

## Short reports

# Refractory hypoxaemia after pneumonectomy: diagnosis by transoesophageal echocardiography

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### Abstract

**A patient with normal oxygenation before operation became very breathless and hypoxaemic after a right pneumonectomy. Transoesophageal echocardiography showed right to left interatrial shunting due to a patent foramen ovale. Transoesophageal echocardiography should be included in the assessment of patients who became inappropriately dyspnoeic after pneumonectomy.**

Dyspnoea after pneumonectomy is usually attributable to loss of alveolar volume and restriction of the pulmonary vascular bed. Chronic severe postoperative dyspnoea is almost invariably due to coexistent chronic airflow limitation. Very unusually, severe dyspnoea and hypoxaemia after right pneumonectomy have been ascribed to right to left shunting through a patent foramen ovale.<sup>1-3</sup> Previous reports of this extremely rare syndrome either have not included data

on preoperative arterial blood gases or lung function or have described patients with pre-existing hypoxaemia.

We describe a patient with normal preoperative oxygenation and exercise tolerance who became profoundly breathless after right pneumonectomy. This was due to right to left interatrial shunting through a patent foramen ovale, discovered only by transoesophageal echocardiography.

### Case report

A 64 year old ex-smoker underwent a right pneumonectomy for a T<sub>2</sub>N<sub>0</sub>M<sub>0</sub> squamous cell bronchogenic carcinoma. Preoperative assessment had shown normal arterial oxygen tension (Pao<sub>2</sub> 11.7 kPa) and carbon dioxide tension (Paco<sub>2</sub> 5.1 kPa), with minimal impairment of lung function (FEV<sub>1</sub>/FVC 2.3/3.5; transfer factor for carbon monoxide (TLCO) 74% predicted). Postoperatively he was persistently breathless at rest and his effort tolerance was limited to 20 metres. Arterial blood gas analysis while he was breathing air in the supine position showed a Pao<sub>2</sub> of 4.7 and Paco<sub>2</sub> of 3.4 kPa. His symptoms were exacerbated in the sitting position, when his oxygen saturation fell from 71% to 55%. Despite 40% oxygen there was little improvement in the hypoxaemia (Po<sub>2</sub> 6.5 kPa). An isotope lung perfusion scan of the left lung showed no perfusion defects, and he was transferred to our hospital for further assessment. On admission he was breathless at rest and centrally cyanosed when breathing room air. The left lung was normal clinically and radiologically. Penetrated views of the left main bronchus showed it to be normal. Assessment of lung function showed a forced vital capacity (FVC) of 2.15 litres (50% of predicted). Arterial blood gas tensions when he was breathing 100% oxygen were Pao<sub>2</sub> 13.0 and Paco<sub>2</sub> 4.3 kPa; oxygen saturation was 98.8%. The anatomical shunt was therefore more than 30% of the cardiac output.<sup>4</sup>

Right heart catheterisation showed that the mean right atrial pressure was 5 mm Hg, right ventricular pressure 35/0 mm Hg, and pulmonary artery pressure 35/12 mm Hg. Pulmonary angiography showed normal left pulmonary vasculature with no evidence of thromboembolic disease. The interatrial septum was not crossed during the catheterisation so a right to left shunt at atrial level could not be excluded. Transthoracic echocardiography failed to show the intracardiac structures as the heart and mediastinum were shifted to the right as a consequence of the pneumonectomy and lay beneath the sternum. Transoesophageal echocardiography was performed with a transoesophageal phased array probe (Toshiba PEF-511SA) with the patient in the left lateral position. This showed a defect in the interatrial septum (fig 1), across

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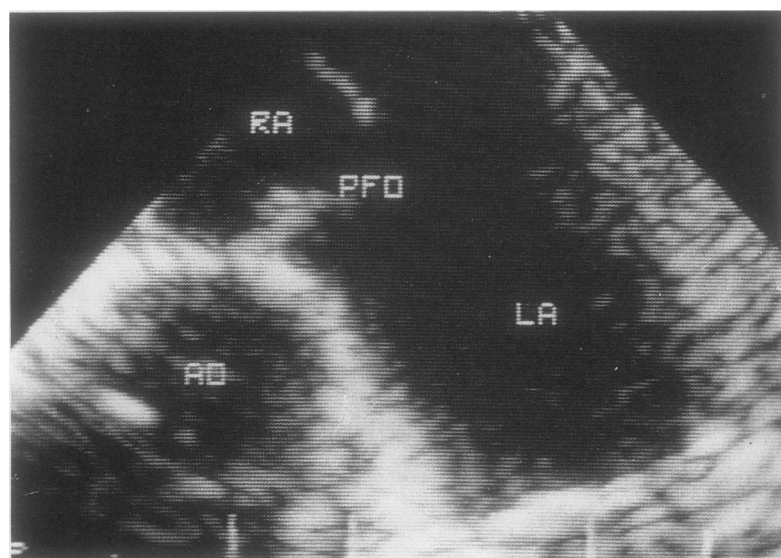


Figure 1 Transoesophageal echogram showing a patent foramen ovale (PFO). RA—right atrium; LA—left atrium; AO—aorta.

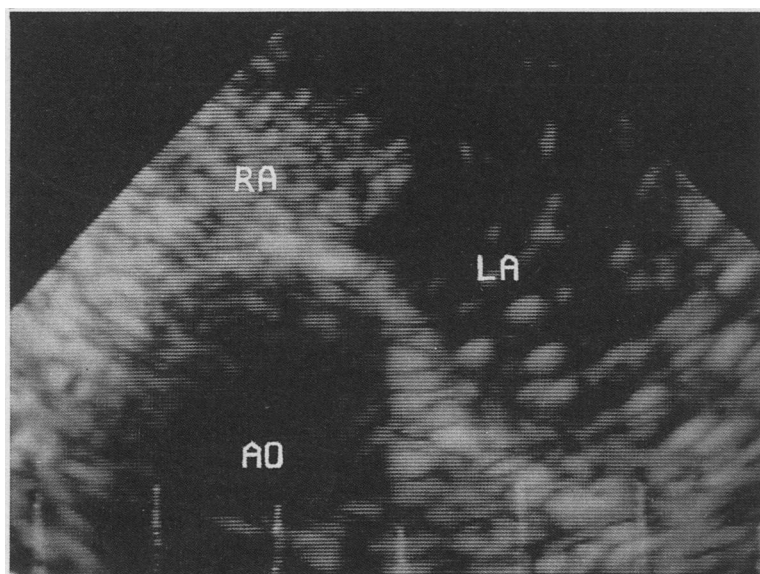


Figure 2 Passage of air micro-bubbles across the interatrial septum into the left atrium with a contrast injection. RA—right atrium; LA—left atrium; AO—aorta.

which air micro-bubbles could be seen to pass freely during a subsequent study with contrast medium (fig 2).

The patient underwent thoracotomy and at operation a widely patent foramen ovale was found and closed uneventfully. Immediately after operation a dramatic improvement in oxygenation was found ( $\text{FiO}_2$  0.4;  $\text{PaO}_2$  16.2 kPa) and a further transoesophageal echocardiographic examination confirmed closure of the defect. Despite an excellent technical result, his postoperative course was complicated by sepsis and subsequently respiratory failure and he died of multisystem organ failure four weeks after closure of the defect.

### Discussion

This patient presented after right pneumonectomy with gross hypoxaemia, despite persistently normal clinical and radiological appearances of the left lung. The differential diagnoses for these findings included pulmonary thromboembolic disease, major airway narrowing, respiratory muscle weakness, and right to left shunting. Investigations, including FVC when he was seated and supine, excluded the first three possibilities. The refractory nature of the hypoxaemia indicated a right to left shunt, which was confirmed by calculation of the anatomical shunt.

Confirmation that shunting is occurring at atrial level may be obtained by right heart catheterisation, retrograde left heart catheterisation, or echocardiography. Left and right heart catheterisation are invasive procedures and not without complications. Right heart catheterisation alone may be inadequate as it may prove difficult to cross the interatrial

septum, as in this case. Visualisation of the defect has been shown previously only after radio-opaque contrast injection from a catheter in the inferior vena cava.<sup>5</sup> Transthoracic echocardiography may also be inadequate—as in this case—because the heart is shifted to the right as a result of right pneumonectomy. In contrast, transoesophageal echocardiography affords excellent views of the interatrial septum irrespective of previous surgery and when combined with a contrast study is diagnostic.

The syndrome of postpneumonectomy shunting was ascribed in early reports to an interatrial pressure gradient.<sup>3</sup> Latterly, however, as in this case, the phenomenon has been clearly seen with normal right sided pressures.<sup>5-7</sup> The mechanism for the shunt is now thought to be mediastinal distortion caused by right pneumonectomy. The inferior vena cava remains fixed in position but the right atrium is shifted to the right and this favours opening of a foramen ovale. A characteristic feature of the syndrome seen in this patient is breathlessness and arterial desaturation in the upright position (platypnoea and orthodeoxia). This is presumably due to accentuation of mediastinal distortion in the upright position, with further shift of the right atrium and widening of the orifice of the patent foramen ovale.

The incidence of probe patent foramen ovale in adult life is about 25%.<sup>8</sup> The frank syndrome is therefore likely to represent one end of a clinical range of abnormal shunting and hypoxaemia after right pneumonectomy. Lesser degrees of shunt may be more common and should be borne in mind in assessing breathless patients after major right sided pulmonary resections when common causes of postoperative breathlessness have been excluded. This assessment should include a transoesophageal echocardiographic examination.

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