Matters arising

Cerebral glucose utilisation in Parkinson's disease

Sir: We were interested in the paper by Rougemont 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 vivo 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 Rougemont 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


in falsely negative results. In fact, besides being affected by temperature and having easy habituation, the sympathetic skin response is a reflexly evoked response, having efferent autonomic and diverse afferent sensory pathways, and also subject to “central” control. This implies that absent sympathetic skin responses could conceivably follow not only lesions of the efferent autonomic branches, but also involvement of sensory afferences, some of which are affected in neuropathy, or of central nervous system alone. Central nervous system lesions could also simply change the reflex threshold, modifying the supraspinal facilitation of sympathetic skin response. Moreover, sudomotor activity can be affected by a number of agents, such as anticholinergic and antihypertensive drugs; their effects on sympathetic skin responses, at present unknown, might be relevant.

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Sympathetic skin response

Sir: A simple, reliable and non-invasive test to identify dysfunction in non-myelinated peripheral axons would be a great advance and the investigation of peripheral neuropathy. Shahani et al claim that the measurement of the sympathetic skin response, which they believe to be related to eccrine sweat gland activity, provides such a test. They produce evidence which is interpreted as showing that the sympathetic skin response is absent in axonal neuropathy but preserved in demyelinating neuropathies. Our experience suggests that their conclusions should be treated with caution.

We have measured the sympathetic skin response in patients and normal controls using surface electrodes placed on the palm and dorsum of the hand and on the sole and dorsum of the foot. Recordings were made with a DISA 1500 electromyograph using a band pass of 0.5–2000 Hz. Stimuli were sudden inspiratory gasp, cough, sudden loud noise and electric shock. Sympathetic skin responses were easily recorded from both upper and lower limbs of eight normal subjects in response to all stimuli. Latencies varied slightly from test to test in a single patient but all responses were within the range 1.2–1.5 s when recorded from the hand and 1.8–2.2 s when recorded from the foot. Response amplitude was much more variable, showing a tendency to become smaller with repeated testing. Maximum amplitude ranged from 1.0 to 2.5 mV measured from peak to peak. Four diabetic patients were studied. All had evidence both of autonomic dysfunction, judged by abnormalities of tests based on cardiovascular reflexes, and polyneuropathy, judged by clinical examination and reduced common peroneal motor nerve conduction velocities. In addition, all had symptoms of dysautonomia including the characteristic pattern of sweating abnormality found in diabetics: loss of sweating in the lower limbs and a tendency to hyperhidrosis over the upper trunk and head. A sympathetic skin response of comparable latency and amplitude to that found in normal subjects was elicited from the hands of all patients. Sympathetic skin responses were absent in the feet of two of the patients but were easily elicited from the others.

Symptoms occur late in the natural history of diabetic autonomic neuropathy. All our patients were symptomatic and had abnormalities of cardiovascular reflexes in addition; they should be regarded as having advanced autonomic neuropathy. They also showed clear evidence of polyneuropathy which, in diabetics, is known to be axonal in type. In two out of four patients we were able to elicit sympathetic skin responses indistinguishable from those found in our control subjects. Absence of the sympathetic skin response may well indicate dysfunction of non-myelinated peripheral axons but its presence cannot be taken as evidence that these axons are intact. We think that the lack of sensitivity of this test will limit its value in the investigation of patients with peripheral neuropathy.

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