

The Gouaux-Ashman Phenomenon: His Bundle Recordings*

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The differentiation of ventricular premature beats from supraventricular beats with aberrant ventricular conduction is often difficult on the basis of the surface electrocardiogram. The diagnosis of aberrant conduction has been based largely on the presence of a right bundle branch block pattern appearing in a short cycle subsequent to a longer cycle (2). Several other criteria for diagnosing aberrant conduction of a supraventricular beat such as variable coupling time have been used to differentiate these abnormal ventricular complexes from ventricular premature beats which are most commonly followed by a full compensatory pause. This differentiation is often inexact and under some critical clinical circumstances cannot be accomplished with any degree of reliability. In 1947, Gouaux and Ashman wrote a report entitled: "Auricular Fibrillation with Aberration Simulating Ventricular Paroxysmal Tachycardia" (2). It is thus in the clinical context of atrial fibrillation that the diagnosis of aberrant conduction of supraventricular beats as opposed to ventricular tachycardia becomes particularly difficult. Recent reports in the literature have emphasized the use of the His bundle-electrocardiographic technique as a means of differentiating between aberrant conduction and ventricular ectopic complexes (3, 4, and 6). In order to evaluate this method of differentiation, it is important to note the physio-

logical behavior of A-V nodal and His bundle conduction under these two circumstances.

Figure 1, taken from a study by Damato *et al.* (1), shows the response of the His-Purkinje system to atrial premature beats delivered at varying intervals after the previous cardiac cycle. A slight degree of aberration can be seen even with no prolongation of the H-V or H-Q time. However, more marked aberration occurs with the prolongation of His-Purkinje conduction time from 45 to 65 msec. Finally, block is shown distal to the recorded His bundle deflection, and these properties of the His-Purkinje system during premature activation have been confirmed in other clinical laboratories. Therefore, the normal response to atrial premature or His premature beats which result in aberrant ventricular conduction is either an unchanged or prolonged H-V time.

In contrast to the constancy or prolongation of the H-V interval during supraventricular beats conducted with aberration, ventricular ectopic beats or fusion beats show either no His potential preceding the aberrant QRS complex or a shortened H-V time. In figure 2, the His bundle-EKG tracings reveal an H-V time of 50 msec with a wide QRS complex. Note that the P-R interval is 190 msec during sinus rhythm. In the second beat in panel A, a shortening of the P-R interval from 190 to 175 msec with a narrowing of the QRS complex is observed. Also, a shortening of the H-V time to 35 msec occurred. That this beat represents in fact a fusion of a normally conducted atrial impulse and a ventricular ectopic beat is indicated in panels B and C. Here the P-R shortens to 135 msec in the sec-

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ond beat of each panel with a negative H-V time, that is, ventricular activity precedes His bundle activity by 5 msec and 20 msec, respectively. It can be seen that the change in H-V time from normal is appropriately matched by the change in the P-R interval.

Unfortunately, these quantitative aspects of the His bundle recording in regard to ventricular ectopic beats and supraventricular beats conducted with aberration have been essentially ignored in several recent publications. In a report by Lau *et al.* (3), the authors were the first to note that the recording of His bundle activity represents a valid method for diagnosing aberrant conduction, particularly in differentiating supraventricular beats and ventricular premature contractions of ventricular tachycardia. However, in illustrating this point, a case of atrial fibrillation was examined with simultaneous recordings of His bundle electrogram and a standard ECG lead (fig. 3). The authors state that the second complex in this figure is a supraventricular beat conducted from above with aberrant ventricular activation. However, the H-V time is clearly shorter in this beat than in the normally conducted beats, 1, 4, and 6. Note that the H-V time is measured from the H deflection to the earliest ventricular activation which in this case is the standard ECG lead. In the normally conducted beats, the earliest ventricular activation occurs in the His bundle electrogram.

In another recent report by Massumi, a case was presented (fig. 4) in which the first two beats shown are sinus beats showing atrial, His bundle, and ventricular activity. The third beat shows a QRS complex which the author describes as a pattern of "incomplete right bundle branch block and left axis deviation suggesting impaired conduction through the right bundle branch and superior division of the left bundle branch." Massumi localizes the extrasystolic focus as occurring within the His bundle and bases this conclusion on the observation that this extrasystole is preceded by a His bundle potential. However, it should be noted that the H-V time of this third beat is markedly shortened compared to the H-V time seen during sinus beats. Both of the previous records can be interpreted as ventricular beats arising in the proximal portion of the right or left bundle branch which are simultaneously conducted antegrade to the ventricular myocardium and retrograde to the His bundle. Under these circumstances the QRS would be aberrant and if one were only recording His bundle activity, this poten-

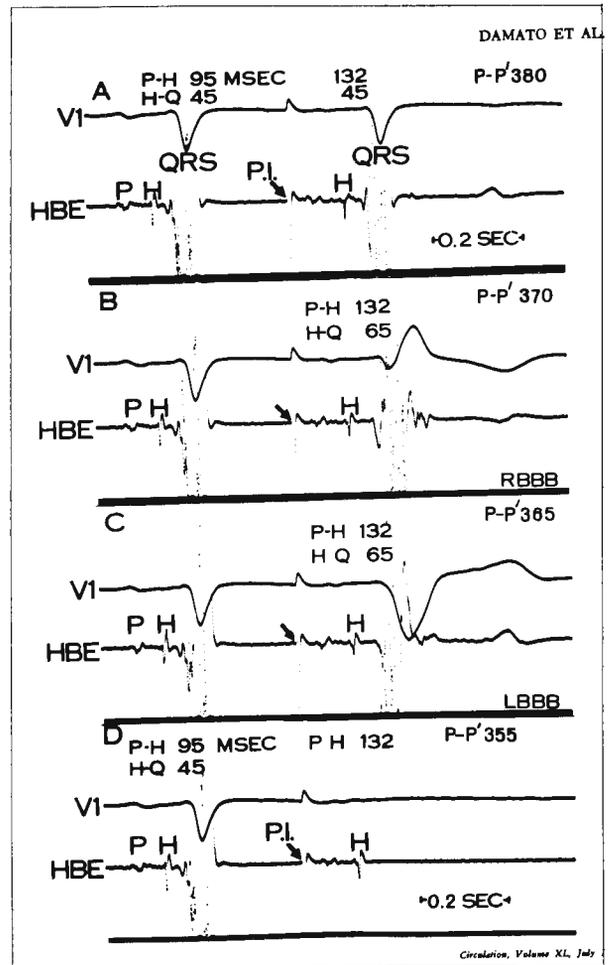


Fig. 1—In panels A through D, the top tracing is a lead V_1 and the bottom tracing a His bundle electrogram (HBE). When the atrium was prematurely stimulated at a coupling interval of 380 msec (panel A), a delay in A-V conduction occurred as manifested by an increase in the P-H interval to 132 msec. The H-Q interval remained constant, and ventricular depolarization was only slightly altered. Shortening the coupling interval to 370 msec (panel B) and 365 msec (panel C) resulted in aberrant ventricular depolarization (RBBB and LBBB) with an associated prolongation of the H-Q interval. At a coupling interval of 355 msec (panel D), complete block below the His bundle resulted. (Reproduced by permission of the American Heart Association, Inc. from Damato, A. N. *et al.* "A Study of Atrioventricular Conduction in Man Using Premature Atrial Stimulation and His Bundle Recordings." *Circulation* 40:64, 1969).

tial would appear before the QRS with a shortened H-V time.

Just such recordings have been made in our clinical laboratory by Narula *et al.* (5). Figure 5 shows a simultaneous recording of His bundle and left bundle activity during sinus rhythm in a patient

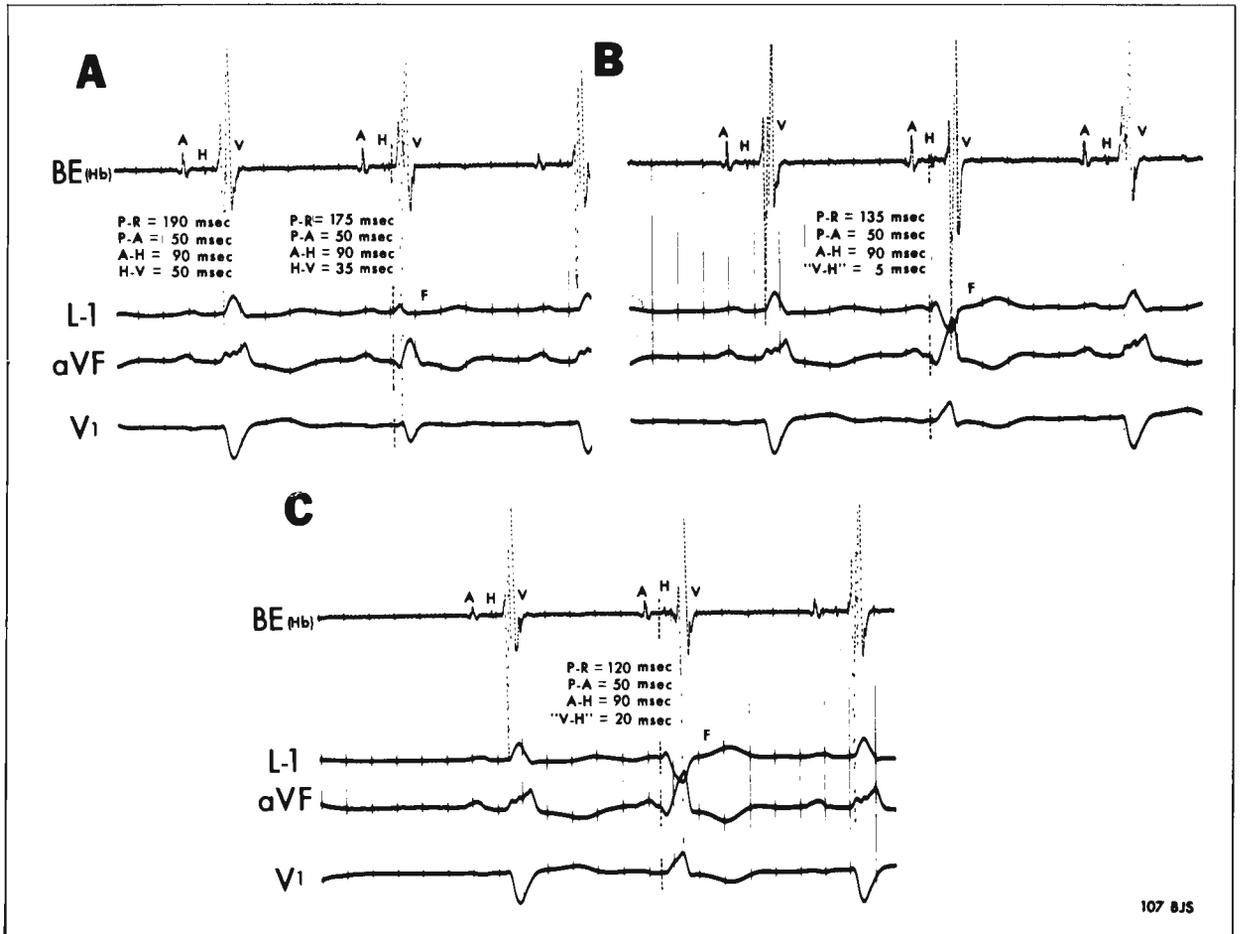


Fig. 2—The effect of fusion beats on the H-V interval in a patient with intermittent ventricular ectopic beats. Traces from above: Bipolar His bundle electrogram (BE, Hb) and 3 simultaneously recorded ECG leads I, AVF, and V₁. A. During sinus beats, the P-R of 190 msec was composed of intra-atrial conduction time (P-A = 50 msec), A-V nodal conduction (A-H) = 90 msec and His-Purkinje conduction (H-V) = 50 msec. A fusion beat (F) caused a shortening of the "P-R" to 175 msec entirely due to H-V diminution of 15 msec (50-35 msec). The vertical dotted line indicates the earliest ventricular activation in all leads. B. Another fusion beat (2nd complex marked F) causes a further shortening of the P-R to 135 msec and a reversal of the H-V with the onset of ventricular activation preceding His bundle activation by 5 msec. C. Another fusion beat (F) produced "normalization" of the QRS complex as compared to other beats. Note that the shortening of the P-R in each case is matched exactly by the alteration in H-V or V-H time; the P-A and A-H interval remain constant throughout. The interval between time lines equals 100 msec.

with a normal QRS complex. The second beat in panel A shows left bundle activity preceding His bundle activity during ventricular premature contractions which probably arose in the area of the proximal left bundle. In panel B, a series of beats arising in the same region of left bundle activity preceding His bundle activity is seen. Without the left bundle recording, one could interpret the aberrant beats with a shortened H-V time as arising in the His bundle and conducting through the ventricles aberrantly.

In conclusion, it is important to state that the use of His bundle-electrocardiography in differentiating supraventricular beats with aberration from ventricular beats requires a critical approach to the interpretations of deflections appearing in the P-R segment. The use of quantitative rather than qualitative tests is essential when only the His bundle recording is used as opposed to the simultaneously recorded activity from the proximal bundle branches. In some cases, His bundle pacing may be helpful in reproducing spontaneously occurring aberrant pat-

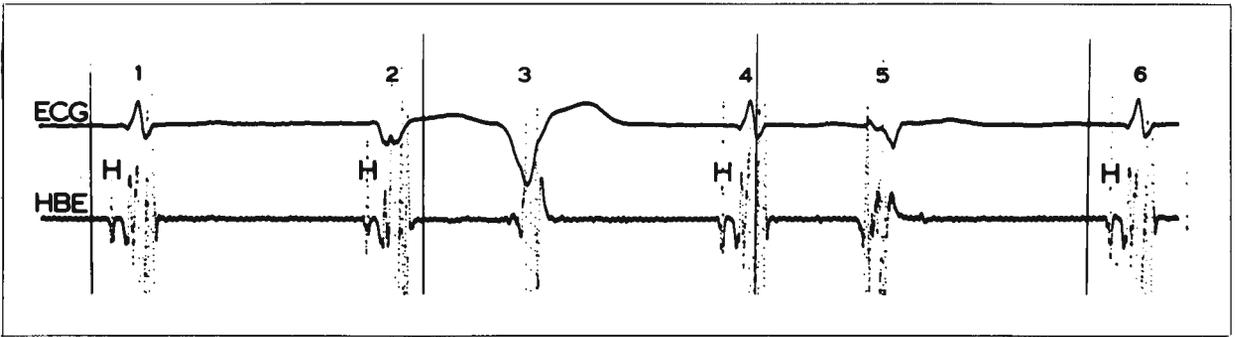


Fig. 3—In this clinical case of atrial fibrillation, the first, fourth, and sixth complexes represent the normally conducted beats, each of which is preceded by a His deflection. The second complex, which is also preceded by a His deflection, represents an aberrant beat. The third complex is a premature ventricular contraction. No His potential precedes this beat. (Reproduced by permission of the American Heart Association, Inc. from Lau, S. H. *et al.* "A Study of Atrioventricular Conduction in Atrial Fibrillation and Flutter in Man Using His Bundle Recordings." *Circulation* 40:73, 1969).

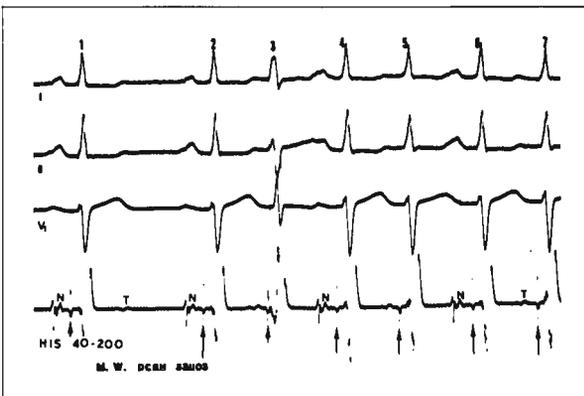


Fig. 4—Simultaneous recording of leads I, II, and V_1 together with His bundle potentials recorded from the main His bundle. The sinus P waves before beats 1, 2, 4, 6, are clearly visible in leads I and II and also in the His bundle recording just before the atrioventricular node potentials marked N. Note that the His bundle potentials, marked by arrows, precede not only the QRS of the sinus beats 1, 2, 4, 6, but also the interpolated extrasystoles 3, 5, and 7. The aberrant intraventricular conduction of the first interpolated extrasystole No. 3, is of the incomplete right bundle branch block and left axis deviation type, suggesting impaired conduction through the right bundle branch and the superior division of the left bundle branch. Our localization of the extrasystolic focus within the His bundle is based on the observation that the extrasystoles 3, 5, and 7 are preceded by His bundle potentials but not by atrioventricular node potentials like the sinus beats. Had they arisen from the atrioventricular node, the atrioventricular node potentials would have also been recorded just before the His potentials of the extrasystoles. This indicates that the extrasystoles originate below the atrioventricular node. The low frequency, low amplitude distortion of the baseline seen before the His potential of the extrasystoles represents the T wave of the preceding beat, two of which are marked with T. Finally, the reason for aberration of the first beat of the run of tachycardia with normal conduction

thereafter is explainable on the basis of the well-known effect of the long preceding cycle, in this case, cycle 1-2. (Reproduced by permission of the *American Journal of Medicine* 49:267, 1970, and R. A. Massumi).

terns. Although one should expect a normal or prolonged H-V time in supraventricular or His bundle beats conducted with aberration, one must rule out the theoretical possibilities of a shortened H-V time due to "super-normality," or a constant or prolonged H-V time due to a beat arising in the bundle branches which is conducted with delay to the ventricular musculature but with normal retrograde conduction to the His bundle.

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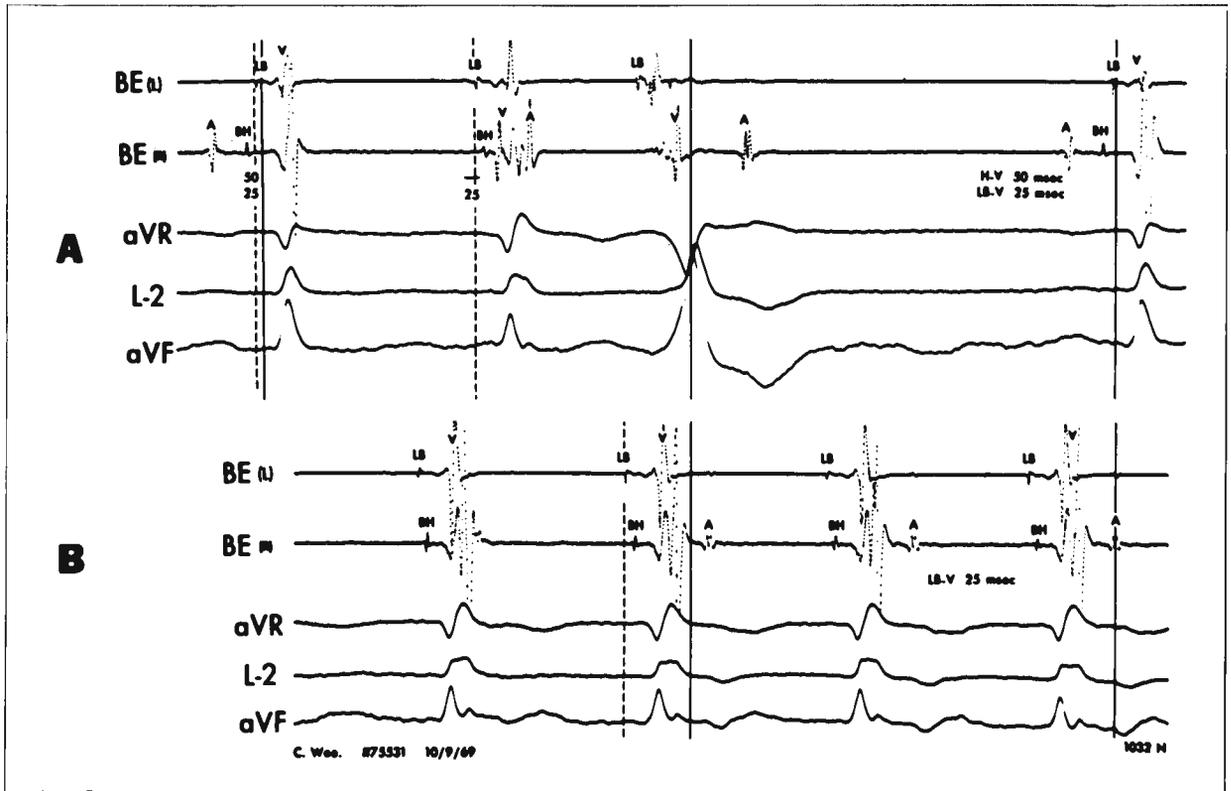


Fig. 5—Case 5: Retrograde activation of the BH by premature “ventricular” beats (probably left bundle beats). (Reproduced by permission of the American Heart Association, Inc. from Narula, O. S. *et al.* “Significance of His and Left Bundle Recordings from the Left Heart in Man.” *Circulation* 42:395, 1970).

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