Concealed Conduction
Further Evaluation of a Fundamental Aspect of Propagation of the Cardiac Impulse

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Recognition of concealed conduction of the cardiac impulse is shown to be indispensable for the understanding of many cardiac arrhythmias. After a historical review of the concept of concealed conduction, records are presented to demonstrate established, as well as hitherto undescribed, aspects of concealed A-V conduction. Then, the manifestations of concealed intraventricular conduction are pointed out and, finally, the relationship of the phase of concealed conduction to the supernormal phase is discussed.

The earliest phase of the relative refractory period of the specific conduction system is characterized by a partial response to the cardiac impulse, inasmuch as the impulse penetrates into the conducting tissues but fails to traverse them completely. This concept, based upon experimental evidence, was clearly enunciated by Engelmann and Erlanger. However, its precise mechanism was investigated only much later by Ashman and Hermann, Drury and Lewis, and Lewis and Master. Scherf and Shookoff, studying the effect of premature beats upon A-V nodal rhythm, postulated a penetration of nonconducted impulses into the A-V junction. Kaufmann and Rothberger and Wenckbach and Winterberg were aware of the significance of such abortive penetration of the cardiac impulse and considered it in analyzing arrhythmias of the human heart. Winternitz and, independently, Luten and Jensen were the first to point out the disturbance of impulse formation in the A-V node as a result of partial conduction of sinus impulses in cases of incomplete A-V dissociation engendered by acceleration of a subsidiary pacemaker. Winternitz and Langendorf demonstrated that penetration of "blocked" sinus impulses has to be postulated to explain the variations of retrograde conduction in cases of A-V block with V-A response. In a previous report from this department\(^\text{16}\) the term concealed conduction was introduced and the various manifestations of the phenomenon pertaining to A-V conduction were described.

Concealed conduction of the cardiac impulse is manifested by its after-effects, involving either the conduction or formation of a subsequent impulse. In the former case, impulse conduction is unexpectedly delayed or stopped; this is due to the creation of refractoriness in conduction pathways by penetration of the preceding "blocked" impulse. In the latter case, discharge of an expected subsidiary impulse is delayed or fails to occur; this is due to premature extraneous discharge of a subsidiary pacemaker located in the path of the penetrating "blocked" impulse.

Since we have focused our attention on these two manifestations of concealed conduction in the human heart, the concept proved extremely valuable in providing the key to the understanding of various otherwise unexplainable features of simple and complex arrhythmias. It has been utilized by us and others in the interpretation of conduction disturbances engendered by premature systoles\(^\text{16}-\text{18}\); of apparent irregularities of the nodal pacemaker in A-V dissociation with or without A-V block\(^\text{19-23}\); to unravel satisfactorily irregularities of auricular and ventricular beating in complex arrhythmias, such as A-V nodal tachycardia with unequal degrees...
Fig. 1. Concealed A-V conduction in a case of A-V block causing blockage of two consecutive auricular premature systoles. The conventions in the diagram are: A-V represents the spread of the impulse through the A-V junction between the auricles (A) and the ventricles (V); oblique lines at different angles indicate varying speed of impulse conduction; the short lines at right angles to the oblique lines indicate stoppage of the impulses; varying length of the oblique lines representing such stopped impulses indicates the varying distance to which the impulses penetrate into the A-V junction.

Two types of P waves are seen: one, tall and peaked (the first, fifth and last), represents sinus impulses conducted to the ventricles at a prolonged P-R interval of 0.28 second, indicating depression of A-V conductivity. The other, smaller and notched (the second to fourth, and the sixth to eighth), represents runs of three successive auricular premature systoles at a rate of 167. Transmission of these ectopic impulses to the ventricles is irregular. In the first group, the first and last is conducted at a P-R of 0.30 second and the middle one is blocked. In the other group, the first two are blocked and the last is conducted at a P-R of 0.30 second.

The cause for this apparently different response of the A-V junction to the rapid stimulation by the two identical triads of ectopic impulses is indicated in the diagram. In both groups the first of the ectopic impulses penetrated into the A-V junction. In the first group it traversed it and reached the ventricles, in the other it failed to do so. Evidently, its deep penetration created refractoriness in the traversed part of the junction of sufficient degree so as to prevent the transmission of the subsequent ectopic impulse in the same way as in the first group. Its partial conduction is concealed and revealed only by the otherwise unexplained failure of the next ectopic impulse to pass. Thus, both triads of ectopic beats actually yield a similar 2:1 response of the A-V junctional tissue: in the first triad this response was complete, in the other incomplete, simulating a 3:1 response.

The cause of success or failure of the ectopic impulses in traversing the A-V junction is revealed by close examination of their R-P intervals. The first premature P wave of the second triad occurred 0.2 second earlier than that of the first triad. Evidently, here the impulse reached the A-V junction at the transition of the absolute into the relative refractory phase, during the phase of concealed conduction.

of forward and retrograde block, or in an unusual case of ventricular parasystole complicating A-V dissociation. The concept proved indispensable for the understanding of the ventricular response in certain cases of auricular flutter and probably also in auricular fibrillation for the interpretation of some cases of reciprocal beating and for the explanation of the failure of A-V nodal escapes to occur at the expected time in cases of auricular fibrillation. Failure to recognize the operation of concealed A-V conduction in cases of second degree A-V block has led, in our opinion, to erroneous interpretations and unwarranted deductions.

The purpose of the present report is: (1) to demonstrate the usefulness of the concept of concealed conduction in explaining certain aspects of impaired A-V conduction in sinus as well as ectopic rhythms, in particular multiple successive blockage of auricular impulses and the failure of A-V nodal escapes to appear during long ventricular pauses; (2) to point out the different mechanism of blockage of successive auricular impulses in auricular flutter or tachycardia associated with group beating of the ventricles; (3) to discuss possible applications of the concept of concealed conduction to the analysis of disturbances of intraventricular conduction, to the interpretation of some peculiarities of the pre-excitation syndrome, and to the understanding of the timing of ventricular premature systoles; and (4) to examine the relationship between the supernormal phase and concealed conduction in the A-V junction.

Material

The material of this report comprises nine records obtained on six patients and illustrates: (1) runs of auricular premature systoles revealing the operation of concealed conduction responsible for blockage of two consecutive impulses and elucidating the mechanism of 3:1 A-V response (fig. 1); (2) incomplete A-V dissociation (without A-V block), in which the occurrence of "abortive" ventricular captures, permits determination in the human heart of the part of the cycle occupied by the phase of concealed conduction (fig. 2); (3) second degree A-V block (involving two levels of the A-V junction) with seemingly irregular discharge of the escaping A-V nodal pacemaker, clarified by the demonstration of concealed conduction (fig. 3); (4) second
degree A-V block with unusually long ventricular pauses, with or without nodal escape, likewise clarified by implication of repetitive concealed conduction, which causes multiple blockage of consecutive auricular impulses as well as multiple consecutive discharge of the subsidiary (A-V nodal) pacemaker (figs. 4, 5 and 6); (5) successive blockage of auricular impulses in auricular flutter with irregular A-V conduction and group beating of the ventricles as a result of varying penetration of the A-V junction by flutter impulses due to two areas of conduction impedance (fig. 7); and finally, (6) auricular tachycardia with group beating (bigeminy) of the ventricles as a result of either of the two mechanisms—two regions of impedance or concealed conduction in the A-V junction (fig. 8A and B).*

The analysis of all electrocardiograms is presented in diagrammatic form and is fully discussed in the respective legends. The conclusions drawn from the interpretation of these records form the basis for the following comment.

**COMMENT**

The portion of the cardiac cycle at the transition of the absolute into the relative refractory phase, during which partial (concealed) penetration of the impulse into the conduction pathways takes place, was termed by Lewis and Master the "phase of interference." We propose to name it the phase of concealed conduction in order to avoid confusion with the term interference used in a different and wider sense. Such a phase of concealed conduction can be demonstrated with normal as well as depressed conductivity (figs. 2 and 3). However, it is not always possible to delineate sharply the time it occupies within the cycle, as was possible in figure 2. In constructing recovery curves of conductivity in such cases, the effect of cycle length upon refractory period must be taken into consideration in addition to the simple determination of R-P and P-R relationships, customarily used for this purpose; and this is true, not only for completed but also for partial (concealed) conduction. Nevertheless, it would appear that the phase of concealed conduction may lengthen, if conductivity is depressed, just as is the case with the absolute and relative refractory phases.

Concealed A-V conduction should not be viewed as an exceptional phenomenon that is helpful in unraveling rare and complex arrhythmias only. Actually, one of its manifestations, namely, the effect on conduction of a subsequent impulse has to be invoked to account for the consequences of the most trivial disturbance of cardiac rhythm, that is, ventricular premature systoles. Here, concealed retrograde spread of the ectopic impulse into the A-V junction is the cause for the occurrence in sinus rhythm of a compensatory pause, or of a prolonged A-V conduction time subsequent to interpolation of the ectopic beats, and in auricular fibrillation for the pauses, following ventricular premature systoles. A less common, but more obvious manifestation of concealed conduction in ectopic impulse formation is the occurrence of unexpected disturbances of A-V conduction during runs of auricular premature systoles, exemplified in figure 1. Under all these circumstances, each concealed conduction of an impulse sets up physiologic refractoriness of A-V junctional tissue, delaying or preventing transmission of a subsequent impulse. Such mechanisms operating in conjunction with depressed A-V conductivity, for example in second degree A-V block, are illustrated in figures 4, 5 and 6 and in figure 8. As a rule the presence of concealed A-V conduction in such records can be considered proven, when in some portions of the same record completed (but delayed) conduction of auricular impulses is seen under comparable conditions, that is, with identical or almost identical R-P intervals.

The other manifestation of concealed conduction is a disturbance it causes in the regularity of impulse formation in a subsidiary pacemaker. This is usually seen in cases with A-V dissociation with first degree A-V block but may occur as a physiologic phenomenon in the absence of impairment of A-V conductivity. An instance of the latter variety is illustrated in figure 2. Detailed analysis of this case, as indicated in the legend and applied to the entire material available, permitted determination of the duration of the phase of concealed conduction, that is, that

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* We are indebted to Dr. S. K. Wilhelm for his kind permission to use the material of figure 8.
Fig. 2. Incomplete A-V dissociation (without A-V block) with successful and attempted ventricular captures. The duration of the phase of concealed conduction in the absence of A-V block. The two strips shown are a continuous portion of a long lead II, the last beat of the upper strip is reproduced as the first of the lower strip. The conventions in the diagram are the same as in figure 1. Impulses arising in the A-V node, spreading towards the ventricles, and in retrograde fashion towards the auricles, are indicated by oblique lines originating in the center of the A-V node. In some of them the distance between the nodal QRS and subsequent P wave (R-P interval) is indicated by numbers representing hundredths of a second.

Upright P waves with uneven R-S spacing, varying between 1.12 and 0.86 seconds, occur throughout the record, indicating a sinus arrhythmia. Some of the P waves coincide with QRS complexes. When isolated, they reveal slight variations in size, ascribed in association with the sinus arrhythmia, to a wandering of the pacemaker within the sinus node. When the sinus rate speeds up to over 68 the sinus impulse is conducted to the ventricles with a normal P-R of 0.18 second (cf. first two beats). When the sinus rate slows below 68 the A-V node escapes. Its retrograde impulses are prevented from activating the auricles by the sinus impulses, occurring simultaneously with, or shortly after, the nodal impulse. There is thus intermittent A-V dissociation caused by simple A-V interference.

Perpetuation of the A-V dissociation is prevented by penetration of some sinus impulses into the A-V junction up to or beyond the area of the nodal pacemaker. The extent of this penetration depends on the timing of the sinus impulse within the ventricular cycle. When a P wave occurs shortly after a nodal QRS, 0.09 second or less, the impulse is stopped high up in the A-V junction and A-V dissociation continues (sixth and last P of the lower strip). When the P wave occurs 0.24 second (0.20 second in one instance, not shown) or more after a nodal QRS, the impulse passes the A-V junction with some delay and activates the ventricles, causing an early ventricular beat with some aberrant contour, aventricular capture (fifth beat of the upper strip). When a P wave follows a nodal QRS at intervals of 0.13 to 0.18 second, no ventricular response ensues but a nodal escape fails to occur at the expected time (eighth P of the upper and seventh P of the lower strip). Instead, following a pause that exceeds the interval (0.88 second) between the two successive nodal beats, a sinus impulse is conducted through to the ventricles. Obviously, the stopped sinus impulse ahead of it must have penetrated into the A-V junction but failed to traverse it completely; yet on its way towards the ventricles it passed the nodal pacemaker and discharged it prematurely, thus shifting its “timetable”. The conduction of these sinus impulses is concealed and becomes manifest by its effect on impulse formation in a subsidiary pacemaker. Sinus impulses occurring 0.12 second after a nodal QRS at one time (fourth P wave in the upper strip) are stopped without concealed conduction, and at another time (third P wave of the lower strip) are stopped after the occurrence of concealed conduction. In the first case A-V dissociation continues, in the other it is interrupted.

This interpretation, fortified by analysis of the entire material, permits conclusions in this instance of incomplete A-V dissociation, with an average sinus rate of 62 and A-V nodal rate of 68 as follows:

(1) The period of complete unresponsiveness of the A-V junctional tissue to auricular impulses (the absolute refractory phase) occupies at least the first 0.09 second of the ventricular cycle, a normal value. (2) A subsequent “phase of concealed conduction”, during which the impulse penetrates partway into the junction (far enough to reach a subsidiary pacemaker), occupies a period of 0.08 second (R-P of 0.12 to 0.20 second). (3) Recovery of A-V junctional tissue so that the impulse can pass completely is attained after the first 0.20 second, again a normal value. (4) The transition of absolute refractoriness to the phase of concealed conduction (at R-P of about 0.12 second) is not sharply delineated. In this short transitional period the response of the A-V junction cannot be anticipated and A-V conductivity may be considered to be in a critical state. (5) A-V conductivity is not depressed since the P-R interval of the conducted sinus impulses is normal (0.18 second). The prolonged P-R interval (0.36 second) of the earliest ventricular capture can be ascribed to a physiologic delay of A-V conduction during the relative refractory phase. (6) The disturbance of rhythm is caused by acceleration of the subsidiary pacemaker in the presence of a sinus arrhythmia. The A-V dissociation can be attributed entirely to normal A-V interference and no abnormal depression (block) of the junction has to be postulated.
During the block. Its otherwise partial averted A-V longest layed, anticipated region for the first A-V nodal junction complex, as the ventricular stretch areas of the first A-V conduction time (P-R) is responsible for the ventricular pauses. During these pauses the A-V node (R') escapes, its impulses coinciding with a sinus P wave. Thus, in some instances two P waves in succession fail to activate the ventricles, the first (Pc) due to A-V block, the other (coinciding with R') due to A-V interference with the escaping nodal impulse.

When the R-R' intervals of the two nodal escapes in the upper strip are compared, it is found that the second is longer (1.28 seconds) than the first (1.20 seconds). This difference neither precludes the correctness of the interpretation nor does it mean an irregular nodal pacemaker. It can be accounted for if the duration of the A-V conduction time (Pc-R) of the last conducted beat ahead of the escape is taken into consideration. The first Pc-R is 0.08 second longer than the other, exactly by the difference of the R-R' distance of the two escapes; or, in other words, the distance Pc-R' is equal (1.56 seconds) in both instances. The explanation is as follows: The delay in A-V conduction time causing prolongation of the first Pc-R takes place, in part, in the A-V junction below the area of origin of the A-V nodal impulse. This delay is more marked at the end of a sequence of conducted beats (as with the first Pc-R) than immediately after a pause (as with the second Pc-R). The longer the delay in this stretch of the A-V junction, the closer the R of the conducted beat comes to the R' of the escape; but the duration of Pc-R' will remain the same. The longer R-R' (1.28 seconds) therefore approaches the actual duration of the nodal interval more closely.

A more complex condition is present in the lower strip. Here, too, two ventricular pauses are caused by intermittent blockage of a P wave; however, nodal escape takes place only during the second pause (containing Pc) with R-R' measuring 1.24 second and Pc-R' 1.58 second (as in the upper strip). During the longer pause that follows the third beat (and contains Pc) no escape occurs at the anticipated Pc-R' interval. This failure of the nodal escape to take place when expected is ascribed to partial penetration of the apparently nonconducted sinus impulse (Pc) into the A-V junction discharging the A-V nodal pacemaker ahead of the time of its spontaneous release of an impulse. This partial conduction of the "blocked" sinus impulse is concealed, and its presence is revealed only by its effect upon impulse formation in the subsidiary pacemaker. Thus, while the nodal discharge is delayed, the next sinus impulse arrives and finds the A-V junction no longer refractory. Hence, the longest ventricular cycle of the entire record is terminated by a conducted beat and not by an escape.

The interpretation is fortified by comparing, in the entire material, the different response of the A-V junction with the timing of "blocked" impulses (Pc and Pc). Thus, R-Pc measures 0.32 second as compared with R-Pc of 0.30 second; expressed in terms of refractoriness and recovery, with an R-P of 0.30 second the impulse falls into the absolute refractory phase of the A-V junctional tissue and is completely stopped, while 0.02 second later it occurs within the phase of concealed conduction, linking the absolute and relative refractory phases, and penetrates partway into the junction.

In summary, in this complex example of second degree A-V block it can be demonstrated that: (1) Two areas of block are present in the A-V junction, one above, the other below the location of the subsidiary nodal pacemaker; (2) the impulse may be stopped in the upper or lower region of block. Its penetration into the lower region is concealed and revealed by a shift in the timing of the otherwise regular nodal pacemaker; (3) success or failure of an impulse in penetrating to the lower region of block is a function of its timing in the ventricular cycle and depends on the momentary phase of recovery of the A-V junction after the passage of a preceding impulse; and (4) in a record with unquestionable A-V nodal escapes, the beats terminating the longest R-R intervals may be conducted beats and not escapes.
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Fig. 4. Concealed A-V conduction in second degree A-V block causing blockage of consecutive sinus impulses and apparent prolongation of nodal escape intervals. The conventions in the diagram are the same as in figure 2; the additional horizontal line dividing the area A-V (representing the A-V junction) indicates the level of the site of the nodal pacemaker. The numbers between horizontal arrows indicate, in hundredths of a second, the distances between P waves and QRS complexes, or between successive QRS complexes, corresponding to the arrow heads at each side. The symbols P', P\(_v\), R and R' are the same as used in figure 3. The four strips are a continuous portion of a long lead II; the last QRS of the first and third strip is reproduced as the first QRS of the second and second QRS of the fourth strip, respectively; the last P wave of the second strip as the first P wave of the third strip.

The presence of sinus rhythm is indicated by regular upright P waves at a rate of 65; the presence of a second degree A-V block by failure of single, or two successive P waves to yield a ventricular response, and by progressive prolongation of P-R when A-V conduction occurs several times in succession (cf. top strip). The ventricular pauses engendered by the blockage of auricular impulses are terminated by nodal escapes interfering with sinus impulses, except in the fourth and last beat of the third strip. These latter two beats are considered to be sinus beats conducted with the shortest P-R intervals (see below).

The distance of the nodal escapes to the last preceding conducted ventricular beat (R-R') varies; it falls into two categories: (1) shorter ones (1.46 to 1.76 seconds), when the escape occurs after a single blocked P wave and (2) much longer ones (2.50 to 2.70 seconds), when the escape follows two blocked P waves. In both categories the intervals from the escape to the penultimate preceding P wave (P_v-R' or P\(_v\)-R') are fairly constant as indicated by the horizontal arrows (ranging from 1.86 to 1.96 second). In one instance (the first two beats of the third strip), two escapes occur in succession, revealing the approximate duration (1.88 second) of the escape interval (R'-R'). This interval would be precisely equal to the actual duration of the nodal cycle were the conduction times of the successive nodal impulses to the ventricles equal. Were the conduction time of the first longer than that of the second, then the actual nodal cycle would be a little longer than the manifest R'-R' interval. Were the conduction time of the second longer than that of the first, then the actual nodal cycle would be a little shorter than R'-R'. The latter seems to be the case here.

Similarly, the differences in the shorter escape intervals (gaged by their P_v-R' distance) which do not exceed 0.12 second, can be explained by variations of the conduction times below as well as above the nodal pacemaker; in addition, a slight instability of the discharge rate of the nodal pacemaker might be present. However, the several long ventricular pauses during which nodal escape fails to occur at the anticipated time cannot be explained on this basis. They are attributed to the phenomenon of concealed A-V conduction. The clue to this interpretation is provided by three facts: (1) the association of these long intervals with two successively blocked P waves; (2) the constancy of the distance between the escapes and the preceding penultimate P wave regardless of whether
Fig. 5. Concealed A-V conduction in incomplete A-V dissociation due to second degree A-V block causing blockage of two consecutive sinus impulses and postponement of an A-V nodal escape. Another record (lead II) of the case illustrated in figure 4, obtained 10 weeks earlier, with the same conventions used in the diagram.

Small upright P waves (rate 67) indicate a regular sinus rhythm. Only the first and fifth sinus impulses are conducted, yielding the first and third ventricular beat. All other ventricular complexes represent beats of nodal origin, interfering with transmission of independent sinus impulses. The nodal pacemaker (rate 34) has come into operation during ventricular pauses caused by a second degree A-V block. On this basis, in the second half of the record, from the fourth to the last ventricular beats, A-V dissociation has developed; one half of the sinus impulses being blocked, the others interfering with impulses of continuous nodal escapes. The nodal interval (1.8 seconds) is twice the sinus interval (0.90 second); hence, in this portion of the record, the position of P within the ventricular cycle is fixed. The unusual feature of the record is the failure of a nodal escape to occur at the expected time during the long pause (2.5 seconds), following the first ventricular beat. Here, two blocked P waves occur in succession and furnish the clue for the delayed appearance of the nodal escape.

The reason for the apparent inconsistency, concealed A-V conduction, is indicated in the diagram. The distance between the first of the two consecutively blocked P waves and the QRS complex terminating the long pause (P_e-R') is similar to that between the conducted beat and the subsequent escape (P_e-R') and that between two consecutive escapes (R'-R'). This suggests that the second sinus impulse, unlike others in the record, is not blocked high up in the A-V junction but only after penetrating beyond the point of origin of the A-V nodal pacemaker. This penetration causes: (1) disturbance in the regular discharge of the nodal pacemaker and (2) refractoriness in the A-V junction of sufficient degree to cause blockage of the subsequent sinus impulse.

Note that both P_e-R' (1.98 seconds) and P_e-R' (1.86 seconds) are longer than R'-R' (1.80 seconds). This is to be expected since the former two intervals include the conduction times from the auricles to the nodal pacemaker and from the nodal pacemaker to the ventricles in addition to the nodal cycle R'-R'. The fact that P_e-R' is shorter than P_e-R' is attributed to the shortening of the conduction time between the nodal pacemaker and ventricles subsequent to the longer period of rest in that area. However, a slight irregularity of the nodal pacemaker must also be present since the difference (0.06 second) between P_e-R' and R'-R' is too brief to cover the entire A-V conduction time.

the latter yields a ventricular response (P_e) or not (P_e); and (3) the occurrence, on occasion, of two nodal escapes in succession, revealing the approximate duration of the simple nodal interval R'-R', which approaches the duration of the P_e-R' and P_e-R' intervals. It thus becomes apparent that the first of two successively blocked sinus impulses penetrates into the A-V junction, beyond the nodal pacemaker, and is blocked somewhere between this pacemaker and the ventricles. In doing so it sets up refractoriness of the A-V junction as if it had traversed the junction completely, and it discharges the A-V nodal pacemaker before it can spontaneously release an impulse and thus shifts the timing of the latter which is expected to occur at intervals of about 1.90 second.

On two occasions (the fourth cycle of the third strip, and the last cycle of the third strip or first cycle of the fourth strip) the long ventricular interval is terminated by a beat which, in view of the whole record, has to be considered a conducted one and not an escape; these beats come too late to represent escapes. Here, too, penetration of and concealed nodal discharge by a preceding sinus impulse took place, permitting termination of a long cycle by a sinus beat (fig. 3).

In this case, therefore, concealed A-V conduction is revealed by both of its usual manifestations; the failure of two successive sinus impulses to reach the ventricles and the failure of a nodal escape to occur at the expected time.
The auricular rate is regular throughout and corresponds to a sinus rhythm at a rate of 62. The ventricular beating is slower and irregular because: (1) many auricular impulses fail to yield a ventricular response (second degree A-V block) and (2) nodal escapes occur at variable R-R' intervals. The conducted beats have P-R intervals varying between 0.20 and 0.40 second. There is only one instance, towards the end of the second strip, where A-V conduction occurs twice in succession with P-R lengthening from 0.20 to 0.32 second (a frequent occurrence in other portions of the long record not reproduced). Elsewhere, one to four consecutive P waves are blocked following a conducted beat before the A-V node escapes.

There are several unexpected features in this record, apparently inconsistent with the usual events in second degree A-V block. One is the occurrence on some occasions of repetitive blockage of consecutive sinus impulses (with ventricular pauses of 2.44 to 4.24 seconds), whereas consecutive conduction takes place in other instances. Another, is failure on many occasions of P-R to shorten subsequent to pauses engendered by blocked P waves. A third, is the variability of the duration of the ventricular pauses with nodal escapes absent when expected. Implication of the mechanism of concealed A-V conduction, as indicated in the diagram, permits a satisfactory interpretation of the record on a uniform basis.

Blockage of impulses penetrating into the A-V junction occurs either above or below the site of the nodal pacemaker. When the impulse is stopped at the upper level, a nodal escape promptly occurs but, unlike figure 4, the actual escape interval cannot be determined since nowhere in the record do two escapes occur in succession. An approximation of this interval, however, is possible by measuring (as in figures 4 and 5) the distance (P_n-R'_n and P_n-R'_o) to the penultimate P wave preceding the escape beat (not counting the P wave which coincides with the nodal QRS). As indicated in the diagram this distance is fairly constant (1.8 to 1.92 seconds, on one occasion 2 seconds), and this is true regardless of whether this impulse yields a ventricular response (P_e) or fails to do so (P_s). It thus becomes apparent that both impulses, P_e and P_s, travel to the nodal pacemaker and, by discharging it, set the time for initiation of a new nodal cycle. P_e then penetrates further to acti-
vate the ventricles, while Pr is blocked in the lower levels of the A-V junction, its conduction remaining concealed. Pr-R’ and Pτ-R’ represent, therefore, the sum of the unknown nodal escape interval plus the time spent by the sinus impulse Pr or Pτ in reaching the nodal pacemaker plus the conduction time of the nodal impulse to the ventricles. The variations in the duration of Pτ-R’ and Pτ-R’ can be ascribed to variations in the conduction times above and below the nodal pacemaker and/or to a slight irregularity of the nodal discharge. The fact that Pτ-R’ (1.80-1.88 seconds) is consistently shorter than Pr-R’ (1.90 to 2 seconds) can mean only that any expected lengthening of the conduction time of impulse Pτ above the site of the nodal pacemaker is outbalanced by a shortening of the conduction time of impulse R’ below the nodal pacemaker as a result of the longer rest period in the latter region (fig. 5). Variations in conduction times below the nodal pacemaker appear to be more significant than those above it and this suggests that the subsidiary pacemaker is located in the upper region of the A-V junction as indicated in the diagrams. Variations of the conduction time Pr-R subsequent to an escape (R’) seem to depend on variations of the R’-Pτ distance.

Partial penetration of apparently blocked P waves, established in this way, can then be implied to account for the repetitive blockage of P waves as well as for the failure of P-R to shorten when expected. Each such penetration of the impulse sets up varying degrees of refractoriness in the A-V junction so as to lengthen the conduction time to the ventricles of a subsequent impulse, to permit only its partial conduction or to stop its conduction altogether. In the long pauses of the second and third strip, three and two successive sinus impulses, respectively, penetrated to the nodal pacemaker but were blocked below it before reaching the ventricles. Thus, the ventricles remained unstimulated because conduction failed and the spontaneous discharge of the subsidiary pacemaker was shifted several times in succession (repetitive concealed conduction).

The conditions determining the great variability of the manifest A-V response can be further unraveled as exemplified diagrammatically in figure 6A. In essence, the conduction disturbance present can be viewed as a second degree A-V block, varying between 2:1 and 3:2 ratios, with several intermediate stages resulting from concealed conduction of the ‘blocked’ auricular impulse. The extent to which this impulse (Pτ) penetrates into the A-V junction, and whether it is stopped above or below the site of the nodal pacemaker, depends on its R-Pτ distance. The speed at which this penetration takes place is determined mainly by the P-R interval of the preceding conducted beat. The interplay of these two factors sets the pattern for subsequent events whether a nodal escape occurs in time, whether its appearance is delayed due to premature discharge by one or several subsequent penetrating impulses, or whether the ventricular pause is terminated by a conducted beat.

The evidence for this interpretation is provided by the following facts: The three Pτ impulses in the record that are blocked high in the A-V junction and do not reach the pacemaker have R-Pτ intervals of 0.56, 0.54 and 0.52 second; all other R-Pτ intervals associated with penetration of impulse Pτ measure 0.54 second or more. Now, one might expect that the blocking effect of the concealed penetration of Pτ upon conduction of impulse Pτ depends on the extent of the penetration of Pτ, and, therefore, increases with lengthening of the R-Pτ distance. Yet, paradoxically, so it seems, amongst the Pτ impulses with concealed conduction those with the shorter R-Pτ intervals exert the greater blocking influence upon impulses Pτ. Thus, an R-Pτ distance of 0.54 to 0.56 second is associated with blockage of impulse Pτ above the nodal pacemaker; an R-Pτ of 0.56 to 0.62 second with penetration of impulse Pτ; and an R-Pτ of 0.60 to 0.68 second with delayed but complete A-V conduction of impulse Pτ. The clue to the understanding of this peculiar relationship is found in the varying duration of the conduction time (Pτ-R) of the impulse preceding impulse Pτ. Since in second degree, A-V block conduction of consecutive impulses is commonly associated with progressive prolongation of the conduction time (until one impulse is blocked), it is reasonable to as-sume that the time for concealed conduction of impulse Pτ to the site of the nodal pacemaker lengthens when the preceding Pτ-R interval is prolonged; and the slower the concealed conduction of Pτ, the smaller are the chances for Pτ to enter, penetrate into or traverse the A-V junction. This is in short a Wenckebach phenomenon of concealed conduction. Hence, ceteris paribus, it is the speed and not the extent of penetration of impulse Pτ that determines whether impulse Pτ can pass to and beyond the nodal pacemaker or is blocked above it.
Fig. 6.1. A diagrammatic representation of the fundamental conduction disturbance, and its principal variations encountered in figure 6. Intermediate stages between 2:1 and 3:2 A-V block. Extent and speed of concealed conduction. A corresponds to a portion of the top strip of figure 6, starting with the tenth P wave; B corresponds to another portion of the same strip, starting with the second P wave; C represents a portion of the third strip starting with the seventh P wave; D starts with the first P wave of the second strip and E with the ninth P wave of the second strip. The conventions and symbols are the same as in figure 6. The varying degree of inclination of the oblique lines indicates the varying speed at which sinus impulses traverse or penetrate into the A-V junction. The first number in column A-V (within an oblique line) indicates the length (in hundredths of a second) of the P-R interval of impulse P1; the number to the right of it the R-P interval of impulse P2.

The five strips are arranged in such a manner as to demonstrate three intermediate stages (B-D) between a 2:1 A-V block with nodal escape (A) and a 3:2 A-V block with the Wenckebach phenomenon (E). The intermediate stages result from quantitative differences of the effect of concealed conduction of impulse P1. In B, P1 penetrates slowly to the site of the nodal pacemaker and causes high blockage of the subsequent impulse P2 so that escape of the node is delayed by about one sinus cycle. In C, P1 penetrates to the same extent but at somewhat faster rate; this in turn permits penetration of impulse P2 to the nodal pacemaker so that the nodal escape is delayed by about two sinus cycles (repetitive concealed conduction). In D, nodal escape is delayed by penetration of P1, but impulse P2 succeeds in slowly traversing the junction so that the pause is terminated by a conducted beat.

These different effects of impulse P1 upon subsequent events, evidently dependent upon both the degree and the speed of its penetration, can be further analyzed in the following manner: the extent to which P1 penetrates into the A-V junction is clearly determined by its R-P1 distance. Thus, in A, where R-P measures 0.54 second, P1 is blocked above the nodal pacemaker. In B-D, with R-P1 distances of 0.56 to 0.62 second. P1 passes beyond the site of the nodal pacemaker but fails to reach the ventricles. In E, with an R-P1 of 0.68 second, P1 traverses the junction completely. The speed at which P1 travels through the junction under these various circumstances appears to depend primarily on the preceding P-R interval. This is demonstrated by comparison of B and C. In B, where P-R is longer (0.4 second) and R-P shorter (0.56 second), P1 travels slower (and has a more marked effect on P2) than in C where P-R is shorter (0.3 second) and R-P1 is longer (0.62 second). Comparison of C and D, however, reveals that R-P1 and P-R intervals cannot be the only factors which influence the effects of P1 upon subsequent impulses. The values are the same in both instances, yet in C, P2 fails to traverse the junction, while in D, it succeeds in doing so. Other factors operating during the "critical phase" of A-V conductivity (see text) may be invoked to account for such variations in the expected events.
part of the ventricular cycle to which partial penetration of the A-V junction by sinus impulses seems limited under normal conditions. However, we realize that with this method of determination, one may underestimate the duration of the phase of concealed conduction. It is conceivable that, when gaged by its other effect, viz., upon transmission of the subsequent impulse, concealed conduction may already become manifest when the impulse has not yet penetrated far enough to reach the site of the subsidiary pacemaker.

In cases of sinus rhythm with second degree A-V block, or auricular fibrillation,

![Diagram](image)

**Fig. 7.** Blockage of consecutive impulses in auricular flutter, with ventricular group beating due to two regions of impedance to A-V conduction. Conventions in the diagram are the same as in figure 1.

Regular continuous undulations of the baseline (F waves), best seen in leads II and III, indicate the presence of auricular flutter (rate 316). The ventricular response is irregular; groups of four or five faster beats are separated by pauses. The R-R of the faster beats is a little longer than two F-F intervals; the R-R of the pauses is shorter than four but longer than three F-F intervals. Within each run of ventricular beats the R-R progressively shortens so that the last R-R before a pause is invariably shorter than the first R-R following a pause. The R-R between the initial beats of two consecutive fast groups is an exact multiple (10 or 12) of the F-F interval. This grouping of the ventricular beating, and recurrence of a characteristic R-R spacing within and between the groups, indicates the operation of the Wenckebach mechanism in the transmission of every other flutter impulse to the ventricles.

As indicated in the diagram, alternate flutter impulses are stopped high in the A-V junction so that the effective auricular rate, that is the number of impulses penetrating deeper into the junction, is reduced to half the flutter rate. Four out of five, sometimes five out of six, of these effective impulses traverse the junction and activate the ventricles, the last of each such group being blocked. Hence, two areas of impedance to A-V conduction are in operation: One, acting at a higher level, reduces the number of flutter impulses to become effective; this may be considered an area of physiologic interference resulting from the normal refractoriness of A-V junctional tissue to rapid stimulation; the other, operating at a lower level, causing Wenckebach periods with dropping out of every fifth or sixth ventricular response to the "effective" flutter impulses; this may be considered a manifestation of block caused by an abnormal depression of conductivity in the lower A-V junction.

Although one of the characteristic manifestations of concealed A-V conduction seems to be present, namely, blockage of several auricular impulses in succession with varying penetration of the A-V junction, this mechanism need not be invoked here. With rapid auricular rates, implication of two areas of impedance, as outlined above, may be sufficient to account fully for the failure of several consecutive auricular impulses to traverse the A-V junction.
Fig. 8. Auricular tachycardia with ventricular group beating (bigeminy) caused by two regions of A-V block (A), and by concealed A-V conduction (B).

The two records were obtained several days apart; conventions in the diagrams are the same as in figure 1. The additional horizontal line at the level of A-V indicates an area of block in the upper part of the A-V junction. The numbers indicate the duration of the P-R intervals in hundredths of a second.

In A, the auricular action is rapid (176) and regular (auricular tachycardia), while the ventricular rate is slower and irregular. Throughout the record there is grouping of ventricular beats, in that short cycles alternate with long ones (bigeminy). All ventricular complexes except the fourth (a ventricular premature systole) are alike and are due to conducted auricular impulses; their QRST contour is abnormal because of intraventricular block. In most of the record the long ventricular cycles are a little shorter than four P-P intervals and the short ventricular cycles a little longer than two P-P intervals; the interval between corresponding beats measures exactly six P-P intervals. In the beginning of the record this consistency in ventricular spacing is disturbed: the second, third and fourth cycles are shorter than corresponding cycles of the ventricular bigeminy seen elsewhere in the strip. However, the interval between the first and fifth ventricular beat is again an exact multiple (10 times) of P-P.

On the basis of these measurements the following interpretation, which is indicated in the diagram, is suggested: A-V conduction of the rapid auricular impulses is impaired by the presence of two levels of block in the A-V junction. Only every other auricular impulse traverses the upper and penetrates into the lower level of block. These impulses, while traveling toward the ventricles are either delayed or blocked. Usually, of three successive penetrating impulses the second is more delayed than the first and the third is stopped; this causes the bigeminy present in most of the record with P-R intervals alternating between 0.36 and 0.38 to 0.41 second. On one occasion (first portion of the record) the third successive penetrating impulse manages to reach the ventricles, though with a very long P-R of 0.76 second, and the next (fourth) penetrating impulse would be expected to be blocked (a 3:2 response being changed to a 4:3 response). But at this time a ventricular premature systole occurs which, by retrograde spread of its impulse, prevents transmission of another penetrating auricular impulse and causes a true compensatory pause. Thus, bigeminy is maintained. Similarly, as in figure 7, the two areas of conduction impedance (both caused by depression of the A-V junction) can account for the unusual A-V ratio and there is, therefore, no need to implicate the mechanism of concealed conduction.

In B, at a slightly slower (167) auricular rate, ventricular bigeminy, on a different basis, is seen in the second half of the record. Its longer cycles measure a trifle more and its shorter cycle a trifle less than two P-P intervals. The distance between corresponding beats equals exactly the length of four P-P intervals. In the first half of the record the alternation of long and short cycles involves only the first three beats. Subsequently, two longer pauses occur, both shorter than three P-P with two
flutter or tachycardia with a slow ventricular rate, delay or failure of appearance of escape beats can be attributed to concealed A-V conduction and discharge of the subsidiary pacemaker by some of the apparently blocked auricular impulses. Under such circumstances, a subsequent auricular impulse, occurring after recovery of A-V conductivity, may cross the junction to activate the ventricles before a delayed escape takes place (figs. 3 through 6). Thus, in a record with unquestionable A-V nodal escapes, contrary to the rule, the longest ventricular interval may be terminated by a conducted beat and shorter intervals by escapes (fig. 3). On occasion, the distinction between the two types of beats may become difficult or impossible, unless they are characterized by differences in contour.\textsuperscript{38, 39}

In figures 3 through 6, a method is demonstrated to determine whether an A-V nodal escape or a conducted beat terminated the pause engendered by concealed conduction shorter cycles in between. Again the comparison of ventricular with auricular cycles suggests that the grouping of ventricular beats is caused by a complex conduction disturbance in the A-V junction as indicated in the diagram.

Fundamentally, a 2:1 conduction ratio is present with varying penetration of the A-V junction by the “blocked” auricular impulses. Ventricular bigeminy is the result of alternation of shorter and longer P-R intervals of the conducted impulses, which is caused by deeper penetration of alternate “blocked” impulses. Little or no penetration permits the subsequent P-R to shorten; this in turn enhances penetration of the following blocked impulse; the more extensive penetration of this lengthens the subsequent P-R and consequently penetration of the following blocked impulse is again reduced; thus the sequence starts anew. During the irregularity with the two longer pauses seen in the first part of the record, a second factor seems in action in addition to varying penetration of blocked impulses, namely, the influence of cycle length upon the refractory period. The details are as follows: the first two ventricular beats show alternation of P-R as seen during the bigeminy at the end of the strip. However, subsequent to the shorter (0.32 second) of the two P-R intervals, two auricular impulses are blocked in succession. Apparently here, for reasons unknown, the first of the two penetrated deeper than usual causing more refractoriness of the A-V junction and thus transiently changed the fundamental 2:1 A-V ratio to a 3:1 ratio—actually an attempt at 3:2 conduction (fig. 1). The result is the first longer ventricular pause which in turn has the following effects: it permits subsequent shortening of P-R to 0.26 second and thus lengthens the R-P of the next auricular impulse; on the other hand, it prolongs the refractory phase so that penetration of the blocked auricular impulse after this short P-R interval becomes minimal. Consequently, the P-R remains also short (0.28 second) in the next conducted beat. Only after this beat does deeper penetration of the blocked auricular impulse take place and, in turn, causes lengthening of the subsequent P-R to 0.38 second. The following pause, too, is caused by a transient change of a 2:1 to a 3:1 A-V response, but here it is due to the failure of an impulse to traverse the junction because of shortening of its R-P distance. As a consequence, the subsequent auricular impulse succeeds in doing so, though at a longer P-R (0.32 second) and this initiates the bigeminy analyzed above.

In this record, in contrast to record A, the ventricular bigeminy cannot be explained merely on the basis of two levels of block in the A-V junction. Here, concealed A-V conduction must be postulated. The two mechanisms are not exclusive and may operate in the same case on different occasions or simultaneously in conjunction (fig. 3).
be equal. Slight differences can be expected and explained by variations in the A-V conduction times above and below the subsidiary pacemaker, and/or some irregularity in the discharge of the latter.\textsuperscript{21} Whenever distances between P of a conducted impulse (P\textsubscript{c}) and R of a subsequent beat are found considerably longer than the established P\textsubscript{c}-R' distance, in other words, whenever the escape fails to appear at the expected time, concealed conduction of the subsequent interposed and apparently blocked P wave has to be postulated (figs. 3, 4 and 5). As stated before, under such circumstances the auricular impulse that follows the concealed discharge may be conducted to the ventricles before the delayed escape is due, so that the long R-R interval is terminated by a conducted beat and not by an escape. Thus, if the distance between P\textsubscript{c} (the “blocked” P following P\textsubscript{c}) and the subsequent R approaches the established P\textsubscript{c}-R' distance, R represents an escape; if it is considerably shorter, the beat terminating the pause engendered by concealed conduction is identified as a conducted beat (figs. 3 and 4). This method can also be applied when blockage involves more than two sinus impulses in succession (fig. 6), by measuring the distance from each of the consecutively “blocked” P waves, to the next R wave and comparing it with an established (P\textsubscript{c}-R') or (P\textsubscript{c}-R') distance. Under such circumstances, partial penetration of impulses into the A-V junction may be responsible for both repetitive extraneous discharge of a subsidiary pacemaker, “ahead of schedule,” and repetitive blockage of consecutive impulses above or below the site of this pacemaker. As indicated in two instances of figure 6, such a mechanism may result in prolonged ventricular asystole with apparent failure of the subsidiary pacemaker to take over.

The record illustrated in figure 6 is of particular interest because, in spite of the great variations in manifest A-V response (varying between 3:2 and 5:1), the disturbance can be shown to represent essentially an A-V block varying between 2:1 and 3:2 with tendency to concealed conduction of the “blocked” impulse. This is demonstrated in figure 6.1. Depending on both the extent and speed of concealed conduction, three intermediate stages between the ratios of 2:1 and 3:2 A-V response can be defined and actually identified in the record. The auricular impulse that follows the partially conducted one may: (1) be blocked above the site of the subsidiary pacemaker, (2) penetrate beyond the subsidiary pacemaker but fail to reach the ventricles (repetitive concealed conduction), or (3) yield a ventricular response after a delay. Evidently, with repetition of concealed conduction, further patterns of A-V response may develop in that repetitive concealed conduction may again be responsible for the same threefold effect upon conduction of the subsequent impulse and discharge of the subsidiary pacemaker.

Failure of several consecutive auricular impulses to cross the A-V junction, one of the principal manifestations of concealed AV-conduction, is encountered commonly in auricular flutter and auricular tachycardia upon exhibition of digitalis. Here, however, the mechanism usually is different and, in the presence of even conduction ratios (for example, 4:1) or group beating of the ventricles, can be explained without implication of concealed conduction. Since the normal refractory phase of the A-V junction is longer than that of the auricles, its reaction to rapid stimulation (that is, at rates over 200) is a 2:1 response which reduces to one half the number of impulses penetrating into the junction. This is a normal protective mechanism of the heart, preventing rapid ventricular rates. On the other hand, digitalis primarily depresses A-V conduction of the “effective” impulses as they spread toward the ventricles. Thus, two areas of impedance to A-V transmission of impulses are created: one, high up in the junction, caused by physiologic interference, the other, at a lower level, caused by abnormal depression with block; the former, with a regular 2:1 response, the latter, showing all varieties of second degree A-V block. Irregularity of ventricular beating or grouping of ventricular beats (for example bigeminy) can, in the majority of cases, be unraveled by identifying the structure of simple Wenke-
bach periods in the ventricular response to the effective impulses occurring at half the actual auricular rate\textsuperscript{26} (fig. 7).

Concealed A-V conduction may, of course, also occur with rapid auricular rates or complicate the mechanism of the double area of conduction impedance. Thus, varying penetration of the region of interference by the “ineffective” auricular impulses has to be implicated to account for alternation of ventricular cycle length in cases of auricular flutter with a persistent 2:1 ventricular response.\textsuperscript{10, 15, 22} More pronounced penetration of every second impulse, representing attempts at 3:2 A-V conduction may result in the uncommon occurrence of a 3:1 conduction ratio in auricular flutter. In auricular tachycardia with less rapid rates (below 200), irregular ventricular response and failure of several successive impulses to traverse the A-V junction can be explained on a similar basis as in auricular flutter; one merely has to postulate two levels of depressed conductivity instead of the combination of one of physiologic interference with one of abnormal block (fig. 8).

Varying penetration of auricular impulses altering refractoriness of the A-V junction to subsequent impulses occurring in rapid succession can, at least in part, account for the complete irregularity of the ventricular action in auricular fibrillation.\textsuperscript{27, 28}

While the phenomenon of concealed conduction is most readily and most commonly demonstrable in the A-V junction, it can be invoked to account for certain features of impaired intraventricular conduction as well. The supraventricular impulse that reaches the site of a blocked bundle branch via the septum, may affect the recovery time of the defective bundle branch system and thus influence propagation of the subsequent supraventricular impulse. Two well known phenomena find their explanation in such concealed intraventricular conduction: (1) the rare manifestation of second degree block of intraventricular conduction,\textsuperscript{10} and (2) the perpetuation of aberrant ventricular conduction, initiated by the resumption of a rapid ventricular rate subsequent to sudden transient ventricular slowing; this occurs in auricular tachycardia with second degree A-V block,\textsuperscript{41} or, more commonly, in auricular fibrillation with rapid ventricular rate.\textsuperscript{42, 24} A somewhat similar mechanism, concealed retrograde intraventricular conduction, can be postulated in cases of pre-excitation with auricular fibrillation\textsuperscript{43, 44} to account for the peculiar grouping of beats with normal and anomalous conduction, with transitional complexes in between. It would appear that in these cases an impulse, conducted over one of the two available conduction pathways tends to enter the other in a retrograde fashion, to render it refractory for the subsequent supraventricular impulse. Finally, the occurrence of concealed conduction of the impulse within a re-entry path was postulated in certain cases of intermittent bigeminy, due to ventricular premature systoles. Such an incomplete penetration of the re-entry path may become manifest in an unexpected prolongation of the coupling of a subsequent premature systole,\textsuperscript{45} or the failure of the premature systole to occur when expected.\textsuperscript{27} A similar mechanism was proposed by Schott\textsuperscript{46} to account for inconsistencies in the occurrence of parasystolic beats.

The short period of concealed conduction during which an impulse penetrated partway into the preformed conduction pathways, ordinarily is sandwiched between the period of absolute refractoriness, causing complete failure of conduction and relative refractoriness, permitting passage of impulses at slow speeds. Clinical records with disturbances of cardiac rhythm can be found in which, as in the controlled animal experiment, the time span within the cycle during which only concealed conduction takes place, can be separated from the periods of absolute and relative refractoriness and expressed in terms of R-P intervals (fig. 2). The demarcation may be sharp or there may be some overlap in either direction. In dealing with cases with depressed A-V conductivity this overlap is usually more marked; in fact, the phase of concealed conduction may become inseparable from the end of the absolute and the beginning of the relative refractoriness, so that within a certain
range of R-P intervals the response of the A-V junction is unpredictable. Thus, a “critical period” may be present during which, at the same R-P interval, the impulse sometimes is blocked, sometimes may pass at a long P-R interval, and sometimes penetrates into the A-V junction without reaching the ventricles.

There are several factors which might influence the extent of passage of an impulse occurring in such a critical phase of conductivity. It may be extraneous influences, in particular, momentary alterations of vagal tone; or it may be the effect of the duration of the preceding cycle, shortening or lengthening refractoriness of the A-V junction (fig. 8B). These two factors may operate with normal as well as depressed conductivity. When conductivity is depressed, a third factor, namely, a supernormal phase of recovery, may come into play to influence the response of the A-V junction to individual impulses.

A supernormal phase of conduction may be responsible for the occurrence of concealed conduction, in that an impulse that otherwise would be blocked may penetrate into the conducting tissues. The phases of concealed conduction and of supernormal conduction can be expected to overlap in various ways, rather than show a fixed temporal relationship, and the manifestations of the supernormal phase can be expected to vary accordingly. Thus, as a result of supernormal conduction, the phase of concealed conduction may be shifted to the left of the recovery curve and widened, abbreviating the absolute refractory phase. If supernormal conductivity affects only the last portion of the phase of concealed conduction, the expected result is a shortening of the phase of concealed conduction and a lengthening of the relative refractory period. On the other hand, there may be no change in the location or duration of the phase of concealed conduction, but, impulses occurring during its earlier portion may penetrate at a faster speed and have a less marked effect on the conduction of subsequent impulses than those occurring during its later portion; for the same reason impulses which occur during the early portion of the phase of concealed conduction may reach and discharge another pacemaker, whereas those occurring later may fail to do so. Evidently, if the supernormal phase of conduction permits not only partial penetration but full conduction of early impulses, and if the phase of concealed conduction outlasts the phase of supernormal conduction, a period of full conduction may precede a period of concealed conduction. In short, the supernormal phase of conduction may manifest itself merely in a change of the duration of the phase of concealed conduction: lengthening it by shortening the absolute refractory period, or shortening it by lengthening the relative refractory period. Under such circumstances the presence of a supernormal phase of conduction may easily remain unrecognized. By contrast, it will become manifest by the occurrence of: (1) more rapid conduction of the earlier fully conducted impulses compared with later ones, (2) full conduction of earlier impulses and only partial (concealed) conduction or nonconduction of later impulses, or (3) deeper or faster penetration of the earlier impulses amongst those which are only partially conducted.

Concealed conduction of impulses is not restricted to forward conduction through the A-V junction, but may affect retrograde spread of impulses as well. The simplest and most common result of concealed A-V conduction in retrograde direction, the compensatory pause or the P-R lengthening after interrupted premature ectopic beats, has been mentioned. Less frequent and more involved manifestations are the occurrence of double blockage of retrograde A-V nodal impulses in A-V nodal tachycardia and the occurrence of concealed re-entry of the nodal impulse in reciprocal beating. On the other hand, concealed retrograde conduction of A-V nodal impulses may also be responsible for a state of supernormality influencing forward conduction of subsequent sinus impulses. An analogous mechanism was postulated for retrograde conduction of ventricular or A-V nodal impulses after concealed conduction of sinus impulses. Under such circumstances an extraordinary condition may result in which concealed conduction may enhance rather than inhibit the conduction of a subsequent impulse.
CONCLUSIONS

1. Conductivity of the A-V junction cannot be gaged by the ratio of the manifest A-V response.

2. When two consecutive auricular impulses fail to traverse the A-V junction the mechanism of concealed A-V conduction has to be considered. Apparent 3:1 A-V conduction may actually represent an abortive attempt at 3:2 A-V conduction.

3. Intermediate stages between 2:1 and 3:2 A-V response can be defined, depending upon the quantitative effect of concealed conduction of the “blocked” impulse. Concealed conduction may cause delayed transmission, partial penetration (repetitive concealed conduction) or complete blockage of the subsequent impulse.

4. The effects of concealed conduction depend not only on the extent, but also on the speed of penetration of the “blocked” impulse. Regardless of the ultimate extent of penetration, the slower the speed of concealed conduction, the more marked is the effect upon conduction of the subsequent impulse.

5. In repetitive concealed conduction involving more than two consecutive impulses, a Wenckebach phenomenon of concealed conduction can be postulated.


7. In second degree A-V block, occasional concealed discharge of the subsidiary A-V nodal pacemaker by a penetrating (“blocked”) auricular impulse may cause the longest ventricular intervals to be terminated by conducted beats and shorter ones by escapes.

8. Repetitive concealed conduction, responsible for repetitive concealed discharge of the subsidiary pacemaker, may account for prolonged ventricular asystole.

9. In the presence of rapid auricular rhythms, the mechanism of a double region of impedance to A-V conduction or the mechanism of concealed A-V conduction may be responsible for group beating of the ventricles.

The two phenomena are not mutually exclusive.

10. Concealed conduction may give rise to hitherto undescribed manifestations of the supernormal phase.

11. When A-V conductivity is depressed, an overlap of the phase of concealed conduction with the absolute and/or relative refractory phase may result in a “critical period” of the cycle, during which the response of the A-V junction cannot be predicted.

12. Recognition of concealed conduction is indispensable for the understanding of disturbances of conduction and formation of the cardiac impulse.

SUMMARY

1. Concealed conduction, a neglected but important phenomenon of impaired conductivity, occurring under physiologic and pathologic circumstances, consists of incomplete penetration of preformed conduction pathways by an apparently blocked impulse. It manifests itself by its effect upon conduction or formation of a subsequent forward or retrograde impulse.

2. Nine selected clinical records with various types of conduction disturbance are presented, illustrating the application of the concept of concealed conduction to the analysis of simple and complex arrhythmias, and demonstrating the usefulness of the concept in the understanding of otherwise unexplainable features.

3. The most common example of concealed A-V conduction affecting transmission of a subsequent impulse is the compensatory pause after ventricular premature systoles and the P-R prolongation subsequent to their interpolation. Concealed conduction is responsible for double or multiple blockage of consecutive auricular impulses occurring at slow, normal or moderately rapid rates. When the auricular rate becomes faster than 200, as in auricular flutter, failure of the A-V junction to respond to consecutive impulses can be explained by a different mechanism, namely, two areas of conduction impedance, one due to physiologic interference, the other due to abnormal block. However, concealed A-V conduction may complicate the latter
mechanism; thus, the two mechanisms may operate in the same case on different occasions or in conjunction.

4. The effect of concealed conduction upon formation of impulses becomes manifest by altering the expected appearance of impulses of a subsidiary pacemaker. Thus, in A-V dissociation, the regular cycle of a nodal pacemaker may be periodically disturbed by unsuccessful attempts of sinus impulses at capturing the ventricles. Or, in second degree A-V block, nodal escapes may fail to occur when expected. When such a concealed nodal discharge occurs several times in succession, long ventricular pauses may ensue and it may be difficult to determine the origin of the beat terminating such pauses, whether ectopic or conducted. A method is demonstrated which permits such a differentiation.

5. The phase of concealed A-V conduction, that is the short period of the cycle between the absolute and relative refractory phases, during which impulses penetrate into the A-V junction without traversing it, can sometimes be sharply delineated in terms of R-P intervals. On occasion, however, particularly with depressed conductivity, there may be a considerable overlap with the absolute and relative refractory phase, and their transition may become a “critical period.” For such overlaps the operation of a supernormal phase of A-V conduction may be invoked.

6. Principles developed in studying concealed conduction in A-V junctional tissues can readily be applied to account for certain phenomena associated with impaired intraventricular conduction. Thus, concealed intraventricular conduction may account for the rarity of second degree bundle branch block, for continuation of aberrant ventricular conduction in supraventricular tachycardia, for the pattern of impulse spread in certain cases of ventricular pre-excitation, and for variations in appearance and coupling of ventricular premature systoles.

**Summario in Interlingua**

Es demonstrate que le recognition de celate conduction del impulso cardiac es indispensabile pro le comprehension de multe arrhyth-


Concealed Conduction: Further Evaluation of a Fundamental Aspect of Propagation of the Cardiac Impulse
RICHARD LANGENDORF and ALFRED PICK

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