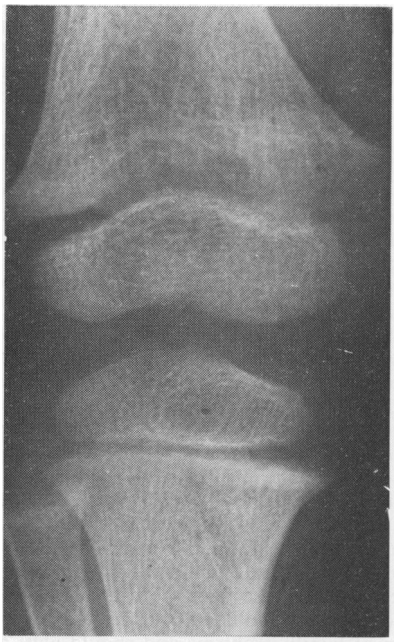

(b)

Dr Seino comments:

As previously stated, the mean plasma levels of $1,25(OH)_2D$ are significantly low in hypophosphataemic vitamin D-resistant rickets and increase after treatment, but remain relatively low in spite of large doses of $1\alpha OHD_3$ (0·4–2·0 μg/kg per day).[1–2] These results suggest that the metabolism of $1,25(OH)_2D_3$ is accelerated in such patients. The relatively low level of $1,25(OH)_2D$, even after administration of a massive dose of $1\alpha OHD_3$, indicates that large doses are necessary in these patients. Moreover, the low level of $1,25(OH)_2D$ before treatment showed a positive correlation with the low level of serum phosphate and TmP/GFR.[2] This finding suggests that massive doses of $1\alpha OHD_3$ are necessary in severe cases. I suppose that the case referred to by Kristiansen and Pedersen was mild, as commonly occurs in females. The risk of hypercalcaemia is always present during treatment with vitamin D metabolites. However, hypercalcaemia or hypercalciuria (urinary calcium/creatinine appears to be the best indicator) is easily controlled by reducing the dose of $1\alpha OHD_3$ because it has a short half-life.

References

[1] Seino Y, Shimotsuji T, Ishii T, *et al.* Treatment of hypophosphataemic vitamin D-resistant rickets with massive doses of 1α-hydroxy-vitamin D_3 during childhood. *Arch Dis Child* 1980; **55:** 49–53.

[2] Seino Y, Shimotsuji T, Ishida M, Ishii T, Yamaoka K, Yabuuchi H. Vitamin D metabolism in hypophosphatemic vitamin D-resistant rickets. *Contrib Nephrol* 1980; **22:** 101–6.

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Somatomedin-C in the Beckwith-Wiedemann syndrome

Sir,

We refer to the paper by Spencer *et al.*[1] We have obtained fasting sera for growth hormone and somatomedin-C on 3 unrelated patients with classical features of the Beckwith-Wiedemann syndrome. The results are shown in the Table.

Case	*Age*	*Sex*	*Growth hormone*	*Somatomedin-C* (U/ml)*	
				Patient	*Normal*
1	3 weeks	F	—	0·1	0·2–2·0
2	1 year	F	2	0·19	0·2–2·0
3	8 years	F	12	0·26	0·4–2·0

*By radioimmunoassay.

The growth hormone determinations were performed by Bio-Science Laboratories, Van Nuys, California, and the Somatomedin-C determinations by Nichols Institute, San Pedro, California, which also supplied the normal range. The very low values of Somatomedin-C in our patients do not support the concept proposed by Spencer *et al.*[1] No explanation for the conflicting findings is offered.

Reference

[1] Spencer G S G, Schabel F, Frisch H. Raised somatomedin associated with normal growth hormone. A cause of Beckwith-Wiedemann syndrome? *Arch Dis Child* 1980; **55:** 151–3.

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Dr Spencer comments:

The results presented by Robinow and Shafer are interesting but it is difficult to explain why they conflict with ours. However, since submitting our article,[1] I have been able to test somatomedin activity in another patient with Beckwith-Wiedemann syndrome. The patient was 7 years old and had a bioassayable plasma somatomedin activity of 1·6 U/ml. This value, although not as high as that in the patient we reported earlier, is still raised compared with normal age-matched children; our range

of values for normal children of this age is 0·7–1·1 which compares with the values given by Phillips and Vassilopoulou-Sellin.[2] Our observations of somatomedin activity in this condition, although conflicting with those of Robinow and Shafer, are supported by those of Ashton and Aynsley-Green.[3]

The difference between our findings and those of Professor Robinow could be due to the difference in the assays used. Both my results and those of Ashton were obtained with bioassays to determine somatomedin activity, while Robinow and Schafer used a radioimmunoassay. Direct comparison of somatomedin activity between the two types of assay used is difficult to make, particularly as the normal range quoted by Robinow and Schafer (0·4–2·0 U/ml) would cover the complete range of somatomedin activity (from hypopituitarism to acromegaly) in most bioassay systems.

No results are presented by Robinow and Shafer for insulin levels; however, the hypoglycaemia in their patients must presumably have been due to hyperinsulinaemia. These results are therefore interesting as, to my knowledge, no other such association between high insulin and low somatomedin has yet been demonstrated. Similarly, it is also difficult to reconcile the gigantism of the Beckwith-Wiedemann syndrome with low somatomedin levels, and I know of no other reported cases of such excessive growth in the absence of somatomedins.

If the difference in somatomedin levels is due to the different assays used, then either there is a factor which interferes in the radioimmunoassay, or the somatomedin-C radioimmunoassay used fails to cross-react with any of the other recognised somatomedins, or there is, in patients with Beckwith-Wiedemann syndrome, a previously unrecognised serum growth factor which is capable of stimulating growth *in vivo* and cartilage growth *in vitro*. This may be a modified (non-immunoreactive but biologically active) somatomedin molecule.

The conflict in these results should be investigated.

References

1 Spencer G S G, Schabel F, Frisch H. Raised somatomedin associated with normal growth hormone. A cause of Beckwith-Wiedemann syndrome? *Arch Dis Child* 1980; **55**: 151–3.

2 Phillips L S, Vassilopoulou-Sellin R. Somatomedins. *N Engl J Med* 1980; **302**: 438–46.

3 Ashton I K, Aynsley-Green A. Plasma somatomedin activity in an infant with Beckwith-Wiedemann syndrome. *Early Hum Dev* 1978; **1**: 357–62.

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Accidental poisoning preceding nonaccidental injury

Sir,

Factors that might indicate children to be at risk from nonaccidental injury have been studied extensively.[1] Birth factors such as young maternal age and admission to a special care unit have been shown to be important, but factors after birth have been difficult to assess. We have studied the medical records of 80 children on the South Glamorgan Area Review Committee Child Abuse Register, particularly in respect of admissions to hospital for poisoning before the episode of abuse. All of the children had been either physically injured or had suffered clearcut neglect before age 5. The records of 80 control children, whose names were obtained from the Cardiff Birth Survey and matched for age and sex, were also studied.

Ten of the 80 abused children had poisoning episodes before their placement on the register for child abuse. Three of them had taken salicylates, three benzodiazapines, two domestic bleach, one sherry, and the other unknown tablets. Two of the control children had been admitted for poisoning episodes, one after ingesting perfume and the other after paediatric metoclopramide (Maxolon). The difference between the cases (10) and controls (2) is significant, $P<0.05$ χ^2 4·41.

Although nonaccidental poisoning is recognised as a type of child abuse,[2] we believe that the episodes we describe were accidental, indicating family stress which has been shown to be a factor in child poisoning[3] and other accidents.[4] These figures are a further indication that child poisoning should be considered as an important symptom of family problems and should alert all those concerned in the primary care of children.

References

1 Lynch M A, Roberts J. Predicting child abuse. Signs of bonding failure in a maternity hospital. *Br Med J* 1977; **i**: 624–6.

2 Rogers D, Tripp J, Bentovim A, Robinson A, Berry D, Goulding R. Non-accidental poisoning: an extended syndrome of child abuse. *Br Med J* 1976; **i**: 793–6.

3 Sibert J R. Stress in families of children who have ingested poisons. *Br Med J* 1975; **iii**: 87–9.

4 Brown G W, Davidson S. Social class, psychiatric disorder of mother, and accidents to children. *Lancet* 1978; **i**: 378–80.

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Should handicapped children attend ordinary schools?

Sir,

I read with interest the annotation by Rosenbloom,[1] but time has changed the significance of much of what was