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Chapter 48

Heart

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Heart disease is common in cats but there are few feline cardiac conditions that are treated surgically. Of these, patent ductus arteriosus (PDA) and symptomatic, medically unresponsive bradycardias are the most frequent indications for performing cardiac surgery. Both conditions are rare in cats.

The commonest cardiac surgical procedure performed in cats is subtotal pericardectomy; the commonest indication for performing this procedure is chylothorax, which has a number of potential underlying causes, only one of which is cardiac disease.

The basic requirements for anesthesia and postoperative care for cats undergoing cardiac surgeries, and many of the potential postoperative complications are as for patients undergoing sternotomy or thoracotomy for other reasons. Specific postoperative requirements will depend on the underlying disease and the patient's clinical status. In this chapter the most commonly performed surgical cardiac procedures are discussed.

SURGICAL ANATOMY

There is little variability in normal thoracic conformation and heart shape between cat breeds. The normal feline heart is slender in appearance on a lateral thoracic radiograph and it appears more horizontal in its orientation within the thorax than in the dog; with increasing age this can become more marked.[1] The heart lies within the mediastinum in the mid thorax, approximately between the 3rd and 6th intercostal spaces.

The right atrium and ventricle form the craniodorsal part of the heart. The right atrium receives blood from the cranial and caudal vena cavae, and from the coronary vessels via the coronary sinus. The right atrial appendage arises from the cranial aspect of the right atrium and extends ventrally. The right ventricle is arciform in cross section; it lies cranial and ventral to the left ventricle. Blood is ejected from the right ventricle through the pulmonic valve into the pulmonary trunk. The initial part of the pulmonary trunk is within the pericardium, the ventral, lateral and caudal surfaces are covered by serous pericardium, and this portion of the pulmonary trunk is usually masked by fat. The pulmonary trunk contacts the aorta along its entire medial surface. The pulmonary trunk divides into the right and left pulmonary arteries. The fibrous pericardium attaches to the distal fourth of the pulmonary trunk and the terminal portion of the pulmonary trunk can be examined without opening the pericardium.

The left atrium and ventricle form the caudodorsal part of the heart. The left atrium forms the left dorsocaudal part of the base of the heart. The left atrial appendage is a cranial extension of the left atrium. It is located caudal to the pulmonary trunk and lies along the right atrial appendage, caudal to and separated from it by the pulmonary trunk. Blood enters the left atrium from the pulmonary veins, which open into its dorsal aspect; the individual pulmonary veins may fuse but usually remain separate and enter the left atrium separately. The left ventricle is cone shaped and its apex forms the apex of the heart. Blood is ejected from the left ventricle through the aortic valve into the aorta, near the center of the heart base. The initial part of the aorta, the ascending portion, is mainly located within the pericardium. It continues for a short distance cranially before passing dorsocaudally and to the left by forming a U-bend, the aortic arch. Two great vessels leave the aortic arch to supply the head, neck, and thoracic limbs: the brachiocephalic trunk and the left subclavian artery. The ligamentum arteriosum, the connective tissue remnant of the fetal ductus arteriosus, arises near the bifurcation of the pulmonary trunk and passes to the aorta. The aorta then continues caudally as the descending aorta.

The right and left vagus nerves pass caudally in the dorsal mediastinal pleura. The left vagus nerve passes over the ligamentum arteriosum, and is a landmark used during the surgical treatment of PDA. The left recurrent laryngeal nerve leaves the left vagus nerve just cranial to the ductus and it passes around the caudal aspect of the ductus.

The pericardium forms a double layered sac that surrounds the heart, the initial portion of the ascending aorta, pulmonary trunk, termination of the vena cavae, and the pulmonary veins. It attaches near/at the base of the heart to the aortic arch, pulmonary trunk, left atrium at the level of the pulmonary veins and dorsal to the interatrial groove on the right side. The outer fibrous layer is termed the parietal pericardium, the inner serous membrane the visceral pericardium, which is adherent to the epicardium. At its apex the pericardium is continued as a flattened band, the caudoventral mediastinal reflection, which attaches to the ventral part of the muscular insertion of the diaphragm. The right and left phrenic nerves run across the dorsal third of the pericardium and must be identified and preserved during pericardectomy. Paired pericardiophrenic blood vessels from the

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internal thoracic arteries run with the phrenic nerves. Pericardial blood vessels, also arising from the internal thoracic arteries, run within the pericardium passing in a caudoventral direction. With pericardial disease or pericardial and/or pleural effusion the pericardium can become thickened and these vessels will become more prominent.

GENERAL CONSIDERATIONS

Specific investigations will depend on the patient's history, clinical signs, findings on clinical examination, and differential diagnoses but will usually include a minimum of hematology, serum biochemistry, and thoracic radiography. Specific blood tests to assess for infections such as feline coronavirus, leukemia, and immunodeficiency viruses may be indicated. For investigation of suspected cardiac disease electrocardiography (ECG), echocardiography and, uncommonly, angiography are indicated. Pleural and/or pericardial effusions should be sampled for biochemistry, cytology, and bacterial and/or fungal culture as appropriate. Blood coagulation screen should be performed if a hemorrhagic effusion is identified. Advanced imaging of the thorax using computed tomography may be appropriate in cases (e.g., chylothorax or other pleural effusions) where intrathoracic neoplasia is a differential diagnosis.

SURGICAL DISEASES

Patent ductus arteriosus

The ductus arteriosus carries blood from the pulmonary artery to the aorta in the fetus. Within hours of the birth the ductus normally closes in response to an increase in the oxygen tension of the blood; failure of closure allows continual shunting of blood between the left and right sides of the heart. After birth, the blood pressure in the pulmonary artery reduces and the systemic blood pressure increases. Typically this pressure differential is such that blood shunts continuously through the cardiac cycle, from the higher pressure aorta to the lower pressure pulmonary artery (i.e., left to right shunting) if the ductus remains patent. The secondary changes that occur with increased blood flowing in the pulmonary artery are pulmonary over-circulation, left atrial dilatation, left ventricular dilatation with eccentric hypertrophy, dilatation of the aortic arch to the level of the origin of the PDA, and dilatation of the pulmonary trunk. As a consequence of the left to right shunting and left-sided volume overload, left-sided congestive heart failure (CHF) commonly develops. The size of the shunt will determine volume of blood shunting, which in turn determines the onset and severity of secondary consequences. As a sequel to the increased blood flow in the pulmonary vessels, resistance to blood flow and pulmonary hypertension can develop. If the pulmonary arterial pressure is greater than the aortic pressure for some or all of the cardiac cycle, blood flow through the PDA will reverse. Blood will then flow from the pulmonary artery into the aorta (i.e., right to left shunting). Right to left shunting can also occur from birth, and usually occurs when the ductus is large and tubular without narrowing at the pulmonary ostium, and this may reflect retention of fetal pulmonary vasculature. Right to left shunting, termed Eisenmenger's physiology, is rarely reported in cats,[2,3] although it is suggested that it is more likely to develop over time in cats than dogs.[4] Surgical treatment of the PDA is contraindicated in patients with right to left shunting.

PDA is a rarely diagnosed congenital heart disease in the cat, with few cases of this condition reported in the English language veterinary literature so it is difficult to make conclusions about the presentation, investigative findings, and the outcome with medical or surgical treatment. This contrasts with dogs, where PDA is one of the more commonly diagnosed congenital heart diseases[5,6] and it is well documented.

Most dogs are diagnosed as puppies, whilst the condition is asymptomatic, by auscultation of a pathognomic continuous murmur. It is suggested that the situation may be similar in cats, and certainly the majority of cats reported with PDAs are less than one year of age. However, in an abstract reporting 21 cases of PDAs in cats approximately two-thirds had a continuous murmur but the remainder were described to have systolic murmurs and approximately only one-third of the cats in the study were asymptomatic at presentation. Their presenting complaints included abnormal respiration, exercise intolerance/lethargy, stunted growth, and poor weight gain.[7]

The murmur is well described in dogs; it may be accompanied by a thrill felt at the heart base, and by hyperkinetic pulses. The point of maximal intensity of the murmur lies over the main pulmonary artery at the dorsocranial heart base and may radiate cranially to the thoracic inlet and to the right heart base. It is suggested that in cats the continuous murmur of a PDA may be best heard slightly more ventrally than this typical location in dogs. Femoral pulses may be more difficult to assess in cats;[8] hyperkinetic pulses may be noted.

Diagnosis

Thoracic radiographic changes are variable and will depend on the size of the PDA and any consequential left-sided heart failure. Cardiomegaly, shifting of the cardiac apex to the left, a ductus aneurysmal bump, and pulmonary over-circulation are all common radiographic findings. If the patient presents in left-sided heart failure, pulmonary venous congestion and pulmonary edema will be present.

Diagnosis is usually confirmed with echocardiography using a right parasternal short axis or left parasternal cranial window (Fig. 48-1). Initially, there may be no changes in chamber size or myocardial contractility but eventually the consequences of left to right shunting may result in dilatation of the left atrium, aorta, and main pulmonary artery and dilatation and hypertrophy of the left ventricle, all recognizable on echocardiography (Fig. 48-2). Left ventricular systolic function may become impaired. If a left-to-right shunting PDA is identified, Doppler studies can be performed and will show characteristic high velocity turbulent flow from the aorta, through the ductus and into the main pulmonary artery (Fig. 48-3). This PDA peak velocity will

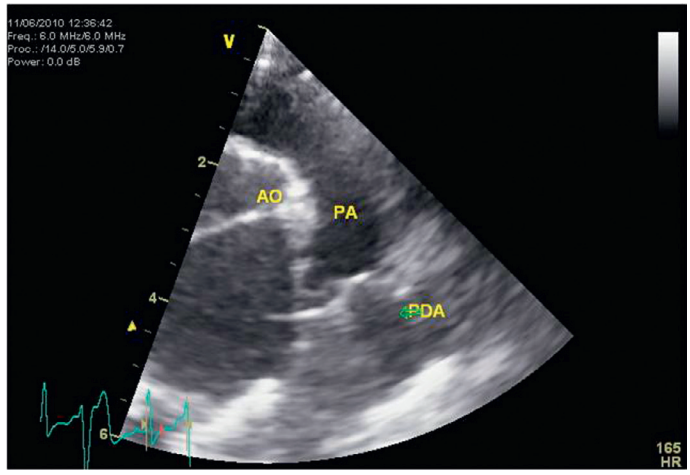


Figure 48-1 Right parasternal short axis view of the heart in a cat with a patent ductus arteriosus (PDA). This image was taken at the level of the pulmonary artery and shows the PDA. *(Courtesy Dr Jo Dukes-McEwan.)*

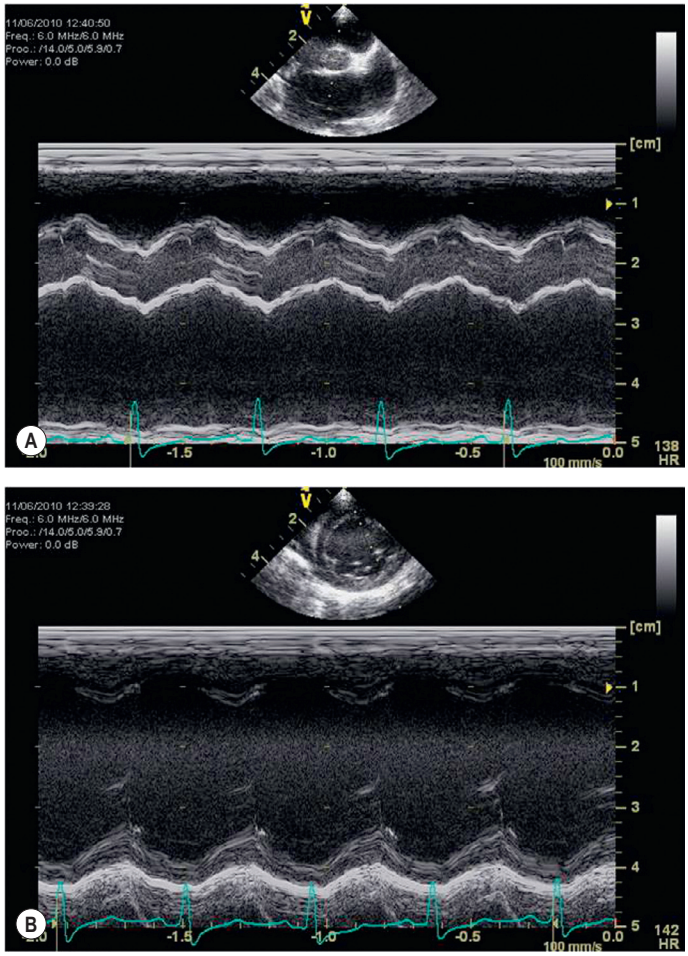


Figure 48-2 A cat with a left-to-right shunting patent ductus arteriosus (PDA). **(A)** Aortic M-mode image showing marked left atrial dilatation relative to the size of the aorta. **(B)** M-mode image of the left ventricle showing left ventricular dilatation, relative wall thinning and impaired systolic function. *(Courtesy Dr Jo Dukes-McEwan.)*

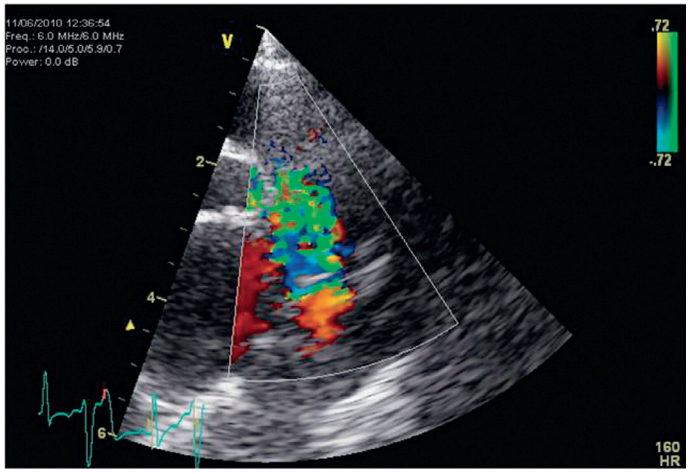


Figure 48-3 Right parasternal short axis view of the heart in a cat with a patent ductus arteriosus (PDA). This image was taken at the level of the pulmonary artery with color flow Doppler showing turbulent flow across the PDA. *(Courtesy Dr Jo Dukes-McEwan.)*

decrease if pulmonary pressures increase, prior to exceeding systemic pressures. However, note that this decrease may be reactive pulmonary hypertension as a consequence of the pulmonary over-circulation and closure of the PDA may resolve the mild pulmonary hypertension when assessed after surgery. Mild increase of velocity in the left ventricular outflow tract and mild aortic and pulmonic insufficiency may also be recognized. Mitral regurgitation is secondary to the left-sided dilation and may be significant.

Thorough echocardiography should be performed in cats because concurrent, but unspecified, congenital heart defects may be present; these were found in 29% of cats in one study.[7]

If the diagnosis of suspected PDA cannot be confirmed by echocardiography, or the presence of other congenital heart anomalies is suspected, cardiac catheterization and angiocardiography may be helpful, but this investigative procedure is rarely required to confirm diagnosis of PDA.

ECG is non-specific; it usually shows tall R waves and wide P waves due to atrial enlargement. Arrhythmias such as atrial fibrillation, supraventricular or ventricular arrhythmias, and ventricular premature complexes may be present.

Treatment

Patients with CHF and pulmonary edema at the time of diagnosis should be treated for several days with furosemide and angiotensin-converting enzyme (ACE) inhibitors before proceeding to anesthesia and surgery. In patients that are older at the time of diagnosis, with a small PDA, conservative management may be appropriate, although generally surgery is still recommended.

Pericardial disease

Congenital pericardial diseases are infrequently diagnosed and most have an excellent prognosis with surgical treatment. The most common condition in dogs and cats is peritoneopericardial diaphragmatic hernia (PPDH);[9] it is more common in cats (see Chapter 45).

Intrapericardial cysts

Intrapericardial 'cysts' are rarely reported.[10–14] These are usually not true cysts but encapsulated adipose tissue or organizing cystic hematomas. The cyst may be attached to the apex of the pericardium by a pedicle. They may arise if a PPDH containing herniated omentum or falciform fat closes before birth, trapping the fat within the pericardial sac. Cystic changes of herniated and incarcerated liver tissue may also occur in association with PPDH in cats. Intrapericardial cysts cause clinical signs either directly associated with their presence, or due to an associated effusion causing cardiac tamponade. Diagnosis can usually be made by radiography (Fig. 48-4) and/or ultrasound examination of the cranial abdomen and heart. Treatment is by subtotal pericardectomy (see Box 48-4), removal of the cyst (and pedicle if present), and repair of the PPDH (Chapter 45, 45.4.3) if present.

Congenital pericardial defects are very rare. Clinical signs may develop if the size of the defect is such that a portion of the heart can herniate through the defect, as cardiac function will then be compromised. This condition is treated by subtotal pericardectomy.

Pericardial effusion

The commonest acquired pericardial disease in both cats and dogs is pericardial effusion (PE), but the incidence, presentation and underlying causes have differences between these species. Clinically significant PE is very uncommon in cats, and this species rarely presents with cardiac tamponade. In both species PEs remain asymptomatic if the

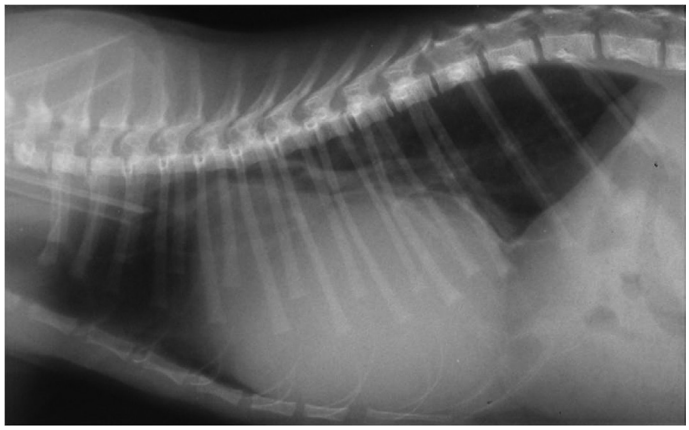
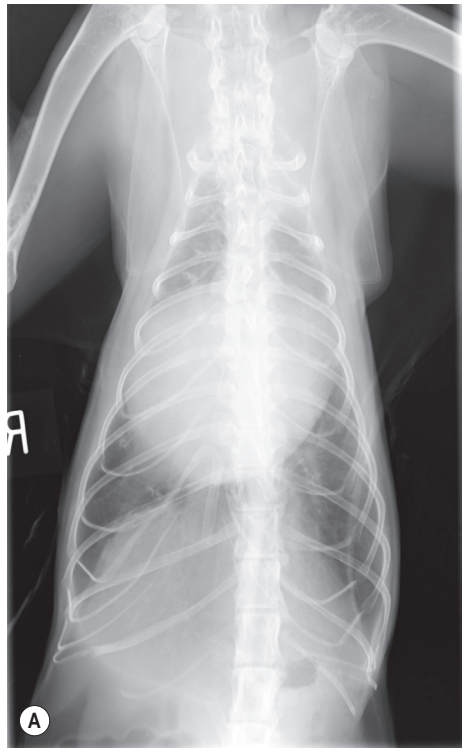


Figure 48-4 Lateral thoracic radiograph of a young, anesthetised cat with a peritoneopericardial diaphragmatic hernia (PPDH). The cardiac silhouette is markedly enlarged and silhouettes with the diaphragm. The distal intrathoracic trachea is displaced dorsally over the cranial border of the cardiac silhouette.



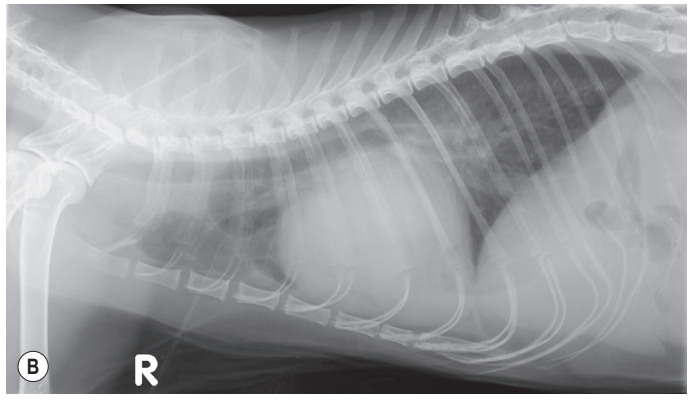


Figure 48-5 **(A)** Dorsoventral and **(B)** lateral thoracic radiographs of a cat with a pericardial effusion. There is a small volume of pleural fluid. The underlying heart disease in this cat was severe hypertrophic cardiomyopathy. *(Courtesy Dr Jo Dukes-McEwan.)*

Box 48-1 Possible causes of pericardial effusion in cats

- Congestive heart failure (various cardiac pathologies including hypertrophic cardiomyopathy, dilated cardiomyopathy, pulmonic stenosis)
- Feline infectious peritonitis
- Peritoneopericardial diaphragmatic hernia
- Cardiac neoplasia (e.g., lymphoma, hemangiosarcoma, rhabdomyosarcoma)
- Heart base tumors
- Pericardial tumor (e.g., mesothelioma)
- Systemic inflammation and infection
- Uremia
- Coagulopathies (e.g., warfarin toxicity, disseminated intravascular coagulation)
- Trauma
- Intrapericardiac cyst
- Bacterial pericarditis

effusion is small and the pericardium remains slightly compliant, or if a larger effusion accumulates slowly allowing the pericardial sac to accommodate to some extent. Cardiac tamponade results when the amount of fluid (or cyst/mass) reaches a volume such that it exerts a pressure equal to or greater than normal cardiac diastolic pressures. The heart is compressed externally and cannot fill effectively during diastole. Right ventricular filling is reduced first, and may progress to compromise left ventricular filling and cardiac output. Systemic venous pressure and capillary hydrostatic pressure increase whilst cardiac output is reduced. Cats with cardiac tamponade usually develop pleural effusion and present with tachypnea and increased respiratory effort.

PE in cats is most commonly part of a more generalized disease (Box 48-1). Myocardial disease is the commonest underlying cause.[15] Small volume clinically insignificant PEs have been reported in approximately 45% of cats with CHF,[16,17] while PEs are rarely reported in dogs with CHF. The other most frequent underlying cause of PE in cats is feline infectious peritonitis. The commonest causes of PE in dogs are idiopathic hemorrhagic pericardial effusion (IPHE), which is unreported in the cat, and cardiac-related neoplasia. In dogs, hemangiosarcoma, usually of the right side of the heart, is the most common neoplastic cause of PE, aortic body tumors also occur with some frequency, mesotheliomas occur less commonly, and other tumors are also reported. These tumors are rarely diagnosed as a cause of PE in cats; lymphoma is the most common underlying tumor associated with the effusion.

The classic radiographic appearance of PE is general cardiomegaly (globoid heart) and sharp borders to the cardiac silhouette (Fig. 48-5). If the PE has accumulated rapidly without time for adaptation, cardiac tamponade may occur without the classic radiographic signs being present. Pleural effusion is usually present in cats with significant PE and may partly obscure the cardiac silhouette.

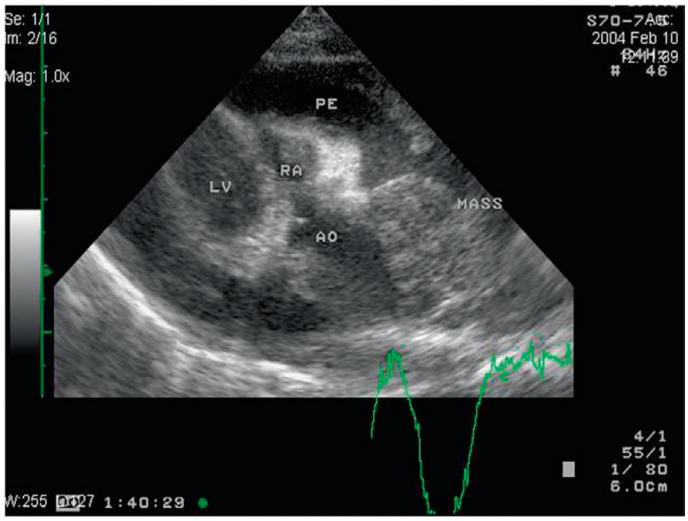


Figure 48-6 Echocardiogram of a cat with a heart base mass and an associated pericardial effusion (PE). *(Courtesy Dr Jo Dukes-McEwan.)*

A definitive diagnosis of PE is made by echocardiography, which allows cardiac disease or mass lesions to be excluded. The pericardial fluid is seen as an anechoic/slightly anechoic circular region surrounding the heart, within the thin hyperechoic line representing the pericardium. Although an uncommon cause of PE in cats, mass lesions affecting the heart may be seen on ultrasound examination. A thorough examination of the heart should be performed prior to pericardiocentesis because masses are most likely to be identified whilst surrounded by fluid (Fig. 48-6).

ECG findings in the dog can include reduced amplitude QRS complexes, electrical alternans and ST segment depression, but low voltage QRS complexes are normal in cats.

Cardiac tamponade should be treated immediately by pericardiocentesis (Box 48-2). Any pleural effusion should also be drained. Cats with significant PE may benefit from subtotal pericardectomy (see Box 48-4).

Chylothorax and fibrosing pericarditis

Fibrosing pleuritis (see Chapter 44) can develop secondary to any prolonged hemorrhagic, exudative, or chylous effusion. In these circumstances the pericardium is subject to the same environment and is also likely to become thickened and less compliant and there may be a consequential increase in venous pressures. Even a modest increase could cause the formation of extra lymphatics in the cranial thorax; these lymphatics may then leak. Thus a thickened, noncompliant pericardium may contribute to the development of chylothorax, and conversely chylothorax may cause pericardial thickening or fibrosing pericarditis. The actual role that the pericardium has in the development of and progression of chylothorax is unknown but pericardectomy, in combination with thoracic duct ligation with or without thoracic omentalization, has been shown in several small case series to achieve a successful resolution of the disease in approximately 75–80% cats.[18,19] Subtotal pericardectomy (see Box 48-4) has been successful in some cats as the sole treatment of chylothorax. It has also been suggested that it may be used to treat and/or prevent the serosanguinous effusions that occasionally occur in some patients after thoracic duct ligation.[18] Further studies are required to investigate these apparent benefits of pericardectomy.

Box 48-2 **Pericardiocentesis**

Pericardiocentesis is performed in the conscious cat if this is tolerated, otherwise sedation is used if required for patient restraint. The cat may be positioned in lateral or sternal recumbency, depending on clinician preference and the tolerance of the patient. Pericardiocentesis can be performed through a right or left lateral approach. ECG monitoring should be performed throughout the procedure. The landmark for pericardiocentesis is found by palpation of the point of maximal intensity of the heart beat; this is typically the ventral 6th-7th intercostal space and is confirmed using ultrasonography. The area is clipped and prepared for surgery and local anesthetic can be instilled into the skin, subcutaneous tissues, intercostal muscles, and pleura at the site of proposed catheter entry through the chest wall. Pericardiocentesis is performed as a sterile procedure using aseptic technique. A tiny stab incision is made in the skin at the point of catheter insertion and a 16–18G 2.5 inch over the needle catheter is introduced through the chest wall in a direction aimed towards the opposite scapula. Extra holes can be cut in the catheter near the tip to increase the efficiency of drainage. The catheter is slowly introduced through the chest wall and through the pericardium. If the tip of the catheter can be felt touching the surface of the heart and/or the patient develops arrhythmias, the catheter should be withdrawn several millimeters before being redirected. Once sanguinous fluid is seen in the hub of the catheter, or the catheter is identified within the pericardial sac if performing the entire procedure under ultrasonographic guidance, the needle is removed and the catheter is connected to a piece of extension tubing and a 20–50 mL syringe. The catheter is advanced several more millimeters into the pericardial sac and fluid is aspirated. To confirm that the fluid is not being withdrawn from a blood vessel or the heart, the fluid can be observed for one to two minutes for the presence of clotting. PE will not clot, whilst blood collected from the heart or a blood vessel will clot. Once the catheter is within the pericardial sac the pericardial fluid is drained. Ultrasonography should be performed to confirm removal of the effusion. At the completion of pericardiocentesis the catheter can be withdrawn.

Constrictive pericardial disease

Rarely, the visceral and/or parietal pericardial thickening can restrict ventricular filling in the absence of a PE, resulting in constrictive pericarditis. In some cases a small PE may be present, which would be insufficient to cause cardiac tamponade if the pericardial changes were absent, a situation that is called constrictive-effusive pericarditis. This condition is rarely diagnosed in cats, but it has been reported in a cat with dilated cardiomyopathy.[20] It is uncommon in dogs; underlying causes have included IPHE, intrapericardial foreign body, chronic septic pericarditis, and traumatic pericardial hemorrhage. Diagnosis can be difficult, and is easier if a small volume of PE is present. Thoracic radiographs may show mild to moderate cardiomegaly, pleural effusion, and distension of the caudal vena cava. Echocardiography may demonstrate suggestive changes such as flattening of the left ventricular free wall in diastole and abnormal septal motion. Invasive hemodynamic studies to measure central venous pressure changes may be needed to confirm the diagnosis.

Treatment is subtotal pericardectomy (see Box 48-4). The surgery is easier, complications fewer, and the outcome good if only the parietal pericardium is involved. If the visceral pericardium (epicardium) is also involved, and is fused to the parietal layer, subtotal pericardectomy is a more difficult and traumatic procedure. Stripping of this layer can result in severe hemorrhage from the myocardium, tachyarrythmias, and pulmonary thromboembolism postoperatively.

Symptomatic bradycardia of cardiac origin

Symptomatic bradyarrhymia is one of the indications for pacemaker implantation in cats. Affected cats may have signs of hypoperfusion, and/or CHF, and/or syncope or seizure-like episodes due to atrioventricular (AV) block and normokalemic atrial standstill. Bradyarrhythmias are often asymptomatic in cats, and hence do not require treatment, as cats can have stable and relatively fast escape rhythms (90–120 beats per minute),[21] unlike dogs.

In dogs, electrodes are usually placed via a transvenous route, whilst in cats, generally, epicardial electrodes are placed using a surgical approach. This is because in cats the transvenous route is technically more difficult, and complications such as thromboembolism and obstruction to venous return causing chylothorax occur more commonly.[22]

A thorough history, clinical examination, and patient assessment are necessary to ensure that any non-cardiac disease is identified and eliminated as a cause of the bradycardia.

An ECG is necessary to diagnose the bradycardia. There is an absence of P waves and a slow supraventricular or ventricular escape rhythm with persistent atrial standstill. High grade second degree AV block is characterized by frequent 'dropped' QRS complexes, and usually does not respond to atropine. Third degree AV block has a complete dissociation of P waves and QRS complexes, there is a slow ventricular escape rhythm, and the arrhythmia is non-responsive to atropine.

PREOPERATIVE PREPARATION

It is essential to prepare thoroughly for cardiac and pericardial surgery. The correct instrumentation should be available for the surgeon (see Chapter 13). The anesthesia technique selected, the ability to be able to continually monitor anesthesia and respond to changes, the need for intermittent positive pressure ventilation (IPPV), and the ability to be able to provide appropriate levels of postoperative care are integral to the success of any thoracic surgery (see Chapters 2 and 3).

Surgical approaches to the heart.

The heart and associated structures can be approached from intercostal thoracotomies (see Chapter 41, Box 41-4) and median sternotomy (see Chapter 41, Box 41-5). The approach will depend on the procedure that is to be performed. Access to the apex of the heart for pacemaker lead implantation can also be achieved through a cranial midline celiotomy combined with caudal median sternotomy or a transdiaphragmatic incision.

Intercostal thoracotomies performed through the left 4th, 5th or 6th intercostal spaces have all been reported to achieve good access for surgical ligation of PDAs in cats.[2,23,24] In dogs a 4th intercostal space is the recommended approach; in cats the 4th or 5th space is generally recommended[25] due to the slightly more caudal location of the heart in this species.

Approach to the pericardium

Pericardectomy can be performed via several approaches. The final decision will depend on the indication for surgery and surgeon preference. If the primary reason for performing thoracic surgery is pericardectomy, a 5th left or right intercostal thoracotomy is recommended; excellent access is also obtained by median sternotomy. If a lateralized heart base mass is confirmed in a patient with a PE, the thoracotomy should be performed on the side of the mass. If a heart base mass is suspected, or has been identified involving both sides of the heart base, a median sternotomy may be the preferred approach.[26] A median sternotomy gives good access to both the left and right sides of the heart and allows easier identification of both phrenic nerves at surgery. Thus, pericardectomy may be technically easier via median sternotomy. A median sternotomy also reduces the requirement for manipulation of the heart during surgery, and thus reduces the likelihood of associated intraoperative arrhythmias and it allows exploration of both the right and left sides of the heart base if required.

When pericardectomy is performed as an adjunct to thoracic duct ligation (TDL) for the treatment of chylothorax it is usually possible to perform pericardectomy from an 8th, 9th or 10th left intercostal thoractomy (i.e., the same approach through which TDL is performed). If the access achieved from the caudal thoracotomies used for TDL is inadequate for pericardectomy a separate 5th left intercostal thoracotomy incision can be made.

Pericardectomy can also be performed thoracoscopically (see Chapter 42, Box 42-2).

Access to the apex of the heart for epicardial pacemaker implantation has been achieved via a 5th or 6th left or right lateral thoracotomy, midline cranial celiotomy and caudal sternotomy, and a cranial midline celiotomy and transdiaphragmatic approach.[27–30] The latter gives adequate access to the apex of the heart and is a less traumatic procedure for the patient, avoiding some of the additional postoperative management considerations and potential complications associated with thoracotomy and sternotomy.

SURGICAL MANAGEMENT AND TECHNIQUES

Ligation of a patent ductus arteriosus

It is widely accepted that prompt surgical treatment (Box 48-3) should be performed in young patients diagnosed with PDA to avoid the development of CHF or pulmonary hypertension. There are two described methods of open PDA ligation: the conventional approach and the Jackson–Henderson approach (Box 48-3).

The use of a vascular clip to occlude a ductus has been reported in a cat where safe dissection around the far wall of the ductus was considered impossible[3] and more recently for PDA occlusion in dogs.[32,33] It appears to be a suitable alternative to suture ligation and avoids 'blind' dissection around the far side of the ductus.

The use of vascular occluding devices is extensively reported for treatment of PDAs in dogs and has a similar rate of complications and outcome whilst avoiding an invasive surgical procedure. The femoral artery is generally used for catheter introduction to place the occluding device. This vessel is very small in cats, restricting the size of introducer or delivery catheter that can be placed and several authors have suggested that feline PDAs are only amenable to surgical ligation.[34,35] Transvenous embolization with detachable coils has been reported for successful occlusion of PDAs in two cats using the femoral vein to gain vascular access, and this technique may prove to be a good alternative to surgical closure of PDAs in cats.[36]

Subtotal pericardectomy

Subtotal pericardectomy is performed for PEs, restrictive pericarditis, symptomatic pericardial cysts, congenital or acquired pericardial defects, with herniation of the heart, and chylothorax. Generally, subtotal pericardectomy (Box 48-4) is performed via a right or left 5th intercostal thoracotomy although a median sternotomy can also be used.

Box 48-3 Ligation of patent ductus arteriosus

A 4th or 5th left intercostal thoracotomy is made (see Box 41-4). The left cranial lung lobe is reflected caudally using a saline soaked swab. The ductus is identified between the aorta positioned dorsally and the pulmonary artery ventrally. If not immediately evident, the ductus should be easily identified by palpation, as obvious fremitus will be present. The vagus nerve is identified as it runs over the ductus, and several stay sutures are placed in the mediastinal pleura above or below the vagus nerve. The mediastinal tissue is carefully incised and the vagus nerve is retracted dorsally or ventrally, respectively. The caudal aspect of the ductus is dissected using roght-angled forceps (Mixter forceps), passed parallel to the transverse plane. Dissection may be entirely external to the pericardium, or extended so that the pericardium is opened, depending on the individual ductus anatomy. The cranial aspect of the ductus is dissected with the tips of the forceps angles approximately 45° caudally. Dissection is continued caudally around the medial ductal wall; this must be performed with extreme care and patience to avoid damage to the vessels. A double strand or loop of 3M or 4M silk is soaked in saline and knotted near one end. Once dissection around the medial aspect of the ductus is complete and the tips of the forceps can be seen at the caudal border of the ductus, the knotted suture material is placed between the tips of the forceps and is very carefully passed around the ductus by slowly withdrawing the forceps. The knot is then cut and removed.

Ligation

The suture on the aortic side is tied first. As the PDA is ligated, in response to the pressure changes there may be a vagally mediated bradycardia (Branham's sign) so an anticholinergic agent such as atropine should be available for administration in this event. It is recommended that the ligature is tied slowly over several minutes to avoid this reflex. The suture on the pulmonic side is then tied and it is confirmed by auscultation using an esophageal stethoscope that the murmur has resolved. There should now be no fremitus on digital palpation. Stay sutures and the swab retracting the left cranial lung lobe are removed and the thorax is lavaged with warm sterile saline. A thoracostomy tube is placed (see Box 41-6) and closure of the thorax is routine.

The Jackson–Henderson approach[31]

The Jackson–Henderson approach is an alternate technique of PDA ligation that avoids dissection of the medial aspect of the PDA. The ductus is dissected on the cranial and caudal aspect as described above. The mediastinal pleura is incised between the origin of the left subclavian artery to the origin of the first intercostal artery dorsal to the aorta and the connective tissues on the medial aspect of the aorta at this level are bluntly dissected. A pair of right-angled forceps is passed immediately cranial to the ductus and around the medial aspect of the aorta so their tips can be seen dorsal to the aorta at the site where the mediastinum has been dissected. A knotted loop of 3M or 4M silk is placed in the tips of the forceps and the forceps are gently removed so that the suture material passes from dorsal to ventral around the medial aspect of the aorta immediately cranial to the ductus. This dissection around the medial aspect of the aorta is repeated immediately caudal to the ductus, the free end of the suture material passed into the tips of the forceps, and as they are withdrawn the suture material is taken around the caudal aspect of the ductus. Ligation then proceeds as described above.

Total pericardectomy

Although total pericardectomy (Box 48-5) may be appropriate in the management of some neoplastic or infectious pericardial diseases, it is very rarely indicated. It is a more challenging surgery with a greater risk of serious complications than subtotal pericardectomy.

Box 48-4 Subtotal pericardectomy

For subtotal pericardectomy the cat is positioned in lateral recumbency and a right or left 5th intercostal thoracotomy performed (see Box 41-4). A T-shaped incision is made in the pericardium: the base of the T extends from the apex of the heart, and the arms of the T extend around the circumference of the heart just ventral to the phrenic nerves. Stay sutures can be placed in the pericardium just ventral to the phrenic nerve to aid dissection and manipulation. If the pericardium is thickened the pericardial vessels can be hypertrophied and hemorrhage from these vessels can be marked so use of electrocautery applied to individual vessels, or in a cutting/coagulating mode, or use of a vessel sealing device prior to cutting the pericardium is recommended (see Chapter 13). The heart can gently be retracted by an assistant to aid access to the contralateral side of the heart if performing pericardectomy via intercostal thoracotomy. The contralateral phrenic nerve must be identified and preserved. This manipulation can cause arrhythmias and temporary reduction in venous return and cardiac output. The caudoventral mediastinal reflection is ligated, or alternatively a vessel sealing device can be applied to this structure to achieve hemostasis and this tissue is then divided.

After subtotal pericardectomy the excised pericardium is submitted for histopathologic analysis, and bacterial and/or fungal culture if considered appropriate. The pleural cavity is lavaged with sterile saline and the remaining pericardium is checked to ensure there is no ongoing hemorrhage. A thoracostomy tube is placed (see Chapter 41) and closure of the thorax is routine. The thoracostomy tube is drained on closure of the thorax, and again once the patient is positioned in sternal recumbency in recovery.

Epicardial pacemaker implantation

Anesthesia can worsen bradycardias and it is thus recommended that patients undergoing pacemaker implantation have temporary pacing under anesthesia until the permanent pacemaker has been implanted. Transthoracic pacing patches attached to an external pacer are useful in small patients, although their use results in patient movement, but neuromuscular blockade reduces this.

The author recommends a cranial midline celiotomy and transdiaphragmatic approach (Box 48-6). This gives good access whilst avoiding the invasiveness and potential complications of sternotomy or intercostal thoracotomy. If access is inadequate, the celiotomy incision can be extended to a caudal sternotomy.

Alternate approaches for epicardial pacemaker implantation include modifying the transdiaphragmatic approach by extending the cranial celiotomy by the addition of a caudal sternotomy or placement via a 5th or 6th left or right thoracotomy. A standard intercostal approach is made and the epicardial lead is secured as described for a midline celiotomy/transdiaphragmatic approach. The lead is exited through a stab incision made in the intercostal muscles at an intercostal site one to two intercostal spaces caudal or cranial to the site of surgical entry into the thorax. Several sites of generator placement have been described including a subcutaneous site overlying the craniolateral abdomen, the caudal thorax, and with and without suture anchorage to local structures such as an adjacent rib. A second incision may be necessary to implant the generator when using an intercostal approach for pacemaker placement. An alternate site for generator placement and anchorage, if the latissimus dorsi was retracted dorsally rather than incised during thoracotomy, is under this muscle adjacent to the thoracotomy site (but not overlying it). The generator can then be sutured to the external thoracic muscles.

Box 48-5 **Total pericardectomy**

This surgery for total pericardectomy is performed with the cat in dorsal recumbency via median sternotomy (see Box 41-5). The phrenic nerves are identified as they cross the pericardium. They are preserved by very careful dissection to free them from the pericardium; fine ophthalmic surgical scissors may be helpful to perform this delicate dissection. The apex of the heart is gently shifted to the right side of the thorax to aid access to the left heart base using the surgeon's or surgical assistant's fingers, or a malleable retractor wrapped in a saline-moistened surgical sponge, and likewise the apex is gently shifted to the left to improve access to the right side. The apex of the heart is gently moved in a cranial direction to access the caudal heart base. The anesthetist should be warned prior to these manipulations as they can result in a reduction in cardiac output or arrhythmias. Dissection should be temporarily halted to allow recovery (and adjustment of retraction if necessary) in the event of reduced cardiac output and/or arrhythmias. Saline-moistened umbilical tape is placed around the phrenic nerves once they have been freed from the pericardium, so they can gently be retracted away from the heart. The pericardium is incised/entered towards the apex of the heart and any PE is removed by suction. The pericardium is then incised towards the heart base. Once nearing the base of the heart, stay sutures are placed in the pericardium on either side of the incision to allow retraction and aid exposure so that the great vessels can clearly be identified and avoided. The pericardium is then dissected circumferentially around the heart base, close to the great vessels[26] leaving a small remaining 'rim' of pericardium (approximately 5 mm). Great care must be taken to avoid the heart base and great vessels being injured by sharp dissection, or by thermal trauma if using electrocautery or a vessel sealing device to achieve hemostasis of the pericardial vessels.

After total pericardectomy the excised pericardium is submitted for histopathologic analysis, and bacterial and/or fungal culture if considered appropriate. The pleural cavity is lavaged with sterile saline and the remaining rim of pericardium is checked to ensure there is no ongoing hemorrhage. A thoracostomy tube is placed (see Chapter 41) and closure of the thorax is routine. The thoracostomy tube is drained on closure of the thorax, and again once the patient is positioned in sternal recumbency in recovery, and thereafter as necessary.

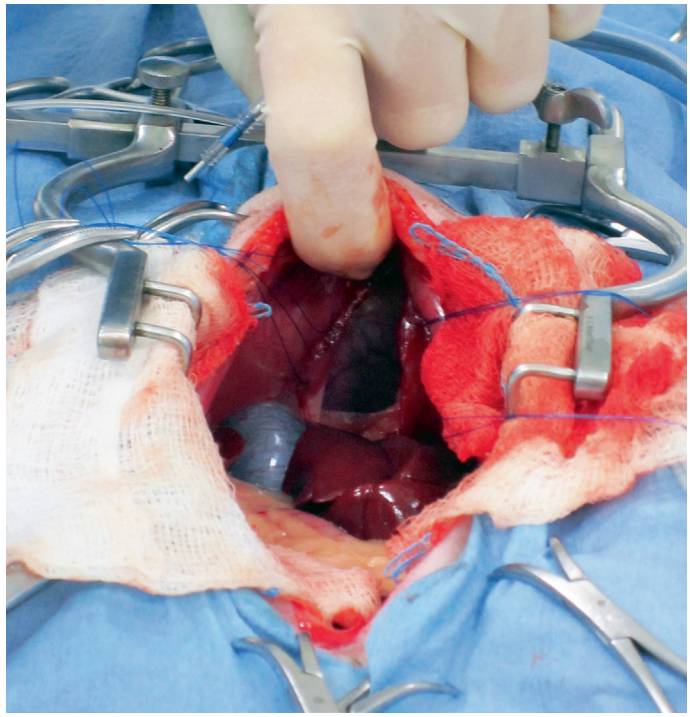


Figure 48-7 Intraoperative view during the approach for epicardial electrode placement by a caudal celiotomy and transdiaphragmatic approach. The liver and gall bladder are seen immediately caudal to the diaphragm which has been incised and is being retracted by stay sutures to aid exposure.

Box 48-6 **Epicardial pacemaker implantation through a cranial celiotomy and transdiaphragmatic approach**

An incision is made from the xiphoid and extending caudally up to the umbilicus. Self-retaining retractors are placed to facilitate access. Neuromuscular blockade may also facilitate surgery. The diaphragm is incised at its midline attachment to the sternum or just to the left of the midline. This incision is extended towards the central tendon. Stay sutures are placed in the diaphragm on either side of the incision to allow retraction and aid exposure (Fig. 48-7). The apex of the heart is readily identified, the pericardium is grasped at its apex using forceps and a 1–2 cm incision is made to allow access to the apex of the heart. Stay sutures can be placed in the pericardium on either side of the incision to aid manipulation. An epicardial pacemaker lead is placed in an avascular area at the apex of the left ventricle using either a non-sutured screw-in corkscrew electrode (according to manufacturer directions) or a sutured electrode (Fig. 48-8). If using a sutured technique, the electrode is anchored to the heart with three or four sutures (depending on the actual electrode being used) of 2M polypropylene; pre-placing the sutures before tying is helpful. If the incision in the pericardium is small and herniation of the heart through this defect is impossible it can be left unsutured, otherwise it is repaired using 1.5M monofilament absorbable material placed in a simple continuous pattern. The end of the lead is exited from the thorax through a small stab incision through the diaphragm lateral to the incision made to enter the thorax, leaving a loop of epicardial lead within the pleural cavity to allow for cardiac and respiratory movements, activity, etc., in the conscious patient and thus avoid tension on the lead. The pulse generator is connected to the lead and covered in a saline-soaked swab, or surrounded by abdominal tissues (e.g., omentum) to activate the system (which is unipolar, requiring contact with the body).

Any external pacing is stopped and it is confirmed that the implanted system is achieving pacing as planned, and threshold tests and impedance can be interrogated with an appropriate programmer. The lead can be anchored with a suture to the diaphragm at its entry to the peritoneal cavity but if doing this care must be taken not to

Box 48-6 **Continued**

damage the insulation layer of the lead. The diaphragmatic incision is closed routinely and the pleural cavity is evacuated via needle or cannula drainage across the diaphragm.

Alternatively, a thoracostomy tube can be placed. Some reports describe leaving the pulse generator free within the abdomen, but generally it is recommended that the generator is anchored in a 'pocket' within the abdominal wall. A pocket just large enough to accommodate the transducer is made in the left cranioventral abdominal wall between the transversus abdominis and internal abdominal oblique muscles (Fig. 48-9). A loop of lead is left within the abdomen to avoid tension on the fixation, as above. Any excess lead is loosely coiled around the generator. Usually the generator has a small hole at an edge to allow it to be sutured and anchored to the local tissues. The incision in the internal abdominal oblique is closed using 2M or 3 M polydioxanone placed in a simple continuous pattern. Abdominal closure is routine. A postoperative radiograph of the lateral and/or dorsoventral thorax/abdomen is taken prior to recovery from anesthesia to confirm the electrode, lead, and pacemaker locations and to confirm that the lead is not kinked (Fig. 48-10).

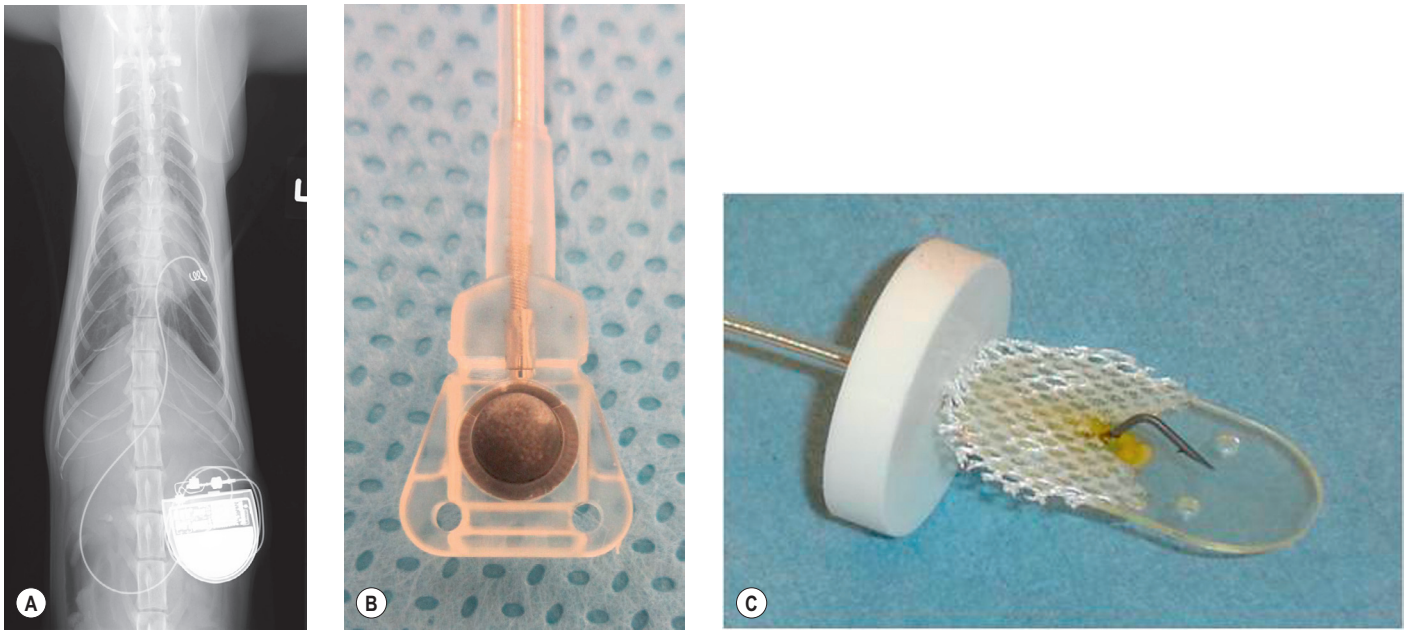


Figure 48-8 Electrodes that can be used for pacemakers in cats. **(A)** A non-sutured screw-in electrode has been used in this cat, that is 'self' anchored to the left ventricular wall at the apex. This postoperative radiograph also shows the transducer placed within the abdomen. Excess lead has been coiled around the transducer. The lead and transducer are then tucked into a pocket made between the internal abdominal oblique and transverse abdominal muscles of the left body wall. **(B)** Epicardial electrode. This type of electrode is placed onto the left ventricle at its apex and is anchored to the heart by sutures placed between holes in the end of the electrode and the left ventricular muscle. An additional suture is placed around the base of the electrode through the left ventricular muscle, providing additional anchorage. **(C)** Epicardial electrode. This type has a barbed point that is embedded into the left ventricular muscle at the apex; the electrode is then secured to the left ventricle by placing sutures through the holes in the end of the electrode to the ventricular muscle. The circular white disk at the base of the electrode is removed prior to implantation.

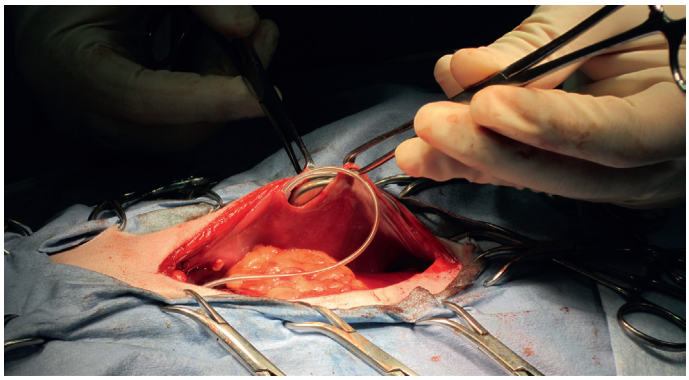


Figure 48-9 A pocket is made in the left cranioventral abdominal wall between the tranversus abdominis and internal abdominal oblique muscles for implantation of the pacemaker.

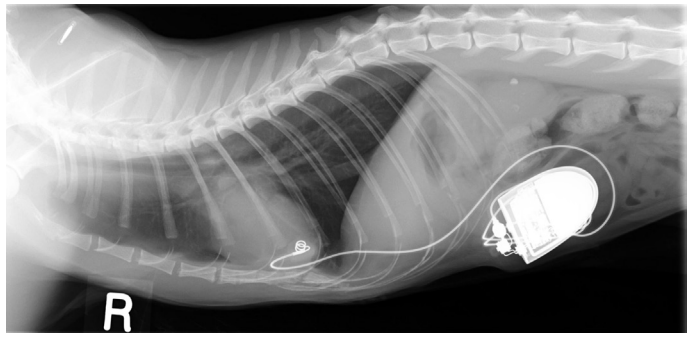


Figure 48-10 A postoperative radiograph is taken to confirm correct electrode, lead, and pacemaker locations and to confirm that the lead is not kinked.

POSTOPERATIVE CARE

General postoperative considerations after heart surgery include monitoring respiration (rate, effort), heart (arrhythmias, murmurs, rate), analgesic requirements, and management of the thoracostomy tube. Postoperative nursing after thoracic surgery and postoperative monitoring are covered in Chapters 3 and 4.

Thoracostomy tube

The thoracostomy tube is drained immediately on closure of the thorax and again once the patient is positioned in sternal recumbency in recovery. The thoracostomy tube is maintained whilst there is a significant volume of fluid being aspirated. For information about when to remove the tube see Chapter 41. Minimal fluid and air production is expected following uneventful PDA ligation and the thoracostomy tube can usually be removed in recovery or within several hours of completion of surgery. Intravenous fluid therapy with crystalloids is continued cautiously (2–4 mL/kg/hour) after heart surgery to avoid volume overload until patients are eating in recovery.

COMPLICATIONS AND PROGNOSIS

Complications after heart and pericardial surgery can range from minor and treatable to serious or even fatal, the most common including hemorrhage, pain, arrythmias, and cardiac arrest.

Patent ductus arteriosus

There is little data available in cats, but the anticipated potential complications are those reported in dogs. The most serious, potentially fatal intraoperative complication is rupture of the PDA, aorta or pulmonary artery during dissection around the ductus. Small tears to these vessels may be managed by the immediate application of digital pressure, but continued dissection may result in further hemorrhage as the tear in the damaged vessel may enlarge. A decision must be made whether to continue with surgery, or abandon the procedure on this occasion but second surgeries can be difficult due to scarring and fibrosis at the surgical site. If hemorrhage is marked, a vascular occlusion clamp (Satinsky) is applied to the aorta cranial to the ductus and caudal to the brachycephalic and left subclavian arteries. This allows blood flow to continue to the head and heart. Another vascular clamp is placed across the aorta caudal to the ductus to prevent retrograde aortic hemorrhage. Hemorrhage from the ductus will now be from the pulmonary artery, which is a lower pressure system so hemorrhage should not be as severe. Various techniques are suggested for resolving intraoperative hemorrhage including vascular repair using 1M polypropylene if the tear can be identified, ductal closure using polytetrafluoroethylene felt pledget buttressed mattress sutures, or ductal division between clamps and oversewing of the ductal edges.

In dogs, surgical mortality of less than 2% is achievable by experienced surgeons.[37–39] There is inadequate information available in cats to report an expected surgical outcome although the mortality rates are higher than in dogs for the few feline cases reported to date.

Outcome in dogs is excellent, but residual shunting has been reported in over 20% of cases.[40] Ductal division and oversewing of ductal ends prevents residual flow but is not recommended because it is more difficult than ligation and the risk of fatal intraoperative hemorrhage is not justified against the potential benefits gained.

Other rare postoperative complications in dogs include rupture of an aortic aneurysmal dilation with fatal hemorrhage, recanalization of the ductus with recurrent blood flow through the shunt, and bacterial colonization of the ductus, none of which have been reported in cats.

Pericardectomy

Failure to seal/ligate pericardial blood vessels cut during pericardectomy will result in postoperative hemorrhage, and occasionally a further surgery may be necessary to address this hemorrhage.

Epicardial pacemaker

Complications associated specifically with epicardial pacemaker implantation include displacement of the pacemaker electrode with accompanying loss of pacing, infection, seroma, and migration of the generator.

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