Implanted-pacemaker-induced dysrhythmia and its management

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Summary

Five cases of implanted-pacemaker-induced dysrhythmia are described and factors precipitating such dysrhythmias are discussed.

The efficacy of practolol in the treatment of such dysrhythmias is shown and its use as a prophylactic antidysrhythmic agent has been suggested.

A NUMBER of artificial-pacemaker-induced dysrhythmias has been described. Quinidine, lignocaine and procainamide are known to be beneficial in the treatment of such dysrhythmias. The use of practolol is described as a valuable anti-dysrhythmic measure in the management of artificial-pacemaker-induced dysrhythmias.

Case reports

Case 1

A 65-year-old diabetic lady had been treated with Saventrine since 1965 for her heart block. In September 1971 she developed frequent Stokes-Adams attacks for which a ventricular inhibited intracardiac pacemaker was implanted. Within 24 hr of implantation she developed frequent ventricular extra-systoles which responded to procainamide. Subsequently she developed lignocaine-resistant ventricular tachycardia (Fig. 1) which responded to 20 mg of practolol, given intravenously. As the ventricular extra-systoles reappeared she was treated with practolol 100 mg twice daily and she has had no dysrhythmia since.

FIG. 1. Case 1.

FIG. 2. Case 2.

Case 2

A ventricular inhibited pacemaker was implanted in the axilla of a diabetic man for heart block with syncopal attacks. He had suffered from heart block for 2 years and was managed medically with Saventrine. Six weeks after the implantation, he developed supraventricular tachycardia (Fig. 2). His dysrhythmia was treated with practolol 100 mg given at 8-hr intervals. No dysrhythmia has been noted since.

heart block. A month after the implantation of the pacemaker, she developed frequent ventricular extra-systoles which inhibited the pacemaker (Fig. 3). This produced syncope. She was treated with practolol 100 mg at 8-hr intervals. Since the institution of this therapy she had no dysrhythmia and no further syncope.

Case 3

A ventricular inhibited pacemaker had to be implanted in a 71-year-old lady as Saventrine failed to restore her haemodynamic instability due to

Case 4

A man who had had a fixed rate pacemaker implanted 4 years before for complete heart block developed 'palpitations' and syncopal episodes. An

FIG. 3. Case 3.

electrocardiogram revealed frequent ventricular extra-systoles (Fig. 4). He was treated with practolol 100 mg twice a day. Since the commencement of practolol therapy, he has had no syncopal attacks and subsequent electrocardiograms showed no extra-systoles.

Case 5

Soon after the implantation of a fixed rate pacemaker in the axilla, frequent ventricular extra-

systoles were observed (Fig. 5). These were treated with practolol and he has showed no other dysrhythmia since.

There was no evidence of recent cardiac infarction in any of the above patients. All the pacing systems were found to be functioning satisfactorily. Saven-trine was discontinued after commencement of artificial pacing. Satisfactory pacing was achieved in all the above cases after controlling the dys-rhythmia. All the above patients are now symptom-free.

Discussion

Since the first successful resuscitation of the heart in ventricular standstill by external electric stimulation by Zoll (Zoll, 1952), artificial pacemakers have been acclaimed as a most useful contribution in the treatment of heart block, and approximately 1800 patients in Britain are at present being treated with implantable mercury-cell pacemakers. Apart from the other recognized complications, fatal dysrhythmias

FIG. 4. Case 4.

FIG. 5. Case 5.

TABLE 1

Case no.	Type of pacemaker	Pacemaker rate	Intracardiac cavity potential (mV)	Dysrhythmia	Time interval between implantation and onset of dysrhythmia
1.	Ventricular inhibited	86	6	VES VT	24 hr
2.	Ventricular inhibited	73	9	Supraventricular tachycardia	6 weeks
3.	Ventricular inhibited	72	10	VES	1 month
4.	Fixed rate	70	Not done	VES	4 years after
5.	Fixed rate	70	Not done	VES	Soon after implantation

VT, Ventricular tachycardia; VES, ventricular extra-systoles.

are known to be induced by the artificial pacemaker. Sowton (1965) has described a higher death rate in those with artificial pacemaker-induced ventricular fibrillation. A rare dysrhythmia resembling ventricular fibrillation has been described after implantation of a cardiac pacemaker, due to repetitive ventricular firing. With the advent of 'demand' pacemakers, such complications seem to be rare. Other associated dysrhythmias are parasystole, ventricular and supraventricular tachycardia, 'escape' pacemaker systoles after blocked atrial premature beats, forms of atrioventricular (interference) dissociation and some bizarre rhythms (Castellanos & Lemberg, 1964).

The danger of implanted pacemaker-induced ventricular fibrillation is greater when the threshold for ventricular fibrillation has been lowered by ischaemia, sympathomimetic drugs or recent operation. It has been thought to be due to an inflammatory reaction around the site of attachment of the electrode to the heart (Sowton, 1965). Competition between the patient's spontaneous rhythm and that produced by the pacemaker commonly results in ventricular fibrillation (Lasser & Julian, 1968). Forbes *et al.* (1968) report a 22.7% incidence of ventricular extra-systoles in long-term pacing. The majority of the patients developed the extra-systoles in the first 48 hr after implantation. In a series of sixty patients, ventricular fibrillation occurred in five patients and one developed ventricular tachycardia while in hospital (Sowton, 1965). Ventricular fibrillation occurred in 5–21% of cases with intracardiac pacing in acute myocardial infarction (Paulk & Hurst, 1966).

In the cases described in this paper, there was no evidence of recent myocardial infarction in any. The intracardiac cavity potentials in the first three patients are high enough to inhibit the artificial pacemaker.

Although the first three patients received a sympathomimetic drug (Saventrine) until the time of the pacemaker implantation, only the first case developed dysrhythmia within 24 hr. The other two developed

dysrhythmia after one month. The fifth case developed dysrhythmia soon after surgical implantation of the pacemaker (Table 1). The appearance of dysrhythmia 4 years after the implantation in the fourth case, proves that the dysrhythmogenic activity may remain dormant for a variable period.

Chatterjee, Harris & Leatham (1969) suggested using lignocaine infusion followed by oral procainamide or quinidine, as a prophylactic measure, in order to diminish competition. We do not follow this practice: we prefer to use an anti-dysrhythmic drug only when the dysrhythmia demands its use. Forbes *et al.* (1968) treated the extra-systoles with either procainamide or quinidine. Procainamide has been shown to increase the threshold (Siddon & Sowton, 1968). Harris *et al.* (1965) mentioned that beta-blockers have been successfully used in suppressing ectopic beats. Previous studies have shown that practolol is effective in the treatment of both supraventricular as well as ventricular dysrhythmias (Jewitt, Mercer & Shillingford, 1969; Jachuck & Husaini, 1971). Barrett (1971) has demonstrated that practolol is a specific, competitive antagonist of catecholamines at beta-receptor sites with a marked selectivity for those in the myocardium.

The cases described in this article demonstrate the efficacy of practolol in the treatment of artificial pacemaker-induced dysrhythmias and also suggests its use as a prophylactic measure, in a dysrhythmia-prone patient.

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