Observations on Concealed Conduction in Atrial Fibrillation

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ABSTRACT

The mechanism of the ventricular dysrhythmia associated with atrial fibrillation in isolated rabbit heart was studied by recording electrograms from right atrium and ventricle simultaneously with transmembrane action potentials recorded from single fibers within the A-V node and right bundle branch. In most experiments, the variation in ventricular cycle length during experimentally initiated atrial fibrillation resulted from concealed conduction within the A-V node. The most frequently recorded ventricular interval during atrial fibrillation was found when a single response was concealed within the A-V node. In several experiments, as many as seven to nine consecutively concealed responses were recorded from the A-V node. When repetitive A-V nodal concealment occurred, the resulting ventricular cycle lengths were prolonged accordingly. In only a few experiments were conduction delay and block observed within the ventricular specialized conduction system (VSCS) during atrial fibrillation. Subsidiary pacemakers were never observed within the A-V node but were noted occasionally within the VSCS following epinephrine administration. Multiple A-V nodal concealments were usually associated with rapid atrial rates.

ADDITIONAL KEY WORDS ventricular dysrhythmia cardiac potentials A-V nodal action potentials A-V block A-V conduction delays aberrant QRS complexes rabbit

The elaborate experimental studies of Moe and Abildskov on atrial fibrillation demonstrated that the ventricular dysrhythmia associated with atrial fibrillation can be interpreted as being due to concealed conduction within the atrioventricular (A-V) node (1). This finding supports previous clinical impressions of the importance of concealed A-V nodal conduction in atrial fibrillation. The occurrence of aberrant QRS complexes in some clinical cases of atrial fibrillation in dogs (2) and man (3) suggests that delays and block of atrial impulses also may occur within the ventricular specialized conducting system (VSCS). By careful analysis of clinical records of atrial fibrillation, Langendorf, Pick, and Katz (4) found evidence that subsidiary pacemakers within the A-V node may be responsible in some cases for variations in the irregular ventricular rhythm that occurs during atrial fibrillation. Recently, computer analysis of electrocardiograms of patients with atrial fibrillation suggested that A-V nodal pacemaking with entrance and exit block accounts entirely for the irregular ventricular rhythm observed in atrial fibrillation (5). In the present investigations, microelectrodes were used to record from single cells within the A-V node and VSCS during experimentally initiated atrial fibrillation. Previously reported studies on the mechanism of the ventricular dysrhythmia in atrial fibrillation did not employ methods for directly recording from the A-V node and VSCS. The present report confirms the importance of concealed conduction within the A-V node in governing the ventricular irregularity observed...
during atrial fibrillation. Evidence is also presented that delays and block can occur within the VSCS in some cases of atrial fibrillation, and could account for aberration of QRS complexes. Subsidiary A-V nodal pacemakers, however, were never observed.

**Methods**

Rabbits weighing 1 to 2 kg were anesthetized intravenously with pentobarbital sodium (30 mg/kg) and the hearts were rapidly removed and placed in oxygenated Tyrode's solution. A right heart preparation containing the right atrioventricular conduction system was prepared as described previously (6). In this preparation the left VSCS is not functional. The temperature of the perfusing solution was maintained at 37°C. Conventional microelectrodes were employed to record from single cardiac cells using unneutralized cathode followers. Atrial and ventricular electrograms were recorded using Grass P5 amplifiers. Signals were displayed on a Tektronix 565 oscilloscope and were photographed with a Grass C4 camera. Experimental atrial fibrillation was initiated by stimulating the right atrium at frequencies of 20 to 50/sec using bipolar silver stimulating electrodes. Stimuli were RA Al A2 A3 A4 AVN RBB RPPJ T

**Figure 1**

RA = right atrial electrogram; AVN = A-V nodal transmembrane potentials; RBB = right bundle branch transmembrane potentials; RPPJ = right Purkinje-papillary muscle junction electrogram; T = time intervals of 10 msec. Vertical bar = 100 mv. A1 was conducted over the A-V transmission system to the ventricle. A2, A3 and A4 were blocked within the A-V transmission system. No subsidiary pacemakers developed during the absence of stimulation. Rapid deflections retouched.

2 to 5 times threshold strength. In some experiments, the effect of a concealed atrial response on conduction of a subsequent response and the development of subsidiary pacemakers was determined by driving the heart at a basic cycle length and then evoking premature atrial responses at various intervals following every sixth or later basic response.

**Discussion of Results**

In clinical bouts of atrial fibrillation, aberrant ventricular complexes most often follow the QRS that terminates a long cycle. It is usually thought that they result from either aberrant A-V conduction or from a subsidiary pacemaker within the ventricles or the A-V node. Figure 1 shows the results of an attempt to assess the frequency of development of subsidiary pacemakers within the A-V node and VSCS following concealment of several premature atrial responses. The first atrial response (A1) was transmitted through the A-V node (AVN) over the His bundle (not shown) to the right bundle branch (RBB) through the Purkinje system and to the right ventricle (RPPJ). The first complex in the RPPJ recording is the Purkinje electrogram, which is followed by the smaller downward-directed right ventricular complex. Following the conducted A1 response, three premature atrial responses, A2, A3, and A4, were evoked; all three failed to be transmitted to the ventricles. The A2 premature response was conducted through the A-V node, as demonstrated by the all-or-none A-V node action potential; however, complete block of A2 subsequently occurred between the His bundle and the right bundle branch as reported previously (6). The A3 and A4 premature atrial responses were both concealed within the A-V node. Block of A3 and A4 occurred at different levels in the A-V node, as indicated by the different amplitudes of nonpropagated A-V node local responses. When A4 was evoked earlier than in this figure, block occurred within in the atrium or upper A-V node. After the A4 response, a period of 400 msec or longer was maintained without electrical stimulation to see if subsidiary pacemakers within the A-V node or VSCS developed. None did in

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this experiment nor in any experiment in which drugs were not added to the perfusate. When epinephrine was added to the perfusate, subsidiary pacemakers did develop during periods when no stimuli were delivered. The pacemakers, however, were observed within the VSCS, and not within the A-V node. Hoffman and Cranefield also reported that they have not observed primary pacemakers within the A-V node (7).

The influence of the A-V node in governing the interval between subsequent ventricular responses during atrial fibrillation is shown in Figure 2. The right atrium was stimulated at a rapid rate to initiate atrial fibrillation and extracellular recordings were obtained from the right atrium (RA) and right ventricle (RV) simultaneously with transmembrane potential recordings from an A-V nodal fiber (AVN) and right bundle branch fiber (RBB). The A-V nodal potentials were recorded from a region in the lower A-V node referred to as NH by Paes de Carvalho (cited in reference 7). It was difficult, and often impossible, to make any correlation between the right atrial electrogram and A-V nodal responses in these experiments on atrial fibrillation. This is not surprising since atrial activity is rapid and chaotic during atrial fibrillation and since the right atrial electrogram was recorded from only one small region. The non-uniform conduction through the A-V node during atrial fibrillation is shown by the prepotentials that occur before the all-or-none A-V nodal action potentials in this illustration. A-V nodal cells appear not to have a definite threshold potential for eliciting an all-or-none action potential. In Figure 2, all-or-none A-V nodal potentials develop following prepotentials of different amplitudes and occur even after the A-V nodal prepotential has started to return toward resting levels. The nonpropagated local A-V nodal response that occurred after three propagated A-V nodal responses (AVN tracing) demonstrates that concealed conduction within the A-V node does occur during atrial fibrillation. When block occurred in the A-V node, the interval between resulting ventricular responses was prolonged. In the experiment recorded in Figure 2, as in most of these experiments on atrial fibrillation, the ventricular dysrhythmia was a result of conduction delays and block within the A-V node. Once an all-or-none response developed within the lower NH region of the A-V node, the impulse usually was conducted to the ventricles and no conduction delays or block were observed within the VSCS. In in-vivo experiments on atrial fibrillation in dogs during cardiopulmonary bypass, Moe and Moore (unpublished experiments) observed that the VSCS did not normally play a role in influencing the ventricular irregularity during atrial fibrillation; that is, the interval between responses in the bundle of His was identical to those between corresponding right and left ventricular electrograms.
In Figures 3, 4, and 5, the dissociation in activation of different A-V nodal fibers during atrial fibrillation was studied. All three figures were recorded in the same experiment. A right atrial electrogram (RA) was recorded simultaneously with transmembrane potentials from two A-V nodal cells (NH and N). The N record was recorded from the middle or decrementing region of the node, and NH was recorded closer to the His bundle in the NH region. Atrial fibrillation was assumed to be present when the RA electrogram showed rapid, irregular atrial activity. Although not shown, it was determined that when all-or-none responses occurred in the NH fiber in this experiment, conduction to the ventricles always occurred at a constant conduction velocity.

In Figure 3 even the shortest interval between conducted responses contained a concealed A-V nodal response. In order to determine the site of conduction delays and block, it was frequently necessary to record from more than one A-V nodal or VSCS fiber. This is particularly obvious between the fourth and fifth NH responses. Although there is the suggestion of a small local response after the fourth NH action potential, it would be impossible, because of artifacts due to microelectrode movement to say definitely that a local response occurred at this instant without the simultaneously recorded action potentials from the N cell. Between the fourth and fifth NH action potential (NH4-NH5 interval), at least two responses were blocked within the A-V node in or above the N region. The variation in duration and amplitude of the all-or-none N action potentials (second, third, fifth, sixth, seventh, etc.) occurring at the time when only local nonconducted responses were recorded from the NH fiber points out the complexity of A-V nodal transmission. When the action potentials recorded from the N fiber were of nearly normal amplitude but of shorter than control duration, conduction to NH fibers still failed in many instances. The N action potentials of short duration and smaller amplitude usually were associated with the smaller local response in the NH fiber.

In Figure 4 the shortest intervals between NH responses, and consequently ventricular responses, occurred when no responses were concealed within the A-V node. Even a small, local, nonpropagated response in the N region of the A-V node resulted in a prolonged NH-NH interval; namely, the NH4-NH5 interval is longer than the NH5-NH6 interval. Although no local responses were recorded from the N fiber between the second and third NH action potentials, the NH2-NH3 interval was still longer than the NH5-NH6 and NH8-NH9 response intervals. Therefore, between

\[ NH = \text{A-V nodal transmembrane potentials recorded from a fiber located within the lower A-V node; } N = \text{A-V nodal transmembrane potentials recorded from the decrementing region of the A-V node.} \]

\[ T = \text{time intervals of 100 msec; other abbreviations and vertical bar as in Figure 1. Experimental atrial fibrillation produced by rapid stimulation of the right atrial septum. Rapid deflections res-touched.} \]
the second and third NH action potentials, an atrial response probably was concealed very high up within the node or within the atrium. Figure 4 supports the conclusions of Moe and Abildskov (1) that the shortest ventricular intervals observed during atrial fibrillation are associated with the absence of A-V nodal concealment; longer ventricular intervals follow concealment of atrial responses.

Figure 5 was recorded when the RA rate was about 600/min. During this rapid atrial activity, eight responses were consecutively blocked within the A-V node. Although action potentials of nearly normal amplitude were recorded from the N fiber, only local non-propagated responses occurred within the NH fiber. Again, the action potentials recorded from the N region during A-V nodal block were of shorter duration than the propagated responses. Mendez and Moe also have observed brief A-V nodal action potentials associated with failure in transmission through the A-V node (8). The rapid atrial rate in this figure was maintained longer than in the other figures. The observation that the ventricular response interval was also longer supports Moe's and Abildskov's suggestion that long ventricular intervals in atrial fibrillation are associated with rapid atrial input and multiple A-V nodal concealment (1).

In several studies on experimentally initiated atrial fibrillation, a delay in A-V transmission occurred within the VSCS as well as within the A-V node. In Figure 6, the first two bundle of His responses (BH1 and BH2) were conducted over the right bundle branch and Purkinje system to the ventricles with a constant transmission time. The third His bundle response (BH3), however, reached the right bundle branch during its relative refractory period. Decreased conduction velocity within the VSCS in this instance is obvious from the increased response interval of His bundle to right bundle branch. The third right bundle branch response exhibited a decreased rate of depolarization, amplitude, and duration. In this instance of aberrant A-V conduction, a QRS complex typical of partial right bundle branch block would likely have been recorded in the intact heart since conduction over the left VSCS probably would not have been comparably impaired. The last bundle of His response (BH3) failed to be propagated to the right bundle branch and
ventricle. The BH₃ response arrived at the impaled RBB fiber earlier than the propagated BH₂ response; that is, depolarization of the BH₃ response occurred during rapid depolarization of the RBB fiber, while depolarization of the BH₂ response occurred during the RBB's plateau. The failure of BH₂ to be conducted to the impaled RBB fiber in this experiment on atrial fibrillation would result in recording of an aberrant QRS complex. Although not shown, in this experiment concealed conduction occurred within the A-V node during the long intervals between His bundle responses, namely between the BH₁-BH₂ and BH₃-BH₄ intervals.

The present studies provide direct experimental evidence for concealed conduction within the A-V node during atrial fibrillation. In most experiments on atrial fibrillation, the variations in ventricular intervals resulted from conduction delays and block at different levels of the A-V node. When only one A-V nodal fiber was impaled, it was often impossible to distinguish conduction block within the atria from concealment within the A-V node. Frequently, all-or-none action potentials were recorded from some A-V nodal fibers when others exhibited only local nonpropagated responses. Thus conduction through different parts of the A-V node during atrial fibrillation is not uniform.

In Figures 7 and 8, the distribution of ventricular cycle lengths during atrial fibrillation was a function of concealed conduction within the A-V node. At ventricular intervals between 350 and 500 msec no evidence of concealed conduction within the A-V node was recorded (Fig. 7). At longer ventricular intervals, between 500 and 650 msec, a single concealed response was routinely recorded from the middle N region of the A-V node. The most frequently recorded ventricular response interval of 600 msec (Fig. 8) occurred when a single response was concealed within the A-V node (Fig. 7). In several other experiments on atrial fibrillation the most frequently recorded ventricular interval also occurred when a single response was concealed within the A-V node. The number of concealed responses within the A-V node increased as the cycle length between ventricular responses increased. At the longest ventricular cycle length in this experiment, nine consecutively concealed responses were recorded within the A-V node.

Conduction delays and block within the VSCS occurred in only a few experiments on atrial fibrillation. When conduction delays and block did occur within the His-Purkinje system, they normally were subsequent to long preceding ventricular cycles. Several factors influenced conduction delays and block within the VSCS. The increase in the functional refractory period of bundle branch and Purkinje fibers following increases in preceding cycle length (9) increased the likelihood of block within the VSCS. Whether transmission delays and block occurred within the VSCS...
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 depended not only on preceding cycle length, but also on the time of arrival of the next conducted A-V nodal response. For example, in the middle of Figure 2, the action potential duration and hence refractory period of the RBB fiber increased following a blocked A-V nodal response. As in most experiments, however, propagated activity from the A-V node arrived too late to encounter partially refractory bundle branch fibers. Another factor that resulted in concealed conduction within the VSCS was increased pacemaker activity in bundle branch and Purkinje fibers. When the RBB fibers exhibited prominent diastolic depolarization, occasionally an impulse would arrive from the A-V node at a time when diastolic depolarization had progressed to a point where the excitability of the RBB was decreased. Hence, conduction delays occurred within the VSCS. Hoffman and Cranefield have previously pointed out the importance of pacemaker activity within the His-Purkinje system in causing concealed A-V conduction.

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The number of concealed responses recorded in the N region of the A-V node are plotted against the corresponding ventricular cycle length: When the same number of concealed responses occurred at the same ventricular cycle length, only a single point was plotted on the graph. Same experiment as Figure 8.

Frequency distribution of ventricular cycle lengths during atrial fibrillation. Same experiment as Figure 7.
The present studies suggest that when conduction delays in the RBB result from decreased excitability due to VSCS pacemaker activity, a resulting aberrant QRS complex develops later than when conduction delays result from differences in the functional refractory periods of VSCS fibers.

In clinical cardiology, aberrant QRS complexes are usually considered to result from subsidiary pacemakers within the A-V node, VSCS, or aberrant antegrade conduction. In these in-vitro experiments on rabbit hearts, no evidence was obtained to support the hypothesis (5) that the ventricular dysrhythmia associated with atrial fibrillation results from A-V nodal pacemaking with entrance and exit block; subsidiary pacemakers were observed only within the VSCS. It is well known that driving a pacemaker at a rate faster than its intrinsic rate tends to depress pacemaker activity. During atrial fibrillation, A-V nodal fibers usually were excited at frequencies close to their functional refractory period. Thus, subsidiary pacemakers within the A-V node would probably be depressed. Repetitive concealment within the A-V node results in correspondingly long intervals between subsequent excitations of VSCS fibers. The decrease in frequency of excitation of VSCS fibers might permit latent pacemakers within the ventricles to become active. If, as suggested previously, an A-V nodal response is propagated into the VSCS when diastolic depolarization has progressed to a point when conduction velocity and excitability of some VSCS fibers are decreased, then A-V conduction delays and block might occur as a result of subsidiary pacemakers within these fibers.

When aberrant QRS complexes occur with antegrade conduction of premature atrial responses in normal animals, either partial or complete right bundle branch block is usually found. Functional left bundle branch block appears to be very rare in normal animals (10). It would seem, therefore, that if an aberrant QRS complex recorded during atrial fibrillation were not associated with right bundle branch block, then a subsidiary pacemaker within the VSCS should be suspected. Premature ventricular beats frequently occur within the left VSCS following administration of substances that enhance pacemaker activity. A left VSCS pacemaker could produce a bizarre ventricular complex due to abnormal ventricular activation. It is possible also to have supraventricular conduction delays and block within the left VSCS when diastolic depolarization occurs in these fibers.

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**References**

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