Psychological Factors and Ischemic Heart Disease

To the Editor:

Rozanski et al., in discussing the role of psychological factors in inducing ischemic heart disease (IHD), indicated that “the pathophysiological mechanisms by which behavior therapies reduce cardiac events need to be identified.” I suggest that behavior therapies operate to prevent stress-induced IHD by countering mechanisms by which stress induces IHD.

The recently described altered homeostatic theory (AHT) asserts that mechanisms that prevent stress-induced IHD directly counter mechanisms that induce such symptoms. Importantly, the AHT is a general theory and includes multiple risk factors besides stress (such as cholesterol, smoking, and homocysteine) and multiple disorders besides IHD (such as hypertension, atherosclerosis, and stroke).

The AHT asserts that multiple risk factors for IHD and other disorders induce disease by the single action of shifting homeostasis inappropriately toward defensive fight/flight and that multiple preventative factors (such as stress reduction, vitamins, aspirin, and exercise) shift homeostasis toward health by an opposite shift of homeostasis. For IHD, chronic stress favors atherosclerosis and acute stress induces symptoms through spasm-of-resistance vessels (S-RV).

The AHT can be considered an expansion of Selye’s stress syndrome to other risk factors. Selye asserted that stress shifts homeostasis toward disease because of overactivity of the general adaptational or defensive fight/flight response. This defensive response is characterized by mechanisms that are generally similar to the mechanisms described by Rozanski et al by which psychological factors such as depression, anxiety, and social isolation favor IHD: the mechanisms described by Rozanski et al include sympathetic nervous system hyperresponsivity, expression of S-RV and a clotting tendency (S-RV/clotting), hypercortisolemia, and neurohumoral arousal.

The AHT grew mainly out of a literature review that provided evidence that risk and preventative factors for IHD and other disorders have opposite actions; major risk factors (such as stress, cholesterol, smoking, and homocysteine) express S-RV/clotting, and major preventative factors (such as vitamins, aspirin, and exercise) express the opposite combination of vasodilation-of-resistance vessels (V-RV) and an anticoagulation tendency (V-RV/anticoagulation).

The opposite actions of S-RV/clotting by risk factors and V-RV/anticoagulation by preventative factors seem consistent with risk factors causing disease by shifting homeostasis inappropriately and preventative factors improving disease by correcting this adverse shift.

Rozanski et al discussed a possible mechanism by which behavior therapies might operate, namely, improvement in coronary endothelial function. This improvement implies reduction of vasoconstrictive tendencies of resistance vessels, thus preventing S-RV. The AHT focuses on vasoconstrictive endothelial dysfunction, and the AHT is based in part on the S-RV concept, which asserts that S-RV induces symptoms in IHD.

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Response

Hellstrom suggests that altered hemostatic theory (AHT) provides a unitary explanation for ischemic heart disease (IHD) and other disease states but provides no empirical data to support this claim. The evidence we reviewed supports an alternative view, viz that diverse psychosocial factors mediated by more than 1 biological mechanism potentiate adverse outcomes, at least with respect to IHD. The attempt to subsume multiple psychosocial risk factors into overarching constructs is liable to at least 3 deficiencies. First, as illustrated by Diamond et al, the “information content” of variables (in this case, that of psychosocial stress) is generally diminished as categories of measurement are diminished. Second, the importance of individual pathophysiological mechanisms (eg, excessive sympathetic responsivity, hyperlipidemia, hypertension, and glucose intolerance) is probably outweighed by interactions between mechanisms in mediating psychosocial influences on IHD; overarching constructs tend to mask such interactions. Third, the efficacy of interventions or preventive measures is hampered by failure to distinguish component psychosocial risk factors in individual patients. For instance, it is more difficult to alter a global factor, such as stress or personality, than individual component psychosocial risk factors, such as depression or social isolation. Just as the targeted treatment of hypertension and hypercholesterolemia diminishes total IHD risk, identification and treatment of individual psychosocial risk factors may improve cardiac outcomes.

Are there useful overarching constructs? One useful concept is the notion that behaviorally evoked, excessive perturbations of the body’s principal axes of neuroendocrine response (ie, pituitary-adrenocortical or sympathetic-adrenomedullary) may produce pathophysiological consequences. This concept is not improved by adding an AHT construct. Furthermore, by focusing on the fight/flight response, AHT ignores the potential role of the pituitary adrenocortical or other stress-responsive systems, such as the renin-angiotensin system, in mediating the pathobiology of behavioral influences on disease.

Finally, in contrast to Selye’s formulation of stress as a “nonspecific response to any demand,” work by Cannon and others has shown that the body makes specific responses to specific psychosocial stressors. The hypothesis that there exists a single mechanistic pathway, as defined by the AHT, does not appear to add materially to the information we have reviewed or to the biological models that we have proposed.

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_Circulation_. 2000;101:e177-e178
doi: 10.1161/01.CIR.101.16.e177

_Circulation_ is published by the American Heart Association, 7272 Greenville Avenue, Dallas, TX 75231
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Print ISSN: 0009-7322. Online ISSN: 1524-4539

The online version of this article, along with updated information and services, is located on the World Wide Web at:
http://circ.ahajournals.org/content/101/16/e177

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