Suicide risk after a suicide attempt

Is highest in people with bipolar disorder and schizophrenia

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Two linked studies explore the risk of non-fatal self harm after discharge from a psychiatric hospital and the factors associated with the risk of suicide after a suicide attempt.^{1 2} Suicide is one of the 10 leading causes of death worldwide and will represent about 2.4% of the global burden of disease by 2020, with about 1.5 million people dying from suicide each year.³ Making the prevention of suicide a health service and public health priority is justified on medical, ethical, and cost effectiveness grounds.^{4 5}

Previous attempts at suicide increase the risk of suicide 30-40 times.⁶ A history of deliberate self harm is the strongest predictor of future suicidal behaviour.⁷ A systematic review found that 16% of patients who attended an accident and emergency department as a result of deliberate self harm repeated this behaviour and 1.8% died by suicide.⁸

On the basis of a large Swedish cohort study of almost 40 000 people admitted to hospital because of attempted suicide, Tidemalm and colleagues report how many suicides were completed over 30 years.¹ They found that the type of co-occurring psychiatric disorder was an important factor for the overall risk of a completed suicide after an attempted one. The strongest predictors of completed suicide throughout the follow-up period were schizophrenia (men: adjusted hazard ratio 4.1, 95% confidence interval 3.5 to 4.8; women: 3.5, 2.8 to 4.4) and bipolar disorder (men: 3.5, 3.0 to 4.2; women: 2.5, 2.1 to 3.0).

The authors also explored the population attributable fractions for suicide. In line with previous evidence, they found that women with depression or bipolar disorder and men with schizophrenia have a significantly increased risk of death by suicide.

The risk assessment of suicidal behaviour should not only include psychiatric comorbidity because other factors such as previous self harm are important. The second linked study, by Gunnell and colleagues, assesses the risk of non-fatal self harm in about 75 000 patients for up to one year after their discharge from psychiatric inpatient care.²

Of the total sample, 6% were admitted to hospital because of self harm within 12 months of discharge. Further evidence of the need for intensive aftercare is that a third of these episodes occurred in the first four weeks after discharge. As expected, admission for self harm to a psychiatric unit during the year before admission was one of the strongest risk factors for self harm after discharge. The risk was higher for women than for men. It was also higher in younger patients and in

those with a shorter length of inpatient stay. Gunnell and colleagues emphasise that patients with personality disorders, depression, and substance misuse have a high risk for self harm.

What are the practical implications for clinicians? The care of patients who attempt suicide or deliberately harm themselves should include routine psychiatric and psychosocial assessment and standardisation. Up to 45% of patients who deliberately harm themselves leave accident and emergency departments without being comprehensively assessed by a suitably qualified healthcare professional or do not receive a specialist psychosocial assessment.^{9 10} Routine psychiatric assessment might be the most important initial step when organising aftercare.

As Gunnell and colleagues report,² depression and substance misuse are important risk factors for self harm after discharge. These problems are often undiagnosed in patients with deliberate self harm as a consequence of non-standardised procedures for assessment and aftercare.^{11 12}

Systematic referral to professional services before and after discharge is therefore recommended. Depression, bipolar disorder, and schizophrenia are the strongest predictors of suicide risk in people who have attempted suicide. For these people, aftercare should also include education of their immediate family and friends. In addition, recent studies found that contacting patients by telephone one month after discharge from an emergency department for deliberate self poisoning may reduce the number of repeat episodes.¹³ Self poisoning is one of the most common methods of attempted suicide, so access to toxic substances needs to be restricted; this will require better regulation by public authorities.

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Screening for colorectal cancer

Should be tailored to available resources, local experience, and population characteristics

GI ASSOCIATES/CUSTOM MEDICAL STOCK PHOTO/SPL

Colorectal cancer is the second most common cause of death from cancer in Europe and the United States.^{1,2} Population screening trials using the guaiac faecal occult blood test have reduced mortality by around 16%.³ National or regional colorectal cancer screening programmes and pilot studies for the general population have recently been introduced in more than 50 countries.⁴ Such organised screening programmes are the best way to reduce the incidence of colorectal cancer.

In the linked study, Malila and colleagues report the test, episode, and programme sensitivities for a new randomised method of introducing population screening for colorectal cancer in 2004 in Finland.⁵ Test sensitivity refers to the proportion of colorectal cancer cases detected by the test in the preclinical phase of the disease. Episode sensitivity is a function of test sensitivity (confirmed by diagnostic testing) that incorporates interval cancers not detected during the time between screening tests. Programme sensitivity is a much broader concept and is based on the proportion of total cancers detected for all people invited to participate in screening (that is, those who do and do not return the test kits).

The participation rate of 71% will be viewed with envy by many other programmes. The higher participation rate in women than in men confirms the findings of other studies of population screening using the faecal blood test. In contrast, uptake for endoscopic screening is widely reported as being higher in men than in women.⁶

Screening aims to diagnose disease while it is curable and so prevent future deaths. It is important therefore to minimise the proportion of interval cancers (those that occur in the time between screening rounds) and maximise the proportion of cancers identified by screening. Sensitivity is defined as the ability of a test to identify a disease. The test sensitivity of 55% for the guaiac faecal blood test reported by Malila and colleagues is similar to that seen in previous European randomised controlled trials.^{7,8}

The authors hold that the reported test, episode, and programme sensitivities are important measures for evaluating the effectiveness of a screening programme. However, the use of programme sensitivity as an outcome measure for a cancer screening programme should be treated with cau-

tion. Programme sensitivity is a direct function of the participation rate, so population uptake (for the faecal blood test and colonoscopy) must be high for programme sensitivity to be similar to episode sensitivity.

A major weakness of faecal blood test screening is its low sensitivity. But other aspects of a programme must also be taken into account to determine its effectiveness. Firstly, specificity—the relatively low programme sensitivity of 37.5% reported by Malila and colleagues might be compensated for by an acceptable specificity, which would reduce the number of false positives that cause unnecessary anxiety and require invasive diagnostic procedures to be carried out. Secondly, attendance at colonoscopy—was the episode sensitivity of 51% a function of accessibility to colonoscopy or did it represent other factors (such as inadequate bowel preparation, particular subgroups being reluctant to undergo the procedure)? Attendance at colonoscopy is fundamental to reducing the incidence of and mortality from colorectal cancer. Thirdly, informed decision making by participants—were participants satisfied that they had been given pertinent information about the benefits and risks of screening (such as the ability of screening to detect colorectal cancer and potential complications associated with colonoscopy)?

The authors report that test and episode sensitivity were considerably higher in men than in women (14% higher for test sensitivity and 11% for episode sensitivity). In contrast, the incidence of interval cancers was higher in women than in men (49 v 42 per 100 000 person years). Do these differences mean that screening for colorectal cancer is more effective in men than in women, or should other factors be considered?

As anticipated on the basis of the epidemiology of colorectal cancer,⁹ evidence shows that the benefits of screening are seen some years later in women than in men. A study in Poland and screening programmes in Italy found that men reach a given prevalence of colorectal cancer and level of detection of advanced adenomas some five to 15 years in advance of women.^{10,11} This has prompted the suggestion that sex specific screening for colorectal cancer may be warranted,¹² although this may be difficult to achieve in practice.

The difference in the location of colorectal cancer in men and women is also important—cancer in the distal

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colon and rectum is less common in women than in men. Consequently, the sex difference in the occurrence of distal colon cancer is even more pronounced than that of the overall occurrence of colorectal cancer. Mean sojourn time—the time before clinical symptoms become apparent but during which the disease is detectable—differs greatly by subsite. The tumour growth rate is considerably slower for distal colon cancer than for proximal or rectal cancers.¹³ It could be unreliable and misleading to consider the sensitivity of the faecal occult blood test without taking into account mean sojourn time, tumour subsite, the screening interval, and sex differences.

The new design for implementing the Finnish colorectal cancer screening programme allows for the screening interval, screening modality (for example, the introduction of an immunochemical faecal occult blood test), and age at beginning of screening to be monitored and modified if necessary. However, many programmes will have to be phased in on the basis of the availability of facilities, the experience of health professionals, and the capacity of the service provider. Also, achieving informed consent from the entire target population for participating in this type of programme implementation may be a problem for many countries. Therefore, logistically, randomisation may not be possible in many situations, and implementation will depend on local circumstances.

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Caffeine intake during pregnancy

Should be minimised, but not replaced with unhealthy alternatives

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In the linked cohort study, the CARE Study Group reports that consuming caffeine during pregnancy is associated with an increased risk of fetal growth restriction.¹ For 100-199 mg caffeine a day the odds ratio was 1.2 (95% confidence interval, 0.9 to 1.6), for 200-299 mg a day it was 1.4 (1.0 to 2.0), and for over 300 mg a day it was 1.5 (1.1 to 2.1).

Coffee and tea contain a variety of chemical compounds, but most of the health concerns relate to caffeine. One cup of coffee contains about 100 mg of caffeine and a cup of tea about half of this amount; the exact amount varies according to cup size, brewing methods, and brands of coffee or tea.² Caffeine is also present in cola, chocolate, cocoa, and some drugs. Most of the caffeine that adults consume comes from coffee,³ but in the present study 60% of the caffeine that pregnant women consumed came from tea.¹

Some studies have shown no negative association between drinking tea during pregnancy and fetal growth,^{4,5} which may indicate that caffeine is not the culprit or that the results for coffee were confounded. However, the present study's finding of an adverse effect in tea drinkers will reinforce the concern that caffeine is a potential fetotoxic substance.

Caffeine easily crosses the feto-placental unit, and in large doses it may cause harm to the unborn child.

Observational studies have shown associations with reduced fetal growth and fetal death,^{6,8} but not with pre-term delivery.⁹ All of these studies are subject to confounding by unmeasured or partially measured factors.

Drinking coffee and tea correlate with other lifestyle factors like smoking, work load, and perhaps also dietary habits. Only one study on drinking coffee has used a randomised design, and this study found no overall reduction in birth weight in women who regularly drank coffee containing caffeine compared with those who drank decaffeinated coffee, except in those who smoked.¹⁰ Unfortunately, Boylan and colleagues did not stratify their results on smoking habits or cotinine concentrations.

The metabolism of caffeine depends on genetic and environmental factors.¹¹ Caffeine is metabolised in the liver primarily by CYP1A2 and NAT2, and people can be classified as slow or fast metabolisers. In observational studies, therefore, the fetuses of women who are slow metabolisers will be exposed to more caffeine than the fetuses of fast metabolisers with an equivalent caffeine intake.

The use of longitudinal sampled biomarkers for caffeine exposure is needed to bypass this problem, but such data have not yet been collected for large samples of pregnant women. Boylan and colleagues rely on self

reported intake of products containing caffeine, so their data on caffeine exposure will have large measurement errors. It could be argued that these errors are probably non-differential and may therefore lead to underestimated effect sizes.

The women in Boylan and colleagues' study did, however, perform a caffeine half-life challenge test, and although this test was not validated, the results show that faster metabolisers have a higher risk than women with a slower clearance rate who have the same caffeine intake. This indicates that it is not caffeine but one of its metabolites that causes harm—this notion is supported by another study based on biomarkers for caffeine exposure.¹² A possible effect modification by clearance rates is important, but as always confounding is a concern. For example, the comparison may be confounded by stress and sleeping patterns or by some of the many other compounds that are metabolised by the same enzyme system. Slow metabolisers with a high intake of caffeine may not be comparable with fast metabolisers with a similar intake.

Boylan and colleagues found less variation in tea consumption during pregnancy than is normally seen for coffee intake. Many women report an aversion for coffee during pregnancy but not for tea. The authors found no indication for a threshold effect or for a larger effect later in pregnancy when the fetal demand for energy is highest.

Their results will probably reopen the debate on what advice we should give to women who are or intend to become pregnant. The authors propose that every effort should be made to stop pregnant women consuming caffeine or to reduce intake greatly, because the association between fetal growth restriction and caffeine is reduced

for those consuming less than 100 mg a day.¹ But we think that this advice is not justified by the current body of evidence, and that such advice may unnecessarily frighten women who have consumed caffeine while pregnant. We do, however, think that pregnant women should be advised to reduce their intake of caffeine products during pregnancy, but that they should not replace caffeine containing beverages with drinks containing alcohol or soft drinks loaded with sugar.

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Auditory processing disorders

Can cause educational, behavioural, and social problems but are often undiagnosed

Hearing impairment is the most common sensory disability worldwide and has a profound effect upon an individual's ability to function at a personal, social, and occupational level. In February 2008, the Royal College of Physicians published a report about the diagnosis and management of hearing and balance disorders.¹

The hearing process is underpinned by the transmission and analysis of sounds from the external ear to the auditory cortex. Disorders of the external, middle, and inner ear are well recognised, but recent advances have made us aware of hearing disorders related to the auditory nerve and brain.

In 1954 it was shown that brain pathology can lead to hearing difficulties that are not identified by conventional audiometry.² More recent work has shown that hearing difficulties can be caused by disordered auditory processing within the brain across all age ranges. Auditory processing disorders result from impaired neural function and are characterised by poor recognition, discrimination, separation, grouping, localisation, or ordering of non-speech sounds.³ They may be developmental

or acquired. The exact prevalence of auditory processing disorders is unknown, but they are estimated to affect around 5% of school aged children and an even higher proportion of adults.

Auditory processing disorders can develop as a result of sound deprivation associated with hearing loss, glue ear, or neurological conditions (tumours, stroke, and demyelination). They may be associated with higher order disorders such as dyslexia and specific language impairment, or they may simply reflect age related changes of the central auditory system in the older population.⁴⁻⁶

Auditory processing disorders manifest as hearing difficulties in the presence of competing sounds, difficulty in localising sounds, and difficulty in following oral instructions or understanding rapid or degraded speech (for example, over a loudspeaker), despite the presence of normal hearing thresholds.⁷⁻⁹ Children with auditory processing disorders may experience difficulty in a wide range of hearing situations (in quiet places, noisy places, and environments with multiple and complex sensory inputs) and may have problems with auditory

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memory and attention.¹⁰ This may lead to inattention and distractibility; academic difficulties; and disorders involving language, reading, and spelling.⁹ Children and adults with auditory processing disorders have a higher than normal chance of having behavioural, emotional, social, and other difficulties,⁹ and they may be perceived as disruptive, uncooperative, or unsociable.

Accurate diagnosis of auditory processing disorders remains a clinical challenge, not least because of the anatomical and physiological complexity of the central auditory nervous system—multiple decussations and relay centres, simultaneous processing in different domains and at different levels, and multimodal input and integration. The design and interpretation of current tests have limitations, and universally accepted diagnostic criteria are lacking.^{3 11} The diagnosis of auditory processing disorders requires detailed medical assessment and is currently based on comprehensive investigations (see box on bmj.com).^{3 7 8}

The appropriate intervention for auditory processing disorders depends on the patient's symptoms and the results of the investigations. Interventions can be implemented by a range of professionals working in different health, education, and social environments.^{3 12} Educational audiologists, teachers of the deaf, and audiologists should collaborate with the diagnosing clinician and oversee the implementation of strategies that aim to improve the quality of the auditory signal in school and work environments. The use of personal or sound-field FM systems (wireless devices that receive, amplify, and transmit the sound to the listener's ear); speaker or teacher based adaptations; and environmental modifications to conform to UK building regulations, which define the upper limit of ambient noise and reverberation (multiple reflections of sounds within a room) may be valuable.

Auditory training is an emerging intervention in all age groups. It aims to ameliorate auditory processing by promoting cerebral plasticity (the potential of the brain to change its sensory representations in response to learning or altered receptors, sensory environment, or use) but might also improve processes such as attention. Auditory training includes simple and complex tasks implemented

by means of commercial computerised game type programmes or with the help of a parent or teaching assistant, speech and language therapist, or hearing therapist. Activities become progressively more difficult as the patient's competence improves. In addition, auditory training may be complemented by compensatory strategies that target higher order processes. Current outcome studies of auditory training are conducted on children with a wide range of developmental disorders that may not necessarily overlap with or be caused by disordered auditory processing. These are mostly non-randomised controlled studies and are hampered by additional methodological limitations, such as lack of uniform criteria for auditory processing disorders, and the wide range of interventions assessed; however, some evidence indicates that the above interventions may be beneficial.¹²

What are future priorities? Reliable diagnostic tools and uniform diagnostic criteria are essential, as acknowledged by several multiprofessional consensus conferences.^{3 7 8} These would facilitate high quality research, which would translate into widespread evidence based practice.

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Coronary stent thrombosis in the perioperative period

Is potentially life threatening, but simple steps can minimise the risk

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The American College of Cardiology and a multidisciplinary French Task Force have recently published guidance on perioperative stent thrombosis.^{1 2} We summarise these guidelines and highlight several simple steps that can be taken to minimise the risk of this potentially life threatening perioperative condition.

The introduction of coronary stents revolutionised percutaneous intervention in patients with coronary heart disease. Apart from preventing abrupt vessel closure, coronary stents reduce vessel recoil and restenosis.³ In 2006, coronary stents were deployed in more than 90%

of the 70 000 percutaneous coronary interventions performed in the United Kingdom, with similar patterns of use reported worldwide.³ This has led to the emergence of two important complications—in-stent restenosis and stent thrombosis. Drug eluting stents, consisting of a stent covered by a thin polymer impregnated with antiproliferative drugs, have reduced the rates of in-stent restenosis by more than 70%, and nearly six million patients worldwide have received a drug eluting stent.³

Stent thrombosis is a serious complication associated with poor clinical outcomes—20% mortality and

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myocardial infarction in 70% of cases.⁴ Initially, rates of acute and subacute (within 30 days of deployment) coronary stent thrombosis were high, complicating 6-12% of percutaneous coronary procedures. Treatment with dual antiplatelet therapy (aspirin and clopidogrel) for four weeks after placement of bare metal stents has reduced this to less than 2%.³ Although drug eluting stents have reduced restenosis, they are associated with a small but significant increase in very late (more than one year), and to a lesser extent, late (30 days to one year) stent thrombosis.⁴ This has been attributed to delayed or incomplete endothelialisation,⁵ hypersensitivity reactions to the drug or stent polymer, and the greater complexity of interventions being performed, often outside of the licensing indication. Discontinuation of antiplatelet drugs is one of the most important independent predictors of stent thrombosis.⁶ Current guidelines recommend 12 months of dual antiplatelet therapy after placement of a drug eluting stent.¹ The increase in very late stent thrombosis has prompted calls to extend this treatment beyond 12 months, although the optimal duration is unknown.

Antiplatelet therapy is often stopped perioperatively because of concerns about excessive bleeding. This, coupled with the proinflammatory and prothrombotic state associated with surgery, increases the risk of perioperative stent thrombosis. Previous work evaluating outcomes in patients undergoing non-cardiac surgery reported extremely high rates (up to 40%) of death, myocardial infarction, and stent thrombosis in those having surgery within four weeks of bare metal stent implantation.⁷ The risks of stent thrombosis in patients with drug eluting coronary stents undergoing non-cardiac surgery are not known, but risk is probably high for longer than four weeks after stent implantation.

So what can be done to minimise the risk of perioperative stent thrombosis? Coronary revascularisation should be avoided in patients being considered for non-cardiac surgical procedures. Data from patients undergoing vascular surgery do not support the use of preoperative coronary artery stenting to reduce cardiac risk in patients undergoing major non-cardiac surgery.⁸ Such intervention may just increase the risk.⁹

If non-cardiac surgery is needed after implantation of a bare metal coronary stent, it should be deferred for four to six weeks.¹² If a drug eluting stent has been implanted, surgery should be deferred for six to 12 months.¹² Where this is not possible, a risk assessment should be made on the basis of the extent and type of surgery proposed, the consequences of bleeding, the complexity and relative timing of previous percutaneous coronary procedures, and the location and type (bare metal or drug eluting) of stent used.

Aspirin and clopidogrel are irreversible inhibitors of platelet activation and, with the exception of neurosurgery, combined therapy seems to increase surgical bleeding by about 50% without affecting morbidity or mortality.^{10 11} The risks and consequences of excess bleeding should be carefully considered before discontinuing dual antiplatelet therapy. Dual antiplatelet therapy need not be interrupted for dental or minor surgical procedures.¹² Where therapy must be discontinued

Coronary artery stent at the level of the anterior interventricular artery

prematurely to minimise the operative bleeding risk during major surgery, trial data suggest that this should be done at least five days before surgery.¹⁰ Aspirin should be continued perioperatively if possible and clopidogrel restarted at the earliest opportunity after surgery.¹²

A cardiologist must be involved in any decision to discontinue antiplatelet therapy prematurely in patients with a recently implanted coronary stent. In patients at high risk of perioperative stent thrombosis in whom an interruption in dual antiplatelet therapy is unavoidable, "bridging" therapy with heparin, direct thrombin, or short acting intravenous glycoprotein IIb/IIIa inhibitors until oral antiplatelet agents can be safely restarted should be considered, although the success of this approach is unclear.¹² For such patients, it might be safest to perform surgery in centres with rapid access to emergency percutaneous coronary intervention.

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