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Positive symptoms of schizophrenia

SIR: Frith & Done (*Journal*, October 1988, **153**, 437–443) propose that the positive symptoms of schizophrenia arise from a failure in transmission, from goal-setting areas to a central monitor, of willed intentions that form the basis of self-generated action. They suggest that when self-generated actions are noted by the monitor, but not understood as such because of failure of advanced warning of willed intention, then these actions are attributed to external events.

Such a mechanism could underlie the "permeability of the ego-world boundary" mentioned by Schneider as possibly causing passivity phenomena (Koehler, 1979). However, there is another implication of this hypothesis which is more difficult to place: to lose awareness of willed intention to an extent sufficient to cause a florid positive schizophrenia may be to lose recognition of oneself as an independent thinking being. If the central elements of a singular identity are retained in the absence of awareness of the self-generated nature of activity, then Descartes' phrase, "I think therefore I am", would need to be restated as, "I respond therefore I am". This is not a correct statement, since the ability simply to respond does not require an individual sense of consciousness.

Jaspers (1959) considered the effect of psychopathology on awareness of existence, and concluded that there are circumstances where *cognito ergo sum* is no longer a valid experience, particularly in the presence of derealisation and depersonalisation.

Such observations lead to the prediction that the more severe are the positive symptoms expressed by a schizophrenic patient, the more likely it becomes that the patient will be experiencing severe symptoms of depersonalisation or derealisation. This is generally not the case. One explanation for this would be that only the transmission of willed intentions relating to selected goals is impaired. This would account for the observation in many cases, particularly of paranoid schizophrenia, that positive symptoms are only experienced in a part of the patient's experience as a whole. The question then becomes: why is there an abnormality in this particular area of self-generated behaviour?

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SIR: Frith & Done (*Journal*, October 1988, **153**, 437–443) draw attention to the lack of a psychological theory for the positive symptoms of schizophrenia. They propose what appears to be a simplistic model to explain the phenomena of auditory hallucinations. They believe that the patient is "talking to himself" but believes the voices he is hearing are from an outside source.

What they do not consider is the form or content of auditory hallucinations commonly seen in schizophrenia. For example, how would this theory explain two voices discussing the patient, one of which may be male, the other female? This would certainly not reflect "normal psychological processes", whether or not it was labelled "my own". Similarly, the content of auditory hallucinations in paranoid schizophrenia is often abusive and derisory; our understanding of this is not advanced by the theory.

It is admirable that experimental tests of monitor failure are possible, but surely the theory must first embrace those symptoms commonly seen in clinical practice.

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Sample Size and CT Scans in Schizophrenia

SIR: Smith *et al* (*Journal*, November 1988, **153**, 667–674) remind us that the use of high-tech research instruments such as computerised tomography (CT) often hides basic methodological flaws, such as the choice of bogus control groups. Ironically, however, the results of the authors' own elegant meta-analysis of published CT studies in schizophrenia contain the seeds for criticism of their own study: the use of sample sizes too small to test hypotheses is another much-perpetrated sin. The figures derived by Smith *et al* from previous studies show that lateral ventricular size (as measured by VBR) in schizophrenic subjects exceeds that of healthy controls by about 30%. Entering these figures and an approximation of the authors' value for overall standard deviation (± 3.1) into a power analysis shows that, in order to be even 80% confident that a two-tailed test will produce a statistically significant difference, at least 60 patients and 60 controls are needed. It is not surprising,

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therefore, that the authors failed to demonstrate larger ventricles in schizophrenic subjects, since their sample size (30 patients, 30 controls) gave them slightly less than a 50–50 chance of doing so.

The authors should be congratulated on a genuinely informative literature review, but it is a shame they felt unable to put their own prescriptions into practice. In illuminating the devil of the Type I error they have slipped into the deep blue sea of the Type II.

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Schizophrenia: Recency Theory and Syphilis

SIR: Hare (*Journal*, October 1988, **153**, 521–531) argued that the historical origins of schizophrenia may have parallels with that of general paresis. He described the earlier global variation in the prevalence of paresis and the more recent increase in prevalence in non-Western countries, and judged from this that the changes in prevalence were real and probably attributable to changes in an environmental factor, i.e. the introduction of a new infectious agent.

The relative rarity of syphilis, and by extension of general paresis, in West Africa before 1950 may have been related to the endemicity of yaws; the rapid increase in the incidence of syphilis since then is thought to be related to the decrease in the prevalence of yaws following the WHO mass treatment campaign of the 1950s and 1960s (Perine, 1987).

Yaws is caused by *Treponema pertenue*, which is morphologically identical to *Treponema pallidum*, the cause of venereal syphilis. The increase in venereal syphilis since the mass treatment programmes may represent a decline in herd immunity to yaws, and thereby to syphilis. This thesis is not incompatible with the notion that venereal syphilis was a new disease. Venereal syphilis may well have derived from the endemic treponematoses (unitarian theory) which include pinta, yaws, bejel, and endemic syphilis (Csonka, 1987). However, the thesis suggests that rapid alterations in prevalence, especially in this case, may be as much to do with changes in herd immunity to a related pathogen as it is to do with introduction of a new pathogen.

I agree with Dr Hare that study of the historical origins of general paresis may throw some light on the schizophrenia debate and suggest that the curious relationship between rheumatoid arthritis and schizophrenia (if true) is reminiscent of that between yaws and venereal syphilis in the African population. Both rheumatoid arthritis and schizophrenia may be caused by related but different pathogens.

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Tactile Extinction Phenomenon in Schizophrenia

SIR: With regard to Dr Cutting's comments (*Journal*, August 1988, **153**, 281) about our interpretation of the tactile extinction phenomenon in his review of *Etiopathogenetic Hypotheses of Schizophrenia: the impact of epidemiological, biochemical and neuromorfological studies*, the direct relationship between QET and "a disorder of some sort in the right hemisphere" is so far to be univocally demonstrated. Taking the neurophysiological pathway of tactile sensitivity into account, there is clinical evidence that both right contralateral and left frontal cerebral lesions cause a left-extinction phenomenon (Schwartz *et al*, 1977).

Attempts to clarify which of these (left homolateral or right contralateral) is responsible for our results have not given consistent results. Even if a neuropsychological interpretation of the QET suggests that left-extinguishing patients performed worse in right-hemisphere tasks (Gambini *et al*, 1986) the interpretation of these results still are not definitive. In fact, QET can be considered to be similar to other dichotic tasks (e.g. dichotic listening and, in general, simultaneous stimulations of the left and right sensorial pathways). Therefore, as Schwartz *et al* (1977, 1979) pointed out, it is reasonable to suggest that when a verbal response is needed, as in the QET paradigm, the two sensorial tactile pathways are quite different; in fact, the left-side stimulus has a longer indirect pathway to CNS in comparison with the stimulus from the right hand, which arrives directly to the left hemisphere. The longer way for the left-side stimulus includes fibres crossing from the right to the left hemisphere mainly through the anterior part of the corpus callosum. Thus it might be reasonably

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