Supernormal Conduction in the Human Atria

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SUMMARY

A supernormal period of intra- and interatrial conduction was observed in five patients during premature stimulation of various atrial sites with driving cycle lengths ranging between 500 and 600 msec. Electrograms were recorded with filtered, 1-mm apart, bipolar catheter electrodes placed in the high right atrium, coronary sinus, and midleft atrium. The supernormal period, which lasted from 90 to 140 msec, was located at the end of the relative refractory period. During this part of the cycle, the response1-response2 (R1-R2) intervals were shorter than the corresponding St1-St2 intervals. As in experiments performed with plunge electrodes, the conduction time of premature atrial responses was shorter than in late diastolic or driven beats. Although a mechanical origin (due to inevitable catheter movement produced with cardiac motion) can be invoked in the genesis of these changes, it is highly probable that supernormality was a true electrophysiologic event involving, predominantly, the specialized atrial tracts.

Additional Indexing Words:
Supernormal conduction  Atrial pacing  Interatrial conduction
Midleft atrial pacing  Intraatrial conduction  Specialized atrial tracts

CHILDERS et al. described a supernormal phase of interatrial conduction in canine hearts.1 Other investigators, however, have not observed this phenomenon either in animal experiments or in man.2-8 Due to this discrepancy, and because of possible electrophysiologic implications for the analysis of His bundle recordings, the present study was performed in order to delineate the various patterns of interatrial and intraatrial conduction during premature atrial stimulation.

Methods and Material

The technic used in our department to record the electrical activity from the His bundle area and other atrial sites has been reported elsewhere.9,10 After explaining the procedure and obtaining consent, a tripolar catheter electrode was introduced percutaneously through the femoral vein and positioned, under fluoroscopic control, across the septal leaflet of the tricuspid valve. The two distal electrodes were separated by a distance of 1 mm while the one at the tip was 11 mm from the most proximal electrode.* Three other catheters with similar characteristics were introduced through an antecubital vein and placed in the high right atrium (HRA) and coronary sinus for recording or pacing from these sites. In one patient the midleft atrium was explored transeptally. Filtered (40-400 Hz) bipolar electrograms were recorded simultaneously with four surface leads at paper speeds of 100 mm/sec. For arbitrary reasons only one standard lead is shown in each figure.

The catheter placed over the septal leaflet of the tricuspid valve recorded an electrogram from the low right atrium (LRA) probably in the vicinity of the A-V node. On the other hand, the coronary sinus electrodes registered the electrical activity of a left atrial site.9 Stimulation through

*Elecath Corporation, Rahway, New Jersey.
this catheter produced negative P waves in the standard leads. If the ventricles were simultaneously or exclusively paced from these electrodes, the catheter was slowly withdrawn until pure atrial stimulation resulted.

The characteristics of the stimulator used in this study have been reported elsewhere. The pulses delivered were slightly underdamped, 2.5 msec in duration. The atria were driven (St1) at a rate fast enough to suppress sinus node activity. By means of a variable delay circuit, premature stimuli (St2) were delivered after every eighth driven beat and the cycle scanned at 10-20-msec intervals. Whereas St1 had twice diastolic threshold intensity, the diastolic threshold intensity of St2 was two times higher. The responses to driven and premature stimuli were identified by the numbers 1 and 2 placed at the end of the corresponding abbreviations. Various intervals were measured (in msec) from (or to) the onset of the rapid deflections in each electrogram, and from the inscription of the stimulus spike. The following intervals were measured during HRA pacing; (1) St1-St2 (driving stimulus to premature stimulus); (2) LRA1-LRA2 (low right atrium driven response to low right atrium premature response); (3) LA1-LA2 (left atrial driven response to left atrium premature response); (4) ST1-LRA1 (driving stimulus to low right atrial driven response); (5) ST2-LRA2 (premature stimulus to low right atrial premature response); (6) ST1-LA1 (driving stimulus to left atrial driven response); and (7) ST2-LA2 (premature stimulus to left atrial premature response).

Three intervals were measured during coronary sinus and transeptal left atrial (LA) pacing: (1) St1-St2; (2) LRA1-LRA2; and, (3) HRA1-HRA2 (high right atrial driven response to high right atrial premature response).

The effects of at least three testing stimuli were analyzed at each St1-St2 interval. Moreover, the intervals between St1 and their responses were measured in the control period as well as in the beat preceding each testing stimulus. Measurements were performed independently by three of the authors. Cases in which the interobserver variations were greater than ±5 msec were excluded from this study. An average was made from the three sets of measurements and the resulting values punched into IBM cards and fed into a computer programmed to bring out the types of graphs presented in the following section.

Atrial, atrioventricular, and intraventricular conduction patterns were studied in 18 patients referred for intracardiac electrophysiologic studies. Some of the results were reported elsewhere. However, studies of atrial conduction were adequate for detailed analysis in only five patients. Three had primary conducting system disease, one atherosclerotic heart disease, and one rheumatic heart disease with mitral regurgitation.

**Results**

A supernormal phase of conduction from the three paced sites was observed in five patients. Two types of graphs were constructed to analyze the patterns of conduction. One, obtained from a patient with an old inferior wall myocardial infarction (figs. 1 and 2), was constructed by plotting the St1-St2 intervals on the abscissa against the intervals between the premature (St2) stimuli and their responses in the LRA and LA on the ordinates. This type of graph was selected so that the results of the present study could be compared with those of Childers et al. which were presented in this fashion. At St1-St2 intervals greater than 475 msec the St2-LRA2 and St2-LA2 intervals showed values similar to those recorded in driven beats. This was the period of full

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*American Optical Corporation, Bedford, Massachusetts.

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Case 1. Intracardiac leads showing supernormal conduction at a St1-St2 interval of 350 msec. At this delay the St2-LRA2 and St2-LRA intervals were shorter than those of driving and late diastolic testing stimuli. In this figure only the electrograms from the low right atrium and left atrium are shown. HBE = His bundle electrogram; CSE = coronary sinus electrogram. Paper speed was 100 mm/sec.

In contrast, when the coupling intervals between driving and premature stimuli ranged between 470 and 350 msec, the St2-LRA2 and St2-LA2 intervals were as much as 25 msec shorter than in late diastolic or driven beats (figs. 1 and 2). This was the period of atrial supernormality. At delays shorter than 350 msec, the intervals between the premature

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<th>Table 1</th>
<th>Electrophysiologic Data in Four Patients with Supernormal Intra- and Interatrial Conduction</th>
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Abbreviations: SN = supernormality; PCSI = primary conducting system disease; RHD = rheumatic heart disease; MR = mitral regurgitation; ERP = effective refractory period of the atria; HRA = high right atrium; CS = left atrium via coronary sinus; MLA = midleft atrium transeptally.

* Shortest LRA-LRA2 and LA1-LA2 intervals obtained to the right of the oblique line.
† Shortest LRA-LRA1 and LA1-LA2 intervals obtained to the left of the oblique line.
stimuli and their responses were longer than in driven beats, indicating that the impulses were propagating through incompletely recovered tissues. Finally, premature stimuli failed to produce propagated responses when the St1-St2 delay was reduced to 270 msec. The intervals between the inscription of the LRA2 and LA2 electrograms were similar in all parts of the cycle.

The graphs in the other four patients were constructed by plotting the LRA1-LRA2 and LA1-LA2 intervals (ordinates) as a function of the St1-St2 intervals (abscissa). Clinical and electrophysiologic information from these four patients is presented in Table 1. The supernormal period of conduction lasted between 90 and 140 msec, extending from a maximum St1-St2 delay of 450 msec to a minimum delay of 260 msec, with driving cycle lengths ranging between 500 and 600 msec. During supernormality the R1-R2 intervals were 10–30 msec lower than the corresponding St1-St2 delays.

A graph of this type showing supernormal atrial conduction is shown in Figure 3. The full recovery time (FRT) occurred at an St1-St2 interval of 370 msec. At delays ranging between 370 and 280 msec the atrial response curves descended slightly below (to the right of) the oblique line of identity. This was the period of atrial supernormality during which the LRA1-LRA2 and LA1-LA2 intervals were

**Figure 5**
Graph relating the St1-St2 intervals (abscissa) to the R1-R2 (response1-response2) intervals during high right atrial (HRA) pacing and (transeptal) midleft atrial (LA) stimulation. Supernormal conduction was observed from both pacing sites as determined by the deviation of the corresponding values to the right of the oblique line of identity.

**Figure 6**
Bipolar surface lead III and intracardiac recordings during high right atrial (HRA) and transeptal midleft atrial (LA) stimulation. Supernormal conduction was observed from both pacing sites since the R1-R2 intervals were shorter than the corresponding St1-St2 intervals.
shorter than the corresponding St₁-St₂ intervals (fig. 4). At St₁-St₂ delays shorter than 280 msec, the atrial response curves ascended above the oblique line (to the left of the line of identity) until reaching the ERP of the atria (240 msec). During this phase of incomplete recovery, the conduction velocity of the premature atrial beats was depressed as indicated by the fact that the R₁-R₂ intervals were greater than the corresponding St₁-St₂ delays.

In one patient atrial stimulation was performed through the coronary sinus (table 1). In another, the mid-LA was paced transeptally (table 1). The graph in figure 5 shows the results obtained from HRA and mid-LA pacing. Only one value was represented for the LRA₁-LRA₂ intervals during HRA stimulation and for the LRA₁-LRA₂ and HRA₁-HRA₂ intervals during mid-LA stimulation since they coincided within ±5 msec. Atrial supernormality occurred when pacing was performed from both sites, at St₁-St₂ intervals ranging between 445 and 335 msec (fig. 6). At these delays both curves were located below (to the right of) the oblique line. At intervals shorter than 335 msec both curves ascended above (to the left of) the oblique line until reaching the ERP of the atria.

**Discussion**

A supernormal phase of conduction between canine atria was demonstrated by Childers et al. during right, as well as left atrial stimulation.¹ It appeared both in conduction of premature beats and as a simple shortening of conduction time with cycle lengths in the range of 200–550 msec. They attributed this phenomenon to the specialized interatrial tracts since a supernormal phase of excitability occurred only in the cells of the Bachman bundle and not in ordinary atrial fibers. Supernormality was not found in the atrial appendages which are probably devoid of specialized cells. The faster conduction velocity appeared between stimulating and recording electrodes, the decrease in conduction time being only 17% lower than the conduction velocity of the driven and late diastolic responses. The small magnitude of this difference, as presented in figures 1–6 and table 1, might explain why supernormal conduction was not observed by other authors who also had studied intraatrial conduction.²⁻⁴, ⁶⁻⁸

In animal experiments the recording electrodes were attached directly to the atria. Obviously, the use of catheter electrodes introduces a potentially significant variable, i.e. catheter movement with heart motion. It can be postulated that the phenomenon observed in this communication had a mechanical, rather than an electrical, origin. However, as suggested by comparing figures 1 and 2 of this article with figures 1 and 2 of the article by Childers et al., supernormality was found in the same part of the cycle as in those experiments where plunge electrodes were used.

It is also possible that this phenomenon could have been due to improper measurements created by the incorrect assessment of the arrival time of excitation at the recording sites. The precise instant of arrival of the propagating wavefront might not be accurately determined even when using bipolar catheter electrodes having an interelectrode distance of 1 mm. Moreover, at times in experiments with plunge electrodes, the responses to very early stimuli can be so distorted that a satisfactory measurement is impossible to obtain.⁴ The significance of this type of error was reduced when the atrial electrograms were sharp and of short duration.

Supernormality in the human atria is likely a true electrophysiologic event rather than an artifactual finding. Unfortunately, the nature of inter-and intraatrial conduction in man has been insufficiently studied under controlled conditions.¹²⁻¹⁶ For instance, that the HRA and LRA were activated at more or less the same time when the midleft atrium was paced transeptally suggests that conduction does not necessarily occur in a linear fashion to the various bipolar electrodes. Hence, intervals between the corresponding responses merely indicate differences in activation time.
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at the explored sites. It was interesting to observe that the R₁-R₂ intervals coincided (with ± 5 m sec) in all parts of the cycle, regardless of the pacing site. In other words, when conduction was depressed on one area, it was also depressed in the other recording site. The same was true when conduction was enhanced or fully recovered.

At short S₁-S₂ intervals delayed arrival of excitation at the proximal electrodes can be due to an increase in latency at the site of stimulation and/or to the slow propagation that occurs early in the cycle.

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