Management of Heart Disease – Part 1: Dogs

This two-part series will review the options available for management of the common acquired cardiac diseases in dogs (part 1) and cats (part 2). Myxomatous mitral valve disease and dilated cardiomyopathy are the most common acquired cardiac diseases in dogs. Here, we review management options in the pre-clinical phase for both diseases as well as acute and chronic congestive heart failure. Examples of echocardiographic and radiographic changes are provided as well as tables and figures covering the mechanism of action for the drugs used.

Key words: Myxomatous mitral valve disease, dilated cardiomyopathy, congestive heart failure, treatment, canine, cardiology

Common acquired cardiac diseases in dogs

The most common forms of acquired heart disease diagnosed in dogs are myxomatous mitral valve disease (MMVD, also known as degenerative mitral valve disease or endocardiosis) and dilated cardiomyopathy (DCM). MMVD is most commonly diagnosed in small breed dogs, including Cavalier King Charles Spaniels, Chihuahuas, Shih Tzus, Yorkshire Terriers, and Border Collies (Mattin et al. 2015). Breeds predisposed to DCM include Great Danes, Doberman Pinschers, Irish Wolfhounds, Newfoundlands, Boxers, Labradors and Cocker Spaniels (Martin et al. 2010) (Figure 1).

Both MMVD and DCM can have long pre-clinical periods. Pre-clinical MMVD is generally easy to identify in a small breed dog as mitral regurgitation will result in the presence of a characteristic left apical, systolic murmur (Boswood et al. 2016). Echocardiography will demonstrate mitral valve thickening and prolapse in addition to mitral regurgitation on colour Doppler (Figure 2). Pre-clinical DCM can be more difficult to identify as the typical quiet, left apical, systolic murmur is not present in all affected dogs and many dogs may not have any abnormalities on auscultation. Dogs with pre-clinical DCM have left ventricular systolic dysfunction (Figure 2) and/or frequent ventricular arrhythmias. Echocardiography and 24-hour ECG Holter screening is therefore often performed in at-risk breeds to identify pre-clinical cases (Wess et al. 2017).

MMVD is a progressive disease, although the rate of progression varies widely between animals. A small amount of mitral regurgitation can be well tolerated, and...
or vasopressin). This results in increased circulating volume and vasoconstriction allowing the body to maintain cardiac output and blood pressure. The same compensatory mechanisms occur in response to the reduction in cardiac output secondary to systolic dysfunction in DCM. While initially having the positive effect of maintaining blood pressure, the net long-term result is increased circulating volume and if the MMVD is slowly progressive the dog may live many years without clinical signs, often dying of non-cardiac disease (Borgarelli et al. 2008). However, with increasing severity of mitral regurgitation, cardiac output is compromised and there is activation of compensatory mechanisms, including: sympathetic nervous system stimulation; activation of the renin-angiotensin-aldosterone system and release of antidiuretic hormone (ADH or vasopresin). This results in increased circulating volume and vasoconstriction allowing the body to maintain cardiac output and blood pressure. The same compensatory mechanisms occur in response to the reduction in cardiac output secondary to systolic dysfunction in DCM. While initially having the positive effect of maintaining blood pressure, the net long-term result is increased circulating volume and venous return to the heart. This leads to increased filling pressures and both left atrial and left ventricular dilation. With increasing left atrial size (Figure 3) and pressures, irrespective of the underlying aetiology, there is increasing pulmonary venous pressure and eventually left sided congestive heart failure (CHF; namely, pulmonary oedema) develops.

Although MMVD and DCM are predominantly diseases of the left side of