The 'Stuck Synapse' as a Model for Schizophrenia

DEAR SIR,

The widespread nature of schizophrenic illnesses and their uniform symptomatology throughout the world seem to imply that a fundamental cerebral mechanism is disturbed in some way. Recent models of cerebral function, attempting to integrate learning theories with synaptic structure, are based on the concept of the working brain as a dynamic network of pathways selectively reinforced by feedback mechanisms (Miller, 1981). Thus, plasticity would be an essential feature of normal cerebration, and I write to suggest that a failure of such plastic mechanisms might underlie the symptoms of schizophrenia.

A loss of plasticity is most obvious in chronic schizophrenia, where poverty of ideation, blunting of affect and so on could easily be explained with such a model. However the positive symptoms of acute schizophrenia are also susceptible to explanation on this basis. In the normally working brain, familiar or repetitive sensations and perceptions are rapidly repressed, and such repression may correlate with a feeling tone labelling them as "mine". In contrast unfamiliar, and thus persistent, sensations have a feeling tone "not mine". A failure of those plastic mechanisms giving rise to the repression of familiar sensations would then result in their being wrongly perceived as "not mine". In this way might arise a large variety of passivity phenomena, as welll as the highly organised hallucinations characteristic of schizophrenia. The defining feature of a delusion of course is its lack of plasticity.

Recent studies of evoked potentials have reported abnormal persistence of the evoked response as the most consistent finding in schizophrenia (Morihisa *et al*, 1983) and it could be suggested that such results may represent a direct measure of impaired plastic mechanisms. To explain biochemical findings it would be necessary to postulate that inhibition of these mechanisms is at least partly mediated by dopamine—thus dopamine hyperactivity could be one possible aetiological factor, and a dopamine blockade could have a general, if partial, therapeutic effect in promoting plasticity.

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References

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Dysmorphophobia or Monosymptomatic Hypochondriasis?

DEAR SIR,

I would like to reply to Dr. Jenike's letter (*Journal*, March 1985, **146**, 326). The two patients he reports had single solitary delusions about their facial appearance with no apparent involvement of the rest of the personality (Jenike, 1984; Brotman & Jenike, 1984). These descriptions fulfill Munro and Chmara's (1982) criteria for monosymptomatic hypochondriacal psychosis. Although this condition superficially resembles dysmorphophobia, it can be distinguished from it.

The principal feature that separates them concerns the quality of the belief. In dysmorphophobia it is an over-valued idea (Thomas, 1984) and in monosymptomatic hypochondriacal psychosis it is a single solitary delusion (Munro & Chmara, 1982). In both the belief is invariably false, but the former is comprehensible in the context of the person's personality and life experiences, as opposed to the latter unshakeable belief which is out of keeping with the patient's social or cultural background and is the product of an internal morbid process.

The importance of such a phenomenological distinction lies in the clinical implications. Pimozide is said to be particularly effective in monosymptomatic hypochondriacal psychosis but not in dysmorphophobia (Riding & Munro, 1975). This compound is a highly specific dopamine receptor blocker but it is not available in the U.S.A. The response of Dr Jenike's subjects to doxepin and tranylcypromine is interesting.

The fifteen year old male that I reported is now eighteen years old (Thomas, 1984). He responded well to supportive psychotherapy and day attendance at the Young Persons Unit and was discharged in July 1983. A year later at the time of his exams he became extremely anxious and experienced a recurrence of his symptoms thinking that his face was mishapen. By January 1985 he was admitted to an adult psychiatric unit and received a course of six ECTs, dothiepin and flupenthixol decanoate with little impact upon this belief.

At the time of discharge he was still asking to see a plastic surgeon. This case illustrates the sensitivity (in the Kretschmerian sense) of the personality, with recurrence of symptoms at a time of stress, and the resistance of dysmorphophobia to physical treatment.

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