Disturbances in Atrial Rhythm and Conduction Following the Surgical Creation of an Atrial Septal Defect by the Blalock-Hanlon Technique

By Sandra D. Hamilton, B.S., Thomas D. Bartley, M.D., Robert H. Miller, M.D., Gerold L. Schiebler, M.D., and Henry J. L. Marriott, M.D.

SUMMARY
Disturbances in atrial conduction or rhythm, or both, were found in 16 of 27 patients undergoing the surgical creation of an atrial septal defect by the Blalock-Hanlon technique. These included P-wave aberrations of intra-atrial block and ectopic atrial rhythm, A-V rhythm, A-V dissociation, sinus bradycardia, atrial flutter, escape-capture bigeminy, and atrial premature beats. Some of these were transient and occurred within 2 weeks after surgery. It seems likely that tissue trauma engendered by the clamp and resection of the atrial septum with possible injury to the internodal conducting pathways may be the genesis of these early postoperative disturbances. Of the eight cases with transient disturbances, six occurred in this period.

Later changes, such as intra-atrial block, sinus bradycardia, and atrial flutter, may be attributed to changes in atrial size secondary to the postoperative pathophysiology. Digitalis intoxication and congestive heart failure cannot be excluded as contributory factors in either the early or the late disturbances.

It was not possible to correlate the incidence and nature of these disturbances with morbidity and mortality. In such severely ill, cyanotic infants, irregularities, however slight, may have altered cardiac function significantly and contributed to their deaths.

Additional Indexing Words:
Arrhythmias
Intra-atrial conducting systems

A
n unexpected number of dysrhythmias and conduction disturbances have occurred in patients at the University of Florida Teaching Hospital, who were undergoing the creation of an atrial septal defect by the Blalock-Hanlon technique. Their incidence has seemed greater than those after various other cardiac operations. We therefore undertook an analysis to determine their nature and relative frequency, with particular emphasis on the time of onset and their possible relation to surgical trauma.

Methods
Forty-two patients have had the Blalock-Hanlon procedure for a variety of congenital malformations between July 1960 and September 1967. Of these, 27 had preoperative and postoperative electrocardiograms and these formed the basis of our study. The remaining 15 died before postoperative electrocardiograms could be taken. Twenty-six had as their primary diagnosis complete transposition of the great vessels; the other was an infant with a ventricular septal defect.
defect and pulmonary atresia ("end-stage" tetralogy of Fallot). The age of the patients at the time of operation ranged from 1 day to 8 months.

All preoperative and postoperative electrocardiograms were evaluated for changes in P-wave duration and contour, atrial conduction rates, pacemaker location, and dissociation phenomena. The initial postoperative electrocardiograms were taken at various times from the day of surgery to 2 weeks after, most being taken during the first postoperative week. Follow-up electrocardiograms were obtained at variable intervals, the longest being 7 years. Thirteen patients were on digoxin preoperatively; all were on maintenance digoxin postoperatively.

All 27 operative reports were surveyed. In addition, seven postmortem records, including four gross specimens, were available for review. The gross specimens were examined under the guidance of Dr. L. H. S. Van Mierop to determine the exact position and extent of the surgical lesion and any associated changes in chamber size or myocardial hypertrophy. Preoperative and postoperative chest x-rays were also reviewed to seek correlation between the size of the chambers and the electrocardiographic changes.

**Results**

All 27 patients had sinus rhythm before surgery. Postoperatively, 16 patients (all with complete transposition of the great vessels) developed disturbances in atrial conduction or rhythm, including atrioventricular (A-V) rhythm, transient A-V dissociation resulting from atrial slowing or A-V acceleration, sinus arrhythmia, and varying abnormal P-wave patterns including intra-atrial block and ectopic atrial rhythms. Of the 16 patients, nine had more than one type of disturbance, yielding a total of 29 changes found in the postoperative electrocardiograms (table 1). Six
Table 1
Total Number of Types of Disturbances Following the Creation of an Atrial Septal Defect by the Blalock-Hanlon Technique

<table>
<thead>
<tr>
<th>Types of disturbances</th>
<th>Number of disturbances</th>
</tr>
</thead>
<tbody>
<tr>
<td>P-wave aberrations</td>
<td></td>
</tr>
<tr>
<td>(A) Intra-atrial block or</td>
<td></td>
</tr>
<tr>
<td>left atrial enlargement</td>
<td>5</td>
</tr>
<tr>
<td>(B) Ectopic atrial rhythm</td>
<td>5</td>
</tr>
<tr>
<td>A-V rhythm</td>
<td>9</td>
</tr>
<tr>
<td>A-V dissociation*</td>
<td>5</td>
</tr>
<tr>
<td>Sinus bradycardia</td>
<td>2</td>
</tr>
<tr>
<td>Escape-capture bigeminy†</td>
<td>1</td>
</tr>
<tr>
<td>Atrial premature beats</td>
<td>1</td>
</tr>
<tr>
<td>Atrial flutter</td>
<td>1</td>
</tr>
<tr>
<td>Total</td>
<td>29</td>
</tr>
</tbody>
</table>

*Since A-V dissociation is not a complete diagnosis, but always a secondary result of a primary disturbance, the mechanisms leading to dissociation in these cases are here detailed:

1. Sinus arrhythmia, rate less than 100; A-V nodal rhythm rate 106.
3. Sinus rhythm, rate 128; A-V tachycardia, rate 130.
4. Isorhythmic dissociation with sinus and A-V rates 98.
5. Isorhythmic dissociation with atrial fusion beats; sinus and A-V rates 76.

†The couplet consisted of an A-V escape beat and an ectopic atrial beat.

patients eventually reverted to a normal sinus rhythm. Both cases of sinus bradycardia occurred as a later change, 3 and 5 years postoperatively. The five cases of intra-atrial block occurred 1 to 18 months postoperatively.

The first onset of these disturbances in the 16 patients (table 2) occurred from immediately to 3 years postoperatively. Eleven patients had early changes, that is, appearing within the first 2 weeks after the operation. The remaining five developed changes after 1 year, except for one patient who developed A-V tachycardia and “lower” A-V rhythm (fig. 1) after 4 months. Of the 11 patients with early onset of changes, five reverted to normal sinus rhythm within 3 months. Of the remainder, one patient developed atrial flutter; four, intra-atrial block; and one, A-V rhythm.

Abnormal P-wave changes are illustrated in figures 2 through 5, where they are compared with the preoperative tracings. The P-wave axis in the frontal plane changed con-
Table 2

Types of Initial Disturbances Following the Blalock-Hanlon Procedure

<table>
<thead>
<tr>
<th>Initial disturbances</th>
<th>Postoperative (0-2 weeks)</th>
<th>Postoperative (4 months or later*)</th>
</tr>
</thead>
<tbody>
<tr>
<td>P-wave aberrations:</td>
<td></td>
<td></td>
</tr>
<tr>
<td>(A) Intra-atrial block</td>
<td>—</td>
<td>1</td>
</tr>
<tr>
<td>(B) Ectopic atrial rhythm</td>
<td>3</td>
<td>1</td>
</tr>
<tr>
<td>A-V (nodal) rhythm</td>
<td>5</td>
<td>1</td>
</tr>
<tr>
<td>A-V dissociation†</td>
<td>2</td>
<td>1*</td>
</tr>
<tr>
<td>Sinus bradycardia</td>
<td>—</td>
<td>1</td>
</tr>
<tr>
<td>Escape-capture bigeminy</td>
<td>—</td>
<td>—</td>
</tr>
<tr>
<td>Atrial premature beats</td>
<td>1</td>
<td>—</td>
</tr>
<tr>
<td>Atrial flutter</td>
<td>—</td>
<td>—</td>
</tr>
<tr>
<td>Total</td>
<td>11</td>
<td>5</td>
</tr>
</tbody>
</table>

*This A-V dissociation occurred 4 months postoperatively; the other disturbances appeared after 1 year.
†For A-V dissociation mechanisms refer to numbers 1, 2, and 3 in table 1.

Figure 4

Tracings taken 1 day before surgery (A) and 2 years after surgery (B). Note wide notched P wave in lead 1 in B with deep wide negative component in leads 3 and V₃R.

Considerably in some cases (for example, figure 2 shows a shift from +60 to −40°). Others had the frontal mean P vector untouched, but showed considerable changes in the horizontal plane (fig. 3). Still others had considerable shifts in both planes (fig. 4).

Suggestions of intra-atrial conduction disturbances, or changes often interpreted as left atrial or bi-atrial enlargement, are seen in figures 2 through 5. Characteristic features of these tracings include wide, negative P-wave components in V₃R and V₁ (figs. 3 to 5), with an associated leftward shift in the P axis (figs. 2 and 4). There is also often a widening and notching of the P wave in limb leads (figs. 2, 4 and 5).

The case illustrated in figure 2 A, B, and C shows three atrial mechanisms: normal sinus rhythm, ectopic atrial rhythm, and intra-atrial block. The P waves change from a sinus contour in A to inconspicuous, low voltage waves in B, suggesting an ectopic mechanism. In C, the P waves have grossly widened and their axis has shifted leftward, suggesting intra-atrial block or left atrial enlargement.
Other striking disturbances in atrial rhythm and conduction were atrial flutter and sinus bradycardia. The patient with atrial flutter was operated on at 2 months of age and developed A-V rhythm 1 week later. Seven months postoperatively, her electrocardiogram showed isorhythmic A-V dissociation between sinus and accelerated A-V rhythm that, 3 months later, reverted to a "mid" A-V rhythm. Two and one-half years postoperatively atrial flutter with 2:1 conduction developed (fig. 6). This persisted for 2½ years until cardioversion resulted in A-V rhythm. Two hours later, isorhythmic dissociation developed between probable left atrial flutter and A-V rhythm. This patient has since had a complete repair of her defect, with the creation of an intra-atrial pericardial tunnel (Mustard procedure). Postoperatively she has had a variety of arrhythmias, including mid-A-V rhythm, lower A-V tachycardia, and atrial flutter with 1:1, 2:1, and 3:2 conduction. All postoperative tracings have shown intraventricular block.

Sinus bradycardia developed in a boy who was operated on at 7 months of age. He retained a normal sinus rhythm, with a rate of approximately 100 beats per minute, until 17 months postoperatively. At that time he developed "lower" A-V escape with transient A-V dissociation. Intermittent A-V dissociation with marked sinus arrhythmia at a rate

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**Figure 5**

Tracings taken 1 day before surgery (A) and 1 month after surgery (B). Note increased width and notching of P wave in B with wide negative component in V_{3R} compatible with left atrial enlargement.

**Figure 6**

Atrial flutter with 2:1 A-V conduction.
of approximately 50 beats per minute continued until 7 years postoperatively. Later, he developed sinus bradycardia with sinus arrhythmia at a rate of 45 beats per minute. The patient subsequently underwent subpulmonary stenosis resection, closure of a ventricular septal defect, and the creation of an intra-atrial pericardial tunnel. His electrocardiograms have since shown a regular sinus rhythm at a rate of 110 beats per minute, with intraventricular block.

Discussion

The Blalock-Hanlon procedure has gained wide acceptance as a palliative operation for complete transposition of the great vessels. Most studies have dealt with postoperative hemodynamic changes, and analyses of disturbances in atrial rhythm and conduction in such patients are limited. Khoury and associates reported that, in 28 patients having the creation of an atrial septal defect for complete transposition of the great vessels, two (14%) developed A-V rhythm; the remainder maintained sinus rhythm. This contrasts with our finding that 9 of 27 (33%) patients developed A-V rhythm. Khoury and associates also noted that 90% of their cases exhibited changes in their P waves that suggested combined or left atrial enlargement. They attributed such changes to either the results of surgery or an increase in atrial “work” secondary to increased bidirectional shunting at the atrial level. Shafer and Kidd in studying many of these same cases, found that bidirectional atrial shunting increased considerably postoperatively but that the pressures in both atria were equal and showed little change from their preoperative values. In our study, four had P-wave changes suggesting intra-atrial block or left atrial enlargement, four had changes suggesting an ectopic atrial pacemaker, and one had both.

The incidence of dysrhythmias and conduction disturbances recorded in our study (tables 1 and 2) is artificially low, since it represents only those found in survivors. Furthermore, no postoperative patient was continuously monitored. Despite this, after the Blalock-Hanlon technique, we encountered a high incidence and wide variety of atrial rhythm and conduction disturbances. No correlation could be found between the incidence and nature of these disturbances with associated defects, such as ventricular septal defect and pulmonary valvular stenosis, the age of the patient at time of operation, or the time of onset after surgery. Some were of a transient nature, soon reverting to sinus rhythm, whereas others persisted. As noted before, these disturbances appeared either within the first 2 weeks postoperatively or after 1 year.

Among the more serious dysrhythmias were included one case of atrial flutter and two of marked sinus bradycardia. Nadas has pointed out that atrial flutter is quite rare in children (less than one in 80 patients with heart disease). All three patients no longer have these serious dysrhythmias and are doing well.

Those disturbances appearing in the early postoperative period may be a consequence of surgical trauma to the sinoatrial node or interatrial conducting systems, or both. In reviewing the operative notes and discussing them in detail with the respective surgeons, it seemed that of the three internodal conducting pathways, both the posterior and middle internodal tracts could conceivably have been either traumatized or interrupted. The posterior tract is subject to injury if the clamp is applied over the crista terminalis of the right atrium; the middle tract, by the excision of the posterior portion of the atrial septum. When Holsinger and associates divided the crista terminalis of five dogs, all developed intra-atrial block, but there was no effect on A-V conduction. Division of the anterior and mid-internodal pathways, however, caused a marked delay in A-V conduction. In three of five instances, A-V rhythm was produced only when all three tracts were divided. It certainly seems unlikely that all three tracts should be damaged during the Blalock-Hanlon procedure.

Perhaps the dysrhythmias produced are the result of injury either to the S-A node or...
its blood supply, or to the A-V node or its blood supply. This is conceivable in view of the fact that the clamp encroaches upon a large portion of the right atrium and could readily damage atrial tissue. Both Selzer and Tung and associates have suggested that surgical trauma to atrial tissue may be responsible for the high incidence of dysrhythmias after cardiac surgery. Selzer found that dysrhythmias occur two and one-half times more frequently in patients after repair of atrial septal defects than those with repair of ventricular septal defects. He also noted that A-V rhythm was the most common disturbance in patients after closure of their atrial septal defect. Tung and associates found a high correlation between atrial dysrhythmias and incidence of injury to and around the area of the sinus node. They demonstrated histologically destruction and scarring of sinus nodal tissue and angiitis and thrombosis of the nodal artery. Why in our two cases of the Mustard procedure, in which the entire atrial septum is removed, the rhythm at least transiently again became normal sinus, remains unexplained.

Congestive heart failure or altered atrial hemodynamics causing right atrial dilation or enlargement, or both, and digitalis toxicity should also be considered as possible causes for the noted dysrhythmias, particularly as many of them proved to be transient in nature and others appeared too late to be the result of surgical trauma. Increased sensitivity to digitalis in the early postoperative period is possible. We encountered such possible effects of digitalis as shifting pacemaker with episodes of A-V rhythm, ectopic atrial beats, and intra-atrial block. In our series, two dysrhythmias were thought to be correlated with digitalis toxicity, in that they disappeared with discontinuance of the drug. Of the five cases that reverted to sinus rhythm, all were continually maintained on consistent or increased digitalis dosages.

In reviewing the postoperative chest x-rays, our findings are consistent with those of Starr and associates and Venables, in that the relative heart size remained fairly constant subsequent to the Blalock-Hanlon procedure. Since 10 of our patients demonstrated intra-atrial block, left atrial enlargement or ectopic atrial pacemakers, all chest x-rays were reviewed to detect changes in atrial size postoperatively. This revealed only two cases in which radiological changes after surgery were consistent with left atrial enlargement. The electrocardiograms of these two cases showed left atrial enlargement in one and A-V rhythm in the other. Even more important, in none of our necropsy material was left atrial enlargement noted. In contrast, we found the “classic” electrocardiographic P-wave pattern of left atrial enlargement in three cases in which necropsy and x-ray findings showed a normal or decreased left atrial size and an enlarged hypertrophied right atrium. Thus, massive right atrial enlargement alone may cause the “classic” P-wave configurations of left or perhaps biatrial enlargement. A possible explanation for this phenomenon is an increase in right atrial tissue thereby increasing conduction time through the atria. Since all five of our cases of intra-atrial block or left atrial enlargement occurred from 1 to 18 months postoperatively, it seems plausible to ascribe these to changes to this hypothesis.

Of course, it is well known that P-wave morphology is not predictably correlated either with the site of impulse formation or with anatomic changes. P-wave patterns considered characteristic of right atrial enlargement have been associated with conditions causing pure left atrial enlargement and it comes as little surprise that changes typical of intra-atrial block or left atrial enlargement—including widened, notched P waves, leftward and posterior displacement of the terminal P forces—should characterize right atrial enlargement, as in several of our cases. Furthermore, changes in P-wave configuration, such as are often interpreted as indicating a shift in atrial pacemaker, may just as well result from altered intra-atrial conduction.

In the absence of continuous electrocardiographic monitoring during and immediately
Glossary of Terms Used with Semantic Commentary

A-V Rhythm: Rhythm presumably arising in the A-V junction; synonymous with A-V nodal rhythm, nodal rhythm, and junctional rhythm; "lower" A-V rhythm—with P wave following QRS; "mid" A-V rhythm—with P wave lost within QRS.

Intra-atrial block: Abnormally wide P waves. In adults, the upper limit of normal P-wave duration is generally accepted at 0.11 second. In infants and children the limit has been less clearly defined. We use the term here when the P wave has clearly widened over its previous preoperative form. There is obvious overlap with the patterns of left atrial enlargement.

Ectopic atrial rhythm: Is here applied to obvious changes in P-wave contour that do not conform with the accepted patterns of atrial hypertrophy or intra-atrial block. It is well recognized that such changes might just as well represent a change in intra-atrial conduction.

References


Human Pulmonary Artery Pressures (1945) (Cournand)

"... For me this tracing holds a unique place, since it is the first demonstration that the tip of a catheter was placed in the pulmonary artery of man in order to record pressure pulses. Subsequent progress in our knowledge of the dynamics of the pulmonary circulation in man owes much to the technique of catheterization of the pulmonary artery."—A. Cournand: Description of the Prize-Winning Work. In Sourkes, Theodore L.: Nobel Prize Winners in Medicine and Physiology 1901-1965. London, Abelard-Schuman, 1966, p. 336.
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Circulation. 1968;38:73-81
doi: 10.1161/01.CIR.38.1.73
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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