

Stillbirth – psychological impact on fathers

Although there is a lot of information on the psychological impact of stillbirth on mothers, data on the effect on fathers is very rare. We often fail to acknowledge that fathers can have a difficult time after a stillbirth in separating their own grief from that of the mother. Their role in supporting the mother through this ordeal cannot be overemphasised and the recent article by Turton *et al* (2006) is important because it describes the psychological stress and needs of fathers during subsequent pregnancy and the puerperium. However, I would like to raise a few points which need further discussion.

Social support from family or partner following such a life event can have a substantial impact on subsequent mental and physical well-being, which may also determine the subsequent level of coping. Turton *et al* measured support from partner and family as a dichotomous (yes/no) variable, which does not seem entirely appropriate. Social support is a multidimensional construct and should have been analysed in terms of quantity and quality. Various questionnaires such as the Norbeck Social Support Questionnaire; <http://nurseweb.ucsf.edu/www/NSSQ-Instrument.pdf> are available to evaluate social support in a holistic and objective manner. Second, Turton *et al*, relaxed the inclusion criteria by including four couples after the safe arrival of their babies. This might have skewed the final result.

Interestingly, the fact that fathers often refused to take part in the interview could have led to underestimation of the psychological impact of stillbirth and the underlying psychiatric morbidity. It would have been informative if the authors had identified the reasons for their refusal. This is particularly important since it is well accepted that fathers generally tend to minimise their problems, put on a 'brave face' and refuse to speak out. There is no mention of the reliability or validity of the scale used for the assessment of marital satisfaction. Moreover, exclusion of Black participants and those from minority ethnic groups limits the application of the results to a wider general population.

However, I think this is a relevant and significant study which may prove to be beneficial for a wider understanding of this poorly recognised problem. It highlights the importance of actively encouraging fathers

to be more forthcoming about their problems and also helps health professionals to focus on high-risk couples.

Turton, P., Badenhorst, W., Hughes, P., et al (2006)

Psychological impact of stillbirth on fathers in the subsequent pregnancy and puerperium. *British Journal of Psychiatry*, **188**, 165–172.

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Authors' reply: We would like to expand on the points raised relating to sampling and assessment tools. We accept the criticism that relaxing the inclusion criteria might have skewed the final results, but have already explained our rationale for this decision. Unfortunately it is not possible to make any inference about the psychological morbidity of non-participants. Non-responding fathers fell into two groups: those who were persistently unavailable and those who declined to take part. Only one father gave a reason for his refusal: rejection of what he perceived as a false assumption that it was possible for a parent to 'recover' from a stillbirth. Although we were active in seeking fathers' participation, ethical considerations did not permit us to persist in questioning fathers who declined to take part. Black couples and those from minority ethnic groups were not excluded from the study; rather they were underrepresented as a result of higher non-participation rates.

Two factors contributed to our use of a single dichotomous variable for the presence or absence of appropriate social support. First, social support at the time of loss was not a primary focus of the study and we felt it appropriate to limit the number of questionnaires that participants had to complete. Second, research in this field has relied on a range of assessment tools (e.g. Zeanah *et al*, 1995; Lin & Lasker, 1996). The use of multiple complex tools limits the comparison of data across studies. However, we accept the view that elaborating on the quality of support would deepen the findings. The Golombok Rust Inventory of Marital State, which was used to assess marital satisfaction, is a short and easy-to-administer assessment which has high face and content validity and good reliability (Rust *et al*, 1988).

We hope that continuing research in this field will lead to greater awareness of the needs of parents experiencing stillbirth.

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Promotion of psychiatric drugs

Dr Moncrieff (2006) attacks the pharmaceutical industry for promoting the idea that depression is 'caused by imbalances in brain chemicals' and berates it for the fact that people are taking more prescription drugs than ever before. She implies that because the biochemical basis of depression is not known, the promotion of antidepressants is a plot to encourage profiteering.

The history of medicine teaches us that many crucial and life-saving drugs were and still are used despite a lack of knowledge of their scientific action (e.g. the use of steroids in asthma). In fact, there are very few instances where the scientific basis of action of crucial medicines is fully understood. The history of psychiatry teaches us that before antidepressant medication there was no treatment for depression except waiting for natural recovery: frequently a long and painful process during which the patient often starved to death or ended life by suicide.

One can easily take, like Moncrieff, an extreme view of the pharmaceutical industry, emphasising how it controls research and uses advertising to influence clinicians, and I note that the current issue of the *Journal* has no fewer than 12 full-page colour advertisements promoting psychotropic medication. The alternative view, however, would be that the industry has helped us to move out of the dark ages when all we could offer was asylum and

restraint. The reality is probably somewhere between these extremes.

As a general psychiatrist with a special interest in psychological treatments (especially cognitive-behavioural therapy) I am not proposing that medication has all the answers or is even the preferred choice in all cases. However, I have to persuade many patients on a regular basis to take antidepressant medication before improvement can occur. The 'chemical imbalance theory' is a useful working hypothesis for one cause for depression. There is a current climate of opinion among those who regularly surf the internet that medication is all bad, dangerous and addictive. Clinical psychiatrists like me have an uphill battle to persuade patients to take life-saving medication which articles such as those by Moncrieff, and the websites she directs us to, make even harder.

Moncrieff, J. (2006) Psychiatric drug promotion and the politics of neoliberalism. *British Journal of Psychiatry*, **188**, 301–302.

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Author's reply: By mentioning the use of steroids in asthma, Dr Stern highlights an important contrast between our understanding of how drugs work in general medicine and how drugs work in psychiatric conditions. In general medicine the effects of drugs can usually be understood by their actions on some level of the pathological process that generates the symptoms. Thus, steroids reduce the inflammatory response that gives rise to some of the symptoms of asthma. In contrast there is no evidence that drugs used in psychiatric conditions act on specific neuropathological processes. No specific physical pathology has been established for any major psychiatric condition and other evidence that drugs might be specific is lacking. Instead I have suggested elsewhere the alternative hypothesis that psychiatric drugs do not correct pathological brain states or chemical imbalances but create them (Moncrieff & Cohen, 2005, 2006). These drug-induced states might sometimes prove useful in psychiatric conditions, but the negative aspects of such states are often likely to outweigh the benefits that can be gained. However, drug companies and the psychiatric profession have presented psychiatric drugs as

disease-specific treatments that correct chemical imbalances. This view helps to downplay the disadvantages of long-term drug use and may help to create the context for the expansion of markets for psychiatric drugs.

As far as antidepressants are concerned, there is little evidence that they have specific antidepressant effects (Moncrieff & Cohen, 2006) or that they are 'life-saving' in terms of reducing suicide (Moncrieff & Kirsch, 2005). There is no evidence that there is a chemical imbalance in people with depression, and I do not understand how we can be justified in persuading patients to see their problems in this way. Doing so runs the risk of undermining patients' own coping mechanisms and thereby increasing chronicity, dependence on services and use of prescribed drugs.

Declaration of interest

I am co-chairperson of the Critical Psychiatry Network.

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Moncrieff, J. & Cohen, D. (2006) Do antidepressants cure or create abnormal brain states? *PLoS Medicine*, **3**, e150.

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Initial rate of improvement in major depression

Dr Mitchell (2006) suggests that it may be pertinent to re-examine another commonly quoted recommendation – that an antidepressant trial must be at least 6 to 8 weeks before switching drugs. The evidence on which switch guidelines are based is weak but these guidelines are applied frequently in daily clinical practice. In previous studies symptom improvement at earlier time points in relation to *response* has been investigated (e.g. Koran *et al*, 1995) but the ultimate goal of depression treatment is complete *remission*. Remission takes longer than 4–6 weeks to achieve but substantial improvement is unlikely after 10–12 weeks

(Trivedi *et al*, 2006). Quitkin *et al* (2003) investigated the relationship between initial change in symptoms and remission by week 12 and demonstrated that even when there was no improvement after 6 weeks of treatment, an antidepressant trial should be continued because the proportion of patients attaining remission by week 12 was still considerable (i.e. greater than 30%). They argued that a switch of antidepressant medication would be unlikely to have resulted in higher remission rates. Furthermore, large studies are required in which change in symptoms is frequently measured at uniform time-points and dimensions other than those measured by conventional questionnaires for depression are assessed. These might be more sensitive to early change following the initiation of antidepressant treatment (Harmer *et al*, 2004), and therefore might better predict which patients will attain remission. Calculation of the sensitivity, specificity, area under the receiver operating characteristic curve, and positive and negative predictive power to assess the likelihood of remission for various levels of symptom change at different time-points would help clinicians to decide on clinical applicability. Results from such studies will improve the evidence on which switch guidelines are based.

Harmer, C. J., Shelley, N. C., Cowen, P. J., et al (2004) Increased positive versus negative perception and memory in healthy volunteers following selective serotonin and norepinephrine reuptake inhibition. *American Journal of Psychiatry*, **161**, 1256–1263.

Koran, L. M., Hamilton, S. H., Hertzmar, M., et al (1995) Predicting response to fluoxetine in geriatric patients with major depression. *Journal of Clinical Psychopharmacology*, **15**, 421–427.

Mitchell, A. J. (2006) Two-week delay in onset of action of antidepressants: new evidence. *British Journal of Psychiatry*, **188**, 105–106.

Quitkin, F. M., Petkova, E., McGrath, P. J., et al (2003) When should a trial of fluoxetine for major depression be declared failed? *American Journal of Psychiatry*, **160**, 734–740.

Trivedi, M. H., Rush, J., Wisniewski, S. R., et al (2006) Evaluation of outcomes with citalopram for depression using measurement-based care in STAR*D: implications for clinical practice. *American Journal of Psychiatry*, **163**, 28–40.

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Author's reply: I agree that the evidence base for strategies for treatment-resistant