Proceeding of the NAVC
North American Veterinary Conference
Jan. 8-12, 2005, Orlando, Florida

Reprinted in the IVIS website with the permission of the NAVC
http://www.ivis.org/
CARDCARRHYTHMIAS:
WHAT TO TREAT, WHEN AND HOW

Rebecca L. Stepie, DVM, MS, DACVIM
School of Veterinary Medicine
University of Wisconsin, Madison, WI

THE PATIENT-ARRHYTHMIA INTERACTIONS

Cardiovascular pathology and clinical situations interact to cause or aggravate arrhythmias. A frequent cause of arrhythmias is primary cardiovascular disease, but other systemic abnormalities may directly cause or contribute to the occurrence of arrhythmias. In any case, the best “therapy” for most arrhythmias is to eliminate the underlying cause (e.g. therapy of pulmonary edema to reduce hypoxia-related arrhythmias). In the case of systemic disease-related arrhythmias, it is especially important to treat the disease, not necessarily the arrhythmia (e.g. hyperkalemia-related bradyarrhythmias require fluid therapy and sodium bicarbonate or calcium, not a pacemaker). The clinician can usually define the cause of arrhythmias based on history, clinical examination findings, blood tests and other routine diagnostic tests.

The presence of heart disease does not always mean it is the cause of the arrhythmia, but chronic heart disease patients may have more risk factors for arrhythmia development than other patients. Systemic derangements common in chronic heart failure patients (e.g. acid/base or electrolyte disorders and alterations in renal function due to medications given for heart failure management) may make drug toxicity more likely (e.g. hypokalemia) or may lead to decreased elimination of medications and accumulations of toxic plasma drug concentrations.

APPROACH TO ANTIARRHYTHMIC THERAPY

Correct identification of the arrhythmia is the most important step in the process, but given the rhythm a name is just part of the puzzle. Some characteristics of arrhythmias (e.g. bradycardia vs. tachycardia, supraventricular vs. ventricular origin of ectopic complexes) lead the clinician to a specific choice of medication. Other characteristics (e.g. effect on cardiac output, probability of sudden death) help the clinician to decide if an arrhythmia is life-threatening (i.e. should be treated immediately), symptom-aggravating (i.e. may be involved in clinical signs of other disease) or clinically unimportant (i.e. requires only monitoring). This information is used to decide if complete resolution is needed to avert death (e.g. high rate ventricular tachycardia) or control but not complete resolution is sufficient (e.g. decrease in frequency or repetitiveness of paroxysmal tachycardia or administration of anticholinergics in vagally-induced bradycardias).

A final and important step in therapy of arrhythmia occurs if the therapy does not appear to be working. At this point, the diagnosis should also be reconsidered. If the diagnosis is correct, factors that limit response to medication (e.g. hypokalemia) should be investigated. On occasion, a change in the arrhythmia during therapy can lead the clinician to reconsider the original diagnosis.

Choice of antiarrhythmic therapy

Beyond therapy or resolution of any contributory clinical situation, antiarrhythmic therapy can include direct treatment of the arrhythmia with medical (drug) or mechanical (e.g. electrical conversion) therapy or palliative methods that control the clinical signs without attempting to address the origin of the arrhythmia directly (e.g. pacemaker implantation for 3rd degree AVB). The clinician can choose the optimal therapy based on origin (e.g. supraventricular vs. ventricular) or proposed mechanism of the arrhythmia (e.g. reentry vs. increased automaticity). In addition, choices regarding choice of medication and route of delivery are affected by rapidity of onset of effect.

THERAPY OF BRADYCARDIC ARRHYTHMIAS

Sinus Bradycardia

Bradycardias are heart rhythms that occur at a rate slower than the normal rate. Sinus bradycardias are frequently associated with identifiable physical causes including use of sedatives or anesthetic agents, antiarrhythmic medications (e.g. beta blockers) and medications or conditions that alter electrolyte balance. In addition, conditions that intensify vagal tone (hypothermia, deep sleep, neurologic, gastrointestinal or pulmonary diseases) may be associated with slow but normally conducted heart rhythms. These sinus bradycardias are usually self-limiting, not associated with over clinical signs of low cardiac output and resolve with removal of the underlying cause. If sinus bradycardia is associated with clinical signs of weakness or known hypotension, the heart rate (HR) can be supported with medications (e.g. anticholinergics, IV catecholamines, Table 1) until diagnostics can be pursued. If the clinical importance of a bradycardic rhythm is in doubt, an atropine response test may be helpful. A baseline ECG is recorded, 0.04 mg/kg of atropine is given IM and a follow-up ECG recorded 30 minutes later. A normal response includes a HR increase of ≥ 50% with normal conduction. If this response does not occur, sinus node disease rather than high vagal tone may be suspected as the cause of bradycardia.

Sinus Arrest, Bradycardia/Tachycardia Syndromes (Sick Sinus Syndrome)

Periods of sinus arrest without development of an escape rhythm for greater than approximately 3-5 seconds lead to neurologic signs (e.g. staggering, syncope) in dogs with sick sinus syndrome. The sinus arrest seldom leads to sudden death, but frequency of events may severely affect quality of life. Short-term therapy is the same as that for sinus bradycardia, but immediate pacing may be required if non-responsive to anticholinergics or if the animal is severely symptomatic. Although theophylline or terbutaline therapy are frequently recommended for management of this disease, therapy with these drugs alone is usually not effective. In the long term, sinus arrest and bradycardia/tachycardia syndromes are most effectively treated with pacemaker implantation.

Atrial Standstill (Atrial Asystole)

Atrial standstill (asystole) may be associated primary disease of the atrial muscle (atrial myopathies) or occur more commonly secondary to hyperkalemia. The atrial myopathy-related atrial standstill is treated with pacemaker implantation if the rhythm is bradycardic. Hyperkalemia at a concentration high enough to lead to atrial standstill is an emergency. Immediate therapy includes anticholinergics, IV saline and IV calcium administration along with definitive therapy to reduce potassium concentrations (sodium bicarbonate, insulin etc.). A rare cause of atrial standstill is toxic concentrations of digoxin; these cases respond to therapy for digoxin toxicity.
Bizarre escape rhythms associated with atrial standstill should not be mistaken for ventricular tachycardia; suppression of these life-sustaining rhythms may be lethal.

Atrioventricular Blocks (AVB)
The most common cause of 1st or 2nd degree AVB is high vagal tone or use of vagomimetic drugs (e.g. digoxin). In these cases, the underlying cause should be treated, rather than the AV block. If second degree AV block is symptomatic and does not respond to anticholinergics, pacing may be required. Symptomatic "high grade" 2nd degree AVB (long strings of P waves without any QRS complexes) require immediate pacing. Third degree AVB usually reflects primary nodal disease and anticholinergics are typically ineffective. Immediate therapy is warranted because 3rd degree AVB can lead to sudden death if the escape focus fails. If the patient is symptomatic, IV catecholamines may induce subsidiary pacemaker activity in the short term but immediate temporary or permanent pacemaker implantation is recommended.

**Figure 1:** Atrial standstill due to severe hyperkalemia

**Table 1: Medications Commonly Used in the Therapy of Arrhythmias (2004)*

<table>
<thead>
<tr>
<th>Category</th>
<th>Medication</th>
<th>Dose Information</th>
</tr>
</thead>
<tbody>
<tr>
<td>Parasympatholytics</td>
<td>Atropine</td>
<td>(D,C) 0.02-0.04 mg/kg IV, IM, SC</td>
</tr>
<tr>
<td></td>
<td>Glycopyrrolate</td>
<td>(D,C) 0.005-0.01 mg/kg IV, IM, SC</td>
</tr>
<tr>
<td>Sympathomimetics</td>
<td>Dobutamine</td>
<td>(D) 2-15 mcg/kg/min (start low and titrate up)</td>
</tr>
<tr>
<td></td>
<td>Dopamine</td>
<td>(D) 2-10 mcg/kg/min (start low and titrate up)</td>
</tr>
<tr>
<td></td>
<td>Epinephrine</td>
<td>(D) 5 mcg/kg IV, 0.1-0.4 mcg/kg/min (start low and titrate up)</td>
</tr>
<tr>
<td></td>
<td>Isoproterenol</td>
<td>(D) Dilute 1 mg in 500 cc of 5% dextrose or Ringer’s, infuse IV at 0.5-1 ml/min (1-2 ug/min) or to effect</td>
</tr>
<tr>
<td>Antiarrhythmics</td>
<td>Amiodarone</td>
<td>(D) 10-20 mg/kg q 12 hr</td>
</tr>
<tr>
<td></td>
<td>Atenolol</td>
<td>(D) 6.25-25 mg q 12-24 hr, start low/titrante, (C) 6.25-12.5 mg q 12-24 hr</td>
</tr>
<tr>
<td></td>
<td>Diltiazem</td>
<td>(NOT sustained release): (D): 0.5-1.5 mg/kg PO q 8 hr, 0.25 mg/kg IV (over 3 min), (C): 1.5-3 mg/kg PO q 8hr</td>
</tr>
<tr>
<td></td>
<td>Esmolol</td>
<td>(D/C) 0.5 mg/kg IV bolus, 25-200 mcg/kg/min</td>
</tr>
<tr>
<td></td>
<td>Lidocaine</td>
<td>(D) 2-4 mg/kg IV bolus, 30-80 mcg/kg/min, (C) 0.2-0.4 mg/kg IV slow bolus</td>
</tr>
<tr>
<td></td>
<td>Mexiletine</td>
<td>(D) 5-8 mcg/kg q 8 hr</td>
</tr>
<tr>
<td></td>
<td>Procaainamide</td>
<td>(D) 5-15 mg/kg IV, IM q 6 hr, 10-20 mg/kg PO q 6 hr, 10-40 mcg/kg/min</td>
</tr>
<tr>
<td></td>
<td>Propranolol</td>
<td>(D) 0.1-2 mg/kg PO q 8 hr, start low/titrante, 0.04-0.06 mg/kg IV slowly, (C) 2.5-5 mg total dose q 8 hrs, start low/titrante</td>
</tr>
<tr>
<td></td>
<td>Sotalol</td>
<td>(D,C) 1-2 mg/kg PO q 12 hr</td>
</tr>
<tr>
<td></td>
<td>Verapamil</td>
<td>(D,C) 0.05 mg/kg q 30 min up to 0.15 to 0.5 mg/kg total</td>
</tr>
</tbody>
</table>

*The most recent editions of Current Veterinary Therapy (X, XI, XII) are recommended as references for drug doses and additional medical information. These doses are suggestions only and any medical therapy must be tailored to the patient based on the clinician’s judgement.
SUPRAVENTRICULAR TACHYARRHYTHMIAS

Sinus Tachycardia
Sinus tachycardia is almost always the result of clinical derangements not related to primary cardiac disease. The underlying situation commonly involves hypotension, but pain may also contribute. The underlying disease should be treated; sinus tachycardia is not a fatal arrhythmia and usually resolves without direct therapy.

Atrial Premature Complexes, Atrial Tachycardia (AT), Supraventricular Tachycardia (SVT)
Single atrial ectopic depolarizations (APCs) may not require therapy if they occur at low frequency, are not related to CHF or known structural heart disease and are not associated with clinical signs. They often resolve with systemic problem resolution. Supraventricular tachycardia or atrial tachycardia often occurs at extremely rapid HR (~400 bpm in dog) and may severely decrease cardiac output. A “vagal maneuver” (ocular pressure or carotid sinus stimulation) may abruptly abolish the tachycardia, but recurrence is common if no other method is used. The first choice in medical therapy of AT or SVT is IV beta-blockers (BB, esmolol or propranolol) or calcium channel blockers (CCB, diltiazem or verapamil). These medications may be given orally if no IV access is available. Chronic medical therapy of frequent APCs, SVT or AT usually consists of BB or CCB (do not combine) if no definable underlying disease is present, or digoxin plus BB OR CCB (one or the other) if cardiac disease present.

Atrial Fibrillation (AF)
If spontaneous AF is diagnosed, underlying heart disease is usually present. A new diagnosis of AF may be the cause of sudden decompensation in a previously stable CHF patient. The therapeutic goal is control of the ventricular response to AF. Acute decreases in HR are recommended if ventricular rate is > 180 bpm (dogs) or > 200 bpm (cats). The most rapid and effective therapy presently used is IV diltiazem. Oral diltiazem or BB can be used if IV is not available. BB may lead to acute worsening of CHF and should be used with caution in patients without CHF and should not be used when overt CHF is present. Oral digoxin therapy can be started concurrently with either BB or CCB. The chronic therapy of AF is the same as that used for SVT. In severe heart failure, the patient may be dependent on an elevated HR to support cardiac output; antiarrhythmic drugs used to slow the ventricular response rate to atrial fibrillation are often “titrated” to the desired clinical effect. A typical acceptable HR in AF is 140-160 bpm (dogs) or 160-180 bpm (cats).

Table 2: Differentiation of accelerated idioventricular rhythm vs. ventricular tachycardia.

<table>
<thead>
<tr>
<th></th>
<th>Ventricular Tachycardia</th>
<th>Accelerated Idioventricular Rhythm</th>
</tr>
</thead>
<tbody>
<tr>
<td>Depolarization Rate</td>
<td>Usually &gt; 160 bpm</td>
<td>Usually &lt; 160 bpm</td>
</tr>
<tr>
<td>First abnormal complex</td>
<td>Premature (early diastolic)</td>
<td>Late diastolic</td>
</tr>
<tr>
<td>Relationship to sinus rhythm</td>
<td>Significantly faster</td>
<td>Within ~ 5-10 bpm</td>
</tr>
<tr>
<td>Associated with pulse deficits</td>
<td>Yes</td>
<td>Usually no</td>
</tr>
</tbody>
</table>

Figure 2: Ventricular tachycardia in a dog

Figure 3: Accelerated idioventricular tachycardia in a dog
VENTRICULAR ARRHYTHMIAS:
Accelerated Idioventricular Rhythms (AIR)
Accelerated idioventricular rhythms (alternative terms: “fast idioventricular rhythm” “slow ventricular tachycardia” or “idioventricular tachycardia”) are very common in canine critical care patients, and are commonly confused with ventricular tachycardia. Misdiagnosis may lead to administration of toxic doses of antiarrhythmic medication in an unsuccessful attempt to convert “VT”.

Because AIR is frequently noted as a complication of severe systemic illness and is usually not associated with evidence of impaired cardiac performance, therapy is aimed at the underlying problem. Hydration, oxygenation, electrolyte and acid-base abnormalities should be rectified and specific therapy for the underlying disease instituted. Pain should be assessed and managed appropriately.

No direct antiarrhythmic therapy is indicated if ALL of the following criteria apply:
- If HR and blood pressure are within normal ranges and stable
- If no clinical signs are apparent
- If no other ventricular ectopics interrupt AIR

Direct antiarrhythmic therapy (as for VT) may be indicated if ANY of the following occur:
- HR > 130-160 bpm
- If hypotension or unstable blood pressure
- If premature ventricular ectopics initiate or interrupt AIR, or if R wave of ectopic occurs on the T waves of the previous depolarization (“R-on-T”)  
- Clinical signs of poor cardiac output present

VENTRICULAR PREMATURE COMPLEXES (VPCS) OR VENTRICULAR TACHYCARDIA (VT)
The decision to treat ventricular arrhythmias depend on the arrhythmia’s effect on cardiac output and electrical instability of the arrhythmia. Ventricular arrhythmias significantly decrease cardiac output if they occur concurrently with CHF, in the setting of severe myocardial dysfunction or if the arrhythmia is highly repetitive or provides large proportion of total HR. Ventricular arrhythmias may be electrical unstable (and lead to sudden death) if the ventricular ectopics occur at such a high HR that the ectopic falls on the T wave of the previous sinus complex (“R on T”). If cardiac output is compromised or electrical instability is suspected, therapy is warranted. The cornerstone of acute therapy of ventricular arrhythmias remains IV lidocaine. If lidocaine is unsuccessful or contraindicated, IV procainamide or BB may be used. Be sure to administer significant doses of the medication before diagnosing lack of response. If lidocaine is administered as a bolus and is effective, a continuous rate infusion (CRI) should be started immediately. Intermittent boluses of lidocaine may be needed until CRI reaches therapeutic blood levels. When the patient is stable and can tolerate oral medications, these drugs can be started while the CRI is still running. Once oral medications are started, the rate of the CRI can be decreased by 50% every 6 hours. In cats, lidocaine can be used but with great caution. IV or oral BB may be a better choice in cats that are not critically unstable.

Choices for therapy of chronic therapy of ventricular arrhythmias include oral drugs such as sotalol (class II [beta-blocker] and Class III [K channel blocker] effects) and mexiletine (class I effects, similar to lidocaine). Sotalol is very effective and usually well-tolerated, but should be used with caution with patients with heart failure or that are hypotensive. Mexiletine is effective against many ventricular arrhythmias, especially in short term, and may be combined with oral BB (e.g. atenolol) for added efficacy. Amiodarone is used less frequently to treat ventricular arrhythmias; amiodarone is highly effective against resistant arrhythmias but more worrisome toxicity profile than the other medications suggested.