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## **IS IT IN FAILURE? THE TRANSITION FROM THE COMPENSATED TO DECOMPENSATED STATE.**

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### **Lecture Summary**

Heart disease in dogs and cats is often characterised by a prolonged pre-clinical phase during which the patient has evidence of cardiac disease detectable on either clinical examination or diagnostic testing, and yet has no outward sign of heart failure. Only when outward signs associated with loss of function of the cardiovascular system have developed can an animal be said to be in heart failure.

There are various definitions of heart failure a useful one to consider is as follows "Heart failure is the inability of the heart to maintain a normal cardiac output at normal filling pressures". This allows for forward failure where there is inadequate output but normal filling pressures and backward failure when there may be normal output (at rest) but increased filling pressures. Usually animals in heart failure have some combination of the two with peripheral underperfusion and elevated filling pressures. Clinically the outward manifestations of heart failure are usually either signs associated with congestion – due to elevated atrial pressures - or signs of low output. The first manifestation of heart failure in dogs may be an inability to sustain normal levels of exercise. Although this may seem simple enough to determine, in practice it is difficult to detect because few dogs are exercised to their maximal capacity and a lot of older dogs have co-morbid conditions that may also affect exercise tolerance e.g. osteoarthritis.

Using canine mitral valve disease as an example we can derive rough estimates for the period of time dogs with mitral valve disease have mitral insufficiency prior to developing heart failure from the SVEP study [1]. In this study dogs were enrolled when they were found to have evidence of mitral insufficiency and were considered to have reached the endpoint when they went into heart failure. The estimated mean time to the development of heart failure in dogs with a mitral valve murmur but no evidence of cardiomegaly was 1290 days i.e. 3.5 years. By contrast, in the LIVE study [2] when dogs with heart failure were treated their average period before treatment failure was 159 days i.e. less than half a year. If we therefore take treatment failure as rough correlate of survival we can see that dogs with mitral valve disease may spend several years asymptomatic prior to the development of heart failure but once in heart failure deteriorate rapidly and typically survive between six and twelve months. These figures would suggest that approximately four fifths (80%) of dogs with mitral valve disease, at any one time, will not be in heart failure. Similar observations (in terms of relative proportions rather than absolute periods of time) have been made in human patients with heart disease. These observations can probably be generalised to other cardiac diseases, and other species.

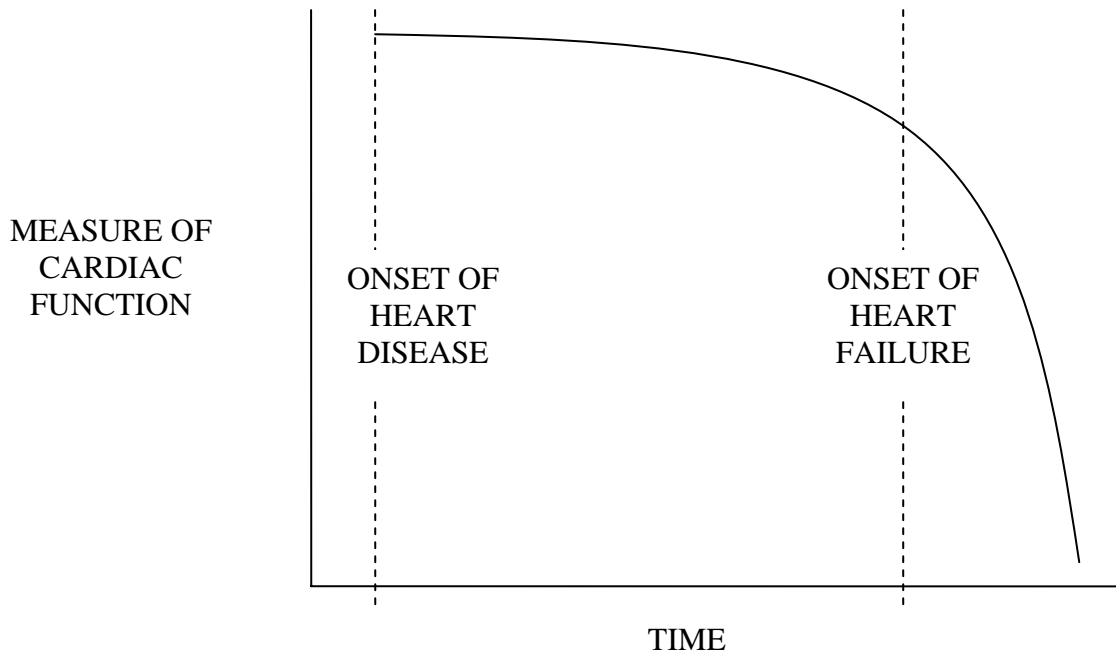


Fig 1. Simple graphical representation of cardiac function against time.

Why is it important for us to distinguish animals that have only heart disease from those that have heart failure? There are several possible reasons two being; firstly it enables us to identify those animals most in need of, and most likely to benefit from therapy; secondly it allows us to prognosticate more accurately.

Most Veterinary studies that have documented efficacy of therapy in patients with heart disease have only done so for patients once they have developed signs of heart failure. Thus if we are able to identify signs of heart failure more accurately we can administer therapy only to those animals that are most likely to benefit. Some studies have been conducted to see whether therapy prior to the onset of heart failure is beneficial and few conclusive results have been obtained. These studies are inherently more difficult to conduct and will almost inevitably take longer to undertake.

The importance of prognostication is one that has been poorly appreciated in the veterinary literature. The prognosis for a patient is probably of greatest importance for their owner. The diagnosis, although of interest to the clinician may be of limited interest to the owner.

If we understand what causes heart disease to progress to the point where heart failure develops we may better appreciate the clinical findings that will distinguish patients in heart failure from those with heart disease. The progression from heart disease to heart failure is mediated by a combination of factors. The cause of the underlying diseases we encounter such as dilated cardiomyopathy and myxomatous mitral valve disease is at best poorly understood. Although these underlying diseases themselves have a tendency to progress the progression of any heart disease to the point of heart failure tends to be caused by a similar series of neural, endocrine and growth pathways; irrespective of the original cause. There are several texts and articles that have reviewed these mechanisms in both man and animals [3, 4] and I do not intend to review them in

detail here. In summary several potentially detrimental systems are activated including the sympathetic nervous system, the renin-angiotensin- aldosterone system, the endothelin system and arginine vasopressin. Some counter-regulatory systems are down-regulated e.g. the parasympathetic nervous system, whereas others are up-regulated but overwhelmed e.g. the natriuretic peptide system.

On clinical examination or with ancillary diagnostic aids we can detect some of the consequences of activation of these systems and better characterise the nature of a patient's heart disease [5].

### Physical examination findings

The signs on physical examination that may indicate the presence of heart failure are those of either sympathetic stimulation, poor peripheral perfusion or congestion. Sympathetic stimulation is characterised by an increased heart rate and decreased heart rate variability; characterised in dogs by an absence of respiratory sinus arrhythmia. Cats are much less predictable in the change of their heart rate on development of heart failure. Some cats with advanced disease present with a bradycardia. Poor peripheral perfusion at rest tends to be found in animals with advanced heart failure or failure of an acute onset. This can be characterised by pallor and cold extremities. Pulse quality may also be poor. Signs of congestion will manifest with evidence of tachypnoea, dyspnoea or ascites. Sub-cutaneous oedema is a rare finding in small animal patients with heart failure.

### Ancillary diagnostic aids

Routine blood tests may provide clues as to the possible presence of heart failure but none of the abnormalities that may arise is specific to heart failure. Typically heart failure is characterised by worsening renal perfusion and therefore urea and creatinine may increase. Advanced heart failure is indicated by the presence of hyponatraemia due to non-osmotic release of ADH leading to water being retained in excess of sodium. These findings are confounded by the influence of therapy which also has effects on electrolyte homeostasis and renal perfusion [5].

A number of specific blood tests (biomarkers) are available which may help to determine if patients are in heart failure. NTproBNP is an accurate marker of the presence of heart failure, the concentration of which can be assessed in the blood of both dogs and cats (requiring species specific assays). Troponin I has been recommended for use in the diagnosis of heart disease and failure. It is a marker specific for cardiac muscle and is a very sensitive marker of myocardial injury however it did not prove to be very accurate when used for discriminating dogs with heart disease from dogs with heart failure [6].

Electrocardiography does not offer any findings specific for heart failure although if respiratory sinus arrhythmia and a low heart rate are evident in a dog, it is very unlikely that the dog is in heart failure (high negative predictive value).

Echocardiography, although an excellent diagnostic aid for the demonstration of abnormalities of cardiac structure and function, is not a very good test for the determination of the presence or absence of heart failure. Individual diseases

tend to have characteristic progressions. Certain findings have reasonable negative predictive value i.e. animals in which they are found are very unlikely to be in heart failure. It is very unlikely that a dog or cat with a normal sized left atrium has left-congestive heart failure. Other more sophisticated techniques have been developed using combinations of spectral and tissue Doppler measurements to obtain indirect estimates of left atrial pressure. These require considerable expertise to measure in a reproducible and clinically useful fashion.

As mentioned above individual diseases tend to have typical echocardiographic progressions and for instance a dog with mitral valve disease is more likely to have congestive heart failure if it has an enlarged left atrium, enlarged left ventricle and a higher E-wave velocity and E to A ratio during diastole.

Thoracic radiographs are widely regarded as the “gold-standard” for the definitive diagnosis of left-sided congestive heart failure. They are the only widely available means where by pulmonary oedema or pulmonary venous congestion can be definitively demonstrated. If however we apply our broad definition of heart failure above it can be seen that not all dogs with heart failure necessarily need have signs of congestion and therefore thoracic radiographs can be considered to be specific but not sensitive.

In conclusion the most appropriate treatment of and prognostication for patients with heart disease relies upon the staging of the severity of their disease and particularly in the discrimination of dogs in heart failure from dogs not in heart failure. The gold standard for the diagnosis and monitoring of left sided congestive heart failure remains thoracic radiography but newly emerging blood tests may ultimately rival thoracic radiography in their accuracy and utility.

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