McLellan, D. L., Brice, J. (Wessex Neurological Centre, Southampton University Hospital, Southampton): *A Double Blind Controlled Trial of Chronic Cerebellar Stimulation in Patients with Epilepsy.*

There have been many reports of the success and otherwise of cerebellar stimulation in the treatment of epilepsy. Much of these reports is anecdotal and uncontrolled, and the reports are at times contradictory. Basic animal work suggested that stimulation of the paleocerebellum inhibited epileptic impulses produced by experimental lesions. More recently other work in primates has found contradictory results.

Four years ago we decided to set up a carefully controlled double blind trial of cerebellar stimulation. Twelve patients were selected with stable uncontrolled generalized epilepsy who had had adequate forms of medical treatment and who were totally disabled by repeated epilepsy. An electrode ray of eight silver electrodes was placed over each anterior lobe of the cerebellum under the tentorium through posterior fossa craniectomies. Biopsies of the neocerebellum were taken at the same time. These electrodes were connected subcutaneously to receivers on the front of the chest, and a specially modified Avery external power source was given to the patients. These power sources were regularly serviced, and at intervals unknown to the patients and the clinical assessors the power source was made to provide continuous stimulation alternating between one cerebellar hemisphere and the other, or intermittent on demand stimulation provided by an external button, or no stimulation whatsoever. The patients were carefully assessed over two-month periods during these three separate modes of stimulation. Careful epileptic charts were maintained throughout the period of the trial, and the results were assessed. They were found to be disappointing.

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Thomas, D. G. T., Wise, R. J. S., Beaney, R., Rhodes, C. G., Lammertsma, A., Jones, T. (Gough Cooper Department of Neurological Surgery and MRC Cyclotron Unit, Hammersmith Hospital, London): *Glucose Metabolism in Patients with Cerebral Glioma: Application of Positron Emission Tomography.*

It is generally accepted that glucose metabolism in normal brain is almost exclusively by aerobic respiration. However, it is probable that in cerebral glioma glucose metabolism is by anaerobic pathways.

Patients with cerebral glioma have been examined by positron emission tomography following injection of $^{18}$FDG glucose, as well as during inhalation of $^{15}$O and $^{17}$O. Quantitative values have been obtained for regional glucose metabolism, as well as for regional blood flow and oxygen metabolism.

The results show mismatching of regional glucose and oxygen metabolism in gliomas. The glucose extraction ratio in the tumours was close to normal, while the oxygen extraction ratio was reduced. This pattern of mismatched glucose and oxygen metabolism suggests that anaerobic metabolism is occurring in cerebral glioma in vivo.


There have been reports suggesting that very high doses of Naloxone might reverse clinical stroke, and improve outcome in experimental spinal cord trauma and endotoxic shock. We have used the gerbil model of profound cerebral ischaemia to assess morbidity, mortality, and cerebrovascular parameters. In this model, 60 minutes' bilateral carotid occlusion followed by reperfusion resulted in 50% mortality at 3 hours; 93% were dead at 24 hours. Pretreatment with Naloxone 10 mg/kg I.P. significantly delayed death. In a less severe model (30 minutes carotid occlusion) mortality was again reduced, and survivors had fewer permanent neurological deficits with Naloxone pretreatment. In other animals cerebral blood flow and brain water have been measured. Naloxone had no effect on CBF or cerebrovascular responses in normal gerbils but significantly increased reperfusion blood flow without a change in blood pressure in those subjected to cerebral ischaemia.

It is suggested that Naloxone has a local effect in ischaemic tissue and that the increase local CBF might improve outcome.


The majority of patients with primary brain tumours develop both humoral and cell-mediated antitumour responses, and lymphoreticular infiltrates are frequently observed. In spite of this the prognosis for patients with malignant gliomas is poor. Many patients, although sensitized against their tumours, show