Implantation of Permanent Transvenous Endocardial Pacemaker in a Dog with Atrioventricular Block

Masayuki KOBAYASHI¹⁾, Katsuichiro HOSHI¹⁾, Hidehiro HIRAO¹⁾, Miki SHIMIZU¹⁾, Shunsuke SHIMAMURA¹⁾, Midori AKIYAMA¹⁾, Ryou TANAKA¹⁾, Kohji MARUO¹⁾ and Yoshihisa YAMANE¹⁾

¹⁾Department of Veterinary Surgery, Faculty of Agriculture, Tokyo University of Agriculture and Technology, 3-5-8 Saiwai-Cho, Fuchu, Tokyo 183-8509, Japan

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ABSTRACT. A 10-year old male mongrel dog was presented to the University Veterinary Teaching Hospital with a two-month history of episodic syncope. Twenty-four hr Holter electrocardiographic (ECG) recording revealed frequent episodes of advanced atrioventricular block with long periods of ventricular asystole. The cause of syncope was determined to be Adams-Stokes syndrome exhibited bradyarrhythmia. After the animal failed to respond to medical therapy, permanent transvenous pacemaker implantation was performed. Postoperative Holter ECG showed 100 beat per min programmed pacemaker rhythm, which indicated successful capture of the artificial pacing. The dog recovered smoothly from the operation and syncopal episodes completely disappeared. Six months after the surgery, no complications were observed and the dog's quality of life has dramatically improved.

KEY WORDS: atrioventricular block, canine, pacemaker.

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Implantation of artificial cardiac pacemaker is commonly used in humans for the treatment with bradyarrhythmias. In small animals, as well as in humans, pacemaker therapy has been used for the naturally occurring bradyarrhythmias, including sick sinus syndrome, persistent atrial standstill, and complete or advanced second-degree atrioventricular (AV) block [5, 6]. Although reports in humans are quite extensive of pacemaker therapy, there are limited studies on the use of pacemakers in small animal practice. Thus, the indications, surgical techniques, complications and long-term outcome of pacemaker therapy in small animals have not been clearly defined. This report describes a case of pacemaker therapy in a dog with symptomatic advanced AV block.

A 10-year old male mongrel dog weighing 17.0 kg was referred to the Veterinary Teaching Hospital of Tokyo University of Agriculture and Technology with a primary complaint of frequent episodes of syncope. The syncopal episodes were first seen about two months before presentation, which were increasing in frequency in spite of medical treatment with atropine and dexamethasone by a previous veterinarian. Upon presentation, the dog appeared in good health and leaned towards obesity. Findings upon neurologic examination were normal. Femoral pulses were of normal intensity but extremely irregular and the rate was below 50 beats per min (bpm). Auscultation of the heart and thoracic area did not reveal any apparent abnormalities. The dog showed sudden onset of syncope many times during the conduct of physical examination. These episodes were characterized by blinking and head shaking followed by transient loss of consciousness and collapse, sometimes accompanied by urinary incontinence, and then full recovery within 30 sec. Electrocardiography (ECG) revealed second-degree AV block which varied from 2:1 to more than

3:1 (high-grade) AV conduction (Fig. 1). The ventricular asystole persisted approximately 10 sec, and then recovered to normal AV conduction. During these periods of ventricular arrest, progressive shortening of P-P interval occurred and P wave rate exceeded 180 bpm. These periods of ventricular arrest coincided with the syncopal episodes observed during ECG monitoring. Hematologic and serum biochemical analyses revealed the elevation of hepatic enzymes [alanine aminotransferase (ALT); 560 U/l, aspartate aminotransferase (AST); 122 U/l]. Thoracic radiography showed slightly enlarged heart size [Cardiothoracic ratio (CTR); 58%, Vertebral heart score (VHS); 11.1V], hypervascularity of lung field, and distention of caudal vena cava which suggested circulatory congestion. Echocardiographic findings revealed mild dilatation of the right side of the heart and tricuspid regurgitation, which also suggested bradycardia-induced right-sided heart congestion. A 24-hr Holter ECG (Quick Corder QR2100, Fukuda M-E Kogyo Inc., Tokyo, Japan) showed high-grade AV block leading to long periods of ventricular asystole (Fig. 2). One of the periods of ventricular asystole reached 13 sec in this figure. There were more than 100 times of ventricular arrest which lasted for over five sec during a 24-hr period. The longest duration of ventricular asystole was 20 sec. The mean and minimum heart rate was 64 bpm and 20 bpm, respectively.

Fig. 1. Lead II, 50 mm/sec, Electrocardiography (ECG) recording on the day of first presentation showed second-degree atrioventricular block followed by long period of ventricular asystole.

Fig. 2. Twenty-four hr Holter ECG recording before pacemaker implantation revealed advanced atrioventricular block associated with long period of ventricular asystole. The longest period of ventricular asystole in this figure was 13 sec (arrow).

Based on the above findings, a diagnosis of Adams-Stokes syndrome resulted from advanced (high-grade, second-degree) AV block was made. The cause of this arrhythmia was presumed to be idiopathic primary disorder of AV node or His bundles because no underlying cardiac disorder was observed through detailed examinations. Medical treatment using isoproterenol (15 mg/head, orally, every 8 hr) had no effect. The syncopes progressively increased to reach more than 100 times per day. Two weeks after the first admission, pacemaker implantation was performed.

The dog was premedicated with atropine (0.04 mg/kg, subcutaneously), acepromazine (0.5 mg/kg, intramuscularly), and butorphanol (0.1 mg/kg, intramuscularly). Anesthesia was induced through administration of thiamylal sodium (5 mg/kg, intravenously) and maintained with isoflurane and oxygen. Heparin sodium (100 U/kg, intravenously) was given for the intravascular procedure. The dog was placed in right lateral recumbency and the left jugular vein was incised. A bipolar, steroid-eluting, tined endocardial lead (width; 6 Fr, length; 58 cm, Cupsure SP 5054, Medtronic Inc., Minneapolis, U.S.A.) was inserted transvenously through the left jugular vein and advanced to right ventricle under a fluoroscopic guidance. The tip of the pacing lead was positioned at the trabecular muscles in the apex of the right ventricle. The lead was fixed by 1-0 silk suture at the point of the insertion into the jugular vein after ensuring successful lead capture (Table 1). Subsequently, a subcutaneous small pocket in left side of the neck was made and a pulse generator (weight; 27.2 g, SIGMA SD303, Medtronic Inc., Minneapolis, U.S.A.) was implanted. The generator was connected to the pacing lead exiting from the subcutaneous tunnel at the neck. The subcutaneous tissue and skin were closed in a routine manner and the pulse generator was programmed adequately (Table 2). The pacing rate at 100 bpm was determined empirically by the dog's body size.

Table 1. Lead implant data

Polarity	Bipolar
Pulse width	0.5 ms
Pacing threshold	0.2 V 0.3 mA
Lead impedance	834 ohms
Sensing threshold	R-wave 25 mV

Table 2. Pacemaker program parameters

Pacing mode	VVI
Pacing rate	100 bpm
Amplitude	4.0 V
Pulse width	0.4 ms
Sensitivity	2.8 mV
Pace polarity	Bipolar
Sense polarity	Bipolar
Ventricular refractory	230 ms

a) VVI represents fixed rate, single-chamber, ventricular-inhibited pacing mode.

The dog was recovered from the operation and postoperative monitor ECG showed that pacing beats were started after the animal's sinus rhythm fell below 100 bpm, thus, the heart rate was appropriately maintained above 100 bpm without evidence of AV block. The pacing spikes preceding each ventricular beat indicated good capture of the pacing lead and accurate functioning of the pulse generator (Fig. 3). After implantation, the dog showed no clinical abnormalities and the syncopes completely disappeared. The dog was discharged from the hospital five days after surgery. When the dog was re-evaluated three weeks postoperatively, the heart rate was observed to be maintain at 100 bpm and pacing malfunction was not detected by monitor and Holter ECG. The tip of the pacing lead was well positioned at the apex of the right ventricle as seen through chest radiography (Fig. 4), and heart enlargement was reduced (CTR; from 58% to 53%, VHS; from 11.1 V to 10.3 V). Moreover,

Fig. 3. Lead II, 50 mm/sec, ECG recording at three weeks after the implantation. Note that the pacing spikes (arrow) preceding ventricular beats which indicated good function of the pacemaker system.

Fig. 4. Lateral radiographic findings at three weeks after the implantation. The tip of pacing lead well positioned in the apex of the right ventricle. The pulse generator was situated in the neck.

Echocardiographic findings did not show dilatation of the right side of the heart and tricuspid regurgitation. Serum biochemical examination revealed that the values of hepatic enzymes returned to normal range (ALT; from 560 U/l to 32 U/l, AST; from 122 U/l to 18 U/l). These results indicated that the artificial pacing improved the bradycardia-induced circulatory congestion. Mild seroma formation within the implanted site of the pulse generator was found several days after the operation, but disappeared clearly at that time. Six months after implantation, the dog was brought to the hospital again for evaluation of the pacemaker function. During the same periods, the dog's quality of life improved dramatically due to complete loss of syncope, and the owner was very satisfied with this treatment. Upon examination, the pacemaker was found to be completely functioning and the output voltage was reprogrammed from 4.0 V to 2.5 V in order to extend the life span of generator battery. The estimated battery life span is up to ten years and the pacemaker should be checked every six months.

Although pacemaker therapy in small animal medicine has been increasing since 1968 in which first reported for a dog with complete AV block [1], it still has not gained wide acceptance because of the poor results obtained, the need for specialized surgeons, and the high cost of pacemaker equipment.

Surgical techniques for permanent pacemaker implantation were reported previously as epicardial [1, 4] or endocardial [5, 6] implantation, each having distinct advantages and disadvantages. Endocardial implantation through the jugular vein has been more commonly used because it is a less invasive procedure, can be performed more rapidly, and results in lesser complications than epicardial implantation which requires a thoracotomy or celiotomy [5].

Complications associated with pacemaker implantation occurred in 55% of the cases in a study of 154 dogs [5]. Major complications considered lifethreatening comprised 33% of the cases and include lead dislodgement, generator failure, cardiac arrest during surgery, and infection. Minor complications occurred in 31% of the cases include seroma formation, muscle twitching, and inconsequential arrhythmias. In the present case, no major complication was observed in the dog for six months after surgery, and only mild seroma at the implanted site was found as a minor complication. Lead dislodgment from the endocardial wall is the most frequently occurring major complication in endocardial implantation procedure [5]. To avoid this problem, screw-in type pacing lead should be selected or the procedure implanting the generator in the lateral thoracic wall via costocervical vein approach should be used [3], especially if large breed of dog with excessive movement of the neck. In this case, creating enough slack for the pacing lead in the cervical pocket can minimize tension and dislodgement of the lead.

Although single-chamber pacing mode (VVI) is commonly selected for pacemaker therapy in dogs, as well as in this case, the use of this mode might result in a so-called "pacemaker syndrome" due to low cardiac output induced by mismatch AV synchrony [5]. Thus, if a more physiological pacing is required, dual-chamber pacing mode (DDD) should be selected which has normal AV synchronous pacemaker systems. However, DDD requires the use of dual pacing leads which might lead to more complications. In a dog with complete AV block, it was observed that the use of single pass lead atrial synchronous ventricular pacing (VDD) resulted in greater improvement in cardiac output and function than VVI pacing [2]. In the present case, the use of VVI pacing resulted in satisfactory resolution of the clinical signs and improvement of the quality of life of the animal. However, further studies are needed to evaluate the indications for pacing mode in dogs.

For long-term outcome, it was reported that the prognosis in using pacemaker implantation depended mainly on the presence or absence of congestive heart failure before implantation; 60% and 25% of the dogs with and without heart failure, respectively, one year after surgery [5]. In the present case, although several signs of bradycardia-induced mild circulatory congestion was detected before the surgery, the pacemaker implantation before the development of congestive heart failure is expected to result in a good prognosis.

In conclusion, implantation of pacemaker in the dog resulted in dramatical improvement of clinical signs and a

very satisfied owner. It is suggested that this procedure be used more widely in dogs with symptomatic bradyarrhythmias.

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