bral grey matter was normal. The posterior borders of these lesions were formed by the caudate nuclei, the anterior parts of the internal capsules being spared. The lesions reached the parietal periventricular areas. The necrotic areas were characterised by extensive loss of myelin with myelinladen gitter cells on both sides a substantial num-
ber of neurons in the ventromedial parts of the putamen and in the nuclei accumbens showed central chromatolysis. No signs of neuronal degeneration were found in the rostral parts of the putamen or in the caudate nuclei on both sides.

The extensive coagulative necrosis of cen-
tral white matter with spongiosis, sparing the U fibres, is in agreement with the diagnosis of delayed neuronal death. The presence of gitter cells and reactive glio-
sis is compatible with onset of the disease about six to eight weeks before the patient’s death. The absence of CSF pleocytosis at the onset of delayed necrotising leuko-
cephalopathy suggests that a CNS leukemic relapse was not important in its evolution.

In the present case all symptoms of ante-
terior cingulate syndrome as described by Cummings were present, but the clinical picture did not deteriorate to the most severe form of anterior cingulate syn-
drome—that is, the akinet mutism strik-
ingly characteristic of Cairns et al. as “a state of motionless, mindless wakefulness”.4 In our patient verbal communication remained possible by writing and reading excluding global aphasia as the cause of his mutism.

In children mutism is a rather non-spe-
cific symptom, which may occur in a wide variety of neurological diseases. The present finding suggests that in children with ante-
terior cingulate syndrome mutism is associ-
ated with an extreme loss of initiative. This selectively affects the modalities of commun-
ication of which spontaneous speech and spontaneous writing seem to be the most vulnerable.

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Unilateral auditory hallucinations in a boy with ipsilateral conductive hearing loss

Unilateral auditory hallucinations in adults and adolescents are associated with con-
tralateral CNS lesions and ipsilateral periph-
eral lesions.1 A nine year old boy was admitted to the child psychiatry inpatient unit after exhibit-
ing self destructive and aggressive behaviour for two weeks at school. He attempted to burn himself on a radiator and to jab a pen-
cil into his head. He regained his head, punched one teacher, and threw a chair at another without provocation.

Four years before admission he had tried to set a tree on fire. Two years before admis-
sion he had tried to damage and to set fire to his bedroom on fire. He often shoppedlised from the neighbourhood shop and often fought siblings and peers. One month before admission his mother’s intoxicated boyfriend hit him hard on the chest, leaving marks.

He was conceived when his father raped his mother. The umbilical cord was tightly wrapped around his neck at birth. His Apgar scores were 6 and 8. At birth he had a cleft soft palate, hyperbilirubinemia, and sepsis with Streptococcus viridans and diphtheroides. At two and a half years of age his cleft palate was repaired. Later myringotomy tubes were placed to treat multiple episodes of otitis media. At four years of age a right hydrocele was repaired. At four and a half years of age audiological examination disclosed a right sensorineural hearing loss, and a mild to moderate right conductive hearing loss. Both parents used street drugs. His father is a violent, abusive man who has not been living in the family for many years. His mother was abandoned by her own mother and was raised in orphanges. His mother had psychiatric admissions to hospital beginning in childhood, resulting in treatment with an antidepressant and a psychoactive drug. No other family member was reported to have a hearing loss.

On mental status examination he had a moderately severe articulation deficit. He heard the voices of devils outside his head at night and during the day telling him to jump off the building and to kill himself. He heard the voices only in his right ear. He saw faces of devils, a rag doll that his mother had given him, Jason, a character in a horror movie, cockroaches that turned into red dev-
ils, and hell that looked like a fire sur-
ronded by cockroaches.

His right tympanostomy tube was dismis-
pacted from the cerumen and was removed. His left myringotomy tube was patent and in place. The visual and auditory hallucinations continued unabated. Audiograms showed normal left hearing and a mild right conductive hearing loss at 1 kHz and 4–8 kHz and improved to normal hearing at 1.5–3 kHz. His EEG was normal.

The visual and auditory hallucinations and suicidal and homicidal ideation stopped a few days after starting treatment with the antipsychotic drug molindone (5 mg twice daily). He denied having visual or auditory hallucinations during the subsequent treatment with molindone and, later, with haloperidol. His discharge diagnoses were brief reactive psychosis and conduct disorder.

This case report is reminiscent of the occurrence of schizophrenic in elderly people with peripheral auditory disease. Typically, unilateral hearing loss is associ-
ated with auditory hallucinations in adults with severe hearing loss. Our case is novel because the patient is a child with a mild conductive hearing loss affecting only a portion of the acoustic frequencies which are significant in speech perception.

The disappearance of auditory hallucina-
tions in our patient coincided with the reso-

nution of his psychosis when he received antipsychotic medication.

The occurrence of hallucinations has been considered to be caused by (1) stimulatory phenomena in the CNS—for example, elec-
tric excitation by electrodes, seizures, myoclonus,1 and pharmacological agents,1 or (2) release of inhibitory phenomena on sensory neurons—for example, sensory deprivaton,2 the visual hallucinations in blindness (the Charles Bonnet Syndrome), and the phantom limb hallucinations after damage to peripheral nerves in amputees.3 We propose that mild conductive hearing loss, even if limited to only a portion of the acoustic frequencies which are significant in speech perception, may predispose vulner-
able children to develop ipsilateral auditory hallucinations during psychotic episodes.

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4 Ketter TA, Andreasen PJ, George MS, et al. Anterior parietal limbic activation in a pro-
caine-induced emotional stimulation paradigm: An event-related fMRI experi-

MATTERS ARISING

Low dose interferon-a is safe in patients with myasthenia gravis

Piccolo and colleagues1 have recently reported that interferon-a (IFNα) could induce myasthenia gravis in patients with hepatitis C virus infection and briefly reviewed the incidence of patients with myasthenia gravis induced by IFNα. The therapeutic efficacy of IFNα treatment in myasthenia gravis has been shown in experi-
mental studies.2 We performed a prospective study that aimed to evaluate the efficacy of IFNα (Roferon-2b, (3 µa subcutaneously three times a week, for six months)) in seven
myasthenic patients. No appreciable clinical deterioration or myasthenic crisis was noted during the IFNα treatment; clinical grading according to modified myasthenia gravis scoring showed improvement in four, no sig-
nificant change in two patients, and the score was worse than that before treatment in one patient. Findings from single fibre
Matters Arising

and correlated autoantibodies reported and myasthenic symptoms that develop during treatment. Thus, inhibition attributed persisting and whether activity disappears.

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Habitual snoring, sleep apnoea, and stroke prevention

I read with interest the recent review articles on stroke prevention by Khaw1 and Bronner et al2 and was surprised that snoring and sleep apnoea were not mentioned as risk factors for stroke. Several cross sectional and case-control studies have shown that habitual snoring represents an independent risk factor for stroke, with odds ratios ranging from 2-1 to 3-5. Based on a 10%-30% prevalence of habitual snoring and a 2%-4% prevalence of sleep apnoea3 the risk of stroke associated with habitual snoring may be of the same magnitude as the risk associated with diabetes mellitus and dyslipidaemia.4

Several physiological aberrations associated with obstructive apnoeas including hypoxaemia, cardiac arrhythmias, and pronounced variations in blood pressure and cerebral blood flow may contribute to the increased risk of stroke in patients with disordered sleeping.

Although it is not known if treatment of sleep apnoea reduces the risk of stroke, it seems to reduce vascular morbidity and mortality.5,6 As sleep apnoea is a treatable condition, sleep apnoea and habitual snoring should be included in discussions of modifiable risk factors of stroke.

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The basis for behavioural disturbances in dementia

In her editorial, The basis for behavioural disturbances in dementia, Esiri reviews some possible neurochemical and pathological correlates of behavioural changes in dementia with particular reference to alterations in noradrenergic, serotonergic, and dopaminergic transmission.7 These data, offering some pathophysiological explanations for behavioural disorders in demented subjects are of great current interest but unfortunately, this review is not complete and even presents some incorrect impressions that deserve the following comments:

Noradrenaline

Despite substantial neuronal loss in the noradrenergic locus coeruleus in Parkinson’s and Alzheimer’s diseases,8,9 markers of noradrenaline metabolism in brain tissue are reported to be unchanged or increased.1 A non-significant increase in Alzheimer type senile dementia has been reported by Yates et al10 whereas most other authors demonstrate a significant decrease in noradrenaline values ranging from 29% to 52% of controls in the striatum, hypothalamus, and several cortical areas.11 In non-cortical projection areas there was no evident decrease in noradrenaline.12

On the other hand, Zubenko et al13 found a specific and pronounced loss of noradrenaline in the middle frontal area, superior temporal cortex, and hippocampus (90% to 95%) in demented patients with major depression along with a relative preservation of choline acetyltransferase activity in several subcortical regions. These data in patients with Alzheimer’s disease suggest that dysfunction of the noradrenergic system is also related to mental changes and depression in parkinsonian patients.8

Serotonin

Degeneration of serotoninergic systems in both Alzheimer’s and Parkinson’s disease results from neuronal losses in the dorsal raphe nuclei ranging in Alzheimer’s disease from 10% to 76%,14,15 and in Parkinson’s disease containing many neurofilibrillary tangles that may involve up to 90% of the neurons;16 cell depletion in Parkinson’s disease averages 40% to 40%.17 This contrast to the selective, dopaminergic, and cholinergic reduction of 5-HT and 5-HIAA in some cortical and hippocampal regions of Alzheimer disease brain ranging from 54% to 77% and a reduction of 5-HT, its

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