panic. Therefore, Marks *et al*'s findings (*Journal*, June 1993, **162,** 776–787) that exposure therapy, with or without alprazolam, is no better than placebo and relaxation in reducing panic is of interest. Since Marks *et al* affirm that relaxation is only a credible psychological placebo, the failure of exposure to serve as a specific anti-panic measure is evident.

However, this apparent confirmation of my views (Klein, 1988) is somewhat mitigated by the fact that in this study alprazolam also was of no benefit against panic. Marks *et al* believe this impugns the value of alprazolam, but to others (Spiegel *et al*, *Journal*, June 1993, **162,** 788–789) it calls the validity of his study into question. Unusual treatment findings are usually due to unusual samples. Marks *et al* state that at week 0, of the 144 intent to treat cases, the mean number of major panics per week was 7.4 with a standard deviation of 11. Since the standard deviation far exceeds the mean, it is possible that approximately 50% of the sample had no major panic attacks at all when entering the trial. We would appreciate it if Marks *et al* would provide the baseline frequency distributions of major and spontaneous panic attacks for the entire sample of 154 patients, as well as the 144 intent to treat cases. It could be illuminating concerning sample composition.

The authors use analyses of covariance, but apparently never do the necessary preliminary analysis for heterogeneity of slope. This would have spoken directly to the relevance of initial clinical status to treatment outcome. The use of *post hoc* comparisons, "controlled for experiment-wise error due to multiple comparisons" is also dubious, as it leads to an unnecessarily insensitive test for treatment differences. We have discussed both these crucial issues recently (Klein & Ross, 1993).

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AUTHORS' REPLY: Dr Klein seems to have missed key points in our paper (*Journal*, June 1993, **162,** 776–787) and in our replies to Spiegel *et al* and Klerman *et al* (*Journal*, June 1993, 790–794; 795–796): "... exposure therapy, with or without alprazolam, is no better than placebo and relaxation in reducing panic". In our study, exposure was twice better than alprazolam in reducing most problems of panic disorder with agoraphobia, and even more so compared with placebo. Like other symptoms, panic also improved markedly with exposure, but unlike other symptoms, panic also improved markedly with placebo and with alprazolam. In any case, both patients' and clinicians' independent global ratings of improvement correlated with reduction not in panic but in avoidance.

"Unusual treatment findings are usually due to unusual samples." Our findings were not 'unusual', and in fact agree with results from previous multicentre studies, for example Upjohn Phases I and II. Firstly, those studies also found a good response of panic symptoms to placebo, with no drug effect (not even for imipramine, which Dr Klein has long espoused for panic) on more complete analyses. Placebo is so effective for panic (as opposed to other) symptoms that no treatment does significantly better, whether it is alprazolam, imipramine, or exposure. Secondly, the previous studies also found a huge variance for panic measures.

These two findings are common in the literature and make panic problematic as an outcome measure. Such problems do not beset other measures such as avoidance, anxiety, depression, work and social disability. Dr Klein's belief that panic is the key to panic disorder with agoraphobia may need reconsideration.

"It is possible that approximately 50% of the sample had no major panic attacks at all when entering the trial." As we wrote, all our patients met DSM–III panic and other criteria for panic disorder at trial entry, including "at least one three-symptom panic a week, and at least two past unexpected three-symptom panics". We pointed out that our patients had even more panics at baseline than did patients in previous multicentre studies, and that Spiegel *et al* (*Journal*, June 1993, **162,** 788–789) were mistaken in writing that our sample differed from others with respect to baseline depression. Brevity precludes publication here of all the data Dr Klein asks for, but we would be glad to send these to him.

"Call for yet more analyses." In the London/Toronto study the results were similar with a wide variety of analytic methods. Apart from ANCOVA, MANCOVA and *post-hoc* analyses (whether or not controlled for experiment-wise error), the same pattern emerged for: effect size, survivor analyses and inspection of means, and across sites, raters, measures (including ten different criteria of panic symptoms), and intent-to-treat and evaluable patients.

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This testifies to the robustness of the main Anglo–Canadian findings.

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The importance of severity as a factor in post-traumatic stress disorder

SIR: There are several points that we would like to raise about the recent single case report by Spector & Huthwaite (*Journal*, July 1993, **163,** 106–108), on the treatment of Y, a patient reported to have post-traumatic stress disorder (PTSD) following a road traffic accident. We do not feel there is sufficient evidence in the report that the subject's experience fulfils the part of the DSM–III–R criteria A, of being outside the range of normal human experience. There is no mention of the subject thinking that her life was in danger, or that she witnessed any horrific scenes. Without such information we are not convinced that this case satisfies this criteria for a diagnosis of PTSD although it clearly is an experience which would be markedly distressing to most people.

Secondly, PTSD is a condition which presents with a wide spectrum of severity and this can be overlooked when employing a categorical diagnosis which can depend on the presence or absence of a single symptom. Neither DSM–III–R or ICD–10 enables one to measure symptom intensity on a continuous scale. The severity of Y's PTSD is not considered in the article and we believe that this variable is vital in assessing treatment efficacy.

One research tool developed by the National Centre for PTSD, USA, which has been incorporated in validation studies and outcome research by the PTSD Unit at RAF Wroughton (Neal *et al*, 1993), is the Clinician-Administered PTSD Scale-1 (CAPS-1; Blake *et al*, 1990). This is based on DSM–III–R criteria but in addition has an intensity scale assessing severity. We would suggest that clinicians involved in PTSD research consider the use of an instrument using both dichotomous and continuous scales. A revised computerised version of the CAPS-1 has been developed and validated at RAF Wroughton.

There is a need for well designed controlled studies into the treatment of this common and disabling condition. In our experience at the PTSD Units at RAF Hospital Wroughton and RN Hospital Haslar, even non-specific interventions, such as a simple psychiatric assessment, can result in a marked clinical improvement. Such an improvement could be misattributed to treatment methods in case reports or research studies where this is not considered.

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Right hemisphere damage v. dysfunction in Tourette's syndrome

SIR: "Diagnosing a cerebral dysfunction on the sole basis of neuropsychological test results" is a questionable clinical method as Lanser *et al* point out in their report of absence of right hemispheric dysfunction in Tourette's syndrome (*Journal*, July 1993, **163,** 116–118). They criticise previous reports interpreting Tourette's syndrome (TS) patient's poor performance on visual-perceptual tasks as suggestive of right hemispheric involvement in the pathogenesis of TS. To support their point, they report a neuropsychological comparison of individuals with TS and with "proven lesions of the right hemisphere (RH dysfunction)". Their "unexpected results" indicate that 7 to 16 children with "RH dysfunction" do not show any disturbance in right hemisphere functions (as assessed by their battery), and all of the children with TS perform "free from any neuropsychological signs or organic impairment". Since there are not many significant differences between the groups, they claim that this absence of difference should not lead us to identify TS as a RH dysfunction. However, there are many caveats which they do not discuss in the text.

Firstly, comparing two clinical groups in the absence of a normal control group is not "fair". Since there is not a one-to-one correspondence between

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