Phenomenon of Supernormality in the Human Heart

By Rashid A. Massumi, M.D., Ezra A. Amsterdam, M.D., and Dean T. Mason, M.D.

SUMMARY
This report documents five cases of supernormal (SN) conductivity in the sense of unexpected normalization of the previously existing bundle-branch block (BBB) and three cases of SN excitability in which impulses delivered to the ventricles propagated to the myocardium only during a well-defined interval in the cardiac cycles corresponding to the U waves of the preceding beat. In none of the patients could SN be abolished by atropine, suggesting absence of an important vagal role. In the five patients with SN conductivity there were three examples of right BBB in which spontaneous, as well as electrically induced premature atrial beats, were followed by normal QRS complexes only if they reached the right bundle during its SN phase. In the other two patients with bilateral BBB, strategically placed sinus P waves and electrically induced atrial impulses were conducted to the ventricles with normal P-His and His-Q intervals. Analysis of the electrocardiograms and His bundle electrograms indicated that SN conductivity resided in the bundle branches, and not in the A-V node. In one of two patients with bilateral BBB and SN conductivity in the right bundle branch, the SN-conducted beats occurred after both left and right ventricular escape beats, but were coupled to the preceding escape beats by longer intervals after left ventricular escape beats than after right ventricular escape beats. The difference was accounted for by the later activation of the right bundle branch when the preceding escape beat originated from the contralateral left ventricle. These observations favor an electrophysiologic explanation for the phenomenon of supernormality which is most probably related to the increased voltage of the transmembrane action potential in the period commonly known as the period of negative afterpotentials. This study provides no support for the hemodynamic or vagal theory of supernormality.

Additional Indexing Words:
Supernormal conductivity
Supernormal excitability
Bundle-branch block
Bilateral bundle-branch block

SUPERNORMAL CONDUCTION, or more precisely, the unexpected improvement or normalization of conduction in the face of a persistently depressed atrioventricular (A-V) or intraventricular (I-V) conduction has been observed clinically in a variety of circumstances. SN excitability, in the sense that a subthreshold stimulus produces a propagated impulse only when it occurs in a well-defined period of the ventricular cycle, has also been described in the human heart. Much doubt has, however, been expressed as to the very existence of supernormality, and alternative explanations have been advanced. It should be noted that, in clinical electrocardiography, it may not always be possible to ascertain whether an observed SN phenomenon in reality pertains to conductivity or

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excitability. This is so because, on the one hand, the fibers of the conduction system must be excited before they can conduct. On the other hand, an impulse originating supernormally somewhere in the Purkinje system must be conducted out into the surrounding myocardium before it can give rise to a manifest QRS complex. Therefore, it may be stated that, unlike the physiologist working with a nerve or myocardial fiber and using precise tools with which to measure conduction velocity or to deliver stimuli of varying strengths, the clinician's use of the terms conductivity and excitability is based on whether he observes the phenomenon of supernormality in the parameters of A-V and I-V conduction or in the appearance of manifest QRS complexes after discharge of impulses which have proved ineffective elsewhere in the cycle. Nonetheless, in describing the cases in the present report, the common practice of separating SN conductivity from SN excitability will be followed.

The purpose of this article is to present our experience with eight cases in which SN conductivity or excitability have occurred and to indicate that the phenomenon does indeed exist and must be considered causal in producing certain unusual electrocardiographic features not explicable by any other phenomenon.

Methods

Eight patients, seven males and one female, were studied (Table 1). Seven patients had clear-cut evidence of heart disease and one was suspected of having angina pectoris. Of the eight cases studied, the first five are placed in the category of SN conductivity, while the last three conform to the commonly described SN excitability. Observations were based on routine electrocardiograms and long rhythm strips in all of the cases, while His bundle electrography was performed in four. Supernormality was considered to be present when conduction or excitation was absent both before and after a definite interval near the end of the preceding T wave. An attempt was made to gather long rhythm strips covering several hours of observation and make certain that coincidence did not play a role. Recordings were made both with the usual direct-writing electrocardiograph machines and with photographic recorder, Electronics for Medicine DR-12. All His bundle electrograms were obtained using the percutaneous-femoral approach and recordings were made at paper speed of 100 mm/sec.

Supernormality was spontaneous in five cases, and related to electrically induced premature atrial beats in three cases. In two cases of right BBB and the two cases of bilateral BBB with SN

<table>
<thead>
<tr>
<th>Case</th>
<th>Age (yr)</th>
<th>Sex</th>
<th>Cardiac diagnosis</th>
<th>ECG diagnosis</th>
<th>Supernormality</th>
<th>Interval from preceding Q to onset of SN (sec)</th>
<th>Duration of SN (sec)</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>61</td>
<td>M</td>
<td>Aortic regurgitation</td>
<td>Rate-related RBBB</td>
<td>Cond, RBBB</td>
<td>0.54–0.62</td>
<td>0.08</td>
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<td>2</td>
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<td>F</td>
<td>Ischemic heart disease</td>
<td>Persistent RBBB</td>
<td>Cond, RBBB</td>
<td>0.50</td>
<td>?</td>
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<tr>
<td>3</td>
<td>55</td>
<td>M</td>
<td>? Angina pectoris</td>
<td>Persistent RBBB</td>
<td>Cond, RBBB</td>
<td>0.63</td>
<td>?</td>
</tr>
<tr>
<td>4</td>
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<td>M</td>
<td>Heart block</td>
<td>Bilateral BBB</td>
<td>Cond, RBBB</td>
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<td>0.06</td>
</tr>
<tr>
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<td>82</td>
<td>M</td>
<td>Ischemic heart disease</td>
<td>Bilateral BBB</td>
<td>Cond, LBB</td>
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<tr>
<td>6</td>
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<td>M</td>
<td>Ischemic heart disease</td>
<td>Bilateral BBB</td>
<td>Excit RV pacemaker</td>
<td>0.62–0.64</td>
<td>0.02</td>
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<tr>
<td>7</td>
<td>78</td>
<td>M</td>
<td>Hypertensive heart disease</td>
<td>Bilateral BBB, depressed sinus node</td>
<td>Excit RV pacemaker</td>
<td>0.68–0.70</td>
<td>0.02</td>
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<tr>
<td>8</td>
<td>39</td>
<td>M</td>
<td>Acute inferior wall infarct</td>
<td>Parasystole</td>
<td>Excit, inferior fascicle LBB</td>
<td>0.58–0.64</td>
<td>0.06</td>
</tr>
</tbody>
</table>

**Table 1**

Biographic and Clinical Data in Eight Cases Showing SN Phenomenon

*Abbreviations: R = right; L = left; BB = bundle branch; RV = right ventricle; BBB = bundle-branch block; Cond = conductivity; Excit = excitability.*

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conduction through one bundle branch the phenomenon of supernormality was reproduced by coupling atrial stimuli to the preceding R wave in such a way as to reach the bundle branches during the SN phase, and thus be conducted. Atropine was administered intravenously in all the cases in order to ablate the vagal influence and study the effect on the appearance and frequency of SN beats.

Presentation of Selected Cases

Case 1

This 61-year-old man with aortic regurgitation and left ventricular failure was found to have a rate-related right BBB (fig. 1). QRS complexes were narrow and normal in configuration at heart rates under 60 beats/min (beats 1, 2, 4, 5, 6, and 11), and under 68 beats/min (beats 16, 18, 24, and 26) after the administration of intravenous atropine. At slightly higher rates RBBB was present (beats 7, 8, 9, and 12 before atropine, and 13, 14, and 19–22 after atropine). Frequent premature atrial beats were encountered in all tracings. Very early premature atrial or junctional beats were conducted aberrantly with RBBB configuration (beat 10) while slightly later premature beats were conducted normally (beats 3, 15, 17, 23, and 25).

Figure 2 (case 1) shows QRS configuration plotted against the preceding R-R intervals measured from the beginning of the preceding QRS to the beginning of the beat under consideration. To be noted is that the I-V conduction system or the ventricular myocardium is completely refractory to impulses arriving within 0.40 sec after the onset of the preceding QRS. With coupling intervals ranging from 0.40 to 0.52 sec, RBBB is present. The area of supernormality extends from 0.54 to 0.62 sec, while all beats occurring later than 0.62 but before 0.98 sec show RBBB. Normal QRS is again noted when coupling intervals exceed 0.98 sec.

Case 2

A.F. is a 72-year-old woman with a history of previous inferior wall myocardial infarction and the sequela of persistent RBBB. Figure 3 shows His bundle recordings for two control sinus beats and during right atrial pacing. It was noted that at the pacing rate of 124 beats/min alternate QRS complexes became normal. This was easily reproduced so long as the pacing rate was unchanged, but disappeared when the atrium was
paced at faster or slower rates. His bundle electrography shows that all beats are conducted from the atrium. They are preceded by atrial potentials and by His spikes (arrows). The P-R, P-His, and His-Q intervals using lead II as reference for the P and Q show only minimal prolongation of the P-His intervals during atrial pacing. The His-Q intervals remain fixed at 65 msec. This observation indicates that in normalized beats conduction velocity through the right bundle branch improved and equaled that present in the left bundle branch, resulting in excitation of the ventricular myocardium in a normal fashion.

Case 3

Case 3 was identical with case 2 except that the RBBB was not associated with any demonstrable heart disease. This patient was suspected of having angina pectoris.

Comments

The three cases described show SN conductivity in an already depressed right bundle branch. The improved conduction could not be explained either by a long delay in the A-V node or by an equal delay in the left bundle, for the expected prolongation of the P-R (and of His-Q in case 2) did not accompany the normalization of the QRS complexes. The failure of SN conductivity to disappear after administration of atropine suggests that the vagus did not play an important role.

Case 4

A 61-year-old man, previously in excellent health, developed LBBB approximately 1 year before admission and was found to be in complete heart block in May, 1971. This sequence of events suggested bilateral BBB as the basis for the complete heart block. In figure 4, leads I, II, and V₁ show the prevalent rhythm to be one of complete heart block with left ventricular escape beats having features of RBBB. Beats 4, 9, and 15 are preceded by a normal sinus P and a P-R interval of 0.17 sec. These are SN-conducted beats. The QRS complexes in these beats show complete LBBB and a normal axis. The normal P-R interval indicates unimpeaded conduction through the A-V node and the main His bundle, while the presence of LBBB signifies that propagation to the ventricle proceeded along the right bundle branch. The SN conductivity, therefore, occurred in the right bundle branch.

In figure 5, His bundle electrograms with both left ventricular escape beats (beats 1 and 2) and right ventricular escape beats (3 and 4) show His spikes after all blocked P waves. The P-His intervals are normal at 95 msec indicating unimpaired transmission through the A-V node. In the lower strip, electrical stimuli (S) are delivered to the right atrium resulting in premature atrial beats. All such P waves were blocked except those delivered 500–540 msec after onset of the preceding QRS. These were conducted to the ventricles with a P-R interval of 145 msec, P-His interval of 105, and His-Q interval of 40 (beat 7). The resulting QRS complexes had features of LBBB. Supernormality persisted after atropine, and in fact SN-conducted beats occurred more frequently.

In figure 6 (top), lead V₁ shows two SN-conducted beats (3 and 5). They are identical with respect to their P-R intervals and their QRS.

Figure 3

Case 2. His bundle recordings for two control sinus beats and during right atrial pacing.
However, they differ in one important parameter, namely their coupling intervals. Beat 3 is coupled to the onset of the preceding escape beat 2 (from the left ventricle) by 0.76 sec, while beat 5 is coupled to beat 4 (from the right ventricle) by 0.66 sec. This observation indicates that the supernormality which resides in the right bundle branch occurs 0.10 sec later, after a left ventricular QRS rather than after a QRS originating from the right ventricle. The drawings below the electrocardiogram are purported to explain the mechanism. A left ventricular QRS in the left-hand portion and a right ventricular QRS are followed by SN-conducted beats in the drawing of lead V1-marked ECG. Drawings marked TAP, LBBB, and TAP, RBBB portray transmembrane action potentials of the left and right bundle branches, respectively, and the related carotid pressure pulse is drawn beneath each of the two escape beats. In a left ventricular beat, activation of the right bundle is delayed and occurs near the end of the QRS, i.e., after the impulse has traversed the interventricular septum and reached the right ventricle. The TAP of the right bundle is therefore drawn later, with its upstroke beginning near the end of the QRS. The period of supernormality at the end of such TAP (marked SN) is therefore late in appearance, and consequently the period of SN conductivity in the right bundle branch is late. Conversely, in a beat originating from the right ventricle activation of the right bundle branch is early as is its TAP and its SN period. The difference of 0.10 sec in the coupling intervals of the two SN-conducted beats observed in the electrocardiogram (fig. 6, top) corresponds well with the delay in activation of the right bundle branch following a left ventricular escape impulse. The arterial pressure pulse, on the other hand, occurring after left ventricular contraction, will have an earlier onset after a left ventricular escape beat than after a right ventricular escape beat. It may be expected, therefore, that vagally mediated pressoreceptor reflexes related to ejection of blood into the aorta and the carotid arteries would also occur earlier. If the SN conductivity in this case was ventriculophasic, i.e., related in some way to pressoreceptor reflexes, it should have appeared earlier after left ventricular escape beats and later after right ventricular beats. In reality, however, the pattern was the exact opposite.

Comments

In this case, SN conductivity in the right bundle branch could not be explained on the basis of hemodynamic events and pressoreceptor reflexes, but was easily explained by attention to

Circulation, Volume XLVI, August 1972
SUPERNORMALITY IN THE HUMAN HEART

the electrophysiologic events and the transmembrane action potentials.

Case 8

Acute inferior wall myocardial infarction occurred in a 39-year-old man previously in good health. On the twelfth hospital day, and after an episode of chest pain, extrasystoles with aberrant but narrow QRS complexes, and not preceded by atrial depolarization, were noted. These beats were shown subsequently to arise from the inferior fascicles of the left bundle branch.

The extrasystoles (fig. 7) occurred at regular intervals of approximately 6 sec, and study of longer strips indicated the existence of a parasystolic focus discharging at the rate of about 60 beats/min. Beats under numbers 3 and 9, and marked by asterisks in each lead, possess QRS complexes of 0.11-sec duration with features of left-axis deviation and RBBB. To be noted is that the intervals by which they are coupled to the preceding normal QRS are either short, ranging from 0.56 to 0.65 sec, or long, between 0.88 and 0.98 sec. Several hours of observation and recording failed to disclose extra systoles occurring earlier than 0.56 sec or in the interval between 0.64 and 0.90 sec. Discharges occurring very late in the R-R cycle (beats under number 9) propagated to the myocardium because of complete recovery of the conducting tissues. Early discharges (beats under number 3) became manifest because of SN excitability in the focus. The fourth beat in the third strip is of sinus origin with minor aberrancy of its QRS because of its proximity to the preceding extrasystole.

Figure 8 shows His bundle electrogram in one sinus beat numbered 1 and one SN-conducted beat numbered 2. P-His and His-Q intervals in the control sinus beat are normal. The His spike coincides with the onset of the QRS in beat 2 indicating a focus equidistant between the ventricular myocardium and the main His bundle.

Figure 5
Case 4. His bundle electrograms with both left ventricular escape beats (beats 1 and 2) and right ventricular escape beats (3 and 4) showing His spikes after all blocked P waves. In the lower strip electrical stimuli are delivered to the right atrium resulting in premature atrial beats.
Case 4. (Top) Lead V₁ shows two SN-conducted beats (3 and 5). (Bottom) A left ventricular QRS in the left-hand portion and a right ventricular QRS are followed by SN-conducted beats in lead V₁. TAP LBBB and TAP RBBB portray transmembrane action potentials of the left and right bundle branches, respectively, and the related carotid pressure pulse is drawn beneath each of the two escape beats.

Figure 6

Taking note of the RBBB and left-axis deviation, the ectopic focus can be localized in the inferior division of the left bundle (large asterisk). From this focus, which is related to the area of the acute myocardial ischemic injury, impulses arise and propagate peripherally toward the myocardium of the left ventricle, and, centrally reaching the superior division (small asterisk 2), and a little later, the mainstem and the right bundle (small asterisk 3). The asynchronous and delayed activation of the myocardium supplied by the superior division of the left bundle branch accounts for the left-axis deviation, and the delayed activation of the right bundle branch explains the RBBB pattern.

Comments

In this case of acute myocardial infarction a parasystolic focus situated in the inferior division of the left bundle branch became active. However, impulses from this focus propagated antegrade to the surrounding myocardium and retrograde to the His bundle only during the SN phase or very late in the diastole period. It is very probable that supernormality involved excitability of the focus.

Case 5 was an example of supernormality in the left bundle branch in a case of complete heart block secondary to bilateral bundle-branch block. Cases 6 and 7 were examples of permanently implanted fixed-rate transvenous pacemakers in which pacemaker discharges were effective only when they appeared near the end of the T wave of the preceding escape beat.

Discussion

The concept of supernormality as introduced by Adrian and Lucas in 1912 applied
SUPERNORMALITY IN THE HUMAN HEART

Figure 7
Case 8. Leads II, F, and V1 show the extrasystoles occurring at regular intervals of approximately 6 sec; the longer strips indicate the existence of a parasystolic focus discharging at the rate of about 60 beats/min. Beats under numbers 3 and 9, and marked by asterisks in each lead, possess QRS complexes of 0.11 sec duration with features of left-axis deviation and RBBB.

to nerve and muscle. It was based on the observation that in nerve and muscle preparations propagation of one impulse through a depressed region causes the conductivity in that region “to pass through a course of recovery which includes a period of supernormal ease of propagation; a second disturbance entering the region during that period will be propagated over a greater distance than the first . . .” The authors described supernormality for both processes of excitation and propagation, and made a clear distinction between them. Nine years later Adrian found supernormality in the animal heart and described an “overswing” in the recovery curve of excitability during which subliminal stimuli became effective initiating a propagated impulse. The first human examples of supernormality were published by Lewis and Master in 1925. Review of these cases reveals that both displayed slow heart rates probably related to a malfunction of the sinoatrial node, and the paper was subsequently criticized by Wenckebach and Winterberg, as representing examples of A-V dissociation with capture beats, and not of heart block with supernormality.

The second report of supernormality in the human heart was by Ashman and Hermann in 1926, and this too was criticized by the same workers because of a similar misinterpretation. Ashman, working about the same time with the turtle heart, found supernormality both in excitability and conductivity and suggested that the supernormal conductivity may reflect nothing more than a supernormal ease with which conducting fibers are excited. In other words, SN conductivity may in reality be a manifestation of SN excitability. Following these early observations, interest in supernormality waned and only a few reports dealing with SN excitability in cat hearts and human examples appeared. The first report describing an SN phase of recovery in a bundle branch was that of Scherf and Scharf and the subsequent case published by Pick and Fishman. Many of these early human cases may find alternative explanations in light
of more recently acquired knowledge. It should be observed that as early as 1938, Hoff and Nahum noted in experiments in cat hearts that the SN-excitability period coincided with the U wave of the surface electrocardiogram which in turn was thought to be related to the negative afterpotentials of the kind recorded in nerve tissue. Similar speculations were made by Segers. This relationship was extensively reviewed by Lepeschkin and Rosenbaum. Simon and Langendorf also noted that in the presence of preexisting bundle-branch block a premature beat may display normal QRS configuration probably because of SN conductivity in the involved bundle branch. Similar observations had been made earlier by Wilson and Hermann and Hewlett who suggested an entirely different explanation. They thought that the normality of the QRS indicated that impulses arose from the interventricular septum and spread to both ventricles in quick succession, thus overcoming the effect of bundle-branch asynchrony. Clinical reports of supernormality continued to appear. In a review of the literature from 1923 to 1960, Pick, Langendorf, and Katz were able to cull 48 reported cases to which they added 11 of their own. Some of the cases from the literature had not been recognized as examples of supernormality, and, as indicated earlier, several others had been criticized for error of interpretation.

A new surge of interest in the phenomenon of supernormality from the midfifties onward

Figure 8

Case 8. His bundle electrogram in one sinus beat, numbered 1, and one SN-conducted beat, numbered 2. P-His and His-Q intervals in the control sinus beat are normal.
SUPERNORMALITY IN THE HUMAN HEART

273

came about as a result of the fresh interest in electrophysiology created by the microelectrode technic for recording transmembrane action potentials. This technic was originally developed by Ling and Gerard in 1949 for recording muscle fiber potentials and was subsequently adapted for the myocardium by Woodbury in the U.S.A. and Hodgkin in England (cited by Weidman).

This important technic opened a new avenue of investigation which made it possible to reexamine many of the electrophysiologic mechanisms in light of the events occurring at the level of the cell membrane. Thus, it was through this approach that Weidman was able to demonstrate in sheep Purkinje fibers a short period of fall or a dip (negative afterpotentials) in membrane electrical resistance corresponding to the region just preceding phase 4, or the electrical diastole. During this period excessive quantities of potassium ions may escape the interior of the myocardial cell leading to an unusually or supernormally high transmembrane potential, and consequently to enhanced excitability. The action potential resulting from excitation in this region displays a very high rate of rise, a phenomenon known to be associated with increased velocity of conduction. According to Hoffman enhanced excitability is found immediately at the end of repolarization. This region corresponds with Weidman's period of dip in membrane electrical resistance.

The advent of technics for pacing the human heart provided new opportunities for observing SN excitability. Thus, Lewis et al. were able to demonstrate this phenomenon in a few cases during cardiac catheterization in 1961. Linenthal, Zoll, and Blumgart were the first to demonstrate SN excitability in patients who had implanted pacemakers. Similar reports by Burchell et al., Dressler et al., and Siddons and Sowton described examples of implanted pacemakers with weak batteries or broken lead wires in which ventricular capture was achieved only when spikes occurred at or near the end of the T wave of the preceding beat, i.e., in the SN period of excitability.

In an important paper reviewing the pros and cons of the very existence of SN A-V conduction, Moe, Childers, and Meredith suggested that most, if not all, the published examples of supernormality could be explained by alternative mechanisms. Of the many mechanisms described, four are particularly germane to the cases presented in this article. They include: (1) so-called "peeling back" of the refractory barrier by a premature ventricular beat; (2) A-V conduction along alternate pathways possessing different effective refractory periods; (3) in a case of bundle-branch block, a premature atrial beat may be delayed sufficiently in the A-V node for the previously blocked bundle to recover, thus giving rise to a long P-R interval and a normal QRS; a mechanism also observed by Damato and Lau; and (4) the ventriculophasic supernormality in cases of heart block, wherein the usually large stroke volumes initiate a powerful vagal surge after each beat, thus perpetuating the block by means of depressing conduction across the A-V node. Only those P waves which occur early enough after ventricular contractions, i.e., before the vagal surge, will be conducted. In the cases described in this article, these possibilities were excluded and true supernormality is believed to have existed.

Thus, in case 1 of the present study premature atrial beats reaching the bundle branches 0.54-0.62 sec after the preceding R waves were conducted with normal QRS. The P-R intervals of these beats were not prolonged and therefore mechanism 3 did not apply. Premature atrial beats reaching the bundle branches earlier displayed RBBB, again with normal P-R intervals. Cases 2 and 3 showed SN conduction in the right bundle branch. The premature atrial beats displaying SN I-V conduction were not associated with long P-R or P-His intervals. Mechanism 3 cannot explain the supernormality in the right bundle branch in either of these two cases.

In case 4 the ventriculophasic supernormality (mechanism 4) may be suggested. However, several reasons can be marshalled against vagal effect in this case: (1) The block
was documented to be at the level of the bundle branches and therefore not likely to be profoundly affected by vagal influences. (2) It was shown that the period of supernormality bore different temporal relationships to the preceding QRS depending on the ventricle of origin of the latter, and that events at the level of the cell membrane explained the difference. (3) The administration of atropine failed to eliminate the SN beats. (4) The brevity of the SN period does not exceed a few hundredths of a second after correction has been made for the shifting origin of the QRS, as was done in figure 6. A vagally mediated reflex would be expected to last considerably longer. (5) There was an absence of any other manifestations of vagal activity such as variations in the sinoatrial rate similar to that observed by Rosenbaum and Lepeschkin. It would be paradoxical, indeed, to have so much vagal effect on the right bundle branch without any parallel changes in the rate of discharge from the sinoatrial node.

Cases 6, 7, and 8 exemplify SN excitability. Cases 6 and 7 resembled those described by a number of authors, and showed that subthreshold pacemaker impulses were effectively propagated only when they occurred at a specific distance following the preceding QRS. The relationship to the preceding P waves was inconstant, thus providing no support for the thesis that unusually strong atrial contractions may have thrust the catheter deeper into the myocardium and allowed the weak impulse to be effective. In cases 6 and 7 intravenous atropine administration failed to abolish the supernormality. The inability of atropine injections to abolish the supernormality in any of the cases and the absence of any demonstrable relationship between the supernormality and the hemodynamic events cast doubt on such mechanisms as vagally mediated ventriculophasic supernormality. In the cases reported here observations favored an electrophysiologic explanation based on the presence of negative afterpotentials and the consequent enhancement of conductivity.

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