

Valvular Diseases
 Mitral Regurgitation (MR)
 Tricuspid Regurgitation (TR)
 Aortic (Subaortic) Stenosis
 Pulmonic Stenosis
 Aortic Insufficiency

MITRAL REGURGITATION (MR)

ETIOLOGIES	<p>Primary etiologies are those involving changes in the mitral valve leaflets or chordae tendineae:</p> <ul style="list-style-type: none"> • Endocardiosis (chronic myxomatous valvular degeneration) is a common lesion seen in older dogs (especially small breeds). It is characterized by an accumulation of collagenous and myxomatous deposits in the valve leaflets resulting in thickened, nodular, stiff valve leaflets. • Ruptured chordae tendineae (seen in conjunction with endocardiosis): Large primary or smaller secondary and tertiary chordae tendineae break, causing a sudden worsening of regurgitation in proportion to the structural support that is lost. • Bacterial endocarditis is bacterial infection of the valves. • Mitral dysplasia is a congenital malformation of the valves. <p>Secondary etiologies involve malpositioning of the valves:</p> <ul style="list-style-type: none"> • Left ventricular dilation (e.g. dilated cardiomyopathy) changes the positioning of the papillary muscles and consequently pulls the valve leaflets apart, creating a gap. • Left ventricular concentric hypertrophy (e.g. HCM in cats)
PATHO-PHYSIOLOGY	<p>Mitral regurgitation causes volume overload:</p> <p>Each time the left ventricle contracts, a portion of the total stroke volume (TSV) flows in the normal forward direction (forward stroke volume, FSV) and a portion leaks backwards (reverse stroke volume, RSV) through the defect, into the low pressure left atrium. As a result, cardiac output initially decreases, thus activating the sympathetic nervous system which triggers an increase in heart rate and cardiac contractility to bring cardiac output back to normal. This sympathetic stimulation effect diminishes over several days. Meanwhile, a different system, the RAAS (Refer to p. 3.6) is activated to help the heart compensate.</p> <p>The activated RAAS leads to an increase in water retention, which in turn results in increased preload (end-diastolic volume) for the heart to pump. Thus the total stroke volume increases. As the TSV increases, the amount that regurgitates also increases, but so does the volume of blood ejected forward. In the early stages, enough blood is ejected forward to bring cardiac output back up to normal.</p> <p>In order for the left heart to compensate for the increase in end-diastolic blood volume without having to increase left ventricular pressure (e.g. if you fill a balloon with more air, the pressure in the balloon increases), the ventricle undergoes eccentric hypertrophy. In eccentric hypertrophy, sarcomeres are laid down end-to-end so that the ventricular walls get longer and the left ventricular chamber dilates (analogous to getting a bigger balloon).</p> <p>With chronic mitral valve disease, congestive heart failure may be the result of one of two separate entities:</p> <ul style="list-style-type: none"> • Congestive heart failure: Once the heart has undergone maximal eccentric hypertrophy, the chamber stops enlarging. As preload continues to increase, pressure in the left ventricle also increases. During diastole, the left ventricle, left atrium, and pulmonary veins freely communicate (the mitral valve is open) so that the pressure is the same in all three regions. This means that left atrial pressure and pulmonary venous and capillary pressures are also elevated. The end result is pulmonary edema. • Myocardial failure: In end-stage mitral regurgitation, the cardiac muscle may begin to fail so that the heart no longer has the strength to pump fluid forward. This occurs infrequently in small dogs and when it does, it tends to be mild. Myocardial failure is more common in large breed dogs.

MITRAL REGURGITATION continued

CLINICAL SIGNS	<p>Signs are due to congestive heart failure:</p> <ul style="list-style-type: none"> • Cough is due to pulmonary edema or compression of the left mainstem bronchus by an enlarged left atrium. Rule out collapsing trachea and lower airway disease. • Tachypnea and increased respiratory effort are due to pulmonary edema leading to hypoxia. • Exercise intolerance and lethargy are due to low cardiac output. • Syncope: With moderate to severe disease, some animals develop baroreceptor abnormalities which result in hypotensive events and consequently episodes of fainting.
PHYSICAL EXAM	<p>Holosystolic or pansystolic murmur due to turbulent blood flow into the left atrium. The murmur is heard best over the left apex but it radiates dorsally, caudally, and to the right. A third heart sound is sometimes present (volume overload).</p>
RADIOGRAPHIC FINDINGS	<ul style="list-style-type: none"> • Early in disease, radiographs may be normal. As the disease progresses, radiographs may reveal left atrial enlargement which appears as loss of the caudal cardiac waist (mild disease) or left atrial bulge ± elevation or narrowing of the trachea at the carina or mainstem bronchi. These tracheal changes can cause a cough. Left atrial enlargement also appears on the DV radiographs. Left ventricular enlargement appears on lateral radiographs as elevation of the trachea or a "tall" heart. • Pulmonary congestion and edema appear when congestive heart failure is present. Earlier in the course of disease, the pulmonary veins may just look distended.
ECG	<ul style="list-style-type: none"> • Symptomatic patients often have long P waves (> 0.04 s) due to the enlarged left atrium. • The QRS complex may be tall due to left ventricular enlargement (ECG is not a sensitive indicator of ventricular enlargement but is quite specific when abnormalities are present). • Atrial arrhythmias become common as the left atrium enlarges (atrial fibrillation, etc). • Ventricular arrhythmias can occur in advanced disease. • Increased heart rate: Heart rate increases in patients with heart failure in order to compensate for the low stroke volume.
ECHOCARDIOGRAM	<ul style="list-style-type: none"> • Eccentric hypertrophy of the left ventricle + dilated left atrium • Hyperdynamic contraction of the left ventricle • Doppler echocardiography reveals mitral regurgitation
TREATMENT	<p>Mitral insufficiency often progresses slowly and the left ventricle and atrium have time to adapt. Consequently, in cases where radiographic or echocardiogram evidence of failure exists (e.g. severely enlarged left atrium, pulmonary edema), it is controversial as to whether animals should be treated. Some studies have found early treatment with ACE inhibitors decreases disease progression whereas other studies have found no difference. The conflict in research findings may be due to the fact that heart disease has strong genetic components and genetics contribute to the onset and progression of disease. Thus far, studies have not controlled for genetics.</p>

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