because latencies may vary between subject groups that consistent nomenclature is essential. Evoked potentials are conventionally identified according to the polarity and modal latency of their peaks. Thus when potentials from patients and normal subjects are compared, like is compared with like, and changes in the characteristics of the potentials may be related to changes in the processes or structures generating them. Unfortunately, when the present results are quoted by future authors, confusion will arise. For instance, it would seem reasonable to suppose that in a paper on early cortical-evoked potentials, the wave labelled N1 (their Fig. 1) would refer to the initial negative-going cortical potential (which is conventionally called N19 or N20 since its modal latency is about 20 ms). A peak corresponding to N20 is clearly present in Fig. 1 but is unlabelled and distinct from the peak labelled N1.

The authors note considerable variability both within and between subjects in the amplitudes of their cortical potentials. At least some of this variability is likely to arise from the type of stimulus used. Vibrotactile stimulation of the skin of the finger may excite impulses in a wide range of sensory fibres of differing conduction velocities (Iggo, 1982); together with the 10 ms duration of the stimulus this produces a diffuse and temporally dispersed afferent volley. Furthermore, spread of vibration is a potent stimulus to muscle receptors of the intrinsic muscles of the hand and forearm (Davies, 1987). Since the amplitude of excursion of the vibrator is not quoted in their paper, the extent of these effects cannot be assessed.

Finally, a further source of variability might arise from combining data from patients suffering different forms of schizophrenic illness. Until proved otherwise, it should not be assumed that sensory processing or its dysfunction is similar in acute v. chronic patients, or in those with positive v. negative symptoms.

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Clozapine in patients with NMS

SIR: Szabadi & Cashman (*Journal*, April 1991, **158**, 577) suggest that clozapine is the drug to consider when a patient has suffered neuroleptic malignant syndrome (NMS). However, it would be inappropriate to generalise their experience with clozapine to other patients who have suffered NMS. The first case report linking clozapine (in combination with lithium) to NMS appeared more than four years ago (Pope *et al*, 1986). This was followed by another case in which NMS was presumed to have resulted from a combination of clozapine and carbamazepine (Muller *et al*, 1988). There might have been some scepticism about these reports as clozapine by itself could not be implicated. More recently, a series of case reports suggested that clozapine alone can cause NMS (e.g. DasGupta & Young, 1991). It would not be unfair to say that the strategy suggested by Szabadi & Cashman (1991) has also been adopted in the past without recurrence of NMS. Despite its different structure and pharmacological profile, clozapine does affect the dopaminergic system; recently the gene for a human dopamine D4 receptor with high affinity for clozapine has been cloned (Van Tol *et al*, 1991). In view of these observations it may be rather premature to generalise about the potential safety of clozapine in patients with NMS who need further antipsychotic treatment.

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Outcome indicators in mental handicap

SIR: We read with interest Jenkins' article (*Journal*, October 1990, **157**, 500–514), particularly on outcome indicators for mental handicap. Although we fully agree there is an urgent need for specific outcome indicators for mental health care, we were dismayed to see that many of the suggested indicators for mental handicap bore little relationship to the clinical practice of the 'psychiatry of mental handicap'. Many indicators given seemed to be more related to the clinical practices of obstetrics, paediatrics,