Infected endocarditis in neonates

Sir,
We read with interest the recent review of five patients with neonatal infective endocarditis by O'Callaghan and McDougall.1 We wish to report a further case where echocardiography had an essential role in the early diagnosis of pericarditis and endocarditis in a newborn infant where clinical signs specific to endocarditis were absent.

A boy weighing 3200 g was born vaginally at term to a primagravida who had loose bloody stools due to Shigella sonnei infection during the last trimester of pregnancy. The infant developed shigella enteritis with intermittent gaseous abdominal distension. Ultrasound examination showed an abdominal abscess but during this procedure the pericardial effusion was incidentally detected. Although the infant was ill and looked septic, arterial pulses and blood pressure were normal. The heart sounds were normal with no evidence of a murmur or pericardial rub. The chest radiograph showed cardiomegaly but the electrocardiogram was normal. A septic spot was noted on the sternum. A peripheral blood count showed chromytopenia but no petechial lesions were present. There was no haematuria.

Cross sectional echocardiography confirmed the presence of a large anterior pericardial effusion as well as a vegetation on the anterior leaflet of the mitral valve. Thirty ml of pus was obtained by needle aspiration of the pericardial space. Subsequent surgical exploration was performed and the pericardial space was left on continuous drainage for one week. An identical strain of Staphylococcus aureus was isolated from the pericardial fluid, blood cultures, and the septic lesion on the sternum. Gentamicin and cloxacillin to which the organism was sensitive were given intravenously for six weeks. After one week of treatment the pericardial effusion had resolved but the vegetation persisted. Umbilical arterial or venous lines were not used at any time during his management. He was discharged from hospital at the age of 3 months. At that time his cardiac examination was normal; in particular there was no evidence of mitral valve insufficiency. Echocardiography confirmed persistence of the mitral valve vegetation which had become more echodense and possibly calcified. After discharge from hospital the infant was lost to follow up.

Clinical detection of bacterial endocarditis in the newborn who is already ill from sepsis is difficult. Embolic phenomena, congestive cardiac failure, and changing heart murmurs are important clues but are infrequently found.2 Cross sectional echocardiography permits earlier diagnosis of endocarditis provided vegetations are at least 2 mm in diameter.2 We surmise that survival of our patient was due to early diagnosis and prompt treatment with appropriate antibiotics in high doses, and the maintenance of normal tissue perfusion and blood pressure in the presence of overwhelming septicemia. We concur with O'Callaghan and McDougall in the usefulness of echocardiography in the diagnosis of neonatal endocarditis. We have shown that with early diagnosis recovery is possible.

References
Patient triggered ventilation in premature neonates.

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