Chronic Cardiac Compression (Chronic Constrictive Pericarditis)

A Critical Study of Sixty-one Operated Cases with Follow-up

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Sixty-one patients with chronic cardiac compression were studied critically, the largest series in the literature. All patients had pericardiectomy performed. The operative mortality was 18 per cent. Satisfactory result was obtained 72.1 per cent of the patients operated. Tuberculosis was the cause in 28.2 per cent. In most of the remaining cases the etiology was obscure. The symptoms, physical signs, and laboratory examinations are reported. The physiologic studies after operation demonstrate improved cardiac filling and emptying.

The importance of diagnosis and the desirability of pericardiectomy are stressed. With antibiotic therapy there is less risk in operating patients with active infection and the compression syndrome.

In the past 20 years the many cases of chronic compression operated at University Hospitals of Cleveland offered an opportunity for careful study. This report is based upon 61 operative cases from the service of Dr. C. S. Beck, with a study of pre- and post-operative observations. To our knowledge, this is the largest series ever reported.

Chronic cardiac compression occurs as the result of a contracting pericardial scar which encroaches upon the diastolic filling and the systolic emptying of the heart, thereby reducing output. Usually the entire pericardium is converted into a compressing, nondistensible sac. Occasionally a localized fibrous band constricts the inflow or outflow tract of either ventricle. This is the basic pathologic and physiologic disturbance causing the various clinical and laboratory findings.

Excellent historical reviews have been written by White, Blalock and Burwell, Heuer and Stewart, Armstrong, Beck, and others. The reader is referred to these papers.

Beck\(^8\), \(^7\) evolved the concept of "chronic cardiac compression." In 1935 Beck\(^8\) presented the two diagnostic triads of cardiac compression: the acute (low arterial pressure, high venous pressure, and a quiet heart) and the chronic (high venous pressure, ascites, and a quiet heart). In a review in 1939 Beck\(^8\) found satisfactory results in 65 per cent of the cases operated by 51 different surgeons.

Incidence. Clinically and pathologically, chronic cardiac compression must be considered an uncommon disease, although chronic adherent pericarditis is present in about 2 per cent of all autopsies. Cabot,\(^10\) in 114 cases of adherent pericarditis in 4,000 autopsies (2.8 per cent), found only one case of Pick's disease. Smith,\(^11\) in 62 cases of chronic adherent pericarditis in 3,053 autopsies (2.03 per cent), found three cases which could be considered chronic cardiac compression. Sprague,\(^12\) in 1900 autopsies, found 43 cases of pericardial adhesions of which 25 showed complete obliteration of the pericardial sac, and not one case of Pick's disease. Wells\(^13\) found adherent pericarditis in 57 of 1048 autopsies. Armstrong\(^4\) reported 114 cases of complete adherence of the pericardium, 10 of which showed constriction. Essentially a disease of young men, the incidence among men in our series (78.7 per cent) is similar to that of Paul and associates\(^14\) and of Harrington.\(^15\)
CASES IN THIS STUDY

All cases here reported were treated surgically and a definite pericardial scar was found in each. The 61 patients were operated 69 times.

Follow-up data has been obtained on all but two. The data was gathered through re-examination when possible, questionnaires from the patients, their immediate families or their personal physicians and reports from various clinics.

Age and Sex. The average age was 31.7 years ranging from 6 to 54 years. Forty-three (70.1 per cent) were between 20 and 49 years with the greatest number, 16, in the decade 30 to 39. Forty-eight of the patients were males (78.7 per cent).

HISTORY

Primary Complaints. Dyspnea, ascites, edema, orthopnea, or a combination of these and, in a few instances, fatigue, were primary complaints. Some had had previous diagnoses of nephrosis, cirrhosis of the liver, chronic nephritis, and cardiac decompensation.

The average duration of the primary complaint was two and a half years. Twenty-four cases had symptoms for one year or less, 24 had symptoms for two or more years and of this latter group, seven had symptoms for eight or more years.

Accepting dyspnea, ascites, edema, and orthopnea as the most common symptoms of chronic cardiac compression, the cases were analyzed to determine how often each was the first symptom. Dyspnea led in 31 cases (50.7 per cent); 21 (34.5 per cent) reported abdominal swelling as the first symptom; eight (13.1 per cent) reported edema; and one (1.6 per cent) reported orthopnea.

Past History. Only four had a history of tuberculosis. Subsequently, this proved the cause of their compression. Ten patients had a history of acute rheumatic fever, preceding the symptoms of compression by three months to 29 years.

Symptoms

Dyspnea. The most common symptom was dyspnea, occurring in 54 cases (88.5 per cent). Varying in severity from mild to completely incapacitating, it was usually moderately severe. The average duration of dyspnea was 28.3 months, ranging from two weeks to 156 months.

Abdominal Swelling. The next most common symptom was abdominal swelling, occurring 49 times (80.5 per cent), and varied from slight to very marked. The average duration was 28.7 months, ranging from 1 to 144 months.

Edema. Forty-three patients (70.5 per cent) gave a history of peripheral edema of moderate severity with the average duration of 15.1 months, ranging from 1 to 144 months.

Orthopnea. The least common of these four symptoms was orthopnea, occurring in 21 cases (34.5 per cent). The average duration was 21.5 months with a range of 1 to 120 months.

Dyspnea and abdominal swelling were the most frequent symptoms, and the symptoms of longest duration. Edema occurred almost as frequently but was of slightly shorter duration. Orthopnea occurred in fewer cases with still shorter duration. In view of the fact that orthopnea is generally considered an uncommon symptom of chronic cardiac compression, note that it occurred in 34.5 per cent with an average duration of 21.5 months.

Fatigue was quite common and in a few instances was incapacitating. Other symptoms of some severity were, in one, dysphagia; in two, cough; in five, substernal discomfort and palpitation; and in two, swelling of the face.

Paracenteses. Thirty-nine patients had some type of paracentesis prior to operation, varying in individual cases from 1 to 55 taps. One had 53 pericardial paracenteses in one year. Following the instillation of gentian violet into the pericardium, the necessity for paracentesis ceased. During this period he developed signs and symptoms of chronic cardiac compression. In the next six years this patient required only two paracenteses.

Only four cases required thoracenteses alone; 16 had thoracenteses in combination with abdominal or pericardial taps.

PHYSICAL EXAMINATION

In general, the patients were well developed and moderately well nourished. A few were described as "chronically ill." Twenty-four (39.3 per cent) had some degree of cyanosis, varying from slight to severe. Of these, five (20.8 per cent) died postoperatively. Almost
one-half of the patients who died in the hospital were cyanotic preoperatively. Two showed very slight icterus on admission.

**Vital Signs.** In general the temperature was normal. Eight patients had a preoperative temperature of 38°C. or above. Of these, only one died while still hospitalized, five months after operation. Four of these febrile cases proved later to have tuberculous pericarditis. One patient had staphylococcal mediastinitis.

The average resting pulse rate was 91 with a range of 60 to 124. Twenty-four cases had a pulse rate in excess of 100.

The average systolic blood pressure was 112 mm. Hg with a range of 80 to 150 mm. Eleven patients had a systolic pressure below 100 mm.
The average diastolic pressure was 80 mm. with a range of 60 to 110 mm. There were seven with a diastolic pressure of 100 mm. or more. The average pulse pressure was 33 mm. with a range of 10 to 70 mm.

**Venous Distension.** Distension of the neck veins was a universal finding, generally prominent, and frequently associated with distension of the veins of the forehead, arms, and chest.

**Cardiac Examination.** A description of precordial activity was given in 58 cases. In 18, the precordial activity was absent on inspection and palpation. In 26, it was decreased. Thus, in 48 cases (78.7 per cent) there was decreased precordial activity. In only one case was a systolic retraction of the intercostal spaces described (Broadbent’s sign). An apical systolic murmur was heard in four and a basal systolic murmur in one. In one, a pericardial friction was heard associated with staphylococcal mediastinitis. The character of the heart sounds was normal in 24, distant in 35, and almost inaudible in two cases. A paradoxical pulse was described in eight.

**Abdominal Examination.** Distension of the abdomen with ascites was a common finding. Some degree of ascites was present in 52 cases (85.3 per cent), but only 27 patients required abdominal paracentesis. In 17, the abdominal veins were distended.

The spleen, palpable in six, was usually at the costal margin. Umbilical hernias occurred seven times and in three, inguinal hernias were present. All of these hernias were associated with ascites. The liver was enlarged and palpable in all but one patient. In this patient, the abdomen was tensely distended with fluid and palpation was unsatisfactory. The liver edge was usually palpated at the umbilicus, the iliac crest, or below the iliac crest.

**Peripheral Edema.** Edema was present in 41 (67.2 per cent). Of the remaining cases, ascites was present in 17. Six cases had edema without ascites. Thus, ascites and/or edema was present in 58 (95 per cent).

Varicose veins with or without ulcers were present in 16 (26.2 per cent).

**Laboratory Studies**

Routine urine analysis revealed no consistent abnormality. Fifteen patients showed a trace of albumin at times. Only two had a hemoglobin of less than 70 per cent and only six had a red blood cell count of less than 4 million, the lowest being 3.3 million. The average white blood cell count was 6,870 per cu. mm. with a range of 4,300 to 13,300. There were only four with an initial white count of 10,000 or more. The differential blood smears were all within normal limits, and only one patient had serologic evidence of syphilis. In 52, the average blood urea nitrogen was 12.2 mg. per 100 cc. with a range of 6.0 mg. to 22.5 mg.

**Serum Proteins.** The total serum proteins were determined in 44 instances. The average value was 6.01 Gm. per 100 cc. Six patients had total serum protein of less than 5 Gm. per 100 cc. of blood. All had ankle edema. The serum albumin was determined in 39. The average was 3.46 Gm. per 100 cc. of blood. Eleven patients with edema had less than 3 Gm. The serum globulin was determined in 39 patients, the average being 2.61 Gm. per 100 cc. The albumin-globulin ratio was 1.0 or less in only eight patients.

Analysis of our data of total serum proteins showed that in the cases without edema it was 1 Gm. higher than in the group with edema. In the group without edema there were no cases with a total protein less than 5 Gm. per 100 cc. or a serum albumin less than 3 Gm. In the group with edema 21.4 per cent had a total protein less than 5 Gm. per 100 cc. and 57 per cent with albumin less than 3 Gm. per 100 cc.
Sedimentation Rate. The preoperative sedimentation rates were recorded in 37; nine determinations were 20 mm. or more in one hour (normal 10 mm. in one hour). Rapid sedimentation rate may be evidence of disturbed liver function. The sedimentation rate was inconclusive evidence of active infection.

Circulatory Studies

The preoperative circulatory status was evaluated on the basis of pulse rate, pulse pressure, vital capacity, venous pressure, circulation time, blood volume, basal metabolic rate, and cardiac output.

Circulation Times. The preoperative circulation time was studied in 44 of the patients. The average value, arm to tongue, was 25.5 seconds with a range of 13 to 45 seconds. Twenty-five cases (59 per cent) had a circulation time of 24 seconds or greater.

Vital Capacity. Vital capacities were recorded in 49 cases. The average value was 1885 cc. with a range of 575 to 4000 cc. Thirteen patients (26.5 per cent) had a vital capacity of less than 1500 cc.

Venous Pressure. Venous pressures were recorded in 60 of the 61 cases. The values confirm the opinion previously expressed by Eeck and by others that elevated venous pressure is an integral part of this clinical diagnosis. The average value was 26 cm. of saline with a range of 15 to 41 cm. No case had a venous pressure of less than 15 cm. and only five had a value of less than 20 cm. At the other extreme, six patients had a venous pressure of 35 cm. or greater.

Cardiac Output. The cardiac output determinations were done by the Grollman acetylene method. This method is accurate and valid for comparative studies though absolute values are consistently lower than those obtained by catheterization of the right heart. Preoperative stroke volume was determined in 36 cases. The average value for the group was 34 cc. with a range of 19 to 66 cc. The normal value for stroke volume by the Grollman method is 60 cc. Thus the average stroke volume was 56 per cent of the accepted normal.

Blood Volume. Blood volume was determined in 19 preoperatively by the vital red method. The range of normal for this method is 70 to 100 cc. per Kg. of body weight. The average value for this group was 93.5 cc. per Kg. of body weight with a range of 71 to 112 cc. Six values were over 100 cc. per Kg. and most of the patients had values toward the upper normal limit.

Electrocardiograms

The electrocardiograms of all 61 cases have been studied. Most of the records made before the use of unipolar limb and chest leads make this primarily a study of the standard bipolar limb leads. Eleven of the cases were previously reported by Cushing and Feil. The behavior of the heart during surgery will be reported by Jaruszewski and associates.

Rhythm. Forty-two (68.8 per cent) had regular sinus rhythm throughout the entire period of observation including the follow-up. Fourteen (22.9 per cent) had auricular fibrillation throughout the entire period of observation. Four (6.5 per cent) had auricular flutter at some time. Two of these cases were converted to sinus rhythm with digitalis and/or quinidine preoperatively. This sinus rhythm was maintained throughout the period of observation. One patient was converted from auricular flutter to auricular fibrillation with digitalis. Ten years later he still had auricular fibrillation. The fourth case of flutter developed during the induction of anesthesia (GOE) and two weeks postoperatively was converted to sinus rhythm with digitalis and quinidine. Only one patient with sinus rhythm, when first seen, developed permanent auricular fibrillation 10 hours postoperatively. This patient is still living 14 years later. One patient had transient paroxysmal auricular tachycardia on the third postoperative day and one had transient auricular fibrillation.

P-R and QRS Intervals. The P-R interval in general was within normal limits. In four, it was greater than 0.20 second, the longest being 0.23 second. The QRS complexes were within normal limits except for reduced amplitude.

T-Wave Changes. All had primary T-wave changes. In two all the T waves were upright in the standard bipolar limb leads, with the T waves low in amplitude.
Electrical Axis. The electrical axes were generally in the intermediate position. The mean axis for the group was +68 degrees. The normal axis for males in this age group is +58 degrees. Two patients had axes of less than 0 degrees, these being -20 and -25 degrees. Both had normal blood pressure and no history or signs of rheumatic heart disease. Eight patients had an axis of greater than +90 degrees.

Voltage. The maximum voltage was measured by taking the height of the tallest R wave in any of the three bipolar limb leads. In many, the voltage was characteristically abnormally low. The average height of the R wave was 5.7 mm. with a range of 1 to 13.5 mm. The amplitude of QRS in the chest lead was usually reduced, but not to the same degree as in the bipolar limb leads.

Duration of Electrical Systole. The duration of electrical systole was determined by calculating the value of k in the formula QT = K√R - R. The average value for k was 0.380 (within the limits of normal) with a range of 0.307 to 0.480.

P Waves. The P waves were broad and notched in 20 cases (42.6 per cent). In some, this was marked enough to suggest “mitral P waves,” though no cases of mitral stenosis were present.

In the whole series the most consistent electrocardiographic findings were broad, notched P waves, low voltage of QRS, and primary T-wave changes.

Postoperative and Follow-up Electrocardiograms. Electrocardiographic patterns in the immediate postoperative period (first 30 days) were difficult to evaluate. With no consistent change in voltage, it was impossible to separate those T-wave changes due to altered posture and to epicardial injury from the surgical procedure itself, from those due to intrinsic changes in the myocardium. A study of 23 cases with follow-up electrocardiograms obtained 1 to 22 years postoperatively (with only five cases less than two years), showed a fairly consistent increase in voltage (average increase, 4.5 mm.). The T waves remained inverted and the mean axis shifted 18 degrees to the right. In four, the T wave became normal. In eight, there were minor T-wave changes associated with shift in electrical axis. There was no significant change in the value of k and only one had a change in rhythm from that present at the time of discharge from the hospital.

Serial electrocardiographic changes before and after operation are shown in figures 1, 2, and 3.

Roentgenoscopy

Moderate or marked diminution in pulsations occurred in all cases, with none visible in some; in others the restriction was localized. Localization occurred when the calcified or thickened pericardium formed a ring about the apex of the heart. This caused a localized pulsation with adjacent areas appearing immobile. During inspiration, fixation and lack of change of the cardiac silhouette were observed. Calcification, marked in some patients, was so minute in others that it could only be identified on the roentgenograms. Where calcification was extensive it appeared to encase the heart completely except at the base. In others, it was localized to one of the margins. Motion of some of the calcified mass indicated that the scar was not rigid. Calcification in a healed myocardial infarct must be included in the differential diagnosis. It is frequently difficult to determine the exact location of the calcium densities.
Changes extrinsic to the pericardium include those in the lungs, pleura, diaphragm, and great vessels, resulting in varying degrees of fixation of the heart and pericardium. Normally, as the diaphragm descends in inspiration, the heart posterior mediastinum become more radiolucent and the distance between the heart and sternum increases. In our patients no change in contour or position of the heart and mediastinum could be identified during inspiration.

Fig. 2. Serial electrocardiograms. The record of 4/3/35 was taken one day prior to pericardiectomy. Note the persistence of primary T-wave changes despite the increase in voltage.

Fig. 3. Serial electrocardiograms. The record of 7/28/35 was taken one week after the onset of acute purulent pericarditis associated with pneumonia. The record of 11/20/36 was taken after the development of chronic compression, one day prior to pericardiectomy. Note the broad P waves prior to operation.

and mediastinum decrease in transverse diameter, the lungs become more radiolucent, and the costophrenic angles are sharply defined. The heart may shift slightly with a change in the position of the patient. The lateral projection reveals a slight decrease in the size of the heart, the inferior portions of the anterior and posterior mediastinum, and the costophrenic angles.

Freedman found that with the patient in the lateral recumbent position, there was some degree of fixation. The retrosternal space may remain obliterated. With inspiration an "upward tug" on the heart may be noted. Diaphragmatic fixation and adhesions, though
identified, are frequently obscured by extensive pleural effusion.

The size of the heart was estimated from the charts of Ungerleider\textsuperscript{26} using the transverse diameter and height and weight. In younger patients the cardiothoracic ratio was determined by the method of Lincoln and Spillman.\textsuperscript{27} The heart size was regarded as normal with the measurement within $+10$ per cent and $-10$ per cent of the mean. The transverse diameter represents the combined heart and pericardial shadow rather than a true measurement of the heart. Heavy exposure roentgenograms were useful in some instances. Small calcifications in the periphery of the lungs as well as paratracheal calcium shadows may be obscured by the pleural shadow. Frequently, the roentgenograms in the oblique positions showed abnormalities which escaped detection at roentgenoscopy.

*Kymograms.* Johnson,\textsuperscript{28} the first in the United States to report on the value of the kymogram in diagnosing pericardial scars, showed absence of the diastolic thrust of the cardiac silhouette throughout the right and left lower borders, with a marked decrease in the region of the left auricle and descending aorta. Freedman\textsuperscript{29} believes the kymographic examination holds no specific diagnostic value but can be used as an objective record for postoperative studies to compare the improvement in the pulsations. Stewart and co-workers,\textsuperscript{30} however, believed it was of considerable aid in diagnosis, especially in the study of the pulsations along the aorta and the right border of the heart where the amplitude was found to be most regularly reduced. They believed the flattening and irregularity of the diastolic peak of the wave was helpful but less important. The kymogram in the frontal projection may be a composite of two chambers of the heart. This is especially true of the right border. Additional studies in the oblique projections enhance the value of this method.

*Roentgen Studies in This Series.* Our roentgen study consisted of a review of the roentgenograms and roentgenoscopic observations in 58 cases with evaluation of the preceding criteria. Twenty-six of these were reported by Freedman in 1937.\textsuperscript{24} Kymograms were taken in 43 patients prior to operation.

The transverse diameter was measured in 52 patients. In five, pleural fluid or thickening obscured the cardiac margins. It was within normal limits in 24 patients and enlarged in 28. The contour was classified as triangular in 21 patients, globular in 26, boot-shaped in six, and normal in four. Calcification, identified in 27 patients, was frequently quite extensive and appeared to encase the heart almost completely. The base of the heart was usually spared. The most common locations were the posterior portion of the pericardial sac, the apex and the left cardiac margins.

Roentgenoscopic observations and kymograms revealed a decrease in the amplitude of the pulsations in all. Absent in some throughout the entire silhouette, in others it was confined to one of the cardiac margins. An increase in the amplitude of pulsations was occasionally noted. This usually occurred in the apical region where the calcification formed a firm ring about the apex. With a change in the position of the patient fixation of the heart caused a diminished lateral shift in 33 of the 40 patients so studied. In these there was no respiratory change in contour. The behavior of the diaphragm during respiration was variable, with decrease in excursion or with portions appearing fixed. In a small number of patients, a paradoxical movement was observed.

The superior mediastinum appeared normal in 20 patients and widened in 38, probably due to engorgement of the superior vena cava and associated mediastinitis. Normal segmentation of the aorta and pulmonary artery was obscured in all but seven. There were diminished pulsations in the aorta in 36, representing the thickening of the mediastinal pleura and a decrease in blood flow secondary to reduced cardiac output. Stewart and Heuer\textsuperscript{31} noted an increase in the size of the aorta following operation not extending to this region.

Active infiltration was identified in four patients, fibrosis in six, and parenchymal calcification in 13. Vascular markings of the lungs were increased in 20 patients indicating some degree of passive hyperemia.
Fig. 4. Roentgenogram prior to (left) and subsequent to (right) pericardectomy demonstrating marked increase in pulsations, especially over the left border, after removal of scar.

Fig. 5. Lateral views demonstrating (left) thick layer of calcium prior to operation. Following operation (right) a small posterior scar remains.

There were pleural shadows (effusion and thickening) in 49 patients with the costophrenic margins sharply defined in nine. The distribution was bilateral in 24 patients, unilateral in
25. In many instances, the effusion was extensive, obscuring the cardiac margin. Only after repeated pleural taps could an adequate study be performed.

Postoperative roentgen studies were obtained in 45 patients. Improvement in the amplitude of pulsations occurred in 41. In some, it was localized to the area where the scar was removed, in others, generalized. The heart size was unchanged in 18 patients, enlarged in 16, and decreased in 11. The pericardial calcification was not completely removed in many patients and was identified in 21 in the postoperative roentgenograms.

Typical x-ray studies are shown in figures 4, 5, and 6.

**Surgical Procedures**

Every effort was made to avoid surgery in the presence of active infection.* No case was refused surgery because of being a “poor risk.” Paracenteses were performed preoperatively and diuretics were given freely. For a short period quinidine was given immediately prior to the operation to reduce myocardial irritability. This was done in 17 patients and then abandoned. Gas, oxygen, and ether produced the most satisfactory anesthesia. The operation was smoother when respiration was maintained by a mechanical respirator. The left anterior approach in the third or fourth intercostal space

![Fig. 6. Posteroanterior and oblique views showing a layer of calcium originally considered to represent pericardial calcification. Subsequently proved to be calcification of myocardial aneurysm resulting from myocardial infarction.](image-url)

*Holman and Willett* recently obtained satisfactory results in pericardiectomy during active tuberculous pericarditis in five patients. Three patients were cured and two patients were improved. Streptomycin was administered.

was used in all instances excepting those requiring removal of scar from the extreme right side of the heart. The pericardium was split from base to apex and removed anteriorly and laterally. When possible, the pericardium was separated from the heart by blunt dissection, occasionally by sharp dissection. A second operation was necessary in some instances to clear the extreme right side of the heart. Rest periods of one to three minutes were frequently employed when the heart became irregular or when the blood pressure fell.

The usual scar was thick, fibrous, and diffuse. In two patients the compression was due to...
thick fibrous bands encircling the heart. One patient had a “tight band about the base.” Another had two bands, one at the apex and one at the atrioventricular sulcus with the myocardium “bulging in between.” A third had a thick band about the superior vena cava in addition to a diffuse scar. In 26, calcium in the pericardium was easily discernible to the operator. In one, the calcium was thick enough to make complete excision impossible. In another, rongeurs were used to remove the calcified pericardium. In 12 instances, signs of active infection in the pericardium were present, manifested by a small amount of pus, tubercle-like formations, or inflamed or edematous scars. The scar invaded the myocardium in 19 patients, making the dissection difficult. Slight bleeding occurred.

Operative Difficulties. Jaruszewski, Hellerstein, and Feil described the treatment of arrhythmias developing during cardiac surgery and Beck discussed the treatment of ventricular fibrillation and standstill. In 69 operations on 61 patients in this series, some type of surgical difficulty occurred 12 times with four deaths.

The surgical complications were: hemorrhage from incision of right auricle in three, hemorrhage from left auricle in two, ventricular fibrillation in two, cardiac arrest in one, bilateral pneumothorax in one, hemorrhage from branch of anterior descending coronary artery in two, and tear of a coronary vein in one.

Postoperative Status

The postoperative course of patients was generally good with few complications. They were given oxygen, general supportive measures, and antibiotics. Serious postoperative complications appeared in four. Two had postoperative bleeding requiring surgical intervention. One had postoperative wound infection and subsequently died. Another had wound infection, which apparently was a manifestation of the underlying disease. One developed acute cardiac failure on the twelfth postoperative day but responded satisfactorily to the usual measures. Twelve patients required one to four paraventeses postoperatively, all making an uneventful recovery. Three had postoperative arrhythmias, previously mentioned. No instances of embolic phenomena occurred.

Deaths. Eleven (18 per cent) died in the hospital. This included one patient who died in his fifth postoperative month, never having been well enough to be discharged. Only one patient died on the operating table; six died within the first 48 postoperative hours. The other three patients died at 5, 23, and 40 days.

1. During operation a 15 year old white boy developed bilateral pneumothoraces. These were controlled satisfactorily, but toward the end of the operation, the heart dilated acutely and ceased to beat. All attempts at resuscitation were unsuccessful. No autopsy was performed.

2. A 37 year old white woman was in extremely poor condition preoperatively. The calcium on the pericardium was so thick that it was removed with a rongeur. During operation the heart stopped but was revived satisfactorily. Postoperatively the patient was in fairly good condition with a systolic pressure of 88 mm. Hg and a pulse of 105. The patient suddenly died 24 hours postoperatively. Autopsy revealed acute pulmonary edema. There were no embolic phenomena.

3. A 23 year old white woman was in good preoperative condition. During operation both pleural spaces were accidentally opened, and a massive hemorrhage from the right auricle occurred. The postoperative blood pressure was 60/30, and in spite of transfusions the patient died 16 hours later. Autopsy revealed hemopneumothorax (900 cc.), atelectasis of the left lung, and acute pulmonary edema.

4. A 40 year old white man developed ventricular fibrillation which persisted for 20 minutes during the operation before it was successfully treated. The patient was in good condition for a few hours following operation and then began to fail. One thousand cc. of fluid and then air were aspirated from the left chest. A catheter was placed in the chest through a stab wound and the respirations maintained by a positive pressure mechanical respirator. The wound was not “sucking air,” but as air was forced into the lungs it came freely out of the catheter, giving evidence of a bronchopleural fistula. The patient died 18 hours after operation. An autopsy was not performed.

5. A 36 year old white man had an uneventful operation. Three hours postoperatively the blood pressure fell. He died six hours later. Autopsy did not reveal the cause of death.

6. A 26 year old white man had active tuberculosis. Surgery was delayed. At operation the pericardium was thick, edematous, and showed signs of active infection. The operation was uneventful, but 36 hours after the operation he suddenly became...
cyanotic and died. Autopsy revealed a massive hemopneumothorax and tuberculosis of the pleura, pericardium, and mediastinal nodes.

7. A 27 year old white man had a septic pre- and postoperative course. At operation a creamy pus-like material was present in the pericardium. This was negative for organisms by culture, smear, and guinea pig inoculation. The postoperative course was septic, requiring catheter drainage of the pericardium. A hemolytic streptococcus was cultured from the pus draining from the wound. In the fifth postoperative month, there was a sudden hemorrhage from the wound and the patient died. Autopsy revealed a perforation of the right ventricle due to suppuration and abscess formation of the ventricular wall. Organisms resembling pneumococci were seen in the microscopic sections. The ventricular perforation may have been caused by constant pressure of the catheter.

8. A 26 year old white man withstood the operation. Postoperatively he was febrile, and he died on the fifth day. Autopsy revealed left hemopneumothorax (3000 cc.), cavitary tuberculosis of the mediastinum and pericardium, and confluent bronchopneumonia of the right lung. In addition there was miliary tuberculosis involving the lungs, liver, spleen, peritoneum, and brain.

9. A 14 year old white boy had an uneventful operation, though his blood pressure was unobtainable for 12 hours postoperatively. He then appeared well until the eighth day when he developed a fever which persisted until his death on the fortieth postoperative day. Autopsy revealed miliary tuberculosis involving the lungs, pleura, lymph nodes, spleen, liver, kidneys, and peritoneum.

10. A 36 year old white man withstood the operative procedure well but postoperatively the right lung did not expand. Chest aspiration and bronchoscopy were of no avail. The patient died 24 hours later. Autopsy did not reveal the cause of death. It was attributed to "acute dilatation of the heart."

11. A 30 year old white man withstood the operation well but developed postoperative wound infection. The wound drained profusely; the patient grew steadily worse, dying on the twenty-third day after operation. Autopsy was not performed.

**Results of Treatment**

Following recovery from the immediate trauma of surgery, most cases showed an early objective and subjective improvement associated with a gradual improvement of physiologic function tests over a period of several weeks. Six patients in this series did not follow this course but required one or more operations. One case was operated three times; one, four times; and one, five times. The other three were each operated twice.

In a check of the 50 patients discharged from the hospital, 11 were examined by the authors, 13 by physicians or clinics in other cities, and 24 answered questionnaires. Personal letters frequently accompanied the questionnaires amplifying the answers. Two cases have been followed for one year (the most recently operated), 13 were followed for two to six years, 7 for 6 to 11 years, and 22 for more than 11 years. The longest follow-up in a single case is 22 years.

Two cases have been classified as unimproved. One, unimproved at the time of discharge, we have been unable to trace. The other died of progressive disease one year and two months later. This patient had been operated four times previously.

Of the six improved cases, three died since discharge (one died of unknown causes 11 years and six months after operation, one died of a double hernia two years after pericardectomy, and one died 11 years after operation from ventricular fibrillation during operation in another hospital for recurrence of compression symptoms). Of the remaining three in this group, one was improved at the time of discharge with follow-up data not available; one is improved 13 years after the first operation, seven years after the second operation, but is again developing symptoms; and one has been steadily improving in the year and eight months since operation.

Of the 38 patients with excellent results, three have died of unrelated causes; two, five and six years postoperatively. The remaining 35 patients are well and are carrying on full activity. Thus, a satisfactory result (excellent or improved) has been obtained in 44 (72.1 per cent) of the 61 cases operated.

**Pulse Rate.** The average preoperative pulse rate was 91, with a range of 60 to 124. The late postoperative pulse rate averaged 81 with a range of 58 to 103. Only three patients had pulse rates over 100, as compared with 24 in the preoperative period.

**Venous Pressure.** Preoperative venous pressures were obtained in 60 cases, the average value being 26 cm. of saline with a range of 15 cm. to 41 cm. There were no values below 15 cm. and only five below 20 cm.
Many cases showed a drop in venous pressure almost immediately after operation. The average value in the 48 cases, with determination done in the first 30 postoperative days, was 17.5 cm. with a range of 5 cm. to 34 cm. of saline. There were 31 cases (50.7 per cent) with venous pressures less than 20 cm. and 12 cases (32.7 per cent) with pressures less than 15 cm.

In the late postoperative period there were 24 observations. The average value was 14.3 cm. of saline with a range of 6 cm. to 25 cm. Fifteen (62.5 per cent) had pressures less than 15 cm. at this time; however, three of the 24 observed in this late period had pressures greater than 20 cm. of saline. The average decrease in venous pressure for individual cases was 11.4 cm., with only five failing to show a fall at the time of last observation.

**Pulse Pressure.** The average preoperative pulse pressure was 33.2 mm. Hg, with a range of 10 to 70 mm. Hg. In the early postoperative period 27 observations were made. The average value was 43 mm. Hg, with a range of 25 to 58 mm. Hg. In the cases with late postoperative observations, there has been very little change. The average value of the 23 cases in the late group was 43 mm. Hg, with a range of 25 mm. to 55 mm. Hg.

**Stroke Volume.** In the 36 preoperative determinations of stroke volume, the average value was 34 cc. per beat, with a range of 19 to 66 cc. Stroke volumes were determined in the early postoperative period in 28. There was a definite increase in most cases. The average value at this time was 45 cc. per beat with a range of 29 cc. to 70 cc. As in the pulse pressures, the greatest change in stroke volume seemed to occur in the early postoperative period. The average value for the nine cases seen in the late postoperative period was 48 cc. per beat, with a range of 31 to 62 cc. per beat.

**Blood Volume.** Blood volume was determined in 19 cases preoperatively. The average value was 93.5 cc. per Kg. of body weight, with a range of 71 cc. to 111.6 cc. per Kg. There were six cases with values greater than 100 cc., and the group as a whole showed a tendency toward increased blood volume. Early postoperative determinations of blood volumes were made in 14 cases; there were no late determinations.

The average value postoperatively was 88.6 cc. per Kg. of body weight with a range of 59 to 104 cc. Of the 15 cases having pre- and postoperative studies the average decrease in blood volume was 7 cc. per Kg. Only three cases showed postoperative increase in blood volume.

**Circulation Time.** Arm to tongue preoperative circulation times were determined in 44 patients. The average value was 25.5 seconds, with a range of 13 to 45 seconds. In the early postoperative period circulation times were done on 27 patients. The average value was 20 seconds, with a range of 10 to 36 seconds. In the late postoperative period the average circulation time was slightly longer than in the early period. The average late value in 18 cases was 23 seconds, with a range of 13 to 40 seconds.

**Basal Metabolic Rate.** The basal metabolic rate was determined in 26 patients prior to operation. In most cases multiple determinations were done. The basal metabolic rate was calculated from the oxygen consumption and based upon estimated dry weight. (Usually the weight after recovery was used as the dry weight.) The average basal metabolic rate for the 26 cases was −7 per cent. In both the early and the late postoperative period, the oxygen consumption increased and was within normal limits in almost every case studied.

**Pathologic Findings**

In chronic cardiac compression, the heart and pericardium appear as a single mass of scar tissue which is so frequently adherent to the diaphragm, pleura, and mediastinal structures. The density of the scar and the amount of contraction determines the presence or absence of compression. The scar may vary in thickness from 2 mm. to over 1 cm. Varying amounts of calcium or even bone formation may be present (fig. 7). The scar frequently invades the myocardium itself. Occasionally signs of active inflammation may be present in the form of pericardial fluid, edema, hyperemia, and tubercle formation. With long-standing compression, atrophy of the heart occurs histologically and grossly.

In all cases a thick, fibrous, adherent scar was found. In approximately 50 per cent there
was an appreciable amount of calcium. The scar was diffusely distributed in all cases, except in two with localized bands. There were signs of active pericardial infection in 12. The hearts of the eight patients examined at autopsy were small or normal (from 175 to 300 Gm.), with one exception. This patient had associated rheumatic mitral disease with definite cardiac hypertrophy. The ventricular walls were usually thinner than normal. In three cases there was slight dilatation of the right ventricle.

Histologically, the typical picture was that of an "acellular fibrosis with hyalinization consistent with chronic inflammation." Many sections showed fibroblastic proliferation, some showed vascularity, and a few inflammatory cells.

In 16 patients the surgical specimens were of particular interest. Sections of these hearts have been re-examined and new sections prepared. Eight were diagnosed as tuberculosis of the pericardium on the basis of tubercle formation. Tubercle bacilli were identified in only one. The other eight specimens showed chronic inflammation even though the diagnosis of tuberculosis was established by other pathologic or bacteriologic evidence obtained before or after operation. Thus, tuberculosis cannot be excluded on the basis of a negative pathologic examination of the excised pericardium. Figure 7 shows representative specimens of the removed pericardium.

Eight patients were autopsied; all had been operated and died while still hospitalized. Two had widespread miliary tuberculosis involving the lungs, pleura, mediastinum, liver, spleen, peritoneum, and pericardium. One had not had a previous diagnosis of tuberculosis despite two operations. Another showed tuberculosis involving the pericardium, right pleura, and the mediastinal nodes; the fourth showed tuberculosis of the hilar mediastinal nodes and of the spleen without a previous diagnosis of tuberculosis.
In two no specific cause for the compressive scar was found at autopsy. One other showed an old tuberculous mediastinal node and chronic mitral valvulitis with calcification and insufficiency. Neither of these lesions could be definitely established as the etiology of the compression. The eighth showed suppurative myocarditis associated with a longstanding suppurative pericarditis.

Chronic passive hyperemia of the lungs, liver, spleen, and viscera was found in all. Three patients had tuberculous pleuritis and the other five, obliterative pleuritis. A diagnosis of “chronic productive peritonitis” was made in half of the cases autopsied.

Examination of the liver revealed a constant pattern of varying degree in each case. Capsular thickening was marked in only two, less in the others. Sectioning of the liver revealed evidence of nodularity (subcapsular).

Histologic examination revealed moderate passive hyperemia in only one case. The other seven had various degrees of damage to liver cells, occasional necrosis, and proliferation of connective tissue and bile ducts, with alteration of the architectural pattern. In two, the histologic section was indistinguishable from advanced Laennec’s cirrhosis.

These findings emphasize the fact that increased venous pressure not only gives central involvement but also produces portal fibrosis and alteration of architecture. The predominance of subcapsular involvement may result from the pressure effects at the periphery of the liver substance, due to greater anoxia there.

**Etiology**

There has been difference of opinion as to etiology. Since the pathologic examination of the specimen removed at surgery is so frequently inconclusive, it is often necessary to determine the etiology from the type of onset, associated diseases, previous history of tuberculosis or other disease, examination of pleural or pericardial fluid, or the presence of tuberculosis elsewhere.

In 31 patients (50.8 per cent) the onset was vague and insidious. In these patients, there was no history of any acute episode related to the onset of symptoms. In 10 patients (16.4 per cent) the onset was associated with acute infection, which in five consisted of an upper respiratory infection, bronchitis, or grippe. In one the onset followed an acute nonspecific pericarditis and in one, “gripe and acute pericarditis.” In the other two patients the onset were associated with illnesses manifested by fever, headache, and occasionally chills. Four of these 10 patients were diagnosed as having acute pericarditis during this initial infection. Nineteen (31.1 per cent) had delayed onset following an acute infection, the delay varying from one to several months. In two patients the onset followed pneumonia with pericarditis.

Even though in 29 (47.9 per cent) patients the onset could be related to some febrile illness, a slightly larger group of 33 (52.1 per cent) patients had no history of a precipitating episode and only nine had a history of any type of pericardial disease prior to the onset.

Tuberculosis, based on histologic or bacteriologic evidence, was the cause in 16 patients (28.2 per cent). There was no significant type of onset in the tuberculous cases. Two of these had normal chest plates, four had Ghon complexes, and in only one instance was active pulmonary tuberculosis suggested. Four cases (6.5 per cent) were considered as of possible tuberculous origin, because of suggestive evidence of tuberculosis in the pathologic specimen.

Two (3.3 per cent) were pyogenic in origin. In one patient, there was x-ray evidence of pericardial calcification prior to the onset of the pyogenic episode. One case illustrates the pitfalls of making a diagnosis on the basis of history alone. There was a history of septicemia with metastatic abscesses two years prior to the onset of the compression syndrome. At autopsy, it was of tuberculous origin.

In three patients (4.9 per cent) the onset followed acute nonspecific pericarditis. Of the 16 cases proved to be tuberculous in origin, three had the onset of symptoms following acute pericarditis.

Although 10 patients had a past history of rheumatic fever, in only one could this be considered as a possible etiologic agent. This patient had recurrent rheumatic fever preceding and following the development of com-
pression. In view of the findings in this series and those of others,\textsuperscript{34} it is probable that rheumatic fever was coincidental. One case illustrated the hazard of diagnosing a rheumatic etiology. This patient had rheumatic pericarditis 16 months prior to the onset of compression. Pathologically the pericardium removed at operation showed fibrocaseous tuberculosis.

Thirty-five cases (57.7 per cent) are classified as of unknown etiology. On occasion the etiology cannot be established even with a complete autopsy, and an autopsy can present some data for two causes, but be insufficient for diagnosis.

Most observers believe that tuberculosis is the single most important etiologic agent; however the published incidence varies widely. Blalock and Levy\textsuperscript{35} reported positive evidence for tuberculosis as the etiology in 57 per cent, while Harvey and Whitehill\textsuperscript{14} reviewed 95 cases of tuberculosiis of the pericardium and concluded that “we have never seen a patient who recovered from the active process develop chronic constrictive pericarditis.” Andrews, Pickering, and Sellors\textsuperscript{37} found that of 18 patients with acute tuberculous pericarditis, 16 developed chronic compression, concluding that tuberculous pericarditis results in compression.

Sellors\textsuperscript{34} found tuberculosis in 16 of 20 cases. Mortensen and Warburg\textsuperscript{28} found histologic evidence of tuberculosis in only three of 20 operated cases. Harrington\textsuperscript{19} found a 15 per cent incidence of tuberculosis in 34 operated cases. Paul, Castlen, and White\textsuperscript{14} found tuberculosis in nine of 53 cases. Heuer and Stewart\textsuperscript{1} could prove tuberculosis in only one of their 18 operated cases. Armstrong\textsuperscript{4} concluded that “probably there are many more cases of tuberculous origin than can be proven by histological or bacteriological methods.”

The role of rheumatic fever has long been controversial. Harrison and White\textsuperscript{49} in a follow-up of 1500 cases of rheumatic heart disease found no cases of chronic compression. In Harrington’s series\textsuperscript{15} the one case thought to be due to rheumatic fever was later proved to be tuberculous. Armstrong\textsuperscript{4} excluded rheumatic fever as a cause of compression.

There are many reports of chronic compression following acute nonspecific pericarditis. Five of Heuer and Stewart’s\textsuperscript{3} 18 cases had a previous pericardial effusion; and Paul and associates\textsuperscript{14} reported that previous pericarditis had been present in six cases (11.3 per cent) and a history suggestive of pericarditis was obtained in three additional cases (5.7 per cent). Hunter and East\textsuperscript{40} and Griswold\textsuperscript{41} reported chronic compression following acute pericarditis after a long interval.

Occasionally chronic cardiac compression has been reported to follow pulmonary infections, nonspecific infections, and septicemia.\textsuperscript{42–44} Recently, two cases of compression due to \textit{Pneumococcus tularensis} were reported.\textsuperscript{43, 44} Rarely, compression may be due to tumor.\textsuperscript{45}

The relationship of tuberculosis to compression scars is further emphasized in the pathologic studies of our cases. Of the eight patients studied at autopsy, four had tuberculosis. Of these four, only two were diagnosed as such prior to autopsy. This, in conjunction with the finding of negative surgical specimens in eight cases considered to be of tuberculous origin, emphasizes that more are due to tuberculosis than can be proved by clinical diagnosis. Six of the 11 postoperative deaths were in patients with tuberculosis, and six of the 16 cases proved to be tuberculous died. Even though 10 of the 16 tuberculous patients are living one year and eight months to 17 years after operation, it appears that a preoperative diagnosis of compression due to tuberculosis gives a poorer prognosis.

**Physiologic Changes**

Compression occurs when the rigid encasing scar contracts and encroaches upon the heart preventing adequate filling and ejection. A diffuse scar alone causes no physiologic derangement, as is illustrated by the relatively high incidence of adherent pericardium without compression. Occasionally a thick scar containing calcium may be present without evidence of compression.\textsuperscript{4}

The basic derangement of function, Regardless of the type of scar, is limitation of stroke volume.\textsuperscript{3, 46–50} The decreased cardiac output initiates the factors acting in congestive heart failure; abnormal salt retention, increased venous pressure, and increased blood volume.
Since the stroke volume cannot be increased with physiologic demands, the immediate form of compensation is tachycardia. As a result of the decreased cardiac output, tissue oxygenation is maintained by increased arteriovenous difference as the tissues extract more oxygen per unit of blood. At these lower partial pressures of oxygen, oxygen transfer to the tissues is impaired and the efficiency of the transport system is decreased.

Elevated venous pressure is one of the most striking and constant findings in this disease. It is higher than is usually found in congestive failure and shows little fluctuation in a single patient. The high venous pressure is due to obstruction of inflow, increased blood volume, and decreased cardiac output.

Figure 8 summarizes the alterations in the physiologic aspects of the disease following pericardial resection.

The severe passive hyperemia of the liver leads to portal hypertension and eventually to cardiac cirrhosis. Universal impairment of liver function occurs. Edema and ascites result from impaired formation of serum protein and from faulty absorption of protein from the intestinal tract.

The principle electrocardiographic findings are low voltage, P-wave deformity, primary T-wave changes, and arrhythmias. Low voltage of the QRS complex is due to the insulating effect of the pericardial scar and short circuiting by the pleural and peritoneal effusions. Atrophy of the heart is another factor in producing the low voltage.

The T-wave changes are due to injury of the superficial myocardium with resulting changes in the direction of repolarization. Postoperatively the primary changes remain because of the injury to the epicardial surface. A change in the ventricular gradient may be a factor altering the form and direction of the T wave.

The heart encased in a chronic constricting scar undergoes atrophy. With the removal of this rigid case, associated with augmented venous pressure, an increased burden is placed on the heart, and the heart dilates. In our series at least two postoperative deaths occurred as a result of “cardiac dilatation.” The myocardial fibers operate against an increased initial tension and are stretched beyond optimum initial length.

The immediate postoperative dynamic changes are due to release of the mechanical constriction. The later changes are due to improved function, reversal of atrophy, return of blood volume to normal, normal tissue oxygenation, increased renal flow, and decreased venous pressure. Their gradual return depends upon the restitution of cardiac muscle and function. Electrocardiographically, the early increase in voltage is due to removal of the insulating pericardial scar and disappearance of excess extracellular fluid.

**Differential Diagnosis**

While the clinical picture of chronic cardiac compression is clear-cut in the majority of patients, some errors are made when the clinical triad—so well emphasized by Beck—is not

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*With subsequent recovery of the myocardium, there is further increase in voltage.*
recognized. The diagnosis of Concato's disease may lead to error unless the importance of the compressing pericardial scar is considered.

The early occurrence of ascites may lead to the diagnosis of cirrhosis of the liver. The obvious increase in venous pressure in cardiac compression and the normal venous pressure in hepatic cirrhosis enables one to differentiate the two. Congestive heart failure due to congenital heart disease, rheumatic heart disease, cor pulmonale, hypertensive and arteriosclerotic heart disease may simulate in some respects the picture of chronic cardiac compression. The moderate dyspnea, the small heart, and the very high venous pressure usually make the diagnosis of cardiac compression clear-cut. The high venous pressure of congestive failure falls with diuresis while the venous pressure remains elevated constantly in cardiac compression. Chronic cor pulmonale (due to chronic pulmonary disease, pulmonary arteritis, or chronic pulmonary artery thrombosis) may produce signs suggesting cardiac compression. Peritoneal metastasis may produce ascites, but the normal venous pressure is of diagnostic importance.

The heart is large and the circulation times are rapid in cases of beriberi heart disease. Thrombosis of the portal vein (in cirrhosis of the liver, malignant liver disease, cholangitis, pancreatitis, and subphrenic abscess) produces ascites, splenomegaly, abdominal pain, and hematemesis. The patient may survive for months or years with the development of collateral circulation. Occlusion of the hepatic veins in polycythemia vera, neoplasm, or cirrhosis of the liver results in abdominal pain, nausea, vomiting, a tender rapidly enlarging liver and spleen, ascites, and collateral superficial abdominal veins. Thrombosis of the inferior vena cava results in sudden increase in the venous pressure of the lower extremities and edema. Rarely, metastases to the pericardium may cause the symptoms of chronic compression.

**Summary and Conclusions**

1. A critical pre- and postoperative study of 61 cases operated for chronic cardiac compression has been presented.

2. In this series, the patients were predominantly young men. The most common symptoms, in order of frequency of occurrence, were dyspnea, ascites, edema, and orthopnea.

3. The typical physical findings were venous distention, decreased precordial activity, distant heart sounds, pleural fluid or thickened pleura, an enlarged liver, and ascites and/or edema.

4. The electrocardiographic findings were low voltage and primary T-wave changes. Thirty-one per cent of the cases had some type of arrhythmia. Broad, notched P waves were found in 42.6 per cent of the cases.

5. Roentgenologic studies showed diminished cardiac pulsations. The hearts were of normal size or slightly smaller. Pleural thickening and/or fluid were present in 84.5 per cent. Pericardial calcification was demonstrated in 46 per cent.

6. In the cases having edema, 21.4 per cent had total serum proteins less than 5 Gm. per 100 cc., and 37 per cent had serum albumin less than 3 Gm. per 100 cc.

7. Circulatory studies showed elevated venous pressure, slight tachycardia, slight decrease in pulse pressure, decreased vital capacity, prolonged circulation time, decreased stroke volume, increased blood volume, and normal or a slightly less than normal basal metabolic rate.

8. The operative mortality was 18 per cent.

9. A satisfactory result was obtained in 72.1 per cent. The operative results in this series may be compared with those of other surgeons (table 1). Of a total of 415 operated cases, satisfactory results have been obtained in 258 (62 per cent).

10. Postoperatively all circulatory function tests tend to return to normal with the exception of persistent primary T-wave changes.

11. Tuberculosis was the cause of the compressive scar in 28.2 per cent. No etiologic agent could be demonstrated in 57.7 per cent of the cases. Rheumatic fever was not proved to be the cause in any of the cases.

12. Follow-up studies indicate that the majority of the patients return to full normal activity.

13. The outcome was less favorable in those
Table 1.—Results of Pericardiectomy Collected from the Literature

<table>
<thead>
<tr>
<th>Authors</th>
<th>Total Cases</th>
<th>Results</th>
<th>Mortality</th>
<th>Etiology</th>
<th>Remarks</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td></td>
<td>Cured</td>
<td>Improved</td>
<td>uncertain</td>
<td>Op</td>
</tr>
<tr>
<td>Heuer &amp; Stewart (Total of cases to 1939)⁴⁴</td>
<td>131</td>
<td>45</td>
<td>24</td>
<td>8</td>
<td>18</td>
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<tr>
<td>Schmieden (Westerman) 1944⁵⁵</td>
<td>54</td>
<td>20</td>
<td>15</td>
<td></td>
<td>8</td>
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<tr>
<td>Blalock &amp; Burwell 1941²</td>
<td>20</td>
<td>12</td>
<td>5</td>
<td>3</td>
<td>14</td>
</tr>
<tr>
<td>Heuer &amp; Stewart 1946³</td>
<td>18</td>
<td>8</td>
<td>7</td>
<td>3</td>
<td>1</td>
</tr>
<tr>
<td>Harrington 1947¹⁶</td>
<td>34</td>
<td>18</td>
<td>5</td>
<td>7</td>
<td>4</td>
</tr>
<tr>
<td>Mortensen 1948³⁸</td>
<td>20</td>
<td>3</td>
<td>4</td>
<td>2</td>
<td>1</td>
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<tr>
<td>Andrews et al. 1948³⁷</td>
<td>18</td>
<td>2</td>
<td>11</td>
<td>2</td>
<td>3</td>
</tr>
<tr>
<td>Paul et al. 1948¹¹</td>
<td>42</td>
<td>15</td>
<td>11</td>
<td>3</td>
<td>4</td>
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<tr>
<td>Lawrence et al. 1948³⁸</td>
<td>8</td>
<td>4</td>
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<tr>
<td>Holman &amp; Willett 1948³⁹</td>
<td>9</td>
<td>5</td>
<td>3</td>
<td>1</td>
<td>4</td>
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<tr>
<td>Present Series 1951</td>
<td>61</td>
<td>38</td>
<td>6</td>
<td>2</td>
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<tr>
<td>Totals</td>
<td>415</td>
<td>170</td>
<td>88</td>
<td>12</td>
<td>27</td>
</tr>
</tbody>
</table>

patients who were cyanotic preoperatively or who had a preoperative diagnosis of tuberculosis.

14. Physiologic factors involved in the production of the clinical picture of chronic cardiac compression are discussed.

REFERENCES

¹ WHITE, P. D.: Chronic constrictive pericarditis (Fick's disease) treated by pericardial resection. Lancet 2: 539, 1935.


⁶ —: A new conception of pericardial disorders.


Chronic Cardiac Compression (Chronic Constrictive Pericarditis): A Critical Study of Sixty-one Operated Cases with Follow-up
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