Strategies to prevent suicide

Should target methods that are commonly used, highly lethal, and readily accessible

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Suicide is a leading cause of premature mortality in low and high income countries. Despite this, information to guide prevention strategies is limited. One of the few approaches with a strong evidence base is preventing access to methods that are highly lethal and commonly used in suicidal acts.[1] The rationale for this approach is based in four observations: suicide attempts are often impulsive, crises are often fleeting, prognosis is good after non-fatal attempts (<10% of people go on to die by suicide), and acts are more likely to be fatal when highly lethal methods are used.

Placing barriers on bridges and other sites from which suicide by jumping is common therefore seems sensible. Jumping is highly lethal and deaths are often very public,[2] leading to media reporting and possible contagion.[3] Reviews and national guidelines have called for safety measures at "suicide hotspots."[4-6]

In the linked study, Sinyor and colleagues describe the apparent failure of the Bloor Street Viaduct barrier in Toronto to reduce suicide by jumping.[7] They assessed the yearly rates of suicide by jumping at the Bloor Street Viaduct from 1993 to 2001 (nine years before the barrier) and from July 2003 to June 2007 (four years after the barrier). Although the barrier prevented suicides from the bridge—deaths fell from 9.3 a year to zero—this had no effect on the rates of suicide by jumping in the region as a whole (although suicide rates overall fell); indeed, there was a possible compensatory rise in suicides from other bridges.

What do these findings mean for the efficacy of preventive barriers? A few notes of caution are important before abandoning their use at selected sites. Firstly, the relatively small number of suicides from the Bloor Street Viaduct (about 9.3 a year before the barrier; <4% of suicides in Toronto) make it impossible to draw any conclusions about the net effect of the barrier on suicide deaths overall. Secondly, suicides from other bridges may have been increasing in the years before the barrier was erected.[7] Thus, the rise in suicides from other bridges may have resulted from an increase in the popularity of bridge jumping rather than substitution. Thirdly, other studies of bridge barriers (with similar sample size limitations) have been more favourable[5]: barriers on the Clifton Suspension Bridge (United Kingdom) led to a halving of deaths from the bridge and a reduction in overall suicides by jumping in the area by males (90% of those who jumped from the Clifton Suspension Bridge before the barriers were put in place were male).[8] Fourthly, as the authors point out, people who jump from bridges that, like the Bloor Street Bridge, span hard surfaces (rather than water) and do not have the iconic status or aesthetic distinction of the Golden Gate or Clifton Suspension Bridge, may differ from those who jump from iconic sites. Moreover, jumpers may be less impulsive than people who use common suicide methods. Data from the US National Violent Injury Statistics System, for example, show that jumpers are less likely to have same-day crises, such as interpersonal conflicts on the day of the suicide, than people who use methods other than jumping (most of whom die in their homes); this suggests that restricting access to highly lethal household methods may, on average, be more effective.[9] Lastly, suicide barriers reduce the number of people who witness gruesome, public suicides and the kind of high profile coverage of these violent deaths that may, in turn, trigger additional suicides.[3]

From a population perspective, with the exception of city states, such as Hong Kong and Singapore, jumping is a relatively rare (<10% total) method of suicide[4]; deaths from specific hotspots are fewer still. If the ultimate goal of means restriction is to reduce the incidence of suicide, the most promising targets are therefore not hot spots, important as these may be, but methods that are commonly used, highly lethal, and readily accessible in or near the home (such as toxic pesticides in developing counties and firearms in the United States).

To be effective in reducing the overall incidence of suicide, however, substitute methods must also be less lethal. Thus the detoxification of the domestic gas supply in the UK, bans on highly toxic pesticides in Sri Lanka, and decreases in household firearm ownership in the United States were followed by marked reductions in method specific suicides and overall suicide rates in these countries.[10-12] These reductions occurred because there was limited substitution by other methods, or if substitution did occur, less lethal methods were used. Moreover, these reductions were measureable and significant at the population level because the restricted method accounted for a large proportion of deaths from suicide.

Sinyor and colleagues' study reminds us that means restriction may not work everywhere, that characteristics of the means targeted are important to consider, and that we have much to learn about the determinants of the choice of method in suicidal acts. Yet, where and when means restriction works, it may save more lives than other suicide prevention strategies, especially in children and young adults, who tend to act impulsively in fleeting suicidal crises.

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Completed suicide after attempted suicide

Methods used in attempts may indicate the degree of risk

FOTOLIA

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Listen to Professor Bo Runeson from the Karolinska Institutet in Sweden discuss how the method of an attempted suicide relates to a later successful attempt at www.bmj.com/podcasts

An increasing number of nations are developing national strategies to try to prevent some of the estimated million suicides that occur annually throughout the world. Detection of people at risk is a key component of such programmes. Although attempted suicide (or self harm) is an important risk factor,[1] the risk of suicide after attempted suicide seems to differ between countries. In the United Kingdom, the risk of suicide in the first year after self harm, although 60-100 times greater than in the general population,[2-4] is much lower than that reported from other countries.[4]

In the linked cohort study, Runeson and colleagues assess the effect of the method of the attempted suicide on the risk of subsequent completed suicide.[5] Using data from Swedish national case registers, the authors found that one in 10 people (12%) admitted to hospital after attempted suicide between 1973 and 1982 died by suicide during long term follow-up (to the end of 2003), 4.2% within the first year. These figures are three to four times greater than those found in UK samples.[2-4] Possible explanations include differing sources of patient samples (all those in the Swedish study had been admitted for inpatient medical care), variations in characteristics of patient populations, and differences in general population levels of suicide.

The most important finding from the Swedish study was that the risk of completed suicide varied considerably according to the methods used in earlier attempts. Compared with patients who had tried to poison themselves, those who used hanging (strangulation or suffocation) had a particularly high risk of subsequent suicide, with more than half eventually dying by suicide (and over two thirds of these people dying in the year after the index attempt). Risk was also higher in people who had tried to gas themselves, jumped from a height, used firearms, and attempted to drown. However, relative lethality should not be assumed from the general method used for self harm—in another Scandinavian study of patients who had attempted suicide by poisoning, those who had used more lethal poisons had a greater subsequent risk of suicide.[6]

Although the absolute and relative levels of suicide risk after attempted hanging reported in Runeson and colleagues' study are far greater than found in a recent UK study,[7] the important point is that people who use more lethal methods in non-fatal attempts have a greater risk of subsequent suicide. One question that Runeson and colleagues could not answer is whether this is because people who use more lethal methods have greater suicidal intent (wish to die). Assessment of suicidal intent is a key element of the psychosocial assessment of patients who self harm.[8] Higher suicidal intent indicates increased risk of eventual suicide, especially in the short term,[9] although this measure can be a poor predictor in individual patients.[10] Assessment of lethality and suicidal intent, which may be poorly correlated in some patients,[11] is likely to be the most informative.

As shown elsewhere,[12] Runeson and colleagues found that patients who had psychiatric disorders diagnosed after suicide attempts had a greater risk of suicide. But within diagnostic categories the method used in attempts was also indicative of relative risk. Patients with psychotic disorders who used hanging in their attempts had a very high risk of eventual suicide (84.1% of men and 84.4% of women died during follow-up). Risk was also increased in patients with affective disorders (a more common diagnosis in patients who self harm) who used hanging. This highlights the importance of carefully looking for signs of psychiatric disorder during psychosocial assessment of patients with self harm.[8]

Most people who died by suicide during follow-up in the Swedish study used the same method as in their non-lethal attempt, except where the initial method had been self cutting. This phenomenon was particularly pronounced for hanging, with more than nine of 10 people who used this method in their initial attempt doing so in their fatal act. Continuity of method was also high for drowning, jumping, and, in men, use of a firearm. This has relevance to restricting availability of methods that might be used for suicide in those at risk.

The results of Runeson and colleagues' study have important implications for assessment and aftercare of patients who self harm; however, caution is needed in their interpretation. Self poisoning is the most common method of self harm in people who present to general hospitals, and most people who subsequently died by suicide in the Swedish study had taken overdoses in the index attempt. Self cutting is the second most common method used for self harm in patients seen in general hospitals. In the Swedish study, such patients had a similar risk of subsequent suicide to those who self poisoned. Although use of more lethal methods of self harm is an important index of suicide risk, it should not obscure the fact that self harm in general is a key indicator of an increased risk of suicide.

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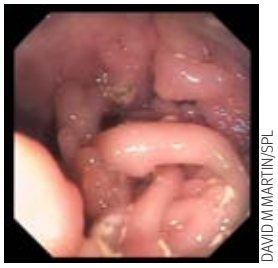
Faecal calprotectin for the diagnosis of inflammatory bowel disease

A useful test in secondary care but not enough evidence to support its use in primary care



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RESEARCH, p 188

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For the family doctor presented with a patient who gives a short history of bloody diarrhoea the clinical diagnosis of ulcerative colitis may be straightforward, particularly if there is a family history of inflammatory bowel disease or recent cessation of smoking. However, making a diagnosis of Crohn's disease can be challenging, especially in younger patients, when symptoms of recurrent abdominal pain and intermittent diarrhoea may be indistinguishable from those of irritable bowel syndrome (a more likely diagnosis). Referral to specialist services for diagnosis usually involves either colonoscopy or flexible sigmoidoscopy, which may be uncomfortable for patients with irritable bowel syndrome who have visceral hypersensitivity. The linked meta-analysis by van Rheenen and colleagues highlights a new approach to the diagnosis of inflammatory bowel disease that might reduce the number of patients referred for endoscopy.[1]

Calprotectin is a non-covalently associated complex of two S100 (A8 and A9) proteins that is released from phagocytes and inflamed epithelia as part of the initial innate immune response. It is resistant to intestinal degradation and is distributed throughout the stool, where it can be detected readily using standard enzyme linked immunosorbent assays (ELISAs). Since the initial reports from Fagerhol's group in Norway,[2] several groups have published data on the accuracy of testing for faecal calprotectin using different assays. The findings have been variable, but with standardisation and experience results are now more consistent. Three assays are commercially available, two of which are based on immunorecognition of the same molecular epitope, either by monoclonal or polyclonal antibodies.

The meta-analysis by van Rheenen and colleagues is timely and notable for two reasons. Firstly, meta-analysis is an unusual method for assessing studies of a diagnostic test but using the QUADAS (QUality Assessment of studies of Diagnostic Accuracy included in Systematic reviews) checklist resolves initial inconsistencies in the published data by prespecifying standards of quality for including studies in the meta-analysis. In this case the authors included only studies in which biopsies had been taken from the right colon or ileum to ensure that microscopic inflammation was not missed. Secondly, the main finding, that the ELISA test for faecal calprotectin has a sensitivity of 93% and a specificity of 96% for the diagnosis of inflammatory bowel disease in adults, is remarkable considering the diverse and complex antigenic environment of faeces.

Interestingly, the authors found that the test had a lower specificity in children. They suggest that the case mix of the study populations may have been responsible for this, but the false positives could actually have been true positives resulting from undetected small bowel pathology only detectable using capsule endoscopy. Alternatively, given that the study protocol allowed for a delay of four weeks between stool sample collection and endoscopy, the stool samples may have come from children with "infectious" diarrhoea that had resolved by the time of endoscopy.

What are the implications of van Rheenen and colleagues' study for routine clinical practice? Our own experience, and that from other centres,[3-5] supports more widespread use of the test in secondary care, not only to reduce the need for colonoscopy in patients referred with symptoms suggestive of inflammatory bowel disease, but also to help tailor immunosuppressive treatment in patients with established disease. The NHS recently commissioned a review of faecal calprotectin from the Centre for Evidenced based Purchasing (CEP) in an attempt to find out what effects major new technologies might have on the NHS.[6] The comprehensive review includes studies of the other commercial assays (the meta-analysis included only the original Phical assay or an in-house assay) and covers technical aspects such as intra-assay variation and limits of detection. Its main findings were that for the NHS, even at current costs, faecal calprotectin was cheaper and more accurate than measuring C reactive protein or erythrocyte sedimentation rate in a diagnostic algorithm to differentiate inflammatory bowel disease from irritable bowel syndrome in primary care.

However, we cannot yet recommend the use of faecal calprotectin as a diagnostic test for inflammatory bowel disease

in primary care, mainly because the results of the current study apply only to patients referred to secondary care. We have no good evidence of how the test performs in primary care, where patient characteristics and populations are probably different, thereby affecting the negative and positive predictive value of the test. This is why van Rheenen and colleagues emphasise the importance of study populations and pretest probabilities when discussing their findings. Certainly, our data from a large number of patients from primary and secondary care suggest that cut-off points might need to be raised to maintain the high negative predictive value.[3] In addition, data on head to head comparisons of the different faecal calprotectin assays are scant. We also need to consider how to avoid overinvestigating patients with infectious diarrhoea, in whom falling faecal calprotectin titres offer a quantitative test that may provide a diagnosis when stool cultures are negative.

If studies conducted in primary care find a high diagnostic accuracy of the faecal calprotectin test it will be an important step forward in how inflammatory bowel disease is diagnosed.

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Reorganisation of the NHS in England

There is little evidence to support the case for yet more structural change

OBSERVATIONS, p 180

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For many healthcare professionals and managers working in the NHS, last week's publication of the white paper *Equity and Excellence: Liberating the NHS* brought unwelcome but familiar news—that the new government plans to reorganise the NHS in England.[1] Despite having promised just two months ago in the coalition government's agreement to "stop the top-down reorganisations of the NHS that have got in the way of patient care,"[2] the new secretary of state, Andrew Lansley, announced plans to abolish strategic health authorities and primary care trusts; to create about 500 new general practitioner consortiums to handle healthcare commissioning; to hand over public health responsibilities to local authorities; to strip the Department of Health of many of its functions and to create an independent NHS board to take them on; to force all NHS providers to become NHS foundation trusts; and to restructure arrangements for healthcare regulation. Little of the current architecture of the NHS will survive these changes unscathed. The white paper, written at breakneck speed in about six weeks, is long on rhetoric but short on detail and specifics. It promises at least seven further strategy or consultation papers on various topics and another white paper, on public health, in the autumn.

For someone who has spent more than six years mastering the health brief in opposition, Andrew Lansley seems to have learnt little from the history of NHS reorganisation, which was analysed recently in detail.[3] Over the past 30 years, governments have reached repeatedly for structural reorganisations of both the NHS and the Department of Health. They have created, merged, and abolished health bodies and distributed service, functional, and geographical responsibilities in different ways.[4] Reorganisation has often been cyclical, with new governments or ministers reinventing structural arrangements that their predecessors abolished, seemingly unaware of or uninterested in past reorganisations. Reorganisation has happened frequently—with at least 15 identifiable major structural changes in three decades, or one every two years or so. And reorganisation has been rapid, with changes often being initiated in advance of formal legislative approval, the details of reforms being worked out as they are implemented, and the timetable for hasty consultation and implementation being a matter of weeks or months. This latest reorganisation looks likely to make all these mistakes again.

We have little evidence that these reorganisations have produced much, or any, improvement.[5] Few NHS reorganisations have been properly evaluated, but a recent study from the National Audit Office of the reorganisation of central government is highly relevant to the Department of Health and the NHS.[6] It makes dismal reading. In the four years up to 2009 the study identified more than 90 reorganisations of central government departments and agencies. The costs of 51 of these reorganisations for which data could be found were £780m (€935m; $1200m), although the authors think this is a substantial underestimate of the true costs. They point out that the benefits of reorganisation were unclear, that the process was often poorly managed, and that its impact on performance was often adverse.

In brief, the government should learn three things from the history of NHS reorganisation. Firstly, structural reorganisations don't work. Although NHS performance may be problematic, there is often little evidence to show that the causes of poor performance are structural or that the proposed structural changes will improve performance. For example, during the 20 years since the internal market was introduced to the NHS, we have seen a bewildering variety of forms and structures put in place to run primary care and commission secondary care[7]—family practitioner committees, health authorities, GP fundholders, total purchasing consortiums, GP multifunds, primary care groups, primary care trusts, and external commissioning support agencies—and now the new government proposes another round of changes and the creation of around 500 new GP

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Listen to Chris Ham, King's Fund, and Edward Davies, *BMJ Careers*, discuss their immediate impressions of the white paper at www.bmj.com/podcasts

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- Editorial: The coalition government's plans for the NHS in England (*BMJ* 2010;341:c3790)
- Observations: GPs have reasons not to be so cheerful over commissioning plans (*BMJ* 2010;341:c3839)

consortiums to undertake commissioning. Yet we have little evidence to suggest that any of these organisational structures for commissioning are better or worse than others, or that the proposed new consortiums will work any better than the current arrangements. Indeed, some would argue that the perceived failures of healthcare commissioning result not from any particular structure but from these repeated reorganisations and the discontinuity and disruption they produce.[8]

Secondly, the transitional costs of large scale NHS reorganisations are huge, although they are often discounted or ignored, and the intended or projected savings from abolishing or downsizing organisations are rarely realised. Closing down or merging organisations produces a round of expensive redundancies, early retirements, and redeployment, while new organisations find new premises and appoint lots of new staff. On the basis of the National Audit Office's survey data,[6] I estimate that the proposed NHS reorganisation will cost between £2bn and £3bn to implement, at a time of unprecedented financial austerity. Reorganisations are often presented as an exercise in cutting bureaucracy, and this one is no exception, with the astounding claim being made that NHS management costs will be reduced by 45%. It remains to be seen whether these changes, which involve abolishing 162 organisations and creating 500-600 new ones, will produce higher or lower management costs, but throughout the past two decades the numbers of NHS managers and the management costs of the NHS have grown steadily, regardless of reorganisation.[9]

Thirdly, and most importantly, reorganisation adversely affects service performance.[10] It is a huge distraction from the real mission of the NHS—to deliver and improve the quality of health care—and it can absorb a massive amount of managerial and clinical time and effort. It saps morale and creates uncertainty for many people about their careers and futures. In addition, new or merged organisations take time to become established and start to perform well.[11] Reorganisation can also destabilise organisations or services and result in poor performance or failure.[12]

The new government's proposals deserve careful scrutiny and debate, both inside and outside of parliament. The government needs to produce empirical evidence—not ideological platitudes—to support the case for change. If this reorganisation proceeds, the government should commit to following the recommendations of the recent National Audit Office report on government reorganisations. This would mean making the intended costs and benefits of NHS reorganisations explicit and measurable in a statement to parliament. They would then be identified and accounted for separately by NHS organisations so that they could then be measured properly. A systematic analysis of the impact of the reorganisation should also be produced within two years of its implementation and presented to parliament.

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New WHO guidelines for the treatment of malaria

Quality assured diagnosis of malaria in Africa is a major challenge

ANALYSIS, p 182

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The publication of a second edition of the World Health Organization's guidelines for the treatment of malaria in March 2010 just four years after the first is testament to how quickly malaria control has developed in the past few years.[1] This is not so much the result of new tools for control, but rather the changing use of existing tools, whose more effective application over the past 10 years has resulted in a marked reduction in the global burden of malaria.[2]

Both the first (2006) and new (2010) editions of the guideline provide a clear account of evidence and recommendations for the treatment of severe and non-severe malaria caused by *Plasmodium falciparum* and the other four species of *Plasmodium* known to infect humans (*P knowlesi* now being recognised as an important zoonosis).[3] In addition, annexes provide scientific detail and references, and the new edition has used the GRADE system, which details the strength of evidence behind each recommendation. The 2010 edition differs from the first edition in four important areas—refining and improving treatment of malaria, minimising the risk of resistance to artemisinin-based combination treatments, using drugs to reduce transmission, and malaria diagnosis.

The choice of partner drug with artemisinin derivatives continues to be refined, and piperaquine, which is attractive because of its low cost and co-formulation, is now recommended alongside lumefantrine, amodiaquine, sulfadoxine-pyrimethamine, and mefloquine. The new

WILLIAM DANIELS/PANOS

***Artemisia* being collected in Uganda, which is used to make artemisinin combination therapy**

guideline places greater emphasis on completing courses of these combined treatments to minimise selection pressure for resistance to artemisinins, which is now a major concern after reports of partial resistance to artemisinin monotherapy in South East Asia.[4]

For severe malaria, intravenous artesunate is recommended in preference to quinine in adults, and it has at least equal preference to quinine in children. The results of the AQUAMAT trial (quinine *v* artesunate for severe malaria in African children), which are expected in early 2011, may guide a more definitive policy.[5] The 2006 guideline included useful advice on the use of antimicrobials in severe malaria, but this section has been removed, possibly because of the lack of evidence from clinical trials. However, this leaves an important gap in recommendations for the treatment of malaria-bacterial co-infection, which is present in 14-25% of inpatient deaths from malaria in children.[6 7]

In areas where reduction in transmission is a priority, a single dose of primaquine is now recommended at the end of a course of artemisinin based combination treatment. This recommendation is based on its historical use and a single trial that showed excellent clearance of gametocytes, although a transient fall in haemoglobin was noted.[8] Mass drug administration to reduce transmission is not supported because it has only ever resulted in sustained malaria control on the small Pacific island of Aneityum.

The single most important operational change in the new guideline is the replacement of "presumptive treatment" in young children (treatment of any childhood fever with no obvious alternative cause as malaria, a cornerstone of WHO policy for the past 20 years) with parasitological diagnosis wherever possible. There is already a consensus that parasitological diagnosis of malaria is highly desirable, but opinion differs on the speed with which this can be achieved in resource poor settings.[9] The evidence needed to guide this decision is limited, and being largely operational, might be inconclusive even if it did exist. So the new guideline has taken a bold step and the question now has to shift from whether we are ready to abandon presumptive treatment to how we can provide a quality assured parasitological diagnosis where infrastructure is weak and the burden of disease is high.

The challenges to achieving parasitological diagnosis of malaria are immense, especially in many areas of Africa where routine slide results compare badly with malaria rapid diagnostic tests or quality assured microscopy.[10] The scaling up of rapid diagnostic tests has progressed rapidly in recent years. Many countries now have substantial rapid diagnostic test programmes supported by the Global Fund, but important areas still need urgent attention. These include selection and cost of tests (more than 80 are now available), establishment of quality assurance systems, and prescriber use and adherence to results.[11]

Slide microscopy remains a key element in parasitological diagnosis of malaria, both for clinical care and quality control of malaria rapid diagnostic tests, but the limited evidence available suggests that the quality of slide results in routine care in Africa is poor, with estimates of sensitivity and specificity often falling below 70% (WHO sets a minimum standard of 90% specificity and 95% sensitivity). Although quality assurance schemes for routine slide microscopy in Africa exist in policy, they rarely operate in practice, and major efforts are urgently needed to correct this.

Targeting antimalarial treatment on those who actually have malaria is an important objective, and the 2010 WHO guideline sets a challenging task to provide accurate parasitological tests for malaria at all levels of the health system. However, the difficulties in translating the aspiration to reality should not be underestimated. Strong leadership is needed from WHO, international funders, and ministries of health if it is to succeed.

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