Erratic prolongation of the pacing spike interval with presyncope as the only manifestation of minute bipolar lead insulation failure

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A break in lead insulation is thought to be a potential cause of erratic prolongation of the pacing spike interval. We report the case of a patient with reappearance of presyncope two months after a VVI bipolar pacemaker implantation. A lead insulation defect, which was invisible but resulted in erratic prolongation of the pacing spike interval, was indirectly inferred to be the only possible cause.

A 73 year old woman was admitted because she reported a presyncope attack two months after implantation of a VVI bipolar pacemaker (Vista 6, 444, CPI, St Paul, Minnesota, USA). The pacemaker was implanted because of sick sinus syndrome with sinus pauses resulting in presyncopal attacks. One month postimplantation, the pacemaker was found to work satisfactorily. The electrocardiogram recording showed appropriate sensing and capture but the spike to spike or the spike to intrinsic QRS complex intervals were erratically prolonged (fig 1). Since no cause of this abnormality could be identified non-invasively, surgical exploration of the pacemaker generator, the electrode generator connection site, and the proximal part of the lead (CPI endocardial bipolar tipped ventricular lead, model 4261, with silicone rubber, CPI HP-Plus, as the insulation material) was carried out. The only noteworthy finding was a region of bloody fluid intrusion in the proximal part of the lead (fig 2). However, no definite crack in the outer insulation coating was obvious. Proximal electrode (anode) impedance was found to be 310 ohms, whereas the distal electrode (cathode) impedance was 800 ohms. The combination of the fluid intrusion and the disproportionately low impedance of the anode and its wire—which going as always outside the wire that was attached to the cathode—was in contact with the fluid, led us to extract the lead and replace it with a new one of the same type and model. Proximal and distal electrode impedance of the new lead were 400 and 820 ohms, respectively. The patient was discharged asymptomatic with a normally functioning pacing system.

In contrast to this case, lead insulation breakdown usually results in a more generalised pacemaker malfunction with capture and sensing abnormalities with or without erratic prolongation of the pacing spike interval. The last abnormality was the only one...
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We thank Vivi Ordoli of CPI greek representatives for her technical assistance.


Images in Cardiology

Chemodectoma in a patient with a single ventricle

A 52 year old man with a single ventricle and severe pulmonary valve stenosis (peak systolic gradient 91 mm Hg) proven by echocardiography and cardiac catheterisation, complained of a large but slowly growing pulsatile mass in his left neck. He had chronic hypoxaemia. For more than 20 years he had intermittent venesection for symptoms of hyperviscosity resulting from secondary polycythaemia; his pretreatment haemoglobin concentration was 217 g/l, and haematocrit 0·63. Cardiac symptoms were stable during this period. There was a thrill over the mass. A colour Doppler ultrasound scan at the level of the bifurcation of the common carotid artery showed splaying of the internal and external carotid arteries by a highly vascular mass demonstrating multidirectional flow. A post-intravenous contrast computed tomography section at the same level showed a vividly enhancing mass (arrow) with maximum dimensions 6 × 6 × 9 cm (identification of the left internal and external carotid arteries was not possible because of the high contrast enhancement of the vascular tumour). These investigations confirmed that the mass was a chemodectoma which we postulate resulted from a hyperplastic response to prolonged severe hypoxaemia. The patient was treated with radiotherapy because he was considered unfit for surgical resection.

Carotid body tumours are most common in individuals chronically exposed to low partial pressures of oxygen by living at high altitude1 and less common in those with chronic hypoxia due to lung disease.2 In both these situations it is believed that hypoxaemia stimulates a hyperplastic response.3,4 Chemodectomas are described rarely in patients with cyanotic heart disease.4,5

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*Heart* 1997 77: 384-385
doi: 10.1136/hrt.77.4.384

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