Psychological Risk Factors for Cardiac Events
Could There Be Just One?

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There is abundant evidence that depression, anxiety, and anger increase the risk for cardiac events in patients with coronary heart disease. Denollet and Brutsaert are to be commended for contributing a generally well-conducted study of psychological predictors of cardiac end points to this rapidly growing literature. They have found that the combination of "negative affectivity" and social inhibition predicts cardiac events independently of established medical risk factors. Prospective studies such as theirs, in which potential confounders are adequately measured and cardiac end points carefully documented, are needed to substantiate the effects of psychological factors on medical outcomes after acute myocardial infarction (MI).

Unfortunately, the authors may have gone beyond their data in asserting that they have identified a personality trait that both predicts cardiac events and explains why such disparate mood states as depression, anxiety, and anger predict cardiac events as well. It is not at all clear that they have substantiated this claim or that it is time to abandon research on depression, anxiety, and anger in favor of this personality trait.

Absent from the authors’ discussion is any reference to an established theory of personality. It is by no means universally accepted among personality theorists that a single trait accounts for every negative mood state (cf Cloninger et al) or that the combination of negative affectivity and social inhibition defines a persistent “distressed personality type.” In this light, it makes more sense to conclude simply that patients with coronary heart disease who are both emotionally distressed and socially inhibited are at greater risk for cardiac events than are patients without both characteristics. This more conservative interpretation does not diminish the importance of the study or the need for further research on the relationship between social inhibition and emotional distress in cardiac patients.

The authors’ argument that the distressed personality type accounts for the relationships between cardiac events and depression, anxiety, and anger rests, in large part, on the finding that negative affectivity and social inhibition remained in a multiple regression model after the negative mood state variables dropped out. Unfortunately, this is not particularly convincing evidence, because the statistical power of the model was too low to test the hypothesized relationships among personality, mood state, and cardiac events.

Furthermore, the measures of negative mood states used in this study were not the same ones that have been found in other studies to predict post-MI mortality. For example, most previous studies of depression as a predictor of cardiac mortality have either used standardized clinical interviews or well-validated questionnaires such as the Beck Depression Inventory or the Zung Depression Scale. Instead of one of these established depression measures, the authors used the Pessimism and Despair scales from the Millon Behavioral Health Inventory as indicators of depression. Pessimism and despair may be common features of depression, but they are not equivalent to depression. In fact, these two Millon scales correlate better with the authors’ measure of negative affectivity than they do with the Beck Depression Inventory.

Even if a single personality factor were found to account for a variety of negative mood states in post-MI patients, it would not necessarily do so for clinical depression, a known predictor of cardiac events. The authors argue that this is unimportant because depressed mood, even in the absence of a clinical depressive disorder, increases the risk for the post-MI period. They cite the study by Frasure-Smith et al to support this position, but this is somewhat misleading. Although Frasure-Smith and colleagues did find that the number and severity of depressive symptoms (as measured by the Beck Depression Inventory) was a better predictor of 18-month mortality than was a diagnosis of major depression, their previous report showed that major depression was the better predictor of 6-month mortality. Furthermore, they found that patients who were free of major depression at index but who nevertheless had high Beck Depression Inventory scores were at high risk of developing major depression during the follow-up period. This is similar to our study of patients with stable coronary heart disease, in which nearly half of those who had minor depression at index developed major depression in the course of the following 12 months. In short, some post-MI patients with depressed mood or minor depression will subsequently develop major depression, and others will not. We do not yet know whether the dysphoric or mildly depressed patients who do not go on to develop major depression are at any higher risk for mortality than are otherwise comparable nondepressed patients.

It may or may not be easier for clinicians to assess a single personality factor than three separate mood states. However, personality factors are, by definition, much more stable and much more difficult to change than are mood states such as depression or anxiety. There are well-established, short-term psychotherapeutic and pharmacological treatments for the most common forms of depression, anxiety, and pathological...
anger as well as social inhibition. Thus, even if it were easier to assess personality traits than mood states, this advantage would be thoroughly offset by the challenge of changing the patient’s personality.

The authors have attempted to bypass this obstacle by proposing clinical trials of “comprehensive treatment programs” aimed not at changing personality but rather at reducing general emotional distress. However, two recent randomized trials cast doubt on the wisdom of that approach. These studies found that nonspecific interventions for general distress failed to improve survival in post-MI patients and may even have decreased survival in female patients.

Finally, it is not clear that all negative emotions affect all patients and all cardiac end points in the same way or to the same degree. For example, although numerous studies have established a link between hostility and anger and the development of coronary artery disease, there is little evidence that anger has prognostic significance after an MI. As another example, chronic, low- to moderate-intensity anxiety, such as that seen in generalized anxiety disorder, may have different effects on both cardiac physiology and cardiac end points than transient, high-intensity anxiety such as that seen in phobias and panic disorder. Preserving such distinctions is essential if we are to identify the mechanisms that underlie the relationships between these mood states and cardiac end points. Identification of the specific mechanisms is, in turn, necessary if we are to refine our ability to identify and treat patients at risk.

The history of research on cholesterol as a cardiac risk factor provides an instructive analogy. If lipid researchers had studied only total serum cholesterol to the exclusion of specific cholesterol fractions, much less would have been learned about the mechanisms by which cholesterol contributes to atherosclerosis or about how to most effectively treat hypercholesterolemia.

Again, Denollet and Brutsaert are to be commended for their work. Their findings should stimulate a much-needed debate about the direction for future research in this area. However, I submit that it is too early to abandon research on specific mood states in favor of a single personality trait. Studies of the effects of specific mood states on specific cardiac end points are still needed. Whether successful treatment of depression, anxiety, or pathological anger can reduce cardiac event rates is still unknown. Clinical trials could help answer this question, but only if these mood states are adequately characterized.

References


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