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GLUCOSE TOLERANCE AND INSULIN SECRETION IN VERY SMALL BABIES

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Nineteen exchange transfusions were performed via the umbilical artery using blood preserved with acid-citrate and dextrose in eight term /34-40 weeks gestation/ and nine preterm /26-33 weeks gestation, 850-560 g body weight/ infants. Plasma glucose rise was similar in both groups during transfusion $\Delta 93 \pm 7$; $\Delta 77 \pm 7$ mg%/. The glucose disappearance was $1,33 \pm 0,20$ in terms and $1,47 \pm 0,19$ in prematures. Term infants responded to glucose with a sharp rise in mean plasma insulin $\Delta 24 \pm 3$ uU/ml/ which was seen three hours following the transfusion. $\Delta 41 \pm 2$ uU/ml/. Insulin secretion did not change in prematures during the transfusion thereafter rose slightly $\Delta 11 \pm 1$ uU/ml/. Correlation was found between the maximum post-transfusion plasma insulin and the birth weight of the infants $r=0,769$ $p<0,001$ /. FFA decreased during transfusion in terms /max 609 ± 257 $\mu\text{mol/l}$ /, increased in both groups afterwards. Term infants secreted more HGH than prematures $6,34 \pm 1,31$; $4,07 \pm 0,51$ ng/kg net HGH/. Correlation was found between the HGH values and the gestational age of the infants $r=0,5186$ $p<0,05$ /.

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DEFICIENCY OF THE EXTRAMITOCHONDRIAL FRACTION OF HEPATIC PHOSPHOENOLPYRUVATE CARBOXYKINASE IN NEONATAL HYPOGLYCAEMIA

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The activation of gluconeogenesis is an essential part of the adjustment to extrauterine life. At birth this activation is completed with the occurrence of phosphoenolpyruvate carboxykinase /PEPCK/ in the liver. This enzyme occurs partly in the mitochondria and partly in the cytosol, gluconeogenesis being started at birth with the induction of extramitochondrial PEPCK. The case to be presented developed a therapy-resistant hypoglycaemia 24 hours after birth. Isotope studies revealed a marked reduction in labelling of glucose after i.v. injection of 14C -alanine. Plasma alanine, glycine and glutamine seemed somewhat high whereas lactate and pyruvate were not elevated. A liver biopsy showed fatty infiltration. Studies of the gluconeogenic key enzymes in liver showed normal activities except PEPCK. The total activity of this enzyme was normal, but there was an abnormal subcellular distribution, virtually no activity being detected in the extramitochondrial fraction.