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 (P1A) 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 (P2), and the beginning of the H wave (the P2H 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 (Macruez, 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.
2. No patients were receiving cardiac drugs.

The following measurements were made in milliseconds:

1. The duration of the P wave in lead II, P1P2 interval.
2. The conduction time from sinus node to atrioventricular (AV) node or internodal conduction time, P1A interval.
3. The AV nodal conduction time, AH interval.
4. His-Purkinje-system (HPS) conduction time, HV interval.
5. P2H interval, expressing the temporal relation between the completion of atrial activation (P2) and main His bundle depolarization H.
**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 interval is negative. P1A interval, internodal conduction time is identical in both.

**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.

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

<table>
<thead>
<tr>
<th>Group</th>
<th>No. of cases</th>
<th>PR interval (msec)</th>
<th>Duration of P wave (P1P2 msec)</th>
<th>Internodal conduction time (P1A msec)</th>
<th>AV nodal conduction time (AH msec)</th>
<th>P2H (msec)</th>
<th>His-Purkinje system conduction time (HV msec)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Normal heart</td>
<td>30</td>
<td>130–185</td>
<td>70–115</td>
<td>10–45</td>
<td>60–110</td>
<td>(+45)–(-10)</td>
<td>30–50</td>
</tr>
<tr>
<td>P mitrale</td>
<td>19</td>
<td>140–200</td>
<td>140–165</td>
<td>20–45</td>
<td>60–125</td>
<td>42</td>
<td>35–45</td>
</tr>
</tbody>
</table>

*Note: AV = atrioventricular, P = atrial, H = His, P2H = P2-H interval.*
His bundle electrogram in P mitrale

FIG. 2 Bundle of His electrogram in normal P wave. The $P_1 P_2$ interval is upper limit of normal, 120 msec. The $P_2 H$ interval is positive, +30 msec. Distance between two light divisions = 40 msec.

interval is negative. In the Table the $P_2 H$ interval varied significantly in P mitrale from the control group ($P < 0.0005$). Fig. 2 illustrates a normal positive $P_2 H$ interval measuring 30 msec in a normal heart, and Fig. 3 a negative $P_2 H$ interval, −30 msec in P mitrale. When the PR interval is normal, the $P_1 P_2$ duration determines the $P_2 H$ interval. Fig. 4 illustrates the relation between the $P_1 P_2$ interval and $P_2 H$ interval in the control group and P mitrale. As the $P_1 P_2$ 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
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 P2H interval described above are incidental to the duration of the P1P2 interval and the sum of the PA and AH intervals. It could be expressed as a ratio, P1P2/P1H, which exceeded unity in the series studied when P1P2 was greater than unity. While this ratio will always be altered when P1P2 is prolonged, the graphic relation of P2 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 P2H will be altered. Thus, the changes in the P2H 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 P2H interval in P mitrale. It was normal in the cases studied (Table).

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mitrale.

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