there was no significant excess of NSS in the subgroup of patients with an early onset of illness. Another possible interpretation would be that NSS may be the evidence of an early cerebral insult and the resultant central nervous system dysfunction may lead to poor educational attainment even before the onset of illness. The brain dysfunction may also act as a predisposing factor, at least in a subset of patients, and may be responsible for the clinical expression of schizophrenic syndrome later on. Although these hypotheses sound attractive there does not seem to be enough evidence as yet to provide definitive answers.

Magnetic resonance brain imaging (DeMyer *et al*, 1988) of schizophrenic and normal subjects have shown that brain size is positively correlated with education. Lewis (1990) after reviewing a number of CT scan studies concluded that a range of demographic factors including educational level were important in determining various structural brain parameters. Such studies highlight the importance of controlling for the effect of education in studies of human subjects where brain size is the dependent variable. Although there are no definitive studies to correlate NSS and brain pathology using the newer techniques of imaging, this area needs to be pursued. If indeed there is a correlation, assessment of NSS could be considered as an inexpensive, simple method of studying neurological impairment. Issues raised by Rossi *et al* regarding the methods of assessment of NSS need careful consideration.

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Brain lesions and cognitive function in late-life psychosis

SIR: Miller *et al* (*Journal*, January 1991, **158**, 76–82) published the results of their study of 24 patients with late-life psychosis. Patients had significantly more brain abnormalities on magnetic resonance imaging (MRI) scan than controls. In particular, despite normal neurological examinations in all but two of the patients, 25% of this group had cortical or white matter infarctions on MRI, compared with 7% of controls. Although the patient group had more abnormalities on neuropsychological testing, their mean Mini Mental State Exam (MMSE) score was 28/30 and none met clinical criteria for dementia.

These findings are very similar to our own (Flint *et al*, 1991). We studied 33 elderly patients with normal neurological history and examination, and a mean MMSE score of 27/30, meeting DSM–III–R criteria (American Psychiatric Association, 1987) for delusional disorder, schizophrenic disorder (late-onset type), and schizophreniform disorder. Of the patients undergoing CT brain scan, 31% were found to have cerebral infarction, mostly affecting the frontal-subcortical system. In addition, the presence of infarction was inversely correlated with social risk factors (failure to marry, social isolation) previously described for 'late paraphrenia', suggesting that stroke, by itself, is a potent mechanism in the pathogenesis of late-life psychosis.

Although Miller *et al* did not specifically examine treatment response, their impression was that many of their patients were treatment resistant. They speculated that the underlying brain disease contributed to a poor prognosis. In our study, subjects with brain infarction were indeed significantly less likely to respond to treatment. Because of the appearance of disabling side-effects, patients with CT evidence of stroke could only tolerate, on average, half the dose of antipsychotic medication compared to those without infarction. Had this 'organic delusional' group been able to tolerate higher doses of medication they may well have shown more symptomatic improvement. Interestingly, several studies, including Holden's (1987) all found significantly worse outcome in late-onset paranoid patients with cerebral organicity.

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