SHORT CASES IN CARDIOLOGY

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

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Figure 1 Electrocardiographic leads V1 to V3 and a rhythm strip depicting all proper and improper pacemaker functions (top to bottom): capture is complete; sensing is appropriate and the pacemaker fires at the programmed rate (60 beats/min) after the sensed intrinsic QRS complex; the spike to spike intervals between the 4th and 5th paced beats and the 8th and 9th paced beats are longer than the programmed pacing spike interval, that is, the interval between the 7th and 8th paced beats; the spike to intrinsic QRS complex interval (between the 5th, paced, and the 6th, intrinsic, beat) is also longer than the programmed pacing spike interval.

Figure 2 The extracted lead. The bloody fluid intrusion and staining beneath the outer insulation coating is shown between the arrowheads.

A break in lead insulation is thought to be a potential cause of erratic prolongation of the pacing spike interval.¹ 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 tined 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 that was 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.¹ The last abnormality was the only one

found in the bipolar pacing system of our patient, probably because the insulation break was a tiny one confined to the outer insulation coating and allowing just a small amount of fluid intrusion during the two month period postimplant, and minimal leakage of current. Additionally, the impedance of the anode and the external wire, over which the fluid was accumulated, was not very low since an impedance lower than 250 ohms is considered to be definite evidence of a lead failure.² However, this invisible insulation defect resulted in the described erratic pacing spike

interval prolongation or "pacemaker pauses" similarly to the sinus pauses of sick sinus syndrome before the pacemaker implantation and with the same symptoms.

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- 2 Erickson S, Sweesy M, Johnson D, Forney R. The benefit of in office follow up in identification of lead failure [abstract]. *Eur J Cardiac Pacing Electrophysiol* 1996; 6(suppl 5):224.

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.^{1,2} Chemodectomas are described rarely in patients with cyanotic heart disease.^{3,4}

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- 4 Hirsch JH, Killien FC, Troupin RH. Bilateral carotid body tumors and cyanotic heart disease. *Am J Radiol* 1980; 134:1073-5.