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Cardioversion of Supraventricular Tachycardia Using Lidocaine in Five Dogs

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Sustained supraventricular tachycardia (SVT) may lead to life-threatening complications such as tachycardia-induced myocardial failure. We report the use of intravenous lidocaine[a] in 5 dogs with SVT. Two dogs had evidence of an accessory conduction pathway, 2 were suspected of having an accessory pathway, and the mechanism of SVT was unknown in the remaining dog, which subsequently developed dilated cardiomyopathy 2 years later. In all cases there was rapid conversion to normal sinus rhythm, which was then maintained with oral mexilitene[b] (4 dogs) or mexilitene combined with propranolol[c] (1 dog).

Key words: Cardiomyopathy; Cardioversion; Congestive heart failure; Mexilitene; Pre-excitation.

Untreated supraventricular tachycardia (SVT) can lead to myocardial failure and subsequent congestive heart failure. Hemodynamic changes occur within 1 day of tachycardia with progressive deterioration in function and heart failure over 3–5 weeks.[1] A variety of classes of antiarrhythmic drugs such as calcium-channel blockers, beta-blockers, or more recently, class 3 drugs (sotalol, amiodarone) have been used to terminate SVT in dogs. Traditionally, class 1b antiarrhythmic drugs (Vaughan Williams classification), which include lidocaine and mexilitene, have been used in the treatment of ventricular rather than supraventricular tachyarrhythmias. Lidocaine is considered a safe and effective drug for the treatment of ventricular tachycardia (VT) because it has a short half-life and a rapid onset of action when administered intravenously (IV).[2]

Lidocaine has been reported to have little effect on atrial fibers and conduction in accessory pathways, and therefore is of little value in the treatment of SVT.[2,3] The action potential duration of atrial tissue is short when compared with that of the ventricles. Class 1b agents are therefore considered to be ineffective in atrial arrhythmias because they have a particular affinity for binding with inactivated sodium channels with rapid onset–offset kinetics. There are limited reports on the use of lidocaine to treat SVT in dogs.[4]

Lidocaine has been reported to be effective in some forms of SVT in humans and, in some cases, subsequent therapy with oral mexilitene was successful in maintaining sinus rhythm.[5–9] In this article we report on the successful termination of SVT in 5 dogs using lidocaine administered IV.

Materials and Methods

All cases were referred to the Veterinary Cardiorespiratory Center for the investigation of tachycardia and, in 2 cases, concurrent congestive heart failure. Dogs underwent physical examination, and a complete blood profile that included CBC and biochemistry, including measurement of serum total thyroxine concentration, electrocardiography, echocardiography, and thoracic radiography.

Lidocaine for pharmacological cardioversion was administered IV as a rapid bolus at 2–3 mg/kg over 5 seconds. This dose was repeated after 2–3 minutes if the initial dose was unsuccessful. A final dose of 2–3 mg/kg was repeated after a further 2–3 minutes if necessary. The total dose did not exceed 8 mg/kg.

Results

Case 1

A 2-year-old female Golden Retriever presented with a history of multiple episodes of collapse with tachycardia over the previous 6 months. During these episodes she appeared lethargic, adopted sternal recumbency, and was tachypneic. These episodes persisted for 2–4 hours. In between, no abnormalities were detected.

No CBC or serum biochemical abnormalities were detected. Thoracic radiographs and an echocardiogram did not detect abnormalities.

At electrocardiography, there was normal sinus rhythm at 100 beats per minute. The PR interval was short, at 50 milliseconds, and there was notching of the initial portion of the QRS complexes. These changes were consistent with ventricular pre-excitation (Fig. 1). Sustained SVT (320/min) developed during the recording of the electrocardiogram. The SVT did not resolve over the following 25 minutes.

Lidocaine (2 mg/kg IV) was administered and this dose was repeated after 3 minutes. Within a further 2 minutes, a return to normal sinus rhythm was established. The dog was then maintained on mexilitene (6 mg/kg IV q8h). Two brief episodes of tachycardia persisting for less than 20 minutes have been noted by the owner over the succeeding 3 years.

Case 2

A 2-year-old male, neutered Cocker Spaniel was referred with a history of collapse with tachycardia, tachypnea, and pallor for 24 hours. At examination the heart rate was 310 beats per minute. Sustained SVT was evident at electrocardiography. Moderate pulmonary edema in the caudal lung fields was present on thoracic radiographs.

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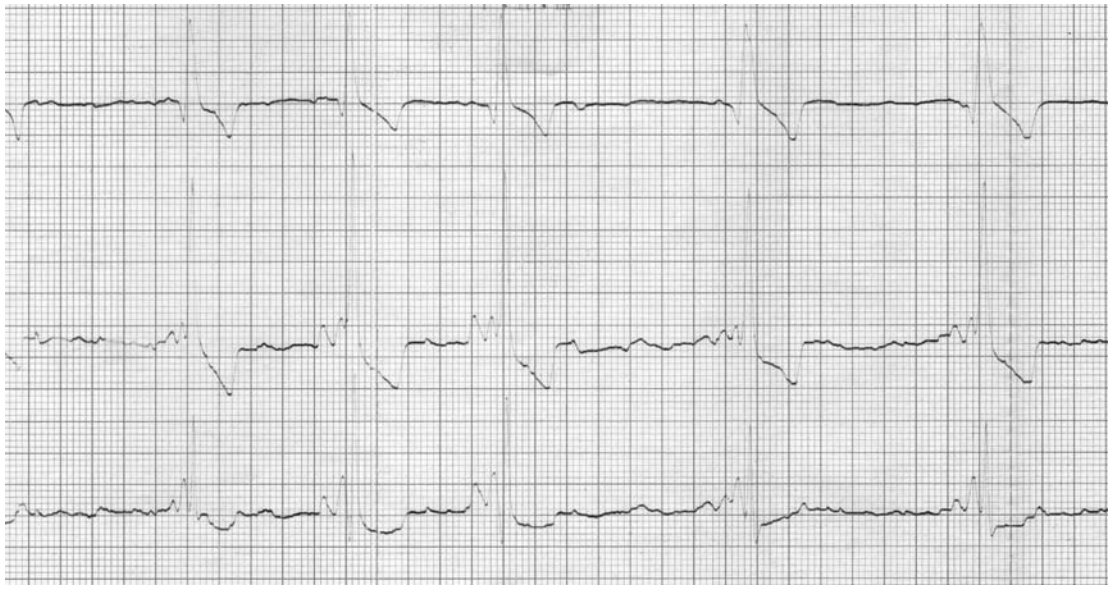

Fig 1. Case 1 after conversion to normal sinus rhythm. The short PR interval and notched QRS complex is suggestive of pre-excitation. Leads 1, 2, and 3 are 50 mm/s, 1 mV/cm.

Lidocaine (3 mg/kg IV) caused conversion to normal sinus rhythm at 150/minute within 60 seconds. SVT recurred at 2 hours and 4.5 hours. On each occasion there was a prompt return to normal sinus rhythm after a single IV bolus of lidocaine (3 mg/kg). Oral mexilitene (6 mg/kg q8h) was then administered. Furosemide[d] (2 mg/kg PO q12h) and benazapril[e] (0.5 mg/kg PO q24h) were also provided to control signs of congestive heart failure. Both medications were discontinued after 4 weeks.

A rapid improvement was noted and the dog remained stable for 2 years, until it developed lethargy and exercise intolerance due to dilated cardiomyopathy. One month later he relapsed with SVT and was again converted to normal sinus rhythm with 1 bolus of lidocaine (2 mg/kg IV). The dose of mexilitene was increased to 8 mg/kg q8h. He has remained stable for the next 2 years and has shown only 4 clinically apparent bouts of SVT over this period. On each occasion these were associated with omission of treatment by the owner, and there was a return to normal sinus rhythm when oral administration of mexilitene resumed.

Case 3

A 6-month-old female Labrador was referred with a history of lethargy, dyspnea, and tachycardia for 4 days. Thoracic radiographs demonstrated a mild perihilar alveolar lung pattern consistent with pulmonary edema and left heart enlargement with a prominent left atrial wedge. Moderate mitral and tricuspid valvular regurgitation were detected at echocardiography. The left atrium was markedly dilated. Left ventricular diastolic and systolic dimensions were increased, and fractional shortening was reduced to 17%.

Sustained SVT was apparent at electrocardiography. Conversion to normal sinus rhythm was achieved with a single bolus of lidocaine (3 mg/kg IV). Mexilitene was administered (8 mg/kg per os q8h) daily, and normal sinus rhythm was maintained for the next 12 months. Furosemide (2 mg/kg per os q12h) was discontinued after 1 week.

The dog was clinically normal at 1 year after treatment. A final diagnosis of mitral and tricuspid valvular dysplasia was made on the basis of continuing valvular regurgitation and enlarged left atrium and left ventricle.

Case 4

An 8-year-old male Boxer presented with a history of episodic weakness due to intermittent SVT over the previous 2 years. Results of CBC and serum biochemistry were unremarkable. No abnormalities were detected on thoracic radiographs and echocardiography. Electrocardiography had demonstrated frequent episodes of nonsustained SVT at 300 beats per minute. The dog had been maintained on mexilitene (6 mg/kg per os q8h) and atenolol[f] (0.3 mg/kg per os q12h) over the previous 6 weeks. The day before presentation the dog became weak, tachypneic, and tachycardic.

The dog had sustained SVT at 310 beats per minute. Lidocaine was administered (3 mg/kg IV) and this dose was repeated after 3 minutes. Within 1 minute after the second dose there was conversion to normal sinus rhythm. Evidence of pre-excitation was present after conversion with a reduced PR interval and a notched upstroke on the QRS complexes (Fig. 2).

Sustained SVT recurred 1 year later and again there was a response to 2 boluses of lidocaine IV at the same dose. Mexilitene (8 mg/kg per os q8h) controlled the

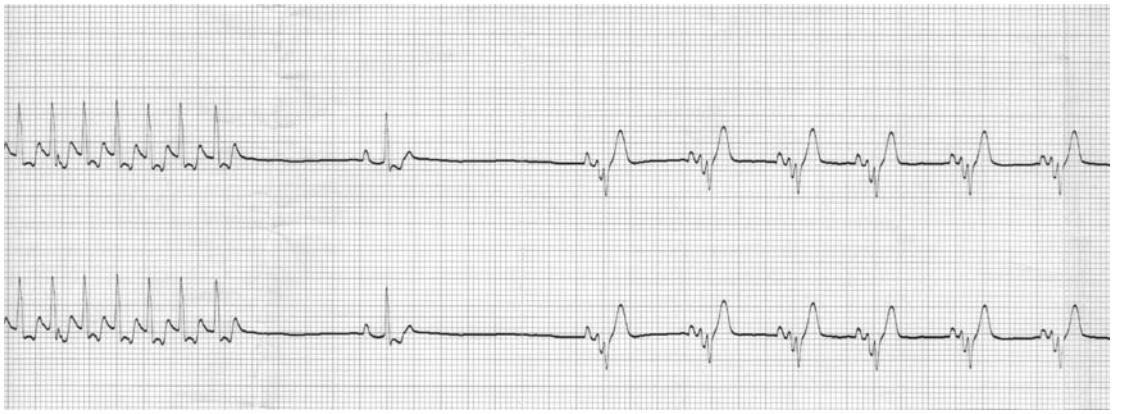

Fig 2. Case 4. Conversion to normal sinus rhythm. A normal sinus beat follows the SVT. Subsequent sinus complexes show pre-excitation with a short PR interval and notched QRS complexes. Leads 2 and 3 are 25 mm/s, 1 mV/cm.

arrhythmia satisfactorily until death from noncardiac related causes at 12 years of age.

Case 5

An 18-month-old Labrador Retriever was referred with a history of intermittent weakness, tachypnea, and tachycardia over the previous 2 months. At examination a tachycardia was present. No abnormalities were detected on a blood profile, thoracic radiology, or echocardiography. Electrocardiography revealed sustained SVT at 300 beats per minute.

A single bolus of lidocaine (3 mg/kg IV) was successful in conversion to normal sinus rhythm. Lidocaine was administered on 3 further occasions over the next 6 hours due to hourly relapses of SVT. Mexilitene (8 mg/kg per os q8h) and propranolol (1 mg/kg per PO q8h) terminated the SVT with only 3 relapses over a 2-year follow-up period. On each occasion, brief relapse was due to poor owner compliance.

Discussion

Lidocaine is advocated for use in frequent or sustained wide complex tachycardia, which is most commonly due to VT. It has been successful in termination of these arrhythmias in humans and animals.

The successful use of lidocaine in conversion of atrial tachycardia due to Wolff-Parkinson-White (WPW) syndrome was documented in 1969.[5] This is due to lidocaine slowing conduction and prolonging refractoriness across the accessory pathway.[10] Several case reports subsequently confirmed the efficacy of this drug in humans presenting with SVT due to the presence of accessory pathways.[5,6,8,9,11] A larger study involving 99 people documented the success of lidocaine in conversion of SVT in cases of automatic atrial tachycardia, but not in cases of atrial flutter, atrial fibrillation, or in patients with tachycardia due to the WPW syndrome.[7] Antiarrhythmic drugs used in treating WPW syndrome may act on the AV node (digoxin, beta-blockers), accessory pathway (procainamide and possibly lidocaine), or both (amiodarone).[12] A more recent report in humans documents a lack of efficacy of lidocaine in conversion of atrial fibrillation in a series of patients.[13] However, another study confirmed demonstrated conversion of 8 patients with an undetermined mechanism of atrial tachycardia.[3]

A short communication involving the successful treatment of 2 dogs with SVT using lidocaine was presented at a veterinary cardiovascular society meeting in 1996, but the use of lidocaine to treat SVT in dogs is not common.[4]

A mechanism for the tachycardia is not evident in many dogs presenting with SVT without performing electrophysiologic studies. However, recent information based on electrophysiologic studies in dogs suggests that accessory pathways are responsible for the majority of cases of SVT in dogs. A minority have focal atrial tachycardia.[14] Due to poor retrograde conduction in the canine AV node, it seems unlikely that AV nodal reentry tachycardia is a common mechanism of SVT in dogs, unlike the case in humans.[14]

Although electrophysiologic testing is necessary to diagnose the specific mechanism underlying SVT, lidocaine may be effective in cases of SVT due to accessory pathways or focal atrial tachycardia.[7,8] Success is unlikely in cases of SVT due to AV nodal reentry.[8] Lidocaine's efficacy should be rapidly apparent, and if unsuccessful, alternative drugs could be used.

All 5 dogs in this study presented with narrow complex SVT. None had electrophysiological studies performed to categorize the mechanism involved. Cases 1, 4, and 5 involved a primary arrhythmia alone without evidence of structural heart disease on echocardiography. Cases 1 and 4 showed pre-excitation due to the presence of an accessory pathway based on the electrocardiographic findings. In case 5, SVT may have been due to an accessory pathway given the young age and breed (Labrador), but evidence of this could not be demonstrated on the surface electrocardiogram. However, many such pathways are occult. Case 3 involved a young Labrador Retriever with dysplasia of the AV valves. In this breed, an association between tricuspid

dysplasia and accessory pathway tachycardia has been noted.[15] Therefore this dog's SVT may have been due to a concurrent accessory pathway. The remaining case (case 2) presented with tachycardia-induced congestive heart failure and 2 years later was diagnosed with dilated cardiomyopathy. It is unknown whether the SVT and dilated cardiomyopathy were related, although cardiomyopathy had developed in the absence of persistent tachycardia. In cases 2 and 3, it seemed unlikely that tachycardia-induced cardiomyopathy had developed, as prolonged rhythm control did not prevent the subsequent onset of cardiomegaly (case 2) or lead to a reduction in cardiac dimensions (case 3).

In all cases there was a response to bolus administration of lidocaine. Cases 2 and 5 relapsed within hours of cardioversion but again responded to repeat bolus administration. The effect of lidocaine could be prolonged by slow infusion if necessary, but this was not performed in this series of cases. All dogs were successfully maintained on oral mexilitene or mexilitene and propranolol (case 5), with only intermittent, usually brief, relapses of SVT over the maintenance period.

Adverse effects of lidocaine were observed in case 4 only. These included sedation, salivation, and tremors, but these were resolved rapidly and did not require specific therapy. In these and other cases we have not encountered any serious adverse effects after lidocaine administration.

Class 1b agents inhibit the fast sodium current while shortening the action potential duration in nondiseased tissue.[16] Lidocaine has rapid onset and offset kinetics and does not affect normal sinus node automaticity but does depress abnormal forms of automaticity, as well as early and late after-depolarizations in Purkinje fibers in vitro.[17] Lidocaine has little effect on atrial fibers and does not affect atrioventricular or intraventricular conduction.[18] In Purkinje fibers the action potential is shortened.[19] Class 1b agents act selectively on diseased or ischemic tissue where they are believed to promote conduction block, thereby interrupting re-entry circuits.[16] Lidocaine may reduce the rate of rise of phase 0 of the action potential and hence conduction velocity in re-entry pathways in addition to increasing the refractory periods. Unidirectional block may then be converted to bidirectional block.[20] Some cases of SVT due to macro re-entry involve the ventricles, and this may be a reason for the efficacy of class 1b agents in such patients.[21]

Caution has been advised in the use of lidocaine in certain circumstances such as when SVT exists in the presence of atrioventricular block. In such cases, therapy with lidocaine could cause acceleration of the ventricular rate due to a reduction in the atrioventricular block.[22] Lidocaine can decrease the refractoriness of the accessory pathway, which could lead to ventricular fibrillation. Likewise, disease of the sinoatrial or atrioventricular nodes may be contraindications for the use of lidocaine.[20]

Dogs presenting with broad complex tachycardia are usually assumed to have VT. However, some cases may be due to SVT conducted with aberrancy. Successful conversion of a broad-complex tachycardia by lidocaine, whether due to VT or SVT, is possible, and successful cardioversion in such cases does not necessarily prove an underlying VT.

Footnotes

[a] Lidocaine, Lignocaine hydrochloride injection 2% w/v, Martindale, Essex, UK
[b] Mexilitene, Mexitil capsules, Boehringer Ingelheim, Berkshire, UK
[c] Propranolol, Inderal tablets, Zeneca, Cheshire, UK
[d] Furosemide, Frusemide tablets, Millpledge Pharmaceuticals, Retford, UK
[e] Benazapril, Fortekor tablets, Novartis, Basel, Switzerland
[f] Atenolol, Atenolol tablets, Tillomed, St. Neots, UK

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