removed. The operator did not find that the transducer encroached on the fontanelle nor did it impede his ability to perform the ventricular tap.

Discussion

Real-time ultrasound studies have shown that ventricular dilatation occurs in up to 40% of cases of intracranial haemorrhage during the neonatal period.² Papile *et al.*³ suggest that ventricular decompression by lumbar puncture may reduce the incidence of this condition. Decompression by the lumbar route, although obviously preferable, is not always possible due to basal arachnoiditis and cisternal obstruction. In this condition, non-communication of CSF between the cerebral ventricles and the lumbar subarachnoid space precludes the use of lumbar puncture and direct ventricular taps may be necessary to remove CSF.

Lateral ventricular taps are a potentially dangerous procedure which should be undertaken only by experienced staff. Risks include infection, cyst formation, and damage to important areas within the brain. Intraventricular haemorrhage as a result of a ventricular tap has been reported.⁴ In order to minimise the risks of unnecessary iatrogenic problems by the needle passing through the ventricle, a method of performing ventricular taps under direct vision is described. No stereotactic method for

needle insertion into the lateral ventricles has been previously reported. This method is simple and requires little ultrasound experience. The operator is given instructions on where the tip of the needle is in relation to the ventricle and insertion of the needle through the ventricle into the caudate nucleus can be avoided. Although the infant described here was fairly immature, it is likely that this procedure can be performed in most infants up to age 6 months, by which time progressive ossification of the skull prevents adequate visualisation of the ventricular system by ultrasonic methods.

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Congenital dislocation of the hip and short maternal stature

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SUMMARY The heights of 33 consecutively selected mothers of children in hospital for treatment of congenital dislocation of the hip (CDH) were compared with the heights of 54 mothers of children in hospital for conditions other than CDH. The mothers of the CDH children were found to be shorter than those of the control group by a mean of 4.4 cm. This difference was statistically significant. Short maternal stature is associated with a narrow pelvis and presumably also with reduced uterine size which may cause restriction of space for the fetus.

The aetiology of congenital dislocation of the hip (CDH) has been the subject of numerous investigations and is still considered multifactorial. Several

mechanical factors have been noted to correlate with CDH—such as breech presentation,¹ increased birthweight and length,² and a greater number of labours with fetopelvic disproportion.^{1,2} A restriction of space for the fetus *in utero* has also been suggested.³

Since maternal factors play an important role in affecting the available space for the fetus *in utero* and in view of the clinical observation that several mothers of children with CDH were short, we decided to investigate the association between maternal height and CDH.

Patients and methods

The subjects of this study were 33 infants and children with clinical and radiological evidence of

CDH who had been consecutively admitted to our hospital during the period 1 January to 3 June 1981 with their mothers. Each patient had been examined and diagnosed as having CDH by a senior orthopaedic surgeon and was in hospital for traction or surgical correction of the condition. There were 7 (21%) boys and 26 (79%) girls. Thirty-one had been vertex presentations and 2 breech presentations at birth. The ages of the mothers ranged between 19 and 35 (mean of 26.3) years.

The control group comprised 54 infants and children who had been in hospital consecutively in the paediatric department between 1 April and 10 May 1981 with their mothers. Any patient who had CDH himself or in a sibling was excluded from the control group. The referral pattern of children with CDH was similar to that of other children.

The ethnic composition of the mothers in both groups is given in the Table. The height of each mother in each group was measured, and the difference between the mean heights of the two groups was assessed by the Student's *t* test.

Results

Maternal height. The height of the mothers of the

Table Ethnic composition of mothers of patients with CDH and mothers of control group

	CDH group		Control group	
	No	%	No	%
Ashkenazi jewish	6	18.2	13	24.1
Sephardi jewish	15	45.4	22	40.7
Arabic	12	36.4	19	35.2

Figure Height of mothers of the CDH and control groups with mean height for both groups.

patients with CDH ranged between 143 and 168, mean ($\pm$ SEM) 154.8 ± 1.2 cm. The height of the mothers in the control group ranged between 143 and 176, mean ($\pm$ SEM) 159.2 ± 1.1 cm. The difference between the means was 4.4 cm and was statistically significant ($P < 0.01$). The individual measurement of maternal height in both groups is given in the Figure.

Discussion

Mothers of children with CDH were significantly shorter than mothers in a control group. Since the referral pattern and the ethnic composition were similar in both groups, the two groups were comparable. Several features have been previously demonstrated in the aetiology of CDH. A high proportion of first born was present in several studies^{2,4} and was related to the reduced stretching of the uterus in first pregnancies which increases the pressure on the fetus.⁴ An increased proportion of mechanical problems in labour, other than breech presentation, has also been noted.¹ Fourteen per cent of mothers of infants with unstable hips and vertex presentation underwent caesarean section, compared with 8% of mothers of infants with stable hips and vertex presentation. The CDH group showed a higher prevalence of fetopelvic disproportion at birth compared with the overall population of children born at the same hospital.¹ A narrow pelvis was shown to correlate with short stature⁵ and this possibly increases the chance of undergoing caesarean section.

In a thorough search we could find no data pertaining to the relationship of maternal height with CDH. In this context it is interesting to note that the incidence of CDH is high in Japan compared with elsewhere⁶ and the average stature of Japanese women is shorter than that of American and English women.⁷ Short maternal stature is probably correlated with decrease in fundal height and this might increase the pressure to which the fetus is exposed.

Other mechanical factors which act in the same way are increased birthweight and length of infants with CDH and increased prevalence of oligohydramnios in pregnancies of children with CDH.⁸ This results again in limitation of space for the fetus *in utero*.

We found that mothers of children with CDH have significantly shorter stature than mothers in a control group. We suggest that short maternal stature is another possible aetiological factor in CDH and that offspring of mothers with very short stature should be considered at risk for the development of CDH.

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Congenital isolated thyrotrophin releasing hormone deficiency

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SUMMARY A 4½-year-old girl with congenital thyrotrophin-releasing hormone (TRH) deficiency is described. Oral TRH administration led to normal thyroid hormone and TRH levels in the blood; favourable growth and development was achieved.

Deficiency of thyroid-stimulating hormone (TSH) and hypothyroidism can occur in any condition that is associated with developmental defects of the pituitary or hypothalamus, or in children with idiopathic hypopituitarism. However isolated deficiency of TSH, or thyrotrophin-releasing hormone (TRH) is rare.^{1,2} We describe in detail a case of isolated TRH deficiency.

Case report

A 4½ year-old girl was referred to us because she was short.

She was the third child of healthy, intelligent parents. Pregnancy and delivery had been normal, as had been the neonatal period. Birthweight was 3200 g and length 50 cm. Growth to age 1 year had apparently been normal but retardation of growth was noticed at 2–3 years of age.

Her parents were not related, but her maternal grandparents were consanguineous. There was no family history of thyroid disease, dwarfism, or mental abnormality.

Physical examinations showed height 93.0 cm (–2.0 SD of the mean height for Japanese girls as reported by the Japanese Ministry of Health and Welfare), weight 13.0 kg (–1.6 SD), with normal body proportions. The thyroid gland was not enlarged, and the remainder of the physical examina-

tion was normal. Roentgenograms of the skull were normal and bone age was 2¾ years.

Haematological studies, urine analysis, and blood chemical determinations were normal. An electroencephalogram was normal. Computerised tomography scans of the skull were normal. Her developmental quotient was estimated to be 112.

At the initial visit, the serum thyroxine (T4) value was 4.1 µg/100 ml (52.8 nmol/l) (normal 4.5–13.5 µg/100 ml), serum tri-iodothyronine (T3) 177 ng/100 ml (2.7 nmol/l) (normal 98–202 ng/100 ml), and serum TSH 14 µU/ml (normal <2–8). Thyroid uptake of ¹²³I sodium iodide was 7.1% at 24 hours (normal 10–40%). A thyroid scan gave normal results. Tests for antithyroglobulin antibody and antimicrosome antibody were negative. The serum thyroxine binding globulin determined by radioimmunoassay was 20.3 µg/ml (338.3 nmol/l) (normal 15.7–43.3 µg/ml).

A peak TSH level of 70 µU/ml was obtained after administration of 130 µg (10 µg/kg) of TRH intravenously (normal 10–40 µU/ml). Two additional TRH tests showed similar positive responses. Luteinising hormone and follicle-stimulating hormone showed normal responses to intravenous injection of 40 µg of luteinising hormone-releasing hormone. Serum growth hormone was demonstrable in the fasting state (3.2 ng/ml), and an increase was obtained during insulin-induced hypoglycaemia (20.5 ng/ml). The plasma level of adrenocorticotrophin was 16 pg/ml (normal 15–85) in the early morning and a peak level of 27 pg/ml was obtained by insulin-induced hypoglycaemia. Urinary 17-OHCS were 3.9 mg and urinary 17-ketosteroids 0.1 mg/24 hours. Serum prolactin level was 16.4