

Matters arising

Cerebral glucose utilisation in Parkinson’s disease

Sir: We were interested in the paper by Rougement et al. in which no alteration of local cerebral glucose utilisation was found between treated and non-treated Parkinsonian patients. However, the same parameter was found to be moderately increased in the basal ganglia of these patients compared to controls. In a recent study one of us demonstrated that low concentrations of dopamine combined with insulin in vitro increased glucose transport in the isolated rat adipocytes. However high concentrations of dopamine combined with high insulin concentrations inhibited glucose transport. If this occurred in vivo, then alterations in dopaminergic function (for example decreased dopaminergic activity) could result in impaired glucose transport in neuronal cells. This would be in agreement with the findings by Lenzi et al. who demonstrated decreased glucose metabolism in the parietal lobe of patients with hemi-Parkinsonism. Moreover Rougement et al. demonstrated slightly increased glucose metabolism in the basal ganglia of Parkinsonian patients. This, we postulate, could result from reduction of dopamine content in these areas with resultant compensatory enhancement of insulin activity in these areas. It is thus possible that increased glucose utilisation in the basal-ganglia of Parkinsonian subjects could reflect impaired dopaminergic activity. The degree of the regional glucose utilisation could thus serve as a marker for loss of dopaminergic activity in these areas.

Dementia is a common associated symptom of Parkinson’s disease. It is possible that by normalising glucose transport into the cortical cells which have been shown to have decreased utilisation in Alzheimer’s type dementia, that the condition can be improved. This could possibly be achieved by administration of insulin, glucose and levodopa.

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References


Sympathetic skin response

Sir: Techniques for evoking the psychogalvanic response and determining conduction velocity along autonomic nerve fibres have long been available but have met with limited interest in electrophysiology. The simplicity of Shahani et al.’s technique of eliciting the sympathetic skin response makes it particularly suitable in the study of the autonomic nervous system during routine EMG sessions. In effect, psychogalvanic responses can be easily induced by any internal or external stimulus of sufficient “novelty”: comparable sympathetic skin response in one hand can be obtained by electrical stimulation of the ipsi- or contralateral wrist, of the glabella and by a sudden auditory burst applied by earphones (fig). Thus, exploring several eliciting modalities of sympathetic skin response may have a localising value. The technique has however some drawbacks, which, if unrecognised, could result...

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Correction

"Mitochondrial malic enzyme in Fried- reich’s ataxia: failure to demonstrate reduced activity in cultured fibroblasts" J Neurol Neurosurg Psychiatry Vol 48 Page 70-74.

Page 71—Methods

Column 2, Line 8 should read ‘‘The cells from a 175 cm² flask were harvested, washed, frozen and thawed once in 20 µl of distilled water and sonicated on ice with two 20 second bursts’’

Fig. Sympathetic skin response evoked in the left hand by (a) stimulation of left median nerve; (b) stimulation of right median nerve; (c) stimulation at glabella; (d) auditory burst.