Case reports

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Pulmonary artery pressure during acute pulmonary oedema in patient with myocardial infarction

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A patient with myocardial infarction was studied before, during, and after the development of acute pulmonary oedema. The haemodynamic measurements indicated poor left ventricular function before pulmonary oedema. Mean pulmonary artery pressure rose from 38 to 53 mmHg during the acute episode and fell to 25 mmHg as clinical improvement occurred. The fall in pulmonary artery pressure was closely related in time to the administration of 3 mg morphine intravenously.

Acute pulmonary oedema is a rapidly changing and unpredictable condition which is difficult to study in man. Occasionally haemodynamic measurements have been made when pulmonary oedema has occurred during cardiac catheterization, and these studies have shown a large rise in pulmonary artery pressure (Scebat, Lenegre, and Maurice, 1949; Finlayson *et al.*, 1961; Yu, 1969). Few measurements are available in patients with myocardial infarction and acute pulmonary oedema, and these were carried out several hours after the first signs of acute pulmonary oedema and after energetic treatment had been given. Nixon (1968) reported a patient with normal left atrial pressure and Knutsen and Broch (1968) found stroke index to be reduced in the 5 patients they studied but they did not measure left heart or pulmonary artery pressure.

During a recent study into the effects of intravenous diuretics in patients with myocardial infarction, one patient developed acute pulmonary oedema. This paper presents the clinical and haemodynamic findings before and during this episode and after treatment.

Case report

The patient, aged 64, had experienced three episodes of chest pain between 1963 and 1968; these were diagnosed as myocardial infarction by his doctor and treated at home. The present admission followed 4 weeks of increasing angina and dyspnoea on exertion and was precipitated by severe chest pain lasting 20 minutes. The

electrocardiogram showed widespread recent antero-septal infarction. On admission he had no pain and was slightly breathless but not distressed. He was in sinus rhythm (80 beats/min), blood pressure was 140/100 mmHg, the apex beat was in the 6th intercostal space, and heart sounds were normal. There were no signs of heart failure and no treatment was given. The following morning he was more dyspnoeic and had a sinus tachycardia (120 beats/min) and bilateral basal lung crepitations.

After obtaining informed consent from the patient a fine catheter (PE60) was floated into the pulmonary artery. Right atrial pressure was measured while the catheter was in the right atrium. A short cannula was inserted into the adjacent brachial artery. Pressures were measured using midthoracic point as reference zero. Cardiac output was measured by the dye dilution technique using indocyanine green. After a half-hour rest, baseline pressure measurements of cardiac output, and arterial blood gas tensions were made in duplicate over one hour with the patient breathing air. Frusemide (80 mg) intravenously was then given and the same measurements were repeated 1 and 2 hours later. The results are given in the Table. In the two hours after intravenous frusemide the patient passed 500 ml urine. There was no change in his clinical condition.

Fifteen minutes after completion of the 2-hour measurements he developed rapidly increasing dyspnoea and orthopnoea with clinical signs of acute pulmonary oedema. He was given 0.25 mg ouabain and 250 mg aminophylline intravenously with very little benefit. Venous tourniquets were then applied to both legs. At this time it was possible to record pulmonary artery pressure which had risen from 37 mmHg to 53 mmHg (Fig.). Morphine (3 mg) was given slowly intravenously. Five minutes after the start of the injections the mean

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TABLE Haemodynamic and respiratory measurements: 80 mg frusemide given at 12 a.m.; acute pulmonary oedema developed at 2.30 p.m.

Time	Before frusemide		After frusemide			
	11-12 a.m.		1 p.m.	2 p.m.	2.30 p.m.	6 p.m.
Cardiac index (l./min per m ²)	1.3	1.45	1.4	1.5		
Stroke index (ml/beat per m ²)	11	12	12	12		
Heart rate (beats/min)	125	120	120	128		
Right atrial pressure (mmHg)	8					
Pulmonary artery pressure (mmHg)	50/32 (38)	48/32 (38)	45/31 (35)	47/35 (37)	(53)	38/25 (29)
Brachial artery pressure (mmHg)	115/80 (90)	112/76 (88)	120/85 (95)	107/77 (90)		
Arterial oxygen tension (mmHg)	53	54	48	49		51
Arterial carbon dioxide tension (mmHg)	32	32	30	32		29
Arterial pH	7.49	7.50	7.50	7.52		7.52

Figures in parentheses indicate mean pressures.

pulmonary artery pressure had fallen from 53 to 25 mmHg. Clinical improvement was then evident and continued with digitalis and further diuretic therapy. Mean pulmonary artery pressure 4 hours later was 29 mmHg and the following day was 31 mmHg. Gradual deterioration occurred however despite treatment, and he died 6 days after admission with intractable left ventricular failure. Serum potassium was normal throughout his illness.

Necropsy showed a large heart (430 g) with a large, recent anterior septal and lateral infarct, subendocardial infarction extending to the aortic valve, and an old fibrous inferior infarct. All the coronary arteries were grossly atheromatous, with 80 per cent narrowing and recent occlusive thrombosis of the left anterior descending coronary artery. The lungs were heavy (930 and 780 g) and congested. There was a small right lower lobe pulmonary embolus with no definite lung infarction.

FIG. Changes in mean pulmonary artery pressure during acute pulmonary oedema.

Discussion

This patient demonstrates that a large gradient may occur between pressure in the right atrium and pulmonary artery in patients with myocardial infarction. Right atrial pressure was only 8 mmHg and, clinically, jugular venous pressure was not raised before the onset of pulmonary oedema despite the pronounced raised pulmonary artery pressure of 50/32 mmHg. Though we have found a significant correlation between right atrial and pulmonary artery pressure in myocardial infarction (Tattersfield, McNicol, and Sillett, 1972), normal central venous pressure does not exclude severe pulmonary hypertension.

During the acute episode of pulmonary oedema there was a rise in mean pulmonary artery pressure from 37 to 53 mmHg. The maximum rise was probably greater as the latter measurement was made approximately 10 minutes after the development of acute pulmonary oedema and after the administration of ouabain and aminophylline and the application of tourniquets. This finding is similar to those observed in patients with rheumatic heart disease during acute pulmonary oedema where the most distinctive feature has been the pronounced rise in both pulmonary artery pressure and pulmonary wedge or left atrial pressure (Scebat *et al.*, 1949; Finlayson *et al.*, 1961; Yu, 1969).

There is now much evidence to show that the raised pulmonary artery pressure in myocardial infarction is primarily reflecting increased left ventricular end-diastolic pressure (Kirby, McNicol, and Tattersfield, 1968; Hodges *et al.*, 1969; Russell *et al.*, 1970; Rahimtoola *et al.*, 1972). The high pulmonary artery pressure and very low stroke index in this patient indicates extremely poor left ventricular function before the onset of acute pulmonary

oedema. In patients with such poor left ventricular function, it has been shown that changes in filling pressure are associated with little or no change in stroke index (Russell *et al.*, 1970; A. E. Tattersfield, M. W. McNicol, and R. W. Sillett, unpublished observations). Further increases in filling pressure will eventually be associated with a fall in stroke volume (Ross *et al.*, 1966). This is an inherently unstable situation in which left ventricular filling pressure will rise rapidly. We believe that our patient reached this situation which caused the very rapidly developing pulmonary oedema.

In this patient the factors precipitating acute pulmonary oedema are not known. In vulnerable patients with poor left ventricular function a small increase in venous return or in peripheral vascular resistance or slight further impairment of left ventricular function could precipitate pulmonary oedema. Changes in venous return and peripheral vascular resistance occur with such everyday activities as changing posture or micturition (Marshall and Shepherd, 1968). It is unlikely that the diuretic was related to the development of pulmonary oedema as diuretics usually produce a fall in pulmonary artery pressure (Sjögren, 1970; Tattersfield *et al.*, unpublished observations). None of the other 34 patients in the same study developed pulmonary oedema.

It is impossible to say categorically which therapeutic measure, if indeed any, contributed to the rapid reduction in pulmonary artery pressure. There was very little clinical improvement following ouabain, aminophylline, and the venous tourniquets, and the close time relation with intravenous morphine suggests that morphine was probably mainly responsible. This is consistent with observations on the pulmonary circulation in man (Roy *et al.*, 1965; Yu, 1969) and on the peripheral circulation in animals (Henney *et al.*, 1966), which suggest that morphine causes a redistribution of blood toward the peripheral circulation. It may be judicious to give intravenous morphine with caution in this situation so that the reduction in filling pressure is not excessive.

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