LETTERS TO THE EDITOR

Trapezius muscle atrophy after whiplash injury: accessory nerve or cervical plexus lesion?

We have observed the rare occurrence of trapezius muscle atrophy after whiplash injury. Only one similar case has been reported previously.¹

A 24 year old woman was waiting at traffic lights in her car and was changing a cassette with her right arm outstretched when another car ran into the back of hers. She was not wearing a safety-belt. Immediately she felt pain and tingling on the back of the neck but no other symptoms. Later that day she developed nausea and dizziness. The same day a neurological examination was normal. There was no radiological evidence of fracture or dislocation of the cervical spine. A soft collar did not control the neck ache.

One month later, she noticed a lowering of her right shoulder. Six months later examination revealed weakness and atrophy of the right trapezius: with her arms at her side, the right shoulder drooped and the scapula was slightly winged and laterally displaced. Abduction of the arms made these defects worse and increased the prominence of the rhomboid muscles. Muscle tests showed a global involvement of the right trapezius, more marked in the middle and lower parts. The sternomastoid and the other shoulder girdle muscles were not affected. Slight hypoaesthesia for touch and pinprick was appreciable in the region of the supraclavicular branches of the cervical plexus. Thoracic fluoroscopy showed normal diaphragm motility. CT and MRI of the cervical regions were normal. EMG examination revealed total denervation of the middle and lower parts of the right trapezius and a reduced recruitment of low amplitude, and polyphasic potentials in the upper part at full effort. There was no spontaneous activity. Conduction studies along the right accessory nerve² showed a slight delay of the motor action potential derived from the upper part of the trapezius (4.2 ms; normal range 2.1-3.7 ms), whereas no motor responses could be evoked in the middle and lower parts. There was no clinical improvement the following year.

The accessory nerve, after supplying the sternomastoid muscle with the contributions from C2 or C2-C3, often divides and forms a plexus with the fibres from C3-C4, or C4 alone, before entering the deep surface of the trapezius.³ A complete lesion of the accessory nerve in the posterior triangle, frequently observed in neck surgery, results in a partial denervation of the whole muscle, the upper trapezius being clinically more affected.¹ The third and fourth cervical roots, in addition to containing the afferent fibres, provide motor innervation mostly to the lower trapezius through the cervical plexus.⁴ There is, however, some variability as occasionally the whole trapezius is supplied by these roots.⁵

In this case, the clinical and electrophysiological findings are consistent with a prominent lesion of the radicular contribu-

tions. The unusual position of our patient at the time of the collision, implying torsional components in addition to the usual hyperextension-flexion movement of the neck, may explain the rarity of this condition. The combination of these rapid movements may have produced a stretch-induced lesion of the cervical plexus fibres, which suggests a poor prognosis.

L TOSI
G ZANETTE
Servizio di Neurologia,
Ospedale "Sacro Cuore" Negrar,
Verona, Italy

- 1 Mumenthaler M, Schliak H. *Lesionen peripherer nerven. Diagnostik und therapie*. Stuttgart-New York: Thieme, 1987:214.
- 2 Petrera JE, Trojaborg W. Conduction studies along the accessory nerve and follow-up of patients with trapezius palsy. *J Neurol Neurosurg Psychiatry* 1984;47:630-6.
- 3 Soo KC, Hamlyn PJ, Pegington J, Westbury G. Anatomy of the accessory nerve and its cervical contributions in the neck. *Head Neck Surg* 1986;9:111-5.
- 4 Coleman CC, Walker JC. Techniques of anastomosis of the branches of the facial nerve with spinal accessory for facial paralysis. *Ann Surg* 1950;131:960.
- 5 Fharer H, Ludin HP, Mumenthaler M, Neiger M. The innervation of the trapezius muscle. An electrophysiological study. *J Neurol* 1974;207:183-8.

Cysticercus immunoblot assay in Indian patients with single small enhancing CT lesions

Single small enhancing lesions (SEL) are frequently seen on CT scans of Indian patients with seizures.¹ Their aetiology has been disputed for a number of years. Recently it has been shown that most if not all these lesions are caused by cysticercosis.^{2,3} An excision biopsy of the lesion following CT guided stereotaxic localisation is the preferred method of obtaining a definitive pathological diagnosis. Since this procedure carries some morbidity and the disease process in most patients is benign and self-limiting, biopsy may not be desirable in the majority of individuals with SELs. Attempts have been made to use a serological test in the diagnosis of these lesions, the ELISA test being the one most frequently used.³ In early 1989, the Centers for Disease Control (CDC), Atlanta, reported an improved assay for detecting anti-cysticercus antibodies in the serum or CSF of patients with cysticercosis.⁴ The test uses lentil-lectin affinity purified antigens in an immunoblot assay. We have assessed its use in the diagnosis of patients with SEL.

Serum samples were drawn from 36 Indian patients whose clinical data are shown in the table. The lyophilised samples were analysed at CDC using the immunoblot assay and the results are shown in the table. Of the 18

Table Clinical data of patients and results of the assay

Diagnosis	Number	Serology	
		Positive	Negative
Healthy volunteer	5	0	5
SEL*	18	1	17
Healed SEL†	5	0	5
Multiple			
neurocysticercosis	7	4	3
Tuberculoma	1	0	1
Total	36	5	31

*SEL = Single, small, enhancing CT lesion.

†Healed SEL = "Disappeared" or calcified.

patients with SEL only one had a positive result. Two patients with SEL had an excision biopsy and in both the biopsy showed a cysticercus granuloma. The other four positive results were in patients with multiple lesions. There were three patients with multiple lesions who had a negative result.

Preliminary evaluation of the CDC immunoblot assay had shown very high sensitivity.⁴ One or more of seven major glycoprotein bands was recognised in serum specimens of 97% of 108 parasitologically confirmed cases of neurocysticercosis. The serum of 376 patients with heterologous infections did not show any bands (specificity = 100%). Subsequent prospective experience with the assay has shown that the number of intraparenchymal cysticerci is the most important factor determining test sensitivity. Seropositivity in patients with single, active lesions has been 71% and 36% in serum and CSF, respectively (M Wilson, unpublished data). One reason for the low sensitivity of this test in patients with single lesions could be the poor systemic immune response. It is possible that the serum antibody levels in these patients are below the threshold required to obtain a positive result with the test.

The serological results reported here on seizure patients with CT abnormalities and other characteristics identical to those previously identified as neurocysticercosis² demonstrate that the immunoblot assay is of little use for diagnostic confirmation of SEL. A negative result in a patient with SEL does not exclude a diagnosis of cysticercosis.

In Indian patients with seizures and a well defined, rounded SEL a confident diagnosis of cysticercosis can be made. However, it is in patients with a lobulated, ill-defined lesion larger than 10 mm that the diagnosis becomes doubtful. Even in the latter group anticonvulsants alone can be given and a CT scan obtained after eight to 12 weeks to look for a change in the size or configuration of the lesion and the surrounding oedema. An early diagnosis is the ideal and our results indicate that serological tests do not provide an answer in the majority of patients. Thus in patients with negative serology and doubtful diagnosis, excision biopsy following CT guided stereotaxic localisation seems to be the only method of arriving at a diagnosis.

We thank Drs Anna Oommen and V Sivakumar for their help in the collection and processing of the sera.

VEDANTAM RAJSHEKHAR*
Department of Neurological Sciences,
Christian Medical College and Hospital,
Vellore, India
MARIANNA WILSON
PETER M SCHANTZ
Parasitic Diseases Branch,
Parasitic Diseases Division
Center for Infectious Diseases,
Centers for Disease Control,
Atlanta, Georgia 30333, USA

*Present address: Section of Neurosurgery, Dartmouth-Hitchcock Medical Center, Hanover NH 03756, USA.

- 1 Wadia RS, Makhale CN, Kelkar AV, et al. Focal epilepsy in India with special reference to lesions showing ring or disc-like enhancement on contrast computed tomography. *J Neurol Neurosurg Psychiatry* 1987;50:1298-301.
- 2 Chandy MJ, Rajshekhar V, Prakash S, et al. Cysticercosis causing single small CT lesions in Indian patients with seizures. *Lancet* 1989;i:390-1.
- 3 Ahuja GK, Behari M, Prasad K, Goulatia RK, Jaikhani BL. Disappearing CT lesions in epilepsy: is tuberculosis or cysticercosis the

cause? *J Neurol Neurosurg Psychiatry* 1989;52:915-6.

- 4 Tsang VCW, Brand JA, Boyer AE. An enzyme-linked immunotransfer blot assay and glycoprotein antigens for diagnosing human cysticercosis (*Taenia solium*). *Journal of Infectious Diseases* 1989;159:50-9.

Chronic hemidystonia following acute dystonic reaction to thiethylperazine

Thiethylperazine is a phenothiazine neuroleptic drug that blocks postsynaptic dopamine D2 receptors and is extensively used for the treatment of vertiginous syndromes.¹ Its extrapyramidal side effects include acute and tardive dystonic reactions and a Parkinsonian syndrome.² The acute reactions are usually self-limiting and brief and nearly all respond to anticholinergic therapy.² We report an unusual case of a chronic hemidystonia which began immediately after treatment with thiethylperazine.

A 47 year old man, with no previous personal or family history of neurological illness, was evaluated in December 1988 in our unit because of involuntary movements and postures of his right limbs. When he was aged 40 he developed a vertiginous syndrome (retrospectively diagnosed as "vestibular neuritis") and was treated with thiethylperazine 6.5 mg three times daily. When he was given the third dose (total dose = 19.5 mg) there was a clinical picture of generalised dystonic posturing including oculogyric crises, that was alleviated with biperiden 5 mg intravenously. Twenty four hours later, however, he developed dystonic postures and movements of the right limbs, neck and trunk that persisted until the day of his admission. Apart from his movement disorder, the general and neurological examination were normal. A blood cell count, biochemistry, urinalysis and copper studies in plasma and urine, were all normal, and serological tests for syphilis were negative. EEG, cranial CT and MRI showed no abnormalities. The dystonic movements improved moderately with biperiden 8 mg/day, although he has a persistent action dystonia of his right limbs, which is more noticeable during walking.

Dystonia has been classified according to distribution as focal, multifocal, segmental, or generalised, and when its distribution involves the ipsilateral arm, leg, and face, the term "hemidystonia" is applied.³ The presentation of a dystonic syndrome as hemidystonia has an important aetiological significance because in most cases there is CT evidence of contralateral basal ganglia damage.⁴⁻⁶ The lack of evidence of a structural lesion in the basal ganglia in our patient on CT and MRI, together with the onset of this clinical picture immediately after thiethylperazine exposure, implies that hemidystonia may be caused or at least unmasked (in a subject possibly already predisposed to develop idiopathic dystonia) by phenothiazine.

Acute dystonic reactions after exposure to phenothiazines or other neuroleptic agents usually disappear after withdrawal of the relevant drug and/or anticholinergic therapy.^{2,3} Nevertheless, some cases have been reported in the literature of prolonged or chronic dystonia.⁷⁻¹⁰ The series of tardive dystonia by Burke *et al*¹¹ includes 42 patients with dystonia after exposure to antipsychotic drugs. Dystonia developed after a mean interval of exposure of 3.7 years.

In none of these cases nor in recent series of tardive dystonia,^{11,12} was the distribution ever hemidystonic. Our case therefore appears to be the first report of hemidystonia occurring as a direct or indirect side effect of a neuroleptic drug.

FELIX JAVIER JIMENEZ-JIMENEZ
ANTONIO VAZQUEZ
PEDRO GARCIA-RUIZ
DANIEL GARCIA-URRA
EDUARDO VARELA DE SEIJAS
Movement Disorders Unit,
Service of Neurology,
Hospital Universitario San Carlos,
Madrid, Spain

Correspondence to: Dr Jiménez-Jiménez, C/ Corregidor José de Pasamonte, 24, 30 D, 28030 Madrid, Spain

- 1 Oosterveld WJ. Vertigo: current concepts in management. *Drugs* 1985;30:275-83.
- 2 Weiner WJ, Lang AE. Drug-induced movement disorders (not including tardive dyskinesia). In: Weiner WJ, Lang AE, eds. Mount Kisco, New York: Futura, 1989:599-644.
- 3 Fahn S. Concept and classification of dystonia. *Adv Neurol* 1988;50:1-8.
- 4 Pettigrew LC, Jankovic J. Hemidystonia: a report of 22 patients and a review of the literature. *J Neurol Neurosurg Psychiatry* 1985;48:650-7.
- 5 Marsden CD, Obeso JA, Zarranz JJ, Lang AE. The anatomical basis of symptomatic hemidystonia. *Brain* 1985;108:463-83.
- 6 Marsden CD. Investigation of dystonia. *Adv Neurol* 1988;50:35-44.
- 7 Walsh TD. Chronic dysarthria and metoclopramide. *Ann Neurol* 1982;11:545.
- 8 Dabbous IA, Bergman AB. Neurologic damage associated with phenothiazine. *Am J Dis Child* 1966;111:291-6.
- 9 Angle CR, McIntyre MS. Persistent dystonia in a brain-damaged child after ingestion of phenothiazine. *J Pediatr* 1968;73:124-6.
- 10 Leopold NA. Prolonged metoclopramide-induced dyskinetic reaction. *Neurology* 1984;34:238-9.
- 11 Burke RE, Fahn S, Jankovic J, *et al*. Tardive dystonia: late-onset and persistent dystonia caused by antipsychotic drugs. *Neurology* 1982;32:1:335-46.
- 12 Kang UJ, Burke RE, Fahn S. Tardive dystonia. *Adv Neurol* 1988;50:415-29.

Bilateral metastases in the cerebellopontine angle

Only 0.2% of the pontocerebellar angle (PCA) tumours are metastases.¹ To our knowledge we report the first case of bilateral metastasis.

In 1974, a 64 year old woman presented with a malignant melanoma of the left leg which was treated by exeresis and chemotherapy. An inguinal adenopathy in 1976 followed by a subcutaneous metastasis on the right deltoid region six years later were treated surgically.

From June to July 1987 the patient experienced a right-sided hearing loss, an unsteady gait with vertigo, a swallowing impairment and dysphonia. The brain CT scan and the CSF analysis were unremarkable. The patient was admitted to our hospital on 20 July because of a right facial palsy and left hypoaacusia.

On the right side, there was involvement of the VII-IXth cranial nerves. On the left, the Vth, VIIth and VIIIth cranial nerves were involved slightly. The patient appeared ill but on skin examination there was no evidence of malignant melanoma.

The CT scan (fig a) showed two hyperdense lesions which on MRI (fig b) were isointense during T1 and hyperintense during T2. The first image, which was round, was in the right PCA and displaced the brain stem and the cerebellar without invading

Figure 1A CT scan with contrast: two hyperdense lesions of the right and the left CPA; B: MRI (T2 weighted): the two lesions are hyperintense.

them; the second, which was oval and smaller in size, was in the left PCA, without mass effect.

On 10 August 1987 a soft, haemorrhagic, yellowish, self contained (2 cm x 3 cm) tumour was found in the right PCA, disrupting the last cranial nerves, forcing back the brain stem on the median line, and spreading onto the jugular foramen. An identical tumour on the left (1.5 cm diameter) did not press on the brain stem but spread along the last cranial nerves without disrupting them.

The histopathological examination showed that they were metastases of an undifferentiated cancer; cells of epithelial type were associated with long cells which looked like sarcoma. On electron microscopy, there was no melanosoma, but small stick inclusions in the ergastoplasm cisterns (75% of cells), of variable orientation, often crossed and made from straight microtubules, suggesting a melanoma origin.

The patient died in October 1987. A necropsy examination could not be performed.

Cornil² reported the first case of a metastase developed in the PCA; the primary lesion was an oropharynx epithelioma. Since then, most of the unusual published cases are isolated ones, originating from histologically differing types such as: lung, breast, oropharynx carcinoma,¹ carcinoma of probably colon origin,³ histiocytic malignant lymphoma,⁴ malignant prolactinoma,⁵ malignant fibrous xanthoma.⁶ No case of melanoma was reported.

In our case, there were several inclusions of straight microtubules in the ergastoplasm cisterns on histopathological examination, similar to those observed in 6% of cases of