features of Addisonian crisis. He was successfully resuscitated and commenced on corticosteroid replacement therapy. The diagnosis was confirmed by a short Synacthen test. Despite pleural thickening seen on the chest radiograph there was no evidence that his disease was tuberculous; adrenal antibodies were negative. He complained coincidentally of a 5 year history of progressive difficulty in walking and was noted to have signs of spastic paraparesis. Skull radiograph, isotope brain scan, CSF examination, B12 and serological tests for syphilis were normal and no definite diagnosis was made. He remained comparatively well on replacement therapy and was able to continue his job, working 7 days a week, for a further 6 years. In 1981 he was forced to retire through immobility. Always abrupt in his manner, he became increasingly cantankerous and given to swearing. In December 1983 his dementia became overt with worsening quadriplegia, cerebellar signs, confusion, faecal and urinary incontinence. There was no clinical evidence of peripheral sensory neuropathy; ankle reflexes were brisk and associated with clonus. The diagnosis of adrenomyeloneuropathy was suspected. EEG showed bilateral slow wave activity with no specific features. Peripheral nerve conduction studies suggested axonal neuropathy as previously reported by others.⁶ Accumulation of long-chain fatty acids (C26) was determined in skin fibroblast culture; the C26:C22 ratio was 0.90 (normal 0.55–0.90). Serum testosterone was low (4.1 nmol/l), while LH and FSH were borderline high (11.3 U/l and 8.6 U/l, respectively). He was not investigated further and died in July 1984 following admission to hospital for terminal nursing care. Post-mortem appearances of brain, adrenal cortices and gonads were characteristic with widespread cerebral demyelination with perivascular cellular infiltrates and foamy vacuolation of adrenal cortical and Leydig cells.⁴

Schaumburg and colleagues¹ have drawn attention to late onset adrenomyeloneuropathy as a variant of the better-recognised childhood form. In their report of five cases and review of four others, there were eight males and one female, and age of onset ranged from 9 to 44 years. In one adult there were no affected family members, and in two others family history was unknown. Detailed family history had not been sought at first in our patient, but subsequent enquiry revealed that his maternal grandfather may have been paralysed, although not demented, when he died 50 years earlier, age 80. The patient's mother was said to

have taken to her bed at the age of 57 years and remained there for the last 18 years of her life. However she was not thought to be demented or paralysed, and it is not clear if her confinement was for medical or social reasons. There were no other relatives with either demyelination of adrenocortical failure. He had no children.

The possibility of adrenomyeloneuropathy should be borne in mind in clinical practice. Indeed it may be more common than is generally recognised and may account for some cases of adrenocortical failure even in the absence of neurological signs. Although typically a disease of early life, occasional cases present very late.

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Improved phonation during fever in brainstem dysarthrophonia

Sir: Dysphonia and dysarthria are common sequelae of brainstem pathology as it is in this part of the brain that the direct neural control of phonation, articulation, breathing and their complex coordinated execution during speech takes place. Thermoregulatory disturbances are also not infrequent after brainstem damage. Recently we treated a patient who after sustaining a severe brainstem insult, exhibited a combination of dysphonia, dysarthria and relapsing fever most probably of central origin. There was a remarkable improvement in the patient's phonatory ability during fever. This phenomenon is new in our clinical experience and we could not find such a case reported in the literature.

A 21 year old male sustained a head injury in a road accident rendering him immediately unconscious. Brain and cervical spine CT failed to reveal fractures; however, brain oedema and intracranial air bubbles were noted. About 2 weeks later he contracted pseudomonas meningitis which responded to antibiotic therapy. He became afebrile and his CSF remained normal thereafter. Neurological examination 2 months after injury revealed spastic tetraplegia, anisocoria, hypoaesthesia on the right side of the face with reduced corneal reflex, right sixth and peripheral seventh cranial nerve palsies, bilateral attenuation of the gag reflex and deviation of the tongue to the right. Repeated CT revealed widening of the ambient cisterns, interpeduncular and quadrigeminal cisterns.

Fig CT scan showing widening of the quadrigeminal cistern.

terns and of the 4th ventricle, secondary to posttraumatic atrophy in the bulbomesencephalic regions (fig).

The patient was conscious but easily fatigued and sleepy. He was aphonic and dysarthric and had an apparently preserved comprehension of surrounding events and speech. Soon after admission we noticed a dramatic improvement in the patient's phonatory ability during episodes of high fever. Pyrexia was almost always associated with signs of general excitation in this otherwise lethargic patient. Over 20 episodes of fever, reaching 39–40°C and lasting less than 24 hours, occurred during the first year at irregular intervals. The phenomenon occurred less often thereafter and disappeared altogether 18 months after injury. On three occasions fever was due to infection but in the other episodes no apparent cause could be found in spite of extensive laboratory investigations. The patient's speech was non-functional owing to severe dysarthrophonia. Although facial gesticulations as well as tongue and soft palate movements during articulation were impaired, the dysarthric component was mainly of a "suprasegmental" nature, that is, it was exhibited not so much in the production of individual speech sounds as in the production of longer units.¹ The major obstacle for communication was the phonatory impairment. Phonation length ("ah") was only 1–2 s. Loudness was inconsistent, the usual state being aphonia with occasional marked hypophonia. Phonation improvement with fever was as prominent during the infectious episodes as in the more than the 20 unexplained ones. We constructed a quantitative measure of speech intelligibility by submitting the patient to read from lists of monosyllabic and bisyllabic words in front of "judges" who tried to decipher what was said. This was done repeatedly during febrile and afebrile states. Basic pulmonary function tests were measured concomitantly as the patient suffered from a severe restrictive disturbance contributing to the phonatory problem. The improvement in speech intelligibility during fever was found to be statistically significant. There was also some change in respiratory parameters during fever. Respiratory rate increased from 20/min to 36/min, tidal volume from 0.31 to 0.42 l and forced vital capacity from 0.49 l to 0.61 l. Peak expiratory flow rate remained very low (95 l/min). Administration of moderate doses of methylphenidate, caffeine and epinephrine, in an attempt to reproduce the excitatory effect of fever, failed to improve phonation or to induce mental excitation although an improvement in lung functions did occur.

Transient mutism and aphonia are quite common in head injured patients, especially in those suffering from damage to the upper brainstem. Gradual recovery of speech functions, including the phonatory ability, is the rule in these patients.² On the basis of data derived from animal experimentation, in which the major role played by the periaqueductal grey and the lateral tegmental area was shown,^{3,4} it was speculated that lesions in these regions might be responsible for aphonia and perhaps other phenomena seen in posttraumatic mutism.² The persistence of aphonia in our patient suggests that apart from the damage to brainstem phonatory control systems, other factors operated to produce the phonatory problem. It is known that head injured patients may develop ventilatory disturbances of a restrictive nature.⁵ As gross alteration of the respiratory pattern takes place during speech production,⁶ the disturbances in ventilatory mechanics found in our patient could further aggravate his phonatory problem. A correlation was found between the improvement in speech intelligibility during fever and the occurrence of thermal hyper-ventilation and a general excitatory effect. The drugs used failed to induce such an excitation and although an increment of considerable magnitude in tidal volume was obtained, the patient's phonation remained unchanged. It seems therefore that simple ventilatory augmentation cannot in itself account for the entire effect of fever upon phonation. Efficient sound production demands a rapid and precise neuromuscular coordination of ventilatory and laryngeal musculature.^{6,7} Most probably this complex activity depends, as other motor activities do,⁸ on an optimal level of arousal for its proper execution. It is proposed that the elevation of body temperature, through its effect upon the level of arousal⁹ improved our patient's sleepy state and thereby his ability to coordinate respiratory, laryngeal and oral muscles functioning during vocalisation. Search for the possible existence of a relationship between body temperature and phonation in other aphonic brain damaged patients may shed further light on the exact mechanism and the clinical significance of this phenomenon.

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Unusual drug interactions between monoamine oxidase inhibitors and tricyclic antidepressants

Sir: Monoamine oxidase inhibitors and tricyclic antidepressants are both capable of producing severe side effects when taken in excess and these have been well documented in the past.¹ It is less well recognised that combining these two drugs, in either therapeutic or excessive dosages, may have potentially lethal side effects. The following case report is an unusual example of the dangerous side effects that may result from combining these two drugs.

A 34 year old depressive woman took an overdose of monoamine oxidase inhibitors (MAOI) and tricyclic antidepressants (TCA). The medications taken were tranylcypromine (40 mg), clomipramine (150 mg) and trazadone (400 mg). Cyclizine (150 mg) and oxazepam (60 mg) were also taken. Four hours after taking the drugs, the patient had a grand mal seizure, went into respiratory failure and was intubated and ventilated. On examination the patient was