factors, possibly immunological, must be involved in determining the clinical response to infection.

MARY E. C. HORN, PAUL TAYLOR, and
SUSAN J. YEALLAND

South London Hospital for Women and Children,
Clapham Common, London SW4
and Department of Virology,
Brompton Hospital,
London SW3.

REFERENCES

- Gardner, P. S., McQuillin, J., McGuckin, R., and Ditchburn, R. K. (1971). Observations on clinical and immunofluorescent diagnosis of parainfluenza virus infections. *British Medical Journal*, **2**, 7.
- Horn, M. E. C., Brain, E., Gregg, I., Yealland, S. J., and Inglis, J. M. (1975). Respiratory viral infection in childhood. A survey in general practice. Roehampton, 1967-1972. *Journal of Hygiene*, **74**, 157.
- Jacobs, J. W., Peacock, D. B., Corner, B. D., Caul, E. O., and Clarke, S. K. R. (1971). Respiratory syncytial and other viruses associated with respiratory disease in infants. *Lancet*, **1**, 871.
- Neligan, G. A., Steiner, H., Gardner, P. S., and McQuillin, J. (1970). Respiratory syncytial virus infection in the newborn. *British Medical Journal*, **3**, 146.

Exercise as a physiological stimulus to growth hormone release

Sir,

In comparing methods of stimulating growth hormone release, Wise *et al.* (1975) included exercise, but reported that levels exceeding 14 μ U/ml were only obtained in 50% of cases who were not growth-hormone deficient. The technique they described involved blood sampling 10 minutes after completion of exercise to mild exhaustion by stair climbing. Extensive studies on several subjects (Buckler, 1972) have shown that plasma growth hormone levels associated with exercise do not begin to rise for 10-15 minutes and do not reach peak values until 25-30 minutes after the start of exercise. Unless the children in the report by Wise *et al.* climbed stairs for at least 15 minutes, it is unlikely that the greatest response in plasma growth hormone levels would be detected by their method. It is possible that others who report poor responses with this method of exercise testing may have failed to take the blood sample at the correct time, namely 25-30 minutes after the start of a short (5-10 minute) period of intensive exercise.

J. M. H. BUCKLER
University of Leeds,
Department of Paediatrics and
Child Health,
27 Blundell Street,
Leeds LS1 3ET.

REFERENCES

- Buckler, J. M. H. (1972). Exercise as a screening test for growth hormone release. *Acta Endocrinologica*, **69**, 219.
- Wise, P. H., Burnet, R. B., Geary, T. D., and Berriman, H. (1975). Selective impairment of growth hormone response to physiological stimuli. *Archives of Disease in Childhood*, **50**, 210.

Dr. Wise and colleagues comment as follows:

We agree entirely with the comments of Dr. Buckler. Since submission of our original paper, and in an attempt to provide a more consistently effective stimulus, we have modified our exercise test to a 10- to 20-minute period (depending on endurance) workload of 450 kilopond (4410 N) m/min using a bicycle ergometer (Monark). Samples for growth hormone assay were taken at 1 and 15 minutes after completion of exercise, so that a minimum interval of 25 minutes from the start of exercise to final sampling applied to all cases. Over 90% of 24 subjects with short stature, and who were not growth-hormone deficient exceeded 14 μ U/ml in one or both of these samples.

P. H. WISE, R. B. BURNET, R. D. GEARY, and
H. BERRIMAN

Endocrine Unit, Royal Adelaide Hospital,
North Terrace, Adelaide,
South Australia 5000.

Maternal histidinaemia

Sir,

Recently two questions have been raised concerning the inherited biochemical trait histidinaemia. (1) Is the trait a benign metabolic disorder (Popkin *et al.*, 1974; Levy, Shih, and Madigan, 1974)? (2) Are children born of histidinaemic mothers at risk of harm during gestation (Neville *et al.*, 1971; Lyon, Gardner, and Veale, 1974)? The answers to these questions are important, since the unnecessary administration of a histidine-deficient diet to pregnant women or to young infants with biochemical histidinaemia might result in a harmful nutritional imbalance. We report a normal histidinaemic mother with 4 normal children.

Plasma and urine amino acids were measured with an amino acid analyser. Imidazolepyruvic acid was determined spectrophotometrically by the borate-arsenate method (Lin *et al.*, 1958). Imidazolelactic acid, imidazoleacetic acid, and N-acetylhistidine were estimated by visual comparison of colour produced by diazotized sulphanilic acid with materials in aliquots of urine and with known amounts of authentic compounds after two-dimensional paper chromatography on Whatman No. 3MM filter paper in pyridine-acetone-3 N ammonium hydroxide (50:30:25), followed by isopropyl alcohol-formic acid (88%) -water (8:1:1).

The mother was on our staff; her plasma amino acids were measured during collection of data on plasma amino acid levels of normal children and adults. Biochemically she had typical histidinaemia. Her fasting plasma histidine levels were 0.73, 0.52, and 0.79 μ mol/ml in 3 samples collected over a 6-month period.