BREED-RELATED VENTRICULAR ARRHYTHMIAS


In human beings more of the 10 % of the ventricular arrhythmias are evident in the absence of apparent structural heart disease. Ionic channel disease like the Brugada's syndrome and the Long QT syndrome, the catecholaminergic polymorphic VT, the idiopathic RVOT VT and LVOT VT, the idiopathic Torsades de pointes are often inherited. In veterinary medicine has been demonstrated only few disease with inherited arrhythmic disorder.

German Shepherd inherited arrhythmias.

The arrhythmias range from infrequent unifocal ventricular premature depolarizations through single runs of monomorphic ventricular tachycardia and from multifocal ventricular premature depolarizations through polymorphic repetitive ventricular tachycardia and ventricular fibrillation. The dogs show no evidence of structural heart disease. Electrocardiograms, echocardiograms, and serum electrolyte levels do not differ from those of normal dogs; neither do endocardial monophasic action potential durations differ between these and German shepherd dogs having no ventricular arrhythmias. There is evidence of inadequate sympathetic innervation in portions of the apical, anterior, septal, and lateral regions of the left ventricole. The arrhythmias are catecholamine sensitive and exercise induced, and has been demonstrated attributable to early after depolarization. These rhythm disturbances usually occur in young dogs (13-24 weeks). After the first 7 month of life the innervations system developed in a normal maturation with a reduction of the frequency of the disease.

Ventricular tachycardia during dilated cardiomyopathy

Dilated cardiomyopathy is characterized by ventricular dilatation, especially of the LV featuring a generally progressive impairment of ventricular function that frequently leads to HF. Different ventricular arrhythmias are present in process of dilated cardiomyopathy, from rare VPCs to sustained VT. The arrhythmogenic mechanisms of these arrhythmias are either enhanced automaticity or reentry or trigger activity. A particular type of VT is produced by a reentry where both the branches of the specific conduction system are involved. This VT is called Bundle branch reentry VT. In this case, the VT is initiated by a right PVC that ascends through the left bundle branch and descends over the right bundle branch, thus depolarizing the left ventricle from right to left and showing the typical morphology of the LBBB. In some exceptions this
In reentrant circuit may be reversed and the VT morphology will be that of a RBBB. Ventricular tachycardias originating in an interfascicular reentry circuit have also been described. In Dobermann Pinscher breed an inherited form of dilated cardiomyopathy has been described, due to alterations in chromosome 5 and 14.

**Ventricular tachycardia during arrhythmogenic right ventricular cardiomyopathy**

Arrhythmogenic right ventricular cardiomyopathy (ARVC) is a hereditary cardiac disease characterized by anatomical and functional alterations of the right ventricle leading to an abnormal electrical conduction, changes in heart rhythm, and ultimately hemodynamic consequences. It is characterized by areas of progressive myocyte death, with fibro-fatty replacement. Arrhythmogenic right ventricular cardiomyopathy may be either diffuse, throughout the right ventricle, or segmental and localize in the so-called 'triangle of dysplasia', that includes the subtricuspidal region, the right ventricle apex and outflow. Only in rare cases the condition involves also the interventricular septum. The structural changes of the cardiac muscle may then cause reentrant ventricular arrhythmias with possible sudden death. The human form of ARVC is hereditary and caused by mutations in genes coding desmosomal protein. A hereditary form of ARVC associated with sudden death has been described also in dogs. In boxer breed the rhythm disturbances are PVCs with RBBB type morphology, either isolated or organized in pairs, triplets, non sustained, sustained, repetitive, paroxismal VT that can developed in VF and SD. In English bulldogs has been demonstrated a particular pattern of the disease localized RVOT aneurysm. Right ventricular aneurysms, whether single or multiple are considered a pathognomonic features of ARVC. The electrocardiographic appearance of the VT described in the English Bulldog presents similarities with the RVOT-VT found in an experimental canine model of idiopathic RVOT-VT reported in humans. RVOT-VT usually manifests as wide QRS complexes with a LBBB configuration and inferior axis, with the QRS complex positive in leads II, III, and aVF, negative in lead V1 and positive in leads V2-V6. A standard 12 lead electrocardiogram is useful to determine the site of origin within the outflow tract of the RVOT–VT: negative QRS complexes in lead I suggest an anterior (toward the left arm) (cranial) focus, while positive complexes indicate a more posterior (toward the right arm) (caudal) focus. Lead aVL is usually negative if the tachycardia arises within 2 cm of the pulmonic valve, and R wave notching in the inferior lead occurs in cases of RVOT free wall circuits.
Ventricular Tachycardia during hypertrophic cardiomyopathy

Hypertrophic cardiomyopathy (HC) is a disease characterized by alterations in proteins of the myocardial cell leading to myocardial fiber disarray, hypertrophy of the heart and an increased SD incidence. This hypertrophy may often cause a dynamic obstruction of the left ventricle (LV) outflow tract, and in some occasions it leads to delayed cardiomyopathy (DC) and heart failure (HF). Pathologic ECGs are observed in approximately 95% of cases, although there is no characteristic ECU alteration indicative of a specific mutation. Dogs frequently show ECG signs of left ventricular enlargement, which are impossible to distinguish from those found in other heart diseases. In these cases, imaging techniques play a major role in establishing the correct diagnosis. The QRS voltage is usually increased, whereas low voltage is associated with a higher incidence of dilated cardiomyopathy (DC) during follow-up. Often dogs with HC present arrhythmias in the ECG. The most frequent supraventricular arrhythmia is atrial fibrillation. The presence of PVCs is also common, runs of non-sustained VT are found. The presence of sustained VT is infrequent. Hypertrophic cardiomyopathy dogs show different risk markers of SD. The trigger usually is ventricular tachycardia (VT) generally related to exercise and/or an intraventricular gradient increase.

Ventricular tachycardia during congestive heart failure

Heart failure (HF) is the pathophysiologic state in which the heart is unable to pump blood at the rate required by the metabolizing tissues, or when the heart can do it only with an elevated filling pressure. HF represents a complex clinical syndrome that may occur in patients with idiopathic myocardial disease (idiopathic cardiomyopathy) or appear in the course of many heart diseases. Ventricular tachycardia may occur as a consequence of myocardial hypertrophy, fibrosis, local ischemia, electrolyte abnormalities, catecholamines or myocardial stretch. With congestive heart failure prolonged action potential duration and the abnormalities in the ripolarization phase can due in part to a reduction of the rectifier K+ current I_{k1} and I_{k0}. These changes that are non uniform in the ventricular myocardium can cause increased dispersion of refractoriness that predispose to re-entrant arrhythmias.

References upon request