reported a tendency for their infants to sleep long periods, especially at night, from the early weeks. They were happy with their infants' progress throughout and were feeding 'on demand', being reassured by the knowledge that 'breast is best' and 'a hungry baby is a crying baby.'

Three mothers were encouraged to increase milk supply by feeding every 3 to 4 hours night and day. This succeeded in one infant (Case 4). The other infants gained weight rapidly only on starting bottle feeds.

Discussion

The case histories described in this paper are clear examples of infants who uncomplainingly fail to thrive from underfeeding. Furthermore, we believe from discussion with other paediatricians and nurses in other parts of the UK that this problem might be more common than is generally appreciated. Doctors and nurses should be alerted to the 'wasted' but contented breast-fed infant. This will only be achieved if the emphasis in infant welfare clinics is on accurate measurement of naked weight with a clinical estimate of fat folds and careful use of up-to-date weight charts.

Why some apparently normal infants should uncomplainingly starve is an intriguing question. Our 4 infants shared a tendency to accept long intervals between feeds which dated since birth. Presumably they had a defect in appetite control in the opposite direction to infants who demand feed in excess of their needs and become obese. Breast-fed infants tend to get a diminishing supply with a diminishing demand and may enter a vicious circle ending in irretrievable failure of lactation. In Case 4 we think that we were able to break this cycle by 'forced feeding'.

Summary

Four apparently normal breast-fed infants failed to thrive when fed on demand. They all shared a tendency to accept long intervals between feeds from birth. Infant welfare clinics should be alerted to this problem. If infants who attend these clinics are accurately weighed and their progress carefully recorded on weight charts this insidiously developing complaint could be detected earlier and with appropriate advice, a potentially serious condition averted.

We thank Professor O. P. Gray for advice and for permission to describe a patient under his care.

References


T. J. Evans and D. P. Davies
Department of Child Health, University Hospital of Wales, Cardiff.

Correspondence to Dr. D. P. Davies, Department of Child Health, Leicester Royal Infirmary, Leicester LE1 5WW.

Retention of urine in the neonate possibly due to anticonvulsant drugs

Congenital infravesical obstruction of urine is readily suspected when an infant has a distended bladder and palpable kidneys. Temporary retention of urine, however, has not been well documented. Shearer et al. (1972) reported a case of maternal ingestion of nortriptyline during pregnancy causing acute retention which required urethral catherization. Robson and Davies (1974) discuss 3 cases of transient retention of urine in the neonate with no obvious aetiology. None of their infants suffered any neurological damage; none were given drugs in the first few days of life. Reported here are 3 recent cases of neonatal acute retention of urine, associated with severe birth asphyxia, neonatal convulsions, and the administration of various anticonvulsant drugs.

Case reports

Case 1. A boy, weighing 4.04 kg, was born at term by emergency caesarean section due to fetal distress and a prolonged second stage. No drugs had been taken during pregnancy. Apgar score at one minute was 1 and the baby required intubation for 5 minutes. At 13 hours the baby had his first convulsion. Treatment was started with phenobarbitone (22.5 mg/24 h), and dexamethasone (2 mg/24 h) was added the next day for continued seizures. Further prolonged convulsions required 1 mg diazepam repeated four times in the period of 36 hours.

Short reports 975
On the 4th day the baby was found to have a distended bladder and bilaterally palpable kidneys. Suprapubic aspiration of urine was sterile on culture. Twelve hours later the bladder was still palpable and the blood urea 20.8 mmol/l (125 mg/100 ml). A urethral catheter was inserted and the bladder was slowly decompressed, in addition phenobarbitone was withheld for 48 hours. Four days later blood urea had fallen to 7.8 mmol/l (47 mg/100 ml). IV urogram showed that both kidneys functioned and were normal in size; cystourethrogram was normal with complete emptying around the urethral catheter. The catheter was removed after 5 days and the baby passed urine spontaneously. He has had no subsequent urinary problems.

Case 2. A girl of 3.32 kg was born at term by caesarean section due to brow presentation with fetal distress. No drugs had been taken during pregnancy. Apgar score was 1 at one minute and she required intubation for 3 minutes. The first convulsion was noted at 7 hours and regular and frequent seizures occurred for 3 days. Treatment included phenytoin (30 mg/24 h), diazepam (total of 2 mg in four doses), and dexamethasone (2 mg/24 h). On the 4th day the bladder was noted to be at the level of the umbilicus and both kidneys were easily palpable, but not thought to be enlarged. Slow suprapubic decompression of sterile urine was required on two occasions before spontaneous micturition returned. At 2 weeks the kidneys were no longer palpable and there have been no subsequent urinary problems.

Case 3. A boy of 2.5 kg was born at 36 weeks by caesarean section after severe maternal toxaemia. Apgar score was 3 at one minute and he was intubated for 3 minutes. At 4 hours he became apnoeic and required ventilation for 4 hours. The first generalized convulsion occurred at 14 hours. Seizures continued for 4 days with 20 to 30 convulsions per day. Phenobarbitone (16 mg/24 h), diazepam (total of 1 mg), and dexamethasone (2 mg/24 h) were given in an attempt to control the convulsions. At 72 hours the baby was noted to have a large tense bladder and 40 ml sterile urine was slowly removed by suprapubic aspiration. Three further taps were required to decompress the bladder before normal urinary function returned. The peak blood urea at this time was 64 mmol/l (386 mg/100 ml). There have been no subsequent urinary problems.

Discussion

There are several possible explanations for the acute retention of urine in these 3 cases. In a personal communication, W. J. Robson and R. H. Davies report that an indwelling catheter can lead to the disappearance of obstructing posterior valves. In 2 of the cases a urethral catheter was not inserted and the infants passed urine normally after a period of between 1 and 2 days. In the other, a urethrogram excluded the presence of valves.

The common features reported here include caesarean section, severe birth asphyxia, poorly controlled neonatal seizures, and the need for one or more anticonvulsant drugs. The first three factors are relatively common events in the neonatal period, and there have been no reports of any associations with acute urinary retention. Dexamethasone and diazepam were the only drugs used in all 3 cases. It is unlikely that dexamethasone could cause acute retention of urine, leaving diazepam as the drug most likely to be responsible.

The act of micturition is mediated through a spinal reflex arc with synapses in the sacral segment of the spinal cord. Bladder distension above a critical level raises these cells from a subliminal to an active state, and bladder emptying is triggered by the efferent motor fibres stimulating bladder contraction. The effects of certain drugs on this mechanism are well known. Atropine acts on the postganglionic nerve endings and diminishes the frequency of urinary bladder contractions. Imipramine, and other drugs with anticholinergic actions, also lower intravesical pressure and increase the capacity of the bladder.

Diazepam is a member of the benzodiazepine group of drugs and has a central skeletal muscle-relaxing action. It has no autonomic effects and does not relax smooth muscle. As well as causing profound central depression it may conceivably interfere with the spinal reflex of micturition when given with other anticonvulsant drugs. The central depression with skeletal muscle relaxation would allow palpation of normal-sized kidneys.

In view of the return to normal functioning of the bladder in iatrogenic urinary retention of the neonate, management should include a reduction in sedation and caution when using diazepam in conjunction with other anticonvulsants. Manual expression of the bladder may be effective in decompressing the bladder, but suprapubic aspiration was required in all our 3 cases. In one, a urethral catheter was inserted after normal voiding failed to return with repeated suprapubic taps.

Summary

Three cases of neonatal acute retention were associated with convulsions and heavy sedation. Inhibition of micturition may be due to diazepam-induced depression of the nervous system, in association with other anticonvulsant drugs.
I am grateful to Dr. Herbert Barrie for help in preparing this report.

References


M. I. Levene
Department of Paediatrics, Charing Cross Hospital, Fulham Palace Road, London W6.

Neurological complications of β-thalassaemia major

A variety of neurological complications following vascular thrombotic crises have been documented in patients with sickle cell anaemia (Weatherall and Clegg, 1971), but β-thalassaemia major neurological manifestations are uncommon. We report 2 cases of β-thalassaemia major with transient major motor seizures; in one there was in addition transient hemiparesis. The probable pathogenesis of these neurological episodes is considered and the literature on the matter briefly reviewed.

Case reports

Case 1. An 8-year-old Indian boy with homozygous β-thalassaemia major on regular blood transfusions every 6 weeks, was seen on 10 January 1975 with Hb 2·2 g/dl. He was given 70 ml packed cells and Hb rose to 13·9 g/dl.

After the transfusion he developed fever and cough; 5 days later he complained of headache and vomiting. On 18 January he had twitching of his right hand followed by a generalized major fit. He had further fits at 10-minute intervals; they were controlled with intravenous diazepam. On examination he was unresponsive; there was no meningism, localizing neurological signs, or other abnormalities apart from an enlarged liver and spleen. Blood pressure was 120/80 mmHg, temperature 37·2°C, and heart rate 140. Hb was 12·2 g/dl, total white cell count 7100/mm³ (7·1 x 10⁹/l) with polymorphonuclear leucocytes 80%; platelets were adequate, and a blood film for malarial parasite was negative. Cerebrospinal fluid (CSF) contained red blood cells 1160/mm³, WBC 4/mm³ (lymphocytes), sugar 96 mg/100 ml (5·33 mmol/l), and protein 0·45 g/l. Serum iron was 268 μg/100 ml (48 μmol/l), transferrin saturation 87·5%, and TIBC 306 μg/100 ml (54·8 μmol/l).

An electroencephalogram (EEG) on 4 February showed medium-voltage 6–8 cps waves over the right posterior head region: intermittently a 6 cps rhythm was observed over the corresponding area of the left side. Bursts of high-voltage rhythmic 3–4 cps waves intermixed with arrhythmic 2–3 cps delta-waves were seen over the left hemisphere maximally over the posterior quadrant. High-voltage single sharp waves occurred over the left posterior and middle temporal regions.

No further fits were noted after admission. The patient recovered later the same evening and was discharged home after a few days.

Case 2. A 6-year-old Malay girl with homozygous β-thalassaemia major and on regular blood transfusion every 6 weeks, was seen on 23 July 1975 with Hb 3·1 g/dl. She was given 450 ml packed cells after which Hb rose to 11·3 g/dl. On 27 July she had vomiting and diarrhoea; she went to bed and was later unable to walk. On examination there was nystagmus to the right, and left hemiparesis with twitching of the left hand and foot. Temperature was 37·2°C, heart rate 180, BP 110/90 mmHg. Liver was 4 cm, spleen 3 cm; both plantar responses were extensor. Hb was 9·9 g/dl, total white cell count 12 600/mm³ (12·6 x 10⁹/l), polymorphonuclear leucocytes 72%, lymphocytes 21%; platelets 83 x 10⁹/l. CSF was normal. She recovered later the same day and received a further blood transfusion which raised Hb to 15·3 g/dl.

On 3 August 1975 she developed fever, twitching of the right hand followed by a generalized major fit. She was found unresponsive with BP 115/65, and no abnormal findings apart from hepatosplenomegaly. Hb was 12·9 g/dl, total WBC 8700/mm³ (8·7 x 10⁹/l), and platelets adequate. CSF was normal. Serum iron was 200 μg/100 ml (35·8 μmol/l), transferrin saturation 84%, and TIBC 238 μg/100 ml (42·6 μmol/l). Serum antibody to Arbou virus groups A and B and Japanese B were negative. Serum antibody titre to Herpes simplex was <1/8. She was treated with dexamethasone and diazepam for 48 hours and had no further fits.

The first EEG on 30 July showed low- to medium-voltage rhythmic 4–6 cps rhythms over the posterior head regions, more persistently over the left side. β-rhythms were more prominent over the left hemisphere. High-voltage arrhythmic 0·75–3 cps delta-waves were seen intermittently over the right posterior temporal, mid-temporal, and parietal regions. During sleep high-voltage single sharp waves oc-