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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 Stern[8] 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%).

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There may be many more people with depression (with high or low Patient Health Questionnaire (PHQ) scores) who were unidentified because a SCID was not applied to the 780 and the screen depends wholly on the PHQ–9 on a single application. At a typical (medium-risk) prevalence rate of 20% there would be around 156 cases of depression in the group of 780, but in a high-risk group where the prevalence may be 35% this would mean around 273 true cases. Just relying on a single application of the PHQ–9 on its own (either by algorithm or recommended cut-off) is probably insufficient. Assuming (like the authors) that the sensitivity of the PHQ–9 is a generous 0.88,[2] then there might be 33 missed cases in a high-risk sample. However, a meta-analysis from Wittkampf *et al*[3] found a pooled sensitivity of 0.77 and Gilbody *et al*[4] found 0.81 (both in primary care), which would translate into 52–63 missed cases. Of course there is offset by the issue of false positives which should also be examined in a screening implementation study. However, this remains a speculation without the SCID data from the parent 780 sample which is not reported (but perhaps available to the authors?).

In summary, I suggest this is a genuinely useful paper about the hazards of screening implementation but it is not really about screening success, for which a screening randomised controlled trial or pre–post screen design is needed. A simple guide to interpretation of screening studies can be downloaded from www.psycho-oncology.info/education.htm.

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Author's reply: Dr Mitchell raises two interesting issues: (1) the way we calculated results of the screening procedure, in particular the number needed to screen to treat one additional patient; and (2) the PHQ–9 as a screening instrument.

As Dr Mitchell mentions, our study was indeed a screening implementation study. We wanted to learn whether screening in high-risk groups could detect a substantial number of so far not detected and treatable patients with depression. For this we conducted a pragmatic study and determined the gain of a stepped care screening (and treatment) programme in real practice, with real doctors and real patients. A GP who wants to screen his patients can read what to expect. Failures (refusals, no-shows, misclassifications) are inherent to such conditions and should be incorporated in the calculations.

We defined our target population and included patients ($n = 2005$) from the GPs' medical files and surgery. Our screening cascade showed 17 new patients that could be treated for depression. Perhaps this calculation is a bit optimistic because treatment was directly available without costs, which is not always the case.

Dr Mitchell makes a comparison with a drug trial. Unfortunately, we do not think this comparison makes the interpretation of our data easier. We consider as our screen the PHQ and use the SCID as the reference diagnostic standard. So the 780 patients who returned the PHQ and gave informed consent form the screened population. From there we count downwards to the number of detected cases and upwards to the number needed to be invited for the screening to be able to screen those 780 patients.

There can be discussion about the way we corrected for patients that did not adhere to the programme. We presented each step (with number of people who refused, did not attend and the reasons therefore), so that readers can make their own judgements, as Dr Mitchell has done. However, we disagree with his interpretation. If we use his analogy of a drug trial, then an intention-to-treat analysis is the best analysis. That means that non-adherence should be incorporated in the number needed to treat (or screen). Starting the analysis with the number of patients that completed the SCID (as Dr Mitchell does) provides the GPs with the number of patients they have to see, after a pre-screen with the PHQ–9.

It is correct that the PHQ–9 misses some cases, although not as many as Dr Mitchell supposes. We used the PHQ–9 in the screening mode (a cut off score of 10) and not in the diagnostic mode (algorithm). Sensitivity of the screening mode is 0.93, not 0.77.[1] However, a GP who uses the PHQ–9 will follow the results of the screening and invite patients with a positive score for clinical evaluation, thus also missing patients with a score below threshold. We unfortunately do not have SCID data of those who scored negative on the PHQ.

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