Electrophysiologic Demonstration of Concealed Conduction in the Human Atrium

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SUMMARY In the course of electrophysiologic evaluation of six patients with sick sinus syndrome, two patients with chronic conduction system disease and four patients with paroxysmal supraventricular tachycardia or atrial flutter-fibrillation, the phenomenon of concealed conduction within the atrium (At) was repeatedly observed. One pair of intracardiac electrodes was used to deliver electrical stimulus (St) to the high right At and two additional pairs of electrodes were utilized to record high right and low septal right atrial electrograms. The interelectrode distances were 10 mm apart. In all 12 patients, high right atrial capture could be accomplished at a pacing rate of ≥200 beats/min. Concealed intra-atrial conduction was evident when the intra-atrial conduction time of the propagated St during 2:1 St-At block was more than 40 msec longer than that during 1:1 St-At conduction at half the St frequency. This indicated that the nonpropagated St during 2:1 St-At block partially penetrated the At and in turn, delayed conduction of the subsequently propagated St. Further observations revealed that a gradual increase in the St frequency resulted in a progressive prolongation of the intra-atrial conduction time and a shifting of 2:1 to 3:1 St-At block (alternating Wenckebach periodicity in the At) in five patients. These findings clearly demonstrate the occurrence of concealed conduction in the human At.

CONCEALED CONDUCTION is defined as the effect of a non-propagated impulse upon conduction of a subsequently propagated impulse. This electrophysiologic phenomenon can be validated in animal experiments with programmed electrical stimulation as well as microelectrode recordings of action potentials from various levels of the conduction system. In the human heart, Langendorf et al. and Carleton and Graettinger have experimentally induced concealed atrioventricular (AV) conduction with atrial stimulation. Damato and coworkers have subsequently used electrode catheter recordings of intracardiac electrograms to localize the sites of concealment.

As observed in animal experiments, concealed conduction in the human heart usually occurs in the AV node or His-Purkinje system or both, and to our knowledge, has not been previously demonstrated in the human atrium. In the present study, we have repeatedly observed its occurrence in the human atrium, using the techniques of intra-atrial stimulation and recordings.
Materials and Methods

Data were analyzed for 35 patients in whom successful atrial pacing capture could be accomplished at an atrial pacing rate of > 200 beats/min (cycle length ≤ 300 msec) during electrophysiological studies. The phenomenon of intra-atrial concealed conduction could be repeatedly observed in 12 patients (34.3%). The electrophysiological findings in these 12 patients are the basis of this report.

After obtaining an informed consent, all cardio toxic and antiarrhythmic medications were discontinued 48–72 hours before the study. The study was performed in a postabsorptive, nonedated state. Using a conventional technique, the His bundle electrogram was obtained with a tripolar electrode catheter (Castillo, Elecath No. 07315, 5F) placed across the tricuspid valve, from which low septal right atrial (LRA) activity was recorded as well. A hexapolar electrode catheter (Berkovits-Castellanos, USCI No. 003932, 6F) was introduced via an antecubital vein, and its distal pair of electrodes were placed at the right ventricular apex for programmed right ventricular stimulation. The most proximal pair of electrodes was placed at the junction of the superior vena cava and the right atrium, and was used for pacing the high right atrium (HRA). Its adjacent pair (located 20 mm distally of electrodes was used for recording the HRA electrogram. The interelectrode distances for recording were 10 mm. The bipolar intracardiac electrograms were then displayed simultaneously with standard surface electrocardiographic leads I, II, and V6 on a multichannel oscilloscopic photographic recorder (Electronics for Medicine, DR-16, White Plains, New York) and recorded at a paper speed of 100 mm/sec, using a filter setting of 40–500 Hz.

Following programmed atrial and ventricular premature stimulation, incremental atrial pacing with a progressive decrease in the pacing cycle length was performed through the most proximal pair of electrodes of the hexapolar electrode catheter in the HRA. The electrical stimuli were delivered via a programmed digital stimulator and were 2 msec duration at approximately twice diastolic threshold. The position of the catheter electrodes was adjusted so that successful HRA capture could be accomplished. Patients in whom constant atrial capture could not be obtained were excluded from the study.

A protocol of incremental atrial pacing was designed to observe the occurrence of concealed conduction in the atrium. 1) Atrial pacing was begun at a rate slightly faster than that of the sinus rhythm to assure constant 1:1 stimulus (St)-atrial (At) capture. 2) Incremental atrial pacing with a gradual decrease in the atrial pacing cycle length was then performed until the development of 2:1 St-At block. 3) Finally, the atrial pacing rate was then abruptly reduced to one-half, or was further gradually increased in an attempt to induce alternating Wenckebach periodicity in the atrium. All 12 patients tolerated the procedures well, and even when the protocol was repeated, no complications were observed.

For the purpose of this study, only pertinent data pertaining to the electrophysiological properties of the atrium were analyzed. St, At, and H represented stimulus signal, atrial and His bundle electrograms, respectively. Definitions of terms are as follows:

1) Effective refractory period of the atrium: the longest stimulus premature coupling interval (St1-St2) that failed to generate an atrial response.


3) Intra-atrial conduction time: A) P-A interval measured from the onset of the P wave to the beginning of LRA electrogram in the His bundle electrographic lead (HBE) during sinus rhythm (normal: 20–50 msec in our laboratory); B) St-HRA interval measured from the onset of stimulus signal to the beginning of the HRA electrogram; and C) HRA-LRA interval measured from the beginning of the HRA electrogram to the beginning of the LRA electrogram in the HBE. The atrial refractory periods so measured were then compared to previously published data by Denes et al.

Results

Clinical Profiles

Table 1 summarizes the pertinent clinical findings in these 12 patients. There were seven males and five females ranging in age from 38–64 years (mean 53.3 years). Six patients presented with the sick sinus syndrome, two had chronic conduction system disease and four had episodes of paroxysmal supraventricular tachycardia or atrial flutter-fibrillation. In these last four patients, subsequent electro-

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Abbreviations: F = female; M = male; SSS = sick sinus syndrome; CCSD = chronic conduction system disease; PSVT = paroxysmal supraventricular tachycardia; AF = atrial flutter-fibrillation; WPW = Wolff-Parkinson-White syndrome; ASHD = arteriosclerotic heart disease; MI = myocardial infarction.
physiological evaluations suggested that three had AV nodal reentrant tachycardia related to dual AV nodal pathway conduction, and the remaining patient (case 9) had atrial flutter-fibrillation associated with the Wolff-Parkinson-White syndrome. Three patients had associated hypertension and four arteriosclerotic heart disease with or without prior myocardial infarction. None of these 12 patients had atrial enlargement by echocardiography and/or chest x-ray.

Electrophysiological Findings

Table 2 lists the pertinent electrophysiological data pertaining to the functional properties of the atrium.

### P-A Interval

The P-A interval during sinus rhythm ranged from 30–70 msec. Five patients (cases 2, 7, 8, 9, and 10) had prolonged P-A intervals (>50 msec). The P-R interval in the one patient (case 9) with associated Wolff-Parkinson-White syndrome was relatively long (140 msec), consequent to a prolonged P-A interval (70 msec) associated with the presence of a left-sided accessory AV pathway.

### Atrial Refractory Period

With respect to the atrial driving cycle lengths, the effective refractory period was prolonged in four patients (cases 2, 6, 8, and 11), and the functional refractory period was prolonged in seven patients (cases 2, 3, 6, 7, 8, 9, and 11) (table 2).

### St-HRA and HRA-LRA Intervals

In all 12 patients, 2:1 St-At block developed at atrial pacing rates between 222–400 beats/min (St-St intervals between 150–270 msec) (table 2). Intra-atrial conduction time (the St-HRA and HRA-LRA intervals) of the propagated St during 2:1 St-At block was significantly longer than that during 1:1 St-At conduction at half the atrial pacing rate (stimulus frequency).

As illustrated in figure 1, there was 2:1 St-At block at an atrial pacing cycle length (St-St) of 180 msec. After the fourth St, the atrial pacing rate was abruptly reduced to one-half (St-St interval lengthened to 360 msec). This resulted in 1:1 St-At conduction. Note that the atrial rate remained the same as that during 2:1 St-At block. However, intra-atrial conduction time of the propagated St (St-HRA = 45 and HRA-LRA = 30 msec) during 2:1 St-At block was longer than that (St-HRA = 30 and HRA-LRA = 15 msec) during 1:1 St-At conduction at half the atrial pacing rate. The HRA electrogram was more fractionated during 2:1 St-At block than during 1:1 St-At conduction.

Figure 2 shows another example. Panels A and B were not continuous. In panel A, there was 2:1 St-At block at an atrial pacing cycle length of 130 msec. The corresponding St-HRA and HRA-LRA intervals of the propagated St were 50 and 65 msec, respectively. In panel B, the atrial pacing cycle length was 260 msec (twice that in panel A). This was followed by development of 1:1 St-At conduction. Note that the At fre-
Alternating Wenckebach Periodicity in the Atrium

Further shortening of the atrial pacing cycle length (St-St) after development of 2:1 St-At block resulted in conversion of 2:1 to 3:1 St-At block in five patients (cases 1, 2, 7, 10 and 11). One example is illustrated in figure 3. An atrial pacing cycle length of 228 msec appeared to be critical, as it resulted in conversion of 2:1 to 3:1 St-At block (following the 8th pacing St). This was preceded by gradual prolongation of both the St-HRA and HRA-LRA intervals (from 20 to 40 msec and from 95 to 190 msec, respectively). In addition, the HRA electrogram was noted to be fractionated before the conversion of 2:1 to 3:1 St-At block. Similarly, figure 4 demonstrates conversion of 2:1 to 3:1 St-At block resulting from a gradual decrease in the atrial pacing cycle length from 230 to 180 msec.

Discussion

In isolated preparations of rabbit atrium, Klein et al. illustrated that a premature electrical stimulus delivered to the atrium could reach and reset sinus nodal activity without being propagated to the nearby atrial recording site. This observation provided experimental electrophysiological evidence of concealed conduction in the rabbit atrial tissue. Langendorf et al. elegantly postulated, from the surface electrocardiograms of a patient with atrial parasystole, that an atrial premature impulse could induce atrial concealed conduction with penetration into the sinus node, thereby producing prolongation of subsequent
sinoatrial conduction. Similar clinical observations were made by Fleishmann and Louvros et al. Nevertheless, Langendorf recently pointed out that the occurrence of sinoatrial reciprocation, in those instances, remains a possibility. Thus, concealed conduction at the atrial level in man requires further confirmation.

Using the methodology introduced by Lewis and Master and Langendorf et al., along with the utilization and recordings of atrial electrograms, we demonstrated that intra-atrial conduction time (St-HRA and HRA-LRA intervals) became significantly lengthened during 2:1 St-At block compared with that during 1:1 St-At conduction at half the stimulus frequency (table 2 and figs. 1 and 2). This indicates that the nonpropagated St partially penetrated the atrium and in turn, delayed conduction of the subsequent propagated stimulus. One might ascribe prolongation

FIGURE 2. Intra-atrial conduction time during 2:1 stimulus (St)-atrium (At) block at an atrial pacing cycle length (St-St) of 130 msec (panel A) and during 1:1 St-At conduction at an atrial pacing cycle length of 260 msec (case 5). Panels A and B are not continuous. Atrial frequency is the same in both panels despite the fact that the St frequency in panel A is twice that in panel B. Note the HRA electrograms are more fragmented during 2:1 St-At block in panel A. Abbreviations: same as figure 1.

FIGURE 3. Alternating Wenckebach periodicity in the atrium (case 9). The presence of a left-sided accessory atrioventricular pathway accounts for predominantly positive QRS deflection in lead V. At a critical atrial driving cycle length (St-St) of 228 msec, 2:1 stimulus (St)-atrium (At) block converts to 3:1 St-At block after the eighth St. Note progressive fragmentation of the high right atrial electrogram accompanied by prolongation of intra-atrial conduction time before the conversion. Abbreviations: same as figure 1.
of the St-HRA interval during 2:1 St-At block to atrial latency. Nevertheless, simultaneous lengthening of the HRA-LRA interval unequivocally reflects prolongation of intra-atrial conduction time (table 2 and figs. 1 and 2). We realize that impulse transmission in the atrium is a non-lineal phenomenon, and that the HRA-LRA interval may represent only the difference in time of the onset of activation at two different atrial sites. Consequently, prolongation of the HRA-LRA interval during 2:1 St-At block could be the result of two independent manifestations of a same event — prolongation of the St-HRA and the St-LRA intervals respectively.

The occurrence of alternating Wenckebach periodicity is believed to imply multiple levels of conduction disturbance with various degrees of concealment in a conduction tissue. In this study, the demonstration of alternating Wenckebach periodicity occurring in the atrium in five of the 12 patients provides further evidence of concealed conduction in the human atrium. Unfortunately, the site of intra-atrial concealment could not be precisely determined, because only a limited number of atrial electrograms (HRA and LRA) were recorded. However, the following observations suggest that concealed atrial conduction was a local event occurring nearby the HRA stimulation site: 1) only the HRA electrogram became fractionated during 2:1 St-At block; and 2) both 2:1 St-At block and alternating Wenckebach periodicity occurred between the St and the HRA electrogram, but not between the HRA and LRA electrograms.

Concealed atrial conduction so demonstrated requires appropriate adjustment of the electrode catheter position during programmed atrial stimulation. Since the phenomenon of concealed atrial conduction could be observed in patients with and without abnormal functional properties of the atrium (prolongation of the P-A interval as well as the effective and/or functional refractory period of the atrium), the present study suggests that the occurrence of concealed conduction in the human atrium may not be uncommon, and subsequently it may be related to the genesis of specific atrial arrhythmias. For instance, AV nodal concealment frequently results in an irregular ventricular response during atrial flutter-fibrillation. An analogous situation is that a rapid ectopic atrial rhythm may electrocardiographically manifest itself an irregular atrial rate as a result of concealed atrial conduction. Additionally, when this occurs in association with prolongation of intra-atrial conduction time, the P wave may accordingly change its morphology, thereby mimicking a chaotic atrial rhythm. Further studies are necessary to substantiate these hypotheses.

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