Cardiac arrhythmias can cause or contribute to morbidity and mortality in critically ill dogs and cats. Successful management of arrhythmias often involves investigation and correction of an underlying non-cardiac disorder. In animals with primary cardiac disease, arrhythmia management is usually accomplished with drug therapy, however cardiac pacing is required in animals with certain bradyarrhythmias.

IDENTIFICATION OF INCITING CAUSES

Intrinsic cardiac disease (e.g., cardiomyopathy or conduction system disease) is the most common cause of symptomatic cardiac arrhythmia. Echocardiography, thoracic radiographs, electrocardiography, serum troponin I concentration, complete blood count and routine biochemical testing, serum thyroxine concentration, a careful history and physical examination are used to establish whether significant underlying cardiac disease is present. Many critically ill dogs, and some cats, develop cardiac arrhythmias in association with severe systemic disease. Contributors to arrhythmia formation include sepsis, hypotension, hypoxemia, myocardial hypoxia, electrolyte disturbance, acid-base alterations, pain, hypovolemia, and coagulopathy leading to myocardial hemorrhage or infarction. In addition, some drugs can lead to arrhythmia formation. Theophylline, terbutaline, phenylpropanolamine, and thyroxine supplementation can all contribute to ventricular or supraventricular tachyarrhythmias. Beta-blockers, calcium channel blockers, and digoxin can cause or contribute to bradycardia. Elimination of the cause, treatment of the underlying disease and correction of these physiologic alterations may be sufficient to control arrhythmias in some dogs and cats. In most dogs and cats with underlying cardiac disease and symptomatic cardiac arrhythmia, drug therapy is needed to control clinical signs and reduce the risk for sudden death.

ANTIARRHYTHMIC DRUGS

The most common classification system for antiarrhythmic drugs is the Vaughan-Williams classification scheme. In this scheme, there are four major classes of drugs. Many drugs act by more than 1 major mechanism of action. Other drugs are not well accounted for by this classification system, including digoxin and most of the drugs used for management of bradycardia. Most antiarrhythmic drugs have significant potential for side effect and the most common side effects for each drug should be well understood at the time drug therapy is initiated. While use of antiarrhythmic drugs for ventricular arrhythmias can reduce clinical signs, they have not been demonstrated to delay or reduce the risk of death in humans or animals with ventricular ectopy.

SURGICAL THERAPIES FOR ARRHYTHMIAS

There are certain arrhythmias that are better managed with surgical interventions, catheter – directed therapies, or electrical defibrillation. Advanced AV block is best managed with cardiac pacing. Sustained supraventricular tachycardia resulting from an accessory pathway can be managed with catheter ablation. The mainstay of therapy for people with serious ventricular arrhythmias is an implantable cardioverter-defibrillator, and these devices have been clearly shown to improve survival and reduce the risk of death. Unfortunately, these devices are expensive and the diagnostic algorithms designed for people often lead to inappropriate shocks if these pacemaker-like devices are implanted into dogs.

IS TREATMENT INDICATED?

There is still considerable debate within the opinion leaders in veterinary cardiology relative to the indications for antiarrhythmic therapy and the merits of one drug strategy vs. another for specific arrhythmias. In general, arrhythmias require treatment when they result in clinical signs such as weakness, syncope or collapse, if they are associated with sudden death (i.e., sustained ventricular tachycardia or advanced AV block), or if they contribute to CHF (e.g., atrial fibrillation). Many cardiologists would also agree that arrhythmias that occur in animals with cardiomegaly and advanced cardiac disease are often of much greater concern than arrhythmias in dogs with relatively normal hearts and minimal underlying heart disease.

MANAGEMENT OF SPECIFIC ARRHYTHMIAS

It is very important to establish the correct electrocardiographic rhythm diagnosis. When the rhythm diagnosis is in question then the following suggestions may be of help. If a lead II rhythm strip has been obtained and the diagnosis is unclear then additional limb leads are recommended. A chest lead may also help in identification of small P waves or in differentiation of supraventricular vs. ventricular arrhythmias. If the distinction between ventricular and supraventricular tachycardia persists then a bolus of lidocaine is usually safe and may help to change the rhythm. Finally, a longer period of ECG recording may help in identification of the rhythm.

Ventricular Tachycardia

Ventricular arrhythmias cause in interruption in sinus rhythm by a wide-complex QRS-T waveforms of unusual appearance. The ventricular premature contractions (VPCs) typically have a large T wave in the opposite direction of the QRS complex, no ST shelf, and no discrete relationship to P waves. Many clinical associations exist for ventricular arrhythmias in dogs and cats either with or without underlying cardiac disease. Ventricular arrhythmias are most commonly seen in association with significant cardiac disease in cats. Isolated VPCs, by themselves, pose little risk for mortality and have minor effects on cardiac performance, therefore cause few clinical signs and are not treated. In the Doberman pinscher, Boxer, and in cats, VPCs can be an early indicator of cardiomyopathy and may identify individuals at risk for anesthetic complications. Sustained ventricular tachycardia (e.g., ventricular tachycardia lasting >
30 seconds in duration) is associated with a medium to high risk for sudden death due to development of ventricular fibrillation. Ventricular tachycardia also significantly decreases cardiac output and can worsen CHF and contribute to weakness or collapse. Ventricular arrhythmias in systemically ill animals (e.g., vehicular trauma, splenic disease, GDV) that occur at a slow rate of 100-150 beats per minute (also termed idioventricular tachycardia or accelerated ventricular rhythm) rarely pose a clinically significant risk and usually do not create problems during anesthesia – in fact these arrhythmias are often less frequent during anesthesia than they are in the awake animals. Identification and elimination of inciting factors is recommended (see above). Treatment of ventricular tachycardia is usually initiated in an awake animal with clinical signs resulting from the arrhythmia (weakness, worsening CHF, collapse, or syncope) and in animals with sustained (> 30 seconds) and rapid (> 180-200 beats per minute) ventricular tachycardia. Treatment may be initiated if the above criteria are not present in animals with DCM who are known to be at high risk for sudden death (i.e., Doberman pinchers and Boxers). For chronic therapy, with the exception of beta-blockers, antiarrhythmic drugs have dubious efficacy in improving survival in human cardiac patients, even if they reduce arrhythmia frequency. Hemodynamically unstable ventricular tachycardia in the awake dog may be treated with IV lidocaine (bolus(es) at 2-4 mg/kg IV slowly, followed by 40-80 ug/kg/minute as a CRI). If this is ineffective then procainamide can be used (IV 20-50 ug/kg/min or IM 6 to 15 mg/kg q 4-6 h). Limited experience is available for slow IV amiodarone administration in the setting of severe ventricular arrhythmia (3-5 mg/kg boluses up to 10 mg/kg). Clinical experience and several reports indicate that IV amiodarone, in the dog, can be associated with hypotension, collapse, and allergic or anaphylactic-like reactions with hyperemia or urticaria, so the author is reluctant to use amiodarone IV in a dog that is conscious and alert, but more willing to use the drug in the setting of CPR or pulseless ventricular tachycardia. For chronic oral therapy, a variety of medications can be used including procainamide (slow release formulation), beta-blockers (sotalol, atenolol, metoprolol or propranolol) or other class I antiarrhythmic drugs (i.e., mexiletine, or tocainide). Clinical experience is growing with amiodarone, and this drug is often successful for management of rapid, sustained ventricular arrhythmias that have not responded to lidocaine, procainamide or sotalol. Current preferences of the author for long-term management of sustained ventricular tachycardia in dogs include amiodarone (8 to 15 mg/kg BID for 7 days, then 5 to 10 mg/kg BID), sotalol (1-3.5 mg/kg BID), or mexiletine (5-8 mg/kg TID) combined with atenolol (0.25-0.6 mg/kg BID). Doberman pinchers are prone to a hepatotoxicity when treated with amiodarone. Drug combinations which have proved effective in Boxers with arrhythmogenic right ventricular cardiomyopathy include sotalol, mexiletine with atenolol, or mexiletine with sotalol.

**Ventricular Fibrillation**

Immediate electrical defibrillation is indicated for the treatment of ventricular fibrillation. The likelihood of success is inversely proportional to the amount of time in ventricular fibrillation, with successful defibrillation being reduced by approximately 50% for every 3 to 5 minutes of time delay. Drug therapy alone has been proposed but is virtually ineffective for ventricular fibrillation (unless CPR can be continued for 15 minutes or longer after administration of KCl). However drug therapy can be combined with electrical defibrillation to reduce the defibrillation threshold, the energy required to electrically defibrillate the heart. Intravenous amiodarone (3-5 mg/kg) and magnesium (0.15-0.3 mEq/kg) have been used in animals that are refractory to repeated attempts at electrical defibrillation. In dogs with ventricular fibrillation, the author prefers to use low doses of epinephrine as higher doses of epinephrine seem to be associated with difficult defibrillation and recurrent ventricular fibrillation.

**Supraventricular Tachycardia**

Supraventricular tachycardia is recognized by tachycardia with QRS-T complex that is (usually) of similar appearance to the normal P-QRS-T. This arrhythmia typically results in a narrow QRS complex tachycardia (unlike ventricular tachycardia), and P waves may be identified but can be superimposed in the QRS or T wave. Supraventricular tachycardia can sometimes be differentiated from sinus tachycardia based on the response to a vagal maneuver, with sinus tachycardia responding with a gradual slowing in heart rate and rebound to the prior rate following termination of the vagal maneuver. Supraventricular tachycardia typically does not respond with a change in heart rate or the result is an abrupt drop in heart rate with termination of the arrhythmia. Atrial stretch/dilatation can contribute to the development of supraventricular tachycardia (esp. dogs with endocardiosis), and hypoxemia, respiratory disease, or intoxication with digoxin, theophylline, or thyroid hormone can also cause this arrhythmia. In dogs and cats with sustained arrhythmia an accessory pathway connecting the atria and the ventricle (i.e., WPW) should be considered. While SVT is usually associated with a low risk for mortality due to the arrhythmia, sustained arrhythmia can lead to weakness, syncope and CHF. Dogs with chronic valvular disease and supraventricular tachycardia often have advanced heart disease and may be prone to anesthetic complications. Drug therapy is appropriate for animals with rapid supraventricular arrhythmias (rates above 260 beats/minute in the cat or above 220 beats/minute in the dog) or for slower or non-sustained SVT when seen in association with CHF or severe systemic disease. In cases where emergency treatment is required, IV diltiazem can be administered in an initial 0.25 mg/kg IV bolus over 2 minutes. Subsequent 0.25 mg/kg boluses can be repeated at 15 minute intervals until conversion occurs or to a maximum dose of 0.75 mg/kg. Alternative drugs include verapamil (0.05 mg/kg slow IV boluses up to a total dose of 0.15 mg/kg), propranolol (0.02 to 0.06 mg/kg slow IV q 8 h) and a short acting beta-blocker named esmolol (incremental doses of 0.05 to 0.1 mg/kg q 5 min up to a maximum dose of 0.5 mg/kg). Esmolol's effects are short-lived, and if arrhythmia conversion does not occur then other drugs with negative inotropic properties (i.e., diltiazem or verapamil) can usually be safely given 30 minutes after esmolol administration. For chronic management of SVT, oral therapy is accomplished using either digoxin, calcium channel blockers (diltiazem, verapamil) or beta-blocking drugs (propranolol, atenolol, metoprolol, sotalol). A recent article described conversion of supraventricular tachycardia in dogs following treatment with lidocaine. Digoxin is usually the drug of choice when SVT is accompanied by heart failure. High doses of procainamide have been reported to be effective in Labrador retrievers with SVT as these dogs often have an accessory pathway.
Atrial Fibrillation

Atrial fibrillation is recognized by a rapid, irregularly irregular rhythm with upright QRS complexes (supraventricular appearance) in Lead II and (usually) chaotic undulations in the baseline. This rhythm is typically seen with significant cardiac/atrial enlargement in cats and dogs less than 50#. In large breed dogs, atrial fibrillation may occur with or without underlying heart disease. Animals with atrial fibrillation are at increased risk for mortality, but mortality risk is probably due more to the degree of underlying cardiac disease instead of specifically due to atrial fibrillation leading to arrhythmic sudden death. The goal of therapy is different in dogs with underlying heart disease compared to those without underlying myocardial or valvular disease. In animals with underlying heart disease, the goal of many cardiologists is not to convert back to sinus rhythm but simply to slow the heart rate to a rate that is slow enough that cardiac filling will be optimized for most cardiac cycles. Oral digoxin is initiated and if adequate rate control is not achieved with digoxin alone then additional drugs such as β-blockers (propranolol, atenolol or metoprolol) or calcium channel blockers (diltiazem) can be used to reduce the heart rate to between 100-160/minute. For treatment in animals without underlying heart disease (e.g., normal thoracic radiographs and echocardiogram) the goal is conversion from atrial fibrillation back to sinus rhythm. Some veterinary cardiologists feel that attempts to convert back to sinus rhythm in all dogs may be the preferable approach. Quinidine (6-11 mg/kg IM, Q 6 hr) can be attempted, and diltiazem (0.5 to 1.5 mg/kg PO q 8-12 hr) or sotalol may convert atrial fibrillation to sinus rhythm in some dogs. Amiodarone can also be used successfully in some dogs to convert the rhythm back to sinus rhythm or control the heart rate. 3,4 ECG synchronized cardioversion may be attempted, although this technique has significant risk of inducing a worse arrhythmia and requires heavy sedation or anesthesia to be performed.

Third Degree AV Block and High Grade Second Degree AV Block

Third degree AV block is recognized by a total lack of relationship between the P waves and the QRS-T complexes. High grade second degree AV block has some conducted and some non-conducted P waves with a fixed P-R interval. From the perspective of clinical risk, clinical signs, and clinical management, high grade second degree AV block is managed in the same fashion as third degree AV block. With both arrhythmias, the QRS-T complexes may originate from ventricular or junctional tissue. Typically, these cardiac rhythms are due to primary pathology of the AV node or bundle of His. There is a medium to high risk for sudden death and the arrhythmias commonly causes weakness, modest weight loss, lethargy, and syncope. Longstanding third degree AV block can lead to congestive heart failure. Pacemaker implantation should be recommended to all clients as the treatment of choice as this is the therapy which is associated with a superior clinical outcome. 5 When pacemaker implantation is not possible, anticholinergic therapy (propantheline), theophylline or sympathomimetic therapy (terbutaline) can be considered. There is some evidence that use of sympathomimetic drugs may be associated with an adverse outcome. If inflammation is suspected in recent onset AV block, therapeutic trial with corticosteroids may also be attempted. Lyme disease has been associated with disease of the conduction system and serology can be performed is dogs from endemic areas. Animals with AV block are at very high risk of anesthesia complications and transthoracic or transvenous cardiac pacing should be available for immediate use in all cases. Temporary transvenous pacing can be used in dogs with congestive heart failure that are judged to be unsuitable for immediate anesthesia for pacemaker implantation. Third degree AV block is better tolerated by some cats as they often have a faster escape rhythm. Some elderly cats with third degree AV block do well with medical therapy and may not require pacemaker implantation.

Sinus Arrest and Sick Sinus Syndrome

Sinus arrest is identified by a pause of greater than 2 P-P intervals without evidence of depolarization of the sinus node. This arrhythmia is part of the sick sinus syndrome, a syndrome characterized by bradycardia due to sinus node dysfunction, typically accompanied by short bursts of supraventricular tachycardia. Individuals with sinus arrest or sick sinus syndrome are at modest increased risk for mortality; however they are at very high risk for bradycardia and circulatory collapse during anesthesia. Many dogs with sick sinus syndrome are asymptomatic at rest and only rarely experience syncope. The transient nature of this rhythm disturbance can lead to misdiagnosis as a seizure disorder and many cases have been previously treated with anticonvulsant therapy. An atropine response test may be useful to determine the potential effectiveness of anticholinergic drugs. The combined effects of sinus node disease and anesthesia can readily lead to deterioration and symptomatic bradycardia. In these cases, transthoracic pacing can be used, but it can be difficult to continue transthoracic pacing in a dog that is recovering from anesthesia – temporary transvenous pacing or permanent pacemaker implantation may be required. Catecholamine infusion can also be used to increase heart rate, but cardiac pacing is usually safer and more predictable. For long-term treatment, permanent pacemaker implantation is preferred for animals with repeated syncope, especially those who do not respond favorably to drug therapy with propantheline or theophylline. The prognosis following pacemaker implantation is good.

References