Labile Variations of Intraventricular Conduction Unrelated to Rate Changes

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SUMMARY
An example of unstable intraventricular conduction is presented. Several unusual features of this type of conduction are described. These are: (1) variable intraventricular conduction which was sensitive to multiple stimuli but not clearly related to changes in heart rate, (2) mid and terminal QRS changes reflected only in the left lateral chest leads and in the mid and terminal QRS portions of the vectorcardiogram, (3) constant association of prolonged P-R interval suggesting that this is a variety of bilateral bundle-branch block, and (4) apparent persistence of the peculiar intraventricular conduction for at least 4 years. Multiple involvement of the conduction system is probably present, and functional variations in distal portions of the left ventricular conduction system account for these labile changes. Predictable QRS patterns can be produced by various stimuli which alter intraventricular conduction.

Additional Indexing Words:
- Bilateral bundle-branch block
- Left bundle-branch conduction
- Intraventricular conduction disturbances
- Computer electrocardiogram

Although electrocardiographic patterns associated with disturbances of the main bundle branches are well known, a large number of tracings show intraventricular block within the left ventricle where the location or locations of faulty conduction are uncertain. These include a wide variety of changes in the QRS complexes usually with a duration of greater than 0.10 sec, notching, and slurring, and often abnormally directed terminal forces. Intermittent changes of intraventricular conduction are well known, and are usually attended by changes in heart rate.1,2 We wish to report an example of varying intraventricular conduction which is influenced by a wide variety of stimuli and is largely independent of changes of heart rate.

Report of Case
The patient was a 71-year-old male Negro with a clinical diagnosis of primary myocardial disease based on the presence of biventricular failure, mitral regurgitation, prominent atrial and ventricular gallops, and a history of chronic alcoholism. Coronary artery disease could not be excluded although there was no history of chest pains or hypertension. He was ambulatory and was being treated with digitalis, a diuretic, and a low sodium diet. There was no history of foreign travel. Complete blood count, cholesterol, electrolytes, blood sugar, and urinalysis were normal. Chest x-rays showed a large globular heart and increased pulmonary vascular markings.

Electrocardiogram
The resting 12-lead electrocardiogram showed nonrespiratory sinus arrhythmia with frequent premature ventricular contractions, first degree A-V block (P-R interval, 0.22 sec), marked left axis deviation (QRS, $-60^\circ$), left ventricular hypertrophy, and a left atrial abnormality. An intraventricular conduction defect was diagnosed on the basis of widened, slurred, QRS complexes (0.11 sec), and Q waves were small or absent in leads I, aV_L, V_5, and V_6. The T waves were abnormal (frontal plane axis $+120^\circ$) (fig. 1A). This pattern was compatible with incomplete

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left bundle-branch block. A second tracing taken shortly afterward showed a change to an RS pattern in leads V5, and V6 (QRS to 0.11 sec) without marked change in the limb leads (fig. 1B).

Methods

All medications were discontinued for 3 weeks prior to further study. Various procedures were performed during ECG monitoring (V6 chest lead), in order to define the factors associated with the two types of intraventricular (IV) conduction. Exercise was performed on a motorized treadmill at 2 mph for 3 min. Several vagal maneuvers, including carotid sinus stimulation, ocular pressure, Müller and Valsalva maneuvers,
and deep breathing were performed at various times. The electrocardiogram was also recorded during pain stimulation, drinking ice water, postural changes, various body movements, eating, sleeping, and deep breathing. Atropine was administered intravenously (total dose, 1.2 mg), amyl nitrate by inhalation, and 100% oxygen, by a face mask for 5 min. Each procedure was repeated several times, allowing sufficient time for equilibrium between studies. All of these tests, except for those with the drugs were repeated on 3 separate days. Constant monitoring was performed for 24 hours in the hospital to detect spontaneous changes in the ECG. Vectorcardiograms and simultaneous orthogonal XYZ leads were recorded (Frank lead system) in recumbent and sitting positions.

Standard 12-lead electrocardiograms were also recorded on ¼-inch magnetic tape, with the patient in the recumbent and sitting positions, by means of a Dat-EK data acquisition console. Each lead was recorded sequentially for 3.72 sec. This data-acquisition console accepts ECG voltages from a patient and properly codes and conditions these signals to be acceptable for computer analysis. The patient identification code and electrocardiographic voltages are presented on a conventional strip chart record and stored similarly on magnetic tape.

The magnetic tape containing our patients' electrocardiograms was digitized at 500 samples/sec by a special analog to digital converter and then processed on a 160-A digital computer using the system developed by Caceres and associates. Changes in mid and terminal QRS forces were quantitated by this method.

**Results**

The results of these maneuvers and observations are summarized in table 1. The patient remained asymptomatic throughout the period of observation and study, and his temperament remained placid.

Two basic patterns of IV conduction were seen throughout the study. One was a pattern of small or absent Q wave in V₆, tall R wave, or tall R and tiny s, QRS duration of 0.11 to 0.12 sec, and terminal slowing (pattern A). The other pattern consisted of an RS complex, absence of the Q wave, and QRS duration of 0.11 sec (pattern B). Several intermediate forms were also seen: Rs and rS. Recumbency was usually associated with pattern A, and pattern B appeared with an upright position. These changes were less apparent in the orthogonal XYZ leads. They appeared as a slight change in shape of the R wave in the X lead and as an alteration in the synchrony of the maximal deflections in leads X and Y (fig. 2). Throughout exercise testing the RS conduction pattern persisted (fig. 3). The patient was unable to walk more than 3 min at 2 MPH. Various body movements were associated with an alteration of ventricular conduction (fig. 4). Protrusion of the tongue consistently diminished.
or abolished the S wave (fig. 5). This particular response could be produced five or six times in succession and then would require a rest period of approximately 1 min for recovery. If the test was repeated at intervals of 30 sec, the response could be produced many times. Carotid sinus pressure produced marked vagal effects but was not associated with consistent changes in ventricular conduction. Effects of posture were characteristic: lying down produced the R pattern (A), and sitting or standing was associated with an RS pattern (B). The Valsalva and the Müller maneuvers produced the A pattern during straining. Frequently, alteration in QRS pattern followed a premature ventricular contraction. Sometimes, however, ventricular conduction changed spontaneously. There was no apparent effect from change of heart rate, ingestion of food, ice water, sleep, or pain. Changes with deep breathing were slight, without change of conduction from the A to a B type. Amyl nitrate increased heart rate but did not alter IV conduction. Oxygen administration similarly had no apparent effect on ventricular conduction.

Changes from one pattern to another were associated with only slight changes in the initial parts of the QRS complex. This was seen as development of a small Q wave in V5, and in V6 during the A pattern and reduction or disappearance of the Q wave during B pattern. The major change in QRS pattern occurred as terminal slowing and slight QRS prolongation (0.01 sec) in changing from the B to the A configuration.

Slight variability in heart rate occurred after some procedures. The direction of change was inconsistent except for carotid sinus pressure (slowing of heart rate). Furthermore, as much variability in heart rate was seen without any intervention and with-

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**Table 1**

**Observations and Interventions: QRS Patterns and Heart Rates**

<table>
<thead>
<tr>
<th></th>
<th>Initial response</th>
<th>Response</th>
<th>Heart rate</th>
<th>QRS pattern</th>
<th>Heart rate</th>
<th>QRS pattern</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Heart rate</td>
<td>QRS pattern</td>
<td></td>
<td></td>
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<td></td>
</tr>
<tr>
<td>Exercise (treadmill)</td>
<td>80 RS</td>
<td>107 rS</td>
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<tr>
<td>Valsalva: recumbent sitting</td>
<td>67 qRs</td>
<td>62 qR</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Ocular pressure: recumbent sitting</td>
<td>64 Rs</td>
<td>60 qR</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Müller: recumbent sitting</td>
<td>64 Rs</td>
<td>64 qR</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Posture: recumbent to sitting sitting to recumbent</td>
<td>67 R</td>
<td>65 RS</td>
<td>68 RS</td>
<td>68 qR</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Carotid sinus stimulation: recumbent</td>
<td>55 Rs</td>
<td>46 qR</td>
<td></td>
<td></td>
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<td></td>
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<tr>
<td>Shoulder movement: recumbent sitting</td>
<td>67 qR</td>
<td>67 Rs</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Shoulder movement: sitting</td>
<td>68 RS</td>
<td>69 R</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Amyl nitrate (inhalation): sitting</td>
<td>62 qRs</td>
<td>80 Rs</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Ice water: sitting</td>
<td>64 RS</td>
<td>64 RS (no change)</td>
<td></td>
<td></td>
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<td></td>
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<tr>
<td>Atropine (1.2 mg): recumbent</td>
<td>70 Rs</td>
<td>86 Rs (no change)</td>
<td></td>
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<td></td>
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<tr>
<td>100% O2: sitting</td>
<td>70 RS</td>
<td>70 RS (no change)</td>
<td></td>
<td></td>
<td></td>
<td></td>
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<tr>
<td>Tongue protrusion: recumbent sitting</td>
<td>68 Rs</td>
<td>67 qR</td>
<td></td>
<td></td>
<td></td>
<td></td>
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<tr>
<td>Shaking hands: sitting</td>
<td>76 Rs</td>
<td>76 qRs</td>
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</table>
out alteration of QRS pattern. Changes in blood pressure following procedures displayed a similarly inconsistent pattern.

A review of previous electrocardiograms revealed that similar changes in IV conduction were occurring in 1963 when two ECGs were taken fortuitously on successive days which showed the two patterns described.

Vectorcardiograms were recorded to illustrate the two types of ventricular depolarization (fig. 6). The sagittal plane loop showed little change while the frontal and horizontal planes showed changes in the loops with changes in position. The mid and terminal forces were directed more superiorly and posteriorly with sitting. The major changes in conduction seen in both standard ECGs and the VCGs were in the mid and late forces in the horizontal plane.

Results of the computer-processed electrocardiograms are shown in table 2. Specifically, the onset, end, location, and value of minimum slope (first derivative) of each QRS complex was identified for each lead. The parameters, QRS duration (sec), Q-wave amplitude (mv), Q-wave duration (sec), and “intrinsicoid interval” (time from QRS onset to minimum slope) were calculated for each QRS and then rounded to the nearest 0.01. A mean value was determined for each of the various QRS parameters from a specific lead. The lateral precordial leads (V5 and
V₆ showed pattern A in the recumbent position and pattern B in the sitting. A tiny q was measured in the recumbent position while none was found in the sitting position. The differences in the intrinsicoid interval represent the two types of conduction (A and B).

Discussion

The mechanisms of these changing patterns of depolarization are not known. They persisted after the abolition of vagal influences. They are not explained by anatomic shifting of the heart position because deep inspiration with descent of the diaphragm was accompanied by a QRS pattern that was unlike that associated with an erect posture. QRS changes invoked by such minimal stimuli as motion of the shoulders or protrusion of the tongue are completely unexplained but are suggestive of a neurological trigger mechanism. Although the underlying anatomic lesions in this case remain unknown, the presence of the same ECG abnormalities for at least 4 years suggests chronic pathological changes such as areas of fibrosis. Left ventricular hypertrophy is also undoubtedly present, and this may contribute to the conduction delay.

Varying intraventricular conduction, especially intermittent or transient bundle-branch block, is not an uncommon occurrence. However, most of these reported cases have been associated with either slowing or increasing the heart rate. Wallace and Laszlo\textsuperscript{11} reported a case of intermittent

Figure 4

Lead V₆. In A, B, and C, slight motions of the shoulders (irregularities of base line) are followed by the appearance of small s waves for a few beats.
bundle-branch block not primarily influenced by heart rate. They stated that "hemodynamic, neural, and nutritional factors" might play a significant role in determining the mode of IV conduction. Predictable QRS patterns in our patient resulted from postural change, body movement, and Valsalva and Müller maneuvers. Also premature beats were occasionally followed by a change from one QRS shape to another. A functional disturbance of the sympathetic nervous system can be suggested by the limited increase in heart rate after the stimulation of exercise, atropine, and amyl nitrate.

The combination of a prolonged P-R interval and unilateral bundle-branch block suggests the possibility of bilateral bundle-branch block. Lepeschkin reviewed the ECG criteria for bilateral bundle-branch block, and stated that the concept of first, second, and third degree A-V block can be applied to lesions affecting bundle branches. Our patient may have first degree block of the right bundle, and second degree or more

Table 2
Results of Computer-Processed Electrocardiograms

<table>
<thead>
<tr>
<th>Lead and position</th>
<th>QRS duration (sec)</th>
<th>Q wave duration (sec)</th>
<th>Q wave amp (mv)</th>
<th>Interval from QRS onset to intrinsicoid deflection</th>
<th>Absolute value of minimum derivative</th>
</tr>
</thead>
<tbody>
<tr>
<td>I Lying</td>
<td>0.13</td>
<td>0.01</td>
<td>0.03</td>
<td>NA*</td>
<td>122</td>
</tr>
<tr>
<td>I Sitting</td>
<td>0.13</td>
<td>0.01</td>
<td>0.03</td>
<td>NA*</td>
<td>133</td>
</tr>
<tr>
<td>V6 Lying</td>
<td>0.12</td>
<td>0.01</td>
<td>0.03</td>
<td>0.07</td>
<td>263</td>
</tr>
<tr>
<td>V6 Sitting</td>
<td>0.12</td>
<td>0</td>
<td>0</td>
<td>0.05</td>
<td>302</td>
</tr>
<tr>
<td>V6 Lying</td>
<td>0.12</td>
<td>0.01</td>
<td>0.03</td>
<td>0.07</td>
<td>88</td>
</tr>
<tr>
<td>V6 Sitting</td>
<td>0.12</td>
<td>0</td>
<td>0</td>
<td>0.05</td>
<td>250</td>
</tr>
</tbody>
</table>

*NA = not applicable.
INTRAVENTRICULAR CONDUCTION

Figure 6

Vectorcardiograms. (1/2 standardization-dash intervals, 2.5 msec.) Upper loops recorded with patient recumbent; lower loops, sitting. The left sagittal loop, inscribed counterclockwise, shows no significant change with position. The frontal and horizontal loops demonstrate decreased ratio of length to width and a more superior and more posterior afferent limb with the sitting position.

extensive (unequal) first degree left-bundle involvement. It could be that partial block of the right bundle would produce prolonged P-R while involvement in branches of the left bundle would produce the labile IV conduction patterns with QRS prolongation. A definite diagnosis of bilateral bundle-branch block, however, can be made only if both right and left bundle-branch block patterns appear in the same patient accompanied by consistent change of the P-R interval.

Although a single lesion of the right bundle may produce right bundle-branch block, this is not as likely in the left bundle because of early and multiple branchings into the anterior and posterior divisions and widespread ramifications throughout the left ventricle. In our patient most of the labile conduction changes were seen in the mid and terminal QRS forces in the frontal and horizontal planes and in leads V5 and V6 of the electrocardiogram. This in conjunction with slight QRS prolongation, increased P-R intervals, and persistent left axis deviation indicates multiple defects in the ventricular conduction system, proximal as well as peripheral. The proximal conduction defect(s) account for the more constant conduction abnormalities (increased P-R, left axis deviation, QRS prolongation), while left intraventricular conduction block (or delay) produces the labile QRS changes which are sometimes called "arborization blocks," "intraventricular blocks," "focal blocks," or "parietal blocks." Regardless of the terminology, intraventricular conduction is not necessarily a fixed situation, and blocks or delays in the distal branches of the left bundle can explain these variable...
changes. Also, because of the multiple branchings of these distal ramifications, a wide variety of ECG configurations can be expected.

Because the surface electrocardiogram records only general information concerning the voltage time course of individual fibers, conduction velocity and sequence of excitation in different areas of the heart, a precise explanation for these unusual intraventricular conduction patterns is not possible. It is possible that marginally functional peripheral conduction fibers might be further compromised by local circulatory or other changes producing these varying patterns.

References
