Correspondence

Edited by Kiriakos Xenitidis and Colin Campbell

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High-risk strategies v. universal precautions against suicide

The recent paper by Gunnell et al1 and the accompanying editorial by Pitman & Caine2 clearly outline the practice and principles of a contemporary approach to suicide prevention in mental health settings. However, I do not think the policy initiative that every patient with a serious mental illness or a recent episode of self-harm should be followed up within a week of discharge is really a high-risk approach to suicide prevention. Patients who self-harm and those with serious mental illness must constitute the vast majority of people who are admitted to psychiatric hospitals and therefore this recommendation is more like a universal precaution against suicide than a targeted intervention based on a high-risk model.

In my view there are compelling reasons to doubt the usefulness of high-risk categorisation for future suicide at the point of discharge from psychiatric hospitals. It is known that discharged patients have about a 100-fold increased risk of suicide compared with the general community in their first few weeks at home.3 However, those categorised as at high risk of suicide after discharge are only about four times more likely to take their own life than discharged patients categorised as at low risk of suicide.4 Hence, compared with the risk of just being a discharged patient, being at high risk or low risk is virtually meaningless.

If the English guideline for early follow-up of patients has been successful, this is almost certainly because it approximates a universal precaution against suicide and not because of the success of a high-risk approach. We need to acknowledge that all those admitted to psychiatric hospitals have a very high absolute risk of suicide and that we are unable to tell who will be safe.

Authors’ reply: Large highlights two issues in relation to suicide prevention: (a) the differing terminology used internationally in relation to models of suicide prevention; and (b) the difficulties inherent in assessing suicide risk following discharge from psychiatric hospital.

Whereas in the UK the terms high-risk (or targeted) approach and population (or mass) approach are used commonly,1 terminology in the USA and elsewhere differs, referring to universal, selective and indicated interventions.2 A universal intervention corresponds to the population approach, in that it is applied to a broad population irrespective of the risk of individual members, in order to change norms and values, to influence unidentified members of the population who may carry more risk and, ultimately, to shift the risk of the entire population. At the other end of the spectrum, an indicated intervention corresponds to a high-risk approach, in that it is applied to identified symptomatic individuals. It is much the same as a clinical intervention except that public health approaches proactively reach into communities and diverse settings to engage such persons, whether or not they present in clinical settings.

Selective interventions equate to a form of high-risk approach, but one which addresses groups with a significantly higher-than-average risk of developing mental disorders or adverse outcomes.2 Such groups are described in the 2012 suicide prevention strategy for England as those ‘with particular vulnerabilities or problems with access to services’ (p. 21).3 The groups listed include children and young people; people with a history of childhood abuse; minority ethnic groups and asylum seekers; and people with untreated depression. These are distinguished from groups regarded as high risk for completed suicide on the basis of clear epidemiological evidence, which in the English strategy include people under the care of mental health services; people with a history of self-harm; people in contact with the criminal justice system; adult men under 50; and specific occupational groups. Whereas effectiveness studies tend to concentrate on proximal interventions for these highest-risk groups, less evidence describes the effectiveness of selective interventions, but this situation is likely to evolve.

In relation to the second issue that Large raises, also highlighted in his recent letter to The Psychiatrist,4 it would be fair to say that anyone admitted to hospital for a major mental disorder, or a substance use disorder, has a greater degree of risk for suicide than non-hospitalised individuals with mental disorders or the general population. However, people in contact with mental health services in the year prior to death account for 27% of general population suicides in England.5 Gunnell et al’s study6 found that 10% of all suicides in England occurred within the year following psychiatric discharge. Applying the term ‘high risk’ to this group of patients describes their overall risk in relation to the general population, ignoring the wide degree of variation in risk between individuals within this group. One could argue that integrated aftercare constitutes high-quality care for all but, on the basis of the above taxonomies, we would not regard this as universal because it is indicated for all such discharged patients.

References


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Assessing the role of cerebrovascular disease in the incidence of geriatric depression

The association between vascular disease and late-life depression is an example of how a common ‘medical’ disorder could have clinically significant neuropsychiatric sequelae. With cerebrovascular disease, a prominent cause of mortality and disability in the aged population, this is a major public health issue. In their longitudinal study of white matter changes and depression incidence, Firbank et al note that the cross-sectional nature of existing work prohibits conclusions about the direction of causality, with their prospective investigation a welcome contribution to this exciting field. A temporal sequence of white matter disease before depression supports the use of neuroimaging in screening ‘at risk’ individuals and implicates cardiovascular risk factors in the pathogenesis of geriatric mood disorder. However, other recent studies have suggested that this sequence could be bidirectional. As I argue elsewhere, the relationship between physical disease and mood disorder in the elderly is likely to be aetologically complex and characterised by reciprocity.

Firbank et al present results from the LADIS study and conclude that in their patients, progression of white matter disease was associated with depression incidence. However, I believe the analytical methods used by the authors affect the significance of this finding and warrant discussion.

A cohort study of harm typically compares individuals exposed to a risk factor (white matter changes) with those unexposed. The two groups are followed to monitor the incidence of the adverse effect (depression incidence), which allows for the calculation of a relative risk (the hazard ratio). However, in this study the authors used the equivalent of a t-test for non-parametric data to compare the level of white matter changes between groups of patients according to their depression status. When the results are presented in this manner, depression status effectively becomes the exposure. Therefore, although it is possible for the authors to conclude that exposure to depression at baseline did not lead to white matter changes, their claim that white matter changes predict depression incidence seems less certain. The presence of overlapping 95% confidence intervals between cohorts also introduces doubt about whether the true value of white matter changes between populations is significantly different, although a wide confidence interval in those patients with depression onset in year 3 of the study is likely related to the small number of patients in this group.

Firbank et al then make a careful attempt to identify and control for potential confounders in their regression analysis. Here, however, the 95% confidence interval for the relationship between white matter changes and depression includes 1 (unity). With such marginal significance, the fate of those patients who were lost to follow-up (over 30%) seems increasingly relevant. Moreover, I wonder why the authors chose to use the Folstein Mini-Mental State Examination as a correlate of cognitive impairment, when executive dysfunction is often most problematic in these patients.4

Future studies might dichotomise patients into ‘high white matter changes’ and ‘low white matter changes’ exposure cohorts to more accurately quantify risk and demonstrate a biological gradient for the effects of vascular disease on mood disorder.


Authors’ reply: We thank Dr Mosley for his interest in our paper. As noted in the discussion to the paper, we agree with him that the relationship between physical disease and mood disorder is complex. However, we do not agree regarding the other points raised.

Our two-group statistic presented in the paper is quite appropriate. We compared progression of white matter changes between those with and without depression at the 3-year point, and demonstrated with a high significance that those with depression had a greater progression of white matter changes prior to depression. If, as suggested by Mosley we dichotomise the white matter change progression, then high white matter change progression is still significantly associated \( (P<0.01) \) with both depression at 3 years, and any depression over the 3 years.

Patient drop-out is a problem in any longitudinal study, and our drop-out rate is fairly typical for the study population. However, participants who drop out are typically less well than those who do not, and it is likely that those developing depression are more likely to drop out. The effect of drop-out is thus more likely to have weakened the association between white matter changes and depression than otherwise.

We agree that executive dysfunction is associated with depression. However, it is also associated with white matter changes,\(^1\) and the purpose of the regression analysis was to investigate whether depression could be accounted for by factors other than white matter changes, rather than attempting to identify the best risk factors, and hence we used the Mini-Mental State Examination as a well-recognised measure of general cognitive ability.

When combined with the previous findings in our cohort\(^2\) demonstrating that baseline white matter changes predict incident depression, we feel confident that, in this cohort at least, our findings robustly demonstrate that vascular disease as measured by white matter changes is a risk factor for depression.

Declaration of interest
J.T.O. is an editorial board member for Psychological Medicine, is Deputy Editor of International Psychogeriatrics. He has been a consultant for GE Healthcare, Servier and Bayer Healthcare, and has received honoraria for talks from Pfizer, GE Healthcare, Eisai, Shire, Lundbeck, Lilly and Novartis.

\(^1\) Debette S, Markus HS. The clinical importance of white matter hyperintensities on brain magnetic resonance imaging: systematic review and meta-analysis. BMJ 2010; 341: c3666.


Corrections
Where are the hypotheses when you need them? BIP, 201, 178–179.
Reference 4 should read:

Association between maladaptive parenting and child self-control over time: cross-lagged study using a monozygotic twin difference design. BIP, 201, 291–297. Figures 1 (p. 293) and 2 (p. 294): the outcome, top right of each figure, should read ‘Emotional difficulties’.

do: 10.1192/bjp.201.5.412a
Assessing the role of cerebrovascular disease in the incidence of geriatric depression

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Access the most recent version at DOI: 10.1192/bjp.201.5.411a

References
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