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

The article by Kanthar 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 marketed 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 Kanthar 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 neuroradio logical (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 in mind that other extraneous factors were not explored in our case. However for the record, our patient was not hypoxic, hypotensive or hypothermic 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 case illustrates 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” sensations in environments 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 added onsets of excito-toxic syndromes, like 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 psychiatric disorder may be missed. There are no other indicators of neurological disease. The same consideration applies to “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 Lingworth 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. Authority and legal scrutiny should properly remain focused on the contentious ethical, social, and legal dilemmas arising from this interdisciplinary matter.

The extraordinary litigation 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, governed 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. This primordial case, decided by the highest court in the United States, 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 explicitly the termination of treatment. At least in American jurisprudence, the presumption, traditionally, has been that the right to life in a persistent vegetative state is not a 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 state. What then?

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vegetative state, rather than on the shoulders of those wishing to continue such treatment. As pertinent case law continues to evolve in America, however, this pre-sumption, and accompanying burden, may be in danger of disintegrating. In this vein, one commentator has, indeed, espoused the view that it should be presumed that patients in a persistent vegetative state would not want to be kept alive indefinitely; and families objecting to the discontinuation of treatment would have the burden of showing why treatment should be continued after a specified time sufficiently long to establish irreversibility with high certainty. At this time juncture, however, the divisive legal, bioethical, and social issues emanating from cases involving a persistent vegetative state remain far from resolved; and continue to be considered on a piecemeal, or case by case, basis. In several very recent cases involving "medical futility", including one involving Baby K1 and another involving Baby Ryan, American courts have upheld the right of family members to insist on continued treatment over the objections of hospitals and doctors embracing a futility argument. Incidentally, healthcare providers in America who object to providing medical services which they claim are futile run a grave risk of being publicly charged with hypocrisy, or at least gross inconsistency. This is because medical services in the United States traditionally have been regarded as being simply another economic commodity, and have been dispensed based on ability to pay, rather than medical need. To embrace a differing mentality affecting, for example, patients in a persistent vegetative state, would constitute an obvious, and striking, departure from the accepted rule.

British courts have also been wrestling with contentious issues involving patients in a persistent vegetative state. In a case involving a young man named Anthony Bland, in a persistent vegetative state as the result of a disaster at a football ground, the House of Lords sought to clarify the state of the law affecting the care of a patient in such a state. Judges approved the drawing of artificial feeding; and the patient succumbed shortly afterwards. This case represents the first time an English court has riveted attention on the question of circumstances which may lawfully empower a physician to discontinue life sustaining equipment, including hydration and nutrition. Uncertainty lingers, however, regarding the withdrawal of care from patients who are incompetent, but not in a persistent vegetative state.

The weighty ethical, social and legal questions inextricably intertwined with the dilemma of the persistent vegetative state will not be resolved in the absence of embalmed understanding of the attendant issues. The challenge now is to push ahead with robust debate of such issues, in both public and professional forums.

In our opinion, pretarsal injections of BTA into the upper eyelid are sufficient to obtain optimum results in patients with blepharospasm and eyelid freezing. There is usually no reason or need to additionally inject the preseptal or orbital portion of the orbicularis oculi. Our results are similar to those of Aramidieh et al., even though we have not employed an EMG guided approach. It is unlikely that the use of EMG would further enhance the already highly successful response rate.

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NOTICES

Announcement from the British Neuro-psychiatry Association: 1996 summer meeting

The 1996 Summer meeting will be held on 14-16 July at Robinson College, Cambridge. It will include topics on neuro-development, language, and the presentation of short scientific papers and single case videos by members. The Association’s AGM will be held on 16 July.

For further details of these meetings please contact: Sue Garratt, Administrative Assistant, BNPA, 17 Clocktower Mews, London N1 7BB. Telephone/Fax: 0171 226 5949.

For details of membership of the BNPA, which is open to medical practitioners in psychiatry, neurology, and related clinical neurosciences, please contact: Dr Jonathan Bird, Secretary BNPA, Burden Neurological Hospital, Stoke Lane, Stapleton, Bristol, BS16 1QT. Telephone: 01179 701212 ext 2925/2929 or Sue Garratt at the address given above.
Persistent vegetative state.

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