transmitters such as dopamine can be implanted into adult mouse brains. The embryonic cells not only survive but continue to make their transmitter and release it in the new environment. The embryonic cells can secrete transmitter and correct behavioral deficits in the adult mouse. These and other experiments made on the mammalian nervous system raise the possibility that the prospects for repair might not be as hopeless as had previously been thought. The experiments performed in our laboratory are designed to study mechanisms at the cellular level that enable nerve cells to grow, to grow in a particular direction, to stop growing when they reach the correct destination and then reform synaptic connections with the correct cells but not the incorrect cells (3). The animal used is a simple invertebrate, the leech. Upon such neurons of this animal one can make all sorts of manipulations that would be impracticable in mammalian nerve cells. The central nervous system of this animal can repair itself with amazing fidelity, the correct cells being reconnected to their correct target cells after an injury. At the cellular level it has been possible to show that the nature of the substrate on which the cells are plated has a dramatic influence on their rate of outgrowth. Once targets are reached in culture the connections that are made are highly specific. Some pairs of cells make electrical synapses, other pairs make chemical synapses, other pairs make mixed chemical and electrical synapses and other pairs fail to become connected. With this simplified system it becomes possible to investigate molecular events involved in neurite extension and in synapse formation between specific cells. It seems inconceivable that highly complex structures such as the visual cortex or the frontal lobes could rewire themselves after injury. Nevertheless, it now seems possible that with better knowledge of cellular mechanisms, progress may be made towards understanding how neurons in the brain can reform their connections after injury.

References

1. Aguayo JG 1985 Axonal regeneration from injured neurons in the adult mammalian central nervous system. Chapter 15 in *Synaptic Plasticity*, ed Carl W Cotman The Guilford Press, New York
2. Gage FH and Björklund A 1986 Neural grafting in the aged rat brain. *Ann Rev Physiol* 48, 447-459
3. Fuchs LP, Hernderson L and Nicholls JG 1982 Chemical transmission between individual Retzius and sensory neurones of the leech in culture. *J Physiol* 323: 195-210

PATHOPHYSIOLOGY OF CEREBRAL BLOOD FLOW

H. Lou

John F. Kennedy Institute, Dept. of Neuropediatrics, Glostrup, Denmark

We have previously shown in experiments on fetal lamb that autoregulation of cerebral blood flow is extremely vulnerable in utero. Even if autoregulation is more stable in the newborn lamb (apparently due to higher SaO_2), it is still vulnerable to hypoxia, and is abolished for several hours when SaO_2 drops below 50 per cent. Similarly, 19 stressed newborn infants were shown to have a proportional relationship between flow (intraarterial Xe^{133} technique) and arterial blood pressure, indicating absence of autoregulation. This entails a risk of ischemia in even moderate hypotension and, conversely, an increased risk of capillary rupture and germinal layer hemorrhage in

hypertension. Of the 19 infants, 8 had neonatal cerebral blood flow $<20 \text{ ml}/100\text{g}/\text{min}$. Of these three died in the neonatal period. The remaining five were examined repeatedly and were all found to have focal neurologic/neuropsychologic deficits, in contrast to the group with higher neonatal flows who was largely normal.

At 8 years of age single photon emission tomography showed cerebral hypoactivity in watershed areas, in particular in the periventricular region, indicating early ischemic brain damage.

QUANTIFICATION OF ARM-TRACKING PERFORMANCE IN NORMAL AND CLUMSY CHILDREN

Van der Meulen J, Van den Berg R, Oostveen E, Denier van der Gon J, Gooskens RHJM, Gielen C and Willemse J, University of Utrecht, Dept. of Child Neurology and Medical Physics, Utrecht, The Netherlands

We have investigated whether the performance of normal and clumsy children in a tracking task, could be distinguished quantitatively. The term clumsiness refers to a less than normal competence in fine and gross motor control without evidence of neurological disease. Subjects had to track a target moving unpredictably along a straight line with the left or right arm in conditions of variable difficulty (e.g. with and without a preload, with more or less high frequencies in target movement, with and without visual feedback of arm position). Accuracy of tracking performance is expressed by means of mathematical techniques derived from system theory. The mean delay in the tracking task was significantly larger for clumsy (mean=457 ms, SD=65 ms) than for normal children (mean=395 ms, SD=43 ms). Moreover, the clumsy children tended to be less able to track high frequency target movements (mean cut-off frequency at 1.26 Hz (SD=0.26 Hz) and 1.62 Hz (SD=0.50 Hz) for clumsy and normal children, respectively). Also the coherency between target and tracking signal clearly adds to the possibility of discriminating both groups, suggesting more superfluous movements in case of clumsiness. These differences disappeared or became less clear if difficulty of the tracking task was increased. These results appeared to be reproducible. Still, the performance of a part of the clumsy children was indistinguishable from that of normal children.

PROBABILITY OF NEURODEVELOPMENTAL DISORDERS CALCULATED FROM BRAIN ULTRASOUND APPEARANCE IN VERY PRETERM INFANTS

Stewart AL, Reynolds EOR, Hope PL, Hamilton PA, Baudin J, Costello AM de L, Bradford BC, Wyatt JS, University College London, Dept. of Paediatrics, London, England

To determine the ability of ultrasound scanning of the brain to predict early outcome, the neurodevelopmental status of 342 infants born at less than 33w gestation whose brains had been prospectively scanned was assessed at a median corrected age of 52 weeks (range 44-100 weeks). The probabilities and 95% confidence intervals for neurodevelopmental disorders were calculated according to the ultrasound findings. The results showed that the probability of a major or minor disorder was only 10% (6-15%) in infants whose scans did not show periventricular haemorrhage or markedly increased parenchymal echodensities indicating haemorrhagic lesions in the first week