The Role of Left Ventricular Conduction in the Electrogenesis of Left Ventricular Hypertrophy

An Electrophysiologic Study in Man

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SUMMARY Various electrocardiographic and vectorcardiographic (ECG-VCG) patterns of ventricular conduction disturbances are inducible by premature right atrial stimulation (PRAS). These patterns are a consequence of different degrees of refractoriness in the specialized ventricular conduction system. We observed that the intermediate phases of left bundle branch block (LBBB) induced by PRAS in 20 subjects with normal basal QRS (complexes) were similar to those of left ventricular hypertrophy (LVH). In 18 patients with basal ECG-VCG signs of LVH, right bundle branch block (RBBB) induced by PRAS produced a progressive disappearance of these signs and the "normalization" of the tracings. The initial maximum QRS vector decreased, disappeared or remained absent in the patients with LBBB induced by PRAS, and appeared (when absent in the basal VCG) and remained unchanged (when present) in patients with RBBB induced by PRAS.

In this paper we discuss the electrogenetic implications of these data. The ECG-VCG signs of LVH are probably dependent on a slowed conduction in the left bundle branch system, while anatomical hypertrophy per se probably plays a less important role.

THE ELECTROCARDIOGRAPHIC and vectorcardiographic signs of left ventricular hypertrophy (LVH) are well known, but their electrogenesis is still uncertain.1-19 Some findings suggest a mechanism of left bundle branch system impairment.7, 8, 12, 15, 17, 18, 20-23 but no clearcut evidence has been presented.

Premature right atrial stimulation (PRAS) and LVH produce similar QRS configurations.24 Thus, we hypothesize that if electrocardiographic signs of LVH depend on left ventricular conduction disturbances, one would expect that 1) the induction of such signs in patients with normal basal QRS would be the effect of slowed left bundle branch conduction, and 2) the disappearance of these signs in patients with electrocardiographic patterns of LVH would be the effect of slowed right bundle branch conduction. To determine the validity of these assumptions, we examined the results of an electrophysiologic study in man.

Materials and Methods

We divided 38 patients into two groups before beginning electrophysiologic studies. Group 1 (14 males and four females, aged 24-72 years, mean age 47.5 years) consisted of patients with clinical or angiographic and electrocardiographic-vectorcardiographic signs of LVH. The data concerning these patients are listed in table 1 and the electrocardiographic14, 15 and vectorcardiographic1, 19 criteria for LVH diagnosis are summarized in tables 2 and 3. The second group (13 males and seven females, ages 19-66 years, mean age 42.5 years) included subjects with cardiac failure or right ventricular hypertrophy and with normal QRS and without clinical and radiological signs of LVH. We excluded patients with electrocardiographic or vectorcardiographic evidence of bundle branch or fascicular block.24

All patients were in sinus rhythm at the time of the electrophysiologic study, and cardioactive drugs were discontinued at least 48 hours before. The study was performed with patients in the resting, postabsorptive, nonsedated state. All patients gave informed written consent. Two #6F bipolar electrode catheters were passed percutaneously via the femoral veins into the right heart; the first was positioned in the high right atrium for the PRAS and the second was advanced to the tricuspid orifice to record His bundle activity according to the method of Scherlag et al.26 Single premature atrial stimuli were delivered after every eighth spontaneous beat at progressively decreasing 10-msec coupling intervals until the atrioventricular (AV) nodal or ventricular specialized conduction system effective refractory period (ERP) was reached. We used the bipolar atrial electrogram as the trigger signal. Stimuli were approximately twice diastolic threshold, 2.5 msec in duration and elicited by a programmable digital stimulator (El Desi CD 6). The PRAS was performed during spontaneous sinus rhythm and not during atrial driving because in this way it is easier to find the refractoriness of the His-Purkinje system. The His bundle electrogram, the surface ECG (leads I, II, III and V1), and the frontal, horizontal and left sagittal planes of VCG (Frank method)26 were simultaneously recorded during PRAS, using an eight-channel direct writing recorder (Elena Schönander Mingograf `81) at a paper speed of 50 or 100 mm/sec and an ICR 1001 Instant VCG vec-
torcardiograph, respectively. When the ERP of the AV node was longer than that of the His-Purkinje system, 0.25 mg atropine i.v. was generally sufficient to reduce the AV nodal ERP, without significant changes in the heart rate and the ERP of the His-Purkinje system.27

At the end of the study the localization of the activation delay in the His-Purkinje system was extrapolated on the basis of the changes in morphology and duration of the induced ventricular aberrances. We considered 1) anterior displacement of the QRS horizontal loop as a right ventricular conduction disturbance (see discussion), and 2) the reduction or disappearance of the initial QRS vectors without an increase of the QRS duration as a possible conduction disturbance of the left septal fascicular branch (LSFB) or mid-septal network. In some induced QRS loops the same vectorcardiographic parameters analyzed in the basal tracings for LVH diagnosis were then calculated (tables 2 and 3). Since voltage and direction of the initial QRS vectors are related variables in different cases of LVH, the voltage, direction and time of inscription of the maximum horizontal initial vector in the basal and induced QRS loops were also calculated. Normal values of this vector are listed in table 4.19 The basal and induced HV interval were determined by His bundle recording.

Results

Table 5 shows the changes induced by PRAS in the LVH group (18 cases): in four cases (22.2%) a RBBB developed after a progressive anterior displacement of the QRS horizontal loop, associated with disappearance of LVH signs, reduction of QRS duration (14, 10, 14 and 10 msec, respectively) and "normalization" of the tracing (table 6, figs. 1 and 2). Leftward shift of the initial vectors was the earlier aberrance in one case (fig. 2). The HV interval lengthened in three cases and remained unchanged in one. At shorter coupling intervals, PRAS induced in two cases a complete LBBB pattern through intermediate phases of QRS prolongation with different patterns of LVH. In three cases (16.7%) only an anterior displacement of horizontal QRS was induced, not followed by the classic patterns of RBBB. In two of these it was preceded by a leftward deviation of the initial vectors. Basal signs of LVH and QRS duration tended to normalize in all three cases (table 6), without significant changes of the HV interval. In five cases (27.9%) a progressive left ventricular conduction disturbance like LBBB but indistinguishable from other aspects of LVH was induced. One of these patients presented an early disappearance of the initial vectors and the other two later developed RBBB with disappearance of LVH and LBBB signs. In the remaining six cases (33.2%) the PRAS induced leftward deviation of the initial vectors followed by left posterior hemiblock in one case, left anterior hemiblock in one case, disappearance of initial vectors in one case and indeterminable small changes in the other three cases.

In group 2, patients with normal basal QRS (20 cases), the earlier ventricular aberrances induced by PRAS were RBBB in 13 cases (65%), LBBB in four cases (20%) and left anterior hemiblock in one case (5%). No appreciable changes occurred in two cases (10%). The QRS duration of the intermediate phases with anterior displacement in induced RBBB did not decrease, unlike in the patients with basal LVH. When LBBB developed, the intermediate QRS aspects simulated those of LVH (fig. 3).

The hemodynamic overloading of the left ventricle (table 7) in the LVH group was diastolic in six cases, systolic in seven cases, systolic and diastolic in three cases and undefined in two cases. The maximum initial QRS vector in the vectorcardiographic horizontal plane was increased in one case, normal in two cases and absent in three of six cases with diastolic overloading of the left ventricle; increased in one case, normal in two cases, decreased in three cases and leftward oriented in one of the seven cases with systolic overloading of the left ventricle; normal in two cases and leftward oriented in one of the three cases with mixed overloading of the left ventricle.

The initial maximum horizontal QRS vector decreased, disappeared or remained absent in patients with LBBB induced by PRAS. It appeared, when absent, in the basal ECG, or remained unchanged, when present, in those with RBBB induced by PRAS (table 7). In the correlation with His bundle recording, the changes of initial maximum horizontal QRS vector in the PRAS induced LBBB were never associated with lengthening of the HV interval.

Discussion

The electrocardiographic-vectorcardiographic analysis of the QRS aberrances induced by PRAS and simultaneously recorded with His bundle electrogram is a recent technique for studying intraventricular conduction disturbances in man.21, 28, 29 We have used this method to evaluate the role of ventricular conduction in the electrogensis of LVH. PRAS resulted in classic conduction disturbances, as well as other morphologies which were difficult to interpret. Among these the anterior displacement of QRS horizontal loop is very important for the interpretation of our data. We considered any evident anterior shifting of the centrifugal QRS horizontal loop compared with the basal tracing as QRS anterior displacement. We interpreted this finding as a right ventricular conduction disturbance because 1) it represented the typical evolutional pattern before reaching the classic RBBB pattern, while the progression toward the induced LBBB or toward left hemiblocks presented other typical morphologies; and 2) in cases of basal incomplete LBBB, the induced anterior displacement was always associated with a reduced QRS duration and frequently with a prolonged HV interval (our unpublished observations). Other investigators thought that the anterior displacement of the QRS horizontal loop observed in clinical cases depended on left conduction disturbances in the anterior subdivision of the
left bundle branch\(^\text{20}\) or in the left septal fascicles.\(^\text{31}\) However, 1) Hoffman and co-workers\(^\text{20}\) argued on the basis of the indirect data (angiography) that pathology of the anterior descending coronary artery may result in a right bundle branch involvement; and that 2) Nakaya et al. based their hypothesis on a marked fibrosis of the left septal fascicle observed at post-mortem examination in only one case, in which marked fibrosis of the right bundle branch was also

<table>
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<th>ID</th>
<th>ST-T</th>
<th>AQRS</th>
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<td>+</td>
<td>2.55</td>
<td>2.45</td>
<td>_</td>
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<td>M</td>
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<td>-</td>
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<td>+</td>
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<td>1.96</td>
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<td>18</td>
<td>74</td>
<td>F</td>
<td>AH</td>
<td>38</td>
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<td>+</td>
<td>+10°</td>
<td>+</td>
<td>2.75</td>
<td>2.05</td>
<td>-36°</td>
</tr>
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</table>

Abbreviations: AI = aortic insufficiency; AS = aortic stenosis; MI = mitral insufficiency; CM = cardiomyopathy; AH = arterial hypertension; SLI = Sokolow-Lyon index; ID = intrinsecoid deflection; ST-T = depression of ST segment and/or flattening or inversion of T wave; LAE = left atrial enlargement; MSV = voltage of maximum spatial vector; MHV = voltage of maximum horizontal vector; MHVd = direction of MHV; MPV = voltage of maximum frontal vector; MPVd = direction of MPV; MVH = time of inscription of MHV; QRSD = duration of QR; MVd = direction of maximum horizontal initial vector; MVd = direction of maximum horizontal terminal vector; MT = angle between MHV and MV; MPV = voltage of maximum posterior vector; DA > PA = distal QRSH area > proximal QRSH area; STHV = direction of horizontal ST vector; MTHV = direction of maximum horizontal T vector; + = present; _ = absent; ind = indeterminable.

The LVH diagnosis is considered probable when the Sokolow-Lyon index is the only positive sign and certain when one or more of the other criteria are also positive.

**Table 2. Electrocardiographic Criteria for Left Ventricular Hypertrophy (LVH)**

1. Sokolow-Lyon index (SV\(_1\) + RV\(_1\) - V\(_6\)) \(\geq 35\) mm.
2. QRSD duration \(\geq 0.09\) second (max 0.11–0.12 second).
3. Onset of the intrinsecoid deflection in the left precordial leads = 0.04 second (without Q wave) and = 0.05 second (when Q wave is present).
4. ST-segment depression and T-wave flattening or inversion in the left precordial leads.
5. QRSD axis between +30° and -30°.

**Table 3. Vectorcardiographic Criteria for Left Ventricular Hypertrophy (LVH)**

1. Maximal spatial vector \(\geq 2.10\) mV.
2. Maximal horizontal vector \(\geq 2.0\) mV (1.90 mV over 40 years).
3. Maximal frontal vector \(\geq 2.40\) mV (2.00 mV over 40 years).
4. Time of inscription of the maximum QRSH vector \(\geq 47.5\) msec.
5. Duration of the QRSD loop \(> 90\) msec \(< 120\) msec.
6. Leftward orientation both of the initial and terminal deflections.
7. Direction of the terminal horizontal deflection anterior to \(-65°\).
8. Angle between the maximum QRSH vector and terminal deflection \(< 35°\).
9. Maximum leftward posterior horizontal force \(> 1.20\) mV.
10. Distal QRSH loop area larger than the proximal one.
11. Direction of the ST\(_F\) vector to the right of 120°.
12. Direction of the maximum T\(_F\) vector to the right of 70°.

The LVH diagnosis is considered probable when the voltage criteria are fulfilled and certain when one or more of the other criteria are also present.
TABLE 1. (Continued)

<table>
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<tr>
<th>MFV</th>
<th>MFVd</th>
<th>MHVt</th>
<th>QRS</th>
<th>MIVd</th>
<th>MTVd</th>
<th>MT</th>
<th>MPVv</th>
<th>DA &gt; PA</th>
<th>STHV</th>
<th>MTHV</th>
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<tr>
<td>1.50</td>
<td>+0.60</td>
<td>0.37</td>
<td>0.06-0.37 mV (mean 0.20 mV). Direction: +90° +149° (+64° +90° when QRS frontal loop is vertical). Duration: 4-16 msec (mean 11 msec).</td>
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</table>

TABLE 4. Normal Values of the Initial Maximum QRS Vector in the Horizontal Plane

Our results seem to confirm our introductory hypothesis. In fact, we obtained both some ventricular aberrances similar to LVH in normal basal QRS and an increase of the basal LVH signs when the PRAS evoked a slowed conduction in the left bundle branch system. However, we obtained the disappearance of basal LVH signs and the “normalization” of the electrocardiographic-vectorcardiographic tracings when the PRAS evoked a slowed conduction in the right bundle branch system. Furthermore, disappearance of the basal LVH signs in the course of PRAS always coincided with evident reduction of QRS duration. These findings suggest that an induced slowed conduction in the right bundle branch balanced a preexistent conduction disturbance in the main stem of the left bundle branch (table 5, fig. 2).

These results seem to demonstrate that electrocardiographic-vectorcardiographic signs of LVH, at least in our cases, are dependent on a slowed conduction in the left bundle branch system, while the anatomic hypertrophy per se probably plays a less im-

TABLE 5. QRS Aberrances Induced in Patients with Basal Left Ventricular Hypertrophy

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<th>Second</th>
<th>Third</th>
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<tbody>
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<td>RBBB</td>
<td>LBBB</td>
</tr>
<tr>
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<td>AD</td>
<td>RBBB</td>
<td>LBBB</td>
</tr>
<tr>
<td>3</td>
<td>LSFB</td>
<td>AD</td>
<td>RBBB</td>
</tr>
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<td>AD</td>
<td>RBBB</td>
<td></td>
</tr>
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<td>LSFB</td>
<td>AD</td>
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</tr>
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<tr>
<td>10</td>
<td>LBBB</td>
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<td></td>
</tr>
<tr>
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<td>14</td>
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<td>18</td>
<td>NCCh</td>
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Abbreviations: AD = anterior displacement; RBBB = right bundle branch block; LBBB = left bundle branch block; LSFB = left septal fascicular block; NCCh = non-characteristic changes; LAFB = left anterior fascicular block; LPFB = left posterior fascicular block.
Figure 1. Recordings from 49-year-old female with aortic and mitral insufficiency. A) The left panel shows a basal VCG in horizontal (H) and frontal (F) planes with signs of left ventricular hypertrophy. The center panel shows leads I, II, III and V1 with His bundle electrogram (HBE) simultaneously registered with the VCG of the left panel (first beat) and with the VCG of the right panel (second beat). The right panel shows the VCG of the first ventricular aberrance induced by premature right atrial stimulation at a coupling interval (A1-A2) of 445 msec. Note the leftward shifting of the initial vectors and the slight shortening of the maximum spatial (MSV) and horizontal (MHV) QRS vectors. B) Same patients as in figure 1A. At increased coupling intervals (A1-A2 = 390 msec) and 355 msec, the more leftward displacement of the initial vectors, the progressive shortening of left maximum spatial (MSV) and horizontal (MHV) vectors and the widening of the QRS horizontal loop can be seen. C) Same patient as in figures 1A and 1B. At further increased coupling intervals (A1-A2 = 325 msec and 340 msec) a further shortening of left maximum spatial (MSV) and horizontal (MHV) vectors (left panels) and the appearance of right bundle branch block (right panels) can be seen. Note that when this conduction disturbance is manifest, the QRS duration (82 msec) is still less than that of basal tracing (94 msec).
FIGURE 2. Recordings from 46-year-old male with arterial hypertension. Figures 2A–C show, as in the previous case, the electrocardiographic and vectorcardiographic left ventricular hypertrophy "normalization" after right bundle branch block induced by premature right atrial stimulation. Abbreviations and disposition of tracings as in figures 1A–C.

TABLE 6. The Changes in Vectorcardiographic Data in Left Ventricular Hypertrophy Cases in Which Premature Right Atrial Stimulation Induced Right Bundle Branch Block or Anterior Displacement of the Horizontal QRS Loop

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<th>QRS</th>
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<td>RBBB</td>
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<td>+10°</td>
<td>76</td>
<td>40</td>
<td>50</td>
</tr>
<tr>
<td>5</td>
<td>2.50</td>
<td>-8°</td>
<td>112</td>
<td>52</td>
<td>45</td>
<td>AD</td>
<td>1.60</td>
<td>-2°</td>
<td>96</td>
<td>46</td>
<td>45</td>
</tr>
<tr>
<td>6</td>
<td>2.45</td>
<td>0°</td>
<td>96</td>
<td>46</td>
<td>40</td>
<td>AD</td>
<td>1.72</td>
<td>+2°</td>
<td>84</td>
<td>36</td>
<td>40</td>
</tr>
<tr>
<td>7</td>
<td>1.80</td>
<td>-2°</td>
<td>90</td>
<td>44</td>
<td>40</td>
<td>AD</td>
<td>1.60</td>
<td>+12°</td>
<td>80</td>
<td>40</td>
<td>40</td>
</tr>
</tbody>
</table>

Abbreviations: PRAS = premature right atrial stimulation; MHV = voltage of maximum horizontal vector; MHVd = direction of MHV; QRS = duration of QRS; MHVt = time of inscription of the MHV; HV = HV interval; RBBB = right bundle branch block; AD = anterior displacement.
importance role. Particularly, the increased and displaced left ventricular forces in LVH are probably caused by the damaged or stretched left conduction pathways rather than by increased thickening of the left ventricular wall and septum. This electrogenetic mechanism may also explain the low sensitivity and specificity of the clinical electrocardiographic and vectorcardiographic tracings in the LVH diagnosis and the poor correlation between electrocardiographic-vectorcardiographic data and the thickness of the left ventricular wall or the hemodynamic parameters.

With regard to the behavior of initial QRS vectors in LVH, various electrogenetic mechanisms may be invoked. However, we believe that the different changes of the initial vectors could be related to intraventricular conduction disturbances at different sites. On the basis of recent morphologic, electrocardiographic, clinical, and electrophysiological studies we could assume that these conduction disturbances are localized in the main left bundle branch or in the left septal fascicles when the initial vectors are reduced or absent, and in the distal portions of the subdivisions of the left bundle branch when the initial vectors are normal or augmented. Our results seem in part to confirm these possibilities. Indeed, the presence of normal or augmented initial QRS vectors in the basal tracings of some LVH cases and their reduction or disappearance in induced left ventricular arrhythmias probably suggest 1) a slowed conduction in the distal parts of the left bundle branch system in the former, as the effect of a reduced competition of the left ventricular wall during left septal activation, and 2) a slowed conduction in the main left bundle branch or in the left septal fascicles in the latter. However, other hypotheses are also possible: a local modification of septal activation as well as a different involvement of the anterior and posterior subdivisions of the left bundle branch.

Finally, the left axis deviation generally observed in LVH does not seem to be caused by left ventricular hypertrophy per se or by counterclockwise rotation of
the heart on its longitudinal axis. Our data suggest that the left axis deviation is related to a left conduction disturbance and probably to a greater slowing of the conduction in the left anterior subdivision of the left bundle branch.

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