The Electrocardiogram in Muscular Subaortic Stenosis

Effect of a Left Septal Incision and Right Bundle-Branch Block

By E. Douglas Wigle, M.D., and Robert H. Baron, M.D.

Following Brock's recognition that hypertrophied muscle in the left ventricular outflow tract could obstruct left ventricular outflow, much interest has centered on the nature of this hypertrophy. Initial reports suggested that concentric hypertrophy of the left ventricle was the underlying cause of the obstruction of the outflow tract. Following Teare's description of asymmetrical hypertrophy of the heart (hypertrophy centered in the ventricular septum often extending into the anterior wall of the left ventricle), considerable evidence has been accumulated to indicate that asymmetrical, rather than concentric hypertrophy of the left ventricle, is the more common cause for muscular subaortic stenosis.

More recently, a number of observers have noted abnormal Q waves in the electrocardiograms in some cases of muscular subaortic stenosis, and some have suggested that these Q waves may indicate ventricular septal hypertrophy. Tall R waves in the right precordial leads have also been recorded in cases of muscular subaortic stenosis in which there was no cause for, or evidence of, significant right ventricular hypertrophy. In a previous communication, we suggested that both the Q waves in the left precordial and limb leads and the tall R waves in the right precordial leads could be reasonably attributed to ventricular septal hypertrophy. Evidence was provided that both Q-wave and R-wave abnormalities diminished or disappeared as electrocardiographic evidence of left ventricular free-wall hypertrophy emerged. It was suggested that septal hypertrophy caused the muscular subaortic stenosis, which in turn resulted in hypertrophy of the free wall of the left ventricle.

The purpose of the present report is to detail the changes that occurred in these electrocardiographic abnormalities following surgical incision of the left side of the ventricular septum (ventriculotomy), and following spontaneous development of right bundle-branch block.

Activation of the Ventricular Septum

Prior to consideration of the effect of ventricular conduction defects on these electrocardiographic abnormalities in muscular subaortic stenosis, certain pertinent anatomic and physiological considerations regarding the cardiac conduction system and activation of the ventricular septum should be considered. Lev has demonstrated that the bundle of His emerges on the left ventricular side of the septum beneath the noncoronary cusp of the aortic valve (fig. 1). The posterior division(s) of the left bundle branch are given off almost immediately, and the remaining anterior division(s) of the left bundle branch and the right bundle branch divide at or before the commissure between the noncoronary and right coronary cusps of the aortic valve. Thus, only the anterior division(s) of the left bundle branch lies below the right coronary cusp or below the commissure between
the right and left coronary cusps of the aortic valve (fig. 1). The incision of ventriculomyotomy was made in line with the commissure between these cusps, or below the right coronary cusp, both sites being situated at the anterior end of the bulbar part of the ventricular septum.

Previously we reported that when this incision was in this position, the postoperative electrocardiogram revealed left axis deviation due to a left ventricular parietal block or anterolateral peri-infarction block. This postoperative electrocardiographic change was compatible with severance of the anterior division(s) of the left bundle branch. In one case, in which the center of the septum was incised, complete left bundle-branch block developed without deviation of the QRS axis.

It is generally agreed that activation of the mid-left septal surface dominates the initial phase of septal depolarization. This may result from this area’s being activated earlier than the right septal surface or from the spread of excitation being more rapid over the left septal surface than over the right. The authors are unaware of any studies that clearly demonstrate whether the mid-left septal area is predominantly activated by conduction fibers arising from the anterior or posterior division(s) of the left bundle branch or possibly by branches of this bundle arising between these major divisions.

Because the septum lies relatively parallel to the frontal plane of the body with its apex tilted anteriorly, the left septal surface lies posterior and inferior, as well as to the left of the right septal surface. Activation of the left septal surface would thus result in a vector force being directed anteriorly and superiorly to the right, and the superior component being especially prominent with the overall force moving in the apex to base direction. This force, in the presence of hypertrophy of the ventricular septum, could result in pathological Q waves in the left precordial and limb leads (except aVR) and tall R waves in the right precordial leads.

**Methods**

Four patients with muscular subaortic stenosis were observed and form the basis of this report (table 1). The diagnosis for each patient was established by clinical, hemodynamic, and angiographic means. Patients 1, 2, and 3 bear the same case numbers as in a previous report. These three patients underwent ventriculomyotomy for relief of the subaortic stenosis by Dr. W. G. Bigelow; the incision was made in line with the commissure between the right and left coronary cusps. The incision was deepened manually until the cut edges of the incision were felt to retract. No muscle was removed, other than one or two pieces less than 5 mm in maximum dimension for biopsy. Postoperative heart catheterization has demonstrated complete relief of the stenosis in these three patients. Patient 4 has been followed for 6 years, during which time first a left, and subsequently a right, ventricular conduction defect developed spontaneously. There was a 10-mm Hg systolic pressure gradient between the inflow and outflow tracts of the right ventricle both before and after development of complete right bundle-branch block.

Standard 12-lead electrocardiograms recorded on a Sanborn direct writing recorder were made before and after surgery in patients 1, 2,
and 3 and serially over 6 years in patient 4. Left and right ventricular angiograms revealed evidence of ventricular septal enlargement in the four cases. In cases 1, 2, and 3 this was confirmed at surgery and was particularly prominent at the anterior end of the bulb septum.27

Results

The preoperative and postoperative electrocardiograms in cases 1, 2, and 3 are shown in figs. 2 to 4. Preoperatively in each instance there were prominent Q waves in the limb leads (principally leads II, III, and aVF) and in the left precordial leads and tall R waves in the right precordial leads. Initial R waves were also present in aVR. Following ventriculotomy, deep S waves developed in leads II, III, and aVF, indicating left axis deviation, believed due to the surgical incision cutting the anterior division(s) of the left bundle branch. The postoperative conduction defects are identical to either a left ventricular parietal block (case 2) or anterolateral peri-infarction block (cases 1 and 3), depending on the presence or absence of Q waves in aVL (cases 1 and 3).34 There is prolongation of the terminal QRS forces in case 3, but not in patients 1 and 2.

In addition to these left ventricular conduction defects the ventriculotomy incision resulted in the disappearance of the abnormal Q waves in the limb leads and left precordial leads and the R wave in aVR. The right precordial R waves diminished significantly in magnitude, while the left precordial R wave increased in magnitude. Postoperatively, in cases 1 and 2 there was an apparent increase in the degree of left ventricular free-wall hypertrophy (figs. 2 and 3).

The exceptionally tall R waves and inverted T waves in the right precordial leads in case 1 are similar to those reported in several other cases of muscular subaortic stenosis, in which there was no evidence of severe right ventricular hypertrophy.4,15–17,28 The fact that these very tall R waves diminished following the left septal incision tends to confirm that they are to a considerable extent due to a depolarizing force commencing on the left side of the ventricular septum. The exact reason that these R waves were so prominent in this one case is uncertain but could be connected to the anatomic relationship between the septum and the chest wall, or might indicate excessive hypertrophy of the anterior end of the septum, possibly extending into the anterior left ventricular wall.

Serial electrocardiograms in case 4 are shown in figures 5 and 6. In 1959, Q waves were evident in leads II, III, and aVF, and to a lesser extent, in V6 (fig. 5). In 1960, in addition to the Q-wave abnormalities, S waves were evident in leads II, III, and aVF (fig. 6). In 1963, the QRS interval had prolonged to 0.16 seconds (fig. 6). This prolongation was believed due to the spontaneous development of complete right bundle-branch block; the QRS configuration at this time resembled that previously reported to indicate complete right bundle-branch block, plus a lesion in the anterior division of the left bundle branch.35,36 Associated with this right ventricular conduction defect, the Q waves became more prominent and an initial R wave of considerable magnitude became evident in aVR and V1. These latter Q-wave and R-wave changes may reflect increasing dominance of left septal activation associated with decrease in right septal activation secondary to complete right bundle-branch block.

Table 1

Systolic Pressure Difference Between Body and Outflow Tract of Right and Left Ventricle, Respectively

<table>
<thead>
<tr>
<th>Case</th>
<th>Age</th>
<th>Sex</th>
<th>RV Gradient (mm Hg)</th>
<th>LV Gradient (mm Hg)</th>
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<td>M</td>
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</tr>
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<td>M</td>
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<tr>
<td>4</td>
<td>37</td>
<td>F</td>
<td>10</td>
<td>85</td>
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Discussion

The pathological Q waves reported to occur in muscular subaortic stenosis in the limb leads and the left lateral precordial leads are believed to be of considerable significance. Knowledge of their occurrence may be a diagnostic aid in this condition and may prevent an erroneous diagnosis of myocardial infarction.19 There was no evidence of coronary artery disease in the four cases reported on herein, nor in a previous study.19 The age of these patients makes coronary artery disease very unlikely. It is believed that the prominent Q waves in the left precordial and limb leads (except aVR) and the tall R waves in the right precordial leads result from depolarization of an hypertrophied ventricular septum commencing at the left septal surface. The fact that a surgical incision at the anterior end of the left side of the septum diminishes both the right precordial R waves and the left precordial and limb lead Q waves, while
Figure 3

Case 2. The prominent Q waves in leads II, III, aVF, V₃ to V₆, and tall R waves with inverted T waves in leads V₁ and V₂ diminish or disappear following the anterior left septal incision (ventriculomyotomy). In cases 1 and 2, the surgeon felt a particularly prominent muscle bar at the anterior end of the ventricular septum, 1 to 2 cm below the aortic valve.

the development of a right ventricular conduction defect has the opposite effect, tends to support this hypothesis.

In the cases comprising this report, the Q waves in the limb leads have been present principally in leads II, III, and aVF. These deflections, along with the precordial abnormalities, may suggest that in these cases the abnormal hypertrophy is mainly in the anterior end of the septum and in the immediately adjacent anterior left ventricular wall. Surgical observations in cases 1, 2, and 3 suggested that this was so.²⁷

It is tempting to compare the abnormal Q and R waves in these cases with those seen in posterodiaphragmatic myocardial infarction. In the latter instance, because of death of muscle in the posteroinferior aspects of the heart,
the depolarization of normal anteroseptal muscle results in Q waves in leads II, III, and aVF, and occasionally in leads V₅ and V₆, and if strictly posterior damage is present, prominent R waves may be present in the right precordial leads. The genesis of the Q-wave abnormalities in these two grossly different conditions would thus be analogous. In either case, there is a dominance of anterosuperior over posteroinferior electrical forces. In posterodiaphragmatic infarction there is a deficiency of posteroinferiorly directed forces, while in muscular subaortic stenosis there is an excess of anterosuperiorly directed forces.

In some cases of muscular subaortic stenosis, the Q-wave abnormalities may be most marked in leads I and aVL and in the right precordial leads. These abnormal deflections may suggest predominant posteroseptal hypertrophy, and the reason these deflections resemble those seen in anteroseptal infarction may be similar to the reasons suggested for the abnormalities of anteroseptal hypertrophy resembling those seen in posterodiaphragmatic infarction (vide supra). In a number of cases of muscular subaortic stenosis in which Q-wave abnormalities are present, they are maximal in lead II and are also present in leads I and aVF. This pattern may suggest

**Figure 4**

Case 3. Preoperatively Q waves were prominent in leads V₅ and V₆, and also present in leads I, II, III, and aVF. Following ventriculomyotomy (Feb. 27, 1962) the Q waves vanished in these leads and a prominent Q wave appeared in aVL. Postoperatively, the ventricular conduction defect was comparable to anterolateral peri-infarction block with QRS prolongation.
generalized septal hypertrophy. Complicating these generalizations concerning the localization of the myocardial hypertrophy is the frequent occurrence of fibrosis of the septal myocardium. Although there is angiographic, surgical, and postmortem evidence of unusual ventricular septal enlargement in cases of muscular subaortic stenosis in which these Q-wave abnormalities occur, two factors other than septal hypertrophy per se must be considered in the genesis of the abnormal Q waves. First, Burchell has commented on the fact that the electrocardiograms in some cases of defects of the muscular ventricular septum, when the septal musculature has a "Swiss cheese" appearance, may have similar Q-wave abnormalities. Thus, it is considered possible that the bizarre nature of the myocardial fiber hypertrophy in muscular subaortic stenosis may play a role in the genesis of the abnormal Q waves. Whether or not the excessive catecholamine deposits in these fibers could affect the electrocardiogram is unknown. Secondly, Snellen reported that he and Roos have demonstrated that in some cases of muscular subaortic stenosis there is premature activation of basal paraseptal myocardium. This observation may be related to the findings of Lunel (working with Snellen and Roos) of an anomalous conducting bundle arising from the left bundle branch (personal communication). If the base of the septum were also prematurely activated, this factor might result in, or contribute to, the abnormal Q waves.

The exact reason for the postoperative disappearance of the Q waves in these patients is not known but could be related to the cutting of the anterior division(s) of the left bundle branch, to cutting of other septal branches, or an anomalous bundle, from the left bundle branch, or to cutting and spreading of the muscle itself. Similarly, the reason for the postoperative appearance of Q waves in lead aVL (fig. 4) or in leads I and aV₅ is uncertain, but could be due to anterior septal damage or to predominance of middle

Figure 5
Case 4. In 1959 pathological Q waves were evident in leads II, III, and aV₂. See figure 6 for later electrocardiograms.

MUSCULAR SUBAORTIC STENOSIS
Case 4. In May 1960, there was a leftward shift in QRS axis with the development of S waves in leads II, III, and aV\textsubscript{F}, believed to be due to a lesion in the anterior divisions of the left bundle branch (see text). This change in left ventricular conduction pattern did not significantly alter the previously observed Q waves. In October 1963, following development of complete right bundle-branch block, the Q waves became more prominent (see text).

and posterior septal hypertrophy or both following the anterior septal incision.

Although ventriculomyotomy results in the aforementioned electrocardiographic alterations, it is believed that the success of this operation is not in any way related to these changes. Evidence has been provided that the subaortic stenosis is caused by the contraction of the circularly arranged deep bullospiral and sinospiral muscle bundles (which comprise the major portion of the septal musculature) and that the muscle-splitting septal

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incision abolishes the stenosis by cutting perpendicularly across the long axis of these bundles.41, 42

Summary

The electrocardiograms of four patients with muscular subaortic stenosis, in whom there was angiographic evidence of ventricular septal hypertrophy (confirmed at surgery in three), revealed pathological Q waves in the limb leads (except aVR) and the left precordial leads, as well as tall R waves in the right precordial leads. Three patients underwent surgical correction of the stenosis. The left anterior septal ventriculotomy abolished the Q waves and diminished the right precordial R waves while at the same time producing left axis deviation believed to be due to severing of the anterior division(s) of the left bundle branch. In the fourth patient the left precordial and limb lead Q waves and right precordial R waves increased following spontaneous development of complete right bundle-branch block. These observations support the belief that the Q-wave and R-wave abnormalities in the electrocardiogram in patients with muscular subaortic stenosis are the result of septal depolarization commencing at the left septal surface and may be related to the overall hypertrophy of the septum, the bizarre myocardial fiber hypertrophy or premature activation of the base of the septum, or all three.

Addendum

Subsequent to the submission of this report we have carried out postoperative catheterization studies on two patients in whom the ventriculotomy incision failed to produce any ventricular conduction defect. There was complete abolition of the intraventricular pressure gradient in each instance, both at rest and following drug stimulation, providing further evidence that the success of this type of surgery, in muscular subaortic stenosis, is related to the muscle-splitting incision and not to the production of a ventricular conduction defect.

References

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