37 REGIONAL VASCULAR RESPONSES TO ASPHYXIA IN THE RABBIT

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8 spontaneously breathing male New Zealand white rabbits were exposed to a 50 min period of asphyxia. The average pO_2 was 59, pCO_2 57 mmHg, stand.bic. 17.6, pH 7.10. Cardiac output /C.O./ was measured with the dye dilution method, distribution of C.O. and organ blood flow was assessed with left ventricular injection of radioactively labelled microspheres. The results were compared with 8 control animals. C.O., arterial pressure and total peripheral vascular resistance did not change, distribution of C.O. however showed increased flow to heart, brain and diaphragm. Flow to kidneys, spleen and adipose tissue was reduced, gastro-intestinal tract, liver, skeletal muscle, skin and carcass maintained their blood flow. Prolonged moderate asphyxia produces preferential blood supply to vital organs and maintains flow for a ready-to-function musculature. Their blood supply is diverted mainly from the kidneys, whereas the reduced adipose tissue flow has probably metabolic consequences since it facilitates lipolysis through an increased capillary filtration quotient.

38 NEW RADIOISOTOPIC METHOD FOR ESTIMATION OF GALACTOSE-1-PHOSPHATE IN GALACTOSEMIA

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Determination of Gal-1-P was performed by measuring the amount of ^{14}C Glu 1-P formed in the reaction: UDP- ^{14}C Glu+Gal-1-P UDPGal +/ ^{14}C /Glu-1-P, in the presence of exogenous enzyme hexose-1-phosphate uridylyltransferase. The amount of ^{14}C /Glu-1-P is a measure of the amount of Gal-1-P present in the investigated material. This method allows to determine Gal-1-P in RBC in the range: about 1.0 to 25 mg/100 ml RBC. The method covers the expected concentrations range for clinical purposes. The values of erythrocyte Gal-1-P in galactosemic patients cover the range: 3.47-9mg/100ml RBC. In none of the healthy children was found a measurable amount /below 0.8 mg/100ml RBC. The accumulation of gal-P in RBC of galactosemic patients was confirmed by in vitro preincubation with galactose. This method gives reproducible results, and is simpler and less time consuming than other methods.

39 EFFECTS OF GLUCAGON ON PLASMA ADENOSINE 3,5-MONOPHOSPHATE /cAMP/ IN FULL TERM NEWBORNS AND PREMATURES

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cAMP plasma levels in fullterm and premature newborns, during the first day of life, were 3-4 times higher than those of children. Glucagon /50 μ g/kg, i.v./, attains maximum levels of cAMP at 20 minutes. In newborns and prematures the response was similar to that of children.

	cAMP pmoles/ml	
	Before injection	20' after
Newborns /20/	96 - 168 /128/	516 - 792 /620/
Prematures /6/	99 - 160 /123/	535 - 745 /615/
Children /5/	30 - 44 /39/	562 - 912 /662/

In conclusion both fullterm and premature newborns have higher than adults plasma levels cAMP and also have mature hepatic cAMP response to glucagon.

40 HEAT LOSS OF NEWBORN INFANTS OF DIFFERENT BODY SIZE IN THERMONEUTRAL AND COLD ENVIRONMENT MEASURED BY DIRECT CALORIMETRY

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45 newborn infants of different body size was subjected to an ambient temperature T_a of 32°C or T_a = 28°C and their heat loss was measured by direct gradient layer calorimetry at relative humidity of 50% and air flow 30 l/min. Total heat loss per kg body weight /wt/ in the small for date, appropriate-for-date and large-for-date infants at T_a 32°C were 2.93 $\pm$ 0.12 W. kg⁻¹/M $\pm$ SE/, 2.23 $\pm$ 0.05, 2.05 $\pm$ 0.07 respectively and at T_a = 28°C 4.35 $\pm$ 0.15; 3.06 $\pm$ 0.1 and 3.04 $\pm$ 0.09 respectively. This negative correlation between heat loss /W. kg⁻¹/ and the body size was especially apparent below 2.5 kg body weight. Evaporative heat loss was fairly constant in the two different environments irrespective to the body size. It is concluded, that in the higher heat loss in the small-for-date infants at T_a = 28°C, the disadvantageous surface-body mass ratio plays the most important role.

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41 USE AND EXPERIENCES WITH THE MEASUREMENT OF THE TRANSCUTANEOUS PO_2 /tc PO_2 / IN LONG-TERM VENTILATED NEWBORNS

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Recent advances in the treatment of children with severe respiratory disorders have markedly improved the survival of long-term ventilated children in intensive care units up to 70-85%. The development of the transcutaneous monitoring system of oxygen tension, tc PO_2 , and the improvement of the method have essentially contributed to improving the possibilities of continuous monitoring of long-term ventilated patients by means of recording their oxygen tension transcutaneously. The present paper reports the first application of the method in our pediatric intensive care unit. From our large amount of data obtained we discuss the effect on the transcutaneous recording of partial oxygen tension of crying, cramping, arterial sampling, too high oxygen concentration in the inspiratory gas mixture, disconnection from the respirator, PEEP and suction with following forced hyperventilation. Even from the most critical point of view the method is a considerable improvement towards the effort in increasing intensive monitoring while reducing the strain on long-term ventilated patients.

42 DIAGNOSTIC AND PROGNOSTIC SIGNIFICANCE OF SERUM TOTAL CREATINE PHOSPHOKINASE AND BRAIN SPECIFIC ISOENZYME ACTIVITY IN ASPHYCTIC NEWBORN INFANTS

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Elevated total serum creatine phosphokinase /CPK/ activity / 200 U/l/ was found during the first week of life /peak between 24-48 hrs/ in 32 out of 76 newborn infants, having had birth asphyxia. Of the 32 neonates with elevated total serum CPK 27 survived and in 26 the EEG, neuromotor- and mental development were followed at regular intervals up to the age of one year. By the last follow-up examination 13 infants revealed signs of neurological and/or mental abnormality. Isoenzyme separation on DEAE Sephadex A 50, according to Dubo et al., was performed in 23 cases showing elevated total CPK activity during the neonatal period. The brain specific isoenzyme fraction increased above the normal upper limit of 45 U/l in 13 infants /range 45.8 U/l-449.6 U/l/: 9 showed neurological and mental defects at the age of 1 year. It has been concluded that the coincidence of birth asphyxia with an activity of more than 200 U/l in total serum CPK and the increase of the brain specific isoenzyme fraction by 15 per cent or more of the total CPK means a bad prognosis regarding neuromotor and mental development.