Myopotential Inhibition of Demand Pacemakers*

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This report of 20 patients who have R-wave inhibited demand pacemakers, 19 of which are titanium-screened, connected to unipolar electrode systems, suggests that there is a significant incidence of myopotential inhibition. It is therefore possible that considerably more patients may experience fleeting muscular inhibition than is generally believed, particularly in the younger, more active age group. It is worthy of note, that on no occasion did any of the pacemakers in question revert to an asynchronous mode of operation when myopotential inhibition was provoked.

It is now well recognized that external electromagnetic fields may influence implanted cardiac pacemakers.12 Patients with these devices are therefore warned that frequencies within the range of the radio wave bands can cause inhibition of normal, spontaneous pacemaker function. Apparatus emitting such frequencies include medical cautery and diathermy instruments, radiotransmitters, microwave ovens, car ignition systems, large electric motors, etc.

Patients with unipolar electrode systems connected to an R-wave inhibited pacemaker are more susceptible to inhibition problems influenced by these magnetic fields. The long, single electrode configuration presents a large dipole to which electromagnetic fields may be induced. This causes saturation of the sensing mechanism of the demand pacemaker which, electronically, is unable to differentiate the depolarization patterns from those originating from the myocardium. Bipolar electrode systems, connected to R-wave inhibited pacemakers are less vulnerable to external influence, while fixed rate models are infinitely less susceptible since they may be disturbed only by extremely high density radio frequency sources such as those emitted by electrocautery or shortwave diathermy equipment.

Total screening of pacemaker generators with titanium together with sophisticated circuitry switching the pacemaker to an asynchronous mode of action has considerably reduced the problem of external radio frequency interference, but at late it has become apparent that inhibition can also be provoked by myopotentials emanating from the muscles surrounding the pacemaker. We have encountered this complication in 20 of our patients who have R-wave inhibited pacemakers. This report discusses the findings in three patients who developed symptoms and correlates muscular inhibitory activity with electrocardiographic and electromyographic studies of two cases (1 and 2).

Case Reports

Case 1

A 69-year-old man was admitted to the pacemaker clinic with complete heart block in 1966. Since that time, five pacemaker generators have been implanted: the first four into the rectus abdominus sheath and the fifth, a titanium-screened unit, into the left pectoralis region. The last pacemaker was implanted in August, 1971. Previous pacemakers except for one which had to be removed because of component failure, were removed electively as the patient lived 500 miles from this clinic. In April, 1973, during routine testing of pacemaker function3 the patient complained of intermittent lightheadedness. On examination, he was obviously distressed by his condition and was hyperventilating. Pacing function was normal in every respect while the patient lay resting quietly, but electrocardiographic monitoring revealed short periods of asystole without pacemaker spikes when the patient sighed deeply.

To demonstrate the mechanism of this phenomenon an electrocardiogram (ECG) and electromyogram (EMG) from the pectoral muscles were simultaneously recorded during deep inspiration (Fig 1A). The procedure was then repeated with a magnet placed over the pacemaker generator, converting it to a fixed-rate mode (Fig 1B). It was evident that in the demand mode, pacemaker impulses were not emitted during deep inspiration, but that this was effectively corrected by use of the magnet, producing fixed-rate pacing. Isometric adduction of the arm on the same side as the pacemaker produced asystole for as long as forced adduction was maintained and could be prevented by use of the magnet (Fig 2A, B). At no time was there any other evidence of malfunction of the pacemaker or electrode system.

Case 2

A 53-year-old man was seen in August, 1971 complaining of dizziness and palpitations. The patient had complete heart block, with a ventricular rate of 30/min. A titanium-screened demand pacemaker was implanted in the right pectoral region in September, 1971 with a unipolar endocardial electrode. His progress was satisfactory until he attended the pacemaker clinic in April, 1972, when he complained of giddiness when he applied pressure with the outstretched right arm. On many occasions he had felt lightheaded when tying his shoelaces, and once he had a syncopal attack when lifting a heavy weight.

Simultaneous ECG and EMG recordings showed suppression of pacemaker spikes at the height of muscular activity (Fig 3). Conversion of the pacemaker to the fixed-rate mode by use of the test magnet permitted uninterrupted pacemaker

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activity during identical muscular activity.

In order to alleviate his symptoms caused by inhibition of the pacemaker it was decided to electively replace the unit with a nonshielded R-wave inhibited device. Following re-implantation into the same site, no further asystolic episodes could be provoked.

**Case 3**

A 52-year-old man with extreme bradycardia due to complete heart block resulting from sarcoidosis had a titanium-screened R-wave inhibited pacemaker with a unipolar transvenous electrode implanted in the pectoral region in May, 1973. At a recent routine followup he complained of light-headedness when shaving with a safety razor, while using the hand on the side of the pacemaker generator. Repetition of the arm movement revealed pacemaker inhibition which could be prevented by application of the test magnet over the implanted pacemaker.

The phenomenon of muscular inhibition was demonstrated at routine followup clinics in other 17 patients by isometric adduction of the arm on the side of the implanted generator and could not be repeated in any after application of a test magnet. All but one of these patients had a titanium-screened unit implanted in the pectoral region. At no time did any of the 17 patients complain of any symptoms associated with pacemaker inhibition.

**Comment**

Wirtzfeld et al4 who are believed to be the first to have drawn attention to the phenomena of myopotential inhibition reported that more than 50 percent of patients could be shown on routine testing to be prone to the complication of myopotential inhibition. Wirtzfeld6 suggested that a decrease in the detecting sensitivity of the R-wave inhibited pacemaker to 3 mV be recommended to eliminate the problem. Myrmin et al8 reported that 24 percent of patients in their series showed vulnerability to myopotentials.

In our series of 186 patients with R-wave inhibited pacemakers (including 76 with titanium screening), we have shown 20 with the phenomena of myopotential inhibition. Three of our patients were symptomatic, and in 17 inhibition was elicited. This figure of 20 patients represents 11 percent of the total of 186, but as 19 of these patients have titanium-screened devices we have demonstrated a greater susceptibility to inhibition in this group (26 percent).

Although it cannot be denied that the asystolic episodes described could be due to sudden resistance changes in the electrode lead system as reported by Lasseter et al,7 it is believed unlikely because of the absence of any inhibitory influence during normal bodily movement and the absence of gross period variation as often encountered in in-
termittent impedance changes of the electrode system. Unlike external inhibition by radio frequencies, which is of an inductive nature, via the electrode dipole, myopotential inhibition is a more direct artifact influence on the pacemaker itself and is more likely to be caused in unipolar systems connected to an R-wave inhibited pacemaker that is completely covered in titanium, presumably because of the large anodal electrode area of the pacemaker exposed to an equally large area of musculature. Until electronic engineers are able to resolve this problem it is felt that careful consideration should be given to the type of pacemaker implanted, especially in young, active or muscular patients. The use of bipolar electrodes, a unipolar electrode attached to a demand pacemaker without complete metallic screening, or an asynchronous pacemaker would seem least likely to give rise to problems of myopotential inhibition.

The following routine test is now carried out on all patients who have R-wave inhibited devices situated in the pectoralis region: The subject is requested to stand erect and extend the arm on the side of the pacemaker generator in line with the long axis of the body (as if standing at attention). Thus adducted, he then applies hard elbow pressure against the side of the body, which will cause contraction of the pectoralis major. During this procedure the subject's electrocardiogram is monitored and any evidence of temporary or continuous inhibition should be shown.

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REFERENCES


Figure 3. ECG and associated EMG activity (upper) serratus anterior (lower) pectoralis major of patient 2 showing periods of partial and prolonged asystole during application of pressure against outstretched right arm.