SPECIAL ARTICLE

An Appraisal of "Supernormal" A-V Conduction

By G. K. Moe, M.D., Ph.D., R. W. Childers, M.D., and J. Merideth, M.D.

SUMMARY

Certain temporal patterns of A-V and V-A transmission in experimental preparations resemble phenomena attributed to "supernormal" conduction in the clinic. Detailed study of the properties of the A-V transmission system in such experiments reveals alternative explanations in which supernormality is clearly eliminated. By application of similar principles, supernormality can be eliminated as a factor in most if not all of the published examples. Three major categories can be discerned: (1) occult 2:1 A-V block, in which an idioventricular beat "retracts" an otherwise refractory barrier within the A-V node; (2) alternation between dissociated intranodal transmission pathways; and (3) "ventriculophasic" (vagal) depression of nodal conductivity.

Additional Indexing Words:
Alternation   Diastolic depolarization   His-Purkinje system   Idioventricular beat
Occult A-V block   Peeling   1, 2, 3, 4 phenomenon   Premature beats
Ventriculophasic (vagal) depression of nodal conduction

"SUPERNORMAL" CONDUCTION is a term applied to a number of puzzling features of atrioventricular conduction.¹ Because these features are usually associated with depression of A-V nodal conductivity, the term refers, not to conduction which is faster than normal, but to conduction which is "better" than anticipated under the circumstances. If one assumes that the more closely a propagated response follows upon the heels of a preceding response, the more slowly it should be conducted, then any departure from this orderly relationship will be regarded as supernormality, or "relative" supernormality. The example illustrated in figure 1, reproduced from the frequently cited case report by Lewis and Master,² indicates that atrial systoles falling shortly before the peak of the T wave, and within the subsequent 0.26 sec, were propagated to the ventricles, while earlier or later atrial responses were blocked. These authors postulated that the excitatory process within the A-V transmission system exceeded threshold only during this brief period of the cardiac cycle.

Conduction time from atrium to ventricle represents the sum of the serial activation times of all the elements in the conduction pathway. Each individual latency, however small, must be a function of the excitation threshold of the recipient unit, and of the amplitude and rate of rise of the action potential in the elements which provide the input stimulus. Supernormal conduction might therefore be expected in a pathway in which either the excitability or the stimulating efficacy of the action potential is enhanced at an early stage of recovery, relative to the corresponding properties later in the cycle.

Supernormal excitability, in the sense of a lower threshold in early than in late diastole, has been demonstrated in cardiac tissue under abnormal circumstances (ischemia, hypoxia), and appears to be a "normal" property of the specialized conduction system of the
atia and ventricles in anesthetized, open-chest dogs, or in isolated perfused preparations of mammalian hearts.\(^3\),\(^4\) Relatively supernormal conduction, as a function of the rate of depolarization (\(dv/dt\)), may appear in Purkinje tissue of isolated preparations when widespread diastolic depolarization (pacemaker activity) takes place. The rate of rise of the action potential upstroke under these conditions will become progressively slower as the potential difference across the cell membrane decreases late in diastole.\(^5\)

Supernormal excitability has not been demonstrated in preparations of the A-V node in which excitability can be directly assessed. On the contrary, the recovery of "normal" diastolic excitability is delayed appreciably beyond full repolarization of nodal cells.\(^6\) The stimulating efficacy of nodal action potentials, judged by amplitude and rate of rise, has not been shown to be greater at an early stage of recovery than in late diastole. Under usual conditions of perfusion, no feature of nodal behavior would suggest the possibility of supernormal or relatively supernormal conductivity. The response of the node to agencies which depress conductivity (that is, rapid driving frequency, premature responses, acetylcholine) also fails to support the concept.

In spite of these constraints, certain features of transnodal conduction in experimental animals resemble phenomena which have been attributed to supernormality in the clinic. Because the circumstances leading to such complexities of A-V transmission in the laboratory can be more readily controlled, a more complete analysis can be made than is usually possible in the clinic. Accordingly, we have selected a number of examples of events which, while apparently representing supernormal conduction, can be shown on further analysis to have alternative explanations. In the light of these observations, we have also examined some clinical examples of supernormality to which similar explanations may apply.

I. Experimental Studies

A. Apparent Supernormality in A-V and V-A Conduction Induced by Premature Beats of the Ventricle or Atrium

Effect of Ventricular Premature Beats on A-V Conduction

When the heart is driven by stimuli applied at regular intervals to the atrium, an early premature stimulus may initiate a response which fails to propagate to the ventricles. In the dog heart it can be shown that the premature atrial response (A\(_2\)) enters the A-V node but dies within it ("concealed" conduction). The extinction of A\(_2\) in this case is clearly due to residual refractoriness of tissue within the nodal conduction pathway. If the same sequence (for example, 12 rhythmic driven responses, A\(_1\), followed by a premature response, A\(_2\)) is repeated, but if the ventricle is pre-excited by a stimulus applied to

---

**Figure 1**

Chart illustrating "supernormal" A-V conduction in case 1 of Lewis and Master,\(^9\) reproduced with permission from Heart 11: 371, 1924. Atrial responses which followed a QRS complex by less than 0.425 sec or by more than 0.708 sec failed to propagate. Within these limits (the "supernormal phase"), A-V propagation was almost the rule. The authors' explanation, diagrammed below the ventricular complex, indicates that the stimulating efficacy of the atrial action potential exceeds threshold only during this period. An equivalent hypothesis would suggest that the threshold at a critical intranodal junction was lower during this period.
Facilitation of A-V propagation by pre-excitation of His bundle. Records taken from isolated rabbit heart preparation. In each segment, upper trace is a bipolar electrogram recorded from the atrium; lower trace, transmembrane action potential recorded from a cell in the NH region of the A-V node, just above the His bundle. In each record, the first atrial response is the last of a series of seven driven responses at a basic cycle of 720 msec. In A and B, the premature atrial responses occur at an A.A2 interval of 151 msec; in C and D, the A.A2 interval was 152 msec. In B and D, the His bundle was stimulated simultaneously with the last A1 stimulus, resulting in pre-excitation of the NH cell about 80 msec in advance of the expected arrival of A2. Propagation of A2 fails just above the impaled cell in A, but succeeds in B. In C, A2 barely succeeds in traversing the node. Facilitation of transmission in the presence of pre-excitation of the His bundle is indicated by the broken vertical line in C and D. Time and voltage calibrations in A: 50 msec, 50 mv.

it shortly before the appearance of the last driven response from the atrium, then A2 will successfully traverse the A-V transmission system and emerge as a ventricular response, V2.7

It might be suggested that pre-excitation of the ventricle in such an experiment permits the transit of A2 because it penetrates the node and is followed by a supernormal period. What in fact must occur is that A2 (in the absence of ventricular pre-excitation) is blocked within the transmission pathway at a barrier still refractory following the preceding A1V1 response. Pre-excitation of the ventricle results in earlier excitation and earlier recovery of the A-V nodal tissue below the site of block, and therefore permits the passage of A2.

Direct confirmation of this interpretation was obtained in isolated preparations of rabbit heart, as shown in figure 2. In part A, a premature atrial response penetrated the node but gave rise to only a local response in the impaled nodal cell. Propagation must have failed just above this level.8 In part B, pre-excitation of the His bundle permitted the propagation of A2. In part C, the premature response was delayed enough to permit its complete passage with a conduction time of 130 msec from atrium to the impaled cell. In
Facilitation of V-A propagation by pre-excitation of atrium. Open-chest dog preparation; heart driven at cycle length of 500 msec by stimuli applied to ventricle. In A, V1V2 interval is 275 msec; V2 is the latest premature response which fails to reach the atrium. In B, V1V2 interval is 215 msec; pre-excitation of the atrium (A0) permits the passage of V2. Broken lines indicate the time course of V2 in the absence of A0, and of A1 in the absence of V1. The shaded areas represent the duration of the effective refractory period at the site of propagation failure in A. (Based on an experiment described by Moe, Abildskov, and Mendez.?)

the presence of pre-excitation of the His bundle (part D), the conduction time was reduced to 108 msec.

Effect of Atrial Premature Beats on V-A Conduction

An equivalent situation can also be set up in the reverse direction. The events illustrated in figure 3 were described previously as indirect evidence for concealed V-A conduction of a premature ventricular response.9 In part A of the figure, the premature response (V1V2 interval, 275 msec) failed to traverse the node. In part B, following pre-excitation of the atrium at A0, a much earlier V2 succeeded in reaching the atrium. As in the previous illustration, success of the premature V2 was not the result of supernormality following the intranodal penetration of A0, but was achieved because a refractory barrier in the upper node had been “peeled back” by the atrial pre-excitation. In extensive studies of this type, the earliest “facilitated” response was invariably propagated more slowly than later responses; that is, there was no evidence of relatively supernormal conduction in either the normal or retrograde direction.

B. “Repetitive Supernormality” Induced by Ventricular Premature Beats

The events described above would not, of course, be cited as examples of supernormal A-V conduction if they appeared in clinical electrocardiograms, because the premature responses provide a longer R-P or P-R interval preceding the subsequent A2 or V2. Analogous events can, however, occur under conditions in which retrocession of the refractory barrier is not so obvious. Two experimental situations will serve to illustrate the point.

Termination of Functional Bundle-Branch Block

A premature atrial response may occasionally traverse the node quickly enough to reach the His bundle at a time when the right bundle branch is still refractory (functional right bundle-branch block [RBBB]). It has been shown that the site of block may be quite high, perhaps at the origin of the right bundle, and that the major portion of the bundle and its rami are then excited antidromically from the left side.10 Retrograde excitation of the right bundle branch (RBB) throws the refractory periods of the His bundle and the RBB out of phase, and apposes a refractory barrier to the propagation of a subsequent response from above.* The phenomenon of functional block can thus be repetitive, provided the input frequency falls within appropriate limits. An example of repetitive RBBB induced in this manner has been previously published.10 Figure 4 indicates how a single premature ventricular response could restore “normal” intraventricular conduction in such a situation. The premature V5, by engaging the right

*On occasion, the left bundle branch may fail while propagation through the RBB is maintained. A clinical example of this appears in the article by Wellens, H. J. J., and Durrer, D.: Supraventricular tachycardia with left aberrant conduction due to retrograde invasion into the left bundle branch. Circulation. In press.
bundle system prior to its expected retrograde invasion by H₄, would advance its refractory period and permit H₅ to propagate normally over both bundle branches. Once H₅ is enabled to engage both pathways, all subsequent responses, even though the basic frequency remains unchanged, would also be propagated normally.

These events could be ascribed to a supernormal phase in the excitability and conductivity of the RBB. It could be said that once the change of phase occurred in the RBB, each subsequent response fell in the corresponding supernormal phase of its predecessor. The events are, however, comparable to those depicted in figures 2 and 3, and they are amenable to the same explanation.

Termination of 2:1 A-V Block

A second situation demonstrates a somewhat similar facilitation of conduction terminating an episode of 2:1 A-V block. It is well known that the blocked atrial responses enter the A-V node, and that transmission failure occurs within it.⁸,¹¹ The prolonged A-V conduction time of the propagated responses is ascribed to refractoriness in the upper node following its partial invasion by the “concealed” response.¹¹

In the experimental animal, 2:1 A-V block can be easily induced by appropriate acceleration of driving stimuli applied to the atrium.

Figure 4

Mechanism of termination of an episode of repetitive functional right bundle-branch block. Driving stimuli applied to bundle of His (H₁-H₅) at intervals which yielded bundle-branch block. H₁H₂ = 220 msec, H₂H₃ = 200 msec; H₃H₄, H₄H₅, and H₅H₆ = 190 msec. Horizontal lines represent: (1) site of stimulation in His bundle; (2) bifurcation of common bundle; (3) epicardial recording site on right ventricle; (4) epicardial recording site on left ventricle; and (5) simulated bipolar electrogram from right and left recording sites.

Stippled areas between 1 and 2 represent refractory period in His bundle; between 2 and 3, refractory period of right-bundle branch. Abbreviation of refractory periods as a function of reduction of cycle length is indicated; as determined by experiment, frequency-dependent change is greater in the right bundle than in His bundle.¹⁰

H₁ (last of a series of basic driving stimuli at cycle length of 500 msec) activates both right and left bundles in normal pattern (upright QRS in simulated electrogram); H₂ is blocked in right bundle; recurrent excitation of bundle branch occurs from left, and is blocked at His bundle. H₃ and H₄ encounter right bundle which is refractory because of retrograde activation. Pre-excitation of ventricles by S permits normal propagation for H₅ and H₆. (Based on an experiment described by Moe, Mendez, and Han.¹⁰)
Temporal pattern of premature responses initiated in atrium. Open-chest dog preparation. Heart driven by atrial stimulation at basic cycle of 515 msec. Abscissae, A1A2 intervals in msec; ordiates, resulting V1V2 intervals as recorded from right ventricular surface. At A1A2 intervals between 160 and 175, V1V2 was about 235 msec. (that is, the A2V2 conduction interval was prolonged by 60 to 75 msec over basic A1V1). When A1A2 was between 175 and 200, the right ventricular electrogram was inverted, and V2 was delayed by an additional 40 msec (functional block of RBB). Vertical arrows indicate A1A2 intervals at which transmission failed completely.

Inset shows atrial and right ventricular electrograms recorded when A1A2 was just earlier (B) and just later (A) than 175 msec. (Based on an experiment described by Moe, Mendez, and Han.)

If the rate of atrial stimulation is gradually increased, 1:1 transmission will give way to variable ratios of Wenckebach periodicity, followed by a 2:1 rhythm. If the frequency is now gradually diminished, 2:1 block can sometimes be carried back briefly to an atrial frequency which was previously accompanied by 1:1 transmission. The range of atrial frequencies at which 2:1 block can be maintained is thus fairly broad. The reason for this is obvious enough: the effective refractory period of the upper portion of the node is abbreviated because of the short cycle length of its atrial input, while that of the lower node (and of the His bundle), responding at half the frequency, may be considerably longer. The blocked beats are, accordingly, extinguished at a refractory barrier in the lower node. Here, again, a single premature ventricular beat, appropriately timed, could pre-excite the lower node, peel back its refractory period, and permit the passage of the otherwise blocked atrial response. This is, of course, no different from
the phenomenon illustrated in figure 2, and if the A-V transmission of only one atrial response were thus facilitated, the result would be expected on the basis of the induced prolongation of the preceding R-P interval. However, once a single successful nodal transit occurs, one or more subsequent atrial responses at the same frequency should also be transmitted. The refractory period in the lower node will be reduced, and 1:1 transmission will again be possible. The improvement of A-V function does not improve, one or more subsequent responses will be expected on the basis of the induced prolongation of the preceding R-P interval. However, once a single successful nodal transit occurs, one or more subsequent atrial responses at the same frequency should also be transmitted. The refractory period in the lower node will be reduced, and 1:1 transmission will again be possible. The improvement of A-V function does not mean that each successive response falls upon the “supernormal” phase of the preceding one.

C. Apparent Supernormality of A-V Conduction Resulting from Refractoriness in the Intraventricular Conducting System

A somewhat more complex manifestation of “supernormal” A-V conduction was described recently in a study of functional block of the bundle branches; one example is illustrated in figure 5. In this experiment, the heart was driven by stimuli delivered to the right atrium, and responses were recorded from the surface of the right atrium and right ventricle. The interval following the last of a series of driven responses was scanned by premature atrial stimuli, starting at an A1A2 interval (abscissae) of 300 msec; at this interval A-V propagation was nearly fully recovered. As the premature stimulus was advanced (A1A2 abbreviated), A-V conduction time gradually increased; when A1A2 was reduced to 200 msec, an abrupt further increase of 40 msec in the conduction time to the right ventricular recording site was observed. The extra increment in conduction time persisted down to an A1A2 interval of 175, and disappeared with equal abruptness as still earlier premature responses were initiated. The extra delay was shown to be due to functional block of the RBB; it did not occur in electrograms recorded from the left ventricle (not shown in the figure). Occasional atrial responses within the range of RBBB failed to reach the ventricles at all (arrows).

Preliminary examination of the results shown in figure 5 suggests that the earliest atrial premature responses encountered a region (intranodal or subnodal) of supernormal excitability and were therefore facilitated. The fact that the His bundle does indeed exhibit supernormal excitability lends plausibility to the argument. More careful study, however, revealed the actual mechanism. At slow driving frequencies, the effective refractory period of the bundle branches may exceed that of the His bundle, and of the A-V node itself. Late premature responses will, of course, find all parts of the intraventricular conducting system excitable, and the sequence of excitation will be normal. Earlier premature responses, if not excessively delayed within the A-V node, may arrive at the bundle of His before recovery of the RBB, leading to the pattern of RBBB. If the premature response arrives slightly earlier, the LBB or the His bundle itself may also be refractory, and complete failure of ventricular excitation can occur. A very early A2 response, however, will be delayed so long during passage through the node that its arrival at the bundle of His occurs after the expiration of the functional refractory period of the common bundle and both its branches. A detailed exposition of the characteristics of transmission in this and similar experiments was presented in the earlier publication and will not be repeated here.

If supernormality is defined as "unexpectedly" facile propagation of early responses as compared with later ones, then figure 5 is indeed an example of supernormal A-V conduction. The use of this term, however, obscures the physiological mechanism. The observed events were the result of transmission delay, as would be expected early in the relatively refractory period of the A-V node; supernormality played no part.

D. Apparently Supernormal Conduction Resulting from Intranodal Dissociation

The "1, 2, 3, 4 Phenomenon"

An even more complex example of facilitated A-V conduction, dubbed the "1, 2, 3, 4 phenomenon," has been observed frequently in this laboratory, and a detailed analysis has been published. When an early premature A2 response is propagated slowly through the
A-V node, a prolonged period follows during which a subsequent $A_3$ response is concealed within the A-V node and fails to emerge in the ventricles. When $A_3$ is placed as early as possible after $A_2$ and is followed by a fourth atrial response, $A_3$ can be shown to facilitate the passage of $A_1$. That is, $A_1$, within a certain range of $A_2A_4$ intervals, will propagate to the ventricles in the presence of $A_3$, but not in its absence. This could, of course, be readily attributed to a supernormal phase of conductivity following the intranodal penetration of $A_3$.

Not infrequently, it was observed that $A_3$ not only facilitated the passage of a subsequent response, but also that of the prior response, $A_2$. The apparent conduction time, $A_2V_2$, was regularly diminished when $A_3$ was present. A particularly striking example is illustrated in figure 6. In the upper segment of the figure, the early premature $A_2$ response was propagated to the ventricle with a conduction interval of approximately 400 msec. In the lower segment, the $A_1A_2$ interval was the same as before (170 msec), but a third atrial response was introduced 175 msec later. The apparent $A_2V_2$ interval was reduced by 60 msec. We can hardly assume that $A_3$ somehow created a supernormal pathway for the preceding response; we must assume that $A_3$ traversed a pathway which had not been entered by $A_2$; that is, the upper portion of the node must have been dissociated into two pathways, one of which was still inexorable at the time of $A_2$, but had recovered in time to permit the passage of $A_3$. The third atrial response did not facilitate the passage of the prior response; it entered an alternate pathway, now fully recovered, and reached the ventricle ahead of $A_2$. It was shown, in fact, that the $A_3V_3$ conduction time in this experiment remained the same when $A_2$ was omitted.

We have assumed that the interaction of $A_2$ and $A_3$ in this situation is due to the dissociation of two pathways, a conclusion which is supported by direct studies in isolated preparations of rabbit heart tissue. Clearly, the interaction of $A_3$ and $A_1$ must also be considered in terms of intranodal dissociation. This has been done in a detailed analysis of the 1, 2, 3, 4, phenomenon, and it was demonstrated that a dual conduction system provides a logical explanation for both interactions. We have, then, a curious pattern of responses in which an atrial premature beat facilitates the conduction of both a preceding and a succeeding response; in neither case is "supernormality" involved.

Facilitation of Ventricular Echoes

Another example of "supernormality" which also involves intranodal dissociation is shown in figure 7. In each segment, $V_1A_1$ represents the last of a series of driven responses to stimuli applied to the right ventricular surface. In part A, $V_2$ is the latest premature ventricular response which failed to reach the atrium. In part B, the earliest ventricular premature beat which was propagated to the atrium was followed by an "echo." In part C, is shown the earliest $V_2$ which was not accompanied by an echo. The apparent supernormality is illustrated in part D. The premature response $V_2$ was initiated at the same $V_1V_2$ interval as in part A; it could not have reached the atrium. The response $V_3$ was initiated at a time (later than the $V_1V_2$ interval in C) when it should not have yielded an echo, but in the presence of $V_2$ an echo did in fact occur.

One might suggest that the intranodal penetration of $V_2$ in this experiment left the reciprocal pathway supernormally excitable, permitting an echo to appear in response to $V_3$. Collateral evidence indicates that this is not a valid explanation. We can accept the existence of ventricular echoes as evidence for effective intranodal dissociation. During the span of time represented by the $V_1V_2$ intervals in figure 7B and C (290 and 380 msec, respectively), one of the intranodal pathways was unavailable to $V_2$. As the $V_1V_2$ interval increased, however, the $V_2A_2$ conduction time diminished. If the $A_2$ response occurs too promptly, the reciprocal response will be blocked by refractoriness in the lower node (the "final common pathway"). The exposure...
Figure 6

"Facilitation" of A-V propagation of a premature atrial response, 2, by a subsequent response, 3, in open chest dog. Upper trace in each segment, right atrial bipolar electrogram; lower trace, right ventricle. Response 1, last of a series of driving stimuli applied to atrium. Response 2, premature atrial response (A₁A₂ = 170 msec); 3, third atrial response (A₂A₃ = 345 msec). Time calibration in top segment, 400 msec. Note that the apparent V₂ response occurs 60 msec earlier in the presence of A₃. (Taken from an experiment described by Moe, Mendez, and Han.¹³)

of the echo in figure 7D can thus be explained: refactoriness following the nonpropagated (but concealed) V₂ response caused a delay in the retrograde propagation of V₃, which then permitted the reciprocal response to "clear" the refractory period of the final common path. "Supernormality" was not a factor.

Alternation of A-V Conduction

Alternation of A-V conduction time, occasionally observed in clinical tracings and in experimental preparations, has been ascribed to supernormality.¹⁰,¹¹ Because in this situation the briefer P-R interval follows the briefer R-P interval, the propagation pattern is said to be "paradoxical." The paradox is, however, only apparent. If alternation of A-V conduction time occurs when the atrial frequency is constant, the short P-R must follow the short R-P; there is no other possibility. The primary event is the alternation of transnodal conduction; the variation in R-P interval is the result.

The explanation of alternating P-R intervals may be found, as in the phenomena described above, in longitudinal dissociation within the node. When A-V nodal transmission is stressed (as at rapid driving frequencies), or depressed (as with hyperkalemia or digitalis), dissociation may occur; it is unlikely that the conductivity of the nodal pathways will be equally depressed. There are several ways in which unequal depression of dual
Figure 8

Possible mechanisms of alternation of P-R interval. In part A, A₁ and A₂ are assumed to propagate through α and β pathways in upper node, resulting in prompt excitation of final common pathway. Refractory period of β pathway (stippled area) prevents transmission of A₃ and A₄; delayed excitation of final common pathway results. In B, α and β pathways are excited alternately from atrial end; β pathway is assumed to conduct more slowly than α (A₂ and A₄). Diagrams at right indicate dissociation of upper node into two independent pathways converging on a final common pathway. Arbitrarily chosen intervals between successive responses indicated in seconds.

pathways could be expressed as alternation of conduction time. Two of these possibilities are diagrammed in figure 8. In part A, each atrial response is assumed to engage the "α" pathway, but a 2:1 block in the "β" pathway is postulated. Assuming that simultaneous activation of both pathways provides the more effective input to the final common pathway, the latency of activation at the junction should be expected to alternate. The postulation of summation at an intranodal junction is not mere speculation; summation has been demonstrated in isolated preparations of the rabbit A-V node.⁶

A related explanation is illustrated in figure 8B. Here we assume that 2:1 block occurs in each nodal pathway for alternate atrial responses. If the conduction intervals in the two pathways are not equal, alternation of conduction time must occur. If they are equal, the P-R interval need not change from beat to beat, but the pattern of activation of the node would still alternate. It can be shown, in support of this assumption, that concealed alternation may occur.⁷ When the transmission system is stressed by driving the atria at a frequency approaching the maximum for 1:1 A-V conduction, the duration of the “phase of concealment” (that is, the range of A₁A₂ intervals within which A₂ enters the node but fails to propagate to the ventricles) may alternate from beat to beat whether or not the overall P-R interval alternates. In short, alternation of A-V conduction can be readily explained in terms of well-established characteristics of nodal behavior without invoking a hypothetical supernormal period.

E. “Ventriculophasic” Supernormality

A number of examples of occasional A-V (or V-A) transmission during episodes of
otherwise complete A-V dissociation have been described. For example, an atrial beat may propagate to the ventricles only when it falls within a relatively narrow time period shortly after a preceding idioventricular beat, or after a considerably longer recovery time. When, as is often the case, A-V dissociation is at least partially due to digitalis action, we may assume a relatively strong background of vagal discharge. If the ventricular rate is slow, and the stroke volume is correspondingly large, surges of increased vagal activity will be triggered by baroreceptor discharge following each pressure pulse. Cholinergic activity in the sinoatrial pacemaker and in the A-V node will be at a minimum just prior to each vagal burst; at this time, successful penetration of the transmission system will be more likely than at any other time during the ventricular cycle. Such a period of “enhanced” (that is, less depressed) conductivity would be cited as evidence of a supernormal phase following the incomplete nodal penetration of the preceding beat.

Whether or not the apparent supernormal phase of A-V conduction in this situation can be ascribed to reflex vagal discharge depends critically upon the timing of events. On the basis of experiments performed on the dog heart, the total lag between a given QRS complex and the first expression of vagal effects upon A-V nodal conductivity can be fairly precisely estimated. The total latency between QRS and vagally mediated prolongation of the P-R interval includes: (1) the time between QRS and the systolic pulse wave in the pressure-sensitive areas of the arterial tree; (2) the reflex time, including conduction time in the baroreceptor nerves, delay in the central nervous system, and vagal efferent conduction time; and (3) the lag between vagal excitation and the expression of its physiological effects.

The probable time course of reflex vagal effects has been considered in detail by Roth and Kisch18 and by Rosenbaum and Lepeschkin19 in their analyses of ventriculophasic sinus arrhythmia; it can be shown in the laboratory that the concept applies equally well to A-V conduction. The earliest atrial response which will encounter A-V nodal delay as the result of the vagal discharge evoked by a preceding ventricular contraction in the anesthetized dog occurs at an R-P interval of approximately 600 msec.20 Accordingly, any atrial response occurring at a briefer R-P interval should be propagated with greater facility than one generated later than 600 msec.

A number of interesting corollaries to this conclusion are apparent; all of them are subject to experimental test. (1) Phasic changes in A-V conductivity should be more apparent at slow ventricular rates, because of the greater stroke volume and pulse pressure and, accordingly, a more discretely phasic vagal discharge. (2) The ventricular beat following a closely-coupled premature beat should be more likely to expose a reflex change in conductivity; the premature beat itself would not provide a mechanical stimulus to the baroreceptors, but the combination of a long diastole added to the potentiating effect of the premature beat upon the subsequent contraction should result in an enhanced systolic discharge. (3) In experimental preparations in which vagal reflexes are active (morphine-chloralose anesthesia) there should be a range of slow-driving frequencies (cycle length more than 600 msec) at which A-V conduction is slower than at faster rates. (4) During a period of A-V dissociation, an atrial impulse, falling during the period of minimal vagal influence, may effect a ventricular capture. The resulting ventricular contraction, if early enough, will eject relatively little blood. The idioventricular pacemaker will, however, be discharged and its next spontaneous discharge will be postponed. During this period, the level of vagal activity will continue to decline. Accordingly, if the S-A nodal and idioventricular frequencies are in an appropriate relationship with each other, one capture may permit one or a series of subsequent S-A nodal beats to be transmitted to the ventricle. Loosely speaking, vagally mediated changes in A-V conductivity may be cited as examples of
supernormality, for conduction may be possible at an early stage in recovery and impossible at later times, but here again the term obscures the mechanism.

**F. Possible Mechanisms of “True” Supernormality of A-V Conduction**

In the examples described above, apparent supernormality can be ascribed to alternative mechanisms. There remain at least two possibilities, difficult to assess but nonetheless conceivable, which merit consideration.

*Supernormality in the His-Purkinje System*

A phase of supernormal excitability in the specialized conducting tissue of the ventricles can be demonstrated in vitro and in the heart in situ. Presumably, an impulse which reaches the His bundle during its supernormal phase could be propagated more rapidly than one which occurred later in diastole. Conceivably, we could have an idioventricular rhythm initiated from a focus in the His bundle. If A-V nodal function were sufficiently depressed by disease or drugs, the ectopic impulse could fail to enter the A-V node, and impulses of supraventricular origin could also fail to cross the threshold of the His bundle except during the supernormal phase following spontaneous discharge of the pacemaker in the common bundle. If, however, the idioventricular beat penetrates the A-V node for even a short distance, it is unlikely that a response of supranodal origin could cross the node early enough to encounter a supernormally excitable His bundle. The experimental conditions which would permit demonstration of such a mechanism would be difficult to achieve, and the clinical situations would be rare, but the possibility cannot be dismissed.

*Relative Supernormality due to Diastolic Depolarization*

Since the rate of rise of the action potential must be one of the determinants of conduction velocity, and since it is a function of the membrane potential at the time of excitation, one should expect that the slow diastolic depolarization characteristic of pacemaker activity in the His-Purkinje system would be accompanied by a diminution of conduction velocity. Singer and associates have emphasized this mechanism as a possible explanation of “entrance block.” They postulate that an impulse approaching a region of pacemaker activity may decelerate or decrement even to complete block. According to this hypothesis, a supraventricular impulse reaching the His bundle soon after an idioventricular discharge could propagate rapidly and “capture” the ventricles, while a later response would fail.

![Figure 9](http://circ.ahajournals.org/)

**(A) Supernormal A-V conduction in a clinical case described by Lepeschkin and Kimura (redrawn from their figure 2).** (B) A similar plot of the RP-PR relationship obtained from the model illustrated in figure 10A. In part A, the points which fall within the broken lines represent spontaneous idioventricular discharges (A-V nodal “escapes”); that is, PR + RP = a constant of approximately 1.5 sec. Atrial responses occurring at R-P intervals between about 0.47 and 0.7 sec were not propagated to the ventricle. Earlier atrial responses (R-P of 0.34 and 0.46 sec) were commonly propagated with P-R intervals in the range of 0.5 to 0.6 sec.
This might be particularly likely when ectopic pacemaker activity is enhanced, A-V conductivity is depressed, and conduction within the specialized conducting system is also impaired. All three of these conditions are characteristic of digitalis intoxication, and it is conceivable that certain instances of supernormal A-V conduction may be explained on this basis. Here again, however, it would be necessary to postulate that the idioventricular discharges fail to enter the node; otherwise the supraventricular response would be delayed too long to arrive at the appropriate moment in the common bundle. It is also necessary to postulate that the ectopic impulse itself can propagate in partially depolarized tissue which cannot support a response of supraventricular origin, that is, entrance block.

II. Analysis of Selected Clinical Examples
A. Supernormality due to “Peeling”
Facilitation of A-V transmission due to retrocession of the nodal refractory period must occur frequently in the clinic. The simple patterns illustrated in figures 2 and 3 would not be cited as examples of supernormality, but several case reports fit the conditions described in Section I-B (page 8) and we shall consider them in detail.

In a case described by Lepeschkin and Kimura,22 A-V dissociation was present with P-P nodal intervals of about 0.8 sec, and an “A-V nodal escape”* interval of about 1.5 sec. Occa-

---

*We use quotation marks to indicate that the escape may, in fact, represent the accession of a subnodal (His bundle) pacemaker.

---

Figure 10
Postulated mechanism of “supernormality” in A-V dissociation. In each panel, atria respond to sinus node at cycle length of 0.75 sec; idioventricular pacemaker fires with cycle of 1.4 sec. Refractory period of lower portion of A-V node represented by longer shaded parallelograms; briefer refractory period of His bundle above and below hypothetical ectopic pacemaker represented by shorter polygons. In upper panel, retrograde penetration of A-V node from His focus is assumed; in lower panel, retrograde transmission is blocked. Response 5 in upper panel and response 3 in lower panel would be regarded as “supernormal” conduction. Delay of these responses at critical junctions is indicated by shoulders in corresponding conduction lines.

(Based on the observations of Lepeschkin and Kimura*22 (upper panel) and Jonas,23)
sional atrial beats were propagated, with P-R intervals ranging from about 0.2 to 0.6 sec. When P-R intervals were plotted as a function of R-P, a cluster of “supernormally” propagated responses occurred at R-P intervals between 0.34 and 0.46 sec, while transmission regularly failed when R-P intervals were between 0.47 and 0.7 sec (fig. 9A). Some of the atrial responses occurring during the “supernormal phase” failed to propagate; the difference between success and failure during this period was related to the duration of the immediately preceding P-R interval.

Upon closer inspection of the temporal relationships of responses in this case, it became apparent that the underlying pattern was 2:1 A-V block, with a subordinate pacemaker firing at a frequency slightly faster than half the sinus rate. Whenever the idioventricular pacemaker discharged just before or just after a P wave, the following one or two sinus beats (in one instance, four) were propagated. This situation, as described above, can be readily duplicated in the laboratory, and the diagram in figure 10 indicates a mechanism which clearly does not demand supernormality, and is consonant with well-known properties of the A-V transmission system.

In the upper portion of figure 10 we have plotted the interaction of two pacemakers. The frequencies were arbitrarily chosen, but they are close to those observed in the clinical case just described, and also observed by Jonas23 in his case 1. We assume that the S-A node discharges regularly at a cycle length of 0.75 sec, and that a basic 2:1 block is obscured by interference from a subordinate pacemaker (for example, in the His bundle) with a cycle length of 1.4 sec (that is, slightly less than twice the S-A nodal interval). We also assume that retrograde transmission is possible up to and beyond the point at which alternate descending impulses are blocked. The first response, A1, would have been transmitted in the absence of the idioventricular discharge; the collision occurs too late to retract the refractory period in the lower node, and A2 is therefore extinguished. The second ventricular beat occurs 0.1 sec in advance of A3, and the resulting pre-excitation of the lower node permits the passage of A4, which effects a ventricular capture and discharges the His pacemaker. Because the cycle length in the lower node has been abbreviated, the refractory period of the critical nodal area following the transit of A4 may also be shortened. Accordingly, A5 is allowed to pass, although with some delay. With the conventions adopted, the 2:1 intranodal block is then reestablished, but it is apparent that a slightly shorter refractory period within the lower node would permit the passage of one or more additional supraventricular impulses before restoration of the initial response pattern. As diagrammed, the pattern would recur after every twelfth S-A nodal discharge, a cycle which depends, of course, on the precise relationship between atrial and idioventricular frequencies.

The schematic analysis represented in figure 10 was continued, allowing some variation in both S-A nodal and subordinate pacemaker frequency. When the “R-P” intervals were plotted against “P-R,” the relationship was strikingly similar to that plotted by Lepeschkin and Kimura (fig. 9B). A cluster of “supernormally” conducted beats appeared at R-Ps in the neighborhood of 0.45 sec, and a zone of transmission failure lay between the “supernormal” period and longer R-P intervals at which transmission was again possible. The clinical data thus fit the concepts outlined in the Section I-V (page 8), and the postulated supernormality may be rejected.

A related explanation applies to the case described by Jonas.23 The records from his report are reproduced in figure 11. The S-A nodal cycle length varied between 0.74 and 0.78 sec, while an idioventricular pacemaker discharged with a cycle of 1.4 to 1.44 sec; as in the previous example, the “escape” cycle was slightly less than double the S-A nodal period. P waves were conducted only when the R-P interval was in the range of 0.06 to 0.16 sec, which was assumed to represent a supernormal period.

An alternative mechanism is illustrated in the lower half of figure 10. In order to show
the basic similarity with the example just described, we have used the same cycle lengths, refractory period durations, and conduction times. The only difference is the assumption that retrograde transmission fails to reach the level of intranodal 2:1 block. Accordingly, interference at or just above the His bundle obscures the 2:1 block until the idioventricular discharge occurs early enough to allow the passage of the subsequent S-A nodal beat \(A_3\) in the diagram). Since the passage of \(A_3\) resets the His pacemaker, \(A_5\) is also transmitted, following which the "concealed" 2:1 block is reestablished until R again advances to the "supernormal" position. This cycle, with the numerical values chosen for the illustration, would repeat with each tenth S-A nodal beat. In the ECG record published by Jonas, the first, eleventh, and nineteenth P waves were propagated (fig. 11, 2nd and 3rd strips).

It is clear from the diagrams of figure 10 that still earlier positions of the idioventricular discharges would also permit the passage of the subsequent S-A nodal response; in other words, longer R-P intervals would have been followed by conducted beats. However, automatic recycling must occur each time the A-V node is successfully traversed. No opportunity for transmission outside the "supernormal" phase would be provided.

In his description, Jonas suggested that the QRS following a ventricular capture represented escape of the subordinate pacemaker and that the R-R interval was shorter than 1.4 sec because of a difference in the conduction times of the initial and terminal beats between the pacemaker focus and the ventricle. There is,
however, no compelling reason to assume that this response was not also propagated from
the atrium. The slightly different configuration
of the capture and escape complexes would
be expected as a result of the preceding
cycles: the ventricular captures occurred im-
mediately after the end of the preceding T
wave (R-R interval 0.52 to 0.58 sec in the
records shown), while the following ventricu-
lar complexes occurred after periods of 1.30
to 1.38 sec.

The diagrams of figure 10 indicate that
rather slight changes in nodal conductivity,
pacemaker frequency, or refractory periods
would result in striking changes in the tem-
poral pattern of responses. For example, slight
dereleration of the idioventricular pacemaker
or slight acceleration of the sinus node would
expose a 2:1 block. Slightly deeper retrograde
penetration of the node would convert Jonas’
case to one like that of Lepeschkin and Kim-
ura, in which case the “supernormal” period
would be abruptly shifted from an R-P of ap-
proximately 0.1 to an R-P of about 0.5.
Slight abbreviation of the refractory period
in the lower node would result in Wenckebach
periodicity, or 1:1 transmission. Furthermore,
it is obvious that the same fundamental pat-
tern, namely, “dissociation with interference
in the presence of 2:1 atrioventricular block”24
could be present without suggesting the diag-
osis of supernormal conduction. In the upper
half of figure 10 for example, the “super-
normal” period is defined by the V4A5 interval.
If the refractory period following the fourth
response were slightly longer, A5 would fail.
This situation, in fact, is evident in the tracings
published by Dressler and associates,24 who
called attention to the complex pattern of in-
terference but had no reason to invoke super-
normality in their careful analysis of A-V re-
lationships.

Still another comparable situation is pres-
et in one of the earliest reports of super-
normality, case 2 of Lewis and Master.2 The
basic pattern, instead of being 2:1 A-V block
as in Jonas’ case, is one of Wenckebach pe-
riodicity with a 3:2 A-V block. The pause
attending block of each third sinus beat is
occasionally interrupted by an escape beat.
When the escape beat is late, it prevents the
emergence of the initial response of the subse-
quent Wenckebach cycle; when it is early
enough, the sinus beat is propagated. The
pattern is equivalent to the diagram pertain-
ing to Jonas’ case (fig. 11), with the substitu-
tion of 3:2 for 2:1 block as the underlying
pattern.

A somewhat more complex example which
can also be explained on the basis of retro-
cession of the nodal refractory period was
described by Burchell.25 In this case of com-
plete heart block (case 1), occasional S-A
nodal impulses were propagated to the ven-
tricles, but only when the P waves fell in the
range of about 0.56 to 0.88 sec after the onset
of a preceding (idoventricular) QRS com-
plex. The conclusion was reached that a super-
normal phase, following the appropriately
spaced QRS complexes, permitted the suc-
cessful passage.

At first glance the record appeared to be an
element of a vagotropic mechanism. However,
the period during which A-V transmission
was possible encroached upon the time that
phasic vagal discharge should have been at its
peak. Furthermore, the P-P intervals appear to
be almost absolutely constant. Further exam-
ination of the records reveals that when a
QRS of ventricular origin fell just before or
just after the beginning of a P wave, the fol-
lowing sinus impulse was propagated. We
can assume that each sinus impulse penetrated
the A-V node partially. Failure of the non-
propagated responses to reach the His
bundle can be attributed to “decremental”
conduction within the node, which is equiva-
ient to saying that these responses penetra-
ted to a point at which the margin of safety for
propagation dropped below 1 because of the
combined effects of refractoriness following
the prior response and “deterioration” of the
nodal action potential. Pre-excitation of the
weak junction by a retrograde impulse, pro-
perly timed, would permit a longer recovery
time before the arrival of the next S-A nodal
response, and would improve the chance of
successful transmission.
Supernormal A-V Conduction

Figure 12

Schematic explanation of facilitation of A-V transmission by idioventricular beat in “complete” A-V block. Responses of sinus node origin assumed to fail repeatedly within A-V node at a level between cells Na and Nb. Abbreviated (simulated) action potential in Na and electrotonic “local” potential in Nb are characteristic of cells just above and just below a site of block. The “margin of safety,” which is assumed to be a function of the stimulating efficacy of Na and of the excitability of Nb, fails to reach the level of restoration of cells just above and just below a site of block. In the lower panel, pre-excitation from below “resets” the margin of safety curve and allows successful transmission of the subsequent atrial response.

A schematic explanation of Burchell’s case 1, which also applies to a number of other published examples of “supernormality,” including case 1 of Lewis and Master, is presented in figure 12. The illustration is an armchair hypothesis, similar to that proposed by Wolferth, but it is based on observed characteristics of nodal behavior. In part A of the figure, we have assumed that two successive atrial responses are arrested at the same intranodal junction. The abbreviated transmembrane “action potential” of unit Na, and the “local response” in a subjacent unit, Nb, were drawn to represent the electrical events just above and just below the site of block. Similar events have often been described. Below the simulated action potentials is a curve labeled “margin of safety” which represents the recovery of conductivity at the critical junction following the responses to A1 and A2. The horizontal line represents the level of recovery necessary for successful transmission from Na to Nb. It is implicit in this construction that if A2 were slightly delayed, propagation would occur.

In part B of figure 12, we have assumed that an idioventricular response, V1, has occurred sufficiently in advance of A1 to accomplish significantly earlier activation of Na and Nb. This is, again, compatible with well-known properties of the node. Pre-excitation of the cells at the critical junction resets the margin of safety curve, which reaches the value of 1 just prior to the arrival of A2. Although conjectural, these concepts are not different from those presented in figure 10, and experimental support should not be difficult to achieve.

B. “Supernormality” due to Longitudinal Dissociation

At least two of the examples reproduced in the book by Katz and Pick represent alternation of A-V conduction. In one of these (fig. 317) 1:1 transmission occurs with alternation of the P-R interval between 0.56 and 0.64 sec; the alternation can be explained on the basis of intranodal dissociation, as diagrammed in figure 8, without invoking a phase of supernormality. In figure 361 of the same work, illustrating a case of second degree heart block with an A-V ratio of 4:3, the P-R interval of the last propagated response in each group, though grossly prolonged, is shorter than the preceding one. In one of the tracings, five successive beats are transmitted, with P-R intervals of 0.48, 0.64, 0.59, 0.62, and 0.59. This is a special case of...
alternation in which an occasional sinus impulse fails to traverse the A-V node. Again, intranodal dissociation adequately explains the phenomena. Clearly, variations in neural influences may also contribute to the irregularity. When A-V transmission is intermittent, as in Wenckebach periods, quite pronounced phasic changes in arterial pressure must also occur; conversion of “simple” alternation of conduction to second degree heart block could easily result.

In a number of the cases reported by Langendorf, periods of P-R alternation and typical Wenckebach cycles were observed at different times in the same records. Transition between these patterns also appears in one of the illustrations in the study by Fisch and Steinmetz, who induced A-V alternans in dogs by infusion of potassium. In figure 6 of their paper, a period of alternating P-R intervals is succeeded by a 3:2 Wenckebach cycle, which in turn gives way to 2:1 block, and again alternation of P-R. Although transitions of this sort were cited by Pick and associates as evidence against the participation of a dual A-V transmission system, there is in fact no incompatibility. When successive sinus impulses experience progressively increasing delay in the A-V node, the chance that the last propagated response will engage only one of the available pathways is great. This pattern, in fact, quite commonly results in echoes in experimental preparations, whether the Wenckebach periods are imposed by atrial or by ventricular stimulation.

Examples of atrial echoes terminating Wenckebach periods have been published. In a 3:2 Wenckebach structure, it is easily possible that the first propagated response engages both pathways, while the second advances slowly through only one. The propagation pattern of the third response will now depend on the conditions at the time. If the second response traverses only pathway $\alpha$ through the upper portion of the node, but enters pathway $\beta$ from below at the intranodal junction, it may return toward the atrium and be extinguished en route. The third response will then fail. If, however, the second response traverses $\alpha$ but fails to enter $\beta$ either from above or from below, then the third atrial discharge will find the atrial extremity of $\beta$ recovered, and its propagation to the final common pathway and to the ventricle will be relatively “supernormal.” The variation of nodal conductivity which on one occasion causes the third response to be blocked, and on another permits it to pass with “paradoxical” speed need be very slight. In the hypothetical example described, the margin of safety for entrance of the $\beta$ pathway at its midnodal junction with $\alpha$ need shift only from just less to just more than one. In accord with clinical experience, events of this kind could occur only when nodal conductivity is depressed; that is, when the possibility of longitudinal dissociation of $\alpha$ and $\beta$ pathways is present, and when incomplete (“concealed”) penetration of any or all nodal transmission pathways is also possible. The transition from Wenckebach periods to alternating P-R periods can thus be explained without invoking a hypothetical supernormal period. Because other evidence of intranodal dissociation can be obtained under comparable conditions, the intervention of two pathways appears to be by far the more likely mechanism.

C. “Supernormality” due to Vagal Discharge

The possibility of varying or pulsatile changes in vagal discharge as a factor in supernormal A-V conduction has been considered in numerous case studies. Since changes in vagal activity would produce “expected” alterations of A-V conductivity, the cases which have been published are, in general, those in which this explanation has been rejected. In some of these, rejection does not seem to be entirely warranted.

As we have indicated in Section I-E (page 14) the lowest effective level of vagal activity should be expected at about 0.5 to 0.6 sec after a QRS complex. There are a number of published reports in which A-V transmission occurs preferentially when the P wave follows an idioventricular QRS complex by approximately 0.5 sec, and again when the
R-P interval is quite long, but not at intermediate stages. One of these has been published as figure 379 by Katz and Pick. In this tracing, which is reproduced in figure 13, P waves which followed an idioventricular QRS complex at an interval of 0.56 sec, or of 1.6 sec or longer, were propagated to the ventricles, while conduction failed at intermediate intervals. Propagation of the early P waves was ascribed to supernormality following incomplete penetration of the node by the idioventricular impulse; the late ones were assumed to find A-V nodal recovery sufficiently advanced to support conduction.

A reasonable alternative explanation is illustrated in figure 14. The reconstruction is based on the assumption that the lag between a QRS complex and the subsequent effect of the reflexly evoked vagal discharge on the A-V node is of the order of 0.6 sec, and that the effect is largely dissipated within another second. The cholinergic effects within the A-V node would thus be at a minimum at approximately 0.5 to 0.6 sec after an idioventricular discharge, would rise rapidly to a peak, and fall toward the same minimum about 1.6 sec later. The S-A nodal frequency, although said to be practically regular at a rate of 75, also exhibited phasic changes which fit this concept. Although the changes were not great, the P-P interval varied from 0.7 to 0.82 sec in the published records, with the longer intervals occurring for cycles beginning at a time when the vagal effect should have been maximal.

A similar vagotropic mechanism is also apparent in the description of case 1 in the report by Kline and associates. Although the authors stated that vagal effects do not provide an adequate explanation, this is by no means certain in the published records. It should be emphasized, for example, that relatively minor variations in the level of vagal discharge may spell the difference between propagation or no propagation in a depressed A-V node, even though only minor changes in S-A nodal frequency are observed. In case 2 of the same report, V-A transmission is said to occur only when the idioventricular beat occurs relatively early after a P wave. In the

**Figure 13**

An instance of advanced A-V block, reproduced from Katz and Pick, with permission of Lea & Febiger. In lead I, the second ventricular response was assumed to be facilitated by the prior ventricular event. A diagrammatic interpretation of this strip, excluding the first P wave, is presented in figure 14.
...electrocardiograms shown, any later QRS would collide with the next S-A nodal impulse in the upper node. There was, therefore, no test of the possibility that late ventricular responses might also have been propagated to the atria.

When partial or complete restoration of A-V transmission follows the implantation of an artificial pacemaker, interesting interactions between the competing pacemakers may occur, including phenomena attributed to supernormality. In one such case, described as case 1 in a study by Burchell, occasional P waves were followed by QRS complexes, interpolated between ventricular responses to the artificial pacemaker. Propagated responses occurred only when the P waves followed the preceding pacemaker stimulus by 0.36 to 0.40 sec; it was suggested that a supernormal phase permitted transit of the A-V node only during that limited period. If we assume, as in the analysis illustrated in figure 14, that the A-V nodal effect of a vagal surge will begin approximately 0.5 to 0.6 sec after a QRS complex, then the best "chance" for successful A-V propagation will occur for P waves falling early enough to have traversed the upper node before cholinergic block occurs. By the same token the S-A nodal cycle (P-P interval) might be expected to be slightly longer when the initial P wave occurs about 0.6 sec after a QRS complex than when the cycle is initiated...
SUPERNORMAL A-V CONDUCTION

earlier or later.\textsuperscript{18, 19} In the limited tracings reproduced in the paper, this appeared to be true.

At a later stage in the recovery of the same patient, retrograde conduction was observed when the ventricular stimulus fell 0.36 sec after a preceding P wave. This again was suggested as evidence of supernormality, but the published records show that the inverted P waves occurred at precisely the time when a P wave of sinus origin might have been expected. In other words, no \textit{later} impulse of ventricular origin could have reached the atria. In the same record, two beats of sinus origin were observed to propagate to the ventricles when the interval between stimulus and P wave was 0.42 sec. Other comparable intervals without A-V conduction in the same record indicate that the margin between successful passage and failure was precarious; again, the “supernormality” could equally well be attributed to pulsatile variations in the level of vagal activity. This reexamination of the events described by Burchell may not, of course, be taken as proof of the absence of supernormal conduction; it merely indicates that alternative mechanisms have not been eliminated.

In several reports, the intervention of increased vagal discharge is clearly recognized as the precipitating factor in paroxysms of complete heart block, but the resumption of A-V transmission is attributed to “supernormality” following an idioventricular beat. Case 1 of the report by Ashman and Herrmann\textsuperscript{31} is one of the earliest examples. In this patient, repeated episodes of ventricular arrest were preceded by pronounced deceleration of the sinus node. Atrial slowing continued for one or two cycles after the last propagated response, but was followed by progressive acceleration. When, and only when, a ventricular “escape” occurred, A-V transmission was resumed. The authors rejected a vagal mechanism because: “When the auricles accelerate during a period of ventricular standstill, there is no relief from block until an idioventricular beat occurs. If the vagus were responsible for the block, release of the conducting tissues from vagus influence should occur synchronously with the auricular acceleration.” The flaw in this argument was discussed by Mack and associates.\textsuperscript{32} Coronary perfusion must cease shortly after the last ventricular systole (within about 2.5 sec in the case reported by Joossens and associates\textsuperscript{33}). In experimental preparations, ischemia causes impairment of A-V transmission before intraventricular conduction is seriously depressed. Accordingly, vagally initiated block may give way to ischemic block, and resumption of transmission cannot occur until an idioventricular beat restores coronary perfusion. Such episodes should tend to be cyclic. Each time arrest occurs, there will be a reflex sympathetic discharge, together with reduced vagal activity. Resumption of ventricular contractions will result in an overshoot of arterial pressure as the accumulated venous blood is discharged into a constricted arterial tree, whereupon a renewed baroreceptor discharge will initiate another cycle of cardiac inhibition.

D. The Sanctity of the R-P-PR Relationship

The events, described in preceding sections, and the postulates, derived from them, demand reexamination of the dictum that a brief R-P interval should result in prolongation of the subsequent P-R. This relationship has been accorded the status of another law of the heart, to the extent that any deviation is considered a paradox; as a result, exceptions to the rule are cited as evidence of “supernormality.” It is necessary then to consider the circumstances to which the “law” does or does not apply.

When the atria fire at a constant frequency, whether in response to the sinus node or to an artificial pacemaker, and when the P-R interval is constant from beat to beat, we may assume that each propagated response is followed by a period of refractoriness which, for any single element in the conduction pathway, will be approximately constant from beat to beat. If a series of rhythmically driven responses is followed by a premature atrial contraction which successfully traverses the A-V transmission system, the resulting P-R
interval will be related to the prematurity of the atrial impulse. In this orderly relationship, a curve can be drawn which demonstrates that P-R is inversely related to R-P. But does this curve permit the conclusion that R-P is the determinant of P-R? The premature atrial response, A, must enter A-V nodal tissue which is recovering progressively from the refractory trace of A1. The briefer the A1A2 interval, the more refractory will be the upper node. Delay within the refractory tissue permits a longer time for recovery in the lower node; accordingly, it is not the ventricular arrival time of the preceding response which determines the state of conductivity at the time of the premature impulse. In other words, a curve which relates the conduction time to the A1A2 interval (and which will, of course, have the same configuration as the PR versus RP relationship) defines the cause-effect relationship more accurately. Nevertheless, the RP-PR relationship is "normal" under these circumstances; exceptions occur, as outlined in Section I-C (page 11; and fig. 5), when intraventricular rather than A-V nodal tissues appose the refractory barrier to propagation.

A similar but not identical relationship of RP to PR pertains when the atrial driving frequency is increased. As R-P shortens, P-R lengthens; to the effects of "refractoriness" we now add cumulative effects which, for want of a better term, we may ascribe to fatigue. Again it is the interval between successive entries of the node rather than the interval between the last emergence and the succeeding entry which determines how facile will be the transit.

The RP-PR relationship will be further modified if we initiate changes of atrial frequency by altering the level of adrenergic and cholinergic influences. At any given frequency (or any degree of prematurity of an atrial response) the P-R interval will be longer under vagal, and briefer under sympathetic, drive. We have, obviously, a family of RP-PR curves, depending upon the environment and the history of the conducting tissues. Similar variations must, of course, attend the administration of drugs.

None of the variations described cause any difficulty if the basic conditions are recognized; R-P and P-R are inversely related if these are the only variables. Consider now the situation when variations in R-P occur as a result of occasional and randomly placed ventricular premature beats. The effect of an idioventricular beat upon the propagation of a subsequent atrial response will depend not only upon its temporal position in the cycle but also upon whether or not it penetrates the node in a retrograde direction. If a ventricular premature contraction (VPC) occurs late in the cardiac cycle, just prior to the expected arrival of a propagated supraventricular beat, it will certainly block that response. In this sense, the sanctity of the RP-PR relationship is preserved. If the VPC occurs earlier, but does not penetrate the node, it can only block or delay the subsequent response; again the relationship is preserved. If it is still earlier, and if the basic atrial frequency is sufficiently slow, and if retrograde transmission is impossible, the propagation of a subsequent atrial response will be unimpeded; again, the law is inviolate. If, however, the idioventricular response penetrates the node, the law no longer applies. The course of recovery through the stage of relative refractoriness of intranodal elements now depends upon the depth of retrograde and antegrade penetration, upon the speed with which such penetration is effected, and upon whether or not pre-excitation at a given level permits earlier recovery or leads to additional "fatigue"; in short, it depends upon the temporal position of the VPC and upon the previous history of the transmission pathway. As discussed in detail in Sections I-B (page 8) and II-A (page 17), and represented in figure 10, a brief R-P interval may either facilitate or impede propagation, depending upon the preceding events.

We have not attempted to discuss each of the many published examples of so-called supernormality, but all of those we have examined appear to fit one of the categories we have described on the basis of experimental...
SUPERNORMAL A-V CONDUCTION

observations. We have not encountered a single clinical case in which the "paradoxical" relationship between R-P and P-R can be accepted as proof of supernormal A-V conduction. We suggest, therefore, that the alternatives we have proposed should be considered before any complex arrhythmia is accepted as evidence of supernormal conduction.

Acknowledgment

The authors are grateful for the collaboration of numerous associates who participated in the discussions which led to this analysis, and in the conduct of many of the experiments upon which it is based. In particular we thank Drs. Carlos Mendez and Jaok Han, and also the man who, perhaps unaware of his role, provided the initial stimulus, Dr. Louis N. Katz.

References

dependent upon the time relations of auricular and ventricular systole: Supernormal phase. Amer Heart J 17: 524, 1939.


On Teaching

The teacher and student of history—and all human situations have a history—must follow the advice given by the resourceful wife to her husband as he stood on the top of a tall chimney from which the scaffolding had collapsed. All ladders were too short, all efforts to throw up ropes had failed. It was then the wife called, “John, take off your sock and unravel it, but begin at the toe.” That thread would make it possible to lift ropes of greater strength, but to get the thread he must begin his unraveling at the toe. The simplest political or social situation we study, whether in the nation, the state, or the school district, is the toe of a historical sock. Good teaching must at least start the unraveling whose end-result will lift problem and class to the level where long views give that perspective and proportion that are the finest and most permanent result of teaching and study.—Guy Stanton Ford: On and Off the Campus. Minneapolis, University of Minnesota Press, 1938, p. 387.
An Appraisal of "Supernormal" A-V Conduction
G. K. MOE, R. W. CHILDER and J. MERIDETH

Circulation. 1968;38:5-28
doi: 10.1161/01.CIR.38.1.5

The online version of this article, along with updated information and services, is located on the World Wide Web at:
http://circ.ahajournals.org/content/38/1/5

Permissions: Requests for permissions to reproduce figures, tables, or portions of articles originally published in Circulation can be obtained via RightsLink, a service of the Copyright Clearance Center, not the Editorial Office. Once the online version of the published article for which permission is being requested is located, click Request Permissions in the middle column of the Web page under Services. Further information about this process is available in the Permissions and Rights Question and Answer document.

Reprints: Information about reprints can be found online at:
http://www.lww.com/reprints

Subscriptions: Information about subscribing to Circulation is online at:
http://circ.ahajournals.org//subscriptions/