Inhibition of external demand pacemakers during muscular activity

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Following the demonstration of external demand pacemaker inhibition by myopotentials from the pectoralis major muscle, a study was made of the frequency and cause of this phenomenon and its effect on different pacemaker units. Of the 11 patients with complete atrioventricular block studied, 8 showed pacemaker inhibition during arm movement; in 3, with prolonged asystole, this was associated with symptoms. In the remaining 3 patients repositioning of their indifferent electrodes nearer to a large muscle bulk was followed by pacemaker inhibition during contraction of that muscle. All the units tested were equally susceptible to this form of interference, though one offered some protection because its sensitivity to inhibitory potentials could be regulated. It is considered that this is a common form of interference in patients with external demand pacemakers and that close proximity of the indifferent electrode to the main muscle belly allows entry of myopotentials to the pacemaker circuitry and thus to inhibition. Wider use of bipolar pacing electrodes and especially fixed rate pacemakers will protect against this form of interference.

It has been the policy of members of our department to carry out a two-stage procedure in patients requiring a permanent pacemaker system. Initially a unipolar pacing electrode is introduced via a subclavian vein and its tip positioned at the apex of the right ventricle. An indifferent electrode wire, one end of which is fashioned into a loop of approximately 1 cm diameter, is placed subcutaneously near the sternal edge and both wires are connected to a demand or fixed-rate pacemaker unit. Some weeks later the second stage is completed by the insertion of an implantable unit.

In 2 patients with external ‘on-demand’ units, routine tests after the first stage showed that movement of the arm on the same side as the electrodes was associated with loss of pacing. In both cases an unstable electrode tip position was considered likely and repositioning of the pacing electrode carried out. This, however, did not prevent loss of pacing function during arm movement and further studies have shown that the pacemaker was being inhibited by myopotentials generated by the pectoralis major muscle.

Myopotential inhibition of implanted demand pacemakers of the ventricular inhibited type has been reported by Wirtzfeld, Lampadius, and Ruprecht (1972), Mymin et al. (1973), and Ohm et al. (1974). The purpose of this paper is to report similar findings in patients with external ventricular inhibited units and to emphasize the importance of the indifferent position relative to the underlying muscle, in the genesis of this form of interference.

Subjects and methods

Eleven patients with external demand pacemaker units were studied. Three had been known to complain of occasional faintness since insertion of the pacing wire, and they include the two patients referred to in the introduction. The other eight were drawn in a consecutive manner from patients attending the pacemaker clinic. In all of them routine tests of pacemaker function carried out at rest failed to demonstrate any abnormality. The patients' ages ranged from 50 to 76 years and Adams-Stokes attacks were the reason for pacing in 9 of them. All had persistent or intermittent third degree atrioventricular block, and in 6 this was associated with ischaemic heart disease.

Each patient was questioned about recent episodes of faintness or dizziness, and if a history of these symptoms was elicited then an association with movement, particularly of the arm, was sought. Each patient was then studied in a semisupine position and if necessary pacemaker capture was ensured by increasing the pacing rate. In each, a Meditek MP 10 ventricular inhibited pacemaker was used initially and in 3 patients the following units were also tested: Meditek MP 11, a ventricular
inhibited demand unit; Devices 38 to, a ventricular inhibited unit with adjustable sensitivity and also a fixed rate mode; Cardiac Recorders 162, a ventricular inhibited unit, and lastly Cardiac Recorders 163 bench model, a ventricular inhibited unit also with a fixed rate mode.

Each patient was asked to move the arm on the same side as the wires across the chest as far as possible, while a standard lead III electrocardiogram was recorded. If pacemaker inhibition did not occur, then a surface electrode placed on the skin over the main bulk of the pectoralis muscle was substituted for the subcutaneous indifferent electrode and adduction of the arm repeated. In 4 patients the indifferent electrode was also applied to the skin over a biceps muscle, and a record obtained before, during, and after contraction. In 2 patients an electromyogram was obtained from the pectoralis muscle during contraction and a simultaneous record of myopotentials and pacemaker activity made. A Medelec MS 4 electromyograph was used and myopotentials were recorded from two electrodes, one of which was attached to the pacing indifferent electrode lying subcutaneously on the muscle, and the other was applied to the skin lateral to the left sternal edge.

**FIG. 2** Electrocardiogram recorded during arm movement in a patient without symptoms. In A and B arm movement coincided with the 2nd paced beat. In A, on-demand pacing, inhibition occurs but an escape beat prevents prolongation of asystole. In B, fixed rate pacing is unaffected.

**Results**

The 3 patients who had complained of intermittent faintness all considered that arm movement played a part in precipitating symptoms. One man noted faintness on shaving and another patient when she swung her arm while walking. All 3 patients felt faint and demonstrated complete inhibition of the pacing unit with prolonged periods of asystole during adduction of the ipsilateral arm. Normal pacemaker function returned immediately the arm relaxed and fixed rate pacing was uninfluenced by muscle contraction (Fig. 1). Five other patients showed similar pacemaker inhibition but were symptomless, probably because their own natural escape rhythm took over; again fixed rate pacing was unaffected (Fig. 2).

Of the 3 remaining patients, pacemaker inhibition was obtained in one by moving the indifferent electrode to a position between the clavicle and the anterior axillary fold which corresponds to the main pectoralis muscle bulk and the other 2 only produced inhibition by placing the electrode over the biceps muscle as described. The latter manoeuvre brought about inhibition in every patient studied.

**FIG. 1** The electrocardiogram of a patient in whom arm movement was associated with loss of pacing and faintness. In A, B, and C movement coincided with the 2nd paced beat. In A and B, on-demand pacing, 2 paced beats are followed by asystole which persists for duration of muscular contraction. In B an escape beat occurs. In C, fixed rate pacing is unaffected.
entry to the pacemaker electronic circuitry, is quite sufficient to cause inhibition of the majority of both external and implantable ventricular inhibited units (Chamberlain and English, 1973). The way in which myopotential activity can influence these units has not been fully understood, but Mymin et al. (1973) considered that the position of the indifferent electrode relative to the muscle might be of importance. The results reported here strongly support this view and it seems likely that the indifferent wire transmits myopotentials to the generator unit, resulting in inhibition, and this is most likely to occur if the indifferent electrode is placed close to a large muscle bulk. Thus, if loss of pacing during muscular activity was not at first seen in patients with the indifferent electrode sited medially, then repositioning of this electrode over the main bulk of pectoralis or biceps muscle resulted in pacing failure caused by pacemaker inhibition.

Protection from this form of interference could reasonably be expected to follow the use of bipolar pacing electrodes, but if unipolar electrodes are employed with external units then the indifferent electrodes should be placed medially away from the main pectoralis muscle bulk. Of the individual external units tested, the Devices 3810 offers the advantage that its sensitivity to inhibitory stimuli can be regulated, and of course, when necessary, changing to a fixed rate mode will protect the patient from this form of interference.

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References

Requests for reprints to Dr. B. Gribbin, Department of Cardiology, The Radcliffe Infirmary, Oxford.
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