Contribution of depression and anxiety to impaired health-related quality of life following first myocardial infarction

C. M. DICKENS, L. McGOWAN, C. PERCIVAL, B. TOMENSON, L. COTTER, A. HEAGERTY and F. H. CREED

Background   The extent to which depression impairs health-related quality of life (HRQoL) in the physically ill has not been clearly established.

Aims   To quantify the adverse influence of depression and anxiety, assessed at the time of first myocardial infarction and 6 months later, on the physical aspect of HRQoL 12 months after the infarction.

Method   In all, 260 in-patients, admitted following first myocardial infarction, completed the Hospital Anxiety and Depression Scale and the Medical Outcomes Study SF–36 assessment before discharge and at 6- and 12-month follow-up.

Results   Depression and anxiety 6 months after myocardial infarction predicted subsequent impairment in the physical aspects of HRQoL (attributable adjusted $R^2 = 9\%$, $P < 0.0005$). These negative effects of depression and anxiety on outcome were mediated by feelings of fatigue. Depression and anxiety present before myocardial infarction did not predict HRQoL 12 months after myocardial infarction.

Conclusions   Detection and treatment of depression and anxiety following myocardial infarction improve the patient's health-related quality of life.

Declaration of interest   F.H.C. has performed consultancy work for Ely Lilly in a field unconnected with the current study. There are no other conflicts of interest to declare.

Although psychiatrists commonly acknowledge the considerable threat to public health posed by depression, the extent to which depression contributes to mortality and illness burden through interactions with physical illnesses is often underestimated. Taking the example of coronary heart disease, the most common cause of death in the UK, depression increases the risk of incident coronary heart disease 1.6 times, even after controlling for other cardiovascular risks (Rugulies, 2003; Wulsin & Singal, 2003). Furthermore, depression doubles the risk of death in those with established heart disease, such as following myocardial infarction (Barth *et al*, 2004; van Melle *et al*, 2004). As survival after myocardial infarction has increased, attention has also focused on the adverse effects of depression on health-related quality of life (HRQoL) (Rumsfeld & Ho, 2005). Relevant studies, however, have only partially controlled for the influence of potential confounding factors (Brown *et al*, 1999; Failde & Ramos, 2000; Pocock *et al*, 2000; Taira *et al*, 2000; McBurney *et al*, 2002; van Jaarsveld *et al*, 2002; Izawa *et al*, 2004; Kim *et al*, 2005).

METHOD

We performed a prospective cohort study to investigate the extent to which depression and anxiety contributed to impairment in HRQoL 12 months after the patient had been admitted with their first myocardial infarction. We aimed to understand more fully among people with coronary heart disease the burden of illness that is attributable to depression. We controlled for socio-demographic and clinical characteristics at baseline, including comorbid, non-cardiac illnesses and any further cardiac events during the follow-up period. Since depression may develop in the months following myocardial infarction (Blumenthal *et al*, 2003; Poston *et al*, 2003; Dickens *et al*, 2004a), we included depression severity measured at baseline and at 6-month follow-up as predictive variables. We tested the hypothesis that anxiety and depression would predict impairment in HRQoL 12 months following first myocardial infarction, after controlling for possible confounding factors.

The study was approved by the relevant local research ethics committees.

Sample

Consecutive patients admitted to four inner-city hospitals with suspected myocardial infarction were screened for this study, on average 3.6 (s.d.=3.3) days after admission. Individuals were recruited if they had no history of previous myocardial infarction, were aged less than 80 years and met World Health Organization criteria for myocardial infarction (Gutzwiller, 1994). Two of the following were required: history of typical chest pain, characteristic echocardiogram changes, a serial rise in cardiac enzyme creatine phosphokinase (CPK). People with severe mental illness (including severe depression with suicidal ideas) were excluded from the study. Written, informed consent was provided by participants after the procedures involved in the study had been fully explained.

Participants received treatment as usual, as determined by the attending hospital physician and primary care doctors. As this was an observational study, participants did not receive any additional therapy (e.g. antidepressants) based on their research assessments.

Baseline assessments

We recorded age, gender (male=1, female=2), ethnicity (White=1, Black and minority ethnic=0), marital status (single: yes=1, no=0), years of education, smoking history (past or current smoker=1, never smoked=0), past psychiatric history (yes=1, 1, no=0) and comorbid illnesses for which participants were receiving regular treatment (for each illness yes=1, no=0). Socio-economic status, based on current or latest employment, was categorised into high or low according to the criteria of Goldthorpe and Hope (Goldthorpe & Hope, 1974), (lower status=1, higher status=2). Serum cholesterol was measured during admission.

Details of the myocardial infarction were recorded from the notes, with discussion with the attending cardiologist if clarification was required. We recorded site of infarction as determined by echocardiogram, categorised as anterior (including

anterior, anterolateral, anteroseptal and apical myocardial infarctions, scored as 1) or other (scored as 2). Details of treatment, including reperfusion therapy, surgical intervention during index admission (angioplasty or coronary artery bypass graft) and discharge medication were also noted. We used three measures of severity of myocardial infarction. The Killip class, a 4-point scale, rated left ventricular function based on the presence of pulmonary rales, S3 gallop rhythm and peripheral hypoperfusion, with high scores representing worse function (Killip & Kimball, 1967). Creatine phosphokinase levels were measured on admission and on 2 subsequent days as an indication of the extent of myocardial damage. Finally, left ventricular ejection fraction measures (using echocardiogram) were recorded for 217 patients.

Anxiety and depression were assessed using the self-rated, 14-item Hospital Anxiety and Depression Scale (HADS), which scores the severity of the symptoms of anxiety (scored 0 to 21) and depression (scored 0 to 21). This measure was developed specifically for medically ill populations, and excludes bodily symptoms such as sleep disturbance, fatigue and pain that may be owing to physical illness (Zigmond & Snaith, 1983). The HADS has been widely used in people with medical illnesses, including myocardial infarction, and its factor structure has been confirmed (Herrmann, 1997; Herrmann et al, 1998; Mayou et al, 2000). At baseline, participants were instructed to complete the questionnaire to reflect their mood during the week before myocardial infarction, in order to avoid recording mood changes that occur as immediate reactions to the infarction and during time spent on the coronary care unit. In addition to using HADS sub-scores to measure severity of depression and anxiety, a HADS total score of 17 or above was taken to indicate probable major depressive disorder, as we have validated this score against a standardised research interview (Dickens et al, 2004b). We did not aim to measure distress in the period immediately after infarction, and we have recorded the fact that HADS scores rated for the days following admission were significantly higher than those rated for the week before, which were used in this report (Dickens et al, 2004b).

Participants completed the Illness Perceptions Questionnaire (Weinman et al, 1996), which assesses patients' perception of their illness in terms of:

(a) its bodily symptoms ('identity' scale – scored 0 to 14, with high scores indicating more reported symptoms);

(b) its likely duration ('time-line' – scored 1 to 5, with low score reflecting longer anticipated duration);

(c) the perceived consequences (scored 1 to 6, with low score indicating more serious perceived consequences);

(d) the likelihood of the illness being cured or controlled (scored 1 to 5, with low score indicating more perceived control).

This scale has been used previously for people who have experienced myocardial infarction (Petrie et al, 1996, 2002).

We used the self-reported Medical Outcomes Study SF–36 (Ware & Sherbourne, 1992) to assess both the physical and mental aspects of HRQoL at baseline, 6 months and 12 months following myocardial infarction. The SF–36 is a generic measure of HRQoL that provides scores on eight sub-scales, reflecting functioning over the month preceding the assessment. The SF–36 has been well validated in populations with coronary heart disease (Failde & Ramos, 2000; Morrin et al, 2000; Rumsfeld & Ho, 2005). We deliberately chose a generic measure of HRQoL, as opposed to a cardiac-specific measure, as our main outcome; people after myocardial infarction frequently experience multiple health problems, and we wanted a measure that was sensitive to the impact of non-cardiac illnesses on HRQoL. A cardiac-specific measure of HRQoL would be less sensitive to such non-cardiac illnesses.

We used the SF–36 Physical Component Score (PCS) as the primary outcome measure, because it has greater sensitivity to change over the original eight scales of the SF–36, involves fewer statistical comparisons and eliminates floor and ceiling effects (Ware et al, 1994, 1995). The PCS was derived from four sub-scales (physical function, role limitation, physical pain and health perceptions), using the method described by Ware and colleagues (Ware et al, 1994). The PCS is standardised to the general population (mean score, 50: standard deviation, 10), with high scores representing higher levels of functioning. A change on the PCS of between 3 and 5 (equivalent to 0.3 to 0.5 of the standard deviation) is considered to be clinically significant (Beck et al, 2001). We did not use the mental component score as an outcome measure, as its items overlap with those of

the HADS; however, we did use the SF–36 energy and vitality sub-scale as a measure of fatigue, for the purposes of the mediator analyses.

Follow-up assessments

The HADS, Illness Perceptions and SF–36 questionnaires were mailed to our study participants at 6 and 12 months following myocardial infarction. At 12 months, participants were asked whether they experienced chest pain and, if appropriate, whether they had returned to work. They were also asked to complete the Seattle Angina Questionnaire (Spertus et al, 1995). This 19-item self-rated questionnaire assesses cardiac health status as perceived by the patient on 5 scales: physical limitations owing to chest pain, anginal stability, anginal frequency, treatment satisfaction and perceived burden of cardiac illness.

Participants were also asked to give details of hospital attendances during follow-up period (dates, hospitals attended and nature of health problem). Hospital attendances were verified by inspection of the appropriate hospital records, during which we confirmed whether participants had experienced any significant cardiac events during the 12 months following the myocardial infarction. These were defined as admissions to hospital or visits to accident and emergency departments with an acute cardiac problem, confirmed by investigations, e.g. chest pain associated with acute changes on echocardiogram. Both angioplasty and coronary artery bypass graft following an acute deterioration in cardiological state during the follow-up period were included.

Statistics

We used hierarchical multiple regression techniques to identify, at baseline and 6 months, variables that contributed to SF–36 PCS at 12 months after myocardial infarction, which was the dependent variable. The following independent variables were entered in blocks in the order presented below.

Block 1 (socio-demographic factors): age, gender, marital status (widowed, divorced, separated or other), education (more or less than 12 years), socio-economic status and baseline SF–36 PCS.

Block 2 (comorbid, non-cardiac illnesses): current rheumatic, respiratory, gastrointestinal or other disorder (including

neurological, renal, cerebrovascular disease or endocrine disorder other than diabetes).

Block 3 (cardiac factors): ongoing risk factors for further coronary heart disease (diabetes, serum cholesterol, hypertension, previous cardiac disease, previous or current smoking v. never), measures of severity of index myocardial infarction (Killip class, CPK), site of infarction, use of thrombolysis or surgical intervention during index admission, cardiac medication on discharge and cardiac events during subsequent 12 months.

Block 4 (anxiety and depression): HADS anxiety and depression scores at baseline.

This analysis was then repeated using block 4 depression and anxiety scores recorded 6 months after infarction, in place of the same scores measured at baseline.

Collinearity diagnostics were performed and showed that, although the independent variables were associated, the degree of association was not so great as to threaten the reliability of the regression analyses (variance inflation factors all <2.5, tolerances all >0.4) (Field, 2000).

In order to check that we had adjusted adequately for cardiac function, the analyses were repeated including the subgroup of 217 participants who had undergone echocardiogram, with left ventricular ejection fraction (categorised as <30%, 30–50% and >50%) entered into the regression equation in place of Killip class. In addition, we assessed whether chest pain, fatigue (energy and vitality sub-scale of the SF–36), illness perceptions or self-rated cardiac health status at 12-month follow-up could act as possible mediators of any association between depression/anxiety and SF–36 PCS at 12 months. These potential mediators were added after

block 3, to see whether the subsequent contribution of depression/anxiety to the model was reduced, according to the method of Baron & Kenny (1986).

Statistics were performed using the Statistical Package for the Social Sciences for Windows, version 11.5.0 (2002).

RESULTS

Of the 357 eligible patients, 314 (88%) gave informed consent, underwent baseline assessments and were included in the study. The mean age was 57.6 (s.d.=11.2) years and 199 (63%) were men. Site of myocardial infarction was recorded in 297, of whom 118 (37.6%) had an anterior infarction, and the majority did not have signs of cardiac failure after the infarction (Killip class I, n=230, 73.2%). On index admission, 230 patients (73.2%) received thrombolysis and 27 (8.6%) underwent a surgical intervention.

In all, 199 participants (63%) had comorbid, non-cardiac illnesses, most commonly rheumatic (32%), respiratory (15%) or gastrointestinal disorders (13%). The HADS depression score at baseline was 4.9 (s.d.=4.0), and anxiety score was 7.4 (s.d.=4.9). The mean SF–36 PCS score at baseline was 43.2 (s.d.=12.3).

Follow-up assessment

At 6 months, follow-up data were collected for 268 participants. Mean HADS depression score at 6 months was 5.6 (s.d.=4.0) and mean anxiety score was 7.7 (s.d.=4.6). Mean SF–36 PCS score was 39.0 (s.d.=11.3).

At 12-month follow-up, 16 patients had died and follow-up data were collected

from 260 (83% of original sample; 87% of survivors). Participants who did not complete follow-up were significantly younger than those who did, i.e. 53.3 (s.d.=10.4) v. 57.9 (s.d.=11.3) years, respectively, more likely to have low socio-economic status (73.7% v. 54.3%, respectively), and less likely to have received aspirin (90% v. 96%, respectively). There were no statistically significant differences in other demographic variables, medical history, severity of myocardial infarction, depression and anxiety scores or SF–36 PCS between those who completed assessments and those who dropped out of the study.

Mean HADS depression score at 12 months was 5.4 (s.d.=4.2) and mean anxiety score was 7.5 (s.d.=4.7). The mean SF–36 PCS score at 12-month follow-up was 40.4 (s.d.=11.9); participants who were depressed at this time had lower PCS scores than those who were not, i.e. mean scores=32.7 (s.d.=9.9) v. 43.8 (s.d.=11.1), respectively, t=7.8, $P<0.0005$.

At the time of their myocardial infarction, 137 patients were employed (part time or full time). Their mean PCS at baseline (adjusted for age and gender) was 48.7 (s.e.m.=0.86) compared with 31.8 (s.e.m.=1.9) for those on sick leave (n=29, F=43.0, $P<0.0005$). Of those employed at baseline, 3 died during the subsequent 12 months, and we collected complete follow-up data on 100 of the remainder (75%). For the 66 who had returned to work at 12-month follow-up, the mean PCS at follow-up (adjusted for the variables that predicted PCS at 12 months – listed in Table 1) was 46.5 (s.e.m.=1.3) and for the 30 who had not returned to work, it was 39.5 (s.e.m.=1.7, F=9.1, P=0.003).

Table 1 Regression model: predictors of Medical Outcomes Study SF–36 physical component score 12 months[1] after myocardial infarction

Independent	Cumulative adjusted R^2	Significance of change (P)	Variables included in block	Unstandardised coefficients (s.e.)	Standardised beta	t	P
Block 1	0.21	<0.0005	Age in years	−0.23 (0.07)	−0.21	−3.47	0.001
			Baseline physical component score	0.17 (0.06)	0.18	2.86	0.005
Block 2	0.25	0.001	Gastrointestinal problems	−4.37 (1.88)	−0.12	−2.32	0.021
			Rheumatic disease	−3.91 (1.46)	−0.15	−2.68	0.008
			Other medical problems	−4.06 (1.70)	−0.13	−2.39	0.018
Block 3	0.30	0.025	Smoking	−4.44 (1.60)	−0.15	−2.77	0.006
			Site of myocardial infarction	−3.01 (1.42)	−0.12	−2.12	0.04
			Cardiac events during follow-up	−4.16 (1.87)	−0.12	−2.22	0.028
Block 4	0.39	<0.0005	Depression at 6 months	−0.67 (0.23)	−0.21	−2.91	0.004
			Anxiety at 6 months	−0.42 (0.20)	−0.15	−2.09	0.037

1. SF–36 physical component score at 12 months=dependent variable. Non-significant variables have been excluded from this table for clarity.

Predictors of SF–36 PCS at 12-month follow-up

The variables that were independently associated with low SF–36 PCS at 12-month follow-up are shown in Table 1. Addition of the cardiac variables (block 3) increased the proportion of the variance accounted for (adjusted R^2) by 5%, and further addition of depression and anxiety scores at 6 months (block 4) increased the adjusted R^2 by a further 9% (F-change=10.5, $P < 0.0005$). Anxiety and depression measured at baseline and reflecting patient mood in the weeks before first myocardial infarction did not contribute significantly to the regression model after controlling for demographic and medical details (F-change=0.89).

Repeating the above analyses using left ventricular ejection fraction in place of Killip class (n=217) did not change our findings. Depression and anxiety at 6 months continued to contribute significantly to PCS at 12 months (adjusted R^2 attributable to block 4=11%, F-change=18.6, $P < 0.0005$).

Assessment of potential mediator variables

Reported chest pain, SF–36 energy and vitality scores, Illness Perceptions Questionnaire and Seattle Angina Questionnaire scores at 12 months were potential mediators, as they all showed significant univariate associations with both depression and anxiety at baseline and 6 months and with PCS at 12 months after myocardial infarction.

When chest pain was entered as a potential mediator, the R^2 for depression and anxiety at 6 months decreased from 9% to 5.0%, but the R^2 attributable to depression and anxiety remained significant at $P < 0.0005$. When SF–36 energy and vitality score was added, the R^2 for depression and anxiety decreased to 0% (P=0.37). Addition of the Illness Perceptions Questionnaire sub-scores, either singly or together, reduced the R^2 that was independently attributable to the depression and anxiety measures, although in all models the contributions of depression and anxiety remained significant ($P \leqslant 0.01$). Similarly, addition of the scores from the Seattle Angina Questionnaire reduced the magnitude of the R^2 attributable to anxiety and depression, although it remained significant (all $P \leqslant 0.002$).

The effects of changing depression on SF–36 PCS

To clarify the effect of depression severity changing between baseline and 12-month follow-up, we compared changes in the SF–36 PCS of 38 participants who were not depressed at baseline but who became depressed during follow-up, with those of 143 participants who remained undepressed throughout the study period, using HADS total of $\geqslant 17$ to indicate depression. The HADS depression scores of these two groups at baseline were 3.2 (2.2) and 2.7 (2.5), respectively, (t=−1.3, P=0.2), confirming the non-depressed status of both groups at baseline. There was no significant difference in SF–36 PCS at baseline between these two groups: scores were 42.1 (12.8) and 45.5 (11.5), respectively (t=1.6, P=0.12). The HADS depression score at 12 months for individuals who became depressed was 10.0 (3.2) whereas for those who remained non-depressed it was 3.0 (2.2), (t=−15.6, $P < 0.0005$). The SF–36 PCS for these groups were 30.9 (7.4) and 44.1 (10.7) (t=8.8, $P < 0.0005$).

DISCUSSION

We have shown that depression and anxiety recorded 6 months after first myocardial infarction predicted impairment in the physical aspects of HRQoL at 12 months after the infarction. This adverse impact of depression is additional to the effect on HRQoL of the myocardial infarction itself, ongoing angina and cardiac events during the follow-up period. Depression and anxiety in the period immediately preceding the first myocardial infarction did not predict the physical aspects of HRQoL 12 months later, however. Thus our hypothesis was partly proved.

Our method has a number of strengths, according to the quality criteria used in the systematic review of Sørensen et al (2005). First, we screened a consecutive series of patients presenting with first myocardial infarction, and managed to recruit and follow up a high proportion of eligible individuals. Second, we included only people who had not experienced previous myocardial infarction, to avoid any confounding effect of previous infarction on the association between depression and later impairment of HRQoL. Third, we used valid and widely recognised measures of depressive symptoms and HRQoL that were appropriate

for our population, and we used the same measures over three waves of assessment. In our regression analyses, we controlled for a wider range of potential confounders than any previous study, including the effects of cardiac and non-cardiac illness, and for baseline SF–36 PCS before assessing the contribution of anxiety and depression. Furthermore, we assessed rigorously the cardiac status of participants, particularly with regard to the severity of myocardial infarction, using a range of clinical and laboratory techniques. Thus we can be confident that our assessment of the contribution of anxiety and depression to subsequent HRQoL was conservative.

Our study has some weaknesses. Our assessments of depression and HRQoL were retrospective, making them vulnerable to recall bias. However, we had validated the HADS administered in this way against a standardised psychiatric interview that asked in detail about each depressive symptom (sleep loss, appetite difficulty, mood, etc.) for the month before the myocardial infarction (Dickens et al, 2004b). We were also able to show the validity of the SF–36 in relation to employment status. Furthermore, our findings are supported by those of a truly prospective study that showed that onset of coronary heart disease led to deterioration of physical function and role performance, when it was associated with increased anxiety and depression (van Jaarsveld et al, 2002).

Psychiatrists will be familiar with the impairment in the mental component of the SF–36 that occurs with depression, but we have shown that when depression is concurrent with physical illness there is also a reduction of the physical component. The low SF–36 PCS score of participants who were depressed at 12 months represented very marked impairment, with a mean score one standard deviation lower (more impaired) than that for non-depressed participants and nearly two standard deviations lower than the score for the general population.

We have shown previously that fatigue is associated with depression in patients presenting with myocardial infarction (McGowan et al, 2004). This current report indicates that this fatigue mediated the observed association between depression and HRQoL. There are a number of mechanisms by which fatigue might be associated with impairment in HRQoL. Fatigue may occur as the direct result of depression,

and may be mistaken for a symptom of on-going cardiac problems, thus leading to impaired HRQoL by virtue of increasing symptom load. Furthermore, fatigue following myocardial infarction may lead to deconditioning (or possibly, failure to rehabilitate) through reduced tolerance of physical activity, with consequent loss of physical function (Lavoie *et al*, 2004).

It is interesting that depression and anxiety that preceded myocardial infarction did not predict impairment in HRQoL 12 months following infarction, whereas depression and anxiety recorded 6 months after infarction did so. We have shown previously, however, that there are considerable changes in depression scores over the period following myocardial infarction; 45% of those depressed at baseline improved during the 12 months following infarction, whereas 21% of those not depressed at baseline developed depression during follow-up (Dickens *et al*, 2004a). It is possible that these changes in depression reduced the effect of pre-infarction depression on HRQoL 12 months after infarction in our regression analyses.

Our findings have considerable implications for clinical practice. Although depression is common following myocardial infarction, the people recruited for our study very rarely attracted treatment. This may represent hesitancy on the part of general practitioners and other doctors to prescribe antidepressants after myocardial infarction, even though this is generally safe (Glassman *et al*, 2002). It is more likely that depression is rarely recognised and treated in the physically ill, even though such treatment is effective (Gill & Hatcher, 1999).

Psychiatrists should be prepared to treat, or advise general practitioners to treat, depression using antidepressant and cognitive–behavioural therapy. Our findings indicate the importance of detecting and treating depression and anxiety, since it would be expected that reducing depression and anxiety would lead to improved quality of life and reduction in days of restricted activity and days missed from work (Kisely & Simon, 2005). This remains to be demonstrated in a clinical trial, however (Simon *et al*, 1998).

ACKNOWLEDGEMENTS

REFERENCES

Baron, R. M. & Kenny, D. A. (1986) The moderator–mediator variable distinction in social psychological research: conceptual, strategic, and statistical considerations. *Journal of Personality and Social Psychology*, **51**, 1173–1182.

Barth, J., Schumacher, M. & Herrmann-Lingen, C. (2004) Depression as a risk factor for mortality in patients with coronary heart disease: a meta-analysis. *Psychosomatic Medicine*, **66**, 802–813.

Beck, C. A., Joseph, L., Belisle, P., et al (2001) Predictors of quality of life 6 months and 1 year after acute myocardial infarction. *American Heart Journal*, **142**, 271–279.

Blumenthal, J. A., Lett, H. S., Babyak, M. A., et al (2003) Depression as a risk factor for mortality after coronary artery bypass surgery. *Lancet*, **362**, 604–609.

Brown, N., Melville, M., Gray, D., et al (1999) Quality of life four years after acute myocardial infarction: short form 36 scores compared to a normal population. *Heart*, **81**, 352–358.

Dickens, C. M., Percival, C., McGowan, L., et al (2004a) The risk factors for depression in first myocardial infarction patients. *Psychological Medicine*, **34**, 1083–1092.

Dickens, C. M., McGowan, L., Percival, C., et al (2004b) Lack of a close confidant, but not depression, predicts further cardiac events after myocardial infarction. *Heart*, **90**, 518–522.

Failde, I. & Ramos, I. (2000) Validity and reliability of the SF–36 Health Survey Questionnaire in patients with coronary artery disease. *Journal of Clinical Epidemiology*, **53**, 359–365.

Field, A. (2000) *Discovering Statistics using SPSS for Windows*. London: Sage Publications.

Gill, D. & Hatcher, S. (1999) A systematic review of the treatment of depression with antidepressant drugs in patients who also have a physical illness. *Journal of Psychosomatic Research*, **47**, 131–143.

Glassman, A. H., O'Connor, C. M., Califf, R. M., et al (2002) Sertraline treatment of major depression in patients with acute MI or unstable angina. *JAMA*, **288**, 701–709.

Goldthorpe, J. H. & Hope, K. (1974) *The Social Grading of Occupations. A New Approach and Scale*. London: Oxford University Press.

Gutzwiller, F. (1994) Monitoring of cardiovascular disease and risk factor trends: experiences from the WHO/MONICA project. *Annals of Medicine*, **26**, 61–65.

Herrmann, C. (1997) International experiences with the Hospital Anxiety and Depression Scale — a review of validation data and clinical results. *Journal of Psychosomatic Research*, **42**, 17–41.

Herrmann, C., Brand, D. S., Kaminsky, B., et al (1998) Diagnostic groups and depressed mood as predictors of 22-month mortality in medical inpatients. *Psychosomatic Medicine*, **60**, 570–577.

Izawa, K., Hirano, Y., Yamada, S., et al (2004) Improvement in physiological outcomes and health-related quality of life following cardiac rehabilitation in patients with acute myocardial infarction. *Circulation Journal*, **68**, 315–320.

Killip, T. & Kimball, J. T. (1967) Treatment of myocardial infarction in a coronary care unit: a two year experience with 250 patients. *American Journal of Cardiology*, **20**, 457–464.

Kim, J., Henderson, R. A., Pocok, S. J., et al (2005) Health-related quality of life after interventional or

conservative strategy in patients with unstable angina or non-ST-segment elevation myocardial infarction: one-year results of the third Randomized Intervention Trial of unstable Angina (RITA-3). *Journal of the American College of Cardiology*, **45**, 221–228.

Kisely, S. & Simon, G. E. (2005) An international study of the effect of physical ill health on psychiatric recovery in primary care. *Psychosomatic Medicine*, **67**, 116–122.

Lavoie, K. L., Fleet, R. P., Lesperance, F., et al (2004) Are exercise stress tests appropriate for assessing myocardial ischemia in patients with major depressive disorder? *American Heart Journal*, **148**, 621–627.

Mayou, R. A., Gill, D., Thompson, D. R., et al (2000) Depression and anxiety as predictors of outcome after myocardial infarction. *Psychosomatic Medicine*, **62**, 212–219.

McBurney, C. R., Eagle, K. A., Kline-Rogers, E. M., et al (2002) Health-related quality of life in patients 7 months after myocardial infarction: factors affecting the Short Form-12. *Pharmacotherapy*, **22**, 1616–1622.

McGowan, L., Dickens, C., Percival, C., et al (2004) The relationship between vital exhaustion, depression and comorbid illnesses in patients following first myocardial infarction. *Journal of Psychosomatic Research*, **57**, 183–188.

Morrin, L., Black, S. & Reid, R. (2000) Impact of duration in a cardiac rehabilitation program on coronary risk profile and health-related quality of life outcomes. *Journal of Cardiopulmonary Rehabilitation*, **20**, 115–121.

Petrie, K. J., Weinman, J., Sharpe, N., et al (1996) Role of patients' views of their illness in predicting return to work and functioning after myocardial infarction: a longitudinal study. *BMJ*, **312**, 1191–1194.

Petrie, K. J., Cameron, L. D., Ellis, C. J., et al (2002) Changing illness perceptions after myocardial infarction: an early intervention randomized controlled trial. *Psychosomatic Medicine*, **64**, 580–586.

Pocock, S. J., Henderson, R. A., Clayton, T, et al (2000) Quality of life after coronary angioplasty or continued medical treatment for angina: three-year follow-up in the RITA-2 trial. Randomized Intervention Treatment of Angina. *Journal of the American College of Cardiology*, **35**, 907–914.

Poston, W. S., Haddock, C. K., Conard, M. W., et al (2003) Assessing depression in the cardiac patient. When is the appropriate time to assess depression in the patient undergoing coronary revascularization? *Behaviour Modification*, **27**, 26–36.

Rugulies, R. (2003) Depression as a predictor of coronary heart disease: a systematic review and meta-analysis of the literature. *American Journal of Preventative Medicine*, **23**, 51–61.

Rumsfeld, J. S. & Ho, P. M. (2005) Depression and cardiovascular disease: a call for recognition. *Circulation*, **111**, 250–253.

Simon, G. E., Katon, W., Rutter, C., et al (1998) Impact of improved depression treatment in primary care on daily functioning and disability. *Psychological Medicine*, **28**, 693–701.

Sørensenf, C., Friis-Hashe, E., Haghfelt, T., et al (2005) Postmyocardial infarction mortality relation to depression: a systematic critical review. *Psychotherapy and Psychosomatics*, **74**, 69–80.

Spertus, J. A., Winder, J. A., Dewhurst, T. A., et al (1995) Development and evaluation of the Seattle Angina Questionnaire: a new functional status measure for coronary artery disease. *Journal of the American College of Cardiology*, **25**, 333–341.

Taira, D. A., Seto, T. B., Ho, K. K., et al (2000) Impact of smoking on health-related quality of life after

percutaneous coronary revascularization. *Circulation*, **102**, 1369–1374.

van Jaarsveld, C. H., Sanderman, R., Ranchor, A. V., et al (2002) Gender-specific changes in quality of life following cardiovascular disease: a prospective study. *Journal of Clinical Epidemiology*, **55**, 1105–1112.

van Melle, J. P., de Jonge, P., Spijkerman, T. A., et al (2004) Prognostic association of depression following myocardial infarction with mortality and cardiovascular events: a meta-analysis. *Psychosomatic Medicine*, **60**, 814–822.

Ware, J. E. & Sherbourne, C. D. (1992) The MOS 36 item short-form health survey (SF–36). I. Conceptual framework and item selection. *Medical Care*, **30**, 473–483.

Ware, J. E., Kosinski, M., Keller, S. (1994) *SF–36 Physical and Mental Health Summary Scales: a User's Manual* (2nd edn). Boston, MA: The Health Institute.

Ware, J. E., Kosinski, M., Baylis, M. S., et al (1995) Comparison of methods for the scoring and statistical analysis of SF–36 health profile and summary measures: summary of results from the Medical Outcomes Study. *Medical Care*, **33**, 264–279.

Weinman, J., Petrie, K. J., Moss–Morris, R., et al (1996) The Illness Perception Questionnaire: a new method for assessing the cognitive representation of illness. *Psychology and Health*, **11**, 431–445.

Wulsin, L. R. & Singal, B. M. (2003) Do depressive symptoms increase the risk for the onset of coronary disease? A systematic quantitative review. *Psychosomatic Medicine*, **65**, 201–210.

Zigmond, A. S. & Snaith, R. P. (1983) The Hospital Anxiety and Depression Scale. *Acta Psychiatrica Scandinavica*, **67**, 361–370.

CHRIS M. DICKENS, PhD, LINDA McGOWAN, PhD, CAROL PERCIVAL, PhD, BARBARA TOMENSON, MSc, Department of Psychiatry, Manchester University, UK; LAWRENCE COTTER, MD, ANTHONY HEAGERTY, MD, Department of Cardiology, Manchester Royal Infirmary, UK; FRANCIS H. CREED, MD, Department of Psychiatry, Manchester University, UK

Correspondence: Chris Dickens, Department of Psychiatry, Rawnsley Building, Manchester Royal Infirmary, Oxford Road, Manchester M13 9WL, UK. Tel: +44 (0)161 276 5386; fax: +44 (0)161 273 2135; email: chris.dickens@manchester.ac.uk