Supernormal Phase of Conduction in Human Heart Demonstrated by Subthreshold Pacemakers

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Subthreshold stimuli may produce ventricular excitation when they fall within a limited period of the cardiac cycle. This phenomenon, known as the supernormal phase of conduction (Adrian, 1921), was shown in the anaesthetized animal to be from the beginning of the descending limb of the T wave, and the beginning of the U wave (Hoff and Nahum, 1938). Its presence in the human heart was first reported by Lewis and Master (1924). Wenckebach and Winterberg (1927) later doubted its existence, but several authors have since confirmed its presence (Schert and Schott, 1939; Kline, Conn, and Rosenbaum, 1939; Pick, Langendorf, and Katz, 1962; Soloff and Fewell, 1960; Linenthal and Zoll, 1962; Burchell, Connolly, and Ellis, 1964; Linenthal and Zoll, 1965).

This period of hyperexcitability of the heart has been invoked to explain the genesis of a number of ventricular dysrhythmias, including those extrasystoles occurring early in diastole immediately after the T wave (Mack, Langendorf, and Katz, 1947; Soloff and Fewell, 1960; Burchell, 1963).

In this paper, we show the supernormal phase of conduction of the human heart by observation on the effects of subthreshold artificial stimuli from failed implanted pacemakers. The duration of this phase is determined and its relation to ventricular extrasystoles established.

Subjects and Methods

Observations were made in 4 patients with implanted cardiac pacemakers, in whom the unit had failed. Failure was due to fracture of one of the electrodes in 2 patients and to pacemaker malfunction in the other 2.

All the patients, after a trial of medical treatment, had epicardial electrodes implanted in the antero-lateral wall of the left ventricle (midway between the atrioventricular groove and the apex). These were attached to fixed rate pacemaker units.

Our observations were made from long strips of electrocardiograms. Timing of events was from the beginning of the QRS complex. The onset of the supernormal phase of conduction was taken as the earliest point where the pacemaker stimulus would cause ventricular depolarization and its end where this would no longer occur. The time relation of spontaneous extrasystoles was also noted. Pacemaker stimuli and ectopic beats were superimposed onto an accurately reproduced electrocardiographic complex. This was done with an estimated error of a maximum of 10 msec.

Brief case histories of the 4 patients are given below.

Case 1. A 29-year-old man, with two years' history of Adams-Stokes attacks, had lived for many years in an area where Chagas' disease is endemic, and the complement-fixation test for Chagas' disease (Machado-Guerreiro reaction, modified by Freitas) was positive. A diagnosis of chronic Chagas' cardiomyopathy complicated by AV block was made. Despite isoprenaline, the frequency and severity of Adams-Stokes attacks necessitated the implantation of a cardiac pacemaker. The unit failed after 22 months.

Case 2. A 2-year-old boy, with an 8-month history of Adams-Stokes attacks, has been described elsewhere as a familial-congenital complete AV block (Veracochea et al., 1967). Cardiac pacing became necessary because of attacks. Ten months after implantation, pacing ceased because of fracture of one of the electrodes near its insertion into the myocardium.

Case 3. A 45-year-old woman had intermittent complete AV block complicated by Adams-Stokes attacks. The aetiology was not clearly established, but she was probably a case of primary heart block (Zoo and Smith, 1963). The pacemaker failed 20 months after implantation.

Case 4. A 62-year-old woman with ischaemic heart disease developed heart block. Her rhythm varied between complete AV block, 2:1 block, and occasionally
normal conduction. The cardiac pacemaker was implanted because of the increased frequency of Adams-Stokes attacks. It failed 18 months later due to fracture of one of the electrodes.

**RESULTS**

**Case 1** (Fig. 1). The spontaneous ventricular activity of the patient was an idioventricular rhythm of 44 a minute, which was frequently interrupted by coupled ventricular ectopic beats and pacemaker-induced beats. Most artificial stimuli were not effective (open circles); however, those that fell within a narrow range (50 msec.) after the peak of the T wave were capable of originating a response (closed circles). This area was shown by superimposition to be between 420 and 540 msec. after the beginning of the idioventricular QRS complexes, with a duration of 120 msec. Ventricular ectopic beats (X) appeared towards the end of this time interval, occurring from 470 and 540 msec. after the onset of the QRS.

**Case 2** (Fig. 2). The tracing shows complete AV block with a ventricular rate of 42 a minute, and atrial rate of 166. The pacemaker stimuli occurred at a rate of 93 a minute; however, most of them were not effective. Nevertheless, those falling at the end of the T wave, between 470 and 510 msec. after the onset of the QRS, were followed by a response. The figure shows an area in which a subthreshold stimulus is capable of originating a response.

**Case 3** (Fig. 3). This figure shows the effect of a subthreshold pacemaker in a patient with intermittent AV block. The tracing was obtained during temporary resumption of spontaneous sinus rhythm. The superimposition shows a supernormal phase from 420 to 595 msec. after the onset of the QRS. The period where ventricular ectopic beats occur extends from 535 to 585 msec. When the ventricle was “captured” by the subthreshold stimulus falling within the supernormal phase, the

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**Fig. 1.**—Electrocardiogram of Case 1, which shows subthreshold pacemaker stimuli. All the strips (1 to 5) are of lead III, taken at different times. Open circles represent the ineffective artificial stimuli, and closed circles the effective ones. The ventricular ectopic beats are indicated by X. In strip 1, only the second artificial stimulus which fell at 420 msec. from the beginning of QRS was effective. The latest effective one fell at 540 msec. on strip 2. Strips 3 and 4 show other pacemaker-induced beats on the descending limb of the T wave, and some ventricular ectopic beats are seen on strips 3, 4, and 5.

* In all the figures the supernormal phase of conduction is shown in the diagram by the horizontal hatched area and the period where spontaneous ectopic beats occurred by the vertically hatched area.
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next P waves were not followed by a ventricular response for one to three cycles.

Case 4 (Fig. 4). This figure shows 2:1 AV block, and artificial stimuli of a subthreshold pacemaker, most of which are not effective. A supernormal phase was shown between 405 and 495 msec. Ventricular ectopic beats appeared in a short period after the zone which was mapped by the subthreshold pacemaker as the supernormal phase, and occurred between 510 and 565 msec. after the QRS. The duration of the observed supernormal phase was 90 msec., and the area in which ventricular ectopic beats were present lasted 55 msec.

DISCUSSION

There has been controversy with regard to the existence of a supernormal phase of conduction in man (Lewis and Master, 1924; Wenckebach and Winterberg, 1927; Soloff and Fewell, 1960; Lienenthal and Zoll, 1962; Burchell et al., 1964).

On clinical grounds, the occurrence of coupled ventricular ectopic beats at a fixed interval after the previous QRS complexes suggests an increased excitability during this period. Hence subthreshold spontaneous ectopic activity can manifest itself in this phase of lowered threshold. The relation between coupled extrasystoles and supernormality has been stressed by several authors (Mack et al., 1947; Scherf and Schott, 1953). A direct demonstration of the supernormal phase of conduction of the human heart can only be made by artificial subthreshold stimulation of the ventricles. The phenomenon is most commonly observed during the investigation of failure of pacing (Siddons and Sowton, 1967). Observations on ventricular excitability made by introducing variable strength stimuli from an external pacemaker in a patient with an epicardial electrode suggested the presence of a supernormal phase of conduction (Soloff and Fewell, 1960). Other authors have reported similar observations in patients with implanted pacemakers and epicardial electrodes (Dressler, Jonas, and Schwartz, 1965; Dolar and Cammili, 1968).

The supernormal period cannot, however, always be demonstrated with endocardial electrodes. Feldman (1963) was not able to do so, whereas Castellanos et al. (1966b) were. All 4 cases reported here, in which the supernormal phase of conduction was showing, had epicardial electrodes. Other authors have pointed out that this phase does

FIG. 2.—Electrocardiogram of Case 2 showing complete AV block and subthreshold pacemaker stimuli, most of them ineffective. Stimuli falling at 505, 510, 490, 470, and 480 msec. from the onset of QRS complexes are effective.
not exist in the normal human heart (Brooks et al., 1955).

The evidence so far is in favour of the existence of a phase of increased excitability after ventricular repolarization, which can be demonstrated either by artificial subthreshold stimulation or by determining the time of the cardiac cycle when coupled extrasystoles appear. Premature ventricular beats occurring late in diastole are probably caused by another mechanism (Castellanos et al., 1966a). The supernormal phase of conduction coincides with the negative after potential and has been shown in dogs to last from 50 to 200 msec. (Scherf, 1960). Its duration in man ranges from 165 to 195 msec. Ventricular ectopic beats which occur late in diastole, after the end of supernormality (260 msec. after the end of the T wave), are probably related to an increased excitability due to the Wedensky effect (Castellanos et al., 1966a).

The duration of the supernormal phase of conduction and its time of onset have been shown to be related to the strength of subthreshold stimuli by Soloff and Fewell (1960); they showed that the minimal current strength capable of originating a response in a patient with an epicardial electrode was 2-6 milliamperes, and excitation then occurred within 0.04 sec. after the peak of the T wave. When the current was increased to 7 milliamperes, ventricular excitation occurred within 0.02 sec. after the peak of the T wave and also in the PR segment. Changing the rate of the electrical pacemaker from 40 to 120 a minute did not vary the time within the cardiac cycle of the appearance of electrically produced ventricular excitation (Soloff and Fewell, 1960). In our cases, a relation between the strength of the stimuli and the time of onset of the supernormal phase of conduction could not be established since all 4 patients had implanted fixed rate pacemakers.

This mechanism may, however, explain why in Cases 1, 3, and 4 the artificially induced beats began to appear earlier in the supernormal phase of conduction than the spontaneous ectopic beats. This latter was perhaps induced by less strong impulses than the artificial beats.

The time of onset of the supernormal phase of
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conduction and its duration could be determined in all 4 cases. In Case 3, supernormality lasted up to 595 msec. after the previous conducted beat (approximately 240 msec. after the end of the T wave). This figure is very close to that reported by Castellanos et al. (1966b). All premature beats appeared within the period of supernormality, or a few msec. after its end (Case 4).

In patients in whom the electrocardiogram is recorded through a catheter electrode, the timing of the supernormal phase of conduction may be influenced by the relative sites of origin of spontaneous and paced beats. When the tip of an endocardial electrode is positioned close to the idioventricular focus, the depolarization wave of a spontaneous beat will pass the electrode earlier than when the two points are far apart. This may explain why the ventricular ectopic beats appear later than the paced beats.

It is important to emphasize that the supernormal phase of conduction may present different values according to the aetiology of the AV block. Thus, in the patients with Chagas' cardiomyopathy (Case 1) and primary AV block (Case 3), this phase lasted longer (120 and 175 msec., respectively), than in the patients with familial congenital AV block (Case 3) and ischaemic AV block (Case 4) in whom it was only 40 and 90 msec., respectively.

This prolonged phase of supernormality in patients with Chagas' heart disease may account for the frequency and variety of ventricular ectopic activity which may lead to the occasional appearance of ventricular fibrillation.

**SUMMARY**

A supernormal phase of ventricular excitation in man has been shown during follow-up of 4 patients with failed pacemakers. Subthreshold stimuli were only effective if they fell on the descending limb of the T wave or a few milliseconds later.

The duration of the supernormal phase of conduction and its time of onset is directly related to the strength of the subthreshold stimulus.

Its timing may be influenced by the relative sites of origin of spontaneous and paced beats.

The aetiology of the heart block may alter the length of the supernormal phase.
Ventricular ectopic beats occurred only late in diastole near the end of supernormality and in one case a few milliseconds later.

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REFERENCES