measurement of 24 hour creatinine is advised with calculation of the ideal weight from the zero muscle chart.[1] To obtain a practical objective for dieting allowance must be made for the increased fibrous tissue accumulation that takes place in the dystrophic disease process.

How to use the chart

This chart is used in conjunction with the Tanner and Whitehouse longitudinal standards for height and weight.[3]

(1) Find appropriate height centile.

(2) Predicted 'ideal' weight is read off the chart using the height centile line.

(3) A practical objective for diet control is achieved by adding to the ideal weight an extra kg for every year the boy is over 10 years—for example, at 15 years this would be 5 kg extra.

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See Annotation by Hulse on p. 1179.

Does ABO incompatibility matter?

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SUMMARY The incidence and severity (peak serum bilirubin concentration) of clinically detectable jaundice was determined retrospectively in 110 elution positive cases of ABO incompatibility. Neither the incidence nor the severity of jaundice in the study group differed significantly from a control group. In the individual case Coombs positivity and/or a strongly positive elution test may be a helpful predictor of jaundice occurring but not of its severity.

With the declining incidence of Rhesus disease, ABO incompatibility is said to be the commonest cause of haemolytic jaundice in the newborn. This is not surprising in view of the fact that 20–25% of pregnancies are ABO incompatible. Compared with Rhesus incompatibility, however, the jaundice is usually mild and characteristically later in onset. Furthermore, associated anaemia rarely occurs. These observations prompted us to consider the clinical importance of ABO incompatibility by a retrospective case-control study.

Patients and methods

In this hospital cord blood is routinely taken from the infants of all group O mothers. This is tested for blood group and presence of antibodies by an elution test and direct Coombs test. The elution test is performed by washing the infant's red cells twice in saline followed by heating to 56°C to dissociate the maternal antibody from the antigenic sites. The resultant solution is centrifuged and the supernatant (eluate) tested against adult red blood cells of known group. Agglutination of the cells confirms the presence of specific antibody. The elution test detects maternal anti-A or anti-B haemolysins (IgG antibodies) on the infant's red blood cells.[1]

Routine screening was instituted in 1978 after reports that it reduced the need for exchange transfusions in ABO incompatible infants by allowing early detection.[2] Data were collected on 110 ABO incompatible deliveries (94 mother group O/infant group A (O-A), 16 mother group O/infant group B (O-B)) in which the cord blood elution test was positive. The elution test was graded as weakly positive or strongly positive. A control group

consisted of 110 mother group O/infant group O (O-O) deliveries who were born immediately before or after the index case. All were Rhesus compatible. The incidence and severity (peak serum bilirubin concentration) of clinically detectable jaundice was determined for each group. Statistical comparisons were made using the χ^2 and Wilcoxon's rank sum test as appropriate. The protocol for treatment of the jaundice was identical for the two groups. This, therefore, could not have been a confounding variable.

Results

Information about sex, gestation, birth weight, method of delivery, and method of feeding for each group is shown in table 1. Table 2 shows data about jaundice. The difference in the incidence of jaundice between the two groups was not significant ($p>0{\cdot}1$). The peak serum bilirubin concentration of those who were jaundiced did not differ significantly between the two groups. The incidence and severity of jaundice in the O–A and O–B groups did not differ significantly.

The direct Coombs test was positive in 36 (33%) of the study cases. Fourteen (39%) became clinically jaundiced with a peak serum bilirubin concentration range of 111–236 μmol/l (median 162 μmol/l). A higher proportion of patients who were Coombs positive became jaundiced (14 out of 36, 39%) than those who were Coombs negative (15 out of 74, 20%). This did not achieve significance ($0{\cdot}1>p>0{\cdot}05$). When compared with the control group, however, jaundice occurred significantly more often in Coombs positive patients ($p<0{\cdot}01$). There was no significant difference in severity from the control group.

Similarly, jaundice occurred more often in patients with a strongly positive elution test (13 out of 37, 35%) than in those with a weakly positive elution test (16 out of 73, 22%). This did not achieve significance ($0{\cdot}1>p>0{\cdot}05$) but did do so when those with a strongly positive elution test were compared with the control group ($p<0{\cdot}05$). Again, there was no significant difference in severity from the control group.

Oxytocin was used in 28 (16 cases, 12 controls) of 116 deliveries (58 cases, 58 controls). In the remainder this information was not recorded in the labour ward register. The use of oxytocin did not affect the incidence of jaundice.

None of the infants born at term became sufficiently anaemic to require a top up transfusion.

Table 1 *Details of infants studied*

	ABO incompatible group (n=110)	*Control group (n=110)*
Sex:		
Male	53	59
Female	57	51
Mean (SD) gestation (weeks)	39·7 (0·2)	39·7 (0·2)
Mean (SD) birth weight (g)	3400 (50)	3350 (50)
Method of delivery:		
Vaginal	89	83
Breech	1	2
Caesarean section	12	17
Instrumental	8	8
Method of feeding:		
Breast	29	40
Bottle	81	70

Table 2 *Details of jaundice in the two groups of infants*

	ABO incompatible group (n=110)	*Control group (n=110)*
No with clinically detectable jaundice:		
Whole group	29 (26%)	18 (16%)
O–A group	26	
O–B group	3	
Median (range) peak serum bilirubin concentration of jaundiced babies (μmol/l):	*(n=29)*	*(n=18)*
Whole group	171 (11–282)	190 (159–295)
O–A group	166 (111–282)	
O–B group	198 (149–228)	

Discussion

The effects of ABO haemolytic disease have been described as varying from a subclinical compensated haemolytic process[3] to hydrops fetalis.[4] The reasons for this variable clinical expression are not clear. One reason may be that the A and B antigens are expressed more weakly on fetal cells than on adult cells.[5] The strength of expression increases with gestation.

In this group of patients ABO incompatibility (as defined by a positive elution test) has not been a clinical problem. Perhaps a less sensitive screening test than that presently used is required. Jaundice was more likely to occur in those with a positive Coombs test or a strongly positive elution test but it was no more severe than in the control group.

On the basis of these findings we believe that routine screening for ABO incompatibility using the elution test is of no clinical benefit. In an individual case, however, a positive Coombs test or a strongly positive elution test may be useful in predicting the occurrence of jaundice.

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Ataxic cerebral palsy and genetic predisposition

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SUMMARY It was calculated that in the 962 family members of 36 patients with ataxic cerebral palsy there were 75 (8%) with a history of neurodevelopmental disorder and 31 (3%) with a major congenital malformation. This was not significantly greater than expected, and does not support the hypothesis of a genetic non-Mendelian role in the aetiology of ataxic cerebral palsy.

It has been suggested that a genetic non-Mendelian influence may play a part in a multifactorial aetiology in cerebral palsy. This gives rise to a constitutional vulnerability to several risk factors and a predisposition to neural tissue damage, and is expressed as an increase in neurodevelopmental disability and major congenital malformation in the near relatives.[1 2] In ataxic cerebral palsy, the least common of the syndromes of cerebral palsy, the infants are usually of normal birth weight with a prenatal origin of the condition.[3] The family histories of a population with this disorder were therefore examined for the frequency of neurodevelopmental disability and major congenital malformation in order to assess the role of a genetic influence in aetiology.

Subjects and methods

Cerebral palsy is defined as a group of disorders of movement and posture due to a non-progressive defect or lesion of the developing brain.[4] When this disorder is an ataxia, which is not primarily due to weakness, spasticity, dystonia, or choreoathetosis, it is termed ataxic cerebral palsy.

Thirty six cases of ataxic cerebral palsy were ascertained from the West Australian cerebral palsy register and from the records of the Spastics Welfare Association of Western Australia. Each case was individually examined to ensure they fulfilled the criteria for diagnosis. All were older than 5 years of age and had histories consistent with a non-progressive disorder. The number of cases of ataxic cerebral palsy ascertained represented 72% of the metropolitan population (details to be published elsewhere).

A history of neurodevelopmental disability or major congenital malformation in the parents, aunts and uncles, first cousins, siblings, and children of siblings, was recorded. The categories of neurodevelopmental disability used are listed in the table. Details of these were checked against the medical records of several institutions and by correspondence with the relevant personnel who were concerned.

Results

Out of a total of 962 people on whom information was sought, 75 (8%) had neurodevelopmental dis-

Table *Categories of neurodevelopmental disability*

Intellectual handicap
Cerebral palsy
Language delay or disorder ascertained by specialist consultation
Epilepsy of unknown origin
Congenital hearing loss
Severe visual handicap
Congenital strabismus
Special educational requirements–remedial teaching, special class or school (where the reason was not considered to be primarily socioemotional)