While evaluative studies and clinical experience both support a course of 5–12 treatments where generalised tonic–clonic seizures occur, the often-quoted 20–25 second minimum seizure duration is based only upon presumptive evidence (Swartz, 1992) and has not been supported by clinical outcome studies. Furthermore, we contend that this 'guesstimate" is without meaning and largely untestable unless and until a standardised mode of measurement is agreed and added. Motor timings are known to be shorter than EEG timings but, since many studies looking at EEG seizure durations use only a single lead EEG of the frontal/anterior temporal field, there is little information concerning the effects of left–right distribution of seizure activity (Fink & Johnson, 1982).

Clearly there is considerable scope for further work in this area with immediate consideration needed of the standardisation of seizure measurement, and larger, well-controlled studies to test the effect of seizure duration on antidepressive efficacy. Following this, EEG studies of seizure generalisation may prove more illuminating still.

FINK, M. & JOHNSON. L. (1982) Monitoring the duration of electroconvulsive therapy seizures. *Archives of General Psychiatry*, **39**, 1189–1191.

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MITCHELL, P., TORDA, T., HICKIE, L., *et al* (1991) Propofol as an anaesthetic agent for ECT: effect on outcome and length of course. *Australian and New Zealand Journal of Psychiatry*, **25**, 255–261.

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Toxic serotonin syndrome or extrapyramidal side-effects?

SIR: In the review article by Arya (*BJP*, December 1994, **165**, 728–733), a significant omission is the failure to take account of the toxic serotonin syndrome (TSS). There is now a growing literature concerning the extrapyramidal-like side-effects of selective serotonin reuptake inhibitors (SSRIs). However, it has previously been suggested that some of the cases in which extrapyramidal side-effects have been described following SSRI administration may be part of (or a mild form of) TSS (Dursun *et al*, 1993).

It is likely that TSS remains underreported and rather misdiagnosed because the full behavioural profile and clinical features have not been studied and reported widely. In clinical practice most patients receive multiple medication, thereby rendering the identification of the causative drug more difficult. We would like to draw attention to this intriguing syndrome, and suggest that TSS should be kept in mind during SSRI treatment when adverse effects occur. Its early recognition leads to the possibility of correction of the biochemical/receptor dysfunction by discontinuing the SSRI medication and using a non-selective serotonin receptor(s) antagonist: methysergide, cyproheptadine or propranolol (Sternbach, 1991; Dursun *et al*, 1993).

Although at present there is no widely accepted diagnostic criteria for TSS, a set of criteria has been suggested for this purpose (see below). Prospective studies are required to establish a definitive scale for TSS, and the re-evaluation of cases which resemble this syndrome may contribute further to understanding TSS and its differentiation from extrapyramidal symptoms.

Suggested diagnostic criteria for TSS: by Sternbach (1991), with additional clinical features from Insel *et al* (1982) and Dursun *et al* (1993).

(a) Coincident with the addition of or increase in a known serotonergic agent to an established medication regimen, at least three of the following clinical features are present:

- uncontrollable shivering
- incoordination
- restlessness in feet while sitting
- initial involuntary contractions followed by myoclonic-like movements in legs
- hyperreflexia
- frightened, diaphoretic hyperarousal state
- agitation
- oculogyric crisis
- diarrhoea
- fever

(b) Other aetiologies (e.g. infectious, metabolic, substance abuse or withdrawal) have been ruled out.

(c) An antipsychotic drug had not been started or increased in dosage prior to the onset of the signs and symptoms listed above.

DURSUN, S. M., MATHEW, V. M. & REVELEY, M. A. (1993) Toxic serotonin syndrome after fluoxetine plus carbamazepine. *Lancet*, **342**, 442.

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Suicide prevention in Gotland

SIR: Williams & Goldney (*BJP*, November 1994, **165**, 692) are quite right. Moving averages will occlude dramatic effects of an intervention. However, in the case of Rutz *et al*'s (1992) work, the reason that preceding falls in suicide rate seem smaller than the fall during the GP's educational programme is that the former was measured *every year*, whereas the latter drop was spread over *five years*.

As only four post-intervention data points were reported, the smoothest plot possible across the intervention is from a 4-year moving average. Taking Williams & Goldney's advice and ignoring all data from the intervention years themselves, it remains clear that there is no long-term effect of the programme on already-falling suicide rates.

Rutz *et al* (1992) implied that the short-term drop in rates during the programme, followed by a rise, might be evidence of a transient effect; the reader can best judge the likelihood of this by examination of previous annual fluctuations in the raw rates shown in my last letter (*BJP*, August 1993, **163**, 260).

I commiserate with any reader attempting to follow a discussion interrupted by intervals of anything up to 14 months of silence.

RUTZ, W., VON KNORRING, L. & WALINDER, J. (1992) Long-term effects of an educational programme for general practitioners given by the Swedish Committee for the Prevention and Treatment of Depression. *Acta Psychiatrica Scandinavica*, **85**, 83–88.

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