in practice it was readily detected by careful analysis of the nitrous oxide disappearance curve. Our technique has advantages. It is technically fairly simple, the apparatus required is likely to be widely available in departments of anaesthesia and respiratory medicine, and in the concentrations used nitrous oxide is less toxic than 'Freon'.

Serial lactic acid measurements in respiratory distress syndrome. J. P. Beca (introduced by J. W. Scopes) (Hammersmith Hospital, London). Blood lactic acid was measured 4-hourly in 21 newborn infants with respiratory distress syndrome (RDS). 13 survived and 8 died. In general lactic acid levels were higher in babies who died than in survivors, but there were inconsistencies which were uninterpretable if a single estimation was made in a given baby. Analysis of serial determinations done in each baby showed that all patients whose highest lactic acid was always below 35 mg/100 ml survived, and babies with high values but decreasing curves also had a good outcome. Only those who had increasing lactic acid curves, even if initially normal, died.

In most cases a high or normal \( P_{3}O_{2} \) was associated with normal or decreasing lactic acid; but babies with \( P_{3}O_{2} \) below 60 mmHg had often also normal or decreasing lactic acid.

If one intends to use lactic acid levels for prognostication of outcome in RDS, serial determinations are needed and single measurements are of limited value. Furthermore these data support the concept that there may be a wide range of hypoxaemia without oxygen deficit in body tissues and that it is therefore impossible to define a 'lower acceptable \( P_{3}O_{2} \)' enough to achieve adequate tissue oxygenation.

Adrenal response to tetracosactrin in newborn infants. Hugh Price, Theresa Cowley, and E. H. D. Cameron (Welsh National School of Medicine and Tenens Institute for Cancer Research). Current belief holds that the fetal pituitary-adrenal axis is intimately involved in the initiation of labour, and indeed recent studies have shown that antenatal corticosteroid therapy may diminish the incidence of respiratory distress syndrome in prematurely born infants. Our studies were undertaken to provide basic data on the human newborn adrenal response to tetracosactrin (\( \beta^{1-24} \) corticotrophin, Ciba) stimulation.

Four types of study were performed.
(a) Standard 30-minute tetracosactrin tests on 50 infants admitted to a neonatal special care baby unit.
(b) Tetracosactrin tests in 12 normal infants; 6 on day 1 and 6 on day 5 after birth, cortisol and glucose levels being measured at 0, 30, 60, 120, and 180 minutes after intramuscular injections.
(c) Deposynacthen (tetracosactrin zinc complex, Ciba) responses were measured serially to give dose response curves.
(d) Cortisol plasma clearance rates were calculated on 6 newborn infants after intramuscular injection of cortisol. All infants in groups (c) and (d) had been admitted to the special care baby unit.

Plasma cortisol was measured by 'micro'—Mattingly (50 \( \mu \)l—venous plasma/assay) or by competitive protein-binding methods (10 \( \mu \)l peripheral plasma/assay). Correlation curves for these two methods were presented.

The results showed that the newborn infant adrenal responds well to stimulation by tetracosactrin in terms of plasma cortisol concentration, but that the time of maximum response is somewhat delayed compared with that of the adult. No significant difference was observed in the response to tetracosactrin at day 1 and day 5. With Deposynacthen, the peak response time was variable but again was later than that reported for adults which may reflect either a slower absorption of the depot in the infant or a slower adrenal response to stimulation. With all infants receiving Deposynacthen, the maximum cortisol concentration exceeded 100 \( \mu \)g/100 ml plasma. In general, basal levels were not reached until 3 to 5 days after administration of the depot.

Effect of glucose on plasma glucagon, insulin, and growth hormone levels in exchange transfusions. R. D. G. Milner, M. Fekete, R. Assan, and J. S. Hodge (Departments of Child Health and Chemical Pathology, University of Manchester, and Hotel Dieu, Paris)

Insulin excretion in cystic fibrosis. M. C. Goodchild and G. A. Brown (introduced by P. H. W. Rayner) (Institute of Child Health, Francis Road, Birmingham B16 8ET). Reduced tolerance to oral glucose with insulinopenia has been shown in 42% of cystic fibrosis (CF) subjects investigated by Handwerger et al. (1969). Abnormal results have also been reported after intravenous glucose loads. Histological investigation of pancreatic islet tissue has shown it to be substantially normal and reduced tolerance may therefore be a primary feature of cystic fibrosis and not secondary to the fibrotic process.

Significant positive correlation has been demonstrated between plasma insulin levels and urinary insulin excretion. To assess overall insulin secretion in cystic fibrosis therefore, 24-hour urinary insulin excretions were measured on 28 CF children using a double antibody radioimmunoassay. Excretion was also measured in the parents of the CF children. Results were assessed by comparison with results of similar investigations in non-CF families. Mean insulin excretion in the CF children was 257 \( \mu \)U/kg body weight per 24 hours, and was lower than the control children's mean of 323 \( \mu \)U/kg body weight per 24 hours. Mean insulin excretion in the parents of the CF children was 158 \( \mu \)U/kg body weight per 24 hours, and in the control adults 215 \( \mu \)U/kg body weight per 24 hours. The difference between the means of the CF parents and the control adults was significant (P < 0.01).

The tendency to lower insulin excretion in CF children was not unexpected in view of the results of plasma
Serial lactic acid measurements in respiratory distress syndrome.

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