Clinical evaluation of extracellular amino acids in severe head trauma by intracerebral in vivo microdialysis

The article by Kanthan and Shuaib is a timely reminder of the potential importance of clinical applications of intracerebral microdialysis. However, there are several methodological and interpretative shortcomings in their case report. Firstly, the microdialysis probe used (Carnegie CMA-10) is one which is not designed solely for experimental use in animals. It has no human product licence. Conventional techniques for sterilisation of products to be used in humans can damage the components of the probe, alter its microdialysis characteristics, and may even result in the release of toxic substances. There are also examples of the dialysis membrane becoming detached from the probe shaft and being left in situ (R Bullock, personal communication, 1995). These problems, compounded by the lack of a dialysis probe licensed for intracerebral use in humans, are the basis for the lengthy delay in extrapolating this exciting technique from experimental to clinical studies.  

The interpretation offered for the results of Kanthan et al is conjectural. High concentrations of all neurotransmitters analysed were found during a three hour period of microdialysis preceding brain death. Are these consequent to neuronal death or a cause of it? The authors did not describe any in vitro testing of probe recovery rate for the measured compounds on removal of the probe from the patient. Without this vital information and in the absence of any reported correlation between the measured neurotransmitter concentrations with either neuroradiological (CT or MR) findings or neuropathology their results provide information of questionable value. Other important information was lacking from their report—namely, details of probe position in either white or grey matter, the relation of the probe tip to any contusion or other macroscopic intracranial pathology, and the presence of hypoxia, pyrexia or hypotension all of which are “secondary insults”, commonly found in patients with head injury, which aggravate brain damage. This type of information is crucial if we are to explain the large differences previously reported both between and within patients monitored by microdialysis.  

The contribution of excitotoxic neurotransmitters to secondary brain injury is still controversial.  

We agree with Mr Whittle that it is conjectural whether these are the result or the cause of neuronal death. We also agree that the exact role of excitotoxic neurotransmitters to “secondary brain injury” remains unclear. It is precisely with this mind that other extraneous factors were not explored in our case. However for the record, our patient was not hypoxic, hypotensive or febrile during the in vivo microdialysis. Nevertheless, earlier such insults may be contributing factors to the high concentrations noted. This remains a complex issue and we advocate intracerebral in vivo microdialysis as an additional monitoring tool in understanding and unravelling these and other related phenomena of the exact pathogenesis of head trauma.  

Visually induced paroxysmal nausea and vomiting as presenting manifestations of multiple sclerosis

Khan et al reported an interesting patient with visually induced paroxysmal nausea and vomiting as a presenting manifestation of multiple sclerosis.  

Their patient was able to suppress the nausea and vomiting by closing the eyes or avoiding visual motion stimuli. This observation highlights the potential for activation of vestibular and autonomic centres from visual pathways.  

This patient seems to be an extreme case of what in neuro-otology clinics would be called “visual vertigo”. These patients report unsteadiness, dizziness, or “sickish feelings” in environments with confusing visual cues or excessive visual motion (for example, supermarkets, moving crowds, disco lights). A history of vestibular disease, abnormalities in the neuro-otological examination and an added vestibular component, for example, old squints—are common. Khan et al also stress the importance of considering multiple sclerosis in cases like theirs, particularly because the possibility of a psychogenic disorder must be considered. There are no other indicators of neurological disease.  

The same consideration applies to the “dizzy” patients. Symptoms triggered or exacerbated in supermarkets or by people or traffic moving around, not surprisingly can be taken as phobic. Although the name “postural phobic vertigo” has been coined to describe those patients with dizzy or postural symptoms with no vestibular history or findings and a psychogenic background, both the report of Khan et al and our own report emphasise the need for careful investigation in patients with visually induced vertigo, nausea, or postural imbalance.

Persistent vegetative state

The editorial of Kennard and Bilingworth notes, correctly, that the persistent vegetative state syndrome raises immense ethical and social dilemmas. It might be added, further, that the syndrome incites highly litigious questions. Any decision about the appropriateness of scrutiny should properly remain focused on the contentious ethical, social, and legal dilemmas arising from this interdisciplinary matter.  

The extraordinary litigiousness of American society has often been commented on, and the matter of the dilemma of the persistent vegetative state does not depart from this tradition. A body of case law, germane to end of life decision making, has actually grown over the course of the past 20 years or so in the United States, with primal roots extending to the landmark case in 1976 involving Karen Quinlan. More recently, in 1990, the United States Supreme Court decided its first “right to die” case, in a matter involving Nancy Cruzan. In this primordial case, decided by the highest court in the United States, it was held that the state of Missouri may require the continued treatment of a patient in a persistent vegetative state, in the absence of “clear and convincing” proof that the patient authorised explicit the termination of treatment.  

At least in American jurisprudence, the presumption, traditionally, has been that the quality of life in a persistent vegetative state is such that it is the wish to be kept alive; and, in turn, the burden has fallen on those desiring to terminate the treatment of the patient in a persistent
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