Evidence of Concealed Atrioventricular Conduction in Man

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THE TERM "concealed conduction" was introduced by Langendorf\(^1\) to describe the phenomenon of entry of an electrical impulse into the A-V node without complete transmission but with modification of conduction of a subsequent impulse. This concept unified diverse observations of earlier years concerning disturbances of A-V transmission\(^2\) and clarified the mechanisms of many arrhythmias.\(^5\) \(^6\)

Subsequent studies in animals have proved the occurrence of concealed conduction in the form of impulses which only partially penetrate the A-V nodal system.\(^7\) Most recently, Langendorf and his co-workers\(^8\) have conducted experiments in man which conclusively prove the occurrence of concealed conduction in patients with disease of the A-V conduction system.

The present study became possible because 2:1 A-V conduction occurs in many healthy persons with normal A-V nodes when the right atrium is paced at rates only moderately above the intrinsic sinus nodal rate. This phenomenon permits observations of A-V nodal conduction time with and without an interposed atrial depolarization at comparable ventricular rates. The present experimental approach, which is a variant of that originally used by Lewis and Master,\(^2\) is applied here to the study of normal human A-V conduction.

Methods

Five persons from a group of 14 who underwent right atrial pacing studies exhibited 2:1 A-V conduction when right atrial pacing was conducted at rates between that of their sinus node and 150 per minute.

One (A.K.) was a 40-year-old woman with moderate congenital valvular pulmonic stenosis (pulmonic valvular area of 1.3 cm\(^2\)). Another (J.S.) was a 15-year-old athletic boy with a pressure gradient of 15 mm Hg across a coarctation of his aorta. The third (A.M.) was a 15-year-old athlete who had a tiny ventricular septal defect. One (T.H.) was a 16-year-old athlete with an innocent pulmonic flow murmur. The last (M.R.) was a 29-year-old female informed volunteer, who had no heart disease. Their base-line electrocardiograms showed P-R intervals and sinus rates of 0.14 sec at 60 beats per minute, 0.19 at 85, 0.06 at 58, 0.20 at 50, and 0.15 sec at 71 beats per minute, respectively.

The first four were studied during complete diagnostic cardiac catheterization; the last one had right atrial pacing only. In each instance, an electrode catheter was placed in the right atrium with the tip contacting the mid-lateral wall of the right atrium; this position remained constant throughout the study of each individual. In this position, pacing could be conducted with impulses of less than 2 volts from the battery-powered generator, producing a P wave after each impulse.

The impulse rate was changed frequently between levels just above the sinus nodal rate and approximately 150 per minute. An electrocardiographic lead, usually lead II, was chosen which best displayed the pacing impulse and the electrocardiographic P waves. Recordings were made at a speed of 25 mm/second. Measurements were made to the nearest 0.01 sec of the pacing rates, the ventricular rates, and the P-R intervals. The P-R interval was taken as the time between the onset of the pacing impulse and the onset of the electrocardiographic QRS complex. For this reason even the P-R valves at the slowest paced rates were longer than those of the base-line electrocardiograms. The P-R interval changed during the three cardiac cycles which followed each change in rate. Thereafter, the P-R interval remained stable for several minutes at least. All measurements were made at least 10 sec after a change in rate; most were made between 20 and 30 sec after a change.

Each of these five subjects displayed second degree block at some rate above their sinus nodal...
rate. This included diverse A-V ratios in the form of Wenckebach periods and 2:1 A-V block. The arrays of P-R interval plotted against heart rate were treated by regression analysis.

Results

The expected direct linear relationship between P-R interval and heart rate occurred with highly significant correlation in four of the five subjects. In T.H., a highly conditioned athlete, A-V block appeared at a paced rate of 70 per minute and prevented accumulation of sufficient rate and P-R data to prove the same relationship. This phenomenon has been described previously.9

Representative examples of electrocardiograms at differing rates are shown in figure 1. The P-R interval is 0.16 sec at a rate of 75 beats per minute and increases to 0.19 sec at a rate of 88 beats per minute. The bottom electrocardiographic strip demonstrates an episode of 2:1 A-V block. This instance has a ventricular rate identical to that in the top strip, but has a P-R interval which is longer by 0.04 sec.

![Figure 1](image)

Electrocardiographic lead III obtained from patient M.R. At each faster atrial pacing rate, P-R prolongation occurred. With 2:1 A-V conduction (bottom strip) a ventricular rate identical to that in the top strip occurred, but the P-R interval was 0.04 sec longer.

![Figure 2](image)

P-R interval of each patient is plotted in a separate panel. As the paced atrial rate was increased, a linear increase in P-R interval occurred (solid circles). A-V conduction time was longer for a given ventricular rate when 2:1 conduction occurred (denoted by +).
The graphic displays of P-R interval plotted against the ventricular rate are shown in the five panels of figure 2. The P-R intervals with 2:1 A-V conduction are consistently 0.03 to 0.13 sec longer than those expected with 1:1 conduction. This difference is attributable to an effect of the intervening P wave in 2:1 A-V block. Yet the intervening P waves themselves were not detectably different.

Discussion

The data presented show the usual relationship between heart rate and A-V conduction time provided 1:1 conduction prevails during substitution of an external pacemaker for the intrinsic rhythmic tissues. When 2:1 conduction occurs, however, A-V conduction of the effective atrial impulse is inappropriately prolonged.

This excessive prolongation is attributable to an effect originating with the previous atrial depolarization. Hoffman and co-workers have shown in animals that such impulses may penetrate the A-V node and bundle of His system for a variable distance without causing ventricular depolarization. These impulses do, however, modify A-V nodal tissue excitability and, therefore, retard conduction of the subsequent impulse.

The present studies provide evidence of concealed conduction in five individuals, three of whom had normal hearts.

Summary

The occurrence of 2:1 A-V block during right atrial pacing in man provided the opportunity to compare A-V conduction time with and without an intervening atrial depolarization at similar ventricular rates. The presence of the interposed atrial depolarization produced prolongation of the subsequent A-V conduction by 0.03 to 0.13 sec, and thus provided evidence of concealed conduction in the normal human heart.

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References


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