CASE REPORT

Determination of His-Purkinje Refractoriness in Man with His Bundle Pacing

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SUMMARY Consistent His bundle pacing was accomplished in one of 60 consecutive patients. Intracardiac electrophysiologic studies in this patient revealed rapid atrioventricular nodal conduction (AH = 55 msec). While the relative refractory period of the His-Purkinje system as a whole determined by His bundle pacing (using the extrastimulus technique) was identical to that determined by atrial pacing, changes in right bundle branch refractoriness differed. An atrial extrasystolic interval of 370 msec resulted in right bundle branch block, whereas direct His pacing at a shorter extrasystolic interval (360 msec) failed to produce a bundle branch block pattern. The effective refractory period of the His-Purkinje system could not be determined by atrial pacing because of atrial refractoriness, but was obtained by His bundle pacing.

Theoretically, direct His bundle pacing may be of value in determining His-Purkinje system refractoriness when this parameter is unobtainable due to either atrial or atrioventricular nodal refractoriness. This technique appears to have limited clinical usefulness, however, because of the very small success rates. Finally, His-Purkinje system refractoriness may differ, depending on site of stimulation, presumably due to differing inputs into the His-Purkinje system.

HIS BUNDLE PACING has been cited as the most reliable means of validating His bundle potentials during intracardiac recordings. Consistent His bundle pacing in man has been difficult, primarily because of problems in maintaining a stable catheter position in the beating heart. Narula, for example, suggested that only three to five consecutive His bundle paced beats constitute adequate validation of the His bundle recordings. We report for the first time a patient in whom consistent His bundle pacing was accomplished, allowing us to determine His-Purkinje refractory periods.

Materials and Methods

Intracardiac electrograms using standard techniques were obtained. Quadripolar catheters were positioned against the right atrial wall and across the tricuspid valve in order to record the His bundle potential. Intracardiac electrograms and external Frank leads X, Y and Z of the orthogonal system were simultaneously displayed on an oscilloscope and recorded on an Electronics for Medicine (DR-12) recorder (White Plains, New York). Cardiac refractory periods were obtained by the extrastimulus technique by introducing progressively premature stimuli (2 msec wide and at 10-msec decrements) after every fifth beat by means of a programmable stimulator (Bloom Associates, Philadelphia, Pennsylvania). His bundle pacing was accomplished by pacing from the pair of electrodes in which His bundle potentials of greatest amplitude were recorded. Criteria for assessing adequacy of His bundle pacing included a stimulus-to-ventricular depolarization time identical to the infranodal conduction time during spontaneous rhythm and similar QRST morphology between spontaneous and His bundle paced beats.

Results

His bundle pacing was attempted in 60 consecutive patients undergoing electrophysiologic studies. In only one patient was consistent His bundle pacing sufficient to enable determination of the refractory periods of the His-Purkinje system. This patient is the basis of our report.

Case Report

A 42-year-old man was admitted to the San Francisco General Hospital Medical Center on March 16, 1977, for evaluation of a 6-month history of episodic palpitations and dizziness. The surface ECG revealed a PR interval of 100 msec and a narrow QRS, and rhythm strips showed episodic atrial flutter with 1:1 conduction with a ventricular response of 280 beats/min. Control studies showed an atrioventricular nodal conduction time (AH) of 55 msec and an infranodal conduction time (HQ) of 35 msec. Atrial overdrive pacing at a paced cycle length of 290 msec resulted in 1:1 atrioventricular conduction with an AH interval of 130 msec (fig. 1). At an atrial paced cycle length of 500 msec, late atrial extrastimuli were associated with normal infranodal conduction. A plot of atrial test cycles (A1,A2) against corresponding His
bundle depolarizations (H₁H₂) produced a smooth curve, indicating either that there was a single “fast” atrioventricular nodal pathway, or that if a “slow” pathway was present, its refractory period was longer than the fast one. A premature atrial depolarization coupled 370 msec after the last driven atrial complex resulted in slight prolongation of infranodal conduction time associated with aberrant ventricular conduction (fig. 2). More premature stimuli failed to stimulate the atrium. The ventricular effective refractory period determined at a paced cycle length of 580 msec was 220 msec.

Overdrive His bundle pacing resulted in narrow QRS complexes similar to those recorded during spontaneous rhythm except for slurring of the initial portions of the QRS deflection. Progressively premature His bundle depolarizations induced at a paced His cycle length of 580 msec resulted in prolongation of the stimulus-to-ventricular depolarization time at coupling intervals of 370 and 360 msec (figs. 3A, B and C). The effective refractory period of the His-Purkinje system was 350 msec (fig. 3D). The difference in right bundle branch refractory periods comparing atrial vs His bundle pacing should be

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**Figure 1.** The left panel depicts simultaneous recordings of surface leads X, Y and Z of the Frank orthogonal lead system, right atrial (RA), coronary sinus (CS) and His bundle electrograms (HBE) during sinus rhythm in our patient. The atrioventricular nodal conduction time (AH) is 55 msec, while infranodal conduction time (HQ) is 35 msec. The right panel shows atrial overdrive pacing (S₁S₂) at a cycle length of 290 msec with 1:1 conduction and lengthening of the AH to 130 msec.

**Figure 2.** The atria were driven (S₁S₂) at a constant cycle length of 580 msec in our patient. An atrial premature stimulus (S₃) was introduced 370 msec after the last driven beat and results in slight prolongation of infranodal conduction (H₂Q₃) (from 35 msec to 45 msec) and aberrant ventricular conduction. The relative refractory period of the His-Purkinje system was 375 msec. Earlier atrial extrastimuli (not shown) failed to result in atrial depolarization. A₁ = atrial depolarization during the basic drive rate; A₂ = atrial depolarization associated with the premature beat; H₁ = His bundle depolarization during the basic drive rate; H₂ = His bundle depolarization associated with the premature beat; RA = right atrial electrogram; HBE = His bundle electrogram.
noted. An atrial extrasystolic interval of 370 msec (fig. 2) results in right bundle branch block, but direct His bundle pacing at an even shorter extrasystolic interval (360 msec) (fig. 3C) fails to produce a bundle branch block pattern.

In summary, this patient displayed electrophysiologic abnormalities characteristic of accelerated atrioventricular nodal conduction. There was excellent agreement in the measurement of the relative refractory period of the His-Purkinje system

**Figure 3.** The His bundle was driven at a paced cycle length of 580 msec in our patient. The initial portion of the QRS was deformed either due to near-simultaneous atrial activation or to simultaneous activation of the high basal ventricular septum (see text). A) A premature His bundle depolarization was introduced 460 msec after the last driven stimulus. B) An earlier premature stimulus ($S_1S_2 = 370$ msec) fell in the relative refractory period of the His bundle and resulted in prolongation of His-Purkinje conduction ($S_2V_2 = 70$ msec). C) Further prolongation of His-Purkinje conduction results from a premature stimulus 360 msec after the last driven beat. D) Failure of an earlier (350 msec) premature stimulus to conduct to the His bundle is demonstrated. RA = right atrial electrogram; CS = coronary sinus electrogram.
using either atrial or direct His bundle pacing techniques, but there were obvious differences in determination of right bundle branch refractoriness. The latter observation suggests that His-Purkinje system refractoriness may differ according to pacing site.

Discussion

We found that His bundle pacing was sometimes associated with slight changes in QRS morphology during pacing. Initial slurring of the QRS complex was probably related to simultaneous activation of either the ventricular septum or the atrium. This finding suggests that nonselective His bundle pacing was operative in this patient. Thus, in figure 3, during the drive rate and with premature beats late in the cycle, both the His bundle and the adjacent ventricular septum might have been paced, whereas with earlier premature beats, selective His bundle pacing was operative. Williams et al., however, confirmed the validation of His bundle activation in the presence of combined activation of the His bundle and high basal ventricular septum in that ventricular depolarization proceeded via the His-Purkinje system.

Our studies support these observations in that there was a marked difference in refractory periods when His bundle pacing was compared with direct pacing from the right ventricular apex, suggesting that local high septal depolarization was insufficient to depolarize the ventricular muscle mass. Slight changes were also observed in the ST-T complex, as pointed out by Williams et al. In addition, slight changes in QRS contour during His bundle pacing were observed and were most likely related to simultaneous pacing of the high ventricular septum. Finally, if direct His bundle pacing results in asynchronous activation of longitudinally dissociated tracts within the common bundle, then asynchronous activation of the ventricles may be produced.

The relative refractory period of the His-Purkinje system is determined using the atrial extrastimulus technique; it is determined as a whole and defined as the extrasystolic His interval resulting in either HQ prolongation or bundle branch block. In our patient clear differences in right bundle branch refractoriness were apparent between the atrial and His bundle pacing approach. This finding suggests that measurement of His-Purkinje system refractoriness may be influenced by the pacing site. One explanation is that pacing from the atrium results in a different input into the His-Purkinje system compared with that from direct His bundle pacing, leading to differences in right bundle branch block refractory periods. Batsford et al., for example, reported that atrioventricular nodal conduction times and refractoriness were altered by a change in the site of atrial stimulation, and postulated that the atrial stimulation site may influence atrioventricular nodal function by changing the site and/or mode of entry of the impulse into the atrioventricular node. We postulate that differing inputs into the His-Purkinje system may result in differences in refractory period determinations. Thus, different stimulation sites may yield different His-Purkinje system refractory period measurements.

We believe this is the first reported case of determining His-Purkinje refractory periods by direct His bundle pacing in man. Inconsistent pacing of the His bundle probably results from catheter movement. We cannot explain our success in this patient.

Determination of His-Purkinje system refractory periods by His bundle pacing enabled determination of His-Purkinje system effective refractory periods in a patient in whom we could not obtain such measurements by standard methods. This technique, however, appears to be of limited clinical usefulness in view of the very low success rate.

References

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R W Peters, M M Scheinman, S Raskin and J Desai

Circulation. 1979;60:956-959
doi: 10.1161/01.CIR.60.4.956

Circulation is published by the American Heart Association, 7272 Greenville Avenue, Dallas, TX 75231
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Print ISSN: 0009-7322. Online ISSN: 1524-4539

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