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Short Communication

Transvenous Pacemaker Implantation in a Schnauzer with Sick Sinus Syndrome

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Abstract

A fourteen-year-old female neutered Schnauzer was presented to the hospital with multiple syncopal episodes for two weeks. The dog appeared fatigue and sleepy but remained responsive. Clinical examination revealed bradycardia and irregular pulse. Electrocardiography showed sinus rhythm with repetitively long episodes of sinus arrest over 3 seconds, indicating sick sinus syndrome. The dog eventually underwent a transvenous single chamber permanent pacemaker. She was followed up for a month after surgery without recurrent syncope and irregular heart rate.

Keywords: dogs, heart disease, pacemaker, sick sinus syndrome, syncope

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Introduction

Sick sinus syndrome is characterized by impaired electrical impulse generation or cardiac conduction system (Burrage, 2012). An affected sinus fails to discharge and resume regular rhythmicity. Electrocardiogram (ECG) usually indicates a presence of tachycardia or regular sinus rhythms followed by bradycardia and ultimate sinus arrest. The alternation between bradyarrhythmias and tachyarrhythmias of the heart rate in affected dogs is known as "bardy-tachy syndrome" (Bulmer, 2011). During sinus arrest, the animal may experience dizziness and syncopal episodes due to a sudden drop in blood pressure. Predisposing breeds include West Highland White terriers, Miniature Schnauzer, and Cocker spaniels (Bulmer, 2011). An occurrence of sick sinus syndrome was frequently found in certain breeds, suggesting possible genetic involvement of the disease. Recent recovery of a recessive mutation of cardiac sodium channels was linked to a development of sick sinus syndrome in babies and children (Benson et al., 2003).

Implantation of electrical stimulator for the heart provides a long term solution especially for dogs with symptomatic bradycardia (Mac Phail, 2013). Pacemakers can be single-chambered, that can set ventricular rhythm or dual-chambered, that can set both atrium and ventricular rhythms (Kienle, 1998). Transvenous pacemaker is less invasive compared to epicardial pacemaker (Goodwin, 2001) and is the most common route for pacemaker implantation (Mac Phail, 2013). Moreover, application of a rate-adjusted single-chamber pacemaker offers good solution for treatment of cardiac bradyarrhythmias especially in small size dogs. The aim of the present study was to provide a clinical approach for diagnosis and treatment of sick sinus syndrome in dogs. Pathophysiology of sick sinus syndrome was also discussed.

Materials and Methods

Case history and clinical examination

A neutered female Schanauzer, 14 years old, was presented at the hospital with a 2-week history of multiple syncopal episodes. Physical examination revealed that the dog was weak and sleepy but she remained responsive. Capillary refill time was less than 2 sec. Bradyarrhytmia with irregular pulse was detected on clinical examination. No significant change in respiratory sound was observed during clinical auscultation. An average systolic blood pressure was 115 mmHg using doppler flow meter (Model 811B, Parks Medical Electronics Inc, USA).

Diagnostic procedures

Radiography: Radiographic findings indicated that the Vertebral Heart Score was 11. An increased sternal contact of the cardiac sillihouette together with a rounding of the right ventricle were observed, suggesting the presence of right-sided cardiomegaly. The pleural space and the lung fields were clear.

Electrocardiogram: Three-lead (Lead I, II, and III) electrocardiogram (ECG; Electrocardiograph Sanborn Series, Philips Medical Systems, USA) was performed on the dog. In addition, ECG revealed a sinus rhythm followed by sinus arrest (at least 5 sec). Three consecutive ECG records disclosed heart rate of 36, 54, and 78 bpm, respectively, suggesting the presence of sick sinus syndrome (Fig 2A). The ECG records during the syncopal episode for 2 minutes revealed that the sinus pauses occurred twice per minute and each pause lasted for 5 or 6 sec.

Atropine response test: To rule out the influence of vagal stimulation on the heart rate, an atropine (ANB Lab, Co Ltd, Thailand) response test (0.02 mg/kg IV) was conducted. The examination showed that atropine failed to increase the heart rate of the patient (56 bpm before and 58 bpm after atropine administrations).

Hematology and serum biochemistry: Hematological findings were unremarkable (Table 1). Serum biochemistry revealed significant increase in hepatic enzymes, both SGPT and SGOT, together with high serum triglyceride and elevated alkaline phosphatase. Urea, creatinine, and electrolytes (Na^+, K^+ and Ca^{+2}) were within normal limits (Table 2).

Treatment

Prior to the initiation of treatment, the dog was given intravenous fluid (5% dextrose in 0.85% saline) at the rate of 5 ml/kg/hr. After 15 minutes of preoxygenation, the dog was induced with intravenous injection of midazolam (0.3 mg/kg IV, Hexal®; Jenahexal Pharma GmbH, Gena, Germany) and fentanyl (8.6 µg/kg IV, Fenilhm®; Hameln Pharmaceuticals GmbH, Hameln, Germany). Prophylactic antibiotics (20 mg/kg cephazolin IV, Cefaben®; LBS Lab Ltd, Bangkok, Thailand) was also administered. After tracheal intubation, the dog was anesthetized with isoflurane (AttaneTM; Piramal Critical Care Inc, Bethlehem, PA, USA) in oxygen. A transvenous pacing lead was introduced into the right ventricle close to the ventricular septum as shown in the x-ray (Fig 3A and 3B). A pacing lead was inserted via the right jugular vein under the guidance of fluoroscope (Fig 4A). The pacing lead was connected to an external temporary pacemaker to identify an electrical response of the heart. The output voltage from electrical generator was set at 3.5 mV and 0.4 msec. The permanent pacing generator was, then, attached and placed cranial to the right shoulder (Fig 4B). The rate was pre-programmed to have a minimum heart rate of 70 bpm with rate-responsive settings that could pace the heart up to 170 bpm. The subcutaneous fat and skin were closed routinely with 3-0 Polydioxanone (PDS*II, Johnson & Johnson Intl, Belgium) and 3-0 polyamide monofilament (Dafilon, B. Braun Surgical SA, Rubi, Spain), respectively. External wound was dressed and soft bandage were applied around the patient's neck. Surgical operation (Fig 4A and 4B) was at least 40 minutes.

Postoperative care

After the operation, the patient recovered well without post-operative complications. The dog was monitored continuously for further 1.30 h. She was able to stand by herself while other vital signs were within normal limits. With the request of the owner, the dog returned home and a telephone consultation with a veterinarian was applied. A one-week course of antibiotic (20 mg/kg cephalexin BID PO, Sporicef®; M&H Manufacturing Co Ltd, Bangkok, Thailand) and anti-inflammatory drug, 5 mg serratiopeptidase (1 tab PO BID, Unizen®; Unison Laboratories Co Ltd, Chachoengsao, Thailand) were prescribed. Multiple hepatotonic drug (1 tab sid, Lipochol®; Interthai Pharmaceutical Manufacturing Ltd, Bangkok, Thailand), Coenzyme Q10 10 mg (2 tabs PO BID, Decaquinon®; Eisai Co Ltd, Tokyo, Japan), Folic acid 5 mg (0.5 tab PO SID, Folivit®; Siam Bheasach Co Ltd, Bangkok, Thailand), and B1,6,12 (1 tabs PO BID, Vita1+6+12®, Newlife Pharm Co Ltd, Bangkok, Thailand) 1 tab, bid were prescribed.

Results and Discussion

On the first day after operation, the dog was slightly weak but was able to stand and response. The average systolic blood pressure from a Doppler flow device was 130 mmHg (120, 130 and 140 mmHg). The ECG revealed ventricular paced rhythm working together with sinus rhythm (Fig 2B) with a heart rate of 71.3 bpm (69, 81 and 64 bpm). A telemetry checkup for the pacemaker indicated 67.4% paced rhythm and 32.6% sensed rhythm (Fig 5). Parasternal echocardiography exhibited a thickening of the interventricular septum; the ventricular ejection fraction was around 70% (Fig 6). No remarkable change on the right atrium was noticed. On the 7th day of post-operation, the average heart rate was 75.6 bpm (71, 77, and 79 bpm). The average systolic blood pressure was 127 mmHg (120, 130, and 130 mmHg).

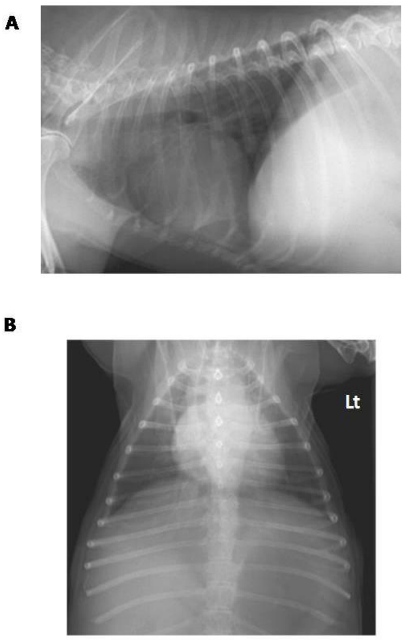

Figure 1 Right lateral (A) and dorso-ventral (B) thoracic radiographic findings revealed an increase in sternal contact of the cardiac sillihouette and rounding of the right ventricle with clear lung fields.

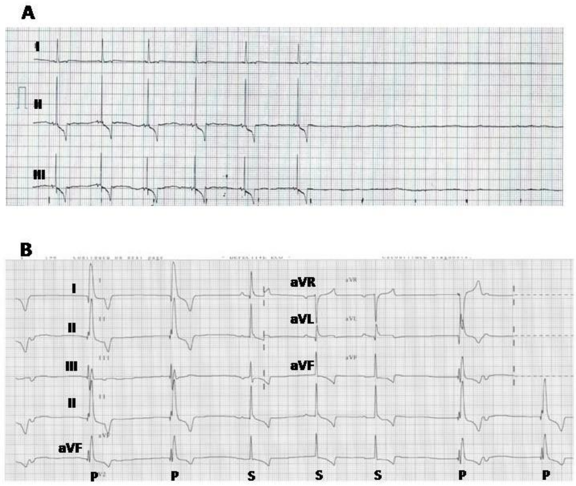

Figure 2 Pre- and post-operative electrocardiography (ECG). A: Three-lead (Lead I, II and III) ECGs showed a sinus rhythm followed by a period of sinus arrest (at least 5 sec/arrest). (paper speed : 50 mm/sec; 1 cm : 1 mV), B: ECGs (Lead I, II, III, aVF, aVR, and aVL) showing ventricular paced rhythms working together with normal sinus rhythm on the 1st day post-operation. Electrical generator provided paced impulse that stimulated the ventricle. The impulse from the generator was inhibited when a natural heartbeat was sensed. S: sensed heartbeat, P: paced heartbeat (50 mm/sec; 1 cm: 1 mV)

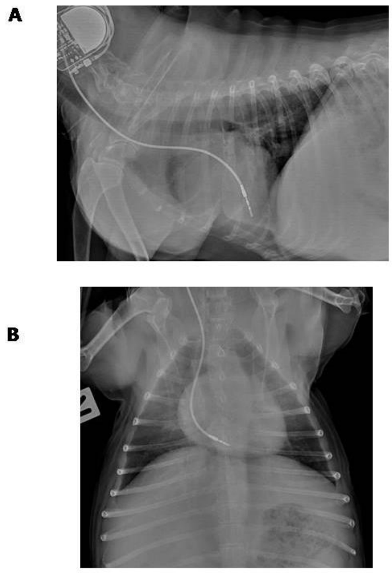

Figure 3 Right lateral (A) and dorso-ventral (B) thoracic radiographic findings revealing the position of the pacemaker lead in the right ventricle. S-bend of the lead helped prevent the strain on the lead during neck movement.

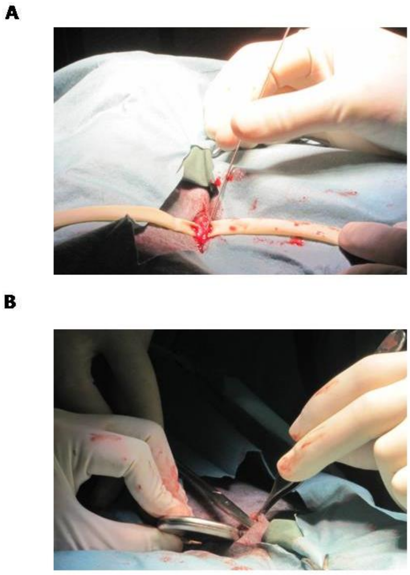

Figure 4 Intraoperative picture during pacemaker implantation. A: A transvenous pacing lead was advancing to the right ventricle through the right jugular vein. B: A permanent pacing generator was inserted underneath the skin at the prescapular area.

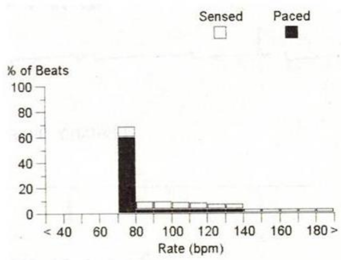

Figure 5 A ventricular long term histogram of electrical stimulation by the pacemaker recorded on the 1st day after operation using a telemetry device

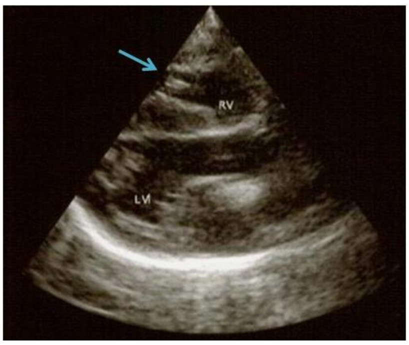

Figure 6 Right parasternal long-axis view (b mode) showing the thickening of the interventricular septum. Transvenous electrode was presented in the right ventricle (an arrow). (LV: left ventricle; RV: right ventricle)

A telemetry checkup for the pacemaker indicated 65.5% paced rhythm and 34.5% sensed rhythm (data not shown). Minimal voltage output that could pace the heart was 0.7 mV. The output voltage from electrical generator was then set back to the original number at 3.5 mV and 0.4 msec. Two-week post-operation, three consecutive ECGs displayed quite regular rhythm with the average heart rate of 76 bpm (71, 77, and 80 bpm) with less than 10% variation (data not shown). An average systolic blood pressure was 127 mmHg (120, 130, and 130 mmHg). The surgical wound was completely healed and stitches were removed after the checkup. The owner was interviewed by telephone at one month after operation. The dog was able to regain regular activities without syncopal episodes and the owner was highly satisfied with the results.

Sick sinus syndrome is a generalized disease affecting the cardiac conduction system, hampering the regular firing rhythm of the sinus. The bradyarrhythmic signs of sick sinus syndrome must be differentiated from condition known as sinus arrest, sinoatrial block and third degree atrioventricular block. The ECG of sick sinus syndrome often shows sinus bradycardia and/or sinus tachycardia with periods of sinus arrest (Burrage, 2012) thus can be differentiated from sinus arrest (Selk Ghaffari et al., 2009) and atrioventricular block (Mak and Allen, 2013). A sinoatrial block is a condition sometimes confused with sinus arrest. In sinoatrial block the pacing impluse is generated but fails to conduct on the way to the atria. A definitive diagnosis of sinoatrial block requires an electrophysiology study to detect electrical impulses from the SA node. Nevertheless, sinus arrest in sick sinus syndrome is not a multiple of normal PP interval and is associated with a long period of sinus pause (> 3 sec), different from a condition known as sinoatrial block. In third degree atrioventricular blocks, the p wave had different bead rates compared to the QRS wave; both waves were not coordinated (Goodwin, 2001; Mak and Allen, 2013). Furthermore, ventricular escape complexes commonly found in dogs with AV blocks are not present in dogs with sick sinus syndrome (Goodwin, 2001).

Animals presented with a history of syncopal episodes are often connected with excitement or exertion that raises oxygen demand and cardiac output (Parent, 2007). Diagnosis of the underlying cause of syncopal episodes may be challenging and requires an amulgamation of information on patient history, physical examination, biochemical profile, radiography, ECK and echocardiography. It was found that cardiovascular dysfunction, neurological disorders, and metabolic abnormality were the major causes of syncopal episodes (Davidow et al., 2001). In the present study, the dog presented with bradyarrythmia and the abnormal ECG findings could further confirmed the presence of cardiovascular dysfunction. A mild elevation of hepatic enzymes with normal serum electrolytes was remarked. The alkaline phosphatase was extremely high (2,000 U/l), and this was likely related to the presence of intrahepatic cysts in this dog (data not shown). Serum cortisol elevated slightly with low urine cortisol to creatinine ratio (UCCR = 0.0008) which was likely due to chronic stress. Taken together, it was unlikely that hepatic disease play role in the syncopal episodes. Neurological problems resulting in syncopal episodes usually affect brain functions. Behavioral changes and alteration of neurological responses related to abnormal brain functions (Davidow et al., 2001) were not observed in the present case. Electrolyte imbalance leading to

Table 1 Complete blood count before and after pacemaker implantation

	Pre-operation	2-wk post-operation	Normal values
Total Protein (Biuret)	6.5	6.6	5.3-7.8 g/dl
Albumin	3.6	3.2	2.3-3.2 g/dl
Globulin	2.9	3.4	1.5-3.9 g/dl
BUN	24	19	10-26 mg%
Creatinine	1.02	0.8	0.5-1.3 mg%
SGPT	230	450	6-70 U/l
SGOT	44	-	10-43 U/l
Alkaline Phosphatase	2,000	3,772	8-76 U/l
Cortisol	4.63	-	0.5-4 µg/l
Cholesterol	245	322	82-218 mg%
Triglyceride	229	379	21-87 mg%
FreeT$_4$	1.24	-	1-2 ng/dl
Ammonia	-	105	45-120 µmol/l
Na$^+$	151	-	138-152 mEq/l
K$^+$	4.74	-	3.5-5.1 mEq/l
Ca^{+2}	10.1	-	9.8-12.0 mg%
Platelets	306	461	200-500 x 10^3 / mm^3
Blood parasite	Negative	Negative	Negative

Table 2 Pre-operative and post-operative blood chemistry profiles of the dog underwent pacemaker

	Pre-operation	2-wk post-operation	Normal values
Hb	12.1	13.8	10.0-18.0 g%
RBC count	5.35	6.12	5.0-9.0 x 10^3/l
Hct	37.4	40.1	35-55 %
MCV	69.91	65.52	60-77 fl
MCHC	32.35	34.41	32-36 g/dl
WBC count	7.17	5.97	6-17 x 10^3/mm^3
Neutrophils	5,807	4,657	3,000-12,500 / mm^3
Lymphocytes	789	657	1,000-4,800 / mm^3
Monocytes	502	478	150-1,350 / mm^3
Eosinophils	72	179	100-750 / mm^3

muscular weakness (Parent, 2007) was unlikely because major electrolytes were found within normal limit (Table 2). Bradycardia was not improved by the application of atropine, therefore the underlying cause of bradycardia was independent from vagal stimulation. The occurrence of abnormal ECG readings together with other clinical findings lead to aconclusion that an abnormality of electrical pulse generation was the underlying culprit of the syncopal episodes in this dog.

Various cardiac arrthythmias that could be treated with pacemaker therapy included atrioventricular block, persistent atrial standstill, and sick sinus syndrome (Monet, 2003). The implantation of pacemaker helps improve cardiac output and decrease the risk of sudden death due to cardiac arrhythmias by maintaining adequate heart rate. In the present study, the transvenous single-chambered pacemaker was used. The pacemaker was composed of a pulse generator and a pacing lead. The pacing lead sensed signals from ventricle and transduced electrical pulse in the absence of heart rhythm lower than 70 bpm. The device was also equipped with a Life-adaptive program that accelerated pulse rate (up to 170 bpm) when the activity of the heart increased. It, thus, provided enough cardiac output according to the patient's activity. The pacing lead is slightly bent to avoid the stress on the pacing lead especially during e neck movement.

Applications of dual-chambered pacemaker have been done in dogs from various studies (Burrage, 2012; Genovese et al., 2013). The dual-chambered pacemaker provids separate pulse generation on the right atrium and the right ventricle. When the heart rate drops below a set-point, the pacing lead will provide signal to the atrium first. If the signal fails to be transmitted to the ventricle, the ventricular pacing lead will generate an electrical pulse to the ventricle. Dual-chambered pacing is more physiologic than single-chambered pacing by providing better left ventricular function (Maisenbacher III et al., 2009). The application of dual-chambered pacing also helps reduce pacemaker syndrome (Burrage, 2012). Nevertheless, size limitations canine patient precludes the use of dual-chambered pacemaker. Furthermore, the insertion of pacing lead on the right atrium could lead into a rupture of the right atrium, resulting in serious complications especially in small breeds of dogs. The application of the single- chambered pacemaker on a senior dog in the present study provided a practical solution, nevertheless, the pacemaker syndrome from asynchronus contraction of atrium and ventricle could develop. In dog, the development of pacemaker syndrome is unlikely due to its shorter lifespan than humans.

According to Canpacers study (Oyama et al., 2001), major complications due to pacemaker implantation included lead dislodgement (6%), generator failure (6%), cardiac arrest during implantation (6%), and infection (5%). Minor complications included seroma formation (12%), muscle twitch (11%), and inconsequential arrhythmias (11%). Magnetic resonance imaging could interfere with the pacemaker device, thus, owners should be informed to avoid accident from device failure. Occurrence of battery failure is uncommon in animals. The electrical generator used in the present study has a long lasting life (5-7 years depending on the activity of the generator), thus replacement of a new battery was unlikely due to the old age of the patient.

In conclusion, we reported a successful application of a transvenous pacemaker in a Miniature Schnauzer affected by sick sinus syndrome. The dog recovered well from the operation and regained the regular activities without experiencing syncopal episodes. The patient resumed its normal activity at monthly follow-ups.

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บทคัดย่อ

การใส่เครื่องกระตุ้นหัวใจผ่านทางหลอดเลือดดำในสุนัขพันธุ์ชเนาเซอร์ที่ป่วยด้วยภาวะซิกไซนัสซินโดรม

อมรรัตน์ ศาสตรวาหา[1] เกรียงไกร จิรสิริโรจนากร[2] จตุพร หนูสุด[1] ทักษอร ดวงอุไร[1]

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สุนัขพันธุ์ชเนาเซอร์เพศเมียทำหมันแล้วอายุ 14 ปี มีประวัติอาการเป็นลมหมดสติหลายครั้งในช่วง 2 สัปดาห์ สุนัขมีลักษณะอ่อน-แรง นอน แต่ยังคงตอบสนอง การตรวจร่างกายพบหัวใจเต้นช้าร่วมกับชีพจรอ่อนและไม่สม่ำเสมอ การตรวจคลื่นไฟฟ้าหัวใจพบลักษณะการเต้นจังหวะไซนัสตามมาด้วยภาวะหัวใจหยุดเต้นนานกว่า 3 วินาทีบ่งชี้ภาวะซิกไซนัสซินโดรม สุนัขได้รับการรักษาโดยการใส่เครื่องกระตุ้นหัวใจห้องเดียวผ่านทางเส้นเลือดดำ ภายหลังการผ่าตัดเป็นเวลาหนึ่งเดือนพบว่าสุนัขไม่มีอาการเป็นลมหมดสติและจังหวะเต้นหัวใจกลับเป็นปกติมากขึ้น

คำสำคัญ: สุนัข โรคหัวใจ เครื่องกระตุ้นไฟฟ้าหัวใจ ภาวะซิกไซนัสซินโดรม หมดสติ

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