103 IMPACT OF HALOTHANE-ANESTHESIA ON CEREBRAL CIRCULATION IN INFANCY.

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Loss of autoregulation of cerebral blood flow (CBF) under halothane-anesthesia has been shown in cats and monkeys. In human adults halothane enhances CBF. However, there are no data about the impact of halothane-anesthesia CBF in infancy. We measured changes of mean blood flow velocity of the right internal carotid artery (MBFV) by transfontanellar Doppler to estimate changes of CBF with induction of anesthesia by halothane 1.5 %. TcpCO2 was kept constant. Mean arterial blood pressure (MABP) was measured oscillometrically. Our study collective consisted of 9 infants (age 15-80, body weight 2.0-5.6 kg) undergoing minor operations. Results (median, range):

parameter	before	5 min after	sign-test
tcpCO2 (mmHg)	34 (27-40)	34 (26-41)	n.s.
MABP (mmHg)	63 (40-106)	51 (33-71)	n.s.
MBFV (cm/s)	18 (16-40)	14 (8-33)	p<0,05

Median decrease of MBFV was 30 % (0-70).
Conclusion: Narcotic doses of halothane may impair cerebral perfusion in young infants.

104 NEUROPEPTIDE Y (NPY) AND CATECHOLAMINE (CA) RELEASE IN THE NEWBORN PIGLET - ENHANCED AFTER THEOPHYLLINE (T).

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NPY is a co-transmitter of noradrenaline (NA) and assumed to potentiate some of the cardiovascular effects of NA. Since NPY is assumed to occur only in the sympathetic nerve terminals and not in the adrenal medulla it might be used as a selective marker of sympathetic nervous activity while the plasma CA also originate from the adrenal medulla and paraganglia. Nine anesthetized 2 w piglets were subjected to 6% CO_2, 12% O_2 and 6% O_2 for 6 min respectively before and after T (20 mg/kg i.v.). Arterial NA, adrenaline (A) and NPY were measured before and during each gas challenge. CA levels were unaffected by CO_2 and 12% O_2 both before and after T. However, there was a moderate increase in A and NA during 6% O_2 which was greatly enhanced after T (bef. T; A, nM: 0.6-11, NA: 25-87, aft. T; A: 1.5-90, NA: 35-158). NPY levels were increased both during 12% and 6% O_2, as was the basal levels of NPY after T (bef. T, pM: 12% O_2, 36-84, 6% O_2; 53-153, aft. T; 12% O_2; 117-146, 6% O_2; 137-168). We conclude that the sympathetic nervous system is substantially activated during hypoxia and theophylline treatment as indicated by the NPY levels, while the adrenal medulla only seem to be considerably activated during hypoxia after theophylline treatment.

105 ACETAZOLAMIDE AND CEREBRAL VASODILATATION IN THE HYPOTENSIVE NEWBORN PIGLET

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A selective cerebral vasodilator could be useful clinically in situations where low blood pressure might lead to cerebral ischaemia.

8 newborn piglets were anaesthetised, ventilated, paralysed, arterial and venous catheters were inserted and a fontanelle made surgically. Cerebral blood velocity was measured from an intracranial vessel by a 5 MHz computerised Doppler (Vingmed SD 100) system held on the fontanelle. Hypotension was induced by arterial bleeding until mean arterial pressure had fallen by at least 30% or was below 45 mm Hg.

Initially the piglets showed a definite cerebral vasodilator response to CO2 but this became minimal or absent when hypotension occured. The cerebral vasodilatation response to acetazolamide 50 mg/kg IV was also minimal or absent in the hypotensive state although the expected rise in arterial pCO2 and fall in end-tidal CO2 was found. Administration of 6%CO2 after the acetazolamide produced no further vasodilatation. Hypotension induces cerebral vasodilatation in an attempt to maintain cerebral blood flow and further dilatation cannot occur with hypercapnoea or acetazolamide.

106 BOTH α- AND β-ADRENORECEPTOR (AR) MEDIATED INOTROPIC COMPONENTS (IC) OF NORADRENALINE (NA) ARE REVEALED BY REVERSING THE RESPONSE IN MYOCARDIUM BY ADRENERGIC BLOCKERS (AB).

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The contribution of α-AR stimulation (S) to the total inotropic effect (IE) of NA during full β-ARS has been questioned. The present study reveals a way of demonstrating an α-AR effect in the presence of full β-ARS by studying the reversal responses to AB at supramaximal NA stimulation in rat papillary muscles. The response was rapidly reversed (t_{50} = 2.8 ± 0.2 min) by simultaneous addition of the β-blocker timolol and the α-blocker prazosin. When AB were added sequentially (5-10 minutes apart), two IC in the inotropic response to NA could be demonstrated: one IC which was sensitive to timolol (73.3 ± 6.9 % of total response) and was taken as β-AR mediated and one IC which was sensitive to prazosin (26.7 ± 5.7 % of total response) and was taken as α-AR mediated. Thus, there is a significant contribution also from an α-AR effect. There is a mutual inhibition of one component upon the other as the expression of both α-AR and β-AR effects was less during combined AR stimulation than when the receptor populations were stimulated separately.

107 DETERMINATION OF SURFACTANT APOPROTEIN AND PHOSPHOLIPIDS IN AMNIOTIC FLUID FOR ESTIMATING FETAL LUNG MATURITY.

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Measurement of disaturated phosphatidylcholine(DSPC) or phosphatidylglycerol(PG) have been proved to be useful in complicated mothers for evaluating the fetal lung maturity. But the procedure is complicated and takes long time. Kuroki et al.(Ped Res 19: 1017,1985) described a simple immunoassay of the surfactant apoprotein having MW of 36 KDa and came up with an immunoassay kit incooperated with TEIJIN inst. With this kit,determination can be done within 1 h. We conducted this study to assess the clinical usefulness of this immunoassay kit (kindly provided from TEIJIN inst.)in the evaluation of fetal lung maturity. Twenty-six amniotic fluid samples were examined for levels of surfactant apoprotein, as well as DSPC, and PG. Results were as follows;

Gestational Age (weeks)	Number	Apoprotein Conc.(ug/ml)
<30 weeks	13	0.412 + 0.08
30-35 weeks	4	6.06 ∓ 2.69
35-41 weeks	9	9.48 ∓ 3.25

The amount of apoprotein was in a statistically significant correlation with gestational age as well as DSPC and PG.
This assay kit will provide rapid and accurate information about fetal lung maturity in the clinical field.

108 CLEARANCE OF ENDOTRACHEALLY INSTILLED SURFACTANT IN PREMATURE SURFACTANT DEFICIENT RABBITS.

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To determine the clearance of endotracheally instilled artificial porcine derived surfactant (APS) from the lung, premature surfactant deficient rabbits were sacrificed at 30, 60, and 120 min. following surfactant instillation. APS was determined in lung tissue specimens using a monoclonal antibody against APS apoprotein a peroxidase staining technique and a counting grid. In 18 rabbits APS was instilled endotracheally, 6 did not receive surfactant (controls). Results: The number of surfactant positive points in alveoli was 38.2±20.0, 13.8±12.0 and 3.3±4.4 at 30, 60, and 120 min. after instillation resp. ($p<0.05$). In the perivascular spaces we found 0, 4.0±0.1 and 14.5±7.3 resp. ($p<0.05$). No surfactant positive points were found in controls. We conclude that endotracheally instilled APS is cleared from the lungs of surfactant deficient premature rabbits within 2 hours. Presumably via the lymphatic ducts in the perivascular spaces.