Supraventricular arrhythmias (SVA) are a group of rhythm disturbances which originate in the atria or require atrial or atrioventricular (AV) nodal tissue as part of their electrical circuit. In people SVA are classified according to their electrophysiological characteristics into: A) Sinus tachyarrhythmias; B) Atrioventricular nodal reciprocating tachycardia (AVNRT); C) Focal and nonparoxysmal junctional tachycardia; D) Atrioventricular Reciprocating Tachycardia (AVRT); E) Focal atrial tachycardia (FAT); F) Macro re-entrant atrial tachycardia. Atrial fibrillation (AF) is not usually included in this group seen its peculiar electrocardiographic and clinical finding. So far in veterinary medicine the orthodromic variant of AVRT, pre-excited atrial fibrillation, FAT, cavotricuspid isthmus (CTI)-dependent atrial flutter have been mapped and the underlining electrogenic mechanism and topographic distribution analysed. Supraventricular arrhythmias can induce periodic weakness and congestive heart failure caused by a tachycardiomyopathy. SVA can be treated acutely with IV drugs and electrical cardioversion and chronically either with oral antiarrhythmic treatment or with radiofrequency catheter ablation (RFCA). Intravenous drugs are usually indicated in case of hemodynamic compromise with still normal systolic function. The most commonly used drugs are lidocaine particularly in case of suspect OAVRT or vagal induced AF, diltiazem, sotalol or amiodarone in case of FAT or high ventricular response AF. Other options that can be used to interrupt fast SVA and particularly AF include biphasic cardioversion. Biphasic external electrical cardioversion can be successfully used in dogs to convert AF into sinus rhythm. However, duration of sinus rhythm after cardioversion is shorter in dogs with structural heart disease than in dogs with lone AF. Several SVA can be treated nowadays with RFCA also in veterinary medicine. Radiofrequency current is applied at the endocardial or epicardial surface and the voltage induces a current to flow between the electrode inside the cardiac cavity and a large, grounded, dispersive electrode on the surface. Once absorbed the energy is converted to heat that warms the tissue. The tissue heating is localised to a very short distance from the electrode-tissue interface.
Temperature at the electrode-tissue interface and the temperature in the tissue can be indirectly assessed during RF application using tip temperature. Tip temperature is a valuable tool that provides important information regarding the adequacy of tissue heating, minimising the development of coagulum and maximizing lesion size. Most electrophysiological (EP) laboratories use a thermocouple-tipped steerable 7F catheter connected to a RF generator to perform RFCA. Maximal catheter tip temperature and power output are usually set respectively at 65°C and 75 watts. Each RF application is performed at a controlled temperature and maximal ablation time for a particular target is 60 seconds. Histological studies run in dogs after catheter ablation of the atrioventricular (AV) junction showed, 4 to 5 days after catheter ablation, well-circumscribed areas of coagulation necrosis surrounded by a peripheral zone of haemorrhage and infiltration by mononuclear cells and neutrophils. Two months after RFCA, areas of fibrosis, granulation tissue, fat cell deposition, cartilage formation and chronic inflammatory cell infiltration have been reported.

In our electrophysiological (EP) laboratory so far we have a global success rate of 86 % with a complication rate of 8 % (1.7% stroke, 3.4% anaesthesia-related deaths, and 5% iatrogenic AV block that required pacemaker implantation) and a recurrence rate of 9%, only in cases of FAT. Only three cases required transeptal puncture with no complications noted. Our success rate of RFCA for accessory pathways is 91% with, in three cases, third degree AV block occurring postablation of 1 antero-septal and 2 mid-septal accessory pathways in three small breed dogs (body weight < 10 kg) with orthodromic AV reciprocating tachycardia resistant to medical therapy and provoking tachycardia-induced cardiomyopathy. Acute success rate of RFCA of focal atrial tachycardia is 74%, since 26% of focal atrial tachycardia was non-inducible with programmed or incremental atrial stimulation, atrial burst or isoproterenol infusion. So far we had only one minor stroke with spontaneous resolution during RFCA of a pulmonary vein ostium automatic focus. Other procedures including a few cases of RFCA of AFL and ventricular tachycardia arising from the right ventricular outflow tract in a dog with segmental arrhythmogenic right ventricular cardiomyopathy showed a high acute success rate with low complication and recurrence rate.

In the veterinary literature accessory pathways are the most common targets of ablation. In dogs most accessory pathways are right-sided, have unidirectional retrograde conduction, and are associated with various arrhythmias, including orthodromic AVRT and atrial fibrillation (AF). Anatomical distribution of accessory pathways reported in dogs includes most commonly posteroseptal and right-free wall locations. For AVRT the ablation catheter is placed at the atrial endocardial insertion of accessory pathways around the tricuspid valve annulus or within the coronary sinus. Ablation procedures are guided by intra-cardiac bipolar recording, searching for the shortest VA interval, or bypass tract potentials, during orthodromic AVRT or ventricular
pacing, and with unipolar recording with sharp and negative waveforms, the pole closest to the bypass tract can be assessed. For ablation within the coronary sinus ostium or body a cool-tipped ablation system should be used.

RFCA of cavo-tricuspid isthmus (CTI) dependent atrial flutter is considered the treatment of choice in people. The target of ablation in the cavo-tricuspid isthmus is an area in the low posterior right atrium between the tricuspid valve orifice and the inferior vena cava and the Eustachian ridge. The endpoint of ablation is the induction of bidirectional conduction block at the CTI demonstrated by a collision of waveforms, while pacing the coronary sinus, with a supero-inferior activation of the right free wall post-ablation, by the presence of slow conduction from the low right free wall and coronary sinus during pacing of the contralateral site, and by recording double potentials at the site of linear ablation at the CTI. So far there is only one communication of a long-term successful ablation of the CTI in two dogs.

The most common sites of origin of automatic tachycardias in dogs were found along the crista terminalis, right atrial appendage, triangle of Koch, pulmonary veins, and the coronary sinus. Mapping these sites of origin is usually performed using both bipolar and unipolar recordings. The goal of mapping is to identify the electrical epicentre of atrial activation characterized by the earliest signal on a bipolar recording, timed to the surface P waves (usually precedes the onset of P waves by a minimum of 20 ms, mean 38 ms). Sharp and negative unipolar recordings are also useful in this settings.

Chronic control of SVA with drugs can be achieved with different antiarrhythmic agent (diltiazem, sotalol, mexiletine, amiodarone, and digoxin) and combinations. So far few studies reported the efficacy of chronic control of SVA with oral drugs such as the combination of digoxin and diltiazem, or amiodarone in dogs with persistent AF. Digoxin should be used at a dose much lower than the one reported in veterinary textbooks (0.003 to 0.005 [max] mg/Kg PO BID). The goal is to use the lowest effective dose of digoxin, not the highest without side effects. Diltiazem can be given at 2-5 mg/Kg sustained release form twice daily in combination with digoxin. Through serum digoxin level (8 hours after pilling) should be obtained 5-7 days after initiation of treatment and target therapeutic value should be 0.5-1.0 ng/ml, which is significantly lower than the ranges reported by laboratory references. This drug association is ideal because contributes to slow down the electrical conduction through the AV node with a beneficial reduction of the ventricular response rate. The efficacy of treatment should be periodically monitored with 24h Holter recording.
References


