Concealed Conduction

By Brian F. Hoffman, M.D., Paul F. Cranefield, Ph.D.,
and Jackson H. Stuckey, M.D.

The concept of concealed conduction was introduced into electrocardiography to explain a lengthening of, or change in, the refractory period of the atrioventricular conduction system caused either by atrial activity that fails to propagate to the ventricles or by ventricular activity that fails to reach the atria. Standard electrocardiograms do not provide a direct demonstration of the partial invasion of the atrioventricular conducting system under such conditions and usually fail to indicate the mechanism or exact site of block. Studies of the electrical activity of single cardiac fibers have shown that a local conduction delay or complete failure of propagation can occur at the junction of two fibers whose action potentials differ in duration. Such disturbances of conduction are observed whenever the propagation of excitation in one fiber precedes complete repolarization of another adjacent fiber. Records of the transmembrane action potentials of single cardiac fibers have shown that rather marked differences in action-potential duration are present at the peripheral junction of Purkinje fibers with papillary muscle and at the junction of fibers of the atrioventricular node and bundle of His. Less abrupt differences in action-potential duration are encountered within the atrioventricular node and within the peripheral Purkinje system. These observations suggest that failure of atrioventricular transmission might take place at any point between the atria and the ventricles because of local differences in the duration of electrical activity. This proposition has been studied by recording the transmembrane potentials of single fibers in isolated preparations of rabbit and dog heart and by recording directly from the specialized conducting system of the in situ dog heart during total cardiopulmonary bypass.

Methods

Isolated Preparations

Preparations of rabbit heart containing the right atrium, the atrioventricular node, and the bundle of His were perfused at 37°C with a modified Tyrode solution. For some experiments this preparation also included the upper part of the interventricular septum. Transmembrane potentials were recorded in the usual manner through intracellular microelectrodes filled with 3 M KCl. In some experiments unipolar or bipolar electrograms were recorded at the same time as the transmembrane potentials. Electrograms were recorded through fine silver electrodes insulated except at the tip and mounted in conventional micromanipulators. Stimulation was applied to the right atrial appendage through a fine steel-pin electrode. Records from single fibers of the atrioventricular node were identified by criteria described previously. Isolated preparations of the right bundle branch and Purkinje-fiber-papillary-muscle junction of the dog heart were studied in the same manner as preparations of rabbit heart.

In Situ Hearts

The methods employed for these experiments have been described in detail. During total cardiopulmonary bypass, multiple small bipolar recording electrodes were attached over the bundle of His, the bundle branches, and the peripheral junctions of Purkinje fibers with papillary muscles. Similar electrodes were attached to the right atrium and right and left ventricles and used to record electrical activity or to apply driving and testing stimuli. Electrical activity of Purkinje fibers located in the false tendons was recorded through electrodes contained in a probe that was held in light contact with these structures. Low-frequency components of the tracings were filtered to increase baseline stability and facilitate identification of electrical activity of specialized tissues. Arterial blood pressure was recorded continuously and body and heart temperatures were measured at frequent intervals.

From the Departments of Physiology and Surgery, State University of New York, Downstate Medical Center, Brooklyn, New York.

Supported in part by grants from the American Heart Association and by Grants H-3316 and H-3916 from the U. S. Public Health Service.

Received for publication September 14, 1960.
Mean blood pressure was maintained at 100 mm Hg or above by pump flows of 80 to 90 ml/Kg/min.; heart temperatures are indicated for each illustration.

Results

Concealed Conduction Within the Atrioventricular Node

Propagation of Premature Beats

Typical transmembrane action potentials recorded from single fibers in the atrium, atrioventricular node, and bundle of His of the rabbit heart are shown in figure 1. A comparison of the duration of action potentials in the atrium and node (fig. 1A) shows that, in spite of the slow conduction within the node, repolarization of the nodal fiber precedes the terminal slow repolarization of the atrial fiber. In contrast, a marked difference in duration is present between action potentials recorded from the atrioventricular node and from the bundle of His (fig. 1B). The differences in action-potential duration shown in these records would suggest that a block of premature atrial activity might take place at the junction between the lower node and the bundle of His. However, if an appropriately timed electrical stimulus is used to produce an atrial extrasystole that, because of its prematurity, fails to propagate to the bundle of His, records of activity within the node usually show failure of transmission between atrial muscle and the site of the nodal electrode (fig. 1C). This failure of transmission presumably results from rapid decrement of conduction in nodal fibers at the atrial margin of this structure, the small depolarization recorded from the nodal fiber after the atrial extrasystole (fig. 1C) and failure of the subsequent atrial action potential to propagate normally will be discussed later.

A somewhat different type of block of premature atrial beats has been observed in several experiments. In these, there was reason to suppose that the preparation was in poor condition, since both the amplitude and maximum rising velocity of action potentials recorded from the bundle of His were decreased. Records from one experiment of this sort are shown in figure 2. It can be seen that an atrial extrasystole, which arises sufficiently late after the preceding nodal response, propagates through the node to the bundle of His with only a moderate increase in delay (fig. 2A); the slowing of nodal transmission shown in the figure results at least in part from the noticeable decrease in the rate of depolarization of the nodal action potential. This change in the rising velocity of the
Isolated preparation of rabbit heart. Transmembrane action potentials recorded from fibers in the atrioventricular node (second trace) and the upper bundle of His (bottom trace), and a bipolar electrogram recorded from the atrium (middle trace). Top trace shows time marks at intervals of 50 and 500 msec. Arrows indicate time of application of stimuli to the atrium; the atrial electrogram shows first a stimulus artifact and then the electrogram complex. Changes in transmission of the atrial extrasystoles are described in the text.

The nodal response is quite marked, even though local activation follows long after repolarization of the preceding action potential.

If the atrial extrasystole arises somewhat earlier in the cycle (fig. 2B), propagation to the bundle of His is markedly delayed. Also, the appearance of the nodal response is quite different. Even though the level of the membrane potential is similar to that preceding normal action potentials, the upstroke of the nodal action potential arises from a slow, steplike depolarization. This step or prepotential accounts for most of the abnormal increase in conduction time from the atrium to the bundle of His. When the atrial action potential is slightly more premature (fig. 2C), concealed conduction within the node is apparent. As in figure 2B, the steplike prepotential does elicit a nodal action potential. However, even though this nodal action potential is of comparable amplitude to that in figure 2B, excitation of the fibers of the bundle of His does not take place. This is true even though the bundle of His fiber is normally polarized; thus failure of transmission again does not result solely from local difference in action-potential duration. The records in figure 2 also show that electrical activity within the node during concealed conduction may last longer (420 msec.) than that caused by premature beats which do propagate to the bundle of His (400 msec.) or than that following normal atrial activity (350 msec.) (figs. 2A, B, and C). Some indication of the cause of prolonged refractoriness is suggested by these same records; although the abortive nodal action potential in figure 2C is of less than normal duration, as would be expected, it is preceded by a slow, steplike depolarization lasting slightly more than 250 msec. The probable mechanism for the slow step-like depolarization is discussed later in relation to the changes in nodal activity seen during atrial tachycardia.

Effects of High Atrial Rate on Nodal Transmission

The records shown in figure 3 were obtained from single fibers of atrium and atrioventricular node at three different rates of atrial stimulation. In this experiment the major change associated with increased frequency of contraction is a progressive decrease in the rate of depolarization and the amplitude of the nodal action potentials. A less marked but similar change in the atrial action potentials also is seen at the higher rates. The augmented delay in activation of the nodal fibers and thus, presumably, of fibers in the bundle of His can be accounted for on the basis of the observed changes in the voltage-time course of the nodal action potentials. In other experiments, again when the preparation was some-
what depressed, the effect of increased atrial rate was quite different. As can be seen in figure 4, at a moderately high atrial rate the nodal action potentials arise out of a slow, steplike depolarization similar to that shown in figure 2. This slow depolarization increases in duration and ultimately fails to elicit a nodal action potential of the usual amplitude (arrow). This nodal response fails to excite the bundle of His and thus constitutes another example of conduction concealed within the atrioventricular node.

Some indication of the mechanisms underlying the changes in nodal transmission associated with the appearance of the slow, steplike depolarization can be obtained from the records in figure 5. In this experiment one intracellular microelectrode was kept in the same atrial fiber and another was used to record from a series of fibers located at different points between the atrial margin of the atrioventricular node and the nodal end of the bundle of His. During sustained atrial tachycardia, transmembrane potentials recorded from a nodal fiber at the atrial margin of the node show that each atrial potential is followed by a nodal response of constant amplitude and duration (fig. 5A). When the recording electrode is located in a nodal fiber somewhat closer to the bundle of His (fig. 5B), small, short-duration action potentials alternate with larger responses of greater duration. The small responses originate prior to the end of repolarization of the preceding action potential. Still closer to the bundle of His (fig. 5C), records of activity show additional changes. The larger responses exhibit a decreased rising velocity and reduced amplitude and a distinct notch on the repolarization limb. The small responses are greatly diminished in amplitude but largely unchanged in duration. The records in figure 5D, obtained through an electrode inserted in a fiber of the bundle of His, show a regular 2:1 response pattern; at this level there is no indication of the alternate "dropped" beats.

Experiments of this sort provide a clear demonstration of the spatial decrement of alternate nodal responses. Also, this decre-
mental conduction is the cause of the concealed conduction that occurs. In this instance decrement seems to result at least in part from excitation reaching fibers that are not yet fully repolarized. The alternate failure of excitation to reach the bundle of HIs is due to concealed conduction within the node. The great increase in nodal delay of action potentials that do excite the bundle of HIs results from the changes in shape and amplitude of the propagated nodal action potentials. The notch on the descending limb of the larger nodal responses in figure 5C most likely results from electronic spread of depolarization from fibers between the recording site and the bundle of HIs.

Discussion

During premature atrial excitation or during atrial tachycardia, records of the electrical activity of single fibers of the atrioventricular node show clear evidence of concealed conduction extending for various distances into the node. During 2:1 block between the atrium and the bundle of HIs caused by high atrial rates, decremental conduction of the dropped beats can be demonstrated directly. Records of the transmembrane potential of nodal fibers suggest that, following the dropped beats, the duration of refractoriness of the nodal fibers is less than after normal propagation. The slowing of the transmission of excitation from the atrium to the bundle of HIs during this arrhythmia results from changes in the nodal action potentials, and these are the changes that would be expected during decremental conduction.

In depressed preparations, concealed conduction has been observed in association with nodal action potentials that arise from a long, slow, steplike depolarization or prepotential. It is thought that these slow depolarizations result primarily from decremental conduction in fibers located between the atrial margin of the node and the recording site. A possible contributing factor is a lowered threshold potential in fibers at the recording site. It is clear, however, that the prolonged, slow depolarization will, of itself, cause decrement of conduction by increasing inactivation in fibers showing this change in membrane potential. When concealed conduction is associated with this particular change in membrane potential, the duration of refractoriness at the site of block may be greater than during normal transmission. Also, the appearance of a slow depolarization of this type, in response to a slight increase in the prematurity of atrial excitation, will produce an abrupt (fig. 2B) and rapidly increasing (fig. 2C) delay in nodal transmission. This response of nodal fibers should be considered in relation to evidence presented for the existence of a dual atrioventricular conduction system. 12

Concealed Conduction Distal to the Atrioventricular Node

Figure 6 shows transmembrane action potentials recorded simultaneously from a single fiber in the bundle of His (top trace) and from another fiber in the right bundle branch of the rabbit heart. Records of this sort show that there is an increase in action-potential duration not only between the lower node and the bundle of HIs (see fig. 1B) but also between the His bundle and the bundle branches. A similar difference in action-potential duration is observed in preparations obtained from dog heart. The records...
in figure 7A were obtained from the isolated right bundle branch and peripheral Purkinje-fiber-papillary-muscle junction from dog right ventricle. The action potential recorded close to the origin of the right bundle branch is considerably shorter in duration than that recorded from the peripheral Purkinje fiber. These records show that, in both rabbit and dog heart, action-potential duration increases throughout a large part of the conducting system. Should a premature atrial action potential propagate through the atrioventricular node, it might thus encounter partially or completely refractory fibers in the His bundle, the bundle branches, or the peripheral Purkinje fibers. Evidence that premature atrial activity can reach the bundle of His prior to the end of repolarization of fibers in the common bundle has been obtained from records of the transmembrane potential of single fibers (fig. 7B). The demonstration of concealed conduction and block within the bundle branches and the peripheral Purkinje system is presented below.

Atrioventricular Block Distal to the Bundle of His

Figure 8 shows bipolar electrograms recorded from the right atrium, the His bundle, and the junction of false tendon with papillary muscle of the in situ dog heart. These records were obtained during total cardio-pulmonary bypass using a pump oxygenator. Identification of the various complexes has been described elsewhere. Atrial extrasystoles were induced at various intervals of

---

Circulation Research, Volume IX, January 1961

---

**Figure 7**

(A) Isolated preparation of dog heart. (Top trace), time marks at intervals of 100 and 500 msec.; (second trace), transmembrane action potential recorded from a fiber in the right bundle branch; (bottom trace), transmembrane action potential recorded from a peripheral Purkinje fiber close to the junction with the anterior papillary muscle of the right ventricle. Vertical bar represents 100 mv. (B) Isolated preparation of rabbit heart. Transmembrane action potentials recorded from the bundle of His (upper trace) and atrioventricular node (middle trace), and a bipolar atrial electrogram (bottom trace). Note that the premature atrial response propagates through the atrioventricular node and excites the bundle of His fiber before it is completely repolarized.

**Figure 8**

In situ dog heart. Bipolar electrograms recorded from the right atrium (top trace), from the region of the bundle of His (second trace), from the Purkinje-fiber-papillary-muscle junction (third trace), and from the epicardial surface of the ventricle (bottom trace). The bundle of His electrogram shows three complexes in the first cycle: the first (a) represents local atrial activity, the second (h) shows activity in the bundle of His, and the third (v) results from activity in the ventricles. The record from the Purkinje-fiber-papillary-muscle junction shows, first, the complex resulting from activity in the Purkinje fibers and, second, the complex resulting from activity in the muscle. Note that the premature atrial complex (E) propagates through the node and elicits a normal action potential from the bundle of His (arrow), but that electrodes located distal to the bundle of His show no propagated activity. Time lines at intervals of 40 msec.
Hoffman, Cranefield, Stuckey

In situ dog heart. Bipolar electrograms recorded from the junction of Purkinje fibers with papillary muscle (top trace), the left bundle branch (second trace), the bundle of His (third trace), and the right atrium (bottom trace). The top trace shows discrete deflections resulting from electrical activity of the Purkinje fibers (p) and papillary muscle; the second trace shows activity of the left bundle (s) and the underlying septal musculature. The third trace shows three complexes; the first results from activity in the atrium (a), the second from activity in the bundle of His (h), and the third from ventricular depolarization. Note the changes in transmission of the atrial extrasystoles (e). See text for description. Time lines are at intervals of 40 msec.

The cardiac cycle by short electrical stimuli applied to the right atrium. During most of the cycle, such extrasystoles either were blocked at the atrioventricular node or propagated throughout the specialized conducting system and reached the ventricles. However, with proper timing of the atrial extrasystole (E), it was possible to demonstrate concealed conduction extending through the entire atrioventricular node and into the bundle of His (arrow) and a complete failure of transmission from the common bundle to the peripheral Purkinje fibers and ventricles. The time required for the extrasystole to traverse the atrioventricular node was appreciably greater than that recorded during normal atrial activity. The bipolar electrogram recorded from the His bundle shows no appreciable change in amplitude, contour, or duration and thus suggests that, at this particular recording site, the extrasystole encountered fully excitable fibers. These records do not demonstrate the exact site of block; however, since there is no sign of electrical activity at the Purkinje-fiber-papillary-muscle junction, it may be assumed that propagation failed either between the bundle of His and bundle branches or within the latter subdivisions of the atrioventricular conducting system.

In several experiments bipolar electrograms were recorded from a greater number of locations within the dog heart, and the actual site of block was localized more exactly. The records shown in figure 9 were obtained from the right atrial appendage, the bundle of His, the left bundle branch, and the peripheral junction of Purkinje fibers with the anterior papillary muscle of the left ventricle. When a stimulus was applied to the right atrium 220 msec. after the preceding response (fig. 9A), the resulting extrasystole (e) was delayed during transmission through the atrioventricular node, as shown by the increased interval between the local atrial activity and the response of the bundle of His. The electrogram recorded from the bundle of His appears normal in amplitude and configuration. However, conduction in the left bundle branch is delayed. This is shown by the widening and change in configuration of the local electrogram and also by the delayed activation of the peripheral Purkinje fiber.

When the atrial extrasystole was initiated 20 msec. earlier in the cycle (fig. 9B), the atrioventricular delay was somewhat increased. Also, the slight decrease in amplitude of the bundle of His complex indicates that excitation has reached this point before completion of recovery of excitability. Premature excitation of the bundle branch by this extrasystole elicited only a small local

Circulation Research, Volume IX, January 1961
CONCEALED CONDUCTION

electrogram complex. Since there is no indication of activity in the peripheral Purkinje fibers and since the local ventricular excitation recorded on the upper two traces is considerably delayed, the records strongly suggest a complete failure of conduction between the bundle branch and peripheral Purkinje fibers. A similar sequence of events follows the postextrasystolic atrial complex. In this cycle the electrograms recorded both from the bundle of His and the bundle branch are more nearly normal in appearance than those in the immediately preceding cycle. This probably results in part from the effect of cycle length on refractory period as well as from the greater time allowed for recovery from the preceding excitation. In this instance concealed conduction clearly extends through the bundle branch, and block occurs in the distal Purkinje fibers.

The records shown in figures 8 and 9 suggest that, in the in situ dog heart, the duration of refractoriness increases progressively from the bundle of His to bundle branch and from bundle branch to Purkinje fibers close to their junction with ventricular muscle. This observation corresponds to the duration of the transmembrane action potentials recorded from single fibers at these same sites in preparations isolated from dog (fig. 7A) and rabbit hearts (fig. 6). One additional finding is in agreement with this conclusion. When the atrium of the dog heart is driven at progressively higher frequencies and electrograms are recorded from the bundle of His and the peripheral Purkinje fibers (fig. 10), the expected progressive increase in atrioventricular delay is shown by the increased interval between activity in the atrium and the bundle of His. In addition, although the bundle of His electrogram shows no change in amplitude or configuration at the rapid rate, that recorded from the peripheral Purkinje fibers is markedly changed. The increased duration of this latter complex, the decreased amplitude, and the polyphasic appearance all suggest that conduction is slower and less synchronous than at lower frequencies. Records of this sort would be expected if the action-potential duration were greater near the Purkinje-fiber-papillary-muscle junction than in the bundle of His.

Discussion

In isolated preparations, records of the transmembrane action potentials of single fibers located in the atrioventricular node, the bundle of His and the bundle branches, and records from single Purkinje fibers located close to the Purkinje-fiber-papillary-muscle junction show that action-potential...
duration increases progressively throughout this part of the specialized atrioventricular conducting system. Also, simultaneous records from the atrium, atrioventricular node, and the bundle of His show that premature atrial activity can, at least under the conditions employed, spread through the atrioventricular node and excite fibers in the bundle of His before they are fully repolarized. Electrograms recorded from the heart in situ show that transmission of premature atrial activity can fail at various points within the specialized conducting system distal to the atrioventricular node and that concealed conduction of such atrial activity may extend as far as the peripheral Purkinje fibers. The results obtained strongly suggest that the slowed conduction and block result directly from the fact that action-potential duration increases from the lower part of the atrioventricular node throughout a major part of the peripheral Purkinje system.

There are certain similarities between the conduction disturbances recorded in these experiments from fibers within the atrioventricular node and from the distal parts of the conducting system. Within the node, failure of transmission does not necessarily result solely from differences in action-potential duration. In many instances excitation fails even though the membrane is fully repolarized. This type of failure of transmission has been termed "decremental conduction" and is thought to result from the anatomical and electrophysiological properties of the fibers at the atrial end of the node.\(^4\) In the bundle of His, the bundle branches, and peripheral Purkinje fibers, failure of conduction of premature activity appears to occur when excitation of one part precedes repolarization of more distal segments. It thus depends directly on local differences in action-potential duration. However, it has been shown that a lowered membrane potential decreases the rising velocity and amplitude of the action potential.\(^11\) It also may lower the threshold potential and thus decrease excitability. These changes make excitation of adjacent fibers less effective, and thus this type of conduction disturbance may also be considered an example of decremental conduction. It is likely that local conduction delay and local block differ only in one respect: in the latter, decrement progresses until excitation fails, while in the former the local slowing of conduction is sufficient to allow more distal fibers to repolarize fully.

**Summary**

The existence of concealed conduction of atrial beats has been demonstrated. Concealed conduction within the atrioventricular node has been studied by recording the transmembrane potential of single nodal fibers. Conduction block in this part of the specialized conducting system depends more upon decremental conduction within certain nodal fibers than upon differences in action-potential duration. Also, the extreme delay of activation that results from such decremental conduction explains certain changes in atrioventricular delay associated with premature atrial beats as well as a prolonged refractoriness of atrioventricular transmission after concealed nodal conduction of an atrial extrasystole.

In the in situ dog heart, local bipolar electrograms recorded from various parts of the bundle of His, bundle branches, and peripheral Purkinje fibers have shown that concealed conduction may extend through the atrioventricular node and the bundle of His and that block may occur between common bundle and bundle branches or between the latter and the peripheral Purkinje fiber. Block in these locations, which results from local differences in the action-potential duration, has been compared to decremental conduction within the atrioventricular node.

**References**


Concealed Conduction
BRIAN F. HOFFMAN, PAUL F. CRANEFIELD and JACKSON H. STUCKEY

Circ Res. 1961;9:194-203
doi: 10.1161/01.RES.9.1.194

Circulation Research is published by the American Heart Association, 7272 Greenville Avenue, Dallas, TX 75231
Copyright © 1961 American Heart Association, Inc. All rights reserved.
Print ISSN: 0009-7330. Online ISSN: 1524-4571

The online version of this article, along with updated information and services, is located on the World Wide Web at:
http://circres.ahajournals.org/content/9/1/194