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. 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.

Schaumburg and colleagues 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 afibrile and his CSF remained normal thereafter. Neurological examination 2 months after the injury revealed spastic tetraplegia, anosmia, 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, interpeduncular and quadrigeminal cisterns.
terns and of the 4th ventricle, secondary to posttraumatic atrophy in the bul- 
bonsencephalic 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 pho-
natory 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 reg- 
ular intervals. The phenomenon occurred 
less often thereafter and disappeared alto-
gether 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 dys-
arthrophia. Although facial gesticu-
lations 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 pro-
duction of individual sound speech as in 
the production of longer units.1 The 
major obstacle for communication was the pho-
natory impairment. Phonation length 
(“ah”) was only 1–2 s. Loudness was in-
consistent, 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 construc-
ted a quantitative measure of speech intel-
ligibility by submitting the patient to read 
from lists of monosyllabic and bisyllabic 
words in front of “judges” who tried to deci-
pher 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 phonautic problem. The improvement in 
speech intelligibility during fever was found 
to be statistically significant. There was 
also some change in respiratory para-
eters during fever. Respiratory rate 
increased from 20/min to 36/min, tidal vol-
ume from 0.31 to 0.421 and forced vital 
capacity from 0.491 to 0.61. Peak expiratory 
flow rate remained very low (95/min). 
Administration of moderate doses of 
methyldenphene, caffiene and ephedrine, 
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 aphony are quite 
common in head injured patients, especially 
in those suffering from damage to the upper 
brainstem. Gradual recovery of speech func-
tions, including the phonautic 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 peri-
aqueductal grey and the lateral tegmental 
area was shown,3,4 it was speculated that 
lesions in these regions might be responsible 
for aphony and perhaps other phenomena 
seen in posttraumatic mutism.2 The persis-
tence of aphony in our patient suggests that 
apt from the damage to brainstem pho-
natory control systems, other factors oper-
ated to produce the phonautic 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 ven-
tilatory mechanics found in our patient 
could further aggravate his phonautic prob-
lem. A correlation was found between 
the improvement in speech intelligibility 
during fever and the occurrence of thermal 
hyper- 
v 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 
function.6 Most probably this complex 
activity depends, as other motor activities 
do, on an optimal level of arousal for 
its proper execution. It is proposed that 
the elevation of body temperature, through 
it 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 

centralisation. Search for the possible existence of a 
relationship between body temperature and 
phonation in other aphasis 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 mono-
amine oxidase inhibitors and tricyclic anti-
depressants

Sir: Monoamine oxidase inhibitors and tri-
cyclic 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 ther-
apotic 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 depressed woman took an 
overdose of monoamine oxidase inhibitors 
(MAOI) and tricyclic antidepressants (TCA). The medications taken were tranxyla-
cypromine (40 mg), clomipramine (150 mg) 
and trazadone (400 mg). Cylazine (150 mg) 
and oxazepam (60 mg) were also taken. 
Four hours after taking the drugs, the 
patient had a grand mal seizure, went into a 
respiratory failure and was intubated and 
vентilated. On examination the patient was