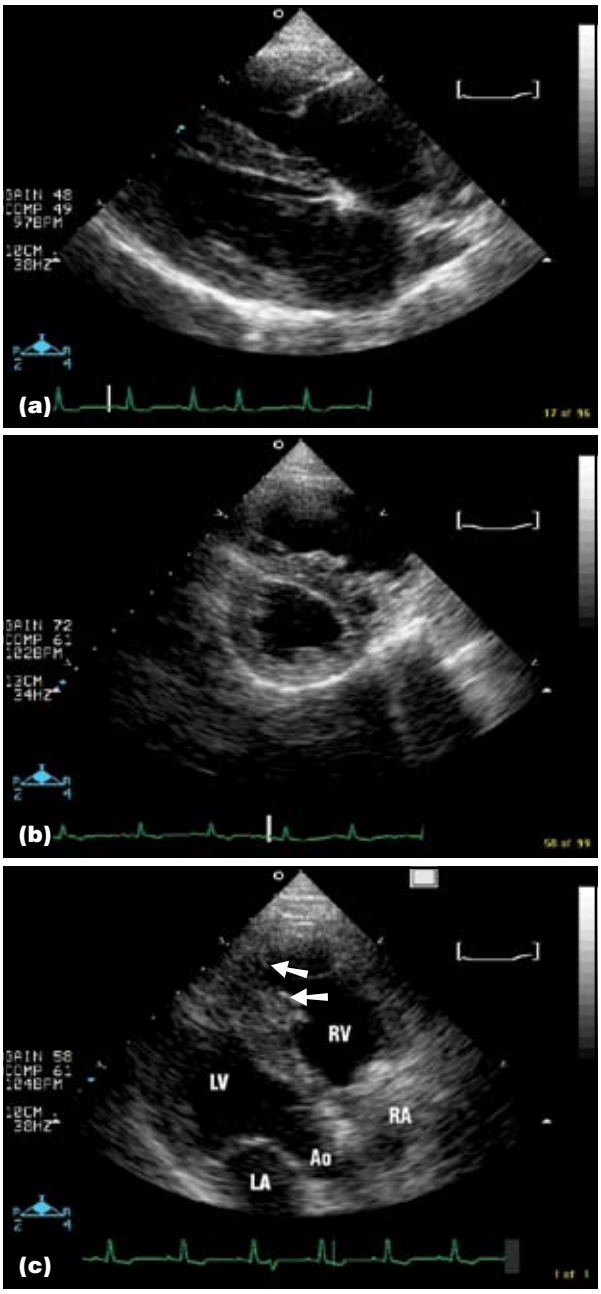
7.98 LV M-mode obtained from a Dobermann with DCM, indicating marked hypokinesis of the LV, proportionately thin walls and a dilated LV chamber. The LV diastolic and systolic measurements can be compared with reference values for breed, where known. This dog has atrial fibrillation. IVS = Interventricular septum; LV = Left ventricle; LVFW = Left ventricular free wall; RV = Right ventricle.



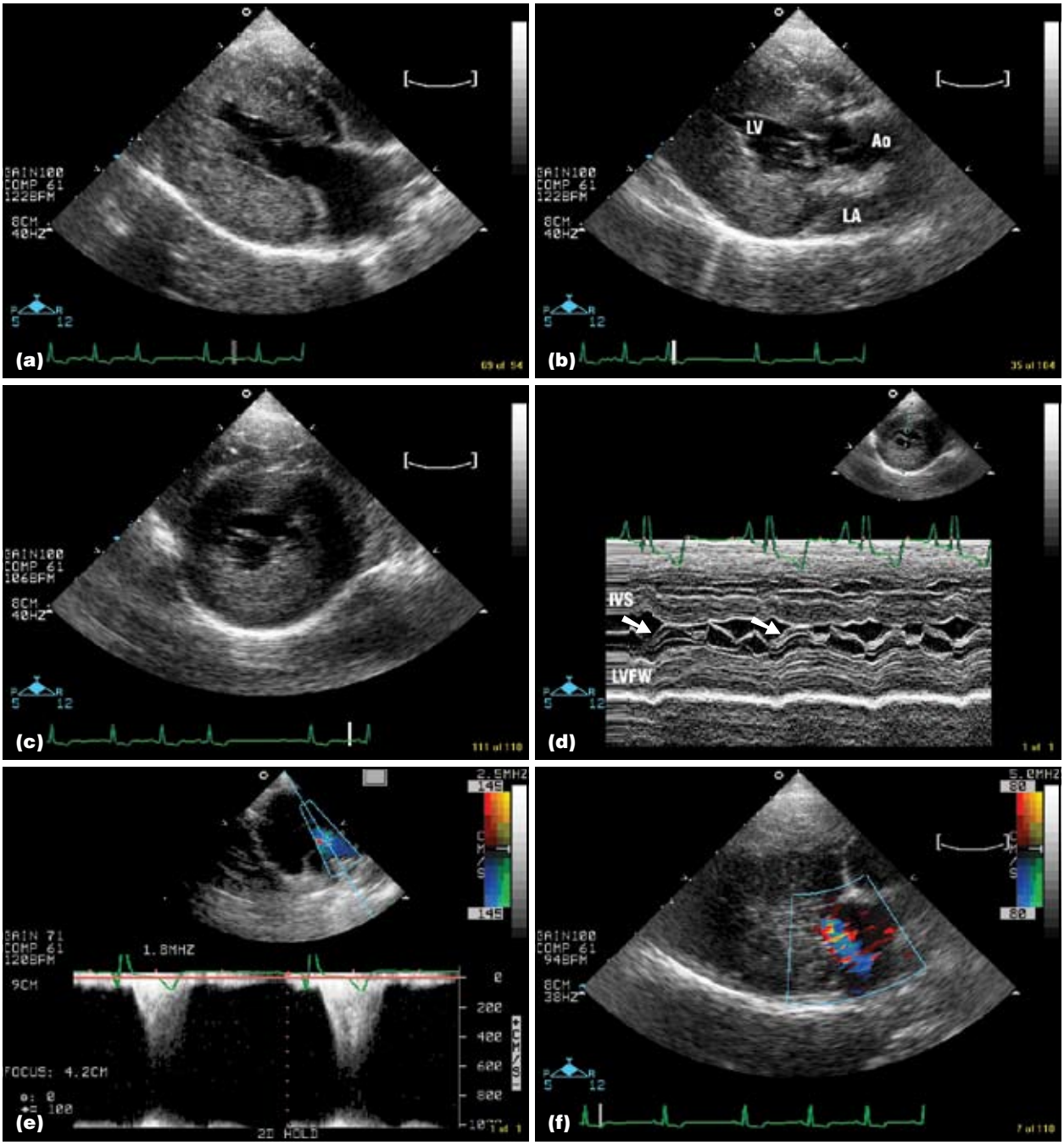
7.97 Simpson's rule to calculate LV volume from a RPS long-axis four-chamber view. Every attempt should be made to optimize the LV chamber length and area. The LV chamber endocardium is traced around, closing at the mitral annulus, in both diastole and systole. A length from the mitral annulus to the LV apex is drawn. The ultrasound machine software divides this length into 20 divisions, considered as discs. The volume of each disc is calculated, and the sum of volumes of the discs gives the overall LV volume, relatively independent of geometrical assumptions. Both **(a)** diastolic volume (EDV) and **(b)** systolic volume (ESV) are calculated. The ejection fraction is calculated as (EDV–ESV)/EDV (usually expressed as a percentage) (normal >50%). The dog's body surface area (BSA) can be calculated. The end-systolic volume index (ESVI) is the ESV/BSA (normal <30 ml/m^2). The ejection fraction is low and the ESVI is increased in DCM, as in this Boxer.



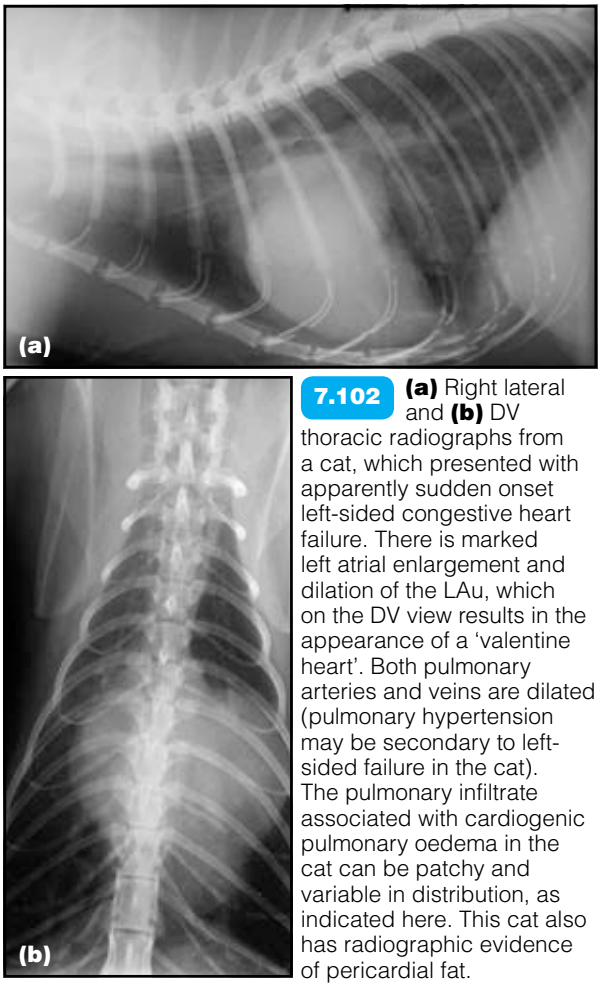
7.99 M-mode obtained at mitral valve level from a Leonberger with preclinical DCM. The anterior leaflet of the mitral valve (amv) moves towards the septum (IVS), and the posterior leaflet (pmv) moves towards the free wall of the LV (LVFW). In normal sinus rhythm, the amv opens twice in diastole: once early (E peak) corresponding to rapid LV filling, and once corresponding to atrial contraction (A peak). The E point to septal separation (EPSS) should not exceed 7 mm in any breed. It increases in DCM due to LV dilation and rounding, and also because of reduced stroke volume.



7.100 ARVC in a Boxer, which presented collapsed with ventricular tachycardia. The arrhythmia was treated. **(a)** RPS long-axis view and **(b)** short-axis view show dilation of the RA and RV. **(c)** Apical sternal RPS view shows dysplastic papillary muscles in the RV apex (arrowed). Ao = Aorta; LA = Left atrium; LV = Left ventricle; RA = Right atrium; RV = Right ventricle.

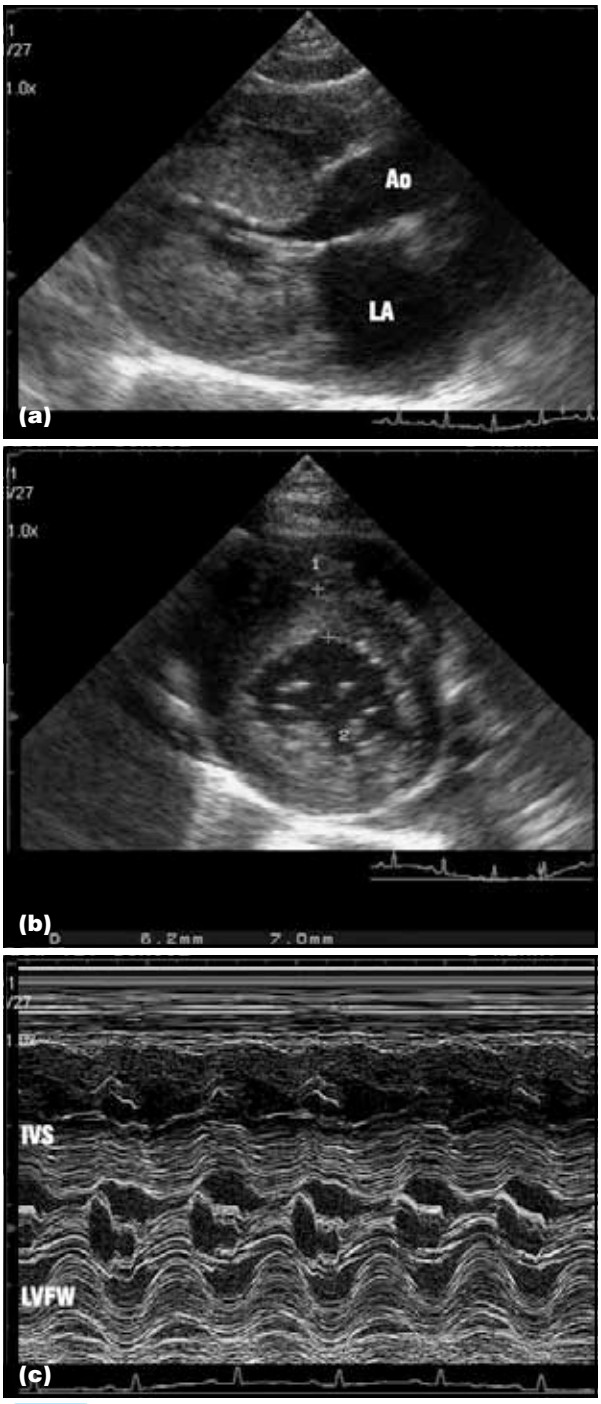


7.101 Images from a 2-year-old Border Terrier with frequent syncopal episodes on excitement and a heart murmur. A diagnosis was made of HOCM. There is marked concentric hypertrophy of the LV, evident from **(a)** the RPS long-axis four-chamber view, **(b)** the five-chamber view and **(c)** the short-axis view. There is no evidence of gross abnormality of the aortic valves, LVOT or the ascending aorta on the five-chamber view. However, the mitral valve anterior leaflet (*) in this early systolic frame is shown moving towards the basal septum (where endocardial thickening may be consistent with a 'kissing' lesion). **(d)** The presence of systolic anterior motion (SAM) of the anterior mitral valve leaflet (**arrowed**) is confirmed by the superior temporal resolution of M-mode at mitral valve level. SAM results in dynamic LVOT obstruction. **(e)** This can be documented from a left apical view. There is increased aortic outflow velocity with a biphasic acceleration slope. In this example, CW Doppler shows aortic peak velocities to be >6 m/s. **(f)** SAM also results in mitral valve incompetence and colour flow Doppler typically shows an eccentric MR jet, coursing towards the posterior-lateral wall of the LA. Ao = Aorta; IVS = Interventricular septum; LA = Left atrium; LV = Left ventricle; LVFW = Left ventricular free wall. **[au: please can you add the * and arrow to parts c and d, respectively – thanks]**



7.102 **(a)** Right lateral and **(b)** DV thoracic radiographs from a cat, which presented with apparently sudden onset left-sided congestive heart failure. There is marked left atrial enlargement and dilation of the LAu, which on the DV view results in the appearance of a 'valentine heart'. Both pulmonary arteries and veins are dilated (pulmonary hypertension may be secondary to left-sided failure in the cat). The pulmonary infiltrate associated with cardiogenic pulmonary oedema in the cat can be patchy and variable in distribution, as indicated here. This cat also has radiographic evidence of pericardial fat.

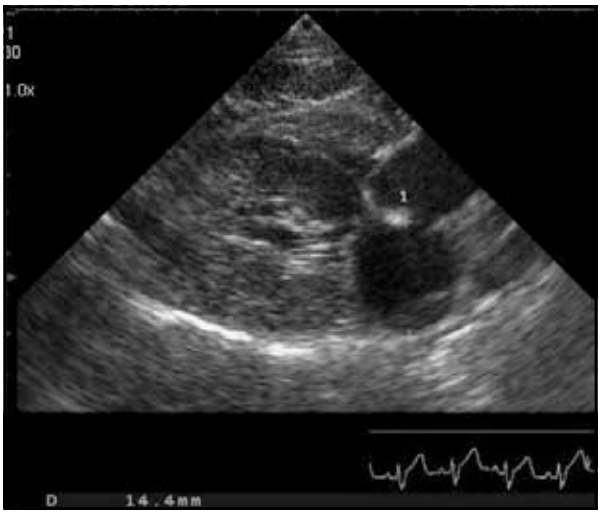
FIGURE 7.103 FOLLOWS >>>>>



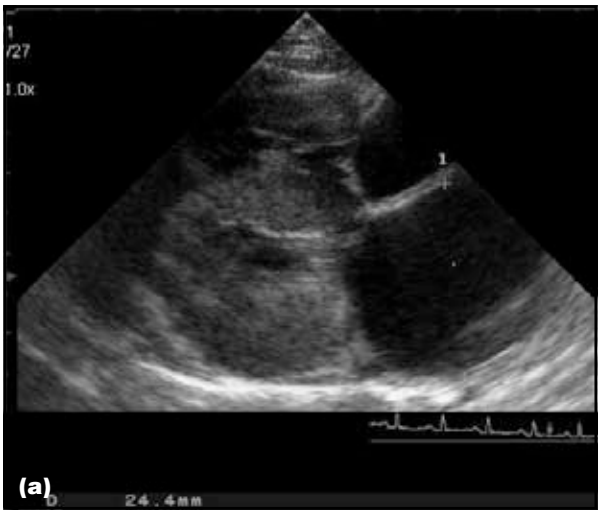
7.104 Images from a 2-year-old British Shorthair cat with HCM. There is marked concentric hypertrophy of the LV. **(a)** The basal septal bulge may result in turbulence or increased velocity of LV outflow. If the basal septal bulge exceeds 6 mm in diastole, this confirms the presence of significant hypertrophy. **(b)** A short-axis view can be used to measure wall thickness at the chordal level, ensuring that papillary muscles are not inadvertently included. Diastolic wall thicknesses over 6 mm confirm hypertrophy, as here. **(c)** The M-mode in feline HCM typically has a cluttered appearance, particularly at mitral valve level. Ao = Aorta; IVS = Interventricular septum; LA = Left atrium; LVFW = Left ventricular free wall.

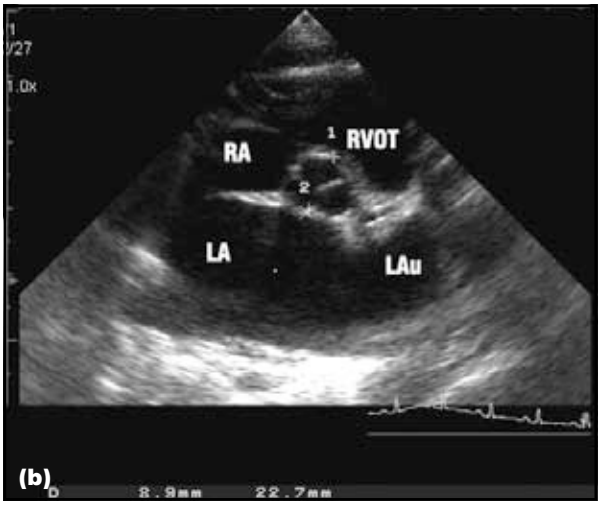
Parameter	Normal	HCM	RCM	DCM
Interventricular septum diastole (IVSd)	3.5–5.0 mm	>6 mm	Normal or mild increase	Normal or reduced
LV free wall in diastole (LVFWd)	3.5–4.5 mm	>6 mm	Normal or mild increase	Normal or reduced
Left ventricular internal dimension in diastole (LVIDd)	14–15 mm	Normal or reduced	Normal or mild increase	>16 mm
Left ventricular internal dimension in systole (LVIDs)	7.0–8.5 mm	Normal or reduced	Normal or mild increase	>9 mm
Fractional shortening (FS)	30–50%	Normal or increased	Normal	<25%
Mitral M-mode	E point (on anterior leaflet of mitral valve) to septal separation (EPSS) <2 mm	Normal EPSS ± systolic anterior motion	Normal or mildly increased EPSS	Increased EPSS
Mitral E:A ratio	>1<2	Impaired relaxation: <1. May be pseudonormal or restrictive, depending on stage of disease	Restrictive filling pattern: E>>A	Normal or restrictive, depending on stage of the disease
E deceleration time	60 ms	>65 ms	<55 ms	Normal or decreased
Isovolumic relaxation time (IVRT)	55–60 ms	>60 ms	<55 ms	Normal or decreased
Pulmonary venous flow (PVF) S:D velocity ratio	<1 (unless aged cat)	S:D>1	S:D<<1	Low S (<0.2 m/s), S:D<<1
PVF Ar velocity	<0.2 m/s	Normal or increased	Normal, increased or decreased	Normal or decreased
PVF Ar duration:mitral A duration	<1	Normal or >1, depending on stage of disease	>1 in cats with chronic heart failure (CHF)	>1 in cats with CHF

7.103 Typical echocardiographic findings in feline myocardial disease based on 2D and M-mode criteria and assessment of diastolic function.



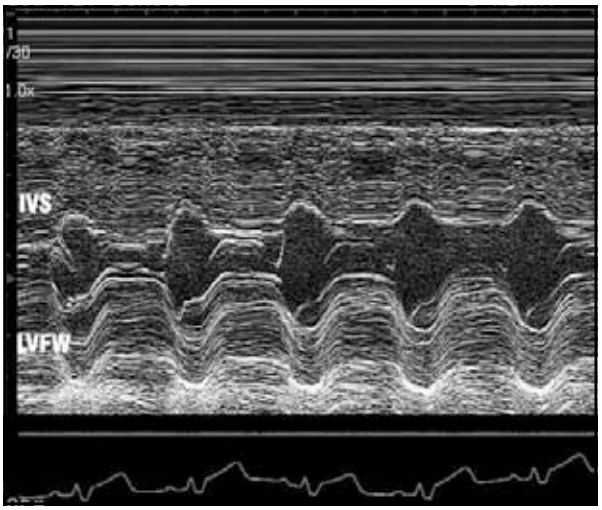
7.105 Left atrial dilation is a consequence of significant diastolic dysfunction and elevated filling pressures in the cat. In cats with asymptomatic HCM, the left atrial size may still be normal. From a RPS long-axis four-chamber view, the maximum width of the LA, parallel to the mitral annulus, can be measured (normal <16 mm).



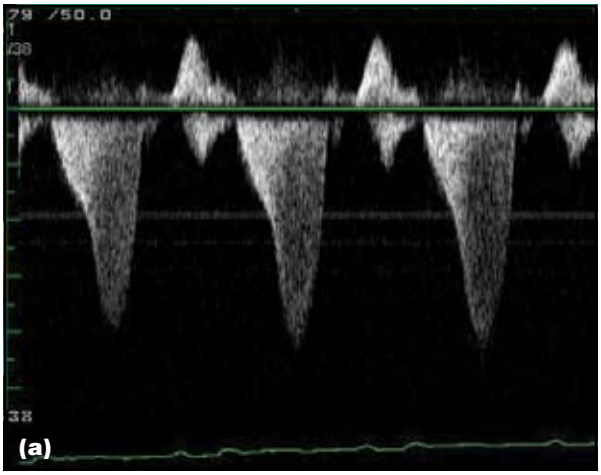


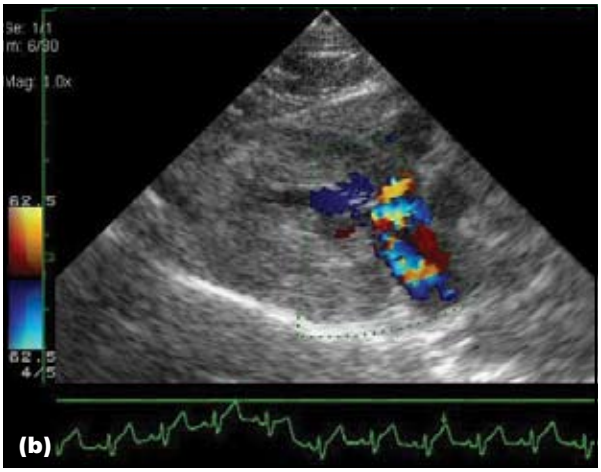
7.106 This is the same cat as in Figure 7.105. The LA is grossly dilated, both measured from **(a)** a RPS long-axis four-chamber view and **(b)** assessing the 2D short-axis LA:aortic root ratio in diastole. The ratio is 2.55 (normal <1.5). LA = Left atrium; LAu = Left auricular appendage; RA = Right atrium; RVOT = Right ventricular outflow tract.

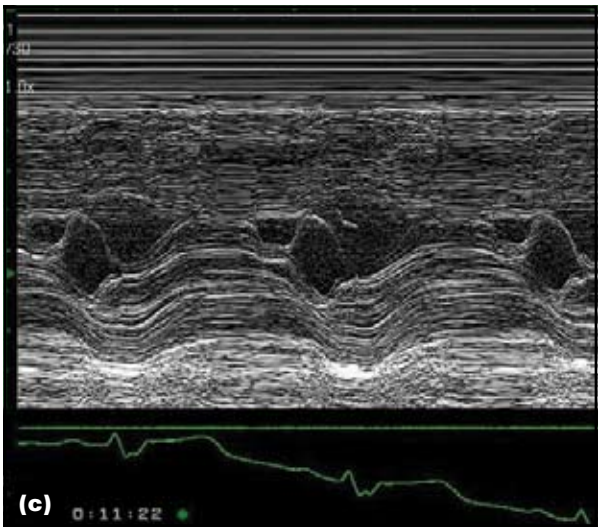
[au: Figures 7.105 and 7.106 are from the same cat but the text/captions state that they are supposed to show a normal LA and a dilated LA – how is this possible? Does the dilated LA appear later in the disease process? Do the captions need rewording to reflect this? Please could you clarify – thanks.]



7.107 M-mode of a cat with HCM, showing the concentric hypertrophy and the apparent hyperkinesis of the LV. IVS = Interventricular septum; LVFW = Left ventricular free wall.



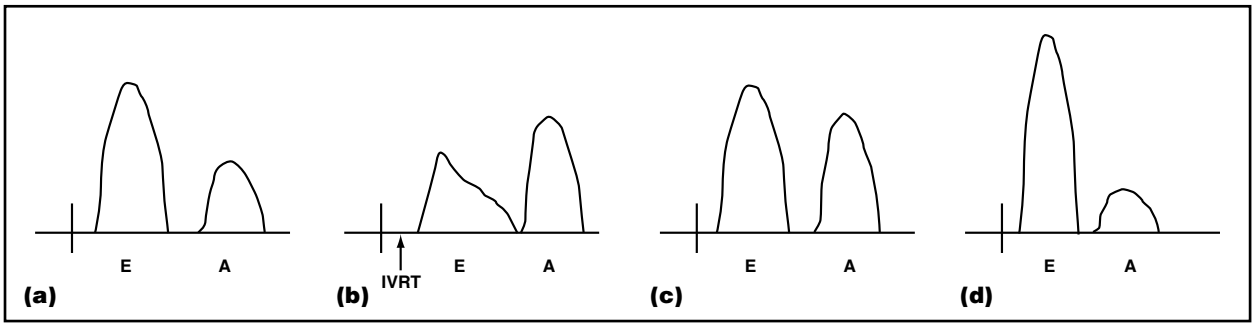




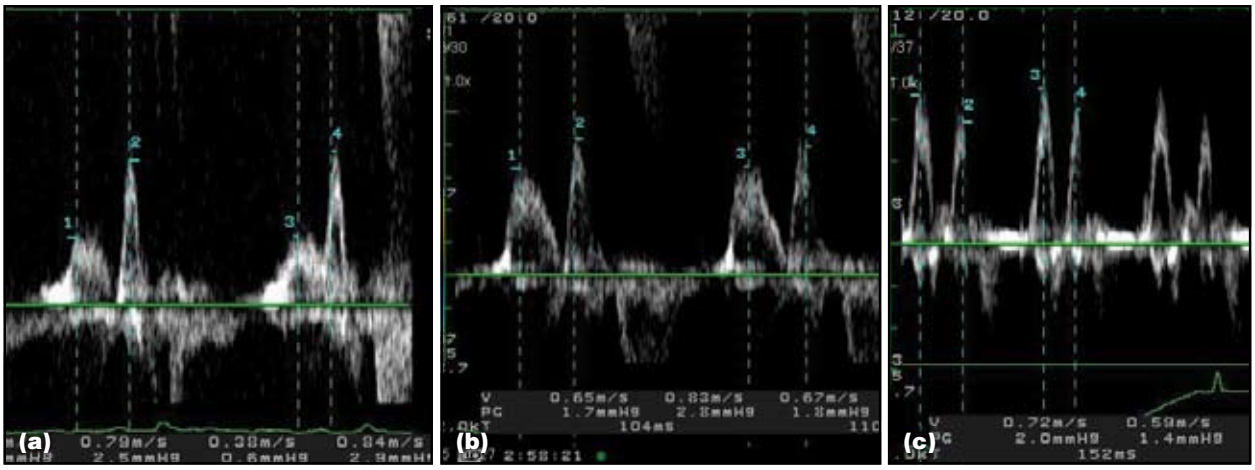
7.108 Evidence of dynamic LVOT obstruction due to systolic anterior motion (SAM) of the anterior mitral valve leaflet, leads to the diagnosis of HOCM. **(a)** There is increased and turbulent LVOT velocity, with a biphasic acceleration slope (so-called 'scimitar' shape); in this example the aortic outflow velocity is around 4 m/s. **(b)** RPS long-axis five-chamber view. The presence of SAM leads to mitral incompetence, with an eccentric mitral regurgitant jet coursing towards the posterior–lateral wall of the LA, as well as colour variance in the LVOT. **(c)** SAM is best confirmed by mitral M-mode (*).
[au: please add a * to part c – thanks]

Condition	Mitral inflow	IVRT	PVF
Asymptomatic HCM. Abnormal LV relaxation	• E:A <1 • Prolonged E deceleration time (>65 ms)	Long IVRT (>60 ms)	• S>D • Increased Ar
Progressive increase in LA–LV PG (increased filling pressures), so E wave velocity increases and exceeds A (pseudonormalization)	• E:A >1 • E deceleration time normal or prolonged	IVRT long or normal	• S>D • Usually increased Ar velocity
With disease progression, LA pressure increases further, and the LV may become less compliant. Results in restrictive physiology	• E:A >2 • E deceleration time short (<55 ms)	IVRT short (<55 ms)	• Low S • Increased D • Ar can be normal if atrial function preserved or low velocity • Prolonged Ar duration (greater than mitral A duration)

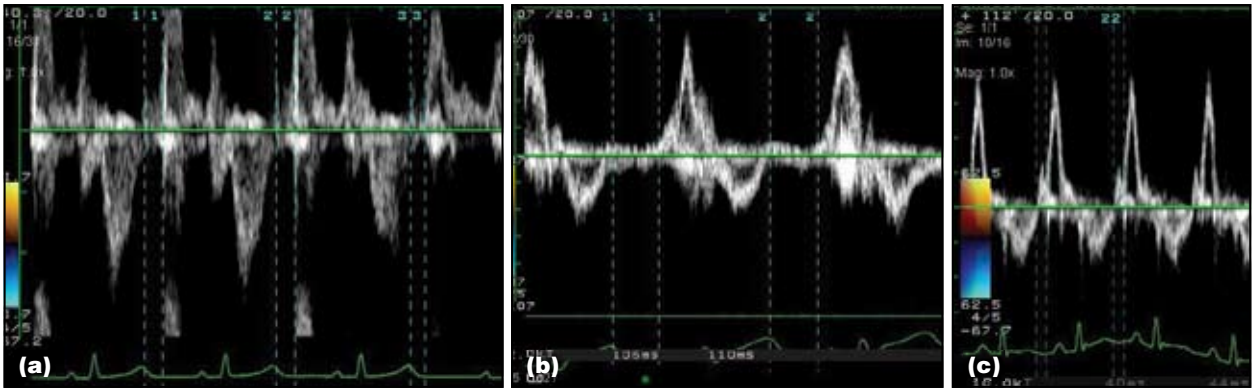
7.109 Criteria for the classification of diastolic abnormalities recognized in feline myocardial disease, such as HCM. Note the theoretical progression of disease from the first row downwards, as LA pressure and filling pressures increase and the LV becomes less compliant.



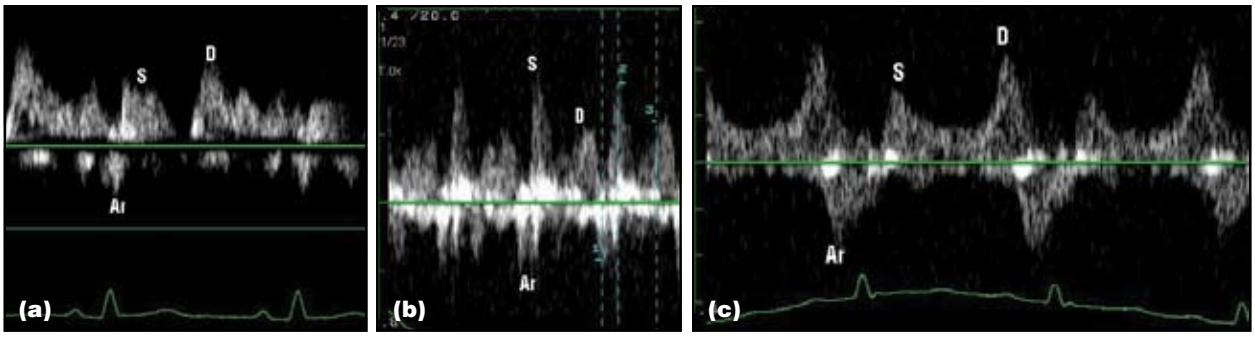
7.110 Mitral inflow patterns. **(a)** Normal mitral inflow with E wave velocity <2 but >1 × A wave velocity. **(b)** Abnormal relaxation. As active relaxation (lusitropy) of the LV is compromised, E wave velocity is reduced and E wave deceleration time is prolonged. IVRT is prolonged. Atrial contraction is important to achieve ventricular filling. A>E wave velocities. **(c)** With evolution of the disease, left atrial pressures increase and E wave velocity increases, giving a relatively normal E:A ratio again (pseudonormalization). **(d)** Further worsening of the disease, with high left atrial pressure and a stiff, poorly compliant LV, can result in a high E wave velocity, short E wave deceleration time and E:A velocity ratio of >2 (restrictive filling pattern).



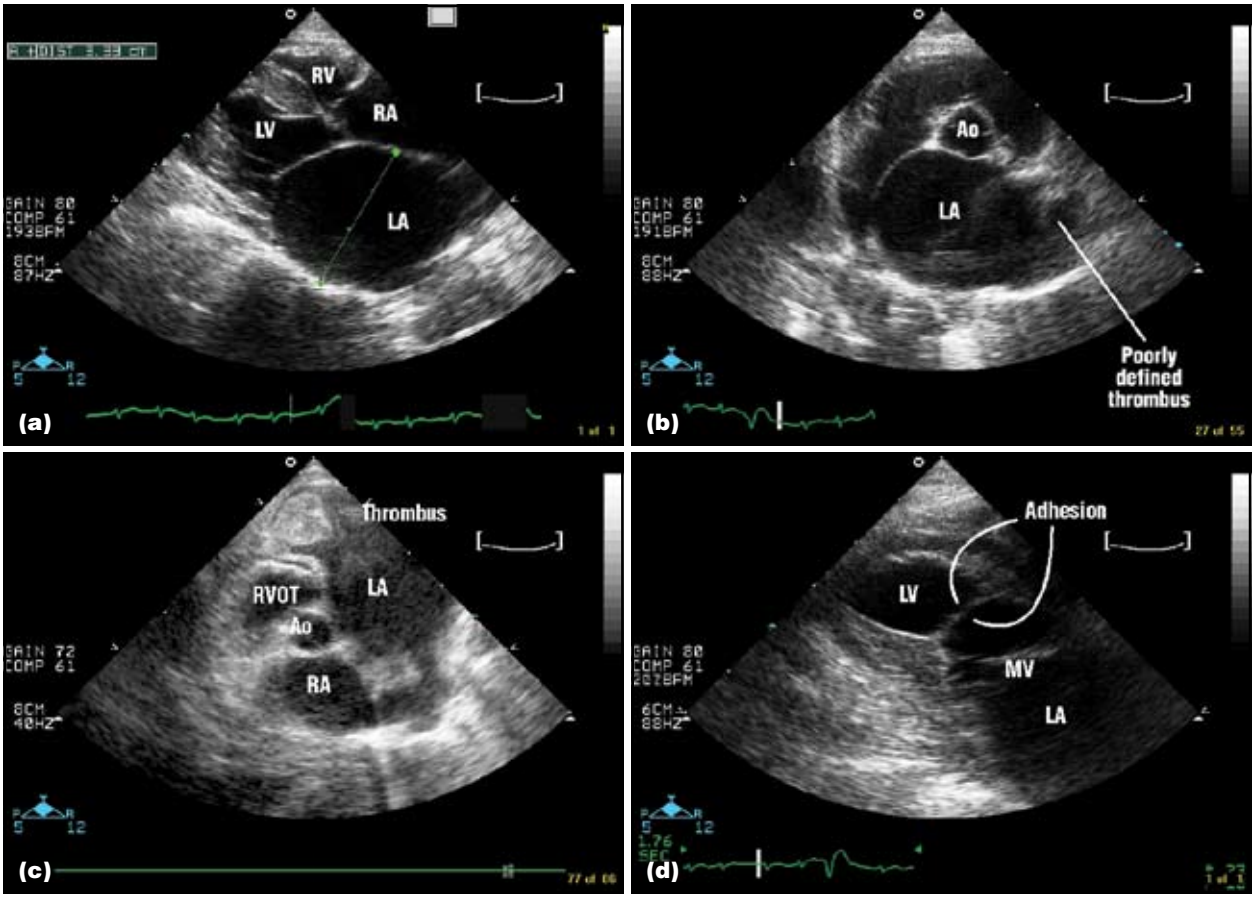
7.111 Mitral inflow patterns from cats with myocardial disease. **(a)** A cat with HOCM, which presented for investigation of an asymptomatic heart murmur. Note the abnormal relaxation pattern (E<A; prolonged E deceleration time). **(b)** Re-evaluation 6 months later. The cat was still asymptomatic but there is evidence of increased LA pressure; the E wave velocity is increased, although, there is still E:A reversal and evidence of abnormal relaxation. **(c)** A cat with pseudonormalization. Note that although the LA pressures have increased, the abnormal relaxation is now masked, and other methods are required to document the diastolic dysfunction. **(d)** A cat with severe biventricular failure and a RCM. A restrictive filling pattern with probable LA dysfunction is shown, with E>>A, low velocity A wave and short E wave deceleration time.



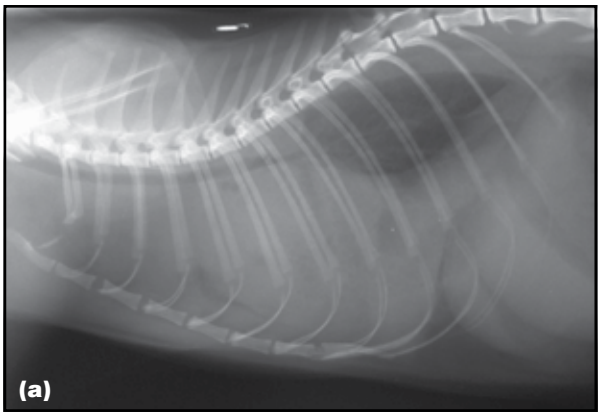
7.112 Measurement of IVRT in cats with myocardial disease. **(a)** Normal cat, with time measurement from aortic valve closure to onset of mitral flow. **(b)** A cat with abnormal relaxation time, showing increased IVRT (>65 ms). **(c)** A cat with a restrictive filling time, showing very short IVRT (<55 ms).

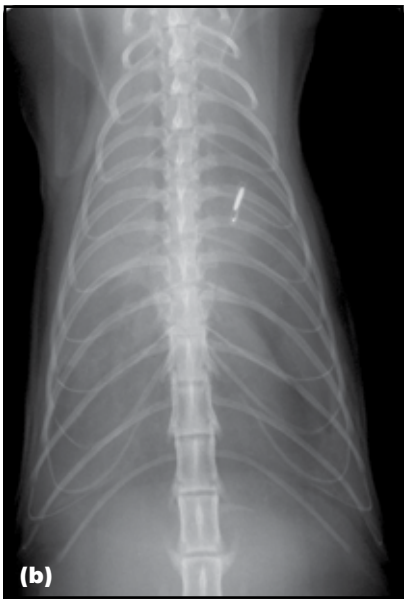


7.113 Assessment of PVF pattern in cats with myocardial disease. **(a)** Normal, middle-aged cat with D>S. **(b)** A cat with abnormal relaxation, with increased S wave (measurement 2) and lower D wave (measurement 3) velocity, but increased velocity of the Ar wave (measurement 1). **(c)** A cat with a restrictive filling pattern, with normal atrial function (increased Ar wave). D wave velocity exceeds S, and D deceleration is rapid.

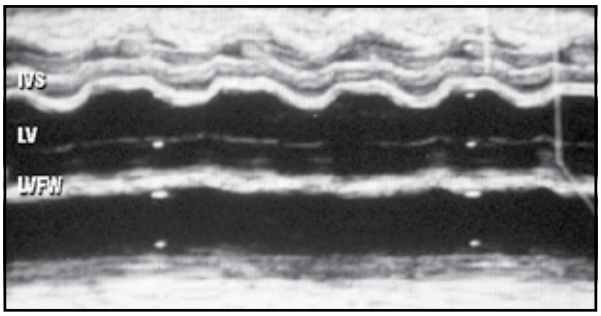


7.114 Marked left atrial enlargement is apparent in this example of a RCM, both from **(a)** the RPS long-axis four-chamber view (33 mm diameter) and **(b)** the RPS short-axis view at the level of the aortic valves. In the short-axis view, there is a poorly defined thrombus in the LAu with a real-time image showing spontaneous echocontrast in the LA. This cat had atrial fibrillation with occasional ventricular premature complexes. **(c)** A modified LPS cranial view, optimized for the LAu. The thrombus in the LAu can be seen with spontaneous echocontrast appearing as 'smoke', swirling in the junction of the LAu and LA. **(d)** RPS four-chamber view of the LV showing an irregular endocardium, particularly on the septum, and a probable adhesion crossing the LV chamber. Ao = Aorta; LA = Left atrium; LV = Left ventricle; MV = Mitral valve; RA = Right atrium; RV = Right ventricle; RVOT = Right ventricular outflow tract.

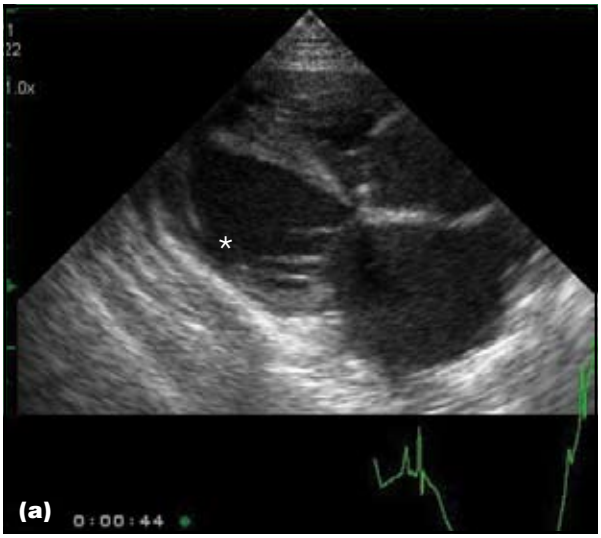


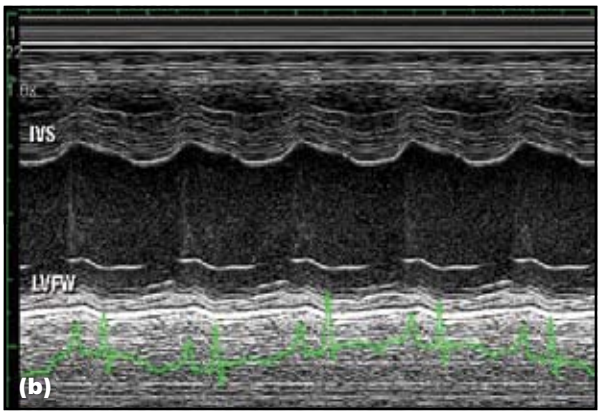


7.115 **(a)** Right lateral and **(b)** DV thoracic radiographs from a cat with DCM. There is a significant pleural effusion, masking detail of the cardiac silhouette and the lung field. From the degree of tracheal elevation, the cardiac silhouette appears to show marked generalized enlargement.

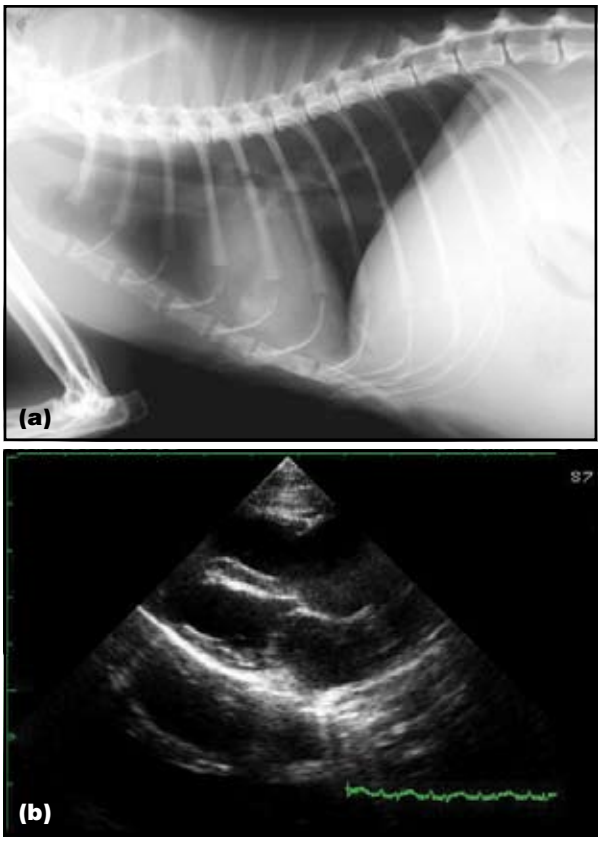


7.116 M-mode of a cat with DCM from 1988, showing a pleural effusion and marked hypokinesis, especially of the left ventricular free wall (LVFW). IVS = Interventricular septum; LV = Left ventricle.

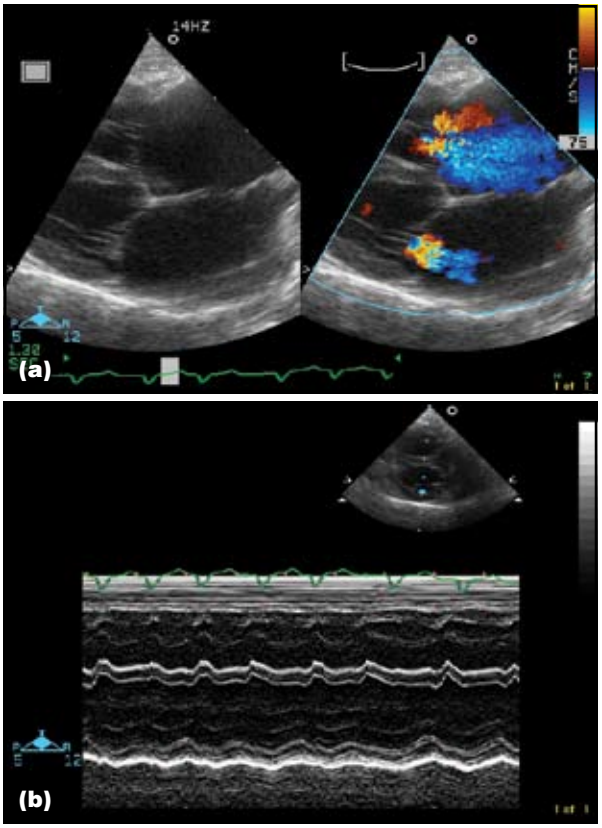




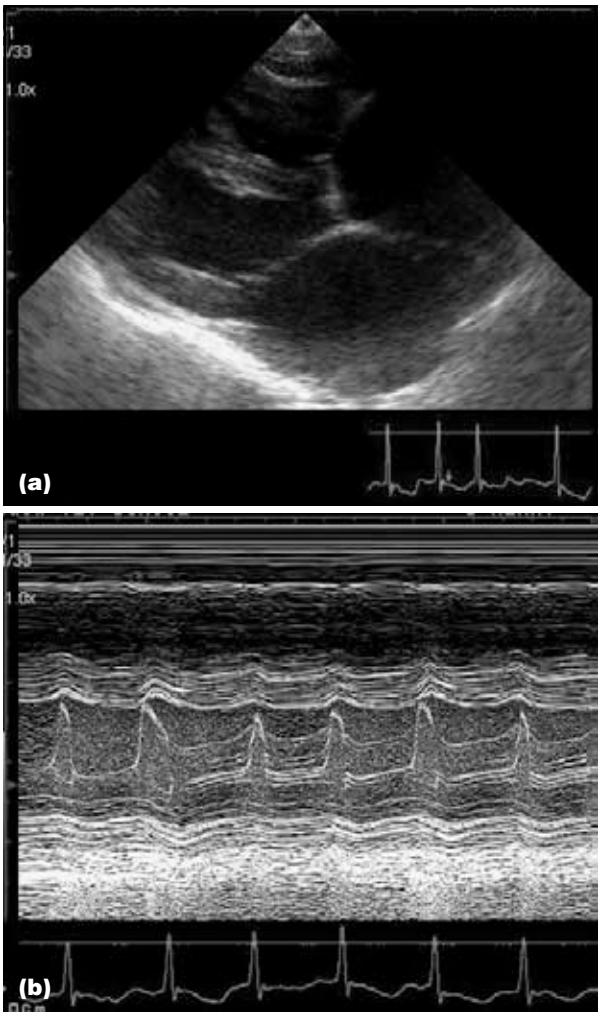
7.117 Images from an elderly cat with untreated, probably long-standing, hyperthyroidism. **(a)** The RPS long-axis four-chamber view shows four-chamber dilation. **(b)** In real time, the LV was noticeably hypokinetic, which is indicated on the M-mode. Notice that the left ventricular free wall (LVFW) is not functioning on the M-mode, and the wall is thin. A segmental region of posterior wall thinning, corresponding to the M-mode sampling, is shown in (a) (*). This may correspond to a myocardial infarct (although postmortem confirmation was not achieved in this cat). IVS = Interventricular septum.



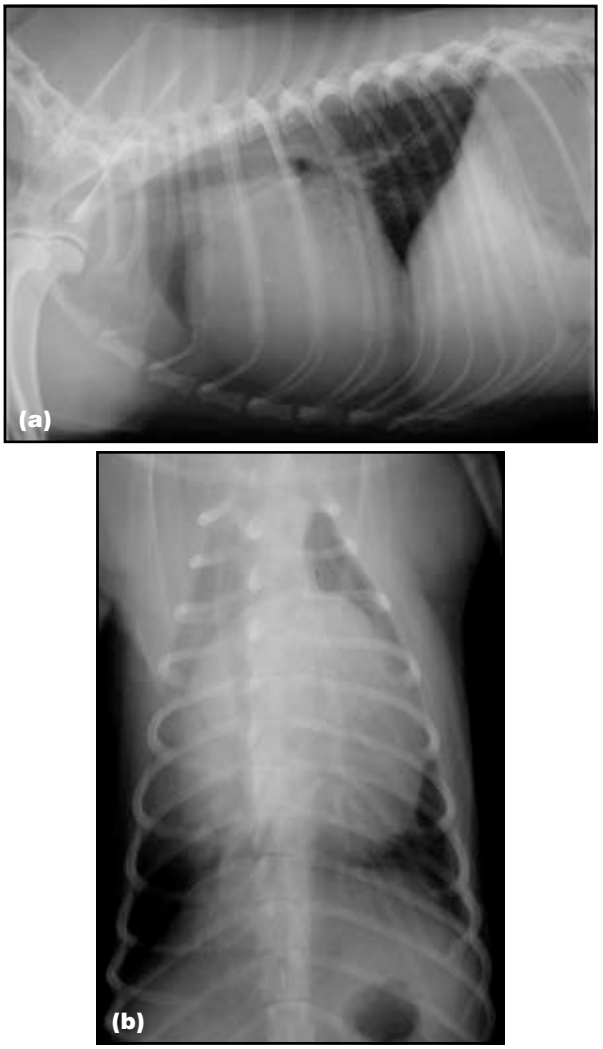
7.118 **(a)** Right lateral radiograph from a cat later confirmed to have ARVC. Ascites with the dilated CdVC indicates radiographic support for right-sided congestive heart failure. From this view (no DV available) there appears to be predominantly right-sided enlargement, resulting in cardiomegaly. **(b)** RPS long-axis four-chamber view, indicating marked right atrial and right ventricular enlargement.



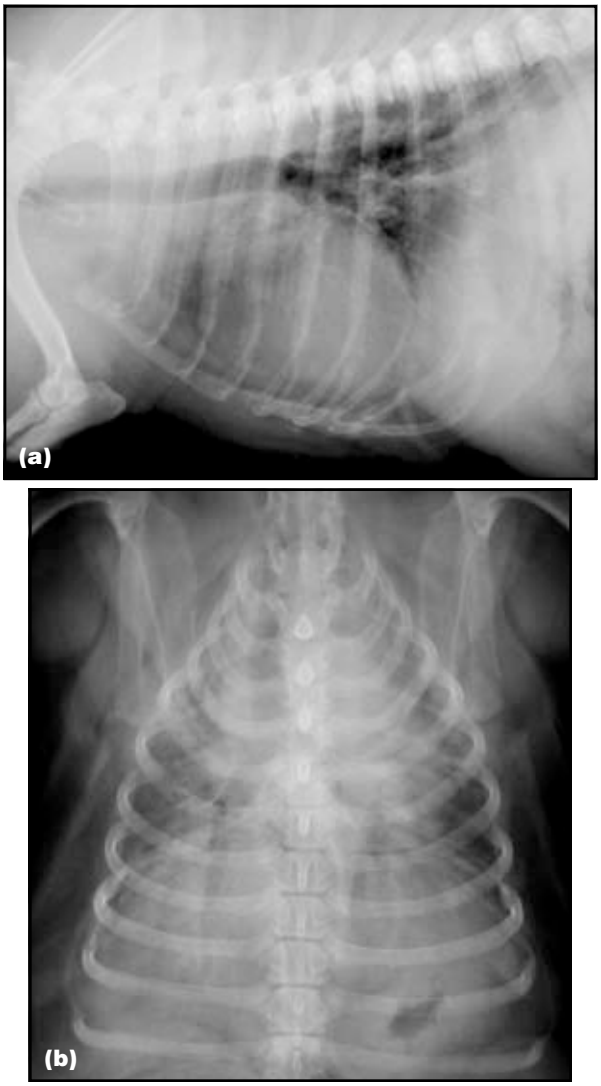
7.119 **(a)** RPS long-axis four-chamber view of a cat with ARVC. Colour flow mapping indicates the presence of tricuspid and mitral regurgitation due to stretch of the atrioventricular annuli, secondary to myocardial disease, although the RV is predominantly affected. **(b)** M-mode, showing dilation of both ventricles and impaired LV systolic function. This cat was in atrial fibrillation.



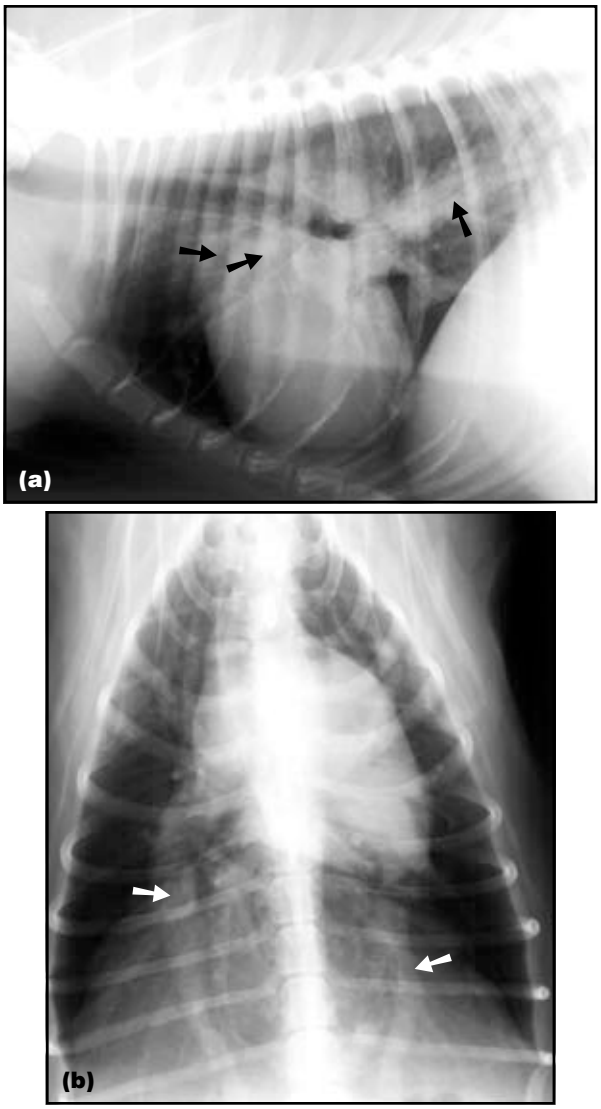
7.120 **(a)** The RPS long-axis four-chamber view and **(b)** M-mode, including the mitral valve, show mild chamber dilation and impaired LV systolic function. There is also a restrictive filling pattern. This cat was categorized as having an UCM. This cat was also in atrial fibrillation.



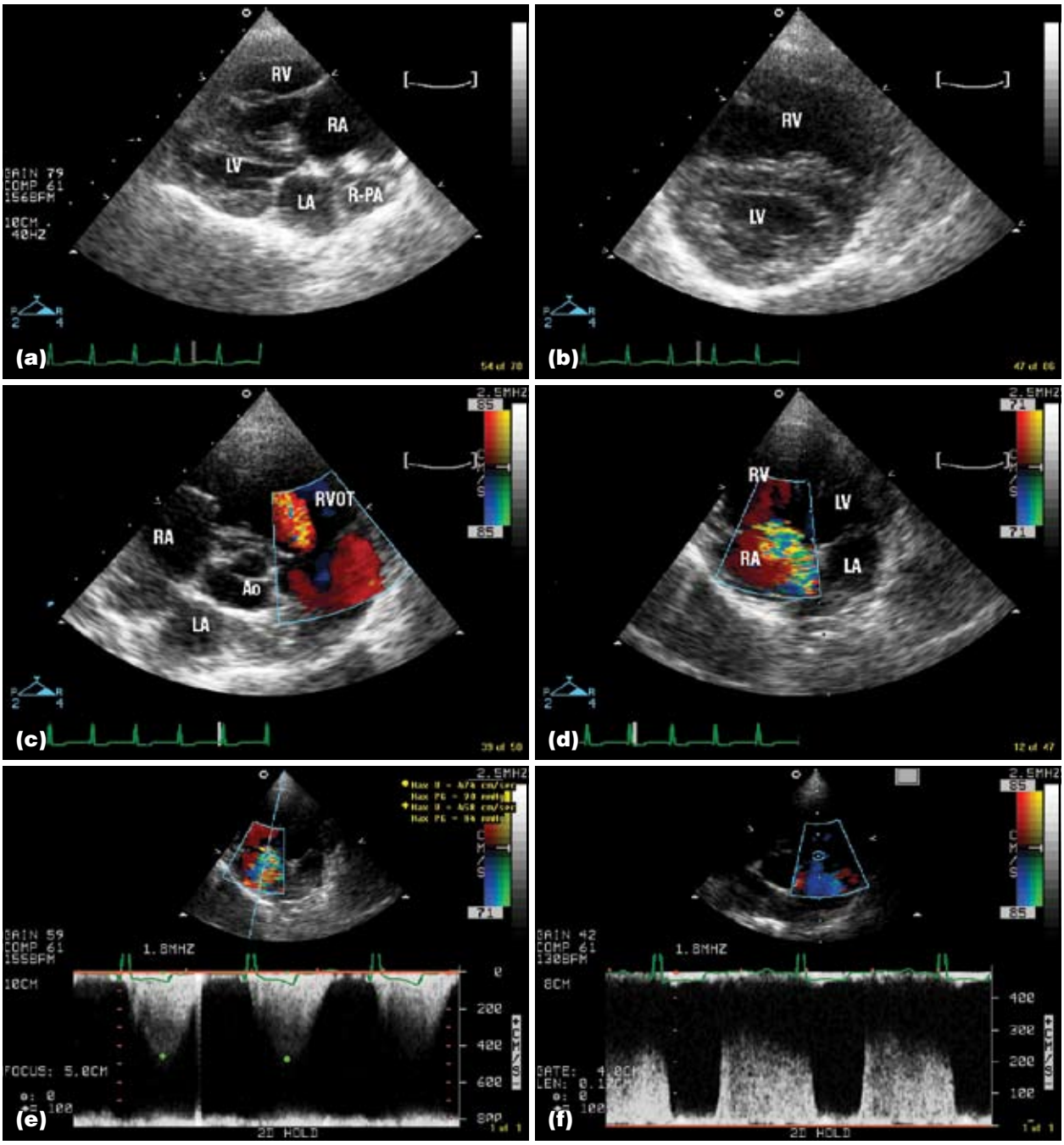
7.121 **(a)** Right lateral and **(b)** DV thoracic radiographs from a Cavalier King Charles Spaniel with presumed idiopathic (essential) pulmonary hypertension, as other common causes of pulmonary hypertension had been actively excluded. There is marked right-sided enlargement, resulting in generalized cardiomegaly. (The DV view is slightly rotated.)



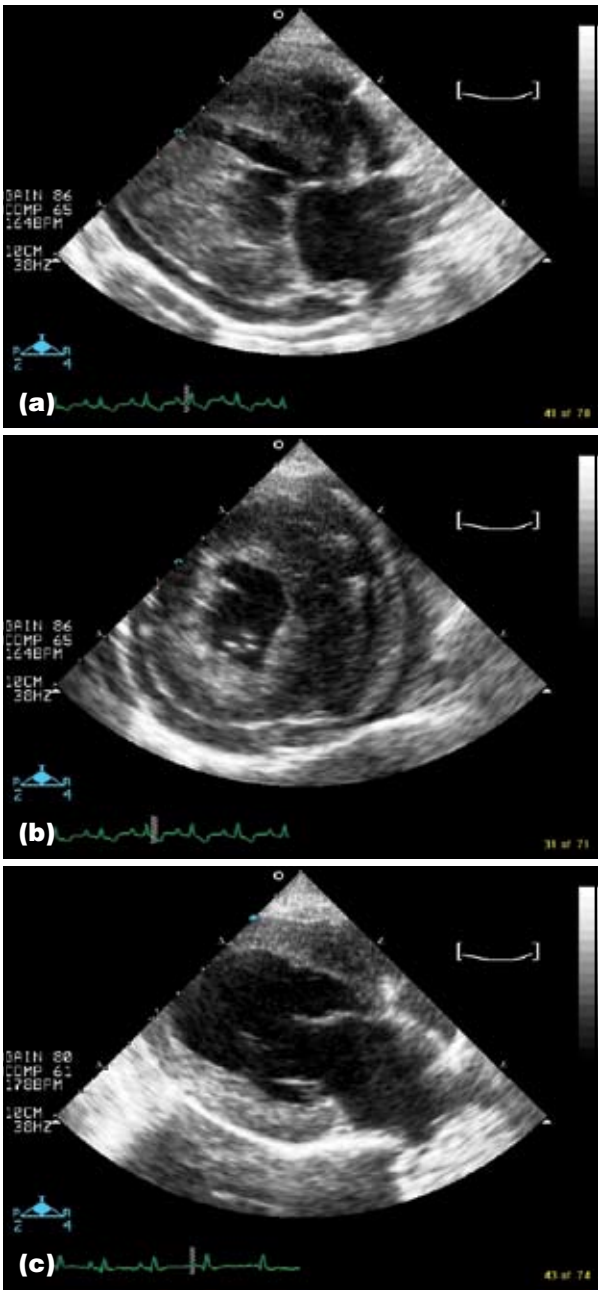
7.122 **(a)** Right lateral and **(b)** DV thoracic radiographs from a West Highland White Terrier with idiopathic pulmonary fibrosis and echocardiographically confirmed pulmonary hypertension. The radiographs show mild right heart enlargement. The cardiac silhouette and pulmonary vasculature are masked by a gross, generalized, interstitial opacity throughout the lung field.



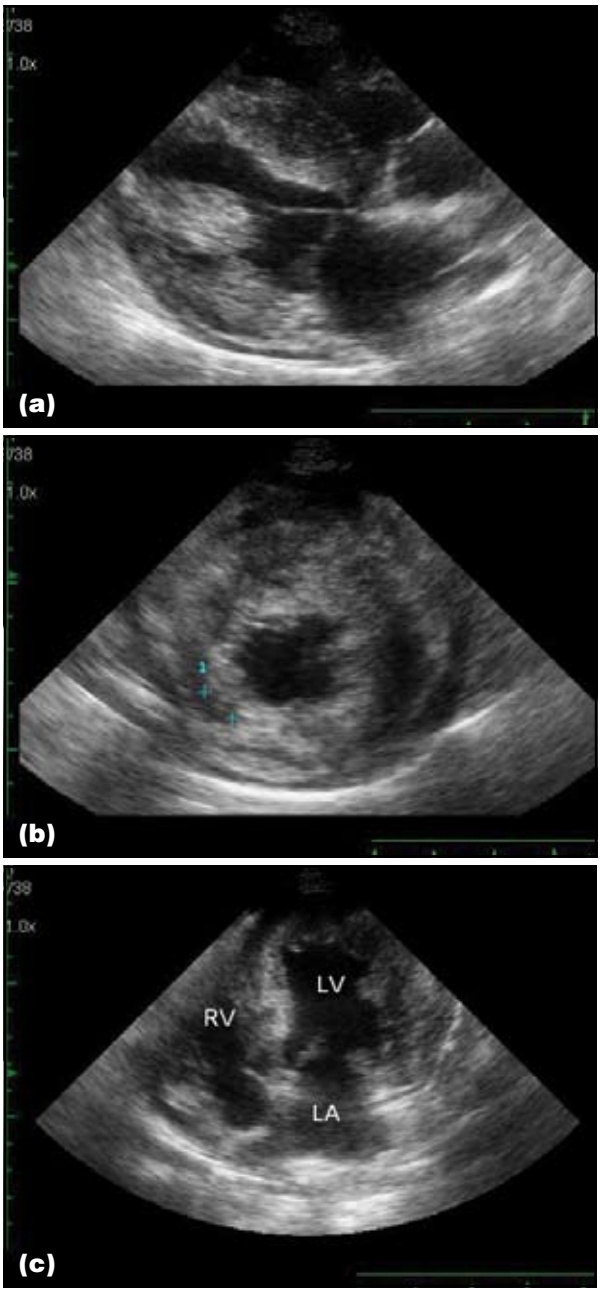
7.123 **(a)** Right lateral and **(b)** DV thoracic radiographs from a dog with *Dirofilaria immitis* infestation (heartworm). The pulmonary arteries are markedly dilated and tortuous (arrowed). (Courtesy of Professor M. Sullivan)

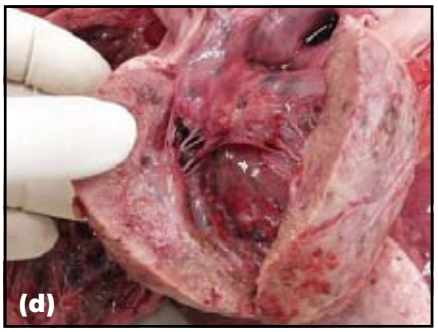


7.124 Images from a 5-year-old Cavalier King Charles Spaniel with a PDA associated with pulmonary hypertension, presented with ascites and other evidence of right-sided congestive heart failure. **(a)** RPS long-axis four-chamber view shows marked right atrial and right ventricular dilation, with concentric hypertrophy of the RV wall. The IVS is flattened, which is also appreciated on **(b)** the RPS short-axis view. The right branch of the pulmonary artery (R-PA) is also markedly dilated as it courses around the base of the heart. **(c)** The RPS cranial view shows the dilated pulmonary trunk with marked pulmonary insufficiency. **(d)** The left apical four-chamber view shows tricuspid regurgitation by colour flow Doppler. **(e)** Mean CW Doppler recorded velocity of the tricuspid regurgitant jet is 4.6 m/s. By the modified Bernouilli equation, there is a systolic PG (4v2) between the RV and RA of 84 mmHg. Therefore, pulmonary arterial systolic pressure is at least 90 mmHg (since normal right atrial pressure is about 6 mmHg). **(f)** The mean velocity of pulmonary regurgitation is 3.4 m/s (recorded from a RPS cranial view). Therefore, the diastolic PG between the main pulmonary artery and RV is 46 mmHg. Thus, the systolic/diastolic pulmonary pressures are estimated to be at least 90/46 mmHg (but this is an underestimate as the dog is in right-sided failure; RA and RV filling pressures will be increased). This has an impact on the PDA flow. There is some left-to-right diastolic flow but in systole, no flow was evident ('balanced' PDA as a consequence of the pulmonary hypertension). Ao = Aorta; LA = Left atrium; LV = Left ventricle; RA = Right atrium; RV = Right ventricle; RVOT = Right ventricular outflow tract.



7.125 **(a)** RPS long-axis four-chamber view and **(b)** short-axis view from a crossbred dog. The myocardium appears thickened but with a heterogenous patchy increased echogenicity. There is a small amount of pericardial effusion. This was sampled and cytology confirmed the diagnosis of cardiac lymphoma. **(c)** After 9 days of staged chemotherapy, reduction in the thickness of the walls could be seen. LA = Left atrium; LV = Left ventricle; RV = Right ventricle.



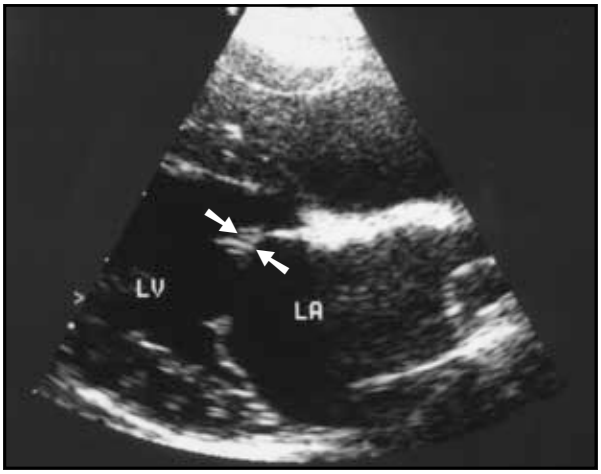


7.126 Images from an 11-year-old male Golden Retriever, which presented collapsed, anaemic and with paroxysmal ventricular tachycardia. **(a)** The RPS long-axis four-chamber view, **(b)** short-axis view and **(c)** left apical four-chamber view all show evidence of thickened myocardial walls, with disrupted architecture, and numerous hypoechoic lesions of various sizes. **(d)** Postmortem examination showed the entire myocardium was affected by a disseminated haemangiosarcoma. (Courtesy of Mr R. Irvine)

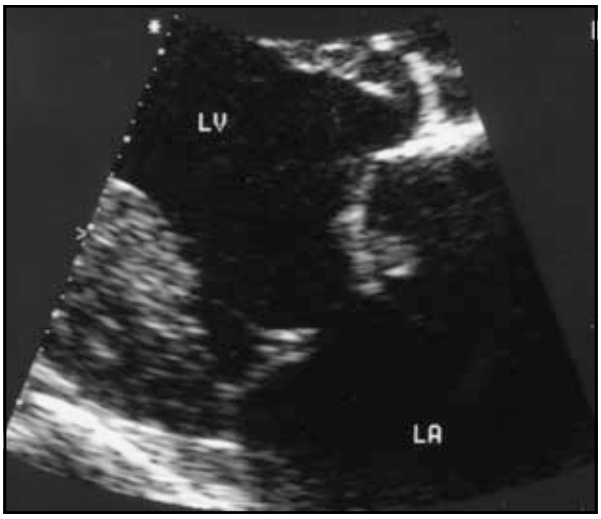
Mitral valve
Small regurgitant jet may be incidental finding
Myxomatous valvular degeneration
Endocarditis
Secondary to left ventricular enlargement
Widespread arteriosclerosis with myocardial infarcts
(Undiscovered congenital heart disease)
Tricuspid valve
Small regurgitant jet may be incidental finding
Myxomatous valvular degeneration
Main pulmonary artery hypertension
Secondary to right ventricular enlargement
Rarely endocarditis
(Undiscovered congenital heart disease)
Pulmonic valve
Low degree as a common incidental finding
Secondary to pulmonary artery hypertension
Secondary to pulmonary artery dilation (e.g. heartworm disease)
(Undiscovered congenital heart disease)
Aortic valve
Low degree as an occasional incidental finding
Endocarditis
Annuloaortic ectasia (idiopathic dilation of proximal aorta and aortic annulus) (very rare)
(Undiscovered congenital heart disease)

7.127 Differential diagnoses for acquired valvular regurgitation.

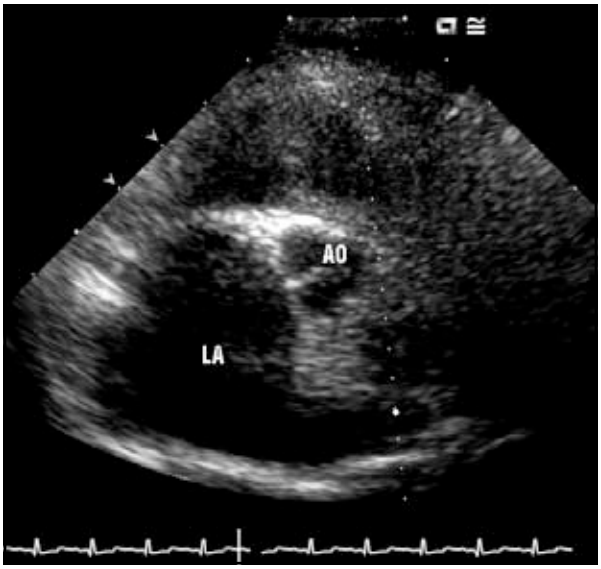
FIGURE 7.128 FOLLOWS >>>>



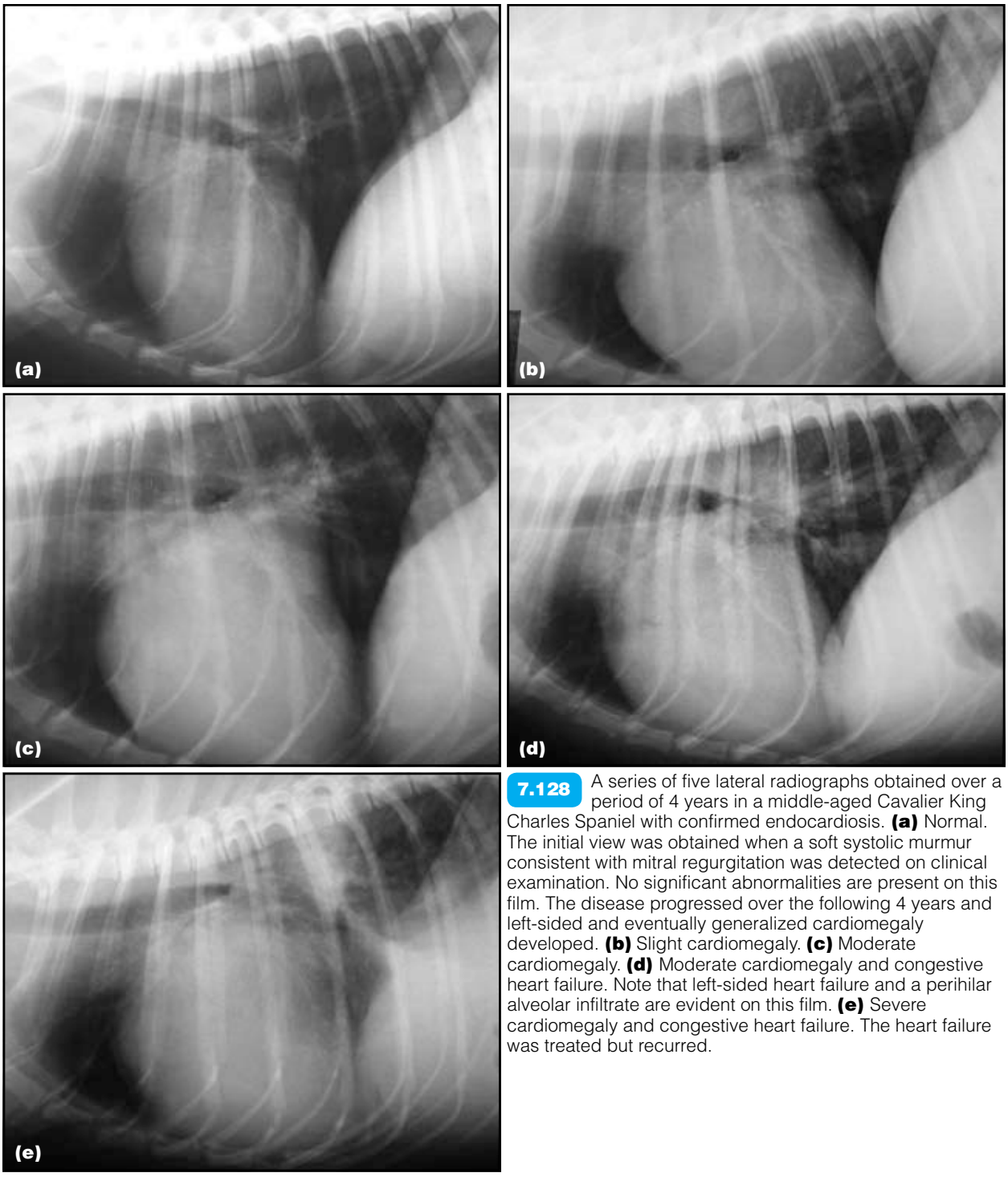
7.129 RPS long-axis echocardiogram, optimized for visualization of the mitral valve in a dog with myxomatous mitral valve degeneration. A thickened region is present at the tips of both valve leaflets creating a 'club-like' appearance (arrows show the thickened region on the septal leaflet).



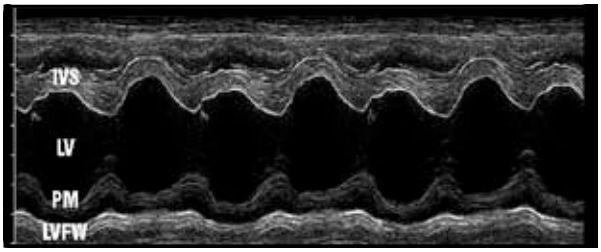
7.130 A flail leaflet is seen in this dog with myxomatous mitral valve degeneration. Note that the septal leaflet is displaced above the level of the valve annulus. This image was acquired during systole. LA = Left atrium; LV = Left ventricle.



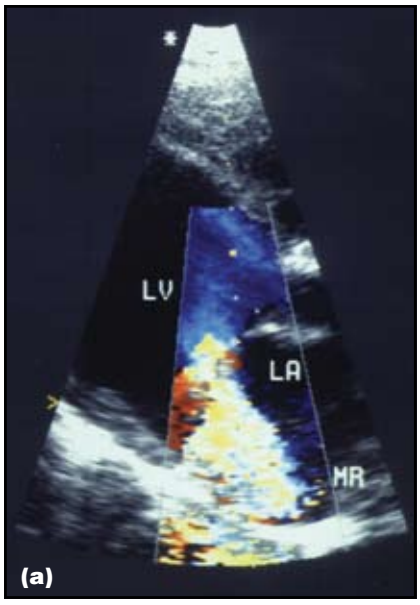
7.131 RPS short-axis view at the heart base in a dog with myxomatous mitral valve degeneration. Normally the ratio of left atrium (LA) to aortic (AO) diameter at this level should be no greater than 1.5:1; in this case the atrium is moderately enlarged. The size of the LA is an important indicator of disease severity.

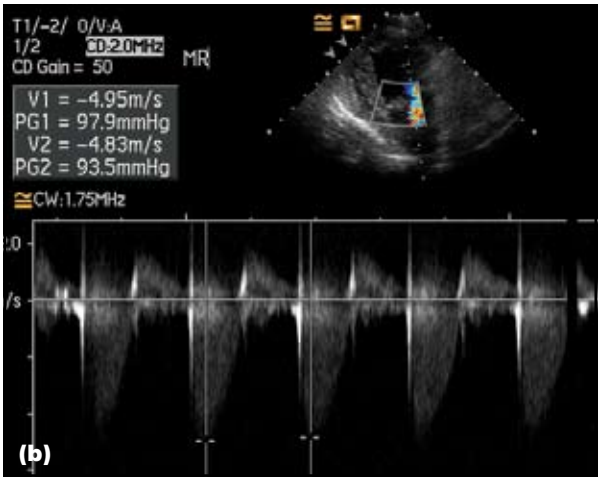


7.128 A series of five lateral radiographs obtained over a period of 4 years in a middle-aged Cavalier King Charles Spaniel with confirmed endocardiosis. **(a)** Normal. The initial view was obtained when a soft systolic murmur consistent with mitral regurgitation was detected on clinical examination. No significant abnormalities are present on this film. The disease progressed over the following 4 years and left-sided and eventually generalized cardiomegaly developed. **(b)** Slight cardiomegaly. **(c)** Moderate cardiomegaly. **(d)** Moderate cardiomegaly and congestive heart failure. Note that left-sided heart failure and a perihilar alveolar infiltrate are evident on this film. **(e)** Severe cardiomegaly and congestive heart failure. The heart failure was treated but recurred.

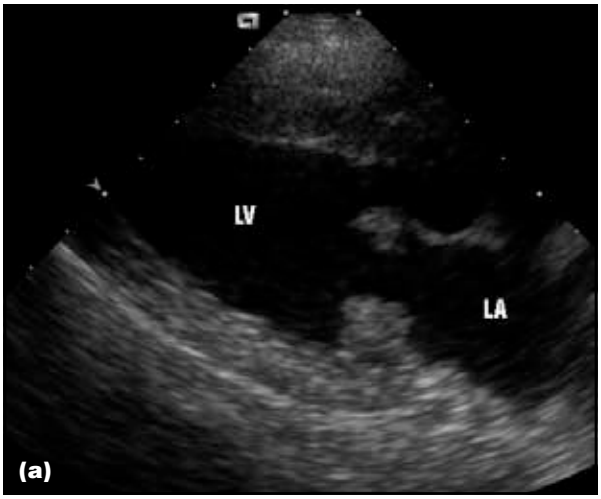


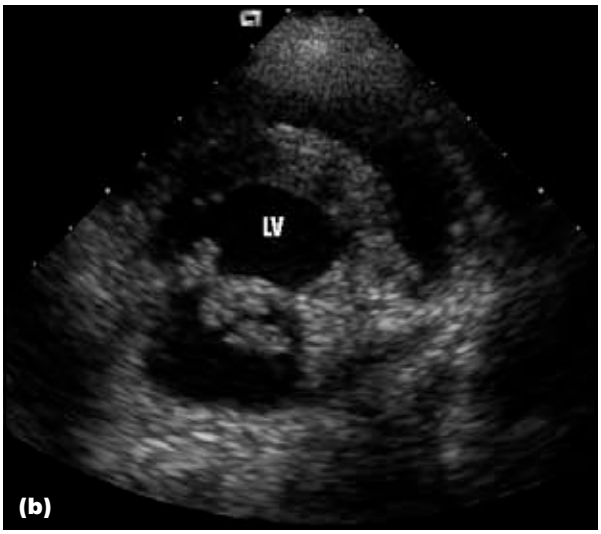
7.132 An M-mode echocardiogram obtained from a RPS short-axis view at the level of the papillary muscles. Note the hyperkinetic motion of the IVS, secondary to the rapid regurgitation of left ventricular blood into the low pressure LA during systole.
IVS = Interventricular septum; LV = Left ventricle; LVFW = Left ventricular free wall; PM = Edge of papillary muscle that was included in the image.



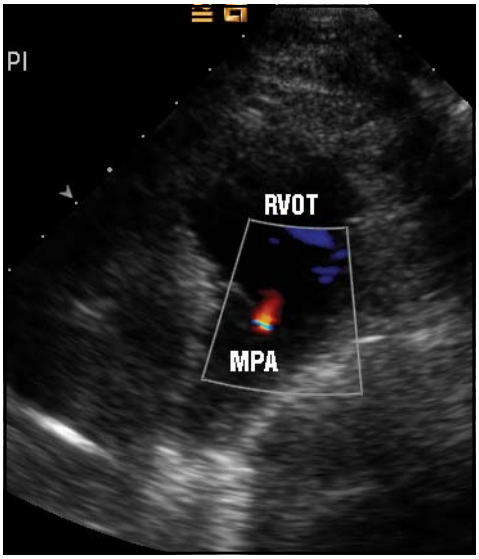


7.133 Colour and spectral Doppler echocardiograms of two dogs with myxomatous mitral valve disease. **(a)** A large jet of mitral regurgitation, extending to the wall of the LA. **(b)** CW spectral Doppler tracing from a dog with mitral regurgitation. The cursor was placed in the mitral regurgitant jet; it measures up to 4.95 m/s.
LA = Left atrium; LV = Left ventricle; MR = Mitral regurgitation.

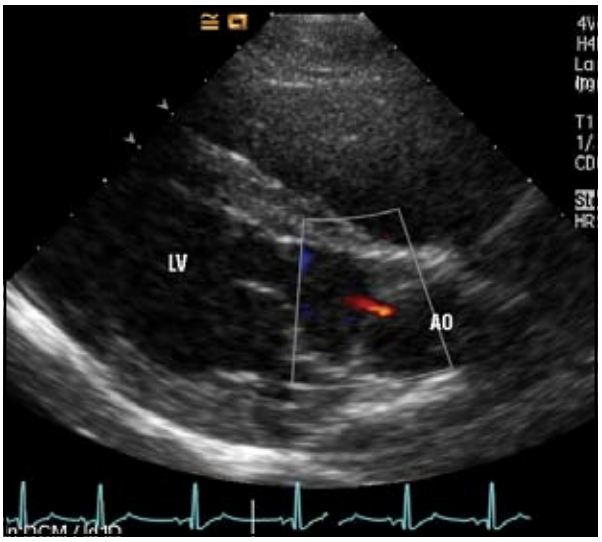




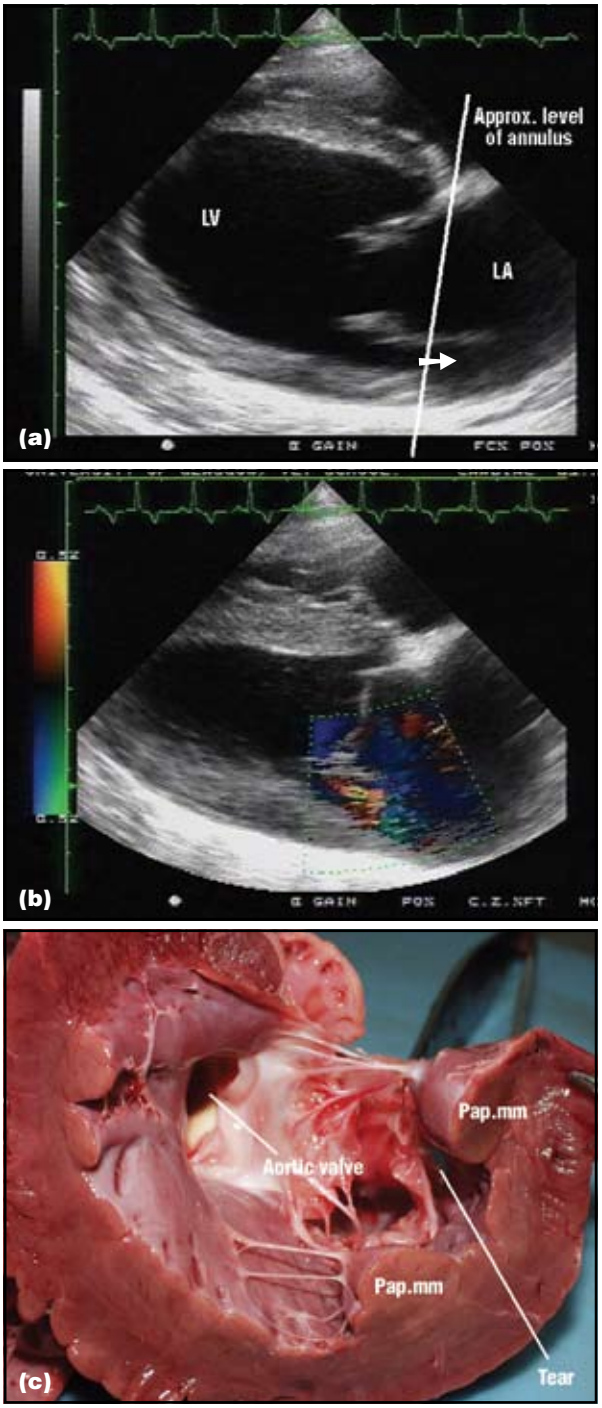
7.134 **(a)** Long-axis and **(b)** short-axis RPS views of a mixed breed dog with bacterial endocarditis of the mitral valve, secondary to a snake bite. Large florid masses are present on the valve leaflets. LA = Left atrium; LV = Left ventricle.



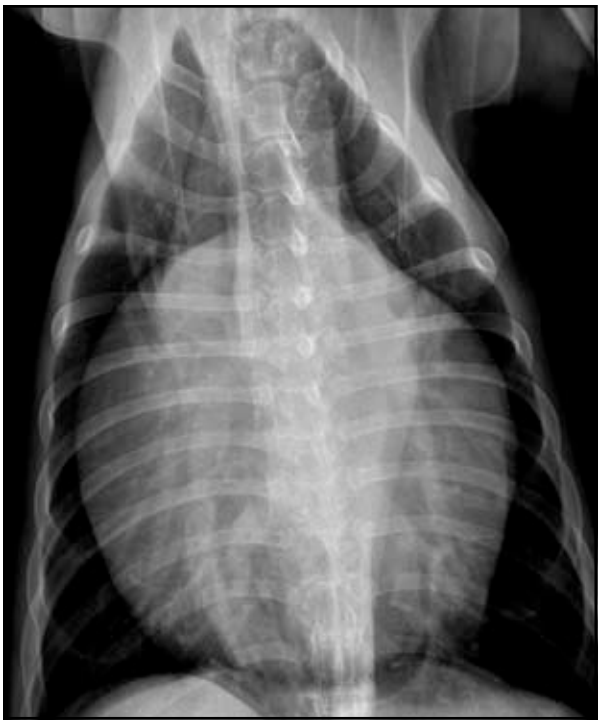
7.135 RPS short-axis echocardiogram at the heart base. A flame-like small jet of pulmonic insufficiency was identified in this 2-year-old Flat Coat Retriever. This was considered to be a clinically insignificant finding. The pulmonic valve and outflow velocity were normal. MPA = Main pulmonary artery; RVOT = Right ventricular outflow tract.



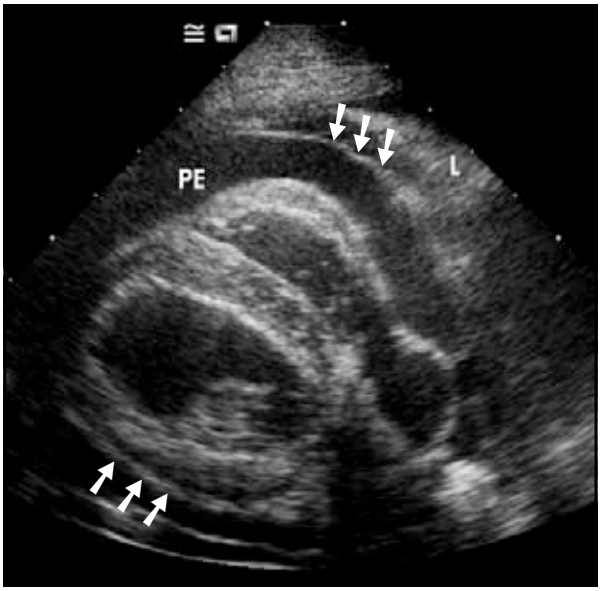
7.136 A tiny jet of aortic insufficiency identified on a RPS long-axis view (optimized for the aorta) in a 7-year-old Border Collie cross. This insufficiency was considered to be a clinically insignificant finding. AO = Aorta; LV = Left ventricle.



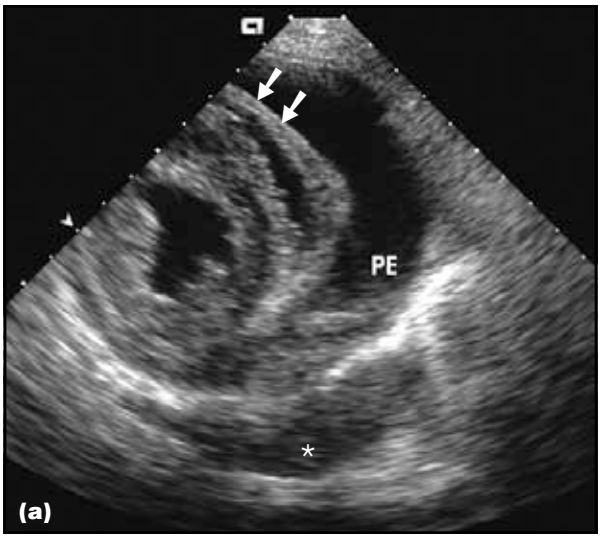
7.137 Echocardiographic and postmortem images of the heart of a 5-year-old male Labrador Retriever. The dog fell heavily on to a gate whilst jumping and developed an acute onset grade 5 holosystolic murmur and pulmonary oedema. **(a)** RPS long-axis view of the heart during diastole, showing separation of the free wall mitral leaflet from the valve annulus. The white arrow shows the direction of blood through the tear. **(b)** Colour Doppler revealed an eccentric jet of mitral regurgitation through this region. **(c)** At postmortem a mitral valve avulsion and left atrial dissecting aneurysm were confirmed. The tear separating the leaflet from the valve annulus was identified (green arrow). LA = Left atrium; LV = Left ventricle. (Courtesy of Dr P. Wotton)

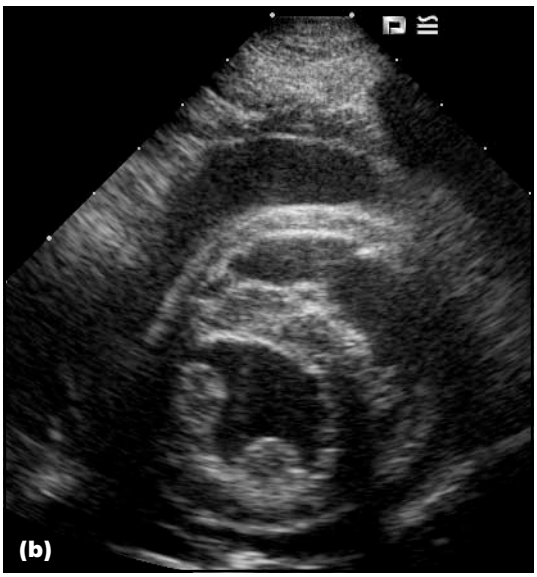


7.138 DV radiograph of a dog with a large volume pericardial effusion. Note the extremely sharp margins of the cardiac silhouette. This is often a clue to the presence of a pericardial effusion in a dog with globoid cardiomegaly.

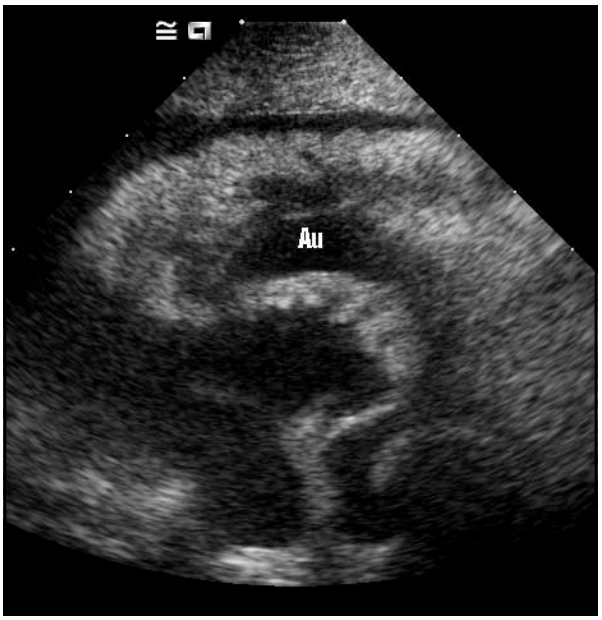


7.139 RPS long-axis echocardiogram of a 7-year-old Golden Retriever with pericardial effusion. The effusion was identified as an anechoic region (PE) surrounding the heart and encased by a thin hyperechoic rim of pericardium (arrowed). A small volume pleural effusion was also present in this dog in association with right-sided heart failure. This is seen as an anechoic region surrounding a collapsed lung lobe tip (L) in the fore field.

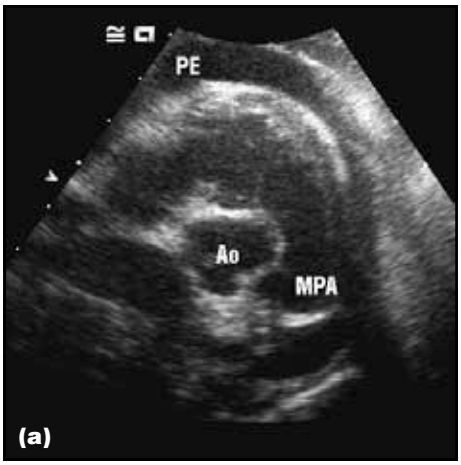


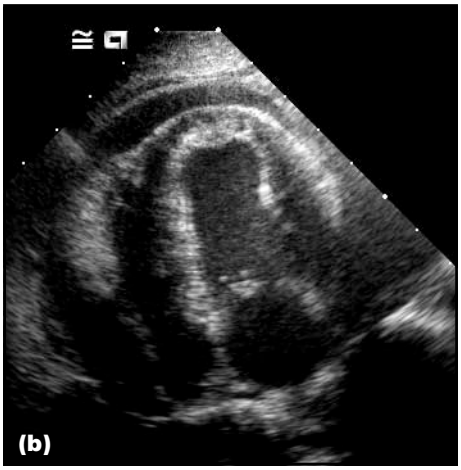


7.140 A pericardial effusion can produce cardiac tamponade. **(a)** RPS short-axis echocardiogram at the level of the papillary muscles in a dog with a pericardial effusion (PE). The image was obtained during diastole and the RV shows collapse (arrowed) due to the pericardial pressure exceeding the intraventricular pressure. This confirms cardiac tamponade. A small pleural effusion was also present in this dog (*). **(b)** RPS short-axis echocardiogram at the level of the papillary muscles in a dog with pericardial effusion. This dog does not show any evidence of tamponade as the RV did not collapse during diastole. Note that the atria should also be examined for evidence of collapse indicating tamponade.

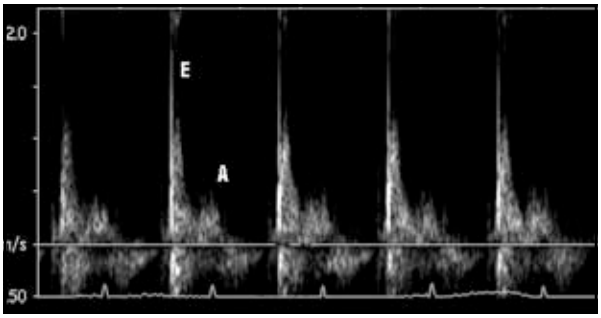


7.141 It is extremely important to visualize the tip of the right auricular appendage (Au) when evaluating patients for neoplastic causes of pericardial effusion. No mass was identified in this dog (compare with Figure 7.149). The auricular wall is thickened and slightly irregular. This is a RPS view of the heart base optimized for the RAu. LPS cranial views are also useful to examine the RAu.

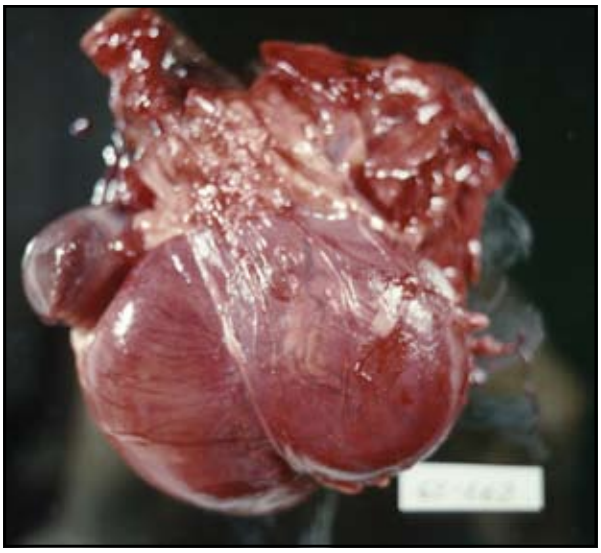




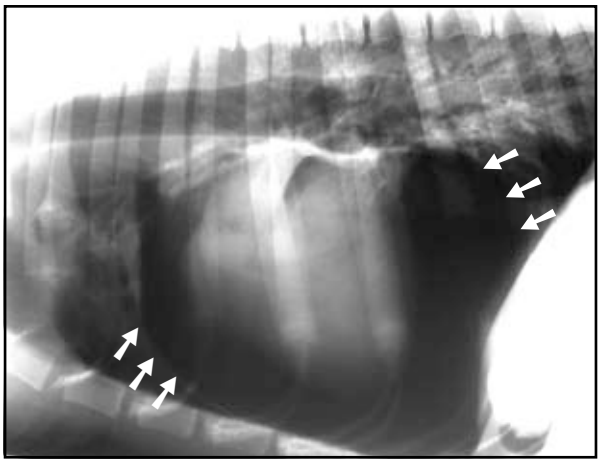
7.142 It is very important to perform a thorough examination in order to identify heart base masses. They often surround the ascending aorta; this is a difficult area to assess due to the surrounding air-filled lung. **(a)** RPS short-axis view of the heart base showing the aorta (Ao) and main pulmonary artery (MPA). The operator should continue to obtain views more dorsally from this location (until imaging is no longer possible) in a search for a heart base mass. This dog had a pericardial effusion (PE) but no mass was identified. **(b)** LPS four-chamber view. It is important to obtain LPS views and optimize imaging for the heart base (in the far field). No mass was identified in this dog.



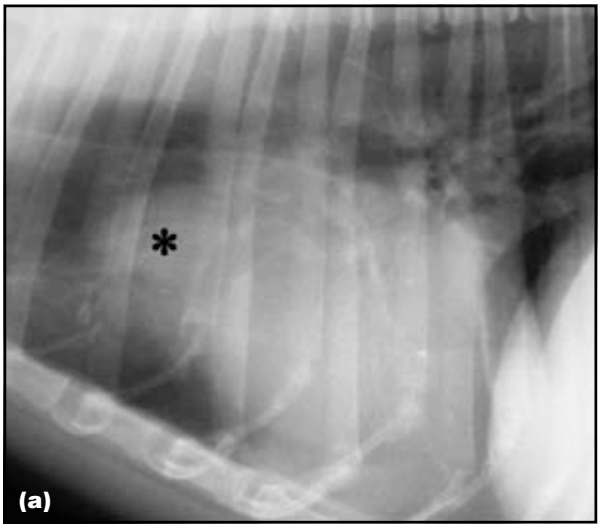
7.143 Constrictive pericarditis will lead to a restrictive pattern of ventricular filling. This image shows mitral E and A waves from a cat with constrictive pericarditis, secondary to a foreign body and pyothorax. The E wave is markedly enlarged compared with the A wave.

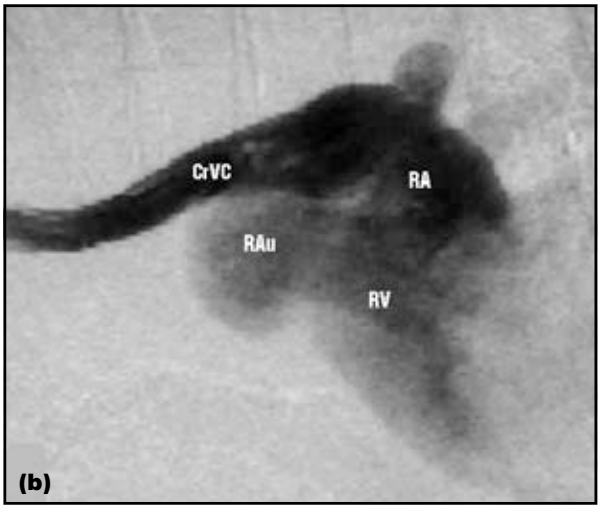


7.144 Necropsied canine heart with herniation and incarceration of the RV through a large pericardial defect. (Courtesy of Dr J. Buchanan)



7.146 Lateral radiograph of a dog with an iatrogenic pneumopericardium (arrowed) after surgery to repair a PPDH. Note that the air is constrained by the pericardial sac and that it outlines the major vessels, i.e. the actual heart is seen.





7.145 **(a)** Lateral thoracic radiograph of a 13-year-old Lhasa Apso with an ovoid mass (*) arising from the cranial cardiac silhouette. This was a herniation of the RAu through a pericardial defect. **(b)** Jugular venography (digital subtraction fluoroscopy) demonstrates vascular contrast medium in the cranial vena cava (CrVC), entering the right atrium (RA) and the right ventricle (RV) and the herniated right auricular appendage (RAu).

Dogs

Primary:
- Haemangiosarcoma (primary or metastatic)
- Chemodectoma
- Mesothelioma
- Others (undifferentiated sarcoma, myxoma, ectopic thyroid carcinoma, fibroma, fibrosarcoma, rhabdomyosarcoma, chondrosarcoma, osetosarcoma, granular cell tumour)

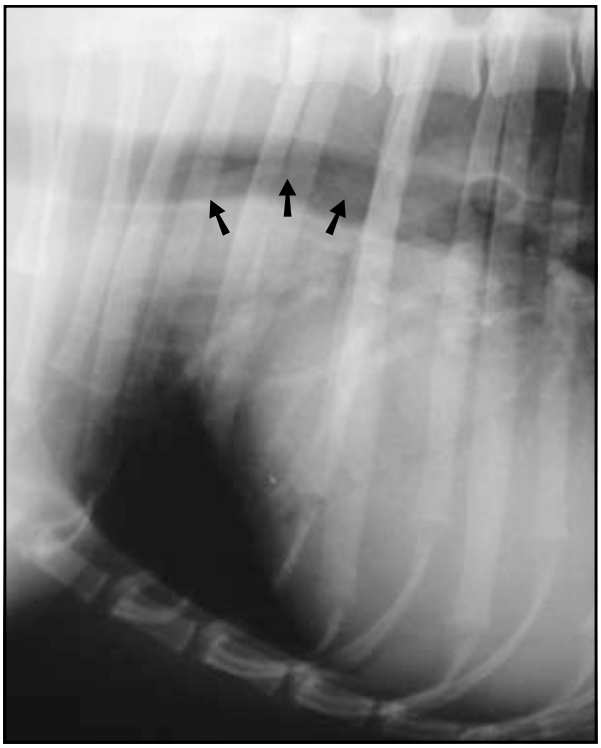
Metastatic neoplasia

Cats

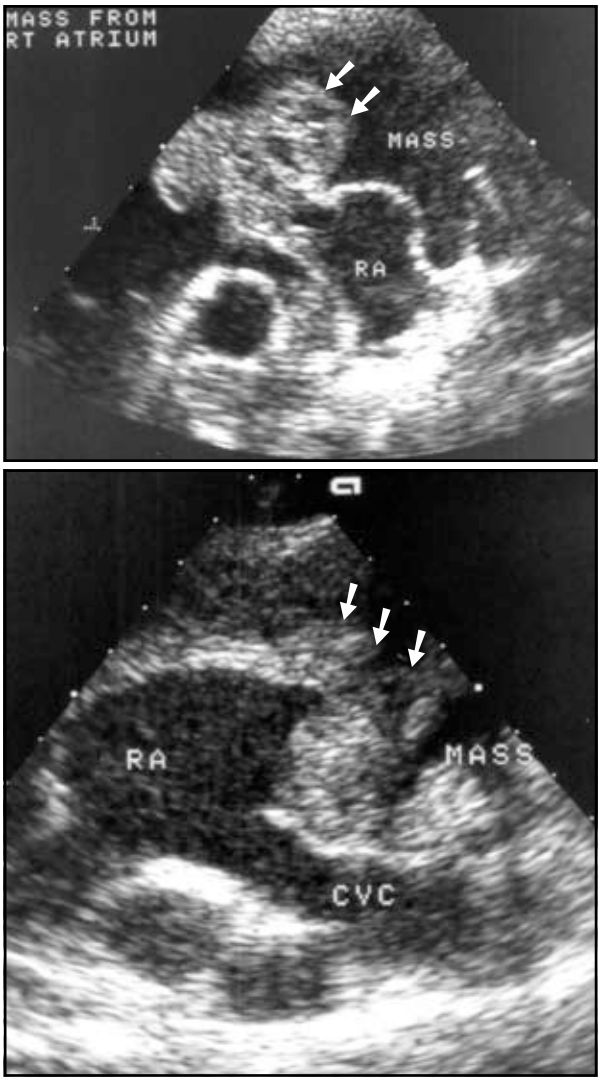
Primary:
- Lymphoma most commonly (generally considered systemic)
- Mesothelioma (very rare in this species)
- Chemodectoma (very rare in this species)

Metastatic neoplasia:
- Haemangiosarcoma (generally not involving the RAu)
- Metastatic carcinomas (pulmonary, mammary gland, salivary gland)
- Oral melanoma and squamous cell carcinoma
- Mast cell tumour

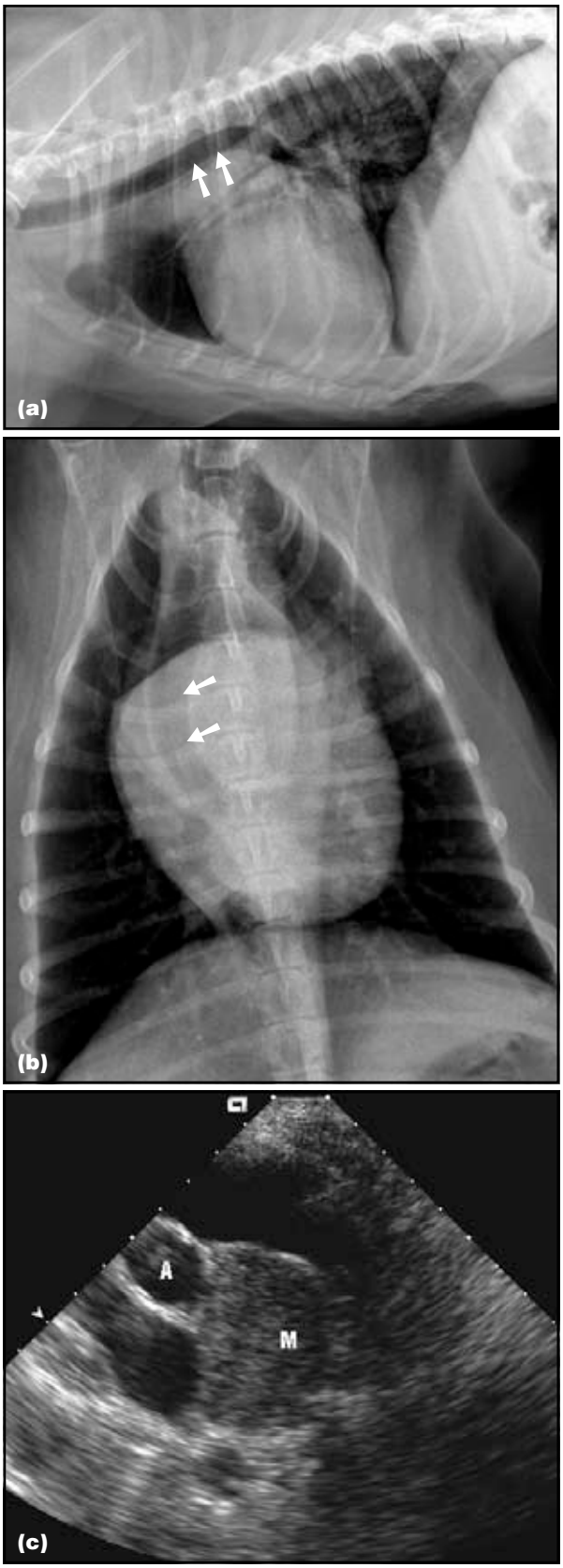
7.147 Differences between canine and feline cardiac neoplasia.



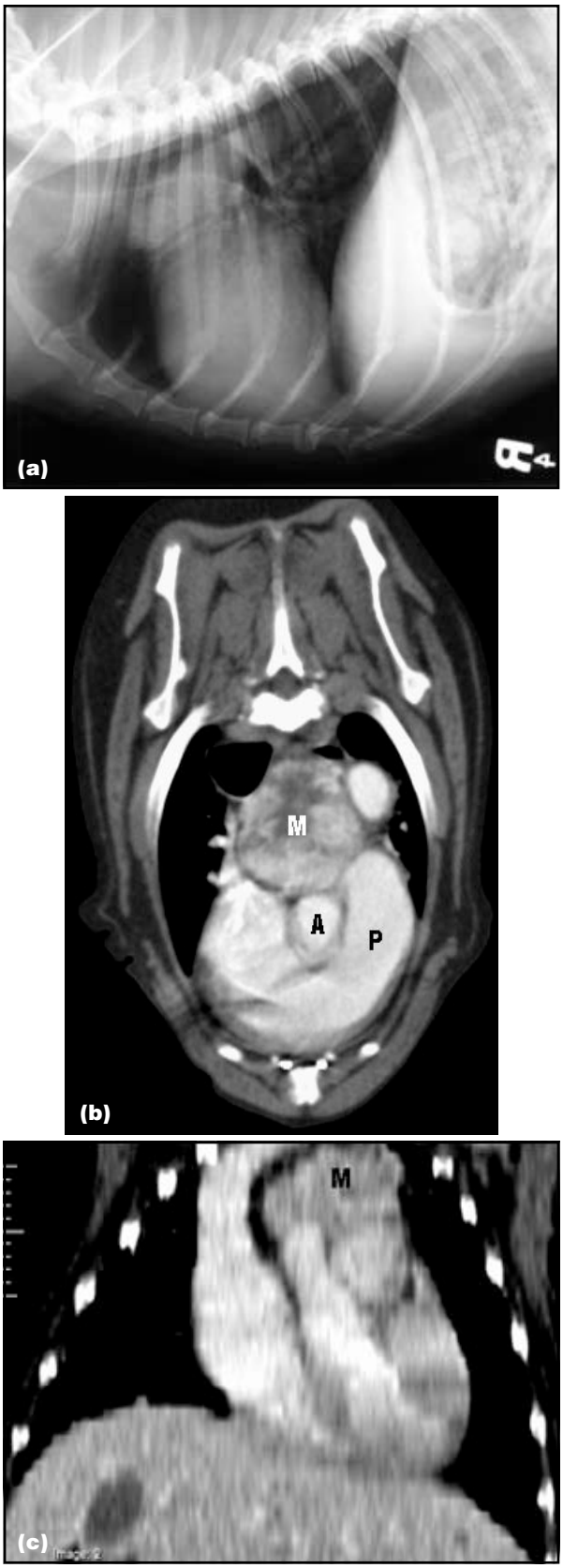
7.148 Lateral radiograph of an 8-year-old Golden Retriever with a right atrial mass. There is a focal dorsal deviation of the trachea (arrowed) just cranial to the carina and over the RA. It is very unusual to see a right atrial mass on a radiograph.



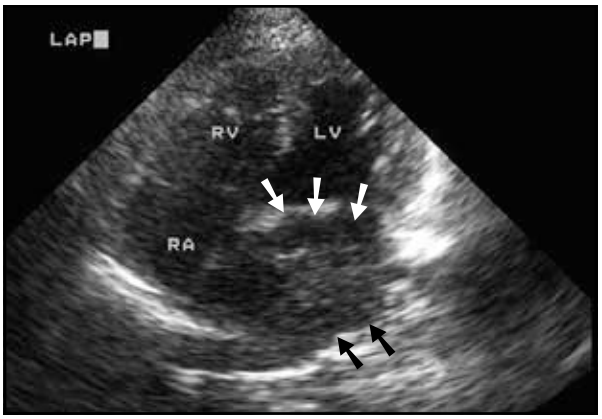
7.149 RPS short-axis echocardiograms from the heart base in two different dogs with RAu tumours. The views have been optimized for the masses (indicated with arrows); both were haemangiosarcoma. Compare these with the normal RAu shown in Figure 7.141.
CVC = Caudal vena cava; RA = Right atrium. (Courtesy of Dr B. Hopper)



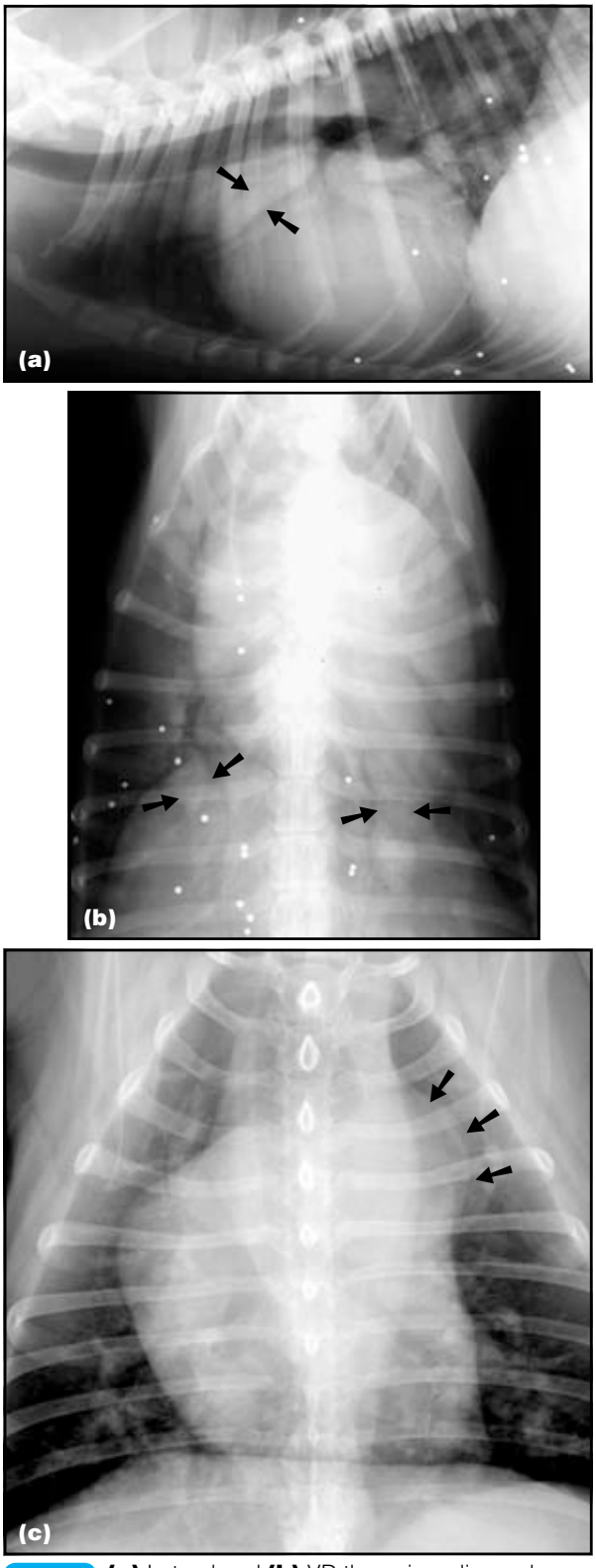
7.150 **(a)** Right lateral and **(b)** DV views of an 11-year-old mixed breed dog with a heart base tumour. Note the focal cranial and right-sided deviation of the trachea at the heart base and cranial to the carina (arrowed). **(c)** RPS short-axis echocardiogram. There is a hypoechoic mass (M) lesion surrounding the ascending aorta (A). This was a heart base mass but not confirmed to be a chemodectoma.



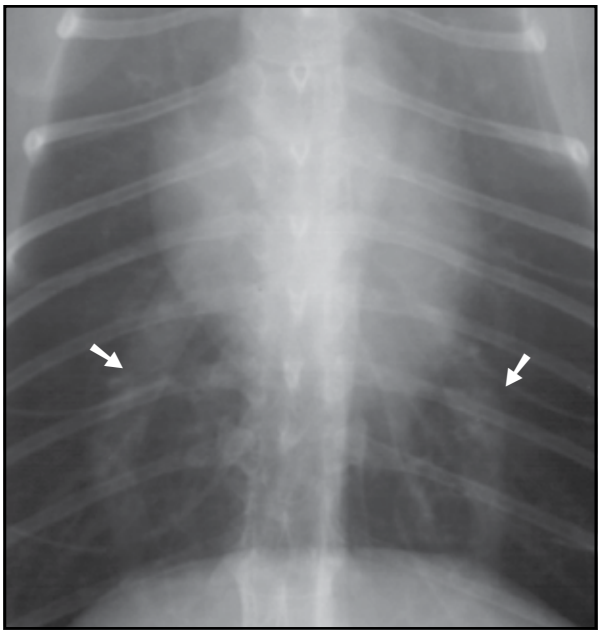
7.151 **(a)** Right lateral radiograph and **(b)** transverse CT (post-contrast) image at the level of the mid-heart from a 10-year-old male castrated Cavalier King Charles Spaniel with a heart base mass. The CT image shows a heterogenously enhancing mass (M) wrapping around the aorta (A) and main pulmonary artery (P). **(c)** The dorsal reconstruction was of further use in defining the margins of the mass.



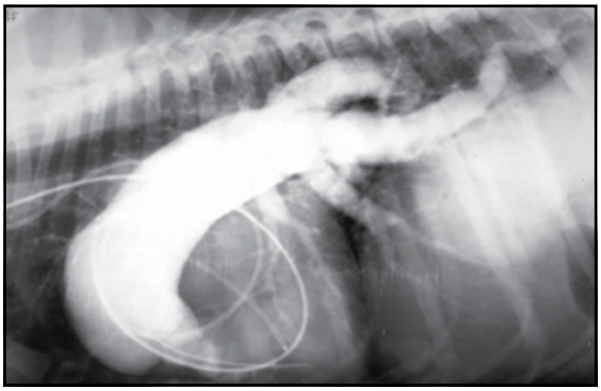
7.152 LPS four-chamber view of a dog with a left atrial myxoma. A homogenous echoic mass is seen within the LA and part of the LAu (arrowed). LV = Left ventricle; RA = Right atrium; RV = Right ventricle.



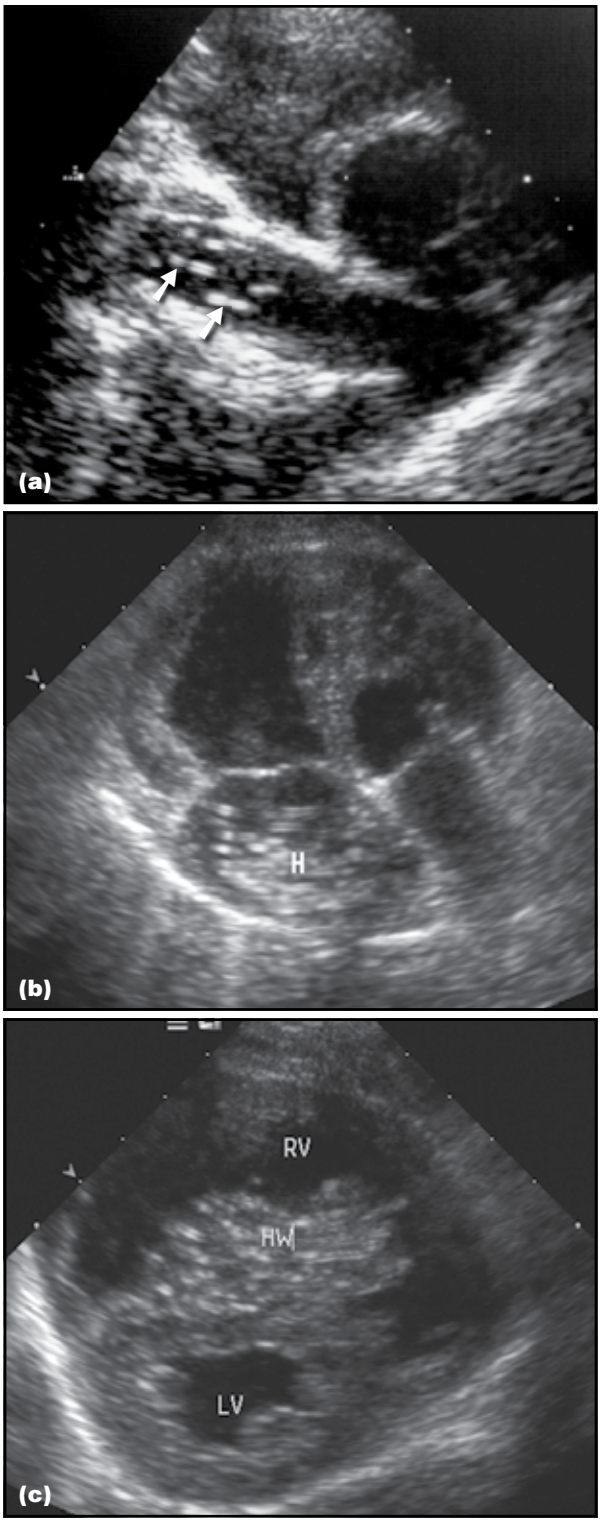
7.153 **(a)** Lateral and **(b)** VD thoracic radiographs of a 12-year-old male Golden Retriever with heartworm disease. The pulmonary arteries are extremely enlarged and tortuous (arrowed). The cardiac silhouette is rounded and right-sided cardiomegaly is present. Incidental tiny metallic opacities represent lead shot from a previous injury. **(c)** DV thoracic radiograph of a 4-year-old Staffordshire Bull Terrier with heartworm disease. This radiograph was acquired 3 months after treatment for caval syndrome and heartworm disease but severe right-sided cardiac enlargement and main pulmonary artery enlargement (arrowed) are still evident.



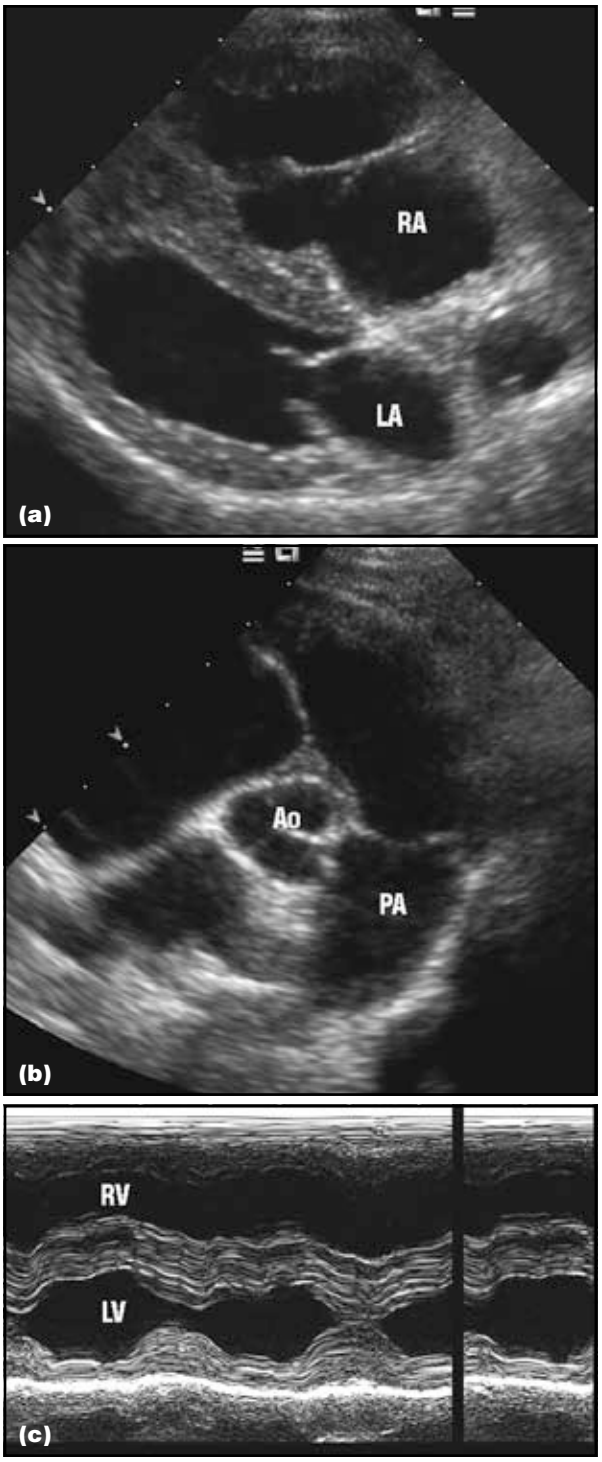
7.154 VD view of a 7-year-old Domestic Medium-hair cat with heartworm disease, showing moderate to severe enlargement and lack of tapering of the caudal lobar pulmonary arteries (arrowed).



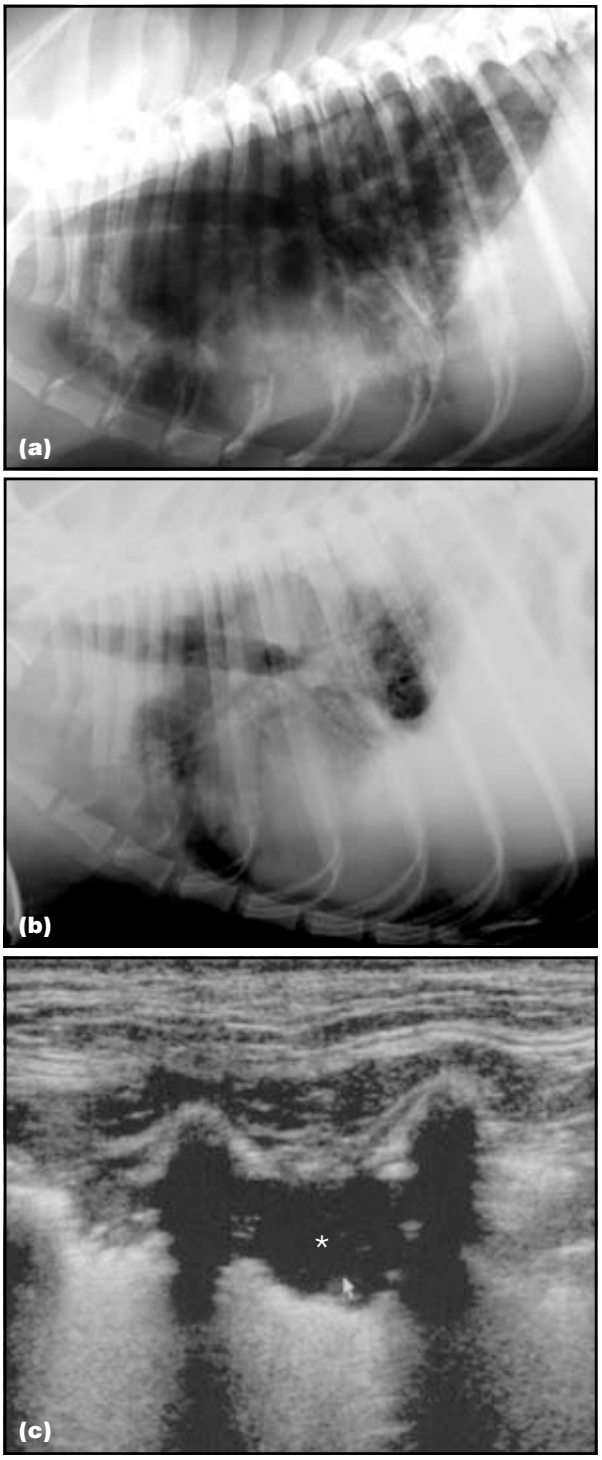
7.155 Selective angiogram in a dog with chronic heartworm disease. Several catheters are present but the angiogram has been performed via a catheter in the main pulmonary artery. The contrast medium fills a markedly dilated main pulmonary artery and its branches. The caudal lobar pulmonary arteries are also tortuous and truncated.



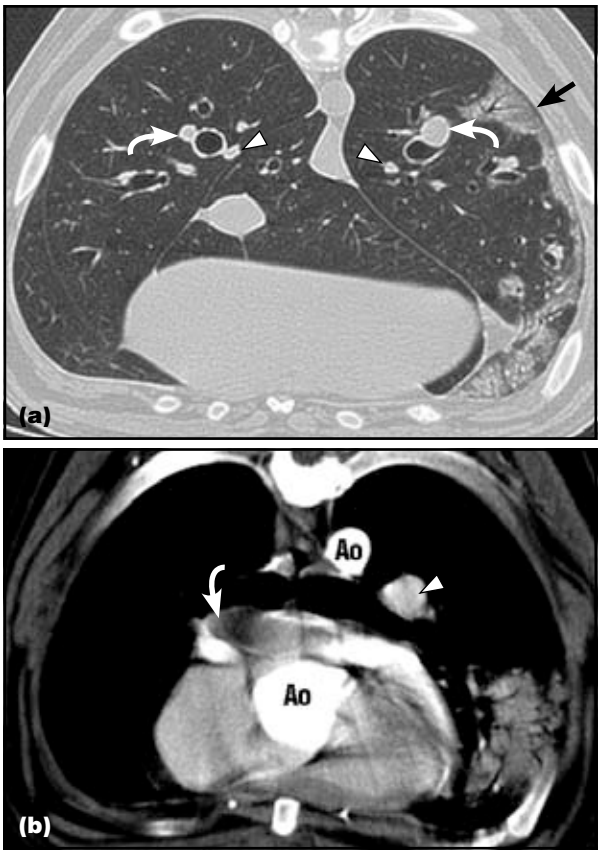
7.156 **(a)** RPS short-axis ultrasound image of the heart base and pulmonary arteries of a 7-year-old male castrated mixed breed dog. Several adult worms are identified in the right pulmonary artery (arrowed). **(b)** LPS long-axis four-chamber echocardiogram of a 4-year-old Staffordshire Bull Terrier with caval syndrome due to heartworm disease. A large mass of adult heartworms (H) is present in an enlarged RA. They are identified as parallel echogenic lines. **(c)** RPS short-axis echocardiogram obtained at the level of the papillary muscles in the same dog as (b). During diastole the worms were seen to move through the tricuspid valve and into the enlarged RV. HW = Heartworm mass; LV = Left ventricle; RV = Right ventricle.



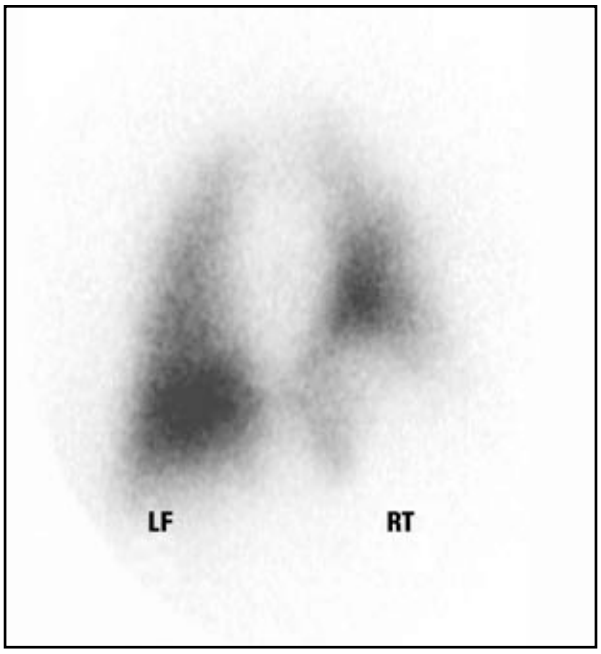
7.157 **(a)** RPS long-axis echocardiogram of the same dog as in Figure 7.156b. Note the enlarged right atrium (RA) and compare its size to that of the left atrium (LA). **(b)** RPS short-axis echocardiogram obtained at the heart base and optimized for the main pulmonary artery (PA). The main pulmonary artery is enlarged both before and after the pulmonic valve. Usually, the main pulmonary artery is close to the diameter of the aorta (Ao) at this level. **(c)** M-mode echocardiogram obtained at the level of the papillary muscles from a RPS location. The right ventricle (RV) is markedly dilated. The left ventricle (LV) is also marked for comparison.



7.158 **(a)** Right lateral thoracic radiograph of a 6-year-old spayed Golden Retriever bitch with angiostrongylosis, showing a patchy, peripheral alveolar pattern. A pleural fissure line can also be seen. **(b)** Right lateral thoracic radiograph in a 5-year-old Cocker Spaniel with angiostrongylosis. The peripheral alveolar pattern is more severe in this case. **(c)** Thoracic ultrasound image from the same dog as in (b) obtained with a linear transducer via an intercostal approach. A peripheral hypoechoic consolidated region of lung (*) is identified. Ultrasound-guided aspiration of this region confirmed the diagnosis of angiostrongylosis.

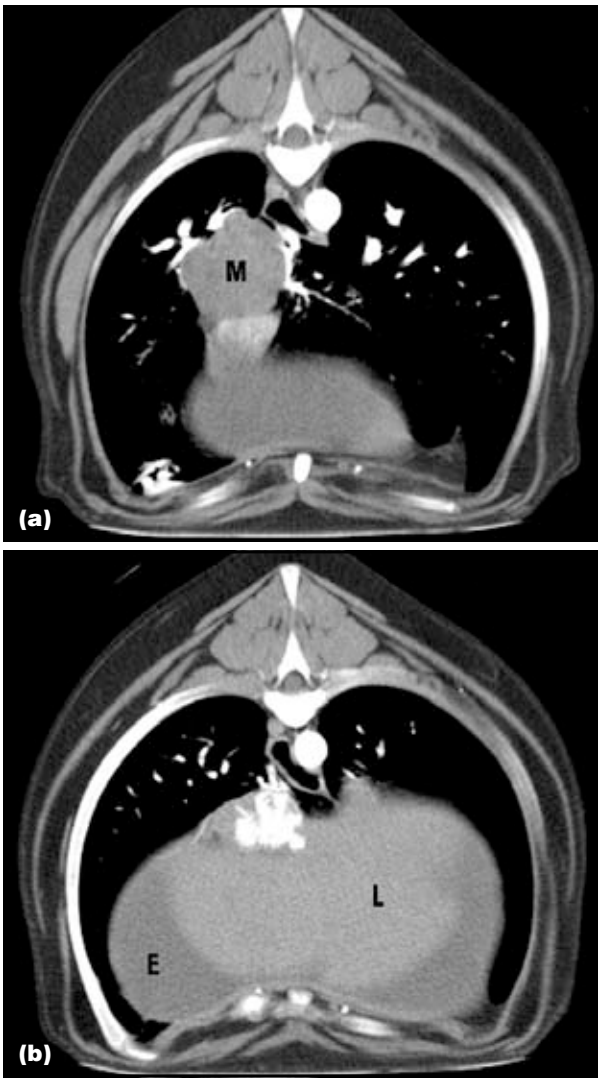


7.159 **(a)** Pre-contrast high-resolution CT image of the lung from a 9-year-old Rottweiler with PTE at the level of the accessory lung lobe. Note the wedge-shaped peripheral pulmonary consolidation (straight arrow) and distended lobar artery (anticlockwise bent arrow) of the left caudal lung lobe, the diminished size of the right caudal lung lobe lobar artery (clockwise bent arrow), and both left and right caudal lung lobe lobar veins (arrowheads). Left of dog is right side of image. **(b)** Post-contrast CT angiography image at the level of the main pulmonary artery. Notice the large contrast medium filling defect in the main pulmonary artery (bent arrow) and right pulmonary artery (arrowhead). This large thrombus extends caudally in the left lung, distending its diameter and causing bilateral oligaemia with reduced vessel size on the pre-contrast images. Ao = Aorta.

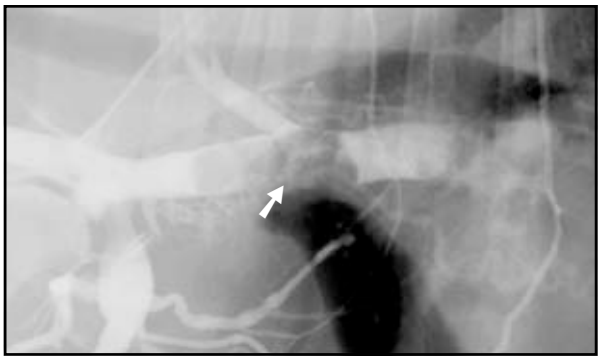


7.160 Dorsal static image of the lungs in a middle-aged, mixed breed dog with Cushing's disease presented for evaluation of possible PTE. The image was obtained 10 minutes after intravenous injection of ^{99m}Tc-MAA. Thoracic radiographs obtained immediately prior to scintigraphy were unremarkable. Note the large wedge-shaped photopenic area, which occupies the majority of the right caudal lung lobe, consistent with a large lobar pulmonary embolus.

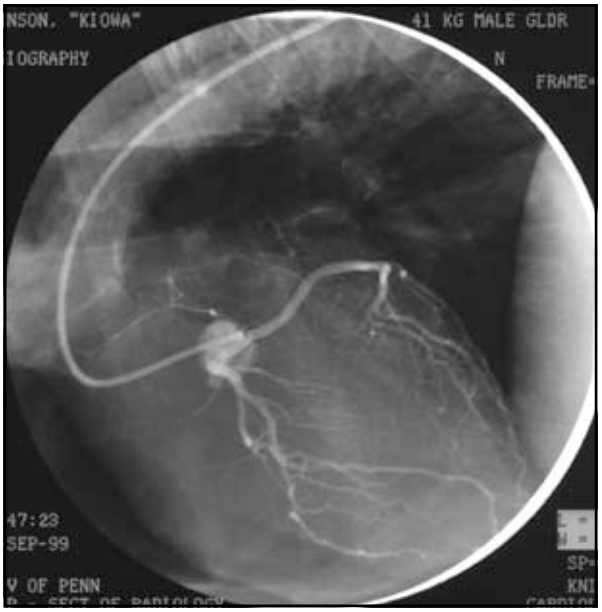
7.161 Ventral (left) and dorsal (right) static images of the lungs obtained 5 minutes after intravenous injection of ^{99m}Tc-MAA in a 4-year-old Beagle with heartworm disease Thoracic radiographs obtained immediately prior to scintigraphy showed no focal pulmonary parenchymal abnormalities. Notice the complete lack of blood flow to the left cranial, caudal and accessory lobes, with minimal perfusion to the right middle lobe. There is also a pleural based, wedge-shaped defect along the diaphragmatic border of the left caudal lobe, best seen in the ventral view.



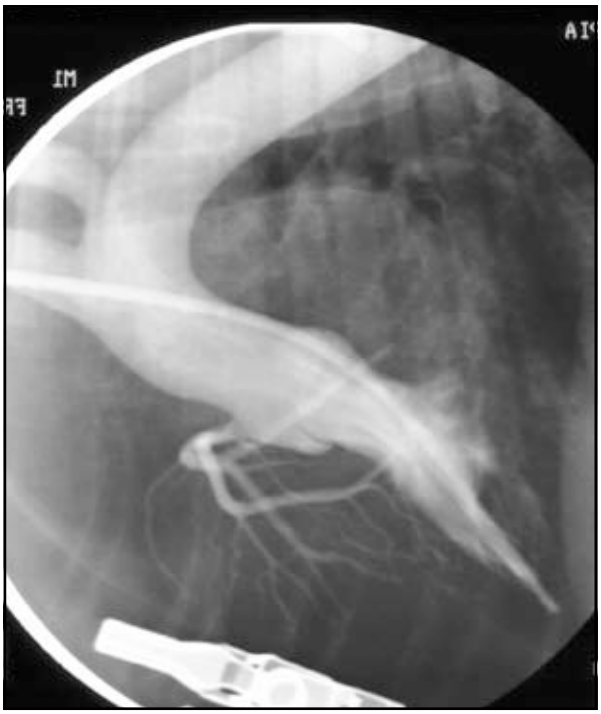
7.162 **(a)** Post-contrast transverse CT scan (soft tissue algorithm and window) of a 6-year-old male castrated Labrador Retriever with a Budd–Chiari-like syndrome due to a CdVC mass. The image was obtained caudal to the heart. The mass (M) is seen as a large soft tissue attenuating structure with a small amount of contrast medium within the CdVC ventral to it. There is also atelectasis of the ventral tip of the right caudal lung lobe**. (b)** A more caudal image from the same CT series. The caudal part of the mass has large regions of mineralization. The peritoneal effusion (E) is evident on this image, surrounding the liver lobes (L).



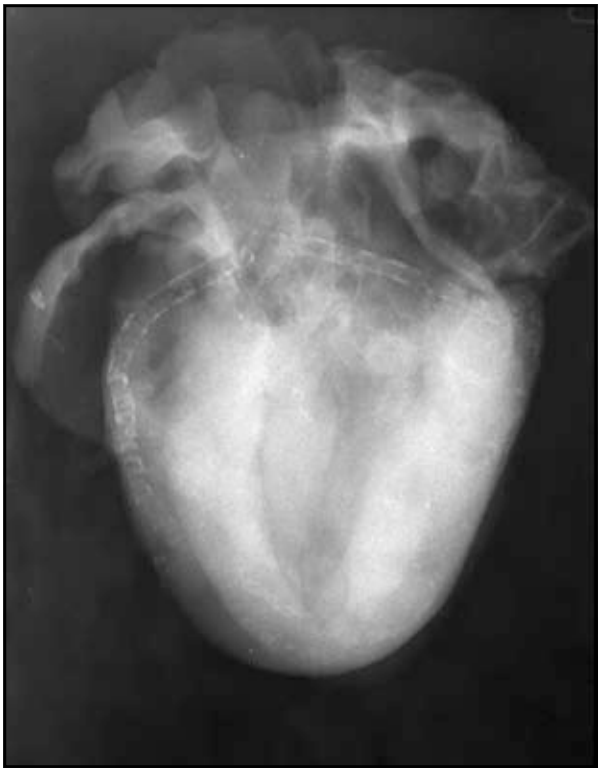
7.163 Lateral view of an 11-year-old spayed Labrador Retriever bitch with recurrence of severe oedema of the head, neck and forelimbs one week after removal of a thymoma. Non-selective angiography via the cephalic vein demonstrated a large intraluminal filling defect (arrowed) consistent with a thrombus in the CrVC at the level of the costocervical vein. A repeat angiogram two weeks later showed the thrombus to be approximately half the size. (Courtesy of Dr D. Davies)



7.164 A selective coronary angiogram with the contrast medium injection made just beyond the level of the aortic valve in a normal dog. This dog has the normal arrangement of left and right coronary arteries. (Courtesy of Dr J. Buchanan)



7.165 A selective coronary angiogram in a 5-month-old Bulldog bitch puppy. A single right coronary artery is present and has resulted in pulmonic stenosis. (Courtesy of Dr J. Buchanan)



7.166 Postmortem radiograph from a dog with mineralized coronary arteries due to atherosclerosis. The dog also had hypothyroidism, which has been strongly linked with this condition.