After my visit to Thailand I have asked myself the question, "Are adult European populations immune to these epidemic psychoses or do they now take more subtle forms?" What, for example, might happen if a rumour started in East Anglia that a new fungicide used on the potato crop caused impotence and genital shrinkage. Once such a rumour started, would it be easily dispelled by medical reassurance? If rabies were reported to have spread from Europe to Kent and, at the same time, a wedding party fell ill after eating pork pie from an allegedly infected pig, would people be more readily reassured by public health experts than their Thai counterparts? What would happen if a Scottish farmer reported that the Loch Ness Monster had emerged from hiding and eaten a couple of cows and several sheep? If such an account was endorsed by a local MP, would it be easily rebutted? My own conclusion is that so-called sophisticated Westerners are not immune to gullibility, remarkable suggestibility, magical thinking and shared delusions leading to irrational behaviour. As Orson Welles found some years ago, the fictional announcement of an invasion from outer space can lead to a dramatic psychic epidemic. Social psychiatrists should perhaps give more attention to shared irrational beliefs in our own society.

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NEUROPATHOLOGY OF THE CORPUS CALLOSUM IN SCHIZOPHRENIA

DEAR SIR,

In their study of ipsilateral/contralateral differences in early somatosensory evoked response, Jones and Miller (*Journal*, 1981, **139**, 553–57) found that the interhemispheric conduction time across the corpus callosum in 12 schizophrenic patients was essentially zero. They concluded that schizophrenia may be a split-brain condition akin to agenesis of the corpus callosum and that neuropathological examination of the corpus callosum, of which there is no report yet in the literature, should test this hypothesis.

We recently completed a post mortem histopathological study of the corpus callosum in 18 chronic schizophrenic and 11 nonpsychiatric control subjects. The thickness of the corpus callosum in the schizophrenic sample was not different from the control group, which does not confirm the findings of Rosenthal and Bigelow (1972). However, the corpus callosum was significantly thinner in the cases with late onset (after age 30 years, usually paranoid, N = 7) compared to early onset (before age 30 years, usually nonparanoid, N = 11) (Bigelow *et al*, 1981).

We examined glial cells on a hematoxylin-eosin stain and callosal fibers on a Bielschowsky stain. There were no differences in the number of glial cells per unit area (high-power microscopy) or the number of fibers (cross sections) per unit area between the schizophrenic and control subjects or between the schizophrenic subtypes.

The slides were then evaluated by a neuropathologist blind to the source of the tissue. He rated gliosis as absent (0) mild (1) moderate (2) severe (3) and very severe (4). There was significantly more gliosis in late onset (paranoid) schizophrenia (Rank Sum Test $P < .04$) than in control subjects. There was no difference in gliosis between the early onset schizophrenia and control groups.

The results suggest that late onset (paranoid) schizophrenia may be associated with a chronic inflammatory process, such as viral encephalitis. There is some evidence for a viral involvement in schizophrenia (Torrey and Peterson, 1976).

The possible disruption of interhemispheric transfer across a diseased corpus callosum is consistent with the findings of abnormal lateralization in schizophrenia (Newlin *et al*, 1981) particularly in the paranoid subtype (Nasrallah *et al*, 1981).

It is possible that the absence of a trans-callosal conduction time reported by Jones and Miller (1981) may reflect compensatory ipsilateral pathways secondary to callosal disease. It would be interesting to know what the subtype composition of the Jones and Miller sample was.

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References

BIGELOW, L. B., NASRALLAH, H. A., RAUSCHER, F. & WYATT, R. J. (1981) Post mortem evidence for pathology in the anterior corpus callosum in schizophrenia. Abstracts of the Society of Biological Psychiatry Thirty Sixth Annual Meeting, New Orleans, page 31.

JONES, G. H. & MILLER, J. J. (1981) Functional tests of the corpus callosum in schizophrenia. *British Journal of Psychiatry*, **139**, 553–7.

NASRALLAH, H. A., KUPERMAN, S., MCCALLEY-WHITTERS, M. & KEELOR, K. (1981) Laterality in paranoid and nonparanoid schizophrenia. New Research Abstracts, American Psychiatric Association 134th Annual Convention, New Orleans, page 32.

NEWLIN, D. B., CARPENTER, B. & GOLDEN, C. J. (1981) Hemispheric asymmetries in schizophrenia. *Biological Psychiatry*, **16**, 561–82.

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ROSENTHAL, R. & BIGELOW, L. B. (1972) Quantitative brain measurements in chronic schizophrenia. *British Journal of Psychiatry*, **121**, 259–64.

TORREY, E. F. & PETERSON, M. R. (1976) The viral hypothesis of schizophrenia. *Schizophrenia Bulletin*, **2**, 136–46.

FUNCTIONAL TESTS OF THE CORPUS CALLOSUM IN SCHIZOPHRENIA

DEAR SIR,

Jones and Miller (*Journal*, December 1981, **139**, 553–57) suggest that schizophrenia is a split-brain condition akin to agenesis of the corpus callosum. They have demonstrated that unlike normal subjects, patients with acute schizophrenia show simultaneous cortical evoked responses to vibratory stimuli applied to each index finger. A more obvious conclusion from their work is that these patients simply have ipsilateral pathways to the CNS of near equal importance to the contralateral pathways. In normals most stimuli going to the CNS are projected preferentially to the contralateral hemisphere, and activity in the corresponding part of the ipsilateral hemisphere is suppressed (Gazzaniga, 1974). Exceptions to this of course arise (e.g. visual stimuli are projected to both right and left cerebral hemispheres without suppression).

A number of studies have tried to demonstrate a causative role in schizophrenia of damage to the corpus callosum. Most of these use as their starting point the study on only 10 chronic schizophrenic patients by Rosenthal and Bigelow, 1972. This study had a number of flaws including a low number of patients and the fact that a number of the controls suffered with alcoholism and 6 had a diagnosis of personality disorder (no criteria given). Even if the corpus callosum is thickened in chronic schizophrenia, this does not mean that this is the primary disorder but might well be secondary to either drug therapy or attempts by the body to compensate for damage of one hemisphere. A major flaw with the study by Beaumont and Dimond (1973) was that, in testing their subject for transfer of information across the corpus callosum, they relied on verbal replies and therefore their results could be interpreted as showing either problems in the corpus callosum or in the dominant hemisphere of schizophrenic patients.

The evidence from Green's (1978) work would on first sight appear to be contradictory to the results obtained by Jones and Miller, for surely if information from one hand is relayed equally to both cerebral hemispheres, then interhemispheric transfer would not be necessary for the other hand to perform a task.

A number of authors (Gruzelier and Venables, 1974; Gur, 1977; Taylor, Greenspan and Abrams, 1979; Schweitzer, Baker and Welsh, 1978; Flor-Henry, 1969) have hinted that in schizophrenia, there might well be a lesion of the dominant hemisphere but so far, neither the investigation of the corpus callosum nor the investigation of the left cerebral hemisphere have thrown much light on the aetiology or treatment of the schizophrenias.

However, the work of Jones and Miller opens a new avenue to the investigation of schizophrenia. The underlying anatomical problems in schizophrenia may be in the presence of unduly large ipsilateral pathways to and from the cerebral hemispheres. These would result secondarily in failure of one hemisphere to suppress function in the other as normally occurs (Gazzaniga, 1974).

Indeed Green (1979) reported that by use of earplugs to occlude stimuli from one ear in schizophrenic patients, they obtained significantly increased levels speech comprehension compared with everyday binaural hearing and perhaps a decrease in auditory hallucinations. By doing this they were clearly giving one hemisphere on advantage in dealing with auditory stimuli over the other, and thus facilitating that hemisphere in suppressing the opposite hemisphere.

It might well be that the genetic predisposition in schizophrenia may be the presence of an excess of ipsilateral sensory and motor tracts and indeed previous research has shown the presence of major ipsilateral nerve tracts in man (Levy, 1976).

This study by Jones and Miller certainly fits in with the theories of attention and perception described by McGhie and Chapman (1961) and with the findings of a disorder of the arousal mechanism (Claridge, 1972). It is also easy to see how the dopamine hypothesis would fit in with this scheme.

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References

BEAUMONT, J. G. & DIMOND, S. J. (1973) Brain disconnection and schizophrenia. *British Journal of Psychiatry*, **123**, 661–2.

CLARIDGE, G. (1972) The schizophrenics as nervous types. *British Journal of Psychiatry*, **121**, 1–17.

FLOR-HENRY, P. (1969) Psychosis and temporal lobe epilepsy. *Epilepsia*, **10**, 363–95.

GAZZANIGA, M. (1974) In *Hemisphere Function in the Human Brain* (eds. S. J. Dimond and J. G. Beaumont). New York: John Wiley. Pp 367–82.

GREEN, P. (1978) Defective interhemispheric transfer in schizophrenia. *Journal of Abnormal Psychology*, **87**, 472–80.

GREEN, W. P. (1979) Schizophrenia and earplugs. *British Journal of Psychiatry*, **135**, 287–8.

https://doi.org/10.1192/bjp.141.1.99 Published online by Cambridge University Press