Left ventricular function and mitral valve opening in massive pulmonary embolism

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SUMMARY M-mode echocardiograms are demonstrated from a patient with subacute massive pulmonary embolism before and after thrombolytic treatment and clinical recovery. Severely impaired left ventricular contraction returned to normal. A reversible reduction in mitral valve opening velocity was also seen and was thought to be in part the result of diminished left atrial filling. This hypothesis was tested experimentally; mitral valve opening velocity was measured in normal subjects and found to be significantly reduced when pulmonary blood flow was impeded during the Valsalva manoeuvre.

M-mode echocardiography can demonstrate characteristic features of right ventricular dilatation in massive pulmonary embolism,¹ and may be used as an aid to the assessment of acute pulmonary hypertension.² Changes in the ratio of right to left ventricular size, and pulmonary artery dilatation, can both be correlated with the severity of pulmonary artery obstruction.² A reduction in mitral valve excursion and mid-diastolic closure rate may also be seen in both acute and chronic pulmonary hypertension,^{3,4} and have been attributed to an alteration in shape and compliance of the left ventricle.⁵ In situations where pulmonary venous return is low, for example pulmonary embolism, haemorrhage, Valsalva manoeuvre, the left ventricular filling pressure will be reduced and this might directly affect left ventricular haemodynamics and mitral valve movement.⁴

Case report

A 44 year old man had a two week history of progressive disabling dyspnoea. Angiography demonstrated 75% obstruction of the main pulmonary arteries, confirming the diagnosis of subacute massive pulmonary embolism.⁶ Streptokinase administration, preceded by a plasminogen infusion, resulted in a dramatic clinical improvement, with repeat angiography after three days showing almost complete clearing of the thrombus.

An M-mode echocardiogram obtained before treatment from the standard left parasternal position showed an unusual pattern of mitral valve movement (Fig.). The opening velocity of the anterior mitral valve leaflet can be measured as the maximum gradient of the

D-E slope. In this patient it was 11.3 ± 1.3 cm/s during inspiration, increasing to 15.2 ± 1.7 cm/s in expiration ($p < 0.001$) when measured on nine cardiac cycles in each of the two phases of respiration, as timed from breath sounds recorded on the phonocardiogram (Table 1). Septal movement was abnormal and left ventricular posterior wall contraction very poor. Calculation of fractional shortening is difficult in the presence of reversed septal motion, but it is obviously reduced by the poor systolic contraction of the posterior wall. (Approximate value 20%; normal $> 30\%$.) The septum moves in a posterior direction during inspiration, and there are small variations in left ventricular transverse diameter in phase with respiration.

An echocardiogram recorded one week later is normal apart from a small pericardial effusion (Fig.). Right ventricular dimension, though not originally above the upper limit of normal, has diminished and left ventricular wall motion and mitral valve opening velocity are normal (33.9 ± 1.8 cm/s), with no respiratory variation. Fractional shortening has recovered to within the normal range at 41%, as normal septal motion is restored and posterior wall contraction has returned to normal. There has been no significant change in left ventricular end-diastolic dimension (4.2 cm).

Experimental results

The opening rate of the anterior mitral valve leaflet measured by echocardiography in 25 normal subjects did not vary with respiration and was 46.2 ± 13.4 cm/s; the pretreatment values observed in our patient lie

Fig. Echocardiograms from a patient with subacute massive pulmonary embolism, before and after thrombolytic treatment. CW, chest wall; RV, right ventricle; IVS, interventricular septum; LV, left ventricle; PLVW, posterior left ventricular wall.

outside the 95% confidence limits for normality (18.5–73.9 cm/s) (Table 1). The hypothesis that the reduction in mitral valve opening velocity was caused by reduced pulmonary venous return was investigated. M-mode echocardiograms were obtained in six normal subjects while they performed a Valsalva manoeuvre. Six measurements of valve opening velocity were made at the beginning and again towards the end of the procedure, a reduction being shown in all cases ($p < 0.001$, paired t test) (Table 2).

Discussion

The echocardiographic features commonly occurring in acute pulmonary embolism have been described by Kasper *et al.*² and include acute dilatation of the pulmonary artery and right ventricle, with an abnormal pattern of septal movement, and decreased mid-diastolic closure rate of the mitral valve. These changes

regress as the embolism resolves.^{1,2} A decrease in left ventricular compliance and a change in shape secondary to the bulging of the interventricular septum around the dilated right ventricle is the explanation for the decreased mid-diastolic mitral valve closure described in both acute and chronic pulmonary hypertension.^{5,7} In addition changes in cardiac output may occur with respiration, possibly because of pooling of blood in the pulmonary veins during inspiration. This may be detected clinically as pulsus paradoxus and by echocardiography as reduced or absent aortic valve opening during inspiration.⁸

The present case showed the expected reduction in right ventricular dimension as embolism resolved, and cyclical variations in ventricular size which may be the result of inspiratory filling of the failing or obstructed right ventricle. Changes in cardiac position in a severely dyspnoeic patient could also be responsible for these phasic alterations.

Table 1 Mitral valve opening velocity in normal subjects, and patient described with subacute massive pulmonary embolism

	25 normal subjects	Pulmonary embolus patient		
		Inspiration	Expiration	After treatment
No. of observations	25	9	9	6
Mean $\pm$ 1 SD cm/s	46.2 $\pm$ 13.4	11.3 $\pm$ 1.3	15.2 $\pm$ 1.7	33.9 $\pm$ 1.8

Table 2 Changes in mitral valve opening velocity in six normal subjects during a Valsalva manoeuvre $p < 0.001$ (paired t test)

Subjects	Quiet respiration cm/s $\pm$ 1 SEM	During Valsalva cm/s $\pm$ 1 SEM
1	36.52 $\pm$ 0.72	28.38 $\pm$ 1.39
2	37.58 $\pm$ 1.36	26.75 $\pm$ 1.06
3	41.83 $\pm$ 2.38	27.3 $\pm$ 0.71
4	21.25 $\pm$ 1.89	13.83 $\pm$ 0.31
5	38.17 $\pm$ 2.17	27.17 $\pm$ 0.53
6	49.0 $\pm$ 2.4	33.08 $\pm$ 1.02

There were two additional echocardiographic features in this patient with massive pulmonary embolism.

(1) Gross reduction of left ventricular wall motion which resolved when the pulmonary vascular obstruction cleared. Probably the main cause for this was myocardial ischaemia resulting from low cardiac output and severe hypoxaemia: both a result of impaired pulmonary perfusion.

(2) The mitral valve opened abnormally slowly. We were able to reproduce this phenomenon in normal subjects during forced expiration against an obstruction, that is the Valsalva manoeuvre. During this procedure pulmonary blood flow is impeded by the high intrathoracic pressure, and as cardiac output falls, a reduction in mitral valve opening velocity is observed. When the Valsalva manoeuvre is performed in normal subjects a reduction in both ventricular diastolic and systolic dimensions occurs.^{9 10} There was no obvious reduction in ventricular minor dimension seen in the acute stage in the case reported here. The Valsalva manoeuvre cannot mimic all the haemodynamic features seen in massive pulmonary embolism, but does severely impede forward flow into the left heart, probably with little effect on compliance.

The mitral valve opening velocity will depend on the rate of development of a pressure gradient across the mitral valve. This is controlled by the left atrial pressure, the rate of change of left ventricular compliance, and the development of "negative pressure" by the relaxing ventricle. In pulmonary embolism a much reduced left atrial inflow occurs and there is a possible reduction in left ventricular compliance caused by ventricular distortion and ischaemia. Mitral valve opening rate, and flow across the valve, is low in early diastole, though augmented later by atrial contraction as seen in the large "a" wave on the echocardiogram.

The lower mitral valve opening velocity seen during inspiration compared with expiration is in accord with

the reported inspiratory reduction in cardiac output in such patients.⁸ The clinical correlate of pulsus paradoxus was not observed.

The discovery of any of the echocardiographic features described in pulmonary embolism in a patient with progressive dyspnoea and echocardiographic reduction in left ventricular wall movement should alert one to the possibility of pulmonary thromboembolism being an alternative diagnosis to heart failure.

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