- 120** SENSITIVITY OF DETECTION OF BRAIN-LESIONS IN VERY PRETERM INFANTS BY ULTRASOUND. Hope, P.L., Gould, S.J., Hamilton, P.A., Costello, A.M. de L., Reynolds, E.O.R. University College London, Depts. of Paediatrics and Morbid Anatomy, London, England.

To investigate how well ultrasound scanning (US) of the brain detected autopsy-proven lesions, abnormalities detected by a sector scanner with 6 and 7.5 MHz probes were compared blind with the autopsy findings in 57 infants born at less than 33w gestation who died aged 0-166 (median 2) days. Germinal layer-intraventricular haemorrhage (GLH-IVH) was found at autopsy in 71 of the 114 hemispheres and always detected by US (sensitivity 100%, 71/71). The 43 hemispheres with no GLH-IVH at autopsy were thought to show US evidence of GLH in 16 infants and IVH in 4, most of whom were extremely preterm (specificity 53%, 23/43).

Parenchymal lesions were present in 50 (44%) of the 114 hemispheres and were predominantly haemorrhagic in 11 and ischaemic in 39. The haemorrhagic lesions were correctly identified in 9 infants (sensitivity 82%, 9/11) and there were 3 false positive diagnoses (specificity 97%, 100/103). Only 11 of the 39 hemispheres with histological evidence of ischaemia were correctly identified by US, mainly because focal periventricular leucomalacia or diffuse gliosis were not detected (sensitivity 28%, 11/39). 10 hemispheres with periventricular echoes thought to represent leucomalacia showed no histological evidence of ischaemia (specificity 87%, 65/75).

US is not a sensitive technique for the early detection of lesser degrees of cerebral ischaemic injury in very preterm newborn infants.

- 121** G. Greisen, L. Hellstrom-Westas, H. Lou, I. Rosén, and N. Svenningsen. EEG DEPRESSION AND GERMINAL LAYER HEMORRHAGE GLH FROM THE DEPT. OF NEONATOLOGY. Rigshospitalet, Copenhagen, Denmark, and the Dept. of Paed. and Clin. Neuropaths., University Hospital, Lund, Sweden.

Amplitude integrated EEG (aEEG) was recorded between 24 and 50 hrs after birth in 32 newborns with 32 weeks gestational age or less. Germinal layer haemorrhage (GLH) was diagnosed by repeated ultrasonography. In the six infants with neither GLH nor hypocalcaemia continuous background activity was present in more than 20% of the time at 30 h, and seizure-like activity was exceptional in contrast to the rest. In 7 infants without GLH but with hypocalcaemia continuous background activity appeared later. In all 4 infants who later developed FLH, EEG was depressed at 30 h. A further EEG depression occurred concurrently with the development of GLH grade II-IV.

In 15 of the infants the EEG recording was started before 6 h in the absence of GLH. In 7 of these, mean internal carotid flow velocity (cmFV, Doppler) more than doubled concurrently with increases in arterial blood pressure (aBP). GLH developed more frequently in this group compared to the remaining 8 infants ($p = 0.039$). The increase in aBP and cmFV was preceded by lower aBP, cmFV, and cmFV.

We suggest that GLH primarily occurs in cerebrovasculature unstable infants (lost autoregulation) with preceding cerebral metabolic and electrophysiologic derangement unrelated to present energy supply, and that further functional deterioration is caused by the most severe haemorrhages.

- 122** HEMOSTASIS AND PERIVENTRICULAR-INTRAVENTRICULAR HEMORRHAGE OF THE NEWBORN

Margot van de Bor¹, Ernest Briet², Frank van Bel¹, Jan H. Ruys¹, University Hospital, Neonatal Unit¹ and Hemostasis and Thrombosis Research Unit², Leiden, The Netherlands.

In a prospective study we analysed the role of coagulopathy in the development of periventricular-intraventricular hemorrhage (PIVH) in 49 consecutively admitted preterm infants of less than 34 weeks gestation by serial ultrasound examinations (every 8 hours during the first week of life) and coagulation assays (prothrombin time, Normotest[®], APTT, factors II, V, VII, fibrinogen, AT III and platelets) on the day of birth and on day 3 and 5. In twenty patients (41%) PIVH was detected. On the day of birth patients with PIVH had significantly lower levels of factor V than the patients without PIVH ($p < 0.05$), but all other clotting factors gave similar results and on the third and fifth day all results were similar including factor V. Even in the small subgroup of infants who subsequently developed grade IV hemorrhage we did not find a more severe coagulopathy than in the other infants although they had significantly lower levels of platelets and of factor VII at birth ($p < 0.01$; versus non PIVH). We conclude that coagulopathy does not play an important role in the etiology of PIVH.

- 123** NIMODIPINE ENHANCES NEWBORN LAMB CEREBRAL BLOOD FLOW. B.M. Mogilner, S. Ashwal, S. Dale and L.D. Longo. ¹Neonatal Unit, Kaplan Hospital, Rehovot, Israel, and ²Department of Pediatrics, and ³Division of Perinatal Biology, Loma Linda University, Loma Linda, CA, USA.

Nimodipine (BAY e 9736) a dihydropyridine type Ca^{2+} channel blocker, has been introduced recently as a selective cerebral vasodilator. In this study the effects of Nimodipine on total and regional CBF, selected organ flow and cardiovascular function were investigated in newborn lambs. After anesthesia with 1% chloralose the lambs were intubated and mechanically ventilated. Blood flow to the brain and other organs was measured by radio-actively labeled microspheres injected into the left ventricle. Nimodipine (0.2, 0.6 and 2.0 $\mu g/kg$) was administered by bolus intravenous infusion to 5 normotensive animals. At 2.0 $\mu g/kg$ the total CBF increased 92.8%. Subsequently, mean arterial blood pressure was decreased in other 6 newborn lambs, from 70.6 to 40.2 mm Hg. Following a 30 $\mu g/kg$ bolus of Nimodipine, CBF increased significantly above control values (total brain 41.5%, gray matter 53%, subcortex 75.6% and brain stem 68.8%). Nimodipine appears to increase CBF in both the normotensive and hypotensive normoxic newborn lamb without significant side effects in blood pressure, heart rate or arterial blood gases. At the blood pressure chosen for hypotension, autoregulation was preserved and Nimodipine had an additive effect of increasing CBF, presumably by further vasodilatation. These findings suggest that Nimodipine may be of therapeutic value in the hypotensive stressed neonate at risk for ischemic brain injury. However, one must also be concerned about the potential for cerebral hyperfusion and the risk of intracranial hemorrhage in such infants.

- 124** PATENT DUCTUS ARTERIOSUS AND CEREBRAL BLOOD FLOW. E. SALIBA, A. CHANTEPIE, M.C. VAILLANT, D. BLOC, L. POURCELOT, J. LAUGIER - Hopital Gatién de Clocheville, 49, Bd Béranger - 37000 TOURS.

Blood flow in the anterior cerebral arteries were studied in 24 preterm infants with patent ductus arteriosus (PDA) and left to right shunt as assessed by physical and echocardiographic findings. Continuous wave doppler technique with spectral analysis were used. We analysed the flow patterns obtained quantitatively by calculating the circulatory index (CI) (peak systolic frequency minus trough diastolic frequency/peak systolic frequency) and area under the curve in order to determine a retrograde/forward flow ratio (R/F). The results were compared to those obtained from 12 control infants without ductal patency. Results are shown with table:

	*p 0.001	CONTROL GROUP n=12	PDA GROUP n=24
G.A. WKS		32 (29-34)	30 (27-33)
Bwg		1460 (1200-1970)	1283(800-1860)
RD (Respiratory Distress)		0	21
APNOEAS		0	3/18
MECHANICAL VENTILATION		0	24
LA/AO (2D. Echo)		1.05 (0.9-1.1)	1.36 (0.9-1.8)
CI		0;73 (à J3)*	0.89*before clos. 0.73*after clos. 0.22(0.14-0.32)
R/F			CI=0.68 after clos

Ductal shunting of CBF could explain the diminished and the retrograde flow in infants with PDA and the return flow patterns following ductal closure.

- 124** PREVENTION OF HYPOXIC-ISCHEMIC CEREBRAL DAMAGE IN PRETERM INFANTS BY PHENOBARBITAL. Ruth V, Virkola K, Paetau R, Raivio KO. Children's Hospital, University of Helsinki, Finland.

We evaluated the effect of phenobarbital (PB) on two sequelae of hypoxic-ischemic insult in preterm infants, early intraventricular hemorrhage (IVH) and late handicap due to neuronal damage. Infants weighing $\leq 1500g$ admitted before 4 hr of age were randomly assigned into a phenobarbital group (PB+) to receive 15 mg/kg iv twice at a 4 hr interval, followed by 5 mg/kg every 24 hr for 5 days, or into a control group (PB-). 45 infants were enrolled into the PB+ and 49 into the PB- group; 7 PB+ and 3 PB- infants died neonatally. IVH was diagnosed by ultrasound (US) in 13 PB+ infants (29%), 4 of them severe (grades III-IV). 28 PB- infants (57%) had IVH, 7 of them severe. Overall incidence of IVH was significantly higher in the PB- group ($\chi^2=8.8$, $p < 0.01$), but when considering only severe IVH there was no significant difference between the groups. At follow-up 13 (39%) PB+ and 17 (40%) PB- infants had ventricular dilatation on US; marked dilatation or hydrocephalus was found in 5 PB+ and 12 PB- infants (NS difference). Neurodevelopmental status at 15 months of age revealed mild abnormality in 4 (11%) PB+ and 4 (9%) PB- infants, whereas it was clearly abnormal in 5 (14%) PB+ and 4 (9%) PB- infants. Our results indicate that early high-dose PB-treatment of small preterm infants decreased the incidence of mild to moderate IVH. However, follow-up so far has failed to show a difference between treatment and control groups in neurodevelopmental outcome or incidence of ventricular dilatation. Routine administration of PB to small preterm infants cannot be recommended.