CARDIOVASCULAR PHARMACOTHERAPY

Hemodynamic Drugs and Antiarrhythmic Agents

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Successful management of the patient presenting with acute cardiovascular compromise requires a thorough working knowledge of the therapeutic agents available for treatment. This article focuses on the indications for, dosing, and monitoring employed for successful use of the vasoactive agents and antiarrhythmic medications in current widespread use in the veterinary intensive care setting.

In the last few decades, solid advances have been made toward an understanding of the underlying hormonal changes occurring in cardiovascular disease, particularly in the heart failure state, allowing a rational approach to the treatment of patients in various stages of heart disease. With the information obtained in the research and clinical settings, medications used to alter the neurohumoral effects of heart failure (particularly angiotensin-converting enzyme inhibitors [ACE-Is] and beta-blocking drugs) have come to the forefront of cardiac therapeutics. Over this same period, key therapeutic trials in people have uncovered some interesting and repeatable results that have called into question many of the conventional approaches to the treatment of patients with heart failure or cardiac arrhythmias. The reader is encouraged to use the reference list to learn about the current approaches to the treatment of chronic heart failure. These continue to change with ongoing medical research and the development of newer therapeutic modalities. Human
medicine has moved from an era of anecdotal discussion to a time of evidence-based medicine. Large randomized therapeutic trials are needed in veterinary medicine to help better define the rational use of cardiotherapeutic agents and to determine which medications are most beneficial. As a critical care veterinarian, one is more concerned about the short-term needs of the particular patient; successful outcome of the initial management period allows there to be a long term. Questions about long-term therapy can be answered after successful negotiation through the crisis period. The indications for some of the therapeutic agents discussed in this article beyond the acute setting are disputed, but these medications continue to have a place in the stabilization period of the patient presenting with intractable heart failure or in a hypotensive crisis.

In all areas of medicine, the successful approach to the individual patient requires an accurate initial assessment of the patient and a diagnosis of the medical condition. In all but the most unstable patients, a brief but thorough physical examination should be carried out to identify existing problems so that a diagnostic and therapeutic strategy may be developed. The basic assessment of the patient should minimally include the evaluation of mucous membrane color and capillary refill time, heart rate and rhythm, respiratory rate and character, and auscultation of the heart and lungs. Pulmonary crackles, muffled heart or lung sounds, or cardiac murmurs may be identified. Brief abdominal palpation can help to identify mass lesions, the presence of fluid, or excessive abdominal splinting suggesting possible extracardiac problems. With this initial information, one can usually formulate a working diagnosis and consider the appropriate diagnostic and therapeutic plan. It is appropriate to obtain an accurate body weight for the patient so that medication dosages can be accurately calculated. If possible, a full laboratory evaluation, including a complete blood cell count, biochemical profile, venous blood gas, and urinalysis, should be obtained. The information derived from an arterial blood gas measurement is helpful in patients with respiratory distress, but the positioning of the patient to obtain the sample may be too stressful for many acute patients. Analysis of "quick assessment tests" (packed cell volume, total solids, blood glucose, and Azostix [Bayer Co., Elkhart, IN]) can give some immediate information.

Armed with the initial physical assessment and a thorough clinical history, the clinician decides if additional tests are necessary to confirm the diagnosis and whether the patient is stable enough to undergo these tests. A risk is taken with the patient only if an experienced clinician is truly unable to make a clinical decision without these tests. An echocardiogram or thoracic radiographs may be necessary to be certain the approach to patient stabilization is appropriate.

Once the diagnosis is established, a treatment plan is instituted. The placement of an intravenous catheter (once patient stabilization permits it) is usually necessary to allow delivery of medications and intravenous fluid therapy where appropriate. The placement of invasive monitoring tools such as an arterial catheter (for direct monitoring of blood pres-
sure), pulmonary artery catheter (for measuring pulmonary artery pressures and pulmonary capillary wedge pressures), or urinary catheter (for monitoring urine output) may be deemed necessary depending on the need for detailed information so as to achieve stabilization of the patient. Although the information obtained with these sophisticated monitoring systems is helpful in tracking the hemodynamic changes and therapeutic responses, all the medications listed can be used successfully without these monitoring parameters. It is important to remember that information derived from monitoring tests should not replace vigilance on the part of the clinician. Always evaluate the patient as a whole, and question the validity of information obtained that does not fit the clinical picture. First, do no harm. Successful management of any critical patient includes accurate clinical assessment, frequent re-evaluation for the response to therapy, and alteration of medications as necessary to achieve the desired response. The accuracy of the initial diagnosis should be questioned if the patient is not responding appropriately to therapy.

**VASOACTIVE AGENTS**

**Inotropic Agents**

Inotropic agents are drugs used to stimulate cardiac contractility and to support the cardiovascular system. These medications have varying effects on blood pressure and heart rate. Clinical trials in human patients have repeatedly shown that long-term use of all the inotropic agents, with the exception of digitalis glycosides, has an adverse effect on survival. Despite this surprising and alarming information, inotropic agents continue to have a place in the acute care setting, where they are most often used to address refractory systemic hypotension, hypovolemic or cardiogenic shock, oliguric or anuric renal failure, fulminant pulmonary edema, or low-output congestive heart failure.

**Catecholamines**

This class of drugs provides the most powerful cardiac stimulation of all the inotropic agents by stimulating the sympathetic nervous system. Individual drugs have variable effects on heart rate and blood pressure as well as variable inotropic strength depending on the type of adrenergic receptors stimulated. It is necessary to briefly review the different types of adrenergic receptors to fully understand the mechanisms of the drugs in this class. The alpha-adrenergic receptors are primarily located on the peripheral arterioles. Stimulation of these receptors effects vasoconstriction, resulting in elevations in the blood pressure with reflex slowing of the heart rate. Alpha-1 (α1) agonists cause either vascular dilation or constriction depending on the intensity of stimulation and the specific vascular beds. The α2 agonists cause pure vasoconstriction. The beta-receptors are also broken down into two subtypes,
with the β₁-receptors being concentrated in the heart and the β₂-receptors concentrated in the smooth muscles of the bronchi, peripheral blood vessels, and uterus. Stimulation of the β₁-receptors results in increases in heart rate, atrioventricular (AV) conduction, and strength and speed of contraction of the myocardium. Stimulation of the β₂-receptors causes bronchodilation and vasodilation (resulting in decreased afterload). The beta-agonist effects are mediated via the stimulation of intracellular G-proteins, resulting in activation of adenyl cyclase, the enzyme that catalyzes production of cyclic adenosine monophosphate (cAMP) from adenosine triphosphate. With the increase in intracellular cAMP, calcium channels are opened, promoting strength of contractility. Calcium reuptake by the sarcoplasmic reticulum is also stimulated, resulting in improved myocardial relaxation (positive lusitropic effects). The sympathomimetics have differing properties with respect to the relative stimulation of the intrinsic receptors.

Dobutamine. Dobutamine is a synthetic adrenergic agent with primarily beta-agonist effects, which more strongly stimulate the β₁-receptors than the β₂-receptors. It also exerts milder alpha-agonist effects. The net effect is for strong increases in cardiac contractility with minimal effects on heart rate and blood pressure. Dobutamine is primarily used in the setting of acute fulminating congestive heart failure, low-output cardiac failure, and cardiogenic shock. Dobutamine therapy is generally initiated at the low end of the dose range (Table 1), 2.5 μg/kg/min in

<table>
<thead>
<tr>
<th>Drug</th>
<th>Formulation</th>
<th>Dose (dog)</th>
<th>Dose (cat)</th>
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</thead>
<tbody>
<tr>
<td>Amrinone</td>
<td>Inocar, 5 mg/mL</td>
<td>1–3 mg/kg slow intravenous bolus; then, 10–100 μg/kg/min continuous rate infusion</td>
<td>No information</td>
</tr>
<tr>
<td>(Inocar)</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Digoxin</td>
<td>0.125 mg, 0.25-mg tablets:</td>
<td>0.01 mg/kg orally divided every 12 hours</td>
<td>¼ of 0.125-mg (or 0.0313 mg) tablet orally every 24–48 hours</td>
</tr>
<tr>
<td>(Lanoxin, Cardoxin)</td>
<td>0.05 mg/mL, 0.15-mg/mL elixir; 0.25 mg/mL for injection</td>
<td>0.0085 mg/kg orally divided every 12 hours</td>
<td></td>
</tr>
<tr>
<td>Dobutamine</td>
<td>250 mg per 20-mL bottle</td>
<td>2.5–20 μg/kg/min</td>
<td>1–5 μg/kg/min</td>
</tr>
<tr>
<td>(Dobutrex)</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Dopamine</td>
<td>40 mg per 5-mL vial</td>
<td>2.5–15 μg/kg/min</td>
<td>2–10 μg/kg/min</td>
</tr>
<tr>
<td>(Intropin)</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Epinephrine</td>
<td>1:1000 ratio, 1 mg/mL</td>
<td>0.2 mg/kg intravenously, 0.4 mg/kg intratracheally</td>
<td>Same</td>
</tr>
<tr>
<td>Isoproterenol</td>
<td>0.2 mg/mL for injection</td>
<td>0.01–2 μg/kg/min</td>
<td>Same</td>
</tr>
<tr>
<td>(Isuprel)</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Milrinone</td>
<td>Not available</td>
<td>50 μg/kg slow intravenous bolus; then, 0.375 μg/kg/min</td>
<td>No information</td>
</tr>
</tbody>
</table>

Table 1. DOSAGES FOR INOTROPIC AGENTS
the dog, with increases made by 2.5-μg/kg/min increments every 15 minutes if necessary based on the clinical response. In cats, a starting dose of 1 to 2 μg/kg/min is used, with 1-μg/kg/min incremental increases; dobutamine dosages greater than 5 μg/kg/min are likely to be associated with central nervous system (CNS) side effects (facial twitching, seizures). While dobutamine is administered, an electrocardiogram (ECG) (ideally a continuous telemetric monitor) is monitored for excessive tachycardia and arrhythmias. Alternatively, the patient can be auscultated, with periodic ECGs obtained if the auscultation is abnormal. The patient is monitored clinically for an improvement in the mucous membrane color and ease of breathing. If a pulmonary artery catheter is in place, one can monitor for a decrease in pulmonary artery pressure and pulmonary capillary wedge pressure. An increase in cardiac output is expected with successful therapy. Dobutamine is relatively contraindi-
cated in patients with atrial fibrillation because of the risk of increased AV conduction, resulting in a faster ventricular response to the fibrilla-
tion. Clinical need may override this contraindication in certain patients. The adverse side effects that may be seen with dobutamine infusion are an increase in heart rate and the development of arrhythmias. Because the half-life of dobutamine is short (minutes), this can be addressed by either decreasing the infusion rate or, in some cases, cessation of therapy. In feline patients, the CNS side effects of facial twitching or grand mal seizures limit dosing. Dobutamine has been studied extensively in nor-
mal dogs and found to be an effective inotropic drug. The remaining information about its use in clinical cases is largely anecdotal or extrapo-
lated from information in human cardiac patients.

**Dopamine.** Dopamine is the immediate precursor to norepineph-
rine. Effects of the drug are related to dose. At lower doses (1-3 μg/kg/
min), dopaminergic effects predominate, resulting in renal and splanchnic vasodilation and theoretically leading to increased renal blood flow and urine output. Traditionally, dopamine has been used as a primary agent for the treatment of oliguric and anuric renal failure. Recently, however, the efficacy of the drug for this use has been questioned, because some studies in people have failed to document a repeatable measurable effect. Similarly, successful use in dogs is based mainly on anecdotal information. At intermediate doses (5-8 μg/kg/min), dopa-
mine is a weak beta-agonist and a weak alpha-agonist. The net results are positive inotropic and chronotropic effects with mild vasoconstric-
tion. At high doses (8-20 μg/kg/min), alpha-agonist effects predominate with resulting strong peripheral vasoconstriction. The main indication for high-dose therapy is for cardiopulmonary resuscitation. At this dose, decreases in renal blood flow and increases in systemic vascular resist-
tance and pulmonary capillary wedge pressure are expected. For this reason, dopamine is rarely indicated for the treatment of congestive heart failure except at lower doses as an adjunctive therapy to dobutam-
ine. Patients receiving dopamine should have their heart rate and rhythm, blood pressure, and urine output monitored. The main potential side effects are hypertension, tachycardia, or the development of ar-
rhythms. Dopamine use is contraindicated in patients with elevated blood pressure. Overall, dopamine is well tolerated.

**Isoproterenol.** Isoproterenol is a pure-adrenergic stimulant. It has potent positive inotropic and chronotropic effects and may be associated with decreases in blood pressure mediated by $\beta_2$ stimulation. The effects of isoproterenol are increases in the sinus rate and in the rate of subsidiary pacemakers (AV junctional, ventricular) as well as increased AV conduction. The main indication for isoproterenol is for heart rate support in patients with third-degree heart block or sick sinus node syndrome while they are awaiting pacemaker implantation or when pacing therapy is not available. The drug does not reliably raise the heart rate significantly in these patients, however. Isoproterenol may be helpful when excessive bradycardia that is unresponsive to atropine develops under general anesthesia. Isoproterenol may be administered to overcome an accidental beta-blocker overdose. The side effects of isoproterenol include the development of arrhythmias, tachycardia, and hypotension.

**Epinephrine.** Epinephrine exerts mixed beta-stimulant effects and, at high doses, alpha-agonist actions. Epinephrine is mainly indicated for cardiac arrest. The beta-stimulant effects provide strong inotropic and chronotropic stimulation, and the alpha-stimulant effects provide blood pressure support. The use of epinephrine for cardiopulmonary resuscitation is addressed in a separate article in this issue.

**Phosphodiesterase Inhibitors**

Phosphodiesterase inhibitors (PDE-Is) are not currently in widespread use in the critical care setting in veterinary medicine. A brief discussion of this class of drugs is justified, however, because interest in these agents may be renewed with the advent of widespread beta-blockade use in cardiac patients. When patients chronically medicated with a beta-blocker present in congestive heart failure, they are likely to be refractory to standard doses of catecholamines. It has been suggested that it may be safer to consider the use of a PDE-I as an inotropic agent instead of risking the side effects that may develop when using the high dosages of adrenergic agents necessary to override the existing beta-blockade.

PDE-Is inhibit the breakdown of cAMP, causing protein kinase activation with resultant phosphorylation of intracellular proteins such as phospholamban. As a result of phosphorylation, calcium uptake by the sarcoplasmic reticulum in diastole is greater; more calcium is available during systole, resulting in stronger cardiac contraction. Peripherally, the result is vasodilation. These effects occur independently of the cellular beta-receptor. Dogs are responsive to the positive inotropic effects of amrinone and milrinone. Milrinone is licensed for intravenous use in people. An oral form of the drug was studied extensively by veterinary cardiologists in the 1980s and seemed to be an effective inotropic agent useful in the treatment of dogs with congestive heart
failure. When PDE-I fell out of favor in the treatment of people, the company researching the oral form of milrinone lost interest in gaining approval for the drug's use in the veterinary sector. The main side effect reported with these drugs in dogs is the development of arrhythmias. Amrinone is a less active PDE-I than milrinone. It is available only as an intravenous agent.

**Digitalis Glycosides**

Digoxin is the only agent in this class that is widely used to treat clinical patients. Digoxin exerts inotropic effects via the blockade of Na⁺-K⁺ adenosine triphosphatase in the cell membrane, resulting in intracellular Na⁺ accumulation. With more intracellular Na⁺ available for Ca⁺⁺ exchange, there is a net entry of calcium, resulting in an increase in the contractile strength. In addition, digoxin exerts antiarrhythmic effects by decreasing AV conduction, allowing slowing of some supraventricular arrhythmias, including atrial fibrillation. Digoxin also has the effect of restoring balance of the autonomic nervous system via its vagomimetic effects. In these ways, digoxin exerts beneficial effects in the patient with chronic cardiac disease. Digoxin is rarely used in the acute care setting because of its slower onset of action and increased potential for intoxication with intravenous dosing (see Table 1). Patients receiving digoxin should be closely monitored for toxicity. Blood levels should be monitored 8 to 10 hours after the dose is administered within 5 to 7 days after beginning therapy or whenever signs of toxicity develop. Although digoxin levels of 0.8 to 2.5 ng/mL are reported to be normal, dosages above 2 ng/mL are frequently associated with side effects. The “ideal” digoxin level in human patients is still being actively debated in human cardiology. The author considers a level of 1 to 1.8 ng/mL ideal in most canine patients. Feline patients do not routinely tolerate digoxin levels in this range. Individual patients may develop toxicity at conservative doses after administration of only a few doses. Successful use of digoxin depends on careful dose calculation, taking into account lean body weight and level of renal function, adequate client education, and vigilance on the part of the owner and clinician. Gastrointestinal signs of toxicity predominate with minor intoxication (inappetence, vomiting, diarrhea), and lethargy is usually noted. If these signs go undetected or are ignored and digoxin continues to be administered, serious arrhythmias may result. Hypokalemia puts patients at higher risk for manifesting toxicity. Digoxin antibodies (Digibind) may be administered in cases of overwhelming toxicity (usually when a pet consumes a bottle of medication inadvertently). In most other scenarios, digoxin toxicity can be managed with discontinuation of the drug and supportive care administered when necessary. Although intravenous digoxin use is generally avoided because of the increased risk for toxicity, it is occasionally indicated (see the section on antiarrhythmic therapy).
Vasodilator Therapy

Nitrates

The nitrate vasodilators effect their action by providing an exogenous source of nitric oxide to vascular cells, which results in the stimulation of guanyl cyclase and the production of cyclic guanosine monophosphate (cGMP). Increases in cGMP lead to a decrease in intracellular calcium, resulting in venodilation, with lesser arterial dilating effects. The development of tolerance is a common property of all the drugs in this class. Although the mechanism for the development of tolerance is not entirely understood, it is thought that depletion of sulfhydryl groups, which are oxidized with chronic exposure to the nitrates, plays an important role. Various dosing schemes providing nitrate-free intervals have been described to avoid the development of tolerance. Nitrates are useful during the acute presentation for congestive heart failure with pulmonary edema.

Sodium Nitroprusside (Nitride). Sodium nitroprusside is a potent balanced arterial dilator and venodilator with a rapid onset and short duration of action. It is administered as a continuous rate infusion (using an infusion pump), which allows rapid assessment of the response to the drug with the ability to reduce the dose or stop the drug if problems develop with its use. The main indication is for the patient presenting with an acute hypertensive crisis or acute fulminant pulmonary edema. Dosing is generally started at 1 μg/kg/min and is increased by 1-μg/kg/min increments every 5 minutes (Table 2). Ideally, drug effects are monitored with direct arterial blood pressure readings and continuous

<table>
<thead>
<tr>
<th>Drug</th>
<th>Dosage (dog)</th>
<th>Dosage (cat)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Benazapril (Lotensin)</td>
<td>0.25-0.5 mg/kg orally every 12-24 hours</td>
<td>Same</td>
</tr>
<tr>
<td>Captopril (Capoten)</td>
<td>0.25 mg/kg orally every 8 hours</td>
<td>Same</td>
</tr>
<tr>
<td>Enalapril (Enaocard, Vasotec)</td>
<td>0.25-1.0 mg/kg orally every 12-24 hours</td>
<td>0.25-0.5 mg/kg orally every 12-24 hours</td>
</tr>
<tr>
<td>Hydralazine (Apresoline)</td>
<td>0.5-2.0 mg/kg orally every 8-12 hours</td>
<td>2.5 mg/kg orally every 12 hours</td>
</tr>
<tr>
<td>Isosorbide dinitrate (Isordil)</td>
<td>0.25-1.0 mg/kg orally every 8-12 hours</td>
<td>0.25-0.5 mg/kg orally every 12 hours</td>
</tr>
<tr>
<td>Lisinopril (Zestril, Prinivil)</td>
<td>0.25-1.0 mg/kg orally every 12-24 hours</td>
<td>0.25-0.5 mg/kg orally every 24 hours</td>
</tr>
<tr>
<td>Nitroglycerine (Nitrol, Nitrobid)</td>
<td>0.25-1-inch strip cutaneously every 6-8 hours</td>
<td>0.125-0.25-inch strip cutaneously every 6-8 hours</td>
</tr>
<tr>
<td>Prazosin (Minipress)</td>
<td>1 mg per 15 kg (only suitable for larger dogs)</td>
<td>No information</td>
</tr>
<tr>
<td>Sodium nitroprusside (Niprid, Nitropress)</td>
<td>0.5-10 μg/kg/min continuous rate infusion</td>
<td>0.5-2.0 μg/kg/min continuous rate infusion</td>
</tr>
</tbody>
</table>
telemetric ECG recording. Noninvasive blood pressure readings using a dinamap (Johnson & Johnson) or Doppler systems are frequently substituted. The goal is maintenance of mean arterial blood pressure above 70 mm Hg or systolic blood pressure above 90 mm Hg. Mucous membrane color and relief of dyspnea are judged to help assess clinical response. Expected changes in hemodynamic parameters are increases in cardiac output with decreases in mean arterial pressure, systemic vascular resistance, and pulmonary capillary wedge pressure. The main side effects seen with sodium nitroprusside use are systemic hypotension and reflex tachycardia. Sodium nitroprusside use is contraindicated in patients who present with systemic hypotension; however, it may be combined with agents with a pressor effect (dopamine, dobutamine) to offset the hypotensive effect where necessary. Although sodium nitroprusside (and all arterial dilators) have been considered to be contraindicated in patients with dynamic left ventricular outflow tract obstruction (seen in cats with hypertrophic obstructive cardiomyopathy), this contraindication is considered relative: in select patients, the potent effect in relief of pulmonary edema may offset any relative contraindications. Because cyanide and thiocyanate toxicity may develop with long-term use, patients receiving the drug should be weaned off it as soon as is feasible. Cats are more sensitive to sodium nitroprusside, and lower doses are recommended (see Table 2).

**Nitroglycerine.** Nitroglycerine is available in a 2% ointment or is alternatively provided as a continuous delivery cutaneous patch system. Oral forms of nitroglycerine are not typically used in veterinary patients. Nitroglycerine is a mild venodilator used mainly in the patient presenting with acute congestive heart failure, where drug application is simple but the effects are questionable. Most agree that because it is safe and easy to use, there is little harm done in using it. Nitroglycerine absorption is theoretically best in a thin-skinned and hairless area of the body; usually, the inner pinna or groin area is selected. Dosage schemes are variable, but the drug is typically only used for 1 or 2 days or until the patient can be started on more effective oral vasodilator medications. The only noted side effects are local skin irritation with topical application. Critical care staff and owners must be careful to avoid skin contact, because the agent can be transferred and absorbed.

**Oral Nitrate Agents (Isosorbide Mononitrate, Isosorbide Dinitrate).** These orally active venodilators are often paired with an arterial dilator (hydralazine) to create balanced vasodilation in patients who are intolerant of ACE-I drugs (see Table 2). Oral nitrates have minimal use in the veterinary critical care setting.

**Angiotensin-Converting Enzyme Inhibitors**

This class of drugs has been shown to be the most effective in addressing the neurohumoral alterations with chronic congestive heart failure. As the name implies, ACE-Is antagonize angiotensin-converting enzyme, which catalyzes the conversion of angiotensin I to its active
form, angiotensin II. Angiotensin II has strong vasoconstrictive effects, which, in addition to maintaining blood pressure, increase afterload on the heart. Angiotensin II also stimulates aldosterone release from the adrenal glands, resulting in sodium retention, volume expansion, and increased preload. ACE-I drugs are expected to lead to decreases in preload and afterload. Angiotensin II also modulates the cellular effects of vascular remodeling and myocardial hypertrophy. Inhibition of this effect may be useful in cardiac disease associated with myocardial hypertrophy. Although ACE-Is have less of a place in the acute care setting, many patients presented to the emergency room have a history of ACE-I therapy. Patients who are presented on ACE-Is should have their blood pressure and renal function evaluated. In the patient with acute chronic heart failure, ACE-I therapy is frequently withheld until the patient negotiates the initial vigorous treatment period and until volume and electrolyte balance is restored in the “recovery period.” Dosing information for the commonly used ACE-Is is provided in Table 2. The main side effect seen with ACE-Is in veterinary patients is decompensation of renal function related to disruption of renal vascular autoregulation.

Other Vasodilators

**Hydralazine.** This potent direct-acting arterial dilator acts rapidly to reduce afterload after a single oral dose. Its use may be indicated in the patient with acute pulmonary edema with refractory congestive heart failure and for patients intolerant of ACE-I therapy (see Table 2). Patients receiving hydralazine should be monitored for hypotension and associated reflex tachycardia, which may be counterproductive in patients with cardiac disease.

**Prazosin.** Prazosin is an alpha-antagonist, and its use results in peripheral vasodilation. This drug is occasionally used to treat systemic hypertension or for acute congestive heart failure (see Table 2). Patients receiving prazosin should be monitored for hypotension, which may be particularly profound after the first dose. Compensatory tachycardia may also be noted secondary to the decreases in blood pressure.

ANTIARRHYTHMIC AGENTS

Antiarrhythmic therapy has become a controversial topic in human cardiology after the results of the Cardiac Arrhythmia Suppression Trial in the late 1980s. In this study, patients surviving myocardial infarction who had ventricular arrhythmias were randomized to receive various class IC antiarrhythmic agents. The trial was prematurely terminated when the patients in the treatment groups had mortality significantly higher than the patients receiving a placebo. These patients suffered increased mortality despite suppression of ventricular ectopy. These results led to a heightened awareness of the proarrhythmic potential of many antiarrhythmic drugs (particularly class I antiarrhythmic agents).
and to a much more conservative approach to antiarrhythmic therapy. The issues are even more clouded in veterinary medicine, where the validity of data extrapolated to veterinary patients is questioned and economic limitations may preclude careful screening and re-evaluation of veterinary patients by continuous ambulatory ECG monitoring (Holter monitoring) to screen for proarrhythmic effects. Although it is unclear how much of this information can or should be extrapolated to animals, the principles of the approach to the arrhythmic patient should be similar. Two important points should be considered. First, double-blind clinical trials on large patient populations are the only way to determine the indications or contraindications for therapeutic agents in the target population. Second, it seems unwise to be overly aggressive in attempting to prophylactically suppress arrhythmias in asymptomatic patients. Effort should be concentrated on careful assessment of the effects of antiarrhythmic medications in those patients with aggressive arrhythmias or in patients displaying symptoms as a result of an arrhythmia.

The successful treatment of a patient for arrhythmia starts with accurate identification of the arrhythmia and with investigation for any possible underlying causes. The hemodynamic significance of the arrhythmia must be considered when deciding whether intervention is indicated and which agent or combination of agents should be selected. Successful identification of the arrhythmia begins with a diagnostic-quality ECG. This requires functioning equipment and careful setup. The patient should be positioned on an insulated surface (towel or cage paper) in right lateral recumbency, and a 10-lead ECG should be obtained if possible. Although the chest leads are not absolutely essential, they may provide clearer waveforms than the limb leads in some patients. The limb leads may be positioned more distally on the legs (closer to where the restrainer is holding the limbs) than standard placement so as to avoid interference from respiration, vocalization, or tremors. The chest leads can be held away from the chest wall to minimize respiratory fluctuations. The amplitude setting is adjusted to optimize the complex size in the individual patient. Using these simple steps, a diagnostic-quality ECG can usually be obtained. A full discussion of arrhythmia recognition is beyond the scope of this article, but a few general points are important in the critical care setting. When presented with a patient with a tachyarrhythmia, it is helpful to break down the diagnosis into wide-complex (in the dog, QRS >0.07 seconds; in the cat, QRS >0.05 seconds) or narrow-complex tachycardia. For all practical purposes, a narrow-complex tachycardia is always a supraventricular rhythm. A wide-complex tachycardia is most likely to be a ventricular tachycardia (VT) but can occasionally be the result of a supraventricular tachycardia (SVT) in a patient with preexisting or rate-related aberrant ventricular conduction. In cats, where preexisting conduction blocks (left anterior fascicular block, right bundle branch block) are more common, the latter explanation should be more strongly considered. Vagal maneuvers may be helpful in slowing down or terminating a SVT involving the AV
node, making definitive diagnosis possible in many cases of SVT. Unless you can be confident of the diagnosis of SVT with aberrancy, it is always safer to treat a wide-complex tachycardia as VT than to administer potentially hypotensive drugs used to treat SVT inappropriately to a patient with VT. Lidocaine administration resulting in resolution of the arrhythmia frequently helps to establish a diagnosis of VT in these patients, allowing more comfortable therapeutic decision making.

The following discussion of the antiarrhythmic agents uses the traditional Vaughan-Williams classification scheme, which is a simple way to organize the antiarrhythmic drugs. The discussion is restricted to the drugs commonly employed for therapy of arrhythmias in the veterinary critical care setting. Detailed pharmacotherapeutic effects of the antiarrhythmic medications are chronicled elsewhere. Drug dosages are listed in Table 3.

**Class I Agents**

Sodium-channel blocking drugs have varying effects on the action potential (AP) duration and repolarization time. These drugs are referred to as membrane stabilizers.

**Class IA Agents**

This subtype of drugs prolongs the AP duration and increases the refractory period.

**Procainamide.** Procainamide is effective against a wide range of ventricular and supraventricular arrhythmias and is frequently selected for the treatment of ventricular arrhythmias that are poorly responsive to lidocaine or for refractory supraventricular tachyarrhythmias. In cases where procainamide fails to terminate the tachycardia, it usually effects slowing of the rhythm, improving overall hemodynamic stability. Intravenous procainamide should be administered slowly, or it may lead to the development of systemic hypotension. Procainamide is occasionally selected for treatment of ventricular arrhythmias...
that are refractory to therapy with either lidocaine or procainamide when newer agents are not readily available. Quinidine is associated with gastrointestinal side effects (vomiting, diarrhea, anorexia). The drug is a mild negative inotrope and may cause systemic hypotension. Theoretically, proarrhythmic effects are more likely to result with quinidine than with most other antiarrhythmic drugs.

**Class IB Agents**

This subclass of class I drugs shortens the AP duration and increases the refractory period.

**Lidocaine.** Lidocaine is the antiarrhythmic agent in most widespread use in the veterinary critical care setting. Lidocaine is quite effective in controlling most ventricular arrhythmias but is generally ineffective in the treatment of supraventricular arrhythmias. It administered only by the intravenous route. The effectiveness of lidocaine is blunted in the presence of hypokalemia. The main side effect of lidocaine is CNS excitement, resulting in twitching, agitation, or tonic-clonic seizures. Nausea frequently precedes the onset of CNS side effects. Lethargy may occasionally be noted. The side effects are generally self-limiting and respond to decreases in the infusion rate of the drug; however, in some patients, the CNS effects may limit dosing significantly enough so that an alternative antiarrhythmic agent needs to be selected. Diazepam can be administered for seizure activity where required. Lidocaine can be administered in cats but must be used carefully and at lower dosages than in dogs (see Table 3). Cats are quite sensitive to CNS side effects, including seizures and excitation.

**Mexiletine.** The effects of mexiletine are similar to those of lidocaine, but this drug is used by the oral route for chronic control of ventricular arrhythmias. Lethargy or CNS side effects may be noted with its use. Gastrointestinal side effects have also been reported with this drug.10, 18

**Tocainide.** Tocainide is an oral analogue of lidocaine. Although this drug is effective, its side effects are somewhat undesirable. Gastrointestinal side effects have also been reported to be common. Neurologic, renal, and ocular side effects have also been reported.10, 18

**Class IC Agents**

This subtype of drugs is not in common use in veterinary patients.

**Class II Agents**

This category of drugs includes all the drugs with beta-blocking effects. The beta-blocker drugs have been used for many years for the therapy of hypertrophic cardiomyopathy and the control of ventricular and supraventricular tachyarrhythmias. They are also used in the treat-
# Table 3. DRUG DOSAGES FOR ANTIARRHYTHMIC AGENTS

<table>
<thead>
<tr>
<th>Drug</th>
<th>Preparation</th>
<th>Dose (dog)</th>
<th>Dose (cat)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Adenosine</td>
<td>Adenocard: 3 mg/mL for injection</td>
<td>1-3 mg/kg rapid intravenous bolus</td>
<td>No information</td>
</tr>
<tr>
<td>Amiodarone</td>
<td>Cordarone: 200-mg tablets</td>
<td>Loading: 10-20 mg/kg orally every 24 hours for 7-10 days</td>
<td>No information</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Maintenance: 3-15 mg/kg orally every 24 hours or 5 mg/kg orally every 48 hours*</td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td>Alternative: 10-15 mg/kg orally every 12 hours for 7 days; then, 5.0-7.5 mg/kg orally every 12 hours for 14 days; then, 7.5 mg/kg orally every 24 hours†</td>
<td></td>
</tr>
<tr>
<td>Atenolol</td>
<td>Tenormin: 25-, 50-, 100-mg tablets; 25 mg/mL oral suspension</td>
<td>0.25-1.0 mg/kg orally every 12-24 hours</td>
<td>6.25-12.5 mg orally every 12-24 hours</td>
</tr>
<tr>
<td>Atropine</td>
<td>0.1 mg/mL</td>
<td>0.02-0.04 mg/kg intravenously, intramuscularly, subcutaneously as needed</td>
<td>Same</td>
</tr>
<tr>
<td>Bretylium tosylate</td>
<td>Bretylol: 50 mg/mL for injection</td>
<td>2-10 mg/kg intravenously; 25-50 mg/kg for cardiopulmonary resuscitation*</td>
<td></td>
</tr>
<tr>
<td>Digoxin</td>
<td>0.125-, 0.25-mg tablets, 0.05-mg/mL, 0.15-mg/mL elixir; 0.25 mg/mL for injection</td>
<td>0.01-0.02 mg/kg orally divided two times daily for tablets; 0.0085 mg/kg orally divided two times daily for elixir; 0.0025 mg/kg for intravenous bolus; not to exceed 0.01-mg/kg total intravenous dose</td>
<td>0.25 of 0.125-mg tablet orally every 24-48 hours</td>
</tr>
<tr>
<td>Diltiazem</td>
<td>Cardiazem: 30-, 60-, 90-, 120-mg tablets</td>
<td>0.5-2.0 mg/kg orally three times daily</td>
<td>7.5-15.0 mg per cat orally every 8-12 hours</td>
</tr>
<tr>
<td></td>
<td>5 mg/mL for injection</td>
<td>0.1-0.2-mg/kg intravenous bolus</td>
<td>Same</td>
</tr>
<tr>
<td></td>
<td>Dilacor XR: 180-240-mg capsules</td>
<td></td>
<td>30-60-mg tablet (from inside capsule orally every 24 hours)</td>
</tr>
<tr>
<td>Esmolol</td>
<td>Brevibloc: 10 mg/mL, 250 mg/mL for injection</td>
<td>50-500-µg/kg slow intravenous bolus, can repeat every 5 minutes as 50-200-µg/kg/min continuous rate infusion</td>
<td>Same</td>
</tr>
<tr>
<td>Lidocaine</td>
<td>Xylocaine: 20 mg/mL for injection</td>
<td>2-mg/kg slow intravenous bolus, up to 8-mg/kg total dose: 50-200-µg/kg/min intravenous continuous rate infusion</td>
<td>0.25-0.5-mg/kg slow intravenous bolus, do not exceed total dose of 4 mg; 10-40 µg/kg/min continuous rate infusion</td>
</tr>
<tr>
<td>Drug</td>
<td>Dosage</td>
<td>Schedule</td>
<td>Notes</td>
</tr>
<tr>
<td>-----------------------</td>
<td>------------------------------------------------------------------------</td>
<td>-----------------------------------------------</td>
<td>------------------------------------------------</td>
</tr>
<tr>
<td>Metoprolol</td>
<td>Lopressor: 50-, 100-mg tablets</td>
<td>0.25–1.0 mg/kg orally every 12–24 hours</td>
<td>Same</td>
</tr>
<tr>
<td>Mexilitine</td>
<td>Mexitil: 150-, 200-, 250-mg tablets</td>
<td>4–8 mg/kg orally every 8 hours</td>
<td>No information</td>
</tr>
<tr>
<td>Procainamide</td>
<td>Pronestyl: 250-, 375-, 500-mg tablets</td>
<td>10–20 mg/kg orally every 6–8 hours</td>
<td>2–5 mg/kg orally every 8–12 hours</td>
</tr>
<tr>
<td></td>
<td>Pronestyl SR, Procan SR: 250-, 500-, 750, 1000-mg tablets</td>
<td>5–15-mg/kg slow intravenous bolus; 25–50 μg/kg/min intravenous continuous rate infusion</td>
<td>0.01–0.1 mg/kg intravenous slow bolus 2.5–10.0 mg total dose every 8–12 hours</td>
</tr>
<tr>
<td></td>
<td>100 mg/mL, 500 mg/mL for injection</td>
<td>0.2–1.0 mg/kg orally every 8 hours;</td>
<td>0.01–1.0 mg/kg slow intravenous bolus</td>
</tr>
<tr>
<td>Propranolol</td>
<td>Inderal: 10-, 20-, 40-, 80-mg tablets</td>
<td>1 mg/mL for injection</td>
<td>No information</td>
</tr>
<tr>
<td></td>
<td></td>
<td>0.01–0.1 mg/kg slow intravenous bolus</td>
<td>No information</td>
</tr>
<tr>
<td>Quinidine</td>
<td>324-mg tablets; 80 mg/mL for injection</td>
<td>5–20 mg/kg orally every 6–8 hours</td>
<td>No information</td>
</tr>
<tr>
<td>Gluconate</td>
<td></td>
<td>5–10 mg/kg slow intravenous bolus</td>
<td>No information</td>
</tr>
<tr>
<td>Quinidine sulfate</td>
<td>100-, 200-, 300-mg tablets; 200 mg/mL for injection</td>
<td>5–20 mg/kg orally every 6–8 hours</td>
<td>No information</td>
</tr>
<tr>
<td></td>
<td></td>
<td>5–10 mg/kg slow intravenous bolus</td>
<td>No information</td>
</tr>
<tr>
<td>Sotalol</td>
<td>Betapace: 80-, 160-, 240-mg tablets</td>
<td>0.5–2.0 mg/kg orally every 12 hours*</td>
<td>3/4 of 80-mg tablet orally every 12 hours</td>
</tr>
<tr>
<td>Tocainide</td>
<td>Tonocard: 400-, 600-mg tablets</td>
<td>5–20 mg/kg orally every 12 hours*</td>
<td>No information</td>
</tr>
<tr>
<td>Verapamil</td>
<td>Calan, Isotin: 80–120-mg tablets; 5 mg/mL for injection</td>
<td>5–20 mg/kg orally every 12 hours*</td>
<td>1–5 mg/kg orally every 8 hours</td>
</tr>
<tr>
<td></td>
<td></td>
<td>0.05–0.1 mg/kg slow intravenous bolus</td>
<td>0.05–0.1 mg/kg slow intravenous bolus</td>
</tr>
</tbody>
</table>


ment of hypertension. In the last few years, beta-blocker therapy has been shown to be effective in attenuating the negative responses, including beta-receptor downregulation, which is related to the chronic sympathetic stimulation associated with heart failure. There is a myriad of research underway to determine which beta-blocker agents are most effective with which types and stages of diseases. It is likely that what is true in human patients can be applied to our veterinary patients; however, hopefully, specific veterinary studies are forthcoming. Beta-blocker therapy promises to be an important part of cardiac therapy in the years to come.

Beta-blockers are subcategorized to receptor specificity (cardioselective agents predominantly provide $\beta_1$ stimulation, whereas nonselective agents generate mixed $\beta_1$ and $\beta_2$ effects) and as to whether they possess intrinsic sympathomimetic activity (ISA). Beta-blockers with ISA have the potential advantage of maintaining heart rate and contractility, which is theoretically beneficial in some patients. The beta-blocker agents in common use in veterinary patients are discussed here. None of these agents possess ISA.

Propranolol

Propranolol is the prototype beta-blocker used in veterinary medicine, although the advent of agents with more cardioselectivity and longer duration of action has led to less common prescription of propranolol. Propranolol is still occasionally used as adjunctive therapy to a class I antiarrhythmic drug in the treatment of refractory ventricular arrhythmias or to decrease AV conduction in SVT. Side effects include lethargy, hypotension, bronchospasm, excessive bradycardia, and the development of AV block. 16

Metoprolol

Metoprolol (Lopressor) is a cardioselective beta-blocker frequently prescribed in canine patients for the therapy of supraventricular or ventricular arrhythmias and to slow the ventricular response to atrial fibrillation. Metoprolol has a longer duration of action than propranolol. Side effects include depression, hypotension, excessive bradycardia, and the development of AV block. 16 Bronchospasm is unlikely to occur with cardioselective beta blockers.

Atenolol

Atenolol (Tenormin) is a cardioselective beta-blocker. It has a longer duration of action than propranolol. Atenolol is frequently used in cats for the treatment of hypertrophic cardiomyopathy and for supraventricular and ventricular arrhythmias. In dogs, atenolol may be selected as a substitute for metoprolol. Side effects are similar to those noted for metoprolol.
Esmolol

Esmolol is a cardioselective beta-blocker administered intravenously. Esmolol has a short duration of action, allowing the effects to be observed and the infusion stopped if adverse side effects are noted. Esmolol is the ideal beta-blocker agent for administration in the acute setting.10, 18

Sotalol

Sotalol is an antiarrhythmic drug with mixed class II and class III effects. At lower dosages, its nonselective beta-blocking effects predominate (for a complete discussion, see the section on class III agents).

Carvedilol

Although this beta-blocker drug is not in widespread use in veterinary patients at this time, it possesses some unique properties that make it a promising drug for cardiac patients. Carvedilol is a nonselective beta-blocker with vasodilator properties mediated by blockade of alpha-receptors. Carvedilol also has antioxidant effects, which are unique to this drug.16 The use of this drug in patients with congestive heart failure is being actively investigated. Carvedilol use may be implemented in veterinary patients as well.

Class III Agents

Pure class III agents, none of which are commercially available, act to block the outward potassium channels, resulting in markedly delayed repolarization.1 Because of the delayed repolarization, there is an increased susceptibility to QT prolongation and associated proarrhythmia. Theoretically, combination with a class I drug is likely to promote proarhythmia. All the agents listed below are mixed class III agents, which have effects in addition to the class III effect. The favorable effects of these drugs may be exerted from actions other than the class III effects, because, to date, experimental drugs with pure class III actions have shown disappointing results.

Sotalol (Betapace)

Sotalol is a nonselective beta-blocking drug with class III effects.16 Sotalol is effective in the treatment of ventricular and supraventricular arrhythmias. The drug is somewhat expensive, so it is mainly selected as a first antiarrhythmic agent in patients with aggressive arrhythmias that are perceived to be potentially lethal. Because sotalol exerts actions within hours of an oral dose, its use should be considered to terminate tachyarrhythmias in the acute setting. The main side effects seen with
sotalol are lethargy and bradycardia. Proarrhythmia related to QT prolongation is possible. Like all the beta-blockers, sotalol has negative inotropic and chronotropic effects, which can decompensate patients with depressed systolic function and narrowly compensated congestive heart failure. These types of patients should be carefully monitored for adverse effects with drug administration.

Amiodarone (Cordarone)

Amiodarone is one of the most frequently prescribed antiarrhythmic agents in the human cardiac intensive care unit. This drug is unique in that it possesses qualities of all four antiarrhythmic groups. It has powerful sodium-channel blocking effects with milder beta-blocker and calcium-channel blocking effects. Amiodarone has been shown to decrease mortality in many subsets of human cardiac patients with arrhythmias. The main drawback to amiodarone is the serious side-effect profile. With chronic use in people, amiodarone can lead to CNS side effects, gastrointestinal side effects, pulmonary fibrosis, corneal microdeposit formation, and interference with thyroid function. In addition, serious bradyarrhythmias may develop. The development of proarrhythmia in human patients seems to be uncommon with this drug. Amiodarone has an extremely long half-life (25–110 days), so various loading schemes have been developed for higher risk patients. Unfortunately, if side effects necessitate cessation of the drug, it can take months to years for the drug to completely leave the body. Amiodarone has been used in a small group of veterinary patients, mainly Doberman Pinschers with dilated cardiomyopathy and ventricular arrhythmias. A few different dosing schemes have been described for canine patients (see Table 3). Hepatotoxicity and neutropenia have been described in dogs after amiodarone therapy.

Bretylium

Bretylium is occasionally used in veterinary patients mainly in the setting of ventricular fibrillation and cardiopulmonary resuscitation. Given intravenously, bretylium results in an initial catecholamine release (norepinephrine release), followed by inhibition of any further norepinephrine release from the terminal sympathetic neurons (“chemical sympathectomy”). Bretylium may be effective where other agents have failed. In human patients, amiodarone has almost entirely replaced the clinical use of bretylium in this setting. In patients who are conscious, bretylium may be associated with nausea and vomiting.

Class IV Agents

The class IV agents are the calcium-entry blockers. Of the two types of calcium-entry blockers, it is the nondihydropyridine agents that have
potent sinoatrial and AV nodal effects and thus usefulness as antiarrhythmic agents. The dihydropyridine agents have greater effects on the peripheral and coronary vasculature and are used more often in the treatment of systemic hypertension or coronary artery disease. The nondihydropyridine agents are mainly used to slow the ventricular response rate in atrial fibrillation and to terminate SVT. They also slow the sinus rate slightly. These agents are ineffective in treating ventricular arrhythmias and are strictly contraindicated in this subset of patients because of their hypotensive effects, which are caused by calcium entry blockage in peripheral vasculature. Combination therapy with beta-blocker and calcium-channel blockers should be undertaken cautiously, because the cardiodepressant effects are additive.

This discussion is limited to the first-generation calcium-entry blockers verapamil and diltiazem, because they are the only agents in the class with significant antiarrhythmic effects.

**Verapamil**

Verapamil is most frequently used intravenously for the cessation of rapid SVT. With intravenous use, excessive AV block or systemic hypotension may result. Caution must be used in the administration of intravenous calcium-channel blockers to patients with depressed systolic function, because the drug has modest negative inotropic effects. Adverse effects related to verapamil may be counteracted by the infusion of intravenous calcium or intravenous fluids. Verapamil is occasionally used orally in cats with hypertrophic cardiomyopathy or to slow a fast ventricular response to atrial fibrillation in the dog or cat. Verapamil may decrease digoxin excretion, so it should be used cautiously, if at all, in combination with digoxin.

**Diltiazem**

Diltiazem is most commonly used in the treatment of cats for hypertrophic cardiomyopathy or in dogs or cats for slowing of the ventricular response rate to atrial fibrillation. Diltiazem does not seem to significantly interfere with digoxin excretion, so it is preferable to verapamil when used in combination with digoxin for the treatment of atrial fibrillation. The main side effects with diltiazem are anorexia, lethargy, and vomiting. Although intravenous diltiazem is less frequently used, it may also result in systemic hypotension or AV blockade. Diltiazem has less negative inotropic effects than verapamil.

**Digitalis Glycosides**

These drugs have no place in the Vaughn-Williams classification scheme despite their antiarrhythmic effects. For discussion of digoxin actions, refer to the section on digitalis glycosides in this article. The
main indication for digoxin as an antiarrhythmic agent in the critical care setting is in the patient with dilated cardiomyopathy presenting with atrial fibrillation with rapid ventricular response. In this setting, the hypotensive and negative inotropic effects of a calcium-entry blocker or beta-blocker are better avoided, and a dose or two of intravenous digoxin may be administered to slow the ventricular response rate and to help stabilize the critical patient. The patient should be closely monitored for digoxin toxicity, and a switch to oral therapy should be made as soon as is feasible.

Adenosine

Adenosine (Adenocard) is frequently subclassified with the calcium-entry blocking drugs, because the indications and effects are most similar to these drugs. Adenosine is a purinergic agent with a short duration of action, which, when administered intravenously, is frequently effective in terminating supraventricular arrhythmias involving the AV node and atrial automatic arrhythmias. This drug has not been used extensively in veterinary medicine, in part, because of its expense.

Atropine

Atropine is a parasympatholytic drug administered to combat bradyarrhythmias. Atropine exerts its main effects by abolishing parasympathetic influences on the sinus and AV nodes. It is effective in patients who have vagally mediated bradyarrhythmias, may be partially effective in patients with sinus node disease, and is generally ineffective in patients with intrinsic disease of the AV node or bundle of His.

Other Considerations in Arrhythmia Control

An important consideration in arrhythmia control is the stabilization of the electrolyte and acid–base balance. Hypokalemia and hypomagnesemia play an important role in the genesis of arrhythmias. In addition, the effects of many of the antiarrhythmic agents listed previously are significantly blunted by electrolyte depletion. Effective oxygen delivery and minimization of stress also help to maximize effective arrhythmia control.

Nonpharmacologic Methods of Arrhythmia Control

It is worthwhile to mention that in light of many of the proarrhythmic effects of antiarrhythmic agents, there has been an increase in the use of nonpharmacologic treatments for arrhythmia, including cardio-
version and pathway ablation in patients eligible for these interventions. There has also been increased use of implantable defibrillators in human patients who have survived aborted cardiac death. The main reason that cardioversion is not used much in veterinary patients outside the setting of cardiac resuscitation is the need for general anesthesia for patient cooperation. With the development of relatively "cardiosafe" anesthetics such as etomidate, defibrillation may be considered as an earlier step in arrhythmia management. Implantation of intracardiac defibrillators (ICDs) is feasible in dogs if the indication for the device can be clearly determined. ICD implantation may be indicated in breeds at risk for sudden cardiac death. Finally, electrophysiologic mapping of accessory pathways responsible for supraventricular tachyarrhythmias has allowed ablation of these pathways, permitting effective therapy in patients with a refractory arrhythmia or the ability to discontinue pharmacologic therapy when desired. Referral to a veterinary cardiologist who has access to an electrophysiology laboratory may be considered for selected cases.

References


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