Supernormal phase of AV conduction

A study during heart block and endocardial pacing

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A distinct, well-defined phase has been identified in a patient with heart block during which sinus impulses were conducted to the ventricles. This phase persisted during pacing but its onset and termination were delayed. No significant difference was found at varying pacing rates. On four occasions while pacing, two consecutive sinus impulses were conducted to the ventricles but this was not seen during the long recordings taken when in spontaneous idioventricular rhythm. This phase of conduction cannot be satisfactorily explained either by a vagal mechanism or refractory variations within the AV junctional tissue, and lends strong support to the recently challenged concept of a supernormal phase of conduction.

The supernormal phase of conduction was first described by Adrian and Lucas in 1912 from their observations on nerve and cardiac muscle in the frog. This early work was confirmed by Hoff and Nahum in 1938 who defined the phase as starting on the descending limb of the T wave and persisting until the beginning of the U wave. The first demonstration of the phenomenon in the human heart was reported by Lewis and Master in 1924. Since this time a supernormal phase of AV conduction has been increasingly recognized, and Pick, Langendorf, and Katz (1962) and Lepeschkin and Kimura (1962) reviewed the published reports, adding personal cases to the series. A supernormal phase of intraventricular conduction has also been claimed, and examples of apparent enhanced conduction in the right bundle-branch (Contro, Magri, and Natali, 1956; Wellens, 1969; Mihalick and Fisch, 1970) and the left bundle-branch (Simon and Langendorf, 1944; Scherf and Scharf, 1948; Wellens, 1969) have been recorded. The application of cardiac pacing has enabled the phases of ventricular excitability to be more accurately studied, and many workers have confirmed the existence of an apparent supernormal phase in this way (Soloff and Fewell, 1960; Linenthal and Zoll, 1962; Burchell, 1963; Hernandez-Pieretti, Morales-Rocha, and Barcelo, 1969). The concept has not, however, been unchallenged, and Moe, Childers, and Merideth (1968) have presented alternative explanations which did not involve a supernormal phase of conduction.

Received 24 October 1972.

Three requirements are necessary to identify such a phase: a situation of impaired conduction must be produced or exist spontaneously, with a primary focus initiating ventricular depolarization during which the effects of further stimulation are observed. Both the initiating pacemaker and the focus of testing stimulation may be either spontaneous or artificial. Electrocardiographically, the phase will be recognized by unexpected periodic conduction of a subthreshold stimulus or conduction with less delay than anticipated. The timing and duration of the phase have been shown to be dependent upon the strength of such subthreshold stimulation (Soloff and Fewell, 1960) but may also be influenced by the proximity of the two stimulating foci; thus the onset may be earlier when the wave of depolarization passes an adjacent rather than a distant focus.

A patient has been investigated who had intermittent sinus conduction during an apparent supernormal period while in heart block and during endocardial right ventricular pacing (Abrams-Lucas inductive method). This unusual situation enabled the timing and duration of this period to be accurately measured.

Subject and method

The patient was a 69-year-old man, who had suffered an uncomplicated myocardial infarction 5 years previously. He had experienced mild angina of effort since this time, controlled by sublingual nitroglycerin. He was found to have a bradycardia of 40/min, the electrocardiogram revealing complete heart block with occa-

sional sinus conduction. Temporary transvenous pacing was instituted and 10 days later was converted to the permanent Abrams-Lucas inductive method. At no time had the patient received digoxin.

All measurements were made two weeks after the insertion of the pacemaker to avoid any early alterations in the refractory periods, as observed by Burchell (1963). The pacemaker was turned off for 30 minutes to establish a stable rhythm before the observations were made. These non-paced records were compared with those taken before pacemaker insertion to ensure that the operation itself had in no way affected the period during which conduction was seen to occur; no such change was evident. Long electrocardiographic records were then taken (simultaneous leads II and V₁) at different pacing rates: 40, 42, 47, 53, 56, 61/min. The intervals from the pacemaker stimulus or the beginning of the QRS in spontaneous rhythm, to the P waves were measured. The onset of the phase was taken as the earliest point at which the P waves were able to elicit ventricular depolarization, and the end at the point where sinus conduction failed. Examples of the records are presented in Fig. 1.

Results

The results are shown in Fig. 2. All measurements, which were made by the same observer and repeated, were taken from the apex of the P waves and accuracy of the readings was not attempted to greater than 0.02 sec.

The duration of the phase in spontaneous idioventricular rhythm was 0.36 sec, with the onset at 0.32 sec and termination 0.68 sec. At pacing rates of 40, 42, 47, 53, 56, and 61/min the onset of this phase was 0.42, 0.38, 0.40, 0.40, 0.40, 0.38 sec, respectively, the termination 0.80, 0.84, 0.82, 0.82, 0.81, 0.82 sec, and the duration 0.38, 0.46, 0.42, 0.42, 0.41, 0.44 sec. Thus there did not appear to be a constant and significant difference between the timings of this phase at different pacing rates. The average onset during pacing was 0.40 sec, average termination 0.82 sec, and average duration 0.42 sec.

Discussion

Analysis of these records taken during spontaneous rhythm and at differing right ventricular endocardial pacing rates has revealed a clearly defined phase during which P waves are conducted to the ventricles with the pattern of right bundle-branch block. This may be due to bilateral bundle-branch block with intermittent conduction through the left bundle-branch pathways, or AV junctional block which allows the passage of critically timed sinus impulses to reach the ventricles disclosing established right bundle-branch block.

The timing of the RP intervals of those sinus impulses whose passage is permitted suggests that

FIG. 1 (A) Simultaneous recordings from leads II and V₁, and II and V₆ while in spontaneous idioventricular rhythm. The majority of P waves are blocked, but occasional sinus conduction is evident.

FIG. 1 (B) Simultaneous recordings from leads II and V₁, demonstrating blocked and conducted P waves at the paced rates studied.

FIG. 2 Timing in 1/100 sec of the intervals (pacemaker stimulus to P wave) studied at the various rates. Zero time length denotes the pacing stimulus, the end of the blocks illustrating the pacing cycle lengths. Dots indicate non-conducted and crosses conducted P waves. The limits of the phases are defined by the vertical lines within the blocks.

conduction has occurred during the so-called supernormal phase and is an example of Type B as classified by Pick *et al.* (1962). There appears to be no significant difference between the times of onset, termination, and duration of these phases at the different pacing rates studied, but when compared with the timings in spontaneous rhythm, the average onset (0.40 sec) and termination (0.82 sec) are later and the average duration marginally prolonged. As the foci controlling the heart in both spontaneous and paced situations were situated in the ventricles and stable, there are two factors that may account for apparent alteration in the timing of this phase: firstly the time at which the ventricular ectopic stimulus reaches the junctional tissue during or after the inscription of the QRS complexes, and

secondly the time required for conduction within the AV node, where it is extinguished. If the time taken for the latter is constant in these two situations, then the site of the foci within the ventricles will determine the onset of this phase when measured from the beginning of the QRS; thus a focus discharging close to the junctional tissue will produce a wave of depolarization followed by a possible phase of enhancement of conduction earlier than that evoked by a more distant focus.

The concept of a supernormal phase of AV conduction has recently been challenged by Moe *et al.* (1968) and they have proposed alternative mechanisms, capable of intermittent AV conduction. These included ventriculophasic (vagal) depression of nodal conductivity, alterations in the refractory periods of

the junctional tissue due to pre-excitation, and alternation between dissociated conducting pathways within the AV node. This arrhythmia has therefore been reappraised to consider the alternative explanations that Moe *et al.* submit.

The phasic influence of vagal activity on sinus rate and AV nodal conduction has been studied by Roth and Kisch (1948) and Rosenbaum and Lepeschkin (1955). The baroreceptor response to each pressure pulse evokes a surge of increased vagal activity which may be manifest as ventriculophasic sinus arrhythmia. This increased activity occurs 0.5–0.6 sec after inscription of the QRS and persists for a further 1.5–1.6 sec when it falls to its resting level. The delay is due to the interval between the QRS and the systolic pulse wave, the reflex time, and the period between vagal excitation and its clinical manifestation. Sinus impulses falling after the absolute refractory period of an idioventricular complex but before the increased vagal activity it produces, may traverse the AV node and suggest a supernormal phase of conduction. The phase of conduction demonstrated extends beyond the point at which the vagal surge has started and no example of conduction was found towards the end of the cycle length when vagal activity should have fallen to its lowest level. Though minor fluctuations in sinus rate suggested some vagal influence after ventricular ejection, its aetiological role in the genesis of the arrhythmia was not convincing.

A concealed 2:1 AV nodal block with a lower pacemaker controlling the ventricles may closely resemble the phenomenon of supernormal conduc-

tion. Under such circumstances, a prolonged refractory period within the AV node would be produced by the first sinus stimulus and the passage of the second sinus impulse would be blocked. If retrograde penetration of the AV node from the lower pacemaker focus was also blocked, this refractory period would not be affected by the subsidiary pacemaker and only alternate P waves would be capable of transmission to the ventricles. Normal conduction would occur if the conducting pathways had not been rendered refractory by the lower pacemaker and supernormality need not be considered. The theory would be tenable if it could be shown that the conducted P waves were always those that initiated the presumed refractory prolongation, rather than those falling during it. If they only delineated the onset of the supernormal phase and did not fall within the latter part of the cycle, the end of the phase would have been artificially shown by the earliest P waves which could not pass the region of intranodal block; thus this period would never have been effectively tested. However, part of the arrhythmia (Fig. 3) showed that P waves which should have been blocked within the AV node were at times able to achieve ventricular capture. Clearly therefore, this is not a satisfactory explanation. If, however, it were assumed that retrograde penetration of the lower pacemaker stimulus into the AV node was able to pre-excite this region and effectively retract its refractory period, then under appropriately timed circumstances, the alternatively blocked P waves would be capable of conduction and capture. Applying these postulates, it has not been possible

FIG. 3 Consecutive recording lead II. Alternate P waves (marked 'a' and 'b') are both capable of conduction to the ventricles at some stage during the rhythm strip (see text).

to produce a temporal scheme which accurately explains the observed arrhythmia. Moe *et al.* (1968) have stressed that alterations in nodal conductivity, pacemaker frequency, or the refractory periods could produce striking changes in the pattern of AV response. No such evidence has been found in this

case while pacing at different rates, and it appears improbable, therefore, that such refractory variations account entirely for this arrhythmia.

On four occasions, once while pacing at 53/min and three times at 61/min, two consecutive sinus impulses were conducted to the ventricles (Fig. 4).

FIG. 4 Examples of conduction of two consecutive sinus impulses during pacing (see text).

FIG. 5 Two consecutive sinus impulses conducted during pacing. PM indicates the pacemaker stimulus, P the first sinus impulse, R the first conducted ventricular complex, P' the second sinus impulse, and R' the second conducted ventricular complex. The calculated intervals are presented in the Table.

This was never seen during the long recordings taken in spontaneous rhythm. Such consecutive conductions might suggest retrograde resetting of the AV nodal refractory period but it has not been possible to construct an idealized scheme to explain this arrhythmia on these principles. If the concept of supernormality is accepted, an explanation can be found. The earlier the P waves fall within the conducting limits of the T waves of the paced complexes the longer tends to be the PR interval of the complex it produces; this is the well-recognized effect of stimulation within the relative refractory period. This latency permits the next P wave to fall within the supernormal phase of the conducted complex and itself be conducted, but with no delay. When the initiating P wave of this doublet falls later after the paced beat, so its conduction is less delayed, and the second P wave falls outside the supernormal phase of the conducted complex and is not transmitted. These intervals for the three examples at a paced rate of 61/min have been calculated (Table) and compared with six other combinations not demonstrating consecutive conductions (Fig. 5).

TABLE Calculated intervals (in 1/100 sec) for 3 examples of consecutive sinus conduction compared with 6 instances of unsuccessful second sinus conduction (see Fig. 5 and text)

PMP	PR	RP'	P'R'
32	34	68	18
34	34	64	18
34	36	60	20
36	30	70	—
38	24	72	—
40	26	76	—
42	24	72	—
46	22	80	—
48	20	72	—

The inability to explain these events by methods other than those invoking a supernormal phase of conduction strongly suggests that this mechanism is responsible for all the arrhythmias observed in this patient. It must be concluded, therefore, that the constant phase of the cardiac cycle in which sinus capture was noted is further evidence of the existence of such a supernormal phase of AV conduction. The differences in timing that were found between the spontaneous and paced rhythms can be readily explained by intraventricular conduction delay due to the site of the ectopic foci, and do not detract from the concept of supernormality.

The author wishes to thank Dr. R. E. Nagle for permission to investigate his patient and Dr. Schott for his valuable suggestions during the preparation of the paper, and acknowledges support from the Endowment Fund, Medical Research Committee, United Birmingham Hospitals.

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