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Acute stress disorder in victims after terror attacks in Mumbai, India

In November 2008, 164 people were killed and at least 308 were physically injured in terror attacks on Mumbai, India.¹ One of the common psychiatric disorders in victims of terror is acute stress disorder. Out of 74 victims admitted to a public hospital, 70 were assessed by a senior psychiatrist (V.P.B.) for the presence of acute stress disorder in the week following hospitalisation. Four patients who were too severely injured were excluded. Victims were directly brought to the hospital because of its proximity to the terror sites or were transferred from other hospitals owing to space, facility and staff (medical/non-medical) constraints.

After obtaining informed consent, patients were individually interviewed and their demographic data (gender, age, address, socioeconomic status (as per B.G. Prasad classification),² religion, education, marital status and occupation), and details of the injuries sustained (initial gravity score)³ were recorded. Patients were specifically evaluated for the presence of acute stress disorder using DSM–IV–TR criteria.⁴ Details of past psychiatric history and family history of psychiatric disorders were also collected. The collected data were then tabulated and analysed using the chi-squared test.

The mean (s.d.) age of the victims was 33.5 (12.95) years. There were 52 males and 18 females. Acute stress disorder was found in 21 (30%) of the 70 victims assessed. Other similar studies on victims of terror attacks have found a prevalence of acute stress disorder varying from 12.5 to 47%.^{5–7} According to Bryant,⁵ human-caused trauma has higher rates of acute stress disorder. According to Stern⁸ and Janoff-Bulman,⁹ this is because the usually indiscriminate and random nature of terrorist attacks create extreme anxiety and helplessness, and destroy individuals' beliefs in their own invulnerability and in the justness of the world.

There were some interesting observations and differences between the patients with and without acute stress disorder on various demographic and clinical variables, although none of the differences reached the level of statistical significance. Acute stress disorder was more common in: females (female, 44.4% v. male, 25.0%); younger victims (<33.5 years, 34.9% v. >33.5 years, 22.2%); victims who were following the Muslim religion (Muslim, 33.3% v. Hindus, 29.6%); residents of Mumbai (residents, 36.6% v. immigrants, 20.7%); divorcees and single victims (divorcees and single, 50.0% and 46.7% v. married and widows, 25.5% and 0%); unemployed (unemployed, 37.5% v. employed, 28.0%); those of low socioeconomic status (low socioeconomic status, 31.7% v. middle socioeconomic status, 20.0%); patients with more than 6.5 years of education (>6.5 years, 39.1% v. ≤6.5 years, 25.5%); and those with severe injury (severe injury, 31.0% v. moderate injury, 25.0%). None of the victims had any past history or family history of any psychiatric disorders.

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Interpretation of screening implementation studies

Baas *et al*¹ report some very valuable findings based on a screening implementation study in Dutch general practice. In particular, they document that converting detections into treatment success is difficult in clinical practice and that many individuals with depression are unable or unwilling to accept help. However, I must disagree with their interpretation that it is necessary to screen 118 (17 of 2005) 'high-risk' people to treat one new case.

Let me illustrate this with an analogy of a drug trial for drug X. Let's say that I conduct a trial of drug X in primary care among 2005 individuals. Of 2005 approached, 780 consent to take X and of these, 226 have an initial response. The main question I would be asked is how many of the 780 actually had depression? I don't have this figure but I can say that of the 226 responders, 173 were given a Structured Clinical Interview for DSM–IV Axis I disorders (SCID) and of these 71 have depression. Further, unknown to me, 36 of the 71 were already receiving treatment (even though the protocol asked general practitioners to exclude those people with depression already known to them) and ultimately only 17 accepted treatment. Can I conclude from my trial of X that it is not a successful drug because only 17 were newly treated? No. I have demonstrated the difficulty of conducting a pragmatic trial in primary care, but I don't really know the success of X and I don't have any comparative placebo (treatment-as-usual) arm. What does this mean for the interpretation of the paper from Baas *et al*? From the authors' data the most critical step for useful interpretation of screening yield is revealed from those who have (a) the screen and (b) the criterion reference (gold standard, i.e. SCID). Thus I suggest that:

- (a) the number of detected cases per screen (who had a criterion diagnosis) = 71/173 (41%);
- (b) the number of newly treated cases per screen (who had a criterion diagnosis) = 35/173 (20%);
- (c) the number of helped cases per screen (who had a criterion diagnosis) = 17/173 (10%).