cycle lasted between 9 and 15 months. His response to lithium therapy was remarkable, and showed in his physical and emotional well-being, as well as in improvement in his learning, social and personal skills.

I hope that Sturmey is not attempting to minimise the importance of the recognition of treatable major mental illness in mentally handicapped patients, many of whom have suffered greatly in the past from the lack of appropriate treatment (mainly psychotropic medication) simply because their mental disorders were unrecognised.

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Is the Positive-Negative Distinction in Schizophrenia Valid?

SIR: The longitudinal study by Johnstone *et al* (*Journal*, January 1987, **150**, 60–64) raises questions on the validity of the concept of positive and negative symptoms in schizophrenia. Their original proposition that this dichotomy represents different pathogenic mechanisms had been predicated largely on the assumption that positive symptoms are treatment responsive, and hence reversible, whereas negative symptoms are not. The current findings indicate, however, that positive symptoms are not uniformly treatment responsive and that negative symptoms are not universally immutable.

Our longitudinal studies of schizophrenia also challenge the validity of the positive-negative model as originally proposed, and suggest that other dimensions, notably chronicity of illness and diagnostic sub-type, may interact with this construct. In a two year follow-up of young patients with acute schizophrenia (Lindenmayer *et al*, 1986), both positive and negative syndromes proved longitudinally unstable and showed changing patterns of external correlates over time. Contrary to assumptions from studies in the chronic phase, it was the base-line negative rather than positive presentation which anticipated a favourable outcome. A cross-sectional survey of 134 patients with schizophrenia classified as acute (up to 2 years history of illness), chronic (3–10 years), and long-term chronic (over 10 years) similarly suggested that an early negative syndrome carries a better prognosis. In patients with acute schizophrenia such a profile was associated with depressive and atypical catatonic features as well as absence of psychosis in first-degree relatives (Kay *et al*, 1986). Thus, the positive-negative dichotomy may have a quite different clinical significance in the acute phase of psychosis to that in the chronic phase.

Recent pharmacological analysis has suggested, moreover, that the positive-negative classification delineates homogeneous psychobiological sub-types in schizophrenia only if considered along with diagnostic sub-type (Singh *et al*, 1987). The addition of anticholinergic drugs to neuroleptic treatment in a mixed population of patients with acute, subacute and chronic schizophrenia produced a significant worsening of positive symptoms overall, but this occurred mainly among patients with catatonic symptoms. In paranoid patients, to the contrary, it was the negative symptoms that worsened significantly, whereas in hebephrenic patients neither syndrome was significantly affected. Cumulatively, our results suggested that the natures of positive and negative syndromes vary according to chronicity and subtype of schizophrenia, and perhaps only in the chronic patients and the non-paranoid forms of illness is the present concept of the positive-negative dichotomy applicable.

The data from Johnstone's group underscore qualifications on the generality of the construct. They observed that delusions – a cardinal feature of paranoid schizophrenia – were significantly more prevalent in neuroleptic-treated than neuroleptic-free patients with schizophrenia, as were also symptoms of anxiety and depression. The latter finding may be understood within the context of a post-neuroleptic emergence of dysphoria and elevation of tonic autonomic arousal, which characterises only non-paranoid patients and portends poor therapeutic response (Singh & Kay, 1979). Furthermore, our work has suggested that non-paranoid neuroleptic-resistant patients with schizophrenia may be distinguished from others by clinical worsening under blind wheat gluten challenge (Singh, 1978; Singh & Kay, 1984). Research on the psychobiology and course of schizophrenia, therefore, casts doubt on the sufficiency of the positive-negative model. Study of its interaction with diagnostic sub-type and phase of illness, on the other hand, may offer new insights not afforded by examining any of these dimensions separately.

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Neuroleptic Malignant Syndrome (NMS) and Tardive Dyskinesia

SIR: Haggerty & Gillette (*Journal*, January 1987, **150**, 104–105) seem to have concluded that NMS developed while the patient was having reserpine and lithium. The details they give do not support such a conclusion, as they mention in their discussion. For example, they do not mention any alteration in consciousness after lithium and reserpine were started: besides rigidity, hyperpyrexia and autonomic dysfunction, alteration of consciousness in varying degrees is a *sine qua non* for the diagnosis of NMS (Caroff, 1980). Here, only autonomic dysfunction was noticed after starting lithium and reserpine. Moreover, the progression of NMS is known to be very rapid after onset. In more than 90% of 120 cases reviewed, the full syndrome developed within 48 hours of the first symptoms (Shalev & Munitz, 1986). It is naïve to conclude that mild NMS developed over a period of two weeks and that the full-blown syndrome began later, when thioridazine and haloperidol were started.

What is of interest in this report is the unusually long duration of the NMS episode (seven weeks). Generally, it is presumed that NMS lasts for 5–10 days after discontinuation of oral neuroleptics (Sternberg, 1986). I have come across another patient in whom the syndrome persisted for three weeks after cessation of haloperidol and remitted after a 17-day course of amantidine (Woo *et al*, 1986). Treatment was continued for a further period of five months. Such an abnormally long duration may have something to do with the patient's metabolic characteristics, as differences in absorption and first pass metabolism are known to cause wide inter-individual variations in the plasma levels of neuroleptics. Moreover, neuroleptics, being highly lipophilic, can be detected for weeks after discontinuation.

Caution is required before concluding that unexplained fever is due to NMS. This is clear when one considers the authors' suggestion that reports by family of transient diaphoresis and elevations of temperature in the months prior to admission were probably because of NMS in "milder form". In the absence of any alteration of consciousness such an interpretation is not justified. At the moment, there is not enough clinical data to justify the spectrum concept of neuroleptic toxicity (Corlon, 1986) which the authors seem to have in mind.

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Continuum of Psychosis and the Gene

SIR: Crow's theory (*Journal*, October 1986, **149**, 419–425) bears a striking similarity to the theory of Britain's best-known psychologist, Hans Eysenck, to the effect that a heritable trait of psychoticism (*P*) underlies much psychotic (and indeed psychopathic) disturbance (Eysenck & Eysenck, 1976, 1985). Like Crow's continuum, Eysenck's *P* has sometimes been considered to be related to cerebral lateralisation of function (Brand, 1981) and to forms of creativity and achievement (Claridge, 1985) that might explain the persistence of high-*P* genes in the population despite the lowered average fertility of people who suffer outright psychotic disturbance.

I wonder, could the theories of Crow and Eysenck by any chance be related?

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