

Letters

- ⁴ Degoes JD, Verrous J, Bouchareine A, Serdaru M, Barbizet J. Asterixis in focal brain lesions. *Arch Neurol* 1979;**36**:705-7.
- ⁵ Donat JR. Unilateral asterixis due to thalamic hemorrhage. *Neurology (Minneapolis)* 1980;**30**:83-4.
- ⁶ Massey EW, Goodman JC, Stewart C, Brannon WL. Unilateral asterixis: Motor integrative dysfunction in focal vascular disease. *Neurology (Minneapolis)* 1979;**29**:1188-90.
- ⁷ Ericson G, Warren SE, Gribik M. Unilateral asterixis in a dialysis patient. *JAMA* 1978;**240**:671.
- ⁸ Chiappa KH, Ropper AH. Evoked potentials in clinical medicine (second of two parts). *N Engl J Med* 1982;**306**:1205-11.
- ⁹ Plum F, Posner JB. *The Diagnosis of Stupor and Coma*. Philadelphia: FA Davis Company, 1980, 3rd ed. 129.
- ¹⁰ Young RR, Shahani BT. Unilateral asterixis produced by a discrete CNS lesion. *Trans Am Neurol Assoc* 1976;**101**:306-7.

Accepted 10 October 1984

Correction

"Mitochondrial malic enzyme in Friedreich'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 175cm² flask were harvested, washed, frozen and thawed once in 20 µl of distilled water and sonicated on ice with two 20 second bursts"

Matters arising

Cerebral glucose utilisation in Parkinson's disease

Sir: We were interested in the paper by Rougemont *et al*¹ 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 study² 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*³ who demonstrated decreased glucose metabolism in the parietal lobe of patients with hemi-Parkinsonism. Moreover Rougemont *et al*¹ 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.⁴ 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

- ¹ Rougemont D, Baron JC, Collard P, *et al*. Local cerebral glucose utilisation in treated and untreated patients with Parkinson's disease. *J Neurol Neurosurg Psychiatry* 1984;**47**:824-30.

- ² Sandyk R. The effects of dopamine-HCL on glucose transport in isolated rat adipocytes. Thesis presented in partial fulfillment for the degree of MSc (Med) at the University of the Witwatersrand, Johannesburg 1984.
- ³ Lenzi GT, Jones T, Reid JL, Moss S. Regional impairment of cerebral oxidative metabolism in Parkinson's disease. *J Neurol Neurosurg Psychiatry* 1979;**22**:59-62.
- ⁴ Hakim AM, Mathieson G. Dementia in Parkinson's disease: a neuropathologic study. *Neurology (Minneapolis)* 1979;**29**:1209-14.

Sympathetic skin response

Sir: Techniques for evoking the psychogalvanic response and determining conduction velocity along autonomic nerve fibres have long been available¹ but have met with limited interest in electroneurography. The simplicity of Shahani *et al*'s² 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

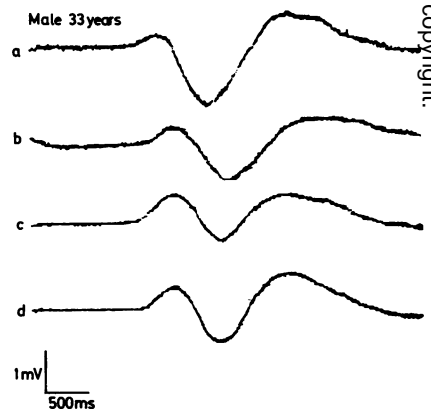


Fig Sympathetic skin response evoked in the left hand by (a) stimulation of left median nerve; (b) stimulation of right median nerve; (c) stimulation at glabella; (d) auditory burst.

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.³ 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 often 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.⁴ 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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References

- 1 Carmichael EA, Honeyman WM, Kolb LC, Stewart WK. Peripheral conduction rate in the sympathetic nervous system of man. *J Physiol (Lond)* 1941;**99**:338-43.
- 2 Shahani BT, Halperin JJ, Boulu P, Cohen J. Sympathetic skin-response—a method of assessing unmyelinated axon dysfunction in peripheral neuropathies. *J Neurol Neurosurg Psychiatry* 1984;**47**:536-42.
- 3 Appenzeller O. *The Autonomic Nervous System*. Elsevier, Amsterdam 1982:92-3.
- 4 Wallin BG, Stjernberg L. Sympathetic activity in man after spinal cord injury. *Brain* 1984;**107**:183-98.

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*¹ 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 between 1.0 and 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,² 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.³ 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.⁴ 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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References

- 1 Shahani BT, Halperin JJ, Boulu P, Cohen J. Sympathetic skin response — a method of assessing unmyelinated axon dysfunction in peripheral neuropathies. *J Neurol Neurosurg Psychiatry* 1984;**47**:536-42.
- 2 Ewing DJ, Clarke BF. Diagnosis and management of diabetic autonomic neuropathy. *Br Med J* 1982;**285**:916-8.
- 3 Clarke BF, Ewing DJ, Campbell IW. Diabetic autonomic neuropathy. *Diabetologia* 1979;**17**:195-212.
- 4 Brown MJ, Greene DA. Diabetic neuropathy: pathophysiology and management. In: Asbury AK, Gilliat RW eds. *Butterworth's International Medical Reviews. Neurology 4. Peripheral Nerve Disorders*. London: Butterworths, 1984.