Etiology of Right Bundle-Branch Block in Patients Undergoing Total Correction of Tetralogy of Fallot

By Henry Gelband, M.D., Albert L. Waldo, M.D., Gerard A. Kaiser, M.D., Frederick O. Bowman, Jr., M.D., James R. Malm, M.D., and Brian F. Hoffman, M.D.

SUMMARY
The electrocardiographic (ECG) pattern of right bundle-branch block (RBBB) occurs routinely in patients after open-heart surgery for tetralogy of Fallot (TF). To determine the etiology of the RBBB pattern, 14 patients with TF, seven with ventricular septal defects (VSD), and one with pulmonary stenosis (PS) were studied during and after cardiac surgery. Bipolar electrograms from 10 selected right ventricular epicardial sites were recorded simultaneously with an ECG. Records were obtained before and immediately after vertical right ventriculotomy, after infundibular resection, and after repair of a ventricular septal defect (VSD). The vertical ventriculotomy alone was always associated with significant prolongation of the time of epicardial activation only to the recording sites lateral to the incision with prolongation of the QRS complex by an average of 39 msec, and with the appearance of an RBBB ECG pattern. Infundibular resection and VSD repair were not associated with any changes in the electrophysiologic parameters measured. A retrospective analysis of 251 patients with TF, VSD, and PS revealed a 100% incidence of RBBB pattern in the electrograms of only those who had undergone ventriculotomy. It can be concluded that the RBBB pattern seen postoperatively in patients with TF is due to changes in right ventricular activation secondary to the vertical ventriculotomy.

Additional Indexing Words:
Vertical ventriculotomy
Right ventricular activation time
Specialized conduction system

Congenital heart disease
Cardiac surgery

The electrocardiographic pattern of right bundle-branch block (RBBB) invariably follows total surgical correction of tetralogy of Fallot (TF). However, to date there is no direct physiologic evidence concerning the etiology of this RBBB pattern. RBBB has also been noted after surgical correction of ventricular septal defects (VSD) and infundibular pulmonic stenosis (PS). Investigators have attributed the RBBB pattern observed following surgical repair of VSD to operative trauma to the right main bundle branch. The

From the Departments of Pharmacology and Surgery, The College of Physicians and Surgeons of Columbia University, and the Surgical Service, The Presbyterian Hospital, New York, New York.

Supported in part by research grant HE 12738 from the National Heart and Lung Institute, U. S. Public Health Service. Work was performed during Dr. Gelband’s tenure as Special Postdoctoral Research Fellow (HE-42457) from the National Heart and Lung Institute and during Dr. Waldo’s tenure as a John Polachek Medical Research Fellow. Dr. Waldo and Dr. Kaiser are Otto G. Storm Established Investigators, American Heart Association.

Dr. Gelband’s present address: Department of Pediatrics, University of Miami School of Medicine, Miami, Florida.

Address for reprints: Albert L. Waldo, M.D., Department of Pharmacology, College of Physicians and Surgeons of Columbia University, 630 West 168th Street, New York, New York 10032.

Received June 22, 1971; accepted for publication August 3, 1971.
ETIOLOGY OF RBBB

etiology of this electrocardiographic pattern following correction of PS was thought to be due to infundibular resection of the subpulmonary area with interruption of distal ramifications of the right bundle branch. However, Coggin and associates, in a published abstract, suggested that the RBBB seen following VSD repair was not due to trauma to the main right bundle branch but was a direct result of interruption of the peripheral ventricular conduction system secondary to the right ventriculotomy.

It had been assumed from electrocardiographic observations, from the clinical studies on other forms of congenital heart disease just cited,7-9 and because the anatomic course of the right bundle branch10 is similar in TF and VSD, that the RBBB pattern observed postoperatively in TF was most likely due to surgical trauma to the proximal right bundle branch.11,12 Because no physiologic data are available concerning the etiology of the RBBB pattern observed postoperatively in TF to support the conclusions drawn from the anatomic and clinical studies, and because right ventriculotomy, infundibular resection, and direct closure of the VSD have all been implicated in the etiology of the RBBB, and all are necessary in the surgical repair of TF, we have conducted a systematic physiologic investigation to determine which is the prime factor in the genesis of this electrocardiographic manifestation.

Methods

All patients were studied during cardiac surgery. The experimental protocol varied slightly for each patient and was determined by the nature and requirements of the surgical procedure. A summary of pertinent clinical data on the 22 patients studied is provided in table 1. The ages of the patients studied ranged from 3 to 19 years, the mean age being 9 years. All patients underwent cardiovascular catheterization and cardiac angiography prior to surgery. Fourteen patients had TF, seven had VSD, and one patient had infundibular and pulmonary valve stenosis. Of the seven patients with VSD, three had undergone pulmonary artery banding in infancy, and therefore at the time of repair of their lesion their condition hemodynamically resembled TF. The VSD of six patients was corrected via a right ventriculotomy and the last patient’s lesion (case 21) was corrected through a right atriotomy. All had membranous or subcristal VSD. Electrocardiograms (ECG) recorded in the operating room prior to thoracotomy were comparable in all respects to those recorded 1 to 3 days prior to surgery. All patients had a QRS duration of 80 msec or less prior to surgery. RBBB was defined13 as (1) QRS duration greater than 100 msec, (2) wide slurred S in leads I and V6, and (3) slurred R' in leads III, aV R, and V1.

All studies were performed during the period of cardiopulmonary bypass. Each patient was studied during spontaneous atrial rhythm or during atrial rhythm produced by pacing the atria through a bipolar electrode placed in the region of the sinoatrial node.14 The atria were paced at a rate slightly in excess of the spontaneous rate. Stimuli were provided by a special digital threshold stimulator (Medtronics 1187). All leads in contact with the heart were isolated both from ground and from the recording instruments by isolation transformers. Using a hand-held probe containing three silver electrodes,14 bipolar electrograms were recorded from 10 selected sites on the epicardial surface of the right ventricle (fig. 1). Recordings were obtained before any surgical

Figure 1

Anterior aspect of the human heart showing the 10 right ventricular epicardial recording sites. Dashed line indicates vertical ventriculotomy incision. Ao = aorta, PA = pulmonary artery, RA = right atrium, RV = right ventricle, LV = left ventricle.
### Table 1

**Clinical Data for 22 Patients Studied**

<table>
<thead>
<tr>
<th>Patient</th>
<th>Lesion</th>
<th>Age (yr)</th>
<th>Sex</th>
<th>Preanesthetic medication (mg)</th>
<th>Anesthesia (mg)</th>
<th>Temperature (°C)</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td>Induction</td>
<td>Maintenance</td>
</tr>
<tr>
<td>1</td>
<td>TF</td>
<td>9</td>
<td>M</td>
<td>S 40</td>
<td>C</td>
<td>N</td>
</tr>
<tr>
<td>2</td>
<td>TF</td>
<td>6</td>
<td>M</td>
<td>S 40</td>
<td>SDC 15</td>
<td>H</td>
</tr>
<tr>
<td>3</td>
<td>TF</td>
<td>3</td>
<td>M</td>
<td>S 40</td>
<td>C</td>
<td>H</td>
</tr>
<tr>
<td>4</td>
<td>TF</td>
<td>13</td>
<td>M</td>
<td>S 100</td>
<td>P 125</td>
<td>H</td>
</tr>
<tr>
<td>5</td>
<td>TF</td>
<td>6</td>
<td>M</td>
<td>S 50</td>
<td>C</td>
<td>H</td>
</tr>
<tr>
<td>6</td>
<td>TF</td>
<td>10</td>
<td>M</td>
<td>S 50</td>
<td>C</td>
<td>H</td>
</tr>
<tr>
<td>7</td>
<td>TF</td>
<td>11</td>
<td>M</td>
<td>S 100</td>
<td>SDC 200</td>
<td>H</td>
</tr>
<tr>
<td>8</td>
<td>TF</td>
<td>8</td>
<td>M</td>
<td>S 30</td>
<td>SDC 30</td>
<td>H</td>
</tr>
<tr>
<td>9</td>
<td>TF</td>
<td>15</td>
<td>M</td>
<td>S 100</td>
<td>SDC 40</td>
<td>H</td>
</tr>
<tr>
<td>10</td>
<td>TF</td>
<td>9</td>
<td>M</td>
<td>S 50</td>
<td>SDC 40</td>
<td>H</td>
</tr>
<tr>
<td>11</td>
<td>TF</td>
<td>15</td>
<td>M</td>
<td>S 100</td>
<td>SDC 40</td>
<td>N</td>
</tr>
<tr>
<td>12</td>
<td>TF</td>
<td>9</td>
<td>F</td>
<td>S 50</td>
<td>SDC 25</td>
<td>H</td>
</tr>
<tr>
<td>13</td>
<td>TF</td>
<td>19</td>
<td>M</td>
<td>S 150</td>
<td>SDC 100</td>
<td>H</td>
</tr>
<tr>
<td>14</td>
<td>TF</td>
<td>7</td>
<td>F</td>
<td>S 40</td>
<td>SDC 20</td>
<td>H</td>
</tr>
<tr>
<td>15</td>
<td>VSD</td>
<td>7</td>
<td>F</td>
<td>S 50</td>
<td>SDC 40</td>
<td>H</td>
</tr>
<tr>
<td>16</td>
<td>VSD</td>
<td>7</td>
<td>F</td>
<td>S 50</td>
<td>SDC 40</td>
<td>H</td>
</tr>
<tr>
<td>17</td>
<td>VSD</td>
<td>9</td>
<td>M</td>
<td>S 50</td>
<td>SDC 30</td>
<td>H</td>
</tr>
<tr>
<td>18</td>
<td>VSD</td>
<td>8</td>
<td>F</td>
<td>S 50</td>
<td>SDC 100</td>
<td>H</td>
</tr>
<tr>
<td>19</td>
<td>VSD</td>
<td>12</td>
<td>M</td>
<td>S 60</td>
<td>SDC 100</td>
<td>H</td>
</tr>
<tr>
<td>20</td>
<td>VSD</td>
<td>7</td>
<td>M</td>
<td>S 50</td>
<td>SDC 40</td>
<td>H</td>
</tr>
<tr>
<td>21*</td>
<td>VSD</td>
<td>12</td>
<td>F</td>
<td>S 65</td>
<td>SDC 40</td>
<td>H</td>
</tr>
<tr>
<td>22</td>
<td>PS</td>
<td>17</td>
<td>F</td>
<td>S 100</td>
<td>P 175</td>
<td>H</td>
</tr>
</tbody>
</table>

*No ventriculotomy.

Abbreviations for lesions: TF = tetralogy of Fallot; VSD = ventricular septal defect; PA band = pulmonary artery banding; PS = pulmonic stenosis (valve and infundibular).

Abbreviations for drugs: A = atropine; C = cyclopropane; H = halothane; MS = morphine sulfate; N = N₂O; P = thiopental (Pentothal); SDC = succinylcholine; S = scopolamine.

**Manipulation upon the heart was performed and then after each of the following surgical procedures, in order: (1) after the vertical right ventriculotomy (an incision usually about 4 to 6 cm long) which always was made between sites 1 to 3 and 4 to 6 (fig. 1), (2) after subpulmonic**

*Circulation, Volume XLIV, December 1971*
ETIOLOGY OF RBBB

infundibular resection, (3) after repair of the VSD, and (4) after the repair of the ventriculotomy. Patients with just a VSD of course had no subpulmonic infundibular resection, and the patient with infundibular and pulmonary valve stenosis had no repair of a VSD. During four studies, the surgical repair required transient cross clamping of the aorta. Recordings after periods of aortic cross clamping were made after a period of reperfusion of the heart such that there was no evidence of conduction abnormalities related to the recent cross clamping.

The electrograms were monitored simultaneously with ECG leads I, aV\(_R\), and either aV\(_L\) or aV\(_F\) on a switched-beam oscilloscope (Electronics for Medicine model DR-12) and recorded on photographic paper moving at a speed of 1 mm/sec. ECGs were recorded at a sensitivity of 2 cm/mv. During periods of data collection, body temperature, measured from the retrocardiac portion of the esophagus or from the rectum, varied from patient to patient as determined by the requirements of the surgical procedure. The range was 32 to 37°C for all patients studied except one in whom the temperature was 30°C during the procedure. For each patient, all recordings were made at the same temperature. The relative sequence of activation of the right ventricular recording sites was determined by measuring the interval from the earliest recorded onset of the QRS complex in the bipolar electrograms recorded from each site. Measurements of all intervals were made from the records using a vernier measuring device having an accuracy of \pm 1 msec.

To obtain data on the incidence of an RBBB pattern recorded following open-heart surgery at our institution, in a retrospective 4-year study (January 1967 to December 1970), the preoperative and postoperative ECGs of patients with the following defects were analyzed: TF, VSD repaired through a ventriculotomy, VSD repaired via an atriotomy, and PS repaired from the pulmonary artery or through a right ventriculotomy.

Results

Relationship of RBBB Pattern to Right Ventriculotomy

An electrocardiographic pattern of RBBB occurred in all patients in whom right vertical ventriculotomy was performed as part of the surgical repair. ECGs for a representative patient recorded 2 days before and 9 days after surgery are shown in figures 2 and 3. Associated with the appearance of the RBBB pattern in this patient, the QRS complex increased 40 msec in duration. An average increase of 39 msec in QRS duration occurred in all patients who had a ventriculotomy. It is of interest to note that the greater postoperative increases in QRS duration occurred in patients with the narrower QRS complexes preoperatively (table 2). In each instance in which the RBBB pattern was recorded after ventriculotomy, prolongation of the QRS complex was associated with the appearance of a slurred S wave in ECG lead I and an R' deflection in lead aV\(_R\). Patient 21, who underwent repair of her VSD through the right atrium, and hence did not require ventriculotomy, did not develop a postoperative ECG pattern of RBBB. A transient increase in QRS duration of 10 msec did occur, but the duration returned to the preoperative value within 3 days.

Epicardial Activation Times in Relation to Ventriculotomy

Activation time measured from onset of the earliest deflection of the QRS complex to each of the 10 right ventricular epicardial recording sites during the various stages of repair for a representative patient with TF is shown in figure 4. Activation times of all 10 sites during control recordings (panel A) in this patient varied from 37 to 43 msec. Immediately after the vertical ventriculotomy (panel B), there was little or no change in the time of activation of sites 1, 2, 3, and 10. However, time of activation of all sites lateral to the ventriculotomy, i.e., sites 4 to 9, increased by 56 to 61 msec. Concomitant with this increase in activation times there was a change in the QRS morphology and duration. The initial QRS duration of 78 msec increased to 118 msec. Associated with the changes in activation time following ventriculotomy was an obvious slurring of the S wave (panel B), which produced the increase in the duration of the QRS complex. Panels C and D represent activation times of the epicardial sites after resection of the subinfundibular pulmonic area and repair of the VSD, respectively. Neither a further increase in activation time of any of the 10 epicardial sites nor changes in the QRS morphology or
duration occurred after these latter two surgical procedures. Similar increases in activation times of sites 4 to 9 occurred in all patients who had a right ventriculotomy as part of their surgery (table 3). Activation times prolonged by 34 to 68 msec, whereas activation time of sites 1, 2, 3, and 10 remained essentially unchanged throughout surgery. Prolongations of the QRS complex and changes in QRS morphology were recorded in all these patients only immediately after the ventriculotomy.

An analysis of the activation times in the one patient who had VSD corrected through the right atrium is shown in figure 5. In panel A, control activation times are noted. Activation times to the epicardial sites ranged from 29 to 33 msec. After repair of the VSD (panel B) activation times to the epicardial sites were similar to control values. They ranged from 29 to 35 msec. Lead-I QRS complexes before and after repair of the VSD are also shown. Note that no changes occurred in QRS morphology or duration.

**Epicardial Activation Time in Relation to the QRS Complex**

A representative example of the changes in QRS configuration and duration which occur after ventriculotomy is shown for patient 9 with TF (fig. 6). Panel A is a lead-I QRS complex recorded prior to ventriculotomy. Lead-I QRS morphology (rS) is typical of a patient with TF.33 QRS duration is 60 msec. Activation of epicardial sites 1 to 10 occurred
ETIOLOGY OF RBBB

Figure 3

ECG of the same patient as in figure 2, taken 9 days after surgery. QRS interval now has increased to 120 msec. A prominent slurred S is present in leads I and aV1, and a slurred R in lead aVR. Precordial lead V1 has the typical broad slurred R', and lead V6 has the slurred S wave observed in RBBB.

between 26 and 39 msec after the beginning of the inscription of the QRS complex. Panel B shows the lead-I QRS complex immediately after the vertical ventriculotomy. The duration of the QRS complex prolonged to 118 msec and was associated with the appearance of a markedly slurred S wave. Activation times of sites 1, 2, 3, and 10 were unchanged from control. Activation times of sites 4 to 9 now were prolonged to 64 to 77 msec when compared to the control activation times. Note that sites 4 to 9 are activated during the inscription of the slurred S wave of the QRS complex.

In contrast, figure 7 demonstrates the time of activation of the right ventricular epicardial sites superimposed on a lead-I QRS complex before and after surgical repair of a VSD which was corrected without a ventriculotomy. QRS morphology was not altered by the surgical procedure; the duration of the QRS complex (78 msec) was identical before and after total correction of the lesion; and epicardial activation times to all sites before and after the repair did not significantly differ, i.e., they ranged from 29 to 33 msec before and 29 to 35 msec after surgery.

Retrospective Analysis of Preoperative and Postoperative ECG

Preoperative and postoperative ECGs of all patients with the anatomic diagnosis of TF, VSD, and PS during the 4-year period
### Table 2

**Electrocardiographic Data**

<table>
<thead>
<tr>
<th>Patient</th>
<th>Lesion</th>
<th>V</th>
<th>QRS axis</th>
<th>RVH</th>
<th>QRS duration in ECG (msec)</th>
<th>Postoperative ECG</th>
<th>RBBB pattern</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td></td>
<td></td>
<td>Preop.</td>
<td>Postop.</td>
<td>Slurred St R' aV F</td>
<td></td>
<td></td>
</tr>
<tr>
<td>1</td>
<td>TF</td>
<td>+</td>
<td>+120</td>
<td>+</td>
<td>80</td>
<td>110</td>
<td>+</td>
</tr>
<tr>
<td>2</td>
<td>TF</td>
<td>+</td>
<td>+120</td>
<td>+</td>
<td>80</td>
<td>110</td>
<td>+</td>
</tr>
<tr>
<td>3</td>
<td>TF</td>
<td>+</td>
<td>+110</td>
<td>+</td>
<td>80</td>
<td>120</td>
<td>+</td>
</tr>
<tr>
<td>4</td>
<td>TF</td>
<td>+</td>
<td>+120</td>
<td>+</td>
<td>80</td>
<td>120</td>
<td>+</td>
</tr>
<tr>
<td>5</td>
<td>TF</td>
<td>+</td>
<td>+150</td>
<td>+</td>
<td>80</td>
<td>120</td>
<td>+</td>
</tr>
<tr>
<td>6</td>
<td>TF</td>
<td>+</td>
<td>+110</td>
<td>+</td>
<td>80</td>
<td>110</td>
<td>+</td>
</tr>
<tr>
<td>7</td>
<td>TF</td>
<td>+</td>
<td>+115</td>
<td>+</td>
<td>80</td>
<td>110</td>
<td>+</td>
</tr>
<tr>
<td>8</td>
<td>TF</td>
<td>+</td>
<td>+100</td>
<td>+</td>
<td>70</td>
<td>120</td>
<td>+</td>
</tr>
<tr>
<td>9</td>
<td>TF</td>
<td>+</td>
<td>+150</td>
<td>+</td>
<td>75</td>
<td>120</td>
<td>+</td>
</tr>
<tr>
<td>10</td>
<td>TF</td>
<td>+</td>
<td>+130</td>
<td>+</td>
<td>80</td>
<td>120</td>
<td>+</td>
</tr>
<tr>
<td>11</td>
<td>TF</td>
<td>+</td>
<td>+110</td>
<td>+</td>
<td>80</td>
<td>110</td>
<td>+</td>
</tr>
<tr>
<td>12</td>
<td>TF</td>
<td>+</td>
<td>+120</td>
<td>+</td>
<td>70</td>
<td>115</td>
<td>+</td>
</tr>
<tr>
<td>13</td>
<td>TF</td>
<td>+</td>
<td>+135</td>
<td>+</td>
<td>80</td>
<td>120</td>
<td>+</td>
</tr>
<tr>
<td>14</td>
<td>TF</td>
<td>+</td>
<td>+120</td>
<td>+</td>
<td>70</td>
<td>115</td>
<td>+</td>
</tr>
<tr>
<td>15</td>
<td>VSD, PA band</td>
<td>+</td>
<td>+110</td>
<td>+</td>
<td>80</td>
<td>120</td>
<td>+</td>
</tr>
<tr>
<td>16</td>
<td>VSD, PA band</td>
<td>+</td>
<td>+105</td>
<td>+</td>
<td>70</td>
<td>115</td>
<td>+</td>
</tr>
<tr>
<td>17</td>
<td>VSD, PA band</td>
<td>+</td>
<td>+105</td>
<td>+</td>
<td>80</td>
<td>120</td>
<td>+</td>
</tr>
<tr>
<td>18</td>
<td>VSD</td>
<td>+</td>
<td>+60</td>
<td>-</td>
<td>60</td>
<td>100</td>
<td>+</td>
</tr>
<tr>
<td>19</td>
<td>VSD</td>
<td>+</td>
<td>+75</td>
<td>-</td>
<td>60</td>
<td>110</td>
<td>+</td>
</tr>
<tr>
<td>20</td>
<td>VSD</td>
<td>+</td>
<td>0</td>
<td>-</td>
<td>60</td>
<td>115</td>
<td>+</td>
</tr>
<tr>
<td>21</td>
<td>VSD</td>
<td>-</td>
<td>0</td>
<td>-</td>
<td>70</td>
<td>80</td>
<td>-</td>
</tr>
<tr>
<td>22</td>
<td>PS</td>
<td>+</td>
<td>+120</td>
<td>+</td>
<td>75</td>
<td>115</td>
<td>+</td>
</tr>
</tbody>
</table>

Abbreviations: those for lesions same as in table 1; V = ventriculotomy; + = present; = = not present; RBBB = right bundle-branch block; RVH = right ventricular hypertrophy.

### Table 3

**Epicardial Activation Times and QRS Duration (Msec*) in Representative Cases following Surgical Procedures**

<table>
<thead>
<tr>
<th>Lesion</th>
<th>Activation times* at recording sites</th>
<th>QRS duration</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>1  2  3  4  5  6  7  8  9  10</td>
<td></td>
</tr>
<tr>
<td>TF†</td>
<td>C 39 36 37 44 42 42 45 44 44 44</td>
<td>76</td>
</tr>
<tr>
<td>After V</td>
<td>41 39 36 104 99 96 102 101 99 42</td>
<td>115</td>
</tr>
<tr>
<td>After I</td>
<td>41 42 39 106 102 102 103 99 101 44</td>
<td>118</td>
</tr>
<tr>
<td>After VSD repair</td>
<td>39 41 40 103 102 103 99 97 97 40</td>
<td>117</td>
</tr>
<tr>
<td>VSD with PA band†</td>
<td>C 40 42 38 39 41 41 43 44 45 44</td>
<td>78</td>
</tr>
<tr>
<td>After V</td>
<td>38 39 41 90 94 91 93 95 90 42</td>
<td>117</td>
</tr>
<tr>
<td>After I</td>
<td>42 41 42 95 96 94 94 95 93 45</td>
<td>121</td>
</tr>
<tr>
<td>After VSD repair</td>
<td>42 43 42 94 99 92 93 93 93 45</td>
<td>122</td>
</tr>
<tr>
<td>VSD‡</td>
<td>C 35 35 34 38 37 36 37 37 36 36</td>
<td>68</td>
</tr>
<tr>
<td>After V</td>
<td>34 34 33 86 89 90 92 92 94 38</td>
<td>105</td>
</tr>
<tr>
<td>After VSD repair</td>
<td>36 38 38 89 92 91 90 91 93 38</td>
<td>108</td>
</tr>
<tr>
<td>VSD‡</td>
<td>C 31 30 29 33 29 29 31 31 31 31</td>
<td>70</td>
</tr>
<tr>
<td>After VSD repair</td>
<td>33 30 29 34 31 30 35 34 34 32</td>
<td>78</td>
</tr>
<tr>
<td>PS†</td>
<td>C 31 29 29 31 31 30 28 30 30 34</td>
<td>76</td>
</tr>
<tr>
<td>After V</td>
<td>34 31 31 68 72 74 71 74 76 34</td>
<td>110</td>
</tr>
<tr>
<td>After I</td>
<td>34 32 33 69 73 73 72 73 72 76</td>
<td>113</td>
</tr>
</tbody>
</table>

*Time of activation in milliseconds from onset of first deflection of the QRS complex.
†Lesion corrected with ventriculotomy.
‡Lesion corrected without ventriculotomy.

Abbreviations: those for lesions same as in table 1; C = control; V = ventriculotomy; I = infundibular resection; VSD = ventricular septal defect.
Etiology of RBBB

Figure 4

Effect of a vertical ventriculotomy on the activation times to the right ventricular epicardial recording sites in a representative patient (case 11). Each panel shows a schematic drawing of the right ventricle with the time of activation to each site illustrated. Below the right ventricular drawing is an electrocardiographic lead-I QRS complex recorded during the period of measurement of the activation times. (A) Activation times made prior to the ventriculotomy ranged from 37 to 43 msec for the 10 epicardial sites. The QRS interval is 78 msec. (B) Immediately after the ventriculotomy (dashed line), epicardial activation times to sites 1, 2, 3, and 10 remained unchanged while activation time to sites 4 to 9 significantly increased (range 99 to 103 msec). The QRS complex has prolonged to 118 msec, associated with the appearance of a slurred S wave. (C and D) Activation times after infundibular resection and repair of the VSD. There are no further changes in epicardial activation time, QRS duration, or morphology after these procedures. Time scale: 100 msec as indicated.

(1/1/67 to 12/30/70) were analyzed to determine the incidence of a postoperative RBBB pattern (table 4). One hundred thirty-nine patients with TF, 55 with VSD, and 12 with infundibular PS had open-heart surgery in which total correction of their lesions necessitated a vertical ventriculotomy. None of these patients had an RBBB pattern recorded in the preoperative ECG, but all developed an RBBB pattern following surgery. On the other hand, there were 26 patients with VSD and 19 with valvular PS who had surgical correction either from a right atrial approach (VSD patients) or via the main pulmonary artery (PS patients); none of these patients developed ECG evidence of a RBBB pattern following surgery. Thus the incidence of an RBBB pattern following right ventriculotomy was 100%, but was 0% when no ventriculotomy was performed, despite the fact that patients with similar anatomic lesions are included in both groups.

Figure 5

Right ventricular epicardial activation times after repair of VSD via the right atrium (case 21). Each panel shows a schematic drawing of the right ventricle with the time of activation to each site illustrated. Below the right ventricular drawing is an electrocardiographic lead-I QRS complex recorded during the period of measurement of the activation times. (A) Activation times to the 10 epicardial sites prior to VSD repair. These activation times ranged from 29 to 33 msec. The QRS interval measured 75 msec before repair of the VSD. (B) Activation times to the 10 epicardial sites are essentially unchanged (29 to 35 msec) after repair of the VSD. The morphology and duration (78 msec) of the QRS complex are also unchanged. Time scale: 100 msec as indicated.
Discussion

The electrocardiographic pattern of RBBB which is recorded postoperatively from patients with VSD has been attributed most often to surgical damage to the proximal right bundle branch,4–7 since the bundle branch lies on the posterior-inferior aspect of the VSD.10 Anatomic studies of the ventricular conducting system in TF demonstrated that in this lesion the conducting system is also positioned in the posterior-inferior portion of the defect.9,15 Hence the RBBB which invariably follows surgical correction of TF has been attributed to injury to the proximal conducting system occurring when the VSD was repaired.11,12 On the other hand, Esmond and others,16 studying the peripheral ramifications of the cardiac conducting system in the human heart, demonstrated that an operative incision in the anterior wall of the right ventricle may result in RBBB due to interruption of the Purkinje fiber network distal to the moderator band. Fisher and associates17 described changes in the postoperative ECG of patients who had their VSD corrected and felt that ventriculotomy was crucial to the development of an RBBB pattern. Coggin and others8 supported the pathologic study of Esmond’s group which described for all patients undergoing VSD repair the onset of an RBBB pattern at the precise moment of incision of the right ventricle.

Indirect support for our observations is provided by experimental studies on canine hearts.18–20 These studies have demonstrated that right ventricular activation in the dog normally begins in the lower and anterior apical area adjacent to the anterior interventricular groove. Activation then proceeds laterally over the epicardial surface. Genender and associates18 have shown that when a

Table 4
Incidence of a Right Bundle-Branch Block Pattern in Postoperative Electrocardiograms: Retrospective and Prospective Study

<table>
<thead>
<tr>
<th>Lesion</th>
<th>Present study</th>
<th>Retrospective</th>
<th>Total</th>
<th>V</th>
<th>% with RBBB</th>
</tr>
</thead>
<tbody>
<tr>
<td>TF</td>
<td>14</td>
<td>139</td>
<td>153</td>
<td>153</td>
<td>100</td>
</tr>
<tr>
<td>VSD</td>
<td>6</td>
<td>53</td>
<td>61</td>
<td>61</td>
<td>100</td>
</tr>
<tr>
<td>VSD*</td>
<td>1</td>
<td>26</td>
<td>27</td>
<td>0</td>
<td>0</td>
</tr>
<tr>
<td>PS</td>
<td>1</td>
<td>12</td>
<td>13</td>
<td>13</td>
<td>100</td>
</tr>
<tr>
<td>PS†</td>
<td>0</td>
<td>19</td>
<td>19</td>
<td>0</td>
<td>0</td>
</tr>
</tbody>
</table>

*VSD corrected via atriotomy. †Valvotomy only.
Abbreviations for lesions same as in table 1; RBBB = right bundle-branch block pattern; V = ventriculotomy.

Figure 6
Temporal relation of the time of activation of the 10 epicardial sites plotted on an electrocardiographic lead-I QRS complex before (A), and immediately after (B), ventriculotomy in patient 9. (A) The QRS duration is 60 msec. Note that activation times to the 10 epicardial sites occurred between 26 and 39 msec. (B) After the ventriculotomy a slurred S wave appeared and was associated with an increase of the QRS duration to 115 msec. Activation times to sites 1, 2, 3, and 10 are unchanged. However activation times to sites 4 to 9 have prolonged to 64 to 77 msec and occur during the inscription of the slurred S wave. Time scale as indicated.

Circulation, Volume XLIV, December 1971
ETIOLOGY OF RBBB

ECGI

Figure 7
Temporal relation of the time of activation of the 10 epicardial sites plotted on an electrocardiographic lead, I QRS complex in patient 21 who had a VSD repaired without ventriculotomy (i.e., via a right atriotomy). Prior to VSD repair activation times to the 10 epicardial sites occurred 29 to 33 msec after the onset of the QRS complex (A). After repair of the VSD, activation times are essentially unchanged, 29 to 35 msec (B). Note also that there was no change in QRS duration or morphology. See text for further discussion. Time scale as indicated.

Ventriculotomy is made lateral to the free-running Purkinje fibers, there is a consistent alteration in the sequence of activation of the right ventricular epicardium as well as a prolongation in the total time required for right ventricular depolarization. However, there were no changes resembling RBBB in the ECG. Hill and others,20 studying the time and sequence of right ventricular epicardial activation in the dog, demonstrated that in experimental and spontaneous RBBB there is a later activation of the right ventricular epicardial sites than in normal dogs.

It is clear from our physiologic data that the vertical ventriculotomy alone was responsible for the occurrence of the RBBB pattern in the ECGs in our cases. Furthermore, the retrospective comparison of the ECGs of patients undergoing TF repair, correction of VSDs with or without a ventriculotomy, or PS repair with or without a ventriculotomy at our institution also indicates that the ventriculotomy was responsible for the RBBB pattern in these patients as well. If the repair of the VSD per se results in trauma to the right bundle branch with consequent interruption of the right bundle branch, then one would expect an equal or similar incidence of RBBB following VSD repair regardless of the surgical approach, but clearly this is not true. An incidence of RBBB of 100% in patients with VSD corrected via a ventriculotomy and an incidence of 0% when no ventriculotomy was necessary suggests the presence of RBBB due to ventricular trauma. Physiologic support our thesis.

It is of paramount importance to emphasize that all 21 of our patients who required ventriculotomy developed the characteristic electrocardiographic pattern of RBBB. QRS duration increased by about 40 msec mainly due to the appearance of slow terminal right ventricular depolarization (slurred S wave in lead I). This occurred simultaneously with the termination of the ventriculotomy. Activation time of those sites that were lateral to the ventriculotomy significantly prolonged (34 to 68 msec) while activation time of those sites that were medial to the ventriculotomy remained unchanged. Since activation time of these latter sites remained unchanged, we assume that no further injury to the right ventricular conduction occurred with further surgical procedures. From the studies by Durrer and co-workers21 on the normal sequence of ventricular activation of the human heart, if trauma to the proximal right bundle branch had occurred at any time during surgery, we would have expected a prolongation in activation time of all sites on the right epicardial surface, not just those sites lateral to the ventriculotomy. The studies of Hill and others20 on the canine heart also support our thesis.
We have demonstrated that when vertical ventriculotomy is employed during cardiac surgery for TF, VSD, and PS, the ventriculotomy is the prime factor responsible for the electrocardiographic pattern of RBBB seen postoperatively. Careful examination of the other reported examples of damage to the A-V conduction system is instructive. Rosenbaum and associates\textsuperscript{12} reported four cases of RBBB and left anterior hemiblock following correction of TF. They suggested that both the right bundle branch and left anterior branch were traumatized at surgery at the time of repair of the defect in the ventricular septum. In light of our data, we would suggest a different interpretation, namely, that the RBBB pattern resulted from the ventriculotomy and the left anterior hemiblock may have resulted from trauma to a portion of the left bundle branch. In fact, it is of interest that in one of these cases the left anterior hemiblock was transient while the RBBB remained. In this case, the right ventriculotomy probably was responsible for the RBBB, and edema for the hemiblock. As the edema regressed, the left anterior hemiblock resolved. If indeed there was surgical interruption of the right bundle and left anterior division, then both electrocardiographic manifestations should have remained.

Our data suggest that it may be necessary to reconsider the significance of the criteria employed in clinical cardiology to diagnose RBBB. It is apparent from our studies, and from the conclusions of Scherlis and Lee\textsuperscript{5} and Scherf,\textsuperscript{22} that these criteria may not be diagnostic of right bundle-branch block. We have shown that a typical electrocardiographic pattern of RBBB can occur without injury or impairment of conduction over the main right bundle branch. A vertical ventriculotomy can result in a significant delay of right ventricular activation which is capable of producing wide slurred S waves in leads I and V\textsubscript{6}, an R' in leads III, aVR, and V\textsubscript{L}, and marked prolongation of the QRS duration.

The conclusions from our data are of important prognostic value because patients who undergo corrective open-heart surgery which includes a ventriculotomy develop a RBBB pattern which is not due to injury to the main right bundle. Therefore, if left bundle-branch block occurs at a future time, these patients will not have complete heart block since the main right bundle branch is still intact.

References
1. Griffiths SP, Malm JR: Medical problems in total correction of tetralogy of Fallot. Progr Cardiol Dis 8: 64, 1965

Circulation, Volume XLIV, December 1971
ETIOLOGY OF RBBB


19. MOORE EN, HOFFMAN BF, PATTERSON DF, STUCKEY JH: Electrocardiographic changes due to delayed activation of the wall of the right ventricle. Amer Heart J 68: 347, 1964


Etiology of Right Bundle-Branch Block in Patients Undergoing Total Correction of Tetralogy of Fallot
HENRY GELBAND, ALBERT L. WALDO, GERARD A. KAISER, FREDERICK O. BOWMAN, JR., JAMES R. MALM and BRIAN F. HOFFMAN

Circulation. 1971;44:1022-1033
doi: 10.1161/01.CIR.44.6.1022
Circulation is published by the American Heart Association, 7272 Greenville Avenue, Dallas, TX 75231
Copyright © 1971 American Heart Association, Inc. All rights reserved.
Print ISSN: 0009-7322. Online ISSN: 1524-4539

The online version of this article, along with updated information and services, is located on the World Wide Web at:
http://circ.ahajournals.org/content/44/6/1022

Permissions: Requests for permissions to reproduce figures, tables, or portions of articles originally published in Circulation can be obtained via RightsLink, a service of the Copyright Clearance Center, not the Editorial Office. Once the online version of the published article for which permission is being requested is located, click Request Permissions in the middle column of the Web page under Services. Further information about this process is available in the Permissions and Rights Question and Answer document.

Reprints: Information about reprints can be found online at:
http://www.lww.com/reprints

Subscriptions: Information about subscribing to Circulation is online at:
http://circ.ahajournals.org//subscriptions/