

While we applaud the approach taken, we find it difficult to interpret the reported results. We are wary of significance levels uncorrected for multiple comparisons, and of the use of controls screened to exclude those with cerebral abnormality as a comparison for scans without such screening. We are particularly concerned by the varying proportion of male and female subjects in the groups compared. Although the VBR measure attempts to correct for varying brain sizes by constructing a ratio of ventricular size to brain size, VBR varies positively as a function of brain size, which is in turn positively related to overall body size. Male subjects, generally larger than female subjects, have significantly larger VBR measures as well (Bridge *et al*, 1985).

An examination of the results of Dr Kaiya *et al* reveals that where differences in VBR are found between groups, there are also differences between the proportion of male subjects in these groups, with a larger proportion of males associated with larger VBR. The strength of this possible confound is indicated by calculating the correlation between the ratio of male to female subjects in a subgroup and the mean VBR, (lateral ventricles VBR) reported for that subgroup; here  $r = 0.994$ ,  $P < 0.005$  for the non-familial, familial (horizontal), familial (vertical), and familial (mix) subgroups, and remains high ( $r = 0.963$ ,  $P < 0.005$ ) after including the control subjects.

It is my hope that by controlling intersubject variability due to gross physical differences such as height, continuing investigation of subtle differences between subgroups of schizophrenic individuals will reveal robust cerebral morphometric differences useful in elucidating the pathophysiological bases of schizophrenic illness.

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#### Reference

BRIDGE, T. P., PARKER, E. S., INGRAHAM, L. *et al* (1985) Gender effects seen in the cerebral ventricular/brain ratio (VBR). *Biological Psychiatry*, **20**, 1136–1138.

SIR: We were interested to read the study by Kaiya *et al* (*Journal*, October 1989, **155**, 444–450). In common with similar studies, the use of high technology in psychiatric research seems to have excused the authors from sticking to the scientific conventions of a plausible, testable hypothesis which is adequately tested. Firstly, the hypothesis of three genetically dis-

tinguishable sub-groups in the aetiology of schizophrenia has little or no precedent to our knowledge, nor much in the way of rationale. Secondly, the hypothesis is not tested properly. The control group was not, as might be expected, healthy volunteers, but neurology patients. They were collected retrospectively, were not matched for age or sex, and most surprisingly were not psychiatrically assessed. In addition, there is nothing to indicate that the multivariate analysis was performed with the intention of making planned comparisons. Consequently, the suggested associations between the CT findings in schizophrenic sub-groups may well be accidental.

It is a pity that with such a topical subject the study failed to be rigorous enough.

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#### Age of onset of depression in the elderly

SIR: The interesting papers by Musetti *et al* (*Journal*, September 1989, 330–336) and Burvill *et al* (*Journal*, November 1989, 673–679) concerning depression in later life and age of onset prompted me to examine, in the light of their findings, data from a previously described cohort of elderly patients with major depression (Baldwin & Jolley, 1986).

Details of whether the age of onset was before or after the age of 60 was available for all but two patients: 77 were late onset and 21 early onset. Late-onset patients were significantly older at the index admission than the early-onset group: 74.7 years compared with 71.5 years ( $t$ -test,  $P < 0.01$ ). Unlike Dr Burvill *et al* I did not find that early-onset patients were more depressed, although the cohort as a whole were more severely depressed than theirs (Hamilton Rating Scale for Depression (17 item) scores: late-onset 27.8, early-onset 27.2; NS). However, like them, I found no significant differences in family history of depression. Twenty-three percent of the late-onset group ( $n = 62$ ) and 21% ( $n = 19$ ) of the early-onset group had a positive history, although this data was missing on 17 patients. Likewise, there were no differences in the numbers dying or developing dementia during the follow-up period or in the overall outcome using the classification of Post (1972). Although adverse life events occurring in the previous 12 months were more common compared with the cohort of Dr Burvill *et al*, as in their study, the proportions did not differ significantly between the groups. Bereavement was the commonest event