Capgras' Syndrome

SIR: Gibbs & Andrewes (*Journal*, December 1988, **153,** 853) contribute further to the correspondence arising from my paper (*Journal*, July 1988, **153,** 117–118), in raising the importance of distinguishing between delusional misidentification and prosopagnosia. They advance the possibility that Capgras' syndrome and prosopagnosia reflect a similar, or the same, underlying process and comment, quite correctly, that a distinction between the two is that Capgras' syndrome is considered to represent delusional misidentification of familiar others. However, they feel that, "this cannot necessarily be claimed in Dr Lipkin's case, as the presence of an organically-based dementia was established".

While welcoming further interest in these matters, I must ask why Drs Gibbs & Andrewes suggest that the delusional nature of my patient's misperceptions is inconsistent with the presence of her organically-based dementia. (This patient experienced delusional misidentification, not "delusional" in quotation marks as per their letter). There are many organic/systemic conditions in which delusional phenomena can arise and indeed delusional Capgras misidentification also, as mentioned in my original paper.

Drs Gibbs & Andrewes also say in distinguishing between prosopagnosia and delusional misidentification that, "When a labelled photograph of the misidentified individual is provided, prosopagnostics benefit from the label, while individuals with Capgras' syndrome would presumably fail to continue to recognise the individual". My original paper did not mention that this particular patient had indeed manifested delusional misidentification of photographs of her second husband and had not in any way, even before the more florid aspects of her dementing process were in evidence, obtained, as Drs Gibbs & Andrewes put it, "benefit from the label" (of photographs of her husband). Her husband related to me that she had actually torn up several photographs of him which had been on her dressing table at home, saying that they were not of him, but of an identical imposter, even though other people (whom she seemingly trusted and did not misidentify) had told her otherwise.

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Unitary Models of Schizophrenia

SIR: I read with interest the article by Frith & Done (*Journal*, October 1988, **153,** 437–443). About three years ago I formulated a unitary view on the aetiology of schizophrenia (*Journal*, August 1986, **149,** 245). Briefly, I thought that some individuals are genetically determined to have weak and vulnerable dopaminergic systems. When subjected to stresses, some of the neurons on which dopaminergic afferents make synaptic contact cease to function. To compensate for the loss, the dopaminergic fibres give off additional nerve terminals and form new D2 receptors. This overreaction gives rise to a relative increase in dopaminergic activity, and manifests itself as the positive symptoms of schizophrenia. In mild cases, the dysfunction is self-limiting and the dopaminergic transmission is able to return to its previous level of functioning, and the process is completed. This psychotic episode is named a schizophreniform reaction. In more severe cases, the dysfunction becomes chronic and progressive. It results in the actual degeneration of the dopaminergic pathways. Negative symptoms occur when the degeneration becomes extensive and when the mesocortical dopamine system is involved. But then how could this cessation of neuronal function and actual degeneration have occurred? The recent studies on the NMDA receptors may provide some clues.

The excitatory amino acids (glutamate, aspartate) act on the NMDA and non-NMDA (kainate, quisqualate) receptors. The non-NMDA receptors, when stimulated, generate fast depolarising responses until a certain level of membrane potential is reached, and then the NMDA receptors are activated. The NMDA-induced current is longer in duration, and Ca^{2+} ions enter along with Na^{+} through the ion channels. Activity of the NMDA receptors are found to be responsible for plasticity in the developing and mature CNS and long-term potentiation (LTP) in the hippocampus. In the event of prolonged and excessive stimulation, excitotoxicity comes into play when there is too much glutamate and too much calcium. It results in nerve cell damage and death. When the neurons are briefly exposed to glutamate, the cells swell transiently and then go on to recover, but when influx of calcium also occurs, the cells slowly degenerate. This excitotoxicity is postulated to be the cause of degeneration in cerebral hypoxia and ischaemia and in some neurodegenerative disorders such as Huntington's disease and Alzheimer's disease.

The excitatory amino acids are major transmitters of cortico-cortical, corticofugal and sensory systems. Corticofugal pathways run from the cortex to the basal ganglia (nucleus accumbens and caudate/putamen), the hippocampus and the dorsal lateral geniculate, the parts of the brain where highest level of NMDA receptors are found. The striatal NMDA receptors are located on the heads of the dendritic