Coronary artery disease, depression and social support: only the beginning

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In this issue, Horsten and colleagues[1] report that symptoms of depression and lack of social integration 3–6 months after discharge for an acute coronary syndrome have independent negative impacts on 5-year prognosis in women 65 years of age and younger. Although finding increased risks associated with depression or with low social support are not new, studies examining the joint impact of depression and measures of social support in cardiac patients are rare.

Of all the negative emotions, including depression, anxiety and hostility, that have been hypothesized to influence outcomes in patients with established coronary artery disease, the epidemiological findings are most consistent for depression[2–7]. Research in post-myocardial infarction samples has documented that depression increases the risk of mortality from two to seven times, depending on the sample characteristics and the duration of follow-up[4–7]. We recently observed similar increases in the combined risk of cardiac mortality and myocardial infarction in unstable angina patients[8]. Post-hoc analyses of several large longitudinal data sets also suggest that depression is associated with the incidence of coronary artery disease in initially healthy individuals[9–12].

What kind of depression increases the risk of cardiac events, and what type of events are involved? The study by Horsten and colleagues[1] suggests an increase in the combined risk of revascularization, cardiac mortality, and myocardial infarction in women reporting two or more out of nine depression symptoms. Most previous research highlighting depression-related risks for mortality and myocardial infarction in coronary artery disease patients has found the risk to be concentrated in the approximately 25 to 30% of patients with the highest level of depression. However, in this study the risk did not increase beyond two symptoms, the level reported by 72% of the women. Two symptoms of depression is...
not the equivalent of major or even minor depression, and is not a condition for which current psychiatric guidelines recommend treatment. The question may not be what a normal level of depressive symptoms is, but at what threshold medical attention is needed. The answer is dependent both on the magnitude of the increased risk, and the costs and benefits of treatment.

There were too few deaths and infarctions over 5 years of follow-up in this sample to assess the link between depression and hard cardiac events. From the data provided, it appears that there were, at most, 26 deaths or myocardial infarctions among the 81 patients with one of the combined events. The risk in this study was primarily a risk of revascularization. What is the clinical significance of these findings? We need to consider what revascularization represents: evidence of myocardial ischaemia with a coronary anatomy amenable to angioplasty or bypass surgery. After hospital discharge what is the process that generates this evidence? In the usual situation, patient complaints of angina or routine procedures to detect residual ischaemia, including exercise stress tests or nuclear medicine evaluations, lead to angiographic assessment. However, in the study by Horsten et al. there was another potential pathway. Some 131 of the 292 patients took part in a quantitative angiography study within 3 months following their baseline interview. According to an earlier report of the study published in this journal in 1998 all angiographies were carried out based on the study protocol i.e. primarily because the patient was a participant in the FemCorRisk study. Thus, the procedures were done on an 'availability basis' rather than based on clinical factors. Although the authors do not describe what happened to patients with significant stenoses, the 1998 report indicates that 53 patients had at least one coronary artery obstructed by 50% or more. It is likely that many of them became the revascularized patients in the present analyses. In this context, it seems that middle-aged women who report less than two depression symptoms, 3–6 months after an admission for an acute coronary syndrome are unlikely to have significant stenoses requiring revascularization. However, it is unclear whether this is a chicken or egg phenomenon. By this point, how many of these women knew or suspected that they were in line for a procedure because of symptoms of residual ischaemia? The authors found that the severity of angina symptoms was highly linked to depression ($P=0.0003$). However, control for angina class did not alter the relationship between depression and their combined end-point. In summary, these results suggest that if a woman coronary artery disease patient aged 65 or younger reports more than one depression symptom, thorough evaluation for revascularization should be undertaken regardless of her angina level.

In addition to their results for depression, the authors also observed a statistically significant increase in risk with decreasing levels of social integration, the degree to which an individual is interconnected with others and has multiple contacts in the family and community. Although women with two or more depression symptoms reported significantly lower levels of this sort of social network contacts, the risk associated with low social integration was present for both depression groups, and so was independent of depression. Previous work by the investigators with middle-aged men free from cardiac disease at baseline demonstrated that social integration predicted myocardial infarctions and coronary deaths during the subsequent 6-year period. So social integration predicts the incidence of cardiac disease in middle-aged men, and revascularization and cardiac events in middle-aged women with already established coronary artery disease. However, in their 1998 quantitative angiography study, involving a subsample of the current study patients, the authors also found that major coronary stenoses were significantly more common in women with low social integration. Thus, it makes sense that the combined end-point of revascularization, myocardial infarction and cardiac deaths would also be linked to low social integration in the full sample.

In comparison, the authors did not find much impact of decreasing levels of what they term 'attachment' on combined outcomes in women with coronary artery disease ($P=0.14$). Again this parallels their 1998 results with coronary blockages. However, their previous work with men initially free of coronary disease found that attachment acted much like social integration to predict hard cardiac events. Their measure of attachment assessed the availability of a close special relationship with one or more people. As the authors suggest, it seems plausible that the lack of stronger findings is due to the scale's very limited range (only 6 points) with the data skewed towards higher scores, possibly because of social desirability response bias.

By what mechanisms might social integration or attachment influence coronary stenoses and via them affect cardiac events and revascularization? The literature suggests two major models to explain the impact of social support: the main effect and the stress-buffering models. In the main effect model, social support has an impact on disease outcomes regardless of other physical or psychosocial risks. This is what the authors observed with depression: integration had an impact independent of depression. In fact, most of the evidence supporting this type of
model has come from studies that assess social integration. There are several plausible explanations. First, people with more inter-connections with the community tend to have healthier lifestyles. In the current study, smoking was less common with higher integration. Second, when people begin to become ill they may reduce their contacts with others because of lower energy levels so that lower social integration may be a proxy measure of greater illness severity. However, control for measures of cardiac risk did not explain the link observed by the authors. It has also been hypothesized that social integration has its impact on cardiac outcomes because integrated patients may be more likely to receive medical treatment, or be more motivated to alter risk factors. In contrast to the main effect model of social support, the stress-buffering model suggests that social support is beneficial only in the presence of ‘high stress’ or high psychological risk. This model is primarily supported by measures of how individuals view their close social support, essentially what was not adequately measured by the attachment scale in the current study. If we consider that depression is a ‘high stress’ situation, the stress buffering model suggests that perceived social support should act to buffer the impact of depression on cardiac events, that is, if properly measured, depression and attachment should interact to predict cardiac outcomes. Although the authors did not report testing this possibility, we recently examined this relationship in a sample of 887 men and women following myocardial infarction[16]. We found that while perceived social support was not directly related to 1-year survival, studies of coronary artery disease patients, the search for the mechanisms has just begun. While there is as yet no scientific proof that intervening on these factors will improve prognosis, the epidemiological data should finally be sufficiently strong to intrigue all of us and trigger a surge of new research.

N. FRASURE-SMITH
F. LESPÉRANCE
McGill University and University of Montreal, Montreal, Quebec, Canada

References