that all patients were free to seek help for their mood problems. Patients may feel tired and low in mood but may not recognise this as depression, for which there are effective interventions available. Is it ethical to withhold information regarding the diagnosis from such patients? Will patients seek help if they are not told they have depression?

Performing research can raise difficult ethical issues and I hope this letter will encourage some debate on this.

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Van Melle *et al* (2007) found no difference in efficacy and cardiac prognosis between treatment with antidepressive medication and care as usual in patients with depression after myocardial infarction. Carney & Freedland (2007) commented that the lack of difference in efficacy prohibits the demonstration that effective treatment of depression improves survival. They emphasised the need for developing highly efficacious treatments for depression following myocardial infarction. Such a treatment, however, already exists, as electroconvulsive therapy (ECT), and has been shown to have superior efficacy compared with antidepressive medication (ECT UK Review Group, 2003).

A trial using ECT as an intervention will more likely find a superior efficacy compared with treatment as usual and may demonstrate that effective depression treatment improves survival. Because of concerns about the cardiac risks some textbooks do not recommend the use of ECT within 3 months of myocardial infarction. Zielinski *et al* (1993) found a higher rate of cardiac complications during ECT in patients with a pre-existing cardiac abnormality compared with patients with no pre-existing abnormality. Most complications, however, were transitory and did not prevent the completion of the ECT course. Rice *et al* (1994) found that ECT increased the risk of minor but not severe complications. They pointed to the advances in ECT techniques which have resulted in improved safety in cardiac patients. The risk of ECT has to be weighed against the risk of an inadequate treatment of depression, which is known to increase mortality (van Melle *et al*, 2007). Considering the high risk of cardiac events of 13% in the 18 months following myocardial infarction (van Melle *et al*, 2007), which may partly be attributable to the inadequate treatment of depression, treatment with ECT could be safer because of its superior efficacy as an antidepressant.

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Authors' reply: To explain why antidepressant treatment with selective serotonin reuptake inhibitors (SSRIs) does not improve cardiac prognosis, Korszun *et al* suggest that SSRIs may not alter the mechanisms through which depression leads to increased cardiovascular morbidity and mortality. However, two other explanations may be more plausible. First, the effects of antidepressant treatment on depression itself are not strong enough. In both the ENRICHD and SADHART trials, the response rates of patients in the active treatment arm hardly exceeded those of patients receiving usual care or placebo. Second, the cardiotoxic effects of depression are limited to patients for whom antidepressant treatment is not effective (Grace *et al*, 2005; de Jonge *et al*, 2006*a*). We have shown that the cardiotoxic effects of depression are concentrated in incident post-myocardial infarction depression, whereas results from the SADHART study have indicated that sertraline is only effective in non-incident depression (of interest, Korszun *et al* mention mechanisms related to recurrent depression, which appears not be to cardiotoxic). If antidepressant treatment is only effective in non-cardiotoxic depression, no effects on cardiovascular prognosis can be expected.

Shetty raises ethical concerns about our study, because we used Zelen's method of randomisation. Controls were not told about their diagnosis of depression and, as argued by Shetty, therefore may have 'missed' an offer of adequate treatment. However, we feel that in 1999, when the study started, Zelen's method was both scientifically useful and ethical because no controlled comparative studies had yet investigated the clinical efficacy and safety of antidepressant drugs in depression post-myocardial infarction. At that time, the proportion of myocardial infarction patients with depression who were treated for their post-myocardial infarction depression was negligible. In addition, serious concerns existed about the safety of antidepressant drugs in cardiac patients. Moreover, in our study patients with a significant risk of suicide or severe depression were excluded from randomisation and referred for psychiatric treatment outside the study. Finally, all patients received usual care, i.e. had cost-free access to all usual treatment facilities such as normal cardiac rehabilitation programmes and healthcare from family physicians. We therefore feel it was ethical to use Zelen's method in our study and scientifically useful as our control patients were truly representative of patients receiving usual care.

We agree with Dr Kho that we need to develop new treatments for depression post-myocardial infarction, but believe it is premature to consider electroconvulsive therapy (ECT) as an effective alternative. In our experience those types of depression that are least similar to those seen in general psychiatry (i.e. incident depression occurring for the first time after myocardial infarction; de Jonge *et al*, 2006*b*) and those that are dominated by feelings of exhaustion rather than negative self-esteem or suicidality (de Jonge *et al*, 2006*a*) are the most cardiotoxic. To our knowledge the mechanism(s) explaining this remain unclear. Similarly, it is not known whether ECT is effective in these subtypes (although it appears that antidepressive medication is not). In fact, the studies mentioned by Dr Kho suggest increased rather than decreased cardiovascular events.

New, effective treatments for depression post-myocardial infarction will improve quality of life but perhaps also survival, as rightfully argued by Dr Kho. Carney *et al* (2004) showed that responders to antidepressive medication had a better cardiovascular prognosis than non-responders,

using data from the ENRICHD trial. In the MIND–IT study we recently confirmed that non-response to mirtazapine and citalopram was associated with more cardiovascular events compared with responders and even untreated controls, a finding that remained after controlling for several confounders, including early cardiovascular events (de Jonge *et al*, 2007). However, as it is unclear what factors are related to response to antidepressive medication (these may well include the presence of somatic symptoms of depression; Tylee & Gandhi, 2005), it also remains uncertain whether it might be an improved state of the heart disease that influences depression or reversely that treatment of depression results in an improved cardiovascular prognosis. However, although causality remains unproven it suggests that more effective treatments may have cardiovascular effects as well. We are not yet convinced that this will be ECT but we encourage researchers to explore this possibility.

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Substance misuse disguised as ADHD?

Attention-deficit hyperactivity disorder (ADHD) is a rather novel disease in adults. It has drawn increasing attention and at present there is no deficit of studies of ADHD in adults (Fayyad *et al*, 2007). Several studies have shown a considerable risk of co-occurring substance misuse in adults given the diagnosis of ADHD (Aanonsen, 1999; Wilson, 2007). Symptoms of ADHD seem to hamper success in methadone maintenance treatment (Kolpe & Carlson, 2007). Fayyad *et al* indicate in Table 5 that in 99% of cases adult ADHD occurs first in patients with a co-occurring substance use disorder but this is not commented upon in the discussion part of their paper. Respondents were classified retrospectively as having met full ADHD criteria in childhood. To ascertain the presence of ADHD in adulthood respondents were asked a single question only, whether they continued to have problems with attention or hyperactivity.

In Norway we have an impression that people with substance misuse tend to ask for a diagnosis of ADHD, as this may lead to better treatment within the psychiatric care system. The finding of Fayyad *et al* of higher prevalences in high-income countries, with purportedly better services for the treatment of ADHD, may be an indication of common presenting symptoms in substance use disorder and ADHD. Could the authors have observed symptoms and behaviour related to substance misuse and not ADHD?

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Authors' reply Dr Berg raises the possibility that respondents in our surveys who reported persistence of ADHD in adulthood might actually have had symptoms caused by some other disorders, such as alcoholism, that are more stigmatising and less likely to be treated than ADHD. Such respondents might consciously have provided incorrect information in an effort to avoid stigma and to increase their chances of receiving treatment. Dr Berg states that such machinations occur in his country. This is an important point in view of the stigma associated with mental disorders and the fact that some healthcare systems discriminate against certain diagnoses. Mental health professionals need to increase their efforts to raise awareness and address these problems.

That said, it strikes us as implausible that our findings are importantly affected by the sort of bias proposed by Dr Berg. First, the World Mental Health surveys are community epidemiological surveys in which no treatment is provided. Second, in a number of the participating countries ADHD is not commonly recognised as an illness, making it unlikely that community respondents would have the sophistication to seek out this diagnosis. Third, we carried out in-depth clinical reappraisal interviews with a probability sub-sample of respondents who reported adult persistence of ADHD. We excluded respondents if concerns existed that another diagnosis might be primary. Although it is possible that some respondents were so familiar with ADHD that they tricked our experienced clinical interviewers, we consider it unlikely that this was widespread. Fourth, treatment-seeking was low in most World Mental Health surveys. When it occurred, the reason for seeking treatment was not ADHD but a comorbid disorder.

Irrespective of whether the type of bias Dr Berg suggested exists in epidemiological surveys, our results imply that clinicians should look more seriously for ADHD in their adult patients than they have before. As more physicians screen for ADHD among adults presenting for treatment of other psychiatric disorders, the extent to which untreated adult ADHD exists among help-seekers will become apparent.

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