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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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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Early Clinical Experiences with Sulpiride

DEAR SIR,

Sulpiride is a neuroleptic, recently introduced into the UK, that appears to have interesting pharmacological properties. It has been available in France for over a decade and has been shown to be singularly free of such severe side-effects as tardive dyskinesia. Its mechanism of action has only recently come under systematic scrutiny; in small doses, it appears to block the pre-synaptic receptors, whereas at larger doses, it acts on the post-synaptic receptors. There should therefore be a differential clinical effect at different dose ranges. It has been suggested from experience in both France and Scandinavia that it has both anti-psychotic and anti-depressant properties. This action, coupled with the absence up to now of any reported case of tardive dyskinesia in association with it, makes sulpiride an important drug to study in clinical psychiatry.

Over the past year we have treated 45 patients with sulpiride; clinical records were carefully maintained so that the data could be looked at as objectively as an open study would allow. No ratings on scales were performed, except that the diagnosis of schizophrenia was cross-checked with DSM-III criteria and a global rating of the clinical state was recorded in the notes at frequent intervals. Patients were prescribed sulpiride only if clinical symptoms warranted its use, and in a significant number the drug was started because other neuroleptics had failed to produce the necessary clinical response.

Data on 37 patients (27 males and 10 females) are presented: three patients had been on this drug for too short a period to make any clinical judgement about its effect, there were four patients who had complicating organic illnesses, and one whose diagnosis was in doubt.

Eighteen patients were treated with sulpiride alone, while the remaining nineteen received the drug in addition to other psychotropic medication. Of the latter, eight patients received sulpiride and conventional neuroleptics; the rest received a variety of other drugs including benzodiazepines, antidepressants, etc. No evidence of adverse interaction with other psychotropics was detected. The dose range varied from 400 mg to 2400 mg daily.

Of the sixteen patients in whom sulpiride was prescribed for negative symptoms ten showed significant improvement, in some cases quite dramatic and unexpected. Many of these also showed a global change for the better. There were twelve patients treated for affective symptoms and ten of these showed a positive response. The five patients with tardive dyskinesia showed improvement not only in the clinical state but also an alleviation of the abnormal movements. Results were less promising in those ten patients in whom sulpiride was used specifically for acute psychotic symptoms. Less than half improved, and in two it produced a state of confusion with catatonia-like features early after the start of therapy. These quickly disappeared after the drug was stopped. In three other patients also, lack of improvement or worsening of symptoms led to the drug being withdrawn.

These early experiences with sulpiride suggest that it is a potent aid to the management of psychotic states. We have found it to have some unique properties, which need to be explored further by carefully controlled studies, especially since the results suggest that it is useful in states where conventional neuroleptics are known to be relatively ineffective, eg, schizophrenic deficit states and in patients who have affective symptomatology. The feeling of increased awareness and drive, and the improvement in concentration noted by several patients during treatment with sulpiride after they had been transferred from conventional neuroleptics, could be an important advantage with respect to social functioning in the community, and may themselves be factors in the beneficial effect on negative and depressive symptoms. Its lack of association with tardive dyskinesia makes sulpiride particularly suitable for managing patients who have developed the condition during prolonged use of conventional neuroleptics; this latter group of patients are also particularly likely to have pronounced negative symptoms. Our colleagues and I are proceeding to examine the role of this drug by controlled trials.

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