Concealed conduction in the atrioventricular (AV) junction in association with coordinated atrial activity is a well-recognized phenomenon, 1-3 recently confirmed with new technics in animal experiments. 4-6 Its operation in atrial fibrillation has received less attention, although it appears to offer the best explanation for the characteristic irregularity of the ventricular responses. 7-11 It is the purpose of this report based on clinical electrocardiograms to demonstrate evidence of concealed conduction in its various manifestations during atrial fibrillation; and to point out how concealed conduction in any type of rapid atrial activity may become a major factor which determines rate and sequence of ventricular responses.

The Equivalent of a Compensatory Pause after Ventricular Premature Systoles in the Presence of Atrial Fibrillation

As illustrated by figure 1 the cycles following each ectopic ventricular premature beat exceed the range of ventricular cycles in a series of consecutive conducted beats in lead III (when bigeminy transiently ceases). The long duration of the postectopic cycles, akin to truly compensatory pauses after premature beats during sinus rhythm, is to be attributed to prolonged refractoriness of the AV junction to antegrade conduction, consequent to its retrograde penetration (concealed VA conduction) by the ectopic ventricular impulse.

Variations of AV Nodal Escape Intervals in Atrial Fibrillation (Figures 2 and 3)

In figure 2, AV junctional—presumably nodal—escapes are indicated by the occurrence of several long cycles of practically equal duration (1.10 to 1.14 sec.) during an over-all irregular ventricular rate; however, in one instance the cycle is considerably longer (1.92 sec.). The cause for the failure or delay in the appearance of the nodal escape in this last instance can be readily accounted for by implicating concealed conduction of (at least) one of the fibrillation impulses up to the site of the nodal pacemaker, leading to a premature discharge of the latter. 12

In contrast to a similar situation when sinus rhythm with second-degree AV block is present, 2 the time at which the subsidiary pacemaker is reached by the penetrating atrial impulse can only rarely be established in atrial fibrillation. It depends on the possibility of identifying the origin of the ventricular complex that terminates the prolonged pause. Assuming that it is a postponed escape beat (as indicated in the upper diagram [a]), the time of resetting of the nodal pacemaker can be determined by subtracting the measurable nodal interval (here averaging 1.12 sec.) from the long interval of 1.92 sec. If, on the other hand, the beat under question were a conducted atrial impulse (as indicated in the lower diagram [b]), the time (and number) of concealed atrial impulses reaching the nodal pacemaker would be unknown. A distinction between these two alternatives is only possible when escape beats differ in shape from conducted ones, due to aberration of intraventricular conduction. 13

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Figure 3 shows a variant of this mechanism. The inherent (escape) rate of a subsidiary supraventricular pacemaker (E) is revealed in lead III by the two long ventricular intervals of equal duration (1.66 sec.). In lead I, the premature discharge of this subsidiary

![Figure 1](Image)

**Figure 1**

Patient with hypertensive heart disease, digitalized (70 year old). Atrial fibrillation with intermittent ventricular bigeminy. Concealed retrograde conduction of ventricular premature systoles causes long postectopic cycles.

![Figure 2](Image)

**Figure 2**

Patient with rheumatic heart disease, digitalized (54 year old). Atrial fibrillation with AV junctional escapes. The longest cycle is due to discharge of the subsidiary pacemaker by concealed (forward) AV conduction; the beat that terminates the long pause may be either a delayed escape (diagram a) or a conducted beat (diagram b). Discussed in text.

![Figure 3](Image)

**Figure 3**

Patient with arteriosclerotic heart disease and atrial fibrillation (70 year old). Idioventricular escape in lead I after prevention of AV junctional escape (E) by concealed AV conduction.
pacemaker by one (or more) penetrating atrial impulses leads to a prolonged ventricular pause of 1.98 sec., during which interval a slower idioventricular pacemaker escapes and takes over control of the ventricles for one beat.

Atrial Rate as a Factor Influencing Concealed AV Conduction (Figures 4-6)

The ventricular response—whether regular or irregular—during atrial flutter and other ectopic atrial tachycardias is more readily accessible to analysis than that associated with atrial fibrillation. The operation of concealed AV conduction has been established in atrial flutter. Clinical experience has shown that it is more difficult to slow the ventricular rate in patients with atrial flutter than in patients with atrial fibrillation. High, or even toxic doses of digitalis may be required to depress AV conduction beyond a 2:1 response unless some degree of AV block existed before the onset of the atrial arrhythmia. Figures 4 and 5 illustrate instances of paroxysmal atrial flutter, one occurring during chronic atrial fibrillation in a digitalized patient, the other during sinus rhythm with a second-degree AV block caused by myocardial infarction. In the former (fig. 4), the decrease in atrial rate (during flutter) is associated with a speeding up of the ventricular rate; contrariwise, in the latter (fig. 5), the increase in atrial rate is associated with slowing of the ventricular rate. It would appear that in the first instance, the existing concealed conduction is eliminated, and in the second case concealed conduction is induced by the temporary change of the atrial mechanism to flutter.

Figure 6 shows the same phenomenon in a paroxysm of atrial tachycardia or atrial flutter which occurred during artificial pacing of a patient via a catheter electrode in the right atrium. After 1:1 AV response was established at a rate of 115, four stimuli were applied to the atrium in rapid succession to study the phenomenon of repetitive concealed conduction. When atrial flutter developed on one occasion, the ventricular rate slowed abruptly to about one half its previous rate. This reduction in rate is clearly the result of repetitive concealed conduction associated with the induced rapid atrial activity.

Distribution of Long and Short Ventricular Cycles in Atrial Fibrillation

In a recent experimental study of the ventricular response in atrial fibrillation Moe and Abildskov postulated that a long ventricular cycle—containing one or more responses concealed in the AV node—would favor the occurrence of a subsequent long cycle if concealed conduction were a major factor in

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**Figure 4**

Patient with rheumatic heart disease, fully digitalized (45 year old). Records A and B (both lead II) were obtained in short succession. Note acceleration of the ventricular rate from 68 to 155 as atrial fibrillation (A) changes to flutter (B). Less rapid stimulation of the AV junction reduces the chance for concealed conduction and permits more atrial impulses—every second or third—to reach the ventricles.
Patient with recent posterior (and atrial) infarction (67 year old). The three portions of lead III are continuous. In leads I and II, during sinus rhythm, AV conduction varies between 2:1 and 3:2; in lead I, the 3:2 response is incomplete (concealed), resulting in a manifest 3:1 ventricular response (compare the two diagrams). The average ventricular rate during this time is 57. In lead III, atrial flutter (rate 250) has developed and continues up to the arrow where sinus rhythm is resumed. During the flutter episode, the ventricular action has slowed to an average rate of 33. Stimulation of the severely depressed AV junction by impulses occurring in rapid succession causes repetitive concealed conduction and thus a reduction of ventricular responses. Discussed in text.

determining the pattern of the ventricular response. The distribution of long cycles was found to occur as predicted, and Moe and Abildskov concluded that the ventricular dysrhythmia associated with atrial fibrillation can indeed be interpreted in terms of concealed conduction. The lower record of figure 7 reveals such a tendency to repetition of “long” ventricular cycles in a digitalized patient with atrial fibrillation. The mechanism is explained, with the help of a diagram, in the upper tracing in which a similar phenomenon was observed in a patient with a paroxysm of regular atrial tachycardia. In this case, the question then may be raised as to why the effect of the lengthened ventricular cycle initiated by concealed conduction of an atrial impulse does not continue beyond the second long cycle. The explanation, it would appear, depends on the assumption that penetration of the “blocked” impulse during the second long cycle is deeper than in the first, deep enough to shorten the refractory period in distal parts of the AV junction, and to prevent, thereby, the occurrence of another concealed conduction after the next atrial impulse. Similarly, in many clinical cases of atrial fibrillation the occurrence of single long cycles preceded and followed by a series of short ones could be accounted for by deep penetration—almost
VENTRICULAR RESPONSE IN ATRIAL FIBRILLATION

Eighty-two-year-old patient with intermittent AV block. Accidentally induced episode of atrial tachycardia and flutter during artificial pacing following application of four atrial stimuli in rapid succession. With increase of the atrial rate from 115 to 230 the ventricular rate decreases from 115 to an average of 60 as a result of repetitive concealed conduction.

The upper record is lead III of a 53-year-old patient with mitral stenosis and ectopic atrial tachycardia with irregular ventricular response; Wenckebach periods (8:7 to 4:3) alternate with 2:1 responses. Thus, there are periods of rapid ventricular beating separated by two longer cycles. Typically the P-R of the last beat of a period is longer than the P-R of the first beat after the intermittence. This abrupt reduction in AV conduction time (a) causes foreshortening of the first of the pair of the longer R-R intervals and (b) permits deep penetration into the lower AV junction of the “blocked” atrial impulse during the second long ventricular cycle. Consequently, the duration of the AV junctional cycle, and with it junctional refractoriness, shorten so that, instead of another 2:1 response, a long Wenckebach period can follow. The lower tracing shows comparable events during atrial fibrillation in a 77-year-old patient with arteriosclerotic heart disease. Note that the longer ventricular cycles are followed by another long cycle, presumably because they contain partial (concealed) responses of the AV junction. Discussed in text.

**Discussion**

The ventricular response to any type of rapid atrial activity is determined by the refractory period of the AV junction. In fibrillation a regular response of the ventricles might be expected, since an atrial impulse is always and immediately available at the termination of the refractory period of the junctional tissues; the rate of ventricular response would be determined by the constant duration of the junctional refractory period in successive beats. However, the duration of the refractory period will be altered, and the regularity of the ventricular rate disturbed, if any of the atrial impulses penetrate into the AV junction without reaching the ventricles. Such concealed conduction of any impulses will affect conduction of the subsequent impulse by delaying it, blocking it entirely, or causing “repetitive concealed conduction.”

Concealed propagation of the atrial impulse may occur during various portions of the heart cycle and may reach different levels within the AV junction. Various depth of penetration will, in turn, result in a disparity of the refractory period duration at several levels of the AV junction. In the distal portions (not reached by the impulse) where
the junctional cycle lengthens, there will be a prolongation of the refractory period: in the more proximal portions where, as a result of concealed conduction, the cycle and refractory period shorten, there will be better responsiveness and a longer phase of concealed conduction.11 These divergent effects at different AV junctional levels will favor "secondary concealment"11 and repetition of long ventricular cycles. The probability of concealment becoming manifest in the form of long ventricular cycles was shown to be directly proportional to the duration of the concealment zone and inversely proportional to the mean cycle length of the atrial input. Since vagal stimulation increases the duration of the concealment zone11 and shortens the atrial refractory period, the vagal effects of digitalization favor concealed conduction and contribute, in this manner, to ventricular slowing. Quinidine, on the other hand, may exert its "atropine like effects" on the AV junction by slowing atrial input, thereby reducing the chances of concealed conduction and accelerating the ventricular frequency.

Depth of concealed conduction within the AV junction may not be the only factor determining the response to a subsequent impulse. Particularly in the case of repetitive concealment, the speed at which successive impulses partially invade the AV junction may decline progressively, and such a Wenckebach type of concealed conduction2 may set the limit to the number of impulses consecutively involved in the repetition of concealment. Finally, a supernormal phase of conduction may also come into play and affect both frequency and regularity of the ventricular response in an unpredictable way.3 Whereas complete AV conduction during the supernormal phase would increase the ventricular rate, concealed conduction would decrease it. The complexity of the interplay of all these influences on AV transmission is reflected in the over-all irregularity of the ventricular response.

Summary

The ventricular response in atrial fibrillation is determined by the long refractory period of the AV junction. Since an atrial impulse is always available for transmission to the ventricles, a regular ventricular rhythm would be expected, the rate of which would reflect the duration of a stable junctional refractory phase. The irregularity of the ventricular action associated with atrial fibrillation, therefore, indicates changes of refractoriness of the AV junctional tissues from cycle to cycle. This can best be attributed to varying degrees of penetration of "blocked" atrial impulses into parts of the AV junction, and to the effect of such concealed conduction on the propagation of subsequent impulses.

The following facts are pointed out and illustrated as evidence of concealed AV and VA conduction during atrial fibrillation: (a) Occurrence of a "compensatory pause" following a ventricular premature systole. (b) Failure of an AV nodal escape to appear at the expected time due to concealed discharge of a subsidiary AV nodal pacemaker by a penetrating atrial impulse. (c) Acceleration of the ventricular rate when atrial fibrillation changes to flutter (elimination of concealed conduction with slowing of the atria). (d) A tendency for two or more long cycles to occur in succession (each containing one or more concealed responses).

The reasons for the variations of the refractory period at different levels of the AV junction as a result of concealed conduction are analyzed. Their complex interplay may readily account for the over-all irregularity of the ventricular response associated with atrial fibrillation.

References


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