Electrophysiological work in monkeys has shown that there are at least two areas of the brain in which neurones are selectively responsive to faces. One of these is a cortical area, the superior temporal sulcus (Bayliss et al, 1985). Neurones here respond with a shorter latency than in the other area, the amygdala (Leonard et al, 1985). This finding has been interpreted as indicating additional processing of sensory information, which, in view of the role of the amygdala in the regulation of social and emotional behaviour (Thompson et al, 1977), probably involves the incorporation of social and emotional cues necessary to identify a particular face as that of a genuine close relative. It is this function which appears lost in Capgras syndrome.

There is an increasing body of evidence that the amygdala may be subject to damage in the early stages of the commonest cause of dementia, Alzheimer's disease. The pathological changes are most severe in the hippocampus and amygdala, and these regions appear to be affected early in the course of the disease (Brun, 1985). Moreover, we have described a number of cases of Alzheimer's disease in which there were marked reductions of the cholinergic innervation of the amygdala in the absence of the characteristic loss of such innervation of the cerebral cortex (Palmer et al, 1986). While it is difficult to extrapolate the results of discrete brain lesions to patients with diffuse brain damage, the case reported by Dr Lipkin and other cases reviewed by him may reflect early damage to the amygdala in dementia.

This syndrome illustrates also that the symptoms of dementia which are most difficult to deal with, both for the patient's relatives and health care professionals, are not necessarily the cognitive impairments, but the deterioration in social behaviours. At least some of these may be a consequence of subcortical disease, and it is now important to investigate these behaviours in dementia and to follow this by pathological and neurochemical studies.

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References

BAYLISS, G. C., ROLLS, E. T. & LEONARD, C. M. (1985) Selectivity between faces in the responses of a population of neurones in the cortex in the superior temporal sulcus of the monkey. *Brain Research*, 342, 91-102.

BRUN, A. (1985) The structural development of Alzheimer's disease. Danish Medical Bulletin, 32, Suppl 1, 25–27.

LEONARD, C. M., ROLLS, E. T., WILSON, F. A. W. & BAYLISS, G. C. (1985) Neurones in the amygdala with responses selective for faces. Behavioural Brain Research, 15, 159-176. MEADOWS, J. C. (1974) The anatomical basis of proposagnosia. Journal of Neurology, Neurosurgery and Psychiatry, 37, 489-501. PALMER, A. M., PROCTER, A. W., STRATTMAN, G. & BOWEN, D. M. (1986) Excitatory amino acid-releasing and cholinergic neurones in Alzheimer's disease. Neuroscience Letters, 66, 199-204.

THOMPSON, C. I., BERGLUND, R. M. & TOWFIGHI, J. T. (1977) Social and non-social behaviours of adult rhesus monkeys after amygdalectomy in infancy or adulthood. *Journal of Comparative and Psychological Psychology*, 91, 533-548.

Dysmorphophobic Avoidance

Sir: I found the behavioural therapy described by Marks & Mishan (*Journal*, May 1988, **152**, 674–678) fascinating. While the results of systematic exposure therapy in their series of five patients were encouraging, the inclusion of two patients who were deluded that they smelled challenges the definition of the term dysmorphophobia. This has usually been reserved to describe patients who complain of a subjective feeling of disfigurement in the absence of any objective abnormality (Hay, 1970; Thomas, 1984; American Psychiatric Association, 1987).

It has been argued that the condition may be primary or secondary to an underlying psychiatric illness (Thomas, 1984). While the authors state that "it feels rather Procrustean to force a diagnosis of what was 'primary'", none of their patients could be considered to be suffering from a primary diagnosis of dysmorphophobia by virtue of the presence of delusions (Thomas, 1984; American Psychiatric Association, 1987).

I was intrigued by the percentage scoring of delusional conviction, and should be interested to know how the score was determined. There is an implication (in case 1) that a score of 50% constitutes an overvalued idea. If that is so, how should the score of 80% (where 100% = total conviction and 0 = none) in case 2 be interpreted, and can this scoring system aid in distinguishing beliefs such as primary delusions from delusion-like ideas or overvalued ideas (Jaspers, 1946)?

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References

AMERICAN PSYCHIATRIC ASSOCIATION (1987) Diagnostic and Statistical Manual of Mental Disorders (3rd edn, revised) (DSM-III-R), pp 255-256. Washington, DC: APA.

HAY, G. G. (1970) Dysmorphophobia. British Journal of Psychiatry, 116, 399-406.

JASPERS, K. (1946) Delusion and awareness of reality. In General Psychopathology (7th edn). Trans. 1963 by S. Hoenig & M. Hamilton. Manchester: Manchester University Press.

THOMAS, C. S. (1984) Dysmorphophobia: a question of definition. British Journal of Psychiatry, 144, 513-516.