Physiologic Evidence for a Dual A-V Transmission System

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A study of the transmission of early premature contractions between atria and ventricles and in the retrograde direction in the dog heart suggests the existence of two parallel A-V conduction pathways communicating with each other over one or more branches. The evidence is based on the excessive delay of very early premature responses in traversing the node, suggesting that a slowly conducting pathway recovers earlier than the normal "fast" pathway; on the echoes back to the chamber of origin of early premature responses; and on ventricular electrograms of "abnormal" configuration obtained during early premature responses. These observations and the hypothesis to which they lead provide a natural explanation for reciprocal rhythm and nodal paroxysmal tachycardia.

Because the A-V node is not easily accessible to direct observation, its properties are defined in terms of time intervals between atrial and ventricular events: estimates of its conduction velocity are made in terms of the P-R interval, and its functional refractory period (FRP) is defined as the least obtainable interval between two successive ventricular responses propagated from the atria. Since it can be shown, at least at frequencies within the physiologic range, that the FRP of the A-V transmission system exceeds that of either the atria or the ventricles, it follows that the temporal behavior of early premature responses is determined by the properties of the junctional tissue itself.

If the conduction pathway were a homogeneous muscular syncytium smoothly bridging the atrioventricular junction, an early premature impulse would be expected to traverse it in much the same manner as an impulse coursing through a relatively refractory axon. The early response, impinging upon relatively refractory and therefore slowly conducting tissue, should be propagated at an initially slow rate; but since the delay engendered by subnormal conduction velocity permits fuller recovery of more distal elements, acceleration of the impulse should occur until, in a sufficiently long pathway, the end of the refractory period is encountered and normal conduction velocity is achieved. In such a system, any impulse entering the conducting bridge, at any time during its relatively refractory period, would reach the ventricle only at the end of the relatively refractory period of the node.

These rules predict that the interval between two successive ventricular responses, R₁ and R₂, should be frozen at the limit of the nodal refractory period, as the atrial premature response, R₂, is advanced earlier in the cycle, up to the earliest atrial response capable of transmission to the ventricle. Krayer and associates, however, found that early secondary responses, unduly delayed in transmission, often yielded longer R₁-R₂ intervals in the ventricles than somewhat less premature impulses. Recognizing that such behavior was inconsistent with a syncytial or continuous conducting pathway, they postulated a synaptic type of transmission at some point of junction between atria and ventricles.

Similar observations of the "abnormal" behavior of early premature responses in this laboratory led to the suggestion that two or more parallel pathways may bridge the atrioventricular gap. The present study was initiated to test this hypothesis.
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Methods

Most of the experiments were performed on dogs of both sexes ranging in weight from 7 to 35 Kg., although substantially similar results were obtained in 3 turtles, 2 sheep and a cat. The animals were anesthetized with sodium thiopental, 15 to 20 mg./Kg., followed by sodium barbital, 180 to 220 mg./Kg. The heart was exposed through a midsternal thoracotomy. Bipolar stimulating and recording electrodes were attached to the margin of the right auricular appendage and to the epicardial surface of the right ventricle near the apex. The S-A node was crushed to reduce the spontaneous rate. In most experiments the vagus nerves were sectioned in the neck; in some experiments one or both stellate ganglia were excised. Heart-lung preparations were employed in a few experiments.

Experimental procedures were similar to those used in a previous study. The heart was driven at various frequencies by stimuli (S) applied through either the atrial or ventricular electrodes. Premature responses were induced by a second (S) and sometimes a third (S) stimulator triggered to fire at controlled intervals after each sixth driving stimulus. To provide a "vacant" period for the exposure of possible reverberating activity of the node following delivery of the premature stimuli, the next recurrent driving stimulus was dropped out by a relay triggered by S. Electric responses of atrium and ventricle were monitored on a dual beam oscilloscope, and were recorded from a companion oscilloscope with a Grass kymograph camera at a paper speed of 10 cm./sec. Responses to S are referred to as "basic" or primary beats, R1; responses to S as premature or secondary responses, R2; and responses to S as third order or R3 responses.

Definitions

Krayer, Mandoki and Mendez defined the functional refractory period of the node as the least interval between two successive ventricular responses both propagated from the atria, but if the A-V conduction pathway is long enough, this interval will bracket the total refractory period of the node; i.e., functional plus relatively refractory periods. In the present study the term FRP designates the least interval between two responses entering the node.

Relatively Refractory Period. The relatively refractory period (RFP) is usually defined as the interval between the end of the absolutely refractory period (which has no precise meaning) and the restoration of normal excitability. Since we have no control over the amplitude of the stimulus delivered to the A-V transmission system, that portion of its relatively refractory period which precedes the end of its FRP has little functional significance. In reference to the node, then, the RFP is defined as the interval between the end of the FRP and the restoration of normal excitability. It is assumed that normal excitability is reached when a response yields the normal, or basic, interval between atrial and ventricular excitation.

Velocity of Transmission. This of course is not known, for we can only guess at path length. We therefore refer to "fast" and "slow" pathways, assuming that impulses which yield a brief A-V interval are propagated more rapidly than those which result in longer intervals.

For brevity the term "A-V node" is applied to the entire A-V transmission system, i.e., the node and A-V bundle.

Results

Time Relations of Early Premature A-V Responses. Premature atrial responses were induced at various intervals after each sixth basic (driven) beat. The early portion of atrial diastole was scanned at intervals of 5 msec. or less, the later portion at intervals of 5 to 40 msec. The resulting atrial intervals, R1-R2 were plotted against the corresponding ventricular intervals as in the studies of Krayer and associates.

Figure 1 illustrates three patterns of relationship encountered with approximately equal frequency. In all three examples, late premature responses were propagated at a speed approaching or equaling that of the primary responses; the atrial and ventricular R1-R2 intervals approached equality; and the resultant points fell on or near a 45° line projected from zero time. Earlier premature responses, transmitted from atrium to ventricle at subnormal speed, yielded ventricular intervals exceeding the corresponding atrial intervals.

In part A of the figure, the temporal relationships between atrial and ventricular responses do not deviate from the behavior expected in a simple homogeneous conducting system interpolated in series between atria and ventricles. Reading the figure from right to left, as the atrial R1-R2 intervals were diminished, a time, X, was reached at which no further shortening of the corresponding ventricular intervals occurred; these remained constant up to the earliest propagated response as the atrial intervals were still further reduced.

In part B, similar conditions prevailed until the atrial R1-R2 interval was reduced to 195 msec., X. At this point, there was an abrupt increase in the ventricular interval, which then remained constant at the new level up to
Fig. 1. Plot of interval between basic and premature responses in the atria $(R_1-R_2)_A$ in relation to the corresponding ventricular interval $(R_1-R_2)_V$. Three examples from different experiments. A: Exp. 1/31/55; dog, 11.5 Kg., heart-lung preparation, basic frequency 104/min. B: Exp. 9/28/54; dog, 15 Kg., both stellate ganglia excised, basic frequency, 164/min. C: Exp. 1/27/55; dog, 17.5 Kg., nerves intact, basic frequency 154/min. Both time scales in milliseconds.

the moment of the earliest propagated response.

In part C the ventricular intervals are seen to have increased gradually and progressively as the atrial intervals were reduced below 180 msec. at $X$.

It may be suggested that part A represents a case in which, while more than one conduction pathway may have existed, only one was exposed under the conditions of the experiment. Part B can be considered as a case in which there existed two parallel pathways, one having a relatively greater conduction velocity but a longer refractory period than the other. In part C there may have been several parallel pathways with varying conduction velocities and refractory periods, or two basic systems communicating with each other through branches of different lengths represented.

In figure 2, similar data are plotted schematically as time against distance. Since we know neither the distance travelled nor the relative time occupied by sequential propagation through the three tissues, the horizontal lines dividing the graphs into atrium, node, and ventricle are drawn at arbitrary levels.

* This appears to be at variance with the well-known fact that the more rapidly conducting axons have the shorter refractory period. There is, however, no reason to suppose that conducting elements within the heart must adhere to the rules of axonal transmission. Furthermore, we must emphasize that "slow" impulses may not actually travel at lesser speed, but may traverse a longer path or be delayed longer at some junctional point in transit.

† The time values allowed for intra-atrial and intranodal conduction correspond approximately with those obtained in a study of spontaneous nodal activity by Rosenblueth. Whether or not they are exact has no significance in the interpretation of events in this or subsequent figures; assumption of different time values alters only the quantitative details of the analyses.
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Figure 3. Exp. 9/28/54; dog, 15 Kg., stellates excised, basic frequency, 104. At left, tracings A, B, and C of original records; atrial responses below, ventricular above. Interval between stimulus artifact and response of atrium in tracing A indicates that S→ was delivered well within the atrial FRP. Diagram constructed as in figure 2. Crosshatched area represents FRP of faster pathway; lined area, FRP of slower pathway. In this and all subsequent figures, transmission through faster pathway is indicated by solid lines; through slower by broken lines. Abscissa, msec.

Basic responses at the left of each segment, travelling at normal velocity through fully excitable tissue, are represented as straight lines drawn from zero time (atrial R1 response) to the time of the resulting ventricular activation. Figure 2A illustrates the simple relationships expressed in figure 1A. The earliest propagated premature response, line a→a, is assumed to enter the node just after the expiration of its functional refractory period and is therefore assumed to travel at an initially slow but accelerating rate until the slope (velocity) characteristic of the primary response is reached (see Mendez, Gruhzit, and Moe,1 figure 6). Responses b and c, encountering less refractory tissue, are shown to travel faster than a, but slower than the primary response. Response d, finding the node fully recovered, is propagated at normal speed along a line parallel with that of the primary response. The ventricular R2-R2 interval for the three early premature responses was constant at about 225 msec.; it is certain that the nodal pathway must have been long enough to permit attainment of normal conduction velocity in all three cases before emergence at the ventricular surface, and the ventricular R1-R2 interval defined the total nodal refractory period, functional plus relative.

Figure 2B, derived from the same experiment as A but under different conditions, illustrates behavior similar to that of figure 1B. Here it is assumed that two parallel pathways having different conduction velocities and different refractory periods connect atria with ventricles. It is postulated that premature responses a, b, and c, finding the “normal” faster pathway inexcitable, engaged the slower tissue and emerged in the ventricle at the time abc, yielding the relatively long ventricular R1-R2 interval of 260 msec. Response d, however, encountered the faster pathway in a state of excitability which, though subnormal, permitted earlier appearance in the ventricle.

Events similar to those of figure 2B are diagrammed in figure 3, together with tracings of the original records, to emphasize the abrupt change of the ventricular R1-R2 interval resulting from a very slight shift in the position of the atrial premature response. The records at the left, labelled A, B, and C, are so aligned that all the primary basic responses in the atrium begin at time zero. In each record the primary response was observed in the ventricle 120 msec. after its initiation. Part A depicts the earliest possible premature atrial responses, a vertical line indicates the moment of its origin relative to the later responses in B and C. A second vertical line, dropped from the moment of arrival of the second A response at the ventricular electrodes, illustrates that the second ventricular response of C occurred more than 30 msec. earlier than the second response of either A or B, although the antecedent atrial response was about 10 msec. later than that of B.

This experiment, lacking further data, also fits the situation illustrated in figure 1B; i.e., two parallel systems having different refractory periods, and either different conduction velocities or different path lengths. The graphic reconstruction at the right of figure 3 illustrates the possible course of the premature responses through the transmission system in terms of this hypothesis. It is assumed that responses 2a and 2b, entering the node at a time when the faster pathway was still refractory, were propagated to the ventricle over the slower route, but that 2c and all later responses found both systems excitable.

Time Relations of Retrograde (V-A) Responses. The propagation of impulses in the retrograde direction was studied in the same experiments...
by similar procedures. The heart was driven by stimuli applied at a suitable basic frequency through bipolar electrodes attached near the septal margin of the right ventricle, and premature responses were evoked at various intervals by secondary stimuli delivered through the same electrodes.

The results of retrograde excitation differed only quantitatively from those obtained in the normal direction. While the earliest possible atrial premature response usually reached the ventricle, the earliest possible ventricular premature beat often failed to pass the node. The V-A conduction intervals for both primary and premature responses were usually appreciably longer than those obtained in the normal direction; and the effective refractory periods of the nodal transmission system were usually longer when studied in reverse. Complete failure of retrograde transmission, observed but rarely occurred following complete thoracic sympathectomy, or late in the life of heart-lung preparations. With suitable correction for these quantitative differences, and with transposition of the axes, the graphs of figure 1 apply equally well to the retrograde transmission process. However, hearts which exhibited behavior of the type shown in figure 1A in the normal direction often showed behavior of the type demonstrated in figures 1B and 1C when studied in retrograde, and vice versa. Indeed, a heart characterized by the time relations illustrated in figure 1A could often be shifted to the type illustrated in 1B or 1C by such procedures as an increase or decrease of pulmonary ventilation, a change in the basic frequency, or by nerve section or stimulation.

The Echo Phenomenon. If the schematic representation of figure 3 approaches accuracy, a return route to the atrium should have been available to responses 2a and 2b, for at the time of arrival in the ventricle the “fast” limb should have recovered its excitability. The sum of conduction times from atrium to ventricle and from ventricle back to atrium would surely have exceeded the refractory period of R2 in the atrium, permitting an echo response in that chamber. More commonly than not, reciprocal excitation of the atrium was indeed observed; figure 4 illustrates such an experiment.

In figure 4 the basic response, R1 is reconstructed as a straight line connecting atrium with ventricle at the normal A-V interval of 140 msec. The FRP of the faster pathway, as represented by the crosshatched parallelogram, lasted 190 msec. (premature responses earlier than 190 msec. arrived in the ventricle at the time indicated as 2a; the response initiated at 192 msec. arrived at the earlier time, 2b). The “normal” conduction interval of the slower pathway was estimated (from the conduction intervals of responses initiated between 2a and 2b, but omitted from the graph) to be about 225 msec., or 85 msec. longer than the fast system. Its FRP is indicated by the diagonally barred parallelogram.

The response 2a, entering the slower path when the faster must have been refractory, induced a third response, 3, in the atrium 495 msec. after R1, and 335 msec. after 2a. This “echo” response had precisely the same configuration as atrial responses resulting from stimulation of the ventricle. The re-entering pathway is represented by an arbitrary diagonal line projected back from the echo, along a time course characteristic of retrograde transmission in this experiment, to intersect the 2a response at a point near the ventricle. Since the point in distance and time at which the re-
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**Fig. 5. Exp. 10/12/64; dog 17 Kg., A-V records (left-hand column) with cardiac nerves intact, basic frequency 118; V-A records after removal of stellate ganglia and section of right vagus at basic frequency 94. Peaks of atrial responses retouched. Short vertical lines 1, 2, and 3, moment of application of respective stimuli in uppermost records. Long vertical lines, time of arrival of ventricular response to earliest atrial S1 (left) and atrial response to earliest ventricular S1 (right). Later responses in each case are echoes.**

Response left the nodal tissue and entered the ventricular conducting system is unknown, it is impossible to say whether the echo rebounded from the ventricle itself, or from some more proximal communication between the two pathways.

Echo responses were also obtained frequently with retrograde excitation of the nodal system (see figure 6B). In many experiments early premature beats reverberated whether evoked by atrial or ventricular stimulation; in other experiments echoes were elicited by atrial or ventricular stimulation, but not by both; only rarely were they unobtainable from either side of the node.

The necessary conditions for production of reverberating responses could not be predicted, but it was apparent early in the course of the study that echoes did not often appear when the time relations of atrial and ventricular excitation were of the pattern shown in figure 1A, and that they could usually be elicited when the pattern shown in figure 1B or 1C was present. In these instances echoes appeared only when premature beats were induced during the time period when, presumably, only the slower conduction pathway could be entered; that is, before the expiration of the refractory period of the faster path. The characteristics of echo responses thus support the concept of two or more A-V conduction paths.

**Multiple Echoes.** If an early atrial premature response can be introduced to the slower of two systems while the other is still refractory and can therefore rebound through the fast path to the atrium, one might expect, if the time relations were appropriate, that the echo response would, on its return to the atrium, find the slow system again available for propagation back to the ventricle. This possibility would be very great indeed if the two paths did not intercommunicate, for the relations of conduction time and refractory periods would be easily compatible. If communicating branches exist, it is much less likely that a self-sustaining circuit could be established, for the likelihood of extinction within several circuits of different lengths becomes great.

In many experiments, nevertheless, we have succeeded in inducing multiple echoes. One of these is illustrated in figure 5. All records in this figure were taken from the same heart; atrial electrograms appear in the upper, and ventricular responses in the lower trace of each portion. On the left are responses to three serial stimuli applied to the right atrium. In each of the parts, A, B, C, and D, the driving stimulus, S1, applied at zero time, was followed by the earliest possible S2. In part A, the third stimulus, S3, was delivered too early to evoke a ventricular response. In part B, the S3 response was transmitted to the ventricle and resulted in an atrial echo which was followed by another ventricular response: i.e., the R3 response made 3 serial excursions through the node. In part C, a later atrial S3 produced a similar response pattern, with somewhat different time relations (note that the ventricular response R3 was recorded earlier than in part B). In part D, the response R3, which must now have activated both conduction pathways, arrived still earlier in the ventricle, and failed to evoke an echo response.

The right of figure 5 portrays four examples of retrograde responses. In part A, the R4 responses failed to traverse the node. In part B a later response reached the atrium and echoed to the ventricle. In part C, a still later response was conducted to the atrium, echoed back to the ventricle, returned again to the atrium, and re-echoed in the ventricle, a total of 4
passages through the node. As in the A-V series, a still later response \( D \), which must have activated both pathways, failed to yield an echo.

**The Electrical Configuration of Early Premature Responses.** If two pathways with different properties communicate between atria and ventricles, it is possible that the two tissues are closely intermingled in space and feed into a final common path, possibly the bundle of His. It is also possible that the tissues are spatially separate, supplying the ventricle through channels which diverge anatomically as well as physiologically. In support of this possibility, the earliest premature response propagated from the atrium often yielded ventricular electrograms of grossly different configuration than those obtained from later responses.

Figure 6 incorporates records and graphic reconstructions of two such experiments. The records \( A-1 \) and \( 2 \), at the left, and the reconstruction \( A \) at the right of the figure, were selected from an experiment of the type shown in figure 1B; i.e., one in which slight abbreviation of the atrial \( R_1-R_3 \) interval resulted in an abrupt increase of the corresponding ventricular interval. Record \( A-1 \) was taken just before, and record \( A-2 \) just after, the sharp break marking the end of the functional refractory period of the faster pathway.

The atrial \( R_2 \) response of record \( A-1 \) was less than 2 msec earlier than that of \( A-2 \) (the corresponding moments have been separated by 10 msec in the diagram to avoid confusion of the lines), but the electrical configuration of the ventricular \( R_3 \) in \( A-1 \) was **opposite in polarity** to that of \( A-2 \). The change of polarity can **not** have been due to any aberration of intraventricular conduction resulting from relative refractoriness in the ventricle itself, for the "abnormal" response occurred a full 50 msec. **later** than the "normal" \( R_2 \) of record \( A-2 \). It can only be concluded that the gross difference in the order of activation of the tissue under the ventricular electrodes in the two cases must be referred to the A-V transmission system.

As might be expected, the major dissociation of the conducting pathways in \( A-1 \) permitted return of an echo to the atrium, and a re-echo to the ventricle. The reconstruction \( A \) illustrates a possible mechanism for the echo phenomena in this experiment. The premature response of record \( A-1 \), shown as the broken line \( 2a-2a \), is assumed to have traversed only the slower of two parallel pathways (\( s \) and \( f \) in the sketch at the extreme right), leading to "abnormal" activation of the ventricle. At some point at or near the ventricular margin of this pathway the impulse \( 2a \) is shown to engage the faster pathway for retrograde transmission to the atrium at 3. At the atrial margin of the node, re-entry of the slow pathway would again have been possible, resulting in a ventricular "re-echo." In harmony with this
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interpretation, the ventricular echo, 3, exhibited the electrical configuration assumed to be characteristic of activation over the slow pathway. The experiment shown in figure 6A thus includes three features which compel the assumption of a dual transmission system: (1) an abrupt shift in the ventricular R1-R2 interval as the atrial interval was altered slightly, (2) multiple echoes, and (3) a change in the electrical configuration of the ventricular response.

Figure 6B illustrates another phenomenon consonant with those of 6A. The record B-1 shows a similar reversal of polarity in the ventricular response to an early atrial premature beat. Normal activation of the ventricle over the faster transmission pathway yielded a negatively directed spike, while premature excitation over the slower route caused an upward spike. The record of B-2 and the accompanying diagram show responses to premature retrograde excitation in the same animal, resulting in a ventricular echo. Assuming that the premature retrograde response must have been transmitted to the atrium over the slower pathway, it follows that re-entry of the ventricle could only occur over the faster, or “normal,” path. The echo response (and this was always true in such experiments) had the configuration and polarity of a basic R1 response propagated from the atrium over the fast system.

Atrial responses to early ventricular excitation also occasionally exhibited “abnormal” configuration, but much less commonly than the ventricular responses to early atrial activation.

Evidence for Intercommunicating Branches

A-V excitation. In many records of early responses, we have been forced to conclude that the postulated parallel pathways cannot be totally insulated one from the other, but must communicate by one or more branches. On occasion, even when the behavior of early responses suggested dissociation of the two pathways (as in figure 1B), an early S2 applied to the atrium failed to evoke an echo. In the experiments of figure 2B and figure 3, for example, the early responses which were assumed to have entered the A-V transmission system when only the slower pathway was available should have been able to engage the faster route at its ventricular extremity for the return journey to the atrium. The absence of echoes in these experiments could not be ascribed to failure of retrograde conduction, for it was established by ventricular excitation that V-A propagation should have been possible at the appropriate moment. It was therefore considered possible that the early premature atrial response, beginning its journey in the slower limb of a dual system, could encounter an excitable branch of the faster pathway at some point of intersection so close to the atrium that its early return to that chamber would be concealed by the persistence of refractoriness in the atrium itself.

Experiments designed to test the “short branch” hypothesis were conducted in three animals which exhibited the proper response pattern. The appropriate conditions were: (1) a sharp break in the curve relating ventricular to atrial intervals (as in figure 1B), (2) a clear cut alteration in the electrical response of the ventricle to early atrial activation, and (3) absence of atrial echoes. It was assumed that if the faster pathway were “silently” occupied by retrograde re-entry over a short branch, then a third response initiated in the atrium soon after its recovery from R1 should again find only the slower system available for transmission. These experiments strongly supported the hypothesis that gross dissociation of the conducting elements was indeed exposed by the third-order stimuli.

It will be recalled that in the experiment shown in figure 2B, early premature responses exhibited the temporal and configurational patterns assumed to be characteristic of a dual transmission mechanism, but yielded no echoes. Figure 7, based on the same experiment, is constructed from a series of four third-order responses, selected from a careful scan of the interval following the earliest possible secondary response, R2. Assuming that the secondary response, 2, reached the ventricle over the slower pathway, it would be expected, in the absence of communications between the two conduction routes, that the earliest pos-
possible atrial R 3 should find the “normal” faster pathway available for transmission. It was discovered, however, that atrial responses initiated during the available 20 msec prior to 3a were not propagated to the ventricle. Both routes must have been refractory. Furthermore, responses 3a, 3b, 3c, and all intervening responses, resulted in the ventricular configuration assumed to be characteristic of the “slow” system. Between 3b and 3c, the A-V conduction interval remained constant at about 190 msec, a full 50 msec. longer than the normal interval for the faster system. It follows that the transmission system traversed by these responses must have been fully recovered at the moment of entry of 3b, and the slope of the parallel broken lines delineating the passage of 3b and 3c must approximate the normal conduction velocity of the system. This prolonged exposure of the slower pathway can have been possible only if the faster system remained unavailable for a prolonged period after R 3; and the faster route can have been made unavailable only if silently activated by R 2. Now, since response 3d, initiated only 10 msec. later than 3c, was propagated to the ventricle at normal speed (conduction interval about 140 msec.) and yielded a “normal” electrogram on its arrival, and must therefore have traversed the faster route; it is obvious that the refractory state resulting from the silent secondary activation must have terminated between 3c and 3d. This datum permits more quantitative analysis of the silent activation pattern.

According to the short branch hypothesis, the portal of entry into the fast system at its atrial origin must have been discharged by retrograde transmission of the response R 2 over a branch communicating with the slower trunk. Since the resulting period of refractoriness at the portal of entry must have ended between 3c and 3d, the line Y was projected back from this moment along a slope corresponding to the retrograde conduction velocity (as determined by study of V-A conduction intervals). This line, Y, defines the end of the refractory period in the recurrent branch, but does not locate the points of its origin and insertion along the course of the two conduction pathways, nor does it place, in time, the course of activation of the branch.

Additional logical assumptions, however, permit an approximate reconstruction. These assumptions are: 1. The retrograde activation of the branch by R 2 must have reached the atrial junction before the expiration of the atrial refractory period (else an echo should have resulted). 2. The R 2 response could not have entered the fast branch from the slow pathway before the expiration of the refractory period of R 1 in the fast tissue. 3. The refractory period of the R 2 response in the fast system must have been shorter than that of R 1 (relationship between cycle length and FRP; see Mendez and associates). 4. The retrograde activation process must have entered the fast system too late to reach the ventricle before the arrival of R 2 over the slower route (R 2 had the “slow” configuration). 5. The response 3d must have travelled for the major part of its journey through tissue which was no longer even relatively refractory, for the resulting A-V conduction interval was within a few milliseconds of the normal value for basic responses. Since the duration of the relatively refractory period of the fast tissue following a basic response was found to be 35 msec. elapsed be-
between the end of the FRP of the secondary response and the subsequent transmission of 3d over the principal trunk of the fast system.

Consonant with these assumptions, the pattern of silent activation was completed. The line Y' was constructed parallel with and 35 msec. earlier than the course of 3d, to represent the end of the FRP of the principal fast pathway following its activation by R2; another line, X', parallel with the first, was projected down from the assumed ventricular margin of the node to represent the course of the excitation process of R4 in this pathway. These two lines, bracketing the stippled area which denotes the FRP of the tissue, represent the maximum possible FRP. Activation could not have occurred earlier, nor could recovery have occurred later.

The point of intersection of the lines X and X', which describe the course of activation of the main trunk and its branch, must approximate an anatomic junction; namely, the point of divergence of the branch from the parent trunk as indicated in the sketch to the left of the diagram proper. The intersection of line X with the broken line Z then represents the point of insertion of the branch into the slow system.

More detailed consideration of the data and of the assumptions to which they lend logical support fixes, within relatively narrow limits, the location of the lines as drawn in the diagram. Further analysis need not be cited to justify the conclusions that: (1) early secondary activation of the A-V transmission system resulted in temporal dissociation of the two conducting pathways for a period exceeding 150 msec., (2) the faster pathway must have been silently activated by the response R2, and (3) the activation of the faster pathway must have been accomplished by retrograde passage over a branch too short to permit an atrial echo.

Before proceeding to analysis of other experiments, we may consider two other characteristics of the experiment of figure 7 which are worthy of mention.

1. Since even the earliest third order response, 3a, failed to appear in the atrium as an echo, it is apparent that it too must have engaged the communicating branch silently, returning to the atrial junction too early to evoke a response.

2. Although the data as presented establish the existence of one branch, they do not deny the possible existence of other junctions between the two systems. Had there been but one, in the position indicated, early reactivation of the system from the ventricular end should have elicited ventricular echoes, for the roundtrip conduction time from ventricle to branch and back should have considerably exceeded the ventricular refractory period. No such echoes were observed in this experiment, and it is therefore likely that a short circuit existed closer to the ventricle than the single branch indicated. Additional branches, more distal than the one shown, could not have been exposed by atrial stimulation under the conditions of figure 7, for activation of the proximal short branch should have spread to the main trunk and from there to any other cross connections, thereby occluding any possible longer echo pathways.

In other experiments evidence was obtained that multiple branches may indeed be present. Figure 8 reconstructs such an experiment in a manner similar to that of figure 7. It portrays a series of 3 records, in which 3 serial stimuli were applied to the atrium in each case. The second stimulus was applied at the earliest possible moment after R1, yielding in each case a propagated response without echoes, comparable with response 2a of figure 3. In part A, S3 was applied to the atrium at the earliest possible moment after R2, yielding response 3a which was propagated after a long delay to the ventricle and which re-entered the atrium as an echo (response 4 at the bottom of the accompanying schema). The shaded quadrangles at

FIG. 8. Exp. 9/28/54; dog, 15 Kg., both stellate ganglia excised, basic frequency 164. Tracings of original records at left, ventricle above, atrium below. A: earliest possible S3 (S = shock artefacts); B and C, later S3. FRP of fast pathway, trunk and branches, indicated by lined areas. Other conventions as in figure 7. Atrial response 4 is an echo resulting from 3a.
the left of the diagram indicate the estimated duration of the refractory period following the response R in the atrium, the faster limb of the nodal system, and the ventricle. Responses reaching the node later than R, but earlier than the broken line at the margin of the shaded area all arrived in the ventricle at the time labelled 2 at the top of the diagram; a response occurring immediately after the broken line was propagated more rapidly, arriving in the ventricle earlier than 2. The response 2 in the chart must therefore have entered the slower pathway.

It might have been anticipated, in the absence of communicating branches, that the subsequent response, 3a, would have found the faster path available for transmission. That it did not was apparent from the pattern of later responses initiated between 3a and 3c. Up to time 380 msec., all these tertiary responses appeared in the ventricle at 3a; i.e., the ventricular R-R interval remained constant at 180 msec. while the corresponding atrial intervals increased from 100 to 215 msec. As the atrial interval was increased stepwise from 215 to 250 msec., the ventricular interval increased by exactly equal increments (in other words, the conduction interval of R remained constant at about 175 msec., which may then be regarded as the normal conduction interval of the fully recovered slow pathway). Just beyond this point, the ventricular R-R interval diminished to the value indicated by 3c in the diagram.

The sudden diminution of the ventricular R-R interval between 3b and 3c can only be interpreted to mean that the faster pathway, unavailable for propagation for more than 330 msec., again became excitable just before 3c. It follows, as in the experiment shown in figure 7, that when the second response must have accomplished its activation "silently." It is again necessary to postulate the existence of branches of the faster tissue, originating near the atrial margin of the node and impinging upon the slower limb in midcourse. R, activating such a "fast" branch at its point of insertion in the slow limb and returning in retrograde fashion, must have found the atrium inexcitable.

With this information, it is possible to reconstruct the probable course of such retrograde activation. Excitation must have returned to the atrium before the expiration of its refractory period, but it could not have entered such a branch before the expiration of the refractory period of the faster tissue, defined by the shaded area. Accordingly, a line was drawn, at the slope ("velocity") characteristic of retrograde transmission in this heart, within the limits thus imposed. The retrograde activation of a short "fast" branch must, of course, have been followed by a period of refractoriness, indicated by the lower shaded parallelogram in the diagram, and must also have spread through other ramifications of the fast tissue. In this experiment, unlike that shown in figure 7, the ultimate activation of the ventricle by R was in fact have been accomplished over the faster route, for the electrical patterns of R and R were identical. (The difference between these and the responses 3a and 3b, though significant, was lost in the retracing procedures.) It therefore follows that the distal extremity of the slow path would have been activated in retrograde from the ventricle; this has been indicated in the diagram by the angulation of the broken line defining the passage of R which indicates how the waves of excitation, normal and retrograde, could have met and extinguished each other. As in the experiment described above, silent activation of the fast path accounts for the prolonged period of time during which only the slower system was available for transmission.

It is necessary to explain how an echo could result from the earliest R response, 3a, but not from those initiated later (including 3b). Had response 3a, moving slowly through the relatively refractory slow limb, encountered excitable tissue at the point of insertion of the short fast branch, it should have led to an atrial echo at approximately 460 msec. Since the echo occurred much later than this (R at time 530 msec.), it follows that the short branch must still have been refractory when encountered by 3a, and that another longer branch must have existed. By projecting a line back from R with the slope characteristic of retrograde transmission, an approximate estimate was obtained of the "length" of the
longer branch. This longer branch must also have been excited silently by $R_2$. Also it must have remained refractory, as indicated by the complex polygon, for approximately the same time as the shorter branch. Since activation of the longer branch by $R_2$ did not elicit an echo, it must be assumed that it stemmed from a common trunk, already blocked by prior excitation of the shorter branch. Yet response $3a$, which did echo, must have encountered the longer branch after its recovery. The reconstruction in the diagram indicates the narrow limits for a successful encounter; slight prolongation of the refractory period of the $R_2$ response in the branches would prevent an echo.

Furthermore, as the $R_3$ response was moved later, the shorter branch would again be found vulnerable; repeating the situation postulated for the early $R_2$, i.e., the retrograde process in the branch would again encounter a refractory atrium.

It has been indicated that the phenomena illustrated in figures 7 and 8 permit an estimate of the "normal" conduction time characteristic of the slower nodal pathway. When the two pathways recover from a basic excitation process, the faster pathway may become excitable within 20 to 50 msec. after the slower pathway. The A-V conduction interval for the slower system recorded during this time will, almost certainly, be longer than normal, for the tissue must be relatively refractory. Furthermore, if the early $R_2$ encounters a "fast" branch on its journey through the node, the recorded A-V interval may not represent the time characteristic of one tissue alone (note $R_2$ of figure 8). In these experiments, however, the two systems were thrown completely out of phase by the early $S_2$, providing in each case a period of about 150 msec. when only the slower path was available to $S_3$.

To illustrate more clearly how such dissociation permits independent assessment of the conduction intervals of the two systems, the conduction times of premature second and third-order responses were plotted against the corresponding atrial intervals, $R_1-R_2$, or $R_2-R_3$. In figure 9, derived from the same data as figure 8, the open circles illustrate the behavior of the $R_3$ responses. It will be seen that at atrial $R_1-R_2$ intervals greater than about 230 msec., the conduction interval was constant, and equal to that observed for the basic response, at about 120 msec. As shorter atrial intervals were imposed, the conduction intervals began to increase as $R_2$ encountered relatively refractory nodal tissue. At 190 msec. the faster limb must have become unavailable, for the conduction interval increased suddenly by 50 msec. and proceeded, as the atrial intervals were still further abbreviated, along a course which must have been characteristic of the slower pathway. With these data, it was impossible to guess at what time the curve characteristic of the slow system might have "flattened out" to give an estimate of its normal conduction time.

The filled circles illustrate the behavior of the $R_3$ responses produced when $R_2$ was introduced as early as possible. The points are plotted as $R_1-R_3$ conduction intervals against the corresponding $R_1-R_3$ intervals in the atrium. Note that very late $R_3$ responses encountered completely recovered nodal tissue, and again were propagated at essentially normal speed ($R_1-R_3$ interval = approximately 125 msec.). As $R_2$ was moved earlier, the curve broke suddenly to a new value about 50 msec. greater, and as $R_3$ was advanced still earlier the $R_1-R_2$ interval remained constant for a time at the new level of about 175 msec., then began to increase along a 45° line. The flat portion of the curve at 175 msec. indicates that the tissue it describes must have been fully recovered, and the normal conduction interval of the slow pathway was therefore about 50 msec. longer than that of the faster limb.

Figure 9 also illustrates the behavior of $R_3$ responses when $S_2$ was delivered, not at the earliest possible moment, but about 40 msec. later; i.e., at a time when both conduction pathways must have
been open to $R_2$. In this case, as represented by the black squares, the relationship between the conduction intervals, $R_1-R_2$, and the atrial intervals, $R_1-R_4$, showed no evidence for the participation of the slow pathway; the curve appears to project without a break toward the normal interval characteristic of the faster tissue. Still, it is apparent that "normal" conditions were not restored by the slight delay of $S_4$, for the curve should then have assumed the form of the curve for $R_2$ (open circles). It is probable that the refractory period of the faster tissue was more sharply reduced by serial excitation (i.e., shortening of the preceding cycle length) than that of the slower tissue, so that in this case the slower tissue recovered later than the fast, following the $R_1$ excitation process.

V-A Excitation. Evidence for the existence of intercommunication between two major pathways was also obtained in experiments in which the response of the transmission system to retrograde excitation was studied. A typical experiment is illustrated in figure 10. Here is represented schematically the behavior of four premature $R_2$ responses, $a$, $b$, $c$, and $d$, introduced at various intervals after the basic response, $R_1$. The earliest possible $R_2$, designated $a$, was transmitted to the atrium with a V-A interval of about 410 msec., and yielded an echo response in the ventricle. The response $b$, initiated about 15 msec. later in the ventricle, arrived in the atrium 40 msec. earlier than $a$, and produced an echo which also occurred earlier than $a$. The later response $c$ arrived in the atrium still earlier and also echoed earlier. All three responses must have entered the node at a time when only the slow system was excitable, but the later response $d$, propagated still faster and unaccompanied by an echo, must have encountered both systems in the excitable state.

Complete analysis of this experiment again leads to the conclusion that communicating branches must connect the two companion pathways. Since the time relationships between the responses $a$, $b$, and $c$ in the atrium and the corresponding echo responses in the ventricle remained constant, it is probable that the return path to the ventricle was common to all three responses. We have therefore shown these as rebounding from a common point, the point of origin of branches connecting the fast with the slow systems. The FRP of the faster system

![Figure 10](http://circres.ahajournals.org/issue/10/1/10/2/10-1.png)
intercepts, and projected toward the atrium to a common junction with the fast trunk as indicated in the sketch at the right of the figure.

Given the approximate length of the branches, and making reasonable assumptions for the slope of the lines representing conduction velocity, the course and direction of activation of the whole structure by R1 is indicated by the arrows. The longest branch must have been activated at its upper (ventricular) end by invasion from the slow trunk; activation of the lower end by invasion from the fast trunk must have occurred somewhat later, and the two excitation waves should have met and extinguished each other near the atrial end. The shorter branches, inserting into the slow tissue closer to the atrium, are shown to have been invaded from their atrial margin. The refractory periods of the branches were assumed to have had the same duration as the main fast trunk; because of the pattern of their activation, the duration of the refractory periods is described by the polygons represented by the shaded areas, the simple parallelogram describing the event in the intermediate branch, and the more complex figure representing the longest branch.

How the premature responses may have engaged the several pathways is now evident. The earliest response, a, found both long branches refractory and was forced to traverse the slow limb down to the short branch. The response b would have found the longest branch still refractory, but the intermediate branch, having recovered, would conduct the impulse over the last part of its course with greater speed. The response c, initiated late enough to encounter the long branch in the excitable state, must have entered this branch and accomplished the rest of its journey at the speed characteristic of the fast tissue.

Although response patterns similar to those depicted in figure 10 have been observed in many experiments, variations have also been recorded. The temporal dissociation of the two conduction pathways was found to be less than 20 msec in some experiments—only responses evoked during this brief period resulted in echoes, and all of these occurred at essentially the same time after the ventricular R2; in other experiments echo responses moved later as the R2 responses from which they were generated were delayed. In both cases, it is possible that only one pathway was functionally available to the early R1 responses.

It must be admitted that the reconstruction in figure 10, together with the reconstructions in figures 7 and 8, does not prove the branch hypothesis. It is nevertheless interesting that analyses of these separate experiments, two in which transmission from atrium to ventricle was assessed with 3 serial stimuli, the other in which retrograde transmission was studied and in which superficially different response patterns were recorded, all compelled the assumption of two pathways communicating by means of branches of different lengths. Similar evidence, which need not be detailed here, has been accumulated in many other experiments.

**DISCUSSION**

The Dual System Hypothesis. Recognizing that excessive prolongation of the A-V transmission time of very early premature beats is inconsistent with a simple homogeneous conducting bridge, Krayer, Mandoki, and Mendez postulated a synaptic mechanism within the nodal system. Similar observations led us to propose the existence of parallel pathways with different properties; other characteristics of A-V transmission, as described in the present study, force the adoption of the latter hypothesis. The reasons for the choice must be considered in greater detail.

The time relations of early premature responses, exemplified by figure 1C, are compatible with a synaptic mechanism. Indeed, the gradual and progressive increase of the ventricular R1-R2 interval with early re-excitation of the atria can be admirably explained by the postulate of Forbes, Ray, and Griffith if one assumes that the excitatory state of the presynaptic "stimulus" outlasts the refractory period of the postsynaptic elements, and decays more slowly than the threshold of the latter structures. The behavior characteristic exhibited in figure 1B, however, can be more adequately explained by the assumption of two parallel systems, the slower of which appears only when the faster is functionally inoperative.
The echo phenomenon, which cannot be conveniently explained by the synapse concept, is adequately accounted for by the dual system hypothesis. Before echoes can be accepted as unassailable evidence, however, other possible sources of such responses must be eliminated.

Since the S-A node was destroyed by crushing, any pacemaker activity in surviving fragments would have been much too slow to appear so early after the secondary evoked response. Furthermore, re-entrant responses of the atrium labeled as echoes always had the electrical configuration characteristic of responses propagated from the ventricle, or from the A-V node itself. Since the echo responses were engendered by early re-excitation of the atrium, it might be argued that they were in truth the result of a circus movement or flutter cycle about the obstacle created by destruction of the S-A node. Such re-entrant flutter beats were sometimes observed, but were always recognizable by their very early occurrence and by their electrical configuration. Further, echoes were often elicited by stimuli applied to the atrium well after its complete recovery from the preceding response, at a time when the conditions for one-way propagation about an obstacle no longer existed (see figure 6A).

It could also be suggested that the echo responses were in reality only an expression of spontaneous pacemaker activity of the A-V node itself. Spontaneous activity of the A-V node was occasionally observed when a recurrent driving stimulus was eliminated to provide a long vacant period after the premature stimulus. The spontaneous nodal activity could always be easily identified by its late appearance and by its irregularity. In successive sweeps of the monitor oscilloscope (triggered to fire with every sixth driving stimulus) spontaneous nodal beats appeared with a random variation of perhaps 20 to 50 msec.; responses identified as echoes occurred early in fixed relation to the preceding activity, or oscillated between two or three fixed values assumed to be indicative of different pathways. It has recently been proposed (Rosenbluth) that rapid re-excitation of the node may enhance its rhythmicity; we submit that the rhythmic responses observed may have been re-entrant echoes.

All of the above considerations apply also to echoes elicited by excitation of the ventricles. In the retrograde direction, S-A nodal or circus movement activity could not have been operative. Early re-excitation of the ventricle can, of course, induce repetitive activity (Moe, Harris, and Wiggers), but the multiple responses resulting from stimulation during the vulnerable period occur at the earliest possible moment (i.e., at the end of the ventricular FRP) and display an electric pattern characteristic of ventricular ectopic beats. Echo responses in the ventricle were of supraventricular origin (see figure 6B), and usually occurred much later than would have been true for idioventricular responses.

Finally, it should be repeated that echoes appeared when other evidence (configuration changes, delay of early responses) indicated the dissociation of two conduction systems. Circumstantial as this evidence may be, it cannot be denied that the data fit the hypothesis.

The most convincing piece of evidence supporting the dual system hypothesis is the common occurrence of gross electric configuration changes in the ventricle. It was clearly established that the "abnormal" configuration of "slow" responses could not have been due to partial refractoriness of the ventricle itself; earlier responses of the ventricle assumed the normal pattern. Furthermore, the configurational changes were unequivocally related to the other criteria of "slow" versus "fast" excitation (time relations; echoes). We can only conclude that very early premature responses must have taken a different course than later responses.

Nothing can be said about the anatomical position and nature of the A-V communications except that they must remain separate well out in the ventricle. This brings up the question whether the dual system is a part of the A-V node at all, or is rather a feature of the intraventricular conduction system. In terms of the events which determine the response of the ventricles to supraventricular excitation, it makes little difference whether the dichotomy occurs in the A-V node or in the bundle of His.
It is quite possible that the atrium makes contact with two distinct intraventricular conduction systems without any intervening "nodal" structure. In this case, for our purposes, the bundle becomes the A-V transmission system. It is even possible, and no purely histologic evidence can gainsay it, that the A-V node is not a device for conduction, but merely a subordinate pacemaker feeding into the conduction system, ready to take over the domination of cardiac rhythm when called upon, but serving no other useful function.

Having established that the ventricular configuration changes are due to activation over separate and discrete channels, we must explain why such behavior was not evident in all experiments.

First, it is probable that the separate conduction pathways insert at different points into a final common pathway (the Purkinje system). If the points of insertion were relatively close together, only a small area of myocardium would be activated aberrantly by early responses; muscle remote from the points of insertion would be activated normally whether A-V transmission was accomplished over the slow or the fast route. In practice, it was found that polarity changes were more easily demonstrated with electrodes applied near the septal margin of the right ventricle than with electrodes attached to areas which are normally activated later. However, the mass of tissue involved is not necessarily small, for reversal of polarity was also demonstrated in a standard lead II electrocardiogram.

Second, if it be accepted that branches communicate between the two systems, it follows that an impulse which begins its journey in the slow pathway may, by passing over an excitable branch, invade the faster pathway and discharge the ventricle over the normal route (figure 8). Polarity changes, then, could only occur by permission of a chance relationship between morphology and function in the two pathways.

Time Course of Excitability Recovery in the Nodal System. It has been mentioned above that the earliest possible secondary atrial response was usually transmitted to the ventricle. We do not know to what extent the prolonged conduction interval of such early responses resulted from true latency at the junction between atrium and node, or to reduced conduction velocity within the transmission system; it is, therefore, impossible to determine whether the true functional refractory period of the node may not have been briefer than that of the atrium itself. It follows that in those few experiments in which dissociation of the two pathways could not be demonstrated, earlier activation, had it been possible, might have exposed the slower system. That is, a response pattern like that shown in figure 1A might have been converted to that of 1B or 1C.

It is also possible, however, that while the slower system usually recovered excitability earlier than the companion pathway, it did not invariably do so. The time zone during which a secondary impulse could enter only the slower system varied, in various experiments, from 0 to approximately 60 msec., and could often be prolonged or diminished by nerve stimulation or section, change of the basic frequency, or other procedures.

For simplicity in illustration of nodal transmission, it has been assumed that the two conduction systems have the same length, that the time course of excitability recovery during the relatively refractory period of the nodal systems describes a smooth curve, and that, as a corollary, the velocity increases with time along a similarly smooth curve.

The first of these assumptions is purely arbitrary and is quite unimportant to the development of the main thesis of the study. It is, however, possible that impulses traversing the "slow" pathway are delayed not because they travel at lesser speed, but because they take a longer course. It is probable that not all observations can be easily fitted into schemata like those of figures 6, 7, 8, and 10, for there is no convenient way of indicating diagrammatically a time lapse resulting from passage through a sinuous path. The frequency with which reasonable "fits" have been obtained suggests that the alternate pathways are of approximately equal lengths.

The second of these assumptions, the smooth
recovery curve, appears to be at variance with what is known of the time course of recovery in other cardiac tissues. Orias and associates have described "dips" in the curve relating threshold to time in the early recovery phase of atria and ventricles. On superficial consideration, it might be supposed that if the A-V conduction pathways exhibit similar irregularities in excitability, then early responses should be propagated at a correspondingly irregular velocity, accelerating as they encounter less refractory tissue in a "dip" and decelerating as they engage more refractory tissue in a hump of the curve. More careful examination, however, suggests that even if the excitability of the nodal tissue recovers over an irregular time course, the velocity of early responses need not exhibit corresponding fluctuations. The excitability curves are obtained with artificial stimuli, and threshold is related to the time of application of the stimulus, not the time of appearance of the resulting response. In atrium or ventricle, the observed irregularities of excitability precede the end of the functional refractory period. Since an impulse cannot arise before the end of the FRP, its conduction should depend on the state of excitability (and the magnitude of the action potential) following, not preceding, this boundary. It is worth emphasizing that the relatively refractory period as determined by the time of application of a stimulus is not identical with the physiologically more significant period which follows the FRP.

While "dips" in the excitability curve of the transmission system should not influence the behavior of responses once initiated, they may nevertheless influence the transfer of activity from atrium to node. An early atrial impulse, encountering the node in a state of relative "supernormality" (i.e., in a "dip") could activate it and be propagated through to the ventricle (after being delayed until the end of the FRP), whereas a later response meeting the node in a less excitable, or subnormal, state might fail to enter. We have obtained suggestive evidence that such phenomena may occur, but detailed analysis would be premature at the present time.

**Relationship to Cardiac Arrhythmias**

**Reciprocal Rhythm.** This study provides a logical explanation for reciprocal rhythm occasionally observed clinically. When an A-V nodal or idioventricular pacemaker governs cardiac rhythm and causes retrograde excitation of the atria, there may occur, occasionally or regularly, a re-excitation of the ventricle, which appears to bear a fixed relation to the retrograde atrial response. The phenomenon, well described and illustrated by Katz, is explained as a re-entry of the ventricle by an impulse which made a roundtrip passage through the node. The explanation implies, of course, that one pathway was traversed by the retrograde impulse, and another by the returning excitation process.

The obvious similarity with events described in the present study extends further. Reciprocal rhythm occurs when the basic frequency slightly overtaxes the retrograde transmission capacity of the node; i.e., when there exists a reverse Wenckebach phenomenon. Clearly, if serial retrograde impulses encroach progressively more upon the relatively refractory period of the node, the time must come when a response can engage only one limb of a dual conducting system, permitting the other to recover in time for the return passage. The echo phenomenon can be induced if the ventricles are driven at a rate which leads to a retrograde block (6:5, 3:2); they may be exposed by cancelling the first recurrent driving stimulus following the "dropped" beat. The same procedure works in the normal direction. We are not aware, however, of clinical descriptions of reciprocal atrial beats associated with partial block in the normal direction, perhaps because the standard electrocardiograph leads do not display atrial excitation with sufficient clarity when P waves are superimposed upon ventricular complexes.

**Wolff-Parkinson-White syndrome.** The demonstration that two pathways communicate between atria and ventricles leads naturally to the supposition that one of these paths may represent the accessory bundle of Kent, and that the aberrant conduction in cases exhibiting the WPW phenomenon may be due to activa-
EVIDENCE FOR A DUAL A-V TRANSMISSION SYSTEM

tion of the "abnormal" bridge. However, it seems probable that the accessory pathway exposed by early re-excitation is always present, at least in the dog, and is not a developmental anomaly. Furthermore, it has been shown that activation of the accessory path yields an unusually long A-V transmission interval, not an "accelerated" interval. We do not believe that the present study provides an explanation for the WPW syndrome.

Supraventricular Paroxysmal Tachycardia. This arrhythmia is usually ascribed to activity of an ectopic atrial or A-V nodal focus (it has, in fact, become popular to consider all tachycardias as expressions of such activity*). However, it was proposed by Barker, Wilson, and Johnston10 that paroxysmal tachycardias might well be examples of circus movements involving nodal tissue. The "silent" or isoelectric interval of the electrocardiogram was explained by the small mass of tissue involved during the slow intranodal transit; the moderate frequency was referred to the presumed long refractory period and slow conduction velocity of nodal tissue; and the regularity of cycle length was presumed to be due to the stability of the circuit.

In many of the cases reported by Barker and associates, the P-waves were normal in contour and polarity and were presumed to be examples of a circuit traversing the S-A node. Another large group, exhibiting inverted P-waves, were considered as examples of an A-V nodal circuit. Whether or not these interpretations are correct, the present study demonstrates the possibility of a self-sustained circus movement traversing the A-V conduction system.

Should a self-sustained rhythm be established experimentally, it can be expected to resemble closely the clinical paroxysm. A single early premature beat, arising in either atrium or ventricle, should be sufficient to start the paroxysm. (Also, a single premature beat, properly timed, should occlude the re-entrant path and terminate the paroxysm.) Vagal stimulation intense enough to cause gross prolongation of the nodal refractory period, or to block transmission in either the normal or the retrograde direction, should terminate the paroxysm. Assuming that the FRP of the nodal system always exceeds that of atria or ventricles, there should be a one-to-one relationship between atrial and ventricular frequency. Finally, it can be predicted that the configuration of the ventricular electrogram in an experimental paroxysm might well be "abnormal," should the impulses reach the ventricle over the slow path and return to the atrium over the fast. Such electrocardiographic abnormalities, when observed in clinical paroxysms, are attributed to "aberrant intraventricular conduction." We may suggest with confidence that the conduction "disturbance" is a normal property of the A-V transmission system, exposed by the tachycardia.

Even with respect to its rarity, the experimental arrhythmia would resemble the clinical entity. If branches communicating between two conduction pathways exist in the human heart, as they surely must in the dog, only the rare individual, human or canine, could be expected to possess by chance an anatomical pattern which would support a sustained circus rhythm without short circuit.

SUMMARY

The propagation of early premature impulses from atrium to ventricle and from ventricle to atrium was studied in the exposed heart of the dog. The temporal relations of atrial and ventricular responses fell into three patterns: (1) as the interval between basic (Ri) and induced premature beats (Rs) was progressively shortened in the atrium, the corresponding ventricular Ri-Rs intervals reached a constant minimum value; (2) as the atrial Ri-Rs interval was diminished, the ventricular Ri-Rs interval reached a minimum value, then abruptly increased to a longer interval which remained constant up to the earliest propagated response; and (3) ventricular intervals lengthened progressively or stepwise as the parent atrial intervals were diminished.

Early premature responses initiated either in the atrium or the ventricle often resulted in reciprocal responses or "echoes" in the chamber of origin.

Responses of the ventricle to early premature atrial excitation were often characterized by gross alterations of electrical configuration.
These observations lead to the conclusion that the A-V transmission system is composed of two pathways, one having a shorter refractory period but a longer conduction interval than the other. Detailed study of the time relations of early responses and the resulting echoes leads to the further conclusion that the two conduction systems must communicate with each other over one or more branches. The dual pathway hypothesis is proposed as an explanation for reciprocal rhythm and paroxysmal nodal tachycardia.

**REFERENCES**


**SUMMARIO IN INTERLINGUA**

Le propagation de precoce prematur impulsos ab atrio a ventriculo e ab ventriculo a atrio esseva studiate in le exopinet corde canin. Le relationes temporal del responsas atrial e ventricular esseva de tres distincte typos: 1. Quando le intervallo inter le pulso basic (R1) e le prematur pulso inducite (R2) esseva progressivemente reducite in le atrio, le correspondente intervallo R1-R2 del ventriculo attingeva un minimo constante. 2. Quando le intervallo R1-R2 del atrio esseva reducite, le intervallo R1-R2 del ventriculo attingeva un valor minimal e posteae se augmentava abruptemente a un intervallo prolongate que remaneva constante usque al prime responsa propagate. 3. Le intervallo ventricular se augmentava progressivemente o passo a passo quando le corrispondente intervallos atrial esseva reducite.

Precoce prematur pulsos de origine atrial o ventricular resultava frequentemente in responsas reciproc o "echos" in le camera originari.

Responsas del ventriculo a precoce prematur excitations atrial esseva frequentemente characterisate per grossier alterationes del configuration electric.

Iste observationes supportava le conclusion que le sistema del transmission atrio-ventricular consiste de duo vias e que le un de iste vias se distinge del altere per un plus breve periodo refractori sec un plus longe intervallo de conduction. Un studio detaliate del relationes temporal de precoce responsas e del echos resultante supporta le conclusion addicional que le duo systemas de conduction debe comunicar le un con le altere a transverso un o plure branças.

Nos propose le hypothese del existentia de un sistema de transmission dual como explication de rhytimo reciproc e nodal tachycardia paroxysmal.
Physiologic Evidence for a Dual A-V Transmission System
GORDON K. MOE, JAMES B. PRESTON and HAROLD BURLINGTON

Circ Res. 1956;4:357-375
doi: 10.1161/01.RES.4.4.357

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