Case reports

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Muscle potentials simulating pacemaker malfunction

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Interference spikes were noted on the electrocardiogram of a patient with an implanted demand pacemaker. Runaway malfunction was suspected and the generator replaced. Subsequent investigation showed that the interference originated from fasciculation in the left leg caused by underlying neuromuscular disease. Skeletal muscle potentials can produce an electrocardiographic appearance closely resembling 'runaway' pacemaker. Such abnormalities should prompt a search for occult neuromuscular disease.

Skeletal muscle activity may be recorded on the electrocardiogram, and the resulting somatic artefact is well recognized. However, multiple discrete signals on a recording which otherwise exhibits a normal isoelectric baseline have not previously been reported. This may be because the phenomenon is very rare or because it has been thought to have no clinical relevance. We report a patient who presented with this electrocardiographic pattern. Its significance was not initially appreciated, and this resulted in an implanted pacemaker being removed because of presumed runaway malfunction.

Case report

In May 1974 a 61-year-old man was transferred to this hospital for insertion of a permanent pacemaker system.

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He had presented elsewhere with a history of four black-outs during the preceding six weeks. Intermittent complete heart block had been diagnosed and temporary pacing had relieved symptoms. A Devices Demand Unit (3821RC), for right ventricular endocardial stimulation, was implanted in the left pectoral region. Diabetes mellitus and hypertension, diagnosed 20 years previously, were controlled with metformin 1 g 8-hourly and methyldopa 250 mg 6-hourly, respectively. At outpatient attendance one month later he was pacing well, with no recurrence of syncope. Three months later he returned with a five-week history of palpitations accompanied by dizziness and dyspnoea. The electrocardiogram showed sinus rhythm with right bundle-branch block and left axis deviation and sharp discrete signals at a rate of 300/min in leads II and III (Fig. 1). Clinical examination revealed no abnormality in the abdomen, cardiovascular, or respiratory systems. The demand pacemaker was activated by a magnet into a fast fixed rate mode and normal pacing was evident. Analysis

FIG. 1 Normal lead arrangement. Presumed inhibition of demand pacemaker by return of sinus rhythm. Artefact seen in leads II and III.

FIG. 2 Modified lead arrangement. Leads I and II now identical, but artefact seen only in II. Artefact clearly seen on lead III.

of the pacing stimuli showed no abnormality. Runaway pacemaker malfunction was considered because of the recent history of palpitations and the abnormal potentials on the electrocardiogram, which resembled those in an established case reported by Harper *et al.* (1974). The manufacturers thought that the normal wave form analysis and response to the magnet made pacemaker malfunction unlikely but, in view of the apparent absence of any other source of interference, urgent removal was advised, and the pacemaker generator was replaced. There was no evidence of a fault in the pacemaker.

Electrocardiographic monitoring showed no interference for the first 12 hours, but subsequently it returned intermittently. Further clinical observation revealed slight generalized muscle wasting and spontaneous fasciculations in the left calf and thigh muscles. Fasciculations could be provoked elsewhere, to a lesser extent, by exercise and percussion. On moving the left leg electrode to different areas on that limb it became obvious that the occurrence of abnormal spikes was related to electrode position. When the left arm electrode was transferred to the outer surface of the left leg (Fig. 2) the modified lead I showed a similar QRS configuration to that of lead II but without interference. In this position the modified lead III represented an axis across the left leg and showed the interference without recording cardiac electrical events. A concentric needle electrode was inserted beneath the original left leg electrode. The electromyogram from this site recorded spontaneous fasciculation potentials synchronous with each spike of interference on the electrocardiogram. Other electromyogram abnormalities found in the patient's muscles were fibrillation potentials, positive sharp waves, diminished recruitment pattern, and giant polyphasic potentials. An anterior horn cell cause for his denervation was proposed. He also had a mild peripheral sensory disturbance which was judged to be the result of long-standing diabetes mellitus.

Discussion

Component failure may result in rapid discharge of pacemaker stimuli. The reduced amplitude of these stimuli usually results in loss of pacing but induced rates up to 300/minute have been reported (Wallace,

Abelmann, and Norman, 1970). Occasionally the phenomenon is intermittent and abnormal pacemaker activity may disappear transiently (Aldridge and Kahn, 1965; Harper *et al.*, 1974). With improved pacemakers, however, this is now rare. We have not experienced such difficulties with the implanted units used at this centre (Devices Implants). The history of attacks of palpitations together with the electrocardiographic appearance led us to suspect and act on the diagnosis of runaway pacemaker in our patient. In retrospect, the fact that the abnormal potentials were seen only in electrocardiographic leads from the left leg should have made us inspect that limb more closely. As it was, fasciculations had not been observed, and we were then unaware that they could produce spikes on the electrocardiogram mimicking those from a runaway pacemaker. It is clearly important to appreciate that discrete fasciculation potentials from muscle underlying an electrode may produce interference spikes on the electrocardiogram which, in patients with an implanted pacemaker, may be misinterpreted as malfunction. Furthermore, when these spikes are seen on a routine electrocardiogram the presence of neuromuscular disease should be suspected.

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