

# The Critical Use of the His Bundle Electrogram\*

BENJAMIN J. SCHERLAG, Ph.D.

*From the Division of Cardiology, Department of Internal Medicine, Mount Sinai Medical Center, Miami Beach, Florida, and the Department of Medicine, University of Miami School of Medicine, Coral Gables, Florida*

The technique for electrode catheter recording of consistent and stable His bundle activity presently used in the clinical laboratory was initially developed as an investigational tool (10, 11). In the clinical laboratory, the His bundle electrogram, in conjunction with the surface electrocardiogram—a technique that has been termed His bundle-electrocardiography by Castellanos (2)—has provided a more accurate means of localizing the site of conduction abnormality in patients with various forms of heart block. In addition, this technique has been utilized to study various physiological and pharmacological interventions (6, 12), arrhythmias (7), and the Wolff-Parkinson-White syndrome (3, 4).

Unfortunately, published reports have contained a variety of terms applied to the various intervals representing conduction through the atrioventricular transmission system. Moreover, the procedures for quantitating critical conduction measurements made with the use of His bundle-electrocardiography have relied heavily upon the shape of a recorded deflection during the P-R segment and its temporal relationships to atrial or ventricular activity. In view of the numerous reports using His bundle recordings that are presently appearing in the literature, a critical evaluation of the His bundle electrogram and some of its applications seems appropriate.

Figure 1 shows an anterior-posterior view of the heart and the position of an electrode catheter during the recording of His bundle electrograms.

---

\* Presented by Dr. Scherlag at the Symposium on Cardiac Arrhythmias, June 8, 1972, at Virginia Beach, Virginia. Please send requests for reprints to: Dr. B. Scherlag, Mount Sinai Medical Center, 4300 Alton Road, Miami Beach, Florida 33140.

In general, the His bundle electrogram is recorded by the use of a standard bipolar pacing catheter with ring electrodes 5 mm or 10 mm apart. The catheter is introduced from the femoral vein into the right heart and stabilized at the A-V ring at the base of the posterior tricuspid leaflet.

In figure 2A, electrograms from the high right atrium and from the His bundle area are shown with simultaneously recorded ECG leads. As opposed to standard electrocardiographic recordings which are usually made with relatively wide frequency response settings, that is, 0.1 to 200 Hz, the His bundle electrogram is commonly recorded with narrower band width limits to accentuate rapid deflections and attenuate slower waves, that is, 40 to 200 or 40 to 500 Hz. For this illustration, another bipolar catheter in the right atrium near the sinus node recorded atrial activity at the onset of the P wave. The His bundle electrogram, in conjunction with the three standard ECG leads, allows a division of the P-R interval into three components. The first is the P-A interval. This is the time from the earliest onset of the P wave (atrial activation in the area of the sinus node), as seen in any of the surface electrocardiograms, to the onset of atrial activity in the area of the A-V junction, as seen in the His bundle electrogram. This interval is taken as a measure of a representative portion of intra-atrial conduction, specifically the conduction time from the area of the sinus node to the area of the A-V node during normal sinus rhythm.

The A-H interval is the time from the beginning of the A wave to the onset of His bundle activity. This interval is taken as a measure of A-V nodal conduction. In our initial study, the term P-H interval was used as a measure of A-V nodal con-

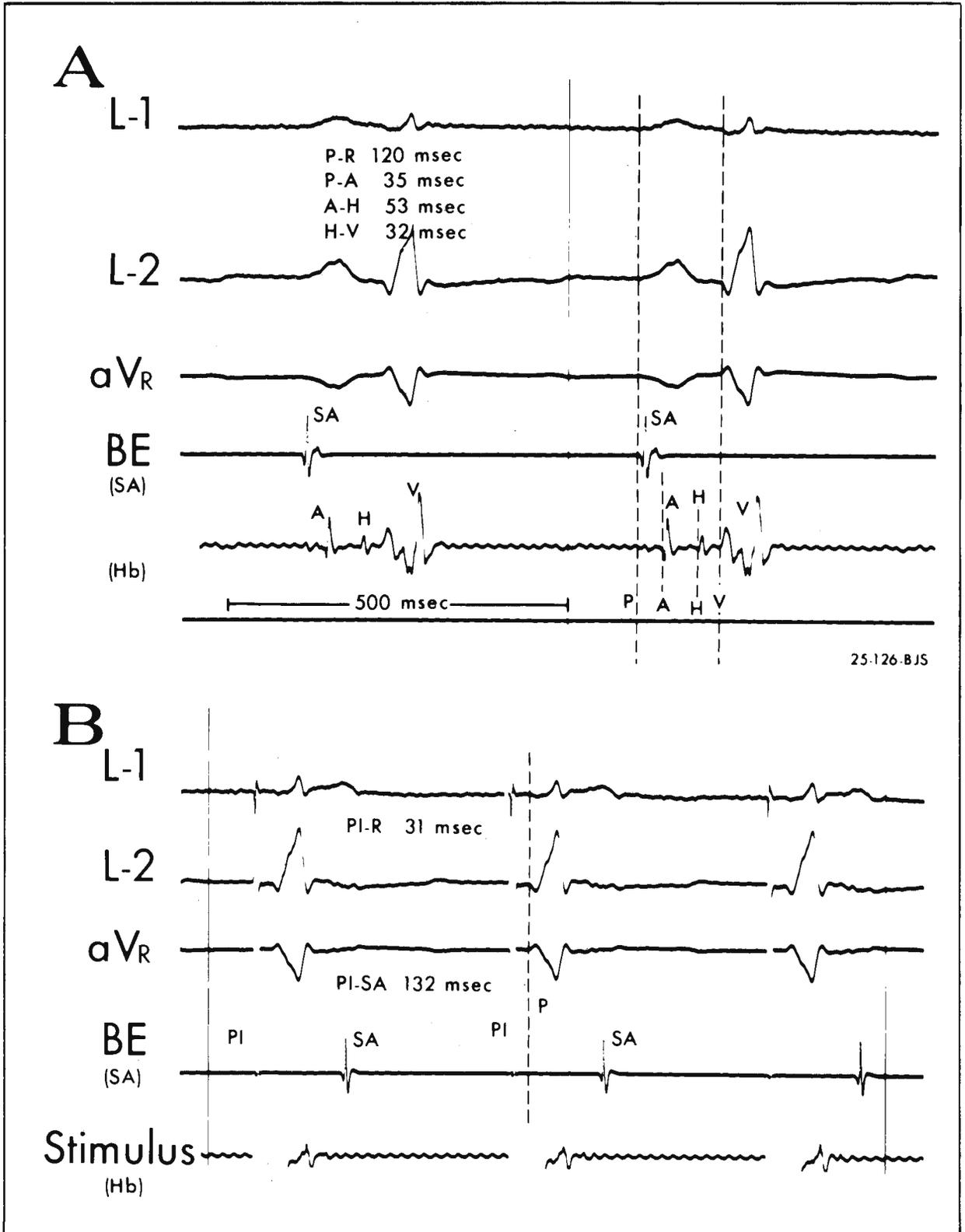


Fig. 1—Recording of His bundle bipolar electrogram (Hb) and simultaneously recorded bipolar electrogram from the sinus node area (BE, SA) with 3 standard ECG leads (L-1, L-2, and AVR). A. The P-R interval (120 msec) is divided into intra-atrial conduc-

tion time (P-A = 35 msec); A-V nodal conduction time (A-H = 53 msec); and His-Purkinje system conduction time (H-V = 32 msec). B. Pacing at a faster rate than the sinus rate, from the recording electrodes (stimulus, Hb), produces an unchanged QRS complex in all leads with a pacer impulse (PI) to R wave interval (PI-R) of 31 msec, directly comparable to the previously measured H-V time, 32 msec (panel A). Pacing the His bundle from the aortic root allowed retrograde activation of the atrium (PI-SA = 132 msec). The interval between time lines equals 1 second. (Reproduced by permission of The American Heart Association, Inc., from B. J. Scherlag, *et al.*, "His Bundle Electrogram," *Circulation* 46:602, 1972.)

duction. Unfortunately, this term has persisted although we now use the term A-H interval since it represents a more accurate measure of transmission through the A-V node. Ordinarily, the A wave of the His bundle electrogram represents local

atrial activity in the immediate vicinity of the A-V node and therefore, eliminates the intra-atrial conduction between the sinus node and the low right atrium. In addition, the A-H interval allows a comparison of A-V nodal conduction during both sinus

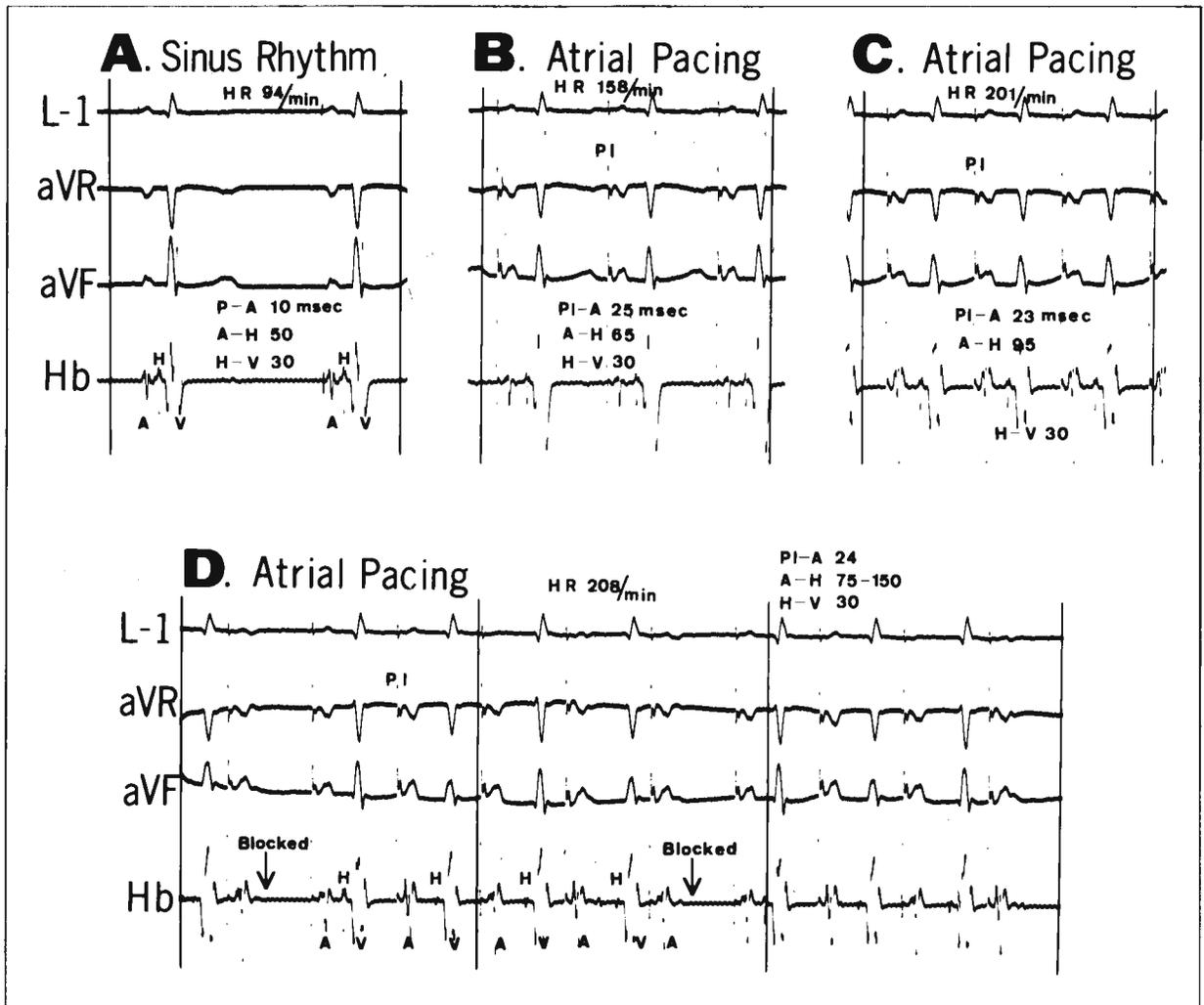


Fig. 2—The effect of atrial pacing on intra-atrial (P-A or PI-A), A-V nodal (A-H), and His-Purkinje (H-V) intervals in the dog. A. During sinus rhythm at a heart rate of 94 beats per minute, the P-A, A-H, and H-V time measured from the His bundle electrogram (Hb) and the simultaneously recorded ECG leads I, AVR, and AVF, are 10, 50, and 30 msec respectively. B and C. Atrial pacing up to a rate of 201 beats per minute produces a progressive increase in the A-H interval to 95 msec while the H-V interval remains constant at 30 msec. The pacer impulse to atrial activity (PI-A) is essentially the same at 23 to 25 msec during atrial pacing. D. At a heart rate of 208 beats per minute, a 5:4 Wenckebach cycle is seen with A-H variation from 75 to 150 msec. PI-A and H-V remain the same at 24 and 30 msec, respectively. Note that in the blocked beat the H and V deflections do not appear after the stimulus and atrial activity.

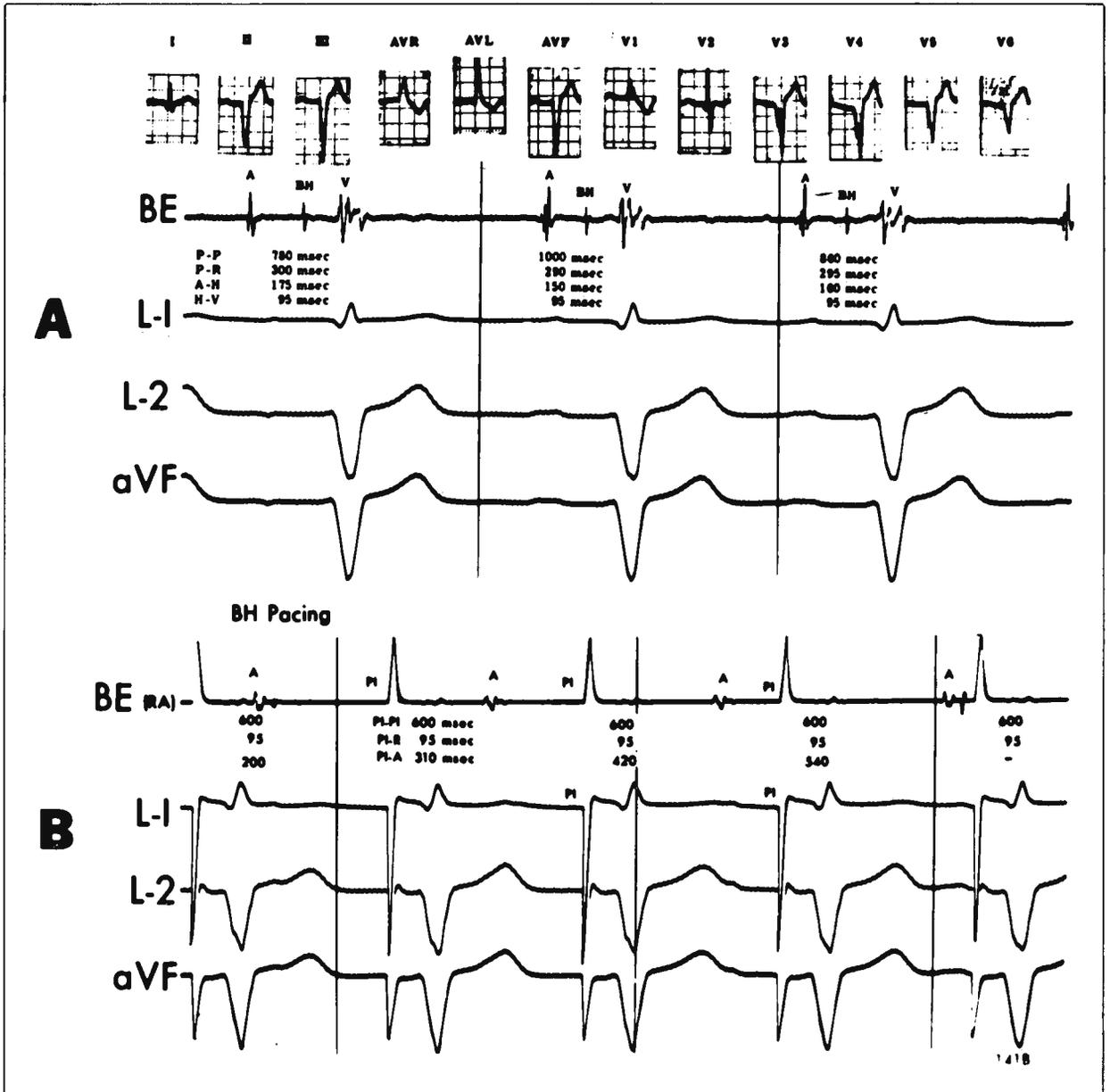


Fig. 3—Case W.A. Twelve standard ECG leads (top) show first degree block with right bundle branch block and left axis deviation. A. Simultaneous recordings of bipolar electrogram (BE) from the area of the A-V junction and standard ECG leads L-1, L-2, and AVF, during normal sinus rhythm. The A-H time varies between 150 and 175 msec, with variations in P-P interval. The H-V time of 95 msec remained constant. B. Simultaneous recordings of bipolar electrogram (BE) from the right atrium (RA) with standard ECG leads L-1, L-2, and AVF, during BH pacing at a rate of 100 per minute (PI-PI = 600 msec). PI-R interval of 95 msec is the same as the H-V time during normal sinus rhythm and the shape of the QRS complex remained unaltered throughout. (Reproduced by permission of The American Heart Association, Inc. from O. S. Narula, *et al.* "Pervenous Pacing of the Specialized Conducting System in Man: His Bundle and A-V Nodal Stimulation." *Circulation* 41:77, 1970.)

rhythm and atrial pacing because the atrial deflection used in the measurement is not ordinarily altered by the pacing site (fig. 3). This is in contrast to the P-H measurement which cannot be accurately

compared during spontaneous rhythm and atrial pacing since the pacer impulse is not usually applied in the area of the sinus node (compare figs. 3A and 2B,C,D).

The H-V interval is the time required for the impulse to traverse the His-Purkinje system, that is, from the onset of His bundle activity to the earliest onset of regular ventricular muscle activity as seen on any of the ECG leads or the His bundle electrogram. The term H-Q interval has been used to describe the conduction time through the His-Purkinje system in several reports. We prefer the term H-V on the basis of the fact that a Q wave may not be present in the ventricular deflection utilized for the measurement. In addition, we have stressed the use of at least three standard ECG leads to accurately determine the time of earliest ventricular activation. The use of only one ECG lead allows the possibility that the onset of ventricular activation is isoelectric in that lead. This would indicate an H-V measurement which is falsely prolonged.

Since the precise anatomic location of the recording electrodes on a catheter cannot be ascertained by fluoroscopy, a recorded deflection within the P-R segment must be verified as truly representing His bundle activity. Several criteria must be met to validate the recording as emanating from the His bundle and not from the atrium, A-V node, or the proximal bundle branches. The independence of a presumed His bundle deflection from atrial activity can be most easily obtained by right atrial pacing or induced premature atrial beats. This procedure uniformly produces a prolongation of the interval from atrial activity to the His bundle deflection (fig. 3). At rapid atrial paced rates, Wenckebach cycles can be elicited with progressive prolongation of the A-H interval and dropped beats indicating block proximal to the site of the recorded His bundle deflection.

Perhaps the most direct means of demonstrating the specificity of the His bundle deflection is the use of stimulation from the recording electrode catheter. In our clinical laboratories, standard safeguard procedures routinely utilized during temporary transvenous ventricular pacing are employed during His bundle pacing. Under these circumstances, we have found His bundle pacing no more difficult or hazardous than pacing in any other portion of the right or left ventricle. Pacing of the His bundle produces capture of the ventricles with the same QRS morphology in all ECG leads as that seen during sinus rhythm or atrial pacing (fig. 2B). This indicates that the point of stimulation was proximal to the right or left bundle branch and must

therefore be located in the His bundle or A-V node. Note also that the interval from the pacer impulse to the onset of ventricular activity is the same as the interval from the recorded His bundle potential to ventricular activation during sinus rhythm or paced atrial rhythm. If this interval is constant over a wide range of heart rates, a pacing site in the A-V node can be eliminated since conduction velocity in any part of the A-V node decreases with increasing rate. On the other hand, conduction in the His-Purkinje system is virtually unaffected by heart rate. We have found that these criteria for validating the His bundle potential by His bundle pacing apply equally as well in patients with normal A-V conduction as in those patients exhibiting severe disease of the His-Purkinje system (9).

Figure 4 shows tracings from a patient exhibiting right bundle branch block with left axis deviation, and an H-V time which was markedly prolonged, 95 msec. An abnormal QRS complex was simultaneously recorded in leads I, II, and AVF. His bundle pacing reproduced the H-V time of 95 msec as well as the same QRS configuration seen in all three ECG leads during sinus rhythm.

In figure 5, this same patient showed alternating bilateral bundle branch block during the study, but there were periods of left bundle branch block again with the characteristic QRS complex in all three leads, and now an H-V time of 90 msec. Pacing from the His bundle at this time reproduced the same QRS configuration and duration in all three leads as seen during sinus rhythm. Note also that the time from the stimulus to the onset of the earliest ventricular activity is the same with this bundle branch block pattern as the measured H-V time seen during sinus rhythm.

At this point, the matter of normal and abnormal H-V time should be mentioned. Normal values for A-V conduction have been battered about somewhat in the recent literature; therefore, we have scrutinized three published studies in which recordings from the His bundle have been obtained from 51 adult patients with so-called normal A-V conduction, that is, a P-R interval of 200 msec or less. Table I is a statistical analysis of the values from these various studies. It can be seen that the values in the study by Narula *et al.* (8) as well as from the study of Bekheit *et al.* (1) from Great Britain are closely comparable. In addition, if one assumes an average P-A time of 40 msec, the data

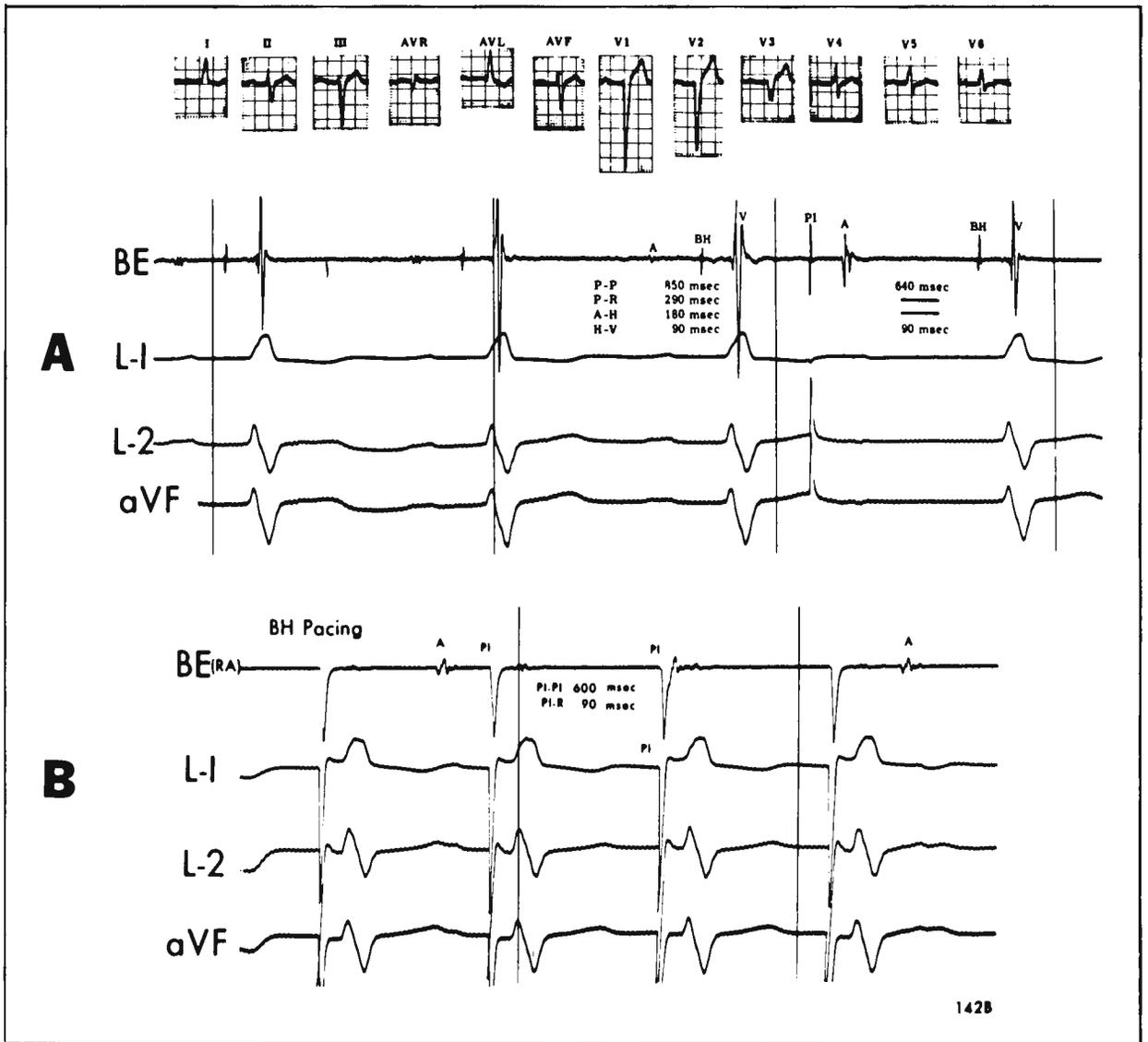


Fig. 4—Case W.A. Twelve standard ECG leads (top) from the same patient as in figure 3, whose pattern changed during study from right bundle branch block to left bundle branch block. A. Simultaneous bipolar electrograms (BE) recorded from the area of the A-V junction and standard ECG leads L-1, L-2, and AVF. The A-H time at P-P interval of 850 msec was 180 msec. The induced (PI) premature atrial systole (A) at P-P interval of 640 msec either blocked and was followed by an A-V junctional escape beat or conducted with an extremely prolonged A-H time. The H-V time of 90 msec was constant throughout. B. Bundle of His pacing at a rate of 100 per minute (PI-PI = 600 msec). The PI-R interval of 90 msec is equal to the H-V time during normal sinus rhythm. The shape of the QRS complex remains unaltered throughout. (Reproduced by permission of The American Heart Association, Inc. from O. S. Narula, *et al.* "Pervious Pacing of the Specialized Conducting System in Man: His Bundle and A-V Nodal Stimulation." *Circulation* 41:77, 1970.)

for heart rate P-R, P-A, A-H intervals in all three studies show good agreement. The lower and upper limits for the H-V interval, that is  $\pm 2$  standard deviations, in the last two studies are also in close accord, giving a range of 33 to 49 msec and 27 to 47 msec, respectively. In the study by Damato

*et al.* (5), the H-V intervals averaged  $51 \pm 12$  with a range of 39 to 63 msec. Other laboratories have reported normal values of 35 to 55 msec, and in a more recent report, Damato and his group have indicated an average value of 45 msec for the normal H-V time in patients. Unfortunately, no tabulated

TABLE 1. "Normal" Values of A-V Conduction Intervals in Man  
(mean and standard deviation)

REFERENCE	BASAL HR (beats/min)	CONDUCTION TIME (msec)			
		P-R	P-A	A-H	H-V
Narula <i>et al.</i> (8)	67 ± 10	172 ± 11	43 ± 14	88 ± 21	41 ± 4
Damato <i>et al.</i> (5)	71 ± 8	167 ± 16	116 ± 14*		51 ± 6
Bekheit <i>et al.</i> (1)	64 ± 15	154 ± 19	37 ± 11	78 ± 18	37 ± 5

Basal HR = normal sinus rate; P-R = intra-atrial, A-V nodal and His-Purkinje conduction time; P-A, A-H, H-V. See text for discussion.

\* This value represents the P-H interval consisting of P-A + A-H. See text for discussion.

(Reproduced by permission of The American Heart Association, Inc. from B. J. Scherlag, *et al.*, "His Bundle Electrogram," *Circulation* 46:606, 1972.)

data have been published to substantiate these ranges and average values. It should be emphasized that the determination of normal limits is of more than academic interest. Basic studies as well as recent clinical reports indicate a close association between prolongation of the H-V time and the existence of partial or complete bilateral bundle branch block. Other reports have shown a correspondence between an abbreviated A-H or H-V interval and anomalous A-V conduction.

It is generally considered that His bundle-electrocardiography has provided a more direct means for electrophysiological and diagnostic study of various clinical problems. However, these interpretations and diagnoses based on His bundle recordings must stand up to critical quantitative verification of the presumed His bundle deflection. Quantitative determinations of the interpretations based on His bundle-electrocardiographic measurements are critical, and in order to facilitate such determinations, greater standardization of measurements and terminology is required.

*Author's note:* We thank Mrs. Marie Ellis for her dedicated assistance in the preparation of this manuscript.

## REFERENCES

1. BEKHEIT, S., MURTAGH, J. G., MORTON, P., *et al.* Measurements of sinus impulse conduction from the electrogram of bundle of His. *Brit. Heart J.* 33:719, 1971.
2. CASTELLANOS, A., CASTILLO, C., LEMBERG, L., *et al.* His bundle-electrocardiography: A programmed introduction. *Chest* 57:350, 1970.
3. CASTELLANOS, A., CHAPUNOFF, E., CASTILLO, C., *et al.* His bundle electrograms in 2 cases of Wolff-Parkinson-White (pre-excitation) syndrome. *Circulation* 41:399, 1970.
4. CASTILLO, C. AND CASTELLANOS, A. His bundle recordings in patients with reciprocating tachycardias and Wolff-Parkinson-White syndrome. *Circulation* 42:271, 1970.
5. DAMATO, A. N., LAU, S. H., HELFANT, R. H., *et al.* Study of A-V conduction in man using electrode catheter recordings of His bundle activity. *Circulation* 39:287, 1969.
6. DAMATO, A. N. AND LAU, S. H. Clinical value of the electrogram of the conduction system. *Prog. in Cardiovasc. Dis.* 13:119, 1970.
7. LAU, S. H., DAMATO, A. N., BERKOWITZ, W. D., *et al.* A study of atrioventricular conduction in atrial fibrillation and flutter in man using His bundle recordings. *Circulation* 40:71, 1969.
8. NARULA, O. S., COHEN, L. S., SAMET, P., *et al.* Localization of A-V conduction defects in man by recording of His bundle electrograms. *Amer. J. Cardiol.* 25:228, 1970.
9. NARULA, O. S., SCHERLAG, B. J., AND SAMET, P. Per-venous pacing of the specialized conducting system in man: His bundle and A-V nodal stimulation. *Circulation* 41:77, 1970.
10. SCHERLAG, B. J., HELFANT, R. H., AND DAMATO, A. N. Catheterization technique for His bundle stimulation and recording in the intact dog. *J. Appl. Physiol.* 25:425, 1968.
11. SCHERLAG, B. J., KOSOWSKY, G. D., AND DAMATO, A. N. Technique for ventricular pacing from the His bundle of the intact heart. *J. Appl. Physiol.* 22:584, 1967.
12. SCHERLAG, B. J., NARULA, O. S., LISTER, J. W., *et al.* Analysis of atrioventricular conduction by direct intracardiac recordings. *J. Mount Sinai Hosp.* 37:266, 1970.