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RAYMOND TRAVERS

Olive Mount Hospital
Old Mill Lane
Wavertree
Liverpool L15 8LW

AUTHORS' REPLY: Dr Travers raises questions but offers few solutions. He is of course correct that there are many influences on the opiate withdrawal syndrome in addition to that described in our paper (*Journal*, May 1991, **158**, 697–699). However, while clinical practice might necessarily involve juggling with all of these simultaneously, the systematic study of these possible modifying factors requires a different strategy. We have previously reported on the differences between 10-day and 21-day withdrawal regimens (Gossop *et al*, 1989) and on the influences of different reduction curves (Strang & Gossop, 1988). Dr Travers rightly draws attention to the difficulty in identifying the relevant measures for the study of the opiate withdrawal syndrome. We are of the view that the SOWS (for development and validation see Gossop, 1990) and the actual measure of completion rates provide two differing and valuable perspectives. When all the criticism is said and done, what does Dr Travers actually suggest?

The most interesting points of the criticism are in the last paragraph where consideration is given to the possible different influence of methadone on heroin or methadone addicts. As we explained in the paper, virtually all of our subjects had originally been using heroin, and were assigned to the two groups according to their most recent opiate of use before admission. Thus if drug cues are being considered, it is unlikely that they would be substantially different across the two groups. Perhaps it is being suggested that the drug cues may relate to the preparation being used – the linctus – but this is unlikely as we use a differently coloured and specially prepared linctus on our inpatient programme. There are also important cognitive cues that influence withdrawal responses. Some of these have been considered by Wikler (1980) as well as in our own previous research.

Finally, Dr Travers questions whether the findings are of any relevance in view of the similar discharge rates. From a clinical management perspective there may be some legitimacy to this point of view, although we would have thought that reducing the levels of within-treatment distress remained a legitimate clinical goal, notwithstanding the similar completion rates. However, we would also draw attention to a more general point: for too long the treatment of addicts has muddled along, driven by chemical whim, with insufficient investment in the systematic study of the treatment being delivered.

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JOHN STRANG
MICHAEL GOSSOP

Drug Dependence Clinical Research and Treatment Unit
The Bethlem Royal Hospital
Monks Orchard Road
Beckenham
Kent BR3 3BX

Cerebral ventricular size and dyskinesia

SIR: McClelland and colleagues (*Journal*, May 1991, **158**, 691–696) report an interesting association between cerebral ventricular size, at the end of 16-years follow-up and the development of facial dyskinesia during the period in a group of 23 patients with functional psychosis. This data adds to 12 previous studies, the results of which divide evenly between supporting and refuting an association between current ventricle:brain ratio (VBR) and current dyskinesia. A smaller group of studies are divided over the importance of cortical atrophy. A confounding problem in McClelland *et al*'s findings may be the effect of ageing on brain structure. While the 18 years from first assessment for dyskinesia to computerised tomography (CT) scan would be accompanied by some ventricular changes, it would also be accompanied by some development of cortical atrophy. This is particularly likely in those patients who converted from dyskinesia negative to positive as their mean age at first assessment was 53 years. Thus it may be equally possible that the development of dyskinesia in this group could be associated with cortical atrophy.

Recent findings of our own would support this view (Cooper *et al*, 1991). We have found no relationship between VBR and the presence or absence of

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