Surviving disaster: what comes after the trauma?†

JONATHAN R. T. DAVIDSON

More than 10 years after the Piper Alpha oil platform tragedy, Hull, Alexander and Klein have assessed the health status and outcome of 36 of the original 59 survivors (Hull *et al*, 2002, this issue). Their important study has several strengths, including its ability to trace and assess a comparatively high proportion of survivors, the lack of ongoing litigation, the sample's relatively good premorbid function, the use of valid and widely used instruments, and the corroboration of much of the information reported by subjects. Included in the description of their findings are information on current psychopathology, characteristics of individual experiences at the time of the event, predictors of long-term outcome, perceived benefits of treatment received after the event, and reported effects of the trauma, both positive and negative. The study touches on several issues which are important to surviving trauma, five of which will be discussed here. These are: the types of psychopathology seen after trauma; early identification of those at risk for post-traumatic stress disorder (PTSD); prevention of PTSD; optimal treatment of established PTSD; and other conditions affecting treatment, such as resilience.

TRAUMA SPECTRUM DISORDERS

Perhaps one of the greatest benefits of including PTSD as an official diagnosis in the DSM and ICD systems is that this highlights the traumatic experience, and officially recognises that exposure to exceptionally severe stress may result in psychopathology that can continue long after the stressor has ended. However, few people have believed that PTSD is the only manifestation of psychopathology following trauma. A family of post-traumatic problems was described by Davidson & Foa (1993), with post-traumatic depression receiving particular attention. Attempted suicide is a significant risk (Davidson *et al*, 1991), and Hull *et al* describe substantial guilt, a feature which we have found to be predictive of a poor response to amitriptyline (Davidson *et al*, 1993). Additional post-traumatic difficulties include excessive alcohol intake. Hull *et al* also observe a high rate (44%) of 'caseness' elicited by the General Health Questionnaire (GHQ; Goldberg & Hillier, 1979), again underscoring the fact that full PTSD by no means accounts for the entire spectrum of post-traumatic psychological problems. Partial PTSD is often found in the community, with a prevalence rate of 4.1% (Davidson *et al*, 2002). We therefore need to be alert to a broad spectrum of psychiatric problems after major trauma.

EARLY IDENTIFICATION OF THOSE AT RISK FOR PTSD

Exposure to a single, discrete and clearly identifiable traumatic event affords the opportunity for early detection. What could help us here? Although no predictive test gives high diagnostic accuracy for PTSD, there are some useful clinical leads. Hull *et al* found that serious physical injury (fractures), persistent anger and witnessing the death or injury of others were all associated with more severe PTSD symptoms at follow-up. Others have found that elevated heart rate within the first week following trauma (Shalev *et al*, 1998) and low urinary cortisol (Resnick *et al*, 1995) increase the risk of developing chronic PTSD. Additional warning signals include severe depression and dissociation. Extent of trauma exposure and lack of social support are also important considerations (Brewin *et al*, 2000). Intensity of exposure to (combat) trauma can influence treatment response as far into the future as 20–40 years (Davidson *et al*, 1993), which suggests that the most severely traumatised group of survivors may be in particular need of vigorous early treatment. Could such treatment alter the trajectory of the disorder? By paying attention to these phenomena, we may be better able to detect and treat post-traumatic stress at an earlier stage.

PREVENTION OF PTSD

Prevention can be primary (preventing exposure to trauma), secondary (preventing development of PTSD immediately after exposure to trauma) and tertiary (prevention of worsening once PTSD has emerged).

Two studies have suggested promising biological approaches in secondary prevention: medication using propranolol for 2 weeks after accident trauma (Pitman *et al*, 2002); and medication with hydrocortisone for 12 days in septic shock (Schnelling *et al*, 2001). Interestingly, each of these treatments targets core central nervous system (CNS) disturbances which are found in PTSD. Beta-adrenergic receptor blockade counteracts the massive sympathetic response seen after trauma. It also lowers the heart rate, and thereby may abolish a symptom which predicts (or maybe leads to) PTSD and which is a symptom of established PTSD. Hydrocortisone, which may compensate for low cortisol status, was successful in preventing the development of PTSD in the Schnelling *et al* study. We do not yet know whether secondary prevention can be effectively accomplished by psychological interventions. Studies by Deahl *et al* (2000), Larsson *et al* (2000) and Gidron *et al* (2001) suggested possible benefit, but standard single-session debriefing (Conlon & Fahy, 2001) is not to be recommended.

Data for prevention of PTSD by treating acute stress disorder are limited but encouraging, both for drug therapy with imipramine in children (Robert *et al*, 1999) and for cognitive–behavioural therapy in adults (Bryant *et al*, 1999).

TREATMENT OF ESTABLISHED PTSD AND RELATED DISORDERS

An unusual, but welcome, finding from Hull *et al*'s study is the high rate of treatment utilisation, suggesting relative

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ease of access to treatment services, greater community support and less personal resistance to accepting help. We must not ignore the finding that non-professional support was perceived as being by far the most helpful. The low levels of perceived benefit from medication (28%) and group therapy (14%) are matters of concern. These more expensive procedures need to be used in the most cost-effective way. Clearly we need to work harder to optimise treatment benefit, and to be cognisant of what our patients think about the treatments in which we are invested. As far as medication is concerned, the study does not report which agents were used, their doses, duration of administration, or whether side-effects were a problem; all these matters need to be addressed because response rates to medications can be good. Recent studies of selective serotonin reuptake inhibitors (SSRIs) and mirtazapine, for example, indicate rates of response above 50%. Cognitive therapy is also highly effective for PTSD, yet it can be associated with a troublesome rate of attrition (Scott & Stradling, 1997).

In thinking about treatment response, we need to define our goals. For many with PTSD full remission is attainable (Connor *et al*, 1999; Hembree & Foa, 2000) and is an expectation which we can set for ourselves and our patients. Although the Piper Alpha follow-up study points to a decline over time in the rates of PTSD, we cannot automatically assume that those without the diagnosis are necessarily well; as the authors state, we need to be attentive to intermediate levels of pathology. It appears that those who survived well the collapse of Piper Alpha had good premorbid functioning and support networks. Lack of neuroticism has been found to predict response to a tricyclic drug in chronic PTSD (Davidson *et al*, 1993).

Comprehensive treatment of PTSD and related disorders can be approached along a continuum of early detection, decision-making about the appropriateness of treatment in the acute phase, selection of such a treatment, decision-making as to whether and when early treatment can be discontinued, and then follow-up. For some individuals long-term treatment will be needed, although others will manage adequately without. Yet others will recover, but relapse later. Non-professional support can pick up an important part of the recovery burden. When people are physically injured, as indicated by Alexander's group, effective liaison needs to be established between professionals involved in the delivery of psychiatric, medical and surgical care. Such a model of care may have especial applicability after a disaster wherein rapid intervention is more feasible and there is the possibility of early detection, secondary or tertiary prevention, and follow-up.

CONCLUSION

Last, but not least, the authors report the subjects' own views of how they were affected by the tragedy. Detrimental effects on the quality of relationships and leisure activities, on well-being and on employment were all noted, serving as a reminder to the clinician that the disorder is more than its defining symptoms. A counterbalancing observation, however, was the fact that 61% perceived some good resulting from their painful experience, whether this was enriched relationships, emotional growth or financial security, reminding us that out of trauma can come further resilience. How much this was the result of self-directed effort and how much a result of treatment is a question worth asking. It may well be that a crucial task of treatment is to help strengthen the resilience of trauma survivors, enabling them to cope more effectively with stressful events and with the general demands of life, as we have previously shown can be achieved (Connor *et al*, 1999). Resilience has been a neglected topic of study in the therapeutics of PTSD, perhaps related to the lack of suitable measures. As there may be a characteristic neurobiology associated with resilience (Morgan *et al*, 2000), the subject deserves further study.

No study can answer all the questions, and the authors acknowledge some very reasonable limitations of their follow-up study. Nevertheless, it is a valuable, carefully conducted survey, which gives us the opportunity to consider a number of major questions concerning outcome after exposure to serious trauma.

JONATHAN R. T. DAVIDSON, MD, Duke University Medical Center, Department of Psychiatry and Behavioral Sciences, Trent Drive, Yellow Zone, 4th Floor, Room 4082 B, Box 3812, Durham, NC 27710, USA. Tel: 919 684 2880; fax: 919 684 8866; e-mail: jonathan.davidson@duke.edu

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DECLARATION OF INTEREST

None.

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