Arrhythmias: a clinician's guide to the identification and therapy of common arrhythmia presentations

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The presentation will discuss:
- anti-arrhythmic drug classification
- supraventricular tachyarrhythmias –recognition and treatment
- ventricular tachyarrhythmias – recognition and treatment
- bradyarrhythmias – recognition and treatment

Anti-arrhythmic Drug Classification

Class 1 : drugs which depress sodium movement in cardiac membranes (membrane stabilizers)
Class 1A – quinidine, procainamide, disopyramide
Class 1B – lidocaine, tocainide, mexilitine, phenytoin
Class 1C – flecainide, encainide, propafenone
Class 2 : sympatholytic drugs – propanolol, atenolol, nadolol, metoprolol, labetalol, esmolol
Class 3 : drugs which prolong action potential duration and refractoriness but not conduction velocity – amiodarone, sotalol, bretylium
Class 4 : calcium channel blockers – verapamil, diltiazem, nifedipine
Class 5 : anionic channel blockers – anilidine

Supraventricular Tachyarrhythmias

Supraventricular premature complexes (SVPCs)
Cause: usually secondary to atrial dilation eg mitral or tricuspid regurgitation, dilated cardiomyopathy, hypertrophic cardiomyopathy (cats). Other causes include digoxin toxicity and anaesthesia. Clinical signs from this arrhythmia are uncommon. Underlying heart disease is usually present.

Treatment: single, even frequent SVPCs do not usually require therapy. Treatment is typically aimed at the underlying cardiac disease and

congestive heart failure – if present.

Supraventricular tachycardia (SVTs)
Cause: usually secondary to atrial dilation as described above for SVPCs. May also occur as a primary defect (eg SVT in young Labradors due to accessory pathway conduction) . Clinical signs from this arrhythmia are common. SVTs may cause a pacing-induced cardiomyopathy (resulting in congestive heart failure), syncope and weakness due to reduced cardiac output. Underlying heart disease is usually present.

Treatment:
Acute therapy:
1) Vagal maneuvers eg carotid sinus massage, ocular pressure - rarely effective ; 2) precordial chest thump – often temporarily effective ; 3) IV drug therapy eg esmolol (ultra short-acting beta-blocker) 0.25 – 0.5 mg/kg IV slow bolus – not recommended if systolic dysfunction present . We currently offer radiofrequency catheter ablation of accessory pathway SVTs at VSS in collaboration with the RBH Electrophysiology Unit

Chronic therapy:
digoxin or digoxin plus diltiazem, digoxin plus atenolol, or diltiazem or atenolol alone, sotalol (digoxin not applicable in feline patients)

Atrial fibrillation (AF)
Cause: usually associated with severe underlying cardiac disease (usually advanced mitral valve disease or cardiomyopathy). In dogs it may occur as a primary condition ie idiopathic or 'lone' atrial fibrillation. In cases of idiopathic atrial fibrillation the ventricular rate is usually normal or mildly elevated. With the more common forms of AF, secondary to underlying heart disease, the rate is rapid, often above 200 bpm.

Treatment: digoxin or digoxin plus diltiazem, digoxin plus atenolol, or diltiazem or atenolol alone, sotalol, amiodarone. Direct current biphasic cardioversion (using a defibrillator) may also have merit in selective cases (typically includes therapy with short term K+ and Mg++ IV plus longer term amiodarone orally). (Only diltiazem, sotalol and atenolol applicable in feline patients)

Ventricular Tachyarrhythmias

Ventricular premature complexes (VPCs)

Cause: May occur associated with cardiac disease (eg aortic stenosis, dilated and hypertrophic cardiomyopathy, mitral valve disease, myocarditis) or many non-cardiac influences , particularly those involving systemic release of inflammatory mediators. Potential causes include trauma, anaesthesia, splenic mass lesions, gastrointestinal disease, metabolic disorders, pancreatitis, digoxin toxicity etc.. Clinical signs from this arrhythmia are uncommon, unless it is very frequent.

Treatment: single, even frequent VPCs may not require therapy, particularly if underlying cardiac disease is not present. Treatment is usually trialled if 1) severe underlying cardiac disease is present or the breed is predisposed to sudden death from ventricular fibrillation eg Boxers and Dobermans); 2) VPCs are frequent, particularly if multifocal and occurring in rapid pairs or short runs (<4 in a row) ie R on T phenomenon is a concern ; 3) there is a history of syncope or weakness that may be due to the arrhythmia. All patients with VPCs would ideally undergo 24 hour Holter monitoring or placement of a longer-term sub-cutaneous monitoring device (Reveal – Medtronics)

Maintenance Drug Therapy includes: sotalol, mexilitine, amiodarone, atenolol or flecainide (mexiletine, flecainide and amiodarone are not applicable to feline patients).

Ventricular Tachycardia (VT)

Cause: Ventricular tachycardia occurs for the same list of cardiac and non-cardiac causes as for VPCs outlined above. It can be sustained (persistent) or paroxysmal (intermittent). VPCs are usually present between episodes of paroxysmal tachycardia. Clinical signs from this arrhythmia are common as the rate is rapid (eg > 200 bpm).

Treatment: Guidelines for when to treat ventricular tachycardia have not been formulated however a guide to therapy will be discussed in this presentation. The most controversial form of ventricular ectopia is accelerated idioventricular rhythm. This has the appearance of ventricular tachycardia, but at a rate between 100-160 bpm. It is typically associated with non-cardiac aetiologies such as splenic mass lesions, pancreatitis, septicaemia etc..and does not cause significant haemodynamic impairment.

It is usually self-limiting over 1-7 days and may not require therapy.

Acute therapy
IV lidocaine – can be difficult to administer safely in feline patients (CNS issues)
OR: Esmolol bolus and CRI +/- concurrent lidocaine CRI. Contraindicated if CHF or myocardial failure present
+/- IV Magnesium

Maintenance Drug Therapy
sotalol
for dogs and cats: can cause a marked deterioration in cardiac output if underlying systolic dysfunction present. Often added orally to therapy while patient is commencing lidocaine boluses/ CRI. Cats: 10 mg bid – observe for signs of XS beta-blockade.

mexilitine (less negative inotropic effects). For difficult to treat arrhythmias, the efficacy may be improved if add sotalol or atenolol. For dogs only
atenolol for dogs and cats (if for feline patients: 6.25 -12.5 mg bid)
amiodarone: dogs only
flecainide: dogs only

Ventricular Fibrillation (VF)
Cause: this is typically a terminal event, resulting from severe cardiac or systemic disease. It may also occur during procedures such as anaesthetic induction or cardiac surgery. During this rhythm there are ventricular contractions. This is a form of cardiac arrest. There is no audible heart beat or palpable arterial pulses.

Treatment: the only effective therapy is electrical defibrillation. Chemical defibrillation is rarely effective and cardiopulmonary resuscitation (CPR) is usually ineffective if not combined with electrical defibrillation. Electrical defibrillation should be applied as soon as possible, taking priority over any other resuscitative measures.

Bradyarrhythmias
Sinus bradycardia
Cause: this arrhythmia is most commonly seen during anaesthesia. It also occurs with increased vagal tone, sick sinus syndrome, hypothermia, severe

hypothyroidism and drugs such as xylazine, beta-blockers and XS digoxin.

Treatment: the need for treatment is determined by the underlying cause and the presence of clinical signs. Under anaesthesia, drugs such as atropine and glycopyrrolate are indicated. Vagally-mediated rhythms are confirmed by the response to atropine at 0.04 mg/kg IV or s/c. The sinus rate should increase to 140-200 bpm if the cause is vagally-mediated. If the rate only increases to 70-130 bpm, sinus node dysfunction is suspected. Chronic anticholinergic medication is indicated only for those patients that are symptomatic and responsive to atropine (eg propantheline).

Sick Sinus Syndrome (SSS)
Cause: this is a primary disorder of the sinoatrial node and conduction system, best described in Miniature Schnauzers (also in Dachshunds, Pugs). Treatment: usually involves implantation of a pacemaker. Some patients will have a temporary response to sympathomimetics (eg terbutaline) or anticholingergics (propantheline) if they have a partial response to an atropine response test. Patients may also experience intermittent SVT due to sinus node dysfunction and therefore may not be candidates for positive chronotropic therapy

Atrioventricular (AV)blocks
First degree AV block
Cause: this occurs when conduction time to the ventricles from the sinus node is increased. This can occur at any point along this pathway. It may occur secondary to increased vagal tone, inflammation or degeneration, drug therapies (eg digoxin, beta-blockers, calcium channel blockers) or hyperkalaemia.

Treatment: no treatment is required. Presentations that are due to direct myocardial disease (eg degeneration/inflammation) may progress to higher forms of block.

Second degree AV block
Cause: this can occur as a normal finding (ie a functional second degree block), as occurs in atrial fibrillation to limit the amount of impulses conducting to the ventricles). It can also be physiological. It is usually pathological in the setting of a veterinary examination. Elevated vagal tone, drug therapies (as per first degree heart block) or primary cardiac disease

are possible causes (eg hereditary Bundle of His stenosis in Pugs). Treatment: no treatment is required, unless clinical signs are present. Clinical signs are rare unless high grade second degree AV block is present (ie multiple non-conducted P waves). For high-grade second degree AV block, treatment is as per third degree AV block.

Third degree (complete) AV block

Cause: usually unknown. Usually patients are middle-aged to geriatric, suggesting a degenerative process may be involved. Some patients are less than 1 year old, suggestive of either a congenital mechanism or myocarditis. Third degree AV block can less commonly be transient or intermittent, suggestive of an inflammatory or immune-mediated cause.

Treatment: Most patients have either syncope, weakness or both. Pacemaker therapy is usually required. A temporary pacemaker is required in some patients with marked clinical signs, prior to permanent pacemaker implantation.

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