His bundle electrogram in P mitrale

S. Bekheit¹, G. Murtagh, P. Morton, and E. Fletcher

From the Cardiovascular Unit, Belfast City Hospital, Northern Ireland

The His bundle electrogram was recorded in 19 cases with P mitrale simultaneously with standard lead II, and compared with 30 cases with normal P waves. Internodal conduction time (P_1A) interval did not differ in P mitrale from the control group, confirming that prolonged atrial activation in P mitrale represents a specific 'left atrial block'. The importance of this observation is discussed with reference to preferential atrial conducting pathways between the sinus node and the atrioventricular node. The end-point of atrial activation is altered in P mitrale so that it approaches the H wave of the His bundle electrogram and 'overrides it'. This graphic feature of the His bundle electrogram is characteristic of P mitrale within the range of normal atrioventricular conduction, the H wave preceding the termination of atrial activation (P_2), and the beginning of the H wave (the P_2H interval) having a negative value.

The notched P wave of mitral stenosis was first described by Lewis (1913), and it is now established as 'P mitrale' in common usage since it was so named by Winternitz (1935). It has usually been ascribed to left atrial enlargement (Alexander, Knight, and White, 1925); Trounce, 1952; Katz and Pick, 1956) but it may occur with normal left atrial size (Saunders *et al.*, 1967), and it may appear and disappear in the same tracing (James and Sherf, 1971). However, from a pathophysiological point of view, P mitrale represents abnormal conduction of the sinus impulse within the atria, and it is with this aspect of it that we are concerned in this paper. The cases selected for study fulfilled the criteria of duration and polarity of the P wave for P mitrale (Morris *et al.*, 1964). In our studies particular attention is paid to the former. In the past, attempts have been made to correlate the duration of the P wave with the arrival of the sinus impulse at the Purkinje myocardial junction using the PR interval of scalar leads to define P mitrale (Macruz, Perloff, and Case, 1958). However, the PR interval is a relatively crude measurement of atrioventricular conduction, which can now be resolved into its component parts more precisely by the His bundle electrogram. When recorded with an appropriate scalar lead, internodal, atrioventricular nodal, and His-Purkinje conduction time can be accurately measured. Accordingly we have

studied the His bundle electrogram in P mitrale to make observations on the nature of intra-atrial conduction, and to define any characteristic features in the His bundle tracing which may be revealed.

Methods

The His bundle electrogram was recorded in 49 patients in all of whom the PR interval was normal in duration (120-200 msec: New York Heart Association, 1940). All of them were undergoing routine cardiac catheterization for assessment, and, after explanation, permission was obtained from each to record the His bundle electrogram as a supplementary study. A bipolar electrode catheter was used as described by Scherlag *et al.* (1969). Lead II was recorded simultaneously at a paper speed of 100 mm/min. The A, H, and V waves of the His bundle electrogram are identified in Fig. 2. The significance of the time intervals given below is in accordance with the authors referred to above. The patients were divided into two groups: (1) 30 cases with normal P waves, as controls, for comparison with (2) 19 cases with P mitrale. No patients were receiving cardiac drugs.

The following measurements were made in milliseconds.

- 1) The duration of the P wave in lead II, P_1P_2 interval.
- 2) The conduction time from sinus node to atrioventricular (AV) node or internodal conduction time, P_1A interval.
- 3) The AV nodal conduction time, AH interval.
- 4) His-Purkinje-system (HPS) conduction time, HV interval.
- 5) P_2H interval, expressing the temporal relation between the completion of atrial activation (P_2) and main His bundle depolarization H.

Received 25 February 1972.

¹ Research Fellow, Northern Ireland Hospitals Authority.

TABLE Comparison of sinus impulse conduction in P mitrale and normal hearts

Group	No. of cases	PR interval (msec)		Duration of P wave (P ₁ P ₂ msec)		Internodal conduction time (P ₁ A msec)		AV nodal conduction time (AH msec)		P ₂ H (msec)		His-Purkinje-system conduction time (HV msec)	
		Range	Mean	Range	Mean	Range	Mean	Range	Mean	Range	Mean	Range	Mean
Normal heart	30	130-185	158	70-115	91.5	10-45	29	60-110	93	(+45)-(+10)	+23	30-50	43
P mitrale	19	140-200	175	140-165	142	20-45	34	60-125	102	(+20)-(-45)	-5	35-45	42

Results

The Table summarizes the range of values and mean values of the intervals measured. The measurements for the control cases correspond with previously published data (Bekheit *et al.*, 1971). Where appropriate, comparison was made with the student t-test. Two distinct deductions can be made from the data, both dependent upon the duration of atrial activation, namely the nature of abnormal intra-atrial conduction in P mitrale, and the graphic features in the His bundle electrogram in P mitrale.

Nature of intra-atrial conduction in P mitrale The P₁P₂ interval of the control group was within the accepted normal range (≤ 120 msec: Graybiel *et al.*, 1944). The P₁P₂ interval was longer in P mitrale than in the control group, the difference being highly significant ($P < 0.0005$). However, the PA interval was not significantly different in the two groups ($P > 0.05$). These observations confirm that internodal conduction was normal in P mitrale. In the normal heart the sinus impulses enter the AV node via the right atrium (Scherf and Cohen, 1964). In P mitrale this pathway of entry appears to remain intact. Also, in the normal heart the left atrium is activated about 40 msec after the right atrium (Hecht and Woodbury, 1950). Thus, the prolonged P₁P₂ interval in P mitrale appears to represent a specific delay in activation of the left atrium. The results of these observations will be discussed below in the light of recent information about specialized Purkinje fibres in the atria.

Graphic features of His bundle electrogram in P mitrale The sinus impulse penetrates into the AV conduction system before the end of atrial activation (Scherf and Cohen, 1964). The degree of penetration can be more precisely measured with the His bundle electrogram using the H wave as a reference point than is possible with a scalar lead. The termination of atrial activation (P₂) and the beginning of the H wave (P₂H interval)

can be used as a measurement for comparison in P mitrale with the normal heart. Fig. 1 is a stylized drawing of the P₂H interval. In the normal heart P₂ is shown preceding H, i.e. the P₂H interval is positive. In contrast, in P mitrale, P₂ follows the H wave and the P₂H

FIG. 1 Schematic drawing of bundle of His electrogram in normal P wave and P mitrale. Note the beginning and end of atrial depolarization in the standard lead (P₁P₂). A = depolarization of atrionodal junction. H wave = depolarization of main bundle of His. In normal P wave, P₂ precedes H, i.e. P₂H interval is positive. In P mitrale, P₂ follows H, and P₂H interval is negative. P₁A interval, internodal conduction time is identical in both.

FIG. 2 Bundle of His electrogram in normal P wave. The P_1P_2 interval is upper limit of normal, 120 msec. The P_2H interval is positive, +30 msec. Distance between two light divisions = 40 msec.

interval is negative. In the Table the P_2H interval varied significantly in P mitrale from the control group ($P < 0.0005$). Fig. 2 illustrates a normal positive P_2H interval measuring 30 msec in a normal heart, and Fig. 3 a negative P_2H interval, -30 msec in P mitrale. When the PR interval is normal, the P_1P_2 duration determines the P_2H interval. Fig. 4 illustrates the relation between the P_1P_2 interval and P_2H interval in the control group and P mitrale. As the P_1P_2 interval lengthens P_2 approaches the H wave and typically overrides it at values of 140 msec or more. In P mitrale therefore the sinus impulse may depolarize the main bundle of His

and penetrate deeply into the His-Purkinje-system before atrial activation is complete.

Discussion

The original concept of Lewis, Oppenheimer, and Oppenheimer (1910) that the sinus impulse activated the atria by a series of concentric circles of depolarization of increasing diameter has been questioned by the confirmation of Purkinje fibres in the atria by both histopathological and electrophysiological studies (James and Sherf, 1971). Our observations of the His bundle electrogram in P mitrale support the view that preferential

FIG. 3 Bundle of His electrogram in P mitrale. The P_1P_2 interval is prolonged to 170 msec. P_2 follows the H wave, and P_2H interval is negative, -30 msec. Distance between two light divisions = 40 msec.

FIG. 4 Relation of P wave duration (P_1P_2 interval) in scalar leads to P_2 (end point of atrial depolarization) and main His bundle depolarization (H wave) expressed as P_2H interval. When P_1P_2 was normal (70–115 msec), P_2 invariably preceded H, and P_2H interval was positive (+10–+45 msec). When P_1P_2 was prolonged (P mitrale) to 140–165 msec P_2 synchronized with H or followed it, P_2H interval, 0–(–45 msec). Intermediate P_1P_2 values (90–135 msec) shortened the P_2H interval which remained positive. P_2 approaches the H wave and overrides it as P_1P_2 lengthens.

conducting pathways connect the sinus and AV node (Wenckebach, 1907; Thorel, 1910; Bachmann, 1916). The specific delay of activation of the left atrium may be explained by focal lesions in the interatrial myocardial band (Bachmann's bundle) which have been shown histopathologically in P mitrale by Beeson and Teabeaut (1969). As the internodal conduction time is normal in P mitrale, the conduction defect may be described as 'left atrial block', akin to left bundle-branch block in the ventricles. Unlike the ventricles, however, the Purkinje fibres in the preferential atrial pathways are interwoven (James and Sherf, 1971) and 'left atrial block' may therefore imply extensive destruction of Purkinje fibres in P mitrale. Hypertrophy of left atrial muscle fibres alone in mitral stenosis would, on the other hand, offer less resistance to the passage

of the depolarization front by increased cross-section of muscle cells, and atrial activation would not be prolonged.

The graphic features of the His bundle electrogram in P mitrale in relation to the P_2H interval described above are incidental to the duration of the P_1P_2 interval and the sum of the PA and AH intervals. It could be expressed as a ratio, P_1P_2/P_1H , which exceeded unity in the series studied when P_1P_2 was greater than unity. While this ratio will always be altered when P_1P_2 is prolonged, the graphic relation of P_2 to H described in this paper applies only to normal AV conduction. If AV conduction is accelerated by anomalous pathways or delayed in the atria or AV node, the relation of P_2H will be altered. Thus, the changes in the P_2H interval in P mitrale described above are non-specific in character and apply when the PR interval is within the normal range. Finally, it may be noted that His-Purkinje-system conduction (HV interval) plays no part in the P_2H interval in P mitrale. It was normal in the cases studied (Table).

References

- Alexander, A. A., Knight, H. F., and White, P. D. (1925). The auricular wave (P) of the electrocardiogram: clinical observations with special reference to pulmonic and mitral stenosis. *Archives of Internal Medicine*, **36**, 712.
- Bachmann, G. (1916). The interauricular time interval. *American Journal of Physiology*, **41**, 309.
- Beeson, C. W., II, and Teabeaut, J. R., II (1969). Bachmann's bundle. Anatomic lesions in P mitrale (abstract). *Circulation*, **40**, Suppl. 3, 42.
- Bekheit, S., Murtagh, J. G., Morton, P., and Fletcher, E. (1971). Measurements of sinus impulse conduction from electrogram of bundle of His. *British Heart Journal*, **33**, 719.
- Graybiel, A., McFarland, R. A., Gates, D. C., and Webster, F. A. (1944). Analysis of the electrocardiogram obtained from 1000 young healthy aviators. *American Heart Journal*, **27**, 524.
- Hecht, H. H., and Woodbury, L. A. (1950). Excitation of human auricular muscle and the significance of the intrinsicoid deflection of the auricular electrocardiogram. *Circulation*, **2**, 37.
- James, T. N., and Sherf, L. (1971). Specialized tissues and preferential conduction in the atria of the heart. *American Journal of Cardiology*, **28**, 414.
- Katz, L. N., and Pick, A. (1956). *Clinical Electrocardiography*. Lea and Febiger, Philadelphia.
- Lewis, T. (1913). *Clinical Electrocardiography*. Shaw & Sons, London.
- Lewis, T., Oppenheimer, B. S., and Oppenheimer, A. (1910). The site of origin of the mammalian heart-beat; the pacemaker in the dog. *Heart*, **2**, 147.
- Macruz, R., Perloff, J. K., and Case, R. B. (1958). A method for the electrocardiographic recognition of atrial enlargement. *Circulation*, **17**, 882.
- Morris, J. J., Estes, E. H., Whalen, R. E., Thompson, H. K., and McIntosh, H. D. (1964). P-wave analysis in valvular heart disease. *Circulation*, **29**, 242.

- New York Heart Association, Criteria Committee (1940). *Nomenclature and Criteria for Diagnosis of Disease of the Heart*, 4th ed. J. J. Little and Ives Company, New York.
- Saunders, J. L., Calatayud, J. B., Schulz, K. J., Maranhao, V., Gooch, A. S., and Goldberg, H. (1967). Evaluation of E.C.G. criteria for P-wave abnormalities. *American Heart Journal*, **74**, 757.
- Scherf, D., and Cohen J. (1964). *The Atrioventricular Node and Selected Arrhythmias*. Grune and Stratton, New York.
- Scherlag, B. J., Lau, S. H., Helfant, R. H., Berkowitz, W. D., Stein E., and Damato, A. N. (1969). Catheter technique for the recording of His bundle activity in man. *Circulation*, **39**, 13.
- Thorel, C. (1910). Über den aufbau des sinusknotens und seine verbindung mit der cava superior und den Wenckebachschen bundeln. *Münchener medizinische Wochenschrift*, **57**, 183.
- Trounce, J. R. (1952). The electrocardiogram in mitral stenosis. *British Heart Journal*, **14**, 185.
- Wenckebach, K. F. (1907). Beiträge zur kenntnis der menschlichen herztätigkeit. *Archiv für Physiologie*, **1-24**.
- Winternitz, M. (1935). Forschungsergebnisse aus Medizin und Naturwissenschaft zur Pathologie des menschlichen Vorhofelektrokardiogramms. *Medizinische Klinik*, **31**, 1575.

Requests for reprints to Dr. Evan Fletcher, Cardiovascular Unit, Belfast City Hospital, Lisburn Road, Belfast, BT9 7AB, Northern Ireland.