reduced during the first six months by non-closure of the defect. Presumably non-closure allows cerebrospinal fluid to escape from the open neural tube, postponing the development of hydrocephalus until epithelium has formed. As deferred shunt operations are associated with a better prognosis,[4] non-closure may in fact be advantageous, delaying the onset of ventriculitis until the child is immunologically more competent.

Thus early closure has no advantage over non-closure with respect to mortality and the incidence of hydrocephalus and ventriculitis. Non-closure not only means that neonates are spared major operations but also reduces the number of shunt operations required in children who have a limited life expectancy. Knowing that non-closure is safe and potentially beneficial means that the family does not have to make an urgent and immediate choice between life and death on the birth of a baby with an open neural tube defect,[5] allowing time for all concerned fully to assess the baby's condition.

1 Mawdsley T, Rickman PP. Further follow-up study of early operation for open myelomeningocele. *Dev Med Child Neurol [Suppl]* 1969;**20**:8-12.
2 Smyth BT, Piggot J, Forsythe WI, *et al.* A controlled trial of immediate and delayed closure of myelomeningocele. *J Bone Joint Surg [Br]* 1974;**56**:297-304.
3 Boston VE, Wilkinson AJ. A retrospective analysis of conservative versus active management in severe open myelomeningocele. *Z Kinderchir* 1979;**28**:340-7.
4 Hemmer R. Surgical treatment of hydrocephalus: complications, mortality, developmental prospects. *Z Kinderchir* 1977;**22**:443-9.
5 Menzies RG, Parkin JM, Hey EN. Prognosis for babies with meningomyelocele and high lumbar paraplegia at birth. *Lancet* 1985;ii:993-5.

(Accepted 14 January 1988)

Royal Belfast Hospital for Sick Children, Belfast BT12 6BE
G T DEANS, FRCS, surgical registrar
V E BOSTON, MD, FRCS, consultant paediatric surgeon

Correspondence and requests for reprints to: Mr Boston.

Nephrotic syndrome after treatment with 5-aminosalicylic acid

Several new derivatives of sulphasalazine that make use of its active moiety, 5-aminosalicylic acid, have recently been introduced for the treatment of inflammatory bowel disease.[1] In rats short term intravenous administration of 5-aminosalicylic acid has been associated with nephrotoxicity.[2] No cases of minimal change nephropathy have been reported with either sulphasalazine or 5-aminosalicylic acid in man. We describe a patient who developed a nephrotic lesion within five months after starting treatment with mesalazine (5-aminosalicylic acid coated with an acrylic based resin).

Case report

A 61 year old white woman with a 30 year history of ulcerative colitis was admitted because of the sudden onset of swelling of her face and fingers. Until five months before admission her colitis had been well controlled with occasional betamethasone retention enemas. Colonoscopy showed a severe colitis up to 30 cm from the anal margin; this responded to a short course of steroid enemas. Because she did not tolerate sulphasalazine she was started on mesalazine 800 mg thrice daily.

Examination showed periorbital, ankle, and sacral oedema. Blood pressure was 120/70 mm Hg. Laboratory investigations showed a full blown nephrotic syndrome: serum albumin concentration 16 g/l cholesterol concentration 11·3 mmol/l, and proteinuria 28 g/24 h (90% albumin as determined by urinary protein electrophoresis). Serum creatinine concentration was 141 μmol/l, and urea concentration 13·3 mmol/l. The urinary sediment was unremarkable. Biopsy of the duodenum and colon showed no amyloidosis. A percutaneous renal biopsy showed normal glomeruli with slight proliferation of the mesangium. Results of immunofluorescence were negative. Electron microscopy showed fusion of the foot processes but no other abnormality. Minimal change nephropathy was diagnosed.

Mesalazine was stopped and prednisone 1 mg/kg/day started. The nephrotic syndrome responded after six weeks of treatment. Prednisone was tapered off until being stopped after four months. The patient experienced a moderate flare up of her colitis three months after stopping prednisone, which again responded to steroid enemas. One year later her nephropathy was in complete remission.

Comment

5-Aminosalicylic acid is released in the large bowel on dissolution of the resin coating of mesalazine tablets.[3] Though it has been shown to induce tubular and renal papillary necrosis,[2] it has not been reported to cause minimal change nephropathy. Non-steroidal anti-inflammatory drugs, in particular the propionic class of these compounds, have been reported to cause acute interstitial renal failure with minimal change nephropathy.[4] Although both these renal lesions are usually present, lipoid nephrosis has occurred as the sole lesion.[5] In this context 5-aminosalicylic acid may be viewed as a non-steroidal anti-inflammatory agent.

After five months of treatment with mesalazine our patient developed a full blown nephrotic syndrome. Percutaneous renal biopsy showed the typical features of minimal change nephropathy. There was a temporal relation between treatment with mesalazine and the onset of the glomerulopathy, and withdrawal of the drug and prolonged treatment with corticosteroids resulted in complete remission of the nephropathy. We did not consider it ethical to rechallenge the patient with the drug. Although the nephropathy may have been coincidental or related to her basic illness, her nephrosis did not recur despite a relapse of the colitis. Given the strong temporal relation, we suggest that the glomerulopathy may have been caused by 5-aminosalicylic acid in a manner similar to other non-steroidal anti-inflammatory drugs.

1 Dew MJ, Cadwell M, Kidwai NS, Evans BK, Rhodes EJ. 5-Aminosalicylic acid in serum and urine after administration by enema to patients with colitis. *J Pharm Pharmacology* 1984;**35**:323-4.
2 Calder IC, Funder CC, Green CR, Ham KN, Tange JD. Nephrotoxic lesions from 5-aminosalicylic acid. *Br Med J* 1972;i:52-4.
3 Dew MJ, Ryder REJ, Evans N, Evans BK, Rhodes EJ. Colonic release of 5-aminosalicylic acid from an oral preparation in active ulcerative colitis. *Br J Clin Pharmacol* 1983;**16**:185-7.
4 Finkelstein A, Fraley DS, Stachura I, Feldman HA, Gandy DR, Bourke E. Fenoprofen nephropathy: lipoid nephrosis and interstitial nephritis. A possible T-lymphocyte disorder. *Am J Med* 1982;**72**:81-7.
5 Chatterjee GP. Nephrotic syndrome induced by tolmetin. *JAMA* 1981;**246**:1589.

(Accepted 9 December 1987)

Meir General Hospital, 44281 Kfar-Saba, Israel
B H NOVIS, MB, MRCP, head of gastroenterology unit
Z KORZETS, MB, BS, staff nephrologist
P CHEN, MD, registrar in medicine
J BERNHEIM, MD, head of nephrology unit

Correspondence to: Professor Novis.

Firefighting and malignant hyperthermia

The complication of anaesthesia malignant hyperthermia occurs in people who have an underlying disorder of the muscle membranes. Such people may also present with heat stroke[1] or with rhabdomyolysis induced by phenothiazine drugs or severe infections.[2] We believe this is the first description of an occupational cause of rhabdomyolysis in someone known to be susceptible to malignant hyperthermia.

Case report

In July 1985 the 43 year old father of a 12 year old girl who had survived an episode of malignant hyperthermia during a general anaesthesia was found to have a raised serum creatine kinase activity of 650 IU/l (normal range 10-200 IU/l). In vitro testing of muscle with halothane and caffeine showed that he too was susceptible to malignant hyperthermia. At that time he was working as a computer salesman, but he subsequently got a job in a factory that made fire extinguishers. One of his tasks was to discharge bromochlorodifluoromethane from the extinguishers before refilling them. This work was usually done in the open air, but it was hard to avoid inhaling some of the gas. In March 1987 he consulted a physician (DGB), complaining of malaise and of stiffness and weakness in his forearms and hands during the 18 months that he had been in this job. The symptoms worsened during the working week, being worst on Fridays, and improved at weekends. Serum creatine kinase activity was 1056 IU/l on one Saturday in March 1987 and 544 IU/l on the following Monday. Physical examination showed no abnormality.

Because of the structural similarity between bromochlorodifluoromethane and halothane (bromochlorotrifluoroethane) the effect of bromochlorodifluoromethane on muscle contraction in vitro was examined. Bromochlorodifluoromethane produced an effect identical with that of halothane in inducing contraction in muscle from both humans and swine susceptible to malignant hyperthermia. He was advised to change his job, which he did; his symptoms immediately improved.

Comment

The clinical and laboratory findings in this patient indicated that he had rhabdomyolysis due to recurrent exposure to bromochlorodifluoromethane.

This was confirmed when he was no longer exposed to the chemical and his symptoms improved.

People susceptible to malignant hyperthermia can lead a normal life provided that they take certain precautions, which include avoiding inappropriate anaesthetic agents and neuroleptic drugs and not taking severe exercise in hot conditions. Avoiding occupational exposure to halogenated hydrocarbons such as bromochlorodifluoromethane should be added to the list as these may cause rhabdomyolysis.

1 Denborough MA. Heat stroke and malignant hyperpyrexia. *Med J Aust* 1982;1:204-5.
2 Denborough MA, Collins SP, Hopkinson KC. Rhabdomyolysis and malignant hyperpyrexia. *Br Med J* 1984;288:1878.

(Accepted 2 February 1988)

Department of Medicine and Clinical Science, John Curtin School of Medical Research, Australian National Univesity, Canberra, Australian Capital Territory 2600, Australia
M A DENBOROUGH, DSC, FRCP, professorial fellow
K C HOPKINSON, AAPPLSC, technical officer

Wynnum Central, Queensland 4178
D G BANNEY, FRACP, neurophysician

Correspondence to: Dr Denborough.

Are routine superficial cultures worth while in neonatal practice?

Financial prudence and the optimum use of scarce resources are assuming increasing importance in medical management. In this context we assessed the clinical value of superficial bacterial cultures taken either routinely on admission of infants to a paediatric unit or as part of a diagnostic investigation to exclude sepsis.

Patients, methods, and results

We reviewed the results of superficial cultures performed for all neonates (age 4-43 days) admitted from home to an infant unit through the outpatient department over six months. We also reviewed the results of superficial and central cultures performed to investigate suspected sepsis in neonatal inpatients (term babies aged up to 2 weeks and preterm babies until age at discharge) over 12 months.

Of 1050 superficial bacterial cultures performed for 273 infants admitted from home, 150 (14%) of 29 throat swabs, 72 nasal swabs, and 49 rectal swabs, yielded pathogenic organisms. *Staphylococcus aureus* (62 nasal swabs) and *Escherichia coli* (12 throat swabs and 14 rectal swabs) were the most common organisms. There was little correlation between results and site: *S aureus* was isolated from several superficial sites in three cases, and *E coli* from throat and nasal swabs but not from a rectal swab in one. In 15 cases the results on culture of routine rectal swabs led to the infants being isolated. No other routine culture performed without a clinical indication affected management.

In all, 241 microbiological examinations to confirm sepsis were performed for 225 patients over 12 months; 1142 cultures (776 superficial, 241 of blood, and 125 of cerebrospinal fluid) were obtained. Organisms were isolated from 51 of the blood cultures, but low colony counts of *S albus* (14 cultures) and *S aureus* (one) suggested possible contamination.

The same organism was grown from superficial and blood cultures in four cases and from blood cultures and rectal swabs in two: in one case *S aureus* was grown from a nasal swab and blood culture, and in one *Proteus mirabilis* was grown from a throat swab and blood culture. Overall the results of superficial cultures agreed with those of blood cultures in four but not in 19 of the 36 infants with septicaemia.

Swabs of the ear or eye were not taken routinely to investigate sepsis, but *S albus* was grown from ear swabs from two infants with septicaemia caused by this organism and *Listeria monocytogenes* was grown from swabs of eyes and ears from an infant with listeria septicaemia.

Five cases of bacterial meningitis were diagnosed from the 125 cultures of cerebrospinal fluid; the findings in 10 other cases suggested viral meningitis. A blood culture and a rectal swab from an infant with *E coli* meningitis also yielded *E coli*, but in the four other infants with bacterial meningitis the results as culture of the superficial swab did not correlate with those from the cerebrospinal fluid.

Comment

The results of culturing superficial swabs did not correlate well with the infecting organism. Gooch and Britt in a study of 9000 newborn babies found that 2·4% of the colonised group became infected as opposed to 0·3% of the non-colonised group.[1] Our results obtained from superficial cultures and blood cultures made simultaneously did not confirm the value of superficial cultures. In only three (8%) of the 36 infants with septicaemia did the results of the superficial cultures alter management. If management had been based on the results of superficial cultures inappropriate antibiotics might have been selected. The only possible value of routine superficial swabs taken on admission was in identifying infants who carried pathogenic organisms in their bowel and required isolation.

Superficial cultures may identify the local bacterial flora in infants and help in selecting antibiotics.[2,3] In practice, treatment is started on the basis of clinical indications at the initial examination and reviewed when the results of cultures of blood or cerebrospinal fluid, or both, become available. The clinical condition of the child largely determines whether antibiotics are used and so limits the usefulness of superficial cultures. Our study further suggests that such cultures are of limited value as a routine procedure when outpatients are admitted.

In an era of financial austerity clinicians should satisfy themselves that the information obtained from routine superficial cultures of bacteria is worth while.

1 Gooch JJ, Britt EM. Staphylococcus aureus colonization and infection in newborn nursery patients. *Am J Dis Child* 1978;132:893-6.
2 Gray OP, Campbell AGM, Kerr MM, *et al*. The newborn. In: Forfar JO, Arneil GC, eds. *Textbook of paediatrics*. 3rd ed. Edinburgh: Churchill Livingstone. 1984:117-258.
3 Klein JO, Remington JS, Marcey SM. Current concepts of infections of the fetus and newborn infant. In: Remington JS, Klein JO, eds. *Infectious disease of the fetus and newborn infant*. 2nd ed. Philadelphia: WB Saunders, 1983:1-27.

(Accepted 2 February 1988)

Department of Paediatrics, Rotunda Hospital, Dublin 1
G P FOX, MB, DCH, paediatric registrar
T A CLARKE, FRCP(C), DCH, consultant neonatologist
T G MATTHEWS, FRCPI, DCH, consultant neonatologist

Correspondence to: Dr Fox.

Anorexia nervosa in a 70 year old man

We report on a 70 year old man with a 40 year history of anorexia nervosa dating from the time he spent as a prisoner of war under the Japanese.

Case report

A 69 year old man presented with abdominal pain. He had needed a partial gastrectomy six years previously for a gastric ulcer. A perforated stomal ulcer was found at laparotomy and a Polya gastrectomy done. Postoperatively he refused to eat or drink and had difficulty with mobilisation. He weighed 31 kg. Neurological examination showed generalised wasting and weakness of his muscles, especially proximally. Reflexes and sensation were normal. Muscle biopsy showed considerable atrophy of type two muscle fibres as seen with disuse and in cachexia. An electromyogram showed a pattern of chronic denervation, and electrodiagnostic studies showed severe sensorimotor neuropathy. Routine biochemical and haematological measurements including creatine kinase were normal.

A year later he was referred again because of his low weight and immobility. He weighed 31 kg and could not sit up in bed or stand unaided. He admitted that his weight had been low and that he had restricted his food intake for years. During the second world war he had been a prisoner of war under the Japanese and had worked on the construction of the Burma to Thailand railway. He avoided talking about his experiences and gave the impression that his captivity had had a profound effect on him. After the war he had worked as a storeman and packer until his retirement at the age of 49. Since his captivity he had been a poor eater. There was no history of vomiting or bulimia. He had married in 1946 but did not have any children. He and his wife were reluctant to talk about their sexual relationship. There was no family history of anorexia nervosa, depression, or any other psychiatric illness.

His serum zinc, iron, phosphate, protein, and albumin concentrations were low. Alkaline phosphatase and γ-glutamyltransferase activities were raised. Other biochemical and haematological variables were normal. Barium studies confirmed an almost total gastrectomy, but no other abnormality was seen. Nasogastric feeding was started. Fifteen days later he weighed 35·2 kg. The next day he complained of abdominal discomfort and distension. Low pressure suction was started, but he died suddenly that night. Permission for a postmortem examination was refused.

Comment

Anorexia nervosa is an underdiagnosed disorder, especially in men. In this case considerable circumstantial evidence favoured a diagnosis of anorexia