Intraventricular Conduction in Man
Studied with an Endocardial Electrode
Catheter Mapping Technique

Patients with Normal QRS and Right Bundle Branch Block

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SUMMARY
The sequence of intraventricular conduction has been studied in a total of 60 patients, 38 of whom had normal QRS morphology and 37 of whom had right bundle branch block (RBBB) either present continuously or produced as functional aberrant RBBB by the introduction of atrial premature depolarizations or by rapid atrial pacing. Activation times were measured with intracardiac electrode catheters positioned at the right ventricular inflow tract (RVIT), right ventricular apex (RVA), right ventricular outflow tract (RVOT), left ventricular apex (LVA) and left ventricular outflow tract (LVOT). The activation after beginning of QRS in milliseconds ± 1 s and the number of patients studied at each location were: RVIT — normal 23 ± 13 (15 patients); RVIT-RBBB 49 ± 16 (15 patients); RVA — normal 18 ± 9 (28 patients); RVA-RBBB 54 ± 16 (30 patients); RVOT — normal 40 ± 10 (28 patients); RVOT-RBBB 78 ± 21 (30 patients); LVA — normal 9 ± 9 (18 patients); LVA-RBBB 6 ± 10 (10 patients); LVOT — normal 45 ± 13 (10 patients); LVOT-RBBB 32 ± 9 (7 patients). Significant differences observed were: RVA-normal versus RVA-RBBB P < 0.001; RVOT-normal versus RVOT-RBBB P < 0.001; RVA-normal versus LVA-normal P < 0.005; LVA-normal versus LVA-RBBB NS, LVOT-normal versus LVOT-RBBB P < 0.05. The LVOT change was unexpected and suggests changes in left ventricular depolarization may occur when right bundle branch block develops. In patients with RBBB the activation of the RVA (r = 0.82) and of the RVOT (r = 0.68) was directly related to the duration of QRS. Changes in activation time when RBBB was induced by rapid atrial pacing or by introduction of atrial premature depolarizations were: RVA (7 patients) 19 ± 11 to 56 ± 16 (P < 0.001); RVOT (9 patients) 41 ± 10 to 77 ± 22 (P < 0.001); LVA (5 patients) and LVOT (2 patients), small insignificant changes.

These data indicate that endocardial activation changes can be evaluated in the catheterization laboratory, that right ventricular conduction becomes slower in RBBB as a direct function of total QRS and that left ventricular conduction may be affected when RBBB develops.

Additional Indexing Words:
His bundle electrograms Septal depolarization Intracardiac electrocardiography
Intrinsic deflection

INVESTIGATORS using electrode catheter recording techniques have concentrated on conduction occurring within the A-V node and His-Purkinje system. For the past three years we have been investigating human intraventricular conduction in the catheterization laboratory, and report here our experiences with 60 patients. Thirty-eight patients had no conduction disturbances and 37 subjects had right bundle branch block (RBBB) which was either present continuously or produced as functional aberrant RBBB by the introduction of atrial premature depolarizations or by rapid atrial pacing. Included are the range of activation times observed in five locations of the right and left ventricles plus an analysis of the changes in activation produced at these locations when RBBB was produced in people with normal QRS morphology.

Patients and Methods
The investigation was performed on patients referred to members of the Cardiovascular Section for hemodynamic,
angiographic or electrophysiological diagnostic studies which were performed before the electrophysiological measurements were made. In our laboratory left heart catheterization is usually carried out through the right brachial artery with the Sones technique used for coronary arteriography. No left heart electrophysiological studies were conducted unless entry into the arterial system was indicated for independent diagnostic reasons.

Patients without apparent bundle branch block had QRS duration equal to or less than 105 msec. RBBB was recognized by the familiar signs of delay in right ventricular depolarization: wide S in lead I and V4; rSR' complex in V4. The QRS duration in the RBBB patients was equal to or greater than 110 msec. In some cases RBBB was produced in patients with normal QRS by the introduction of atrial premature depolarizations or by rapid atrial stimulation. No patients had evidence of pre-excitation. Clinical information about the patients is listed in table I.

Mapping the Left Ventricle

This portion of the study was conducted first to minimize time that the opened artery would be exposed. It followed, in many cases, the completion of left ventricular and coronary angiography. At least ten minutes had elapsed, however, before the measurements were taken. Changes in the electrocardiogram produced by angiography had returned to normal, but some residual effect on conduction produced by the contrast medium may still have been present.

Two points within the left ventricle were selected which could be located reproducibly: the apex and the outflow tract. The electrode catheter was introduced through the right brachial artery and first placed in the apex of the left ventricle (LVA). This location was established from its characteristic appearance on posterior-anterior and oblique fluoroscopic views (fig. 1). Recordings were made at this point (figs. 2, 3, 4). The electrode catheter was then slowly withdrawn with electrical information continually recorded on magnetic tape. The left ventricular outflow tract (LVOT) was identified by its fluoroscopic location and confirmed as the last point where high intensity ventricular myocardial potentials could be recorded before the catheter was withdrawn into the aortic root where the ventricular electrogram disappeared. Atrial signals could often be identified in the aortic root on higher amplification. In selected cases the LVOT was localized by simultaneous recording of pressure through the lumen of the electrode catheter. On slight further withdrawal the pressure tracing changed from ventricular to aortic. At this point the catheter was removed and the brachial artery repaired.

In order to avoid complications, a maximum of five minutes was allowed to gain access to the left ventricle and make the recordings. No embolic episodes were recognized in any patients whose left ventricles were mapped in this manner, and although ventricular ectopic beats were produced during catheter manipulation, ventricular fibrillation never occurred. Furthermore, these studies did not

![Figure 1](image)

Cine-fluoroscopic view of the heart (anterior-posterior projection) with electrode catheters in place. From the femoral vein one catheter has been placed in the right ventricular apex (RVA). The catheter from the right basilic vein has been positioned at the right ventricular outflow tract (RVO), and the arterial electrode catheter is at the left ventricular apex (LVA). The left ventricular outflow tract would have been mapped by withdrawal of the arterial catheter to a position slightly below the aortic valve. Right ventricular inflow tract activation was determined upon withdrawal of the right apical catheter to a position like that where a His bundle recording is usually made.

![Figure 2](image)

Depolarization at the left ventricular apex and right ventricular outflow tract were simultaneously recorded with leads I, II and V1 in a patient referred for evaluation of chest pain. Note the narrow deflections obtained with the close bipolar filtered lead (120-500 Hz). This example also illustrates the discrepancies commonly observed between beginning and completion of ventricular activation as reflected in the surface leads. Recording speed 200 mm/sec.

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Table 1
Clinical and Ventricular Activation Data on Patients with Normal QRS Duration and Right Bundle Branch Block

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INTRAVENTRICULAR CONDUCTION

Mapping the Right Ventricle

This procedure was usually performed after the brachial arterial repair had been completed. In a few cases catheters were inserted on both sides in order to record depolarizations simultaneously from both ventricles. The right ventricular electrode catheter was passed either by cut-down through the median basilic vein of the right arm or percutaneously through the right femoral vein.

Three locations were selected: the outflow tract, apex, and inflow tract (fig. 1). The outflow tract was identified as the point where ventricular electrical activity first appeared upon slow withdrawal of the catheter from the trunk of the pulmonary artery. In some cases this position was confirmed with simultaneous pressure recordings. The right ventricular apex was reached by advancing the catheter to the most distal portion of the body of the right ventricle. The catheter characteristically assumed a slightly caudal bend at this location. The right ventricular inflow tract was located by withdrawing the catheter to the position just below the tricuspid valve where His bundle recordings are made. Upon slight further removal a right atrial signal was obtained.

Catheters and Pacing

Initial studies in this project were conducted with standard bipolar electrode catheters manufactured by United States Catheter and Instrument Company and by Cordis Corporation with electrode separation of 1 cm. We later employed catheters with 1 mm electrode separation (Elecath Corporation), which produce electrograms with a narrower duration. In order to increase the stiffness of these catheters, lumen were included into which wire guides could be inserted.

Interval-dependent right bundle branch block was produced in some patients by the introduction of atrial prematurity beats or by rapid atrial pacing with a right atrial electrode catheter and a digitally programmed multi-

Recording Techniques and Equipment

The electrical signals detected in the electrode catheters were transmitted through a multipolar junction box and specially constructed lead selector to Electronics for Medicine Model EEP amplifiers with filter settings of 120-500 or 400-500 Hz.

In all cases the electrical information was recorded simultaneously at 3 1/2 inches/sec on a Phillips 7-channel or Hewlett-Packard 14-channel tape recorder. Multiple surface electrocardiograms were also recorded, usually leads I, II, and V, in some cases leads III and/or V, were included. Time marking was produced by the multi-impulse generator with intervals of 10 msec recorded onto tape during the procedure.

After the study the tapes were reviewed, often at 1 1/2 inches/sec, which facilitates selection of material for permanent record. The data were then photographed on paper at 200 mm/sec in the Electronics for Medicine recorder.

Measurement of Data

Vertical lines were drawn from the earliest and latest deflections in any of the surface leads to indicate the beginning and termination of measurable ventricular myocardial electrical activity. Local depolarization at the endocardial lead was taken as the point where the first fastest moving deflection crossed the isoelectric line. Measurements were made to the nearest 5 msec after onset of ventricular depolarization from the surface leads.

Initially up to ten different complexes were measured for each point. It quickly became clear that the time relationships between surface ECG and endocardial activation as recorded from the electrodes were stable and that no significant variation occurred once the electrode was stationary. Subsequently three complexes were measured for each point on the map with virtually equal intervals obtained in each case.

In some subjects all five points (LVA, LVOT, RVOT, RVA, RVIT) could be recorded. However, this was not always possible and on several occasions only one to four endocardial positions could be accurately localized. In patients undergoing only right heart catheterization, the maximum obtainable information was necessarily limited to one or three points. Consequently the total number of depolarizations evaluated from each patient is different.

Results

Activation of the Right Ventricle (figs. 2, 3, 4)

The average interval from onset of ventricular depolarization to activation of the right ventricular inflow tract (RVIT) in 15 patients with normal QRS duration was 23 ± 13 msec; in 15 patients with right bundle branch block the interval was 49 ± 16 msec* (table 1; figs. 5, 6, 7).

The average interval from onset of ventricular depolarization to activation of the right ventricular apex (RVA) in 28 patients with normal QRS duration was 18 ± 9 msec; in 30 patients with right bundle branch block the interval was 54 ± 16 msec.

*Data are reported as mean ± one standard deviation.
The average interval from onset of ventricular depolarization to activation of the right ventricular outflow tract (RVOT) in 28 patients with normal QRS duration was 40 ± 10 msec; in 30 patients with right bundle branch block the interval was 78 ± 21 msec. When both points were measured in the same patients, the RVOT was depolarized after the RVA on 44 of 46 occasions, at the same time in one, and before in one.

Activation of the Left Ventricle

The average interval from onset of ventricular depolarization to activation of the left ventricular apex (LVA) in 18 patients with normal QRS duration was 9 ± 9 msec; in 10 patients with right bundle branch block, the interval was 6 ± 10 msec.

The average interval from onset of ventricular depolarization to activation of the left ventricular outflow tract (LVOT) in 10 patients with normal QRS duration was 45 ± 13 msec; in 7 patients with right bundle branch block the interval was 32 ± 9 msec. When both points were measured in the same patient, the LVOT was activated later than the LVA on 12 of 14 occasions and before in two (patient J.B. in normal conduction and RBBB).

The significance of differences between groups of activation times were derived with the t-test system (table 2). In patients with either normal conduction or right bundle branch block significant differences were observed between right and left ventricular apex and outflow tracts. When the same locations were compared in patients with normal conduction versus those
Table 2

<table>
<thead>
<tr>
<th>Location</th>
<th>Comparison</th>
<th>Significance</th>
</tr>
</thead>
<tbody>
<tr>
<td>RVI</td>
<td>normal versus RBBB</td>
<td>P &lt; 0.001</td>
</tr>
<tr>
<td>RVA</td>
<td>normal versus RBBB</td>
<td>P &lt; 0.001</td>
</tr>
<tr>
<td>RVA</td>
<td>versus RVO (normal)</td>
<td>P &lt; 0.001</td>
</tr>
<tr>
<td>RVA</td>
<td>versus RVO (RBBB)</td>
<td>P &lt; 0.001</td>
</tr>
<tr>
<td>RVA</td>
<td>versus LVA (normal)</td>
<td>P &lt; 0.005</td>
</tr>
<tr>
<td>RVA</td>
<td>versus LVA (RBBB)</td>
<td>P &lt; 0.001</td>
</tr>
<tr>
<td>RVO</td>
<td>normal versus RBBB</td>
<td>P &lt; 0.001</td>
</tr>
<tr>
<td>RVO</td>
<td>versus LVA (normal)</td>
<td>P &lt; 0.001</td>
</tr>
<tr>
<td>RVO</td>
<td>versus LVA (RBBB)</td>
<td>P &lt; 0.001</td>
</tr>
<tr>
<td>LVA</td>
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</tr>
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<td>P &lt; 0.001</td>
</tr>
<tr>
<td>LVA</td>
<td>normal versus RBBB</td>
<td>NS</td>
</tr>
<tr>
<td>LVO</td>
<td>normal versus RBBB</td>
<td>P &lt; 0.05</td>
</tr>
</tbody>
</table>

with RBBB the activation times were significantly different at all right-sided points. At the left ventricular apex there was no significant change although the mean LVA activation time was slightly earlier during RBBB (9 ± 9 msec vs 6 ± 10 msec) and at the left ventricular outflow tract activation was also earlier in RBBB and significant at the 0.05 level (45 ± 13 msec vs 32 ± 9 msec).

Relationship of Activation Time and Duration of QRS

In the patients with right bundle branch block the activation of both right ventricular apex and right ventricular outflow tract was directly related to the duration of QRS (figs. 8, 9). At the RVA the expression QRS = 90 + 0.86 (RVA) was derived to describe the relationship. The correlation coefficient was 0.82. At the RVOT the expression QRS = 93 + 0.54 (RVOT) was derived. The correlation coefficient was 0.68. The probability of the straight lines being parallel to each other is greater than 90%.

Changes in Activation Time in the Same Patients

Activation at the right ventricular apex and right ventricular outflow tract was delayed in each patient with normal QRS as right bundle branch block was produced (fig. 10). At the right ventricular apex during normal conduction the activation time in seven patients was 19 ± 11 msec and it increased to 56 ± 16 msec when right bundle block was produced (P < 0.001). At the right ventricular outflow tract during normal conduction in nine patients the activation time was 41 ± 10 msec and it increased to 77 ± 22 msec when right bundle branch block was produced (P < 0.001). In these patients with normal conduction in whom right bundle branch block was produced, the range of values increased during RBBB, at the RVA from 30 msec in normal conduction to 40 msec during RBBB and at the RVOT from 30 msec in normal conduction to 65 msec during RBBB.

Comparisons at the left ventricular apex were observed in five patients with little if any difference recorded. At the left ventricular outflow tract comparisons were made in only two patients during both normal conduction and RBBB and the activation was identical in one and only slightly different in the other.

Changes in RVA-RVOT Activation Interval

In six patients recordings were made at both the right ventricular apex and right ventricular outflow tract during normal conduction and induced right
patients (CAD) vs 5 msec 4 patients (N); LVOT 43 msec 5 patients (CAD) vs 43 msec 3 patients (N). No significant differences were thus present when comparing the coronary patients with those with apparently normal hearts. Nor was there any significant difference between the coronary artery patients and the entire group of 38 patients with normal QRS.

Relationship of Axis Deviation and Activation

No definite relationship could be observed when the activation times at various locations were compared with the axis deviations in the frontal plane in any of the patients studied.

Discussion

Activation when QRS is Normal

Ventricular conduction studies in the dog heart have revealed: 1) that septal depolarization normally is dominated by the electrical activity of the left ventricular component so that activation proceeds for the most part from left to right; 2) that the apices of both ventricles are activated soon after septal activation begins; 3) that the remainder of the endocardium is quickly depolarized via the Purkinje network from below upward so that the basal and outflow tract endocardium are activated after the apices.5-13

Similar data on myocardial activation in man has been obtained from within the thoracic cavity during operations and from the perfused isolated human heart.5, 14-21 The activation times observed with endocardial electrodes in our study are consistent with the data obtained directly in the perfused human heart. The apices were activated before the outflow tracts with the LV apex preceding the right in most cases.

Activation times for the right ventricular apex and right ventricular outflow tract are comparable to those obtained in six patients by Castellanos et al. who reported 15–30 msec (average 18.3 msec) for the RVA and 25–50 msec (average 33.3 msec) for the RVOT.22 By also recording from within the great cardiac vein they obtained an epicardial left ventricular posteroseptal activation time of 50–60 msec (average 56 msec). The activation front thus appears to arrive later at this position on the left ventricular epicardium than at the basal endocardial left ventricular outflow tract as recorded in our patients. Such a finding is expected since endocardial activation precedes epicardial depolarization in neighboring areas.21

Initial human right ventricular endocardial activation occurs 5–10 msec after the onset of the left ventricular cavity potential as recorded from revived perfused hearts.21 We found similar results when the activation at right ventricular apex was compared with left ventricular apex. In only one case (E.W.) from a

Figure 10

The changes in activation when right bundle branch block was produced in patients with normal QRS at rest. In each case endocardial depolarization was delayed at both locations in the right ventricle.

Relationship of Activation to Clinical Diagnosis

The data in the patients with normal conduction was examined to determine whether particular clinical diagnoses affected activation times. In those with coronary artery disease (CAD) the following mean activation times were observed compared with the mean values for patients without demonstrable structural cardiac disease (N): RVA 17 msec 10 patients (CAD) vs 21 msec 7 patients (N); RVOT 43 msec 10 patients (CAD) vs 42 msec 4 patients (N); LVA 11 msec 9

Figure 11

Activation spread between right ventricular apex and right ventricular outflow tract. The interval between activation of RVA and RVOT was greater when right bundle branch block was produced in patients with normal QRS duration in five of six patients.
Activation in Right Bundle Branch Block

Depolarization on the right side of the heart is delayed by varying amounts during right bundle branch block. The range in six patients studied by Castellanos et al. was 50–60 msec (average 51) at the right ventricular apex, and 70–95 (average 84) in the right ventricular outflow tract. The range of activation times in our larger series was certainly greater although the means were quite similar. Delays in the recorded right ventricular points are also the rule when RBBB is produced in dogs.11, 12, 23–26

One would not expect the activation of the left ventricle to be much affected when right bundle branch block develops. However, in our patients, the LVA and LVOT were depolarized slightly earlier during RBBB. These differences were statistically significant at the LVOT but not at the LVA. The comparisons especially at the LVA must be viewed with caution, particularly since differences were slight in the few patients where direct comparisons could be made. Nevertheless, one could conclude that during RBBB some change may occur in the sequence of LV activation so that at least the LVOT is depolarized earlier. Perhaps partial fascicular blocks in the LV during RBBB may cause activation to produce such unexpected results. In the six patients reported by Castellanos et al. no change in the activation of the base of the LV as recorded from within the great cardiac vein was observed when RBBB was produced.22 In dogs the production of right bundle branch block also does not appear to affect the normal activation of the left heart.23, 24

Degrees of Right Bundle Branch Block

Although “complete” right bundle branch block is said to be present when QRS duration is but 120 msec, clinical electrocardiographic findings suggest otherwise. Actually, “complete” RBBB in man may well be quite uncommon, as discussed by Grant.27 Scher has estimated by extrapolation from canine studies that for RBBB to be complete, the QRS width would have to be 160 msec.11 In only two of our patients (I.D. and A.R.) did the total QRS duration exceed this value, and then only to 170 msec.

Our data show that as QRS width increases, activation of the RVA and RVOT occurs later. For reasons that are not immediately apparent, the correlation appears slightly closer in the RVA.

Comparisons in Individual Patients

The expected delay in right side conduction was found whenever activation could be compared in the same patient when QRS was normal and right bundle branch block was produced. The amount of conduction delay varies when the extra-stimulus or rapid atrial pacing method is used, and it seems clear that “complete” RBBB will not usually be achieved.28 The greater range of values at RVA and RVOT when right bundle branch block was produced presumably reflects the differing degrees of RBBB which resulted (fig. 10).

Although the right ventricular apex is activated early and directly from the right bundle branch, the exact source of activation of the outflow tract is not fully understood. We therefore found it particularly interesting that relative to the RVA, the RVOT was activated even later in RBBB than when QRS was normal in five of six patients where the measurements could be compared (fig. 11). Although Castellanos et al. did not specifically comment on this point, the same conclusion could be reached from their data.22 The explanation for this finding may be either that the sequence of activation within the RV is different during RBBB or that the “route” remains the same, and more of the slower muscle-to-muscle conduction is now required. Fascicular blocks might also slow conduction to particular RV locations.

Sources of Variation

The range of values for individual points in our series probably results from: 1) individual variations from patient to patient; 2) different location of the electrode in different patients; 3) the area of myocardium in contact with the electrodes.

During normal conduction, it appears that endocardial activation is reproducible with high consistency between similar locations.23 A somewhat wider variation has been recorded in epicardial activation although the general depolarization pattern was predictable.29 In dogs the endocardial pattern determined with needle electrodes inserted under direct vision also occurs for the most part in a consistent, predictable fashion.3 However, QRS width is narrower in dogs than in man, and changes in time of particular sites are proportionately less. To our knowledge no study has been performed which reveals the range of activation times at the same locations in a large group of normal dogs.

It seems likely that individual differences in activation may be caused by myocardial disease even though total QRS width is normal. For example, some of the later left ventricular apical activation times were seen in patients with severe coronary artery disease. Patient J.B. (age 60) had severe myocardial fibrosis, and he was the only case where the LVA was depolarized after the outflow tract. However, our pop-

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ulation is not large enough to draw definite conclusions in this regard.

Because of the heterogeneous nature of the diagnoses in our patients, we further examined the groups in an effort to determine if the activation in certain subsets of patients differed from the group as a whole. Except for individual variations from the mean, no additional significant patterns of differences emerged based on diagnosis.

Part of the variation is certainly due to placement of the electrodes. Particular care was taken in guiding the catheters into the predetermined locations. Despite these efforts, some differences must have occurred. In the outflow tracts for example, adjacent surfaces at the same level are activated differently, and it is easy to visualize how the apical locations could vary depending upon local hypertrophy of papillary muscles or muscle bundles. We have observed slight differences in endocardial activation time even between unipolar records taken from adjacent 1.5 cm electrodes. Consequently, if close bipolar electrodes were positioned slightly differently, some variations in depolarization time might well be observed. Similar restrictions apply to studies carried out with transmyocardial needle electrodes. Only by direct visualization can the needles be exactly localized.

The actual width of each exposed electrode tip is 2 mm. Although these surfaces are small, they rest against myocardium and/or Purkinje tissue which may be activated over at least a narrow time interval. The catheter technique, therefore, is less precise, on these grounds alone, than the needle electrode technique with its smaller surface area.

Our data show that the range of human endocardial activation times recorded with electrode catheters is quite broad. Part of these variations must reflect subject-to-subject differences, but certainly technical considerations are important. Thus the method is perhaps most useful when comparing activation changes in an individual patient as certain perturbations are instituted such as changes in intraventricular conduction produced by atrial stimulation. However, the activation differences between right bundle branch block and normal conduction are clear and help to support the data from animals and from humans at operation. At this writing, the technique described here is the only one available which permits us to study by direct myocardial contact certain features of intraventricular conduction in the conscious human subject who has not undergone cardiac surgery.

Other Applications of the Technique

Reports have recently appeared which tell of the value to be realized from conduction studies with en-

docardial electrodes. From the laboratory of Castellanos and his associates have come observations on: intra-atrial conduction, atrioventricular and intraventricular conduction, pre-excitation and ventricular tachycardia. These investigations in the catheter laboratory are being performed at a time when provocative new data about endocardial activation in dogs and in man studied during open heart surgery are also appearing. Correlations among these different methods should provide valuable new information about intraventricular conduction in healthy and abnormal hearts.

Acknowledgment


References

1. KASTOR JA, GOLDBREYR BN, SHELBRUNE JC, MANCHESTER JH: Intraventricular conduction in man studied with an endocardial mapping technique. Circulation 45 and 46 (suppl II): II-26, 1972


4. DURBER D, VAN DER TWEEL LH: Excitation of the left ventricular wall of the dog and goat. Ann NY Acad Sci 65: 779, 1957


10. SCHER AM, YOUNG AC: Ventricular depolarization and the genesis of QRS. Ann NY Acad Sci 65: 768, 1957


14. BARKER PS, MACLEOD AG, ALEXANDER J: The excitatory process observed in the exposed human heart. Am Heart J 5: 720, 1930

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Intraventricular conduction in man studied with an endocardial electrode catheter mapping technique. Patients with normal QRS and right bundle branch block.

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