among those delivered operatively than among those delivered normally. The operative deliveries were undertaken for failure to advance in labour due to uterine, pelvic, or fetal factors, the fetal factors being either malpresentation or malposition.

We applied the Apgar score in our study because it remains widely used as a simple clinical index of the condition and behaviour of newborn babies. So far, nothing better has been found to replace it.

This study showed that both the sensitivity and the predictive value of an abnormal cardiotocograph tracing in labour for babies born with an Apgar score of less than 7 were poor, whether or not the babies required intermittent positive pressure ventilation. A normal tracing was obtained in 77% of all babies with an Apgar score of less than 7 and 65% of those who needed intermittent positive pressure ventilation. Cardiotocography gave a false positive prediction in 73% and a false negative prediction in 8% of all babies.

The positive predictive value of cardiotocography was significantly better in those babies delivered by emergency caesarean section compared with those delivered by forceps. Most caesarean sections were performed in the first stage of labour, and all forceps deliveries were performed in the second stage. We do not think that the assessment of the cardiotocograph tracings was different for the two groups, which suggests that cardiotocography is more reliable in the first stage than in the second stage of labour.

The Apgar score will probably continue to be widely used as a clinical criterion for a baby's condition at birth. As factors other than hypoxia may affect it we should not expect it to be closely correlated with intrapartum indicators of hypoxia. It follows that we should re-examine the current overdependence on fetal monitoring by cardiotocography and seek other reliable indicators for non-hypoxic fetal distress.

References

1 Sykes GS, Molloy PM, Johnson P, Stirrat GM, Turnbull AC. Fetal distress and the condition of newborn infants. *Br Med J* 1983;**287**:943-5.
2 Haverkamp AD, Thompson HE, McFee JG, Cetrulo C. The evaluation of continuous fetal heart rate monitoring in high risk pregnancy. *Am J Obstet Gynecol* 1976;**125**:310-20.
3 Kelso IM, Parsons RJ, Lawrence GF, Arora SS, Edmonds DK, Cooke ID. An assessment of continuous fetal heart rate monitoring in labour. *Am J Obstet Gynecol* 1978;**131**:526-32.
4 Renou P, Chang A, Anderson I, Wood C. Controlled trial of fetal intensive care. *Am J Obstet Gynecol* 1976;**126**:470-6.
5 Haverkamp AD, Orleans M, Langendoerfer S, McFee J, Murphy J, Thompson HE. A controlled trial of the differential effects of intrapartum fetal monitoring. *Am J Obstet Gynecol* 1979;**134**:399-412.
6 Beard RW, Filshie GM, Knight CA, Roberts GM. The significance of the changes in the continuous fetal heart rate in the first stage of labour. *Journal of Obstetrics and Gynaecology of the British Commonwealth* 1971;**78**:865-81.
7 Sykes GS, Molloy PM, Johnson P, *et al.* Do Apgar scores indicate asphyxia? *Lancet* 1982;i:494-5.
8 Crawford JS. Fetal distress and the condition of newborn infants. *Br Med J* 1984;**288**:567-8.
9 Steer P. Has the expression fetal distress outlived its usefulness? *Br J Obstet Gynaecol* 1982;**89**:690-3.

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SHORT REPORTS

Generalised epileptic fits in renal transplant recipients given cyclosporin A

Cyclosporin A is widely used to induce immunosuppression after organ transplantation. Reported neurological side effects include tremor, ataxia, confusion, paraparesis, and quadriparesis.[1 2] Epileptic fits have not been reported in patients given renal transplants but may occur after bone marrow transplantation.[3] In our unit cyclosporin A (5-10 mg/kg/day) is administered 12 hourly to achieve predose whole blood concentrations of 200-400 μg/l, as measured by radioimmunoassay.

Case reports

Case 1—Thirty days after cadaveric renal transplantation this patient developed right sided headache and blurring of vision and then had three generalised epileptic fits. On recovery neurological examination showed nothing abnormal. He was metabolically stable (plasma concentration of sodium 138 mmol(mEq)/l, potassium 4·8 mmol(mEq)/l, calcium 2·24 mmol/l (9·0 mg/100 ml), bicarbonate 18 mmol(mEq)/l, urea 33 mmol/l (199 mg/100 ml), creatinine 345 μmol/l (3·9 mg/100 ml)). No acute change in renal function was noted. A computed tomogram (CT) of the brain was normal. Cyclosporin A concentration was 584 μg/l.

Case 2—Four days after cadaveric renal transplantation this patient reported visual flashes and then had four generalised epileptic fits. On recovery neurological findings were normal. His metabolic state was stable (plasma concentration of sodium 139 mmol/l, potassium 4·9 mmol/l, bicarbonate 22 mmol/l, urea 36 mmol/l (217 mg/100 ml), creatinine 892 μmol/l (10·1 mg/100 ml), calcium 2·3 mmol/l (9·2 mg/100 ml)). Electroencephalography showed a normal rhythm. Cyclosporin A concentration was 1075 μg/l.

Case 3—Two months after a second transplant operation this patient felt unwell and saw rainbow bands; she then began jerking her head to the left and had five generalised epileptic fits. On recovery neurological examination elicited weakness of the left side of her mouth and exaggerated reflexes on the right side with clonus of the knee. These signs cleared over the next two days. She was metabolically stable (plasma concentration of sodium 135 mmol/l, potassium 4·2 mmol/l, urea 56 mmol/l (337 mg/100 ml), creatinine 467 μmol/l (5·3 mg/100 ml), calcium 2·55 mmol/l (10·2 mg/100 ml)). CT showed a low density area in the white matter of the right parietal lobe, which had disappeared at the next examination one week later. Cyclosporin A concentration was 533 μg/l.

COMMON CLINICAL FEATURES

Each of these patients had generalised epileptic fits for the first time in their lives while receiving cyclosporin A after renal transplantation. Phenytoin was prescribed as an anticonvulsant measure and the cyclosporin A reduced to achieve blood concentrations of 200-400 μg/l (recommended range). The patients had no further fits. None had an acute rejection episode, although the third patient, whose renal transplant function was poor initially, required chronic ambulatory peritoneal dialysis for two months after the episode. One month later her transplanted kidney was functioning well. These patients were receiving no other drugs which might have been responsible for the fits. Their blood pressures were stable and there was no evidence of hypertensive encephalopathy. The patients were followed up for four, 18, and three months, respectively; there was no recurrence of fits and all three transplants were functioning well (serum creatinine concentration < 300 μmol/l (< 3·4 mg/100 ml)).

When the epileptic fits occurred in cases 1 and 2 an initial diagnosis of rejection was made, but this was not proved.

Comment

Epileptic fits in patients with transplants may be caused by infection, rejection, or metabolic upset. So far as we could tell these were ruled out in our patients: all had normal cerebrospinal fluid findings, the serial serum viral titres did not rise, and their weight and metabolic states were stable. Space occupying lesions and other organic lesions of the central nervous system were also unlikely, as CT scans and electroencephalograms showed no persistent abnormality. Epileptic fits after renal transplantation may occur due to encephalopathy associated with an acute rejection episode,[4] but none of our patients had acute rejection at the time.

Epileptic fits in the absence of a conventional cause in three of our patients over 18 months led us to suspect that cyclosporin A might be responsible or at least a predisposing factor. Although epileptic fits caused by cyclosporin A have not been reported in renal transplant recipients, epileptic fits have been reported in association with cyclosporin A given after bone marrow transplantation.[3 5]

The pharmacokinetics of cyclosporin A are not fully understood,[3] and the present method of monitoring the drug is not always satisfactory[2]; hence concentrations in the blood may not accurately reflect tissue concentrations, particularly in the cerebrospinal fluid and brain. This aspect of cyclosporin A usage needs further investigation. Nevertheless, the epileptic fits were associated with predose cyclosporin A blood values above the recommended range.

We suggest that if epileptic fits occur in patients receiving cyclosporin A after renal transplantation the drug should not be stopped but the blood concentrations brought within the recommended range and the fits controlled with anticonvulsants.

1 Atkinson K, Biggs J, Darveniza P, *et al.* Cyclosporin A associated central nervous system toxicity after allogeneic bone marrow transplantation. *N Engl J Med* 1984;**310**:527.
2 Rolles K, White DJG, Thiru S, *et al.* Cyclosporin A initially as the only immunosuppressant in 34 recipients of cadaveric organs: 32 kidneys, 2 pancreases and 2 livers. *Lancet* 1979;ii:1033-6.
3 Durrant S, Chipping PM, Palmer S, *et al.* Cyclosporin A, methylprednisolone and convulsions. *Lancet* 1982;ii:829-30.
4 Gross MLP, Pearson R, Sweny P, *et al.* Rejection encephalopathy. *Proc Eur Dial Transplant Assoc* 1981;**18**:461-4.
5 Joss DV, Bassett AJ, Kendr JP, *et al.* Hypertension and convulsions in children receiving cyclosporin A. *Lancet* 1982;i:906.

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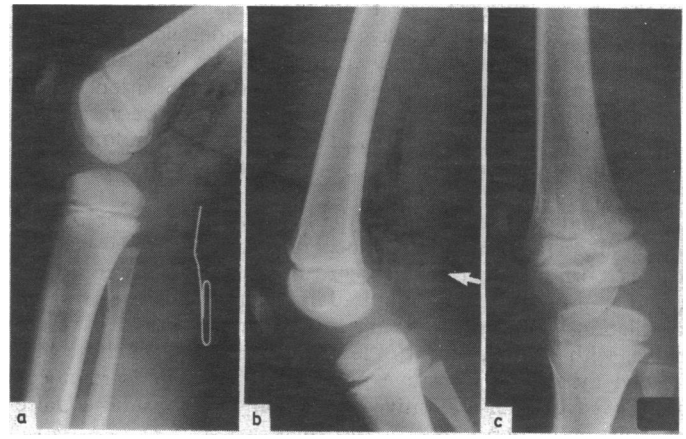
Sucking wound of the knee: not gas gangrene

Sucking wounds develop when a one way valve and negative pressure allow air to enter the tissues; they are commonly seen in chest and neck injuries.[1] We report a case of air entrapment in the leg and discuss the differential diagnosis of gas gangrene.

Case report

An eight year old boy presented in the casualty department with pain in the back of the right thigh. Eight hours earlier, while playing football, he had felt something penetrate the skin at the back of the right knee. He removed a 2 cm splinter and discarded it without paying any attention to its nature. He continued playing football and did not experience any pain until the middle of a karate session seven hours later.

When seen in casualty he was irritable, tired, and mildly feverish (37·6°C). There was a 2 mm puncture wound over the lateral aspect of the popliteal fossa with associated fullness and crepitus extending proximally up the thigh. No foreign body was palpable, and he had slight limitation of knee movements alone. There was no evidence of cellulitis, lymphadenopathy, or neurovascular injury. A radiograph of the knee (figure) confirmed the presence of gas in the soft tissues. This finding raised the suspicion of gas gangrene, but further radiography two hours later confirmed the diagnosis of air entrapment in the tissues (figure). He was treated with parenteral antibiotics and discharged from hospital 48 hours later. On review in the outpatient department four days after injury he was asymptomatic and radiography showed complete resorption of the gas (figure).



Radiographs in boy with small puncture wound (a) at admission, showing gas in soft tissues, (b) two hours later, showing (arrow) air entrapment, and (c) four days later, showing complete resorption of gas.

Comment

Air entrapment in the leg after small puncture wounds is extremely rare. In this case knee flexion may have opened up the skin wound and increased the volume of the popliteal fossa, creating subatmospheric conditions and sucking in air; knee extension may then have sealed the skin wound and forced the air into the tissue planes deep and proximally.

Gas gangrene is rarely seen in civilian practice and is characterised by swollen, crepitant tissues. Its early diagnosis and treatment are essential to avoid serious complications. The incubation period may be as short as four hours but is on average one to two days.[2] Gas forming clostridia may be introduced into wounds as spores, which, in the presence of tissue necrosis or foreign particulate matter, especially calcium and silicates, germinate and produce toxins. It is diagnosed clinically, being characterised by swollen, oedematous, painful tissues with a scanty serosanguinous exudate and crepitus. Rapid spread along the length of skeletal muscles and associated systemic effects, reflected in a rise in temperature and pulse rate, lead ultimately to death. Treatment includes the administration of penicillin, polyvalent antitoxin, surgical debridement, and hyperbaric oxygen.

We thank Mr R A Elson for allowing us to report this case.

1 Harris JH. The significance of soft tissue injury in the roentgen diagnosis of trauma. *C R C Critical Reviews in Clinical Radiology and Nuclear Medicine* 1975;**6**:295-368.
2 Wilson GS, Miles A. Gas gangrene: clostridial infections of man and animals. In: Topley WCC, Wilson GS, eds. Topley and Wilson's *principles of bacteriology, virology and immunity*. London: E Arnold, 1972:2258-77.

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Patterns of ovarian cyst hospital discharge rates in England and Wales, 1962-79

Ovarian cysts are the fourth most common gynaecological cause for admission to hospital in England and Wales, with 14 000 discharges annually. Little is known about their aetiology or distribution in the population. This report summarises the age distribution of women admitted to hospital with ovarian cysts and describes trends in hospital discharge rates in England and Wales from 1962 to 1979.

Methods and results

Hospital discharge data were obtained from the Hospital In-Patient Enquiry (HIPE), a 10% sample of discharges from NHS hospitals. Included were all discharges where the main diagnosis was either retention cyst or benign neoplasm of the ovary (9th ICD codes 620·0-620·2,220). From 1962 until 1979 the all ages hospital discharge rates for ovarian cysts have decreased by 15%—from 59·3 to 51·4 per 100 000—which is highly statistically significant ($p < 0·01$). The age specific discharge rates for the three year periods 1962-4, 1969-71, and 1977-9 are shown in the figure. The age pattern has been stable throughout the years, rates being low until puberty and peaking at 25-34. There is a dramatic decline from ages 45-54, but after 55 there is only a small further decline in the rates. Each age group except those 0-14 years old has experienced decreasing rates since 1962-4. These decreases were significant ($p < 0·01$) except in the 35-44 year olds.