Rupture of the Infarcted Interventricular Septum
Surgical Repair with Survival

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Rupture of the interventricular septum, one of the most dramatic complications of myocardial infarction, has become a well-recognized clinical entity that is readily confirmed by cardiac catheterization. Since the usual course is that of rapid deterioration and early death, surgical repair of the defect is indicated once the diagnosis has been established. The medical literature contains only a few reports of attempts at surgical repair, with prolonged survival in just two cases.

Recently successful repair of a ruptured interventricular septum due to myocardial infarction was accomplished at the University of Oregon Medical School Hospital. The details of this case, including surgical technic and physiologic data, constitute this report. Previously published physiologic studies and surgical treatment of this entity are reviewed.

Case History

A 57-year-old man with angina pectoris, mild hypertension, and a previous myocardial infarction was admitted to the Veterans Administration Hospital, Vancouver, Washington, on May 30, 1961, complaining of severe substernal pain of 3 hours' duration, associated with sweating, weakness, and shortness of breath. The chest was clear, the apical impulse was 2.5 cm. outside the midclavicular line in the fifth intercostal space, and the rhythm was regular. The heart tones were poor; no murmurs, thrills, or friction rubs were audible. The blood pressure was 172/112.

An electrocardiogram revealed an acute inferolateral myocardial infarction (fig. 1). Laboratory studies were within normal limits, except for elevated blood sugar and glycosuria. Initially the patient remained acutely ill and was treated with oxygen, opiates, and sodium coumadin. The diabetes was well controlled with crystalline insulin initially, and then with Tolbutamide. The highest serum transaminase recorded was 350 units on June 1, and his maximal temperature, 103 F., was reached on the third day. He was normotensive after the day of admission. From June 2 to 5, a loud pericardial friction rub was heard. On June 2, an electrocardiogram showed 2:1 atrioventricular block along with deepening Q waves and S-T elevation in the anterior chest leads, indicating anterior, apical, and inferior infarction (fig. 1). He was intermittently confused from June 5 to June 9, and on one occasion walked around the room. On June 9 he had severe high subternal pain for 24 hours. An electrocardiogram on June 12 showed sinus rhythm, and only lead aVp demonstrated the myocardial infarction. The previous large Q waves in the precordial leads were absent and large R waves with asymmetrical, inverted T waves were present in V4 to V6 (fig. 1). This pattern, characteristic of left ventricular hypotrophy or “strain,” was present subsequently on all electrocardiograms with no progressive ST-T changes typical of the usual evolutionary pattern of a myocardial infarction.

On June 26, a loud grade IV, of VI, pansystolic murmur was noted, maximal in the fourth left intercostal space and widely transmitted over the precordium, with an associated thrill. The diagnosis of perforated interventricular septum was made, and the patient was transferred to the Veterans Administration Hospital, Portland, Oregon, on the same day. On admission, his pulse was 120 and regular, the respirations were 32, and the blood pressure was 118/86. The neck veins were not distended, the chest was clear to percussion and auscultation, and the previously described murmur and thrill were noted. The liver was palpable 3 cm. below the right costal margin and a hepato-jugular reflux could be demonstrated. The venous pressure was 15 cm. of water and the Decholin circulation time was 30 seconds from arm to tongue. The patient was maintained at bed rest with salt-restricted diet and digitalis.

Phonocardiogram showed a loud pansystolic murmur (fig. 2). Cardiac catheterization on June 30 was accomplished without complications and revealed a mild increase in the right ventricular and pulmonary artery pressures (fig. 3), and a left-to-right shunt calculated at 14.4 liters per minute (table 1). A dye-dilution curve was grossly

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Serial electrocardiograms. Electrocardiogram in 1949 is consistent with an old inferior infarct and left ventricular hypertrophy. Electrocardiogram on May 31, 1961, shows an acute inferior infarction and extension of infarction on June 2, 1961. Record on June 19, 1961, typical of all tracings taken after June 12, 1961, up to surgery is compatible with left ventricular hypertrophy; only aVF shows infarct. Postoperative electrocardiogram on July 20, 1961, shows late R wave in right precordial leads; signs of left ventricular hypertrophy persist.

abnormal and consistent with a large left-to-right shunt (fig. 2).

On July 1, 1961, the patient was noted to have signs of left ventricular failure with fine and medium moist rales in both lung bases. More vigorous restriction of activity and salt intake was instituted, and mercurial diuretics were added to help control the cardiac decompensation. On this regimen the basilar rales disappeared.

He was transferred to the University of Oregon Medical School Hospital on July 3, 1961, for open-heart surgery on July 5, 1961.

The heart was exposed through a vertical midline sternum-splitting incision. The right femoral artery was cannulated in the groin. The patient's blood pressure dropped sharply on induction of anesthesia, and norepinephrine was necessary to maintain blood pressure until bypass was started. The entire anterior wall of the right ventricle was dark and avascular in appearance and did not bleed when incised. There was a prominent thrill over the lower portion of the right ventricle near the diaphragmatic surface. After institution of cardiac bypass and right ventriculotomy, the ventricular septal defect was seen to lie along the diaphragmatic surface of the septum, the blood having dissected through the septum from above to form a flap (fig. 4). A papillary muscle of the tricuspid valve was divided to expose the defect better. The dissected flap of interventricular septum was sutured in place with interrupted 3-0 silk mattress sutures placed through and tied over a reinforcing patch of Teflon felt. The divided papillary muscle was then reapproximated with interrupted silk sutures and the infarcted right ventricular wall closed with interrupted mattress sutures of 3-0 silk placed so as to suture the margins of the incision and tied over patches of Teflon felt on both sides. This was thought to be the only way to obtain secure closure of the infarcted muscle. Bypass time was 72 minutes. After completion of bypass, the blood pressure was maintained without norepinephrine. Isoproterenol was given until the next morning because of sinus bradycardia. The postoperative course was one of steady improvement. Examination was completely normal, except for a grade I to II systolic ejection murmur. Changes in the chest x-ray are shown in figure 5.

Cardiac output by dye-dilution technique was determined with injection into a peripheral vein, and a low systemic output, 4.1 liters per minute (cardiac index 2.1), was found. The shape of the dye curve was normal (fig. 2), and the brachial artery pressure recording (fig. 3) showed higher pressures with a smaller pulse pressure, associated with loss of the large left-to-right shunt. Repeat phonocardiogram (fig. 2) showed absence of the loud pansystolic murmur. The patient was dis-
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charged on August 3 on digitalis and sodium coumadin therapy.

He was readmitted to Portland Veterans Administration Hospital in September 1961, with acute cholecystitis and cholelithiasis and underwent cholecystectomy without incident.

Six months later the patient reported only occasional mild anginal pain; his exercise tolerance had gradually improved, and he had returned to work. The cardiac murmur, electrocardiogram, and chest x-ray remained unchanged.

Discussion

Reports of septal rupture following myocardial infarction are becoming more frequent. This condition accounts for about 1.5 to 2 per cent of all deaths following myocardial infarction, and about 20 per cent of all ruptures of the heart. Although myocardial infarction was not recognized as a clinical or pathologic entity at the time, Latham described the first case of rupture of the infarcted septum in 1846 with a case history that leaves little to be added. The diagnosis is made by the sudden appearance of a harsh, loud, pansystolic murmur in a patient with a recent myocardial infarction. The murmur is maximal in the fourth or fifth left intercostal space and is widely transmitted over the precordium, but not to the neck vessels. A systolic thrill is palpable in about 60 per cent of the cases. Also present are signs and symptoms of the infarction and major changes in hemodynamics: shock, grayish cyanosis, cold clammy skin, tachycardia, distended neck veins, enlarged liver, and orthopnea. The perforation is often heralded by a fresh bout of precordial pain and usually with dramatic deterioration in the condition of the patient. Less frequently, as in our patient, the rupture is relatively silent with little immediate deterioration in the patient’s status. The differential diagnosis includes ruptured papillary muscle, ruptured chordae tendineae, relative mitral regurgitation due to cardiac dilatation, and friction rub.

Electrocardiograms have not shown characteristic changes after septal rupture. Since the septum is often massively infarcted, various arrhythmias have been noted before and after septal rupture. They include atrioven-

Figure 3

Pressure curves from catheterizations before and after surgery.

Figure 4

Drawing of the heart with the right ventricle opened showing: A. The method of closure of the defect, which passes obliquely upward through the septum. For purposes of illustration, the incised papillary muscle is shown arising from the septum. B. The repaired defect and reattached papillary muscle. C. The method of closure of the right ventriculotomy.
The most striking feature, the loud pansystolic murmur typical of ventricular septal defect, has been confirmed by phonocardiogram in several previous reports. Postoperative phonocardiograms of our patient document the disappearance of the murmur (fig. 2). Dye-output curves demonstrating the left-to-right shunt have been used to confirm the diagnosis previously. Our patient showed a typical dye curve for large left-to-right shunt before operation with return to a normal curve after closure of the defect (fig. 2).

The diagnosis is readily confirmed by cardiac catheterization. A summary of catheterization data from previous reports and from our patient is presented in table 1. All patients demonstrated marked step-up in blood oxygen saturation from right atrium to right ventricle. Calculated left-to-right shunts were moderately large with pulmonary flow approximately twice the systemic flow. In our patient, however, the shunt was unusually great, over four times the systemic flow. The majority of cases showed marked pulmonary hypertension indicative of left ventricular failure. This was confirmed in two instances by high pulmonary capillary "wedge" pressures. Some of the mild elevation of pulmonary pressures in our patient could well have resulted from the huge pulmonary flow. Two cases had completely normal pulmonary vascular pressures.

Life expectancy after rupture of the infarcted septum is very short. Oyamada and Queen reviewed over 200 patients with septal rupture and found survival data in 157. Of these, 24 per cent died on the first day, 65 per cent in the first 2 weeks, and 81.5 per cent in 8 weeks. Only 7 per cent lived a year or more. Similar data are published by other authors, a few patients, however, have lived from 2 years to more than 6½ years. Such survivors always had varying degrees of left and right ventricular failure.

Several attempts have been made to repair the defect surgically. Two prior attempts have been followed by prolonged survival. In 1957, Cooley et al. reported an open-heart
repair with an Ivalon sponge 2 months after septal rupture. The patient lived 6 weeks before dying of pericarditis. At autopsy the Ivalon was well epithelialized, but one suture had torn through the septal wall reopening the defect. A second repair by Cooley24 was attempted 4 days after infarction. At surgery the myocardium was very necrotic and friable, making insertion of a Dacron patch very difficult, and the patient did not survive the operation. Shickman et al.25 reported the repair of a defect by a closed-heart technic 8 weeks after infarction. The defect was closed satisfactorily, and the patient did well for a week, only to die of a fresh myocardial infarction. Rubenstein and Levinson26 reported a closed-heart repair 15 days after infarct and 6 days after septal rupture. The friable myocardium would not hold sutures, the septum tore again, and the patient died on the operating table. A patient of Effler’s reported by Proudfit et al.14 was operated upon 6 months after myocardial infarction by open-heart technic with direct suturing of a defect “the size of a nickel.” Detailed physiologic data are not available although postoperative cardiac catheterization showed a persistent defect of less magnitude than before operation. The patient was living at the time of the report 2 years after surgery. Gerbode, in discussing Cooley’s paper,24 reported the successful repair by open-heart technic of a septal defect in a patient who developed severe congestive failure 6 weeks after rupture. His patient was alive more than 2 years after surgery.27

Myocardial ruptures usually occur early after infarction before tissue repair is adequate. In Oyamada and Queen’s19 series, the time of perforation of the infarcted septum was established in 90 patients. Of these, 21 per cent occurred in the first day, 60 per cent in the first 5 days, and 88 per cent in the first 10 days. This correlates well with the time of rupture of the free ventricular wall after myocardial infarction.28, 29 During the first 4 days the infarcted area is infiltrated from the periphery with leukocytes; after the fourth day capillaries and fibroblasts grow into the area, with removal of the infarcted muscle fibers beginning in the second week. Collagen formation begins in the third week following infarction, and by the sixth week the scar is contracted with decreased vascularity and number of macrophages. By the eighth week collagen is at its maximum density.30

The combination of the high concentration...
of leukocytes and proteolytic enzymes at the periphery and focusing of stresses at the junction of healthy and infarcted tissues due to the paradoxical systolic movement of the infarcted area is considered the basis for the high incidence of rupture at the junction of healthy and infarcted muscle. Many ruptures are sinuous "dissections" through the wall rather than "blowouts."

It is obvious that satisfactory repairs can best be made on well-healed infarcts. Owing to the rapid development of intractable congestive heart failure, however, prolonged waiting may not be possible. From the pathologic data presented it seems advisable to wait at least 8 weeks after infarction for optimum healing. Gerbaux et al. recommended that 12 weeks be allowed for healing, if possible. Cooley et al. believe that 6 weeks is a minimum time, but 3 months is preferable. Two attempts during the first 2 weeks after infarction have ended in operative deaths, whereas our patient tolerated surgery well 5 weeks after myocardial infarction.

Too few cases are available to enable final recommendations to be made regarding surgery. In the early period after infarction when the septal defect causes a rapid deterioration in the condition of the patient, surgery may be the only life-saving procedure,
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despite its acknowledged high risks. This is emphasized by the almost universal failure of medical therapy alone in maintaining these patients for any significant period of time.

Summary

Successful surgical closure of a ruptured interventricular septum following myocardial infarction is reported. The patient developed the loud pansystolic parastrernal murmur characteristic of septal perforation, and cardiac catheterization confirmed an unusually large left-to-right shunt. The patient's course was not the typical rapid deterioration with shock or intractable heart failure, but when myocardial insufficiency developed insidiously 5 weeks postinfarction, surgical closure with a Teflon patch was accomplished during cardiac bypass.

Since early death ensues so frequently, surgical repair has been attempted in only a small number of instances; this patient is one of the very few with successful closure and long-term survival. The pathology, clinical course, and cardiac catheterization data of this entity are reviewed from previously reported cases.

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Addendum

Shortly after this manuscript was submitted the patient died unexpectedly. He had been asymptomatic and working full time as a powerhouse superintendent when he suddenly collapsed and died at his home 8 months after surgery.

Significant findings at autopsy were limited to the heart, which, with its firmly adherent pericardium, weighed 725 gm. The left ventricular wall was 17 mm. thick. There was an aneurysmal dilatation 7 cm. in diameter of the posterior wall of the left ventricle and posterior half of the interventricular septum, bulging 2 cm. into the right ventricular cavity. The septal portion of the aneurysm, 3 cm. in diameter, contained the area of surgical repair. The Teflon patch was well epithelialized and intact. A persistent defect at the anterior margin of the patch admitted a 1.5-mm. probe.

The coronary arteries were all sclerotic with narrowed lumina, and the right coronary was completely occluded from 1 to 5 cm. from its origin by an old thrombus containing areas of attempted recanalization.

Microscopic sections were consistent with the healed myocardial infarction and surgical repair. No new area of infarction was identified, and no anatomic cause for the sudden death was found. Figures 6 to 8 show views of the heart at autopsy.

References


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Three Principles

Not to take authority when I can have facts; not to guess when I can know; not to think a man must take physic because he is sick.—Oliver Wendell Holmes. The Quiet Art: A Doctor's Anthology. Compiled by Dr. Robert Coope. Edinburgh & London, E. & S. Livingstone Ltd., 1952, p. 101.
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