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 perhaps plays a less important role.

THE ELECTROCARDIOGRAPHIC and vectorcardiographic (ECG-VCG) patterns of left ventricular hypertrophy (LVH) are well known, but their electrogenesis is still uncertain. Some findings suggest a mechanism of left bundle branch system impairment but no clearcut evidence has been presented.

Premature right atrial stimulation (PRAS) and LVH produce similar QRS configurations. 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 electrocardiographic and vectorcardiographic 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.

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. 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 electrogam 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 electromogram, the surface ECG (leads I, II, III and V1), and the frontal, horizontal and left sagittal planes of VCG (Frank method) were simultaneously recorded during PRAS, using an eight-channel direct writing recorder (Elema 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.24, 25, 28 We have used this method to evaluate the role of ventricular conduction in the electrogenesis 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 evolitional 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\textsuperscript{20} or in the left septal fascicles.\textsuperscript{31} However, 1) Hoffman and co-workers\textsuperscript{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

\begin{table}
\centering
\caption{Clinical and Electrocardiographic and Vectorcardiographic Data—Left Ventricular Hypertrophy Group}
\begin{tabular}{cccccccccccc}
\hline
Case & Age & Sex & Cardiopathy & SLI & ID & ST-T & AQRS & LAE & MSV & MHV & MHVd \\
\hline
1 & 43 & M & AI + AS & 78 & 0.05 & - & +45° & + & 3.43 & 3.25 & -30° \\
2 & 34 & M & AI & 44 & 0.05 & - & +50° & - & 3.00 & 2.40 & -8° \\
3 & 46 & F & AH & 40 & 0.055 & + & 0° & - & 2.32 & 2.25 & -23° \\
4 & 49 & F & AI + MI & 64 & 0.045 & + & +45° & + & 4.03 & 3.75 & -47° \\
5 & 55 & M & AS & 50 & 0.045 & + & -10° & + & 2.60 & 2.50 & -8° \\
6 & 52 & M & AH & 40 & 0.045 & - & +30° & + & 2.55 & 2.45 & 0° \\
7 & 57 & M & AI & 36 & 0.035 & + & +60° & - & 2.85 & 1.80 & -2° \\
8 & 42 & M & AI & 29 & 0.040 & + & +30° & + & 2.38 & 2.27 & +13° \\
9 & 54 & M & AI + AS & 45 & 0.045 & + & +45° & + & 2.45 & 2.10 & -77° \\
10 & 24 & M & AI & 40 & 0.047 & + & -10° & + & 3.43 & 3.42 & -30° \\
11 & 28 & M & CM & 43 & 0.040 & + & +30° & - & 2.25 & 2.15 & -42° \\
12 & 33 & M & CM & 35 & 0.050 & + & +30° & - & 2.60 & 2.25 & -3° \\
13 & 61 & M & AH & 42 & 0.050 & - & +60° & + & 3.27 & 2.27 & -30° \\
14 & 62 & M & AS & 36 & 0.040 & + & +60° & + & 2.00 & 1.50 & -2° \\
15 & 62 & M & AI + AS & 35 & 0.040 & + & -20° & - & 2.07 & 2.05 & -1° \\
16 & 34 & M & AH & 45 & 0.047 & - & +45° & + & 2.73 & 2.37 & -10° \\
17 & 48 & F & AI + IM & 38 & 0.038 & - & -10° & + & 2.12 & 1.96 & -26° \\
18 & 74 & F & AH & 38 & 0.050 & + & +10° & + & 2.75 & 2.05 & -36° \\
\hline
\end{tabular}
\end{table}

Abbreviations: AI = aortic insufficiency; AS = aortic stenosis; MI = mitral insufficiency; CM = cardiomyopathy; AH = arterial hypertension; SLI = Sokolow-Lyon index; ID = intrasecond 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; MFV = voltage of maximum frontal vector; MFVd = direction of MFV; MHVt = time of inscription of MHV; QRSl = duration of QRSl; MIVd = direction of maximum horizontal initial vector; MTVd = direction of maximum horizontal terminal vector; MT = angle between MHV and MTV; MPV = voltage of maximum posterior vector; DA > PA = distal QRSr area > proximal QRSr area; STH = direction of horizontal ST vector; MTHV = direction of maximum horizontal T vector; + = present; - = absent; ind = indeterminate.

\begin{table}
\centering
\caption{Electrocardiographic Criteria for Left Ventricular Hypertrophy (LVH)}
\begin{tabular}{l}
1) Sokolow-Lyon index (SV_{1} + RV_{1} - V_{6}) \geq 35 \text{mm}.
2) QRSl duration \geq 0.09 second (max 0.11–0.12 second).
3) Onset of the intrasecond 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) QRSl axis between +30° and -30°.
6) Left atrial enlargement.

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.
\end{tabular}
\end{table}

\begin{table}
\centering
\caption{Vectorcardiographic Criteria for Left Ventricular Hypertrophy (LVH)}
\begin{tabular}{l}
1) Maximal spatial vector \geq 2.10 \text{mV}.
2) Maximal horizontal vector \geq 2.0 \text{mV} (1.90 \text{mV over 40 years}).
3) Maximal frontal vector \geq 2.40 \text{mV} (2.00 \text{mV over 40 years}).
4) Time of inscription of the maximum QRSr vector \geq 47.5 \text{msec}.
5) Duration of the QRSl loop \geq 90 \text{msec} < 120 \text{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 QRSr vector and terminal deflection < 35°.
9) Maximum leftward posterior horizontal force > 1.20 \text{mV}.
10) Distal QRSr loop area larger than the proximal one.
11) Direction of the ST vector to the right of 120°.
12) Direction of the maximum T 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.
\end{tabular}
\end{table}
found together with an intermittent, classic RBBB.

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 1. (Continued)

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<th>MTVd</th>
<th>MT</th>
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<th>MTHV</th>
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</table>

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

Voltage: 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).

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

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<th>Case</th>
<th>First</th>
<th>Second</th>
<th>Third</th>
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<tbody>
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<td>1</td>
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<td>RBBB</td>
<td>LBBB</td>
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</tr>
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<td>LSFB</td>
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</tr>
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<td>AD</td>
<td>RBBB</td>
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<td>LSFB</td>
<td>AD</td>
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<td>6</td>
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<td>9</td>
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<tr>
<td>10</td>
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<td>LBBB</td>
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<tr>
<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 V, 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).
VENTRICULAR CONDUCTION IN LVH/Piccolo et al.

B

HBE

A_1 A_2 710
A_1 A_2 390
H_1 H_2 420
H_2 V_2 50
QRS_{ind} 84

M.S.V. 3.15 mV
M.H.V. 3.05 mV

A_1 A_1 705
A_1 A_2 355
H_1 H_2 400
H_2 V_2 50
QRS_{ind} 80

M.S.V. 2.90 mV
M.H.V. 2.80 mV

C

HBE

A_1 A_1 715
A_1 A_2 325
H_1 H_2 395
H_2 V_2 50
QRS_{ind} 80

M.S.V. 2.34 mV
M.H.V. 2.10 mV

A_1 A_2 725
A_1 A_2 340
H_1 H_2 395
H_2 V_2 50
QRS_{ind} 82

M.S.V. 2.15 mV
M.H.V. 1.85 mV
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

<table>
<thead>
<tr>
<th>Case</th>
<th>MHV</th>
<th>MHVd</th>
<th>QRS</th>
<th>MHVt</th>
<th>HV</th>
<th>Induced aberrance</th>
<th>MHV</th>
<th>MHVd</th>
<th>QRS</th>
<th>MHVt</th>
<th>HV</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>3.25</td>
<td>-30°</td>
<td>110</td>
<td>56</td>
<td>40</td>
<td>RBBB + 5°</td>
<td>96</td>
<td>44</td>
<td>50</td>
<td></td>
<td></td>
</tr>
<tr>
<td>2</td>
<td>2.40</td>
<td>-8°</td>
<td>92</td>
<td>50</td>
<td>40</td>
<td>RBBB + 9°</td>
<td>82</td>
<td>44</td>
<td>50</td>
<td></td>
<td></td>
</tr>
<tr>
<td>3</td>
<td>2.25</td>
<td>-23°</td>
<td>114</td>
<td>56</td>
<td>35</td>
<td>RBBB + 3°</td>
<td>100</td>
<td>52</td>
<td>45</td>
<td></td>
<td></td>
</tr>
<tr>
<td>4</td>
<td>3.75</td>
<td>-47°</td>
<td>86</td>
<td>50</td>
<td>50</td>
<td>RBBB +10°</td>
<td>76</td>
<td>40</td>
<td>50</td>
<td></td>
<td></td>
</tr>
<tr>
<td>5</td>
<td>2.50</td>
<td>-8°</td>
<td>112</td>
<td>52</td>
<td>45</td>
<td>AD - 2°</td>
<td>96</td>
<td>46</td>
<td>45</td>
<td></td>
<td></td>
</tr>
<tr>
<td>6</td>
<td>2.45</td>
<td>0°</td>
<td>96</td>
<td>46</td>
<td>40</td>
<td>AD + 2°</td>
<td>84</td>
<td>36</td>
<td>40</td>
<td></td>
<td></td>
</tr>
<tr>
<td>7</td>
<td>1.80</td>
<td>-2°</td>
<td>90</td>
<td>44</td>
<td>40</td>
<td>AD +12°</td>
<td>80</td>
<td>40</td>
<td>40</td>
<td></td>
<td></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.
FIGURE 3. Recordings from 24-year-old male with normal basal ECG and VCG (left panel and first beat of center panel). The second beat of center panel and the VCG of right panel were simultaneously registered during the first ventricular aberrance induced by premature right atrial stimulation (PRAS) at a coupling interval (A₁-A₂) of 550 msec. Note the slight increase of the initial QRS vectors and the leftward shifting of the terminal QRS vectors without significant changes of the maximum QRS vectors. B) Same patient as in figure 1A. At increased coupling intervals (A₁-A₂ = 520 msec and 530 msec), a progressive reduction of the initial QRS vectors can be seen, while the maximum QRS spatial and horizontal vectors increased and the terminal QRS vectors presented a more leftward orientation. C) Same patient as in figures 1A and 1B. At further increased coupling intervals (A₁-A₂ 530 msec and 510 msec), more advanced QRS changes such as incomplete left bundle branch block can be seen. Abbreviations as in figure 1.

Important 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, and electrophysiologic 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 aberrations 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.18 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.

Acknowledgments

The authors express their sincere appreciation to A. C. Pessina, M.D., and M. Villani, M.D., for their critical review of the manuscript, and to Mrs. Loretta Di Natale for her secretarial assistance.

References

13. Postell WN, Rainey RL, Witham AC, Edmonds JH Jr: Vectorcardiographic and electrocardiographic manifestations of

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TABLE 7. Voltage, Direction, and Inscription Time of the Maximum Initial Vector in the Horizontal Plane Before and After the Ventricular Aberrances Induced by Premature Right Atrial Stimulation in Left Ventricular Hypertrophy Cases.

<table>
<thead>
<tr>
<th>Case</th>
<th>LVO</th>
<th>VOLT</th>
<th>DIR</th>
<th>IT</th>
<th>Induced aberrance</th>
<th>VOL</th>
<th>DIR</th>
<th>IT</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>MO</td>
<td>0.18</td>
<td>+130°</td>
<td>16</td>
<td>RBBB 0.20</td>
<td>+126°</td>
<td>12</td>
<td></td>
</tr>
<tr>
<td>2</td>
<td>DO</td>
<td>0.19</td>
<td>+105°</td>
<td>12</td>
<td>LBBB 0.21</td>
<td>+107°</td>
<td>14</td>
<td></td>
</tr>
<tr>
<td>3</td>
<td>SO</td>
<td>0.28</td>
<td>+113°</td>
<td>18</td>
<td>LBBB 0.22</td>
<td>+10°</td>
<td>14</td>
<td></td>
</tr>
<tr>
<td>4</td>
<td>DO</td>
<td>0.40</td>
<td>+80°</td>
<td>16</td>
<td>LSFB ? 0.26</td>
<td>+81°</td>
<td>18</td>
<td></td>
</tr>
<tr>
<td>5</td>
<td>SO</td>
<td>0.11</td>
<td>+115°</td>
<td>12</td>
<td>LBBB 0.24</td>
<td>+80°</td>
<td>18</td>
<td></td>
</tr>
<tr>
<td>6</td>
<td>SO</td>
<td>0.14</td>
<td>+170°</td>
<td>14</td>
<td>RBBB 0.15</td>
<td>+81°</td>
<td>8</td>
<td></td>
</tr>
<tr>
<td>7</td>
<td>DO</td>
<td>0.22</td>
<td>+72°</td>
<td>16</td>
<td>LSFB ? 0.12</td>
<td>+111°</td>
<td>12</td>
<td></td>
</tr>
<tr>
<td>8</td>
<td>DO</td>
<td>0.22</td>
<td>+135°</td>
<td>20</td>
<td>LSFB ? 0.12</td>
<td>+45°</td>
<td>10</td>
<td></td>
</tr>
<tr>
<td>9</td>
<td>MO</td>
<td>0.27</td>
<td>+72°</td>
<td>24</td>
<td>LBBB 0.28</td>
<td>+60°</td>
<td>24</td>
<td></td>
</tr>
<tr>
<td>10</td>
<td>DO</td>
<td>ind</td>
<td>ind</td>
<td>ind</td>
<td>LBBB 0.12</td>
<td>+20°</td>
<td>13</td>
<td></td>
</tr>
<tr>
<td>11</td>
<td>UO</td>
<td>0.15</td>
<td>+118°</td>
<td>14</td>
<td>RBBB 0.22</td>
<td>+121°</td>
<td>14</td>
<td></td>
</tr>
<tr>
<td>12</td>
<td>UO</td>
<td>0.29</td>
<td>+113°</td>
<td>24</td>
<td>LBBB 0.28</td>
<td>+77°</td>
<td>18</td>
<td></td>
</tr>
<tr>
<td>13</td>
<td>SO</td>
<td>0.12</td>
<td>+110°</td>
<td>16</td>
<td>LSFB ? ind</td>
<td>ind</td>
<td>ind</td>
<td></td>
</tr>
<tr>
<td>14</td>
<td>SO</td>
<td>0.09</td>
<td>+157°</td>
<td>12</td>
<td>LCCh 0.09</td>
<td>+157°</td>
<td>12</td>
<td></td>
</tr>
<tr>
<td>15</td>
<td>MO</td>
<td>0.18</td>
<td>+168°</td>
<td>16</td>
<td>LAFB 0.11</td>
<td>+160°</td>
<td>12</td>
<td></td>
</tr>
<tr>
<td>16</td>
<td>SO</td>
<td>0.19</td>
<td>+125°</td>
<td>16</td>
<td>LSFB 0.22</td>
<td>+61°</td>
<td>12</td>
<td></td>
</tr>
<tr>
<td>17</td>
<td>DO</td>
<td>0.12</td>
<td>+25°</td>
<td>10</td>
<td>NCCh 0.12</td>
<td>+25°</td>
<td>10</td>
<td></td>
</tr>
<tr>
<td>18</td>
<td>SO</td>
<td>0.09</td>
<td>+50°</td>
<td>10</td>
<td>NCCh 0.09</td>
<td>+50°</td>
<td>10</td>
<td></td>
</tr>
</tbody>
</table>

Abbreviations: PRAS = premature right atrial stimulation; LVO = left ventricular overloading; VOLT = voltage; DIR = direction; IT = inscription time; DO = diastolic overloading; SO = systolic overloading; MO = mixed overloading; UO = undefined overloading; ind = indeterminable; RBBB = right bundle branch block; LBBB = left bundle branch block; LSFB = left septal fascicular block; LSFB = left anterior fascicular block; LCCh = non-characteristic change; LSFB = left posterior fascicular block.
creasing left ventricular pressure overload. Am Heart J 77: 33, 1969
14. Rombilt DW, Estes EH Jr: A point-score system for the ECG
diagnosis of left ventricular hypertrophy. Am Heart J 75: 752, 1968
16. Upham CB: Simplified clinically applicable vectorcardio-
graphic diagnosis of left ventricular hypertrophy (Frank lead
system). Am Heart J 74: 749, 1967
17. Varriale P, Alfenito JC, Kennedy RJ: The vectorcardiogram of
left ventricular hypertrophy. Analysis and criteria (Frank lead
system). Circulation 33: 569, 1966
18. Wallace AG, McCall BW, Estes EH Jr: The vectorcardiogram
in left ventricular hypertrophy. A study using the Frank lead
system. Am Heart J 63: 466, 1962
Chicago, Year Book Medical Publishers, 1975, pp 38, 776-792
20. Bryant JM: Intraventricular conduction. In Advances in Elec-
trocardiography. New York, Grune & Stratton, 1958, p 117
of aberrant ventricular conduction in man: evidence of isolated
and combined block within the specialized conduc-
tion system. An electrocardiographic and vectorcardiographic
22. Estes EH Jr: Left ventricular hypertrophy in acquired heart dis-
ease: a comparison of the vectorcardiogram in aortic stenosis
and aortic insufficiency. In Vectorcardiography, edited by
Hoffman I. Amsterdam, North-Holland Publishing Co, 1968, p
157
23. Master AM: The relationship between bundle branch block and
cardiac enlargement. Am Heart J 20: 186, 1940
Oldsmar, Florida, Tampa Tracings, 1970, pp 71-96
25. Scherlag BJ, Lau SH, Helfant RH, Berkowitz WD, Stein E,
Damato AN: Catheter technique for recording His bundle ac-
26. Frank E: An accurate, clinically practical system for spatial
vectorcardiography. Circulation 12: 737, 1956
27. Wu D, Denes P, Rosen KM: Refractoriness of atrioventricular
conduction. In His Bundle Electrocardiography and Clinical
Electrophysiology, edited by Narula OS. Philadelphia, PA
Davis, 1975, p 95
28. Kulbertus HE, deLeval-Rutten F, Casters P: Vector-
cardiographic study of aberrant conduction. Anterior displace-
ment of QRS: another form of intraventricular block. Br Heart
J 38: 549, 1976
B: Evaluación de la conducción infravalvular a través de la elec-
rostimulación auricular asociada a la vectorcardiografía. In
Diagnostico y Tratamiento de las Arritimas Cardiacas, edited
by Bayes A, Cosin J. Barcelona, Editorial DOYMA, 1978, p
374
30. Hoffman I, Mahta L, Hilsenrath J, Hamby RI: Anterior con-
duction delay: a possible cause for prominent anterior QRS
forces. J Electrocardiol 9: 15, 1976
31. Nakaya Y, Hisa Y, Murayama Y, Ueda S, Nagao T, Niki T,
Mori H, Takashima Y: Prominent anterior QRS force as a
manifestation of left septal fascicular block. J Electrocardiol
11: 39, 1978
32. Carter WA, Estes EH Jr: Electrocardiographic manifestations
of ventricular hypertrophy; a computer study of ECG-anatomic
correlations in 319 cases. Am Heart J 68: 173, 1964
33. Talbot S, Kilpatrick D, Jonathan A, Raphael MJ: QRS voltage
of the electrocardiogram and Frank vectorcardiogram in rela-
tion to ventricular volume. Br Heart J 39: 1109, 1977
34. Reeve R, Kawamata K, Selzer A: Reliability of vector-
cardiography in assessing the severity of congenital aortic
stenosis. Circulation 34: 92, 1966
35. Soloff LA, Lawrence JW: The electrocardiographic findings
in left ventricular hypertrophy and dilatation. Circulation 26: 553,
1962
36. Toshiba H, Cueto J, Lillehei CW: Vectorcardiographic studies
in acquired valvular disease with reference to the diagnosis of
left ventricular hypertrophy. Circulation 35: 132, 1967
37. Witham AC: Current status of correlations between vector-
cardiogram and hemodynamic data. In Advances in Electro-
cardiography, edited by Schlant RC, Hurst JW. New York,
Grune & Stratton, 1972, p 309
38. Brackbill TA, Shah PM: Vectorcardiographic comparison of
left ventricular hypertrophy in idiopathic hypertrophic sub-
armonic stenosis, aortic stenosis and aortic regurgitation. Am Heart
J 88: 269, 1974
39. Hugenholtz PG, Gamboa R: Effect of chronically increased
ventricular pressure on electrical forces of the heart. A correla-
tion between hemodynamic and vectorcardiographic data
(Frank system) in 90 patients with aortic or pulmonic stenosis.
Circulation 30: 511, 1964
40. Kulbertus HE, Demoulin JC: Pathological basis of concept of
left hemiblock. In The Conduction System of the Heart, edited
by Wellens HJJ, Lie KJ, Janse MJ. Leiden, HE Stenfert Kroese
B.V., 1976, p 287
bifascicular block with normally conducting middle fascicle. J
Electrocardiol 10: 401, 1977
42. Medrano GA, Brenes PC, De Micheli A, Sodi Pallares D: El
bloqueo simultaneo de las subdivisiones anterior y posterior de
la rama izquierda del haz de His (bloqueo bifascicular) y su
asociacion con bloqueo de la rama derecha (bloqueo
trifascicular). Arch Inst Cardiol Mex 40: 752, 1970
The role of left ventricular conduction in the electrogenesis of left ventricular hypertrophy. An electrophysiologic study in man.
E Piccolo, A Raviele, P Delise, F Dainese, P Pascotto, G Totaro, F Sartori and D D'Este

Circulation. 1979;59:1044-1055
doi: 10.1161/01.CIR.59.5.1044

Circulation is published by the American Heart Association, 7272 Greenville Avenue, Dallas, TX 75231
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Print ISSN: 0009-7322. Online ISSN: 1524-4539

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