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PERFORATION OF THE INTERVENTRICULAR SEPTUM FOLLOWING MYOCARDIAL INFARCTION

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Perforation of the ventricular septum may be classified into two groups:—

(1) Congenital and

(2) Acquired.

The congenital form which is commonly known as ventricular septal defect (V.S.D.) is seldom missed on clinical examination and is usually consistent with life.

The acquired form of ventricular septal defect may be secondary to trauma, ulcerative bacterial endocarditis or myocardial infarction with infarction of the interventricular septum. The acquired form of ventricular septal defect is usually fatal and the patient dies of the primary disease rather than from the perforation of the ventricular septum. Though the clinical features of septal perforation following myocardial infarction have been well described the majority of reported cases were not diagnosed until after death.

We describe here two cases of myocardial infarction with perforation of the interventricular septum diagnosed during life and confirmed at autopsy.

CASE REPORTS

Case 1

A lorry-driver, S.K.E., aged 45, was admitted to the Medical Ward in Tan Tock Seng Hospital after an attack of severe retrosternal pain 5 days prior to admission followed by breathlessness and oilguria. He had never been breathless before, and had always enjoyed good health.

Physical Examination

The patient was well-developed, orthopnoeic and slightly febrile (Temp. 99.5°F.). There was no oedema or clubbing of the fingers and he was not anaemic. His fingers and toes were mildly cyanotic. He was sweating profusely and his extremities were cold. There was a tinge of jaundice in his sclera. The jugular venous pressure was raised. The radial pulses were weak and the pulse rate was 100/min. Blood pressure was 60/40 Hg.mm. All the peripheral pulses were felt. The apex-beat was in the 5th left intercostal space in the mid clavicular line. There was no thrill. The heart sounds in all areas were soft, and a pan-systolic murmur of grade 3 intensity was heard in all areas, but the murmur was most marked in the 4th left parasternal area. There were bilateral coarse crepitations in the lungs. The liver was enlarged and it was felt 3 fingers' breadth below the right subcostal margin. The other system was normal.

Investigations

Hb. 87% W.B.C. 14,000 N.81, L.12, E.2, M.5%.

B.S.R. 30/60 mm.

Urine examination showed 100-150 R.B.C. per field, 20-30 W.B.C. per field, albumin + and sugar nil.

Liver function tests: Total protein 6.8 gm.% albumin 5.1 gm.% and globulin 1.7 gm.%.

Thymol turbidity 2 units.

Alkaline phosphatase 15.0 units.

Blood Kahn Test was positive (123).

Blood for S.G.O.T. was unfortunately haemolysed.

The electrocardiograph showed deep Q waves in V_1 to V_5 with raised S-T segments in V_2 to V_4 and inverted T waves in V_1 to V_3 (Fig. 1). Portable X-ray of chest showed a top-normal heart with congestion of the lung bases. Calcified spots were noted in the right upper zone (Fig. 2). Portable X-ray of the abdomen showed two rounded radio-opaque shadows in the gall-bladder region.

Clinical Diagnosis

Myocardial infarction with an acquired ventricular septal defect and congestive cardiac failure.

Clinical Course

In spite of the treatment with Morphia, Digitalis, Nor-Adrenaline and Diuretics, the patent remained orthopnoiec, sweating and restless. He died about 26 hours after admission.

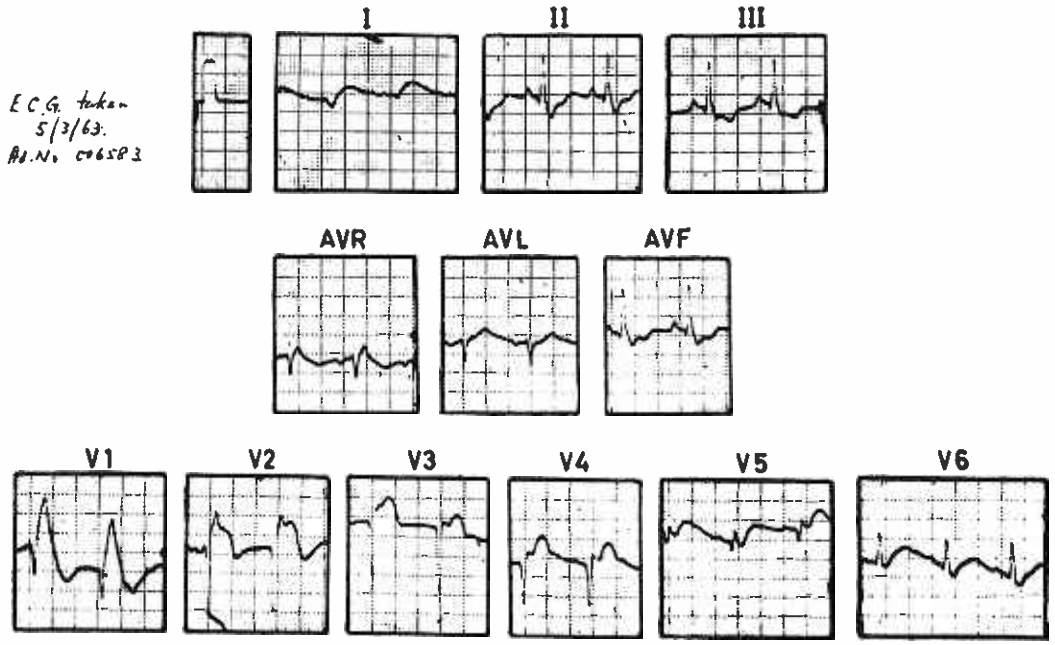

Fig. 1. E.C.G. shows deep Q waves and raised S-T segments from V_1 to V_4.

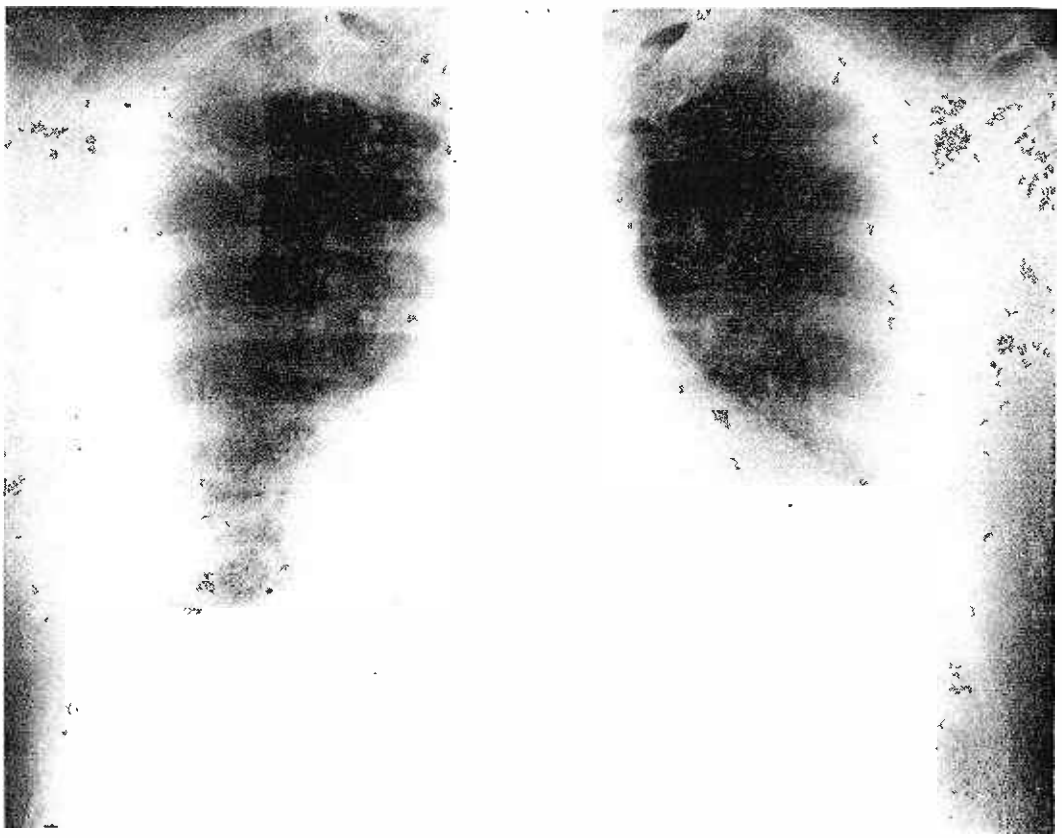

Fig. 2. Chest X-ray shows bi-ventricular enlargement with congestion of the lung bases and calcifications of the right upper zone.

NECROPSY (Dr. K. Sugai, M.B.,B.S., M.D.)

Cardiovascular System

Heart weighed 495 grams. Epicardium of lower-third of left ventricular anterior and lateral walls were hyperaemic, rough and surfaced with yellow areas. Left ventricle dilated. Myocardium showed diffuse yellow soft lesions 7 x 7 x 0.6 cm. involving lower two-thirds of interventricular septum, anterior and lateral walls of left ventricle. There was a longitudinal complete rupture, 3.5 x 0.6 cm. in lower portion of interventricular septum (Fig. 3). The corresponding endocardium of above myocardial necrotic lesion greyish yellow and slightly depressed. Valves — normal. Coronary arteries — left anterior descending branch showed fresh thrombus formation with complete occlusion of lumen for a distance of 3.5 cm. at the point immediately below its orifice (Fig. 4). Left circumflex branch and right coronary artery showed in proximal portions partially calcified atheroma with slight narrowing of lumen. Aorta showed slight degree of fatty streaks.

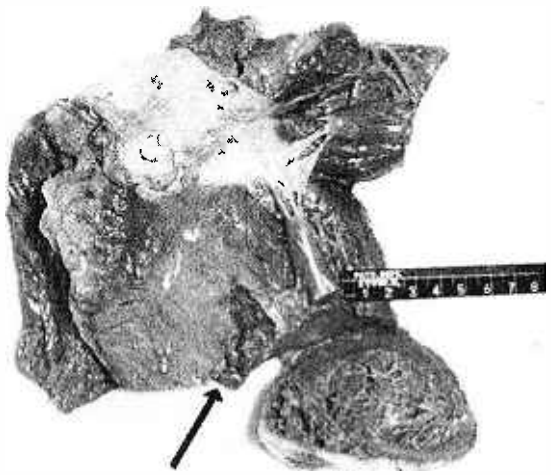

Fig. 3. Shows the interventricular rupture near the apex. (Arrow).

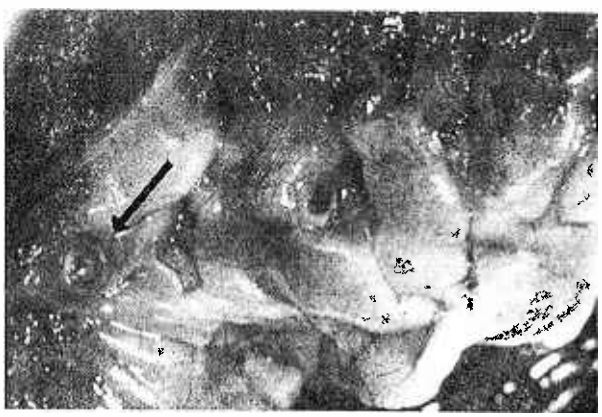

Fig. 4. Shows the lumen of the left coronary artery completely obliterated by the thrombus. (Arrow).

Respiratory System

Left lung weighed 665 gms.; right lung 815 gms. There were numerous fibrocaseous foci in apical portion of right lung. Both lungs congested and oedematous.

Digestive System

Liver weighed 1848 gms. Smooth, thin, tense capsules. Cut surface showed mottling of yellow and reddish-brown and firm parenchyma with uniform lobules.

Gall bladder contained 2 pigment calcium stones with rough surface averaging 1 x 1 0.7 cm. in size, showing bile stained velvety mucosa.

Pathological Diagnosis

(1) Fresh coronary thrombosis in left anterior descending branch.

(2) Diffuse myocardial infarction in lower two-thirds of interventricular septum, anterior and lateral walls of left ventricle.

(3) Complete rupture in lower portion of interventricular septum.

(4) Congestion of lung, liver, kidney and spleen.

(5) Fibrocaseous tuberculous foci in right lung apex.

Cause of Death

Myocardial Infarction following Coronary Thrombosis.

Case 2

C.C., a male Chinese merchant aged 61 years was admitted to hospital with a history of sudden retrosternal pain four days before admission. The pain was moderately severe and associated with cold sweats, palpitations and breathlessness. On the day of admission to hospital he became very weak and tired, breathless with the slighest effort and noticed swelling of his feet.

He had previously been in good health.

On physical examination the patient was observed to be dyspnoeic at rest. A cyanotic tinge was noted in the lips and tongue and there was pitting oedema of both lower limbs. His extremities were cold and sweaty. The jugular venous pressure was raised. All peripheral pulses were palpable but weak: the radial pulse rate was 112 per minute and regular. The blood pressure was 110/70 mm. Hg. The apex beat was located in the 6th left intercostal space in

the anterior axillary line. There was a systolic thrill felt over the mitral area. On auscultation of the heart, both heart sounds were faintly audible in all areas and a loud harsh pan-systolic murmur was heard over the praecordium, maximum in the left 3rd and 4th intercostal spaces. This murmur was also audible in the left axilla. Basal crepitations were present in both lungs and the liver was just palpable below the right costal margin. The rest of the examination revealed no abnormality.

Investigations

Hb. 93%, W.B.C. 17,300, N. 85%, L. 10%, E. 0%, M. 5%.

Urine — albumin +, 1-2 P.C. and 3-4 R.B.C. per field.

The electrocardiograph showed elevation of the S.T. segment in limb leads III and vector leads 1 to 5 especially marked in vector leads 4 and 5. The S.T. segment was depressed in limb lead I and vector leads 6 and 7 (Fig. 5).

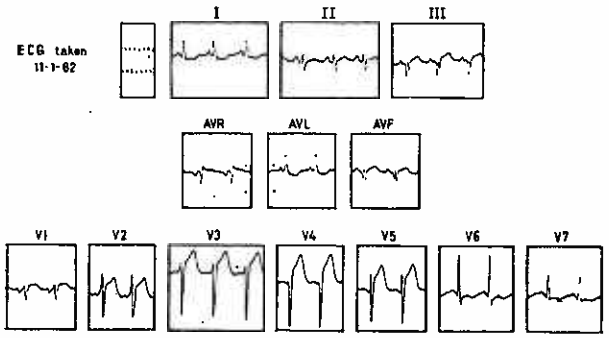

Fig. 5. E.C.G. showing elevation of ST segment in L III and V_1 to V_5.

Clinical Diagnosis

Myocardial Infarction with rupture of interventricular septum.

Clinical Course

Treatment given consisted of complete bed rest, coronary vasodilators and morphia injections. Soon after admission he complained of further retrosternal pain. In spite of repeated morphia injections, his condition deteriorated and the blood pressure fell to 80/60. Methidrine injections were given, but the blood pressure failed to rise. He died the next day, about 22½ hours after admission.

NECROPSY (Dr. E.B. La'Brooy, M.B.,B.S., D.C.P.)

Cardiovascular System

750 ml. of fluid and clotted blood was found in the pericardial cavity. The heart weighed 515 grams. The left ventricle was hypertrophied, the wall being 2 cms. thick. The wall of the left ventricle at the apex and the lower part of the interventricular system showed yellow and reddish brown necrosis with a rupture (2 cms. across) in the lower part of the interventricular septum (Fig. 6). The right ventricle showed haemorrhage into the muscle and epicardium anteriorly, near the apex; and a narrow per-

Fig. 6. Rupture in the lower part of the interventricular septum (Arrow) seen from the left ventricle.

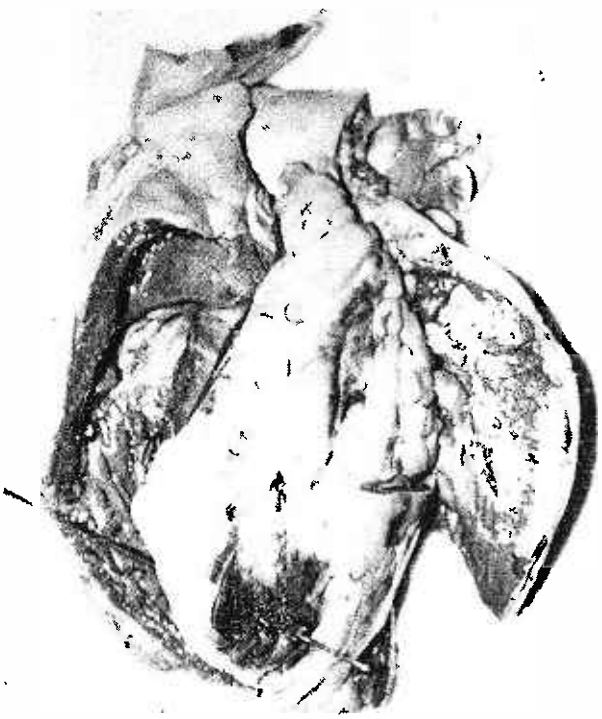

Fig. 7. Right ventricular wall with haemorrhage into the myocardium and epicardium anteriorly and rupture (with probe in situ).

foration was present in the wall at this point (Fig. 7).

The coronary vessels showed atherosclerosis but no thrombus or occlusion was seen. The valves were normal and the aorta showed moderate atherosclerosis throughout.

Respiratory System

Both lungs were congested and oedematous. The right lung weighed 953 grams and the left lung 696 grams.

Digestive System

Liver weighed 1244 grams. Spleen, kidneys and pancreas showed no abnormality.

Pathological Diagnosis

(1) Myocardial infarct of left ventricle at apex, lower part of interventricular septum and anterior wall of right ventricle near apex.

(2) Rupture of:—

(a) lower part of interventricular septum.

(b) right ventricle into pericardial cavity with hemopericardium.

DISCUSSION

Myocardial infarction may be complicated by rupture of the heart at either the ventricular wall or the interventricular septum. The former is reported to be much commoner than the latter, and the infrequency of septal perforation has been attributed to the rather rich arterial blood supply and anastomoses in the septum as demonstrated by Gross (1921).

In both cases reported here there was found perforation of the interventricular septum at post-mortem, the perforation having been diagnosed during life from the clinical history, E.C.G. changes and the pansystolic murmur. There was in addition a rupture of the right ventricle found at autopsy in the second patient. The authors believe that this is the third reported case of perforation of the interventricular septum and rupture of the ventricle following myocardial infarction occurring in the same patient, the first two cases having been reported by Sanders et al (1956).

Jaundice was noted clinically in the first case. This jaundice was probably the result of acute cardiac damage and liver congestion from cardiac failure (Rosin 1961). The occurrence of jaundice has so far not been reported in other previous cases of ruptured interventricular septum complicating coronary thrombosis.

Perforation of the interventricular septum following myocardial infarction was first reported by Sager in 1934 and he was able to collect a further 18 cases from the literature. From 1934 to 1943, 16 additional cases were reported and reviewed together with his own case by Weber in 1943. Since then perforation of the interventricular septum has been increasingly recognised. In 1956, when Sanders et al reviewed their 8 cases of interventricular septal perforations there was a total of 132 cases in the literature.

In the review of the literature most reports have shown that rupture of the ventricles following myocardial infarction is of more frequent occurrence when compared with perforation of the septum. Beresford and Earl (1930-31) in their series of 46 cases of spontaneous rupture of the heart had none with rupture through the septum, Benson et al (1933) reported only one case of rupture through the interventricular septum in their review of 40 cases of spontaneous rupture of the heart. Sager (1934) noted that interventricular septal perforation occurred in only 3% of non-traumatic heart rupture; Edmondson and Hoxie (1942) found, amongst 72 instances of spontaneous rupture through an area of ventricular infarction, only 13 instances (18%) of rupture through the interventricular septum while Dias-Rivera and Miller (1948) in a series of 1250 autopsies at Louisville General Hospital from 1939-1944, found 4 cases of rupture of the left ventricle and one case of interventricular septal perforation in 53 cases of fresh myocardial infarction.

The symptoms presented by the majority of patients are those of the primary disease. Generally a characteristic history of coronary occlusion is given and diagnosis of myocardial infarction is confirmed by E.C.G. changes. The occurrence of interventricular septum rupture is usually associated with a sudden deterioration in the clinical condition of the patient and is indicated by the appearance or presence of a harsh pansystolic murmur with or without a thrill and heard best along the left parasternal area at the 3rd and 4th intercostal space. A previous record of the state of the patients cardiovascular system would help to exclude a preexisting congenital septal defect. Of the cases reviewed by Weber, only one case did not have a murmur or thrill.

The E.C.G. is helpful in confirming the presence of myocardial infarction but appears less helpful in localising the site of septal infarction.

Wilson et al (1946) considered that septal infarction is likely if there were electrocardiographic evidence of both anterior and posterior myocardial infarction. This opinion has been supported by Rossler and Dressler (1947) who considered that septal infarction is likely when E.C.G. shows Q3 T3 pattern together with changes in the right praecordial leads. In reviewing the E.C.G. findings of 31 cases of septal infarcts with perforation, Sanders et al (1956) found that 39% showed conduction defects, 35% had QRS or QS over the right praecordium with elevated ST segments, 13% with Q3 R3 with diagnostic features of anterior infarction and 13% showed posterior infarction with evidence of septal damage. In our two patients the E.C.G. showed changes consistent with anterior and septal myocardial infarction but there was no evidence of any conduction defects.

In the majority of cases of interventricular septal perforation, thrombosis or marked narrowing of one coronary artery is found, associated with arteriosclerosis of the other coronary vessels as well. The anterior descending branch of the left coronary artery is usually involved. Of Weber's 17 cases, 10 cases showed thrombosis or narrowing of the left coronary artery or its branches and 5 cases showed thrombosis of the right coronary artery. In Hochreins' series as quoted by Sanders et al (1956) 74% showed occlusion in the left coronary artery, 21% in the right coronary artery and 8% in both arteries. The first case reported here had fresh thrombus formation in the left anterior descending branch with complete occlusion of its lumen, associated with atheromatous narrowing of the left circumflex branch and right coronary artery, while the second case had no thrombus or occlusion demonstrated but the coronary arteries were atherosclerotic.

The size of the perforation varies from pinpoint to an opening admitting the tip of the finger. The perforations may be single or multiple, and usually found, as in our two cases, in the lower half of the septum towards the apex of the heart. This serves to distinguish it from the congenital form which occurs in the membraneous upper part of the septum. As the left anterior descending branch is commonly involved the perforation is usually found in the anterior half of the lower part of the septum. Of the 18 cases reviewed by Sager (1934), 12 showed perforation in the lower part of the septum. The perforation, which commonly varies from 1 to 3.5 cm. in diameter, is usually too small to cause marked alteration in the haemodynamic state of the heart, but the shunt produced may precipitate cardiac failure in a severely damaged heart.

The time of onset of the perforation is usually 4-11 days after the myocardial infarction. The earliest septal rupture was that reported by Hyman (1930) where the patient developed this complication 4 hours after onset of symptoms. Oblath et al (1952) found that rupture of the heart is three times more common in hypertensive patients than patients with a normal blood pressure. In both our cases the blood pressure before the onset of myocardial infarction was not known. It has also been found by most workers that rupture is rare before the age of 50 years.

The presence of interventricular septal perforation indicates the severity of myocardial infarction as well as the poor state of the coronary vessels. The prognosis of a patient with interventricular septal perforation following myocardial infarction is very poor and death usually follows within a short time after development of this complication. Of the 8 patients reported by Sanders et al (1956) 6 died within 24 hours after perforation, one 19 days after perforation and one is still living 9 months following perforation at the time of reporting. Wood and Livezey (1942) reported a patient surviving 4 years and 10 months after perforation before he died. Probably the longest period of survival by a patient with this complication is the patient reported by Schlappi and Landale (1954) who is still alive $6\frac{1}{2}$ years after her infarction and perforation at the time of reporting. Both our patients had clinical evidence of a perforated ventricular septum when seen on admission to hospital but the time of occurrence of the perforation is not known. The first case died 7 days and the second case 6 days after the onset of symptoms.

SUMMARY

Two patients with myocardial infarction and perforation of the interventricular septum are described. The diagnosis was made during life on the history, the finding of a pansystolic murmur over the left 3rd and 4th intercostal space and E.C.G. changes of anterior and septal infarction. Necropsy confirmed the clinical diagnosis and revealed in addition rupture of the right ventricle into the pericardial cavity with haemopericardium in the second patient.

A brief review of the cases of perforation of the interventricular septum following myocardial infarction reported in the literature is made. The frequency of its occurrence, clinical

picture, diagnosis, E.C.G. changes and prognosis are discussed.

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REFERENCES

1. Benson, R.L., Hunter, W.C., and Manlove, C.H. (1933) Spontaneous Rupture of the Heart. Am. J. Path., 9, 295.
2. Beresford, E.H., and Earl, C.J.C. (1930-31) Spontaneous Cardiac Rupture. A Review of 46 Cases. Quart. J. Med., 24, 55.
3. Diaz-Rivera, R.S., and Miller, A.J. (1948) Rupture of the Heart following Acute Myocardial Infarction. Am. Heart J., 35, 126.
4. Edmondson, H.A., and Hoxie, H.J. (1942) Hypertension and Cardiac Rupture. Am. Heart J., 24, 719.
5. Hyman, A.S. (1930) Spontaneous Rupture of the Heart. Ann. Int. Med., 3, 800.
6. Gross, L. (1921) The blood supply of the heart in its anatomical and clinical aspects. Ed., 85. New York. Paul B. Hoeber, Inc.
7. Oblath, R.W., Levinson, D.C., and Griffith, G.C. (1952) Factors Influencing Rupture of the Heart after Myocardial Infarction. J.A.M.A. 149, 1276.
8. Roesler, H., and Dressler, W. (1947) An Electrocardiographic Pattern of Infarction of the Interventricular Septum extending from the Anterior to the Posterior Aspect of the Heart. Am. Heart J., 34, 817.
9. Sager, R.V. (1934) Coronary Thrombosis — Perforation of the Infarcted Interventricular Septum. Arch. Int. Med., 53, 140.
10. Sanders, Kerns, Blount (1956) Perforation of the interventricular septum complicating myocardial infarction. Am. Heart J., 51, 736.
11. Schlappi, J.C., and Landale, D.G. (1954) Perforation of the Infarcted Interventricular Septum. Am. Heart J., 47, .432.
12. Weber, M.L. (1943) Perforation of the Interventricular Septum following Infarction. Ann. Int. Med., 19, 973.
13. Wilson, Johnston, Rosenbaum and Barker (1946) Einthoven's Triangle, the Theory of Unipolar Electrocardiographic leads, and the interpretation of the Precordial Electrocardiogram. Am. Heart J., 32, 277.
14. Wood, F.C., and Livezey, M.H. (1942) Five Year Survival after Perforation of Interventricular Septum caused by Coronary Occlusion. Am. Heart J., 24, 897.
15. Rosin, A.J. (1961) Liver Function in Acute myocardial infarction. B.M.J. 2, 675.