Supernormality in Bachmann's Bundle

AN IN VITRO AND IN VIVO STUDY IN THE DOG

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ABSTRACT

A supernormal phase of conduction between right and left atria was observed in the exposed hearts of anesthetized dogs. Interatrial conduction time of premature atrial responses was reduced by as much as 17% during the early phase of diastole, relative to the conduction interval of basic driven responses. The supernormal phase lasted from 60 to 140 msec and was greater at slower basic driving frequencies. A brief phase of supernormal excitability was also found in the specialized cells of Bachmann's bundle from puppy hearts in vitro. Supernormal conduction was not observed within the atrial appendages (i.e., in areas not supplied by specialized conducting bands); supernormal excitability was not demonstrable in cells of ordinary atrial myocardium. Although vagal stimulation abbreviated transatrial conduction time, atropine did not abolish the phase of supernormality.

ADDITIONAL KEY WORDS

interratrial conduction, interatrial band, excitability of atrial cells, specialized atrial fibers, vagal stimulation, atropine, Purkinje fibers

Anatomic and physiologic studies have demonstrated bands of specialized conduction fibers, not unlike the intraventricular specialized conduction system, within the mammalian atria. Morphologically distinct bundles coursing from the S-A node to the A-V node have been characterized by Robb and Petri (1) and by James (2). Electrophysiologically, these fibers, and those of Bachmann's interatrial band (3), differ from ordinary atrial myocardial fibers. Their transmembrane action potentials, unlike those of atrial fibers, are reported to have a somewhat faster rise time and a distinct phase 2 plateau (4). They appear to be much more resistant than atrial muscle to increased external potassium; propagated action potentials can be recorded in the specialized tracts at potassium concentrations which cause failure of transmission in atrial myocardium (4, 5). As a result, sinoventricular rhythm can persist in the absence of P waves during hyperkalemia (6).

Like Purkinje fibers, the specialized fibers in Bachmann's bundle conduct impulses at a significantly higher velocity than the surrounding myocardium (4). Unlike Purkinje fibers, however, their action potential durations are significantly abbreviated by ace
tylycholine (4).

In the present study, attempts were made to determine whether the functional refractory period of fibers in the interatrial band of the dog heart could be demonstrated to exceed that of ordinary atrial myocardium, as has been shown in similar comparisons of Purkinje fibers and ventricular muscle (7). In the course of these experiments, it was found that a supernormal phase of interatrial conduction could be consistently demonstrated in the anesthetized dog. In vitro studies were undertaken to determine whether a phase of supernormal excitability, like that observed in Purkinje fibers (8), could be demonstrated in the specialized fibers of Bachmann's bundle.
Methods

IN VIVO STUDIES

Mongrel dogs weighing 14 to 20 kg were anesthetized with intravenous sodium pentobarbital, 30 mg/kg. Artificial respiration was given, the chest was opened through a midsternal incision, and the heart was cradled in the opened pericardium. Cardiac innervation was not disturbed. Stimulating bipolar electrodes were attached to the tips of the right and left auricular appendages. Bipolar recording electrodes were attached to the medial faces of the two appendages 15 mm from the stimulating electrodes.

Driving stimuli from a Tektronix pulse generator were applied to either the right or left atrium through an isolation transformer. Following each sixteenth driving pulse (S1), a precisely timed test stimulus (S2) was delivered through the same stimulating electrodes. The S1-S2 interval was varied progressively by small steps to scan the interval between two successive driving stimuli.

Vagal stimulation, when employed, was delivered by a Grass stimulator through Harvard shielded electrodes applied to the distal end of the cut vagal trunks.

IN VITRO STUDIES

Mongrel puppies aged six weeks and weighing 2 to 3 kg were anesthetized by intraperitoneal sodium pentobarbital, 40 mg/kg. Under artificial respiration the chest was opened in the midline, and the entire heart was quickly excised and placed in Tyrode’s solution or temporarily perfused through the aorta. The aorta and pulmonary artery were retracted, revealing the roof of the atria and the interatrial band. The atrial roof, together with adjacent right and left appendages, was rapidly separated from the rest of the heart. The preparation was then placed in a perfusion chamber. Bipolar silver electrodes were applied to the preparation for driving purposes. Glass microelectrodes of 14- to 20-megohm resistance filled with 2 M potassium citrate were used for intracellular recording and stimulating.

The stimulation circuits consisted of two Tektronix pulse generators triggered by a device which permitted the application of a series of precisely regular basic pulses (S1) applied through the bipolar external electrodes, followed by one or more test shocks (S2, S3) delivered to either the intracellular or external electrodes. Stimulus intervals were counted from a 100-kc crystal oscillator.1 The external circuit consisted of the pulse generator, isolation transformer, and bipolar chlorided silver electrodes applied to the right atrial side of the preparation. This circuit was employed to deliver 11 basic driving pulses of 4-msec duration at a cycle length of 600 msec. The application of test shocks (S2) through the microelectrode was achieved by a gated millisecond relay which prevented short circuit to ground. The recording apparatus was protected from this pulse by a diode short to ground.

Transmembrane action potentials were recorded through an Argonaut negative capacitance electrometer, a Tektronix 565 oscilloscope, and a Grass kymograph camera. The test pulse (S2) was displayed on a second channel. A 100-mv calibrating signal battery and a potentiometer for voltage bucking were connected in a series with the indifferent electrode, a chlorided silver wire submerged in the bath. A differentiator was occasionally used to estimate the rate of rise of cellular action potentials.

To test for supernormal excitability, a test pulse (S2) was delivered through the microelectrode. A second test pulse (S3) was delivered through the external electrodes 30 to 60 msec later. The S3 pulse always succeeded if S2 failed and was seen as an artifact on the plateau of the action potential when S2 was successful. Late diastolic threshold of the impaled cell was established by setting the S1-S2 interval at 400 msec and gradually raising the pulse amplitude of S2 until it succeeded and S3 failed. Using a fine adjustment potentiometer, the pulse strength was then lowered just enough to make S2 repeatedly fail. The S1-S2 interval was then abruptly reduced to 200 msec or to a value at which S2 was successful. To confirm supernormality the S2 was repeatedly shifted to late diastole and back to show its continued failure and success in the late and supernormal phases, respectively.

Results

IN SITU DOG HEART

A supernormal phase of conduction between the right and left atrium was demonstrated in every animal studied. Conduction time of a late premature impulse from the right to left auricular appendage was approximately equal to that of the basic driven responses, but as the S1-S2 interval was reduced, the conduction time diminished by as

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1This instrument was designed by W. J. Mueller and constructed in the Bioelectronics Laboratory of

the State University of New York, Upstate Medical Center, Syracuse, New York.

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Conduction time of premature beats as a function of S1S2 interval. Open-chest dog, pentobarbital anesthesia. Driving stimuli (S1) and premature stimuli (S2) applied through electrodes attached near tip of right atrial appendage. Observations at various basic cycle lengths (S1S1 intervals) of 250 to 340 msec.

A supernormal phase of conduction was also demonstrable when the stimuli were applied to the left atrium. In Figure 2, transatrial conduction times in both directions are shown for both series. At S1S2 intervals of less than 200 msec, conduction delay due to relative refractoriness is apparent in all four curves. From 200 to about 280 msec, transatrial conduction time was less than in late diastole; the maximum reduction was about 10 msec in each direction. Conduction time from the site of right atrial stimulation to the near recording electrodes on the same appendage did not change significantly as the S2 stimulus was delayed from 200 to 400 msec. With left atrial stimulation, conduction to the near site appeared to be somewhat faster for relatively early premature beats. The left atrial recording electrodes in this experiment were attached near the termination of the interatrial band, and it is probable that terminal ramifications of the bundle lay between the stimulating and recording electrodes.

Vagal stimulation abbreviated conduction time between the two atria (Table 1). This effect became more marked as the frequency of vagal stimulation was increased. The minimal conduction time recorded during the supernormal phase was not significantly changed by vagal stimulation; accordingly, supernormality was no longer demonstrable...
when the vagi were stimulated at the higher frequency.

In some experiments, supernormal conduction was demonstrated not only for premature atrial responses, but also when the basic driving frequency was appropriately increased. This was not true, however, in the experiment illustrated in Figure 1. When the basic cycle length was 250 msec in this experiment, the interatrial conduction time for the basic responses was slightly longer than when the cycle length was 350. In those experiments in which conduction time diminished at the higher driving rates, vagal stimulation abolished the change; i.e., the conduction time at slow rates was reduced, and no further reduction occurred when the driving frequency was increased. On the chance that the faster driving rate might have influenced conduction time by liberating acetylcholine from intra-atrial vagal endings, measurements were repeated before and after the administration of atropine, 0.25 mg/kg, in two experiments. In both cases, atropine caused a slight increase in the control conduction time but did not prevent the diminution which occurred at the higher driving

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**TABLE 1**

<table>
<thead>
<tr>
<th>Effect of Vagal Stimulation on Spontaneous Sinus Node Cycle Length and on Transatrial Conduction Time</th>
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<tbody>
<tr>
<td>Cycle length (msec)</td>
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<tr>
<td>---------------------</td>
</tr>
<tr>
<td>Control</td>
</tr>
<tr>
<td>Vagus, 5 cps</td>
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<tr>
<td>Vagus, 10 cps</td>
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</table>

*All measurements of conduction time were made at driven cycle length of 400 msec; minimal values during "supernormal" phase were determined by scanning early diastolic period in each case.

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Characteristic transmembrane action potentials recorded from atrial muscle (A) and from Bachmann's bundle (B) in isolated puppy heart. Time and voltage calibrations, 50 msec, 50mv. Upstrokes retouched.

rate. Atropine also failed to prevent the supernormal phase of conduction for premature beats.

The phase of supernormal conduction ($S_1S_2$ intervals in the range of 200 to 300 msec) occurs during the QT interval of the ventricular cycle. To test the possibility that the electrical field generated by the ventricles might be responsible for the phenomenon, one experiment was conducted after complete A-V block had been produced by acute section of the His bundle. The interatrial conduction time was recorded while the atria were driven with a cycle length of 400 msec, and the ventricles were driven at a cycle length of slightly less than 800 msec. The right-to-left conduction time was 63 msec, and no variations of more than ±1 msec were observed as the ventricular response was allowed to drift through the atrial cycle.

When premature atrial responses were induced at an $S_1S_2$ interval of 200 msec, the right-to-left conduction time was 58 msec; this value was also independent of the temporal position of the ventricular responses.

OBSERVATIONS ON SINGLE ATRIAL CELLS

A large population of atrial cells in the puppy hearts were studied. Cells in the interatrial band showed a distinct overshoot and convex shoulder (phase 2 plateau). Ordinary atrial cells impaled in the appendages tended to have less overshoot and either a concave or straight repolarization phase (Fig. 3).

Responses of cell in Bachmann's bundle to intracellular and external stimuli. In each panel, first action potential is response to last of a series of rhythmic stimuli (cycle length, 500 msec) applied to remote site through bipolar electrodes. In A and C, intracellular stimulus of $3 \times 10^{-7}$ amp was subthreshold. In B, same stimulus was effective. Subsequent coupled stimulus, effective in A and C, was delivered externally through driving electrodes. Action potential amplitude, 107 mv; interval between basic and test (intracellular) stimuli in A and C, 380 msec; in B, 182 msec. Upstrokes of action potentials and current pulses retouched.
The resting membrane potential was often slightly greater in specialized cells of the interatrial band than in atrial cells, but this finding was by no means constant. In general the values obtained for the maximum rise velocity of the action potential were lower than those given by Wagner and his associates (4). This was particularly true of cells in the interatrial band, the average upstroke velocity of which overlapped with that of atrial cells. Transitional cell types were seen which could not be identified by their action potential shape or rise time alone.

The most reliable and distinctive feature of specialized atrial fibers in the interatrial band was the presence of a supernormal phase of excitability. It was repeatedly demonstrated in these cells and was never found in ordinary atrial muscle cells. In a few cases the difference in threshold between early and late diastole was very slight, but it could be regularly demonstrated by shifting the stimulus back and forth. An example is shown in Figure 4; an intracellular stimulus which repeatedly failed late in diastole (A and C), was uniformly successful when applied shortly before complete repolarization (B).

The duration of the zone of supernormal excitability was not precisely determined. It could not have been extremely narrow, however, because the S1–S2 interval which would successfully demonstrate it was guessed by scrutiny of the previous action potential, and the first attempt was nearly always successful.

The phenomenon was present in many transitional cell types not otherwise identifiable. It was repeatedly sought for, but not found, in cells deep to the interatrial band.

In a few experiments, action potentials recorded from cells in Bachmann's bundle were differentiated while driving, and premature stimuli were applied through bipolar external electrodes a few millimeters distant. No significant changes in maximum upstroke velocity were recorded when premature responses occurred within what had previously been demonstrated to be the phase of supernormal excitability.

Diastolic depolarization was not seen in any of the cells impaled. After 8 minutes of perfusion by a Tyrode solution containing 9 mM of potassium per liter, ordinary atrial cellular potentials disappeared while cells in the interatrial band continued to show action potentials. These findings corroborate those of Wagner and his associates (4).

Discussion

The present study provides additional evidence for the presence of specialized Purkinje-like fibers in the atrium. In the isolated preparations, cells of Bachmann's bundle showed on the average a greater resting membrane potential than atrial muscle cells. The upstroke velocity and amplitude of the action potential were greater, and a phase 2 plateau was present. These are largely quantitative differences and, as in the study of Wagner et al. (4), there is considerable overlap in the two cell types. Supernormal excitability was never found in atrial muscle cells and was regularly demonstrated in those of Bachmann's bundle. Supernormality is thus another property which differentiates the two cell types. In the mammalian ventricle a comparable difference between the specialized conducting system and ventricular myocardium is also observed (8). The present study showed that supernormal excitability was present in intermediate atrial cell types, i.e., those in which the descending limb of the action potential was straight rather than convex or concave. These action potentials were seen in the junctional region where Bachmann's bundle arborizes into the left atrial appendage. The intermediate shape may be a reflection of the electrotonic influence of a neighboring population of cells predominantly of the atrial muscle type. Similar influences between adjacent cell groups are apparent in the A-V node (9).

The restriction of supernormal excitability and conduction to specialized atrial fibers explains conflicting reports in the literature. Lewis and Master (10) failed to find supernormal excitability in the dog heart. Orias et al. (11) found it intermittently in both...
SUPERNORMALITY IN BACHMANN'S BUNDLE

The disparity in findings may be a function of the site of atrial stimulation. Should this be far removed from specialized fibers, supernormality is not likely to be demonstrated.

Supernormal conduction was not demonstrated in vitro. Responses to intracellular stimuli were propagated less rapidly than externally evoked responses, perhaps because of the small dimensions of the initial excitation front. Responses propagated from atrium to the impaled unit in Bachmann's bundle, within the limited distances available for study, did not vary significantly in conduction time from one S1S2 interval to another.

Supernormal conduction between the right and left atrium was demonstrated both in the conduction of premature beats and as a simple shortening of conduction time with increasingly rapid atrial driving rates. The latter finding is in contrast to that of Brooks et al. (12) who found no change in trans-atrial conduction time with cycle lengths in the range of 200 to 550 msec. Although the changes observed in the present study were small (less than 17%), the phenomenon was regularly reproducible, and it was never observed to occur between two atrial recording sites except in areas where specialized fiber tracts have been demonstrated. Inasmuch as a significant proportion of the conduction time between the right and left atrial electrodes must be accountable to atrial muscle, the percentage change within the interatrial band itself must be greater than the observed values.

Vagal stimulation shortened interatrial conduction time and diminished supernormal enhancement of conduction velocity. Its effect on supernormal excitability was not studied. The means by which vagal stimulation shortens conduction time is still not precisely known. Brooks et al. (12) reported no change in threshold requirements during vagal stimulation. Cholinergic effects are primarily on K conductance; thus resting membrane potential is increased and repolarization is accelerated (13). If in a given cell the resting potential is changed from −80 to −90 mV, the resulting improvement in action potential upstroke velocity may provide a more effective (and hence faster) propagation wave.

The temporal position of the phase of supernormal interatrial conduction suggested the possibility that reflexly induced phasic changes in vagal discharge (resulting, for example, from the penultimate ventricular beat) might be responsible. The phenomenon was not, however, abolished by atropine. The possibility of an electrical influence of the adjacent depolarized ventricular muscle mass was also eliminated as a factor. There remains the possibility that phasic changes in the immediately extracellular K concentration could be responsible for both the threshold changes recorded in vitro and the accelerated conduction observed in vivo. A convenient test of this possibility is not available.

The clinical counterpart of the findings described here would be difficult to demonstrate. One would expect that when atrial premature beats occur with a short coupling interval, the P wave would be shorter in duration by virtue of supernormal intra-atrial conduction. The most suitable way to demonstrate the phenomenon would be by means of an artificial atrial pacemaker either in competition with the sinus node in a case of 3° A-V block or through paired atrial pacing. The differences in conduction time, however, would be small and would require verification by three plane vectorcardiographic analysis.

Acknowledgment

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