Letters to the Editor

Letters to the Editor will be published, if suitable, and as space permits. They should not exceed 500 words in length, and may be subject to editing or abridgement.

Significance of First Degree A-V Block

To the Editor:

The following comments should be offered about the paper, "Electrophysiological Significance of First Degree Atrioventricular Block with Intraventricular Conduction Disturbance," by Rosen et al. (Circulation 43: 491, 1971).

The H-Q interval was measured from the onset of the H to the onset of the surface ECG QRS complex. This interval varies somewhat with the surface ECG lead utilized. To minimize the error in measurement, multiple surface ECG leads representing all three planes should be recorded simultaneously and the earliest onset of the QRS utilized. This potential variability in measurement technique may explain the wide range of normal H-Q interval in the series published (35–55 msec), as opposed to the normal range in our laboratory (35–45 msec). Furthermore, to provide a meaningful interpretation for the quantitative and qualitative data, the His bundle (BH) electrogram should be validated by the only means available, i.e., His bundle pacing.

Similar comments are in order for the measurement of the P-H interval because the onset of the P wave varies somewhat in different surface ECG leads. In addition, the P-H interval represents the sum total of intra-atrial (P-A) and A-V nodal (A-H) conduction times. The P-A and A-H intervals should be measured separately. First degree A-V block due to an abnormal P-A interval in the face of a normal A-H and H-V time has been described.

The role of lesions in the mainstem BH has been well documented. A prolonged H-Q interval does not necessarily mean delay in both bundle branches but may simply reflect conduction delay within the mainstem BH. This concept has not been stressed with sufficient clarity. The casual reader may misinterpret the concepts expressed as to the meaning of a prolonged H-Q time.

The conclusion that "combination of left bundle-branch block (LBBB) and first degree A-V block usually has significant H-Q prolongation," should be interpreted in comparison with the incidence of abnormal H-Q time in the presence of a normal P-R interval and LBBB. The Rosen study does not provide such data. The same is true for the group with right bundle-branch block (RBBB) and first degree A-V block.

Data from our series of patients are presented below in an effort to give proper perspective to these findings. Our group A consisted of 44 patients with LBBB. In 21 patients with first degree A-V block and LBBB, the H-V (H-Q) time was abnormal in 90%, and combined A-H and H-V abnormalities were present in 52% of the cases. On the other hand, of the remaining 23 patients with a normal P-R interval, the H-V time was abnormal in 70%. Group B with RBBB consisted of 108 patients. In the 25 patients with an abnormal P-R interval and RBBB, 76% had an abnormal H-V time. Two of these 25 patients, despite RBBB, left axis deviation, and first degree block, showed normal H-V times. In the
remaining 83 patients with a normal P-R interval, the H-V time was abnormal in 58%.

In conclusion, in a given patient with first degree block, the delay may be present in one or more of the four regions of the A-V conduction system, i.e., intra-atrial, A-V nodal, mainstem BH, and bundle branches. It is difficult to predict the site of delay from the surface ECG alone.

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References


The authors reply:

To the Editor:

The normal values for P-H and H-Q in our laboratory were established using V1 as a reference lead.1 Subsequent studies in over 40 patients, using a multiple simultaneous lead system (leads I, II, III, and V1) have confirmed these ranges.2 Defining 45 msec as the upper limit of normal for H-Q, as proposed by Narula and Samet, greatly increases the incidence of H-Q prolongation in patients with and without conduction disease.

Narula and Samet seem to have misinterpreted the purpose of the study. We were primarily interested in determining the sites of delay producing P-R prolongation in patients with first degree A-V block (AVB) and bundle-branch block. The incidence of conduction delays in patients with normal P-R and bundle-branch block, although of interest, was not essential to the purpose of the study. We have frequently found H-Q prolongation in patients with left bundle-branch block (LBBB), with both normal and prolonged P-R intervals.3 However, it should be stressed that H-Q was more markedly prolonged in the latter group. Nine of our 12 patients with first degree AVB and LBBB had H-Q intervals ranging from 75–125 msec, which contributed very significantly to P-R prolongation. This was in contrast to our patients with first degree AVB and RBBB, in whom H-Q ranged from only 35–65 msec, contributing much less to P-R prolongation. In both groups, P-H was both frequently and significantly prolonged. Narula and Samet appear to have corroborated the latter finding.

In regard to the sites of conduction delay proposed by Narula and Samet: (1) all of our patients with P-H prolongation had A-H prolongation (P-A [intra-atrial] prolongation is a rare cause of first degree A-V block, and did not contribute significantly to first degree A-V block in our series); and (2) the presence of mainstem BH lesions as well as all other lesions defined by the His bundle technique, cannot be considered “well documented” until there is pathological confirmation of such lesions utilizing serial section of the conduction system.

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