An unusual complication of a myocardial electrode — Apatite mantle on the platinum-iridium spurs

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Summary. A case of a high excitation threshold that occurred 2 years 5 months after the initial implantation of pacemaker electrodes is described in a girl 4 years 3 months of age. This complication was considered to be due to calcification of the platinum-iridium electrode spurs. The calcified material was shown to be a kind of apatite using the X-ray powder diffraction method. This complication is rare, but it must be kept in mind since battery longevity has markedly improved in recent years.

Key words: Pacing failure – Electrode spurs – Calcification – Apatite

Pacemaker therapy for patients with heart block and symptomatic bradycardia has markedly improved since the first implant by Senning in 1958. In spite of improvements in hardware and operative techniques, there still remain some complications associated with the pacemaker. In particular, many complications related to electrodes have been reported [1–6]. The complication described here, however, has been rarely reported.

Case report

The patient, a girl, was operated on at the age of 1 year 9 months for patent ductus arteriosus and congenitally complete atrioventricular block. The operative procedures were ligation of the duct and pacemaker implantation. Myocardial needle biopsy was done at the same time and the specimen revealed no histological evidence of cardiomyopathy. In the implantation, two myocardial electrodes (Medtronic, 6917) were inserted into the left ventricular apex and a bipolar pacemaker (Medtronic, 5984) was placed in an abdominal subcutaneous pocket. By insertion of the two electrodes, a technique of suture fixation was added after screwing in to prevent electrode dislodgement. The postoperative course was smooth and uneventful until diaphragmatic twitching suddenly occurred 2 years 5 months after the operation. An electric leak due to broken insulation of a cable was suspected.

Under operation, the two leads were disconnected from the generator and examined electrophysiologically. The positive electrode showed 352 Ω electric resistance and 2.5 V excitation threshold, while the negative one showed 312 Ω resistance and 4.1 V threshold at 0.5 ms pulse width. From the electric resistance, broken insulation and electric leak were eliminated, but a high threshold was unexpectedly observed. The connections between the two cables were then reversed, i.e., the previous positive lead was used as the new negative one and vice versa. This procedure avoided a more extended operation, though the diaphragmatic twitching remained. At the time, the twitching was considered to be the result of changes in the geometrical relationship between the stimulating sites and the left phrenic nerve due to growth of the patient.

Thereafter, no particular observations were noted except for spontaneous disappearance of the diaphragmatic stimulation. One week after this disappearance, the patient fainted for a few seconds and fell from a chair. On the third admission to hospital, 24-h electrocardiograms revealed a pacing failure over a short period, which occurred several times a day (Fig. 1).

After changing the pulse width from 0.5 ms to 1.0 ms by an extracorporeal programmer, no failures were observed and this was confirmed by 24-h monitoring. The fainting episodes were absent for the following 11 days. Later, however, the same episodes reappeared, and the pulse width was widened again from 1.0 to a maximum of 2.0 ms. The same pacing failure then reoccurred 10 days later.

The patient was admitted to hospital so that the pacemaker and electrodes could be replaced; it was about 5 years after the implantation and the battery source warranty period was 6 years. Under general anesthesia, each lead was disconnected from the pacemaker and studied electrophysiologically by a unipolar use at 0.5 ms pulse width. The negative lead showed 292 Ω electric resistance and 3.6 V pacing threshold; the positive lead showed an astonishingly high resistance of 2667 Ω and a markedly elevated threshold of 7.2 V, which exceeded the battery output of 4.2 V stored at that time. While the extended operation of thoracotomy was being carried out by unipolar pacing using the negative lead, pacing failure suddenly occurred for 1 min, though the negative lead was considered to be functioning normally.

When exposing the portion of the electrode inserted, the positive lead was fixed tightly to the epicardium at a small distance from the left phrenic nerve. The other negative lead was observed to rise slightly from the epicardium and showed a pendulous motion on the inserted spurs, though no abnormal course or tension of the lead was observed. When this lead head was lifted slightly, pacing failure occurred; upon compressing the head against the heart, the failure was immediately discontinued.
Fig. 1a–d. Electrocardiographic findings. a Before pacemaker implantation. b After initial pacemaker implantation. c During pacing failure. Stimulating spikes are recognized, but almost all stimulations fail to induce ventricular excitation. d After second pacemaker implantation. P P wave, S pacemaker spike, Va automatic ventricular contraction

Fig. 2. Calcified spurs of an electrode. Distal part of spurs is coated with a white material

After removing both leads one by one with their spurs, new myocardial electrodes (Medtronic, 6917A-53) were screwed into the left ventricular apical wall; the position was maintained by suture fixation and the distance from the phrenic nerve was kept to 3 cm. With an extracorporeal testing generator, it was confirmed that diaphragmatic stimulation did not occur even with 10 V battery output and 2.0 ms pulse width. The pacemaker was also exchanged (Medtronic, 8402) and placed in the same pocket. The postoperative course of the patient has been smooth and no complication has been encountered.

Close examination showed that the distal one-fifth of the spurs of the positive electrode was coated with a white material (Fig. 2). The material had the appearance of calcification. It was analyzed to determine the lattice dimension by the X-ray powder diffraction method, using copper radiation with a nickel filter, and rotation photographs were obtained. The diffraction fringes and their rotation photographs are shown in Fig. 3 and the observed data are presented and compared in Table 1 with those of hydroxyapatite, a natural mineral, as determined by Harada et al. [7]. Our data for this material fit well with those for apatite, which has relatively strong intensities. It is concluded that the white material was an assembly of fine but incomplete crystals of apatite.

Discussion

Many complications related to the myocardial electrode have been reported, such as fracture, dislocation, twitching of a cable, broken spurs, electric leak, skin erosion, and infections [1–6]. We routinely use bipolar pacemakers, since some of these complications can be overcome by changing the system from bipolar to unipolar using a noninvolved cable. In this way, the prolonged operation of thoracotomy, which we have experienced in other cases, is avoided. In the present case, this procedure was intended to control the complication, since diaphragmatic twitching was considered to have been caused by electric leak due to broken electrode insulation. Broken insulation, however, was disproved by open electrophysiological examination, and the correction of diaphragmatic stimulation could not be achieved at the time. Another cause of the complication could have been electrode dislodgement, though this did not seem feasible since the electrodes had been screwed in and fixed by suture fixation.

The repeated occurrence of pacing failures suggested an increased threshold, since the stimulating spikes were evident on electrocardiograms during the failure (Fig. 1b). A lead fracture was discounted, though a high incidence of myocardial electrode fracture has been reported [1–3]. A high threshold may occur as a result of myocardial fibrosis around the electrode spurs, as observed by Lagergren et al. [8]. Management by increasing the pulse width was tried twice, but it succeeded for a time only.

The operative findings clarified the situation. The negative electrode was observed to rise, just as in cases with endocardial electrodes. This was the cause of the pacing failure. Both electrodes had been fixed in the same way, as described above, but the fixing