Atrioventricular Conduction in Secundum Atrial Septal Defects

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SUMMARY
The atrioventricular (A-V) conduction time in patients with secundum ASD who had left-to-right shunts of 40–65% of their pulmonary flow was evaluated utilizing intracardiac electrograms. The A-V conduction time was divided into three components: (1) P-A interval, from the onset of the P wave to the time of excitation of muscle in the vicinity of the A-V node, (2) A-H interval, from the time of excitation of low right atrial muscle to that of the His bundle, and (3) H-V interval, from the time of the His bundle deflection to the onset of ventricular activation. The mean P-R interval of the ASD group (154 msec) was significantly longer ($P < 0.01$) than that of the control group (135 msec). The internodal conduction time (mean P-A interval) of the ASD group (52.2 msec) was significantly longer ($P < 0.001$) than that of the control group (29.1 msec). The A-H and H-V intervals of the two groups were not significantly different. Both the increased size of the atrium and the increased distance for internodal conduction produced by the defect itself can account for the prolonged internodal conduction time.

Additional Indexing Words: His bundle Atrioventricular node Intracardiac electrograms Intraatrial conduction

Prolongation of the P-R interval in patients with large secundum atrial septal defects (ASD) and large left-to-right shunts remains unexplained.\(^\text{1}\) Roberts and Olley\(^\text{2}\) recorded His bundle electrograms in seven patients with secundum atrial septal defects and found no abnormalities in conduction intervals measured from the onset of atrial activation to the His bundle wave form or from the His bundle wave form to the onset of ventricular activation. However, in their series of patients they were able to obtain His bundle wave forms only in those patients whose left-to-right shunts involved less than 50% of the pulmonary blood flow.

The purpose of this study was to determine, if possible, where the conduction time abnormality occurred in patients with large secundum atrial septal defects and known prolongation of the P-R intervals. Analysis of the electrograms evaluated three component parts of the total time of A-V conduction\(^\text{3}\): (1) from the onset of the P wave to excitation of atrial muscle in the vicinity of the A-V node, (2) from excitation of low right atrial muscle to activation of the His bundle, and (3) from His bundle excitation to the onset of ventricular activation. The results indicate that a significant contribution to the prolongation of the P-R interval is related to prolonged intraatrial conduction, i.e., an increase in time from the onset of atrial excitation to that of muscle in the region of the A-V node.

Methods
Eight children, five to 15 years of age, who had secundum atrial septal defects with left-to-right shunts comprising 40 to 65% of pulmonary flow estimated by Fick and radioisotope techniques\(^\text{4}\), had His bundle electrograms recorded at preoperative cardiac catheterization utilizing the method of Scherlag et al.\(^\text{5}\) which required less than 15 min. The diagnosis of secundum atrial septal defect was confirmed at surgery. To serve as controls, 16 patients, five to 15 years of age at preoperative cardiac catheterization, were studied who had P-R intervals that were normal for their ages. They had the following cardiovascular diagnoses: aortic stenosis, 5; ventricular septal defect, 2; Tetralogy of Fallot, 1; pulmonary stenosis, 4; coarctation of the aorta, 2; bicuspid aortic valve, 1; and pulmonary cyst, 1.

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The intracardiac electrograms were obtained as previously described. They were recorded between two electrodes 1 cm apart on a no. 6 Elecath Hexapolar catheter with an A-C Princeton preamplifier model 113. The output of the preamplifier went to a Sanborn Polybeam oscillographic and tape recorder system. The frequency of the entire system was flat from 30 to 1000 Hz.

The three standard leads were recorded at 50 mm/sec prior to obtaining the His bundle electrogram to ensure that the P wave axis was normal in all the patients included in the study. Lead II was selected as the lead to be recorded simultaneously with the intracardiac electrogram: the P-R interval in lead II was similar in duration or longer than the other leads and the P wave was consistently best delineated in lead II, enabling us to make a more accurate measurement of the onset of the P wave at the high recording speed used for the His bundle electrogram. In addition, we were influenced by animal (dog) studies which have related the epicardial atrial potential distribution to the body surface potential distributions.

For normal atrial excitation in the dog, potentials that were generated in the region of the chest comparable to lead II monitoring were the earliest to occur and were apparent within 4-8 msec of the earliest detectable excitation of atrial muscle. Our approach is given further support by the mean P-A and P-R intervals of our controls being quite similar to those published for children of the same age range.

The method used to measure the three intervals noted above is illustrated in figure 1. These were defined as the P-A interval, from the onset of the P wave in lead II to the first major rapid deflection of the atrial wave form of the intracardiac electrogram; the A-H interval, from the major rapid deflection of the atrial wave form to the rapid deflection between the maximum and minimum of the His bundle wave form; and the H-V interval, from the major rapid deflection of the His bundle wave form to the earliest deflection caused by the onset of ventricular activation recorded in either lead II or the intracardiac electrogram.

The P-A interval represents the time from the earliest detectable evidence of atrial muscle activation to the time of low right atrial activation. The A-H interval represents the time from the low right atrial activation to the activation of some part of the His bundle or proximal bundle branches. The H-V interval represents the time from activation of the His bundle to the onset of ventricular muscle activation. The sum of these intervals measured from the intracardiac electrograms was usually less than the P-R interval measured from the body surface in lead II (table 1) by a few milliseconds. This could be due to the proximity of the extracardiac fields produced by ventricular activation to the intracardiac leads compared to the distant position of the leads used to record lead II so that with onset of ventricular activation selective changes might occur in specific body areas several milliseconds before potentials are perturbed in lead II.

**Results**

The mean P-R interval of the ASD group was 154 msec compared to the control group's mean P-R interval of 135 msec (table 1). This was statistically significant ($P < 0.01$).

The intraatrial conduction time (P-A interval) of the ASD group ranged from 45 to 65 msec with a mean of 52 msec while that of the control group ranged from 22 to 40 msec with a mean of 29.4 msec. The prolonged P-A interval in the ASD group was statistically significant ($P < 0.001$). There was no difference in P-A interval related to size of shunt, and there was no difference in the P-A interval in the control group based on age.

The mean A-H interval of the ASD group (66.2 msec) and that of the control group (69.1 msec) were not statistically different. The mean H-V intervals of both groups are similar: ASD, 31.2 msec; control, 32.9 msec. This was not statistically significant.

**Discussion**

The major finding in this study was that the time of intraatrial conduction (P-A interval)—the time from earliest detectable electrical activity of atrial
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Table 1

Vital Statistics, Hemodynamic Data, P-R Interval, and Atrioventricular Conduction Times from the Intracardiac Electrograms of Patients Studied

| ASD Group | | | | | |
|-----------|-----------|-----------|-----------|-----------|
| Patient   | Age (yr) | % L-R*    | P-R (msec) | P-A (msec) | A-H (msec) | H-V (msec) |
| 1 JW      | 5        | 60        | 160        | 63        | 34         |            |
| 2 JA      | 5        | 50        | 145        | 56        | 32         |            |
| 3 VC      | 6        | 60        | 160        | 73        | 38         |            |
| 4 MF      | 6        | 65        | 165        | 85        | 30         |            |
| 5 DJR     | 7        | 60        | 125        | 50        | 27         |            |
| 6 RS      | 8        | 65        | 165        | 71        | 31         |            |
| 7 DJ      | 11       | 55        | 170        | 82        | 31         |            |
| 8 JB      | 15       | 40        | 145        | 50        | 27         |            |
| Mean 7.9  | ± 3.5    | ± 15      | ± 8        | ± 14      | ± 4        |            |

| Control Group | | | | | |
| Patient       | Age (yr) | dx | P-R (msec) | P-A (msec) | A-H (msec) | H-V (msec) |
| 9 TK          | 9        | VSD| 160        | 88        | 41         |            |
| 10 BH         | 7        | VSD| 145        | 86        | 32         |            |
| 11 CM         | 9        | T/F| 145        | 75        | 34         |            |
| 12 RA         | 9        | PS | 135        | 58        | 36         |            |
| 13 WT         | 6        | PS | 130        | 58        | 38         |            |
| 14 FY         | 7        | PS | 160        | 85        | 40         |            |
| 15 WW         | 15       | PS | 130        | 65        | 40         |            |
| 16 HH         | 13       | AS | 130        | 70        | 31         |            |
| 17 WJ         | 10       | AS | 120        | 57        | 30         |            |
| 18 PM         | 9        | AS | 130        | 75        | 28         |            |
| 19 RP         | 6        | AS | 130        | 68        | 28         |            |
| 20 AS         | 8        | AS | 130        | 66        | 33         |            |
| 21 TB         | 6        | Co | 140        | 70        | 33         |            |
| 22 TT         | 9        | Co | 145        | 82        | 25         |            |
| 23 LE         | 6        | P  | 115        | 50        | 30         |            |
| 24 RL         | 7        | A  | 120        | 58        | 28         |            |
| Mean 8.5     | ± 2.5    | ± 13  | ± 5        | ± 12      | ± 5        |            |
| = 1 sd       | < 0.7    | < 0.01 | < 0.001    | < 0.6     | < 0.4      | |

*Percent L-R is the component of pulmonary flow which is shunted blood.

Abbreviations: dx = diagnosis; A = bicuspid aortic valve; AS = aortic stenosis; Co = coarctation of the aorta; P = pulmonary cyst; PS = pulmonary stenosis; T/F = Tetralogy of Fallot; VSD = ventricular septal defect.

muscle to the time of activation of the muscle in the area of the A-V node—in atrial septal defects was longer than that of the control group which had types of congenital heart disease without anatomical defects of the atrial septum. Although the methods of this study do not allow the actual determination of the cause of the prolonged intraatrial conduction time in patients with secundum atrial defects, two major considerations seem important. First, it is generally agreed that the right atrium is enlarged in secundum atrial defects as compared to normal hearts and as compared to those without abnormalities of the atrial septum. Second, in patients with large left-to-right shunts due to secundum atrial defects, the defect itself produces a different anatomical arrangement over which wavefronts may propagate from the region of the S-A to the A-V node. Both the increased size of the atrium and the increased distance for internodal conduction produced by the defect itself would result in prolonged internodal conduction time if the conduction velocity of the atrial muscle was normal.

Gelband et al.9 showed that in the human atrial appendage the velocity of excitation wavefronts was 0.3 to 0.4 m/sec. At this constant velocity, prolongation of 6 to 8 mm of the shortest pathway between the sinus node and low right atrium would produce an increase in the P-A interval of 24 msec, an increase that is similar to that found in the ASD.
patients as compared to those with no known atrial septal abnormalities.

Although the increased internodal conduction time in these patients can most simply be explained by an increase in distance between sinus and A-V nodes, this explanation does not consider whether or not there are specialized preferential narrow conduction tracts between the sinus and A-V nodes, and if so, how such proposed narrow tracts might be affected by the underlying abnormality. In a large secundum atrial septal defect a major segment of the septum is missing and if a specialized narrow tract exists it would be located in this missing segment. This focuses on the problem as to whether the abnormally long internodal conduction times can be accounted for by the absence of the large area of muscle alone or by the absence of a small segment of the total area, a specialized tract.

Titus and Meredith have pointed out that one of the major difficulties in viewing the problem of the proposed internodal preferential tracts is semantic. An atrial preferential conducting tract must be defined as a narrow area of specialized tissue which conducts faster than and which is insulated from the surrounding atrial muscle. That is to say, a well-defined tract must be well insulated; frequent low resistance connections (the nexus) between cells make it electrically impossible for adjacent fibers to depolarize asynchronously. Detailed excitation maps of the atrial septum of the dog and rabbit showed no evidence of such tracts; rather excitation waves propagated as broad wavefronts which extended to the limits of the anatomical structures involved. In our opinion, there is no functional evidence for internodal tracts thus defined.

A working definition of an internodal pathway is more difficult. The gross anatomy of the atrial septum consists of three broad areas of atrial muscle in the human, chimpanzee, and dog, and two broad areas of muscle in the rabbit. These areas provide the pathways for propagation of excitation waves between sinus and A-V nodes. The histological studies of James and Takayasu showed that if one starts at the sinus node in human and dog atria, there are three general routes that can be traced where cell-to-cell connections are maintained until the three routes merge in the area of the A-V node. As we understand it, James has never said that there were narrow atrial tracts, but others following the definition presented above, have suggested the presence of narrow specialized internodal tracts. We think that the data of James for the dog atrium and the excitation maps of Spach et al. are in agreement as to the presence of three general atrial septal anatomical regions for propagation: these three pathways are the crista terminalis, the midseptum including the limbus, and the anterior arch of the crista and the anterior septum.

Spach et al. have proposed that normal and abnormal sequences of atrial septal excitation can be accounted for by the presence of these three general routes of propagation between sinus and A-V nodes in the dog and two such routes in the rabbit. That is to say, excitation waves propagate throughout these structures (pathways?) which together comprise the gross anatomy of the atrial septum. However, the excitation maps showed that in any specified region of the atrial septum conduction velocity is not constant but varies depending upon the direction in which the excitation wave is propagated through it. For example, one area may be the most rapidly conducting one during antegrade conduction and the slowest during retrograde conduction.

To resolve the question as to whether excitation waves propagate through broad anatomical structures or through narrow specialized preferential tracts requires a large number of detailed measurements throughout the entire area in which there might be a narrow tract. If measurements are made at only a few sites, the total excitation wave cannot be constructed experimentally, and irregularities in the wavefront such as would be produced by a preferential tract cannot be clearly determined.

Clearly, the methods used at cardiac catheterization are inadequate to provide data as to the presence or absence of specialized tracts. However, any large atrial defect of the secundum type alters a large segment of the anatomy of the atrial septum (especially the limbus of the fossa ovalis) and secondarily increases the distance between the sinus and A-V nodes due to the atrial enlargement required to accommodate the hemodynamic overload. In our opinion, the prolonged conduction times can be explained most simply by longer routes (pathways?) without the requirement of a specialized preferential narrow tract that might have been located in the area of absent muscle.

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