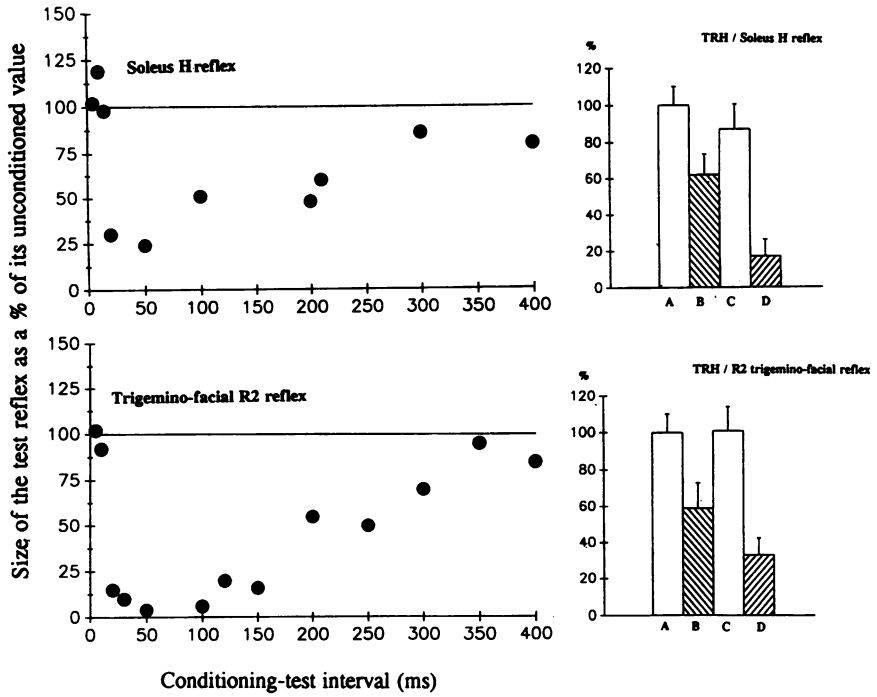

Figure 1 Left panels. Time-course of the inhibition of the soleus H reflex and trigemino-facial R2 response. The sizes of the H (peak to peak) and R2 (area) reflexes (as a % of their unconditioned value) are plotted against the time elapsed after the conditioning stimulation of tibialis anterior Ia and trigeminal afferent fibres respectively. Right panels. Unconditioned (Columns A) and conditioned (Columns B) soleus H and trigeminal R2 reflexes obtained before TRH injection are compared with unconditioned (Columns C) and conditioned (Columns D) reflexes after TRH injection. Each column represents the mean of 15 measurements and the vertical bars 1 SE of the mean. All data from the same subject.

subjects after a single acute intravenous injection of 10 mg of TRH (unpublished observation). This procedure was therefore used in this study. Before and 15 minutes after the end of the injection, 10 unconditioned and 10 conditioned R2 and H reflexes were randomly evoked. The intensity of the conditioning stimuli (applied 100 ms before the test ones) was adjusted to elicit, in pre-infusion control condition, a small amount of inhibition of both H and R2 reflexes (about 60% of their unconditioned values: see columns B). After TRH injection the amount of inhibition of both H and R2 reflex responses was significantly increased (columns D) (Student's test: $t = 4{\cdot}9$, $p < 0{\cdot}001$; $t = 6{\cdot}6$, $p < 0{\cdot}001$ respectively). As no significant changes of unconditioned test responses were present (compare columns A and C) the possibility of some post-synaptic effects on motor neurons could be ruled out. Exactly the same results were obtained in two normal subjects.

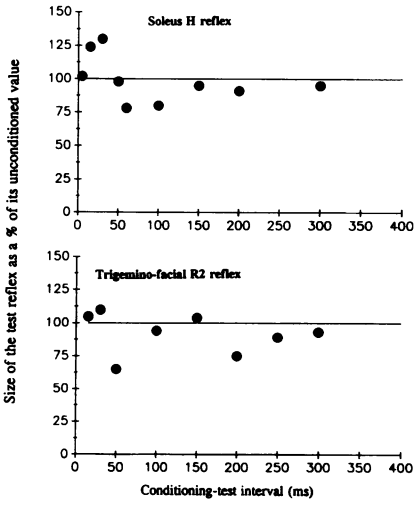

Figure 2 Patient with chronic spino-bulbar spasticity. As in the left panels in fig 1.

Figure 2 shows the behaviour of the conditioned R2 and H reflexes in a patient with chronic progressive spinobulbar spasticity. Conditioning stimulation to trigeminal or pretibial Ia fibres failed to produce depression of the respective test reflexes. It was also verified in two patients with pure spastic paraparesis (Strumplell-Lorrain disease, not illustrated) that, despite the absence of the soleus Ia presynaptic inhibition, the trigemino-facial R2 response was normally depressed when preceded by a conditioning discharge of low-Th trigeminal afferent fibres (not illustrated).

Independent observations support the conclusion that presynaptic inhibition on trigeminal primary afferent fibres is the main factor contributing to depression of the R2 response following conditioning activation of low-Th trigeminal fibres: 1) there is a strict similarity between the time-course of R2 inhibition and that of trigeminal primary afferent depolarisation observed in animals; 2) the time-course of R2 inhibition is identical to the time course of spinal presynaptic inhibition; 3) TRH, a drug able to increase spinal presynaptic inhibition, is equally effective in increasing both Ia presynaptic inhibition and the inhibition of the trigeminal R2 response; 4) lesion of cortico-bulbar and cortico-spinal projections results in depression of both spinal presynaptic inhibition and trigeminal R2 inhibition.

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Intracranial hypertension following psittacosis

Neurological complications are rare in psittacosis. In 1972 a review of 156 cases reported two with encephalitis and one with a lymphocytic meningitis.[1] Since then there have been reports of cerebellar disturbance,[2] sixth nerve palsy,[3] and transverse myelitis.[4] We report a case of intracranial hypertension following psittacosis.

A 46 year old male was referred with deteriorating vision. He had been well until 10 weeks previously when he developed a severe generalised headache with malaise, sweats, anorexia, and weight loss. The headache was of gradual onset and associated with neck stiffness. There were no respiratory symptoms. After 8 days of this illness there was a subacute increase in the severity of the headache with slurring of speech and unsteadiness. He was admitted to a local hospital where he was noted to have mild neck stiffness. Kernig's sign was negative and no other neurological signs were elicited. General examination was normal. A chest radiograph showed an area of consolidation in the right lower lobe. A CT scan of the brain was normal. CSF examination was normal with an opening pressure of 20·5 cm. He was discharged the following day, but the headache persisted for another 10 days. There was then an interval of approximately 2 weeks when he was asymptomatic and well. Over the following 6 weeks he noticed gradual loss of vision, particularly in the left eye, with some variation but no complete obscurations. He was not taking any medication and had not received any antibiotics during the course of the illness. There was no relevant past medical or family history. He was a non-smoker and rarely drank alcohol. He kept ten parrots, one of which had died during the mating season two months before the onset of his illness. The cause of the parrot's death was not established.

On examination he was well and not overweight. Corrected visual acuity was 6/6 on the right and 6/18 on the left. Colour vision was normal. There was mild constriction of the peripheral visual fields and the blind spots were enlarged, more so on the left. There was bilateral papilloedema, worse on the left, with haemorrhages and retinal and choroidal folds. The remainder of the neurological examination was normal, as was the general examination.

The following investigations were normal or negative: full blood count, electrolytes, renal function, liver function, serum glucose, clotting screen, syphilis serology, lupus anticoagulant, protein C and S, antithrombin III, autoantibody screen, monospot, and cold agglutinins. Psittacosis complement fixation test was strongly positive at 1280. A repeat measurement three weeks later was 640. Mycoplasma and Coxiella titres were both < 40. A chest radiograph and CT brain scan were normal. Intravenous digital subtraction angiography showed no occlusion of the major cerebral venous sinuses. At lumbar puncture the opening pressure was elevated at 37 cm; CSF constituents were normal.

A total of 20 ml of CSF was drained with symptomatic improvement and he was given a ten day course of oral erythromycin. Two weeks after the initial lumbar puncture the visual symptoms recurred with intermittent diplopia. Mild bilateral sixth nerve palsies were now present and at repeat lumbar puncture CSF pressure was 37·5 cm. He was started on acetazolamide 250mg twice daily. One week later the CSF pressure was 22·5cm and his symptoms and visual fields had improved. The acetazolamide was continued for a further two weeks but then stopped following an episode of suspected renal colic. Despite this he continued to improve and after nine months, although troubled by occasional headaches, visual acuity was 6/6 on the right and 6/9 on the left. The left blind spot remained slightly enlarged but peripheral fields were now normal. There was no swelling of the optic discs and eye movements were normal. To date there has been no recurrence.

The systemic disturbance at the onset of this patient's illness is compatible with a diagnosis of psittacosis. A review of 135 cases of serologically confirmed psittacosis found the characteristic features to be an abrupt onset of fever, rigors, sweats, a prominent headache, and a mild dry cough which often appeared late. Respiratory symptoms were absent in 18% of patients, but over 90% had an abnormal chest radiograph, or abnormal chest signs, or a combination of both.[5] Despite the absence of respiratory symptoms in our patient, his initial clinical presentation and the chest radiograph are consistent with this diagnosis. As the patient presented to us 10 weeks after the initial febrile illness, we were unable to demonstrate a rise in the complement fixation titre and a raised titre may merely reflect previous exposure. However, a single titre of $\geqslant 256$ without an associated antibody rise to one of the commoner respiratory infections is generally considered to be sufficient evidence for a diagnosis of psittacosis.[5] The initial titre of 1280 in our patient, which subsequently fell to 640, is thus indicative of recent infection. In combination the clinical, radiological and serological features support a diagnosis of psittacosis. The previous death of one of the patient's parrots may also have been relevant. In 25% of cases where there is a firm history of exposure to birds, one or more of the birds had died or been ill.[1]

The subsequent visual symptoms, visual field abnormalities, and papilloedema with an elevated CSF pressure and a normal CT brain scan suggested a diagnosis of so called benign intracranial hypertension. The condition is unusual in males who account for only 16% of cases and obesity is less strongly associated than in females. The cause of the syndrome in most cases remains undetermined. There are several potential mechanisms in this particular patient. Firstly, venous sinus thrombosis occurring at the time of the initial febrile illness could explain the intracranial hypertension and this could possibly have resolved by the time of digital subtraction angiography. This possibility is compatible with the patient's improvement which could have been spontaneous and unrelated to antibiotic and acetazolamide therapy. Tetracycline, the treatment of choice for psittacosis, is associated with benign intracranial hypertension, but our patient received no specific treatment for his original illness. Lastly, CSF absorption may have been impaired secondary to meningo-encephalitis, which is a recognised complication of psittacosis.[1] Our patient had symptoms compatible with meningo-encephalitis but CSF constituents were normal on two occasions. Thus the pathogenesis of intracranial hypertension in this patient is not entirely clear.

Psittacosis appears to be increasing in frequency and this previously unreported complication emphasises the potentially diverse manifestations of the disease.

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MATTERS ARISING

Neurogenic effects on the palatopharyngeal muscle in patients with obstructive sleep apnoea: a muscle biopsy study

Edström *et al*[1] reported neurogenic lesion in the palatopharyngeal muscle of eight patients with obstructive sleep apnoea. Their report is particularly interesting as they suggest a disturbance of the dilating muscle function of the upper airway in the pathogenesis of obstructive sleep apnoea. Surprisingly, the authors did not consider our published data on muscle fibre type distribution of medium pharyngeal constrictor muscle in habitual snorers.[2] In these patients we found an abnormal distribution of fibre types (low percentage of type I and type IIb and high percentage of type IIa fibres) compared with controls. No myopathic or neurogenic changes were found. We proposed two hypotheses to explain the abnormal distribution of fibre types in snorers. First, a constitutionally determined reduction of slow α-motor neurons induces an adaptive transformation of type IIb to type IIa fibres and a hypertrophy, or second motor neuron change their patterns of discharge and, hence, of activation, and modify fibre-type distribution of medium pharyngeal constrictor muscle as an adaptation to the anatomical characteristic of upper airway and habitual snoring.

In patients with obstructive sleep apnoea Edström *et al*[1] found a large variability in muscle fibre size, signs of neurogenic stress and muscle fibre atrophy. However, no neurological evaluation or investigations were performed in patients with obstructive sleep apnoea to exclude a peripheral neuropathy or muscular pathologies. The authors did not specify whether or not the patients had undergone recent pharmacotherapy. It is commonly known that some drugs can change the muscle fibre pattern.[3] Another factor is the site of the muscle sampling in the different patients. Biopsy samples not taken at the same site and same depth tend to vary in fibre type distribution and diameter.[4]

The authors did not give any indication of the fibre type distribution in either of the subjects groups. Such an indication is important as fibre distribution is fundamental to assessing muscle function indexes.[5]

If details of the points we have raised are available there might be a greater understanding of the pharyngeal muscle involvement in patients with obstructive sleep apnoea.

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Edström et al reply:

The principal reason why we did not consider the results from Iannaccone *et al*[1] is because they were not published when the original version of our manuscript was completed. We have, however, considered their comments with interest.

We do not, however, find their results comparable to ours as a different biopsy site was examined. It is possible that different sites in the pharynx are affected in different ways by snoring and obstructive sleep apnoea. Furthermore, it is not known whether the patients in the study by Iannaccone *et al* had obstructive sleep apnoea or not, and snoring was not a medical problem. It is not possible to judge from interviews only whether a patient has obstructive sleep apnoea.[2 4] All patients in our study had sought medical attention because of problems associated with obstruc-