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Correspondence

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THE CORPUS CALLOSUM AND BRAIN FUNCTION IN SCHIZOPHRENIA

DEAR SIR,

Jones and Miller (*Journal*, December, 1981, **139**, 553–7) have added further support to the growing evidence that an impairment of corpus callosum transmission is one feature of the cerebral dysfunction accompanying schizophrenia. Their findings also support a model of brain organization in this disorder discussed by Shaw, Resek and Coulter 'EEG Coherence, Lateral Preference and Schizophrenia' paper submitted for publication.

These latter authors infer from their experimental EEG findings that brain organization in schizophrenia is diffuse like that found in healthy lefthanded individuals. There is evidence that such an organization requires more interhemisphere integration via the corpus callosum than the more common lateralized organization present in healthy right handers.

Jones and Miller interpret their finding of negligible ipsilateral/contralateral latency differences of the early somatosensory evoked response in schizophrenia as follows: First, that the corpus callosum is not conducting at all and second, that the ipsilateral response is produced by ipsilateral pathways from the brain stem. The latter is compatible with the suggestion of Shaw *et al* that brain organization in schizophrenia is like that in lefthanders. This follows from Kinsbourne's (1980) hypothesis that in the lefthander, the diffuse organization develops from bilateral cerebral activation during the adoption of a verbal mental set as a result of a "less laterally polarized brain stem (thalamic) influence that is projected to both cerebral hemispheres". Such organization may account for the sometimes reported, but tenuous, association between lefthanders and schizophrenia (Taylor *et al*, 1980).

Split-brain patients and healthy lefthanders do not show the behavioural disorder of schizophrenia. Jones and Miller's suggestion of bilateral brainstem influence is supported by Shaw *et al's* EEG evidence. It reinforces the latter's conclusion that impaired corpus callosum transmission, together with a brain organized to need it more than most, may contribute to the behavioural and intellectual disturbances of schizophrenia. It would be valuable to apply Jones and Miller's experiment to healthy lefthanders. The outcome may help to decide whether their result was dependent only on faulty corpus callosum transmission or due to the postulated bilateral brainstem projection.

It is of interest that Rosenthal and Bigelow's (1972) finding of an enlarged corpus callosum in schizophrenia has initiated so many studies showing impaired corpus callosum transmission in this disorder. These 1972 results were based on only 10 patients and do not appear to have been replicated.

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DEAR SIR,

I believe the recent article by Jones and Miller (1981) entitled 'Functional Tests of the Corpus Callosum in Schizophrenia' deserves comment as it makes untenable assumptions, presents results indicating, amongst other things, neuropathology in the normal control sample, and reaches conclusions which are unwarranted.

Jones and Miller base much of their introductory opinions on unreplicated results (Beaumont and Dimond, 1973), conclusions since altered by the author (Flor-Henry, 1976) or on interpretation of results contradictory to the interpretation of the original authors (Gruzelier and Venables, 1974). While it is conceivable that there is a callosal transmission problem in schizophrenia it cannot be