# Pericardial Effusion in Dogs

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## Abstract

Pericardial disease in dogs is relatively uncommon, but its presence may be life-threatening. It is an incidental finding or manifestation of a systemic or cardiac disease. The spectrum of pericardial effusions ranges from mild asymptomatic effusions to cardiac tamponade. Pericardial effusion in dogs has an overall prevalence of 0.43% in general and occurs in approximately 7–10% of the dogs with clinical signs of cardiac disease. More commonly seen in dogs, the clinical signs of pericardial disease can be easily overlooked or mistaken for those of other disease processes. In patients with life-threatening pericardial effusion, which has led to cardiac tamponade, it is important to rapidly identify and treat the elevated intrapericardial pressure. Management is guided by the haemodynamic impact, size, presence of inflammation (i.e. pericarditis), associated medical conditions, and the aetiology whenever possible. In this chapter, pericardial effusion in dogs is emphasized.

**Keywords:** dogs, pericardial effusion, aetiology, pathophysiology, clinical signs, diagnosis, pericardiocentesis, management of pericardial effusion

## 1. Introduction

The thoracic cavity contains two pleural cavities and a third serous membrane-lined space, the pericardial cavity. The pericardium composed of fibrous and serous layers of the pericardial cavity is situated within the mediastinum. The parietal pericardium forms the superficial wall of the pericardium and is composed of an external fibrous layer and an internal serous layer. The pericardial cavity normally contains only a thin film of fluid positioned between parietal and visceral layers of serous pericardium [1]. These membranes provide the heart with a sac-like protective compartment. The normal pericardial sac contains 2–10 mL of clear, thin, serous fluid that acts primarily as a lubricant. If the fluid accumulates more than the limit in the pericardial sac, it is defined as pericardial effusion. The effusion usually accumulates slowly and progressively and presents with sign of chronic cardiac tamponade and right-sided heart failure with lethargy, exercise intolerance, or collapse [2].

Further, a rapid accumulation of fluid into the pericardial sac results in a rapid increase in intrapericardial pressure. If the intrapericardial pressure exceeds the pressure within the right atrium, acute compression of the heart (cardiac tamponade) and impairment of ventricular filling may develop. Compression of the heart by this pressure causes a drop in preload and reduces cardiac output [3]. Pericardial effusion usually occurs in middle to older dogs with a mean age of 6 years in large-breed dogs; however, it is found in age groups of 1–14 years. Neoplastic pericardial effusion is seen in brachycephalic breeds and short breeds.

German shepherds, golden retrievers, Great Danes, and Saint Bernards appear to be predisposed to idiopathic pericardial effusion. Both sexes are said to be affected. The most common causes of pericardial effusion in dogs are cardiac neoplasia, right-sided heart failure, cardiac rupture and idiopathic pericarditis and less commonly congenital pericardial disorders, trauma, or infectious processes [4].

## 2. Aetiology

When a clinician is faced with a dog, which is presented with pericardial effusion, the first challenge is to identify its aetiology. The aetiology of pericardial effusion is idiopathic (unknown cause), congestive heart failure, infectious, neoplasia (cancer), congenital and others (atrial rupture, systemic, hypothyroidism, rarely cirrhosis, etc.).

### 2.1 Idiopathic pericardial effusion

The unknown causes (idiopathic) of pericardial effusion in dogs were accounting for 20–75%. This disorder is more common in large- and giant-breed dogs and is thought to be an inflammatory condition affecting the pericardial sac. One-time pericardiocentesis is curative in about half of cases. If multiple taps are required, pericardiectomy is recommended and is usually curative. The diagnosis is arrived after exclusion of possible causes of intrapericardial fluid accumulation thorough echocardiographic examination.

### 2.2 Congestive heart failure

Although congestive heart failure is a common cause of pericardial effusion in cats, it is a less common cause in dogs. When congestive heart failure leads to intra-pericardial fluid accumulation, cardiac tamponade is rare, and pericardiocentesis is almost never indicated. However, in few cases, modification of congestive heart failure medications may be warranted.

### 2.3 Infectious

Pericardial effusion due to infectious cause is not so frequently observed. It may include multiple causes, viz. bacterial infection secondary to migrating foreign bodies. The most commonly isolated bacteria associated with the condition are *Actinomyces*, *Nocardia* [5] and *Coccidioides immitis* [6].

Further, constrictive and septic pericarditis also results in minimal pericardial effusion in dogs, mostly sequelae to infectious diseases.

### 2.4 Neoplasia

In dogs cardiac neoplasia are rare; if present neoplastic pericardial effusion is commonly related with hemangiosarcoma, chemodectoma (common in brachycephalic breeds), and mesothelioma and less commonly related with ectopic thyroid carcinoma, lymphosarcoma and myxosarcoma. Prognosis depends on the type of tumour. Hemangiosarcomas are more aggressive and are likely to metastasize, and these tumours tend to effuse more rapidly. Palliative pericardiectomy is not usually recommended for hemangiosarcoma because the risk of severe and fatal intrathoracic hemorrhage is significant.

However, in patients with aortic body tumours or mesotheliomas, tumours that effuse more slowly than hemangiosarcomas do, pericardiectomy is often palliative, allowing the fluid to be absorbed through the pleural surface and, thus, preventing the development of cardiac tamponade [4].

## 2.5 Other causes

### 2.5.1 Atrial rupture

In chronic mitral valve disease and severe left atrial enlargement, left atrial tearing is a possible cause of pericardial effusion. The pericardial effusion may contain a clot because hemorrhage is acute. The clot is often visible on an echocardiogram as a hypoechoic structure within the fluid-filled (anechoic) pericardial space. The hemorrhage rapidly leads to cardiac tamponade, cardiogenic shock, and possibly death. Pericardiocentesis in these patients may encourage continued bleeding into the pericardial space. Therefore, in general, pericardiocentesis is not recommended in these patients. If the patient destabilizes because of cardiac tamponade, pericardiocentesis is performed, but the prognosis is guarded at best.

Serositis and myocarditis caused by exposure to toxic metabolites that are eliminated by the kidneys result in **uremic pericarditis** [4].

There is a possibility of pericardial effusion caused by **hypothyroidism**, and hypothyroidism in human patients is a well-known cause of pericardial effusion, but cardiac tamponade is not a frequent clinical sign. However, the pathophysiology is not explained [7].

## 3. Pathophysiology

The pericardium normally has minimal elasticity due to its fibrous nature. However, the pericardium can stretch when pressure is slowly placed on it. In pericardial effusion, as the fluid accumulates in the pericardial space, the ability of the pericardium to stretch is eventually exceeded, and further fluid accumulation subsequently results in increases in intrapericardial pressure. When the intrapericardial pressure increases to the pressure of the right atrium and ventricle (normally 4–8 mm Hg), cardiac tamponade develops [4] with variable degrees of haemodynamic collapse. The volume of fluid required to cause cardiac tamponade varies greatly, depending on the speed with which the fluid accumulates. In experimental canine models, as little as 25–100 mL of fluid rapidly injected into the pericardial space can raise intrapericardial pressure high enough to cause tamponade. In contrast, pericardial effusion that slowly increases in volume can result in a volume as high as 2 L in a large-breed dog before cardiac tamponade manifests.

Pericardial effusion is a resultant of several etiological factors, and the pathogenesis may vary depending on the aetiology. In neoplasias/nephritic syndrome/toxins, there is direct irritation of pericardium causing inflammation resulting in building of fluid. In case of systemic inflammatory diseases or infections, the inflammatory cells target and gets collected within the pericardium, whereas in atrial/ventricular rupture, direct addition of blood to the pericardial space results in effusion. In congestive heart failure, the fluid accumulation in pericardium is due to increase capillary hydrostatic pressure and altered starling forces.

Depending on the size of the enlarging pericardium (rapid accumulation of low volumes or slow accumulation of high volumes) and activation of pain fibers that are
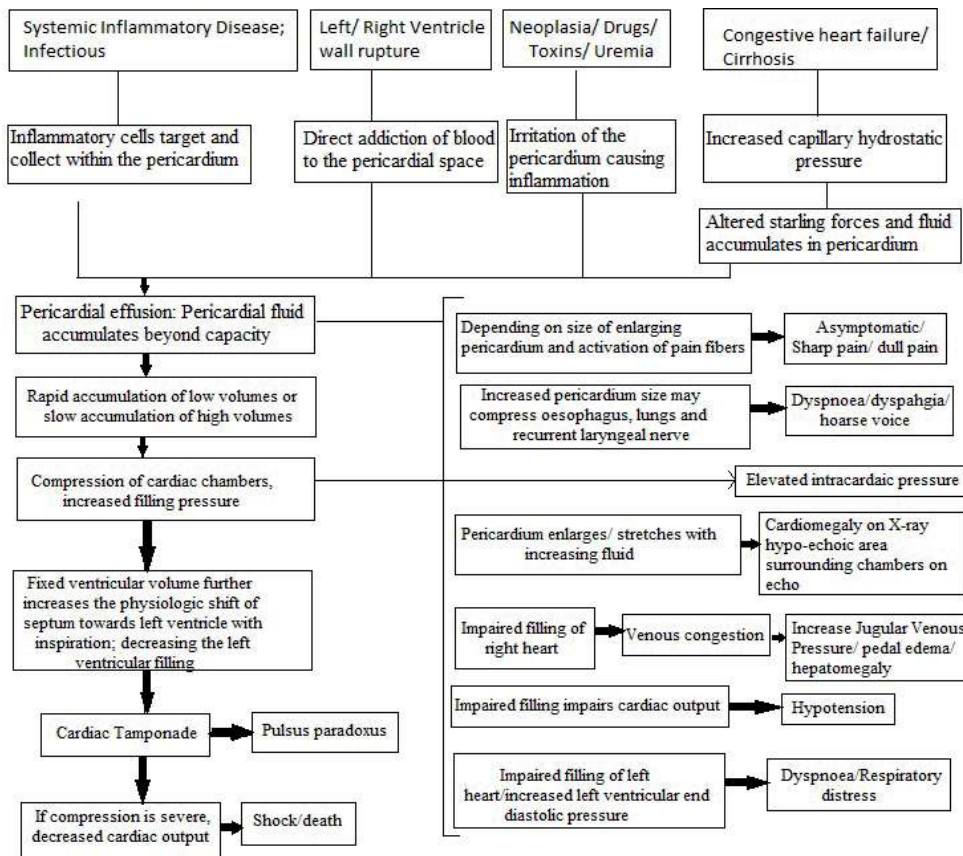
**Figure 1.**
*Pericardial effusion-pathogenesis.*

responsible for expression of signs which may vary viz., asymptomatic or sharp pain with inspiration or dull pain. Increase in heart rate and peripheral vascular resistance can initially compensate these changes, thereby maintaining normal blood pressure. As the intrapericardial pressure rises further, left atrial and left ventricular filling are also compromised. Increased pericardium size (cardiomegaly) may compress the oesophagus, lungs and recurrent laryngeal nerve resulting in dysphagia, dyspnoea and hoarse voice. Further increase in the accumulation of pericardial fluid results in the compression of cardiac chambers, thereby increasing the filling pressures resulting in elevated intracardiac pressure. Impaired filling of the right heart results in venous congestion; thereby elevated jugular venous pressure, pedal oedema and hepatomegaly is evinced. Impaired filling of left heart/increased left ventricular end diastolic pressure results in respiratory distress. Impaired filling also impairs the cardiac output; thereby the blood pressure is decreased (hypotension) [8]. However, fixed ventricular volume increases the physiologic shift of septum towards left ventricle with inspiration, thereby lowering the left ventricular filling resulting in cardiac tamponade (**Figure 1**).

Cardiac tamponade results in decrease venous return, ventricular filling, stroke volume and cardiac output further resulting in shock/death of the patient.

## 4. Clinical signs

Clinical signs of pericardial effusion result from a combination of the volume of effusion, speed with which it accumulated, and underlying cause. Clinical signs may be vague until cardiac tamponade and associated cardiovascular

decompensation develop. The interplay among these factors determines when, in the clinical course, intrapericardial pressure rise high enough to cause cardiac tamponade.

The most common presenting complaint of dogs with pericardial effusion is collapse, weakness, syncope, or lethargy. Dogs may present with abdominal distension and ascites (**Figure 2**) secondary to cardiac tamponade. Heart sounds are muffled, and lung sounds may also be muffled if there is associated pleural effusion. Femoral pulses are weak, and sometimes pulsus paradoxus may be palpated when the pulse is stronger during exhalation and weaker during inhalation. If there is cardiac tamponade, the animal may have signs of cardiogenic shock including pale mucous membranes, cold extremities, hypotension, tachycardia and collapse.

Dogs with chronic pericardial effusion typically have signs secondary to right-sided heart failure, including lethargy, exercise intolerance, respiratory difficulty (**Figure 3**), weight loss and abdominal distention. These signs may be progressive as the ability of the pericardium to stretch is exceeded, whereas dogs with acute pericardial effusion typically present with a history of acute collapse or weakness secondary to decreased cardiac output. Collapse sometimes occurs shortly after physical exertion, and syncope may also be noted.



**Figure 2.**
*Distended abdomen in a dog with pericardial effusion.*



**Figure 3.**
*Pericardial effusion affected dog with respiratory distress.*

## 5. Diagnosis

Physical examination, clinical manifestations, radiography, electrocardiography, echocardiography and estimation of pericardial fluid help in diagnosing the condition. Haematology and serology are not of much significance, and changes in these may reflect the underlying disease process. On physical examination the presence of muffled heart sounds, jugular venous distention, and poor pulse quality or pulsus paradoxus can be suspected for the presence of pericardial effusion. Pulsus paradoxus can best be detected in dogs that are breathing slowly while laterally recumbent. It may not be detected in all affected dogs that are standing or panting. Further, tachycardia, hepatomegaly, ascites, and tachypnoea or dyspnoea can also be noted [9].

### 5.1 Radiograph

Radiographically enlargement of a well-defined cardiac silhouette, globose heart (**Figure 4**) was evident due to the enlargement of the heart caused by pericardial effusion [9]. The size of the cardiac silhouette increases in conjunction with the chronicity of the effusion and the associated fluid volume.

### 5.2 Electrocardiograph

Sinus tachycardia, QRS complexes of diminished voltage (**Figure 5**), and the presence of electrical alternans (**Figure 6**) were the electrocardiographic changes in pericardial effusion. The presence and severity of cardiac tamponade and also inflammatory mechanisms may contribute to the development of low QRS voltage in patients with pericardial effusion [4]. Electrical alternans is the result of a specific type of cardiac motion within the pericardium. The volume of the effusion, viscosity of the fluid, and heart rate are thought to be interrelated in the production of electrical alternans.

### 5.3 Echocardiogram

Echocardiography is the most sensitive method of diagnosing pericardial effusion. It also aids in differential diagnosis from other cardiac diseases.

From the right parasternal view, hypoechoic to anechoic (echo-free space) area between the epicardium and pericardium is evident (**Figures 7** and **8**). In some cases, a left parasternal view may allow better visualization of the right side of the heart, which may aid in identifying right atrial masses. Diastolic collapse of the right atrium or ventricle can be detected and is diagnostic of pericardial tamponade.
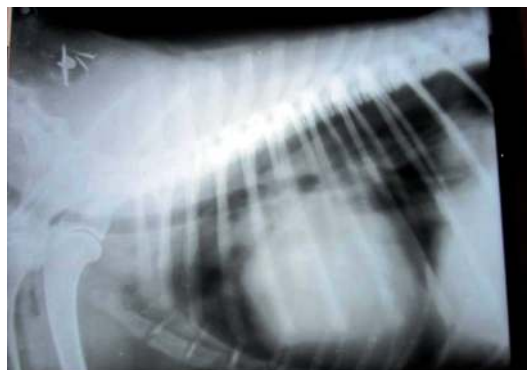


**Figure 4.**
*Globose heart of a dog with pericardial effusion on radiograph—left lateral recumbency.*
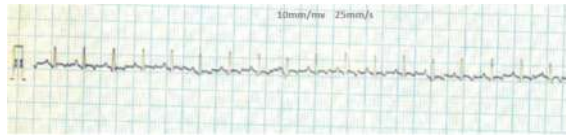
**Figure 5.**
*Low voltage QRS complex (Lead II)—ECG of a dog with pericardial effusion and dilated cardiomyopathy.*



**Figure 6.**
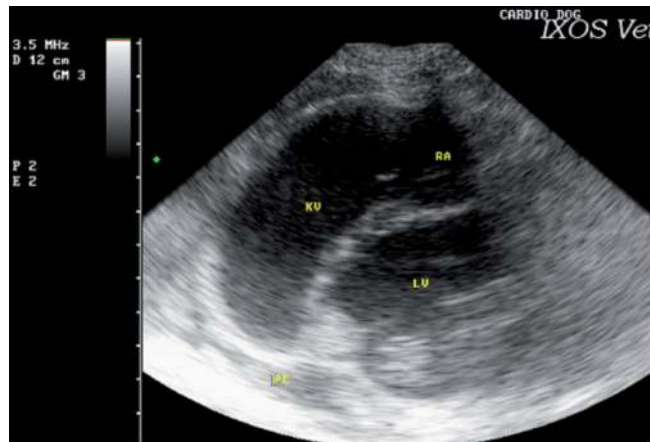*Electrical alternans with slight wide P wave (Lead II).*



**Figure 7.**
*Dilated right and left ventricle with pericardial effusion (short axis view- B mode).*
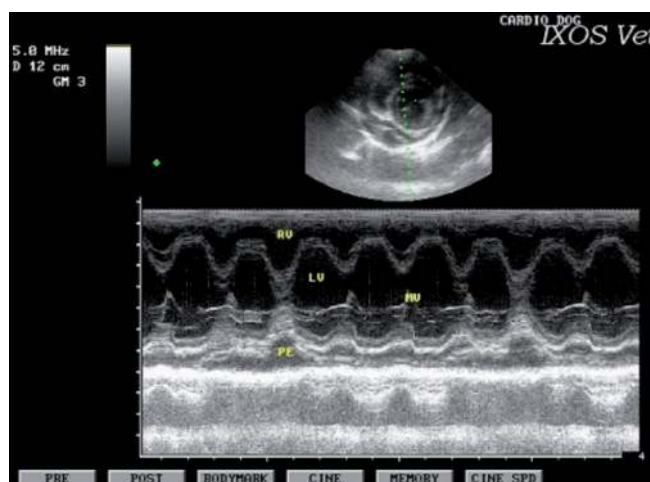


**Figure 8.**
*M-mode image of pericardial effusion dog.*

Transthoracic two-dimensional echocardiography reportedly has 80% sensitivity for a diagnosis of cardiac masses. In most cases, clinicians with basic ultrasonographic skills can readily identify pericardial effusion. However, visualization and definitive

identification of masses often require an exhaustive echocardiographic examination from both sides of the thorax and a higher level of echocardiographic skill.

## 5.4 Pericardiocentesis

Pericardiocentesis is an important procedure as a diagnostic and as a therapeutic standpoint [10]. When cardiac tamponade is diagnosed, pericardiocentesis should be performed as soon as possible. To perform a pericardiocentesis, the patient should be stabilized first.

### 5.4.1 Materials required

To perform a pericardiocentesis:

- Intravenous catheters (for large dogs, 14G, 5-inches angiocatheters are optimal but 16G, 2½-inch over-the-needle catheters will suffice and for small dogs, 16–18G, 2½-inch over-the-needle catheters). Make holes on catheter's side near its tip, so as to maximize the flow if the tip gets occluded while performing the procedure.

- Extension set.

- Three-way stopcock.

- 60-, 12-, and 3-mL syringes.

- 25G ¾-inch needle

- 2% lidocaine.

- Plain (red-top) or serum separator tube.

- EDTA and red-top tubes for cytology samples (and for bacterial culture and antimicrobial sensitivity testing, if indicated).

### 5.4.2 Procedure

Left lateral recumbency (to minimize the movement) and left-side approach are recommended because the right ventricular wall is much thinner than the left ventricular wall, so it is easier to penetrate it unknowingly as you advance the needle and catheter. It is easier to recognize iatrogenic puncture of the left ventricle than of the right ventricle. The oxygenated blood in the left ventricle is bright red. Both right ventricular blood and pleural effusion are dark red, and the high left ventricular pressure usually results in a pulsatile, high-velocity flashback into the catheter, making it obvious if you have penetrated the left ventricle.

If approach is made by the right side, the landmark for performing pericardio-centesis is the "cardiac notch" between the right cranial and caudal lung lobes where the risk of lung puncture is diminished.

### 5.4.3 Site of catheterization

In the seventh and eight intercostal space near the apex beat:

1. Palpate the thoracic wall for the point of maximal intensity (PMI).

2. Clip and scrub that area.

3. Infiltrate 1–2 mL of lidocaine using a 3-mL syringe with a 25G ¾-inch needle to create a local block cranial to the rib, so that the skin, intercostal muscle, and pleural lining are desensitized. Also inject lidocaine as you slowly withdraw the needle along the tract.

4. Slowly introduce the catheter (14 G 5 inch-medium- to large-breed dogs and 16-G, 2½-inch catheter in smaller dogs) through the skin and thoracic wall at the blocked site (**Figure 9**) until you feel a "pop" (catheter's entering the pleural space).

5. Advance the catheter needle slowly into the pericardial space. Monitor the heart rate and rhythm while performing the procedure.

6. A flash of fluid (port wine in colour) is obtained, once the catheter is in pericardial space.

7. Hold the stylet in space when the catheter is well placed inside the pericardial sac. Now remove the stylet, and withdraw small amount of fluid to ensure that it is pericardial fluid and not blood from iatrogenic puncture.

8. Now remove the excess pericardial fluid using a 60-mL syringe. Attach the extension set and stopcock to the catheter (**Figure 10**).

9. Apply suction and evacuate the fluid as soon as possible.

Place the samples of effusion in EDTA and plain tubes for analysis, and record the total volume withdrawn.

The cardiovascular parameters should improve immediately on successful draining of pericardial effusion. The intrapericardial pressure falls, right heart filling improves, cardiac output increases, oxygenation improves, pulse strength improves, and heart rate drops.



**Figure 9.**
*Pericardiocentesis - Introduction of catheter into the pericardial cavity. Adapted from [10].*
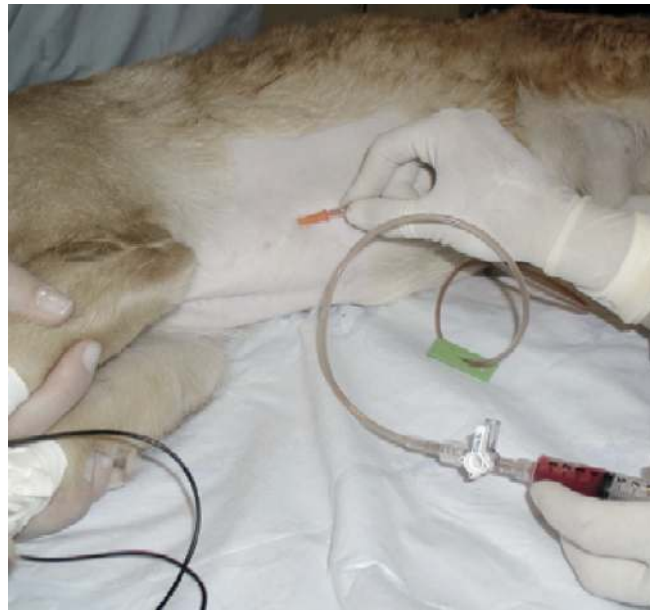
**Figure 10.**
*Pericardiocentesis - Removal of excess pericardial fluid using 60 mL syringe with attachment of the extension set and stopcock to the catheter. Adapted from [10].*

## 5.4.4 Potential complications

- Cardiac arrhythmia due to epicardial irritation or cardiac puncture

- Puncture of the heart or vena cava

- Risk of lung or coronary artery laceration

| Parameter | Pericardial fluid reference range | Pericardial effusion due to tumours/mass | Other aetiologies of pericardial effusion |
|---|---|---|---|
| pH | 7.34–7.47 | 6.75–7.53 | 7.17–7.53 |
| $Pco_2$ (mm Hg) | 34–40 | 21.1–84.4 | 26.7–50.4 |
| $Po_2$ (mm Hg) | 48–56 | 12.0–184.5 | 15.0–201.6 |
| $HCO_3^-$ (mEq/L) | 18–24 | 11.5–24.1 | 17.5–30.8 |
| Sodium (mmol/L) | 147–154 | 131–154 | 127–149 |
| Potassium (mmol/L) | 3.80–4.90 | 3.1–5.5 | 3.5–4.9 |
| Chloride (mmol/L) | 109–120 | 97–120 | 103–121 |
| Ionized magnesium (mmol/L) | 0.14–0.42 | 0.18–0.61 | 0.20–0.50 |
| Ionized calcium (mmol/L) | 1.17–1.38 | 1.09–1.37 | 1.11–1.31 |
| Glucose (mg/dL) | 82–117 | 10–154 | 23–128 |
| Haematocrit (%) | 35–54 | 3–62 | 4–48 |
| Blood urea nitrogen (mg/dL) | 7–28 | 6–74 | 7–30 |
| Lactate (mmol/L) | 0.2–2.4 | 1.9–12.5 | 1.3–12.7 |
| *The table was taken for reference from Ref. [12].* | | | |

**Table 1.**
*Comparison of concentrations of variables in pericardial fluid of dogs with a mass compared with the pericardial fluid of those without a mass.*

### 5.5 A pericardial fluid analysis

Pericardial effusion cytology is believed by many to be of limited value, yet few studies have evaluated its diagnostic utility. Cytologic analysis of pericardial effusion provides an accurate and definitive diagnosis when infectious agents or lymphoma are the causative aetiologies. Conversely, neoplastic diagnoses other than lymphoma (hemangiosarcoma, chemodectoma, mesothelioma) are rarely diagnosed based on cytology alone, and additional diagnostic tests performed on fluid or blood samples have a poor ability to distinguish these from non-neoplastic aetiologies (**Table 1**).

The effusion typically has a haemorrhagic appearance (port wine in colour) despite the underlying cause. Fluid analysis may be diagnostic in cases of infection or lymphosarcoma; therefore, although cytologic evaluation has a low diagnostic yield, it is still recommended [11].

## 6. Treatment

Definitive treatment for pericardial effusion is manual removal of the fluid from the pericardium, i.e. pericardiocentesis. Pericardiocentesis is a method that appears to be of therapeutic value in the management of pericardial effusion patients which are refractory to traditional therapy as the procedure provides immediate relief of cardiac tamponade [8], refer Section 5.4 Pericardiocentesis under Section 5. Diagnosis 5).

Diuretic therapy should be used with caution in cases exhibiting signs of congestive heart failure since their use may result in further reduction in ventricular filling [7]. Mostly, diuretics are not recommended for long-term therapy because diuretics can result in a decrease in preload with a resultant catastrophic decrease in cardiac output if cardiac tamponade recurs.

### 6.1 Pericardiectomy

The surgical removal of part or complete pericardium is pericardiectomy. Pericardiectomy can be a definitive treatment of idiopathic pericardial effusion and a palliative treatment of neoplasia. When pericardial effusion recurs after one or more therapeutic pericardiocenteses, surgical treatment becomes an option. Surgery may be used to remove the pericardium and obtain a biopsy specimen or resect a mass of uncertain origin.

### 6.2 Medical management

The management of pericardial effusion medically is rarely effective. Anti-inflammatory therapy may be used to avoid fluid recurrence. Prednisone in anti-inflammatory doses may be used to treat idiopathic effusions. Chemotherapy for pericardial effusions due to neoplasia vary for different tumour types. However, the prognosis may vary based on the nature of the tumour. Adriamycin may be given for therapy with hemangiosarcoma [13].

## 7. Prognosis

The prognosis for dogs with pericardial effusion varies depending on the underlying aetiology. In case of neoplastic pericardial effusion, it is poor. Prognosis is very good in idiopathic/infectious pericarditis. The prognosis is good in chronic idiopathic pericardial effusion, but tamponade can occur.

## 8. Conclusion

Pericardial effusion is the abnormal accumulation of fluid within the pericardial sac. The effusion usually accumulates slowly and progressively and presents with sign of chronic cardiac tamponade and right-sided heart failure with lethargy, exercise intolerance, or collapse. Diagnosis by echocardiography is the gold standard test. Pericardiocentesis along with diuretic therapy is effective. The prognosis associated with pericardial effusion varies greatly, depending on the underlying cause.

## Conflict of interest

I (the author) hereby declare no conflict of interest.

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