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Lidocaine for chemical cardioversion of orthodromic atrioventricular reciprocating tachycardia in dogs

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Abstract

Background: Typical atrioventricular accessory pathways (APs) are composed of myocardial cells. They provide electrical connections between atria and ventricles separate from the normal conduction system. Accessory pathways can participate in a macroreentrant circuit resulting in orthodromic atrioventricular reciprocating tachycardia (OAVRT).

Hypothesis: Because of ultrastructural similarities of typical AP cells to ventricular myocardial cells, we hypothesized lidocaine would be effective in blocking AP conduction, thus terminating OAVRT.

Animals: Thirty-two consecutive client-owned dogs presenting with narrow complex tachyarrhythmias were confirmed to have OAVRT by electrophysiologic study (EPS).

Methods: Prospective, nonrandomized, single-arm study with lidocaine administered IV to dogs during OAVRT in 2 mg/kg boluses to a cumulative dose of 8 mg/kg or development of adverse effects. Electrocardiograms were monitored continuously. Subsequent EPS was performed to confirm OAVRT and the absence of other tachycardia mechanisms.

Results: Twenty-seven dogs experienced OAVRT cardioversion with lidocaine, before or at the time of adverse effects. Orthodromic atrioventricular reciprocating tachycardia in 5 dogs did not cardiovert before adverse effects, precluding additional dosing. Median total lidocaine dose for cardioversion was 2 mg/kg (interquartile range, 2-5.5 mg/kg). Dogs with right free wall APs had a significantly higher rate of cardioversion than did dogs with right posteroseptal APs.

Conclusions and Clinical Importance: Lidocaine successfully cardioverted OAVRT in 84.4% of dogs in our study before adverse effects precluded additional dosing. In 5 dogs with dose limited by adverse effects, it is unknown whether cardioversion would have occurred at a higher cumulative dose.

KEYWORDS

ablation, accessory atrioventricular pathways, antiarrhythmic drugs, arrhythmia, cardiovascular, ventricular preexcitation, Wolff-Parkinson-White syndrome

Abbreviations: AP, atrioventricular accessory pathway; AV, atrioventricular; EPS, electrophysiologic study; OAVRT, orthodromic atrioventricular reciprocating tachycardia; VPE, ventricular preexcitation.

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1 | INTRODUCTION

The typical accessory pathway (AP) is composed of myocardial cells spanning the fibrous atrioventricular (AV) junction. These cells provide electrical continuity between the atrial and ventricular myocardium at a site electrophysiologically distinct from the AV node and proximal His-Purkinje system.[1] Such connections have been described in the developing human heart, normally regressing by 20 weeks of gestation. It has been inferred that failure of these pathways to regress forms the substrate for APs.[2] Accessory pathways typically are capable of retrograde conduction, from ventricular to atrial myocardium, allowing them to participate in a macroreentrant tachycardia circuit that uses the atrial myocardium, AV node-His-Purkinje system, ventricular myocardium, and retrograde AP. This arrhythmia is known as orthodromic atrioventricular reciprocating tachycardia (OAVRT) and is a differential diagnosis for any regular, narrow QRS complex tachycardia that terminates in the face of second-degree AV block.[3] Rarely, OAVRT can have a wide QRS complex, such as when there is preexisting or rate-dependent bundle branch block.[4,5] Some APs also can conduct in the antegrade direction, electrically preexciting at least a portion of the ventricular myocardium with an atrial signal that bypasses the normal AV nodal-His-Purkinje system during sinus rhythm. The degree of ventricular preexcitation (VPE) depends upon a number of factors, including the conduction properties of the antegrade AP and the antegrade AV node.[6,7]

Lidocaine is classified as an IB antiarrhythmic agent according to the Vaughan-Williams classification system.[8] It binds to and blocks open and inactivated fast sodium channels responsible for phase 0 (rapid depolarization) of the action potential in non-nodal cardiomyocytes. Lidocaine exerts greater effects in depolarized (eg, ischemic) and rapidly driven tissues, making it a use-dependent antiarrhythmic agent.[9] Action potential duration usually is unaffected or shortened, perhaps because of block of the plateau sodium current. Lidocaine is highly effective for ventricular tachyarrhythmias, but largely ineffective for atrial tachyarrhythmias. A study in isolated atrial and ventricular myocytes, however, showed that lidocaine's sodium current inhibition was essentially identical in these 2 cell types.[10] Thus, other mechanisms must be involved in the differential efficacy of lidocaine in ventricular versus atrial tachyarrhythmias. An analysis of the ultrastructural morphology of cardiomyocytes within 3 surgically excised APs identified connexin 43 distribution matching that in ventricular cardiomyocytes, not as found in atrial or AV nodal cells.[11] This finding suggests that APs may have similar responses to lidocaine as do ventricular cardiomyocytes.

2 | MATERIALS AND METHODS

A prospective, nonrandomized, single-arm clinical study was performed. Dogs were included if they had a sustained narrow complex tachyarrhythmia strongly suspected to be OAVRT on baseline 12-lead ECG and after the owner's informed consent for treatment. The clinical tachyarrhythmia had to have regular R-R intervals, a short fixed RP′ interval that was less than the P′R interval, immediate arrhythmia termination if second-degree AV block occurred, and be proven to be OAVRT at subsequent electrophysiologic study (EPS).[12] Lidocaine hydrochloride (Vedco, St. Joseph, Missouri) was injected via a cephalic catheter at an initial dose of 2 mg/kg over 20-30 seconds. If no conversion of the tachyarrhythmia occurred after 2 minutes and no adverse effects developed, an additional 2 mg/kg IV bolus was administered, to a total cumulative dose of 8 mg/kg. Continuous ECG recording (6-lead or single-lead II) was performed during this protocol. Recorded data included (1) cumulative dose administered; (2) cardioversion, adverse effects, or maximal 8 mg/kg dose reached as a cause for cessation of administration; (3) ECG characteristics at the time of cardioversion (including whether or not a P′ wave was present after the last QRS complex of the tachyarrhythmia); and (4) whether dogs with VPE exhibited this finding on the sinus complexes after cardioversion.

Dogs were taken for EPS the next day. The presence of an AP was confirmed using standard techniques.[12,13] Multielectrode catheters (Boston Scientific, Marlborough, Massachusetts) were placed percutaneously under general anesthesia within the high right atrium, right ventricular apex, His bundle region, and coronary sinus. Programmed electrical stimuli (Bloom DTU-215 Programmable Cardiac Stimulator, Fischer Medical Technologies, Wheat Ridge, Colorado) were delivered to the distal poles of the high right atrial and right ventricular catheters to determine antegrade and retrograde conduction properties of the normal conduction system and any APs present. A typical AP was confirmed if ≥1 of these criteria were met: (1) ventricular activation occurring before the His bundle potential (VPE) during sinus rhythm or atrial pacing, (2) eccentric atrial activation during ventricular pacing and narrow complex tachyarrhythmia, and (3) rapid, nondecremental ventriculoatrial conduction during decremental right ventricular pacing with identical retrograde atrial activation at all cycle durations (particularly important if concentric retrograde atrial activation occurs).[14-16] This third criterion does not apply to uncommon APs that exhibit slow decremental retrograde conduction properties. Accessory pathway participation in the tachyarrhythmia circuit was confirmed by advancing (or delaying in the case of a decrementally conducting AP) the identical retrograde atrial activation sequence or terminating the tachycardia without subsequent atrial activation after introduction of a single RV extrastimulus into the tachycardia during His bundle refractoriness.[14-16] Once an AP was confirmed, it was located using fine mapping of the shortest ventriculoatrial time during RV pacing or AV time during sinus rhythm (in the case of APs capable of antegrade conduction), coupled with the identification of AP potentials. If additional tachycardia mechanisms (eg, focal atrial tachycardia) were present during EPS, they also were identified and addressed. Radiofrequency catheter ablation was performed through the distal ablation catheter electrode (Blazer II Temperature Ablation Catheter, Boston Scientific) coupled with a temperature-controlled cardiac ablation unit (EPT 1000TC Cardiac Ablation Unit, Boston Scientific), with a surface grounding pad serving as the indifferent electrode. Antegrade and retrograde conduction properties, as well as location of the APs, were recorded.

3 | STATISTICAL METHODS

Summary data were reported as median and interquartile range (IQR). Two data sets were developed to reflect (1) outcome at final lidocaine dosages (final lidocaine dose data set) and (2) outcome at single (2 mg/kg) as well as multiple (>2 mg/kg) lidocaine doses (2 mg/kg threshold data set). This design accounted for outcome data being recorded for multiple doses in some patients. The final dose data set was used to analyze relationships between lidocaine cardioversion and constant patient variables (eg, age, breed, sex, AP location, AP conduction direction, and fastest OAVRT cycle duration). The 2 mg/kg threshold data set was used to assess lidocaine dose-related effects on cardioversion and adverse effects, as well as relationships with fastest OAVRT cycle duration and AP location for lidocaine responders. The Wilcoxon rank sum test was utilized to compare continuous data, Pearson's chi-squared test was used to compare categorical data, and a Fisher's exact test was employed for categorical data when expected cell counts were insufficient. All analyses were performed using commercially available statistical software (R Programming Language version 3.5.1, Boston, Massachusetts) and significance threshold was designated at $P < .05$.

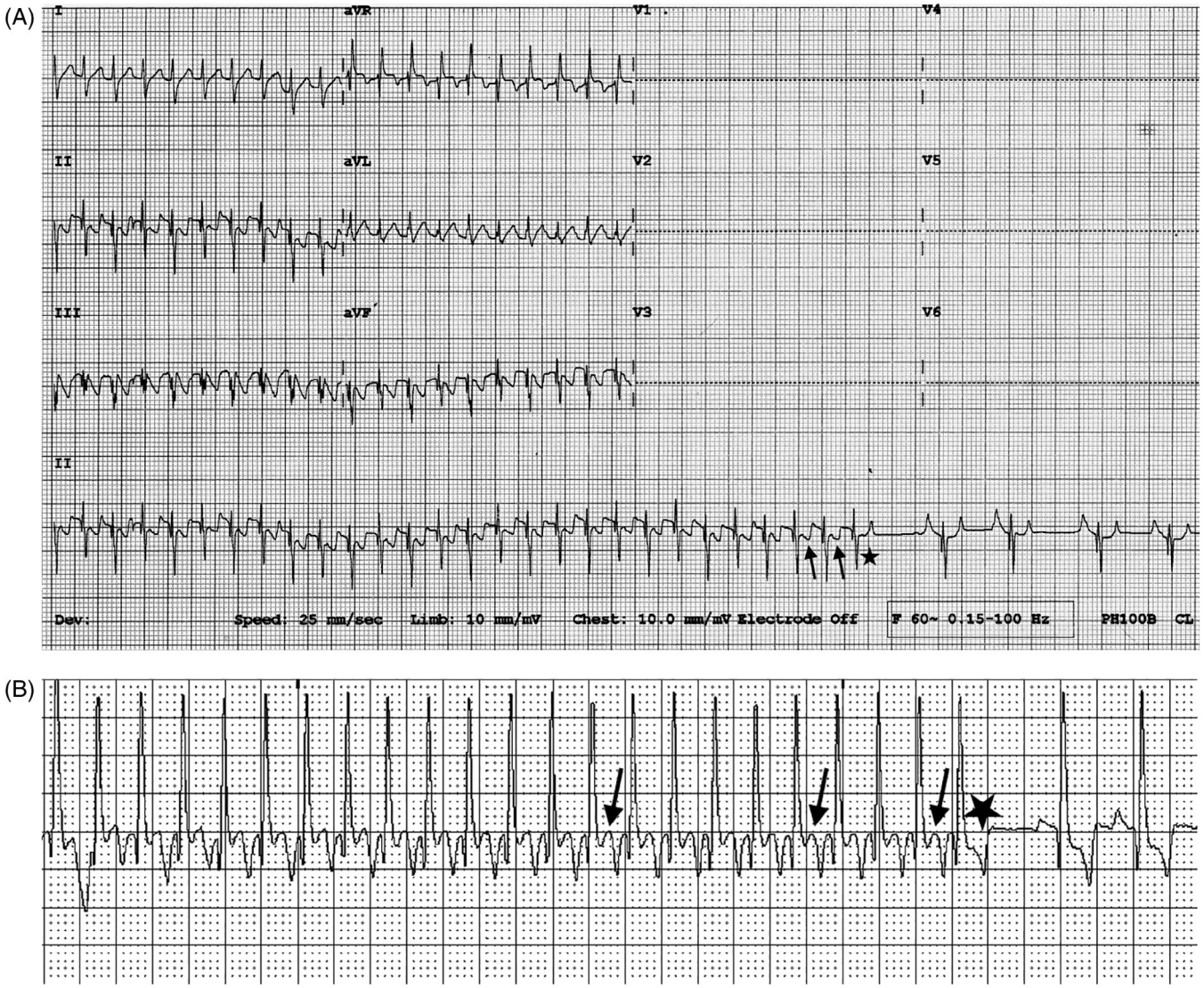

FIGURE 1 A, Six-lead ECG from an 8.3-year-old male Labrador Retriever with a narrow complex tachycardia, proven to be orthodromic atrioventricular reciprocating tachycardia at electrophysiologic study. Lidocaine was administered IV according to the study protocol. Arrows point to representative retrograde P′ waves visible within the early ST segment after each QRS complex. The star denotes the absence of a retrograde P′ wave with subsequent cardioversion because of retrograde accessory pathway block. This dog also has a preexisting intraventricular conduction disturbance, resulting in a right cranial axis shift of the QRS complexes. Paper speed 25 mm/s, calibration 10 mm/mV. B, Telemetric ECG recording at the time of intravenous administration of lidocaine in a 1-year-old male Labrador Retriever. Orthodromic atrioventricular reciprocating tachycardia converts to sinus rhythm when conduction over the retrograde accessory pathway is blocked, as evidenced by the absence of a retrograde P′ wave in the ST segment (denoted by the star symbol). Examples of retrograde P′ waves during orthodromic atrioventricular reciprocating tachycardia are labeled with arrows. Paper speed 25 mm/s, calibration 10 mm/mV

4 | RESULTS

Thirty-two dogs were enrolled in the study, including 16 Labrador Retrievers, 4 mixed breed dogs, 3 Boxers, 2 golden retrievers, and 1 each of German Shorthaired Pointer, Boerboel Mastiff, Border Collie, Doberman Pinscher, German Shepherd, Siberian Husky, and Newfoundland. The median age was 1.63 years (IQR, 0.67-3.98 years), ranging up to 9.7 years. There were 6 intact females, 4 spayed females, 6 intact males, and 16 neutered males. The median fastest sustained narrow complex tachycardia cycle duration recorded for each dog was 180 ms (IQR, 171-200 ms). The range of cycle durations varied from 150 to 205 ms, corresponding to heart rates of 292-400 bpm (Table 1). All dogs had been on antiarrhythmic medications before presentation. Five dogs were on a single antiarrhythmic agent, 20 dogs were receiving 2 antiarrhythmic drugs, and 7 dogs were receiving 3 antiarrhythmic medications. Medications included diltiazem (23 dogs), sotalol (20 dogs), mexiletine (9 dogs), propafenone (8 dogs), digoxin (3 dogs), amiodarone (2 dogs), procainamide (1 dog), and verapamil (1 dog). All oral antiarrhythmic medications were discontinued for 5 half-lives before EPS.

Twenty-seven dogs cardioverted with lidocaine, before adverse effects that precluded additional dosing occurred (Figure 1 and Table 2). Termination occurred with a QRS complex that was not followed by a P′ wave, consistent with block in the retrograde AP, rather than in the AV node. The median total dose of lidocaine needed to convert OAVRT to sinus rhythm was 2 mg/kg (IQR, 2-5.5 mg/kg), ranging up to 8 mg/kg. The 5 dogs that did not cardiovert were dose-limited by lidocaine adverse effects that developed at a median dose of 6 mg/kg (IQR, 6-6 mg/kg), starting as low as 4 mg/kg. When the 5 dogs dose-limited by adverse effects were excluded, higher dosing of lidocaine (>2 mg/kg versus 2 mg/kg) yielded a higher likelihood of cardioversion (P = .003; Table 3).

TABLE 1 Study population characteristics

Age	**1.63 years (0.67-3.98 years)**
Sex	
Female intact	6
Female spayed	4
Male intact	6
Male neutered	16
Breed	
Labrador	17
Non-Labrador	15
Fastest OAVRT cycle duration	180 ms (171-200 ms)
AP Location	
LFW	1
LPS	1
Midseptal	1
RFW	16
RPS	12
RPS, RFW	1
AP direction	
Bidirectional	8
Retrograde only	24

Abbreviations: AP, atrioventricular accessory pathway; OAVRT, orthodromic atrioventricular reciprocating tachycardia.

TABLE 2 Outcome summary

Cardioversion (Y/N)	27/5
Total lidocaine dose at cardioversion	2 mg/kg (2-5.5 mg/kg)
Adverse effects (Y/N)	10/22
Total lidocaine dose at adverse effects	6 mg/kg (5.25-6 mg/kg)

TABLE 3 Dose-related effects for cardioversion and adverse effects

	2 mg/kg lidocaine	**>2 mg/kg lidocaine**	***P* value**
Cardioversion			.003
Yes	14	13	
No	13	0	
Adverse effects			<.001
Yes	0	10	
No	32	8	

Adverse effects associated with lidocaine were encountered in 10 dogs (Table 2). Five of these dogs converted to sinus rhythm at the dose at which adverse effects first developed, whereas the remaining 5 did not cardiovert, preventing them from reaching the maximal protocol lidocaine dose of 8 mg/kg. The median lidocaine dose reached in the 10 dogs with adverse effects was 6 mg/kg (IQR, 5.25-6 mg/kg). Adverse effects were significantly associated with higher lidocaine dosages (P < .001, Table 3). None of the dogs that failed to have OAVRT cardiovert with lidocaine reached the maximal target dose of 8 mg/kg, rather all 5 dogs dropped out because of lidocaine adverse effects. Observed adverse effects included gastrointestinal signs (eg, hypersalivation, lip smacking, and vomiting) in 10 dogs and neurologic signs (eg, sedation, head tremors, opisthotonus, body tremors, and seizures) in 4 dogs. Such adverse effects were brief, and only 1 dog required intervention (ie, IV diazepam for seizures).

Fourteen dogs that cardioverted with lidocaine boluses and did not develop adverse effects were placed on constant rate infusions of lidocaine because of frequent OAVRT recurrence once the bolus effect dissipated. These constant rate infusions ranged from 25 to 60 μg/kg/min, with a median dose of 50 μg/kg/min. Five of these dogs required supplemental lidocaine boluses of 2 mg/kg for OAVRT breakthrough while on their lidocaine constant rate infusions. Three other dogs could be maintained on intermittent lidocaine boluses alone for recurrent in-hospital OAVRT. Eight dogs required only 1 lidocaine bolus to cardiovert their OAVRT and did not develop

subsequent sustained OAVRT before their EPS and AP ablation the next day. Seven other dogs received IV diltiazem for subsequent OAVRT episodes.

Of the 3 dogs with manifest APs in which OAVRT cardioverted with lidocaine, 2 experienced antegrade AP block in sinus rhythm immediately after cardioversion and 1 did not. Another dog with a manifest AP did not have OAVRT cardioversion before the onset of lidocaine adverse effects. Accurate assessment of lidocaine's effects on antegrade AP conduction in dogs exhibiting intermittent pre-excitation was not possible.

A single AP was identified in 31 dogs, whereas 1 dog had 2 APs during EPS. No other tachycardia mechanism was identified during the EPS. The location of these APs was as follows: 17 along the right free wall, 13 in the right posteroseptal region, 1 in the left posteroseptal region, 1 in the midseptal region, and 1 along the left free wall. When comparing the right free wall and right posteroseptal locations, which accounted for >90% of the APs in the study, lidocaine cardioversion was significantly more likely in dogs with right free wall APs (P = .02; Table 4). For those dogs that did cardiovert, lidocaine dose did not affect the likelihood of cardioversion between these 2 sites (P = .19; Table 5).

The fastest OAVRT cycle duration did not differ between dogs that cardioverted and those that did not (P = .4; Table 4), nor between dogs cardioverting at a low dose (2 mg/kg) and those requiring a higher lidocaine dose (>2 mg/kg) (P = .69; Table 5). No differences were identified between cardioverters and non-cardioverters with regard to age at presentation (P = .84), breed (Labrador versus non-Labrador, P = .64), sex (P = .29), or concealed versus manifest APs (P = 1.0; Table 4).

TABLE 4 Assessment of patient and atrioventricular accessory pathway (AP) characteristics on cardioversion of orthodromic atrioventricular reciprocating tachycardia (OAVRT) by lidocaine

Cardioversion	Yes	No	*P* value
Fastest sustained OAVRT CL	180 ms (171-200)	170 ms (160-200)	.40
Age	1.5 years (0.67-4.00)	3.08 years (2.25-3.17)	.84
AP location			.02
RFW	16	0	
RPS	8	4	
Breed			.64
Labrador	15	2	
Non-Labrador	12	3	
Sex			.29
Female	7	3	
Male	20	2	
AP conduction			1.00
Bidirectional	7	1	
Retrograde only	20	4	

TABLE 5 Single versus multidose analysis for dogs with lidocaine cardioversion

Cardioversion dosage	2 mg/kg lidocaine	>2 mg/kg lidocaine	*P* value
Fastest OAVRT cycle duration	180 ms (172-200)	180 ms (171-200)	.69
AP location			.19
RFW	10	6	
RPS	2	6	

Abbreviations: AP, atrioventricular accessory pathway; OAVRT, orthodromic atrioventricular reciprocating tachycardia.

5 | DISCUSSION

Lidocaine generally is considered ineffective in treating narrow complex tachyarrhythmias.[17-19] In this series of 32 dogs with confirmed APs, however, lidocaine was successful in converting OAVRT to sinus rhythm in 27 dogs (84%). Five dogs developed adverse effects precluding additional dosing. Thus, it is not known whether these dogs also would have cardioverted if a higher cumulative dose could have been administered. The dogs that did cardiovert did so at a lower dose than the group of 5 dogs that failed to cardiovert before adverse effects developed. This observation may indicate that the non-conversion group had APs that were less sensitive to the sodium channel blocking effects of lidocaine.

A prior study of 5 dogs with narrow complex tachyarrhythmias reported rapid conversion to sinus rhythm with lidocaine administered as a total IV bolus of 3-6 mg/kg.[20] Two of these dogs experienced VPE after conversion to sinus rhythm, whereas 2 other dogs were suspected of having concealed APs based upon their age and breed (young Labrador Retrievers). None of the dogs in this earlier study underwent EPS to confirm the mechanism of their narrow complex tachyarrhythmia. This is in contrast to the dogs of our study, in which OAVRT was confirmed as the sole mechanism of their narrow complex tachyarrhythmia during EPS. When combining the results of these 2 studies, 5 dogs had manifest APs and also had their OAVRT successfully cardioverted with lidocaine. Three of these 5 dogs experienced VPE immediately upon reestablishment of sinus rhythm, consistent with retrograde AP conduction block only, whereas the other 2 had bidirectional AP conduction block. This observation suggests that lidocaine has differential effects on antegrade versus retrograde AP conduction and that antegrade conduction may be more resistant to class IB drug effects. An additional dog with a manifest AP in our study developed adverse effects before OAVRT cardioversion, precluding assessment of lidocaine's effects on antegrade AP conduction. Additional studies specifically designed to address the effects of lidocaine on antegrade and retrograde AP characteristics are warranted.

The human medical literature regarding lidocaine treatment of AP-mediated tachyarrhythmias has been conflicting, but generally unfavorable. Lidocaine is not currently accepted as a treatment for narrow complex tachyarrhythmias in human medicine.[17,18] Advanced cardiac life support guidelines consistently state that lidocaine is not an

effective or appropriate treatment for supraventricular tachyarrhythmias.[19] A small number of early single case studies demonstrated successful cardioversion of OAVRT with lidocaine administration.[21,22] Other early studies in human patients suggested that lidocaine could be useful in slowing the ventricular response rate in preexcited atrial fibrillation by blocking antegrade conduction over the AP.[23,24] One study demonstrated prolongation of the 1:1 atrial pacing cycle duration that resulted in loss of antegrade AP conduction (loss of VPE) after lidocaine administration.[24] This effect occurred in 4 of 6 patients evaluated, and suppression of antegrade AP conduction by the sodium channel blocking effects of lidocaine was postulated.

Subsequent studies in human patients, however, have not been favorable. Another study evaluated the effects of IV lidocaine on retrograde and antegrade AP conduction in human patients with Wolff-Parkinson-White syndrome.[25] Lidocaine had no statistically significant effect on the AP's retrograde or antegrade effective refractory periods in the 9 (retrograde) and 11 (antegrade) patients who were tested. Lidocaine also had no effect on the ease of OAVRT inducibility or on the tachycardia cycle duration in these patients. Another study found that lidocaine had inconsistent effects on the shortest and average R-R intervals during preexcited atrial fibrillation.[26] The average R-R interval decreased in 5 patients, increased in 2, and did not change in 1. The antegrade AP effective refractory period also was not prolonged in these patients with rapidly conducting antegrade APs. The medical community's general acceptance of the lack of therapeutic effect of lidocaine on supraventricular tachyarrhythmias is echoed in an article reviewing the pharmacologic treatment of narrow complex tachyarrhythmias in children.[27]

Despite these reports in humans, lidocaine proved beneficial in acutely terminating OAVRT in our series of dogs. Lidocaine at therapeutic serum concentrations has no effect on the atrial effective refractory period.[28] In isolated atrial cardiomyocytes, lidocaine is effective in blocking the sodium current that is present.[10] The more depolarized resting membrane potential of atrial myocytes, however, results in less availability of inward sodium channels compared with ventricular myocytes. Thus, a smaller inward sodium current underlies the atrial action potential. The lack of a plateau phase in atrial myocytes also results in fewer inactivated sodium channels to be blocked by lidocaine and similar drugs. These findings can help explain why class IB agents have no clinically relevant electrophysiologic effects on atrial tissue, and there is general agreement that it is ineffective in the treatment of atrial arrhythmias.[28-32]

In rare instances, atrial tachyarrhythmias do terminate with lidocaine. One study reported 8 human patients with an uncommon form of repetitive atrial tachycardia that showed progressive cycle duration prolongation during an atrial salvo, suppression as the sinus rate increased with mild exercise, and prolongation of salvoes with vagal stimulating maneuvers.[33] This form of atrial tachycardia, which appears to be vagally mediated, did respond to lidocaine administration, with abbreviation of the atrial salvoes and eventual restoration of sinus rhythm. This finding is in contrast to the abrupt termination of OAVRT and restoration of sinus rhythm by lidocaine administration in the dogs of our series. With regard to vagally induced atrial tachyarrhythmias, a study in dogs demonstrated efficacy of lidocaine in converting atrial fibrillation to sinus rhythm in a particular setting, namely vagally induced atrial fibrillation.[34] In this same study, 5 episodes of pacing-induced atrial tachycardia occurred in the setting of high vagal tone caused by fentanyl but followed by the vagolytic atropine. Three of these 5 episodes converted to sinus rhythm. The proposed mechanisms in this specific setting include blunting the effects of acetylcholine at the atrial level in addition to sodium channel blockade by lidocaine. Lidocaine's effect in atrial fibrillation, however, appears to be unique to cases occurring in the setting of high vagal tone. A prospective, randomized, double-blind crossover trial in 20 human patients with naturally occurring atrial fibrillation failed to result in cardioversion in any patient.[35]

Based on our experience, we have had only 1 dog with a proven atrial tachyarrhythmia cardiovert with lidocaine, in keeping with the human medical literature documenting its general lack of efficacy for terminating atrial tachyarrhythmias, except in selected circumstances. Lidocaine's termination of a narrow complex tachyarrhythmia, therefore, may have benefit as a diagnostic test, because this drug thus far has been more likely to terminate OAVRT than atrial tachycardia. Additional studies in dogs undergoing EPS to precisely define the mechanism underlying their regular narrow complex tachyarrhythmia are warranted to specifically address lidocaine's diagnostic utility in differentiating OAVRT from other mechanisms.

From a therapeutic perspective, as we transition from IV to PO antiarrhythmic agents to definitive catheter ablation of a dog's AP, the individual's response to lidocaine can help guide PO antiarrhythmic treatment. We found that dogs with OAVRT that cardiovert with IV lidocaine may respond well in the interim period to mexiletine alone or preferably in combination with a negative dromotropic agent, so that both limbs of the reentrant circuit are treated. The lidocaine dose required for cardioversion also has been helpful in providing us a general guideline as to the starting mexiletine dosage. For example, if cardioversion required only a low dose of lidocaine, then starting at the lower end of the mexiletine dosage range is reasonable. If cardioversion required a high dose, then starting nearer the higher end of the dosage range likely will be necessary.

Interestingly, dogs with right free wall APs were significantly more likely to cardiovert with lidocaine than were dogs with right posteroseptal APs. The electrophysiologic basis for this observation is not clear, but it suggests a difference in the sodium channel properties of APs at these 2 locations. Further data should be collected to determine if this significant difference is maintained in larger studies.

A potential limitation of our study is that lidocaine was not administered at the time of, but rather before, the EPS. We did not want lidocaine's effects on the AP to inhibit our ability to map and ablate it; thus, we could not administer lidocaine during the EPS. Hence, one might argue that the narrow complex tachyarrhythmia seen before EPS could have had a different mechanism than OAVRT in these dogs. We dispute that conclusion based on the following: (1) the ECG appearance of the narrow complex tachyarrhythmia in all of these dogs matched that of their electrophysiologically proven OAVRT during EPS the next day, with an identical, short, fixed RP′ interval;

(2) the narrow complex tachyarrhythmia terminated spontaneously immediately upon development of second-degree AV block, which would not be expected to occur with an atrial tachycardia; and (3) no narrow complex tachyarrhythmic mechanism other than OAVRT was identified at the time of EPS in this group of dogs.

In summary, our study supports the use of IV lidocaine for potential chemical cardioversion of a regular, narrow complex tachyarrhythmia in dogs. Successful cardioversion by lidocaine supports a tentative diagnosis of OAVRT as the narrow complex tachyarrhythmia's mechanism. Failure to cardiovert does not definitively rule out OAVRT, particularly if adverse effects limit the maximal dose that can be administered.

CONFLICT OF INTEREST DECLARATION

Authors declare no conflict of interest.

OFF-LABEL ANTIMICROBIAL DECLARATION

Authors declare no off-label use of antimicrobials.

INSTITUTIONAL ANIMAL CARE AND USE COMMITTEE (IACUC) OR OTHER APPROVAL DECLARATION

Authors declare no IACUC or other approval was needed.

HUMAN ETHICS APPROVAL DECLARATION

Authors declare human ethics approval was not needed for this study.

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