taurocyamine at a concentration of 200 nmol. A small peak, perhaps urea, is seen after 40 minutes. Quantitative determination of taurocyamine is not without problems and overestimation can occur. Taurocyamine could not be estimated in any CSF of the patients examined. This means that high levels of taurocyamine as described in CSF of uremic patients can be ruled out for patients with neurological and psychiatric diseases.

References

6 The International Classification of Diseases. 9th rev. division. Department of Health and Human Services Washington DC, 1980, publication No (PMS) 80-1260 US.

Familial carpal and tarsal tunnel syndrome

Sir: The familial occurrence of nerve entrapment at the carpal and tarsal tunnel seems to have been rarely if ever described. Pedigrees of families liable to pressure palsies have included evidence of subclinical abnormalities under the carpal tunnel but these patients had symptomatic palsies predominantly of the sciatic and ulnar nerves. We have recently studied a patient whose family members show a predilection to either the carpal tunnel syndrome or the tarsal tunnel syndrome.

A 61-year-old housewife was referred to one of us (MLS) with a history of pain in the foot on both sides characteristic of the tarsal tunnel syndrome. She was otherwise healthy, of average build without notably abnormal wrists or ankles. Electrophysiological study confirmed local abnormality, and significant asymmetry of the distal latencies to flexor hallucis and abductor quinti minimi. A sural sensory action potential was normal in amplitude and latency. The patient had had successful surgery for bilateral carpal tunnel syndrome. She had four sisters: one was asymptomatic. An elder sister had been treated for unilateral carpal tunnel syndrome, as had one younger sister. The youngest in the family, a woman aged 43 years, had had treatment for a unilateral carpal tunnel syndrome and a unilateral tarsal tunnel syndrome. The patient's mother had just been diagnosed as having the carpal tunnel syndrome. Unfortunately these other members of her family do not live in the UK and could not be examined.

This family show a striking predisposition to entrapment at the carpal tunnel and at the tarsal tunnel. There was no obvious abnormality of the wrists or ankles of the patient examined, and no stigmata of any connective tissue or skeletal disease. The other family members are not thought to be abnormal in any other way. Nevertheless it seems likely that the cause lies in the geometry of the carpal and tarsal tunnels, rather than undue nerve sensitivity to pressure or stretch since none of the family members gave a history of other nerve pressure palsies and the patient studied had no evidence of a diffuse neuropathy. Computed tomography of the wrists shows that patients with the carpal tunnel syndrome have a smaller than normal space. The occasional coincidence of the carpal tunnel syndrome and tarsal tunnel syndrome has been reported before, but not its familial incidence.

CP Fowler*
MJJ Harrison†
ML Snaithe†
Department of Neurological Studies.*
Department of Rheumatology and Rehabilitation,† The Middlesex and University College Hospitals, London, UK

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Table Diagnoses and number of cases

<table>
<thead>
<tr>
<th>Neurological patients</th>
<th>Psychiatric patients</th>
<th>Number</th>
<th>Diagnosis</th>
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<tr>
<td>Sleep disorders</td>
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<td>Multiple sclerosis</td>
<td>Schizophrenic disorders</td>
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<td>Central nervous system neoplasms</td>
<td>Paranoid</td>
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<td>Meningitis</td>
<td>Brief reactive psychosis</td>
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<tr>
<td>Viral encephalitis</td>
<td>Neurones</td>
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<td>Extrapyramidal diseases</td>
<td>Adjustment disorders</td>
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<td>Infantile spinal muscle atrophy</td>
<td>Organic mental disorders</td>
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<td>Peripheral nervous system diseases</td>
<td>Substance use disorders</td>
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<td>Other psychiatric disorders</td>
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<td>Total</td>
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Fig Pedigree of family. Square = male, circle = female, black symbol = propositus, CTS = carpal tunnel syndrome, TTS = tarsal tunnel syndrome, numbers = age in years.
The controls of the time age examined according to that surface skin electrodes. The blink reflex of the group of normal subjects had a mean latency of 49.7 ± 2.2 ms (range 47–53 ms). The visual blink reflex was absent in one control. The mean latencies of the patient and the control group differed significantly (p < 0.001).

Our study demonstrates that the latency of the visual blink reflex of patients with senile dementia is markedly increased. Possibly, the alteration of the visual blink reflex in senile dementia is due to lesions or functional disturbances at the level of the brainstem, which are known to occur in Alzheimer type dementia. In our view these findings imply that further studies on the visual blink reflex in dementia are warranted.

References


Alteration of the visual blink reflex in patients with dementia

Sir: The clinical diagnosis of senile dementia of the Alzheimer type and multi-infarct dementia can be very difficult. Neurophysiological tests are not specific. In a recent study, the EEG and visual evoked potentials were reported to be normal in respectively 34% and 76% of patients with Alzheimer type dementia. Therefore, the development of additional sensitive tests which could discriminate dementia from other disorders, would be of great value.

We have found a simple neurophysiological test, the visual blink reflex, to be abnormal in patients with Alzheimer type dementia or multi-infarct dementia. The visual blink reflex consists of a reflex contraction of the eyelids in response to a bright light which is flashed in front of the eyes of the subject. Normal subjects usually show a visual blink reflex with a constant latency of approximately 50 ms, although the visual blink reflex can be absent in up to 12% of normals. The visual blink reflex is a subcortical reflex but the exact pathway in the brainstem is unknown.

Fifteen patients with senile dementia (Alzheimer type dementia or multi-infarct dementia) (mean age 77.2 ± 7.7 yr) were examined according to the method described by Malin. The EMG activity of the orbicularis oculi muscles was recorded with surface skin electrodes. Patients were not informed that the blink reflex was measured in order to avoid voluntary blinking. In each subject we determined the average latency time of the visual blink reflex from 10 trials. The controls were 13 normal aged persons (mean age 70.5 ± 11 yr).

Twelve out of 15 patients showed a symmetrical visual blink reflex with a mean latency for the group of 104.3 ± 30.0 ms (range 65–158 ms). In three patients no visual blink reflex could be elicited. The visual blink reflex of the group of normal subjects had a mean latency of 49.7 ± 2.2 ms (range 47–53 ms). The visual blink reflex was absent in one control. The mean latencies of the patient and the control group differed significantly (p < 0.001).

Our study demonstrates that the latency of the visual blink reflex of patients with senile dementia is markedly increased. Possibly, the alteration of the visual blink reflex in senile dementia is due to lesions or functional disturbances at the level of the brainstem, which are known to occur in Alzheimer type dementia. In our view these findings imply that further studies on the visual blink reflex in dementia are warranted.

References


Isolated lower motoneuron involvement following radiotherapy

Sir: Progressive transverse myelopathy which follows, after a characteristic latent period, the radiation of tumours in the vicinity of the spinal cord is a well-recognised sequel of radiotherapy. Another myelopathic post-radiation syndrome is characterised by a slowly evolving amyotrophy with preserved and the peroneal, extensor muscles without sphincter disturbances or sensory impairment, and is presumed to be the result of selective damage to the anterior horn cells in the lumbo-sacral spinal cord.2–4

Our case, a 64-year-old male, had a right-sided pachyochromocytoma, encasing the vena cava and the celiac axis. Surgical removal was only partial, so he received 5800 rads to the affected side (December 1983). Seven months after the treatment, he suffered painful cramps in the legs for two weeks, followed by progressive bilateral leg weakness. There were no sphincter abnormalities or sensory symptoms. The course of illness was one of initial worsening followed by a stabilised state until our observation in May 1985. Neurological examination showed diffuse lower limb wasting, more marked in gluteal muscles, and moderate weakness of the peroneal, extensors of the feet and toes and gluteal muscles. Fasciculations were present in the calves and gluteal muscles. Tendon reflexes were absent in the legs and plantar responses were flexor. Sensation was intact. Laboratory evaluations were normal as well as radiographs of the thoracic, lumbar and sacral spine. Magnetic resonance imaging of the thoracolumbar spinal cord was normal, and there was no evidence of intraspinal metastasis. Quantitative EMG3 of the left vastus medialis, peroneus longus, gastrocnemius medialis and lateralis and gluteus maximus showed denervation activity with fasciculations and loss of motor units with pronounced signs of reinnervation in all muscles. Motor conduction velocity along the left peroneal and posterior tibial nerves was reduced by 24% and 14%, and distal latencies were prolonged by 51% and 40% respectively. Amplitudes of motor responses were normal. Amplitude and shape of SAPs sensory thresholds and sensory conduction velocity along the lower peroneal and sural nerves were normal. F-wave latencies were prolonged by 22% and 10% along the left peroneal and posterior tibial nerves respectively; the left H reflex had a prolonged latency, a markedly polyphasic shape, and was constantly associated with late waves at 72 ms. SEPs were obtained in the upper and lower limbs on the...
Familial carpal and tarsal tunnel syndrome.

C P Fowler, M J Harrison and M L Snaith

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