Contrast echocardiographic features of pulmonary hypertension and regurgitation*

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SUMMARY  Linear contrast echo configuration on the pulmonary valve M-mode echogram was assessed in 28 patients with pulmonary hypertension, in 10 with pulmonary regurgitation, and in 10 normal subjects. Contrast echo parallel lines filling the total systolic phase of the pulmonary valve were recorded in normal subjects. Contrast echo lines stopping in early systole around the pulmonary valve mid-systolic notch were seen in all the patients with pulmonary hypertension in relation to changes with the pulmonary flow. Contrast echo lines reversing in early diastole and crossing the pulmonary valve echogram during diastole were detected in all the patients with pulmonary regurgitation, consistent with the reversed flow across the valve. The use of contrast echocardiography to diagnose both pulmonary hypertension and regurgitation may provide further useful information, particularly when the orientation and time of appearance of the contrast echo lines are related to the systolic and/or diastolic phases of the pulmonary valve M-mode echogram.

A flat e-f slope, absent "a" wave, and mid-systolic closure of the pulmonary valve echogram were previously described as indicators of pulmonary hypertension.1-3 These echocardiographic features have been called into question4 and their reliability as a predictor of pulmonary hypertension remains uncertain. On the other hand echocardiographic features of pulmonary regurgitation have been described5 but they seem not to be specific. Koizumi et al.6 previously used contrast echocardiography to evaluate the linear configuration of contrast echoes in patients with pulmonary hypertension and regurgitation. The aim of this investigation was to assess the linear configuration of contrast echoes seen on M-mode tracings of the pulmonary valve in relation to the systolic and diastolic phases of the valve in normal subjects and in cases of pulmonary hypertension and regurgitation.

Methods

STUDY PATIENTS
We studied 30 patients who underwent cardiac catheterisation for various cardiac lesions, and 10 normal subjects. Twenty-eight of the patients had pulmonary hypertension, caused by mitral stenosis in 15, the Eisenmenger syndrome in two, cor pulmonale in five, post-infarction heart failure in three, and combined mitral and aortic stenosis and regurgitation in three. In 16 patients the pulmonary systolic pressure was 40 to 50 mmHg and in 12 patients it was over 50 mmHg. Ten patients had pulmonary regurgitation, in eight associated with pulmonary hypertension, in one with surgically corrected tetralogy of Fallot and the last with a dilated pulmonary artery in atrial septal defect. The diagnosis of pulmonary hypertension was suspected clinically and from the echocardiographic features of the pulmonary valve2,3 and was confirmed during cardiac catheterisation. The diagnosis of pulmonary regurgitation was made by clinical examination and phonocardiogram in all cases. Angiography of the main pulmonary artery performed in nine patients allowed us to diagnose pulmonary regurgitation clearly in four patients, and probably in five.

CONTRAST ECHOCARDIOGRAPHY
In all patients and normal subjects the M-mode echocardiograms were recorded from the parasternal and subcostal position according to previously described techniques.17 A peripheral (antecubital) vein injection of 2 to 10 ml of 5% glucose solution was carried out in all. A No. 19 butterfly needle, 1·1 mm diameter, or a No. 16 catheter 30 cm long and 1·65
mm diameter were used, inserted into the vein for about 10 to 15 cm. The number of injections varied from patient to patient. The pulmonary valve contrast echogram was recorded and evaluated directing the ultrasonic beam towards the pulmonary valve after having obtained a sufficiently clear visualisation of both systolic and diastolic phases of its motion. The pulmonary valve contrast echogram was analysed by assessing the presence, orientation, and time of appearance of the lines of contrast echoes in relation to the systolic and diastolic phases of the valve.

**Results**

A beam of parallel contrast lines was detected throughout the systolic phase of the pulmonary valve with a direction parallel to the b-c slope. In most subjects no contrast lines were recorded during diastole, but in others contrast lines parallel to the e-f slope were recorded during diastole. This pattern was observed in the normal subjects (Fig. 1 and 2).

A beam of contrast lines parallel to the b-c slope was detected during the first part of the systolic phase; in mid-systole the direction of the contrast lines reversed following the contour of the mid-systolic notch of the valve (rebound effect) and in some cases continuing parallel to the e-f slope for one- to two-thirds of diastole. This pattern was observed in all the patients with pulmonary hypertension (pulmonary systolic pressure higher than 50 mmHg) (Fig. 1 and 3). In two of these patients the pulmonary valve was recorded with the transducer positioned at the third to fourth left intercostal space and, therefore, directed upwards, obtaining a pulmonary valve contrast echogram similar to those of the normal subjects, but with contrast lines of turbulence during systole and diastole. By positioning the transducer at the second intercostal space and, therefore, directing the beam perpendicular to the pulmonary valve we obtained, in the same patients, a pulmonary valve contrast echogram similar to those described in most cases of pulmonary hypertension.

A beam of contrast lines was detected in early systole without the rebound effect. This pattern was often recorded in patients with mild pulmonary hypertension (pulmonary systolic pressure lower than 50 mmHg) (Fig. 1 and 4).

A beam of contrast lines parallel to the b-c slope was detected in systole; in late systole and early diastole the direction of the lines reversed following the d-e slope and crossing the diastolic phase of the pulmonary valve perpendicular to the e-f slope. This pattern was found in all the patients with pulmonary regurgitation (Fig. 1 and 5).
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Fig. 3 Contrast echogram of the pulmonary valve (PV) in a case of pulmonary hypertension. The inferior arrow indicates the mid-systolic notch at the level of which the contrast lines stop. The superior arrows indicate that late systole is free from contrast echoes. In some cases contrast echo lines following the contour of the pulmonary valve echogram were seen during this phase.

Fig. 4 Contrast echogram of the pulmonary valve (PV) in a patient with mild pulmonary hypertension. The arrows indicate an unclear mid-systolic notch of the pulmonary valve. The contrast echo lines stop in early systole.

Fig. 5 Contrast echogram of the pulmonary artery (PA) and valve in a case of pulmonary regurgitation. The horizontal inferior arrows indicate the systolic contrast echo lines, whereas the superior arrows indicate the diastolic ones (compare with the Q and T waves of the electrocardiogram). PO, pulmonary opening.

Contrast lines recorded in early systole with the rebound effect and in early diastole crossing the e-f slope of the pulmonary valve were found to be associated in all the patients with both pulmonary hypertension and pulmonary regurgitation (Fig. 1 and 6). Contrast lines crossing the diastolic phase of the pulmonary valve or detected in the right ventricular outflow tract with reversed direction, as compared with that in systole, were recorded in four patients with pulmonary regurgitation using the subcostal M-mode approach (Fig. 1). Using the two-dimensional subcostal view we also observed a to and fro motion of contrast echoes between the pulmonary artery and the right ventricular outflow tract. This feature was not recorded in the other patients with pulmonary hypertension and no pulmonary regurgitation.

Discussion

The M-mode echocardiographic features which suggest pulmonary hypertension are not always reliable. Among those described, the most dependable is the mid-systolic anterior motion of the pulmonary valve, but this is not easily recorded. Correlations between right systolic and diastolic time intervals and pulmonary pressure have been reported; the visualisation of the exact points of opening and closure of the tricuspid and pulmonary valve are, however, not always possible or clear. On the other hand, the angiographic diagnosis of pulmonary regurgitation is not accurate since imperfect closure of the pulmonary valve caused by
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In the occurrence of pulmonary hypertension we studied the contrast echo lines stopped in early systole around the mid-systolic notch (Fig. 1, 3 and 4). This effect can be explained by the fact that the halting of the microbubbles around the pulmonary notch results from a decrease in the pulmonary flow which slows down their diffusion. Afterwards, when the flow increases the contrast lines assume a variable configuration and flatter slope probably because of the decreased velocity of ejection that occurs in this phase of systole. It appears that the rebound effect of the contrast echoes detected in these cases is an expression of the change in pulmonary mid-systolic flow. This situation does not occur in normal subjects in whom the pulmonary flow is uniform. In fact in these cases the contrast lines neither stop nor reverse in systole, filling the total pulmonary systolic phase uniformly.

In pulmonary regurgitation the contrast lines in systole behaved either normally or as in pulmonary hypertension, when this was present. The important contrast echocardiographic features were observed during the diastolic phase of pulmonary valve motion. In the normal subjects there were usually no contrast echoes or contrast lines parallel to the e-f slope during diastole (Fig. 1 and 2) but in the presence of pulmonary regurgitation we observed contrast echo lines which changed direction, starting in early diastole, and in some of the cases in late systole, crossing the early and/or mid-diastolic echogram of the pulmonary valve (Fig 5 and 6). These features are an expression either of the reversed flow or of the gradient which occurs at the level of the pulmonary valve during diastole in pulmonary regurgitation.

Our findings confirm those of Koizumi et al., in suggesting a new noninvasive method for diagnosing pulmonary hypertension and regurgitation, in which the M-mode is superior to the two-dimensional technique.

We thank Dr Sonia Chang, Medical College of Ohio, Toledo, and Dr Graham Leech, St. George's Hospital, London, for reviewing the manuscript and for their advice.

Fig. 6 Contrast echogram of the pulmonary valve (PV) with both pulmonary hypertension and regurgitation. The arrows indicate the systolic and diastolic contrast echo lines.

the catheter may produce regurgitation of contrast into the right ventricle. Echocardiographic features of pulmonary regurgitation have been described but they seem to be neither specific nor reliable.

Because of this we attempted to verify whether there were more specific and reliable echo signs for diagnosing both pulmonary hypertension and regurgitation, assessing, through contrast echocardiography, the linear configuration of pulmonary valve contrast echoes as referred to by Koizumi et al.; we stressed particularly the direction of the contrast echo lines and their time of occurrence in relation to the closure, opening, and diastolic phase of the pulmonary valve.

In the presence of pulmonary hypertension the systolic notch of the pulmonary valve seems to be the result of changes of pulmonary flow which occur during the cardiac cycle. Tahara and Tanaka observed that when a pulmonary mid-systolic notch occurred the pulmonary flow decreased and later when the valve reopened in late systole the pulmonary flow increased. In all the patients with pulmonary hypertension we studied the contrast echo lines stopped in early systole around the mid-systolic notch (Fig. 1, 3 and 4). This effect can be explained by the fact that the halting of the microbubbles around the pulmonary notch results from a decrease in the pulmonary flow which slows down their diffusion. Afterwards, when the flow increases the contrast lines assume a variable configuration and flatter slope probably because of the decreased velocity of ejection that occurs in this phase of systole. It appears that the rebound effect of the contrast echoes detected in these cases is an expression of the change in pulmonary mid-systolic flow. This situation does not occur in normal subjects in whom the pulmonary flow is uniform. In fact in these cases the contrast lines neither stop nor reverse in systole, filling the total pulmonary systolic phase uniformly.

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