J Vet Intern Med 2012;26:558–564

Atrial-Based Pacing for Sinus Node Dysfunction in Dogs: Initial Results

A.H. Estrada, R. Pariaut, S. Hemsley, B.H. Gatson, and N.S. Moïse

Background: An important consideration for the treatment of sick sinus syndrome (SSS) lies in the function of the atrioventricular (AV) node because most patients with SSS retain the ability to conduct atrial impulses.

Hypothesis/Objectives: This retrospective study examined the feasibility of atrial pacing (AAI) in dogs with sinus node dysfunction (SND).

Animals: Sixteen dogs with SND and AAI pacing were identified.

Methods: Retrospective review of medical records.

Results: Follow-up time ranged from 45 to 1,227 days (mean: 292 days). Only 1 dog developed AV block 3 days postoperatively. Complete lead dislodgment occurred in 3/16 dogs 1, 19, and 27 days postoperatively. Lead perforation into the pericardial space occurred in 2/16 dogs. Rising thresholds for pacing with possible lead microdislodgment or fibrosis were suspected in another 3/16 dogs 57, 192, and 1,016 days after implantation. None of these dogs had complete loss of capture but all required higher thresholds for pacing.

Conclusions and Clinical Importance: Based on this small group of dogs, clinically important AV block does not appear to occur in the long-term for dogs with SND. Risks of lead perforation, complete dislodgment, and rising thresholds for pacing, possibly because of microdislodgment, may be related to the initial skill level of the operator or the leads that were used. Use of leads with reduced torque at the lead tip, higher flexibility, increased lead-tip surface of contact with the endocardium or, more likely, use of alternate locations for pacing in the small right atrium of dogs with SND may decrease the frequency of these complications.

Key words: Lead dislodgment; Lead perforation; Sick sinus syndrome; Sinus node dysfunction.

Permanent pacemaker implantation is a fairly routine procedure performed by veterinary cardiologists. The most common indication for permanent cardiac pacing in veterinary medicine is complete, or third degree atrioventricular (AV) block.[1–4] Sinus node dysfunction (SND), or sick sinus syndrome (SSS), accounts for a smaller percentage of patients receiving permanent pacemakers. The importance of maintaining synchronous ventricular contraction and using a functional AV node when possible has been underscored in numerous clinical trials and case reports in the human literature.[5–14] This may be even more important for many of the breeds afflicted with SND that often are affected by degenerative valve disease. Dyssynchrony of ventricular contractions caused by right ventricular apical pacing potentially can exacerbate the degree of valvular regurgitation.[15] Appropriate selection of pacing site (atrial versus ventricular versus dual chamber) should be an important consideration when placing permanent pacing devices in dogs with SND. Dual chamber pacing may be more expensive, and in the small breed dogs most commonly seen with SND, dual leads may increase the risk of cranial vena cava syndrome. Cranial vena cava syndrome has been reported in 2 large breed dogs[16] with dual chamber pacing systems and thus has been considered a reason to avoid multiple leads in small breeds with SND. With single-chamber ventricular pacing intact retrograde ventriculoatrial (VA) conduction may occur. Although the incidence of VA conduction with SND has not been reported in veterinary medicine, its frequency is reported to be as high as 80% in humans with SND.[17–20] Single-chamber atrial-based pacing traditionally has been avoided because retrospective reports in human patients with SND have indicated an increased risk of developing AV block after the implantation of atrial-based pacing systems.[21–23] Other prospective trials evaluating AV conduction in the long-term for human patients with SND have found that AV conduction remains stable.[24–28] In veterinary medicine, single chamber, ventricular-based pacing remains the standard therapy for patients with SND.[a] The aim of this retrospective study was to evaluate AV nodal conduction after permanent single-chamber atrial-based pacing in dogs with SND and to report on both feasibility and complications identified.

Abbreviations:

AAI	atrial pacing
AV	atrioventricular
SND	sinus node dysfunction
VA	ventriculoatrial

From the Section of Cardiology, Department of Small Animal Clinical Sciences, College of Veterinary Medicine, University of Florida, Gainesville, FL (Estrada, Gatson); the Section of Cardiology, Department of Veterinary Clinical Sciences, School of Veterinary Medicine, Louisiana State University, Baton Rouge, LA (Pariaut); and the Section of Cardiology, Department of Clinical Sciences, College of Veterinary Medicine, Cornell University, Ithaca, NY (Hemsley, Moïse). The study was completed at the University of Florida, College of Veterinary Medicine and was presented as an abstract at the 2011 ECVIM conference in Sevilla, Spain.

Corresponding author: Amara H. Estrada, Section of Cardiology, Department of Small Animal Clinical Sciences, College of Veterinary Medicine, University of Florida, Gainesville, FL 32610-0126; e-mail: estradaa@ufl.edu

Submitted December 6, 2011; Revised February 21, 2012; Accepted March 10, 2012.

10.1111/j.1939-1676.2012.00928.x

Materials and Methods

Medical records for dogs diagnosed with SND at Cornell University Companion Animal Hospital and the University of Florida Small Animal Hospital between May 1999 and April 2010 were reviewed. Patients receiving implantation of transvenous permanent atrial-based pacing systems (AAI) were selected and the following information was collected: patient breed, weight and age; presence or absence of AV block on initial ECG, 24-hour Holter monitoring or both, and on subsequent re-evaluation of ECGs, 24-hour Holter monitoring or both; method of lead fixation (active or passive); lead polarity (unipolar or bipolar); lead model number and specifics; lead location; and duration of follow-up after implantation. Complications after permanent pacemaker implantation were identified and recorded.

Results

Animals

Sixteen dogs with SND and AAI pacing were identified. Age at implantation ranged from 3 to 14 years (mean ± SD, 10.2 ± 3.1). Breeds represented included Miniature Schnauzer (n = 7), West Highland White Terrier (n = 4), Mastiff (n = 2), Cocker Spaniel (n = 2), and Dachshund (n = 1). Patient size ranged from 5 to 90 kg (median: 9.1 kg) and all but 2 of these dogs were < 19 kg.

Pacemaker Implantation

All implantations were performed by board-certified cardiologists or cardiology residents under the supervision of a board-certified cardiologist. The role of primary surgeon rotated among several individuals, resulting in a lack of consistency as to who placed the leads. All dogs were successfully implanted with atrial leads and atrial pacing was confirmed in all dogs. Anesthetic protocols were determined by the anesthesia personnel and were not uniform in all patients. Five dogs were implanted with preformed J-shaped atrial leads whereas 11 had straight leads manufactured for implantation in either the atrium or ventricle. In all dogs, use of a straight stylet within the right atrial body to straighten the lead was first used. Once the lead tip was at the edge of the caudal vena cava (but still within the body of the right atrium), either a preformed J-shaped stylet or a straight stylet customized at the surgical table with a J-shaped curve was used to direct the lead into the right atrium. In 14 dogs, the desired lead placement was the right auricular appendage. In 2 dogs, lead placement in the right atrial septum was attempted. Once the preferred location was selected, 7 of 16 dogs underwent atrial pacing to assess the Wenckebach point (ie, the heart rate at which progressive prolongation of AV nodal conduction followed by second degree AV block occurred). In these 7 (anesthetized) dogs, the Wenckebach point occurred at heart rates of 120–130 beats per minute (bpm). In the remaining dogs (9/16), this determination was not performed. Pacing thresholds and atrial sensitivity were assessed in 5/16 dogs at the time of implantation and were assessed in the remainder of the dogs (11/16) approximately 24 hours after implantation. Five dogs were implanted with unipolar leads and 11 dogs were implanted with bipolar leads. Four dogs were implanted with passive fixation leads and 12 were implanted with active fixation (extendable helix) leads. Eleven leads had a steroid-eluting tip (<1 mg dexamethasone sodium phosphate, dexamethasone acetate or both) whereas 5 did not. Follow-up time ranged from 45 to 1,227 days (mean: 292 days).

Electrocardiographic and Holter Recording Results

All dogs had an initial ECG and follow-up ECG at each visit. Five dogs had initial 24-hour Holter monitoring performed to assess AV nodal conduction before placement of a permanent atrial pacing lead. Eleven dogs had subsequent 24-hour Holter monitoring after pacemaker implantation. Atrioventricular block was not present in any dog before permanent pacemaker implantation. One dog (a 13-year-old Miniature Schnauzer) developed complete AV block 3 days PO and required pacing system revision and ventricular pacing. This dog did not have presurgical Holter monitoring performed or Wenkebach point testing while under anesthesia but had normal AV nodal conduction based on a surface ECG (PR interval,120 milliseconds; no episodes of AV block on a 5-minute surface ECG). The remainder of the dogs had occasional 2nd degree AV block detected on either ECG or Holter evaluation, either immediately PO during a period whereby the effects of anesthesia were still present or only during times of sleep and rest on re-evaluation 24-hour Holter monitoring.

Complications

Pacing System Evaluation. All patients had pacing system interrogation at all re-evaluation points. Rising thresholds for pacing, without loss of capture, were identified in 5 dogs. In 3 of these dogs, this rise in threshold occurred 57, 192, and 1,016 days after implantation. Pacing thresholds stabilized after additional exercise restriction and rest in all 3 dogs and did not require pacing system revision. Atrial leads in the other 2 dogs with rising thresholds for pacing were suspected to have perforated the atrial myocardium and are discussed further below. Oversensing, consisting of refractory sensed events, and noise reversion pacing[29] were seen in 5/5 unipolar leads and in 4/11 bipolar leads (Fig 1).

Lead Dislodgement. Complete lead dislodgment occurred in 3/16 dogs at day 1 (90-kg Mastiff), day 19 (9-kg West Highland White Terrier), and day 27 (9.2-kg Miniature Schnauzer) PO. These lead dislodgments all occurred with actively fixed, straight (non-J-shaped) atrial/ventricular bipolar leads. These patients all were converted to ventricular pacing systems and, in some instances, retrograde VA conduction was observed (Fig 2) with ventricular pacing. Three additional patients (mentioned above) had rising thresholds for pacing with stable lead tip position radiographically at

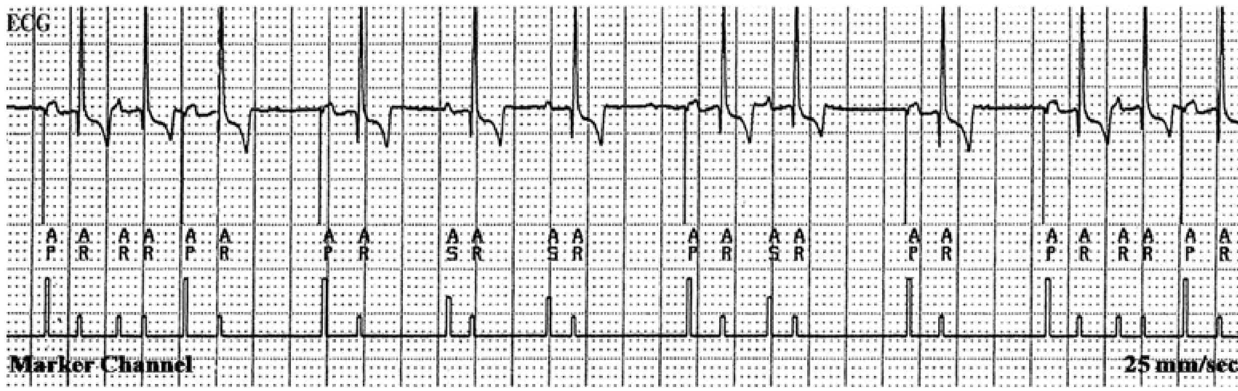

Fig 1. Surface ECG and marker channel recorded a dog with sinus node dysfunction and a single lead atrial pacing system illustrating noise reversion pacing. Repeated refractory sensed events indicated by the labeling "AR" cause asynchronous pacing.

day 57 (9.9-kg West Highland White Terrier), day 192 (23.4-kg Cocker Spaniel), and day 1,016 (11.2-kg West Highland White Terrier) after implantation. All 3 leads had steroid-eluting tips with < 1.0 mg dexamethasone acetate. The cause of the rising pacing thresholds in these dogs was unknown, but either lead fibrosis or lead microdislodgement[30] was considered possible. Two of these dogs had actively fixed, straight (non-J) atrial/ventricular bipolar leads implanted. The third was equipped with a passively fixed, straight (non-J) atrial/ventricular bipolar lead. In this dog, the lead tip was stable, but a loop of lead was seen to cross the tricuspid valve on radiographs and echocardiogram 1,016 days after implantation. In all 3 of these dogs, rising thresholds for pacing were detected on the dates indicated. Pacing thresholds stabilized with higher voltage settings and exercise restriction without requiring revision of the pacing system.

Lead Perforation. Lead perforation into the pericardial space occurred in 2 dogs. Both dogs were Miniature Schnauzers and weighed 7.8 and 8.8 kg, respectively. The first dog was re-examined approximately 3 months after implantation because of lethargy. Intermittent loss of capture was seen electrocardiographically, but increasing the pulse amplitude and duration resulted in complete capture. Thoracic radiographs suggested perforation of the lead when compared to the postoperative radiographs (Fig 3). Echocardiography did not identify any pericardial effusion, and the lead tip could not be visualized in the pericardial space. Thresholds for pacing continued to rise over the course of the next few days to the point where only intermittent capture was achievable even at the highest voltage amplitude and duration. This dog underwent thoracotomy and pericardectomy to successfully remove the atrial lead. No pericardial

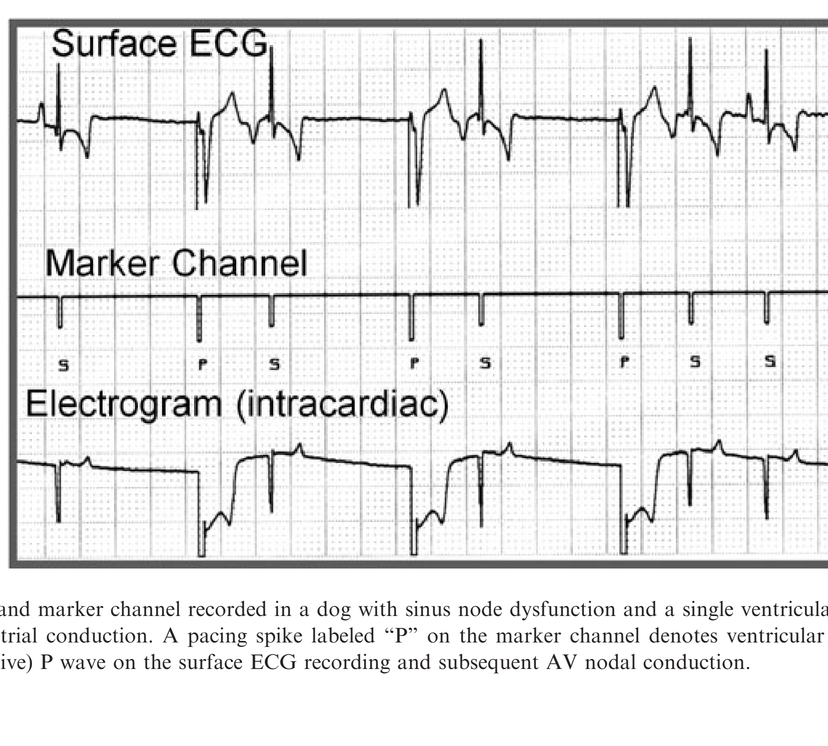

Fig 2. Surface ECG and marker channel recorded in a dog with sinus node dysfunction and a single ventricular lead system illustrating retrograde ventriculoatrial conduction. A pacing spike labeled "P" on the marker channel denotes ventricular pacing. This is followed by a retrograde (negative) P wave on the surface ECG recording and subsequent AV nodal conduction.

Fig 3. Right lateral and VD radiographs (**A**) 24 hours postimplantation and (**B**) at recheck exam. (A) A pacemaker generator is identified within the dorsal subcutaneous tissue of the right cervical region. A single pacemaker lead is identified extending through the right jugular vein and terminating in the right auricular appendage. (B) The dorsal cervical subcutaneous pacemaker generator and single lead passing through the right side jugular vein is again seen. The tip of the pacemaker lead has changed in position and is in a more dorsal orientation than immediately postimplantation, and suspected to be lying in the pericardial space.

effusion was observed, and the lead was removed without bleeding. An epicardial lead was placed on the ventricular myocardium for permanent pacing. In the second dog, rising thresholds 4 months postoperatively prompted radiographic evaluation. Radiographs again suggested perforation into the pericardial space. Pericardial effusion was not apparent on echocardiography. Surface ECG demonstrated that the lead alternated between pacing of the atrial myocardium and pacing of the base of the ventricular myocardium (Fig 4). Pacing threshold remained stable in this dog, and the lead was left in place. Subsequent 24-hour Holter monitoring of this dog did not identify any periods of loss of atrial or ventricular capture. Both dogs with lead perforation had passively fixed J-shaped atrial bipolar leads with a small lead-tip surface area of 2.5 mm^2.

Other Complications. Skeletal muscle tremors and twitching were noted by owners in 3 dogs. All of these dogs had unipolar leads implanted, and the muscle tremors were decreased or alleviated with a reduction in pulse width, amplitude, or both. One Miniature Schnauzer developed right-sided pneumothorax postoperatively. This complication did not cause any clinical signs and was only identified on postoperative thoracic radiographs. Thoracocentesis was performed and pneumothorax did not redevelop. Echocardiography postoperatively, 24 hours later and 4 days later failed to show any evidence of pericardial effusion and no additional complications occurred with the pacing system.

Discussion

Dogs with SND can be successfully paced from the atria, thereby providing a more physiologic approach

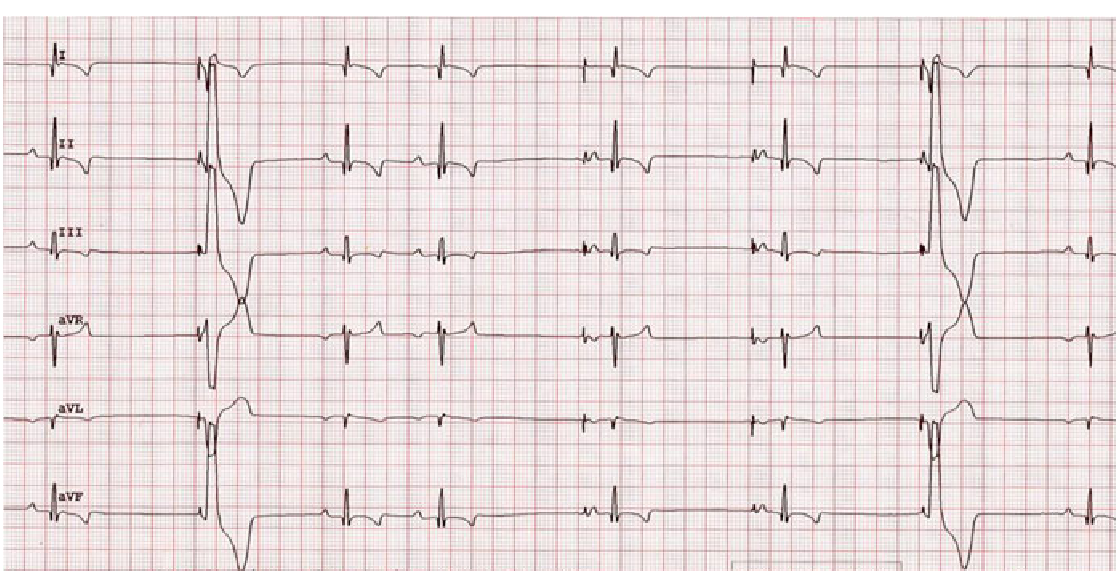

Fig 4. Electrocardiogram from a patient with sinus node dysfunction and perforation of an atrial pacing lead. There is an underlying sinus rhythm present with the pacing lead alternating between capture of the atrial myocardium and the ventricular myocardium. Paper speed 50 mm/s; sensitivity 5 mm/mV.

to management. Although ours is a retrospective study with multiple operators and no individual being proficient at implantating atrial leads in predominantly small dogs, this limited experience has provided some information to improve the technique of atrial pacing. Moreover, in this series of dogs with SND, the long-term risk is low for development of clinically relevant AV block. This is an important finding because the primary indication for single-chamber atrial pacing is the assurance that AV nodal block will not develop.

To insure that dogs with SND do not have coexisting AV nodal block, 24-hour Holter monitoring before pacing may allow acquisition of more information about AV nodal conduction. In humans, during permanent pacemaker implantation, a stepped-up atrial pacing protocol is used to identify the Wenckebach blocking point. A Wenckebach point of < 120 bpm in humans is considered a contraindication for single-chamber atrial pacing.[23,26,27] The corresponding Wenckebach point limit in dogs anesthetized with a variety of drugs is unknown and would need to be established before recommendations can be made.

In this limited series of operators with limited experience in the specific implantation procedure, the incidence of complications related to atrial lead stability appeared high. Perforation of the atrial myocardium, complete dislodgment of the atrial lead, and possible microdislodgment or excessive fibrosis of the lead tip with rising thresholds for pacing were seen commonly in this group of dogs. Atrial leads are placed commonly with rare complications in dogs for studies at companies developing pacing devices (Personal communication, Dr Nancy Rakow, DVM, Senior Veterinary Surgeon, Physiological Research Laboratories, Medtronic Inc, Minneapolis, MN). However, the procedure is performed in large (commonly > 25 kg) young dogs rather than in small breed older dogs with coexisting AV valve degeneration such as those seen clinically with SND. Lead dislodgment with loss of capture resulting in revision and replacement of the pacing system occurred in 3 dogs relatively soon after placement. All of these leads were bipolar, non-J-shaped atrial/ventricular active fixation leads which, in a small atrium, may place tension on the lead arc and tip, leading to subsequent dislodgment. Use of unipolar leads, which often have only 1 conductor wire, and thus are thinner and more flexible, could alleviate this tension. However, far field sensing and noise reversion pacing were uniformly seen with unipolar leads, making them less desirable. Over the last few years, small diameter bipolar leads have been developed that could provide more ideal fixation and sensing for small dogs, but perforations occurred in the 2 dogs that had very small lead-tip surface areas ($2.5\ mm^2$) implanted. These small electrode tips have been theorized to place an increased force per unit area on the thin atrial myocardium, making it more likely for perforation to occur. Also, rather than extruding the contained helix in the leads with a stylet, the small diameter leads that do not use a stylet are secured by turning the entire lead, and these are more prone to perforation.[b] Despite this concern, cardiac perforation is a rare complication, and one that is thought to be more likely with prior steroid use and active fixation leads in addition to the small lead-tip surface area.[36] Possible risk factors with active fixation leads include overscrewing of the lead, distal stylet insertion past the length of the pacing lead, or abrupt withdrawal of the lead with an extended screw. A recent study in adult humans using computed tomography found that atrial lead perforation was more common than previously suspected.[37] Late atrial lead perforations, defined as those occurring > 1 month postoperatively, occurred in 15% of the patients evaluated. Passive fixation atrial leads actually had a greater rate of perforation (25%) than active atrial leads (12%), which is in conflict with previous reports of active fixation leads having a higher perforation rate.[36]

Pacing lead microdislodgment[30] or excessive fibrosis at the lead tip, caused by unstable contact between the electrode tip and tissue interface, was possible in 3 patients. In all of these patients, rising thresholds for pacing with a radiographically stable lead tip were seen. No pacing system revision was performed and exercise restriction along with increases in voltage amplitude resulted in a stabilization of thresholds in all patients. These patients all were implanted with non-J-shaped, active fixation leads, which we hypothesize may have led to excessive torque and motion at the lead tip, particularly in smaller sized patients such as those typically seen with SND.

In reviewing the diameter of implanted leads, the smallest lead was 5.3 Fr with polyurethane insulation. The issue with this lead appears to be the small lead-tip surface area of $2.5\ mm^2$. There are many J-shaped and non-J-shaped active and passive fixation endocardial leads manufactured with a similar small lead body (5–6 Fr) but with a larger lead-tip surface area.[b–f] Even smaller leads (3–4 Fr) are available for placement in the atrial endocardial surface. The latter are placed via a 7 Fr steerable sheath because a central stylet is not possible in this small diameter lead.[g] The requirement of the sheath therefore prevents this option for small dogs unless alternative delivery system catheters are improvised.[h]

This retrospective study of atrial pacing in dogs with SND has several limitations. First, the number of dogs evaluated was small. Moreover, although the operators had performed ventricular pacemaker implantation for years, their experience with atrial lead placement was limited. The type of lead used was varied and this resulted in only a few dogs having a particular type implanted. Therefore, the reasons provided for why complications developed are hypothetical.

Conclusions

The major finding of this retrospective study is that atrial lead implantation is possible in small breeds and that clinically relevant AV block does not appear to occur in the long-term in dogs with SND. Careful selection of patients for atrial-based pacing using Wenckebach testing and 24-hour Holter monitoring before pacing are recommended to assess for pre-existing AV block. In the small number of procedures reported here, lead perforation, lead dislodgment, fibrosis, and possible microdislodgment were more common when compared to published experiences with ventricular pacing in veterinary patients[1–4,37] and atrial pacing in people.[31–36] Leads with decreased torque at the lead tip, use of J-shaped atrial leads, leads with higher flexibility during atrial contraction, and increased lead-tip surface of contact with the endocardium may decrease these complications but will require evaluation. Fixation of the lead in the interatrial septum may be a better alternative to the right auricle in small dogs, but this hypothesis requires investigation. Because of the potential long-term benefits of AAI pacing in dogs with SND, veterinary cardiologists should continue to optimize AAI pacing and compare this method to ventricular pacing for dogs with SND.

Footnotes

[a] Hogan DF. Current pacing practices and future directions. ACVIM 2011 proceedings notes, p 6–8, Denver, CO

[b] Tendril STS Lead, St Jude, Inc, St Paul, MN

[c] OptiSense 1999 Pacing Lead with Optim Insulation, St Jude

[d] Tendril ST Optim Pacing Lead, St Jude

[e] CapSure SP Novus Models, Medtronic, Inc, Minneapolis, MN

[f] CapSureFix Novus Models, Medtronic Inc

[g] SelectSecure Model 3830, Medtronic, Inc

[h] Attain Select II Family of Sub-Selection Catheters, Medtronic, Inc

Acknowledgments

Dr Nancy Rakow at Medtronic Inc for technical assistance.

Conflicts of interest: The authors have no conflicts of interest to disclose.

References

1. Sisson D, Thomas WP, Woodfield J, et al. Permanent transvenous pacemaker implantation in forty dogs. J Vet Intern Med 1991;5:322–331.

2. Oyama MA, Sisson DD, Lehmkuhl LB. Practices and outcome of artificial pacing in 154 dogs. J Vet Intern Med 2001;15:229–239.

3. Domenech O, Santilli R, Pradelli D, Bussadori C. The implantation of a permanent transvenous endocardial pacemaker in 42 dogs: A retrospective study. Med Sci Monit 2005;11:168–175.

4. Wess G, Thomas WP, Berger DM, Kittleson MD. Applications, complications, and outcomes of transvenous pacemaker implantation in 105 dogs (1997–2002). J Vet Intern Med 2006;20:877–884.

5. Prinzen FW, Peschar M. Relation between pacing induced sequence of activation and left ventricular pump function in animals. Pacing Clin Electrophysiol 2002;25:484–498.

6. Albertsen AE, Nielsen JC, Poulsen SH, et al. DDD(R)-pacing, but no AAI(R)-pacing induces left ventricular desynchronization in patients with sick sinus syndrome: Tissue-Doppler and 3D echocardiographic evaluation in a randomized controlled comparison. Europace 2008;10:127–133.

7. Burkhoff D, Oikawa R, Sagawa K. Influence of pacing site on canine left ventricular contraction. Am J Physiol 1986;251: H428–435.

8. Rosenqvist M, Bergfeldt L, Haga Y, et al. The effect of ventricular activation sequence on cardiac performance during pacing. Pacing Clin Electrophysiol 1996;19:1279–1286.

9. Wyman BT, Hunter WC, Prinzen FW, McVeigh ER. Mapping propagation of mechanical activation in the paced heart with MRI tagging. Am J Physiol 1999;276:H881–891.

10. Tse HF, Lau CP. Long-term effect of right ventricular pacing on myocardial perfusion and function. J Am Coll Cardiol 1997;29:744–749.

11. Nielsen JC, Bottcher M, Nielsen TT, et al. Regional myocardial blood flow in patients with sick sinus syndrome randomized to long-term single chamber atrial or dual chamber pacing-effect of pacing mode and rate. J Am Coll Cardiol 2000;35:1453–1461.

12. Sweeney MO, Hellkamp AS, Ellenbogen KA, et al. Mode Selection Trial Investigators. Adverse effect of ventricular pacing on heart failure and atrial fibrillation among patients with normal baseline QRS duration in a clinical trial of pacemaker therapy for sinus node dysfunction. Circulation 2003;23:2932–2937.

13. The DAVID Trial Investigators. Dual-chamber pacing or ventricular backup pacing in patients with an implantable defibrillator: The Dual Chamber and VVI Implantable Defibrillator (DAVID) trial. J Am Med Assoc 2002; 288: 3115–3123.

14. Sharma AD, Rizo-Patron C, Hallstrom AP, et al. DAVID Investigators. Percent right ventricular pacing predicts outcome in the DAVID trial. Heart Rhythm 2005;2Suppl 2:S75–76.

15. Sassone B, De Simone N, Parlangeli G, et al. Pacemaker-induced mitral regurgitation: Prominent role of abnormal ventricular activation sequence versus altered atrioventricular synchrony. Ital Heart J 2001;6:441–448.

16. Van De Wiele CM, Hogan DF, Green HW III, Parnell NK. Cranial vena cava syndrome secondary to transvenous pacemaker implantation in two dogs. J Vet Cardiol 2008;10:155–161.

17. Hayes DL, Furman S. Atrioventricular and ventriculoatrial conduction times in patients undergoing pacemaker implant. Pacing Clin Electrophysiol 1983;6:38–46.

18. Westveer DC, Stewart JR, Goodfleish R, et al. Prevalence and significance of ventriculoatrial conduction. Pacing Clin Electrophysiol 1984;7:784–789.

19. Rubin JW, Frank MJ, Boineau JP, Ellison RC. Current physiologic pacemakers: A serious problem with a new device. Am J Cardiol 1983;52:88–91.

20. Van MechelenRV, Ruiter J, Vanderkerchkhove Y, et al. Prevalence of retrograde conduction in heart block after DDD pacemaker implantation. Am J Cardiol 1986;57:797–801.

21. Sutton R, Kenny RA. The natural history of sick sinus syndrome. Pacing Clin Electrophysiol 1986;9:1110–1114.

22. Bertholet M, Demoulin JC, Fourny J, Kulbertus H. Natural evolution of atrioventricular conduction in patients with sick sinus syndrome treated by atrial demand pacing: A study of 26 cases. Acta Cardiol 1983;38:227–232.

23. Vallin H, Edhag O. Associated conduction disturbances in patients with symptomatic sinus node disease. Acta Med Scand 1981;210:263–270.

24. Adachi M, Igawa O, Yano A, et al. Long-term reliability of AAI mode pacing in patients with sinus node dysfunction and low Wenckebach block rate. Europace 2008;10:134–137.

25. Pioger G, Leny G, Nitzsche R, Ripart A. AAIsafeR limits ventricular pacing in unselected patients. Pacing Clin Electrophysiol 2007;30:S66–S70.

26. Tripp IG, Armstrong GP, Stewart JT, et al. Atrial pacing should be used more frequently in sinus node disease. Pacing Clin Electrophysiol 2005;28:291–294.

27. Nielsen JC, Kristensen L, Andersen HR, et al. A randomized comparison of atrial and dual-chamber pacing in 177 consecutive patients with sick sinus syndrome. J Am Coll Cardiol 2003;42:614–623.

28. Andersen HR, Nielsen JC, Thomsen PEB, et al. Atrioventricular conduction during long-term follow up of patients with sick sinus syndrome. Circulation 1998;98:1315–1321.

29. Moise NS, Estrada AH. Noise reversion in paced dogs. J Vet Cardiol 2002;4:13–21.

30. Kikuchi K, Abe H, Nagamoto T, Nakashima Y. Microdislodgment: A likely mechanism of pacing failure with high impedance small area electrodes. Pacing Clin Electrophysiol 2003;26:1541–1543.

31. Khan MN, Joseph G, Khaykin Y, et al. Delayed lead perforation: A disturbing trend. Pacing Clin Electrophysiol 2005;28:251–253.

32. Irwin JM, Greer GS, Lowe JE, et al. Atrial lead perforation: A case report. Pacing Clin Electrophysiol 1987;10:1378–1381.

33. Wan-Jing H, Chi-Tai K, Kuo-Hong L. Right pneumothorax resulting from an endocardial screw-in atrial lead. Chest 1999;116:1133–1134.

34. Trigano AJ, Taramasco V, Paganelli F, et al. Incidence of perforation and other mechanical complications during dual active fixation. Pacing Clin Electrophysiol 1996;19:1828–1831.

35. Hirschl DA, Jain VR, Spindola-Franco H, et al. Prevalence and characterization of asymptomatic pacemaker and ICD lead perforation on CT. Pacing Clin Electrophysiol 2007;30:29–32.

36. Mahapatra S, Bybee KA, Bunch TJ, et al. Incidence and predictors of cardiac perforation after permanent pacemaker placement. Heart Rhythm 2005;2:907–911.

37. Johnson MS, Martin MWS, Henley W. Results of pacemaker implantation in 104 dogs. J Small Anim Pract 2007;48:4–11.