Electrocardiographic Studies during Cardiac Surgery

By E. J. Jaruszewski, Commdr. U.S.N., H. K. Hellerstein, M.D., and H. Feil, M.D.

The electrocardiographic behavior of the heart was observed in 100 patients undergoing heart operations. Most of the arrhythmias were unrelated to the cardiac operative procedure, but as in other thoracic procedures, were related to hypoxia, level of anesthesia, vagal reflexes, and changes in blood pressure. TA and S-T displacements occurred in all groups and were thought to be related to altered dynamics of the right auricle and ventricle. Prevention, recognition and control of disturbances of cardiac mechanism are discussed.

There are excellent observations concerning cardiac mechanism during anesthesia and surgery but there are few on the behavior of the heart during cardiac and cardiovascular surgery. The present report is a record of the behavior of the heart in four common types of surgical procedures: ligation or section of a patent ductus arteriosus, resection of the aorta in coarctation, increasing pulmonary artery blood flow in congenital cyanotic heart disease and resection of pericardial scar in Pick's Disease. The incidence and nature of operative complications are evaluated. Prevention and control of disturbances of cardiac mechanism are discussed.

Methods and Materials

This study is of 100 patients, operated upon by Dr. Claude S. Beck. Forty-five patients had section or ligation of a patent ductus arteriosus. Eight had surgical correction of coarctation of the aorta. Twenty had resection of pericardial scars due to chronic cardiac compression. Twenty-seven had operations designed to increase the pulmonary artery blood flow either by the Blalock-Taussig or by the Potts procedure. The age distribution is recorded in Table 1.

In all cases a medical cardiologist was present throughout the operation. The observer took frequent records and, in addition, watched the electrocardiogram. Changes in the electrocardiogram were correlated with direct observation of the heart, level of the blood pressure and anesthesia, and the operative procedure. All of the patients had preoperative and postoperative electrocardiographic studies. The observations on the four groups of patients are discussed separately.

The preoperative medication was the same in all groups. This consisted of morphine or codeine and atropine. Congestive failure, if present, was treated. Auricular fibrillation or flutter, if present, was controlled by adequate digitalization. Anesthesia was induced by nitrous oxide or Vinethene in all patients except where cyclopropane was used. Ether was employed for maintenance of anesthesia. The Rand positive pressure respirator was employed with intratracheal intubation in all patients.

Patent Ductus Arteriosus

This group consists of 45 patients who were operated for a patent ductus arteriosus. In 44 patients the ductus was either tied or sectioned. In one patient (with a pulmonic systolic murmur and thrill but no diastolic murmur), exploration did not reveal a patent ductus arteriosus.

Cardiovascular Status before Operation. Cardiac enlargement was noted in 27 of 45 cases and was more marked in patients over 15 years of age. Although signs of impaired cardiovascular reserve occurred in several patients in the older age group, digitalis medication was not prescribed for any reason before or after operation. In general, the cardiovascular status of this group of patients was better than that of the cyanotic group, and was comparable with the coarctation group.

Operative Procedure. In the earlier operations the ductus was isolated and doubly ligated. In the later operations, the ductus has been sectioned, and in several cases, an aneurysmal
dilatation at the site of insertion into the aorta was resected and the aortic wall approximated by sutures. There were no deaths in this group.

Medication during Operation. Two patients received repeated doses of atropine during the operation, one because of frequent ventricular beats, and one for its effect on bronchial secretion. One patient received quinidine lactate, 0.30 Gm. intramuscularly, because of multiple premature ventricular beats which appeared when the chest wall was opened. Four patients received intravenous Cedilanid because of rapid supraventricular rhythms (one because of interference dissociation with a rapid ventricular rate at time of closure; one because of sinus tachycardia prior to administration of the anesthetic; one because of the occurrence of supraventricular tachycardia during dissec-

tion of the ductus; and one because of sinus tachycardia). This operation was shorter in duration than the others, averaging 170 minutes, and ranging from 80 to 305 minutes.

Electrocardiographic Changes

Heart Rate. There was a definite trend for the heart rate to change during various stages of the operation. The average rate before the administration of anesthesia was 92 and increased to 129 after induction. A further increase to 139 was noted when the pleura was opened. During dissection of the ductus the average rate increased to 148 and remained in this range until closure of the chest. Surprisingly, no significant change occurred immediately when the ductus was occluded.

Electrical Position. The patients were in the right or semiright lateral posture. In 24 per cent, the heart became electrically more "vertical."

Mechanism. All of these patients had sinus mechanism before anesthesia and at the time of closure the mechanism was again a sinus rhythm in all, although disturbances of rhythm occurred during the operation. In 26 patients, sinus mechanism persisted throughout the entire operation.

Premature Beats. In nine patients premature beats were noted (20 per cent). As shown previously, anesthesia and incision of the chest wall are often accompanied by the appearance of premature beats. Likewise, in this group two patients had premature beats appear during induction of anesthesia, four when the chest wall or pleura was opened, and three when the ductus was being dissected or sectioned. One patient had both auricular and ventricular beats; one, nodal; one, auricular alone; and six, ventricular. The premature beats occurred singly, and not in runs as in the pericardectomy group. The patients receiving cyclopropane induction showed no disturbance of mechanism.

Complex Rhythms. The following arrhythmias occurred in 13 patients: nodal rhythm, interference dissociation, supraventricular tachycardia, cardiac arrest, and ventricular tachycardia. Analysis of the time of their occurrence and relation to surgical procedures reveals that five occurred during induction; four when the chest was being opened; two when the ductus was being opened; one when the ductus was being sectioned; and one when the operation was completed and the chest was being closed. In five patients the arrhythmia was intermittent and transient. In seven patients the arrhythmias persisted throughout the major part of the operation and throughout various phases. It is important to point out that premature beats and significant arrhythmias occurred more commonly before the cardiovascular part of the operation than during it. Similar arrhythmias have been observed during thoracotomies in general.

Cardiac arrest was encountered in a 7 year old boy and was successfully managed. While under ether anesthesia, the patient developed transient nodal rhythm with auricular pre-
mature beats, and spontaneously reverted to sinus rhythm when the chest was opened. Two hours and 25 minutes later, after the ductus had been sectioned, and while the clamp on the pulmonary end of the ductus was being released, sudden cardiac arrest occurred. The duration of the arrest, electrocardiographically recorded, was 75 seconds. The cessation of cardiac activity was recognized immediately. Manual cardiac massage and intracardiac epinephrine restored the heart beat. The following electrocardiographic changes were noted: sinus rhythm, nodal rhythm, auricular premature beats, cardiac arrest, ventricular tachycardia after massage and epinephrine, then interference dissociation for 10 minutes, followed by a short burst of ventricular tachycardia and finally reversion to sinus rhythm. The patient recovered without residual effects.

\( T_A \) and S-T Segment Changes. One of the most striking electrocardiographic changes noted in all cases of patent ductus arteriosus was the occurrence of depression of the \( T_A \) and the S-T segments. This depression occurred in all cases regardless of the level of these segments in the preoperative preanesthetic records. Thus, in 11 cases there was deviation in the preanesthetic records (S-T depression in leads II and III in seven cases, and in leads I and II in four cases). This preanesthetic depression was considered to be consistent with ventricular hypertrophy. Upon the induction of anesthesia, the depression of the S-T and \( T_A \) segments occurred in leads II, III and aVR in 44 cases and in leads I and II in one case. Elevation of the \( T_A \) and S-T segments occurred in the right arm lead, as would be expected.

The degree of S-T depression increased during the operative procedure and showed definite trends. When the pleura was opened and the patient was put on an artificial respirator, the depression became more marked in 17 patients. While the chest wall was being closed, the depression decreased in three patients, became more marked in one, and in the remainder was unchanged. This depression was a transient phenomenon. In one patient it lasted four hours, but in every patient postoperative records showed regression of this finding.

The Influence of the Auricular T Wave on the Level of the S-T Segment. In the course of the above operations, the effect of the auricular T wave on the level of the S-T segment was clearly demonstrated. Since the auricular T wave may last 0.28 second or longer, it influences the level of the S-T segment. In three patients, there was conspicuous \( T_A \) and S-T depression when the mechanism was a regular sinus rhythm. When middle nodal rhythm occurred transiently (no visible P waves), the S-T segment became isoelectric. Similar changes were noted in the cyanotic group (fig. 6). The depression of the \( T_A \) segment indicates an increased negativity of the auricular T wave. This occurs in other conditions in which there is an increased pressure in the atria, especially the right, as in cor pulmonale due to pulmonary fibrosis.

Changes in the QRS Complexes. Changes in electrical position have already been mentioned. The duration of the QRS complexes remained unchanged during the operation in all patients but one, who received quinidine sulfate intramuscularly during the operation because of ventricular premature beats. Electrical alternans of the QRS complexes was recorded in another patient. This developed immediately after ligation of the ductus and diminished gradually as the operation continued.

Coarctation of the Aorta (Eight Patients)

Condition of Cardiovascular System before Operation. Seven of the eight patients enjoyed excellent health, without restriction of physical activity. The oldest patient (age 44 years) had exertional dyspnea, occasional ankle edema, and severe throbbing headaches associated with hypertension in the upper extremities. Her physical condition improved with rest preoperatively. None of the patients required digitalis therapy.

These patients were excellent surgical risks from the cardiovascular standpoint. There was only slight to moderate ventricular hypertrophy as evidenced by changes in the electrocardiogram in two, and by roentgenographic examination in five. All had regular sinus rhythm. Intraventricular conduction was nor-
mal in seven, and of the Wolff-Parkinson-White configuration in one patient.

**Operative Procedure.** The patients were in a semiright lateral position. The coarctation was exposed and resected. In each patient it was possible to make an end-to-end anastomosis. In addition, one patient had a ductus arteriosus.

**Table 2.—Electrocardiographic Changes during Anesthesia and Cardiac Surgery of 100 Patients**

<table>
<thead>
<tr>
<th>Coarctation of Aorta (8 cases)</th>
<th>Pericardiectomy (20 cases)</th>
<th>Total Pericardiectomy Group</th>
<th>Congenital Cyanotic Heart Disease (27 cases)</th>
<th>Patent Ductus Arteriosus (45 cases)</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td></td>
<td>Sinus Mechanism (11 cases)</td>
<td>Auricular Fibrillation or Flutter (9 cases)</td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td>% SM</td>
<td>% AF</td>
<td></td>
</tr>
<tr>
<td>No change in rhythm</td>
<td>(5) 62.5%</td>
<td>(2) 18.1%</td>
<td>(1) 11.1%</td>
<td>(3) 15%</td>
</tr>
<tr>
<td>Displaced Pacemaker*</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Under anesthesia before chest was opened</td>
<td>(1) 12.5%</td>
<td>(1) 9.09%</td>
<td>—</td>
<td>(4) 20%</td>
</tr>
<tr>
<td>At one time or other during operation</td>
<td>(3) 37.5%</td>
<td>(4) 36.3%</td>
<td>—</td>
<td>(4) 20%</td>
</tr>
<tr>
<td>Premature Beats (excludes Ventricular Tachycardia)</td>
<td>(0) 0%</td>
<td>(5) 45.4%</td>
<td>(7) 77.7%</td>
<td>(12) 60%</td>
</tr>
<tr>
<td>Auricular Fibrillation</td>
<td>(0) 0%</td>
<td>(4) 36.3%</td>
<td>—</td>
<td>(1) 3.7%</td>
</tr>
<tr>
<td>Ventricular Tachycardia</td>
<td>(0) 0%</td>
<td>(3) 27.2%</td>
<td>(3) 33.3%</td>
<td>(6) 30%</td>
</tr>
<tr>
<td>Ventricular Fibrillation</td>
<td>(0) 0%</td>
<td>(0) 0%</td>
<td>(1) 11.1%</td>
<td>(1) 5%</td>
</tr>
<tr>
<td>Cardiac Arrest</td>
<td>(0) 0%</td>
<td>(0) 0%</td>
<td>(0) 0%</td>
<td>(0) 0%</td>
</tr>
<tr>
<td>PTa and S-T segment Displacement</td>
<td>(0) 0%</td>
<td>(2) 18.1%</td>
<td>(6) 66.7%†</td>
<td>(8) 40%</td>
</tr>
<tr>
<td>Preoperative</td>
<td>(0) 0%</td>
<td>(6) 54.5%</td>
<td>(7) 77.8%</td>
<td>(13) 65%</td>
</tr>
<tr>
<td>Under anesthesia</td>
<td>(8) 100%</td>
<td>(6) 54.5%</td>
<td>(7) 77.8%</td>
<td>(13) 65%</td>
</tr>
<tr>
<td>Increased displacement when chest opened</td>
<td>(6) 75%</td>
<td>(3) 27.2%</td>
<td>(0) 0%</td>
<td>(3) 15%</td>
</tr>
</tbody>
</table>

* Includes A-V nodal rhythm, interference dissociation, but does not include supraventricular tachycardia, flutter, fibrillation or ventricular tachycardia.
† No arrests since the use of cyclopropane was discontinued.
‡ Refers to S-T depression only, since mechanism was auricular fibrillation.

with a minute patent lumen, and the ductus was ligated and divided. There were no deaths.

**Electrocardiographic Changes.**

Extreme changes of rate, ectopic rhythms, premature beats, and changes in conduction were absent.

**Rate.** As expected, the rate was faster after induction of anesthesia, with an average increase of 30 beats per minute. Case 5 had sinus tachycardia (160) which remained around 150 throughout the operation. There were general trends in rate which apparently were related to the surgical procedures. When the pleura was entered the rate increased; when the clamps of blood. At the close of operation, which lasted from four to nine hours, the heart rate averaged 140 beats per minute, 27 beats more than the rate during induction.

**Mechanism.** Preoperatively sinus rhythm was present in all cases. Sinus tachycardia was observed in all during the operation. In three patients the pacemaker shifted intermittently to the middle or upper A-V node throughout the operation. In one patient this occurred when clamps were applied to the aorta. This was the sole group in which premature beats were absent (table 2).

With the patient in the semiright lateral posture, the electrical position of the heart.
became more "vertical" in four and did not alter in four. In the latter four, two became electrically "vertical" when the left lung was packed down in order to effect exposure of the coarctation. During the rest of the procedure the position remained constant.

Tₐ and S-T Depression. After induction of anesthesia and before chest incision Tₐ and S-T depression in leads II and III appeared in every patient (fig. 1). Furthermore, this depression became more marked in six of eight patients when the pleura was entered. Some degree of Tₐ and S-T depression persisted throughout the operation, and was unrelated to the length of the operation. The postoperative records several days later showed that the Tₐ and S-T segments had returned to the isoelectric level.

Intraventricular Conduction. There was no change in the duration of intraventricular conduction. The patient with Wolff-Parkinson White complexes withstood surgery uneventfully.

Removal of Compression Scars of the Heart (20 Patients)

The records of 20 consecutive patients with constrictive pericarditis were reviewed. These patients were operated upon during the interval from 1944 to 1949. Since the first reports on the behavior of the heart during pericardial resection¹¹ considerable advance has been made in anesthesiology, and cardiac surgical technique. For this reason, a comparison of our results with those in the earlier series done by the same surgeon (Dr. C. S. Beck) is instructive of these advances.

Condition of Cardiovascular System before Operation. Clinically, these patients had a marked diminution of cardiovascular reserve, apparently due to cardiac compression alone in 18 of the 20 patients. In two patients rheumatic mitral valvular disease was present. These 20 patients had cardiac compression for relatively long periods of time (ranging from six months to two years in eight patients, three years in four patients, and five to 13 years in eight patients). Three patients had been subjected to multiple pericardiectomies previously. Eight patients were maintained on digitalis medication in the month preceding operation. Twelve received mercurial diuretics and/or abdominal paracenteses. There were eight patients with auricular fibrillation and one with auricular flutter. Digitalis was given to the patients with auricular fibrillation and to one case with regular sinus rhythm. Intraventricular conduction was less than 0.09 second, in all cases.

Operative Procedure. Operative details will be found in the publications of Beck.²⁵ The

![Fig. 1. Electrocardiograms of J. L. W., case 5, a 10 year old girl undergoing surgical correction of coarctation of the aorta. Records illustrate marked displacement of Tₐ and S-T segments under anesthesia (B), and increased displacement later during the procedure (C). A is the preoperative control record.](http://circ.ahajournals.org/doi/fig/10.1161/01.CIR.14.4.179)
The improved results are related to experience, technic and preoperative preparation. One patient (case 8) developed supraventricular tachycardia with a rate of 210 while the chest wall was being dissected. Within 10 minutes the rate slowed to 125 per minute, and interference dissociation appeared. Cefilanid* (0.8 mg.) was given intravenously, and the rate remained in the vicinity of 125. The mechanism at the end of the operation and for 17 days thereafter was auricular flutter. Reconversion was then effected by the use of quinidine. The other patient (case 15) had auricular flutter with a preoperative ventricular rate of 100 and had not been given digitalis. While under anesthesia, before surgical incision was made the patient developed a ventricular rate of 200 with obvious cardiac embarrassment. Cefilanid (1.6 mg.) was given intravenously, with slowing of the rate to 125 within five minutes. We now advise that all patients with auricular flutter or fibrillation receive preoperative digitalization to prevent the need of digitalization during the procedure.

**Electrocardiographic Changes**

**Rate.** The rate increased with the induction of anesthesia. The average rate was 90 (65–125) before induction, and after induction rose to 121 (75–200). This increase occurred approximately equally in patients with regular sinus rhythm and in those with auricular fibrillation. Unlike the other groups, opening the chest and the pleura was not accompanied by a marked change of heart rate. About one-half of the cases had an acceleration of 10 beats or more when the chest was opened. The rate remained relatively stable during the process of dissection of the pericardium, except for spurts of short-lived tachycardias (to be discussed in detail later). At the close of the operation, which lasted from three to five hours, the average rate was 10 beats or more faster than the induction rate in 10 cases and unchanged or slower in the remaining ten. This is in contrast to the cases with coarctation who had a closing rate 27 beats faster than at induction. The longer duration of the latter operation (four to nine hours) may account for a greater terminal rate. Postoperative records of the patients with pericardiectomies showed a return of the average rate to 91 per minute.

**Mechanism.** Preoperatively, 11 patients had regular sinus rhythm, eight auricular fibrillation and one patient had auricular flutter. For convenience, the first group is designated as group A, and the patients with fibrillation or flutter as group B.

In table 3, the changes in mechanism during various parts of the operation are summarized. In group A during induction of anesthesia and opening of the pleura and pericardium, 4 of 11 patients developed supraventricular arrhythmias. Similar arrhythmias have been noted by Kurtz and co-workers during noncardiac operations. However, during pericardial resection, there was a definite relationship between the manipulation of the heart, traction on the pericardium as it was being dissected free, and the appearance of ventricular premature beats, singly, in pairs, or in runs (ventricular tachycardia). (See figs. 2 and 3.)

During the periods of dissection (group A) auricular fibrillation of flutter was recorded in four patients and nodal rhythm in two patients, making a total of 6 of 11 patients (54.5 per cent) who developed supraventricular arrhythmias. At the time of closure, two patients still had auricular fibrillation or flutter.

The patients in group B with auricular fibrillation developed no significant arrhythmias prior to resection of the pericardium. During pericardial resection, the same relationship between manipulation and the appearance of ventricular tachycardia was also noted in group B.

In this series, one patient (case 14) developed ventricular fibrillation (fig. 3). While an invasive pericardial scar was being dissected from the myocardium ventricular fibrillation developed and persisted for five minutes. The nature of this arrhythmia was recognized immediately, electrocardiographically and by inspection. Within two to three minutes defibrillation was accomplished by the use of electric shock, manual massage and intracardiac adrenalin.27
The immediate mechanism following defibrillation was idioventricular rhythm with a rate of 50 per minute, showing a marked delay of conduction with normal intraventricular conduction. This patient, in retrospect, was the only one in this group whose preoperative electrocardiogram showed premature ventricular beats. Postoperatively, the premature ventricular beats (electrocardiographically of the same origin) persisted. The patient died 18 hours

intraventricular conduction. Subsequently, this changed to auricular fibrillation with a rapid ventricular rate (190) with varying intraventricular conduction and finally to auricular fibrilla-

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<tbody>
<tr>
<td>Group A</td>
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<td>1</td>
<td>RSR</td>
<td>RSR</td>
<td>RSR</td>
<td>ST</td>
<td>AF with RBBB VT</td>
<td>RSR</td>
<td>RSR</td>
<td>AF with RBBB for 5 min. VT less than 1 min.</td>
</tr>
<tr>
<td>2</td>
<td>RSR</td>
<td>Nodal</td>
<td>ST</td>
<td>ST</td>
<td>ST, occasional VPB</td>
<td>ST</td>
<td>RSR</td>
<td>Nodal rhythm occurred before surgical incision.</td>
</tr>
<tr>
<td>4</td>
<td>RSR</td>
<td>ST</td>
<td>ST</td>
<td>ST</td>
<td>ST</td>
<td>ST</td>
<td>RSR</td>
<td>Sinus mechanism, occasional VPB</td>
</tr>
<tr>
<td>5</td>
<td>RSR</td>
<td>ST</td>
<td>ST</td>
<td>ST</td>
<td>ST</td>
<td>ST</td>
<td>RSR</td>
<td>During dissection, paroxysms VT and transient AF</td>
</tr>
<tr>
<td>7</td>
<td>RSR</td>
<td>ST</td>
<td>ST</td>
<td>ST</td>
<td>ST</td>
<td>ST</td>
<td>RSR</td>
<td>Cenidil (0.8 mg) during operation. A flut persisted 17 days. Converted with digitals and quinidine</td>
</tr>
<tr>
<td>8</td>
<td>RSR</td>
<td>ST</td>
<td>ST</td>
<td>ST</td>
<td>ST, occasional VPB</td>
<td>ST</td>
<td>ST</td>
<td>AF permanent postoperative</td>
</tr>
<tr>
<td>10</td>
<td>RSR</td>
<td>ST</td>
<td>ST</td>
<td>ST</td>
<td>ST</td>
<td>ST</td>
<td>ST</td>
<td>Paroxysms VT (9 sec.)</td>
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<tr>
<td>11</td>
<td>ST</td>
<td>ST</td>
<td>ID</td>
<td>A flut</td>
<td>A flut</td>
<td>AF</td>
<td>AF</td>
<td></td>
</tr>
<tr>
<td>12</td>
<td>RSR</td>
<td>ST</td>
<td>ST</td>
<td>ST</td>
<td>Nodal, ST, paroxysms VT, APB, VPB</td>
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<tr>
<td>13</td>
<td>RSR</td>
<td>ST</td>
<td>ST</td>
<td>ST</td>
<td>ST, paroxysms VT</td>
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<td>RSR</td>
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<td>Group B</td>
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<tr>
<td>3</td>
<td>AF</td>
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</tr>
<tr>
<td>6</td>
<td>AF</td>
<td>AF, occasional VPB</td>
<td>AF, many VPB</td>
<td>AF</td>
<td>AF, occasional VPB</td>
<td>AF</td>
<td>AF</td>
<td></td>
</tr>
<tr>
<td>9</td>
<td>AF</td>
<td>AF, occasional VPB</td>
<td>AF, occasional VPB</td>
<td>AF</td>
<td>AF, occasional VPB</td>
<td>AF</td>
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<td></td>
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<tr>
<td>14</td>
<td>AF, occasional VPB</td>
<td>AF</td>
<td>AF</td>
<td>AF</td>
<td>AF, VF, AF</td>
<td>AF</td>
<td>AF</td>
<td></td>
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<tr>
<td>16</td>
<td>AF</td>
<td>AF, occasional VPB</td>
<td>AF, occasional VPB</td>
<td>AF</td>
<td>AF</td>
<td>AF</td>
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<tr>
<td>17</td>
<td>AF</td>
<td>AF</td>
<td>AF</td>
<td>AF</td>
<td>AF, VF, VP</td>
<td>AF</td>
<td>AF</td>
<td>Bursts of VPB (really &quot;brief&quot; VT)</td>
</tr>
<tr>
<td>19</td>
<td>AF</td>
<td>AF</td>
<td>AF</td>
<td>AF</td>
<td>AF, short VT</td>
<td>AF</td>
<td>AF</td>
<td>Uneventful</td>
</tr>
<tr>
<td>20</td>
<td>AF</td>
<td>AF</td>
<td>AF, occasionalVPB</td>
<td>AF</td>
<td>AF, nodal</td>
<td>AF</td>
<td>AF</td>
<td>Short runs of VT near end of dissection</td>
</tr>
<tr>
<td>15</td>
<td>A flut</td>
<td>A flut or ST</td>
<td>AF</td>
<td>AF</td>
<td>AF</td>
<td>AF</td>
<td>AF</td>
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</tr>
</tbody>
</table>

Key

AF = Auricular Fibrillation
A flut = American Flutter
RSR = Regular Sinus Rhythm
ST = Sinus Tachycardia (more than 100)
Nodal = Nodal Rhythm
VPB = Ventricular Premature Beats

APB = Auricular Premature Beats
VT = Ventricular Tachycardia
VF = Ventricular Fibrillation
SVT = Supraventricular Tachycardia
ID = Interference Dissociation
postoperatively because of a bronchopleural fistula.

Electrical Position of the Heart. The patients were in the supine position. Nine of the 20 patients showed no change in the electrical position of the heart during the entire procedure. This may be due to the relatively fixed anatomical position demonstrated preoperatively by roentgenographic and fluoroscopic studies. Four patients exhibited a change in the electrical position during anesthesia or when the pleura was opened. Of these, three became more vertical and one less vertical. In seven patients, the electrical position changed during pericardial resection and was related to manipulation of the heart. When the surgeon was dissecting the left lateral region of the pericardium, the electrical position became less vertical. When dissection was being accomplished on the right lateral region, the heart became more vertical. At the time of closure, the electrical position reverted to the original preoperative value.

$T_a$ and S-T Segment Changes. Six of 11 patients in group A developed $T_a$ and S-T seg-

![Fig. 2. Serial electrocardiograms of J. K., case 14, a 40 year old man undergoing pericardiectomy, demonstrating restoration of an effective cardiac mechanism following ventricular fibrillation. A. Time 9:00 a.m. Control record (lead II) showing auricular fibrillation with an average ventricular rate of 130. B. Time 11:00 a.m. Record shows ventricular fibrillation. While the invasive scar was being separated from the myocardium, the heart dilated acutely, became cyanotic, and ventricular fibrillation occurred. C. Time 11:05. One minute after the first electric shock. Ventricular fibrillation persists but some complexes are beginning to have form. D. Time 11:15. After the second shock and intracardiac adrenalin an idioventricular rhythm (rate 51) occurred with no visible or electrical evidence of auricular activity. An occasional premature ventricular beat is present. E. Time 11:20. Idioventricular rhythm persists. At 11:25 auricular fibrillation with an average ventricular rate of 187 appeared. The operation was completed. F. 12:35 Time of closure. Auricular fibrillation persists.](image)
leads II and III, while one patient showed marked elevation in lead I and depression in lead III, similar to the injury patterns observed in injury to the anterior wall of the myocardium. The changes in the last patient were transient, and were again transiently observed during a second pericardiectomy. At this time, the changes occurred when the distal end of the anterior descending coronary artery was nicked. Serial postoperative records revealed no evidence of a myocardial infarction. The S-T segment displacement and T-wave inversion which occurred postoperatively was considered to be due either to pericarditis or digitalis therapy.

Changes in the QRS complexes. In this series, the intraventricular conduction time was remarkably constant throughout the procedure. Of the entire group, only two cases had a temporary increase in conduction time. Case I developed right bundle branch block during a period of transient auricular fibrillation and case 14, following the episode of ventricular fibrillation, had a temporary prolongation of the intraventricular conduction time when the mechanism was restored to auricular fibrillation. Both of these patients had normal intraventricular conduction time at the end of the operation.

Amplitude of QRS Complexes. Low voltage of the QRS complexes is found commonly in constrictive pericarditis and is thought to be due to the shielding effect of fibrous pericardial scar and fluid. Since only 25 per cent of our patients had increase of amplitude postoperatively or at the time of discharge from the hospital, it is probable that the low voltage preoperatively is due to both shielding by the fibrous scar and the disuse atrophy of the myocardium. Some of our patients showed delayed increase of voltage, some as late as one year after operation (fig. 4). This would imply that there had been an increase in the muscle mass and electrical potentials and is consistent with the concept that atrophy is reversible.

Cyanotic Heart Disease with Deficient Pulmonary Circulation (27 Patients)

Twenty-four patients had the classic features of the tetralogy of Fallot, one had isolated dextrocardia with pulmonic stenosis, interventricular septal defect and dextroposition of the aorta; one had the pentalogy of the Cuban school (tetralogy of Fallot with an intrastrial septal defect and left ventricular hypertrophy), and one patient had a probable truncus arteriosus.

Condition of Cardiovascular System before Operation. Clinically these patients had cyanosis and marked limitation of physical activity. The diminution of cardiovascular reserve was indicated by voluntary or imposed restriction of activity and exertional dyspnea. However, none of these patients had signs of congestive heart failure or were receiving digitalis. Although surgical intervention is advocated for patients over 2 years of age, in our series three patients below 2 years of age were operated upon because of serious underdevelopment and nutritional failure.

Operative Procedures. In 16 patients, a subclavian-pulmonary artery anastomosis was performed, in eight patients an aortic-pulmonary anastomosis and in the patients exploratory thoracotomy alone was the procedure. In the last group, one patient had severe pulmonic stenosis with poststenotic aneurysmal dilatation of the left pulmonary artery. In
the other two patients a pulmonary artery could not be demonstrated at the time of operation. In each the aorta was extremely large.

Anesthesia. In the earlier part of this series, cyclopropane was used alone in nine cases, and together with ether in eight cases. In the last 10 cases of this series (and routinely in subsequent cases) ether was employed alone or supplemented by nitrous oxide or Vinethene induction. There were 10 deaths in the cyclopropane groups and none in the ether group. All six instances of cardiac arrest occurred in the cyclopropane group. The high incidence of cardiac complications in cyclopropane anesthesia has been noted by others.16,31–34 The mortality rate of the last 10 cases was 10 per cent, and in the subsequent 30 cases, it has remained 10 per cent. The reduction is attributed to the use of ether, atropine, positive pressure respirator, rest periods, and experience of surgeon and anesthetists.

Medication during Operation. The need for repeated doses of atropine during the operation is demonstrated by the observation that cardiac arrest occurred on an average of three to four hours after the preoperative dose of atropine, when its effect had worn off. In six of the last 10 patients, repeated injections of atropine were given. There were no deaths. The indications for additional atropine consisted of the appearance of (1) nodal rhythm, (2) interference dissociation and (3) marked slowing of the sinus rate. These changes were usually related to the opening of the chest wall, retraction and dissection of the pulmonary artery, and retraction of the lungs (figs. 5, 6, 7 and 8). The response to intravenous atropine was immediate, with restoration of regular sinus rhythm and an acceleration of a previously slow rate.

Cardiac Arrest. As mentioned, there were six cases of cardiac arrest; one survived. Electrocardiographic changes before, during and after cardiac standstill are listed sequentially in table 4. In four patients, arrest occurred when the pulmonary artery or aorta was being dissected, or the anastomosis being made. In one patient, cardiac arrest occurred during the incision of the skin, and in another, after the chest had been entered and the lungs were being retracted. In these cases, cardiac arrest was probably produced by reflexes from the operative field, with inadequate protection by atropine administered several hours previously. In each instance sinus mechanism was restored, even though the period of recorded cardiac arrest varied from 33 seconds to five minutes. Three patients died within three hours after completion of operation, two within 24 hours, and one survived without residual effects.

Electrocardiographic Changes

In this group there were more disorders of the heart beat than in operations on patients with
noncyanotic congenital heart disease or constrictive pericarditis. In the latter group, the wave, or "extracardiac"—that is, on the great vessels. More likely alteration of pulmonary pressures incident to inhalation anesthesia and to compression of the lung or pulmonary artery in effecting the anastomosis would increase the right to left shunt and thus the degree of hypoxia.

Fig. 6. Serial electrocardiograms of D. P., case 19, a 15 year old boy undergoing surgery for congenital cyanotic heart disease. The records illustrate the effect of the auricular T wave on the level of the S-T segment. A is the control record (lead aVF) after induction of ether anesthesia, showing sinus mechanism with depression of the T\textsubscript{A} and S-T segment. B, C, and D are continuous records. In B there is a gradual displacement of the pacemaker with the previously depressed S-T segment returning to an isoelectric level. In C nodal rhythm is present with isoelectric S-T segments. In D there is a gradual return of sinus mechanism. Because of the effect of the negative auricular T wave, the S-T segment again is depressed below the isoelectric level.

Fig. 7. Serial electrocardiograms of K. A. B., case 6, an 11 year old girl undergoing surgery for congenital cyanotic heart disease. Complex rhythms unrelated to the surgical procedure itself occurred. A, time 8:40 a.m. Control record (lead 2) under light anesthesia, showing complex arrhythmia, dissociation by interference. B, Time 8:50. P waves are more prominent. There is a sinus rhythm with parasympathetic foci. C, Time 1:10 p.m., an hour after completion of Potts operation. Sinus mechanism resumed spontaneously.

Fig. 8. Electrocardiograms of K. A. B., an 11 year old girl undergoing surgery for congenital cyanotic heart disease, illustrating the effect of anesthesia on the T\textsubscript{A} and S-T segments, and the transient nature of the displacements. A is the preoperative control record. B shows marked T\textsubscript{A} and S-T depression during ether anesthesia. C. Postoperative record showing minimal displacement.

changes in rhythm were due to cardiac manipulation. In the cyanotic group the disturbances were related to hypoxia and less to direct causes since the operations were essentially

Rate. The average preanesthetic heart rate was 116 per minute, which was higher than in the other (older) groups. After induction of anesthesia there was an increase to an average rate
of 130. During the period between the opening of the pleura and dissection of the pulmonary and subclavian arteries and/or aorta, there was a further gradual increase to 140. The maximum average rate (151) occurred when the anastomosis was made. Upon closure of the chest, the rate decreased to an average of 141 per minute.

Mechanism. Preoperatively all patients had sinus rhythm. In addition to the six patients with cardiac arrest, the mechanisms during the dissection of the great vessels in four cases, and at the time of anastomosis in two cases.

There was a similar tendency for nodal rhythm to occur more frequently during induction of anesthesia and incision of the chest wall (five cases) than during surgery of the great vessel (three cases). In only two of the eight patients, did nodal rhythm persist to the time of closure. Atropine was administered intravenously in four patients and re-

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**Table 4.**—Sequence of Electrocardiographic Changes Preceding, During, and After Cardiac Standstill (Six Patients)

<table>
<thead>
<tr>
<th>Case 1. E. R.</th>
<th>Nodal tachycardia, cardiac arrest (complete standstill), multifocal ventricular beats, paroxysms of ventricular tachycardia with standstill, sinus tachycardia with right bundle branch block, sinus tachycardia with normal intraventricular conduction.</th>
</tr>
</thead>
<tbody>
<tr>
<td>Case 2. J. Y.</td>
<td>Sinus tachycardia, sinus rhythm with delayed intraventricular conduction and prolonged P-R interval, sinus bradycardia, complete cardiac arrest (standstill), multifocal ventricular beats in bursts, idioventricular rhythms, rate 70, sinus tachycardia (150), sinus bradycardia with aberrant ventricular conduction, 3:2 S:A conduction, cardiac arrest, sinus tachycardia (150), delayed ventricular conduction, short paroxysms of ventricular tachycardia, electrical standstill, ventricular escapes, idioventricular rhythm sporadic, paroxysms of idioventricular beats, ventricular tachycardia, sinus tachycardia (150), cardiac standstill, coupled idioventricular beats, chaotic heart action, standstill, idioventricular isolated beats, standstill, idioventricular escapes, ventricular tachycardia in paroxysms, nodal rhythm, sinus rhythm with right bundle branch block, sinus rhythm with normal intraventricular conduction.</td>
</tr>
<tr>
<td>Case 3. P. R.</td>
<td>Nodal rhythm with frequent ventricular premature beats, auricular fibrillation with right bundle branch block, auricular standstill with ventricular escapes, complete cardiac standstill, ventricular escapes in pairs and threes, ventricular tachycardia, supraventricular tachycardia, sinus tachycardia.</td>
</tr>
<tr>
<td>Case 12. K. K.</td>
<td>Sinus tachycardia, ventricular tachycardia, cardiac arrest, sinus tachycardia.</td>
</tr>
<tr>
<td>Case 13. O. B.</td>
<td>Sinus tachycardia, cardiac arrest, sinus tachycardia.</td>
</tr>
<tr>
<td>Case 24. P. W.</td>
<td>Sinus tachycardia, interference dissociation, idioventricular rhythm with occasional sinus activity, cardiac standstill, ventricular escapes with irregular sinus activity and short periods of sinus recapture, sinus bradycardia (50), idioventricular rhythm with occasional conducted sinus beat, short bursts of sinus recapture, ventricular fibrillation.</td>
</tr>
</tbody>
</table>

operation were as follows: (a) six patients showed sinus rhythm alone, (b) six patients had sinus rhythm with auricular, nodal or ventricular premature beats, and/or short runs of ventricular tachycardia, (c) eight patients had sinus rhythm with periods of nodal rhythm, and (d) one patient had transient interference dissociation.

Premature beats were recorded in 9 of 21 patients; ventricular in six, auricular in two, and nodal in one. They occurred when the pleura was first entered in four cases, during stored the mechanism to a sinus rhythm. Interference dissociation likewise was transient, occurring during induction of anesthesia, and often spontaneously disappearing when the pleural space was entered. Apparently the appearance of nodal rhythm, interference dissociation and premature beats were causally related to the anesthetic more than the surgical cardiac procedure, since most occurred before the heart was manipulated.

**Tₐ and S-T displacement.** Displacement of the Tₐ and S-T segment was greater than in
the other groups (fig. 8). It occurred during the induction of anesthesia, and had a tendency to become more marked when the pleura was entered and the great vessels were isolated. Depression occurred in leads II, III, aVF, and elevation in lead I and aVR. In eight cases with depression of the S-T segment in preoperative records, additional S-T and TA displacement occurred. Postoperatively, these displacements disappeared, again illustrating the evanescent nature of this charge. Postoperatively, ST-T changes of pericarditis occurred in three patients.

QRS Complexes. In three cases there was delay of right intraventricular conduction during the anastomosis.

DISCUSSION

In the course of cardiac or noncardiac surgical procedures, a majority of patients will show some disorder of the heart beat.\textsuperscript{15, 16, 19, 20} Such abnormalities (premature beats, tachycardia, displaced pacemakers) are usually transient and unaccompanied by clinical signs of embarrassment of the circulation. However, early recognition of significant arrhythmias and other electrocardiographic changes may forestall serious complications.

There were common features in all groups which were usually unrelated to the cardiac surgery, but were related to thoracotomy, anesthesia, hypoxia, and reflexes. These included changes in heart rate, electrocardiographic positional changes, displacement of the pacemaker, and TA and S-T segment displacements.

A progressive increase of heart rate was noted in all four groups as the operation continued. The rate decreased at the end of the operation. This was true regardless of the type of anesthesia used.\textsuperscript{36} In the younger patients the increase of heart rate was greater than in the older patients. In patients under 3 years of age, when the heart rate fell below 125, there was a decrease in blood pressure, and an increase of cyanosis. It is important to correlate the rate under anesthesia with the preanesthetic rate.

The relative innocuousness of rates of 150 per minute in the young should be recognized. Therapy intended only to slow the heart rate, especially at the end of an operation, should be discouraged. In some of our younger patients, 6 to 12 hours have elapsed before the rate returned to lower levels.

Apprehension and fear before operation are occasionally responsible for sinus or supraventricular tachycardia. Mousel\textsuperscript{37} has stressed the importance of preoperative reassurance of the patient.

Mechanism. In our series there was a frequent disturbance of the cardiac mechanism during induction or maintenance of light anesthesia, before the surgical procedure was begun. The most common was displacement of the pacemaker, that is, shift of the pacemaker within the sinoauricular node, nodal rhythm, or interference dissociation. This is in agreement with the reports of others.\textsuperscript{15, 16, 19, 38, 39} We have noted also that at the end of surgery, with the lightening of anesthesia,\textsuperscript{38, 40} nodal rhythms were fairly common.

That anesthetic agents themselves occasion the occurrence of premature beats is generally accepted in that they frequently occur prior to the initiation of surgical procedures. However, it has been difficult to determine whether the incidence of premature beats is related to the type of anesthetic agent. In the 80 patients (exclusive of the pericardectomy group) the over-all incidence of premature beats was 25 per cent (20 cases). Although the number of patients who had cyclopropane anesthesia was relatively small (22 patients), there was no significant difference in the incidence of premature beats in the groups receiving cyclopropane and ether, 27.2 and 24.1 per cent, respectively. This is at variance with the observations of others.\textsuperscript{31, 34} However, in the cyanotic group there was a significant increase in the number of premature beats regardless of the anesthetic used. Hypoxia or other factors are more important than the anesthetic agent in the production of premature beats.

Frequent ventricular premature beats or bursts of ventricular tachycardia are significant enough to warrant temporary interruption of the surgical procedure. This is especially true in pericardectomy and more recently in mitral and pulmonic valvular surgery. Exclusive of the pericardectomy group, we have
found that the occurrence of premature beats during anesthesia does not indicate that they are forerunners of ectopic rhythms. If premature beats are present preoperatively, they are more likely to be forerunners of ectopic rhythms (case 14).

**Displacement of Tₐ and S-T Segments.** A constant effect of inhalation anesthesia on the electrocardiogram was the downward displacement of the S-T segment in leads II, III, aVₑ, and the upward displacement in aVₑ. In many instances there was an increase in the amplitude of the P wave. These changes were noted prior to any significant increase in heart rate and before the operation. There is good evidence that alterations of the cardiopulmonary dynamics may account for most of these changes, which suggest "strain" of the right auricle and right ventricle. Peripheral venous pressure, right atrial and right ventricular pressures have been shown to be elevated by inhalation anesthesia and by positive pressure, controlled or supported respiration. Other procedures which increased the "strain" on the right heart included opening the pleura and packing down the lungs, and temporary occlusion of a pulmonary artery, as during the Potts operation. Increased displacement of the Tₐ and S-T segments occurred at this time. Hypoxia may also account for these displacements. Lindgren and Ohnell noted that depression of the S-T segment occurred during periods of hypoxia and that an increase of oxygen supply was generally followed by partial or complete return to isoelectric levels. Since acute hypoxia produces pulmonary hypertension, the Tₐ and S-T changes may be due to the increased right heart "strain" secondary to hypoxia, or due to subendocardial ischemia.

**Effects of the Surgical Procedure.** The disturbances of cardiac mechanism in nonthoracic surgery are similar but less frequent than in our series. Intrathoracic procedures affect cardiac behavior. For example, the effects of reflex vagal stimulation were commonly noted during certain procedures in all groups. This occurred (1) when the pleura was entered, (2) ribs retracted, (3) the lungs packed away, (4) the hilar vessels dissected, and (5) during dissection in the proximity of the vagi. We have noted that each of the above procedures frequently resulted in sudden bradycardia, a fall of blood pressure, and displacement of the pacemaker or appearance of ventricular premature beats. In each instance, the situation was corrected by intravenous atropine and reinflation of the lungs. As our experience increased, atropine was administered prophylactically in anticipation of the complications just referred to. It is important to be alert for predominant vagotonic effects throughout the surgical procedure and especially near the end of the operation. In many cases, we have noted a reflex vagal effect when the chest wall was being closed. Each time the rib cage was approximated by another suture, there was a sudden drop in pulse rate. Such changes in rate, actually signs of impending cardiac arrest, were readily abolished by atropine.

In the cyanotic patients, the surgical procedure had an especially important effect on cardiac behavior. Serious disturbances such as arrest were apt to occur when a pulmonary artery was partially occluded in order to effect an anastomosis. During this time there is a greater shunting of blood from the right to the left circulation, increasing the systemic hypoxia. Here hypoxia appears more important than vagal influence alone. The potentiation of vagal action or acetylcholine by hypoxia may account for the high incidence of complications in this group.

The pericardietomy group illustrates that manipulation, traction, and displacement of the heart were responsible for the appearance of premature beats and bursts of tachycardia. The greatest incidence of arrhythmias occurred when the lateral aspects of the pericardial scar were resected, necessitating rotation, angulation, and dislocation of the heart. Similarly, manipulation and traction encountered during mitral valvular surgery in our own and others' experience invariably produces ectopic beats and frequently tachycardia.

The ideal anesthetic has not yet been attained for intrathoracic cardiovascular surgery. Cyclopropane and ether are most extensively used. Originally cyclopropane was heralded
as an ideal anesthetic for patients with heart disease because of the high concentration of oxygen administered with it. Actually, with modern technics, an equally high or higher concentration of oxygen can be administered with ether. Most observers1, 2, 16, 31, 34 agree that there is a greater tendency for serious arrhythmias to occur under cyclopropane anesthesia. More recently,35 cyclopropane anesthesia has been modified by the addition of ether, or continuous intravenous procaine. Furthermore, since vagal activity is not depressed by cyclopropane, atropine should be given in addition. The role of the vagus during cyclopropane anesthesia is demonstrated by the abolition and prevention of arrhythmias by large doses of atropine. In our series, most of the serious complications, such as cardiac arrest (a manifestation of excessive vagal activity), occurred under cyclopropane anesthesia. On the other hand ether causes depression of vagal activity and relative dominance of the sympathetic system34 and appears safer, if properly administered.

In view of the divergent reports,7, 48, 49, 50 equally enthusiastic and condemnatory, it would appear that the most important thing is the selection of the anesthetic with which the anesthetist is most familiar. The maintenance of oxygenation and the recognition of danger signs with cyclopropane, and the transfer to another anesthetic (ether) in such instances would constitute a reasonable compromise. Our own preference is ether.*

In considering the management of the patient during the operation, a routine technic for the constant observation of the state of the circulation should be emphasized. The direct writing electrocardiograph and more recently the cathode ray electrocardiograph provide continuous information on the cardiac mechanism. Direct observation of the heart is also valuable in advising therapy and in the timing of rest periods. The ear oximeter35 facilitates the earlier detection of hypoxia, and should be used routinely. The ultimate purpose of these modern electronic adjuncts is to maintain the patient in a surgical anesthetic state, well oxygenated, protected from serious reflexes (atropine, procaine) and with a normal cardiac mechanism and blood pressure. Hypotension is preferably controlled by blood and saline, supplemented by pressor drugs when necessary. Since ventricular fibrillation or cardiac arrest may occur in spite of the above precautions, the operative team should be well-trained and equipped for restoration of the heart beat.8, 9, 10, 13, 26

**Summary**

The electrocardiographic behavior of the heart was observed in 100 patients during anesthesia and during cardiac operations.

Arrhythmias observed during cardiac operations are usually unrelated to the cardiac operative procedure, but are related to the type and level of anesthesia, hypoxia, and reflexes mediated through the vagus. The incidence of arrhythmias in surgery of the heart is about the same as that encountered in thoracic surgery in general. Ventricular fibrillation and cardiac standstill occurred in eight cases. Restoration of cardiac mechanism was affected in six with survival of two patients.

Displacement of the T_a and S-T segments occurred frequently in all groups, with depression in leads II, III, aV_F, and elevation in aV_R. These deviations are thought to be due to changes in the dynamics of the right side of the heart incident to anesthesia and to hypoxia.

There were few significant changes during anesthesia and operation for coarctation of the aorta and for patent ductus arteriosus. Ventricular premature beats, singly or in runs, were related to cardiac manipulation, traction, and resection of pericardial scars. More serious complications occurred in the congenital cyanotic group. The incidence of complications decreased since cyclopropane was replaced by ether.

Rest periods during the operation, reinflation of the lungs, and atropine are important in the prevention of serious complications. The recording and observation of continuous electrocardiograms should be supplemented by direct observation of the heart itself.

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* Recently a combination of procaine, nitrous oxide and Thiental was found satisfactory in mitral commissurotomy.49
**Sumario Español**

El comportamiento electrocardiográfico del corazón se observó en 100 pacientes durante cirugía cardíaca. La mayoría de las arritmias no fueron relacionadas al procedimiento operatorio cardíaco, pero, como en otros procedimientos torácicos, se debieron a hipoxia, nivel anestésico, reflejos vagales, y cambios en presión arterial. Desplazamientos de la T y ST ocurrieron en todos los grupos y se creyeron debidos a los cambios dinámicos alterados de la aurícula y ventrículo derecho. La prevención, reconocimiento y control de estos desordenes del mecanismo cardíaco se discuten.

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E. J. JARUSZEWSKI, H. K. HELLERSTEIN and H. FEIL

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