Cardiac Arrhythmias Associated with the Repair of Atrial and Ventricular Septal Defects

By RIKURO SASAKI, M.D., E. O. THEILEN, M.D., L. E. JANUARY, M.D., and J. L. EHRENHAFT, M.D.

This is a report of cardiac arrhythmias associated with the repair of cardiac septal defects. Electrocardiographic studies showed a high incidence of paroxysmal disturbances of rhythm following surgery.

The remarkable technics of modern cardiac surgery, that permit restoration of normal anatomic and hemodynamic relationships in hearts with septal defects, have created some problems in the identification and control of postoperative arrhythmias. It is not surprising that auriculotomy and ventriculotomy with the closure of septal defects might produce significant disturbances of cardiac rhythm in view of the proximity of some of the specialized conducting tissues to these defects. Previous reports of surgically treated patients do not stress the occurrence of postoperative arrhythmias. This report is a review of the cardiac arrhythmias that have occurred in a series of 110 patients with atrial (62) and ventricular (48) septal defects operated on before January 1, 1958. A significant proportion of these patients had major disturbances of rhythm in the immediate postoperative period, and in the case of patients with atrial septal defects, various paroxysmal arrhythmias occurred during convalescence. Our patients were monitored electrocardiographically during surgery, at frequent intervals during the first postoperative day, and as often as indicated thereafter.

Atrial Septal Defects

All but 2 of the patients with atrial septal defects were operated on under hypothermic conditions. Rapid surface cooling was induced by immersion of the anesthetized patient in a tub filled with cracked ice and water. Cardiomyotomy was performed during occlusion of the large vessels of the in-flow and out-flow tracts to permit operation in a bloodless field. Two periods of circulatory occlusion were used when valvular pulmonic stenosis was associated with an atrial septal defect. Pulmonic valvuloplasty was performed during the first and shorter period of occlusion; then the heart was allowed to recover before the second period of occlusion for repair of the atrial septal defect. A modified atrioseptopexy was done in 2 patients with anomalous pulmonary venous connections; in them the circulation was not obstructed because the nature of the anomaly made open cardiomyotomy and visualization of the defects unnecessary.

Hicks and co-workers¹ and Fleming and Muir² have reported the electrocardiographic changes that occur during hypothermia. Similar changes were observed in our patients. Ventricular fibrillation was a frequent complication of cardiomyotomy under hypothermic conditions, occurring in 25 of 60 patients. The general problem of ventricular fibrillation in hypothermia has been reviewed recently by Badeer.³ The scattergram (fig. 1) shows that the occurrence of ventricular fibrillation in our series was related not only to the length of circulatory occlusion but also to the extent of body cooling as well. Sinus rhythm was restored in 23 of the 25 patients. It could not be restored in 1 child with an ostium primum

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defect and in 1 woman with an associated mitral stenosis. Severe pulmonary hypertension (pressures 80/40 and 95/65) was found in these 2 patients. Defibrillation generally was accomplished with little difficulty, although 6 of the 25 patients maintained the arrhythmia for more than 1 minute. These longer periods of fibrillation were 4, 5, 8, 27, 40, and 120 minutes respectively, despite continued cardiac massage and repeated attempts at electric defibrillation. Sinus rhythm was restored ultimately in each of these 6 patients. Evidence of muscle damage occurred in the 2 patients who fibrillated for 8 and 120 minutes and in 1 who fibrillated for less than 1 minute. The electrocardiographic changes consisted of deep T-wave inversions that gradually regressed (fig. 2). Clinical recovery was complete. It seems likely that more than one factor is responsible for the electrocardiographic evidence of muscle damage after ventricular fibrillation. The duration of the attack, the trauma of cardiac massage, and electric defibrillation are undoubtedly important factors. Peddie et al.4 have shown that structural changes in the heart may result from cardiac massage.

Preoperative cardiac arrhythmias were not common in these patients. Atrial fibrillation was present in a 44 year old woman with an isolated ostium secundum defect and moderate pulmonary hypertension (67/30 mm Hg). Paroxysmal supraventricular tachycardia was documented in a 15 year old girl with an ostium primum defect. It is of interest that first-degree atrioventricular heart block (P-R 0.22 to 0.24 second) occurred only in patients with ostium primum defects. It occurred in 4 such patients, aged 10 to 23 years, none of whom had received digitalis.

The frequency of postoperative cardiac arrhythmias was impressive (table 1). Eighteen patients (30.5 per cent) who survived the immediate postoperative period developed disturbances that were not present preoperatively. All were supraventricular in origin. The following arrhythmias appeared during the first postoperative day in 9 patients: paroxysmal nodal tachycardia, 1; atrial fibrillation, 5; atrial flutter, 1; and paroxysmal atrial tachycardia, 2. With 1 exception the arrhythmias during the first day were transient and responded readily to digitalization. The paroxysmal nodal tachycardia occurred in a child who continued to have difficulty for more than 1 year. She had intermittent paroxysms of supraventricular tachycardia associated with faintness and weakness and episodes of paroxysmal nodal tachycardia that alternated with periods of reciprocal rhythm and wandering pacemaker.

Nine patients with complicated atrial septal defects died, 3 in the operating room. Two were the result of persistent ventricular fibrillation; in the other a complete A-V heart block resulted in an ineffective idioventricular rhythm. Five deaths, all in patients who had ostium primum defects, occurred within 48 hours after operation but were not the result of rhythm disturbances. One child in whom a large patent ductus arteriosus was ligated in addition to closure of the atrial defect died 8 days postoperatively of congestive heart failure.

The appearance of paroxysmal arrhythmia sometimes was delayed. Arrhythmias occurred unpredictably from 3 days to 8 months postoperatively. Atrial flutter with a 1:1 response at a rate of 273 per minute
CARDIAC ARRHYTHMIAS AND REPAIR OF SEPTAL DEFECTS

Fig. 2 Left. Serial tracings of a 28-year-old woman who had intermittent ventricular fibrillation for 120 minutes following closure of an atrial septal defect. Note the extensive T-wave abnormalities after operation. These changes regressed as shown in the tracing 6 months after operation.

Fig. 3 Right. This tachycardia at a rate of 273 per minute occurred on the third postoperative day in a 46-year-old man following repair of an atrial septal defect with anomalous pulmonary venous connections. The rate was halved after digitalis, suggesting that the tachycardia was probably atrial flutter with a 1:1 response.

developed on the third postoperative day in 1 patient who had a modified atrioseptectomy to repair an atrial defect associated with partial anomalous pulmonary venous drainage (fig. 3). Atrial fibrillation appeared 2 weeks after operation in a girl who had an uncomplicated ostium secundum defect. Despite treatment it recurred intermittently and alternated with paroxysmal atrial tachycardia with 3:1 A-V block, a wandering pacemaker, and a nodal rhythm for 3 months before sinus rhythm was re-established and maintained. Two other patients developed short episodes of paroxysmal atrial tachycardia with incomplete A-V heart block 2 to 4 weeks after operation for ostium secundum defects. Neither had received digitalis and neither had other rhythm disturbances. A fifth patient began

Table 1.—Arrhythmias after Repair of Atrial Septal Defects under Hypothermia

<table>
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<tr>
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<tbody>
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<td>Isolated secundum ..............</td>
<td>26</td>
<td>10</td>
<td>7</td>
<td>2</td>
<td>3</td>
<td>1</td>
<td>2</td>
<td>4</td>
<td>1</td>
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<tr>
<td>Ostium primum .................</td>
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<td>9</td>
<td></td>
<td></td>
<td></td>
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<td>7</td>
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<tr>
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<td>3</td>
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<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td>1</td>
</tr>
<tr>
<td>ASD with valvular pulmonic stenosis .......</td>
<td>13</td>
<td>1</td>
<td></td>
<td></td>
<td></td>
<td>1</td>
<td>1</td>
<td></td>
<td>1</td>
</tr>
<tr>
<td>ASD with PDA...............</td>
<td>1</td>
<td>1</td>
<td>1</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td>1</td>
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<tr>
<td>ASD with mitral stenosis........</td>
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<td>1</td>
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<td></td>
<td></td>
<td>1</td>
</tr>
<tr>
<td>Total ........................</td>
<td>62</td>
<td>25</td>
<td>8</td>
<td>2</td>
<td>4</td>
<td>1</td>
<td>3</td>
<td>6</td>
<td>3</td>
</tr>
</tbody>
</table>

ASD, atrial septal defect; PDA, patent ductus arteriosus.
A-V heart block 6 weeks and 5 months after operation. They have not required treatment. Nodal rhythm without tachycardia has been a relatively common finding. It occurred in 5 patients who had no other arrhythmia, and was present intermittently in 3 more who also had other arrhythmias. It has persisted from 1 day to more than 1 year after operation.

The explanation for the supraventricular arrhythmias is unknown. The common denominators have been hypothermia, circulatory occlusion, the incision of the wall of the right atrium, and the placement of sutures for the closure of the atrial defects. There appears to be no correlation with the degree of body cooling, the severity of pulmonary hypertension, or the occurrence of ventricular fibrillation during surgery. Perhaps irritable foci, developing during the healing process of the traumatized atrial tissue, give rise to delayed disturbances of rhythm.

**Ventricular Septal Defects**

Cardiac-bypass procedures were employed in 46 of 48 patients with ventricular septal defects operated on before January 1, 1958. One was successfully repaired under hypothermia alone. The ventricular septal defect was not closed in 1 child, who also had an atrial defect and valvular pulmonary stenosis, although the latter 2 lesions were corrected under hypothermia. Potassium arrest of the

**Table 2.—Arrhythmias after Cardiotomy for Repair of Ventricular Septal Defects (VSD)**

<table>
<thead>
<tr>
<th>Defect</th>
<th>Number</th>
<th>Potassium arrest</th>
<th>Vcntr. fibrill.</th>
<th>A-V heart block</th>
<th>Supraventricular</th>
<th>Atrial</th>
<th>Nodal</th>
<th>Flutter</th>
<th>fibrill.</th>
<th>Deaths</th>
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<tbody>
<tr>
<td>Isolated VSD</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Membranous septum .............</td>
<td>26</td>
<td>3</td>
<td>1</td>
<td>2</td>
<td>5</td>
<td>3</td>
<td>2</td>
<td>Flutter</td>
<td>1</td>
<td>1</td>
</tr>
<tr>
<td>Muscular septum ...............</td>
<td>2</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
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<tr>
<td>VSD with unsupported aortic cusp</td>
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<td>1</td>
<td>1</td>
<td>1</td>
<td>1</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
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<tr>
<td>VSD with tricuspid insufficiency</td>
<td>2</td>
<td>0</td>
<td>2</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>VSD with pulmonary stenosis...</td>
<td>11</td>
<td>4</td>
<td>4</td>
<td>3</td>
<td>3</td>
<td></td>
<td></td>
<td>Fibril.</td>
<td>1</td>
<td>4</td>
</tr>
<tr>
<td>VSD with atrial septal defect and pulmonary stenosis</td>
<td>4</td>
<td>2</td>
<td>2</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td>1</td>
<td></td>
<td>3</td>
</tr>
<tr>
<td>VSD with patent ductus .......</td>
<td>1</td>
<td>1</td>
<td>1</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td><strong>Total</strong></td>
<td><strong>48</strong></td>
<td><strong>11</strong></td>
<td><strong>9</strong></td>
<td><strong>5</strong></td>
<td><strong>9</strong></td>
<td><strong>7</strong></td>
<td><strong>3</strong></td>
<td><strong>2</strong></td>
<td><strong>8</strong></td>
<td></td>
</tr>
</tbody>
</table>
cardiac arrhythmias and repair of septal defects

Fig. 5 Top. Lead I in a 7-year-old boy and (bottom) Lead I on a 4-year-old boy after operation, demonstrating paroxysmal atrial tachycardia at the relatively slow rates of 136 and 139 beats per minute, respectively. These children had ventricular septal defects and infundibular pulmonic stenosis.

Fig. 6 Bottom. Lead II in a 5-year-old boy demonstrating supraventricular tachycardia at a rate of 300 per minute. This may be an example of atrial flutter with a 1:1 response. The patient responded to digitalization.

heart in conjunction with a pump oxygenator was used 11 times.

Ventricular fibrillation occurred in 9 patients during closure of the ventricular septal defects. Sinus rhythm was restored easily in all but 1 who died. This arrhythmia occurred in 8 patients operated on with the pump oxygenator, in 3 of whom potassium arrest also was used. The other patient with ventricular fibrillation, whose defect was repaired under hypothermia, was easily defibrillated. Coronary air embolism was thought to be responsible for ventricular fibrillation in 2 patients with potassium arrest. In one, poor contractions were observed first in an area over the free wall of the left ventricle although the remainder of the heart contracted normally; ventricular fibrillation developed a short time later. Vigorous fibrillatory contractions developed in the area of the left ventricle that had previously been atonic after cardiac massage, and electric countershock re-established regular ventricular contractions.

Eight patients died, 1 from ventricular fibrillation at the time of surgery, and 7 others within 48 hours after operation. One, with an isolated ventricular septal defect and severe pulmonary hypertension (75/36 mm. Hg), and 2, who had an atrial septal defect and pulmonic stenosis as well as a ventricular septal defect, developed complete A-V heart block that persisted until death. Arrhythmias were not clearly related to the deaths of the other 4 patients, although supraventricular
tachycardias at rates of 166, 176, and 180 per minute were observed in 3. However, complete A-V block was not always fatal. It occurred 6 hours after operation in 1 patient, but spontaneously reverted to a sinus rhythm several hours later. Complete A-V heart block with ventricular standstill was observed in a patient with associated infundibular pulmonary stenosis when coronary perfusion was restored following potassium arrest. The block disappeared and sinus rhythm returned when a single suture was removed from the muscular tissue near the anterior margin of the defect.

Most patients had sinus tachycardias for 24 to 48 hours after operation. Rates of 140 to 150 per minute in children have been common. However, ectopic pacemakers were definitely established as responsible for the tachycardia in only 2 patients with heart rates in this range (136 and 139 beats per minute, fig. 5). All tracings in which heart rates were in excess of 160 per minute were examined critically. It was not always possible to determine with certainty whether an arrhythmia originated in an ectopic focus or was an extreme sinus tachycardia. The ectopic origin of the arrhythmia could be demonstrated conclusively in a few instances. Nineteen of 48 patients (39 per cent) had supraventricular tachycardia. Heart rates varied from 166 to 200 per minute except for 2 patients with intermittent paroxysmal atrial tachycardia at rates of 136 and 139 per minute (table 2). Rates slower than 160 with identifiable P waves of normal configuration were not considered to be paroxysmal arrhythmias unless there was electrocardiographic proof of the abrupt paroxysmal nature of the tachycardia. One child with an unusually rapid ventricular rate of 300 per minute probably had atrial flutter with a 1:1 response, although definite proof was lacking (fig. 6). All of the surviving patients who had supraventricular tachycardia and 1 with 2:1 atrial flutter responded to intravenous digitalization. Thus far, none of the patients with repaired ventricular septal defects has developed arrhythmias in the late postoperative period, in contrast to those with repaired atrial septal defects.

**Summary**

Ventricular fibrillation was a frequent complication in the repair of atrial septal defects under hypothermia. It occurred in 25 of 60 patients and was related to the extent of body cooling as well as the length of circulatory occlusion. Restoration of sinus rhythm was accomplished in all but 2 patients who had pulmonary hypertension. Thirty per cent of those patients operated upon successfully for the closure of atrial defects developed supraventricular rhythm disturbances 3 days to 8 months after operation. Supraventricular tachycardias predominated.

Ventricular fibrillation occurred during operation in 9 of 48 patients operated on for ventricular septal defects. Sinus rhythm was restored in all but 1. Coronary air embolism may have initiated ventricular fibrillation in 2 patients. Paroxysmal supraventricular tachycardia responding to digitalization was a common occurrence during the first 24 hours after closure of ventricular septal defects. However, disturbances of rhythm have not appeared later in the convalescent and postoperative period as they have in the patients with atrial septal defects.

**Summario in Interlingua**

Fibrillation ventricular eseva un complicazione frequente in le reparo de defectos atrio-septal sub hypothermia. Illo occurreva in 25 ex 60 patientes e eseva relationate con le extension del frigidation del corpore e tetiam con le duration del occlusion circulatorii. Restauration del rhythmo sinusal eseva complita in omne le patientes con le exception de 2. Iste habeva hypertension pulonar. Trenta pro cento del patientes in qui le opero succedeva a clauder le defectos atrial disveloppava disturbationes de rhythmo supraventricular, 3 a 8 menses post le operation. Tachycardias supraventricular predominava.

Fibrillation ventricular occurreva durante le operation in 9 ex 48 patientes con defectos ventriculo-septal. Le rhythm sinusal eseva
glynn, i. m.: the action of cardiac glycosides on sodium and potassium movements in human red cells. j. physiol. 136: 148 (april 3), 1957.

experiments were done on the fluxes of sodium and potassium across the human red cell membranes to determine whether the glycosides act on the transport mechanism by interfering with the energy mechanism during the "sodium pump" or by interfering with the carrier mechanism in the membrane of the cell. to test whether the action of the cardiac glycosides is one of interference with the transmission of energy to the "pump," experiments were done to study the fluxes in the presence and absence of glucose and with or without the inhibitor, digoxin. the potassium influx was related to the level of external potassium. this was measured by radioactive potassium uptake in red cells. with a fixed external potassium level a given dose of digoxin caused a given degree of inhibition of uptake of potassium. when glucose was removed from the experimental system, the effect of digoxin was unchanged. the efflux of labeled potassium from the cells was not influenced by the presence of glucose or digoxin. the efflux of sodium from cells on the other hand was greatly influenced when digoxin was added to the system. sodium efflux was reduced by 50 per cent in this way, yet the efflux remained unaffected by glucose deprivation. the influx of labeled sodium was reduced 40 per cent when digoxin was added to the external environment, but the influx of labeled sodium was uninfluenced by the absence of glucose in the external environment.

the conclusions drawn from these experiments are that the action of cardiac glycosides cannot be explained by supposing that the drugs interfere with the energy supply to "pump"; their action must interfere with the mechanism by which ions penetrate the membrane. the author further studied the effect of substances of like chemical configuration to the cardiac glycosides on these fluxes. from these experiments it was possible to define certain molecular features that are necessary for such action on ion fluxes. this study shows that the cardiac glycoside competes with potassium ions for a site on the cell membrane where penetration of the ion occurs. from a study of these molecular movements, it is estimated that there are about 1,000 such sites on a red cell membrane.
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