born infants benefited from the monitoring and treatment facilities of an expensive intensive care unit, while those born 15 years earlier were at best kept warm in a heated crib or nursery, given oxygen by face mask or in a tent and fed by tube, pipette, Belcroy feeder or bottle, the small improvement in major handicap is rather disappointing. It is unfortunate that so many workers use my data from the early 1950s to justify the conclusion that (to quote Rawlings et al., 1971 as an example) 'much of the handicap found in surviving children in the past would have been avoidable with modern methods of care', since it seems likely that early starvation, which only operated for a few years, was the main cause of the very high incidence of major handicap in that era. This is not to denigrate recent advances in neonatal care. In 1963 I wrote 'most cases of mental retardation or gross neurological abnormality probably result from developmental defect rather than from the effect of pre- or paranatal damage to a potentially normal central nervous system' and hypothesised that 'such complications (referring to the perinatal period) may have a definite, if less catastrophic, effect on later intellectual functioning and educational ability' (Drillien, 1963). Analysis of the data on early school age status of 1966–70 LBW children is incomplete, but already it seems likely that the benefits of neonatal intensive care will be demonstrated more by a reduction in minor impairment than in major handicap, if those children who suffered from the disastrous iatrogenic effects of early starvation are excluded when making comparisons by date of birth.

References


C. M. DRILLIEN
Armitstead Child Development Centre, 94 Monifieth Road, Broughty Ferry, Dundee.

‘DIDMOAD’ syndrome
Sir,
In the October issue of the Archives (1977, 52, 796), attention was drawn to the fact that the association of diabetes mellitus, diabetes insipidus, optic atrophy, and neurosensory hearing loss is becoming increasingly recognised. We have been following a 17-year-old girl with diabetes mellitus, diabetes insipidus, and optic atrophy. To date our patient has had no evidence of high-tone neurosensory hearing loss, despite repeated audiograms. She does have a neurogenic bladder with hydrenephrosis and hydroureter, a reported concomitant of the syndrome. A family history is not available as she was adopted at 6 weeks of age. She presented in early childhood with optic atrophy; at 6 years she developed diabetes mellitus; at age 15 she was diagnosed as having diabetes insipidus, and shortly thereafter a neurogenic bladder, hydroureter, and hydrenephrosis.

Recently, 97 cases of this syndrome have been reported (Gunn et al., 1976). Of these, 13 had the full combination of optic atrophy, diabetes mellitus, diabetes insipidus, and hearing loss and 18 had optic atrophy, diabetes mellitus, and diabetes insipidus, as did our patient; 31 had optic atrophy, diabetes mellitus, and hearing loss, and 35 had only optic atrophy and diabetes mellitus. We too feel that this condition is more prevalent than suggested by previous literature, and note the increasing frequency of new reports (Carson et al., 1977; Cremers et al., 1977). A high percentage of these cases may have some and not all of the components of this syndrome.

Peritoneal dialysis and peritonitis
Sir,
Day and White (1977) reported that 42% of children dialysed developed peritonitis. All who were dialysed for more than 11 days developed peritonitis, so this is mainly related to the duration of dialysis. We have performed 75 peritoneal dialyses during the last 9 years, with an incidence of peritonitis of 12%. We ascribe this low incidence to a policy whereby we dialyse patients continuously for 40–60 hours until the blood urea is about 80 mg/100 ml (13.3 mmol/l) and other biochemical parameters are normal. The catheter is then removed and the local site dressed. Dialysis is repeated only if indicated by the biochemical parameters and the patient’s clinical course. We have observed that the incidence of peritonitis is unrelated to number of dialyses. After a single dialysis, the incidence of infection was 11%, and with two or more dialyses it was 14%. Leigh (1969) concluded that peritonitis is usually related to the flora of the skin around the catheter site. With daily dialysis such flora may have more time and chance to invade the peritoneum; in our series the incidence of peritonitis was less because dialyses were shorter. Thus we suggest that peritoneal dialysis should be done as and when indicated rather than daily.

Correspondence 605

References

M. FISHER, M. NUSSBAUM, AND L. SUSSMAN
Department of Pediatrics, Long Island Jewish-Hillside Medical Center, New Hyde Park, New York 11040, USA.
Correspondence

References


V. P. CHOUDHRY
Department of Paediatrics,
All-India Institute of Medical Sciences,
New Delhi 110016, India.

Dr R. H. R. White comments:

It is difficult to compare our findings with those of Dr Choudhry in the absence of more detailed information regarding (1) the age range of the patients dialysed, which Dr Day and I found influenced the infection rate, and (2) the causes of renal failure, which would have some bearing on the duration of dialysis. However, we would not disagree with his view that removal of the cannula after a short period of continuous dialysis might reduce the incidence of infection; indeed we suggested that this might be the case but felt that, since reinsertion of the cannula is an unpleasant experience for the child, such perfection might not always be practicable.

In our hands early detection and treatment of infection had a successful outcome except in one child with rapidly progressive glomerulonephritis, whose Candida peritonitis contributed to death at a time when we had no facilities for haemodialysis and transplantation in children.

R. H. R. WHITE
Nephrology Department,
The Children's Hospital,
Birmingham B16 8ET.

Assessment of total body fat in infancy from skinfold thickness measurements

Sir,

We have read the paper by Dauncey et al. (Archives, 1977, 52, 223) and wish to make a few comments. In their paper these authors have related subcutaneous fat layer to skinfold thickness. They refer to the study by Hammond (1955) which examined the relation between uncompressed fat thickness T measured by x-ray and skinfold thickness S measured by calipers. Hammond finds that $T = 0.95S - 0.0074S^2$. For the range values encountered in the newborn, i.e. $S \approx 5 \text{ mm}$, Hammond's results (Tables IV and V) show $T = S - 0.3 \text{ mm}$. If we regard $T$ as representing the true uncompressed subcutaneous fat thickness, i.e. $T$ less the thickness of the dermis, then $t = T - 1$; using Dauncey's value for thickness of the dermal layer (1 mm). Hence $t = S - 1.3 \text{ mm}$.

Dauncey et al. graph $S$ against $T$ using Hammond's data (see curve B, Figure), and by allowing 1 mm for the dermis derive curve C which relates subcutaneous fat to compressed skinfold thickness. Then, by assuming that subcutaneous fat is 2 mm less than the skinfold thickness they show a line $A$, $S = T - 2$, and claim that, since line $A$ lies fairly close to curve C, there is some justification for the empirical relation they have used to derive uncompressed fat thickness. In fact, as shown in our Figure, the line $A_1$ with the equation $S = T - 1.3$ is a better fit to the data which produce curve C since most of Hammond's observations occur with $4.5 < S < 7$; that is within the region of contact of the curve and the straight line. We therefore believe that Dauncey et al. are in error in using $S = T - 2$. As shown in our first paragraph $T$ is, in fact, $S - 1.3$.

Hammond's work shows that the Harpenden caliper, exerting as it does a constant force of 10 g, compresses a fold of skin to almost half its uncompressed value. Just as the calipers compress the fat layer by a factor of almost two, so they also compress the dermis. Knowing this, one would empirically suggest that the true uncompressed subcutaneous fat is 1 mm less (two thicknesses of compressed dermis) than the reading given on the calipers while compressing the fold of skin.

We, therefore, recommend that in using the body fat formula derived by Dauncey et al. one should use $S = T - 1.3$.

Reference


B. RICHARDS and S. W. DE SOUZA
Department of Computation,
University of Manchester Institute of Science and Technology, and
Department of Child Health,
University of Manchester.

Dr S. R. Gairdner and Dr S. Gairdner comment:

We are unable to agree with Richards and De Souza's reasoning for several reasons. Their choice of the figure...