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Correspondence

Editor: Ian Pullen

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Duration of depressive illness

SIR: The influence of premorbid neuroticism on duration of depressive illness, as studied by Scott *et al* (*Journal*, November 1992, **161,** 633–637), seems clinically and intuitively correct. The interval between the onset of the illness and receiving treatment, which was found to be the second major influence, also seems likely to be true, but I have a question about the method, the answer to which I cannot find in the paper. The duration of illness is taken as the time from onset to recovery and it is self-evident that the longer the period before treatment, the longer will be the total duration of illness. A more interesting question is whether the duration of illness before treatment predicts the time taken to respond to treatment once it is given. That result would really support the conclusion of Dr Scott *et al* that major depression should be treated at the earliest stage possible if chronic symptoms are to be avoided. Can they clarify their method in this regard, and do they have data to support the conclusion in favour of early treatment?

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AUTHOR'S REPLY: As indicated, it seems logical to suggest that the longer an illness persists before treatment, the longer the total episode length, and so it is important to know the impact of early or late introduction of treatment on the outcome of depression. Firstly, we would like to consider previous publications that shed light on this problem. Three studies suggest that the longer an episode persists before treatment is introduced, the less likely it is to respond to treatment (Kiloh *et al*, 1962; Deykin & DiMascio, 1972; Paykel *et al*, 1974). By inference, this suggests an extended episode even after the introduction of treatment.

Briefly reanalysing our data, it was found that the length of episode prior to the introduction of treatment was significantly correlated with length of episode after the introduction of treatment (Pearson $r=0.4$; $P<0.05$). However, plotting a regression curve suggested a non-concordance rate of about 35%. A separate analysis of the 50 non-chronic depressives (episode of longer than 2 years) in this study and a group of 50 chronic major depressives has also been undertaken. The difference in the 'no treatment interval' just failed to reach significance ($P<0.06$), but there was a trend for those with a longer 'no treatment interval' to suffer a prolonged illness episode even after the introduction of treatment.

In conclusion, we would suggest that there is tentative evidence to support the hypothesis that duration of illness before treatment predicts response to treatment, once it is given. Further research on the 'no treatment interval' is clearly required. The method of our study can be further improved, firstly by improving the reliability of the estimation of the 'no treatment interval', which was relatively crude, and secondly by careful prospective monitoring of the treatment received by the cohort. In our study, difficulties in collating data meant that 75 mg of a tricyclic antidepressant or its equivalent was the most reliable definition of 'active treatment' that we could achieve. The correlation between 'no treatment interval' and response to treatment after its introduction may be enhanced if the definition of 'active treatment' were for example 150 mg.

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Tourette's syndrome and the amygdaloid complex

SIR: We would like to comment on the paper by Jadresic (*Journal*, October 1992, **161**, 532–534) in which she has proposed that the 'amygdaloid complex' is central to the generation of the symptoms of Tourette's syndrome (TS).

Dr Jadresic advocates a pivotal role for the amygdala on the basis that vocalisations elicited from the amygdala are clearly related to emotional state in animals, and that vehement swearing with angry affect accompanied amygdaloid stimulation in one subject who was suffering from an aggressivity disorder. We would argue that these findings militate against a primary involvement of the amygdala. Tourette's syndrome vocalisations are not necessarily accompanied by aggressive affect; indeed, Nuwer (1982) has stated that "the form of coprolalia (in TS) differs from that of purely emotionally generated obscenity in its cadence, pitch, volume and context, as well as in other ways".

Conversely, Dr Jadresic states that electrical stimulation of the cingulum, pontive tegmentum and the periqueductal grey matter (PAG) trigger vocalisations which are devoid of emotional content. However, it may be noted that her source, a review by Bonnet (1982), took information from a further review by Jurgens & Ploog (1981), which in turn was taken from the experiments of Jurgens (1976). The original work used a form of place-preference test in a shuttle-box to estimate the rewarding or aversive properties of brain stimulation which was accompanied by vocalisation in the squirrel monkey. This is rather different from claiming that the vocalisations so induced were devoid of emotional content. Two groups of areas elicited vocalisation; in the first, which included amygdala, hypothalamus, substantia nigra, and rostral PAG, vocalisation was accompanied by motivational changes, while in the second, including the cingulate gyrus, caudal PAG, and adjacent parabrachial region, motivational changes where absent or variable. In our view the lack of any close coupling between TS vocalisations and affect suggests a possible involvement of this second group of brain areas in TS vocalisation.

With regard to Dr Jadresic's neurobiochemical observations, dopamine antagonist neuroleptics do indeed ameliorate the symptoms of TS, but are effective in only 60–70% of cases and rarely abolish them. There is also a possibility that their effectiveness is related to 5-HT_2 rather than dopamine receptor blockade (for review see Handley & Dursun, 1992). That there is a dopamine input to the amygdala is not an argument for amygdaloid involvement, since such inputs exist to many other parts of the brain. Although an example is quoted of exacerbation of tics and induction of coprolalia by fluvoxamine, it may be noted that exacerbation, improvement, and no change, have been noted in other TS patients treated with selective serotonin-uptake inhibitors (Handley & Dursun, 1992).

Clues to the brain areas and neurotransmitters which may be affected in TS would indeed be of enormous value. However, we do not believe that Dr Jadresic has made a case for a key role of the amygdaloid complex.

BONNET, K. A. (1982) Neurobiological dissection of Tourette's syndrome: a neurochemical focus on a human neuroanatomical model. *Advances in Neurology*, **35**, 77–82.

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Propofol and ECT

SIR: We are able to support the retrospective observations of Mitchell *et al* (*Journal*, December 1992, **161**, 861–862) with the preliminary results of our prospective randomised comparison of methohexitone and propofol in terms of seizure duration and outcome in electroconvulsive therapy (ECT). It has often been demonstrated that seizure duration is

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