retention of urine with overflow, faecal incontinence, drowsiness, and neck stiffness. 17 days from the onset loss of vibration sense, diminution in joint position sensation, and a qualitative loss of sensation to pinprick had developed, involving both legs and extending to the area supplied by the 8th cervical spinal segment. Some return of power in the legs was noted on day 19 of illness, and over the next week there was a steady improvement. 6 weeks later she had only slight weakness of the legs, but after 6 months there were no abnormal signs, and her school reported her academic ability apparently unimpaired.

Lumbar puncture after admission produced clear cerebrospinal fluid under normal pressure containing $15/\text{mm}^3$ white cells, mainly mononuclear, protein 0.36 g/l, and glucose 2.5 mmol/l (45 mg/100 ml), which was sterile on culture. A further tap a few days later showed a slightly higher white cell count ($46/\text{mm}^3$, 90% lymphocytes) but a lower protein. A marked neutrophil leucocytosis (total white cell count $41.0 \times 10^9/\text{l}$ ($41000/\text{mm}^3$) with 87% polymorphs) was present on admission and persisted for 4 weeks. A thorough and repeated search for bacterial infection including a bone scan was negative. Acute and convalescent antibody titres done in parallel did not suggest infection with *Brucella*, Epstein-Barr virus, *Rickettsia*, adenovirus, mumps, cytomegalovirus, measles, herpesvirus, *Toxoplasma*, *Leptospira*, *Staphylococcus*, or *Salmonella typhi* and *paratyphi*. Coxsackie antibody titres also were normal except for B_2 which showed a raised titre on admission (1:1024), falling to 1:256 by day 19 of illness. Viral cultures were negative.

We feel this very high Coxsackie B_2 titre suggests that the illness was caused by this organism, and we cannot find any published reports of this combination, though transverse myelitis has been described in association with B_4 virus (Dery *et al.*, 1974). The persistent neutrophil leucocytosis, which was a worrying feature of the illness, does not seem to have been a feature of the rare cases of transverse myelitis with other viral aetiologies.

T. G. MATTHEWS and SUSAN C. BAILEY
*Department of Paediatrics and
Neonatal Medicine,
Hammersmith Hospital,
Du Cane Road, London W12 0HS.*

Reference

- Dery, P., Marks, M. I., and Shaper, R. (1974). Clinical manifestations of Coxsackie virus infections in children. *American Journal of Diseases in Children*, **128**, 464-468.

Maternal diabetes and congenital malformation

Sir,

May I add to the experience of Drs. Day and Insley detailed in their paper on this subject (*Archives*, 1976, **51**, 935), as it is very similar to my own (Komrower, 1961). I followed 213 consecutive pregnancies where all the mothers had received insulin therapy. There were 20

deaths before the 28th week (14 less than 20 weeks), 16 stillbirths and 20 neonatal deaths. I was able to review 182 children on more than one occasion, some for more than 10 years, and by good chance I adopted similar definitions to Day and Insley for major and minor malformations.

There were 19 significant congenital abnormalities: 5 in stillbirths, 3 in neonatal deaths, and 11 in living children. The system involvement was similar to that described by the Birmingham authors—cardiovascular 5, skeletal 5, urogenital 4, oesophageal atresia 1, central nervous system 2 (one child had both anencephaly and multiple skeletal deformities), and 3 had large haemangiomas. The incidence was 9.75% compared with a hospital figure of 3.2% for a 2-year period during the study (the latter results were obtained from our monthly neonatal report). Unfortunately, I do not have the ages of the mothers concerned but the duration of their diabetes varied between 6 months and 10 years. This study took place before our present experience in the treatment of hyaline membrane disease but even so one was able to show that a diabetic woman reaching the 28th week of her pregnancy had a 75% chance of producing a healthy baby without a congenital abnormality.

G. M. KOMROWER
*The Park Hospital for Children,
Old Road, Headington,
Oxford, OX3 7LQ.*

Reference

- Komrower, G. M. (1961). The fate of the diabetic woman's child. *Manchester Medical School Gazette*, **40**, 166-171.

Fibrosing alveolitis

Sir,

Dr. Hewitt and his colleagues, in their paper on fibrosing alveolitis in infancy and childhood (*Archives*, 1977, **52**, 22), state that all but 2 of their 10 children improved when given corticosteroids, and in their summary they further state that in children fibrosing alveolitis is almost always a corticosteroid-responsive disease. This being the case, I should be interested to learn why 5 of their patients died of the disease, including 4 shown in Table 8 to be steroid-responsive.

S. GODFREY
*Department of Paediatrics and
Neonatal Medicine,
Institute of Child Health,
Hammersmith Hospital,
Du Cane Road, London W12 0HS.*

Prof. D. Hull and Dr. C. J. Hewitt comment:

We anticipated that some readers of the article might raise questions that could be more appropriately answered by full detailed case histories. We would be prepared to send these direct to any interested clinician.

We are grateful to Dr. Godfrey, for his letter has given us the opportunity to emphasize a very important point in the treatment of fibrosing alveolitis. A child with fibrosing alveolitis who initially responds to corticosteroids may deteriorate when the dose is reduced or stopped. As will be seen in Table 8 of our paper, 4 patients, following such a deterioration, improved on one occasion when corticosteroid treatment was restarted or increased. 3 of these subsequently deteriorated when the steroid dose was again reduced or stopped, did not then respond to a further 'adequate' steroid therapy and died. 2 other patients (Cases 7 and 9) responded to multiple courses of steroids after episodes of deterioration and have survived. It is not possible to predict which patients will continue to be steroid-responsive and it was this fact which led us to stress that steroid therapy for fibrosing alveolitis in children should comprise at least a year's treatment and that withdrawal should be cautious and protracted.

D. HULL and C. J. HEWITT
University Hospital and Medical School,
Clifton Boulevard, Nottingham NG7 2UH.

Pericardial effusion complicating umbilical venous catheterization

Sir,

Recently we encountered an unusual complication of umbilical venous catheterization. A 3.4 kg female infant of an insulin-dependent diabetic mother received 4 ml of 50% dextrose by push because of hypoglycaemia (blood glucose of 18 mg/100 ml; 1.0 mmol/l), via an umbilical venous catheter. The infant then received 15% dextrose by continuous infusion. X-ray of the chest and abdomen obtained subsequently showed the catheter coiled up in the heart with the tip in the right atrium. The catheter was pulled back into the inferior vena cava. At 52 hours of age the infant suddenly became dusky, had grunting respirations, and later became apnoeic and bradycardic. She was resuscitated and placed on intermittent positive pressure ventilation. Blood glucose was 103 mg/100 ml (5.7 mmol/l). Chest x-ray showed cardiomegaly. The infant continued to do poorly and died after 15 minutes.

At necropsy the pericardial sac was found to be distended with 28 ml clear yellow fluid. There was a small haematoma measuring 0.2 × 0.2 × 0.1 cm in the anterior wall of the left atrium and adjacent to this haematoma there was a perforation measuring 0.1 cm in diameter. Biochemical analysis of the pericardial fluid showed glucose 3020 mg/100 ml (168 mmol/l), total protein 11.7 g/l, calcium 5.0 mg/100 ml (1.25 mmol/l), and sodium 92 mEq/l (92 mmol/l).

The inordinate amount of glucose in the pericardial fluid and the rent in the left atrium indicate that the fluid reached the pericardial space during the manual infusion of 50% dextrose. It is reasonable to assume that the

catheter tip originally was in the left atrium and that it recoiled into the right atrium after the rapid push of the dextrose. Though simple puncture of the atrial wall by the catheter tip could have been the basis of the observed complication, the cutting force of the jet stream produced by rapid infusion seemed an alternative possibility. We have found *in vitro* that when 50% dextrose is injected rapidly through a 5 French umbilical catheter, pressures as high as 550 mmHg may be produced.

DILIP M. PUROHIT and ABNER H. LEVKOFF
Department of Pediatrics,
Medical University of South Carolina,
80 Barre Street, Charleston, S. C. 29401, USA.

Rapid assessment of gestational age at birth

Sir,

We have assessed the gestational age of 408 newborn babies using the method of Parkin *et al.* (*Archives*, 1976, 51, 259) which is based on four external characteristics. The mean of differences between gestational age, according to the last period, and that assessed by the total score was +1.26 days and the standard deviation ± 8.01 days. Thus, the 95% confidence limits of ± 16 days in our series are very similar to those obtained by the above authors. The relation of total score to gestational age is shown in the Fig. The means of the gestational ages are plotted against total score. 95% confidence limits for prediction of gestational age from the score are also shown by the horizontal lines. The curve has been drawn by free hand. We find the method simple, quick, and quite accurate, so that it can be used in every day clinical practice.

ALEX. PEONIDES
Children's Asylum Maternity Hospital,
Thessaloniki, Greece.
B. KATSOUGIANNOPOULOS
Department of Hygiene,
University of Thessaloniki,
Greece.

Fig. Relation of total score to gestational age.