

†1451 ECONOMIC ANALYSIS OF REGIONALIZED NEONATAL CARE FOR LOW BIRTH WEIGHT INFANTS IN THE STATE OF RI.

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Cost-benefit analysis was utilized to evaluate the economic outcome of regionalized neonatal care of low birth weight (LBW) infants (birth weight 500 to 1500 gm) in RI. In RI, neonatal regionalization began in 1974-75 and is now well established. We selected two study periods (1974-75 and 1977-78) which provided the data base for hospital and long term care costs, the latter were based on neurodevelopmental assessment of survivors who were at least 2 years of age when evaluated at our follow-up clinic. Between the 2 study periods (1974-75 vs 1979-80), neonatal mortality fell significantly 52 vs 36%, morbidity was however unchanged (normal 60 vs 65%, mild handicap 13 vs 12%, moderate handicap 12 vs 10% and severe handicap 15 vs 13%). Cost-benefit analysis (all values converted to 1982 dollars) showed:

	1974-75	1979-80
Total Cost/Survivor	\$48,932	\$46,838
Total Benefits/Survivor	\$60,238	\$67,223
Benefits-Costs/Survivor	\$11,306	\$20,385
Number of Survivors	97	162
Total Benefits-Costs	\$1,096,682	\$3,302,370

We conclude that the increase in absolute number of intact survivors since the establishment of regionalized neonatal care in RI has resulted in a 3-fold increase in economic benefits.

1452 OUTCOME OF INFANTS LESS THAN 29 WEEKS GESTATION.

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Studies on VLBW infants often include outborn and SGA infants and do not describe the effects of short gestation on mortality and morbidity. This study documents the outcome of 138 infants born in a perinatal center (1977-1980) of whom 64 were born at 24-26 weeks gestation (Group I) and 74 were born at 27-28 weeks gestation (Group II). 81 were discharged alive. Neurological status was determined in all 80 longterm survivors and 72 (90%) had psychometric testing at 24 months corrected age. Major handicap included cerebral palsy, blindness, deafness, and Bayley MDI <69 and minor handicap included tone disorder, refractive error, conductive deafness and attention deficit. For Group I infants mean birthweight (gms) was lower (807 v 1049, p<0.0001), hospital survival was reduced (45% v 70%, p<0.005) and rates for major (31% v 8%) and minor handicap (28% v 12%) were increased (p<0.001). Mean Bayley scores were similar (MDI 91 v 92, PDI 82 v 88). Analysis of perinatal variables showed that Group I babies were more critically ill and unstable with significantly lower 5 minute Apgar score, admission temperature, arterial pH and haematocrit; higher FiO₂ requirements and arterial PCO₂ levels. Duration of ventilation and oxygen therapy and incidence of stage 3/4 BPD (45% v 4%) and all grades of RLF (16% v 3%) were significantly increased. This study shows that shortened gestation is associated with increased mortality and morbidity and may be more specific than birth weight in predicting outcome.

●1453 AUTOREGULATION OF CEREBRAL BLOOD FLOW IN THE AWAKE NEWBORN PUPPY.

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Hypertension induced surges in blood flow to the subependymal germinal matrix (GM) may be important in the pathogenesis of neonatal IVH. We measured regional cerebral blood flow (rCBF) to 30 brain regions in 17 awake newborn puppies with quantitative autoradiography and ¹⁴C-iodoantipyrine as tracer. Blood flow was studied during moderate hypotension (MAP 35-50 mmHg) in 4, during normotension (50-70 mmHg) in 6, and during phenylephrine induced hypertension (75-105 mmHg) in 7. An autoregulatory plateau was seen for all brain regions over the range 35-70 mmHg. In gray matter, increases in MAP above 75 mmHg significantly increased rCBF; hypertensive incremental CBF (ICBF) was greater in brainstem nuclei than in cerebral cortex (eg, ICBF .142 ml/min/g/mmHg for oculomotor nucleus, .019-.069 ml/min/g/mmHg for parietal cortex). For white matter, ICBF remained low (.001-.006 ml/min/g/mmHg) even when MAP exceeded 75 mmHg. GM blood flow did not react uniformly to hypertension. Hypertensive ICBF to posterior GM was low and similar to white matter while ICBF to rostral, densely cellular GM was higher (.014 ml/min/g/mmHg) and similar to some cortical regions. From our data we have concluded the following: 1) autoregulation of rCBF is intact for all brain regions including GM for physiologic MAP; 2) at MAP above the autoregulatory plateau, rostral GM blood flow increases more than blood flow to adjacent white matter. These increases in GM blood flow during hypertension may contribute to the genesis of IVH. Differences in hypertensive ICBF between GM and white matter may partially explain the selective vulnerability of the GM for hemorrhage.

●1454 THE EFFECTS OF HYPOXEMIA AND HYPOTENSION ON LOCAL CEREBRAL GLUCOSE UTILIZATION IN THE AWAKE NEWBORN PUPPY.

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As part of an ongoing study to evaluate the pathogenesis of neonatal hypoxic-ischemic brain injury, we have studied the effect of hypoxemia and severe hypotension on newborn brain metabolism. In 5 puppies, severe hypoxemia was produced by having the puppy breathe a 4% O₂/96% N₂ mixture; normal MAP was maintained. In 7, severe hypotension (MAP 20-25 mmHg) was induced by hemorrhage; these animals spontaneously hyperventilated and maintained normal or elevated pO₂. During these insults, local cerebral glucose utilization (LCGU) was studied using quantitative autoradiography and ¹⁴C-deoxyglucose. Hypoxemia caused a marked increase in LCGU to all brain structures: increases of 200-300% occurred diffusely in cerebral cortex and subcortical nuclei; increases of 300-600% occurred in white matter. Severe hypotension caused localized increases in LCGU: increases of 300-500% were seen in parasagittal cerebrum; increases of 150-200% were seen in the subependymal zone. The distribution of the increased LCGU correlated with the presumed distribution of pathologic injury after both insults; i.e. diffuse neuronal necrosis after hypoxemia and watershed infarction after severe hypotension. The increased LCGU probably represents a pathologic disruption of oxidative metabolism. The resultant increase in local tissue lactate may further injure brain elements locally. Thus, increased LCGU may be both a marker of the most vulnerable brain regions after insult and also an important step in the pathogenesis of neonatal hypoxic ischemic brain injury.

1455 CONTINUOUS GLUCOSE INFUSION AND ITS EFFECTS ON PULMONARY PHOSPHATIDYL CHOLINE (PC) AND DISATURATED PC (DSPC) IN FETAL RABBITS.

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It is known that infants of diabetic mothers (IDM) have a higher incidence of hyaline membrane disease (HMD). To determine the effect of continuous glucose infusion on PC and DSPC in lung tissue in vivo, a rabbit model was used. On 26th day of gestation, femoral vein was catheterized in a pregnant rabbit and D25 0.45% saline (group 1) or 0.9% saline (group 2) was infused over 46 to 48 hours. Sham operated pregnant rabbits underwent exposure of femoral vein and attempted catheterization (group 3). C-hysterectomy was performed on 28th day of gestation. Necks of the fetal rabbits were tied at delivery to prevent air breathing. Lungs were removed and analysed for PC and DSPC. Serum insulin and glucose were determined on fetal and doe's blood. Data on 6 rabbits in group 1, 4 rabbits in group 2 and 5 rabbits in group 3 are available. The serum insulin level in does in group 1 was higher compared to other two groups after 24 hours of infusion. The serum glucose in does after 24 hours in group 1 was higher than other two groups. As a % of total phospholipid (TPL), PC and DSPC were not different in all three groups.

Mean + SD	Group 1	Group 2	Group 3
PC % TPL	45.1 ± 5.7	50.2 ± 2.8	45.1 ± 2.9
DSPC % TPL	22.1 ± 6.2	20.7 ± 2.0	20.7 ± 2.7

This preliminary data suggest that infusion of glucose in high concentrations does not affect pulmonary tissue PC and DSPC in fetal rabbits in vivo.

1456 R.L.F.: NOT UNIFORMLY FREQUENT AND/OR SEVERE: FOLLOW-UP OF 206 INFANTS OF LOW BIRTHWEIGHT.

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RLF with a reported incidence as high as 20-25% remains the single most important complication among survivors weighing <1500 gms. From Jan. 1978 to June 1983, 206 infants (101:M, 105:F) <1500 gms were examined by the same ophthalmologist at discharge and on follow-up visits. 41 of them were infants <1000 gms. 20 infants had Gr. I-V RLF at the moment of discharge (9.7%): 15 infants (7.5%) had Gr. I-II RLF (12=Gr. I; 3=Gr. II) and all of these had a normal eye examination by 3 months of age. 5 infants developed Gr. III-V RLF (2.5%) and 2 of them are blind (1%). One child needs glasses and the other 2 have completely recovered. All 3 cicatricially affected infants were <1000gms and all required prolonged ventilation rendering the incidence of cicatricial RLF for <1000gms 7.3% and for >1000gms 0%. Factors found to have an influence on RLF were: 1/ Gest. age: 29.8 vs 26.8 wks. 2/ BW: 1196 vs 1013 gms. 3/ Length of O₂ therapy: 20.8 vs 48.8 d. 4/ Length of ventilation: 3.6 vs 12.8 d. 5/ Highest FiO₂: 27.1 vs 68.1% 6/ Highest pCO₂: 58.5 vs 71 mmHg. 7/ Lowest pCO₂: 30.8 vs 26.4 mmHg. All p values were at <0.05. Anqar scores and highest pO₂ values were found not to be statistically significant factors. On Griffith Score at 24 mos of age no significant difference was found between infants with or without RLF when correction was made for eye motor-coordination. Whether this very low incidence of RLF is due to the careful control of all ventilation parameters or to the extensive use of antenatal steroids remains to be proven.