

## The Wandering Pacemaker: Intraperitoneal Migration of an Epicardially Placed Pacemaker and Femoral Nerve Stimulation

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**Abstract.** A premature child with congenital complete heart block had an epicardial single-chamber pacemaker implanted at 2 days of age. At 21 months of age, while sitting or standing, the patient's right anterior thigh muscles contracted at her pulse rate. Surgical exploration revealed a free-floating pacemaker in her peritoneum. A new dual-chamber pacemaker was implanted into the abdominal wall with resolution of the child's symptoms.

**Key words:** Epicardial pacemaker — Prematurity — Complete heart block

A pacemaker is the treatment of choice in infants with congenital complete heart block and symptoms or significant bradycardia [12]. In infants and children <10 kg, the leads are placed epicardially with the pulse generator implanted in the abdominal wall [2]. We describe a child with migration of the pulse generator into the peritoneal cavity. As a consequence, there was stimulation of the femoral nerve producing contraction of thigh muscles.

### Case Report

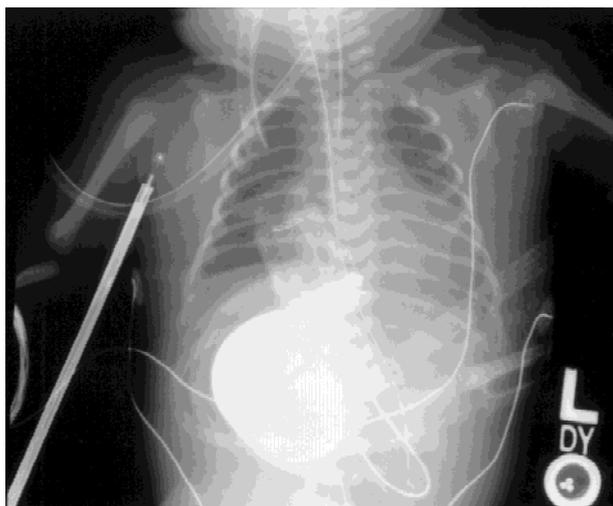
This was the fourth pregnancy of a 44-year-old mother. Her three other children are healthy. During the initial obstetric evaluation, at 25 weeks of gestation, an ultrasound examination demonstrated a female fetus with a ventricular rate of 70–90 beats per minute (bpm). The cardiac anatomy was normal. Maternal serum anti-SS-A antibody levels were >100 EU/ml, and anti-SS-B antibody levels were 50 EU/ml; there were no antinuclear antibodies. There was no family history of congenital heart disease or heart block. Ultrasound follow-up at 28 weeks of gestation detected a 4-mm pericardial effusion, an atrial rate of 160–180 bpm, and a ventricular rate of 57–65 bpm. On ultrasound follow-up 10 days later, the effusion had increased to 8 mm, the ventricular rate was 52 bpm, and the atrial rate was 134 bpm. Five days later, the effusion had increased to 11 mm and the ventricular rate was

42 bpm. Maternal infusion of isoproterenol did not change the fetal heart rate. Because of the development of progressive fetal cardiomegaly and pericardial effusion, a 1940-g female was delivered by cesarean section at 31 weeks of gestation. A poor respiratory effort and a heart rate of 40 bpm necessitated immediate intubation. On examination, she had no gross external anomalies. The heart rate was slow and regular and there were no murmurs. The pulses were normal and the abdomen was soft with no organomegaly. Electrocardiography showed complete heart block with an atrial rate of 130–140 bpm and a ventricular rate of 40 bpm. A chest radiograph showed increased pulmonary parenchymal markings and a normal heart size. Echocardiography demonstrated normal cardiac anatomy and function. There was a patent foramen ovale with bidirectional shunt.

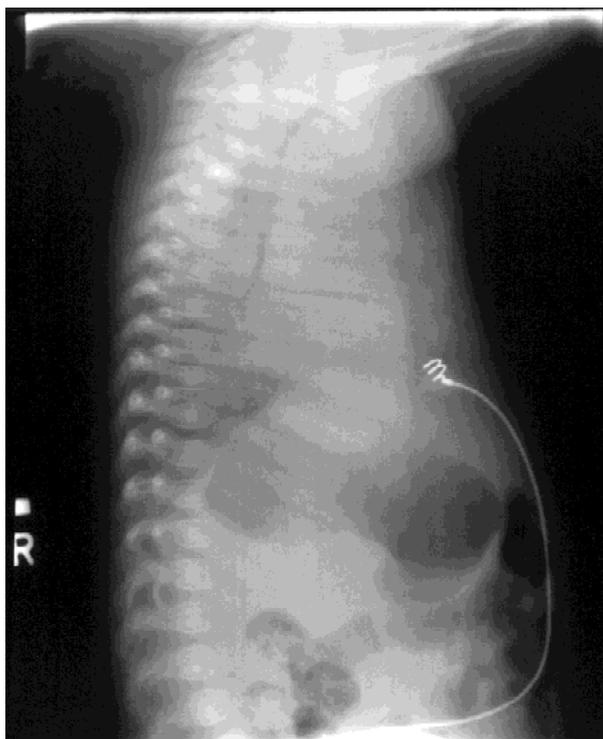
Isoproterenol infusion up to 2 µg/kg/min increased the atrial rate of 150 bpm and the ventricular rate to 50 bpm. Despite three doses of artificial surfactant therapy, she developed severe hyaline membrane disease and pulmonary hypertension. A single chamber epicardial pacemaker (Medtronic Thera SR8940B) was implanted via a subxyphoid approach at 2 days of life. The pulse generator was implanted in the subxyphoid region in the plane between the peritoneum and the posterior abdominal fascia; its rate was set at 140 bpm in the VVI mode. The chronic pacing threshold was 1 msec at 2 V and 0.12 msec at 5 V. The pacemaker was set at a pulse amplitude of 5 V and a pulse duration of 0.5 msec.

After discharge she was maintained at the same pacing rate for several months. Later, the pacing mode was changed to VVIR with a lower rate of 80 bpm. During the period of follow-up, repeat chest radiographs, at 4 months of age, showed that the pulse generator migrated away from the subxyphoid region (Fig. 1) into the abdominal cavity (Fig. 2). However, there were no specific associated symptoms. She had mild left-sided paresis and developmental delay. After an episode of urinary tract infection at 13 months of age, a voiding cystourethrogram was performed. It demonstrated grade 3 vesicoureteral reflux. The pulse generator appeared to be close to the urinary bladder. Filling the bladder with contrast resulted in displacement of the pulse generator superiorly, indicating it was not attached to the abdominal wall (Fig. 3). When the bladder was empty the pulse generator descended into the right iliac fossa (Fig. 4).

At 21 months of age the mother reported that the patient was attempting to walk. However, whenever the child stood and tried to walk or while sitting up and leaning to the right, her right quadriceps muscles contracted in a flexion pattern at a high rate. This was observed during a clinic visit with the patient in a sitting position. The rate of the right thigh flexion equaled the pulse rate. The ventricular pacing thresh-

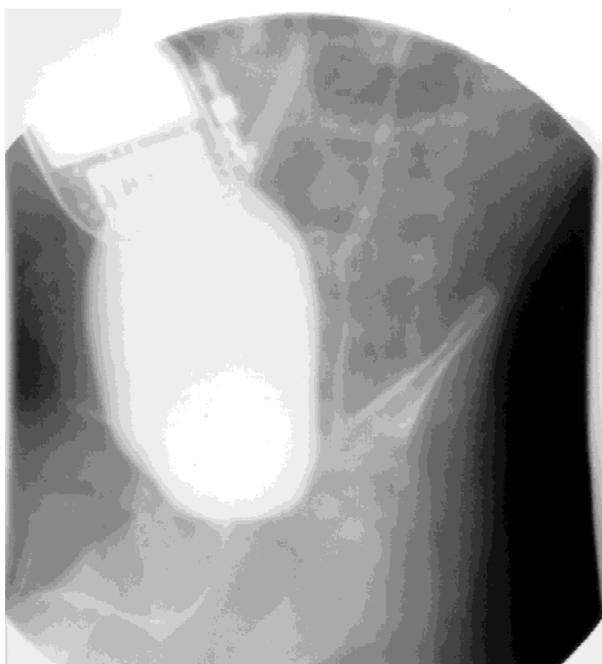


**Fig. 1.** An anteroposterior chest radiograph shortly after pacemaker implant demonstrates the size of the pacemaker and its subxyphoid location.



**Fig. 2.** A lateral chest radiograph, at 4 months of age, demonstrates migration of the pulse generator into the inferoposterior abdominal cavity (note that only the superior border is visible).

olds were unchanged (1 msec at 2 V and 0.12 msec at 5 V) even during the peripheral pacing. The original pacemaker was replaced with a dual-chamber epicardial pacemaker. At operation the original pulse generator was found free floating inside the peritoneum. After surgery



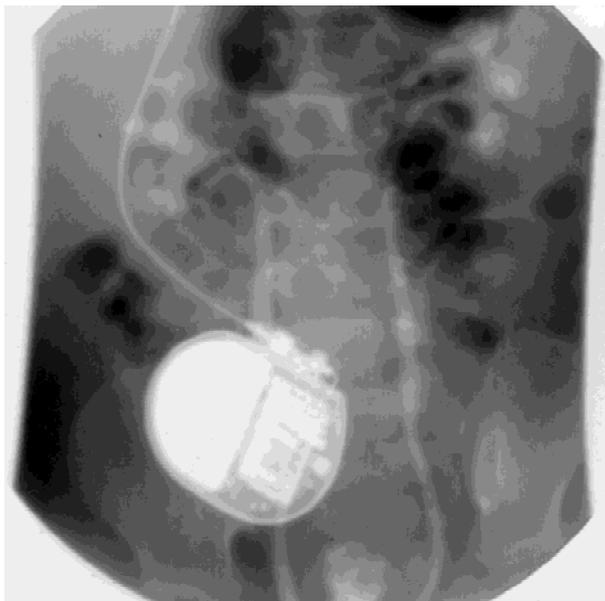
**Fig. 3.** Voiding cystourethrogram. An oblique view with a full bladder demonstrates the pulse generator's proximity to the dome of the bladder.

the contractions of the right thigh muscles disappeared and the child was able to stand and walk without difficulty.

## Discussion

We observed a rare pacemaker complication in a child with congenital complete heart block [1–5]. The migration of the pacemaker into the peritoneal cavity was unusual and unexpected. At the time it was first noted there were no symptoms, so no intervention was deemed necessary. The pulse generator was located inferoposteriorly while upright, in the region above the femoral nerve. In a unipolar lead system, the pulse generator is an active part of the circuit and is able to stimulate the adjacent tissue. Typically, this stimulation manifests as local muscle contraction and, occasionally, the phrenic nerve may be stimulated. To our knowledge, this is the first report of a femoral nerve and peripheral muscles stimulation by a pacemaker.

In our institution the standard abdominal implantation technique for an epicardially placed pacemaker is in the subcutaneous tissue superficial to the rectus abdominis muscle. We believe that patients can and should receive a standard subcutaneous pulse generator's pocket when they have sufficient tissue to allow an adequate barrier to prevent the pulse generator from eroding through the skin. In the absence of adequate abdominal



**Fig. 4.** Voiding cystourethrogram. With the bladder nearly empty the pulse generator descended into the right iliac fossa. Contrast refluxes in both ureters without obstruction of the right ureter.

wall tissue, the pulse generator is placed in a retrofascial, properitoneal space [3]. The size of the patient at the time of the initial implant may have contributed to the pacemaker migration. The external abdominal wall, skin, and subcutaneous tissues of a premature infant are not adequately developed to allow a secure subcutaneous implant position. This necessitated implant in the retrofascial plane.

In previous studies, the pulse generator was reported to migrate into the abdominal cavity [4], the urinary bladder [1], and the pericardium [6]. The implantation technique employed was deep to the rectus abdominis muscle [1, 6], similar to that used for our patient. Despite migration of the pulse generator into the abdominal cavity, our patient had no gastrointestinal symptoms.

Abdominal migration of a pulse generator is a rare complication of epicardial implantation. The surgical technique and/or the size of the patient at the time of implant may modify this risk.

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