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'Slowly progressive schizophrenia'

SIR: By criticising the concept of slowly progressive or sluggish schizophrenia adopted by Snezhnevsky and his school in the USSR, Shafran *et al* (*Journal*, August 1989, **155**, 174–177) reject not only the existence of this concept but also that of simple schizophrenia. The main support for the authors' idea comes from DSM–III. They point out that "Simple schizophrenia by name has disappeared from DSM–III–R", and that the concept of latent schizophrenia which appeared in DSM–II "was, of course, radically changed for DSM–III".

It is amazing that the authors do not even mention ICD–9 which is the internationally approved classification and which describes simple schizophrenia. In the preliminary version of ICD–10 (World Health Organization, 1987) it is included again under F21.1. Black & Boffeli (1989) suggest that it might even reappear in the next version of DSM. It should not be forgotten that the rejection of simple schizophrenia in DSM was due mainly to the overdiagnosis of this disorder in the USA. Even if a diagnosis can lead to mistakes, it does not mean that it does not exist.

An important argument used by Shafran against simple schizophrenia is Schneider's concept of the first-rank symptoms: "... the Schneiderian backlash put an end to his [Bleulers'] concept of schizophrenia". Although invaluable for the diagnosis and for research purposes, the importance of first-rank symptoms is clearly overestimated. It is worth remembering Schneider's (1950) own words: "... which we call first rank symptoms not because we regard them as 'basic disturbances' but because they have special significance for the *diagnosis* both against nonpsychotic mental abnormalities, as well as against cyclothymia.... Nothing is said about the theory of schizophrenia, unlike Bleuler's basic and accessory symptoms or the primary and secondary symptoms of other authors.... First rank symptoms do not have to be present for the diagnosis of schizophrenia..."

Over-reliance on first-rank symptoms (and on positive symptoms as a whole) could lead to diagnosing only one form – paranoid schizophrenia. The characteristic 'negative' features of residual and simple schizophrenia, which are also prominent in the hebephrenic form, are at least as important in diagnosing and understanding schizophrenic illness. If more attention is paid to the variety of symptoms, some of them very subtle, more diagnostic entities will emerge. Not only Snezhnevsky has described many forms. Leonhard (1986), for instance, listed 19 subforms of schizophrenia apart from the three cycloid psychoses. Such efforts for greater diagnostic precision should not be discouraged, even if they make life more difficult.

Simple schizophrenia has the right to exist because here we see the basic symptoms which are more specific of the schizophrenic deterioration than anything else. They include the peculiar change in the personality which is not seen anywhere else in human pathology, namely that the person loses the core of their personality and is completely different in their reactions to important things in their surroundings. In this respect we could probably cite Ey *et al* (1974): "... schizophrenia is not at the beginning of the evolution but at its end".

GEORGE KIROV

Warlingham Park Hospital
Warlingham
Surrey CR3 9YR

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Temporal lobe atrophy versus open operculum in Asperger's syndrome

SIR: The report by Jones & Kerwin (*Journal*, April 1990, **156**, 570–572) concluded that the patient with Asperger syndrome had atrophy of the left temporal lobe. On the basis of the computerised tomography

scan published in their article, we suggest an alternative explanation. The enlarged distance from the temporal tip to the inferior frontal gyrus is consistent with the radiological diagnosis of 'open operculum'.

The diagnosis of open operculum suggests an underlying mechanism distinct from that which results in atrophy. Tatum *et al* (1990) suggest that an open operculum is the result of abnormal neuronal migration during the first part of gestation. They gave case reports of four infants with open opercula. In one infant, examined neuropathologically, they also detected abnormalities in the formation of the cerebral cortical gyri (i.e. macrogyria and micropolygyria). Another infant had Acardi syndrome, known to be associated with micropolygyria and heterotopias. These cerebral cortical anomalies are most likely the result of abnormalities in neuronal migration *in utero* (Larroche, 1984).

We recently detected similar cerebral cortical anomalies consistent with defects in neuronal migration in seven of 13 high functioning autistic males (Piven *et al*, 1990). We performed magnetic resonance imaging (MRI) on 13 autistic men and 13 controls (comparable on age and IQ) and found five autistic individuals with polymicrogyria, one with macrogyria, and one with schizencephaly. In another preliminary study, Berthier *et al* (1990) reported developmental cortical anomalies in two subjects with Asperger's syndrome studied with MRI. One subject was also noted to have a MRI scan consistent with opercular dysplasia.

Thus, the radiological diagnosis of open operculum is consistent with our studies of radiological abnormalities of the brain in autism and Asperger's syndrome. Further, the clinical course which would be expected with a developmental brain abnormality is more consistent with the natural course of both Asperger's syndrome and autism than that which would be expected with the occurrence of atrophy.

JOSEPH PIVEN
SERGIO STARKSTEIN

Department of Psychiatry
Johns Hopkins University School of Medicine
Baltimore, Maryland, USA

MARCELO L. BERTHIER

Institute of Neurological Research
University of Buenos Aires
Buenos Aires, Argentina

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Bipolar affective disorder and anoxic brain damage

SIR: Collins & Jacobson (*Journal*, May 1990, **156**, 736–740) discussed the case of a bipolar patient who developed mild brain damage from anoxia after attempting suicide by hanging. The discussion helped resolve many of the complexities of the diagnosis and treatment of this patient who had concomitant neurological and functional abnormalities. We suggest an additional interpretation of the patient's condition, which may contribute to an understanding of the evolution of her symptoms.

The authors reported that during the two months following the suicide attempt Ms A showed mutism and poor self-care. Although these symptoms can be attributed to depression, they occurred within a constellation of several other neurological abnormalities. Also, the psychiatric staff did not observe frank depressive symptoms. The patient was free from vegetative symptoms of depression. Her sleep and appetite were unremarkable and there was no diurnal variation in her behaviour. It is probable that the patient's depression improved for a period of a few months after her suicide attempt. Although one cannot rule out the effect of the electroconvulsive therapy that the patient was receiving before the suicide attempt, it is possible that the suicide attempt itself had an impact on the patient's mood.

Improvement of mood after a failed suicide attempt is a fairly common but probably temporary event. Lesse (1967) described the positive effects of suicidal behaviour on the mood. These 'apparent remissions' after suicide attempts can be attributed to the cathartic effect of suicide and its role in stimulating an empathic and supportive environment and to the emotional impact of the survival.

We previously reported the observation of manic and hypomanic switches immediately or shortly after a suicide attempt by hanging in four depressed patients. Two of these patients were previously diagnosed as being bipolar (Bourgeois *et al*, 1985). In our review we cited the observation of the 19th-century psychiatrists that melancholics who survived suicide by hanging had their mental state improved. These authors were attributing the improvement to asphyxia. Moreover, 'carbonarcose' or anoxic shock (coma induced by letting patients inhale a mixture of 30% carbon monoxide and 70% oxygen) was