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

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. 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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†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 Chemodectomas are described rarely in patients with cyanotic heart disease.4

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