Demonstration of Diffuse Conduction Disturbance in Sick Sinus Syndrome Utilizing Simultaneous His Bundle Electrogram and Timed Vectorcardiogram*

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The sick sinus syndrome is a clinical entity characterized by disturbances of sinoatrial impulse formation and conduction. Recent studies have demonstrated that abnormalities of conduction and automaticity at other specialized anatomic sites are common in subjects with symptomatic sinus node disease. We describe here the use of combined His bundle electrography and timed vectorcardiography as a method for detecting panconduction tissue disease in such a patient.

Figure 1 shows the simultaneously recorded His bundle electrogram and leads 1, 2, and V1 of the electrocardiogram, along with frontal and horizontal timed biplane Frank vectorcardiograms in a 63-year-old man with coronary artery disease and syncope. The patient manifested a sinus bradycardia which varied from 48 to 56 beats per minute. The QRS complexes so recorded manifested electrophysiologic evidence of inferior and anterior wall myocardial infarctions. The duration of the QRS complex was 130 msec with anterior and rightward delay in inscription, diagnostic of complete right bundle-branch block. The His-ventricle interval was prolonged to 70 msec (normal, 35 to 55 msec), indicating additional conduction delay in the left bundle branch (bilateral bundle-branch block). The second QRS complex in Figure 1 was followed by an unexpected pause with a measured P-P interval of 1.9 seconds. This degree of sinus slowing was diagnostic of a sick sinus node. The third P wave was accompanied by an "A" deflection on the His bundle electrogram yet was blocked in the atrioventricular nodal area, as the "A" was not succeeded by...

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Figure 1. Simultaneously recorded His bundle electrogram (HBE), leads 1, 2, and V1 of ECG, along with timed frontal-plane (FP) and horizontal-plane (HP) biplane Frank vectorcardiograms in 63-year-old man with coronary artery disease. Successive conducted QRS complexes are numbered.
an “H” spike. Another prolonged P-P cycle of 1.6 seconds was observed prior to resumption of atrioventricular conduction (Fig 1, QRS 3).

The tracings described in this report indicate that some patients with disturbances of sinoatrial impulse formation may have diffuse abnormalities of their specialized conduction tissue. Indeed, limited pathologic studies in such subjects have demonstrated fibrosis within and around the common atrioventricular bundle, involvement of the atrioventricular node, and sclerosis of the peripheral left bundle-branch fibers. To our knowledge, the case described here is the first in which simultaneous sinoatrial impulse dysfunction, atrioventricular nodal block, and bilateral bundle-branch block have been demonstrated during the patient's natural rhythm. The association of inferior wall myocardial infarction and sinoatrial disease is established and has been attributed to sinus nodal ischemia or right atrial infarction, or both. The diffuse nature of such disease warrants permanent ventricular pacemaker insertion in symptomatic patients.

In conclusion, simultaneous recording of the His bundle electrogram and timed biplane vectorcardiogram is a useful technique for graphically displaying the panconduction tissue disturbance in selected subjects with the sick sinus syndrome.

REFERENCES