A 27 year old Algerian woman who was 13 weeks into her second pregnancy was referred to the cardiology clinic after being found to have a murmur at her antenatal visit. She had no cardiac symptoms or cardiac history but had miscarried at 10 weeks' gestation in her first pregnancy. Examination showed her to be in sinus rhythm with no signs of heart failure. She had the physical signs of mitral stenosis with a loud first heart sound and a low pitched mid diastolic murmur. Transthoracic echocardiography confirmed moderately severe mitral stenosis with an estimated valve area of 1.3 cm² and anatomical features consistent with a rheumatic cause. The estimated pulmonary artery systolic pressure was 35–40 mm Hg. Left ventricular function was normal and there was slight left atrial dilatation. The patient was managed conservatively through pregnancy with regular review and repeat transthoracic echocardiography. At 36 weeks' gestation, the patient attended a routine review having remained well throughout pregnancy.

Examination showed a mild rise of the jugular venous pressure at 3 cm above the clavicle and mild ankle oedema. Transthoracic echocardiography showed no change in her mitral stenosis (mitral valve stenosis of 1.4 cm²) and there was mild to moderate tricuspid regurgitation (fig 1). The estimated pulmonary artery pressure was, however, grossly increased at about 100 mm Hg and the interventricular septum was flattened suggesting pressure overload of the right ventricle (fig 2). The patient was admitted for close monitoring and a decision on further management. An oral diuretic (furosemide (frusemide) 40 mg once daily) was initiated. Echocardiography repeated after two doses of diuretic showed a dramatic reduction in pulmonary artery pressures to about 25 mm Hg (fig 3). The baby was delivered successfully by elective caesarean section at 37 weeks' gestation with invasive monitoring of the mother's pulmonary pressures. There were no complications.

DISCUSSION
Rheumatic mitral stenosis is the most common clinically significant cardiac abnormality seen in pregnant women worldwide. Stenosis of the mitral valve obstructs left ventricular filling resulting in increased left atrial pressure and reduced cardiac output. In severe mitral stenosis, pulmonary hypertension, pulmonary oedema, and right ventricular failure may develop. During pregnancy several haemodynamic changes exacerbate the cardiovascular aberrations associated with mitral stenosis. Intravascular volumes increase by up to 50% in the last trimester resulting in increased left atrial pressure and increased pulmonary venous filling,¹ and increased heart rate during pregnancy reduces left ventricular diastolic filling time. During natural labour cardiac output and blood pressure increase with uterine contractions, and pulmonary artery catheter measurements during labour record significant increases in left atrial pressure.¹ Immediately after delivery cardiac filling pressures increase dramatically due to vena caval decompression and return of uterine blood.

Pulmonary hypertension is associated with greatly increased maternal risk and moderately increased neonatal risk, with peripartum mortalities of 30–56% and 10–13%, respectively.¹ Elective caesarean section is therefore appropriate in conditions where pulmonary pressures are increased. Severe mitral stenosis with significant symptoms is associated with increased maternal and neonatal mortality (3.5% and 7.5%, respectively, in one large series²) and may require balloon mitral valvuloplasty.³ In this high risk group mortality and morbidity increases incrementally with maternal functional class and severity of stenosis.² Mortality among pregnant women with mild or moderate mitral stenosis and minimal symptoms, however, is less than 1%.⁴ Therefore management of asymptomatic patients with mild to moderate mitral stenosis is usually conservative, with
diuretics and β blockers recommended to minimise volume overload and prolong the diastolic filling period, respectively, if symptoms develop during pregnancy.

This case illustrates the dramatic physiological impact of volume overload and diuresis on pulmonary pressures in the peripartum setting in a patient with moderate mitral stenosis.

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