SUMMARY  The occurrence of dysrhythmias after the Mustard operation for transposition of the great arteries was compared in 70 patients operated upon before and 58 patients operated upon after January 1972 when surgical modifications aimed at preserving the sino-atrial node and its arterial supply were initiated. The surgical modifications included changing the site of the superior vena cava (SVC) cannulation away from the SVC-right atrial junction, incision into the right atrial wall anterior to the sulcus terminalis, and sewing of the superior part of the baffle patch away from the sino-atrial node area.

A significant decrease in the incidence of dysrhythmias occurred in the group of patients operated upon after the surgical modifications. The modifications in surgical technique have reduced but not avoided dysrhythmias.

A HIGH INCIDENCE of dysrhythmias early and late postoperatively after the intra-atrial repair of transposition of the great arteries by the Mustard procedure has been reported by several authors. Because many of our postoperative patients had dysrhythmias similar to the brady-tachycardia syndrome described in adults, we suspected that the main cause of dysrhythmias after the Mustard operation was injury to the sino-atrial (S-A) node rather than to the atrioventricular (A-V) node or inter nodal tracts as proposed in two reports. This clinical impression was confirmed by our pathological and electrophysiological studies. The pathological studies included histologic examination of the S-A node in patients who died in either the early or late postoperative period, presumably from dysrhythmias. In hearts recently operated upon, fresh hemorrhage and acute inflammation were seen in and around the S-A node surrounding the sutures placed in the atrial myocardium. The S-A node showed degeneration and the S-A node artery was partially or completely occluded by intimal sclerosis and medial hypertrophy in those patients whose deaths occurred at four months, one year, and five years after operation.

Electrophysiological studies showed that there were no abnormalities in conduction in the atria, A-V node, or His Purkinje system and that the pacemaker recovery time, corrected for cycle length, was significantly prolonged in four of nine patients studied. The pathological changes in the S-A node and the abnormal corrected pacemaker recovery time confirmed the hypothesis that injury to the S-A node is the main cause of dysrhythmias after Mustard's operation.

To protect the S-A node and its arterial supply several modifications in the surgical technique have been made since January 1972. Cannulation of the SVC is now performed through the right atrial appendage or upper part of the SVC rather than through the superior vena caval-right atrial junction. The incision into the right atrial wall is made anterior to the sulcus terminalis to avoid injury to the S-A node artery. The superior part of the pericardial patch is sewn as far as feasible from the area of the S-A node.

The purpose of this investigation was to tabulate and compare the occurrence of early and late postoperative dysrhythmias and conduction defects presumed to have resulted directly from the surgical procedure in 70 patients operated upon before January 1972 (group A) and 58 patients operated upon after January 1972 (group B) when the surgical modifications were instituted.

Material and Methods

In the ten year period from January 1965 to December 1974, intra-atrial repair by the Mustard technique for transposition of the great arteries was performed in 179 patients. Before the surgical modifications were begun in January 1972, operation was performed in 96 patients with a mortality of 27%. After January 1972 operation was performed in 83 patients with a mortality of 19%.

Group A included all 70 patients who survived the operation before January 1972 and group B included 58 of the 67 patients who survived the operation after January 1972.

All patients were treated with digitalis postoperatively. If sinus or junctional bradycardia developed, digitalis was stopped. If no improvement was noted, digitalis was restarted.

The preoperative and postoperative electrocardiograms were analyzed for maximal amplitude of P wave, as well as for rhythm and A-V conduction. Postoperative electrocardiograms were routinely taken several times in the first two weeks, at six weeks, three months, six months, one year, and yearly. Patients who developed arrhythmias on one or more routine ECGs were followed more closely, as were patients who developed hemodynamic complications. The recording apparatus was standardized during the recording of each lead.

The results of 12 and 24 hour tape recorder ECG monitoring were not included in this study, since it was infrequently performed in the early group.

The dysrhythmias observed after the Mustard operation were classified into three groups. Those due to failure of initiation or propagation of the sinus impulse were designated passive dysrhythmias. Rhythm disturbance characterized by rapid atrial or A-V junctional impulses were termed active dysrhythmias. A-V conduction defects made up the third group. Rhythm disturbances were further categorized according to the time of appearance in the postoperative
course — first week, 2 to 6 weeks, 6 weeks to 1 year, 1 to 3 years, 3 to 5 years, and 5 to 8 years.

Results

Group A

During the three to eight year follow-up of the 70 patients in group A operated upon before January 1972, the following changes were observed:

1. P Waves with Sinus Configuration

In 54 patients atrial depolarization waves having the scalar configuration of sinus P waves were seen at one time or another postoperatively. Comparison of these P waves with the preoperative sinus P waves revealed a highly significant decrease in the maximum P wave amplitude without significant change in the maximum width. The greatest postoperative sinus P wave amplitude in any lead was less than 0.05 mv in 29 patients, between 0.05 and 0.1 mv in 20 patients, and more than 0.1 mv but usually less than 0.15 mv in five patients (table 3).

Before modifications of surgical technique only 34%-40% of patients had sinus rhythm in the first postoperative year (fig. 1). In later follow-up sinus rhythm was less frequent. There were only ten patients available for the five to eight year follow-up; thus the apparent rise in percentage with sinus rhythm in figure 1 is insignificant.

2. Passive Dysrhythmias

Table 1 and figure 1 show that the incidence of passive dysrhythmias remained nearly the same in the six postoperative time periods observed, ranging from 38% to 50%. The dysrhythmias consisted of A-V dissociation by default, slow junctional rhythm, and S-A block.

3. Active Dysrhythmias

Table 1 and figure 1 show progressive increase in the frequency of active dysrhythmias from 11% in the first week to 18% in five years. The incidence of active dysrhythmias in the ten patients seen five to eight years postoperatively decreased to 7%.

4. A-V Conduction Defects

The incidence of A-V conduction defects was low and occurred only as grade I A-V block. Partial or complete A-V block was not seen at any time, either early or late postoperatively.

Group B

During the follow-up of 58 patients operated upon after January 1972, which extends to three years in some patients, the following changes were observed:

1. P Waves with Sinus Configuration

In all patients atrial depolarization waves having the scalar configuration of sinus P waves were seen at one time or another. Comparison of these P waves with the preoperative sinus P waves revealed a highly significant decrease in the maximum P wave amplitude without significant change in the maximum width. The P wave amplitude was less than 0.05 mv in 15 patients, between 0.05-0.1 mv in 27 patients, and more than 0.1 mv in 16 patients (table 3).

Table 2 and figure 2 show that the incidence of sinus rhythm ranged from 57% to 81% during follow-up.

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Table 1. Group A: Patients Operated Upon from Jan. 1965 to Dec. 1971

<table>
<thead>
<tr>
<th>Number of patients and rhythms</th>
<th>Follow-up period</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>First week</td>
</tr>
<tr>
<td>Patients</td>
<td>70</td>
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<tr>
<td>Different rhythms</td>
<td>79</td>
</tr>
<tr>
<td>Patients with sinus rhythm</td>
<td>27</td>
</tr>
<tr>
<td>Passive dysrhythmias (total)</td>
<td>36</td>
</tr>
<tr>
<td>A-V dissociation by default</td>
<td>20</td>
</tr>
<tr>
<td>Slow junctional rhythm</td>
<td>14</td>
</tr>
<tr>
<td>S-A block</td>
<td>2</td>
</tr>
<tr>
<td>Active dysrhythmias (total)</td>
<td>9</td>
</tr>
<tr>
<td>Supraventricular tachycardias</td>
<td>8</td>
</tr>
<tr>
<td>Atrial flutter</td>
<td>1</td>
</tr>
<tr>
<td>A-V conduction defects</td>
<td>7</td>
</tr>
<tr>
<td>1° A-V block</td>
<td></td>
</tr>
</tbody>
</table>
2. Passive Dysrhythmias

The incidence varied from 15% to 22% in the three years of follow-up (table 2 and fig. 2).

3. Active Dysrhythmias

The incidence was very low and varied from 3% to 15% in the years of follow-up (table 2 and fig. 2).

4. Conduction Defects

The incidence was also low and varied from 4% to 8% and was only in the form of first degree A-V block.

Figure 3 compares the incidence of sinus rhythm, passive dysrhythmias, and active dysrhythmias in the first postoperative three years in groups A and B. The incidence of sinus rhythm was much higher and the incidence of both passive and active dysrhythmias much lower in group B than in group A. Forty-five percent of patients in group B remained constantly in sinus rhythm after the first postoperative week in contrast to only 10% of group A.

Discussion

Postoperative electrocardiographic evaluations carried out after the Mustard procedure showed a significant increase in the incidence of sinus rhythm and a significant decrease in the incidence of dysrhythmias in the group of patients operated upon after the surgical modifications.
aimed at protecting the S-A node and its arterial supply. In spite of these significant changes, dysrhythmias are still an important sequela of the Mustard operation. Only 45% of the children remained in sinus rhythm at each examination during the one to three year follow-up. Modifications in the baffling procedure to lessen the incidence of dysrhythmias were also described by different authors. A recent paper by Champsaur et al. emphasized that their technique has been basically unchanged since first described. They reported a mortality rate of 31% in the first year and an incidence of dysrhythmias of 30% in the survivors at the last follow-up, but they did not report the percentage of those who remained only in sinus rhythm postoperatively.

The fact that these surgical modifications did reduce the incidence of dysrhythmias lends support to the mechanism of the dysrhythmias which we proposed in our previous publications on this topic. Further electrophysiological studies performed on a larger series of patients also support the same hypothesis.

An important postoperative change in both groups is the decrease in the maximum amplitude of the sinus P waves, without change in the width or the mean frontal plane P axis. Although the amplitude of the postoperative sinus P waves is higher in group B than in group A, the postoperative decrease in the sinus P wave amplitude is still highly significant also in group B. There is still no definite explanation for the decrease in P wave amplitude.

The involvement of the A-V node was not considered significant in our cases since partial or complete A-V block was not seen postoperatively. This was confirmed by our electrophysiological studies which showed that there were no abnormalities in conduction in the atria, A-V node and His Purkinje system even with rapid atrial pacing. Champsaur et al. reported that impaired A-V conduction leading to death occurred only in their early cases. Aberdeen and Carr described one patient with partial and nine with complete A-V block in 49 patients of surgically treated transposition.

Although fewer arrhythmias occurred after the surgical modifications aimed at protecting the S-A node and its arterial supply, still the incidence of dysrhythmias is high, the decrease in the amplitude of P wave is significant, and the function of the S-A node as measured by corrected pacemaker recovery time was impaired in several patients.

References
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Circulation. 1976;53:788-791
doi: 10.1161/01.CIR.53.5.788

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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