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 myelin laden gitter cells. On both sides a substantial number 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 rostrolateral parts of the putamen or in the caudate nuclei on both sides.

The extensive coagulative necrosis of central white matter with spongiosis, sparing the U fibres, is in agreement with the diagnosis of delayed necrotising leukoencephalopathy.[1] The presence of gitter cells and reactive gliosis is compatible with onset of the disease about six to eight weeks before the patient's death. The absence of CSF pleiocytosis at the onset of delayed necrotising leukoencephalopathy suggests that a CNS leukaemic relapse was not important in its evolution.

In the present case all symptoms of anterior cingulate syndrome as described by Cummings[2] were present, but the clinical picture did not deteriorate to the most severe form of anterior cingulate syndrome—that is, the akinetic mutism strikingly characterised by 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-specific symptom, which may occur in a wide variety of neurological diseases.[5] The present finding suggests that in children with anterior cingulate syndrome mutism is associated with an extreme loss of initiative. This selectively affects the modalities of communication of which spontaneous speech and spontaneous writing seem to be the most vulnerable ones.

We are grateful to Dr RM Egeler for critically reading the text and for helpful comments.

CORIENE E CATSMAN-BERREVOETS
HUGO R VAN DONGEN
Department of Child Neurology
JOHAN M KROS
Department of Pathology, Neuropathology Laboratory, University Hospital Rotterdam-Sophia, Rotterdam, The Netherlands

Correspondence to: Dr CE Catsman-Berrevoets, Department of Child Neurology, University Hospital Rotterdam-Sophia, dr Molewaterplein 60, 3015 GJ Rotterdam, The Netherlands.

1 Gutling E, Landis T, Kleihues P. Akinetic mutism in bilateral necrotizing leucoencephalopathy after radiation and chemotherapy: electrophysiological and autopsy findings. *J Neurol* 1992;**239**:125–8.
2 Cummings JL. Frontal-subcortical circuits in human behavior. *Arch Neurol* 1993;**50**: 873–80.
3 Luteijn F, Van der Ploeg FAE. *GIT, Groninger intelligence test.* Lisse: Swets en Zeitlinger, 1989.
4 Cairns H, Oldfield R, Pennybacker JB, Whitteridge DC. Akinetic mutism with an epidermoid cyst at the third ventricle. *Brain* 1941;**64**:275–90.
5 Van Mourik M, Van Dongen HR, Catsman-Berrevoets CE. The many faces of acquired neurologic mutism in childhood. *Pediatr Neurol* 1997(in press).

Unilateral auditory hallucinations in a boy with ipsilateral conductive hearing loss

Unilateral auditory hallucinations in adults and adolescents are associated with contralateral CNS lesions and ipsilateral peripheral lesions.[1]

A nine year old boy was admitted to the child psychiatric inpatient unit after exhibiting self destructive and aggressive behaviour for two weeks at school. He attempted to burn himself on a radiator and to jab a pencil into his hand. He banged 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 admission he had tried to strangle a cat and to set his bedroom on fire. He often shoplifted from the neighbourhood store 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, hyperbilirubinaemia, and sepsis with *Streptococcus viridans* and *diphtheroids*. At two and a half years of age his cleft palate was repaired and bilateral 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 evaluation disclosed a mild left conductive 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 involved with the family for many years. His mother was abandoned by her own mother and was raised in orphanages. His mother had psychiatric admissions to hospital beginning in childhood, resulting in treatment with dopamine receptor blocking drugs. 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 devils, and hell that looked liked a fire surrounded by cockroaches.

His right myringotomy tube was disimpacted 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 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 schizophrenia in elderly people with peripheral auditory disease. Typically, unilateral hearing loss is associated with auditory hallucinations in adults with severe sensorineural 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 hallucinations in our patient coincided with the resolution 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, electrical excitation by electrodes, seizures,[2] schizophrenia,[3] and pharmacological agents,[4] or (2) release of inhibitory phenomena on sensory neurons—for example, sensory depravation,[5] the visual hallucinations in blindness (the Charles Bonnet syndrome), and the phantom limb hallucinations after damage to peripheral nerves in amputees.[2] 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 vulnerable children to develop ipsilateral auditory hallucinations during psychotic episodes.

JAMES R BRASIC
RICHARD PERRY
Division of Child and Adolescent Psychiatry, Department of Psychiatry, Bellevue Hospital Center, and the New York University School of Medicine, New York, New York, US

Correspondence to: Dr J R Brasic, Department of Psychiatry, New York University School of Medicine, 550 First Avenue, New York, New York 10016–6497 USA.

1 Toulouse E. Les hallucinations unilatérales (unilateral hallucinations). *Gazette de Hôpitaux* 1892;**65**:609–18.
2 Keshavan MS, David AS, Steingard S, Lishman WA. Musical hallucinations: a review and synthesis. *Neuropsychiatry, Neuropsychology, and Behavioral Neurology* 1992;**5**:211–23.
3 Silbersweig DA, Stern E, Frith C, *et al.* A functional neuroanatomy of hallucinations in schizophrenia [letter]. *Nature* 1995;**378**: 176–9.
4 Ketter TA, Andreason PJ, George MS, *et al.* Anterior paralimbic mediation of procaine-induced emotional and psychosensory experiences. *Arch Gen Psychiatry* 1996;**53**:59–69.
5 Petrella F, Petrella AM. Le esperienze di privazione sensoriale alla luce di alcuni problemi neuropsichiatrici (the experience of sensory deprivation in the light of some neuropsychiatric problems). *Rassegna di Neuropsychiatria e Scienze Affini* 1968;**22**: 424–56.

MATTERS ARISING

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

Piccolo and colleagues[1] have recently reported that interferon-α (IFNα) could induce myasthenia gravis in a patient 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 experimental studies.[2] We performed a prospective study that aimed to evaluate the efficacy of IFNα (Roferon-2b, (3 mu 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 significant change in two patients, and the score was worse than that before treatment in one patient. Findings from single fibre