Repair of Ventricular Septal Defect Following Myocardial Infarction *

TERRY DAVISON, M.D., GEORGE A. DEGENSHEIN, M.D.,
YUSUF Z. YUCEOGLU, M.D., ANDRES L. REYES, JR., M.D.,
BERNARD LEVOWITZ, M.D., DAVID T. DRESDALE, M.D.,
ADRIAN KANTROWITZ, M.D., F.A.C.S.

From the Departments of Medicine and Surgery, Maimonides Hospital, Brooklyn, N. Y., and the Departments of Medicine and Surgery, State University of New York Downstate Medical Center, Brooklyn, N. Y.

THE ANTE-MORTEM diagnosis of ruptured ventricular septum is becoming more common and, with the advent of extracorporeal circulation, surgical correction of this defect has been attempted in a number of patients.

Latham [19] is credited with the first pathologic description of a ruptured ventriculum septum (1846). The first ante-mortem diagnosis was made by Brunn [6] 77 years later. Sager's comprehensive review [29] of the world literature in 1934 revealed 18 cases, while Lee [21] in 1962 reported 220 cases.

Documentation of each successful repair of a ruptured septum is an important step toward recognition of clear-cut indications for the procedure. The purpose of this paper is to review the recent literature and to describe successful repair in a patient who had intractable heart failure for 11 months.

Case Report

A 53-year-old white man was admitted to another hospital on January 9, 1962, complaining of severe substernal chest pain of several hours' duration associated with profuse perspiration, general weakness, and shortness of breath. Findings of the physical examination were essentially normal. Blood pressure was 120/70 mm. Hg; no cardiac murmurs were heard. The electrocardiogram was consistent with an acute posterolateral wall infarction; the serum transaminase level was 84 units. Anticoagulants were withheld because of the patient's previous history of hematemesis. Therapy included complete bed rest for 20 days after which the patient was allowed to sit in a chair. He was discharged on February 20, 1962.

The patient was fairly well until April, 1962, when he developed ankle edema, external dyspnea, orthopnea, and ascites and was readmitted to the hospital. Physical examination revealed a blood pressure of 120/70 mm. Hg; pulse 80 per minute and regular. A harsh holosystolic murmur was heard along the left sternal border, loudest at the 4th and 5th intercostal spaces. A systolic thrill was easily palpable. The heart was enlarged to the left. The liver was palpable 4 cm. below the right costal margin. The electrocardiogram was unchanged. Perforated ventricular septum was suspected. Intractable right- and left-sided heart failure persisted despite vigorous diuretic therapy and digitalization.

On September 9, 1962 the patient was transferred to Maimonides Hospital. The electrocardiogram revealed the old posterolateral wall infarction (Fig. 1). A protodiastolic gallop at the apex was noted in addition to the previously described murmur and thrill. The neck veins were markedly distended. The liver was palpable 6 cm. below the right costal margin. The venous pressure was 28 cm. of water and the Decholin circulation time, arm to tongue, was 24 seconds. The patient was maintained on Digoxin, 0.5 mg. daily; salt and fluids were sharply restricted. Mercurial diu-

* Submitted for publication August 1, 1963.

A film of the operation, prepared with the aid of U. S. Public Health Service Grant H-6510, is available.

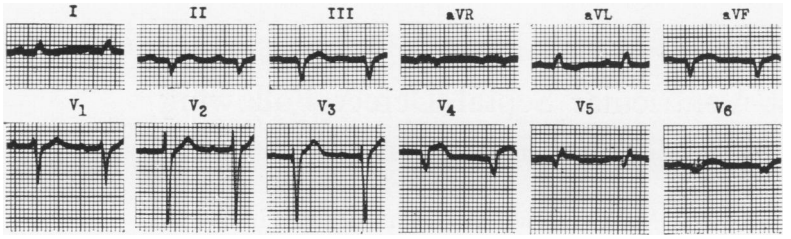

FIG. 1. Electrocardiogram on patient's admission to Maimonides Hospital showing evidence of old posterolateral wall infarction.

retics, alternated with L-lysine monohydrochloride, Aldactone, and thiazide derivatives reduced his weight by 57 lb. within 84 days. A phonocardiogram showed a loud pan-systolic murmur (Fig. 2). Cardiac catheterization on October 11, 1962, revealed a left-to-right shunt of 4.8 liters per minute, and a slight right-to-left shunt at the ventricular level. The data were consistent with a high degree of pulmonary hypertension, right and left ventricular failure, and tricuspid insufficiency (Table 1). Surgical correction of the septal defect was advised.

Surgical Procedure. The operation was performed on December 5, 1962, by three of us (A. K., B. L., and G. A. D.). A median sternotomy was made, and the pericardium was opened. A marked thrill was palpated over the lower portion of the right ventricle. An aneurysm 4 cm. in diameter was identified in the left ventricle, adhering closely to the pericardium laterally and posteriorly. The vena cavae and a previously exposed femoral artery were cannulated. A sump drain was placed in the left atrium. With the patient on total cardiopulmonary bypass, the field was flooded wtih CO_2 and a right ventriculotomy was carried out. A single defect 2.5 cm. in diameter was found anteriorly near the apex (Fig. 3). The ventricular septum was replaced by firm

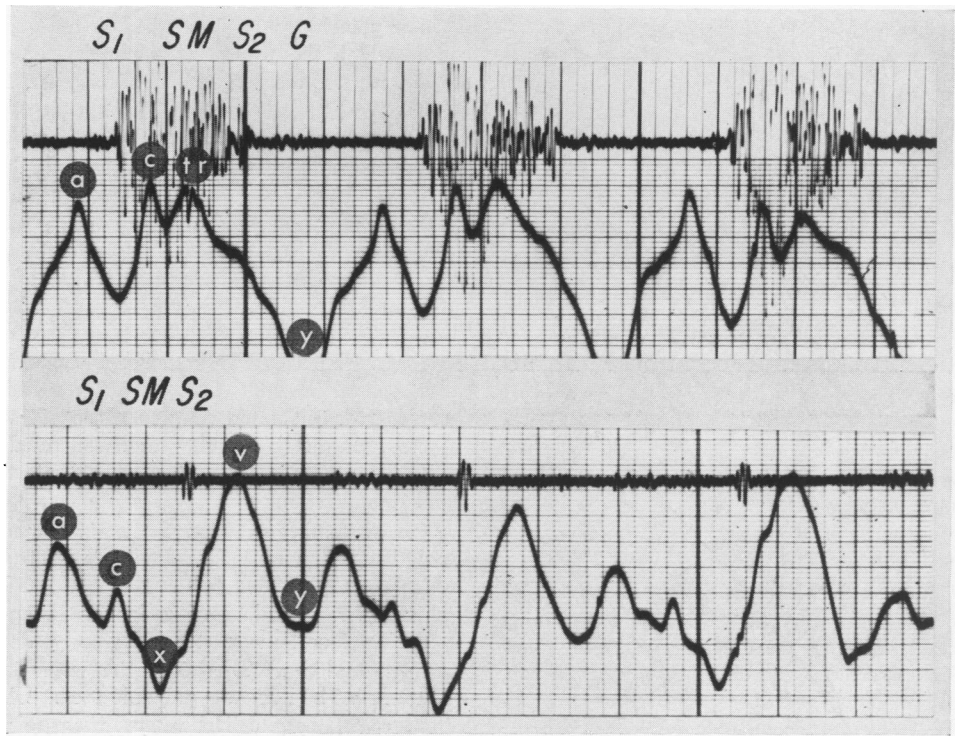

FIG. 2. Phonocardiograms taken at 4th intercostal space simultaneously with jugular pulse tracing. (Top) Pansystolic murmur (SM) with protodiastolic gallop (G) and marked tricuspid regurgitation (tr) seen preoperatively. (Lower) Following surgery, murmur had virtually disappeared, tricuspid regurgitation and gallop rhythm were no longer evident, and intensity of second sound (S_2) had diminished.

TABLE 1. *Hemodynamic Data Before and After Operation*

	Oxygen Content (Vol %)			Pressure (mm Hg)	
	Before	After		Before	After
SVC, average	8.80	11.90			
IVC, average	9.30		Right atrium	(14)	(−1.0)
Right atrium, low	10.25				
Right atrium, mid	10.40	11.70	Right ventricle	81/14	25/0
Right atrium, high	10.70				
Right ventricle, inflow	12.40	11.70	Pulmonary artery	81/29 (44)	23/8 (11)
Right ventricle, mid	14.90	11.80			
Right ventricle, outflow	14.60	11.90	*Pulmonary capillary*	(21)	(4)
Pulmonary artery	14.00	11.90			
Pulmonary capillary	92%*		Brachial artery	114/83 (91)	122/71 (90)
Brachial artery	16.30 87%*	16.00 95%			

	Flow Data (L/min)			Resistance (dynes/sec/cm^{-5})	
	Before	After		Before	After
Systemic blood flow	3.32	5.27			
Cardiac index	2.02	3.31	Systemic	2,195	1,365
Pulmonary blood flow	9.12	3.31	Total pulmonary	385	150
Left to right shunt	5.80	none	Pulmonary arteriolar	195	90

SVC, superior vena cava; IVC, inferior vena cava.
* By oximetry, samples taken simultaneously; ( ) mean pressure.

fibrous tissue. Direct approximation of the defect margins with sutures was impracticable because fibrosis had made the perifistular tissue rigid. An appropriate Teflon felt patch was applied with two continuous 3-0 silk sutures (Fig. 4). The left atrial sump drain was clamped before the last suture was closed, and blood was allowed to well up in the left ventricle to eliminate any remaining air or carbon dioxide. The ventriculotomy incision was closed, and the patient was taken off bypass. At no time did the heart cease to beat. The total time on bypass was 27 minutes. When the heart had been sutured the thrill was no longer palpable. After decannulation and opening of the left pleural space to drain effusion fluid, chest tubes were inserted bilaterally and the chest was closed. The sternum was approximated with wire threaded through punch holes. As the last skin sutures were placed the patient was conscious, moderately coherent, and moving actively.

The postoperative course was uneventful. The patient was ambulatory on the second day. Edema and hepatomegaly abated, and he showed marked clinical improvement. The murmur was no longer audible. He was discharged on the 29th postoperative day in good condition.

When re-admitted for cardiac catheterization on March 26, 1963, the patient said he was able to carry on normal activities and had experienced no dyspnea even after walking several blocks or climbing a flight of stairs. Catheterization data (Table 1) showed no evidence of a shunt and the restoration of normal hemodynamics. The phonocardiogram revealed no murmur (Fig. 2B).

Discussion

Etiology. Although various investigators have reported that 8 per cent[21] to 9 per

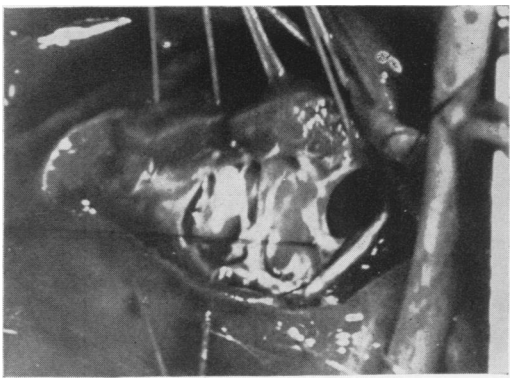

FIG. 3. Ventricular septal defect, located near apex, has fibrotic rim.

FIG. 4. Final sutures secure Teflon patch.

cent[13] of all deaths from myocardial infarction are due to rupture of the heart, and that the septum is involved in 70 per cent of these cases,[24] perforation of the ventricular septum is uncommon because of its satisfactory collateral circulation. Septal rupture is believed to have occurred in 1 per cent[21] to 2 per cent[11] of patients whose death was attributed to acute myocardial infarction.

Septal rupture generally occurs four to 12 days (an average of seven days[11]) after acute myocardial infarction. These figures correlate closely with the progression of necrotic and degenerative changes in the infarcted area as described by Mallory.[23]

Among factors that may contribute to septal perforation following myocardial infarction are: 1) massive necrosis, 2) hypertension, 3) undue physical activity, and 4) anticoagulant therapy. Edmonson's findings[13] suggest a higher incidence of myocardial rupture in patients with blood pressure of 140/90 mm. Hg or above. Bean[2] noted that 81 in a series of 179 patients experiencing myocardial infarction and rupture had a history of previous hypertension. The patient whose case is presented here had no history of hypertension.

Beresford and Earl[3] as well as Jetter and White[18] observed a high incidence of spontaneous cardiac rupture among mentally ill patients, and felt that continued physical activity following infarction might be a contributing factor.

According to Lee and O'Neil,[20] the incidence of myocardial rupture complicating acute infarction was five times higher in a series of anticoagulant-treated patients than in those not so treated. On the other hand, Maher[22] found no significantly increased incidence in the group given anticoagulant therapy.

Pathology. Thrombosis in the left coronary artery occurs at least three times as often as in the right.[17] Since the left coronary artery supplies the anterior two-thirds of the septum, that portion is most commonly infarcted.

Among 82 cases of ruptured ventricular septum reported by Swithinbank,[33] "the perforation site was in the lower part in 66 per cent, in the posterior part in 17 per cent, in the middle part in 13 per cent, and in the superior part in only 4 per cent."

The perforations vary greatly in size and may be single or multiple. Lee,[21] in an analysis of more than 200 cases of ruptured ventricular septum, found multiple perforations in 80 (40%). They varied from a few millimeters to 3 cm. in diameter, averaging 1.0 cm.

Clinical Features. The age range and sex distribution of patients with ruptured ventricular septum correspond to those of other patients with myocardial infarction. The patient's condition usually deteriorates suddenly during the course of infarction. The most striking clinical feature is the onset of a loud, harsh, holosystolic murmur to the left of the sternum, best heard at the 4th and 5th intercostal spaces. In 66 per cent of the cases reviewed by Swithinbank[33] this murmur was accompanied by a systolic thrill palpable in the same area. The intensity of such a murmur appears to depend largely upon the size of the rupture. Severe pulmonary hypertension may foreshorten the murmur.

Ruptured ventricular septum is often associated with pain and, usually, with peripheral circulatory failure. Signs of right-heart failure tend to appear suddenly

or, if present, tend to become more pronounced. Nausea, vomiting, and abdominal tenderness are at times noted, in which case both intra-abdominal pathology and pulmonary embolism must be ruled out. If the communication is small, cardiovascular dynamics may not be significantly affected.

A mid-diastolic murmur, which is occasionally heard, has been ascribed by some investigators to a septal aneurysm.[16, 24, 34] Lee[21] attributed this rumbling murmur to increased flow into the left ventricle during diastole as a result of increased pulmonary blood flow.

Differential Diagnosis. The diagnosis is confirmed by cardiac catheterization and demonstration of a left-to-right shunt at the ventricular level. The sudden appearance of a loud, harsh, systolic murmur in a patient with a recent myocardial infarction may suggest three other possibilities: 1) rupture of a papillary muscle, 2) a pericardial friction rub, or 3) functional mitral insufficiency.

The clinical features of a ruptured papillary muscle and ventricular septal perforation are sufficiently similar to make differential diagnosis difficult. The murmur associated with a ruptured papillary muscle is usually musical, diastolic, loudest at the apex rather than at the left sternal border, and is transmitted into the axilla. Askey[1] stressed the absence of a thrill in papillary-muscle rupture, whereas Swithinbank[33] reported a palpable systolic thrill in two-thirds of the cases of septal perforation. The posterior papillary muscle is usually the site of the rupture, and the myocardial infarction is posterior as a rule. The sudden, severe mitral insufficiency usually produces intractable left-heart failure which is fatal within a short time.

A pericardial friction rub may be located over the left sternal border but can usually be differentiated from a septal perforation by its transient nature and by the auscultatory findings.

Acute dilatation of the left ventricle produces the systolic murmur of mitral insufficiency, which has been noted in about 38 per cent of patients with acute infarction.[30] Less intense and lower in pitch than the murmur associated with septal rupture, it is located nearer the apex and commonly radiates into the axilla.

Electrocardiographic Pattern. Thus far no typical electrocardiographic pattern has been observed in patients with ruptured ventricular septum. Myers[25] believes that the following features point to such a diagnosis: 1) a QS configuration in the right precordial leads without evidence of right ventricular hypertrophy; 2) a QS configuration in the right precordial leads with either a) S-T segment changes in these leads, b) normal R waves farther to the right, or c) abnormal waves farther to the left; and 3) a Q_3-T_3 pattern with right precardial changes typical of infarction. Among various arrhythmias noted before and after septal rupture are all degrees of atrioventricular block, right and left bundle-branch block, and atrioventricular nodal rhythm.[26, 33]

Clinical Course. The prognosis for patients with a ruptured septum is very poor. More than 50 per cent die within the first week; about 13 per cent survive for two months.[30] Oyamada and Queen[26] found that only 7 per cent of more than 200 medically treated patients were living at the end of the first year, and all had varying degrees of left and right ventricular failure.

Surgical Considerations. The first attempt to repair a ruptured ventricular septum was made by Cooley[8] in 1957. This and the other surgical experiences that could be found are briefly described in Table 2.

It is evident that criteria for surgical correction must still be established. Cardiac catheterization is of prime importance; repair should probably not be attempted unless a significant left-to-right shunt is demonstrated. Another critical prerequisite is assiduous medical care during the pre-

TABLE 2. *Data on Patients with Repair of Ruptured Ventricular Septum*

	Date of Oper.	Age	Infarct to Rupture	Rupture to Operation	Type of Incision	Location and Size of Defect
Cooley[8]	4/ 5/56	49	5 days	66 days	Bilat. anterior thoracot.	Posteroseptal 3 × 2 cm.
Cooley[9]	11/ 7/58	47	4 days	1 day		Anterior, 3 cm.
Effler[14]		54	6 mo.			Low posterior 1.5 cm.
Gerbode[15]		56		6 weeks		
Payne[27]		60	7–14 days	13 mo.		Anterior, 0.9 cm.
Shickman[31]		61	53 days		Bilat. anterior thoracot.	Mid-postero-septal 1.5 cm.
Rubenstein[28]			9 days	6 days		
Dennis[10]	3/16/61	60	5 days	5 days	Median sternot.	"large"
Dobell[12]	5/10/61	61	14 days	101 days	Median sternot.	Anterior, 2 cm.
Boicourt[4]	7/5/61	57	10–27 days?	9–26 days?	Sternal split	Diaphrag. surface of septum
Bressie[5]	9/20/61	61	<10 days	Approx. 6 mo.	Median sternot.	Apical 2 × 1.5 cm.
Collis[7]	2/8/62	49	17 days	91 days	Median sternot.	Apical 3 × 1.5 cm.
Kantrowitz	12/5/62	51	12 days?	Approx. 11 mo.	Median sternot.	Anterior, 2.5 cm.

operative period. The value of painstaking, aggressive treatment of patients in chronic failure cannot be overstressed.

Of the 13 operated cases (including those treated within a few weeks of infarction), three patients had survived two years, one 19 months, two others almost six months when reported. One died at eight months and the other six within 32 days. The one-year survival rate in the operated group was about 31 per cent, compared with 7 per cent in the medically treated cases.[26] The absence of congestive failure in the successfully operated group is noteworthy.

Attempts to repair the defect during the first few weeks following rupture have failed. It is difficult to define the area of necrosis, and sutures tend to disrupt in the friable septum. It is now generally agreed that repair should be deferred for at least two or three months to permit scar forma-

TABLE 2—(*Continued*)

Min. on Bypass	Type of Repair	Aneurysm	Shunt	Survival	Comment
	Ivalon patch 3–0 silk	Present not repaired	L to R	45 days	Pericarditis at P.M. 0.5 cm. opening at suture site.
	Dacron patch	None			Died on table.
	Direct suture	None	L to R	>2 yrs. when reported	Catheterization 1 yr. postop. showed shunt.
60				>2 yrs. when reported	
	Direct suture, silk		L to R	2½ yrs. when reported	No failure, no angina 2½ years.
Closed method	4 sutures septum to R.V. wall	None	L ro R	1 week	P.M.–fresh myocardial infarction.
					Sutures disrupted: died on table.
N.B.*	2-0 silk with Ivalon mattresses		"large"		Died on table 2 hrs. later.
	Teflon patch	Noted in L.V. at P.M.	L to R	32 days	P.M.–cause of death uncertain.
72	Teflon patch, 3–0 silk	Noted in L.V. at P.M.	14.4 L L to R	8 mo.	Autopsy inconclusive, 1.5 mm. opening at anterior patch margin.
38	Direct suture 3–0 silk	None	L to R	22 mo. to date	No shunt 8 mo. postop.
21 total 71 partial	2 layers 5–0 silk	Repair via 8 × 5 cm. aneurysm (excised)	L to R	22 weeks when reported	
27	Teflon patch	Present, not repaired	4.8 L L to R	>8 mo. when reported	Well and active.

* N.B. Patient, who developed peripheral circulatory collapse shortly after admission, had been supported on left-heart bypass 12 hours when right-heart bypass was also instituted and ventricular septal defect was closed. Sutures disrupted; attempts at repair unsuccessful.

tion and delineation of the infarcted area. Current experimental work on extracorporeal support of the myocardium during infarction [32] suggests the future possibility of salvaging some of the patients now being lost during the first two months (an estimated 87%) so that they can undergo surgery later.

Although Shickman [31] has reported a closed method, it is felt that repair utilizing bypass is more effective. Direct suture is desirable wherever possible. In three of the *long-term* survivors (Table 2) closure was effected with silk sutures. Dacron, Teflon, and Ivalon patches have been inserted. Dobell [12] stressed the importance of placing

the patch against the left side of the septum with a rim to withstand pressure.

In the case reported here, the patient had a ventricular aneurysm with a paradoxical pulsation—a frequent complication of ruptured ventricular septum. In two other instances (Table 2) the aneurysm was discovered postmortem. Further experience may point to the advisability of repairing the aneurysm at the same time. Collis [7] carried out ventriculotomy through an aneurysm, the walls of which were excised after closure of the septal defect. With the development of new technics in cardiovascular surgery, a method of improving the myocardial vasculature may ultmately be included in the surgical procedure.

Summary

1. The successful repair of a ruptured ventricular septum following myocardial infarction is reported.

2. Assiduous preoperative medical management contributed markedly to the successful outcome.

3. A review of the literature indicates the advisability of delaying the procedure for at least two months to permit scarring.

Acknowledgment

The authors are indebted to Drs. Ludwig W. Eichna and Clarence Dennis of State University of New York Downstate Medical Center, and to Drs. David Grob and Alfred Hurwitz of Maimonides Hospital for valuable suggestions during preparation of this paper.

References

1. Askey, J. M.: Spontaneous Rupture of a Papillary Muscle of the heart. Amer. J. Med., **9**:528, 1950.
2. Bean, W. B.: Infarction of the Heart. II. Symptomatology of the Acute Attack. Ann. Intern. Med., **11**:2086, 1938.
3. Beresford, E. H. and C. J. C. Earl: Spontaneous Cardiac Rupture–A Review of 46 Cases. Quart. J. Med., **24**:55, 1930.
4. Boicourt, O. W., L. W. Ritzmann, J. D. Chase, A. Starr and C. W. McCord: Rupture of the Interventricular Septum. Circulation, **26**: 1321, 1962.
5. Bressie, J. L., D. D. Snyder, G. R. Williams, R. H. Smiley and G. S. Campbell: Ruptured Interventricular Septum–Successful Repair After Myocardial Infarction. J.A.M.A., **182**: 1042, 1962.
6. Brunn, F.: Zur Diagnostik der erworbenen Ruptur der Kammerscheidewand des Herzens. Arch. Inn. Med. (Wien), **6**:533, 1923.
7. Collis, J. L., J. Mackinnon, J. C. A. Raison and S. R. F. Whittaker: Repair of Acquired Interventricular Septal Defect Following Myocardial Infarction. Lancet, **2**:172, 1962.
8. Cooley, D. A., B. A. Belmonte, L. B. Zeis and S. Schnur: Surgical Repair of Ruptured Interventricular Septum Following Acute Myocardial Infarction. Surgery, **41**:930, 1957.
9. Cooley, D. A., W. S. Henly, K. H. Amad and D. W. Chapman: Ventricular Aneurysm Following Myocardial Infarction–Results of Surgical Treatment. Ann. Surg., **150**:595, 1959.
10. Dennis, C., E. Carlens, A. Senning, D. P. Hall, J. R. Moreno, R. R. Cappelletti and S. A. Wesolowski: Clinical Use of a Cannula for Left Heart Bypass Without Thoracotomy. Experimental Protection Against Fibrillation by Left Heart Bypass. Ann. Surg., **156**:623, 1962.
11. Dias-Rivera, R. S. and A. J. Miller: Rupture of the Heart Following Acute Myocardial Infarction–Incidence in a Public Hospital with Five Illustrative Cases Including One of Perforation of the Interventricular Septum Diagnosed Ante-Mortem. Amer. Heart J., **35**:126, 1948.
12. Dobell, A. R. C., H. J. Scott, R. F. P. Cronin and E. A. S. Reid: Surgical Closure of Interventricular Septal Perforation Complicaitng Myocardial Infarction. J. Thorac. Cardiov. Surg., **43**:802, 1962.
13. Edmonson, H. A. and H. J. Hoxie: Hypertension and Cardiac Rupture–A Clinical and Pathologic Study of 72 Cases in 13 of Which Rupture of the Interventricular Septum Occurred. Amer. Heart J., **24**:719, 1942.
14. Effler, D. B.: Reported in discussion of paper "Rupture of the Ventricular Myocardium Complicating Acute Myocardial Infarction" by W. L. Proudfit, F. A. Tarpia, L. J. McCormack and D. B. Effler (Circulation, **20**:128, 1959).
15. Gerbode, F.: Reported in discussion of paper "Ventricular Aneurysm Following Myocardial Infarction–Results of Surgical Treatment" by D. A. Cooley, W. S. Henly, K. H.

Amad and D. W. Chapman (Ann. Surg., **150**:595, 1959).

16. Gross, H. and S. P. Schwartz: A Case of Acquired Interventricular Septal Defect Associated with Long-Standing Congestive Heart Failure. Amer. Heart J., **11**:626, 1936.
17. Hochrein, M.: Der Myokardinfarkt; Erkennung, Behandlung und Verhuetung. Kreislaufbucherei (Deutsche Gesellschaft fur Kreislaufforschung). Band I., ed. 2, Dresden and Leipzig, Theodor Steinkopff, 1941, p. 278.
18. Jetter, W. W. and P. D. White: Rupture of the Heart in Patients in Mental Institutions. Ann. Intern. Med., **21**:783, 1944.
19. Latham, P. M.: Lectures on Subjects Connected with Clinical Medicine Comprising Diseases of the Heart. Vol. 2, London: Longman, Rees, Orme, Brown, Green, and Longmans, 1845–46.
20. Lee, K. T. and R. M. O'Neil: Anticoagulant Therapy of Acute Myocardial Infarction. Amer. J. Med., **21**:555, 1956.
21. Lee, W. Y., L. Cardon and S. J. Slodki: Perforation of Infarcted Interventricular Septum. Arch. Intern. Med., **109**:731, 1962.
22. Maher, J. F., G. K. Mallory and G. A. Laurenz: Rupture of the Heart After Myocardial Infarction. New Eng. J. Med., **255**:1, 1956.
23. Mallory, G. K., P. D. White and J. Salredo-Salgor: The Speed of Healing of Myocardial Infarction–A Study of the Pathologic Anatomy in 72 Cases. Amer. Heart J., **18**:47, 1939.
24. Moolten, S. E.: Prolonged Survival After Perforation of the Infarcted Interventricular Septum in Coronary Arterial Disease. Arch. Intern. Med., **69**:108, 1942.
25. Myers, G. B., H. A. Klein and T. Hiratzka: Correlation of Electrocardiographic and Pathologic Findings in Infarction of the Interventricular Septum and Right Ventricle. Amer. Heart J., **37**:720, 1949.
26. Oyamada, A. and F. B. Queen: Spontaneous Rupture of the Interventricular Septum Following Acute Myocardial Infarction with Some Clinico-Pathological Observations on Survival in Five Cases. Presented at Pan Pacific Pathology Congress, Tripler U. S. Army Hospital, October 12, 1961.
27. Payne, W. S., J. C. Hunt and J. W. Kirklin: Surgical Repair of Ventricular Septal Defect Due to Myocardial Infarction. J.A.M.A., **183**: 603, 1963.
28. Rubenstein, P. and D. C. Levinson: Acquired Interventricular Septal Defects Due to Myocardial Infarction and Nonpenetrating Trauma to the Chest. Amer. J. Cardiol., **7**: 277, 1961.
29. Sager, R. V.: Coronary Thrombosis–Perforation of the Infarcted Interventricular Septum. Arch. Intern. Med., **53**:140, 1934.
30. Sanders, R. J., W. H. Kern and S. G. Blount, Jr.: Perforation of the Interventricular Septum Complicating Myocardial Infarction. Amer. Heart J., **51**:736, 1956.
31. Shickman, M. D., J. Fields and M. L. Pearce: Repair of Ruptured Interventricular Septum Complicating Acute Myocardial Infarction. Arch. Intern. Med., **103**:140, 1959.
32. Stuckey, J. H., M. M. Newman, C. Dennis, E. H. Berg, S. E. Goodman, C. C. Fries, K. E. Karlson, M. Blumenfeld, S. W. Weitzner, L. S. Binder and A. Winston: The Use of the Heart-Lung Machine in Selective Cases of Acute Myocardial Infarction. Surg. Forum, **8**:342, 1957.
33. Swithinbank, J. M.: Perforation of the Interventricular Septum in Myocardial Infarction. Brit. Heart J., **21**:562, 1959.
34. Zucker, R., S. Leibowitz, H. Brody and R. M. Sussman: Perforation of the Interventricular Septum. A.M.A. Arch. Intern. Med., **89**:899, 1952.