

# Psychological debriefing for acute trauma – a welcome demise?

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Psychological debriefing (PD) has until recently been widely regarded by the public and by health professionals as a necessary and effective prophylactic treatment strategy for trauma victims. This view may have its origins in Rachmann's 'emotional processing' hypothesis<sup>1</sup> that the earlier intervention occurs, the less opportunity there is for maladaptive cognitive and behavioural patterns to develop. Over the years the theory has gained support from different quarters.

Contemporary belief in the need for counselling post trauma, media attention to disasters and fear of litigation from victims are amongst the many influences that may have contributed towards the longevity of PD following trauma. Debriefing has been seen as a mandatory prescription and quick fix for all persons experiencing traumatic events. However, in this age of evidence based practice it is important to note that little empirical evidence exists to support this view: that debriefing may have damaging effects now appears more likely.

Debriefing was initially developed by Mitchell in 1983 as an intervention strategy for emergency personnel with the aim of lessening the impact of trauma (critical incidents) and helping them return to routine functioning (Critical Incident Stress Debriefing).<sup>2</sup> The process provides opportunity for group discussion about an incident with focus on how personnel have managed and currently are coping. It was hoped that the procedure would lessen the impact of trauma by encouraging processing of the traumatic experience in a supportive and confidential environment.

The technique has been modified by others and 'psychological debriefing' describes a similar therapy in disaster situations.<sup>3</sup> Apart from use with emergency workers and military personnel, proponents of debriefing have advocated the procedure for civilians involved in trauma: hence the birth of what some have called a 'disaster industry' led by different professional groups including counsellors, psychologists, social workers and psychiatrists, not to mention lawyers who advertise for clients. The basis for this practice appears to have derived from anecdotal and single case reports of good outcome, and from positive comments by individuals who received debriefing.<sup>4</sup> The 1990s saw a steady increase in scepticism amongst therapists disappointed by the poor outcome of many cases.

Following repeated calls for properly conducted research

studies<sup>5,6</sup> a number of randomised controlled trials of debriefing were published in the latter half of the past decade.<sup>7-9</sup> A recent Cochrane Review by Wessely *et al*<sup>10</sup> found only eight trials which fulfilled criteria for randomisation and single session debriefing. Analysis of these trials suggested that single session debriefing neither reduced psychological distress nor prevented the subsequent onset of post traumatic stress disorder (PTSD), the most feared long-term psychiatric sequel. There was also no evidence that debriefing reduced general psychological morbidity, depression or anxiety. Two studies suggested that debriefed subjects might even fare worse than controls.<sup>7,9</sup> The Cochrane Review concluded that there is no current evidence that single session debriefing is useful for the prevention of PTSD and that compulsory debriefing of trauma victims should now cease.

These conclusions of the Cochrane review were strengthened by a recently published randomised controlled trial of debriefing by Mayou *et al*<sup>11</sup> which reported on the three year outcome of a sample of hospitalised road traffic accident victims previously documented in follow-up to four months. The intervention group had a significantly worse outcome at three years in terms of general psychiatric symptoms, travel anxiety when a passenger, pain, physical problems, overall level of functioning, and financial problems than the control group. Conlon *et al*<sup>8</sup> in a smaller Irish study of very early intervention found that 9% of minor road traffic accident victims developed PTSD. They confirmed earlier findings that the best predictor of short-term morbidity was high initial distress levels soon after trauma.<sup>12-14</sup> Also they found that randomised debriefing showed no benefit. Shalev's finding<sup>15</sup> that high pulse rates recorded at A&E departments soon after trauma significantly predicted PTSD is in keeping with these observations. Mayou *et al* noted that for subjects with low initial stress scores, it did not make any difference whether they were debriefed or not. Amongst subjects with high initial stress scores, however, post traumatic symptom outcome was significantly worse with debriefing, both at four months and three years, compared with controls. It follows that those most at risk of developing post-trauma symptoms may be the very ones most likely to be adversely affected by debriefing and, paradoxically, may be the ones who most vigorously seek and obtain treatment.

## Medical litigation

If, as now appears, debriefing may do more harm than good, it seems possible that the debriefing of trauma victims in the course of litigation following accidents may also have harmful effects. Constant, involuntary rehearsal of traumatic experiences at the behest of lawyers and medical experts might compound symptoms, raise levels of distress and put victims at increased risk of chronic psychi-

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atric disability. No systematic data are yet available in this area, but the need for further inquiry is now apparent.

### Recovered memories

There is a parallel between involuntary debriefing and the problems of recovered memories of childhood sexual abuse highlighted by the well known and highly influential Brandon Report.<sup>16</sup> Long held beliefs that abreaction and verbalisation of traumatic memories are clinically helpful must now be balanced against a growing number of accounts of distress experienced by subjects required to 'recover memories' or 'relive trauma' by over-enthusiastic therapists. Brandon et al refer to a study of 26 cases of recovered memories of abuse from a Washington Victim Compensation Programme where the 'recovery' and abreaction had serious adverse effects for the patients in terms of suicidality, hospitalisation, self-mutilation and marriage break-up.<sup>17</sup> Public concern in this area in North America and Europe is reflected in the multiplicity of overlapping internet sites which readily appear in response to requests keyed in as *false memory syndrome* or *psychological debriefing*.

### PTSD

One of the most disabling psychiatric sequelae of trauma of any type is chronic PTSD particularly if complicated by depression. Whilst the precise aetiology of PTSD remains elusive, the possibility that it may have a biological substrate seems increasingly likely.<sup>18</sup> The latent interval before onset and the recent finding of a resurgent PTSD in dementing war veterans<sup>19</sup> are remarkable phenomena awaiting explanation. We know little about the efficacy of psychological or pharmacological treatments. Methodological concerns include the difficulties of obtaining homogeneous subject samples and ethical concerns about the use of control subjects. Randomised controlled trials have been mostly confined to war veterans. There is some evidence supporting the use of cognitive behavioural therapy for PTSD, but not all patients benefit.<sup>20</sup>

Antidepressants have been shown to reduce symptoms in some patient groups.<sup>21</sup> Virtually nothing is known of prophylactic strategy and current pessimistic views are still

confined to single session interventions. Until we know more psychiatrists should be slow to condemn the possible benefits of ordinary Rogerian counselling in single cases and disaster situations. Meanwhile, it appears clear that psychological debriefing for trauma victims is now widely considered to be neither appropriate nor safe.

### References

1. Rachmann S. Emotional Processing. *Behav Res & Ther* 1980; 18: 51-60.
2. Mitchell JT. When disaster strikes...the critical incident debriefing process. *J Emergency Medical Services* 1983; 8: 36-9.
3. Dyregrov A. Caring for helpers in disaster situations: Psychological Debriefing. *Disaster Management* 1989; 2 (1): 25-30.
4. Hytten K, Hasle A. Fire fighters: a study of stress and coping. *Acta Psych Scand* 1989; 80(Suppl. 355): 50-5.
5. Bisson JI, Deahl MP. Psychological debriefing and prevention of post-traumatic stress. *Br J Psychiatry* 1994; 165: 717-20.
6. Raphael B, Meldrum L, McFarlane AC. Does debriefing after psychological trauma work? *BMJ* 1995; 310: 1479-80.
7. Hobbs M, Mayou R, Harrison B, Worlock P. A randomised controlled trial of psychological debriefing for victims of road traffic accidents. *BMJ* 1996; 313: 1438-9.
8. Conlon L, Fahy T, Conroy R. PTSD in ambulant RTA victims: a randomised controlled trial of debriefing. *J Psychosomatic Research* 1999; 46 (1): 37-44.
9. Bisson LI, Jenkins PL, Alexander J, Bannister C. Randomised controlled trial of psychological debriefing for victims of acute burn trauma. *Br J Psychiatry* 1997; 171: 78-81.
10. Wessely S, Rose S, Bisson J. A systematic review of brief psychological interventions ('debriefing') for the treatment of immediate trauma related symptoms and the prevention of post traumatic stress disorder. London. The Cochrane Library 1998; 2: 1-17.
11. Mayou RA, Ehlers A, Hobbs B. Psychological debriefing for road traffic accident victims: Three year follow-up of a randomised controlled trial. *Br J Psychiatry* 2000; 176: 589-93.
12. Shalev AY, Peri T, Canetti L, Schreiber S. Predictors of PTSD in injured trauma survivors: a prospective study. *Am J Psychiatry* 1996; 153:219-225.
13. Feinstein A, Dolan R. Predictors of post-traumatic stress disorder following physical trauma: an examination of the stressor criterion. *Psychol Med* 1991; 21: 85-91.
14. Shalev AY, Freedman S, Peri T, Brandes D, Sahar T. Predicting PTSD in trauma survivors: prospective evaluation of self-report and clinician-administered instruments. *Br J Psychiatry* 1997; 170: 558-64.
15. Shalev AY, Sahar T, Freedman S, Peri T, Glick N, Brandes D, Orr SP, Pitman RK. A prospective study of heart-rate response following trauma and the subsequent development of PTSD. *Arch Gen Psychiatry* 1998; 55(6): 553-9.
16. Brandon S, Boakes J, Glaser D et al. Recovered memories of childhood sexual abuse. Implications for clinical practice. *Br J Psychiatry* 1998; 172: 296-307.
17. Loftus EF. Repressed memory accusations: Devastated families and devastated patients. *Applied Cognitive Psychology* 1997; 11: 631-47.
18. Nutt D.J. The psychobiology of posttraumatic stress disorder. *J Clin Psychiatry* 2000; 61: suppl 5: 24-9.
19. Johnston D. A series of cases of dementia presenting with PTSD symptoms in World War II combat veterans. *J Am Geriatric Society* 2000; 48 (1): 70-2.
20. Foa E. B. Psychosocial treatment of post traumatic stress disorder. *J Clin Psychiatry* 2000; 61: suppl 5: 43-8.
21. Davidson JRT. Pharmacotherapy of post traumatic stress disorder: treatment options, long-term follow-up, and predictors of outcome. *J Clin Psychiatry* 2000; 61: suppl 5: 52-6.

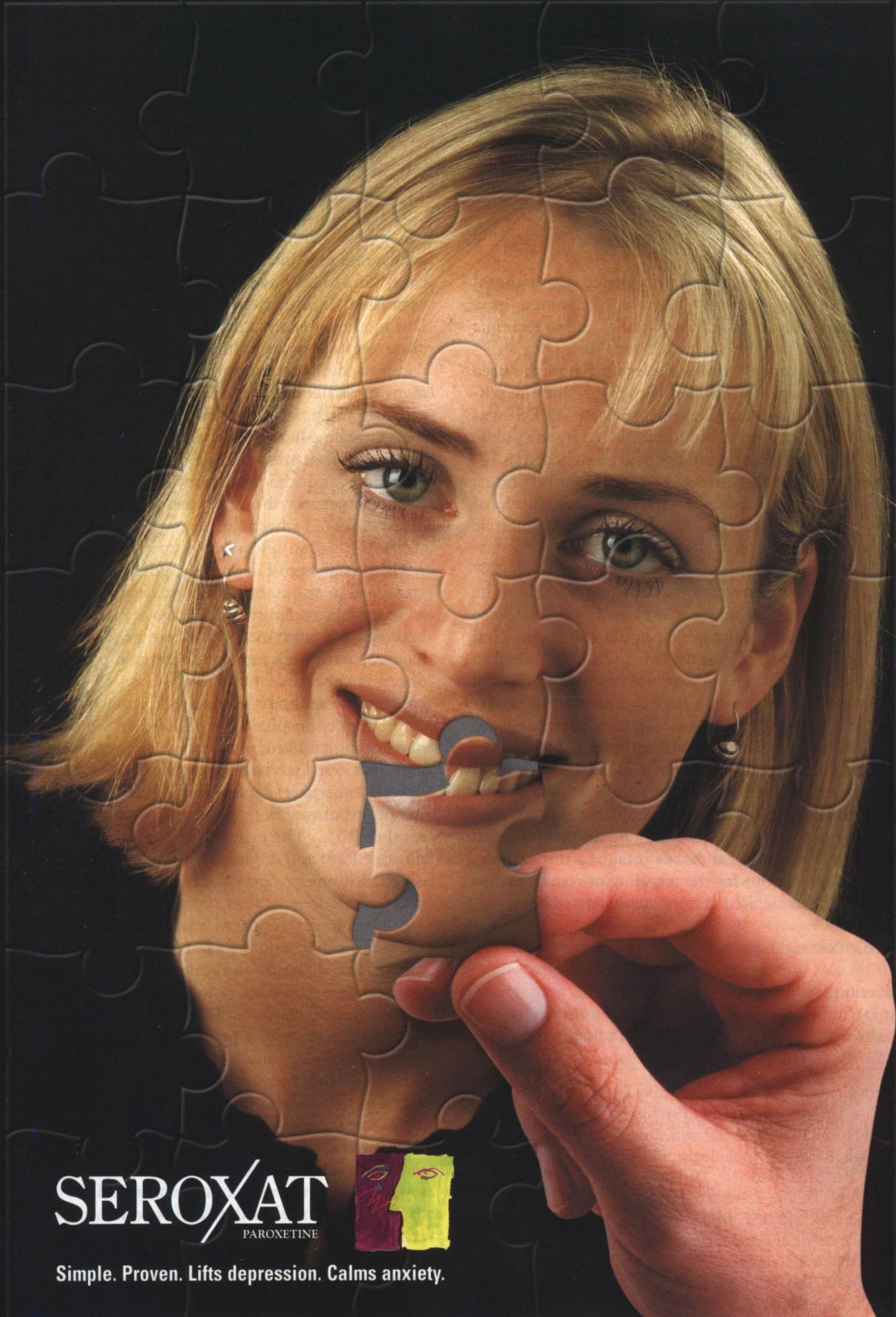
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