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.

References

COLMAN, W. S. (1894) Hallucinations in the sane associated with local organic disease of the sensory organs, etc. *British Medical Journal*, *i*, 1015–1017.

HAMMEKE, T. A., MCQUILLEN, M. P. & COHEN, B. A. (1983) Musical hallucinations associated with acquired deafness. *Journal of Neurology, Neurosurgery and Psychiatry*, **46**, 570–572.

MILLER, T. C. & CROSBY, T. W. (1979) Musical hallucinations in deaf elderly patients. *Annals of Neurology*, **5**, 301–302.

RHEIN, J. H. W. (1913) Hallucinations of hearing and diseases of the ear. *New York Medical Journal*, **97**, 1236–1238.

ROSS, E. D., JOSSMAN, P. B., BELL, B., *et al* (1975) Musical hallucinations in deafness. *Journal of the American Medical Association*, **231**, 620–621.

ROSS, E. D. (1978) Musical hallucinations in deafness revisited. *Journal of the American Medical Association*, **240**, 716.

ROZANSKI, J. & ROSEN, H. (1952) Musical hallucinosis in otosclerosis. *Confina Neurologica*, **12**, 49–54.

*George W. Fenton, MB, FRCP(Ed), FRCPsych, MRCP(Lond), *Professor of Psychiatry, University of Dundee, Ninewells Hospital and Medical School, Dundee DD1 9SY*; Duncan A. McRae, MB, ChB, *Registrar in Psychiatry, Royal Dundee Liff Hospital, Dundee DD2 5NF*

**Correspondence*

British Journal of Psychiatry (1989), **155**, 403–405

Social Phobia Secondary to Pathological Sweating

MATTHEW J. EDLUND

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.

Agenesis of the corpus callosum in association with recurrent hypothermia and attacks of sweating and chills, also known as Shapiro's syndrome, is a rare disorder whose psychiatric sequelae have yet to be reported (Shapiro *et al*, 1969; Noel *et al*, 1973). The cause of the recurrent hypothermia is unclear. The case we report here is of a man with agenesis of the corpus callosum whose hypothermia was not recurrent, as it has been in most previously reported cases (Johnson & Jones, 1985), but was constant. Eventually, severe sweating in times of emotional stress led him to avoid certain social encounters in which others might scrutinise him.

Case report

The patient had a normal course until the age of 19, when he developed severe sweating attacks that would lower his core body temperature 3 °C or more at a time. Simultaneously, he lost the ability to keep his body temperature within normal range. In his early 20s, he spent a total of three years as an in-patient at the National Institute of Health, during which time his temperature would sometimes drop as low as 21 °C, and he suffered frequent attacks of status epilepticus. His seizures were felt to be secondary to his very low temperature. Except for diffuse slowing at times of low temperature, his EEG was normal, and he never showed any clinical evidence of the syndrome of complex partial seizures. His seizures and to some degree his hypothermia were finally controlled on phenobarbitone (180 mg) and cyproheptadine (12 mg) daily, allowing him to leave the hospital. However, he found work difficult, as excessive sweating would result in drops of body temperature, with accompanying cognitive deficits.

He entered our out-patient psychiatric clinic complaining that he could not find a girlfriend. When first seen by us, he still had difficulty controlling his temperature. He would wake up in the morning with his temperature in the high 20s °C, and then take a series of hot baths to increase his temperature to over 30 °C, when his ability to speak coherently or do simple calculations would improve appreciably. However, if his temperature fell below 30 °C, he experienced cognitive deficits and generalised weakness.

A gregarious individual before the onset of illness, the patient remained sociable for some years. Occasionally, he suffered sweating attacks for no apparent reason. However, some years after his long period in hospital, he began to note that emotional stress would sometimes precipitate such attacks, something which had never occurred in hospital. Within a period of five to ten minutes, he would sweat so much he often lost up to 2 kg body weight, while his temperature might decrease 3 °C or more. When he finally stopped sweating, he became exceedingly sluggish, and was unable to function mentally or physically. He began to fear the recurrence of seizures if the sweating attacks became too severe. The emotional stressors that provoked the sweating were going out with women and queuing to pay in cafeterias or shops, situations where he could feel people were watching him.

The patient was seen in group and individual therapy. In group therapy, he would sometimes feel 'emotional' and start to sweat. Embarrassment at his appearance, and the further scrutiny he felt the group would then give him, caused him to sweat even more. The patient found himself becoming more self-conscious of his sweating, and discovered it was progressively harder to engage in social situations where others might be watching him.

Nonetheless, the patient craved social contact. Over time, however, group therapy grew intolerable, and exposure therapy for his fear of queuing in cafeterias and grocery shops was unacceptable to him because of the physiological threat to his temperature and seizure control. He appears to have been socially conditioned to his phobia, which led to his social avoidance and fears of sweating attacks.

His social phobia is unusual, in that it began at a relatively late age in an otherwise normal individual, more clearly linking its development to a socially conditioned response to his pathological sweating. Pharmacological intervention had to be ruled out, as beta-blockers had not helped control his temperature in the past, and his present regimen of phenobarbitone and cyproheptadine was the only one of many interventions that had forestalled what had been a life-threatening condition.

Discussion

There are many organic causes of anxiety states (Goldberg, 1988). There are also unusual phobias that may be directly related to organic causes (Pratt & McKenzie, 1958; Marks, 1981). However, there has been little to link organic disorders to social phobia. In DSM–III–R (American Psychiatric Association, 1987), social phobia is defined as a persistent fear of one or more situations in which the person is exposed to possible scrutiny by others and fears that he or she may say something or act in a way that will be humiliating or embarrassing. Our patient avoided these situations not merely because they were embarrassing, but because this embarrassment further led to pathological sweating with potentially severe physiological repercussions. This fear of social situations fed on itself, as the patient would sweat more, become more embarrassed, sweat yet more, and then his temperature would drop so low that he sometimes felt he would not have the physical energy to escape the sweat-provoking situations.

Although this case is highly unusual, it does provoke consideration of some theoretical difficulties in defining phobias. First, there is the possibility that there are some people with intervening medical conditions for whom social phobias may in some broad sense be adaptive, that is, they will avoid situations where anxiety or embarrassment might cause considerable physiological difficulties. Originally, our patient did not have such a physiological

response to social situations, but developed a phobia to them over a period of approximately one year. Before developing this social phobia, he could face similar social situations with equanimity.

Secondly, this case points up the difficulty of easily dismissing patients' claims that their avoidance of certain situations may be rationally justified by the overwhelming anxiety and strong emotion caused by their phobias and fears. There is now considerable evidence linking panic disorder and increased cardiovascular mortality (Coryell *et al*, 1982), and data that this may possibly be related to subgroups of patients with covert organic disorders are just beginning to appear (Kahn *et al*, 1987). Although the most frightening experience of a phobia may be the fear itself, the physiological sequelae of such fears may also be important. Thus, although phobias may begin as irrational fears, they may later exacerbate real physiological difficulties, that may, as in this case, provide 'rational' reasons to avoid experiences that might potentially cause physical harm.

References

AMERICAN PSYCHIATRIC ASSOCIATION (1987) *Diagnostic and Statistical Manual of Mental Disorders* (3rd edn, revised) (DSM-III-R). Washington, DC: APA.

CORYELL, W., NOYES, R. & CLANCY J. (1982) Excess mortality in panic disorder. *Archives of General Psychiatry*, **37**, 701–703.

GOLDBERG, R. J. (1988) Anxiety in the medically ill. In *Principles of Medical Psychiatry* (eds A. Stoudemire & B. S. Fogel), pp. 177–204. New York: Grune & Stratton.

KAHN, J. P., DRUSIN, R. E. & KLEIN, D. F. (1987) Idiopathic cardiomyopathy and panic disorder: clinical association in cardiac transplantation. *American Journal of Psychiatry*, **144**, 1327–1330.

JOHNSON, M. H. & JONES, S. N. (1985) Status epilepticus, hypothermia and metabolic chaos in a man with agenesis of the corpus callosum. *Journal of Neurology, Neurosurgery and Psychiatry*, **48**, 480–483.

MARKS, I. (1981) Space "phobia": a pseudo-agoraphobic syndrome: fear of absent visuospatial support (open spaces) and of falling. *Journal of Neurology, Neurosurgery and Psychiatry*, **44**, 387–391.

NOEL, P., HUBERT, J. P., ECTORS, M., *et al* (1973) Agenesis of the corpus callosum associated with relapsing hypothermia. *Brain*, **96**, 359–368.

PRATT, R. T. & MCKENZIE, W. (1958) Anxiety states following vestibular disorders. *Lancet*, *ii*, 347–349.

SHAPIRO, W. R., WILLIAMS, G. H. & PLUM, F. (1969) Spontaneous recurrent hypothermia accompanying agenesis of the corpus callosum. *Brain*, **92**, 423–436.

Matthew J. Edlund, MD, *Director, Consultation Liaison Psychiatry, Rhode Island Hospital, APC Building, 9th Floor, 593 Eddy Street, Providence, RI 02903, USA*

British Journal of Psychiatry (1989), **155**, 405–408

A Family with Alzheimer's Disease

S. V. PATEL

A family with clinically diagnosed Alzheimer's disease in three siblings is described. The inheritance of Alzheimer's disease in this pedigree is consistent with autosomal dominant inheritance.

Historically, Alzheimer's disease has been divided into pre-senile and senile dementia of Alzheimer type (PDAT and SDAT respectively), depending upon whether the age at onset of the illness is before or after 65. While many investigators (e.g. Terry & Katzman, 1983) hold the view that the disease is essentially identical irrespective of the age at onset, this is by no means universally accepted, and Sourander & Sjogren (1970) believe that distinctions can be made. However, PDAT and SDAT may not be a unitary disorder, although the clinical features may be quite similar: neurohistological (Tagliavini & Pilleri, 1983) and neurochemical (Rossor *et al*, 1984) differences point towards a distinction between the two.

Various modes of transmission have been suggested, including multifactorial inheritance (Sulkava *et al*,