pursuers, and Stenersen (1972) describes how the artist used to assault complete strangers in response to the derogatory comments that he attributed to them.

The biographies are less clear about whether such symptoms can be totally attributed to Munch's undoubted misuse of alcohol, but whatever their cause the case of Edvard Munch helps to illustrate how great artistic achievement may result from both personal suffering and abnormal experience.

HELLER, R. (1984) *Munch: His Life and Work* (pp. 195–198). London: John Murray.
STENERSEN, R. (1972) *E. Munch: Close-up of a Genius* (p. 30). Oslo: Gyldendal Norsk Forlag.

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Lack of care in Rwanda

SIR: Rene Stockman's description (*BJP*, August 1994, **165**, 145–148) of psychiatric services in Rwanda is now tragically dated. I have recently returned from a mission to Rwanda on behalf of Physicians for Human Rights (UK). Many people will have seen the BBC report of our second visit to the hospital at Ndera near Kigali. We found only 22 surviving patients, most of whom appeared to be suffering from a recrudescence of psychotic symptoms. The last member of staff had left four days before our visit and the patients had received no medication for over a week.

Evidence of the ferocity of the attack on the hospital abounded. Grenade damage to floors and roofs was extensive and automatic fire had raked the building. Unburied human remains littered the ground to the rear of the hospital. Among the most shocking findings was the condition of three allegedly violent, psychotic patients who had been incarcerated by the refugees who had taken over most of the building. Confined to tiny cells, their chances of survival seemed slim.

The scale of psychological disturbance among the survivors of the recent genocide compounds the tragedy. We carried out a survey of psychiatric morbidity, in the towns of Rwamagana and Gahini in east central Rwanda. This area was chosen as being one of the most settled in the country. The massacres only occurred for two to three weeks before the RPF over-ran former government forces.

The instrument used was the 20-question neurotic subscale of the WHO Self Report Questionnaire (Hardinge *et al*, 1980). Preliminary scrutiny of the responses suggests a 'caseness' rate of over 90%. We deliberately excluded the psychotic subscale, since the first question in particular ("Do you feel that people are trying to harm you?") seemed so wholly inappropriate to the circumstances. Indeed the scale of the disaster that has befallen Rwanda almost defies quantification.

It is to be hoped that in the rush to meet the immediate and pressing physical needs of the people of Rwanda, some 70% of whom are either refugees or internally displaced, their equally urgent psychological needs are not ignored. In particular, appropriate models for the treatment of severely traumatised children, and training in their application, will be needed if the cycle of violence which has beset this country for so long is to be broken.

HARDINGE, T. W., DE ARANGO, M. V., BALTAZAR, J., *et al* (1980) Mental disorders in primary health care: a study of their frequency and diagnosis in four developing countries. *Psychological Medicine*, **10**, 231–241.

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Cognitive therapy in panic disorder

SIR: The study by Clark *et al* (*BJP*, June 1994, **164**, 759–769) poses problems concerning its claims for cognitive therapy in panic disorder.

1. The patients had relatively mild panic disorder. Apart from having no marked agoraphobia, they had only about 2.7 panics a week, compared to over four a week in larger multinational studies (Cross-National Study, 1992; Marks *et al*, 1993). The study gives no work or social disability measure.

The authors write of "the need for a psychological treatment for the less phobic panic disorder patients whose attacks were thought unlikely to be completely eliminated by situational exposure alone". Exposure is such a psychological treatment, and eliminated 96% of panics in severe panic/agoraphobia (see below). As it works best in less severe cases (Basoglu *et al*, 1994*a*), exposure would have been suitable for the 81% of Clark *et al*'s cases who had agoraphobic avoidance and for some of the remaining 19% who had situational panics.

2. The study's 'control' group was simply on a waiting list, which did not control for the non-specific factor of attendance and rating over 10 sessions – omission of such a placebo control is crucial. In the studies cited above placebo had a major anti-panic effect (but not an anti-phobia effect). Six months post-entry, although panic

frequency fell by 88% in Clark *et al*'s study, in the placebo group of Marks *et al* (1993) it fell by 85% (96% if the criterion was spontaneous major panics) despite panics having been more frequent at the start than in the Clark *et al* study.

3. The authors aimed to test how "cognitive therapy compares with alternative, established psychological ... treatments for panic disorder", but for that comparison used a weak form of exposure therapy – applied relaxation – which only "introduced exposure after 4 sessions". The authors do not note having asked patients to do self-exposure homework for at least an hour a day or to keep self-exposure homework diaries for review at each session. The outcome of exposure therapy depends on how systematically it is given.

The authors report no check of whether cognitive therapy patients had firmer exposure instructions in doing "behavioural experiments" than did the applied relaxation and imipramine groups, which is a possible reason for the differences.

4. "Cognitive measures at end of treatment were significant predictors of outcome at follow-up". The authors' analyses are insufficient to gauge the value of cognitions as predictors. A multiple regression analysis was desirable to control for the effects of pre-treatment severity, treatment condition, and non-cognitive outcome measures, and to test which variables gave the strongest predictions. Clark *et al* did not exclude the possibility that their non-cognitive measures were as predictive as cognitive ones.

"Sustained improvement ... will depend on cognitive change having occurred during the course of therapy." After rigorous testing the effective ingredients of treatments often turn out to be different from those postulated. The authors have not excluded the role of exposure and/or sense of control regardless of misinterpretations concerning bodily sensations. Basoglu *et al* (1994*b*) found that panic fell dramatically without cognitive therapy, so panic patients can improve without trying to change their interpretations of bodily sensations. Such change may arise because of (i) indirect exposure to such sensations through exposure to external situations or (ii) direct exposure to such sensations (interoceptive exposure) as in Clark *et al*, without a need to do cognitive restructuring.

5. The authors report no test of whether the assessor actually remained blind throughout the study.

6. The cost implications of the lack of balance of therapist time are not addressed. Although imipramine patients had more sessions after six months, their total time with the clinician since the start was $(10.5+1.6+2.8) \times 24=358$ min, whereas cognitive therapy patients had $(10.5+2.2+0.4) \times 58=760$ min, and applied relaxation patients had $(10.3+2.0+1.5) \times 57=787$ min.

7. The authors report 88% of cognitive therapy patients becoming panic-free yet their Table 2 shows a reduction at six months of only 27% in agoraphobic cognitions and 43% in bodily sensations interpretations.

BASOGLU, M., MARKS, I. M., KILIÇ, C., *et al* (1994*a*) Relationship of panic, anticipatory anxiety, agoraphobia and global improvement in panic disorder with agoraphobia treated by alprazolam and exposure. *British Journal of Psychiatry*, **164**, 647–652.

——, ——, ——, *et al* (1994*b*) Alprazolam and exposure for panic disorder with agoraphobia: attribution of improvement to medication predicts subsequent relapse. *British Journal of Psychiatry*, **164**, 652–659.

CROSS-NATIONAL COLLABORATIVE PANIC STUDY, SECOND PHASE INVESTIGATORS (1992) Drug treatment of panic disorders. Comparative efficacy of alprazolam, imipramine and placebo. *British Journal of Psychiatry*, **160**, 191–202.

MARKS, I. M., SWINSON, R. P., BASOGLU, M., *et al* (1993) Alprazolam and exposure alone and combined in panic disorder with agoraphobia: A controlled study in London and Toronto. *British Journal of Psychiatry*, **162**, 776–787.

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AUTHOR'S REPLY:

1. Milder panic disorder Marks *et al* claim that the patients in our study "had only 2.7 panics a week, compared to over four a week in larger multinational studies". This is incorrect. It seems that Marks *et al* failed to read our article carefully. The figure of 2.7 refers to a 0–4 point rating scale (see p. 762), not to absolute panic frequency. Absolute panic frequency is a problematic measure as it is never normally distributed and for this reason we made an *a priori* decision not to include it as an outcome measure. In view of Marks *et al*'s comments, we have gone back to the patients' files and can confirm that the absolute panic frequency for patients included in the study is 4.3 per week (range 1 to 40), making it comparable with the other studies cited by Marks. Marks *et al* also claim that our patients were milder because none had "marked agoraphobia". Marked agoraphobia is not a defined term. We used DSM-III-R which has four categories of agoraphobic avoidance (none, mild, moderate, severe). Using these categories 81% of our patients had some degree of agoraphobic avoidance: 48% fell into the mild category and 33% fell into the moderate category. We excluded patients