HYPOTHYROIDISM IN CONGENITAL RUBEbLA

Sir,—Several manifestations of intrauterine rubella may develop months or years after birth. This late-onset disease may be due to direct action of the persisting infection or to an immunopathological process.

A small-for-dates girl was born by spontaneous breech at 40 weeks menstrual age in May, 1972. Birthweight 2.0 kg and occiputofrontal circumference 30 cm. Rubella-antibody titre was 1/256, but there were no signs of congenital rubella. At age 2 years she was still small but bone age was normal. Over the next year her growth velocity slowed further and she was admitted for investigation. Her height was 80-4 cm and weight 8-2 kg, both below the third centile. Her skin was dry and scaly; there was no goitre. Fundoscopy showed evidence of rubella retinopathy.

Thyroxyne 37 mmol/l (normal 59–138); serum thyroid-stimulating hormone >64 µU/ml (normal 1-0-3-5); thyroglobulin (tanned-red-cell) titre 1/2560; thyroid microsomal haemagglutination-titre 160, rubella antibody 1/320 (aged 3 years 11 months); bone age 2 years 2 months (chronological age 3 years 4 months). She became biochemically euthyroid (T.s.h.<1-0 µU/ml) on l-thyroxine 0-05 mg three times daily. Her height has increased by 9-4 cm in 10 months.

The very rare association of poor intrauterine growth and juvenile hypothyroidism in this patient has been emphasised at a recent clinical meeting of the Royal Society of Medicine, Paediatric Section. Two recent reports from overseas of the association between hypothyroidism and congenital rubella have prompted this letter. The raised thyroid-antibody titres showed evidence of a thyroiditis caused by continuing rubella infection acquired in utero. Cooper reported a case of hypothyroidism in congenital rubella and one other case in the United Kingdom is known.5

If growth and development are slower than expected in children with congenital rubella, thyroid function should be assessed, but in such a clinical state other endocrine disorders may be responsible, such as growth-hormone deficiency.6

I thank Dr W. C. Marshall for his encouragement and advice in writing this report, and Dr David Harvey for his permission.

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TESSY K. HANID

FATAL INHALATION OF METHYLFLUOROSULPHATE

Sir,—Methylfluorosulphate (CH₃OSO₂F) is used in many laboratories as a methylating agent. A fatal accident in the department of chemical technology at this university highlights the hazard of inhaling only a minute quantity of this very toxic substance.

On March 25, 1976, at 11.30 a.m. a 25-year-old chemist split a few millilitres of a fluid containing methylfluorosulphate on his laboratory coat and pullover. He immediately took off the coat and cleaned his pullover with water. Although he had no complaints, the man was sent to hospital at about 1 P.M. No abnormality was found, and he was allowed to go home. At 5 P.M. the man began to cough a little and had some difficulty with deep breathing. At 7 P.M. he was admitted to hospital in a moribund state due to pulmonary oedema. He died the next day despite intensive treatment.

5. Grant, D. G. Personal communication.

Protein denaturation seems to be the major contributor to the development of pulmonary oedema. Moreover, hydrogen fluoride and sulphuric acid are generated when methylfluorosulphate makes contact with water.

This case should be a serious warning to everyone working with methylfluorosulphate. Immediate administration of corticosteroids should be considered if this or other alkylating agents is thought to have been inhaled. Continuous close observation during the first 24 h is necessary.

B. HALLIWELL

ASCORBIC ACID AND PARAQUAT TOXICITY

Sir,—Paraquat is extremely toxic, and even the most vigorous treatments may not be able to prevent death after exposure to the chemical.1 Paraquat toxicity seems to be due to the ability of the reduced form of this compound to generate the superoxide radical,2,3 which can undergo several reactions leading to lipid peroxidation and tissue damage.4 The enzyme superoxide dismutase catalyses the breakdown of the superoxide radical,5 and attempts have been made to use this enzyme to treat paraquat-poisoned animals. Promising results in preliminary experiments6 have not been confirmed, and the lack of effect of superoxide dismutase seems to be due to its inability to penetrate cells.7 What is therefore required is a compound, transported into lung tissue, which can react with superoxide. Such a compound is ascorbic acid.8 It enters the lung from the blood and appears to accumulate in the fluid lining the air spaces,4 and its reaction with superoxide has been directly demonstrated.9 The toxicity of hyperbaric oxygen seems to be due to increased formation of superoxide,10 and in rats exposed to hyperbaric oxygen prior administration of ascorbic acid lessened the toxic effects.11

Since massive doses of ascorbic acid do not seem significantly harmful to man,12 it might be possible to use this treatment in paraquat poisoning.

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B. HALLIWELL

BLAST-CELL TRANSFORMATION CHRONIC GRANULOCYTIC LEUKÆMIA

Sir,—Howes and Emerson13 described a patient having blastic crisis in chronic granulocytic leukaemia with features suggesting lymphoblastic rather than myeloblastic transformation.

We have seen a 38-year-old man who was admitted after a fall which caused a massive haematoma of his leg without bone injury. Hb 6-8 g/dl, white-cell count 200 x 10⁹/l with granulocytes at all stages of maturation, and platelets 800 x 10⁹/l. The neutrophil alkaline-phosphatase score was 7 and Philadelphia chromosome was demonstrated in the peripheral blood. The spleen was enlarged.