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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 antidepressive efficacy than either high energy pulse (40–55 joules) or high energy sinusoidal (70–100 joules)

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stimulus waveforms. There are, however, several statements in this report that are vague and potentially misleading.

These investigators summarize their findings by saying (p. 357), "It is suggested that the quantity of current, as well as the induction of a convulsion, is relevant to therapeutic outcome with ECT". This and a similar statement at the very end of their discussion may lead readers to conclude that the quantity of current or energy *per se* is partly responsible for ECT's therapeutic efficacy. Such a conclusion is supported neither by the convulsive therapy literature (Fink, 1979) nor by Robin and de Tissera's own data analyses. Regarding the latter, Robin and de Tissera did *not* report a positive correlation between electrical energy and anti-depressive efficacy, which would be required if one were to argue for a therapeutic effect directly related to electrical energy. If the therapeutic differences across Robin and de Tissera's three groups is not due to a difference in electrical energy, what is the critical variable influencing therapeutic outcome?

Seizures induced with ECT do not necessarily occur in an "all-or-none" fashion (Liberson, 1948). Clinical observations from several studies (discussed by Daniel and Crovitz, 1983, p. 3) suggest that low energy pulse electrical stimuli (as used in Robin and de Tissera's study) may produce *less generalized* seizures than those produced by higher energy electrical stimulations. These former seizures may be less effective in alleviating depression than the latter more highly generalized seizures (Ottosson, 1962a, 1962b; Cronholm and Ottosson, 1963; Fink, 1979).

While Robin and de Tissera measured and found no difference in *duration* of seizures among their three treatment groups, they apparently did not examine differences in clinical or EEG seizure *patterns* among the three ECT groups. These investigators cannot therefore rule out the possibility that a difference in seizure generalization (reflected in differing patterns) was responsible for the inter-group anti-depressive difference they found, a hypothesis Cronholm and Ottosson (1963) formulated to explain results in a similar study.

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DEAR SIR,

Robin and de Tissera conclude that low energy pulse ECT stimuli are not as therapeutic as high energy pulses or high energy modified sinusoidal waveforms. Their findings, however, do not allow such a general conclusion to be reached. The low energy pulse stimulus used by these investigators has an ultrabrief pulse width (0.3 msec) which has already been shown to be less effective than stimuli with a wider pulse width (Cronholm and Ottosson, 1963; Pippard and Ellam, 1981). A brief pulse stimulus characterized by a pulse width in the range of 0.75 to 1.5 msec, (MECTA ECT device) on the other hand, has been shown to be equally as effective as an unmodified sine wave stimulus for both unilateral nondominant and bilateral electrode placement (Welch *et al,* 1982; Weiner *et al,* in press). This ongoing study involved random assignment to both stimulus waveform and electrode placement. Seizure duration, as monitored by EEG, was equivalent for all combinations, indicating that stimuli were probably equivalent with respect to seizure threshold.

By making a generalization about the efficacy of high and low energy ECT stimuli, Robin and de Tissera may be leading the reader to believe that all forms of brief pulse stimuli are not as effective as high energy stimuli, and that their use should, therefore, be avoided. Such a conclusion, however, is premature, not only for the reason delineated above, but also because there is evidence that higher energy stimuli may be associated with more adverse central nervous system effects (Weiner *et al,* 1982; in press; Daniel *et al,* 1983).

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