

## Post-trauma debriefing: the road too frequently travelled

See page 766

The meta-analysis by Arnold van Emmerik and colleagues in today's *Lancet* about the efficacy of post-trauma psychological debriefing stands among the more potent entries in an increasing litany of reports, reviews, and consensus statements. The latest report raises significant concerns about this ubiquitous intervention. Despite limitations from poor data-quality and uneven design in the studies assessed, the analysis is consistent with those of other researchers, indicating that debriefing: (1) yielded no demonstrable effect on subsequent resolution of traumatic exposure and may inhibit or delay resolution for some participants; (2) showed a smaller effect than calculated for non-intervention controls, suggesting that natural proclivities toward resilience may be more potent than this style of intervention and (3) yielded lower effect-sizes than alternative interventions against which it was compared, raising the strong likelihood that other approaches are more likely to help.

The findings echo and extend assessments of multiple randomised trials in Cochrane Collaboration reviews.<sup>1</sup> Although such meta-analyses have been criticised for lacking studies of group debriefing within the specific occupational settings in which the practice originated (rather than in traumatised individuals more generally), well-designed quasi-experimental field studies in those contexts have also yielded negative or equivocal findings,<sup>2-4</sup> which leaves the burden of proof about efficacy with proponents of debriefing.

The implications for practice are unequivocal. Calls for caution and restraint have been heard from many responsible scientists and practitioners,<sup>5-9</sup> and are underscored in conclusions from consensus panels<sup>10</sup> and empirically-based practice guidelines that have recommended limitation<sup>11,12</sup> or contraindication.<sup>13,14</sup> But despite direct and publicised warnings from well-established researchers in trauma response and intervention,<sup>15,16</sup> reports from New York City after the attacks on the World Trade Center indicated that more than 9000 purveyors of debriefing and other popularised interventions—more than three counsellors for every person believed to have died in the attack—swarmed there, advocating intervention for any person even remotely connected to the tragedy.<sup>17</sup>

Given the evidence, why should use of debriefing techniques not only persist but also seemingly flourish? Post-traumatic stress disorder is much debated.<sup>18</sup> Progressive dilution of both stressor and duration criteria has so broadened application that it can now prove difficult to diagnostically differentiate those who have personally endured stark and prolonged threat from those who have merely heard upsetting reports of calamities striking others. Moreover there are few systematic data about the normal course of resolution after traumatic exposure or the inherent variability of that course within and between individuals, a fact that leaves discernment between symptoms of arrested or abnormal processing and normal signs of sometimes profound but ultimately transient discomfiture a subject of speculation.

The problem is compounded further in practice, where the enterprise of debriefing has become dominated by a prolific and parochial subculture of secondary providers whose understanding of these highly complex and elusive issues is often limited to proprietary workshops, trade magazines, and paperback books rather than the peer-reviewed venues of empirically guided professional practice. This has, in turn, created entrenched enclaves of self-

identified debriefers within various organisations—initially in public safety and military concerns, but now extending into schools, hospitals, and a widening range of other enterprises—who earnestly strive to help but stand severely hampered by the tools they have been sold.

Although immediate debriefing has yielded null or paradoxical outcomes, the value of contemporaneous instrumental assistance and support—those kinds of practical help often learned better from grandmothers than from graduate training—has increasingly been found to be useful in disaster response.<sup>19</sup> Structured interventions, however, may be better embedded in models of stepped care, where the nature and level of intervention is conservatively tailored to the needs, context, and course of individual resolution.<sup>20,21</sup> Preliminary epidemiological data from New York City have revealed levels of post-traumatic stress disorder that, whilst clearly significant, fell below even conservative early prognostications<sup>22</sup> and which had dropped by more than two-thirds within 4 months.<sup>23</sup> These findings underscore the counterproductive nature of offering a prophylaxis with no demonstrable effect, but demonstrated potential to complicate natural resolution, in a population in which limited case-conversion can be anticipated, strong natural supports exist, and spontaneous resolution is prevalent.

Promising approaches are emerging, with high sensitivity and specificity, allowing straightforward and relatively non-intrusive assessment to identify those at greatest risk of clinical progression to post-traumatic stress disorder.<sup>24</sup> These approaches are designed for implementation 2–4 weeks post-impact, when brief-series cognitive behavioural therapy has efficacy in treating post-traumatic stress disorder in high-risk populations.<sup>21</sup>

\*Richard Gist, Grant J Devilly

\*Kansas City, Missouri Fire Department, and University of Missouri-Kansas City, Kansas City, MO 64106, USA; and Departments of Criminology and Psychology, University of Melbourne, Melbourne, Australia (e-mail: Richard.Gist@kcmo.org)

- Rose S, Bisson J, Wessley S. Psychological debriefing for preventing post traumatic stress disorder (PTSD) (Cochrane Review). In: *The Cochrane Library*, issue 3. Oxford: Update Software; 2001.
- Carlter IVE, Lamberts RG, van Uchlen AJ, Gersons BPR. Disaster related post traumatic stress in police officers. *Stress Med* 1998; **14**: 143–48.
- Gist R, Lubin B, Redburn BG. Psychosocial, ecological, and community perspectives on disaster response. *J Personal Interpersonal Loss* 1998; **3**: 25–51.
- Macnab AJ, Russell JA, Lowe JP, Gagnon F. Critical incident stress intervention after loss of an air ambulance: two-year follow-up. *Prehospital Disaster Med* 1998; **14**: 8–12.
- Bisson JI, Deahl MP. Psychological debriefing and the prevention of post-traumatic stress: more research is needed. *Br J Psychiatry* 1994; **165**: 717–20.
- Deahl MP, Bisson JI. Dealing with disasters: does psychological debriefing work? *J Accid Emerg Med* 1995; **12**: 255–58.
- Raphael B, Meldrum L, McFarlane AC. Does debriefing after psychological trauma work? Time for randomised controlled trials. *Br J Psychiatry* 1995; **310**: 1479–80.
- Gist R, Lohr JM, Kenardy JA, et al. Researchers speak on CISM. *J Emerg Med Serv* 1997; **22**: 27–28.
- Kenardy JA. The current status of psychological debriefing. *BMJ* 2000; **321**: 1032–33.
- Ritchie, EC. Draft consensus document of the Mass Violence and Early Intervention Workshop. Washington, DC: US Department of Defense 2001.
- Bisson JI, McFarlane AC, Rose S. Psychological debriefing. In: Foa EB, Keane TM, Friedman MJ, eds. *Effective treatments for PTSD*. New York: Guilford Press; 2000: 39–59.
- Raphael B. *Mental health disaster training manual*. Sydney: New South Wales Department of Health, 1999.
- Perry G. *Evidence based clinical practice guidelines for treatment choice in psychological therapies and counselling*. London: UK Department of Health, 2001.
- Wessely S, Krasnov V. NATO-Russia advanced workshop on social and

psychological consequences of chemical, biological, and radiological terrorism: preliminary notes. NATO HQ, March 25–27, 2002. <http://www.nato.int/science/e/020325-arw2.htm> (accessed Aug 30, 2002)

- 15 Gist R. A message of caution. *APS Observer* 2001; **14**: 16–17.
- 16 Herbert JD, Lilienfeld S, Kline J, et al. Psychology's response: PRIMUM NON NOCERE. *Monitor Psychol* 2001; **32**: 4.
- 17 Kadet A. Good grief! *Smart Money* 2002; **11**: 108–14.
- 18 Summerfield D. The invention of post-traumatic stress disorder and the social usefulness of a psychiatric category. *BMJ* 2001; **322**: 95–98.
- 19 Gist R, Lubin B, eds. Response to disaster: psychosocial, community, and ecological approaches. Philadelphia: Brunner/Mazel, 1999.
- 20 Bisson, JJ, Roberts N, Macho G. The Cardiff Traumatic Stress Initiative: an evidence-based approach to psychological intervention following traumatic events. *Psychiatr Bull R Coll Psychiatry* (in press)
- 21 Litz BT, Gray, MJ, Bryant, RA, Adler AB. Early intervention for trauma: current status and future directions. *Clin Psychol Sci Pract* 2002; **9**: 112–34.
- 22 Galea S, Ahren J, Resnik H, et al. Psychological sequelae of the September 11 terrorist attacks in New York City. *N Engl J Med* 2002; **346**: 982–87.
- 23 Galea S, Boscarino J, Resnik H, Vlahov D. Mental health in New York City after the September 11 terrorist attacks. In: Manderscheid RW, Henderson MJ, eds. Mental health United States, 2001. Washington, DC: US Government Printing Office (in press).
- 24 Brewin CR, Rose S, Andrews B, et al. A brief screening instrument for post-traumatic stress disorder. *Br J Psychiatry* 2002; **181**: 158–62.

## Sperm mRNA—what does daddy do?

See page 772

7–10% of men have male-factor infertility, mostly idiopathic. Whether sperm counts are declining<sup>1</sup> and whether environmental factors (eg, oestrogens, pesticides, underwear) are among the culprits is controversial. Advanced paternal age influences pregnancy success as well as birthweights.<sup>2</sup> Infertile couples are increasingly seeking assisted reproduction technologies (ART) and conception by ART accounts for about 5% of births in some European countries and probably for about 1% in the USA.<sup>3</sup> There used to be no ambiguity about good sperm: swift, normal shape, explosive acrosome release to get through the egg's zona pellucida, and the more sperm, the better.

In 1992, Palermo et al<sup>4</sup> revolutionised the treatment of male infertility with intracytoplasmic sperm injection (ICSI): a single sperm is microinjected into the egg, and the rates of fertilisation and pregnancy success are astonishing. Now men with immotile, misshapen, or few sperm can father children. Furthermore, men with no sperm in their ejaculates or testicles can be fathers by the injection of immature spermatids obtained by testicular aspiration or biopsy (certainly by injection of elongated spermatids, although reports of injections of round spermatids remain controversial<sup>5</sup>). ICSI is a renaissance for infertile men since pregnancies are now routine even with single immotile, immature, dysfunctional, and dysmorphic sperm. Ironically ICSI, by rendering traditional sperm-assays nearly obsolete, has created a void in diagnosing male-factor infertility.

What does daddy do? This question, even before the cloning of Dolly the sheep is considered,<sup>6</sup> is now more answerable with today's report in *The Lancet* by Charles Ostermeier and colleagues. Successful reproduction requires perfect complementation between sperm and egg, and several paternal contributions: the properly imprinted haploid genome,<sup>7</sup> activation signal<sup>8</sup> or signals,<sup>9,10</sup> the sperm's centrosome,<sup>11,12</sup> and now, with the Ostermeier report, perhaps also vital mRNAs. ICSI's success suggests that sperm motility, the acrosome reaction,<sup>13</sup> and morphology are not vital; nor might sperm mitochondria be needed.<sup>14</sup> Since 1997, seven mammalian species have been cloned by somatic-cell nuclear transfer (SCNT). Reproduction by SCNT violates the requirement for exactly two parents of opposite sexes but it is inefficient, possibly because of the absence of some vital RNA from sperm which, according to

Ostermeier and colleagues, include several involved in fertilisation and early embryogenesis. Oocytes microinjected with RNA interference (RNAi), antisense to these identified human sperm mRNAs, studies in either mice or nonhuman primates will provide answers. However, the discovery of mRNA in sperm raises questions about RNA devoid of poly A tails. The mRNAs were sorted by their distinctive poly A tails, but not all mRNAs have such tails. There is more to RNA than just mRNA, rRNA, and tRNA. Small nuclear RNAs (snRNAs)<sup>15,16</sup> may prove to be the most exciting molecular regulators during development.

Less than 3000 different mRNAs define fertile sperm, according to Ostermeier and colleagues, and these mRNAs may become invaluable for: new diagnostics for idiopathic infertility; discovering paternal influences to both the fetus and the placenta; ascertaining if there are generational consequences of environmental exposures of boys and men; new strategies for male contraception; and even potentially new ARTs (eg, specific mRNA supplementation during ICSI of mRNA-impaired sperm). Are sperm mRNAs remnants of their past lives during spermatogenesis, especially spermiogenesis, or vital packets essential to energise embryos? Such information is especially important for its prognostic value when evaluating each sperm's reproductive potential. Although mRNA detection is non-invasive for the man, it destroys the sperm, so population analysis (not individual detection) will be required.

Gerald P Schatten

Departments of Obstetrics, Gynecology and Reproductive Sciences, and Cell Biology and Physiology, Pittsburgh Development Center of the Magee-Womens Research Institute, Pittsburgh, PA 15213, USA (e-mail: pdcgs@mail.magee.edu)

- 1 Jouannet P, Wang C, Eustache F, Kold-Jensen T, Auger J. Semen quality and male reproductive health: the controversy about human sperm concentration decline. *APMIS* 2001; **109**: 333–44.
- 2 Wong WY, Thomas CM, Merkus JM, Zielhuis GA, Steegers-Theunissen RP. Male factor subfertility: possible causes and the impact of nutritional factors. *Fertil Steril* 2000; **73**: 435–42.
- 3 Schieve LA, Jeng G, Wilson LS. Use of assisted reproductive technology—United States, 1996 and 1998. *MMWR Morb Mortal Wkly Rep* 2002; **51**: 97–101.
- 4 Palermo G, Joris H, Devroey P, Van Steirteghem AC. Pregnancies after intracytoplasmic injection of single spermatozoon into an oocyte. *Lancet* 1992; **340**: 17–18.
- 5 Silber SJ, Johnson L, Verheyen G, Van Steirteghem A. Round spermatid injection. *Fertil Steril* 2000; **73**: 897–900.
- 6 Wilmut I, Schnieke AE, McWhir J, Kind AJ, Campbell KH. Viable offspring derived from fetal and adult mammalian cells. *Nature* 1997; **385**: 810–13.
- 7 Surani MA. Reprogramming of genome function through epigenetic inheritance. *Nature* 2001; **414**: 122–08.
- 8 Saunders CM, Larman MG, Parrington J, et al. PLC zeta: a sperm-specific trigger of Ca(2+) oscillations in eggs and embryo development. *Development* 2002; **129**: 3533–44.
- 9 Whitaker M, Swann K. Lighting the fuse at fertilization. *Development* 1993; **117**: 1–12.
- 10 Runft LL, Jaffe LA, Mehlmann LM. Egg activation at fertilization: where it all begins. *Dev Biol* 2002; **245**: 237–54.
- 11 Schatten G. The centrosome and its mode of inheritance: the reduction of the centrosome during gametogenesis and its restoration during fertilization. *Dev Biol* 1994; **165**: 299–335.
- 12 Simerly C, Zoran SS, Payne C, et al. Biparental inheritance of gamma-tubulin during human fertilization. *Molec Biol Cell* 1999; **10**: 2955–69.
- 13 Hewitson L, Dominko T, Takahashi D, et al. Unique checkpoints during the first cell cycle of fertilization after intracytoplasmic sperm injection in rhesus monkeys. *Nat Med* 1999; **5**: 431–33.
- 14 Sutovsky P, Moreno RD, Ramalho-Santos J, Dominko T, Simerly C, Schatten G. Ubiquitin tag for sperm mitochondria. *Nature* 1999; **402**: 371–72.
- 15 Prather R, Simerly C, Schatten G, et al. U3 snRNPs and nucleolar development during oocyte maturation, fertilization and early embryogenesis in the mouse. *Dev Biol* 1990; **138**: 247–55.
- 16 Sempere LF, Dubrovsky EB, Dubrovskaya VA, Berger EM, Ambros V. The expression of the let-7 small regulatory RNA is controlled by ecdysone during metamorphosis in *Drosophila melanogaster*. *Dev Biol* 2002; **244**: 170–79.

# Single session debriefing after psychological trauma: a meta-analysis

Arnold A P van Emmerik, Jan H Kamphuis, Alexander M Hulsbosch, Paul M G Emmelkamp

## Summary

**Background** Despite conflicting research findings and uncertain efficacy, single session debriefing is standard clinical practice after traumatic events. We aimed to assess the efficacy of this intervention in prevention of chronic symptoms of post-traumatic stress disorder and other disorders after trauma.

**Methods** In a meta-analysis, we selected appropriate studies from databases (Medline Advanced, PsychINFO, and PubMed), the *Journal of Traumatic Stress*, and reference lists of articles and book chapters. Inclusion criteria were that single session debriefing had been done within 1 month after trauma, symptoms were assessed with widely accepted clinical outcome measures, and data from psychological assessments that had been done before (pretest data) and after (post-test data) interventions and were essential for calculation of effect sizes had been reported. We included seven studies in final analyses, in which there were five critical incident stress debriefing (CISD) interventions, three non-CISD interventions, and six no-intervention controls.

**Findings** Non-CISD interventions and no intervention improved symptoms of post-traumatic stress disorder, but CISD did not improve symptoms (weighted mean effect sizes 0.65 [95% CI 0.14–1.16], 0.47 [0.28–0.66], and 0.13 [–0.29 to 0.55], respectively). CISD did not improve natural recovery from other trauma-related disorders (0.12 [–0.22 to 0.47]).

**Interpretation** CISD and non-CISD interventions do not improve natural recovery from psychological trauma.

*Lancet* 2002; **360**: 766–71

See Commentary page 741

## Introduction

After traumatic events such as the Sept 11 attacks, offers of emotional and practical support to victims are thought to be appropriate and caring human responses. Psychological debriefing is a formal type of post-traumatic care, for which several models have been developed in the past two decades. Everly and colleagues<sup>1</sup> describe three stages in the development of these models. The earliest forms of debriefing included many individually applied techniques, termed “crisis intervention approaches”.<sup>1</sup> “Group psychological debriefing”<sup>2</sup> has been used to reduce immediate distress, prevent later adverse psychological sequelae including post-traumatic stress disorder,<sup>3</sup> and identify individuals who were at risk of development of chronic problems and who needed referral for further treatment. There are three types of group psychological debriefing: critical incident stress debriefing (CISD) also known as the Mitchell model,<sup>4</sup> the Raphael model,<sup>5</sup> and process debriefing.<sup>6</sup>

In typical CISD, within 1 week of a traumatic event, a group of victims are led through seven stages in a single 1–3 h session. Process debriefing and the Raphael model are variations on the CISD model, differing in their emphasis on structure and in certain aspects of content.<sup>2</sup> CISD was integrated in the more comprehensive critical incident stress management model (CISM).

Psychological debriefing has received increasing attention from the scientific community. A search of the PsychINFO-database for English language journal articles with the word “debriefing” in the title identified 206 hits for the 1990s, compared with 79, 47, and 11 hits in the 1980s, 1970s, and 1960s, respectively. Many interventions are offered as treatments and described as debriefing, including CISD or CISD-like interventions, interventions that share only some elements with CISD, and interventions that have very little to do with CISD in its original form. Furthermore, these interventions are delivered by professional and non-professional workers with different backgrounds, at different time-intervals (sometimes months after a traumatic event), and are assessed with different instruments.

Despite the large number of research publications on this issue, debate continues on the efficacy of single session debriefing in prevention of symptoms of chronic post-traumatic stress disorder and other negative psychological outcomes after trauma. Several narrative reviews have been published on single session debriefing.<sup>1,2,7</sup> Conclusions varied from “there is no current evidence that psychological debriefing is a useful treatment for the prevention of PTSD [post-traumatic stress disorder] after traumatic incidents”<sup>7</sup> to “crisis intervention procedures, group debriefings, and especially CISM approaches are effective in reducing the negative psychological aftermath of a variety of critical incidents”.<sup>1</sup> Thus, there is still no consensus on whether single session debriefing can contribute to the prevention of symptoms of chronic post-traumatic stress disorder.

**Department of Clinical Psychology, University of Amsterdam, Amsterdam, Netherlands** (A A P van Emmerik MA, J H Kamphuis PhD, A M Hulsbosch MA, P M G Emmelkamp PhD)

**Correspondence to:** Arnold A P van Emmerik, Department of Clinical Psychology, Roetersstraat 15, 1018 WB Amsterdam, Netherlands (e-mail: kp\_emmerik@macmail.psy.uva.nl)

Narrative reviews of research have several limitations;<sup>8</sup> meta-analysis is a useful alternative. However, an earlier meta-analysis on the efficacy of psychological debriefing also had several limitations.<sup>9</sup> First, only studies of group psychological debriefing were included, although in clinical practice individual debriefing is the rule rather than the exception. Thus, the conclusions drawn by the authors cannot be generally applied to clinical practice. Second, in two studies, psychological debriefing was done at 6 and 9 months after trauma, and at 6 months after trauma, respectively, thus, these interventions could hardly have been preventive.

We have done a meta-analysis of studies designed to assess the efficacy of single session debriefing in preventing post-traumatic stress disorder and non-post-traumatic stress disorder psychopathology. We included studies of group and individual debriefing interventions that had been administered within 1 month of a traumatic event. The fourth edition of the *Diagnostic and Statistical Manual of Psychiatric Disorders (DSM-IV)*<sup>3</sup> states that to meet criteria for a diagnosis of post-traumatic stress disorder, symptoms have to persist for at least 1 month. Interventions done more than 1 month after trauma are therefore curative rather than preventive.

## Methods

### Procedures

We searched for studies on databases: Medline Advanced (1973–2000), PsychINFO (1967–2000), and PubMed (1970–2000). Keywords used were “posttraumatic”, “stress”, “debriefing”, “prevention”, and “intervention”, and names of authors working in debriefing. We also did a manual search of all volumes of the *Journal of Traumatic Stress*. We searched reference lists of articles and book

chapters identified by the searches for other relevant studies.

Inclusion criteria were that single session debriefing been done within 1 month after a traumatic event, psychological symptoms had been assessed with widely accepted clinical outcome measures, and data from psychological assessments that had been done before (pretest data) and after (post-test data) interventions and were essential for calculation of effect sizes had been reported for at least one outcome measure. Since we were not only interested in the effect of single session debriefing on symptoms of post-traumatic stress disorder, but also in the effects on general psychopathology, we included studies that contained reliable and valid psychological outcome measures for symptoms other than those of post-traumatic stress disorder. Outcome measures assessing symptoms of post-traumatic stress disorder included the impact of event scale,<sup>10</sup> clinician-administered post-traumatic stress disorder scale,<sup>11</sup> and post-traumatic stress disorder symptom scale.<sup>12</sup> Outcome measures assessing non-post-traumatic stress disorder symptoms included the hospital anxiety and depression scale,<sup>13</sup> brief symptom inventory,<sup>14</sup> and state-trait anxiety inventory.<sup>15</sup> Because of the ethical and practical difficulties in doing research after traumatic events and the resulting scarcity of such studies, we also included studies that fell marginally short of the highest methodological standards (eg, non-randomised allocation of participants to intervention and control groups). In studies that included more than one post-test assessment, data for the last measurement are presented.

We grouped interventions into CISD-type interventions and non-CISD interventions (30-min counselling, education, and historical group debriefing).

| Study                 | Intervention                | n* | Age (years, mean (SD)) | Sex male | Dropouts       | Measures                     | Type of trauma         | Intervention                        |                         |  |  |  |                        |
|-----------------------|-----------------------------|----|------------------------|----------|----------------|------------------------------|------------------------|-------------------------------------|-------------------------|--|--|--|------------------------|
|                       |                             |    |                        |          |                |                              |                        | Timing (first)                      | Length                  | Timing (final)   |  |  |                        |
| Bisson <sup>18</sup>  | CISD†                       | 57 | 37.9 (13.1)            | 74%      | 26%            | IES, HADS-A, HADS-D          | Burns                  | Mean 6.3 days (SD 3.6) after trauma | Mean 44.3 min (SD 17.4) | 13 months after trauma                                     |  |  |                        |
|                       | No-intervention control     | 46 | 36.7 (13.9)            | 76%      | 18%            |                              |                        |                                     |                         |  |  |  |                        |
| Carlier <sup>19</sup> | CISD                        | 86 | 28.9 (5.6)             | 70%      | NR             | STAI-S                       | Misc (police officers) | About 24 h after trauma             | Mean 41.4 min (SD 24.9) | 24 h after trauma (shortly after first debriefing session) |  |  |                        |
|                       | No-intervention control     | 82 | 31.7 (7.1)             | 65%      | NR             |                              |                        |                                     |                         |  |  |  |                        |
| Conlon <sup>20</sup>  | 30-min counselling          | 18 | 32.9 (10.8)            | 39%      | 0 (40 on CAPS) | IES, CAPS                    | Road traffic accident  | Mean 7 days after trauma            | About 30 min            | Mean 99 days after trauma                                  |  |  |                        |
|                       | No-intervention control     | 22 | 34.7 (13.2)            | 55%      | 0 (5 on CAPS)  |                              |                        |                                     |                         |  |  |  |                        |
| Mayou <sup>21</sup>   | CISD                        | 30 | 29 (NR)‡               | 57%‡     | 44%            | IES, BSI                     | Road traffic accident  | Within 24–48 h of the accident      | 1 h debriefing          | 36 months  |  |  |                        |
|                       | No-intervention control     | 31 | 26 (NR)‡               | 67%‡     | 40%            |                              |                        |                                     |                         |  |  |  |                        |
| Lee <sup>22</sup>     | CISD                        | 21 | NR                     | 0        | 7 (overall)    | IES-I, IES-A, HADS-A, HADS-D | Early miscarriage      | About 2 weeks after miscarriage     | 1 h session             | About 4 months after miscarriage                           |  |  |                        |
|                       | No-intervention control     | 18 | NR                     | 0        | ..             |                              |                        |                                     |                         |  |  |  |                        |
| Rose <sup>23</sup>    | CISD                        | 29 | 35.4 (13.8)            | 69%      | 46%            | IES, PSS                     | Violent crime          | Mean 21 days (SD 5.6) after trauma  | Debriefing about 1 h    | 11 months  |  |  |                        |
|                       | Education                   | 35 | 34.9 (13.2)            | 75%      | 33%            |                              |                        |                                     |                         |  |  |  |                        |
|                       | No-intervention control     | 28 | 37.3 (13.8)            | 82%      | 45%            |                              |                        |                                     |                         |  |  |  | Education about 30 min |
| Shalev <sup>24</sup>  | Historical group debriefing | 39 | 19.4 (1.8)             | NR       | 5              | STAI-S                       | Combat exposure        | Within 48–72 h of combat            | Mean 2.5 h (SD NR)      | Immediately after debriefing                               |  |  |                        |

NR=data not reported. IES(-I/-A)=impact of event scale (-intrusion/-avoidance). HADS(-A/-D)=hospital anxiety and depression scale (-anxiety/-depression). BSI=brief symptom inventory. CAPS=clinician-administered post-traumatic stress disorder scale. STAI-S=state-trait anxiety inventory-state version. PSS=post-traumatic stress disorder symptom scale. \*Number of participants who completed assessment. †CISD according to Mitchell's seven-stage model, or closely corresponding to CISD. ‡Reported at 4-months' follow-up, not available at 36-months' follow-up, but no significant differences between intervention and control groups at 36 months or between people who did and did not complete preintervention assessments.

Table 1: Description of studies included in the final sample

Measures used to assess symptoms were grouped into those used to assess symptoms of post-traumatic stress disorder and those used to assess other symptoms (mainly of general anxiety and depression).

#### Statistical analyses

Before calculation of mean weighted effect sizes and comparison of 95% CIs, we investigated whether effect sizes should be weighted on quality of the study and duration of intervention as well as on sample size. The analytic strategy was based on work by Van Etten and Taylor.<sup>16</sup> Within-study effect sizes refer to the magnitude of change assessed with continuous measures between preintervention and postintervention assessment results within each intervention and control group (ie, rather than differences in post-test results across interventions). Effect sizes were calculated for each measure using Cohen's *d* statistic,<sup>17</sup> with the magnitude of change defined as the difference between preintervention and postintervention assessment group means divided by the pooled SD. Positive effect sizes indicate reductions in symptom severity; negative effect sizes indicate worsening of symptoms. If a study included more than one assessment after the intervention, effect sizes were calculated from results of all assessments. Long-term outcome was considered to be most relevant to our study. Therefore, reported effect sizes are for means and SDs obtained from the last assessment. Since most studies reported data only for participants who had completed both preintervention and postintervention assessments, effect sizes were based on these participants rather than

on end-point or intent-to-treat analyses. If participants had completed more than one measure in a symptom group, effect sizes for these measures were averaged to obtain an aggregate effect size.<sup>16</sup>

Mean effect sizes were calculated across intervention types (CISD and non-CISD) and no-intervention control groups for both symptom groups. Since effect sizes of large studies are more likely to be reliable estimates of the efficacy of single session debriefing than those of small studies, sizes were weighted by the number of participants who completed assessments in each intervention group. 95% CIs were calculated for these weighted mean effect sizes to establish whether they were significant at  $p < 0.05$ . Weighted mean effect sizes without overlapping 95% CIs differ significantly at  $p < 0.05$ . Fail-safe *N* statistics were calculated<sup>16</sup> to investigate whether significant mean effect sizes might have been inflated by a publication bias—ie, a bias towards publication of studies reporting significant findings and large effect sizes.

#### Role of the funding source

The sponsors of the study had no role in study design, data collection, data analysis, data interpretation, or writing of the report.

## Results

We identified 29 relevant outcome studies. We excluded 22 studies in which the intervention consisted of more than one session (three studies), the interval between the traumatic event and intervention was more than 1 month or was unclear (six studies), the intervention was

| Study                 | Intervention                | Measures | Effect size | Aggregate effect size (95% CIs) |                        |
|-----------------------|-----------------------------|----------|-------------|---------------------------------|------------------------|
|                       |                             |          |             | PTSD symptoms                   | Other symptoms         |
| Bisson <sup>18</sup>  | CISD                        | IES      | -0.18       | -0.18 (-0.80 to 0.44)           | -0.13* (-0.29 to 0.03) |
|                       |                             | HADS-A   | -0.19       | ..                              | ..                     |
|                       |                             | HADS-D   | -0.07       | ..                              | ..                     |
|                       | No-intervention control     | IES      | 0.39        | 0.39 (-0.27 to 1.05)            | 0.24* (0.08 to 0.40)   |
|                       |                             | HADS-A   | 0.21        | ..                              | ..                     |
|                       |                             | HADS-D   | 0.27        | ..                              | ..                     |
| Carlier <sup>19</sup> | CISD                        | STAI-S   | 0.38        | N/O                             | 0.38 (0.18 to 0.58)    |
|                       | No-intervention control     | STAI-S   | 0.01        | N/O                             | 0.01 (-0.21 to 0.23)   |
| Conlon <sup>20</sup>  | 30-min counselling session  | IES      | 0.99        | 0.99 (-1.28 to 3.26)            | N/O                    |
|                       |                             | CAPS     | 1.36        | ..                              | ..                     |
|                       | No-intervention control     | IES      | 0.73        | 0.73 (-0.87 to 2.33)            | N/O                    |
|                       |                             | CAPS     | 0.50        | ..                              | ..                     |
| Mayou <sup>21</sup>   | CISD                        | IES      | -0.07       | -0.07 (-1.15 to 1.01)           | -0.31 (-0.35 to -0.27) |
|                       |                             | BSI      | -0.31       | ..                              | ..                     |
|                       | No-intervention control     | IES      | 0.19        | 0.19 (-0.77 to 1.15)            | 0.13 (0.11 to 0.15)    |
|                       |                             | BSI      | 0.13        | ..                              | ..                     |
| Lee <sup>22</sup>     | CISD                        | IES-†    | 0.63        | 0.62* (-0.50 to 1.74)           | 0.37* (-0.15 to 0.89)  |
|                       |                             | IES-A    | 0.61        | ..                              | ..                     |
|                       |                             | HADS-A   | 0.25        | ..                              | ..                     |
|                       |                             | HADS-D   | 0.48        | ..                              | ..                     |
|                       | No-intervention control     | IES-I    | 0.57        | 0.53* (-0.84 to 1.90)           | 0.37* (-0.32 to 1.06)  |
|                       |                             | IES-A    | 0.49        | ..                              | ..                     |
|                       |                             | HADS-A   | 0.28        | ..                              | ..                     |
|                       |                             | HADS-D   | 0.46        | ..                              | ..                     |
| Rose <sup>23</sup>    | CISD                        | IES      | 0.79        | 0.61* (-0.49 to 1.71)           | N/O                    |
|                       |                             | PSS      | 0.43        | ..                              | ..                     |
|                       | Education                   | IES      | 0.46        | 0.47* (-0.44 to 1.38)           | N/O                    |
|                       |                             | PSS      | 0.48        | ..                              | ..                     |
|                       | No-intervention control     | IES      | 0.80        | 0.66* (-0.53 to 1.85)           | N/O                    |
|                       |                             | PSS      | 0.50        | ..                              | ..                     |
| Shalev <sup>24</sup>  | Historical group debriefing | STAI-S   | 0.36        | N/O                             | 0.36 (0.12 to 0.60)    |

N/O=data not obtained. IES(-I/-A)=impact of event scale (-intrusion/-avoidance). HADS(-A/-D)=hospital anxiety and depression scale (-anxiety/-depression). BSI=brief symptom inventory. CAPS=clinician-administered post-traumatic stress disorder [PTSD] scale. STAI-S=state-trait anxiety inventory-state version. PSS=post-traumatic stress disorder symptom scale. \*Aggregate effect size across measures. †Scores reported for the IES subscales only; we combined these to an aggregate effect size, as with the other aggregate effect sizes. Hence IES total scores are not reported.

Table 2: Aggregate effect sizes and 95% CIs for type of intervention and symptom group

| Intervention            | n | PTSD symptoms<br>M (95% CIs) | Other symptoms<br>M (95% CIs) |
|-------------------------|---|------------------------------|-------------------------------|
| CISD                    | 5 | 0.13 (-0.29 to 0.55)         | 0.12 (-0.22 to 0.47)          |
| Non-CISD interventions  | 3 | 0.65 (0.14 to 1.16)          | 0.36 (-)*                     |
| No-intervention control | 6 | 0.47 (0.28 to 0.66)          | 0.13 (-0.02 to 0.28)          |

M=mean weighted effect sizes for the difference between results of preintervention and postintervention assessments. Since studies used different measures, mean weighted effect sizes were calculated from different numbers of separate effect sizes. \*95% CI could not be calculated because only one effect size was available.

Table 3: Effects of interventions on post-traumatic stress disorder (PTSD) and other symptoms

exclusively pharmacological (one study), no preintervention psychological assessment was done (9 studies), or the data needed to calculate effect sizes (eg, means and SDs) were not available (three studies); studies excluded for multiple reasons are counted under the main reason for exclusion. A list of the excluded studies is available from the authors. The final sample included seven studies (table 1)<sup>18-24</sup> with eight interventions and six control groups in which no intervention had been done (ie, control studies showed spontaneous recovery or assessment only). Five studies were randomised controlled trials, one was a non-randomised controlled trial,<sup>19</sup> and one did not include controls.<sup>24</sup> One study<sup>24</sup> was of group debriefing and six were of individual debriefing. Carlier and colleagues<sup>19</sup> did a three-session intervention, thus their study was initially excluded. However, as the first CISD-type session was done and followed up by a preliminary post-test within 1 month after the traumatic event, the preintervention assessment and first post-test results met our criteria for inclusion.

Weighting effect sizes by relevant aspects of research quality was considered; specifically the effect of design and duration of intervention. Study designs were very similar, whereas other quality indices were too scarce or unclear to calculate meaningful weights. Duration of intervention and effect sizes were not correlated. Accordingly, additional weighting of effect sizes was dismissed.

Table 2 shows aggregate effect sizes and 95% CIs. For the study by Conlon and colleagues,<sup>20</sup> an aggregate effect size across the results from the IES (impact of event scale) and CAPS (clinician-administered PTSD [post-traumatic stress disorder] scale) could not be calculated since different numbers of participants completed each measure. Since the CAPS was the only observer-rated measure with a 40% dropout rate in the intervention, its effect size was not included in further analyses.

Table 3 shows mean weighted effect sizes. One way to interpret effect sizes is to consider values of 0.2, 0.5, and 0.8 as corresponding to small, medium, and large effects, respectively.<sup>17</sup> In accordance with this rule, the no-intervention condition (controls) resulted in a medium reduction in the severity of symptoms of post-traumatic stress disorder and a small reduction in other symptoms. Non-CISD interventions resulted in a medium-to-large reduction in the severity of symptoms of post-traumatic stress disorder and a small-to-medium reduction in other symptoms (but only one effect size was available for other symptoms). CISD interventions resulted in a small reduction in severity of symptoms of post-traumatic stress disorder and other symptoms.

95% CIs show that the effect sizes for CISD were not significant (table 3). The 95% CI for the non-CISD interventions was positive (ie, symptoms improved) and

did not contain zero. The 95% CI for the no-intervention control category was greater than zero for symptoms of post-traumatic stress disorder, but contained zero for other symptoms. Comparisons between 95% CIs showed that they all overlapped—ie, there were no significant differences between effect sizes for CISD, non-CISD, and no intervention for both symptom groups. Fail-safe N statistics indicated that significant effect sizes were unlikely to have been inflated by a bias towards publication of studies reporting significant findings and large effect sizes.

## Discussion

Despite the intuitive appeal of the technique, our results show that CISD has no efficacy in reducing symptoms of post-traumatic stress disorder and other trauma-related symptoms, and in fact suggest that it has a detrimental effect. In both groups of symptoms, 95% CIs for CISD overlapped with those for non-CISD interventions and no intervention controls. Thus, CISD was no more effective than non-CISD interventions or even than not intervening at all. In fact, the mean weighted effect size for symptoms of post-traumatic stress disorder was lower for CISD than for non-CISD interventions and for not intervening.

In the other group of symptoms, mean weighted effect sizes for CISD and for no intervention were equal. Stated differently, effect sizes for CISD were not significant in either symptom group, whereas effect sizes for non-CISD interventions and for no intervention indicated improvement in symptoms of post-traumatic stress disorder. This finding suggests that CISD does not improve psychological outcome after traumatic events.

A more lenient analysis with 90% CIs did not change the pattern of results. At  $p < 0.10$ , mean weighted effect sizes for CISD were again not significant for either symptom group. Also, analysis of data obtained in the first rather than the last psychological assessment done after the intervention did not substantially change results. This result is not surprising since only three studies<sup>18,21,23</sup> included more than one such test and there was no significant correlation between effect size and duration of the interval between intervention and assessment. The only change in results was that the mean weighted effect size for symptoms of post-traumatic stress disorder was not significant for no intervention.

Most events in the studies included in our meta-analysis are major life events, and qualify as potential traumatic events. However, whether early miscarriages<sup>22</sup> are traumatic events is disputed, but exclusion of this study did not change the pattern of findings. In sum, the findings were robust even with varying statistical stringency, timing of assessment after the intervention, or stringency of the definition of trauma.

There are several explanations for the lack of efficacy of CISD. CISD might interfere with the alternation of intrusion and avoidance that characterises the natural processing of a traumatic event.<sup>25</sup> It might also interfere with natural processing in a broader sense—ie, inadvertently leading to victims bypassing the support of family, friends, or other sources of social support. CISD probably increases awareness of normal manifestations of distress after trauma. Although normalisation of these reactions is the aim of CISD, the suggestion that such reactions warrant professional care and must therefore be maladaptive might be an unintended result. Alternatively or additionally, exposure to trauma-related internal and external stimuli in CISD might not allow victims

adequate time for habituation, thereby further sensitising them to these stimuli.<sup>8</sup> This hypothesis can be tested by collecting data that reflect habituation (eg, by use of subjective units of disturbance),<sup>26</sup> but to our knowledge no such data exist.

A third explanation could be that if CISM is offered after trauma, both victims at risk and victims not at risk for chronic psychological symptoms can participate. This factor might obscure a true beneficial effect of CISM on the development of chronic symptoms for individuals at risk. Although increasing numbers of risk factors for chronic symptoms after trauma are being identified,<sup>27</sup> their clinical and practical use is untested. Studies should be done to assess whether targeting the CISM intervention to at-risk individuals is warranted. Finally, CISM was never designed to be a stand-alone intervention, but rather part of a broader, multi-component CISM-type intervention that included training in being prepared for a crisis, follow-up, and referral.<sup>9,19</sup> The efficacy of this type of intervention was not the subject of our meta-analysis, and we suggest that it be convincingly proven in empirical research before large-scale implementation.

A limitation of our study is that, similarly to the meta-analysis by Everly and colleagues,<sup>9</sup> our analysis includes only a small number of studies because of our exclusion criteria and because some studies did not report preintervention assessment data, rendering impossible calculation of within-effect sizes of change in symptom severity. However, we realise that preintervention assessment is difficult in the aftermath of trauma. A possible solution to the small number of studies would have been to widen our inclusion criteria to include studies in which more than one intervention session was done or in which interventions were done more than 1 month after trauma. However, we do not think that this solution would have been useful, since it would not have answered our original question about the efficacy of single session debriefing in preventing chronic symptoms. Furthermore, meta-analyses based on small numbers of studies are not unusual and do not preclude drawing meaningful conclusions.<sup>8</sup>

Adaptations to enhance single session debriefing have been suggested. Symptoms of post-traumatic stress disorder in victims of robbery markedly improved after immediate CISM (<10 h), whereas participants in delayed debriefing (>48 h) benefited only slightly.<sup>28</sup> Brief cognitive behavioural programmes have produced promising results.<sup>29,30</sup> These programmes typically consist of four to five weekly individual sessions, starting within the first month after the traumatic event, and homework assignments. Interventions include education, imaginary and real (but introduced on a graded scale) exposure to traumatic situations, and cognitive therapy.

Should single session debriefing be made available routinely after trauma? Prevention of later adverse psychological sequelae such as post-traumatic stress disorder is only one aim of psychological debriefing. Other aims include reduction of immediate distress and identification and referral for further treatment of individuals at risk for development of chronic problems.<sup>2</sup> The decision to provide debriefing is not necessarily based on findings from only empirical research. Reports of satisfaction<sup>17</sup> or perceived helpfulness by participants<sup>18,22</sup> might be sufficient reasons to continue to offer debriefing. However, claims that single session psychological debriefing can prevent development of chronic negative psychological sequelae are empirically unwarranted.

#### Contributors

P M G Emmelkamp, A A P van Emmerik, J H Kamphuis, and A M Hulsbosch conceived and designed the study. A M Hulsbosch and P M G Emmelkamp did the literature search and identified eligible studies. A A P van Emmerik, J H Kamphuis, and A M Hulsbosch coded articles and decided on their inclusion. J H Kamphuis and A A P van Emmerik did statistical analyses and interpreted results. All drafts of the report, including the final version, were written by A A P van Emmerik and J H Kamphuis and revised by P M G Emmelkamp and A M Hulsbosch.

#### Conflict of interest statement

None declared.

#### Acknowledgments

The report was written as part of a project funded by the Netherlands Organisation for Health Research and Development (ZON).

#### References

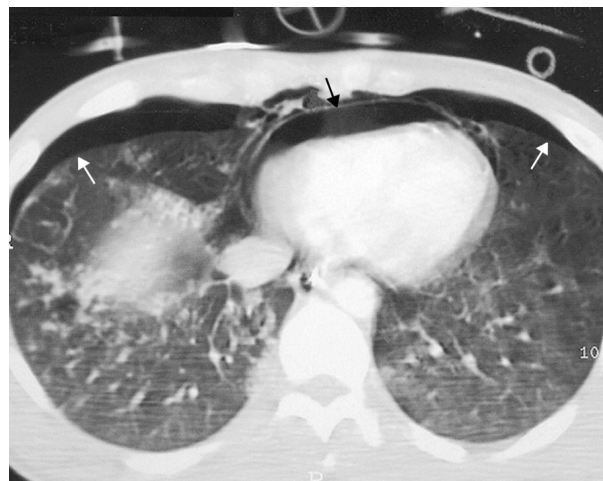
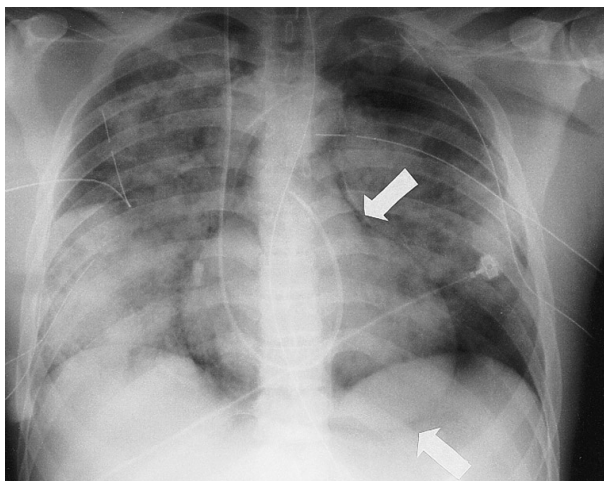
- 1 Everly GS, Flannery RB, Mitchell JT. Critical incident stress management (CISM): a review of the literature. *Aggression Violent Behav* 2000; **5**: 23–40.
- 2 Bisson JL, McFarlane AC, Rose S. Psychological debriefing. In: Foa EB, Keane TM, Friedman MJ, eds. *Effective treatments for PTSD*. New York: The Guilford Press; 2000: 39–59.
- 3 American Psychiatric Association. *Diagnostic and statistic manual of mental disorders: 4th edn (DSM-IV)*. Washington: American Psychiatric Association, 1994.
- 4 Mitchell JT. When disaster strikes . . . the critical incident stress debriefing. *J Emergency Med Serv* 1983; **8**: 36–39.
- 5 Raphael B. *When disaster strikes: a handbook for caring professions*. London: Hutchinson, 1986.
- 6 Dyregrov A. Caring for helpers in disaster situations: psychological debriefing. *Disaster Management* 1989; **2**: 25–30.
- 7 Rose S, Bisson J, Wessely S. Psychological debriefing for preventing post traumatic stress disorder (PTSD) (Cochrane Review). *The Cochrane Library*, Issue 2, 2002. Oxford: Update Software.
- 8 Kramer SH, Rosenthal R. Meta-analytic research synthesis. In: Bellack AS, Hersen M, series eds, Schooler NR, volume ed. *Comprehensive clinical psychology: vol 3—research and methods*, 1st edn. Oxford: Pergamon, 1998: 351–68.
- 9 Everly GS, Boyle SH, Lating JM. The effectiveness of psychological debriefing with vicarious trauma: a meta-analysis. *Stress Med* 1999; **15**: 229–33.
- 10 Horowitz M, Wilner N, Alvarez W. Impact of event scale: a measure of subjective stress. *Psychosom Med* 1979; **41**: 209–18.
- 11 Blake DD, Weathers FW, Nagy LN. A clinician rating scale for assessing current and lifetime PTSD: the CAPS-1. *Behav Therapist* 1990; **18**: 187–88.
- 12 Foa EB, Riggs DS, Dancu CV, Rothbaum BO. Reliability and validity of a brief instrument for assessing post-traumatic stress disorder. *J Trauma Stress* 1993; **6**: 459–73.
- 13 Zigmund AS, Snaith RP. The hospital anxiety and depression scale. *Acta Psychiatr Scand* 1983; **67**: 361–70.
- 14 Mayou RA. A British view of liaison psychiatry. *Gen Hosp Psychiatry* 1987; **9**: 18–24.
- 15 Spielberger CD, Gorsuch RL, Lushene RE. *STAI manual for the state-trait anxiety inventory*. Palo Alto: Consulting Psychologists Press, 1970.
- 16 Van Etten ML, Taylor S. Comparative efficacy of treatments for post-traumatic stress disorder: a meta-analysis. *Clin Psychol Psychot* 1998; **5**: 126–44.
- 17 Cohen J. *Statistical power analysis for the behavioral sciences*, 2nd edn. Hillsdale: Erlbaum, 1988.
- 18 Bisson JL, Jenkins PL, Alexander J, Bannister C. Randomized controlled trial of psychological debriefing for victims of acute burn trauma. *Br J Psychiatry* 1997; **171**: 78–81.
- 19 Carlier IVE, Voerman AE, Gersons BPR. The influence of occupational debriefing on post-traumatic stress symptomatology in traumatized police officers. *Br J Med Psychol* 2000; **73**: 87–98.
- 20 Conlon L, Fahy TJ, Conroy R. PTSD in ambulant RTA victims: a randomized controlled trial of debriefing. *J Psychosom Res* 1998; **46**: 37–44.
- 21 Mayou RA, Ehlers A, Hobbs M. Psychological debriefing for road traffic accident victims. *Br J Psychiatry* 2000; **176**: 589–93.
- 22 Lee C, Slade P, Lygo V. The influence of psychological debriefing on emotional adaptation in women following early miscarriage: a preliminary study. *Br J Med Psychol* 1996; **69**: 47–58.

- 23 Rose S, Brewin CR, Andrews B, Kirk M. A randomized controlled trial of individual psychological debriefing for victims of violent crime. *Psychol Med* 1999; **29**: 793–99.
- 24 Shalev AY, Peri T, Rogel-Fuchs Y, Ursano RJ, Marlowe D. Historical group debriefing after combat exposure. *Mil Med* 1998; **163**: 494–98.
- 25 Horowitz MJ. Stress response syndromes. New York: Aronson, 1976.
- 26 Wolpe J. The practice of behavior therapy. New York: Pergamon Press, 1973.
- 27 Brewin CR, Andrews B, Valentine JD. Meta-analysis of risk factors for posttraumatic stress disorder in trauma-exposed adults. *J Consult Clin Psychol* 2000; **68**: 748–66.
- 28 Campfield KM, Hills AM. Effect of timing of critical incident stress debriefing (CISD) on posttraumatic symptoms. *J Trauma Stress* 2001; **14**: 327–40.
- 29 Bryant RA, Sackville T, Dang ST, Moulds M, Guthrie R. Treating acute stress disorder: an evaluation of cognitive behavior therapy and supportive counseling techniques. *Am J Psychiatry* 1999; **156**: 1780–86.
- 30 Foa EB, Olasov Rothbaum B, Riggs DS, Murdock TB. Treatment of posttraumatic stress disorder in rape victims: a comparison between cognitive-behavioral procedures and counseling. *J Consult Clin Psychol* 1991; **59**: 715–23.

## Clinical picture

### Pneumopericardium

Isabelle Gerard, David Verhelst



A 27-year-old man was admitted to the hospital after a fall from approximately 10 m. He had multiple bone fractures, head trauma (Glasgow Coma Scale: 4/15), bilateral pulmonary contusions and pneumothoraces. We placed bilateral tube thoracostomies, and treated his other injuries. 1 day later, because of severe haemodynamic instability (hypotension and low cardiac output with high central venous pressure), we did transoesophageal echocardiography and found right ventricular compression

in the absence of a pericardial effusion. Repeat chest radiographs (figure, left) showed the existing bilateral lung contusions and a new lucent outline of the heart (arrows). Computed tomography of the chest confirmed the diagnosis of post-traumatic pneumopericardium (figure, right, black arrow), bilateral pneumothoraces (white arrows) and lung contusion. The pneumopericardium resolved after we repositioned the left-sided interthoracic tube.

Department of Intensive Care, Cliniques Universitaires Saint-Luc, Université Catholique de Louvain, 1200 Brussels, Belgium (I Gerard MD, D Verhelst MD)