

Indications and Technique for Permanent Cardiac Pacemaker Implantation in the Dog

Susan M. Kimm, BS*
Brian L. Hill, DVM, MS**

Electrical pacing of the heart has been utilized in human medicine for more than two decades. Cardiac pacing was used initially for cases of complete heart block that were unresponsive to medical therapy. During the 14-year period following the first human implant in 1958, an estimated 120,000 units were implanted with 60% of patients being over 65 years old and with a 50-60% survival rate five years after implantation.¹ Permanent pacemakers are also utilized in dogs at various veterinary practices. The purpose of this paper is to review clinical indications for pacemakers in the dog as well as potential complications encountered with their use; to identify the types of units available and indicate how to select a given pacemaker for a given clinical syndrome; and to review the surgical technique for implantation along with follow-up surveillance.

In a 1979 survey of dogs with pacemakers, miniature schnauzers were by far the most common breed, comprising 26% of total cases.² Other breeds were evenly distributed with German shepherds, poodles, Boston terriers, Yorkshire terriers, and mix-breed dogs represented. The mean age was 8.5 years with a range from 3 to 15 years, and the male to female ratio was nearly equal (52% to 48%). Third degree heart block was diagnosed electrocardiographically in 55% of the dogs. Sick sinus syndrome was diagnosed in 26% of the dogs with electrocardiographic abnormalities including marked sinus arrhythmia or bradycardia and temporary sinus arrest or sinus exit block. The remaining 19% of the dogs had persistent atrial standstill. The primary presenting complaint was syncope which was

cited in 81% of cases. Nonspecific weakness was a complaint in 71% of the dogs, and 29% presented with ataxia.

INDICATIONS FOR CARDIAC PACING

Electrical cardiac pacing is most frequently indicated as a treatment for bradyarrhythmias that are accompanied by such clinical signs as syncope, weakness, and decreased exercise tolerance.^{3,4,5} Complete atrioventricular (AV) block, sick sinus syndrome, permanent atrial standstill, and persistent ventricular arrhythmias are the primary cardiac conditions in which pacemaker implantation is warranted;^{3,4,5} each will be discussed in detail. Acute myocardial infarction resulting in bradyarrhythmias due to conduction disturbances has also been advocated as an indication for cardiac pacing in man,⁵ although myocardial infarction is quite infrequent in the dog.⁶

Complete (third degree) heart block may be caused by fibrosis and degeneration of the atria or AV junctional tissue, congenital damage to the conduction system, natural or idiopathic myocardial trauma, vagal stimulation, hypoxia, inflammation or infectious processes such as bacterial endocarditis, toxemias, hyperkalemia, infiltrative cardiomyopathies due to neoplasia or amyloidosis, hypertrophic cardiomyopathy, or severe digitalis toxicity.^{4,5,7,8} Complete AV block, except when caused by digitalis toxicity, is almost always associated with syncope and is a definite indication for cardiac pacing.^{2,3,4,5,7} Syncopal episodes due to AV block have been referred to as Stokes-Adams syncope.⁶ With classical Stokes-Adams syncope, a permanent ventricular rate of 30 to 40 beats/min is controlled by a slow, relatively unstable idioventricular pacemaker that may be affected by electrolyte changes, hypoxia, or acidosis.^{8,9}

*Ms. Kimm is a fourth-year student in the College of Veterinary Medicine, Iowa State University.

**Dr. Hill is an associate professor of Veterinary Clinical Sciences at Iowa State University.

Syncope may then result due to decreased cerebral arterial perfusion pressure when cerebral blood flow is interrupted for 6 to 8 seconds during an unstable ventricular rhythm.⁸ A second type of complete AV block occurs less commonly in dogs which is transient and alternates with either normal sinus rhythm or first or second degree heart block.⁸ During the transition between the two alternating rhythms, the latent pacemaker may fail to initiate stable ventricular contraction resulting again in episodes of syncope.⁸

Sick sinus syndrome (also called bradycardia-tachycardia syndrome or sinoatrial (SA) syncope) is a second major indication for permanent cardiac pacing in the dog. The etiology may involve disease of the SA nodal artery, infiltrative or inflammatory lesions around the SA node, or replacement of the SA node with fibrous tissue. It is suspected that a genetic mode of inheritance exists for the syndrome in female miniature schnauzers.^{4,10,11} Hamlin documented two forms of sick sinus syndrome in dogs, both of which may cause syncope.¹⁰ In the first form, the sinus node does not depolarize, and presumably due to coexisting disease of the AV junction and peripheral Purkinje system, no ectopic pacemaker develops to initiate an escape rhythm.^{4,9,10} The result is cardiac standstill, cerebral ischemia, and syncope.^{9,10} In the second form of sick sinus syndrome, sinus arrest occurs due to overdrive suppression by a burst of supraventricular tachycardia immediately preceding the arrest. After sudden cessation of the rapid atrial pacing by the tachycardia, the sinus node and junctional pacemaker remain suppressed and arrested for up to 20 seconds before an escape pacemaker arises.^{8,10} Once again, permanent cardiac pacing is the treatment of choice for symptomatic patients.^{2,3,4,5,7}

A third indication for pacemakers in the dog is persistent atrial standstill, or "silent atrium."⁴ Electrocardiographic characteristics include absence of P waves and a regular supraventricular escape rhythm that is usually less than 60 beats/min.^{4,7} Clinical signs may include syncope, weakness, and congestive heart failure, and if present, warrant electrical pacing.⁴

Fourth, life threatening ventricular arrhythmias that are refractory to medical therapy may often be controlled by overdrive electrical pacing.^{12,13,14,15,16} Increasing heart

rate decreases refractory period, diminishes heterogeneity in repolarization of adjoining fibers, and increases the threshold for fibrillation; as a result, suppression of automaticity occurs and the frequency of ectopic ventricular beats decreases.¹² Ectopic beats accompanying bradycardia due to advanced degrees of heart block,¹³ Stokes-Adams seizures due to ventricular tachycardia and heart block,¹⁴ reciprocating ventricular tachycardia,¹⁵ and recurrent ventricular fibrillation¹⁶ have been effectively controlled by overdrive cardiac pacemakers.

PACEMAKER UNITS: COMPONENTS, MODES, AND SELECTION

Permanent pacemakers consist of two major components: a pulse generator and an electrode wire that connects the pulse generator to the heart. The power source for the 100 to 200 gm pulse generator is usually a battery of 4 or 5 individual mercuric oxide zinc cells in series or series-parallel.¹ With the unipolar electrode system used in dogs, only one electrode, the cathode, contacts the heart. The other "indifferent" electrode is a large metal plate surrounding the pulse generator.¹ Pulse generators are nonallergenic, nontoxic, and noncarcinogenic, so they generally are well accepted by the body,¹ although tissue rejection has been documented in both man and dogs.²

There are currently over 30 types of pacemakers manufactured, with each being designed for a specific syndrome or combination of clinical signs.^{1,4,5} The various types fall generally into two categories: asynchronous (fixed-rate) and synchronous (non-competitive). Synchronous pacemakers are further subdivided into ventricular inhibited (demand) pacemakers and ventricular triggered (standby) pacemakers. A 3-letter code has been suggested to identify the mode of operation of a pulse generator with the first letter representing the chamber(s) paced (A = atrium, V = ventricle, and D = both), the second letter representing the chamber sensed, if any, (A = atrium, V = ventricle, and 0 = not applicable), and the third representing the mode of response (I = inhibited, T = triggered, and 0 = not applicable).¹ Using the code, VOO units pace the ventricles at a fixed rate without any sensing activity, while VVI units sense intrinsic R waves and inhibit a pacing spike for a short delay.

The earliest type of pacemaker developed

was the fixed-rate (asynchronous, competitive) pacemaker which stimulated the heart at a constant rate, regardless of inherent cardiac rhythm.^{1,11,17} The rate in adjustable asynchronous units may be reset by needle control potentiometers or magnetic or encoded impulses to increase or decrease rate, but the rate is insensitive to physiologic signals.^{1,17}

Fixed-rate pacemakers are most appropriately used in cases where the return to sinus rhythm is unlikely or where the ventricles do not receive any intrinsic supraventricular impulses. When improperly used in other conditions, competition between inherent and paced cardiac rhythms may present a danger if the paced stimulus falls within the "vulnerable period" of a preceding cycle and evokes a serious dysrhythmia such as ventricular fibrillation.^{9,10} When dealing with dogs that have intermittent tachycardia, as in certain cases of sick sinus syndrome, asynchronous pacemakers cannot be recommended. Instead, ventricular inhibited pacemakers should be utilized since the pacing circuitry in these units resets until after the dangerous vulnerable phase of the sensed beat.^{9,10} Although asynchronous pacemakers normally have a safety margin in that the energy delivered for pacing (5 to 10 joules) is only 10% of that required for fibrillation threshold, electrolyte imbalances or drug toxicities may negate this margin, increasing potential for serious dysrhythmia.^{1,17}

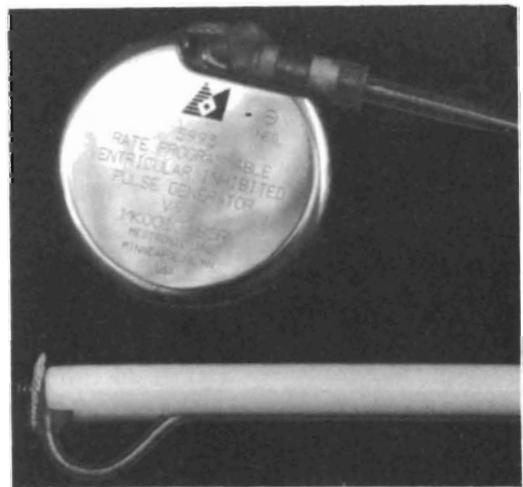
In animals with spontaneous cardiac activity, synchronous (noncompetitive) pacemakers are indicated because of their ability to detect intrinsic cardiac rhythm and alter their output accordingly.¹ In this way, potentially dangerous competition between endogenous and paced rhythms in cases of sick sinus syndrome with intermittent tachycardia or persistent ventricular arrhythmias is avoided. The output of a pacemaker operating in a noncompetitive mode is either inhibited or triggered by a sensed signal, which may be detected in either the atrium or ventricle depending on the position of the sensing electrode. Ventricular inhibited (VVI) units are the most commonly used type of demand pacemaker in dogs to this date.³ These units pace the heart only when the patient's ventricular rate drops below the preset rate of the unit; when the heart rate is above that level, the presence of an endogenous QRS complex with an amplitude of at least 0.5 to 2 mV sup-

presses the output of the pulse generator for 1 cycle.^{1,5} A more complex unit, the biofocal or AV sequential demand pacemaker (DVI), senses ventricular activity and sequentially paces the atrium and then the ventricle on demand.^{1,5} Although AV synchrony is restored by the DVI unit, the fact that both atrial and ventricular electrodes are utilized usually precludes its use even in human medicine since atrial electrodes are difficult to position and likely to dislodge.¹

The second category of synchronous pacemakers includes the ventricular triggered (standby) pacemaker (VVT). This type of unit avoids competitive rhythms by sensing an intrinsic QRS complex and immediately discharging into the refractory period of the heart.^{4,5,17} If no QRS complex is detected, the VVT unit discharges after its preset escape interval to assure a minimum ventricular rate.^{1,5,17} In contrast to the VVI pacemaker, the VVT pacemaker is not inhibited by extraneous electromagnetic interference.^{1,5}

SURGICAL IMPLANTATION

Preoperative evaluation of surgical candidates for permanent pacemaker implantation should include a thorough physical exam, complete blood count, serum biochemical profile, and thoracic and abdominal radiographs.² Yoshioka found that temporary cardiac pacing for 24 to 48 hours before surgery via a transvenous pacing electrode in the right ventricle and an external pulse generator was important both in stabilization



Pacemaker with myocardial electrode inserted in handle for implantation.

of the patient pre- and intra-operatively, and in determining the success of electrical pacing by monitoring clinical response and repeated electrocardiograms.²

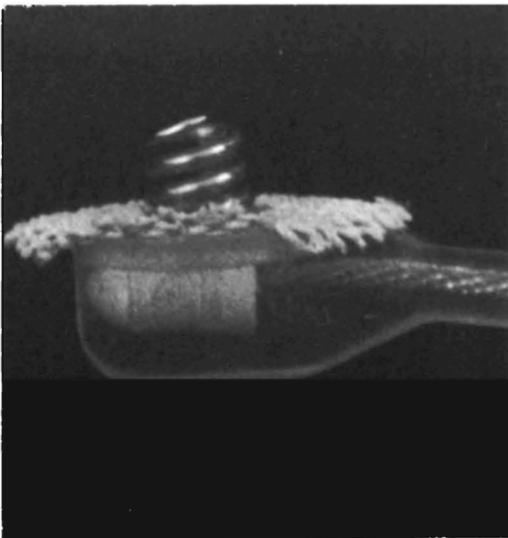
There are two basic methods in which electrical stimulation is applied to the heart.^{2,11} The electrode may either be inserted transvenously, via the external jugular vein, into the endocardial surface of the right ventricle,^{11,17} or it may be sutured or screwed into the epimyocardial surface of the left ventricle, necessitating a thoracotomy.^{2,4,11} The pulse generator is connected to the electrode and placed in a pocket between muscle layers, either in the left flank between abdominal obliques or dorsal to the scapula.^{2,11,17} In animals weighing 5 kg or less, the pulse generator may be too large to fit in these sites, so it can be placed in the cranial abdomen and allowed to sit unattached.²

The transvenous implantation technique has been described by Musselman, et al.¹⁷ No preoperative temporary pacemaker was available and general anesthesia was considered hazardous in the patient described, so the dog was heavily sedated. In this technique, the left jugular vein is isolated by blunt dissection, ligated distally, and a temporary ligature is placed around the proximal segment. A stab incision is made between the ligatures and the electrode is passed, with the aid of fluoroscopy,

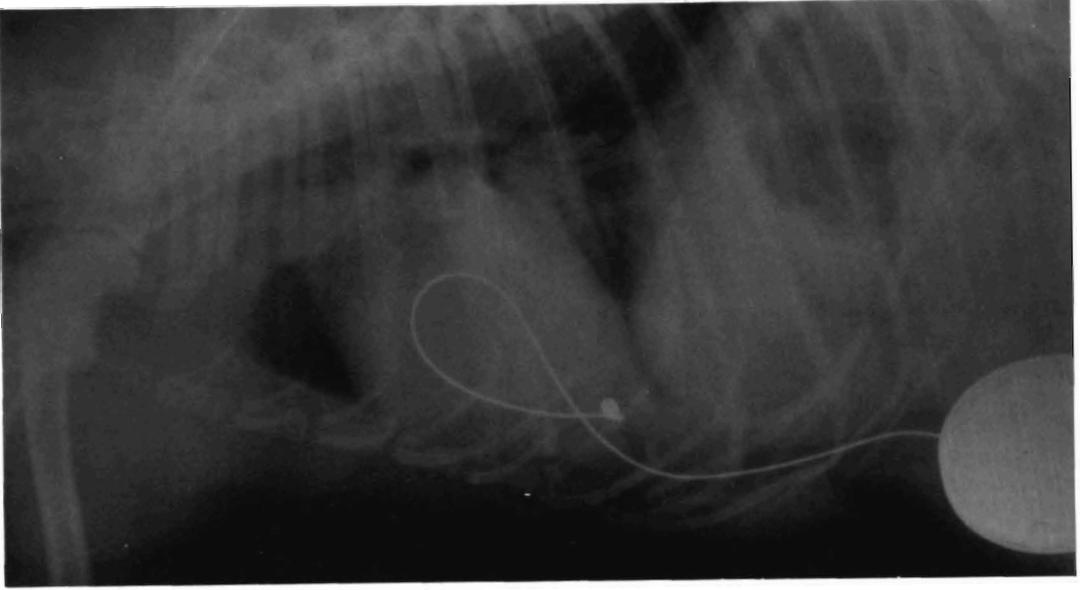
into the right ventricular apex and wedged under a trabeculum. Two nonabsorbable ligatures are then placed around the proximal jugular segment that contains the electrode. After making a subcutaneous pocket near the dorsal scapula and a subcutaneous tunnel to the proximal jugular segment, the pulse generator and electrode are connected and secured with nonabsorbable sutures. Finally, the pulse generator is secured with nonabsorbable sutures in its pocket, and the skin is closed routinely.¹⁷ Care must be taken to avoid excessive tension on the muscles and skin overlying the pulse generator, as pressure necrosis with secondary infection has led to fatal septicemia and chronic fistula formation necessitating removal of infected electrodes in man.^{2,11,18}

Although a transvenous electrode does not necessitate a thoracotomy, there are problems that make it less desirable than an epimyocardial lead. First, the incidence of dislodgment is greater with transvenous electrodes.¹⁹ The canine right ventricle has less trabecular network than the human heart so there is more difficulty wedging the electrode tip under trabeculae to capture good pacing contact.³ Also, the normal movement of quadrupeds increases the likelihood of dislodgment with endocardial leads.^{2,19} The second major problem with transvenous electrodes is that they may interfere with cardiac and pacemaker function due to consistent morphological changes they induce.^{18,19} Pathologic cardiac changes, which appear to be due to the presence of foreign material in the heart rather than external stimulation of the heart, include connective tissue sheath formation around the electrode, endocardial papillary thickening, interatrial septal changes, and myocardial damage. Histologically there is cartilaginous metaplasia in the endocardium and underlying myocardium with marked cellular proliferation.¹⁹ In addition to interfering with function, the entrapment of the electrode and catheter makes withdrawal dangerous to impossible after approximately 6 to 8 weeks due to potential complications such as right ventricular rupture or ventricular fibrillation.¹⁸ In the event of persistent infection or migration of transvenous electrodes, a thoracotomy is recommended for removal.¹⁸

For implantation of myocardial electrodes, entry into the thoracic cavity is made at the ventral third of the left fifth or sixth intercostal



Enlarged photo of the electrode of a myocardial implantation-type pacemaker.



Radiograph of implantation-type pacemaker in place.

space to expose the left ventricle.² A sutureless "corkscrew" electrode^a is available that is mounted on a special handle for rotation into the myocardium.^{2,4} The pericardium is incised 2 to 3 cm to visualize coronary vessels, and the electrode is screwed clockwise into the left ventricle avoiding the exposed vessels.² The electrode is held in place by fibrous attachments to a polyester netting above the electrode tip.⁴ Once implanted, the electrode is gently released from the handle and attached to the pulse generator through the seventh intercostal space with care that no tension is placed on the lead.² Alternatively, epimyocardial leads may be sutured to an avascular area of the left ventricle by 2 to 4 preplaced dacron polyester sutures approximately one-half way between the apex and base of the heart.²⁰

FOLLOW-UP SURVEILLANCE

Follow-up care is very important in pacemaker recipients to determine proper function of the pulse generator and electrode. In the immediate postoperative period, dogs are placed on a broad-spectrum antibiotic for 7 days, and an ECG is run every 12 hours for the first 48 hours.² Once stable, dogs may be discharged and kept initially in strict confinement at home.² Clients are shown how to take a pulse, and instructed to keep accurate daily records of pulse rate with weekly notes about appetite,

^aMedtronic sutureless myocardial pacing lead, model 6917, Medtronic, Inc., Minneapolis.

body weight, water consumption, and activity.^{2,3} At 2 weeks sutures are removed, and an ECG is run.² One month postoperatively thoracic radiographs and an ECG are taken to determine integrity and function of the pacemaker and to check for migration of lead or pulse generator components.² Thereafter, dogs should be examined electrocardiographically every 3 to 6 months to monitor function and anticipate battery exhaustion, and radiographs should be taken every 6 months.²

Caution must be taken for pacemaker recipients with regard to the use of electrosurgical instruments during subsequent operative procedures and the use of direct-current defibrillators.¹ Electrocautery or cutting instruments as well as their ground plates should be kept at least 2 inches from implanted pulse generators, and electrical currents should only be applied in bursts separated by intervals of several seconds.^{1,3} The instruments may otherwise inhibit VVI pulse generators.¹ In addition, although pulse generators are relatively resistant to direct-current defibrillation shock, they may be damaged by such currents.¹ It is therefore recommended that paddles be positioned as far from the pulse generator as possible.¹

PACEMAKER MALFUNCTION AND COMPLICATIONS

Pacemakers are often purchased from human hospitals or donated by manufacturers

after the shelf life has expired in order to keep costs practical for veterinary medicine.^{2,4} The most common cause of pacemaker failure is battery exhaustion.⁴ Therefore, it is essential to detect signs of impending failure to allow for elective pulse generator replacement. In general, standard output units have a 24 to 36 month life expectancy; however, improvements in battery cells are increasing longevity.¹ Specific end-of-life characteristics of an individual pulse generator should be obtained from the manufacturer.²

Indications of impending battery failure include a reduction in artifact voltage, change in the shape and contour of the artifact, and change in pacemaker rate.¹⁷ A declining pacemaker rate is universally accepted as evidence of battery end-of-life.¹ The sensing circuit of a synchronous pulse generator can be deactivated by application of an external magnet over the unit, thus converting it to an asynchronous ("magnet") rate.¹ This magnet rate is the most reliable parameter for detecting battery exhaustion in noncompetitive units.¹ The 15 to 30 seconds of competition produced by the magnet is not dangerous clinically.¹

In addition to replacement of the battery pack, other complications with implanted pacemakers may include migration of the pulse generator or electrode, wire breakage, electromagnetic interference (EMI), abscessation or infection, and postoperative arrhythmias.³ If a wire fracture is suspected, radiography with an overpenetrating technique may reveal the site, but more sophisticated systems such as electronic waveform analysis are usually necessary to confirm a break.¹ Although noncompetitive pacemakers with a sensing circuit may pick up electromagnetic interference from sources such as microwave ovens, small electric motors, and skeletal muscle potentials, these sources do not constitute a significant clinical problem due to shielding of pulse generators with titanium or stainless steel.¹ Postoperative arrhythmias encountered in pacemaker recipients are usually easily controlled with antiarrhythmic drugs and do not persist.³

REFERENCES

1. Parsonnet V, Furman S, Smyth N: Implantable cardiac pacemakers: Status report and resource guideline. *Am J Cardiol* 34:487-500, 1974.
2. Yoshioka MM, Tilley LP, Harvey HJ, Wayne ES, Lombard CW, Schollmeyer M: Permanent pacemaker implantation in the dog. *J Am Anim Hosp Assoc* 17:746-750, 1981.
3. Lombard CW, Tilley LP, Yoshioka MM: Pacemaker implantation in the dog: Survey and literature review. *J Am Anim Hosp Assoc* 17:751-758, 1981.
4. Tilley LP: *Essentials of Canine and Feline Electrocardiography*. St. Louis, CV Mosby Co, 1979.
5. Webb TJ, Clark DR, McCrady JD: Artificial cardiac pacemakers and some clinical indications for pacemaking. *Southwest Vet* 28:91-99, 1975.
6. Ettinger SJ, Suter PF: *Canine Cardiology*. WB Saunders Co, 1970, pp. 400-401.
7. Ettinger SJ: Cardiac arrhythmias, in Ettinger SJ (ed): *Textbook of Veterinary Internal Medicine*. Philadelphia, WB Saunders Co, 1983, pp. 980-1028.
8. Beckett SD, Branch CE, Robertson BT: Syncopal attacks and sudden deaths in dogs: Mechanisms and etiologies. *J Am Anim Hosp Assoc* 14:378-386, 1978.
9. Clark DR, Gross DR, Hobson HP, Humphries, JP, Knauer KW: Artificial pacemaker implantation for control of sinoatrial syncope in a miniature schnauzer. *Southwest Vet* 28:101-107, 1975.
10. Hamlin RL, Breznock EM, Smetzer DL: Sinoatrial syncope in miniature schnauzers. *JAVMA* 161:1022-1028, 1972.
11. Brown KK: Bradyarrhythmias and pacemaker therapy, in Kirk RW (ed): *Current Veterinary Therapy VI*. Philadelphia, WB Saunders Co, 1977, pp. 354-357.
12. Han J, Detraglia J, Millet D, et al.: Incidence of ectopic beats as a function of basic rate in the ventricle. *Am Heart J* 72:632-639, 1966.
13. Zoll PM, Linenthal AJ, Zarsky LRN: Ventricular fibrillation: Treatment and prevention by external electric currents. *New Engl J Med* 262:105-112, 1960.
14. Dressler W: Observations in patients with implanted pacemaker, III: Frequency of ventricular tachycardia as a cause of Adams-Stokes attacks and rate of pacing required for its prevention. *Am Heart J* 68:19-24, 1964.
15. Sowton E, Leatham A, Carson P: The suppression of arrhythmias by artificial pacemaking. *Lancet* 2:1098-1100, 1964.
16. Lown B, Kosowsky BD: Artificial cardiac pacemakers, part II. *New Engl J Med* 283:971-977, 1970.
17. Musselman EE, Parker AJ, Rouse GP: Permanent pacemaker implantation with transvenous electrode placement in a dog with complete atrioventricular heart block, congestive heart failure and Stokes-Adams syncope. *J Small Anim Pract* 17:149-162, 1976.
18. Rettig G, Doenecke P, Sen S, Volkmer I, Bette L: Complications with retained transvenous pacemaker electrodes. *Am Heart J* 98:587-594, 1979.
19. Fishbein MC, Tan KS, Beazell JW, Schulman JH, Criley JM, Hirose FM: Cardiac pathology of transvenous pacemakers in dogs. *Am Heart J* 93:73-81, 1977.
20. Buchanan JW, Dear MG, Pyle RL: Medical and pacemaker therapy of complete heart block and congestive heart failure in a dog. *JAVMA* 152:1099-1109, 1968.