The veterinarian should be familiar with common cardiac malformations, its associated clinical signs, the repercussions for breeding and the possibilities of curative or palliative treatment.

In the feline species the most common congenital defects are atrioventricular dysplasia (with the mitral valve more commonly affected than the tricuspid valve) and ventricular septal defects. Pulmonic and aortic stenosis are relatively rare. Patent ductus arteriosus is extremely uncommon. However one has to be aware that cats often have multiple congenital defects and do present as adults with congenital heart disease. They do amazingly well with complex cardiac malformations. Tetalogy of Fallot is the most frequently recognised, but others like AV-canal defects, and cor triatriatum sinister are, with the common availability of echocardiography, more frequently diagnosed. This paper is the third in a series on congenital heart disease in small animals.

MITRAL VALVE DYSPLASIA

Epidemiology

Mitral dysplasia (MD) is the number one congenital heart disease (CHD) in cats. There appears to be a male predominance. It is well recognised in Siamese cats.

Types

Mitral valve dysplasia is defined as an abnormally formed mitral valve that results in mitral regurgitation. It represents a complex of thickened valve leaflets, abnormally short or long chordae tendineae, abnormal papillary muscles and abnormally low implanted atrioventricular valve (Fig. 1).

Clinical findings

The murmur of mitral insufficiency is typically of mixed frequency and harsh sounding, but it may be high-pitched or musical in quality. Although this murmur is usually loudest over the mitral valve area and left atrium one has to be aware that the sternum often enhances the acoustics of feline murmurs. Clinical signs of left-sided congestive heart failure (CHF) (e.g. dyspnœa due to pulmonary oedema and/or pleural effusion) might be present in severe cases.

ECG findings

The ECG findings of MD are those of left atrial (widened P-wave) and ventricular enlargement (tall QRS). Supraventricular arrhythmias are more common than ventricular arrhythmias, and atrial fibrillation in the feline species often indicates massive left atrial enlargement.

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Radiographic findings
The radiographic findings vary according to the degree of left-sided congestive heart failure (enlarged left atrium, pulmonary venous congestion, interstitial or more generalised alveolar pulmonary oedema with a ‘cotton wool’ appearance). The dorsoventral view often gives a better appreciation of left atrial/auricular size compared to a lateral thoracic radiograph (Figs. 2a and 2b).

Echocardiographic findings
Two-dimensional echocardiography is helpful in assessing chamber size and anatomical morphology (Fig. 3), but Doppler interrogation is essential to confirm and to estimate the severity of the valvular regurgitation. Obvious anatomical and functional mitral valve apparatus abnormalities indicate primary congenital valve disease, and this should be differentiated from the acquired cardiomyopathies that might occur at a very young age and give secondary mitral valve insufficiency.

Treatment and outcome
In mitral dysplasia medical therapy is palliative, and aimed at treating heart failure (ACE-inhibitors and diuretics). Beta-blockers such as propranolol and atenolol remain the drug of choice for controlling the ventricular response rate in feline atrial fibrillation. Thrombo-embolic disease is a common complication of feline cardiac disease, especially when there is marked chamber enlargement. Anti-coagulation therapy such as aspirin or heparins might be indicated in some cases.

TRICUSPID VALVE DYSPLASIA
Epidemiology
With the fairly recent histopathological recognition of a new entity (arrhythmogenic right ventricular cardiomyopathy, ARVC) as a reason for tricuspid valve insufficiency and right atrial enlargement, tricuspid dysplasia might have been overdiagnosed in the past. In France Chartreux cats seem to be predisposed. There appears to be a male predominance. It is commonly associated with mitral dysplasia.

Types
Tricuspid dysplasia (TD) represents a complex of thickened valve leaflets, abnormally short or long chordae tendineae, abnormal papillary muscles and abnormally low implanted atrioventricular valves (Ebstein anomaly).

Clinical findings
The murmur of TD is a systolic plateau murmur of...
tricuspid insufficiency and can be easily confounded with a ventricular septal defect murmur; however the latter is often louder and hence not uncommonly associated with a thrill. In cases of severe TD the regurgitant flow might become laminar and no murmur will be audible. Clinical signs of right-sided congestive heart failure (e.g. dyspnoea due to pleural effusion) might be present in severe cases. The effusions are often chylous in right-sided CHF. However, ascites is less common in cats with right-sided congestive heart failure than in dogs.

**ECG findings**

Splintered QRS complexes and signs of right ventricular (deep S-waves and R-axis deviation) and right atrial enlargement (tall P-wave) are the most commonly found ECG abnormalities. Supraventricular arrhythmias are uncommon.

**Radiographic findings**

The radiographic findings are those from progressive right-sided congestive heart failure (right atrial enlargement, widened caudal vena cava, hepatomegaly, pleural effusion).

**Echocardiographic findings**

Two-dimensional echocardiography is helpful in assessing chamber size and anatomical morphology, but Doppler interrogation of blood velocities is essential to confirm and to estimate the severity of the valvular regurgitation. Obvious anatomical and functional valve apparatus abnormalities indicate primary congenital valve disease. It should not be confused with ARVC which often also shows an additional aneurysmal dilation of the right ventricle (Fig. 4).

**Treatment and outcome**

In tricuspid dysplasia medical therapy is palliative, and aimed at treating heart failure (ACE-inhibitors and diuretics).

**VENTRICULAR SEPTAL DEFECTS**

**Epidemiology**

There is no known breed predisposition although the author has seen it quite a few times in Maine Coon kittens (Fig. 5).

**Types**

Most ventricular septal defects (VSD) are high in the septum just below the aortic valves and peri-membraneous. Muscular ventricular septal defects are uncommon in small animals.

**Clinical findings**

The murmur caused by a restrictive VSD is loud, harsh and typically best heard on the right cranial thorax. The shape and quality of these murmurs vary tremendously, but they are often associated with a thrill. Murmur intensity and duration may be reduced if the VSD is large and pulmonary hypertension due to pulmonary overperfusion develops (so there is an inverse relationship between murmur grade and severity).

**ECG findings**

The findings can be variable but in small VSDs the R-waves can be tall suggestive of left ventricular enlargement. The Q wave can be deep and notched indicating a conduction disturbance high up in the interventricular septum. In larger VSDs right axis enlargement, widened caudal vena cava, hepatomegaly, pleural effusion).

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**Fig. 4a:** Two dimensional echocardiography showing the features of tricuspid dysplasia (right atrial enlargement) and arrhythmogenic right ventricular cardiomyopathy (right atrial enlargement and right ventricular aneurysmal dilation).

**Fig. 4b.**

**Fig. 5:** Maine Coon kitten with a VSD.
deviation might occur suggestive of pulmonary hypertension or pulmonic stenosis.

**Radiographic findings**
The radiographic changes depend of course on the size of the VSD with pulmonary overcirculation left atrial and ventricular enlargement associated with right ventricular enlargement. Signs of left-sided CHF are uncommon.

**Echocardiographic findings**
VSDs are most commonly localised at the level of the aortic valve and best imaged from the right parasternal left ventricular outflow view. VSDs are classified as restrictive and non-restrictive depending on the pressure gradient across the two ventricles (small defect = restrictive: v > 4m/s; ΔP > 64 mm Hg).

**Treatment and outcome**
Treatment is not warranted in animals with small, restrictive VSDs and they often live a normal life span (v > 4m/s; ΔP > 64 mm Hg). With large VSDs medical therapy is palliative, and aimed at treating heart failure (ACE-inhibitors and diuretics). Interventional closure remains an option, depending on the localisation of the defect. Pulmonary artery banding can reduce shunt flow.

**TETRALOGY OF FALLOT (TOF)**

**Epidemiology**
TOF is the most common cyanotic congenital defect in the cat.

**Types**
TOF is a combination of a VSD, pulmonic stenosis, dextraposition of the aorta and secondary right ventricular hypertrophy.

**Clinical findings**
Cyanosis varies from absent to severe. A cardiac murmur is commonly but not always present. The murmur is most commonly due to pulmonic stenosis and is usually loudest at the left heart base. Because the VSD is often large and there is polycythaemia (increased blood viscosity due to right to left shunting) it does not create a murmur. Femoral artery pressure is normally normal.

**ECG findings**
Prolongation of the P-R interval (first degree AV-block) may be seen with all types of atrial septal defects. Bundle branch blocks are also common findings probably because the large defect interferes with normal bundle branch growth (Fig. 6).

**Radiographic findings**
Thoracic radiographs show right ventricular enlargement associated with pulmonary hypoperfusion.

**Echocardiographic findings**
Two-dimensional echocardiography is helpful in assessing chamber size and anatomical morphology. Doppler interrogation or a bubble study (echo-contrast) is helpful in confirming the presence of a right to left shunt.

**Treatment and outcome**
Medical therapy aims at controlling the polycythaemia by phlebotomy or chemotherapy (hydroxyurea). Beta-blockers (propranolol) might be useful in reducing hypoxaemic episodes. Palliative surgery can be performed. Prognosis is guarded and sudden death is not uncommon. However some cats live for many years, even without treatment.

**AV-CANAL DEFECTS**

**Epidemiology**
AV-canal defects are more commonly seen in Persians, and a familial form has been recognised in Japan.

**Types**
AV-canal defects comprise a range of malformations caused by varying degrees of incomplete development of the inferior portion of the atrial septum (ASD), the inflow portion of the ventricular septum (VSD) and the AV-valves (single 5-leaflet valve instead of a separate mitral and tricuspid valve). They have also been called endocardial cushion defects.

**Clinical findings**
The clinical findings are variable depending on the severity of the malformations. A murmur of mitral regurgitation is the most common finding. There is often growth retardation.

**ECG findings**
Prolongation of the P-R interval (first degree AV-block) may be seen with all types of atrial septal defects. Bundle branch blocks are also common findings probably because the large defect interferes with normal bundle branch growth (Fig. 6).

**Radiographic findings**
There is often marked generalised cardiomegaly, associated with signs of left-sided CHF (pulmonary oedema, pleural effusion).

**Echocardiographic findings**
Echocardiography is helpful in determining the abnormal morphology (Fig. 7) and Doppler will
identify valvular insufficiencies and the left to right shunting defects.

**Fig. 7:** Two dimensional echocardiography (left parasternal four-chamber view) showing a big atrial septal defect and a 5 leaflet AV valve (incomplete AV canal defect).

**Treatment and outcome**
Cats generally die or are presented in left-side CHF failure at a young age. Treatment is palliative (controlling heart failure by ACEI and diuretics). Hydralazine reduces the shunt flow and mitral regurgitation in children with AV canal defects but no reports are available in veterinary medicine.

A list of breed predispositions and heritability can be downloaded from www.acapulco-vet.be

**FURTHER READING AND REFERENCES**