These findings offer support for the logic of broad-based treatment procedures, and suggest that a cognitive dimension may be used to complement traditional behavioural methods in the management of paedophilia.

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Musical Hallucinations in a Deaf Elderly Woman

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Musical hallucinations had caused an 86-year-old deaf woman to become anxious and depressed. She was admitted, and tranquillising and hypnotic drugs afforded some slight improvement. The use of her hearing aid to increase ambient noise levels reduced the intensity of the hallucinations, and the patient improved.

References have rarely been made in the English scientific literature to formed auditory hallucinations related to acquired peripheral deafness (Colman, 1894; Rhein, 1913; Rozanski & Rosen, 1952). Ross *et al* (1975) presented two further cases, both with musical hallucinations, and commented that all the described cases conformed to an almost stereotyped pattern. We report a further case.

Case report

An 86-year-old married woman was admitted to our unit with a two-month history of increasing anxiety, agitation, and restlessness, and constant depression with loss of interest, insomnia, poor appetite, and early morning wakening. She complained bitterly that she had been hearing music for the previous nine months. Its onset had been sudden, and she thought that it had first occurred after underoing audiometry and wax aspiration at an ENT outpatient clinic.

The hallucinations consisted of an unaccompanied and unknown male baritone voice singing melodies either to 'la la' or to the words of traditional Scottish folk-songs. She heard them with her left ear only, describing the sounds as being located at times within her ear, and at times outwith her body. They were annoyingly repetitive: lines or verses repeated over and over again, the songs occasionally being sung *in toto*. They were fairly constantly heard, with infrequent short pauses, could not be stopped or altered by the patient, and were more intense at night and at other quiet times. They decreased in intensity with her (left) hearing aid *in situ* and switched on.

The patient was fully aware of the hallucinatory nature of her experiences. She was only marginally temporarily disorientated, with a moderately poor short-term memory, an inability to perform complex arithmetical subtractions, and a poor knowledge of current affairs. There was no previous psychiatric history or relevant family history.

The patient was the youngest in a family of five children and was raised at home by her parents, both jute workers. She left school aged 14 to enter the jute industry and married at 20. The union was happy but childless. She retired from full-time work at 60 but continued to work as a home-help for another 10 years. Her husband was killed in a traffic accident when the patient was aged 67 and she remarried aged 73, her second husband being 15 years younger. Some five years previously they had moved to their present abode.

Total deafness, of unknown cause, had been present in her right ear since early childhood. For the past 20 years she had experienced tinnitus and increasing perceptive deafness on the left side. Two years before admission she had been issued with a hearing aid.

At age 67 she sustained a head injury, with a five-minute loss of consciousness, and had post-traumatic amnesia, of uncertain duration, from which she made an uncomplicated recovery.

Routine general physical examination was unremarkable. The patient exhibited a degree of ambidexterity, but the right hand was considered to be dominant. There was no left handedness in the family. Neurological examination was normal, except that some nominal dysphasia was exhibited, in particular with increasingly uncommon objects (for the patient, e.g. a drawing pin), and she confabulated when identifying these. Drawing a clockface from memory caused some problems and was abandoned. There was, however, no evidence of visual field neglect. Some difficulties in spatial orientation were also noted, with the patient inverting the arrowheads in drawing a series of parallel arrows, in all the cardinal directions. A slight, but inconsistent, right–left disorientation was manifest on tests of finger agnosia.

On the Kendrick Battery both object-learning and digit-copying scores were low (age-scaled quotients = 70 and 60 respectively), placing the patient in the dementing category. A design could not be drawn correctly from memory on the Benton Visual Retention Test, again suggestive of a significant deficit. With both these tests there were some perseverative responses. Further drawing tests suggested diffuse impairments in functioning.

Routine haematological and biochemical investigations were normal. TPHA was negative. Chest X-ray showed cardiomegaly and signs consistent with incipient cardiac failure. Skull films revealed increased right frontal bone density, but computerised tomography (CT) showed only a minor degree of generalised cerebral atrophy. An EEG produced a low-amplitude, poorly rhythmic recording, and there was evidence of right hemisphere dysfunction involving mainly the frontotemporal area.

ENT examination confirmed the existing hearing loss but could detect no new pathology.

The patient was treated with thioridazine in low, divided dosage (25 mg thrice daily) and a short-acting benzodiazepine hypnotic (temazepam 10 mg) with some little improvement. The use of her hearing aid on the affected side, with an increase in ambient noise levels, resulted in a marked decrease in her hallucinosis and on discharge, seven weeks after admission, her agitation and dysphoria had settled. Follow-up in the community, six months after discharge, revealed that the patient was physically well. She remained active and affectively settled and was continuing to use her hearing aid. She reported her hallucinations still present but less bothersome.

Discussion

The history presented by our patient conformed closely to the almost stereotyped pattern described by Ross *et al* (1975), Miller & Crosby (1979) and Hammeke *et al* (1983) of highly organised, vivid and intricate musical hallucinations consisting of a voice or voices, or an instrument, band or orchestra, which almost always reflect past musical memories. Such hallucinations are generally preceded by a long history of progressive unilateral or bilateral sensorineural deafness together with tinnitus, are experienced predominantly in the deaf or deafer ear, and are often of acute onset at a time of a further decrement in hearing. The ear or head is commonly named as the site of supposed origin. Most patients seem to hear a repetition of the same melody or melodies, which begins on awakening and continues throughout the day. Awareness of the music varies with the sufferer's attentiveness, being intensified by mental inactivity and with low ambient noise levels. They can be very disturbing, but are sometimes pleasing, with the patient harmonising or singing along. In the cases described by Miller & Crosby (1979) and Hammeke *et al* (1983), the patients were able consciously to alter the tune or its speed or volume.

Our patient's EEG and CT findings echo those of Miller & Crosby (1979) and Hammeke *et al* (1983), whose patients all showed EEG evidence of abnormalities in the temporal regions, and in whom CT revealed findings of mild diffuse cerebral atrophy. Neuropsychological testing in our patient, although not exhaustive, suggested diffuse impairments in functioning similar to those found by Hammeke *et al* (1983), one of whose patients showed evidence of mild, probably diffuse, cerebral dysfunctioning with impairment in memory and learning tasks.

In none of the reported cases have these hallucinations been associated with a psychosis or a previous history of mental illness.

There is disagreement about the possible anatomical substrate for the phenomenon. Rozanski & Rosen (1952) state that musical hallucinations have been described in various organic diseases, including tumours and vascular lesions, involving the temporal lobe, as well as in epilepsy, diffuse cerebral diseases, and in drug-induced intoxicated states. They are at pains to point out, however, that cerebral disorders seem to play no part in the causation of their patient's condition which, they assumed, arose from

pathological changes of the aural end-organ alone and they cite, as a point of differentiation, the paroxysmal nature of the hallucinations in the formed conditions compared with the persistent nature of them in the latter. Ross *et al* (1975) hold to this view, citing similar phenomena produced by other end-organ disease, in particular visual hallucinations associated with blindness – the Charles Bonnet syndrome. Their main postulate is that the formed hallucinations result from distortions in the normal processing of sensory information so that abnormal perceptions occur centrally, and that such distortion need not imply neuronal damage.

Hammeke *et al* (1983), in discussing these views, use the construct of 'sensory deprivation' as the primary neurophysiological mechanism so that normally, sensory input is said to suppress much non-essential information but, in conditions of reduced sensory input, disinhibition of perception-bearing circuits may occur, thereby 'releasing' perceptual traces, including previously acquired memories, which are then re-experienced. Other investigators, they say, argue for a combination of peripheral and central dysfunction, supported by the fact that these hallucinations are shown to occur most frequently in an elderly population. Direct evidence of central dysfunction is provided by EEG abnormalities in the majority of cases studied, CT abnormalities in all cases studied, and neuropsychological abnormalities in their own patients. In support, our patient has abnormal findings in all these spheres also. Whether this dysfunction is central to, contributory, or coincidental to the phenomenon is as yet unknown.

Although rarely reported, Ross *et al* (1975) predicted that the incidence of this disorder might well be higher than generally appreciated since patients' self-reports were low because of the fear that the symptom might suggest psychiatric disorder. This prediction was found to be correct (Ross, 1978).

Interestingly, although our patient was unable to alter or abolish her hallucinations consciously, the use of her hearing aid to increase the ambient noise level resulted in better control and tolerance of her hallucinosis and this echoes the experiences of other patients who could suppress their hallucinations by playing the television or radio loud. Nevertheless, this result may be fortuitous since a measure of tolerance to the experience seems to develop in time (Ross, 1978). In our own experience and that of Miller & Crosby (1979), there would seem to be a place for pharmacological treatment using tranquillising or hypnotic drugs. However, Hammeke *et al* (1983) report that anticonvulsant, antipsychotic, and vitamin supplement therapy fail to produce major benefits.

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Social Phobia Secondary to Pathological Sweating

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An individual with agenesis of the corpus callosum associated with recurrent severe sweating and hypothermia developed a social phobia. Phobias may be adaptations to real and potentially dangerous physiological events.