

Isolated ventricular septal defect caused by nonpenetrating trauma to the chest

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These ruptures happen oftenest from violent blows; then the individual who is attacked, passes from a state of perfect health to that of incurable disease, and in general soon mortal. . . .

—J. N. CORVISART, 1806 (1)

Nonpenetrating trauma to the chest often results in injury to the heart, which may vary in severity from immediately fatal cardiac rupture to asymptomatic cardiac bruises. Contusion of the myocardium, as evidenced by electrocardiographic abnormalities, has been the most common lesion found clinically, and rupture of the myocardium has been the most common injury found at autopsy following nonpenetrating chest trauma. The rupture usually involves the free wall of either or both ventricles and rapidly leads to death. On occasion, the ventricular septum is ruptured without perforation of a ventricular free wall. This report describes a patient who survived isolated rupture of the ventricular septum following blunt trauma to the chest and summarizes pertinent features of some cases that have been reported.

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A 24-year-old laborer was in excellent health until October 1960, when he was involved in an automobile accident. He struck his chest against the steering wheel after hitting the rear of a truck and was knocked unconscious. Upon awakening approximately 1 hour later, he heard and felt a “purring” sensation over his precordium and was hospitalized. The patient had had no previous history of cardiac disease, and no precordial murmur

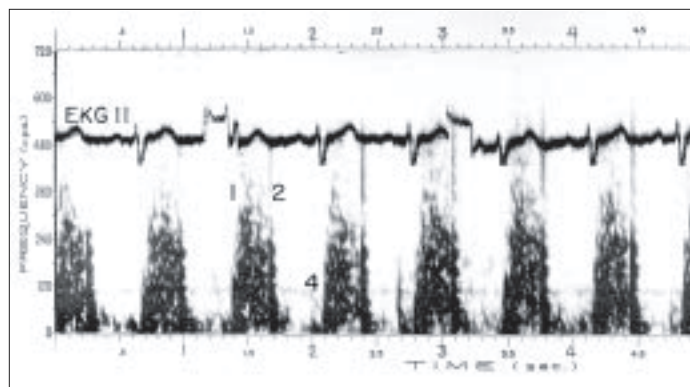


Figure 1. Spectral phonocardiogram recorded at the apex and lead II of the electrocardiogram taken at the first hospitalization in October 1960.

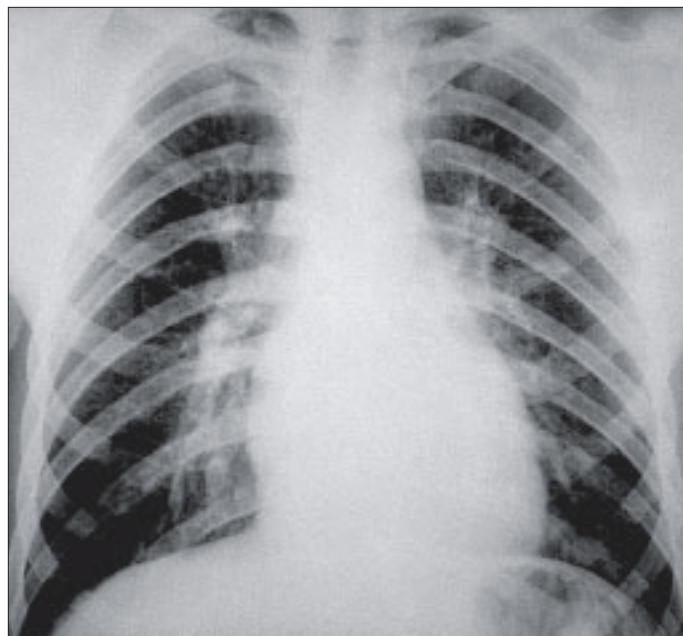


Figure 2. Chest radiograph at the first hospitalization in October 1960.

had ever been detected. His most recent physical examination had taken place in July 1959 upon discharge from the Armed Services.

On admission, several superficial cuts and bruises were present on his face, knees, and chest wall. His blood pressure was 110/75 mm Hg; heart rate, 80 beats per minute; and temperature, 38.4°C (101°F), which became normal after 3 days. Precordial examination disclosed a prominent systolic thrill and a grade 5/6 harsh holosystolic murmur, loudest along the left sternal border but audible over the entire anterior chest, back, and left axilla (Figure 1). A grade 1/6 presystolic rumble was heard along the lower left sternal border and at the apex. Admission chest radiograph (Figure 2) showed a normal-sized heart, an enlarged pulmonary arterial segment, increased pulmonary vascularity, and an incomplete fracture of the left fourth rib. Repeat chest radiograph 15 days later revealed an increase in the size of the cardiac silhou-

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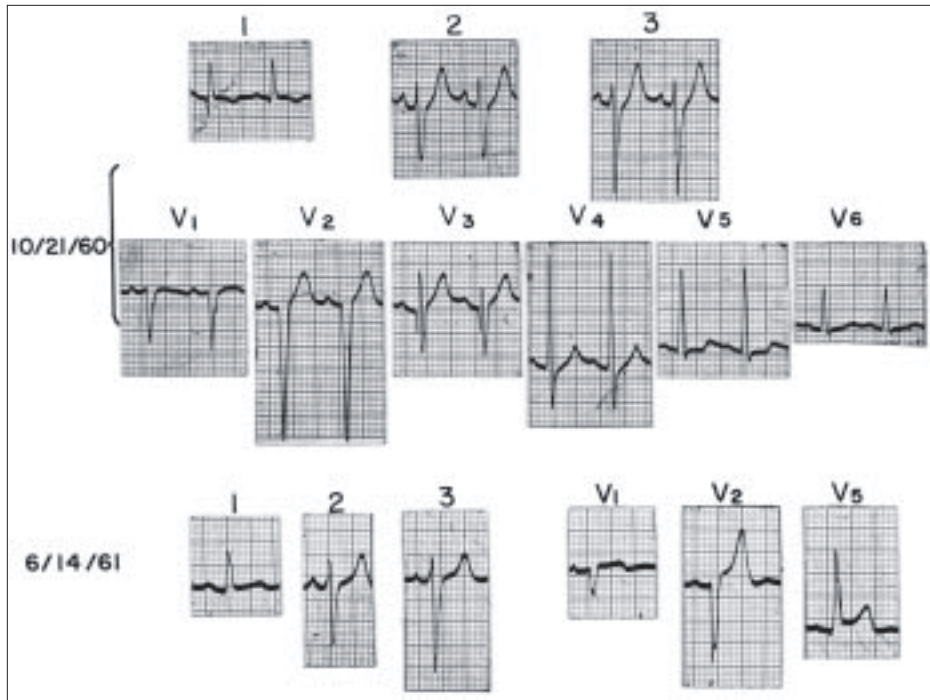


Figure 3. Electrocardiograms recorded in October 1960 and June 1961.

ette. Serial electrocardiograms (Figure 3) showed left axis deviation and changes compatible with anterior wall acute myocardial infarction. The hematocrit was 51%, and the white blood cell count, 13,500/mm³. Leukocytosis (13,500–19,000) remained throughout his 23 days in the hospital. The corrected erythrocyte sedimentation rate ranged from 28 to 34 mm in 1 hour. Over a 4-day period, aspartate aminotransferase levels changed from 58 to 70 and then to 17 units. Throughout the hospitalization, the patient continued to be aware of a noise in his anterior chest. At no time, however, did he have chest pain or signs or symptoms of congestive cardiac failure.

The patient was readmitted to the hospital in June 1961 for cardiac catheterization. During the 8-month interval he had worked as a hard laborer, lifting sheetrock on houses. At no time had he experienced dyspnea, chest pain, palpitations, or limitation of any sort. Physical examination was entirely unchanged. Chest radiograph revealed a slight decrease in cardiac size compared with the radiograph of November 1960. Repeat electrocardiogram continued to show left axis deviation and small R waves in the initial precordial leads, but the T waves in the precordial leads now had reverted to an upright position (Figure 3).

Right-sided cardiac catheterization disclosed a right ventricular pressure of 37/2 mm Hg, with an oxygen step-up from the right atrium to the right ventricle. No oxygen step-up was seen from the vena cavae to the right atrium. Several runs of ventricular tachycardia occurred when the catheter was positioned in the outflow tract of the right ventricle, and consequently the pulmonary trunk was not entered. Retrograde aortic catheterization disclosed a pressure in the left ventricle of 103/10 mm Hg. A left ventricular cineangiogram revealed early opacification of the right ventricle, compatible with a defect in the ventricular septum, and moderate regurgitation into a normal-sized left atrium, compatible with rupture of one or several mitral chordae tendineae. Because the left-to-right shunt was relatively small

and the patient was asymptomatic, surgical repair of the ventricular septal defect was not considered necessary, and he was discharged and subsequently lost to follow-up.

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Analysis of this patient and 28 reported patients (2–28) with isolated ventricular septal defect caused by blunt chest trauma disclosed the following: 25 (86%) were men; 28 (97%) were young (aged 5 to 38 years [mean, 19 years]), and the remaining patient, a woman, was 75 years old. The trauma in 17 (59%) involved an automobile, and in all but 2 the steering wheel compressed the anterior chest. Other types of trauma included a kick in the chest by a horse, a fall from a moving truck, a crush between a 3-ton bucket and a steam shovel, a fall of a heavy bookcase, a 20-m fall to the roof of a car, a crush under a cattle drop gate, a crush by an auto engine dislodging from its attachments, a forceful throw from a moving sled, a kick

in the chest, a crush by the fall of a heavy tree limb, and a fall from 9 floors.

A precordial murmur, systolic in time and loud in intensity, occurred in 25 (86%) of 29 patients, but it was not always present immediately. The time of appearance of the precordial murmur following the accident varied considerably: in 15 the murmur was audible immediately; in the others the murmur was absent initially but appeared anywhere from 6 to 10 days later; and in 1, it appeared 4 months later. When the murmur is delayed, it may be that the ventricular septum is initially only contused; the contused myocardium then becomes necrotic, sloughs, and finally ruptures. The presence of shock also may account for the absence of a precordial murmur immediately.

Abrasions or lacerations of the skin of the chest wall or fractures of the thoracic bones occurred in 17 (61%) of 28 patients in whom associated injuries were described. Rib fractures occurred in only 6 patients (21%), and 11 patients were completely free of injuries to the external chest. An electrocardiogram recorded in the immediate postinjury period in 24 patients was normal in 1 patient and abnormal in 23 (96%), showing changes of acute myocardial ischemia or infarction in 15 patients, non-specific ST-segment and/or T-wave changes in 4 patients, and conduction disturbances (bundle branch block [5 patients] or complete heart block [2 patients]) in 7 patients. Cardiac catheterization in 19 patients showed left-to-right shunts at the ventricular level in each and pulmonary hypertension (pulmonary systolic pressures, 37–70 mm Hg) in 9 patients.

The ventricular septal defect in each of the patients who underwent operation (15 patients) or autopsy (9 patients [2 died postoperatively]) was muscular in type in all but one and usually linear in shape, varying from 1.0 to 5.5 cm in longest diameter. Multiple defects in the ventricular septum occurred in 2 patients. The patients who died tended to have larger defects than those who underwent operative closure of the ventricular

septal defect. Most patients having operative closure of the defect were asymptomatic months after the procedure.

Since the traumatic septal perforations are nearly always of the muscular type, it is possible that these defects may close spontaneously. Eddying of the blood adjacent to the defects tends to produce local endocardial proliferation ("jet lesions"), and this fibrous thickening in itself may at times close defects of the muscular type. Of the 5 patients who survived nonpenetrating rupture of the ventricular septum as proved by cardiac catheterization and did not undergo operation, 4 were asymptomatic >8 months following the accident and the other one had only mild congestive cardiac failure 18 months later.

Since many of these patients do well with temporary medical assistance alone, operative closure of the defect appears to be warranted only in the acutely or progressively deteriorating patient. Even when operation is indicated, it would seem reasonable to delay the procedure for several weeks following the accident if possible to allow healing of the edges of the defect so that sutures can be well secured. These ventricular septal defects often can be closed directly without the aid of a patch.

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