

lethargy, ataxia, dyspnea, syncope, or death. The goal of treatment is to improve the patient's hemodynamic condition either by terminating the arrhythmia or increasing cardiac output by slowing the heart rate and increasing diastolic filling time.⁴ Methods of treating supraventricular tachycardia include vagal maneuvers to slow the heart rate, administration of pharmacologic agents, or electrical defibrillation. Medications used to treat atrial fibrillation include digoxin, class II drugs (eg, β -adrenergic receptor antagonists), class III drugs (eg, sotalol and amiodarone), and class IV drugs (calcium channel blockers), all of which slow conduction of impulses through the atrioventricular node, thereby reducing the ventricular rate. In 1 report,³ hypothermia-induced atrial fibrillation in dogs was successfully converted to sinus rhythm via administration of quinidine, a class Ia drug that blocks sodium channels and prolongs the duration of the action potential. Conversion of atrial fibrillation to sinus rhythm in dogs is best accomplished with class III drugs or via electrical methods and is usually reserved for dogs with apparently normal-sized atria.

In the dog of this report, hypothermia likely played an important role in the development of atrial fibrillation. However, underlying cardiac disease, such as dilated cardiomyopathy or atrial enlargement secondary to mitral or tricuspid valve regurgitation, and subsequent volume overload cannot be completely ruled out as the source of the supraventricular tachyarrhythmia. Additionally, the dog's neurologic disease may have been a contributing fac-

tor. Dogs undergoing decompressive surgery of the cervical portion of the spinal cord have an increased risk of developing dysrhythmia, ventricular premature contractions, and bradycardia.⁵ Alternatively, the dog's neurologic disease may have had a direct impact on the myocardium. Myocardial necrosis may have been the inciting cause of the supraventricular tachyarrhythmia.⁶ Thus, the potential contribution of the underlying neurologic disease cannot be discounted as a possible cause for the dog's arrhythmia. Follow-up echocardiography and Holter monitor assessments would be indicated to determine whether the supraventricular and ventricular tachycardia persist in the dog of this report.

References

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Correction: Dietary fats and the skin and coat of dogs

In the report “Dietary fats and the skin and coat of dogs” (*J Am Vet Med Assoc* 2007;230; 1641–1644), the conversion for the dosage in the fourth line from the end of the section “Dietary Supplementation of n-3 Fatty Acids for Skin Disorders” on page 1642 was incorrect. The correct conversion for supplementation of a diet with marine fish oil should read as follows: 1 g of fish oil/4.54 kg (1 g of fish oil/10 lb) of body weight.