36,4% of the sample. Social classes I - III in the sample correspond to the upper stratum in the report of the Theron Commission.[16]

The fathers of the children in the sample were generally better educated than the mothers. It is alarming, however, to find that 3,2% of fathers and 5,2% of mothers had no formal education, and that a further 35% of fathers and 60% of mothers were not educated further than Standard V. Regarding income, a quarter of the families were below the poverty datum line and over half below an effective minimum level. The Theron Commission[16] estimated that in 1975, 38,3% were below a supplemented living level.

Overcrowding remains a problem among Coloured families in Cape Town. Over half of the families in the sample lived in overcrowded conditions (occupational density over 150) and more than a quarter lived in unsatisfactory accommodation. These findings agree with the statement in the report of the Theron Commission,[16] that in a survey of attitudes among urban Coloureds, housing shortage headed the list of problems.

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Post-infarction Ventricular Septal Rupture Combined with Acute Right Ventricular Infarction

A Case Report

J. Z. PRZYBOJEWSKI, T. ROSENFELD

SUMMARY

An elderly White woman suffering from an acute transmural inferior myocardial infarction, with possible true posterior extension, is presented. Her holosystolic cardiac murmur and hypotension are attributed to rupture of the interventricular septum which occurred between the 2nd and 3rd days after infarction. Strong evidence for concomitant right ventricular infarction is put forward and therapy is discussed.

As far as the authors can determine, this combination of cardiac lesions has not been documented ante mortem previously. The apparent beneficial use of intravenous hydrallazine, for the first time in right ventricular infarction, is discussed.

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Date received: 26 March 1980.

CASE REPORT

A 75-year-old White woman was admitted to Tygerberg Hospital in February 1980 with a history that 2 days previously she had experienced the sudden onset of severe burning precordial pain radiating into her neck and down both arms. This pain lasted several hours and was associated with nausea, vomiting and slight sweating. Her doctor gave her morphine which brought relief. On the day before admission she again experienced mild precordial pain, which was somewhat relieved by intravenous diazepam.

There was no past history of chest pain or dyspnoea. However, she had noticed increasing tiredness during the few previous months. She had previously had a thyroidectomy, appendicectomy and herniorrhaphy. There was no history of hypertension or diabetes. She was a non-smoker. Two of her brothers suffered from ischaemic heart disease and a son was known to be diabetic.

In his referral note the GP commented on a systolic murmur over the heart which he had not been aware of at past examinations.

On examination she was not acutely ill. The radial pulse was regular at 75/min and all the peripheral pulses were equal. The jugular venous pressure was not elevated and blood pressure was 80/60 mmHg. The apex beat was in the fifth left intercostal space just lateral to the midclavicular line. A pansystolic thrill could be palpated over the left parasternal region. The heart sounds were normal but there was a fairly harsh pansystolic murmur of 4/6 intensity, maximal at the fourth left intercostal space radiating to the parasternal region but very poorly to the axilla. There were no diastolic murmurs. Bilateral basal crepitations were heard, but there was no visceromegaly. The rest of the examination was negative.

The ECG (Fig. 1) showed sinus rhythm of 90/min with a PR interval of 0,20 s (first degree atrioventricular block). The mean QRS axis was 90° and there were features of complete right bundle-branch block. Small q waves were seen in leads III and aVF and there was ST segment elevation in the inferior leads with reciprocal ST segment changes in the anterolateral leads as well as T-wave flattening. A diagnosis of acute transmural inferior myocardial infarction with possible true posterior extension was made.

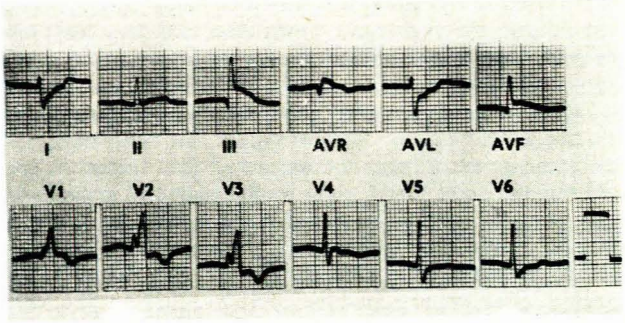

Fig. 1. ECG showing acute transmural inferior infarction with possible true posterior extension. Complete right bundle-branch block and first-degree atrioventricular block are also seen.

The chest radiograph (Fig. 2) showed cardiomegaly, pulmonary congestion, possible pulmonary plethora and small bilateral effusions.

There were serum enzyme changes typical of an evolving myocardial infarction. The urea level was elevated at 9,5 mmol/l and rose further to 19,8 mmol/l. The serum creatinine level varied between 80 and 138 mmol/l and that of sodium between 119 and 131 mmol/l. The full blood count was normal.

Course and Management

On the day after admission many ventricular ectopic beats and episodes of ventricular tachycardia (partially bidirectional) appeared, but were treated with lignocaine and mexiletine intravenously with good effect.

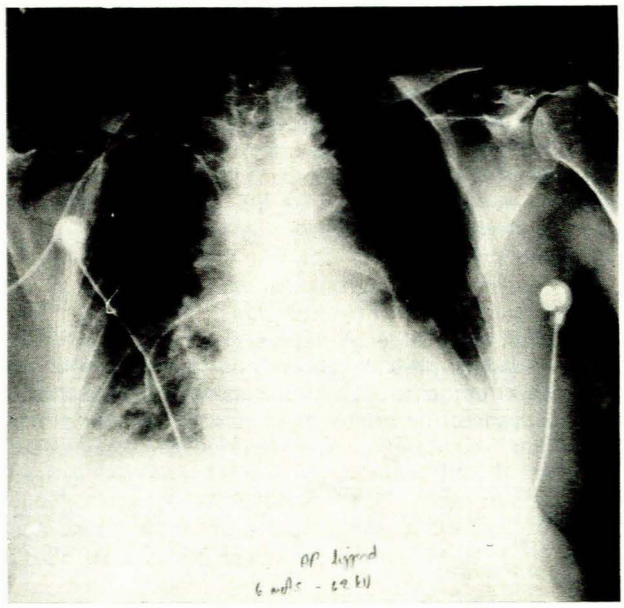

Fig. 2. Chest radiograph showing cardiomegaly, pulmonary congestion, possible pulmonary plethora and small pleural effusions.

During the second day of hospitalization the jugular venous pressure rose, the liver enlarged to 3 cm below the costal margin, and many more basal crepitations appeared. The urine output also fell to 590 ml. A Swan-Ganz catheter was inserted via an antecubital vein and positioned in the pulmonary artery. This pressure was 42/12 mmHg (mean 25 mmHg) and the mean pulmonary capillary wedge pressure (PCWP) was of 10 mmHg. Oxygen saturations were then recorded: superior vena cava 60%; right atrium (mid) 54% and (low) 52%, inferior vena cava at diaphragm 62%; right ventricle (mid) 86% and at tricuspid valve 72%. The systemic blood pressure was 75/50 mmHg and radial artery saturation 96%. Oxygen consumption was 175 ml/min. Rupture of the interventricular septum with a calculated left-to-right shunt of 66% was therefore diagnosed (Table I).

Therapy with fluids (saline) and dobutamine (Dobutrex) was started. When the systemic blood pressure rose to 100/70 mmHg treatment with sodium nitroprusside (1 μg/kg/min) was initiated. The possibility of a right ventricular infarction, in addition to the ventricular septal defect, was considered on account of the configuration of the right atrial, right ventricular and pulmonary artery pressures. On this therapy the urine output was kept at approximately 1 l/24 h for the next 2 days. On the following day the patient was cold and clammy, the jugular venous pressure rose even further, and dullness was noted

TABLE I. HAEMODYNAMIC CALCULATIONS

Parameter	Result
Oxygen consumption	175 ml/min
Pulmonary blood flow (Fick)	8,3 l/min
Systemic blood flow (Fick)	2,8 l/min
Left-to right shunt (by flows) $\left(\frac{8,3 - 2,8}{8,3} \times 100\right)$	66%
Left-to-right shunt (by saturations) $\left(\frac{84 - 60,5}{96 - 60,5} \times 100\right)$	66%
Pulmonary vascular resistance $\left(\frac{25 - 10}{8,3}\right)$	1,8 units

at the right lung base. A repeat chest radiograph confirmed a larger pleural effusion on the right. At this stage a short mid-diastolic murmur as well as a third heart sound appeared at the apex, indicative of greater left-to-right shunting via the ventricular septal defect. The mean PCWP rose to 16 mmHg and the systemic blood pressure fell to 90/70 mmHg. After rapid administration of 200 ml of saline the mean PCWP rose even further to 18 mmHg and the blood pressure to 100/70 mmHg.

In view of this deterioration it was decided to commence with intravenous dihydrallazine in a dose of 0,25 mg/min (360 mg/24 h). The blood pressure did not change and the urine output remained at 16 ml/h. The dose of dihydrallazine was therefore increased to 0,5 mg/min, which caused the patient to complain of flushing, but the urine output increased to 60 ml/h and the blood pressure remained at 100/70 mmHg. The PCWP fell to 16 mmHg. After 3 hours of this therapy the blood pressure fell to 70/55 mmHg and the urine output decreased again to 15 ml/h. The dose was then reduced to 0,25 mg/min, and later to 0,10 mg/min for the next 12 hours. No further change in blood pressure or urine output was noted.

After the dihydrallazine had been stopped the patient was again given dobutamine (4 μg/kg/min) and intravenous fluids. The mean PCWP remained at 15 mmHg, the pulmonary artery pressure at 30/15 mmHg and the urine output steadily diminished with a rise in blood urea to 19,8 mmol/l.

On the 7th day of hospitalization the patient went into cardiogenic shock with complete heart block and could not be resuscitated. Permission for postmortem was refused by the family.

DISCUSSION

The sudden appearance of a systolic heart murmur following an acute myocardial infarction is usually due to either a papillary muscle dysfunction of the mitral valve (often the result of rupture of the anterolateral or posteromedial papillary muscle), or rupture of the interventricular septum. The murmur is usually accompanied by a thrill localized to the lower left sternal edge, with no prominent 'v' waves in the PCWP tracing.

Septal perforation is a relatively uncommon complication of acute myocardial infarction, occurring in approximately 1,3% of patients dying from an infarction.[1-3] There is usually a sudden onset of hypoperfusion and the diagnosis can be established by right heart catheterization at the bedside or by echocardiography.[4] In the main, septal perforation complicates large transmural infarcts produced by total coronary artery occlusion and there is little time for the development of collateral blood flow. All of the 10 patients described by Hutchins[3] showed this uniform pathological picture. Like our patient, all his patients developed hypotension, which was usually severe and progressive. Our patient had a transmural inferior wall myocardial infarction with complete right bundle-branch block and possible true posterior extension of the infarction.

Recently, Sharpe *et al.*[5] and Gewirtz *et al.*[6] have drawn attention to the frequency with which inferior myocardial infarction is complicated by right ventricular infarction. The typical haemodynamic feature of this entity is a raised mean right atrial pressure which equals or exceeds mean pulmonary artery wedge pressure.[7] In our patient, the consistently low PCWP in the presence of a transmural inferior myocardial infarction was very suggestive of severe right ventricular dysfunction secondary to right ventricular infarction.

In the unfortunate combination of rupture of the intraventricular septum and right ventricular infarction, the value of the mean right atrial pressure probably has little haemodynamic significance for the diagnosis of the infarction. In a large left-to-right shunt with early signs of right heart failure, the high right ventricular end-diastolic pressure will lead to an obligatory higher mean right atrial pressure that can equal or exceed PCWP. In our patient the jugular venous pressure rose shortly after admission, with the appearance of hepatomegaly. Unfortunately, the right atrial pressure was not measured (for technical reasons) but the right atrial, right ventricular and pulmonary artery pressure curves appeared very similar in configuration. This was due to the raised mean right ventricular diastolic pressure with a diastolic 'dip' and 'plateau' appearance. The clinical picture therefore led to the conclusion that a second Swan-Ganz catheter insertion was unnecessary because of the confusing haemodynamic information that would be obtained.

The persistent hypotension in our patient can be explained on the basis of the inability of the severely infarcted right ventricle to pump an adequate volume of blood into the left side of the heart. Gewirtz *et al.*[6] showed that the PCWP may be low even in the presence of extensive left ventricular infarction. Right ventricular dysfunction in this situation may cause relatively low left-sided filling pressure, and this may be a cause of error in the clinical and haemodynamic assessment of the extent of left ventricular damage.

The recommended treatment in right ventricular infarction is volume loading in order to increase the left ven-

tricular pressure. When the interventricular septum ruptures, a 'natural' right ventricular overload is present. The chest radiograph of our patient showed pulmonary plethora without overwhelming respiratory distress. This apparent discrepancy may be explained by the low PCWP at this relatively early stage after rupture.

In spite of additional fluid administration and an increase in PCWP the clinical picture in our patient only altered slightly, suggesting that the left ventricle in this patient was severely damaged.

The conclusion seems to be that, in right ventricular infarction complicated by ventricular septal rupture, fluid overload is not indicated in treatment. The large left-to-right shunt should have increased the PCWP by a right ventricular overload mechanism. However, if this did not occur then additional fluid administration could not possibly improve the haemodynamic status; in fact, it would probably cause a deterioration.

Afterload reduction can play a useful role in maintaining patients with rupture of the ventricular septum in a compensated state until definite surgical repair can be accomplished.[8,9] The goal of therapy should be to reduce the left-to-right shunt and increase the forward left ventricular output.

Sodium nitroprusside has proved to be the most effective and best tolerated drug available for the management of heart failure and other states with vasoconstriction. The drug causes relaxation of arterial and venous smooth muscle, has a remarkably specific effect on vascular smooth muscle with a rapid onset and offset in action, and is almost uniformly effective in achieving the desired degree of dilatation provided the dosage is carefully titrated. Cardiac output increases only in the setting of left ventricular failure, when the reduction in outflow resistance predominates over the effect in reducing preload, which might otherwise lower cardiac output. Indeed, when the drug is administered to patients with normal left ventricular filling pressure or when infusion of the drug causes a precipitous fall in filling pressure to low normal levels, cardiac output may actually fall despite the concomitant reduction in impedance.[10]

In our patient, nitroprusside was used when the PCWP rose to 17 - 18 mmHg, but no clinical or haemodynamic improvement could be observed. The preload-reducing effect of the drug may explain this. Since left-to-right shunting at ventricular level is usually continuous throughout the cardiac cycle, preload reduction will, if proportionately greater on the right side of the circulation than on the left, favour more left-to-right flow by increasing the transventricular gradient.

Since the original papers of Chatterjee *et al.*[11] and Franciosa *et al.*[12] were published, hydrallazine has been used more and more in the therapy of chronic heart failure. This drug, unlike nitroprusside, has little if any effect on ventricular preload.[13] Mathey[14] evaluated the haemodynamic effects of hydrallazine in 13 patients with chronic left heart failure. The heart rate remained virtually constant, and the arterial blood pressure fell by an average of 12%. The end-diastolic pulmonary artery pressure was elevated before therapy and did not change significantly after administration of hydrallazine. The cardiac output was increased significantly, by an average of 55%, and by up to 100% in the individual patient. The significant improvement of cardiac output and stroke volume was caused by marked decrease in systemic vascular resistance.

Friedlander and Schwartz[15] were the first to use oral hydrallazine for treatment of interventricular septal rupture in a patient with a myocardial infarction. The degree of shunting through the defect was significantly reduced. Their patient eventually died because of severe ventricular arrhythmia before surgical repair could be carried out. Our patient was on treatment with intravenous hydrallazine and a significant clinical and haemodynamic improvement was noted, but after several hours the systemic blood pressure and urinary output dropped and made continuation of treatment difficult. It is postulated that the sudden decrease in the left-to-right shunt caused a decrease in the PCWP and left ventricular filling pressure and, as a consequence of this, caused a drop in the systemic blood pressure and urinary output. The unfortunate combination of rupture of the interventricular septum and right ventricular infarction thus seems to be a 'therapeutic stalemate' situation.

Hutchins,[3] in his clinicopathological work on septal rupture after myocardial infarction, reached the conclusion that, since the defects usually occur several days after infarction, survival for a few more days brings the patient to a period when repair has begun in the area of the infarction. He suggested early operative intervention.

Intra-aortic balloon counterpulsation can be carried out for a short period of time. In the presence of severe left ventricular damage and right ventricular infarction the early consideration of this treatment appears warranted, followed by early operative intervention for closure of the ventricular septal defect and infarctectomy. We unfortunately did not have the facilities for intra-aortic balloon counterpulsation to use in this patient. However, further clinical data are necessary to evaluate the best therapeutic approach to these complicated haemodynamic disturbances.

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