Experimental Demonstration of Concealed AV Conduction in the Human Heart

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The term concealed conduction was introduced in a report from this department to define in a concise and uniform manner the after effects of partial penetration of atrioventricular (AV) conduction pathways by impulses originating in the atria or ventricles. This concept proved indispensable in the proper analysis and understanding of a great number of arrhythmias encountered in routine electrocardiography. Recently, the validity of our assumptions has been firmly established by others in the experimental laboratory with the help of modern techniques, particularly the recording of action potentials directly from the different portions of the conduction system.

With the advent of artificial cardiac pacing as an accepted method of treatment of Stokes-Adams disease, it became feasible to produce in the human heart conditions prone to reveal the phenomenon of concealed conduction. Simple experimental procedures can be applied at the bedside without danger or discomfort to the patient and various disturbances of cardiac rhythm can be reproduced at will. Such observations, some of which shed new light on the ways concealed conduction influences subsequent impulse formation and conduction, are the subject of this report.

Material and Methods

All records were obtained on patients in need of artificial pacing because of frequent Stokes-Adams attacks. Except in one instance (fig. 7), the observations were made during the use of a transvenous catheter pacemaker, prior to implantation of a permanent one. The experiments consisted in changing the rate of single stimuli of constant duration and suprathreshold strength, or in altering the spacing of paired stimuli according to a method described elsewhere. In one group of experiments the catheter tip was kept in the right atrium, in another group the right ventricle was stimulated. In the case illustrated in figure 7 a permanent pacemaker had been implanted in the left ventricle.

Results

Catheter Electrode in the Right Atrium

Figures 1 to 4 are records (lead I) obtained in a patient with arteriosclerotic heart disease, permanent left bundle-branch block, and varying degrees of AV block. The four types of experiments were performed when, during artificial pacing, the AV conduction disturbance had almost subsided. In figures 1 to 3, the atria were driven at frequencies exceeding those of the natural (sinus) pacemaker, which was completely suppressed. In figure 4, at a lower stimulation rate, the two types of impulses were in competition for atrial control.

Figure 1 shows a replica of the classical

![Figure 1](image-url)

Concealed AV conduction during 2:1 ventricular response to rapid pacing of the atria. Each stimulus artifact (S) is followed by a P wave (P) (lead I).
Atrial stimulation by paired artificial impulses. The duration of the phase of concealed AV conduction and the effects of induced premature impulses on atrial excitability (lead I).

Basic experiment of Lewis and Master on concealed AV conduction. Driving the atria at a fast rate of 168/min. produced a 2:1 ventricular response, which changed to a 1:1 response when the frequency of artificial atrial stimulation was suddenly reduced to 84/min., one half the previous rate. Thus, the ventricular rate remained constant but, as alternate “ineffective” atrial impulses were eliminated, the P-R interval shortened from 0.24 sec. during 2:1 conduction to 0.19 sec. during 1:1 conduction. The most likely explanation of the longer P-R intervals is penetration into the AV junction (concealed conduction) of the apparently blocked impulses. An alternative interpretation, operation of two AV junctional pathways with different refractory periods and conduction speeds appears less likely in view of subsequent experiments in this patient.

In figure 2 are assembled six selected seg-
Repetitive concealed AV conduction. Note that in lower panel the ventricles fail to respond to three consecutive atrial stimuli (X₁-S-X₂) (lead I).

Figure 3

ments of long records illustrating the effects on AV conduction of intermittent paired stimulation of the atria in this patient. While the basic driving cycle (S-S) was kept constant (at a rate of 94/min.) the position of the interpolated extra stimulus (X) was progressively shifted from S₁ toward S₂. In all instances (panels A-F), X elicits an atrial response but only in panel F, after the longest S₁-X interval, is it propagated through the entire AV junction to reach and activate the ventricles. In panels A to D, the extra stimulus, while stopped within the AV junction, progressively slows (panels A and B), and finally prevents (panels C and D) AV conduction of S₂. In panel E, failure of the atria to respond to S₂ (see below) seems to preclude demonstration of concealed conduction of X, but the latter can be postulated in view of the findings in panels D and F. Thus, there is concealed AV conduction of impulse X over a period of at least 0.11 and probably over 0.14 sec.

The response of the atria to basic and premature artificial stimulation is prompt throughout panel A to C. In panel D, there is a latency of 0.08 sec. of the P wave after S₂; in panels E and F, this P wave is absent. Retardation and failure of atrial excitation by S₂ can be attributed to the progressively shorter X-S₂ interval: in panel D, although the atrial myocardium is partially refractory, it still permits interpolation of X (as in panels A to C); in panels E and F complete refractoriness of the atria at the time of S₂ results in a compensatory pause after X. An obvious analogy is thus apparent to the effects of ventricular premature systoles on AV junctional tissues with regard to interpolation or production of a compensatory pause.¹²

Figure 3 illustrates the artificial production of repetitive concealed AV conduction. During a faster basic driving rate of 120/min., which lengthened the AV conduction time to 0.28 sec., extra stimuli X₁ and X₂ were interpolated into two consecutive basic cycles, so that the

Discharge of a subsidiary AV junctional pacemaker by concealed antegrade AV conduction in artificial atrial parasystole. P¹ represents atrial responses to artificial pacemaker stimuli indicated by dots in the diagram and broken lines at A (atrial) level. S-A indicates impulse propagation from and to the S-A node; A-V indicates complete or partial impulse propagation in the A-V junction (which is divided by a horizontal line into an upper and lower portion) (lead I).

Figure 4

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atria received five stimuli in rapid succession: S-X₁-S-X₂-S. With S-X intervals of 0.21 sec. (upper panel), the proximal AV junction was completely refractory to the premature impulse and hence regular ventricular responses to S stimuli, as well as the corresponding P-R intervals, were undisturbed. However, when S-X intervals were increased to 0.24 sec. (lower panel), a long ventricular pause occurred after the response to the first (S) of the group of five rapid stimuli. Evidently, X₁ penetrated into the AV junction to prevent conduction of the subsequent S. Yet this stimulus must likewise have entered the AV junction over the same pathway, for in its wake X₂ was stopped before regular ventricular responses to driving stimuli were resumed (with shortening of the P-R interval of the first conducted beat). Conceivably, during the preceding repetition of concealed conduction not only depth but also speed of partial penetration of the AV junction by atrial impulses may have declined progressively as is indicated in the diagram (a “Wenckebach phenomenon of concealed conduction”³).

Figure 4 shows the effect of concealed AV conduction on impulse formation in the AV junction. By driving the right atrium at a rate slower than that of the natural (sinus) pacemaker an atrial parasystole has been produced artificially. Of the five pacemaker stimuli that fall outside the refractory phase of the atria and elicit an atrial response (P¹) only the first, fourth, and fifth produce ventricular responses; the second and third are stopped in the AV junction and ventricular pauses of different length ensue. The first (shorter) one (1.16 sec.) is terminated by a junctional escape interfering with a simultaneously occurring sinus impulse, the sinus P wave being superimposed on the QRS of the escaped beat. In the second (longer) pause (1.28 sec.), such an escape failed to occur at the expected time, indicating premature extraneous discharge of the subsidiary center by concealed conduction of the third P¹ impulse. This impulse penetrated deeper than the second because its later occurrence after the preceding conduction of the sinus impulse permitted partial recovery of the AV junction. This interpretation was fortified by the facts that (1) the duration of the escape interval was quite constant (1.16 sec.) and (2) whenever the ventricular cycle exceeded this value it contained a nonconducted P¹, with a R-P¹ interval of at least 0.24 sec.

**Catheter Electrode in the Right Ventricle**

Figure 5 shows a case of complete AV dissociation caused by advanced AV block in another patient with arteriosclerotic heart disease. There is competition for ventricular control between responses to an artificial ventricular pacemaker rate (54/min.) and an AV junctional pacemaker discharging spontaneously at a faster rate (60/min.); the QRS complexes of the latter are prolonged due to right bundle-branch block. Whenever a pacer impulse falls outside the refractory period following spontaneous ventricular activation, it produces a premature ventricular

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**Figure 5**

Discharge of an AV junctional pacemaker in complete AV dissociation by concealed retrograde conduction from an artificial ventricular parasystole (with fixed coupling intervals) (lead II). Impulses of the ventricular pacemaker are indicated by dots in the diagram, the retrograde ventricular impulse by broken lines at V (ventricular) level.

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beat. Concealed backward propagation into the AV junction of these effective artificial stimuli is disclosed by a shift in the appearance time of the subsequent spontaneous junctional beat.

The parasystolic complexes in this case recur at a precisely fixed "coupling" interval after every fourth spontaneous beat. This is the case because (a) each effective artificial impulse discharges the natural pacemaker and shifts its timing by a constant interval, (b) the rates of the two pacemakers are regular, and (c) the speed of propagation of the two types of impulses is constant within the AV junction in both forward and retrograde directions. A coincidence of these three factors must result in a constant and repetitive coupling of artificial to spontaneous beats.

The records of figure 6 (all lead II) were obtained in a patient with advanced and unstable AV block. A record before artificial pacing, with AV conduction varying between 4:1 and 3:1 during sinus rhythm, is shown in panel a. When the ventricles are paced at a rate of 88/min. (panel b) each ventricular complex is followed, at a constant interval of 0.16 sec., by an inverted P wave (1:1 retrograde conduction). Sinus activity is completely suppressed and the entire heart is controlled by the artificial pacemaker. After reduction of the driving rate to 55/min. (panel c), sinus activity is restored and incomplete "reversed" AV dissociation has developed. All antegrade (sinus) impulses are blocked but artificial ventricular impulses capture the atria completely (-P) or partially (P_F) when they occur within a certain time interval after a sinus P wave.

On the basis of these facts, the following conclusions can be drawn in this case:

1. The block in the AV junction is unidirectional, permitting complete antegrade conduction only after long recovery times (panel a); this effect on antegrade conduction contrasts with the ease of retrograde conduction even at a relatively fast driving rate (panel b).

2. Proximal to this unidirectional block, there is a pathway common to, and used by, both antegrade and retrograde impulses. The state of recovery of this region following penetration (concealed conduction) of a sinus impulse determines whether a retrograde impulse will succeed or fail in reaching the atria.

Figure 6
Concealed antegrade conduction in unidirectional AV block; a before, b and c during transvenous ventricular pacing (all lead II). The shaded area in the diagram indicates a region of unidirectional conduction in the AV junction.
CONCEALED AV CONDUCTION

Electrode Implanted in the Left Ventricle

In figure 7 are reproduced parts of a long record in a patient after implantation of a permanent pacemaker. The driving rate of the ventricles is slower (68/min.) than the sinus rate (120/min.) and incomplete AV dissociation has developed, with partial captures of the atria by some of the artificial impulses (P₁, i.e., atrial fusion beats), and total capture of the ventricles by some of the sinus impulses; the latter occurs exclusively and predictably whenever a sinus impulse falls, within a certain limited time, shortly after an artificial ventricular beat.

The analysis of this type of complex mechanism is indicated in the diagram and has been described in detail previously. As in the preceding case, it requires the assumption of a region of unidirectional block (shaded area); in addition, however, a supernormal phase of conduction has to be postulated to develop in the wake of retrograde conduction across the region of unidirectional block. Such supernormality following concealed retrograde conduction then permits passage of an appropriately timed sinus impulse. Here, in contrast to the depressant effect illustrated in all preceding figures, concealed conduction acts to enhance subsequent conduction.

Discussion

While Lewis and Master in their classical experiments demonstrated that an apparently blocked impulse may delay or prevent conduction of a subsequent impulse, analysis of spontaneously occurring clinical arrhythmias has revealed a number of other manifestations of concealed AV conduction. Thus concealed conduction may (1) cause a repetition of concealed conduction and thus impair conduction of several subsequent impulses ("repetitive concealed conduction"); (2) disturb the regular action of a subsidiary pacemaker ("by concealed discharge"); (3) enhance, rather than inhibit, subsequent conduction by creation of a supernormal phase. These after effects of partially penetrating impulses—with the exception of the last one—have been confirmed, and their mechanism has been elucidated in detail, in recent animal experiments. In the course of our observations during artificial pacing, all known manifestations of concealed conduction could be reproduced in man by proper adjustment of the driving rate of the artificial pacemaker controlling atria or ventricles.

Antegrade concealed conduction in its simplest form was produced in a case of intermittent AV block, during a transient period of restored AV conductivity, by rapid stimulation of the atria (fig. 1) or by reduction of the atrial driving rate to less than the natural (sinus) rate (fig. 4). A scan of the junctional cycle by paired artificial atrial impulses (fig. 2) yielded values for the duration of the phase of concealed (antegrade) conduction corresponding to data found experimentally in the dog heart. Retrograde concealed conduction was clearly demonstrated in complete AV block during artificial stimulation of the ventricles at a rate slower than that of a spontaneous junctional pacemaker (fig. 5). Stimulation with faster rates has been shown

Figure 7

Concealed retrograde conduction of left ventricular pacemaker stimuli causing a supernormal phase for antegrade conduction of SA impulses in a unidirectional AV block (lead II). The first two pacemaker signals are retouched. (Courtesy of Dr. Samuel Goldfein, Chicago.)
to be followed by depression of impulse formation in, and conduction from, the junctional pacemaker. Furthermore, a well-known manifestation of concealed retrograde conduction was observed during artificial ventricular pacing in patients who have preserved or resumed AV conduction of sinus impulses. If, under such circumstances, the artificial beat becomes interpolated, the postectopic sinus beat may show prolongation of its P-R interval.

By varying the rate of ventricular pacing in an instance of AV block of high degree, a simple mechanism was readily changed to a complex one attributable to unidirectional block with an interplay of both antegrade and retrograde concealed conduction (fig. 6, b and c). Observations of this kind not only justify the assumption of concealed conduction of antegrade impulses in complete AV block but also provide evidence that unidirectional block is indeed the explanation of retrograde P waves under such circumstances, a concept still questioned by some investigators.

Facilitation of antegrade conduction in advanced AV block by a supernormal phase induced by concealed retrograde conduction was postulated to account for the exclusive occurrence of ventricular captures after an idioventricular beat. Actual occurrence of retrograde or fusion P waves, as illustrated in figure 7, supports the same assumption in cases in which such direct evidence of traverison of a region of unidirectional block by retrograde impulses is missing.

The distinction as to whether concealed conduction causes a total block of a subsequent impulse or its partial conduction (repetitive concealed conduction) cannot always be made. In the case of figure 3, penetration of impulse S following X₁ could not have become manifest without the presence of impulse X₂. Repetitive concealed conduction seems to play an important role in the ventricular response to rapid atrial rates, particularly atrial fibrillation and atrial flutter, and can be the mechanism of prolonged ventricular asystole in cases of second-degree AV block.

Finally, two ancillary aspects of our experience with pacing of atria and ventricles at varying rates merit comment, because of their bearing on our understanding of mechanisms and consequences of single premature beats. In figure 2 is illustrated an experimental model that permits an estimation of the atrial refractory period gauged by the length of the returning cycle after an atrial premature systole, comparable to conditions created by ventricular premature beats during idioventricular rhythms. Figure 5 demonstrates the fallacy of too strict separation of parasympathetic rhythms from coupled "extrasystoles."

**Summary**

Artificial pacing of patients with Stokes-Adams disease provided an opportunity to study experimentally the ways of operation of concealed antegrade or retrograde conduction, or both, in the AV junction.

With a catheter electrode in the right atrium the classical experiment of Lewis and Master was repeated, revealing, during a 2:1 ventricular response to an atrial tachycardia, the delaying effect of seemingly blocked atrial impulses on subsequent AV conduction.

Shifting the position of a single premature atrial impulse within a constant driving cycle of the atria produced graded effects of "blocked" atrial impulses on AV junctional refractoriness, permitting an estimation of the duration of the "phase of concealed AV conduction."

Interpolation of such premature atrial impulses into successive driving cycles resulted in "repetitive concealed conduction."

In an artificially produced atrial parasystole there was observed "concealed discharge" of a subsidiary (escaping) AV junctional pacemaker by an apparently nonconducted atrial impulse.

With a catheter electrode in the right ventricle in a case of advanced AV block, concealed retrograde conduction of pacer stimuli disturbed the rhythmicity of a spontaneous AV junctional pacemaker.

In a case of advanced AV block with pre-
served retrograde conduction (unidirectional block), evidence of penetration of the upper AV junction by the "blocked" antegrade impulse was found.

With electrodes implanted in the left ventricle in a case of advanced AV block, concealed retrograde conduction of the artificial pacemaker stimuli enhanced antegrade conduction by transiently changing an area of unidirectional block to one of supernormal conduction.

Thus, all known manifestations of concealed atrioventricular and ventriculo-atrial conduction, occurring spontaneously in clinical records or induced in animal experiments, were artificially reproduced in the human heart.

References
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