References

[1] Earl CJ, Fullerton PM, Wakefield GS, Schutta HS. Hereditary Neuropathy with liability to pressure palsies. *Q J Med* 1964;**33**:481–98.
[2] Oh SJ, Sarala PK, Kuba T, Elmore RS. Tarsal tunnel syndrome: electrophysiological study. *Ann Neurol* 1979;**5**:327–30.
[3] Fu R, Delisa JA, Kraft GH. Motor nerve latencies through the tarsal tunnel in normal adult subjects: standard determinations corrected for temperature and distance. *Arch Phys Med Rehab* 1980;**61**:243–8.
[4] Dekel S, Coates R. Primary carpal stenosis as a cause of "idiopathic" carpal tunnel syndrome. *Lancet* 1979;**i**:1024.
[5] Lam SJS. Tarsal tunnel syndrome. *J Bone Jt Surg* 1967;**49B**:87–92.

Accepted 2 December 1985

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.[1 2] 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.[3] 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.[4] The visual blink reflex is a subcortical reflex but the exact pathway in the brainstem is unknown.[5 6]

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.[4] 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 47–53·3 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.[7] In our view these findings imply that further studies on the visual blink reflex in dementia are warranted.

DLJ TAVY
TCAM VAN WOERKOM
HHE MORRÉ
JPJ SLAETS
Municipal Hospitals, The Hague, Departments of Clinical Neurophysiology and Clinical Neurology and Psychiatric Centre Rosenburg, The Hague, Departments of Neuropsychiatry and Psychogeriatrics 2545 CH, The Netherlands

References

[1] Ron MA, Toone BK, Garalda ME, Lishman WA. Diagnostic accuracy in presenile dementia. *Br J Psychiatry* 1979;**134**:161–8.
[2] Caine ED. Pseudodementia. *Arch Gen Psychiatry* 1981;**38**:1359–64.
[3] Visser SL, Van Tilburg W, Hooijer C, Jonker C, De Rijke W. Visual evoked potentials (VEPs) in senile dementia (Alzheimer type) and in non organic behavioural disorders in the elderly; comparison with EEG parameters. *Electroencephalologr Clin Neurophysiol* 1985;**60**: 115–21.
[4] Malin JP. Der optisch evozierte orbicularis oculi und reflex: methodik, normalwerte, habituation. *Z EEG-EMG* 1982;**13**:101–7.
[5] Brierly JB, Graham DI, Adam JH, Simpson JA. Neocortical death after cardiac arrest. *Lancet* 1971;**2**:560–5.
[6] Tavy DLJ, Van Woerkom TCAM, Bots GTAM, Endtz LJ. Persistence of the blink reflex to sudden illumination in a comatose patient. *Arch Neurol* 1984;**41**:323–4.
[7] Terry RD, Katzman R. Senile dementia of the Alzheimer type. *Ann Neurol* 1983;**14**:497–506.

Accepted 10 May 1985

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.[1] Another myelopathic post-radiation syndrome is characterised by a slowly evolving amyotrophy with paresis and areflexia of legs 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 pheochromocytoma, 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 peronei, extensors of the feet and toes and gluteal muscles. Fasciculations were present in the calf 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 EMG[5] 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 left peroneal and sural nerves were normal. H-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

left, with stimulation of the median (2 series of 512 addresses) and peroneal (3 series of 2048 addresses) nerves and recording sites at Erb's point, Cv_7, C_3', C_4', and L_3, T_{12}, T_6, Fpz respectively. In all cases the amplitude and shape of the SEPs as well as their latencies and peripheral and central conduction velocities (in particular along the segments: Knee-L_3; L_3-T_{12}; L_3-T_6, T_3-T_{12}) were normal. The electrophysiological findings thus indicated chronic denervation in the leg muscles, with slightly reduced motor conduction velocity probably due to loss of fast motor fibres. The completely normal sensory pathways, both distal and proximal to the dorsal ganglia and intraspinal, and the marked signs of reinnervation with fasciculation, implied an anterior horn cell lesion.

There has been controversy as to the exact location of the lesion in clinically similar cases. Some authors[6 7] have suggested a damage to the lumbosacral plexus on the basis of retroperitoneal fibrosis on necroscopy. However their patients also had marked sensory symptoms. Others[8-10] have reported cases with exclusively motor signs suggesting that the probable site of damage lies either in the anterior horn cells of the lower spinal cord and/or in the lumbar and sacral motor roots. Clinically our case had bilateral exclusively motor signs and detailed electrophysiological studies demonstrated only chronic denervation and reinnervation with normal sensory pathways. In our opinion this observation confirms that isolated lower motor neuron involvement may occur after radiotherapy.

P DE CAROLIS
P MONTAGNA
M CIPULLI
A BALDRATI
R D'ALESSANDRO
T SACQUEGNA
Institute of Neurology,
University of Bologna, Italy
Via U. Foscolo 7
40123 Bologna, Italy

References

[1] Palmer JJ. Radiation myelopathy. *Brain* 1972; **95**:109–22.
[2] Greenfield MM, Stark FM. Post-irradiation neuropathy. *Am J Roentg* 1948;**60**:617–22.
[3] Maier JC, Perry RH, Saylor W, Sulak MH. Radiation myelitis of the dorsolumbar spinal cord. *Radiology* 1969;**93**:153–60.
[4] Laguany A, Aupy M, Aupy P, Ferrer X, Henry P, Julian J. Syndrome de la corne antérieure post-radiothérapique. *Rev Neurol (Paris)* 1985;**141**:222–7.
[5] Rosenfalck P, Rosenfalck A. Electromyography. Sensory and motor conduction findings in normal subjects. Copenhagen, Rigshospitalit, 1975.
[6] Klana M. Radiogenic peripheral neuropathies following telecobalt irradiation in abdominal cavity. *Radiobiol Radiother (Berl)* 1974;**15(4)**: 459–64.
[7] Ashenhurst EM, Quartey GRE, Starreveld A. Lumb-sacral radiculopathy induced by radiation. *Can J Neurol Sci* 1977;**4**:259–63.
[8] Sadowsky CH, Sachs E, Ochoa J. Post-radiation motor neuron syndrome. *Arch Neurol* 1976; **33**:786–7.
[9] Kristensen O, Melgard B, Schiødt AV. Radiation myelopathy of the lumbo-sacral cord. *Acta Neurol Scand* 1977;**56**:217–22.
[10] Horowitz SL, Stewart JD. Lower motor neuron syndrome following radiotherapy. *Can J Neurol Sci* 1983;**10**:56–8.

Accepted 18 November 1985.

Notice

The Volvo awards for low back pain research 1987

The Volvo Company of Göteborg, Sweden, this year sponsors three prizes, now increased to US$7.000 each. Awards will be made competitively on the basis of scientific merit in the following three areas: (1) Clinical studies, (2) Bioengineering studies, (3) Studies in other basic science areas.

Enquiries should be addressed to Professor Alf L Nachemson, Department of Orthopaedic Surgery, Sahlgren Hospital, S-413 45 Göteborg, Sweden.

Matters arising

Phaeochromocytoma and intracranial aneurysm

Sir: I read the interesting letter by Jha and Lye published recently in your journal.[1] They indicated that they were not aware of any previous reports on an association between intracranial aneurysms and phaeochromocytomas. I would like to supplement the references in their letter with two articles in which an association between aneurysm and this tumor were discovered.[2 3] These two references were uncovered previously while searching the literature for a chapter on conditions associated with intracranial aneurysms.[4]

JL FOX
Division of Neurosurgery,
Georgetown University Hospital,
3800 Reservoir Rd, NW,
Washington DC 20007 USA

References

[1] Jha AN, Lye RH. Transient hemiparesis: a cautionary tale: coexistence of phaeochromocytoma and intracranial aneurysm [Letter]. *J Neurol Neurosurg Psychiatry* 1985;**48**:188–9.
[2] Browne EF, Meyer JS. Pheochromocytoma with rupture of an intracranial aneurysm. Report of a case. *N Engl J Med* 1952;**247**:671–2.
[3] Gibbons JRP, Brookes JPCM. Phaeochromocytoma associated with multiple neurofibromatosis and aneurysm of the circle of Willis. *Br J Clin Pract* 1967;**21**:360–2.
[4] Fox JL. *Intracranial aneurysms*. Springer-Verlag, New York, 1983:396–417.

Scotland: the birthplace of surgical neurology

Sir: The temporal coincidence of two important events, the death of Dr Irving S Cooper in October 1985 and the simultaneous appearance of the fine historical review of neurosurgery by Dr Paul C Bucy,[1] has prompted the following comments. To the list of distinguished pioneers in world neurosurgery, so eloquently outlined by Dr Bucy, I wish to append the name of Irving Cooper. As CP Snow said, Cooper was "one of the most remarkable men alive" and "one of the great brain surgeons of the world."[2] He developed new techniques to alleviate positive symptoms of movement disorders without producing any negative symptoms. This was something which the experts continued to claim was an impossibility even after Cooper had demonstrated his results in