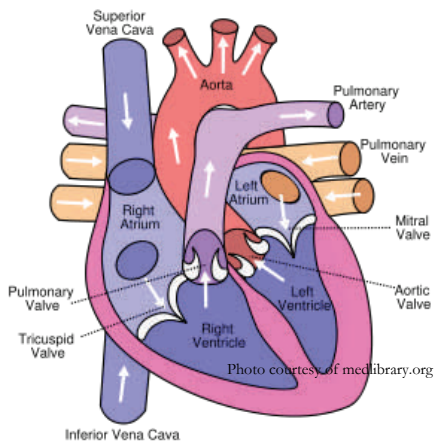


## What is Systolic Anterior Motion?

In normal conditions, when the heart starts contracting, the leaflets of the mitral valve (the valve that regulates the blood flow from the left atrium to the left ventricle) move together towards the left atrium to seal the communication (ostium) between atrium and ventricle. This allows a one-way direction of the blood flow from the left ventricle into the aorta. After completion of the heart contraction, the valve leaflets move away from one other to re-open the ostium and allow blood flow from the atrium to the ventricle. In Systolic Anterior Motion (SAM) there is an abnormal movement of the anterior leaflet of the mitral valve at the beginning of the heart contraction (systole). This leaflet

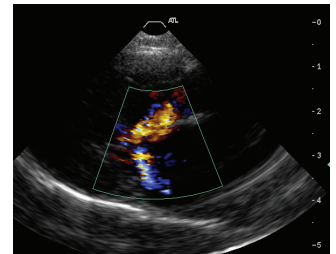


interferes with the blood flow entering the aorta (dynamic obstruction) causing flow

turbulence in the outflow tract. Moreover, since the leaflet is not in the right position to seal the valve, there is a backflow of blood from the ventricle to the atrium (mitral regurgitation), which is also accompanied by flow turbulence. The two turbulences are responsible for an audible heart murmur on chest auscultation. Dynamic obstruction and systolic murmurs can be present at rest or can become audible when the heart rate and cardiac contractility increase, like during stress or excitement.

SAM is present in approximately 50% of cats affected by hypertrophic cardiomyopathy (HCM), a heart disease characterised by an abnormally thickened (hypertrophic) heart muscle (myocardium). However, SAM is not necessarily associated with myocardial hypertrophy, having been convincingly documented in people and dogs in the absence of significant ventricular hypertrophy.

Therefore, in the absence of thickened myocardium, it is difficult to determine whether SAM is caused by an early-stage HCM or not. Intrinsic valvular disease



SAM appearance on echocardiography

(i.e. degeneration or malformation) may also play a role in some patients with SAM, especially when accompanied by severe mitral regurgitation. The mechanism of SAM is not fully understood and several hypotheses have been suggested. However, deformation of the mitral valve architecture (leaflets, chordae tendineae, papillary muscles) and an exuberant heart contraction (e.g. HCM, hyperthyroidism, over-excitement) seems to be the most plausible explanations.

The severity of the outflow gradient can be reliably estimated with Doppler examination and is directly related to the duration of the contact between the valvular leaflet and the ventricular septum.

## What should be expected now?

Cases of SAM not accompanied by echocardiographic evidence of thickened heart muscle should be monitored on a regular interval (12-18 months) to ensure that there are no cardiac changes associated with this abnormality. Severe cases of SAM, those that produce a high turbulence and blood flow acceleration in the aorta, may benefit from pharmacological treatment. It has been demonstrated that human patients affected by SAM respond positively to a class of drugs called “beta

blockers”. Beta blockers (e.g. propranolol or atenolol) act by slowing the heart rate and reducing the force of contraction. This will result in a less vigorous contraction, improved ventricular filling and more regular movement of the mitral valve leaflets. Although some veterinary surgeons advocate the use of beta-blockers to reduce the degree of SAM, the clinical efficacy of

these medications is dubious since it should be assessed when the heart is beating fast and vigorously (e.g. exercise or excitement), which is not always feasible in veterinary cardiology. Furthermore, potential side-effects of beta-blockers should be carefully considered before starting a long-term therapy with these medications.

*This handout provides a general overview on this topic and may not apply to all patients.*

*Please do not hesitate to contact us if you require any additional information. ([www.cardiospecialist.co.uk](http://www.cardiospecialist.co.uk))*