not common.²⁻⁶ Early in the course of the disease it is rare to find any laryngeal abnormality. The voice is sometimes flat, expressionless and monotonous with tendency to improve as talking continues. On examination at this stage of the disease no abnormality can be detected.^{2,3}

Our case presented with unilateral vocal cord paralysis. To the best of our knowledge such a clinical picture has never been reported before. The pronounced asymmetry with left pharyngeal weakness and unilateral atrophy of the left neck and shoulder, is also unusual in a disease where the symmetry of appearance is one of the typical features. Early diagnosis of myotonic dystrophy is important in the light of genetic counselling.

N GILADI
I BAZAK
D HAREL

Department of Neurology,
Lady Davis Carmel Hospital,
7 Michal Street,
34362 Haifa,
Israel.

References

- 1 Harper PS. *Myotonic Muscular Dystrophy*. Philadelphia: WB Saunders, 1979.
- 2 Welsh JD, Haase GR, Bynum TE. Myotonic muscular dystrophy systemic manifestation. *Arch Int Med* 1964;114:669-79.
- 3 Spector GJ. Neurologic disease of the larynx. In: Ballenger JJ, ed. *Disease of the Nose, Throat and Ear*. Philadelphia: Lea and Febiger, 1985:544-5.
- 4 Mesolella C, Pezzarossa G. Pharyngolaryngeal manifestation in myasthenia and myotonic dystrophy. *Arch Ital Laryngol* 1968;Supp 1:1-15.
- 5 Carceni T, Zibordi F. Electromyography in Otolaryngology. *Arch Ital Otol* 1966;Supp 1:48.
- 6 Piquet J, Teprracol J. Laryngeal paralysis of vocal cords in Steinert disease. *Les Maladies du Larynx*. Paris: Masson, ed. 1958.

Accepted 1 July 1988

Sixth cranial nerve palsy complicating psittacosis

Sir: Neurological complications in psittacosis are uncommon. We report the occurrence of an acute VIth cranial nerve palsy complicating psittacosis.

A previously healthy 49 year old English lady presented with a 2 week history of malaise, sweats, fever, chills and severe, intermittent headache. Three days preceding

admission she developed a non-productive cough. She did not own any pets. On examination she was acutely ill, febrile at 39.5 °C, with a blood pressure of 130/90 mm Hg. There were crackles in the left mid-zone posteriorly. She was fully conscious, orientated, with no neurological signs. Eye examination was normal. Initial investigations showed a haemoglobin of 11.8 g/dl, increased white cell count at $16.1 \times 10^9/l$ (neutrophils 89%, lymphocytes 8%, monocytes 3%), and an elevated plasma viscosity at 2.33 cp. The urea and electrolyte levels, liver function tests and serum electrophoresis were within normal limits. A chest radiograph showed patchy left mid-zone shadowing. A diagnosis of an atypical pneumonia was made and the patient commenced on erythromycin 750 mgm 6 hourly by mouth. Two days after admission, when she felt significantly better, the patient developed acute diplopia. Examination revealed an isolated, complete, left VIth nerve palsy, with no papilloedema or other neurological signs. CT of the brain, skull radiograph and CSF findings were normal. Subsequent results showed a positive immunofluorescence *Chlamydia psittaci* titre at 1/256 and a CFT titre at 1/80. Upon close questioning, a history of incidental contact with two parrots and a sick budgerigar was obtained. Serological tests for mycoplasma, legionella and coxiella were negative. Erythromycin was continued for 10 days.

The patient became extremely distressed by the diplopia and was unwilling to use alleviating measures with an eye patch or special spectacles. With no precedence to guide us, the prognosis was guarded. Recovery of the VIth nerve palsy commenced 8 days after its onset and was complete by the fifth week. The patient remains well 3 months later.

Cranial nerve involvement complicating infection with *Chlamydia psittaci* is unusual.¹ IInd,² supranuclear VIIth,³ and XIIth⁴ cranial nerve palsies have been reported in severe encephalitic cases of psittacosis. Unlike these, there were no signs of encephalitis or raised intracranial pressure in our patient. Rare as it may be, the prognosis appears excellent.

A ZUMLA
G LIPSCOMB
D LEWIS

Infectious Diseases Unit,
Rush Green Hospital,
Romford,
Essex RM7 0YA
UK.

References

- 1 Christie AB. Psittacosis in: *Infectious Diseases*.

Vol. 2. Edinburgh: Churchill Livingstone, 1986:1116-30.

- 2 Denmark JC. Encephalitis due to the psittacosis lymphogranuloma venereum group of virus. A case of ornithose encephalitis. *Br Med J* 1957;2:1531.
- 3 Carre-Locke DL, Mair HJ. Neurological presentation of psittacosis during a small outbreak in Leicestershire. *Br Med J* 1976;2:853-4.
- 4 Wilde JA. Un cas d'encéphalite engendré par le virus due l'ornithose. *Rev Neurol (Paris)* 1951;84:348-50.

Motor axon loop as a source of error in the measurement of motor nerve conduction velocity

Sir: In 1984 Roth and Egloff-Baer described the motor axon loop, a new kind of response of the motor unit to stimulation of the peripheral motor nerve.¹ We would like to report a case in which a motor axon loop may have caused an error in the motor nerve conduction velocity measurement.

A 72 year old man developed weakness and atrophy in his right hand muscles in January, 1985. The weakness and atrophy slowly progressed, and in September, 1986 when he visited our outpatient clinic, he had severe weakness and atrophy in his right upper extremity muscles and moderate weakness and atrophy in his left hand muscles. Sensation was intact. Electromyography revealed neurogenic abnormalities in the facial, sternocleidomastoid, tongue, and upper and lower limb muscles. A diagnosis of amyotrophic lateral sclerosis was made. A motor nerve conduction study was carried out on the right median nerve. Recording was made with surface electrodes placed on the belly of the thenar muscle (cathode) and the base of the thumb (anode). Electrical pulses of 0.2 ms in duration were delivered. Only one motor unit potential (MUP) was evoked in the thenar muscle, in an "all-or-none" manner. When the median nerve was stimulated at the elbow, the latency of the MUP shortened abruptly by increasing the intensity of the stimulus to a critical level (figure inset). Serial stimulation from the wrist to the upper arm in 3 cm increments revealed that this shortening phenomenon occurred only between 16.5 and 25.5 cm from the wrist, the difference between the latencies of the late and brief responses being constant. When the stimulus was delivered outside this segment, the latency of the MUP did not show any change by increasing the stimulus intensity. No F-waves were observed. When the latencies of the MUP were plotted

Fig Latencies of the M-response plotted against the distance of the stimulation site from the wrist. Shortening of the latency by increasing the stimulus intensity is observed only with stimulation over the segment between 16.5 and 25.5 cm from the wrist. The points obtained by weak stimulation within this segment and at a more proximal site (28.5 cm) are on a straight line, and those obtained by stronger stimulation within this segment and at more distal sites (4.5–13.5 cm) are on another straight line (fig). Inset: M-responses recorded from the thenar muscle. The median nerve is stimulated just below the elbow (19.5 cm from the wrist). The latency of the response shortens abruptly from 10.8 ms (upper trace) to 8.5 ms (lower trace) as the stimulus intensity is increased to a critical level.

against the distance of the stimulation site from the wrist, the points obtained by weak stimulation between 16.5 and 25.5 cm and at a more proximal site (28.5 cm) were on a straight line. The points obtained by stronger stimulation between 16.5 and 25.5 cm and those at more distal stimulation sites (4.5–13.5 cm) were on another straight line (fig). Motor nerve conduction velocities calculated from the gradients of the two regression lines were 68.0 m/s and 56.5 m/s, respectively.

The motor unit potential obtained in this patient is in complete accordance with the description of the motor axon loop by Roth and Egloff-Baer.¹ In their report, the motor units with a motor axon loop were recorded as a late potential preceded by the main M-response. The present case is unique in that the MUP with a motor axon loop is the only recordable motor response. The motor axon loop is considered congenital, and it appears that the last remaining motor unit in this case happened to have a looped axon. In this particular situation, the motor nerve conduction velocity of the segment containing the loop is calculated to be erroneously low. For example, motor nerve conduction velocity between 13.5 and 28.5 cm from the wrist is calculated as:

$$\frac{28.5 - 13.5 \text{ (cm)}}{12.2 - 7.2 \text{ (ms)}} = 30.0 \text{ m/s,}$$

which is obviously wrong, but one cannot know that it is wrong unless one gives weak and strong stimulations over the looped segment of the motor axon. It should be borne in mind that the motor axon loop can be a source of error in the measurement of motor nerve conduction velocity when the number of surviving motor units is extremely small.

TOMOYUKI SHIMPO
YOSHIKAZU UGAWA
TORU MANNEN
Department of Neurology,
Institute of Brain Research,
School of Medicine,
University of Tokyo,
7-3-1 Hongo,
Bunkyo-ku, Tokyo,
113 Japan

Reference

- 1 Roth G, Egloff-Baer S. Motor axon loop: an electroneurographic response. *Muscle Nerve* 1984;7:294–7.

Accepted 28 July 1988.

Matters arising

Computed tomographic findings of brain and skull in myotonic dystrophy

Sir: We read with great interest the article by Avrahami *et al*¹ about computed tomography findings of brain and skull in myotonic dystrophy and the letter which appeared later on the same topic.² We have seen a 30 year old patient with a family history of myotonic dystrophy (mother and sister) diagnosed on a clinical and EMG basis who had sudden right ear deafness with transitory vertigo. A round osteolytic lacuna of the upper end of the internal auditory canal and of the bony wall of the first cochlear turn was seen by radiographic tomography of the right petrous pyramid. CT of the petrous temporal bone confirmed this lesion (fig) and air-CT-cisternography excluded a small intracanalicular tumor.

Other causes of sudden deafness, such as virus diseases, bacterial labyrinthitis or meningitis, endolymphatic hydrop, vascular diseases, head trauma, were excluded.

Calcium and phosphorus metabolism and parathormone level were normal. Audiological findings were: normal hearing in the left and a complete hearing loss in the right ear, a decreased response of the labyrinthine end organ to caloric tests on the right, startle reflexes present at normal intensity only on stimulation of the left ear, normal ABR amplitude and latency in the left ear, ABR absent in the other one. A 3 year clinical and CT of the petrous temporal bone follow-up showed the same picture.

To our knowledge an osteolytic lacuna, which could be the cause of the sudden deafness in our patient, has not been recognised in patients with myotonic dystrophy.

Fig CT of the petrous temporal bone showing the osteolytic lacuna of the upper end of the internal auditory canal and of the bone wall of the cochlear basal turn (arrow).