Syncope During Standby Cardiac Pacing

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An electric pacemaker may, under certain circumstances, compete with an intrinsic cardiac rhythm and share control of the heart. Should an electric stimulus fall in the vulnerable phase of the ventricular cycle, potentially dangerous arrhythmias may be provoked (Sowton, 1965; Siddons and Sowton, 1967; Bilitch, Cosby, and Cafferky, 1967). "Ventricular-programmed" or "demand" pacemakers, which can detect and respond to intrinsic ventricular depolarization, have been developed (Goetz, Dormandy, and Berkovits, 1966; Parsonnet *et al.*, 1966; Furman *et al.*, 1967; Zuckerman *et al.*, 1967; Sowton, 1967). The following case illustrates an example where stimuli from one such unit were repeatedly delivered into the T wave of ventricular extrasystoles, with strong inferential evidence that transient cerebral symptoms resulted from a rapid arrhythmia and ineffective cardiac action.

CASE REPORT

A 49-year-old white man was admitted to the Veterans Administration Center, Wood, Milwaukee, Wisconsin, with a history of frequent recurrent syncopal attacks for 8 years. There was moderate essential hypertension, minor calcific aortic stenosis, and some left ventricular hypertrophy. Electrocardiograms showed sinus rhythm with right bundle-branch block. This was frequently punctuated by periods of ventricular asystole of up to 15 seconds, terminated by a ventricular escape beat and return of atrioventricular conduction. Treatment with an implanted asynchronous, epicardial electrode pacemaker system was attended by limited success. Reliable repeated ventricular capture was only sporadically obtained, even with the use of a 22 mA Medtronic generator (No. 5852). Despite this unusually poor technical result, syncope did not recur after the insertion of the first unit.

Three years later both lead wires were broken. A Cordis Standby Ectacor unit was implanted into the right ventricular endocardium. Capture of the ven-

tricles could be obtained through the electrode catheter with ease at 1.2 mA. The function of the Cordis Standby unit is illustrated (Fig. 1). Ten days later, while eating his breakfast at home, the patient suddenly became unable to speak, and his left thumb was numb: this abated in perhaps 20 minutes. There was no loss of consciousness or other symptom. Physical examination an hour later showed no fresh sign. Lumbar puncture, electroencephalogram, and brain scan were all normal. Two weeks later the patient again noticed that he could not speak, though his mind was clear. A reliable nurse with experience in cardiac problems noted no heart beat or pulse for about 30 seconds, after which a regular heart beat at about 100 a minute returned spontaneously. However, the right pupil was dilated for 15 minutes, and aphasia persisted for 2 hours. The electrode catheter was noted at fluoroscopy, and later at operation, to be firmly in place in the right ventricle. The Cordis pacemaker was removed and replaced by a Medtronic Demand unit (No. 5841); its action is shown in Fig. 2. Syncope did not recur during a follow-up period of 9 months. The Ectacor unit has been examined by Cordis Corporation. No malfunction was found.

Comment. The two transient cerebral episodes do not suggest any primary neurological disorder, but strongly resemble the many earlier episodes in their suddenness of onset and in the absence of permanent sequelae. The differences in the level of consciousness and the transient localizing signs suggest that different cardiac mechanisms may be responsible. In the earlier attacks, sudden and prolonged asystole resulted in unconsciousness. The later attacks were most likely the result of a ventricular tachyarrhythmia, leading to lowered cerebral perfusion, so that minor arterial narrowings became critical. The electrocardiograms (Fig. 1) show that pacemaker stimuli were delivered into the latter part of the T wave of ventricular extrasystoles on many occasions. Since the coupling interval of the extrasystoles was not fixed, there is a relatively wide range during which the pacemaker stimulus may occur during inscription of the T wave; and, perhaps, the vulnerable phase. The absence of any similar episodes, when a different pacemaker is *in situ*, supports this as a likely mechanism in this case.

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FIG. 1.—Selected strips from the record, with the Ectacor Standby Pacer *in situ*. The pacemaker stimulus artefacts have been retouched for clarity. Note such stimuli falling upon the late part of the T waves of extrasystoles in each lead and producing a response. A basic sinus rhythm is present with P—R=0.24 sec. and right bundle-branch block. Pacemaker stimuli deform the QRS of many beats, sometimes without effect and sometimes (lead II) producing ventricular fusion beats. Ventricular extrasystoles with varying coupling occur. At least once in each lead a pacemaker stimulus falls in the T wave of the extrasystole, evoking a response and indicating that the extrasystole was not sensed by the device.

DISCUSSION

Demand Pacing Mechanisms. The Cordis Ectacor Standby unit and the Medtronic Demand unit, as used in this case, have differing features which are important in the analysis of the electrocardiographic tracings. Furthermore, many published descriptions (Parsonnet *et al.*, 1966; Furman *et al.*,

1967), concerning Cordis Standby pacing, refer to the now discontinued Ventrivor II unit. This is very different from the Ectacor. A brief description of these details is, therefore, apposite.

All "demand" units employ a sensing circuit intended to detect only intrinsic ventricular activity (QRS complexes, but not P or T waves). On

FIG. 2.—Demand pacing. A basic sinus rhythm is present, rate 67 to 72 a minute. The pacemaker rate is 59 a minute, and is manifest after atrial or ventricular extrasystoles in the abortive compensatory pause. When the sinus rate is fastest, lead V1, the pacemaker is inhibited.

FIG. 3.—Standby pacer stimuli in T waves. The rectangle below each diagrammatic electrocardiographic strip shows the relation of the Ectocor principle to premature beats. Each arrow represents a pacer stimulus. The striped areas represent the pacer "refractory period". In (a) the extrasystole occurs after the end of the pacer refractoriness; it is sensed and the stimulus is issued; the subsequent cycle is the longest possible or "escape cycle". In (b) the extrasystole occurs shortly before the end of the pacer refractory period and is not sensed; the stimulus is then delivered after the expiration of the fixed escape cycle, at point X. If AQ (the coupling interval) + $Q-T_{max} \geq AX$, the impulse at X may fall in the vulnerable period of the ventricle.

sensing such a signal, the Medtronic Demand unit and the earlier Cordis Ventricor II unit are inhibited, and the basic pacer cycle—the "escape cycle"—starts again. This is true "demand" action. These sensing units may, however, respond to extraneous electromagnetic signals. Enough external signals may be present in some circumstances to affect the efficiency of the system (A. J. Adducci, Medtronic Inc., Minnesota, personal communication, 1967). The newer Cordis Ectocor unit avoids this particular problem. It is designed to emit an impulse on detecting a "QRS"—either true or false. If the QRS is genuine, the impulse will be ineffective because the ventricles are refractory. Since the pacemaker output is not suppressed, this is not "demand" action in the strict sense. A pacer refractory period is incorporated (Fig. 3), lest the unit responds to frequent impulses with untoward effects, and during this the pacemaker will not respond to any impulse. In the unit used in this patient the refractory period was 0.50 sec., but in later models is 0.40 sec. The maximum or "escape" interval of the Ectocor is fixed at 0.86 sec.

The duration of the pacer refractory period effectively prevents the unit from discharging into the vulnerable phase of any beat that the unit has, in fact, detected. Nevertheless, it is still possible for the Ectocor unit to deliver its impulse into the T wave of a premature beat that it did not detect because it began during the pacer refractory period.

The circumstances in which an Ectocor pacer may discharge its impulse into the T wave of a premature beat are illustrated diagrammatically in Fig. 3. In Fig. 3a the extrasystole begins *after* the end of the refractory period of the pacer. The stimulus is evoked, but is ineffective because of ventricular refractoriness. In Fig. 3b, on the other hand, the extrasystole begins shortly *before* the end of the pacer refractory period and is, therefore, not detected. The pacer stimulus then occurs after the maximum delay permitted, i.e. after 0.86 sec. Under this circumstance the pacer stimulus may invade the T wave of a suitably timed premature beat. It must also be pointed out that the pacemaker detects an intracavitary electrocardiogram. In particular, the magnitude and rate of change of

potential of the endocardial electrocardiogram are not accurately reflected in the surface tracing. This may lead to some beats being detected and others not, even though they have the same prematurity when judged by standard electrocardiograms.

Vulnerable Phase. The question now arises whether a stimulus delivered into the T wave of a premature beat may fall upon the vulnerable phase. Lown *et al.* (1963) have provided data for the timing of the vulnerable phase of sinus beats in 5 mammalian species, including subhuman primates, and it may be assumed that their evidence could also apply to man. Their derived relation for the vulnerable phase is that the ratio of "Q to shock" interval to Q-T duration equals 0.65 to 0.80. Now, if the sum of the pacer refractory period and 80 per cent of the longest observed Q-T duration of any beat is equal to or greater than the escape interval of the unit, then it is possible that the pacemaker impulse may be delivered into the vulnerable phase of the ventricle. Furthermore, there is evidence that the vulnerable phase of *extrasystoles* extends later than is the case with sinus beats (Wégria, Moe, and Wiggers, 1941), and may extend to the end of the T wave (Fig. 1c in Dressler, 1964).

The possibility exists that a unit employing the Ectacor principle may deliver its impulse into the vulnerable phase of an extrasystole, if the sum of the pacer refractory period and the longest Q-T duration of the extrasystoles exceeds the pacer escape interval. In this patient, the longest Q-T of an extrasystole was 0.50 sec. As the pacer refractory period was 0.50 sec., it is clearly possible that a pacemaker stimulus did fall in the vulnerable phase and provoke a tachycardia.

The possibility that a pacemaker stimulus may induce a serious rapid ventricular arrhythmia has produced divergent opinions. The evidence for and against has been recently reviewed by Bilitch *et al.* (1967) who added 5 cases of their own of fatal ventricular fibrillation during competition between intrinsic and electric rhythms. In one of their cases, the precipitation of ventricular fibrillation by a critically timed pacemaker impulse is convincingly shown. The evidence incriminating pacemaker impulses delivered in the vulnerable phase, in some

cases at least, must now be regarded as overwhelming. The present case emphasizes that a similar effect may also result from a standby pacemaker.

SUMMARY

A case is described in which there is a strong inference that minor Adams-Stokes attacks were provoked by a standby pacemaker of particular design characteristics. Replacement by a unit of different design was followed by disappearance of attacks. The designs of the types of "ventricular-synchronized" units currently available are briefly analysed. It is pointed out that a unit that includes a pacer refractory period may discharge its impulse into the vulnerable phase of the ventricle after a critically timed premature beat. This possibility may be checked in other cases by analysis of the pacer refractory period and escape interval, and the Q-T durations of the intrinsic beats.

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