the children of diabetic mothers and suggested that the problem in the infant results from impaired intestinal motility, possibly promoted by glucagon, rather than from an intraluminal plug. In several of their patients an element of obstruction persisted after complete evacuation of meconium and it has been shown that the small calibre of the left colon can persist for variable periods despite normal bowel activity (Davis *et al.*, 1974). Their hypothesis is that an increase in glucagon excretion occurs in babies in the perinatal period either secondary to hypoglycaemia associated with maternal diabetes in a complex way, or with stress. Glucagon is known to decrease bowel motility in the jejunum and sigmoid colon. Conversely hypoglycaemia can also increase bowel motility by vagal and sympathetic stimulation, and consequently Stewart *et al.* (1977) have proposed that the immature small intramural nerve cells are unable to respond to increased sympathetic stimulation. The results of further studies are awaited.

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Acute necrotising fasciitis due to streptococcal infection in a newborn infant

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SUMMARY A 3-day-old baby girl developed septicaemia, meningitis, and necrotising fasciitis due to group A β-haemolytic streptococcus, type M52, which was also cultured from the mother's cervix. Necrotising fasciitis is a severe infection of the skin and subcutaneous tissues with infarction, necrosis, and sloughing of the affected areas. Early recognition of this condition is essential so that appropriate treatment can be given.

Acute necrotising fasciitis is a rare but severe bacterial infection of the subcutaneous tissue and fascial planes, producing extensive destruction of tissues (Hammar and Wagner, 1977). It is often associated with blood stream invasion by the causative pathogen, and the mortality is high. The condition is familiar to the surgeon, but it appears to be rare in children (Wilson and Haltalin, 1973), and only 3 cases have been reported in newborn infants (Weinberger *et al.*, 1972; Ramamurthy *et al.*, 1977).

We report a newborn infant who developed septicaemia, meningitis, and necrotising fasciitis caused by group A β-haemolytic streptococcus.

Case report

A term baby girl of 2·7 kg was born to healthy non-related parents after a pregnancy complicated only by a heavy leucorrhoea during the 3rd trimester. The membranes ruptured spontaneously 14 hours before a normal delivery. The Apgar score at one minute

was 9, and the initial examination was normal. On the 3rd day of life it was noted that she nursed poorly, and an erythematous lesion was observed on the right cheek. Within a few hours the baby's condition deteriorated. She became apathetic, comatose, and her rectal temperature rose to 39·5°C. The rash had spread to the scalp, the back of the neck, and the left upper arm. Initially the rash was indurated, erythematous, and warm. No regional lymphadenitis was noted. A severe infection was suspected.

The blood count showed a leucopenia of $1 \cdot 5 \times 10^9$/l and a thrombocytopenia of $55 \cdot 0 \times 10^9$/l. The next day the leucocyte count increased to $29 \cdot 6 \times 10^9$/l with 39% band cells and 5% metamyelocytes. Chest *x*-ray and urine analysis were normal. The spinal fluid was bloody. Cultures from the nose, skin, CSF, blood and from the mother's cervix grew β-haemolytic streptococcus group A, type M52. A repeat lumber puncture 2 days later showed clear CSF with cells $0 \cdot 028 \times 10^9$/l (mainly polymorphonuclears), a protein level of 220 mg/100 ml (2·2 g/l), and glucose of 47 mg/100 ml (2·6 mmol/l). The culture was negative.

Treatment was immediately started with IV penicillin, gentamicin, and chloramphenicol, and a double volume exchange transfusion was performed. When the results of the cultures were received treatment was continued with only penicillin and frequent transfusions of blood and plasma.

The infant's condition improved rapidly. She became alert and responsive. However, the skin rash changed to a dusky bluish-grey colour, vesicles appeared, and the rash spread along the fascial planes of the upper arm. Subsequently the skin became a purplish colour in some areas and extremely indurated (Fig. 1). The same process was seen in the neck but the rash on the right cheek and the scalp faded.

Gradually areas of necrosis appeared on the skin of the neck and left upper arm and the skin sloughed (Fig. 2). Debridement was performed and the skin was treated with dilute solutions (0·5%) chlorinated lime and boric acid solution (BPC) dressings. Granulation tissue appeared very slowly and it was therefore decided to treat the skin of the arm with pig skin treated with glutaraldehyde. There was rapid growth

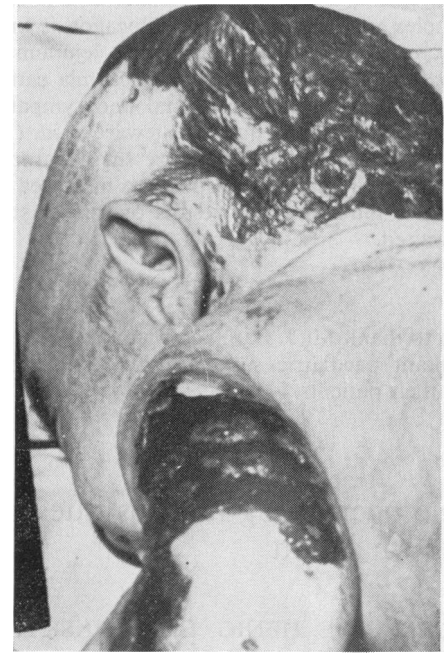

Fig. 2 *Necrotic areas of skin on left upper arm.*

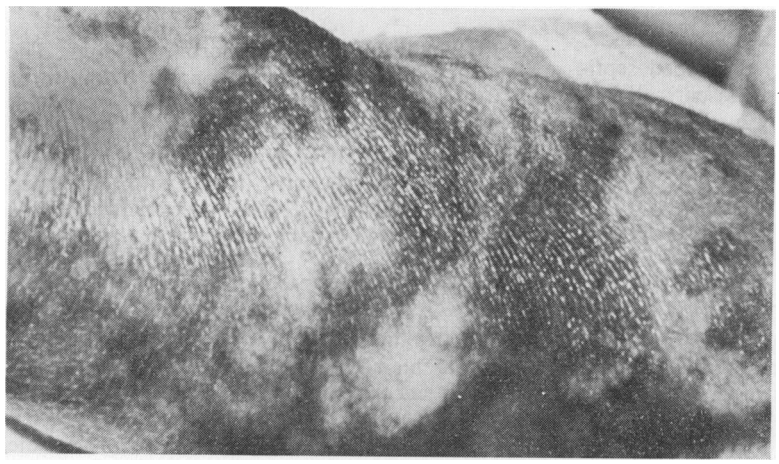

Fig. 1 *Lateral view of left upper arm showing the marked induration of the skin with areas having a purplish-black colour.*

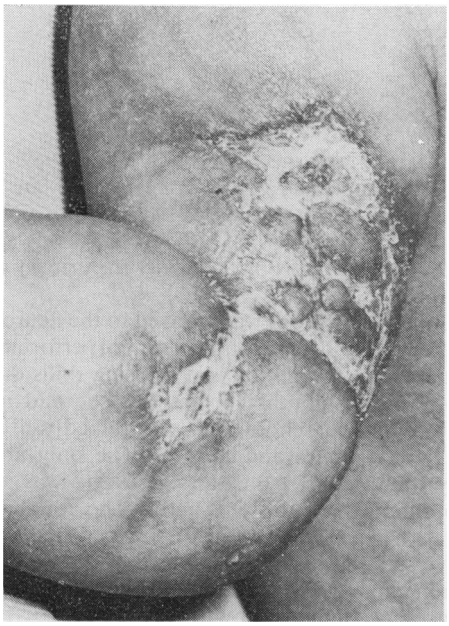

Fig. 3 *Scarring and cicatrisation of skin on left upper arm.*

of granulation tissue and the skin healed with good scarring (Fig. 3). No side effects were noted from the treatment with boric acid solution. The infant was discharged at age 2 months in good health; she was neurologically normal.

Discussion

Streptococcal septicaemia in neonates is usually associated with strains of Lancefield group B. Although no longer the scourge of maternity units, group A streptococci can still cause severe neonatal infection (Peter and Hazard, 1975). The infant may be infected by the mother who carries the streptococcus in her respiratory tract or in her vagina, as was the case in this infant, or by hospital staff.

Group A streptococcus is the most common cause of necrotising fasciitis, although other Gram-positive or Gram-negative organisms have also been reported. Joseph Jones (1871), an army surgeon during the American Civil War, first described this condition. Meleney (1933) gave a clear description of it. The area first becomes swollen, erythematous, warm, and painful. Unlike erysipelas, the border is not raised or well defined and the development of lymphangitis or lymphadenitis is unusual. The overlying skin develops a dusky bluish-grey colour and vesicles may appear. Fluid aspirated from these vesicles may be clear or turbid and usually contains the infecting organisms. The infection spreads rapidly along the fascial planes producing thrombosis of nutrient vessels with resultant necrosis of the overlying subcutaneous tissue and sloughing of the skin. Although the infection usually begins in an operative wound, it may develop without any apparent cause. Unchecked the process spreads rapidly, leading to debility, septicaemia, metastatic abscess, and death.

Therapeutic success depends on prompt recognition of the syndrome, early isolation of the infecting organism, treatment with appropriate antibiotics, vigorous debridement of superficial necrotic tissue, and incision of deep fascia to drain the underlying exudate. Supportive therapy with frequent transfusions of blood and plasma is essential. In our patient there was a rapid response to antibiotic treatment and the infant's general condition greatly improved. After debridement there was, however, very little granulation tissue formation and it was necessary to cover the area of the left upper arm with porcine skin. This resulted in rapid formation of granulation tissue and cicatrisation of the wound.

The infant was discharged at age 2 months, and at one year was found to be normally developed.

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