

His Bundle Recordings*

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When we record atrial activity on the His bundle electrogram we are recording from the area of the atrium which is around the tricuspid valve, that is, the lower part of the right atrium. The normal range of conduction time from the region of the sinus node to the low right atrium is about 25 to 45 milliseconds. When we are at the A-V junction we can record the local atrial activity, the His bundle potentials, and the ventricular activation. Normally, we record this simultaneously with three surface EKG leads. We are using this technique for quantitative measurements, because it enables us to know where the P wave and the QRS complex begins, and depending upon what EKG lead you chose, it could vary the onset of these complexes. The three leads are Lead I, AVF, and V1, which give us the three plains combination: frontal, sagital, and horizontal. A breakdown of the PR intervals which we measure are: (1) the PA, or the intra-atrial conduction from the sinus node down to the low right atrium; (2) the AH, or the atrial activation to the His bundle and represents conduction through the A-V node (a normal range for this time in our laboratory is 50 to 120 milliseconds); and (3) the HV, which is measured from the beginning of the His bundle to the earliest ventricular activation seen either on the EKG or on the bipolar electrogram. In our laboratory the normal values for this are about 35 to 45 milliseconds. The His bundle deflection per se, is about 15 to 20 milliseconds in duration, indicating how long it takes for the impulse to go through the main His bundle before it bifurcates.

Up to now it has been taught that the left bundle activates the myocardium earlier than the right bundle branch or that the septum gets activated earlier from the left side than the right. The present data which we have collected by recording from both right and left heart in man as well as from original experimental work done by Hoffman's laboratory and by Rosen in a Chicago laboratory indicates that impulse arrives at the terminals of the left and the right bundle branches at the same time. Also, the anterior and posterior divisions of the left bundle receive the impulse at

the same time. This means that if we were to damage one bundle branch, there should be no delay in the impulse arrival to the ventricle. Thus it would follow that if we have a unilateral bundle branch block the HV time should not increase. It is considered now that we have three branches: the main right bundle and two divisions of the left. Therefore, if we were to damage either of the two of these three, we still should have normal HV time because the undamaged branch would take the impulse to the ventricle without any delay. We found clinical evidence to support this new data when we studied about 29 patients who had left axis deviation with a narrow QRS complex yet maintained normal HV time. There were also indications that if we produced rate-dependent right bundle branch block through atrial pacing, the HV time would not change. In one patient atrial pacing was done at a rate of 150 beats per minute. This produced second-degree Wenckebach type of block at the A-V node and right bundle branch block. The QRS complex following the blocked P wave was narrow. The H-V time in this beat with a narrow QRS complex was the same as in the following beat which showed right bundle branch block. In studies of the left side, the same phenomena was observed. We had a patient who had a rate-dependent left bundle branch block and the HV time did not change when the QRS complex changed from narrow to left bundle.

Further, we have studied about 25 to 30 patients with pure right bundle branch block. Up to this time the feeling was that most of the patients with right bundle branch block should have a normal HV time. But 20 percent of the patients with pure right bundle branch block show abnormal HV time. This indicates that these patients have additional disease either in the main stem His bundle or in the left main bundle or its two subdivisions. This would explain why statistically 70 percent of patients with right bundle, left axis deviation show abnormal HV time. They are presumed to have additional disease elsewhere. Since this is not reflected in the EKG, what we see is the dominant one-bundle lesion which is the most diseased one.

For years it has been said that whenever you see a combination of first-degree block and right bundle branch block, it indicates bilateral bundle branch

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block. But this is not always the case. The major delay which produces first-degree block occurs in the A-V node, not in the His-Purkinje system. Patients may have additional disease in the Purkinje system, but the major part comes from the delay in the A-V node. So first-degree right bundle doesn't always mean bilateral bundle branch block. The only way to validate the His bundle potential is by His bundle stimulation. Right bundle deflections look like His bundle, and there is no way to differentiate these except by this means. This is very important when we are making quantitative analysis and diagnosis, because if you have a wrong deflection the diagnosis is worthless.

We studied a patient who for several years had right bundle branch block and a normal PR. On the day of admission, he showed 2:1 A-V block with right bundle branch block. That evening he showed a transient episode of complete heart block, and again the QRS complex showed right bundle branch block. The EKG made us suspect that the block was occurring somewhere in the A-V node, because the complex was identical and the patient had only right bundle without any axis deviation. When we studied him four days afterwards, he was showing 2:1 A-V block, the same as when he was admitted. On the EKG, every P wave was blocked beyond the His bundle deflection, and the conduction time through the A-V node, that is the AH time, was completely normal—60 milliseconds. In the beats which were conducted, the HV time was prolonged at 85 milliseconds. The His bundle deflection was about 35 milliseconds. What was happening was

that the lesion which produced the block was in the main stem His bundle. Unfortunately, His bundle has been ignored for a long time as far as lesions are concerned because they do not show anything on the EKG. And for that reason some people have never made a diagnosis of His bundle blocks. But if right and left bundle produce lesions, why should the His bundle be immune to pathology since it is the same tissue. I believe about 40 percent of the patients with bundle branch lesions have disease in the main stem His bundle, and I think that as we collect more data we will be able to stress this.

We have studied about 90 patients with right bundle branch block, left axis deviation. As I suggested earlier, if there is a lesion in either of the two divisions the HV time theoretically should be normal. His bundle records in a patient with right bundle, left axis and normal PR showed a normal HV time of 40 milliseconds. In another patient under the same conditions we observed the HV time at 75 milliseconds. This indicates that these patients are not a homogeneous group and may explain why some patients with right bundle, left axis have gone for ten years without an A-V block and some develop it within a year. It follows that some of them have a significant disease which is not reflected in the EKG's and others have no disease in the third division.

We have observed that when the H-V time is abnormal, the third division is damaged. A patient came to the E.R. with second-degree A-V block. We brought him to the cath lab with 1:1 conduction and at that time the first beat on the surface EKG showed right bundle, left axis deviation. The His recording revealed an abnormal H-V time of 75 milliseconds which indicated that we should expect disease elsewhere in the conduction system. Our expectations were corroborated when the EKG spontaneously showed us right bundle, right axis deviation in the next two beats.

We have studied 25 patients with right bundle branch block, right axis deviation, and all of them except one showed abnormal HV times. Statistically this indicates that in patients with right bundle, right axis deviation the posterior division is damaged and there is additional disease which extensively involves the main His bundle or the anterior division.

PANEL DISCUSSION

Questioner: Dr. Narula, did I understand you to say that the majority of patients with right bundle, left axis had abnormal HV times, and does this mean that they have disease here more than the right bundle itself?

Dr. Narula: Clinically, it is felt from the EKG pattern that right bundle, left axis deviation means right bundle branch block and damage of the anterior division. Right bundle branch block with right axis

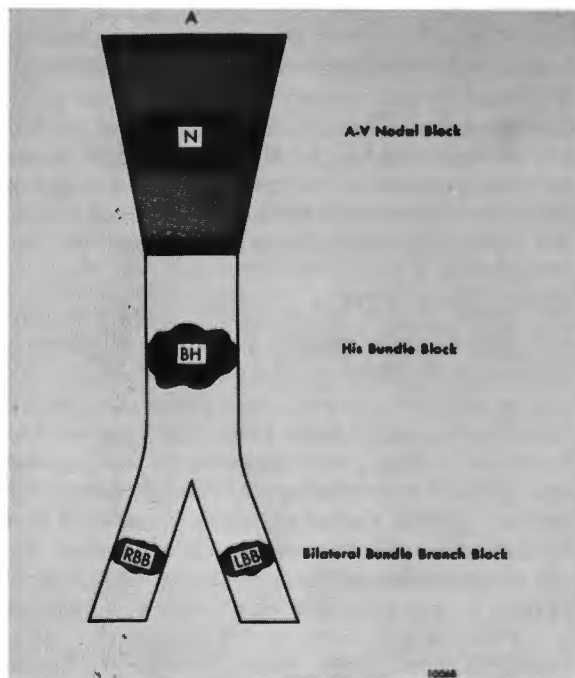


Fig 1—Illustration of His bundle block.

deviation means damage in the right bundle and the posterior division. This is what has been proposed by Rosenbaum, and theoretically it is right. In reality this is not always true, however. It is misleading to assume from the surface EKG pattern that a right bundle, left axis deviation indicates that a third division, i.e. the posterior division, is okay. Seventy percent of these patients have additional disease elsewhere, and probably in the posterior division itself, which we can't see from the EKG. The right bundle, right axis theoretically could be normal, but statistically this is unlikely, and the majority of the patients have additional disease elsewhere involving either the anterior division or the main stem His bundle. Just to reemphasize that point again: there are three divisions. If disease is present in only two of the three divisions, the HV time should be normal. Ninety-five percent of the patients with right bundle, right axis have abnormal HV time; therefore, disease is probably present in the third division as well, despite the fact that the EKG doesn't show it.

Questioner: Are you giving pacemakers to patients who are asymptomatic but the EKG shows right bundle, left axis deviation?

Dr. Narula: As yet, we do not make a decision to implant a pacemaker based on His bundle recordings. We rely instead on the clinical symptoms the patient is having, a history of syncope, and an abnormal HV time. Some day I hope we will have enough data to show that patients with abnormal HV time develop complete heart block in three years. Then we can give pacemakers prophylactically.

Dr. Samet: What if we have a patient with right bundle, left axis, right bundle, right axis and a normal heart rate, and he has a syncopal episode? Can we automatically assume that his syncopal episode is related to his conduction disturbance? We have had at least two patients with right bundle, left axis and one patient with right bundle, right axis who have come in with syncopal episodes and the referring physician was pressing for the insertion of a pacemaker. In these three cases, His bundle study revealed a normal HV time, but in none of them was a pacemaker implanted on this basis. We think this is a correct decision and we are following this approach now.

Dr. Dreifus: I think that what Dr. Samet said is right. The patient should have symptoms before you put in the pacemaker whether it is first-, second-, third-, or fourth-degree A-V block. Lenégre did a good deal of anatomic studies, and he makes it clear that it doesn't make much difference whether block is above or below. It is the importance of the symptoms based on block. There are many patients walking around with high-grade block in which there is no conduction from atria to ventricle who do not have symptoms. The minute symptoms develop and block is present, then

I think it is time to put in pacemakers. If you do not prove block is the cause of the symptoms the decision rests on a clinical diagnosis. I think there are other ways to prove whether the symptoms are due to block. You can use exercise stress tests, drugs, or tape recordings, and a number of these patients will show intermittent periods of nonconducted beats. Then the decision whether or not to implant a pacemaker can be made on that basis.

Dr. Narula: I just want to clarify a point. Dr. Dreifus just said that you can stress the conduction system to bring out the block whether by drugs or by exercise. The His-Purkinje system is so immune to some of these stresses that if you were to increase the rate by pacing or by giving atropine, the majority of these patients would still conduct 1:1, despite severely prolonged HV time. For some strange reason it acts in such a capacity that either it conducts or it doesn't, so that neither atrial pacing nor drugs will stress the system. When patients with 1:1 conduction do develop second-degree A-V block at high atrial pacing rates, it almost always occurs in the A-V node. Only a few cases develop block distal to the His bundle with increase in atrial rate and most of these latter cases even spontaneously manifest second-degree block. The degree of block in the patient with His-Purkinje system lesions will increase, but if he has 1:1 conduction it usually doesn't. We have had patients who came to the E.R. with complete heart block. We brought them to the cath lab, put in a temporary pacemaker, and within two hours we were able to pace them 1:1 up to 160, and they had HV times of 75 milliseconds. So I think atrial pacing might be misleading in checking the condition of the conduction system.

Questioner: Dr. Narula, regarding control of the HV interval which you have been placing so much reliance on. Have you studied the same patient a number of times, knowing what the previous HV interval was, so that you could understand what variance you are getting?

Dr. Narula: We had follow-up studies on the patients with normal conduction times and also on the patients with abnormal ones. For some strange reason the HV interval seems to be exactly reproducible, but not the AH. The AH interval varies from sitting to sitting, and from time to time you recatheterize the patient. But the HV is the same whether you recatheterize them a week or a month afterwards. We have studied at least 100 patients in this length of time where the purpose was to observe the progress of disease. The majority of them have not shown even a 5 millisecond change as yet. So the test, as far as HV is concerned is reproducible, provided you validate that the His bundle is what you are recording.