balloon dilatation of the mitral valve using the Inoue technique in young patients who are critically ill because of severe mitral valve stenosis.[1] We note that the procedure was performed while three of their patients were on assisted ventilation. Three patients had atrial fibrillation, but it is not clear whether this group overlapped with the patients who required ventilatory support.

In an earlier study percutaneous balloon dilatation of the mitral valve was associated with a 2–4% incidence of arterial emboli.[2] We believe that patients at high risk for embolic events should have transoesophageal echocardiographic (TOE) screening before valve dilatation because it has been shown that TOE is significantly superior to transthoracic imaging in detecting of important left atrial thrombus.[2 3]

We have successfully carried out Inoue balloon mitral valve dilatation in two patients (aged 61 and 59 years). Both had atrial fibrillation with severe left ventricular failure secondary to critical mitral stenosis and could not be weaned from intravenous inotropes or ventilatory support. In both patients transoesophageal assessment before the procedure showed spontaneous contrast in the left atrium but no left atrial thrombus. The mitral valve echocardiographic scores (Wilkins *et al*[4]) were 8 and 10. A significant reduction was obtained in the transmitral end diastolic gradient (50% and 80% respectively) and there was a 2–3 fold increase in the mitral valve area on echocardiography. Both patients were successfully weaned off ventilatory support within 24 hours of valve dilatation.

We suggest that percutaneous dilatation of the mitral valve using the Inoue balloon technique is as valuable in older patients who cannot be weaned from assisted ventilation as a result of critical mitral stenosis as it is in younger patients. Older patients are more likely to be in atrial fibrillation and should have transoesophageal echocardiography.

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1 Patel JJ, Munclinger MJ, Mitha AS, Patel N. Percutaneous balloon dilatation of the mitral valve in critically ill young patients with intractable heart failure. *Br Heart J* 1995; 73:555–8.
2 Thomas MR, Monaghan MJ, Smyth DW, Metcalfe JM, Jewitt DE. Comparative value of transthoracic and transoesophageal echocardiography before balloon dilatation of the mitral valve. *Br Heart J* 1992;68: 493–7.
3 Manning WJ, Reis GJ, Douglas PS. Use of transoesophageal echocardiography to detect left atrial thrombi before percutaneous balloon dilatation of the mitral valve: a prospective study. *Br Heart J* 1992;67: 170–3.
4 Wilkins GT, Weyman HF, Abascal VM, Block PC, Palicous IF. Percutaneous balloon dilatation of the mitral valve: an analysis of the echocardiographic variables related to outcome and the mechanism of dilatation. *Br Heart J* 1988;60:299–308.

This letter was shown to the authors, one of whom replies as follows:

SIR,—I appreciate the concern voiced by Davis and his colleagues regarding the role of transoesophageal echocardiography to exclude left atrial thrombus before percutaneous balloon dilatation of the mitral valve. At our hospital we have stopped performing routine transoesophageal assessment because in our experience the yield, following on a carefully performed transthoracic cross sectional echocardiographic study, was low.[1] This may be because most of our patients are young (mean age 30 years) and in sinus rhythm. Only 16% of all our patients undergoing balloon dilatation of the mitral valve have transoesophageal echocardiography. I do not, however, underestimate its important role in elderly patients who are in atrial fibrillation.

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1 Patel JJ, Dyer RB, Pillay R, Munclinger MJ, Mitha AS. Do all patients undergoing Inoue balloon mitral valvotomy need transoesophageal echo (TEE) to exclude left atrial thrombus? [Abstr]. *J Am Coll Cardiol* 1995; 89A.

Strut fracture of the convexo-concave Björk-Shiley mitral valve prosthesis

SIR,—Goodfield *et al* (*Br Heart J* 1995;73: 391) recommend an overpenetrated chest radiograph in posteroanterior and lateral or oblique views if strut fracture is suspected. This takes time when there is no time. Also, in a breathless patient with pulmonary oedema it would be difficult to acquire the high quality penetrated views required to show absence of the occluder.

Sudden total mitral regurgitation transforms a person from normal health to desperate straits virtually instantaneously. In a patient with this prosthetic valve, for practical purposes, there is no differential diagnosis.

Valve thrombosis has a more gradual onset and is less disastrous, with auscultatory signs of mixed stenosis and regurgitation. Loss of a mitral occluder produces pulmonary oedema without murmurs (no pressure gradient between ventricle and atrium). Prosthetic clicks are absent in both. Echocardiography is fast and fully informative but should only be done en route to the operating room.

Goodfield *et al* successfully treated their patient. The *x* rays which they show were taken with the chest open on the operating table.

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This letter was shown to the authors, who reply as follows:

SIR,—Professor Oakley is of course correct in her comments about our dramatic image in cardiology showing an intraoperative chest *x* ray with the disc occluder of a mitral Björk-Shiley convexo-concave valve lying within the aorta. The other *x* ray showed the fractured strut lying within the femoral artery. If a patient who is known to have a convexo-concave (CC) valve presents with sudden cardiovascular collapse and pulmonary oedema to a centre with cardiothoracic surgery, then of course the valve must immediately be replaced.

Many patients with implanted Björk-Shiley CC valves have not been identified and therefore do not carry information stating that they have this valve in place.[1] Most patients presenting to hospital with acute pulmonary oedema do not present to one that has cardiac surgery immediately available and with an experienced cardiologist to see that individual in that casualty department and make the diagnosis. Patients with prosthetic valves in situ who develop sudden pulmonary oedema should always of course be suspected of having valve dysfunction but this may also have other causes such as dehiscence secondary to endocarditis, or may be related to ventricular dysfunction, for example associated with arrhythmia. The absence of prosthetic clicks in a patient in extremis in a noisy accident and emergency department may be apparent only to very experienced observers. Cardiac surgeons would be unnecessarily overwhelmed if all such patients were transferred on presentation for immediate valve replacement.

There is a need for immediate and accurate diagnosis of outlet strut fracture and Hiratzka *et al* in a review of the subject have recommended "the single most expeditious study that should be readily obtainable in virtually any hospital or emergency treatment centre and that is highly effective in making the diagnosis of outlet strut fracture is an overpenetrated chest radiography, preferably in two views (posterior anterior and lateral or oblique)." We believe that this remains sound advice. Patients presenting with cardiovascular collapse caused by strut fracture of the valve may need immediate ventilation and a penetrated chest *x* ray may then permit the diagnosis. An intraoperative *x* ray can help identify the displaced components of the prosthesis and facilitate their removal, as was the case in the patient presented in our illustration.

Almost 100 000 patients are seen annually in the casualty department in our hospital and there are many cases of acute pulmonary oedema. The diagnosis may be obvious to an experienced cardiologist looking at a single illustration after the diagnosis has been made, but what is needed is straightforward advice for those dealing with a common medical emergency with an uncommon but catastrophic cause, and we believe that advice to obtain a penetrated radiograph in such cases remains sound.

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1 Hiratzka LF, Kouchoukos NT, Grunkemeier GL, Miller C, Skully HE, Wechsler AS. Outlet strut fracture of Björk-Shiley convexo-concave valve: current information and recommendations for patient care. *J Am Coll Cardiol* 1988;11:1130–7.