



Cardiovascular Emergencies Mini Series

Session Two: Pericardial Diseases Leading to Collapse

Rachel James MA VetMB CertSAM DVC MRCVS
RCVS Recognised Specialist in Veterinary
Cardiology





James Specialist Veterinary Cardiology

Pericardial Disease

Drugs don't work

Rachel James MA Vet MB Cert SAM Cert VC DVC MRCVS

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Pericardial disease is much more widely recognised these days due to the easy access to ultrasound, however the diagnosis is still a difficult one as this family diseases are still relatively uncommon and frequently overlooked in practice. A through clinical examination is mandatory in these cases and then echocardiography. The experienced clinician will always suspect pericardial effusion in any dog or cat with ascites and pleural effusion especially when the heart sounds are muffled and there is jugular distension present.

Anatomy of the Pericardium

The heart is enveloped in a tough outer fibroserous membrane, the pericardium which is composed of two layers:

- Fibrous pericardium
- Serous pericardium

Fibrous pericardium

- Stronger
- Attached to the diaphragm and adventitia of the large vessels
- Phrenopericardial ligament

Serous pericardium

- Two layers (visceral or epicardium & parietal)
- Line the inside of the fibrous pericardium and covers the heart

Pericardial cavity

- Between the two layers of the serous pericardium
- Small amount of serosanguineous fluid (about 0.5 to 1.5 ml), rich in phospholipids and this lubricates the interior surfaces of the pericardial sac. It is not uniformly distributed within the pericardial space, rather it tends to collect at the heart base and in the atrio-ventricular grooves with only a very thin layer over the ventricular free walls.

The parietal pericardium's blood supply is from branches of the internal thoracic artery, the aorta and musculo-phrenic arteries. The epicardium is supplied by branches of the coronary arteries. Lymphatic drainage of the pericardial sac occurs from vessels emptying into the pre-tracheal and cardiac lymph nodes, vessels running alongside the phrenic nerve and vessels returning to the sternal lymph node. The processes controlling pericardial fluid production and removal are poorly understood. In most circumstances pericardial effusion occurs due to haemorrhage or increased capillary permeability rather than reduction in lymphatic drainage.

The function of the pericardium is not completely understood. It protects the heart from surrounding infection or malignancy and helps maintain the heart in its correct position within the chest. It also helps diastolic ventricular coupling and therefore prevents cardiac over-distension. Because the pericardium is non-compliant and its reserve volume is small, intra-pericardial pressure rises rapidly with a small increase in volume especially in an acute situation. Increased pericardial pressure leads to a reduction in diastolic filling of the ventricles and hence a reduction in cardiac output. If the increase in pericardial volume is slow and chronic then the pericardium can hypertrophy to accommodate this change in volume with subsequently only small increases in intra-pericardial pressure. However there is still a small increase in intra-pericardial pressure which is compensated in part by an increased central venous pressure in order to maintain cardiac filling pressures (thus leading to right sided heart failure) and an increased heart rate; with these compensatory mechanisms the dog can maintain an adequate cardiac output in the short term.

Pericardial disease can be congenital or acquired but the pathophysiology is very similar in both cases.

- Impaired ventricular filling as a consequence of increased intrapericardial pressures
 - Fluid accumulation, presence of a mass or pericardial constriction.

This become significant when

- Cardiac tamponade occurs
 - Intrapericardial pressure equilibrates with the right atrial and right ventricular filling pressures
 - Continuum that ranges from subclinical to fulminant heart failure
 - Congestive right heart failure results from chronic tamponade (where there is an increase in central venous pressure to try and maintain cardiac filling)
 - Low cardiac output and shock results from acute tamponade (ie when there is a sudden increase in intra-pericardial pressure which is very close to the filling pressures making cardiac filling very difficult)
- Pericardial constriction
 - Decrease in pericardial compliance so it is difficult for the ventricles to fill beyond a certain volume.

What can go wrong with the pericardium?

- Pericardial sac can fill with fluid:
 - Blood
 - Transudate
 - Exudate
- Mass can be present:
 - Within the pericardial space
 - Within the heart
 - Within the pericardium
- The pericardium can become stiff – **constrictive**

Fluid within the pericardium

- Blood
 - Lower PCV typically 6% - Common
 - No correlation with causative pathology
 - Same PCV as systemic blood
 - Coagulopathy
 - Rupture of the LA, laceration of coronary arteries, RTA trauma
- Transudate
 - CHF especially in cats – do not need draining
 - FIP
- Exudate
 - Bacterial – FB, bite wounds, pulmonary infections, perforated oesophagus
 - Fungal

Pericardial disease maybe acquired (usually neoplastic) but can be idiopathic, iatrogenic, secondary to coagulopathies, trauma or infectious agents. Congenital pericardial disease does occur though rarely.

Pathophysiology of pericardial effusion

- This is a pressure game NOT volume
- The heart must fill with blood before it has any to pump out
- Impaired ventricular filling as a consequence of increased intrapericardial pressures (not volume)
 - Fluid accumulation
 - presence of a mass
 - pericardial constriction

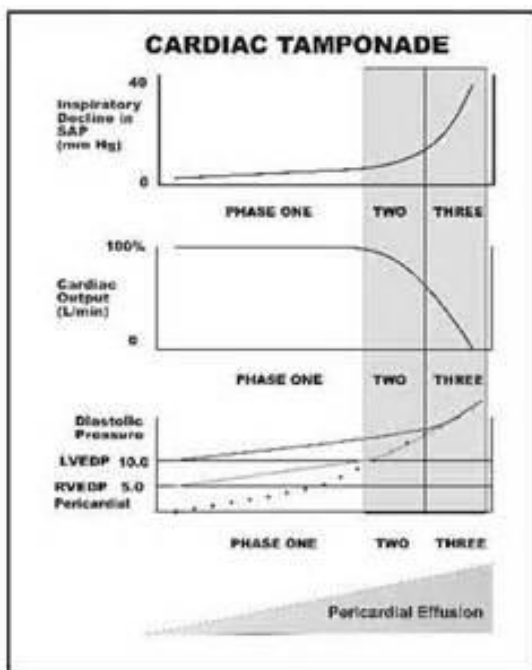
Cardiac Tamponade

This is not an all or nothing phenomenon. It is a graduated process. There are haemodynamic consequences as soon as the pressure within the pericardium is more than zero, as the pericardial effusion (PE) accumulates it leads to an increase in PE pressure which then leads to an increase in the filling pressures in RV and LV.

There are three main phases of cardiac tamponade

- Mild compromise
 - Increase in pericardial pressure but lower than right sided filling pressure

- Right heart tamponade
 - PP is $>$ or $=$ to right sided filling pressures
 - Exercise intolerance , signs of systemic venous congestion
- Left sided tamponade
 - PP is $>$ or $=$ to left sided filling pressures
 - Severe compromise of CO
 - Weak femoral pulses
 - Pulsus paradoxus



A diagram to show the 3 phases of cardiac tamponade

Pericardial constriction

This is a restriction of pericardial filling secondary to reduced pericardial compliance. This can be extremely difficult to diagnose as the heart and pericardium at first appearance can appear normal and a detailed echocardiographic examination is required for diagnosis. Measuring central venous pressure in these cases can also be helpful.

Congenital pericardial diseases

These are rare in general practice and include peritoneopericardial diaphragmatic hernia, intra-pericardial cysts and intra-pericardial cysts (dogs only) and complete or partial defects in the pericardium. Most of these are amenable to surgical correction as clinically indicated.

Peritoneopericardial diaphragmatic hernia (PPDH)

This is the most common congenital pericardial abnormality, the prevalence is difficult to be sure about as many animals are never diagnosed as they are often asymptomatic.

It results from an abnormal development of the dorsolateral septum transversum or from failure of the lateral pleuroperitoneal folds and the ventromedial pars sternalis to unite. It can occur as an isolated deformity or in association with umbilical hernias and other congenital cardiac abnormalities. The clinical signs vary depending on which organs have been displaced into the pericardial sac and how compromised their function and blood supply are.

Diagnosis is usually straightforward with thoracic radiographs and ultrasound and treatment is surgical correction.

Acquired Pericardial diseases

These lead to pericardial effusion and cardiac tamponade. The most frequent cause is neoplasia

- Haemangiosarcoma, mesothelioma, chemodectoma, metastatic tumours – thyroid, osteosarcoma, mast cell tumours, lymphoma etc
- Lymphoma – cats

Idiopathic pericardial effusion does occur (40%-50%) of cases and for recurrent cases pericardectomy is curative.

Other less common causes would include coagulopathies, infection (bacterial and fungal), uraemia (these PE tend to be small and unlikely to require drainage) iatrogenic and atrial rupture.

Clinical presentations

The typical history of these dogs depends on the pathophysiology but two common types of presentation:

- Acute cases
 - Sudden onset exercise intolerance, weakness, collapse, shock, rapid death if untreated
- Chronic cases – more common
 - 2 week history of ascites, progressive exercise intolerance, lethargy, GIT signs, collapse

Physical examination usually shows the following:

- Jugular distension
- Positive hepatjugular reflex
- Ascites
- Tachycardia
- Muffled heart sounds
- Weak femoral pulses (+/-pulsus paradoxus)
- Pale mucous membranes
- Tachypnoea / dyspnoea
- GIT signs

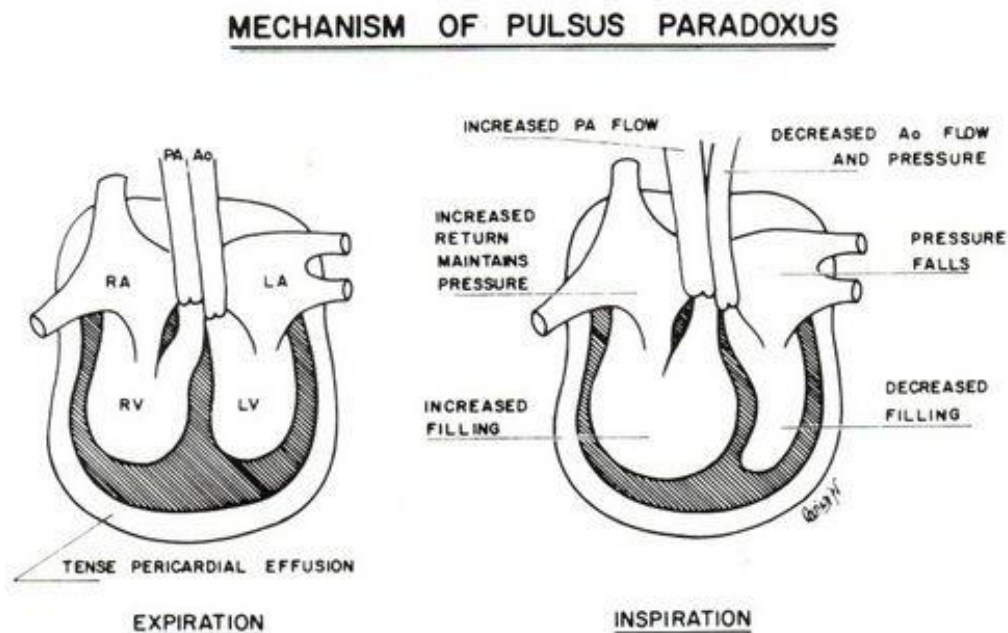
With the classical triad of clinical signs:

- Muffled heart sounds
- Right sided heart failure
 - Ascites
 - Distended jugular veins
 - Positive hepatojugular reflex
- Forward failure
 - Poor peripheral pulses
 - Paradoxical pulse or *pulsus paradoxus*

Pulsus Paradoxus

This change in pulses is not “paradoxus” and not pathognomonic of cardiac tamponad but just an exaggeration of a normal process

- During inspiration there is a decrease in intrathoracic pressure leading to preferential blood flow into the RA, RV and pulmonary veins (these are the most compliant blood vessels and cardiac chambers) this leads to reduced pre load to the left side of the heart.
- Due to ventricular interdependence there is a fixed volume for the ventricles to fill. As they fill the volume they fill to depends on the filling in each ventricle.



The correct definition of pulsus paradoxus is:

A difference of more than 10mmHg in the systemic blood between inspiration (lower) and expiration (higher).

Electrocardiography (ECG)

Typically on ECG we would see:

- Tachycardia
- Small complexes
- Electrical alternans



Echocardiography

This is the gold standard for diagnosis or not only the presence of pericardial disease but the exact aetiology.

- Effusion, mass, hernia, cyst
- Important to perform echo prior to drainage IF patient is stable enough

In the acute collapse dog a DIAGNOSIS is needed IMMEDIATELY these cases wont wait

- Brief history, PE and emergency US
- Shock rate i/v fluids
- Oxygen
- Prepare for pericardiocentesis
- NEED TO BUY TIME FOR:
 - Detailed ultrasound scan
 - Discussion with the owner

Radiographs

- Globoid enlargement of the cardiac silhouette with a sharp outline
- Herniation of intestinal organs or cystic lesion



These cases need pericardiocentesis, drugs won't work. The technique for drainage is straightforward.

Pericardiocentesis

- Therapeutic & diagnostic

Drainage can be from either side with arguments for and against both methods as shown below:

- Left lateral recumbency
 - If penetrate the ventricle the left will heal faster and will be obvious as high pressure well oxygenated blood will be apparent.
- Right lateral recumbency
 - Avoid laceration of main coronary vessels
 - The right side has a larger cardiac notch

With either side the technique is otherwise the same:

- Between the 4th and 6th intercostal space at the level of the costochondral junction (elbow crosses the costochondral junction) of use ultrasound to guide you to the largest pocket at the CC junction.
- GA (or Sedation- but this is usually more time consuming and the clinician has less control and requires the use of local anaesthetic), aggressive fluid therapy, ECG monitoring
- Surgical prep and (local anaesthetic if sedated rather than GA)
- Several different catheter types may be used.

Pericardiocentesis is a safe procedure (risk inversely related to the amount of effusion)

- Complications
 - Cardiac puncture
 - Arrhythmias
 - Dissemination of infection or neoplasia
 - Atrial fibrillation
 - Myocardial stunning

See lecture for detailed method.

Post Pericardiocentesis

There is rapid filling and dilation of the RA and RV post pericardiocentesis. There is likely to be an increased blood volume due to heart failure and i/v fluids and therefore you may need to give one dose of furosemide to reduce fluid overload (1mg/kg). In some dogs there is a risk of arrhythmias post drainage – AF and ventricular arrhythmias. Very rarely myocardial stunning may occur. This is characterised by poor systolic function for a few days post drainage and may require treatment with pimobendan and other cardiac failure medications depending on the severity and presence of pulmonary oedema or not. This is usually transient and resolves after a few days – weeks.

Recurrence of the pericardial effusion often occurs depending on the aetiology but most with a neoplastic origin will recur and some idiopathic cases. What options are available?

- Repeat pericardiocentesis
 - Risk of constrictive pericardial disease developing
 - Risk of the procedure
 - For neoplastic aetiology can use a balloon to make a pericardial window – palliative procedure only
- Pericardial strip

Normally I would advise a maximum of 3 pericardiocentesis and then pericardectomy.

Pericardial disease is an uncommon disease in small animal practice, but it is so important in any collapsed or ascitic dog, that it is considered as a differential, as drugs at best will do no good and at worst can be fatal especially furosemide in these cases. Diagnosis with ultrasound is relatively straightforward although differentiating the different causes can be more problematic. Pericardiocentesis is a straight forward procedure and is life-saving.