Unusual fracture of cardiac pacing electrode

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This report describes a patient with an epicardial demand pacing system in whom pacing failure two months after implantation was found to be due to a fracture of the helical wire electrode within its supporting structure. We know of no report of this site of failure of a Cordis 323-451 or similar electrode.

Case report

When first admitted on 18 September 1973 the patient, a 72-year-old married woman, complained of progressively worsening effort dyspnoea and intermittent claudication for the past two years. She had also suffered from dizzy spells but had not actually lost consciousness. On examination she was found to be in complete heart block with a right bundle-branch block pattern. The ventricular rate was 44/min. She was treated initially with a Devices endocardial electrode (model LC 90) inserted into the apex of the right ventricle via the right cephalic vein and connected to a Devices current-limited demand pacemaker (model 3821 RC) placed subcutaneously over the right rectus muscle. Considerable difficulty was experienced in obtaining a stable position with a threshold of 0.5 V.

Initial progress of the patient was satisfactory. At cardiac pacer checks in the General Infirmary at Leeds (Hepburn, 1975a) a routine electrocardiogram is recorded and the pacer artefact measurement (rate, duration, amplitude) are recorded for the Einthoven leads using a Nukab Pacemaker Impulse Analyser, manufactured by Svenska Radio AB, Stockholm (Rydén, Hedström, and Leijonhufvud, 1970). Artefact wave forms in leads I and III are also observed using an oscilloscope and are photographed on Polaroid film. Our experience has shown that similar results are to be expected when either of our usual endo- or epicardial leads is in use, though they are of quite different type. Measurements on two occasions (27 September and 19 December 1973) after switching to fast fixed-rate pacing gave artefact durations of 1.04 ms and amplitudes of +150 and +144 mV in lead III, with current limitation (Hepburn, 1975b) for the first quarter of the duration (cf Fig. 1b). Demand pacing rates were not obtained as pacing was inhibited by recovery of normal sinus rhythm. However, on 17 April 1974 the patient again presented at a pacer clinic in complete heart block with a ventricular rate of 50 bpm. The endocardial

electrode was found to have retracted into the right atrium.

Because of previous difficulty in achieving a stable endocardial position it was decided to employ an epicardial lead. The unipolar myocardial electrode of a Cordis 323-451 epicardial lead was inserted with a stab incision into the anterolateral surface of the left ventricle near the apex and secured by sutures through the fixing pad in the usual manner. The lead was brought through the intercostal space below the thoracotomy incision and taken by a subcutaneous tract to the existing pacer, which was replaced in the same site. Two months later at a follow-up clinic an electrocardiogram showed failure of the system either to inhibit or to pace (Fig. 2). Measurements taken after this second failure indicated an unexpectedly long duration of 1.08 ms and an artefact (leading edge) amplitude of only 32 mV in lead III. Moreover, the oscilloscope display (Fig. 1a) showed the presence of a long spike component in the wave form and the apparent absence of any current limitation of the pacer output.

Further evidence was sought from fluoroscopic examination. The 8-mm myocardial termination of the Cordis electrode is set at right angles to the epicardial lead approach (Fig. 3). Under x-ray screening in this patient it appeared that the termination was pivoting through an angle of 90° or more about the bend. Erroneously it was deduced that the fixing pad had become detached from the myocardium and that the myocardial portion of the electrode was being deflected by the contracting heart. However, at reoperation the fixing pad was firmly attached but it was not possible to stimulate the heart successfully through the electrode. Examination after removal revealed a fracture of the helical wire electrode-lead 6 mm from the end, within the silicone rubber of the reinforcing conical frustum and at the termination of the supporting wire insert. Replacement of the defective electrode by one of the same type restored the artefact wave form and measurements close to their previous values (Fig. 1b). The stimulus duration

FIG. 1 a) Pacer artefacts in leads I and III with break in a Cordis electrode bridged by body fluid. b) Pacer artefacts in leads I and III after replacement of the Cordis epicardial lead.

FIG. 2 ECG of patient fitted with ventricular inhibited pacing system, having a broken myocardial electrode with resultant failure to inhibit or stimulate effectively.

was 1.04 ms, the average artefact amplitude in lead III was +123 mV, and current limitation was noted for about 15 per cent of the stimulus duration.

Discussion

The increase of stimulus duration and reduction of current limitation occurring with electrode failure had correctly indicated an increased electrode circuit resistance (Hepburn, 1975b). The decrease in artefact amplitude more specifically located the large increase within the stimulus source – that is, pacer, lead, electrode, and immediate tissue (Hepburn, 1975c). Unfortunately the fluoroscopic evidence, which had appeared so conclusive, was misconstrued, despite the indication of a large increase in circuit resistance not found with a previous detachment of a similar electrode.

The increase in circuit resistance was due to an actual fracture rather than to a mere detachment of

FIG. 3 Myocardial electrode termination of a Cordis 323-451 epicardial lead, indicating point of fracture of wire helix at end of reinforcing wire insert.

the electrode. Since the break in the helical wire was so near its open end in the myocardium, body fluid could bridge the fracture and reduce the very high resistance obtained with persistent dry breaks. Bench testing has shown that high resistance, fluid-filled breaks (with the associated capacitive connexion transmitting the rapid increase in voltage as a spike component) may result in artefact wave forms similar to the shape reported.

The revised conclusions from the fluoroscopic evidence are particularly significant. The electrode termination and supporting frustum together had been flexing about the area of attachment of the frustum to the fixing pad. For this to occur there must have been great shearing of the myocardium in the region in which the electrode was implanted. The resultant straining of the wire had rapidly produced unexpected fatigue and failure of the helix. Regions of the myocardium subject to great shearing must be avoided when implanting myocardial electrodes. Also in cases of pacing failure associated with increased circuit resistance the high

probability of wet lead fracture must be considered.

Addendum

Since preparing this paper for publication five further cases of the same type of failure have been collected. A Devices model 3821 demand-type pacer was in use in each instance. Whenever significant measurements were obtained the characteristic changes have confirmed an increased resistance in the pacing circuit. These changes are a slight rise in the pacing rate of 0.3 to 0.5 bpm, a slight increase in the stimulus duration of 0.01 to 0.03 ms, large decreases in the artefact amplitudes of 50 to 75 per cent (95% in one case), and cessation of any current limitation. In two cases the increase of resistance was intermittent so that alternative sets of results were obtained during one examination. For one of these the fracture was confirmed on removal at reoperation but for the second, as for one of the cases of persistent high resistance, no direct confirmation was obtained since the pacing system was abandoned. In two other cases

fluoroscopic and radiographic examination revealed lead fractures at the myocardial termination.

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