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First Pacemaker in a Dog: A Historical Note

Abstract

The 1st Pacemaker used clinically in a 10-year-old male Basenji dog was implanted in 1967 because the dog had recurrent congestive heart failure because of complete heart block. A fixed-rate (now called VOO) 70-beats per minute (bpm), bipolar pacemaker was placed in the left paralumbar fossa and 2 leads were passed subcutaneously and attached to the left ventricular epicardium through a separate thoracotomy.

Disciplines

Animal Diseases | Cardiology | Cardiovascular Diseases | Comparative and Laboratory Animal Medicine | Surgery

Case Reports

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First Pacemaker in a Dog: A Historical Note

James W. Buchanan

The 1st pacemaker used clinically in a 10-year-old male Basenji dog was implanted in 1967 because the dog had recurrent congestive heart failure because of complete heart block.¹ A fixed-rate (now called VOO) 70-beats per minute (bpm), bipolar pacemaker^a was placed in the left paralumbar fossa and 2 leads were passed subcutaneously and attached to the left ventricular epicardium through a separate thoracotomy (Fig 1). The pacemaker had been removed from a human patient who died 6 months after its implantation. The pacemaker was manufactured in 1966 and had a design life of 2 years. The remaining battery life of the pacemaker was estimated to be 18 months on the basis of radiographic clarity of insulation in the self-contained mercury batteries (Fig 2; W.M. Chardack, personal communication). Electrocardiograms were recorded every 6 months and the pacemaker functioned normally at 70 bpm for 5 years and 4 months, even though the battery insulation showed progressive, extensive corrosion (Fig 2). The dog began fainting at this time, and its heart rhythm became irregular. The pacing rate increased from 76 to 124 bpm over the next 2 months, indicating battery failure according to the manufacturer. Fainting, sometimes described as convulsions, occurred several times a day. An electrocardiogram confirmed that the heart rate increase was due to an accelerated pacing rate and fainting was due to intermittent loss of capture by the pacemaker characterized by periods

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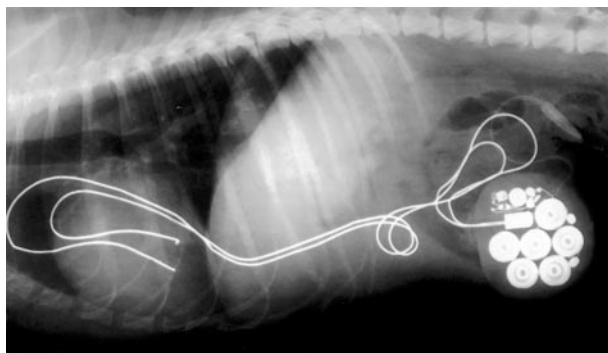


Fig 1. Lateral radiograph made 5 weeks after surgery in 1967. The pacemaker was positioned between the internal and external abdominal oblique muscles in the left paralumbar fossa. (*J Am Vet Med Assoc* 1968;152:1099–1109; with permission.)



Fig 2. Radiographs of pacemaker in situ 15 months after implantation (left) and 5½ years after implantation (right). The radiolucent central cores and insulating rings between electrode cylinders in the batteries became filled by corrosion material (arrows) leading to battery failure.

of asystole lasting 10 to 20 seconds followed by a ventricular escape rhythm.

The dog (Fig 3) was still in good physical condition at 15½ years of age, and surgery was performed to replace the pacemaker. No adhesions were observed on the silicone-covered pulse generator, and it was lifted easily out of a connective tissue pocket (Fig 4). A temporary pacemaker was not available and cardiac arrest occurred as expected when the pacemaker was disconnected (Fig 5). The heart stopped for 22 seconds before the 1st ventricular escape beat occurred. A 2nd escape beat occurred 5.5 seconds later and was followed by subsequent beats after progressively shorter intervals stabilizing at 25 beats/min within the next 30 seconds. The replacement pacemaker was attached to the existing leads and placed in the old generator pocket after cutting small holes in the ventral aspect of the fibrous pocket to



Fig 3. Photograph of the dog at 15½ years of age 1 week after pacemaker replacement.



Fig 4. Photograph at surgery. The silicone-coated pacemaker on the left was removed easily from its fibrous pocket and replaced by the urethane-coated replacement pacemaker.

prevent fluid accumulation. The new demand VVI pacemaker^b was programmed for 100 bpm and functioned normally for the remaining 6 months of the dog's life. At age 16, the dog stopped eating, began vomiting, and lost weight. Renal, cardiac, and pacemaker function were normal. An abdominal mass was palpable and the dog was euthanized. Postmortem examination identified a pancreatic carcinoma with widespread metastasis. The heart was normal grossly except for expected pericardial adhesions over the lead attachments. Step-serial histologic examination of the atrioventricular (A-V) conduction system disclosed replacement of the A-V node by fibrous connective tissue and arteriosclerosis of several coronary arteries. A small coronary arteriole in the upper part of the interventricular septum near the A-V node was

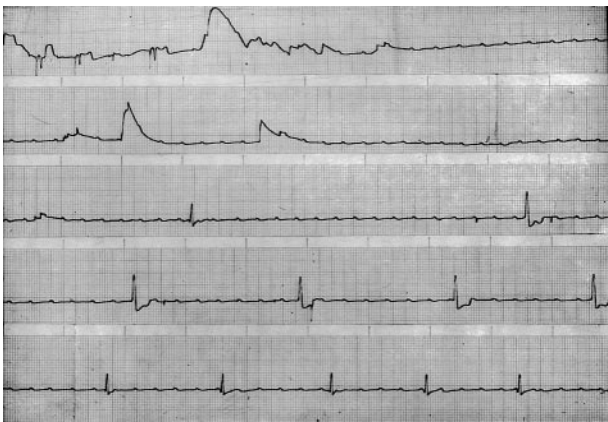


Fig 5. Continuous electrocardiogram recording when the pacemaker leads were disconnected. The 1st ventricular escape beat occurred after a 22-second period of arrest, followed by a 2nd beat in 5.5 seconds and progressively more rapid ventricular beats (25 mm/s, 1 cm/mV).

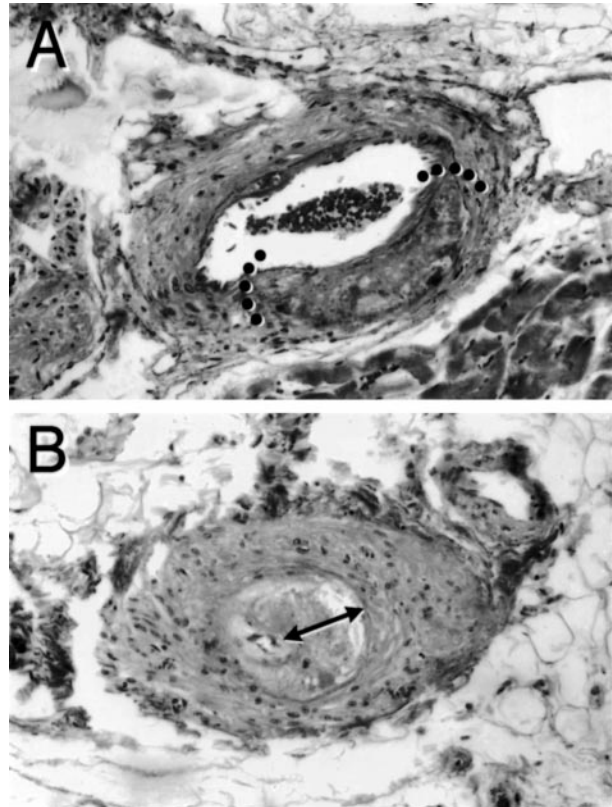


Fig 6. (A) Small coronary artery in the basilar portion of the interventricular septum. Note the crescent-shaped area of medial necrosis between dotted lines. (B) Coronary arteriole in the interventricular septum near the bundle of His. Note the extensive intimal plaque with necrosis (arrows), which nearly occludes the vessel. Pentachrome stain 100 \times .

almost completely occluded (Fig 6). Although at least 6 years had elapsed since the onset of heart block, it is probable that coronary artery disease and ischemia or infarction of the A-V node was the cause of complete heart block in this dog.

Footnotes

^a Chardack-Greatbach implantable cardiac pacemaker, model 5860, Medtronic, Inc, Minneapolis, MN

^b Chardack-Greatbach implantable cardiac pacemaker, model 5842, Medtronic, Inc, Minneapolis, MN

Reference

1. Buchanan JW, Dear MG, Pyle RL, Berg P. Medical and pacemaker therapy of complete heart block and congestive heart failure in a dog. *J Am Vet Med Assoc* 1968;152:1099-1109.