

Correspondence

Correspondents should note that space is limited and shorter letters have a greater chance of publication. The Editors reserve the right to cut letters and also to eliminate multitudinous references. Please try to be concise, strictly relevant and interesting to the reader, and check the accuracy of all references in Journal style.

THE CORPUS CALLOSUM AND BRAIN FUNCTION IN SCHIZOPHRENIA

DEAR SIR,

I feel obliged to reply briefly to the letter of Jones and Miller (*Journal*, 141, 535–37) lest readers assume they have in any way answered the criticisms I have made (Connolly, 1982) of their original report (Jones and Miller, 1981).

Firstly, they have addressed themselves to only a subset of the points I raised. Nevertheless, they go on to say that the fact that their evoked potentials differed from Salamy's (1978) is "to be expected". This is a surprising admission; if they expected this then why did they base their report on the Salamy technique and interpret their results on the basis of Salamy's results? Do they also "expect" that the same fibres involved with Salamy's technique are stimulated with their method? Also, their citing of research pertaining to finger displacement (Papakostopoulos *et al*, 1974) reinforces my confusion as to what Jones and Miller (1982) believe their original report was about. In fact, examination of the displacement data (Papakostopoulos *et al*, 1974) suggests that Jones and Miller (1982) would have been better off maintaining their original position that they were basing their position on Salamy's work. For example, Papakostopoulos *et al* (1974) state that, "To contralateral displacement the pre- and post-central cortical areas showed an initial positivity with a delay of 34 ± 6 ms to peak and 42 ± 4 ms, respectively (p 582) . . . the ipsilateral responses started with a positive deflection with a latency of 60 ms or more (p 583)". The contralateral-ipsilateral differences were indeed longer (after all it is a different experiment altogether) but again ipsilateral responses were longer than contralateral *not vice versa*. How does this support Jones and Miller (1981) and their shorter ipsilateral responses? Also, I fail to see the relevance of their "large scatter" explanation for ipsilateral-contralateral differences seen in their sample. All my criticisms still apply.

Their description of the assessment of evoked potentials as involving an "insurmountably subjective judgement" is not reassuring; again, a surprising

admission. Also, the use of sophisticated technology does not guarantee data; in fact, problems multiply unless those using the technology are fully conversant with its methods and the phenomenon upon which it is being used.

Finally, it is all too easy for discussions of this sort to go on interminably. Only correctly collected and analysed data can provide the final answer. Fortunately, Shagass *et al* (this issue, pp 471–76) have done this—and have failed to replicate the findings of Jones and Miller (1981).

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HOW DOES ECT WORK?

DEAR SIR,

Robin and de Tissera (*Journal*, October 1982, 141, 357–66) conducted an important ECT experiment in which a very low energy (5.5–13 joules) pulse electrical stimulus waveform was found to have less anti-depressive efficacy than either high energy pulse (40–55 joules) or high energy sinusoidal (70–100 joules)