gave the correct name of the hospital, he located it in another city. Despite being repeatedly informed that the hospital was in Málaga, he denied it and was convinced that it was a new branch in the city he named. Although he remained in the same room during his admission to hospital, he insisted he had been moved daily to identical hospitals (all with the same name) that were located in neighbouring cities (Torremolinos, Fuengirola, Marbella).

One week after onset, the patient had a detailed neuropsychological examination. He was found to have a WAIS verbal IQ of 94 and a performance IQ of 51. On the Wechsler Memory Scale (WMS), his memory quotient was normal (99 points); his scores on immediate history recall and associated learning were average though his performance on the visual reproduction subtest was below average. There was no evidence of confabulation in response to items of the Mercer's confabulation battery, but he did poorly on tests thought sensitive to frontal lobe dysfunction (Wisconsin Card Sorting Test (categories achieved = 0; normal range = 4–6), Trail-making test (part A) (below percentile 10)). He also showed a severe impairment on visual-perceptual tests (Visual Form Discrimination (14 points; normal range 23–32), Facial Recognition (15 points; percentile rank = 1). His language was almost intact, except for a mild visual naming impairment (Boston Naming Test, 38 points (maximum = 60)).

CT and MRI scans showed a right thalamic haemorrhage with extension into the posterior limb of the internal capsule, corona radiata and ventricular system. Mild symmetrical periventricular white-matter changes compatible with leukoaraiosis were also observed. Regional cerebral blood flow was studied with ^{99m}Tc -HMPAO and SPECT, using an Elscint Apex 609 RG gamma camera. Focal blood flows were analysed semiquantitatively in twelve circular regions of interest which were placed over the cortical mantle in three successive slices. Asymmetry indices (AI) for each lobe were calculated using the following formula: $(R - L)/(R + L) \times 200$.³ A marked decrease of perfusion was observed in the right thalamus and basal ganglia as well as in the left cerebellum. Hypoperfusion was also noted in widespread cortical regions of the right hemisphere affecting mainly the frontal lobe (AI = -29.6), and to a lesser extent the parietal (AI = -10.8) and temporal (AI = -10) lobes (negative AI values indicate left sided hyperactivity relative to the right sided activity).

The assessment of neuropsychological functions in our case of subcortical environmental reduplication revealed more pervasive deficits than those observed in the patient reported by Nighoghossian *et al.*,¹ but similar to those of previous cases showing evidence of environmental reduplication and cortical involvement. Moreover, the combination of deficits on nonverbal memory, awareness, visual-perceptual skills and reasoning abilities supports the view that environmental reduplication is a complex, multifactorial delusional misidentification syndrome.^{4,5}

Some previous reports emphasised the association between environmental reduplication and the superimposition of bilateral frontal-lobe and right hemisphere cortical involvement, while others suggested that unilateral (right) lesions of either the fronto-

parietal or parieto-temporal cortices are sufficient to cause it.^{4,5} In our patient, right thalamocapsular damage may have induced functional depression of various distant but anatomically connected cortical areas. Data from SPECT, however, revealed secondary cortical deactivation affecting mainly the right frontal cortex. In this context, we suggest that functional deactivation of the right cortical mantle, in addition to thalamocapsular injury, may underlie environmental reduplication and its associated neuropsychological deficits. In addition, given that environmental reduplication probably requires preexisting brain pathology (for example, cortical atrophy) besides the specific sites of brain damage in the right hemisphere,^{1,2} the presence of leukoaraiosis in the case by Nighoghossian *et al.*¹ and in our own patient might be another risk factor for developing it after acute stroke.

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- 1 Nighoghossian N, Trouillas P, Vighetto A, *et al.* Spatial delirium following a right subcortical infarct with frontal deactivation. *J Neurol Neurosurg Psychiatry* 1992;55:334–5.
- 2 Leiguarda RC. Environmental reduplication associated with a right thalamic haemorrhage. *J Neurol Neurosurg Psychiatry* 1983;46:1154.
- 3 Pozzilli C, Passafiume D, Bernardi S, *et al.* SPECT, MRI and cognitive functions in multiple sclerosis. *J Neurol Neurosurg Psychiatry* 1991;54:110–5.
- 4 Patterson MB, Mack JL. Neuropsychological analysis of a case of reduplicative paramnesia. *J Clin Exper Neuropsychology* 1985;7:111–21.
- 5 Kapur N, Turner A, King C. Reduplicative paramnesia: possible anatomical and neuropsychological mechanisms. *J Neurol Neurosurg Psychiatry* 1988;51:579–81.

Evidence for presynaptic inhibition on trigeminal primary afferent fibres in humans

In a recent study we have shown that a conditioning electrical stimulus applied to the trigeminal afferent fibres of intensity below the reflex threshold (Th) (0.90–0.95 times the perceptive Th) produces unexpected changes of test trigemino-facial reflex responses.¹ In particular, for the R2 response, there is a monotonic depression starting at 20–30 ms of interstimulus interval, reaching a maximal value at 50–100 ms and recovering within 300–400 ms (figure 1). Based on the similarity of the time-courses of the R2 inhibition and of the trigeminal primary afferent depolarisation described in the cat,² we propose that presynaptic inhibition from low-Th trigeminal afferent fibres was the primary factor contributing to the depression of the test response. We describe experimental evidence to support this.

Two complementary experimental

designs, approved by the Local Ethical Committee, have been employed: 1) the time-course of the R2 inhibition was compared, in the same subject, with that of the soleus Ia presynaptic inhibition; 2) the effect of intravenous administration of thyrotrophin releasing hormone (TRH), a substance shown to increase presynaptic inhibition in humans,³ has been tested in parallel on both spinal Ia presynaptic inhibition and R2 reflex inhibition. In addition, spinal Ia presynaptic inhibition and trigeminal R2 inhibition were studied in a patient affected by chronic progressive spino-bulbar spasticity, a rare disease presenting with spastic paralysis of facial, laryngeal and lower limb muscles, due to a progressive and parallel involvement of corticobulbar and cortico-spinal projections.⁴ Many patients with spasticity show a reduced or absent presynaptic inhibition on the soleus Ia primary afferent fibres, probably due to the loss of a tonic supraspinal excitatory drive on interneurons acting presynaptically on Ia terminals. If inhibition of the R2 reflex is of presynaptic origin and it is subjected to such a descending control, then it could be expected that a lesion of the corticobulbar and cortico-spinal projections would also result in a parallel reduction of presynaptic inhibition on both spinal Ia and trigeminal primary afferent fibres.

Figure 1 shows the time-course of presynaptic inhibition of the soleus H reflex in a normal subject. We used a method introduced by Morin, Pierrot-Deseilligny and Hultborn in 1984,⁵ which we briefly describe. The test response was a soleus H reflex evoked by stimulating the posterior tibial nerve. To elicit Ia afferent discharge from pretibial muscles, a short lasting vibration (10 ms; 3 cycles; 300 Hz) was applied over the tibialis anterior tendon by an electromagnetic hammer, 10–400 ms before the test stimulus. Under this experimental condition, evidence shows that the resulting long-lasting depression of the soleus H reflex is due solely to presynaptic inhibition from pretibial Ia afferent fibres to those mediating the afferent test volley.⁶ In fig 1 the time-course of the inhibition of the trigemino-facial R2 reflex following a weak conditioning stimulation (0.95 times the perceptive PTh) of the first trigeminal branch is also reported. There is a clear strict similarity of the curves of the R2 and soleus H reflex inhibition. Identical findings have been observed in two other normal subjects. By exploring the upper curve in fig 1 it is apparent that the long-lasting inhibition of the soleus H reflex is preceded by an early short-lasting facilitation (see also the upper curve in fig 2). This early facilitation is due to mechanical spread of the vibration to soleus spindles causing homonymous Ia facilitation in the soleus motor neurons.⁶ This explains why this early facilitation is lacking on the R2 reflex curve (lower curve in fig 1).

The histograms on the right in fig 1 show the enhancement of the R2 and H reflex depressions after acute intravenous administration of TRH. It has been demonstrated that a single subcutaneous injection of high-dose TRH (1.2–2.5 mg/Kg) produces dramatic and long-lasting (1–2 hours) increase of the soleus presynaptic inhibition in patients with amyotrophic lateral sclerosis.³ We have observed a similar but short-lasting (10–20 minute) enhancement of the soleus Ia presynaptic inhibition in normal

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: $t =$

4.9, $p < 0.001$; $t = 6.6$, $p < 0.001$ 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 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 corticobulbar and cortico-spinal projections results in depression of both spinal presynaptic inhibition and trigeminal R2 inhibition.

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

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- Rossi A, Scarpini C. Gating of trigemino-facial reflex from low-threshold trigeminal and extratrigeminal cutaneous fibres in humans. *J Neurol Neurosurg Psychiatry* 1992;55:774-80.
- Stewart DH, Scibetta CJ, King RB. Presynaptic inhibition in the trigeminal relay nuclei. *J Neurophysiol* 1967;20:135-153.
- Morin C, Pierrot-Deseilligny E. Mécanisme spinal de l'action antispasmodique de la TRH chez les malades atteints de sclérose latérale amyotrophique. *Rev Neurol* 1988;144:701-3.
- Gastaut JL, Michel B, Figarella-Branger D, Somma-Mauvais H. Chronic Progressive Spinobulbar Spasticity. *Arch Neurol* 1988; 54:509-13.
- Morin C, Pierrot-Deseilligny E, Hultborn H. Evidence for presynaptic inhibition of muscle spindle Ia afferents in man. *Neurosci Lett* 1984;44:137-142.
- Hultborn H, Meunier S, Pierrot-Deseilligny E, Shindo M. Assessing changes in presynaptic inhibition of Ia fibres at the onset of voluntary contraction in man. *J Physiol* 1987;389:757-72.

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.