

**17** Somatomedin-C/Insulin-like Growth Factor I Released by the Human Foetal Pancreas. M.A. CSER, D.J. HILL\*, P.K. WIRDNAM\*, R.D.G. MILNER. Department of Paediatrics, University of Sheffield.

Insulin has been implicated in the foetus as the primary anabolic hormone and as a regulator of somatomedin production in foetal and postnatal life. Somatomedin-C (SM-C) was measured by specific RIA in 6 foetal human pancreas explants (15-17 weeks gestation) maintained in culture for up to 12 days. Pancreatic tissue contained very little SM-C ( $< 1\text{mU}/\mu\text{g}$  tissue DNA) either in fresh uncultured or in tissue cultured for 8 days. In 3 experiments insulin and SM-C released into conditioned serum free medium exposed to the tissue for 12-24 h periods at different times during the culture were measured. Insulin (up to 100 mU/ml) did not crossreact in the SM-C RIA. Pancreas released substantial amounts of SM-C into the serum-free medium (14-26 mU/ $\mu\text{g}$  DNA per 24 h). The release of SM-C by the pancreas increased with the length of explant culture and was positively associated with insulin release. SM-C release did not appear to depend on the concentration of various nutrients in the culture medium. Conclusion: Human foetal pancreas is a source of SM-C but does not appear to store the peptide. At present the cellular origin of SM-C in the human foetal pancreas is not clear.

**18** Plasma Arginine Vasopressin measured serially in the preterm newborn. NEIL McINTOSH & ALBERTO SMITH\*. Dept. of Child Health, St. George's Hospital Medical School, London, SW17 0QT

This study serially measured the plasma Arginine Vasopressin (AVP) in 11 preterm infants who had umbilical arterial catheters placed at birth for blood gas measurements either for respiratory distress syndrome or immaturity such that ventilatory support was required. Plasma samples of 20-200  $\mu\text{l}$  were taken 4-12 hourly for up to 100 hrs. after birth. Plasma AVP was measured by a cytochemical assay with a sensitivity of 2 femtograms per ml of plasma\*. In only 4 infants was a significant correlation found between plasma AVP and plasma osmolality. The normal "resting" level of plasma AVP was between 0.5 and 2 pg/ml. In 4 infants apparent bursts of plasma AVP secretion were seen. These could not be correlated either with changes in arterial oxygen, blood pressure, or ventilator pressures - all of which were being monitored continuously or the frequently measured values of plasma sodium or osmolality or urine specific gravity. One spontaneously breathing infant with respiratory distress had continuous very high levels of plasma AVP (12-25 pg/ml) recorded over the 1st 100 hours of life and this was associated frequently with low plasma osmolality. This was obviously inappropriate secretion but the renal response was upset as there was continuous production of dilute urine! Serial measurements of plasma AVP proved possible and showed that contrary to previous evidence even the very preterm newborn is capable of producing high levels of this hormone.

\*Smith, A. & McIntosh, N. Bioscience Reports 1984 in press

**19** The effect of intermittent positive pressure ventilation (IPPV) on cerebral blood velocities in the newborn infant. F. Cowan\* & M. Thoresen\* (S. Halvorsen) Depts. of Physiology, University of Oslo & Neonatal Paediatrics, Ullevål Hospital, Oslo, Norway.

We have used a pulsed bidirectional doppler ultrasound system to study the possible effects that IPPV may have on cerebral arterial and venous blood velocities in the newborn. The signal obtained from these vessels (usually the superior sagittal sinus and an intracerebral artery) is analysed by computer and the results are presented as velocity per consecutive heartbeat. 26 babies have been studied, several on more than one occasion and they represent a broad spectrum of babies requiring IPPV. The results indicate that the babies fall into 3 main groups: 1. those in whom we could find no effect on cerebral blood velocities related either to the rate of ventilation or the peak inspiratory pressure (PIP), 2. those in whom venous velocities diminished intermittently in time with the rate of ventilation and 3. those in whom both arterial and venous velocities were influenced by the rate and the PIP. Most babies were in group 2 but a few showed large swings in arterial velocities related to IPPV and in general the higher the PIP the greater the beat to beat variation in the velocities. By lowering the PIP even by 1-3 cm  $\text{H}_2\text{O}$  the effect on venous velocities lessened and disappeared at a certain critical pressure. These findings could vary from day to day depending on lung disease and spontaneous respiratory effort. We have been able to show that it is possible to avoid some of the largest fluctuations in cerebral velocities by only small reductions in PIP whilst still adequately ventilating the infant.

**20** SUDDEN INFANT DEATH SYNDROME IN FINLAND 1969-80. PERTTI RINTAHAKA \* CHILDRENS CASTLE HOSPITAL, 00250 HKI 25 FINLAND

During 1969-80 infant mortality in Finland decreased from 14.3 to 7.6/1,000 live-born. Postneonatal mortality decreased from 4.1 to 1.7/1,000 live-born until 1979. While in 1980 it was 2.6.

Three hundred and eleven (0.41/1,000 live-born) SIDS occurred during 28 days to eleven months of age. During 1969-79 the incidence was 0.25-0.43, and in 1980 it was 0.79, the SIDS percentage being 30.9 of total postneonatal mortality. If so called borderline cases and children dying under 28 days of age were included the incidence was 0.48/1,000 live-born, one of the lowest figures in the world.

Three hundred and three cases and 297 controls were included in the case-control study. Matching was done by sex, birth place and birth date during the collection of the material, although analysis was done unmatched.

One of the most interesting findings was that maternal hemoglobin was lower during the third trimester in case as compared to control pregnancies ( $p=0.0001$ ).

During the last six years 57.4 % of the case mothers smoked and altogether 76.5 % of the children was exposed to tobacco smoke, while normally in Finland 21 % of the mothers smoked during pregnancy.

The confluence of smoking and mild anemia as a possible etiological factor needs further investigations.

**21** Measurement of fatty acid oxidation in low-birth-weight infants with the  $^{13}\text{C}$ -triolein breath test

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The  $^{13}\text{C}$ -triolein breath test presents a non-invasive method that gives evidence on extent and rate of the fatty acid oxidation. Triolein marked with the stable carbon isotope  $^{13}\text{C}$  is used as tracer. The  $^{13}\text{CO}_2$  resulting from the fatty acid oxidation is exhaled via the lungs and, at spontaneous breathing, collected in a bag by means of a mask and a valve. The  $^{13}\text{CO}_2$  concentration is determined by use of a ratio mass spectrometer (Finnigan MAT 251), and the result is defined as cumulative  $^{13}\text{C}$  elimination in per cent of the dosage administered. The  $^{13}\text{C}$  elimination is directly correlated with the fatty acid oxidation during the examination period.

Values of cumulative  $^{13}\text{CO}_2$  elimination in 21 low-birth-weight infants (870-2390 g birth weight) have shown that after intravenous administration of 10 mg  $^{13}\text{C}$ -triolein  $38.4 \pm 1.8$  % of the administered dose are oxidized in 6-8 hours. The oxidation rate of 24 to 30 % in 4 hours reveals a positive correlation to the maturity rate of premature infants and a negative correlation to the carbohydrate intake. Premature infants with septicemia and hypotrophic premature infants show significantly lower  $^{13}\text{C}$  elimination rates (16.0 %). These patients therefore require a reduced intravenous fat supply.

**22** Studies on Fat Digestion and Lipase Activity in the Gastric Contents of Preterm Infants.

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Preterm newborn infants have to adapt to a high-fat diet despite relatively low pancreatic lipase levels and low bile-salt concentrations. In such infants non-nutritive sucking appears to enhance weight gain. Lingual lipase (L.L.) is known to be active in the stomach of newborn infants. L.L. activity was studied by the hydrolysis of  $^3\text{H}$  triolein in vitro and the products of gastric lipolysis in vivo were determined by densitometry. In 10 infants who had not been milk-fed and 10 infants who had been milk-fed mean ( $\pm$  S.E.) L.L. activity in gastric contents rose from 1.59 ( $\pm$  0.70) before feeding to 4.68 ( $\pm$  0.86) in the milk-fed group ( $p < 0.02$ ). In groups of 1 hourly fed ( $n = 5$ ), 2 hourly ( $n = 4$ ) and 3 hourly ( $n = 4$ ) infants L.L. activity rose in the course of feeding and was associated with triglyceride hydrolysis to mainly diglyceride and free fatty acids. In 4 infants on 3 hourly feeds, alternately tube and bottle-fed on 2 successive days, the mean ( $\pm$  S.E.) maximal L.L. activity was 16.71 ( $\pm$  3.71) when tube-fed and 24.41 ( $\pm$  3.42) when bottle-fed. In preterm infants L.L. has an important role in fat digestion and sucking appears to enhance its secretion.