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Patient triggered ventilation in premature neonates

Sir,

Greenough and Greenhall¹ recently reported their experience of the trigger ventilator.² They modified our original circuit by switching the inspiration detector from its site on the abdomen to the oesophagus. We had considered this approach but felt that it had a limited role because it could not be used on a routine basis by untrained staff. In their paper they allege that 'gross body movements' made the abdominal sensor prone to false triggering. This has never been a problem in my experience over the last three years. While it is obviously theoretically possible to interfere with the signal from the capsule, in practice the design of the circuit largely eliminates this problem. This is best illustrated by the example of an infant on triggered ventilation with an inspiratory time set at its recommended 0.5 seconds. When a breath occurs there is a delay of up to 100 msec before the trigger fires. The ventilator is refractory to further signals from the time of firing, through the inspiratory time setting (0.5 seconds) and for 0.25 seconds after this interval (to allow enough time for expiration). Most infants breathe at 60 breaths (approximately) per minute with this inspiratory time (unpublished work). Thus on average the ventilator is refractory for 0.85 seconds out of the 1 second between breaths and only 0.15 seconds remain as a susceptible period before the next breath is due. The only consequence of such a false signal is to make the next breath occur fractionally early.

I submit therefore that the criticism of our original approach is invalid and that triggered ventilation using an abdominal sensor is an effective means of assisting the newborn's respiratory efforts.³

References

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Infective 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 primigravida 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 chromocytopenia 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 septicaemia. 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

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Varicella gangrenosa

Sir,

We read with great interest the account of a case of varicella gangrenosa by Kidney *et al*¹ and would like to report our experience with a similar case associated with an unusual complication.

Case report

A 3½ year old girl developed pain in her left leg eight days after chickenpox, when her chickenpox lesions were actually drying up. A couple of days later her parents noticed a few large, dark bruises appearing on her left leg. On examination she looked rather pale with a normal pulse rate and blood pressure. She had three large, dark, and tender ecchymotic areas on her left leg and similar, smaller lesions, one on each side, just above the ankle. The left leg was obviously swollen and she was unable to bear weight on that leg. Her peripheral pulses were normally palpable.

Initial investigations showed a mild anaemia (haemoglobin 100 g/l) with normal white cell and platelet counts. The prothrombin activity was reduced to 70%. Cephalin-kaolin time increased to 52 seconds and fibrinogen degradation products were >40 mg/l (normal <10 mg/l).

Despite fresh frozen plasma (10 ml/kg) her bruises increased in size in the 24 hours after admission. She was, therefore, started on prednisolone 2 mg/kg/day which seemed to control the spread of the skin lesions and the leg swelling. Her steroid dose was reduced, from the fourth day after admission, but on the fifth day she suddenly developed severe abdominal pain requiring a pethidine injection. She had tenderness in both renal angles and urine examination showed a few red blood cells but no protein. An intravenous pyelogram showed enlargement of both kidneys (left more than right). The right nephogram appeared immediately, the left slowly evolved over 5-10 minutes and the pyelogram on the left was also delayed, beginning to appear after 20 minutes. These findings added support to the clinical suspicion of a renal vasculitis and, probably, a renal vein thrombosis.

She failed to pass urine for 10 hours but, subsequently, produced urine normally. Blood pressure, urea, and electrolytes also remained normal. Plasma proteins and complement concentrations were normal and antiDNA antibodies were negative. Her symptoms quickly improved with intravenous dextrose saline infusion to maintain hydration, continued use of steroids, and intravenous ampicillin. We did not give anticoagulant treatment. The skin lesions became blistering after a few days; this was followed by thick eschar formation. This needed applica-

tion of streptokinase-streptodornase to remove the scab. It took about six weeks for the lesions to heal completely and they had left rather unsightly scars on the left thigh, tethered down to the underlying fascia.

Reference

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Very young children and the Family Fund

Sir,

When the Family Fund, which gives modest grants to families with severely handicapped children, started in 1973 its advisers almost ruled that no child under 2 years of age could satisfy the medical criteria.¹ Fortunately the possibility was left open and a small number of multiply handicapped babies, being nursed at home, have always been accepted.

In recent years, however, the Fund is receiving applications for and accepting as eligible an increasing number of such babies. In 1981 there were 920 applications on behalf of children under 2 and 50% of these infants were regarded as sufficiently handicapped to be eligible. By 1987 the number of applications had almost doubled and two thirds of them were accepted.

Developments in medical techniques and the increased possibility of litigation if a consultant is not seen to do his utmost to save life mean that more babies born with disabilities are surviving, and more who are born prematurely survive but develop disabilities in their struggle to hang on to life. More significant, from the point of view of the Family Fund, is the fact that such babies are often returned to the care of their parents, even when they require 24 hours nursing.

Research evidence has shown that there are considerable financial costs in caring for a disabled child at home,² and the government, in recently announcing the possible extension of attendance allowance to children under 2, has acknowledged that the actual problems of care of such a child are significantly more than that of a normal baby.³ It is vital that professionals should realise that the Family Fund has always recognised this and that families are not discounted because the child is not yet 2 years old

References

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