

Reversible hypovolaemic shock and myocardial ischaemia caused by contrast medium administered during diagnostic cardiac angiography

A case report

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Summary

A 65-year-old white man with severe symptomatic four-vessel atherosclerotic coronary artery disease underwent selective coronary arteriography. Two hours after this procedure he developed hypovolaemic shock secondary to the hyperosmolar contrast medium, as well as severe angina pectoris accompanied by myocardial ischaemia. This diagnosis was established with the aid of Swan-Ganz catheterisation and the patient was successfully managed with intravenous fluid replacement and emergency coronary artery bypass graft surgery. Pathophysiological aspects are discussed with comments on the possible prevention of such a potentially life-threatening complication of selective coronary angiography.

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Case report

The patient was a 65-year-old white man who had been a heavy cigarette smoker until 7 years previously. His essential hypertension had been controlled by a β -blocker, atenolol. A younger brother had significant ischaemic heart disease (IHD). Classic effort-induced angina pectoris was first noted 2 months before his admission to the Cardiac Unit, Tygerberg Hospital, on 8 January 1985 for further investigation. His general practitioner had prescribed diltiazem, a transdermal nitroglycerine preparation and sublingual isosorbide dinitrate when required, together with atenolol 100 mg daily. However, his angina continued on effort and with emotion and he was referred for coronary bypass surgery. A stress test was strongly positive.

On admission he was overweight with stigmata of possible hypercholesterolaemia (abnormal achilles tendons and arcus cornealis). Pulse rate was 56/min with all peripheral pulses normal; blood pressure was 160/90 mmHg and there were grade II hypertensive changes on fundal examination. Although there was no clinical evidence of cardiomegaly, the apical impulse was quite forceful and sustained suggesting left ventricular hypertrophy. A loud fourth heart sound was audible and there were no cardiac murmurs. There were no signs of cardiac failure.

A resting 12-lead ECG demonstrated sinus rhythm at 60/min, a P-R interval of 0,17 second and a mean QRS axis of $+75^\circ$.

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Borderline left ventricular hypertrophy by voltage criteria was also seen. High lateral (leads I and aVL) and apical (leads V3 and V4) asymmetrical T-wave inversion was in evidence. Poor R-wave progression in the anteroseptal leads, insignificant Q waves and early ventricular repolarisation in the inferior leads was also noted.

Routine chest radiography delineated a normal cardiac silhouette. The lung fields appeared clear.

Echocardiography (M-mode and cross-sectional) demonstrated marked symmetrical left ventricular (LV) hypertrophy with a high ejection fraction of 70%. There were no pathognomonic features of hypertrophic obstructive cardiomyopathy (HOCM) but there was evidence of LV systolic overload from long-standing hypertension.

A stress ECG result was interpreted as strongly positive and suggestive of a left mainstem coronary artery (LMCA) stenosis.

The patient continued on his medication and serum enzyme studies were recorded as normal. Repeat resting ECGs did not demonstrate any features of acute myocardial ischaemia or a possible myocardial infarction related to the stress test. Cardiac catheterisation was performed on 10 January 1985.

Cardiac catheterisation

Central aortic and LV pressures were within the normal range. LV cine angiography (right anterior oblique (RAO) projection) demonstrated a hypercontractile and markedly hypertrophic chamber, the angiographic features suggesting the possibility of HOCM (Fig. 1). There was no evidence of mitral-valve prolapse or insufficiency. The right coronary artery (RCA) was studied in both the left anterior oblique (LAO) and RAO view. The RCA was seen to be a dominant and ectatic vessel with three significant stenoses in the first half (Fig. 2). No right-to-left collateral blood flow could be seen, the aortic pressure was stable, and the patient did not complain of angina. The left coronary artery (LCA) was injected in four different views (steep LAO, shallow LAO with cranial angulation, shallow RAO with cranial angulation, shallow RAO with caudal tilt), each injection consisting of a bolus of approximately 8 ml of Urographin-76%. Adequate time was allowed between injections for stabilisation of the aortic pressure (normal with catheter engagement of the LCA ostium). There was no angina and there were no cardiac arrhythmias. The cine angiograms delineated two subtotal obstructions in the mid-portion of the LMCA, the distal one just proximal to the origin of the left anterior descending (lad) and left circumflex (lc) coronary arteries (Figs 3 and 4). Further significant stenoses were seen in the lad coronary artery distal to the origin of its first diagonal branch, and in the lc coronary artery proximal to its mid-lateral branch (Figs 3 and 4). The cardiac catheterisation was completed within 30 minutes and the patient was haemodynamically stable with no angina pectoris.

Course after cardiac catheterisation

In view of the severity of the coronary artery disease, especially the LMCA stenoses, as well as the fact that the vessels were operable, emergency coronary artery bypass graft (CABG) surgery

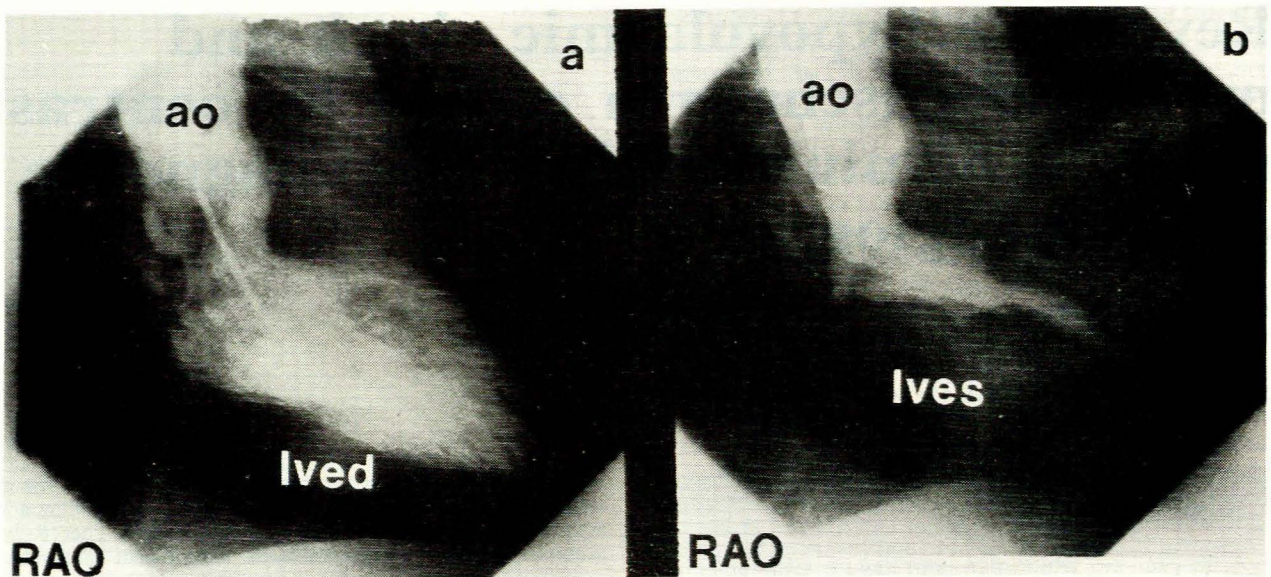


Fig. 1. LV cine angiograms in the RAO projection. The ventricle is severely hypertrophic and hypercontractile ('pseudo-HOCM'). There is no mitral insufficiency or prolapse. The ascending aorta (ao) is dilated. (a) LV in end-diastole; (b) LV in end-systole.

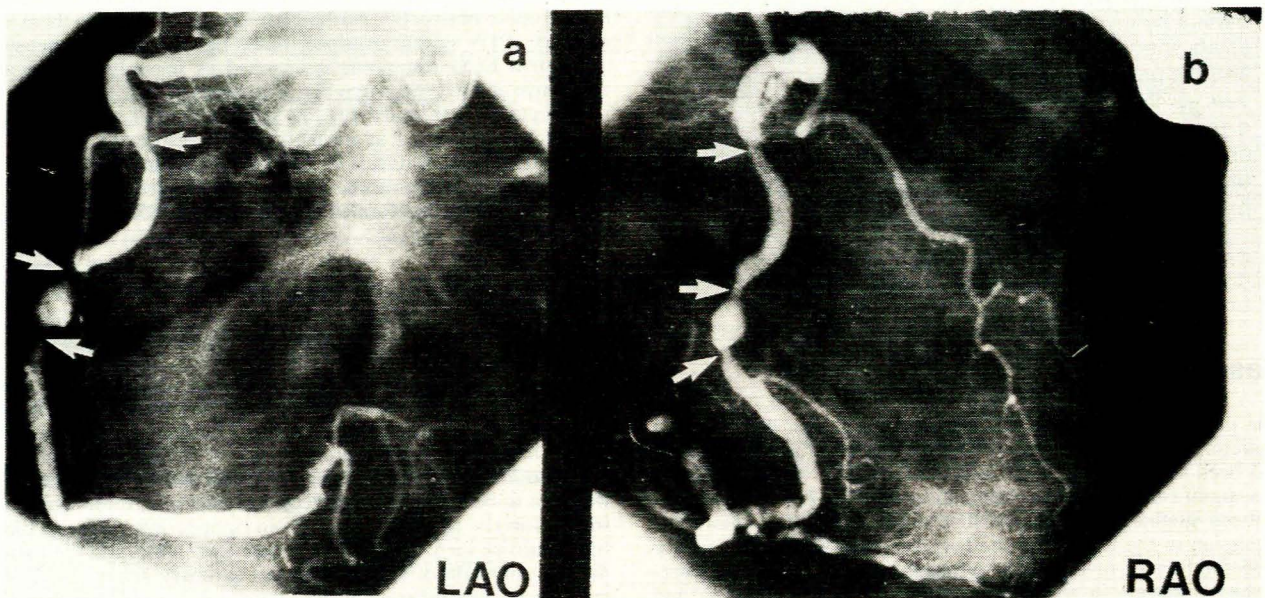


Fig. 2. Right coronary cine angiograms in (a) LAO; and (b) RAO views. The vessel is dominant and ectatic. There are two 80% stenoses in series in the mid-portion and a less severe stenosis distal to the origin (arrowed). There is no evidence of right-to-right or right-to-left collateral flow.

was discussed and arrangements were made for the patient to return to the Cardiac Unit for monitoring until such time as the theatre could be prepared. The patient was also instructed to take oral fluids freely. On returning to the Cardiac Unit a resting 12-lead ECG was unchanged from that before cardiac catheterisation.

About 2 hours later the patient complained of sudden severe precordial pain. A resting ECG was immediately recorded and demonstrated 3 mm downward sloping ST-segment depression in the anterolateral leads. The patient was also found to be shocked with a blood pressure of 75/20 mmHg and a pulse rate of 64/min. An intravenous line was immediately set up and Plasmalyte-B was slowly infused, together with the inotrope, dobutamine. A Swan-Ganz catheter was inserted percutaneously via the left subclavian vein and the right heart pressures recorded. Main pulmonary artery pressure was normal at 22/5 (mean 12) mmHg with a low mean pulmonary capillary wedge pressure (PCWP) of 4 mmHg. A diagnosis of intravascular fluid depletion, probably caused by the osmotic effect of the contrast medium, precipitating myocardial

ischaemia, was made; acute myocardial infarction was also a possibility.

The patient received 3 litres of Plasmalyte-B intravenously within 1 hour with continuous haemodynamic monitoring. Inotropic drug therapy was discontinued; fluid replacement raised the blood pressure to 130/80 mmHg and the mean PCWP to 8 mmHg. All the signs of shock had now disappeared, and the patient began to pass a small volume of concentrated urine. His chest pain also disappeared and the ECG now demonstrated resolution of the previous myocardial ischaemia and no signs of acute myocardial infarction. Intravenous fluid was continuously infused with the PCWP as a guide to volume required.

In this stable clinical condition the patient was taken to the operating theatre where his cardiac output was recorded as 6.8 l/min, cardiac index 3.9 l/min/m², blood pressure 130/80 mmHg, mean PCWP 13 mmHg and pulmonary artery pressure 21/6 (mean 12) mmHg. Emergency CABGs were then successfully inserted into the posterior descending branch of the RCA, the lad

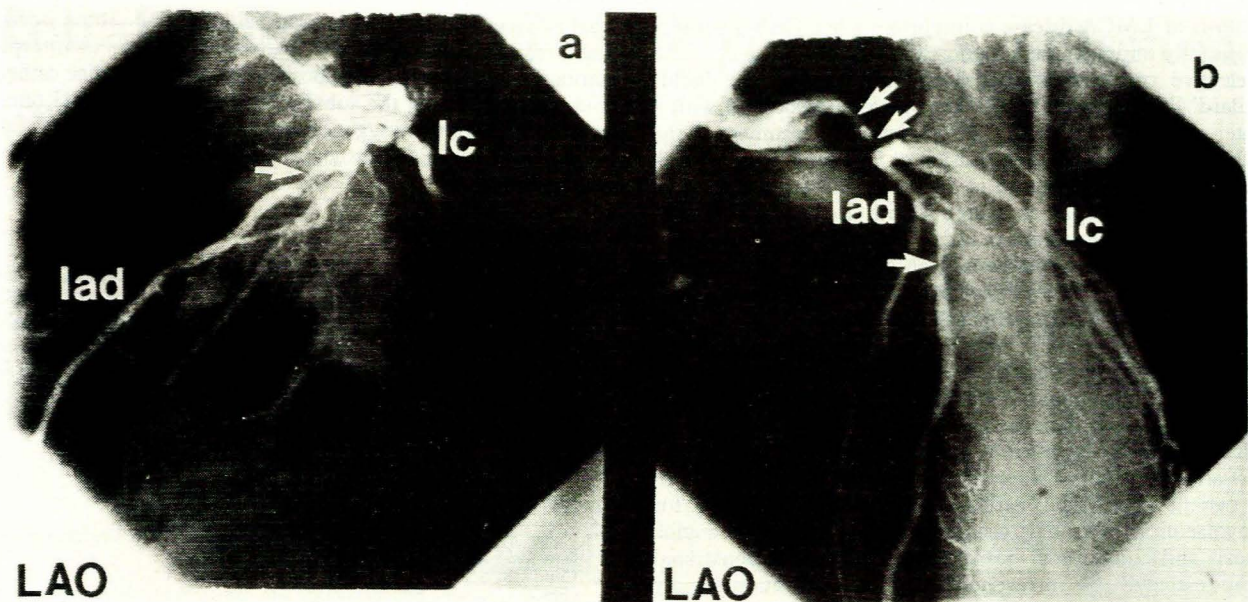


Fig. 3. Left coronary cine angiograms in (a) steep LAO view; and (b) shallow LAO projection with cranial angulation. Two subtotal obstructions (arrowed) are seen in the mid-portion of the left mainstem coronary artery (b). There is also a significant obstructive lesion (arrowed) in the left anterior descending (lad) and left circumflex (lc) coronary artery, the latter not being clearly evident in these projections.

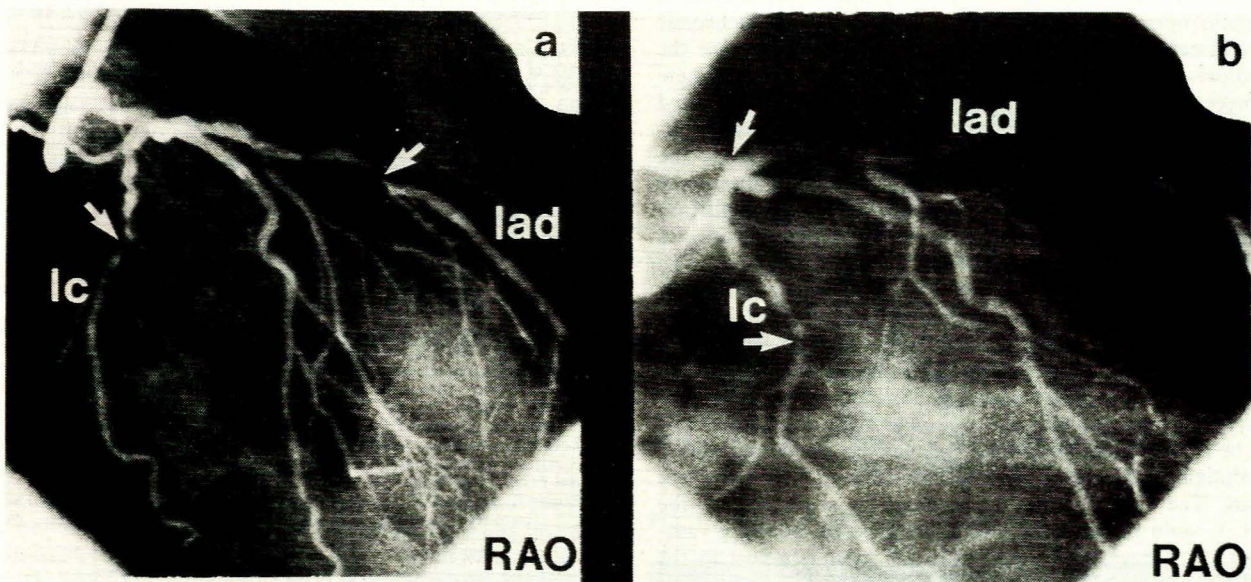


Fig. 4. Left coronary cine angiograms in (a) shallow RAO with cranial angulation; and (b) shallow RAO with caudal angulation. Subtotal obstructions (arrowed) are visible in both the lad and lc coronary arteries. One of the stenoses (arrowed) in the left mainstem coronary artery is evident in (b).

coronary artery both proximal and distal to the stenosis, and the anterolateral branch of the lc coronary artery.

The patient made an uneventful recovery and was discharged on 22 January 1985 on atenolol 100 mg daily, prazosin 2 mg 3 times daily, dipyridamole 100 mg 3 times daily and aspirin 300 mg daily. Follow-up documented the absence of any angina pectoris and adequately controlled essential hypertension. Repeat treadmill stress testing was negative.

Discussion

This experience highlights several important aspects of the management of symptomatic atheromatous LMCA disease.

The concomitant presence of significant narrowing of the RCA, as well as severe symmetrical LV hypertrophy secondary to chronic essential hypertension, had an important influence on the clinical picture.

Several authors¹⁻⁶ have attempted to define the clinical characteristics of patients with LMCA involvement with a view to timely coronary arteriography and possibly urgent surgical intervention. Unfortunately, no clinical picture pathognomonic of underlying LMCA obstruction has been established, although marked ST-segment depression on stress ECG, as seen in our patient, is suggestive. Intermittent pulmonary oedema secondary to global myocardial ischaemia has also recently been encountered in such cases.⁷ The early

diagnosis of LMCA disease is important since CABG surgery significantly improves the prognosis.^{8,9}

Selective coronary arteriography still remains the 'gold-standard' for diagnosis, but is known to be associated with a greater risk.^{4-6,10-15} Recently, Przybojewski¹⁶ documented the death during cardiac catheterisation of 5 patients with LMCA ostial stenosis over a period of 10 years. Mortality related to coronary arteriography in patients with LMCA obstruction has been reported by Wolfson *et al.*¹⁵ to be as high as 20%, but most recently Kron¹⁷ commented on a decrease in frequency in this subgroup of patients. Intravascular dehydration, secondary to the hyperosmotic effect of the contrast medium used during cine angiography, may be a precipitating factor for increased risk in cases of LMCA disease. Researchers therefore recommend a minimum of contrast injections and administration of intravenous fluid during the procedure.

Haemodynamic assessment by Swan-Ganz catheterisation 2 hours after successful completion of coronary arteriography in our patient established dehydration as the mechanism for hypovolaemic shock. This was accompanied by severe angina pectoris and objective myocardial ischaemia as demonstrated on the resting ECG. Intravenous fluid administration rapidly reversed this shock.

Recently Coetzee *et al.*¹⁸ showed experimental evidence for a hypotension-induced fall in coronary perfusion pressure (CPP) giving rise to severe regional myocardial dysfunction in the presence of fixed critical coronary artery obstruction, and commented that 'it appears to be of primary importance to maintain normal blood pressure in patients with ischaemic heart disease, and it may even be preferable to increase the blood pressure slightly during the peri-operative period'. These findings are of direct relevance to our case, since the calculated CPP was 33 mmHg, a figure significantly below normal, considering that effective autoregulation takes place when the CPP ranges from 50 mmHg to 150 mmHg in the absence of coronary artery obstruction.¹⁹ In the presence of significant coronary artery stenosis there is maximum autoregulation, as represented by maximal dilatation of the artery segment distal to the stenosis; under these circumstances coronary blood flow will depend solely upon the perfusion or driving pressure.

Other important factors have to be considered. Hoffman²⁰ has demonstrated that an increase in blood viscosity, as well as LV hypertrophy, can contribute to subendocardial ischaemia. Dehydration is a potent cause of increased blood viscosity and may be an important stimulus to increased platelet aggregation and sludging, a precursor of vaso-actor-induced coronary artery spasm. The two very severe stenoses in series within the LMCA could be ideal sites for superimposed coronary vasospasm.^{21,22} I have been unable to find another report in the English-language literature documenting two significant obstructions within the LMCA, a situation which almost certainly increases the risk of selective coronary arteriography. The presence of significant RCA disease and the relative lack of collateral vessel formation also add to the haemodynamically unfavourable setting.

An additional pertinent factor is the presence of marked symmetrical LV hypertrophy secondary to long-standing hypertension. The myocardial oxygen requirement would be much greater under these circumstances, despite the fact that hypotension could reduce this increased need. Coetzee *et al.*¹⁸ have shown experimentally that LV dysfunction still occurred in the presence of hypotension and critical coronary artery stenosis despite a fall in myocardial oxygen demand.

In the light of this experience, I now routinely begin to administer intravenous fluids on the cardiac catheterisation table as soon as a stenosis of the LMCA is recognised. This policy must be tempered by the presence of an abnormally elevated LV end-diastolic pressure, a haemodynamic finding indicative of LV failure, in this instance secondary to myocar-

dial ischaemia or previous myocardial infarction. Some workers have recommended the use of intra-aortic balloon counterpulsation in cases of LMCA stenosis, both during cardiac catheterisation, and during the subsequent emergency CABG operation.²³⁻²⁵ Again, this suggestion must be considered in the light of the known significant complications caused by the use of this technique.

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REFERENCES

- Cohen MV, Gorlin R. Main left coronary artery disease: clinical experience from 1964-1974. *Circulation* 1975; **52**: 275-285.
- Conti CR, Selby JH, Christie LG *et al.* Left main coronary artery stenosis: clinical spectrum, pathophysiology and management. *Prog Cardiovasc Dis* 1979; **22**: 73-106.
- Plotnick GD, Greene HL, Carliner NH, Becker LC, Fisher ML. Clinical indicators of left main coronary artery disease in unstable angina. *Ann Intern Med* 1979; **91**: 149-153.
- Lavine P, Kimbiris D, Segal BL *et al.* Left main coronary artery disease: clinical, arteriographic and hemodynamic appraisal. *Am J Cardiol* 1972; **30**: 791-796.
- Cohen MV, Cohn PF, Herman MV, Gorlin R. Diagnosis and prognosis of left main coronary artery obstruction (abstract). *Circulation* 1972; **45**: suppl 1, 65.
- Khaja F, Sharma SD, Easky RM *et al.* Left main coronary artery lesions: risk of catheterisation, exercise testing and surgery (abstract). *Circulation* 1974; **49**: suppl II, 140.
- Przybojewski JZ, Rossouw J. Severe isolated left mainstem coronary artery stenosis: a case report. *S Afr Med J* 1986; **69**: 133-136.
- Gersh BJ, Kronmal RA, Frye RL *et al.* Coronary arteriography and coronary artery bypass surgery: morbidity and mortality in patients ages 65 years or older: a report from the Coronary Artery Surgery Study. *Circulation* 1983; **67**: 483-491.
- CASS Principal Investigators and their Associates. Coronary Artery Surgery Study (CASS): a randomized trial of coronary artery bypass surgery: comparability of entry characteristics and survival in randomized patients and nonrandomized patients meeting randomization criteria. *JACC* 1984; **3**: 114-128.
- Conti RC, Brawley RK, Griffith LSC *et al.* Unstable angina pectoris: morbidity and mortality in 57 consecutive patients evaluated angiographically. *Am J Cardiol* 1973; **32**: 745-750.
- Webster JS, Moeberg C, Rincon G. Natural history of severe proximal coronary artery disease as documented by coronary cineangiography. *Am J Cardiol* 1974; **33**: 195-200.
- De Mots H, Bonchek LI, Rosch J, Anderson RP, Starr A, Rahimtoola SH. Left main coronary artery disease: risk of angiography, importance of coexisting disease of other coronary arteries and effects of revascularization. *Am J Cardiol* 1975; **36**: 136-141.
- Rosch J, De Mots H, Antonovic R, Rahimtoola SH, Judkins MP, Dotter CT. Coronary arteriography in left main coronary artery disease. *Am J Roentgenol* 1974; **121**: 583-590.
- Cabin HS, Roberts WC. Fatal cardiac arrest during cardiac catheterisation for angina pectoris: analysis of 10 necropsy patients. *Am J Cardiol* 1981; **48**: 1-8.
- Wolfson S, Grant D, Ross AM, Cohen LS. Risk of death related to coronary arteriography: risk of left coronary arterial lesions. *Am J Cardiol* 1976; **37**: 210-216.
- Przybojewski JZ. Left main coronary artery stenosis — death after angiography. *S Afr Med J* 1986; **70**: 832-837.
- Kron J. The case for continued scrutiny of catheterization-related complications (Editorial). *Chest* 1985; **87**: 707-708.
- Coetzee A, Foëx P, Holland D, Ryder A, Jones L. Coronary blood flow during variation in coronary perfusion pressure. *S Afr Med J* 1985; **68**: 15-18.
- Berne RM. Regulation of coronary blood flow. *Physiol Rev* 1964; **44**: 1-29.
- Hoffman JIE. Determinants and prediction of transmural myocardial perfusion. *Circulation* 1978; **58**: 381-391.
- Murphy ES, Rosch J, Boicourt OW *et al.* Left main coronary artery spasm: a potential cause for angiographic misdiagnosis of severe coronary artery disease. *Arch Intern Med* 1976; **136**: 350-351.
- Tzivoni D, Merin G, Milo S *et al.* Spasm of the left main coronary artery. *Br Heart J* 1976; **38**: 104-107.
- Gold HK, Leinbach RC, Sanders CA, Buckley MJ, Mundth ED, Austin WG. Intraaortic balloon pumping for control of recurrent myocardial ischemia. *Circulation* 1973; **42**: 1197-1203.
- Rajai HR, Hartman CW, Innes BJ *et al.* Prophylactic use of intraaortic balloon pump in aortocoronary bypass for patients with left main coronary artery disease. *Ann Surg* 1978; **187**: 118-121.
- Cooper GN, Singh AK, Christian FC *et al.* Preoperative intraaortic balloon support in surgery for left main coronary stenosis. *Ann Surg* 1977; **185**: 242-246.