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Richard Ketai

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Richard Ketai, MD*

The importance of mind, emotion, and behavior as they relate to the heart, particularly coronary artery disease, is growing in recognition. A review of the research shows increasingly sophisticated demonstrations of links unifying heart function and mind. "Psychocardiology" would be an appropriate term for the diverse areas of interest which focus on the psychosocial correlates of heart disease. This paper illustrates this concept through a review of psychosocial and behavioral aspects of coronary artery disease. (Henry Ford Hosp Med J 1986;34:56-60)

Coronary artery disease continues to be the foremost cause of mortality in the United States, claiming over 600,000 victims each year. The incidence has been falling appreciably in the past two decades, attributable largely to an increased health consciousness among Americans regarding exercise, diet, and smoking and to steadily improving medical interventions (1).

Another frontier in the understanding and treatment of patients with coronary artery disease involves the study and application of psychosocial factors as they interface with this condition. At the simplest level it is obvious that potentially changeable self-harmful behaviors contribute significantly to the genesis of coronary morbidity. These behaviors include smoking, overeating (especially fatty foods), and physical underactivity. At a more complex level psychosocial factors have been extensively studied and implicated in a variety of ways as being contributory to morbidity from cardiac sudden death or slowly progressive coronary degeneration. This review will address the areas linking emotions, behavior, stress, and coronary artery disease.

The idea of a link between emotions and the heart is deeply embedded in human consciousness. The link is found in a myriad of symbols, especially in our vocabularies: the heart as a symbol of the feeling of love and courage, the broken-hearted lover, the caring warm-hearted person, the cruel person with a heart of stone, among many others. The heart surpasses the brain as a symbol of importance in all emotional matters and spirituality. Gantt (2) observed in respect to the psychophysiological link, "though the observed actions of men hide their thoughts and feelings, these are revealed by the observations of their hearts."

People who are somatically perceptive are sensitive to the subtle shifting sensations of cardiac rate, rhythm, and contractile force in situations of both physical and emotional arousal. An obsession with these sensations may be evidence of a so-called "cardiac neurosis." The heart may also be subject to disturbances of function, acutely or chronically, as a result of emotional duress in constitutionally vulnerable individuals. Carruthers (3) indicates that perhaps the earliest recorded example of cardiac sudden death is found in the Bible (I Samuel 25:37 [KJV]). Here, Nabal, described as a temperamental, churlish, and evil man, discovers that only his wife's intervention saved him after he insulted King David, whereupon "his heart died within him"(3). Likewise, the 18th Century English surgeon, John Hunter, who was known for his quick temper and also suffered from angina, was quoted to say "my life is in the hands of any rascal who chooses to annoy and tease me." In fact, when a colleague disagreed with him at a meeting, Dr Hunter became angered, clutched his chest, and expired (4). In 1628 William Harvey wrote "every affection of the mind that is attended with either pain or pleasure, hope or fear is the cause of an agitation whose influence extends to the heart"(5).

The mechanisms by which emotional agitations may contribute to cardiac malfunction have been widely studied. These may be considered in respect to 1) acute stress responses leading to immediate cardiac dysfunction, 2) psychosocial precursors slowly leading to increased risk of coronary degeneration, and 3) chronic behavioral-characterological traits associated to early coronary artery disease. None of these conditions is assumed or proven to be a sufficient cause for cardiac pathology in humans without additional or concommitant biological vulnerability. They may, however, be viewed as possible contributors to cardiac disease.

Acute Events

Engel, a leading spokesman for the "biopsychosocial" approach to disease, has used this model to study intense emotional arousal as an immediate precipitant of sudden death, presumably of cardiovascular origin (6). He collected hundreds of newspaper clippings that described the collapse and death of

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*Department of Psychiatry, West Bloomfield Division, Henry Ford Hospital.
Address correspondence to Dr Ketai, Department of Psychiatry, Henry Ford Hospital, 6777 W Maple Rd, West Bloomfield, MI 48033.

people in highly emotionally charged situations. The most frequent circumstances were upon hearing of the death of a loved one, exposure to threat of death or physical danger, and loss of status or self-esteem. The important common denominator in all these situations was concluded to be overwhelming exposure to major loss or threat which could not be avoided or controlled. In other work, Engel implicated the possibility of combined vasodepressor syncope and arrhythmia resulting in cardiac sudden death as a consequence of the simultaneous activation of physiologic fight-flight (sympathetic) and conservation-withdrawal (parasympathetic) responses as a form of "giving up" in the face of emotional arousal and psychologic uncertainty (7).

In another example of acute cardiac changes provoked by emotional stress, Taggart and others (8) have shown the precipitation of excessive premature ventricular contractions and tachycardia in normal persons delivering public presentations. Subjects with known coronary artery disease developed "prolific ectopic activity" and myocardial ischemia patterns on EKG in public speaking situations.

Using an animal model, Lown et al (9,10) have shown that significant lowering of the threshold for ventricular fibrillation could be induced in dogs who, before coronary ligation, had been exposed to a highly stressful environment. When reexposed to the same environment after myocardial infarction, the ventricular fibrillation threshold for these dogs fell dramatically in comparison to dogs whose coronaries were also ligated but had not been stressfully conditioned. This response could be eliminated by beta-blocking drugs and by bilateral sectioning of the sympathetic cardiac nerves. This model appears to have significant ramifications for humans recovering from myocardial infarction and subjected to excessive stress.

Another possible cause of acutely emotionally induced cardiac malfunction may be coronary artery spasm (11). In fact, Heberden (12), who first described angina pectoris, emphasized that this condition at rest was "increased by disturbances of the mind." In 1960 Prinzmetal predicted coronary spasm as an explanation for angina at rest (13). Schiffer et al (14) claim to have demonstrated inducing such spasm in angina patients solely by administering a test requiring mental effort. It would also be easy to imagine that the increased demand for oxygen in a heart accelerated by emotional arousal may cause angina in any person with sufficiently stenotic coronary arteries.

The well-known "fight-flight" response, first described by Cannon (15) in 1911, has a pronounced effect on cardiovascular functioning. This neurocardiovascular reflex in situations of perceived danger produces significantly increased blood flow to the musculature and brain at some temporary expense to perfusion of the viscera. In the process, heart contractility, heart rate, and blood pressure increase, stimulated by norepinephrine and epinephrine, release from nerve endings and adrenal glands, respectively. Clotting time is also shortened as a defense against exsanguination. High norepinephrine levels have been reported as a stimulus to platelet aggregation; furthermore, Haft and Fani (16) reported in animal research that stress may cause intraarteriolar platelet aggregation.

Acute emotional arousal has also been demonstrated to be a stimulus to transient but significant increases in plasma lipids. Dimsdale and Herd (17) have reviewed 60 studies covering a wide variety of acutely stressful situations that provoked elevations of cholesterol to as much as 65% above baseline. Free fatty acid levels were similarly elevated in such situations. Combining knowledge about physiological changes seen in acutely stressful ("fight-flight") emotional responses, the direct and indirect cardiovascular consequences are seen to be rapid rises in blood pressure, heart rate, and plasma lipids and stimulation of the clotting mechanism through platelet aggregation. In similar situations, acute changes may also include coronary spasm, myocardial ischemia, and ectopic ventricular contractions. According to Freidman (4), the hemodynamic changes of fight-flight arousal that reduce blood flow to the liver may additionally retard the rate at which the liver clears lipids from the blood. He has demonstrated sludging of red blood cells in retinal vessels during periods of blood lipid elevation; the same process might be presumed to occur in myocardial arterioles as well.

The extent to which repeated acute emotionally induced cardiovascular and hematologic disruptions ultimately contribute to progressive coronary atherosclerosis, thrombosis, or cardiac sudden death by vascular occlusion or electrical conduction abnormalities can only be a matter of hypothetical speculation at this time. Provocative potential explanations, these mechanisms are difficult to implicate clearly. Further research will help to explain the role of these possible links.

Psychosocial Correlates of Coronary Artery Disease

Numerous possible psychosocial correlates of coronary artery disease have been examined, revealing some interesting findings yet no hard data on psychopathophysiology. One provocative animal experiment revealed that rabbits fed a high cholesterol diet were significantly protected from arterial cholesterol deposition when they were handled, hand-fed, petted, and spoken to. By contrast, hypercholesterolemic rabbits left alone in their cages developed far greater cholesterol arterial plaque formation (18). Monkeys subjected to severe social stressors and uncertainty, even though normocholesterolemic and normotensive, developed marked coronary atherogenesis by comparison to an unstressed control group (19). The factors mediating between the social stressors and coronary degeneration could not be accounted for in this experiment.

In human beings, social class, work stress, social incongruities, and social isolation have been studied in relation to coronary artery disease vulnerability. Rose and Marmot (20) demonstrated that in the British civil service a man's employment status was a stronger predictor of his risk of dying from coronary artery disease than were smoking, body habitus, blood pressure, glucose tolerance, and exercise. Physical work activity was described as light in all work grades. Specifically, these researchers found that in the lowest employment grade, coronary mortality was 3.6 times higher than in the top grade over a 7.5-year period. The assumption might be made that the relative lack of autonomy and decision-making authority in the lower grades serve as stressors for many.

In more specific respect to work stress factors, Karasek et al (21) have examined the effect of different work conditions on physiological responses which may have cardiovascular im-

plications. These authors point especially to work situations in which a fast work pace is demanded in a setting of low sense of control over the task, mandatory overtime work, and when piece wage replaces regular salary. The physiological responses found in these situations include increased excretion of both epinephrine and cortisol by comparison to nonstressed control workers. In situations where the worker was subjected to a controllable and predictable stressor, cortisol excretion remained low while epinephrine alone was elevated. Elevation of cortisol is considered more a reflection of an ongoing high strain phenomenon. Epinephrine, by contrast, is more associated with a transient active behavioral response compatible with immediate coping, returning quickly to baseline. This response is not detrimental to health unless frequent and excessive. This cortisol-strain model invites consideration of cardiovascular and hematological alterations in response to steroids, namely hypertension, hyperlipidemia, and acceleration of the atherosclerotic process.

In a five-year prospective study of 1500 men initially free of overt coronary artery disease, Shekelle et al (22) studied a variety of social incongruities in relation to the later development of this disease. Incongruities pertained to changes in social status from class of origin to the present and differences between the subject's status and those of his wife. The measured variables included education, occupation, income, neighborhood, dwelling, religion, membership in organizations, and class of origin and present class of subject and wife. These researchers found a significant correlation between several of the social incongruities and development of coronary artery disease. They also found that men with four or five identifiable social incongruities from this list had 6.5 times the risk of developing coronary artery disease compared to men with no incongruities. Correlations do not necessarily imply cause and effect. They do, however, stimulate considerations of possible links or common denominators between the correlates. An interesting sidelight of this study is that no relationship was found between numbers of incongruities and known physical risk factors, which included serum cholesterol, blood pressure, blood sugar, age, weight, and cigarette smoking. In fact, social incongruities were found to have a higher correlation with later coronary artery disease than any of the usually accepted risk factors.

In a landmark prospective study, Ruberman et al (23) followed 320 male survivors of acute myocardial infarction for three years who were members of the Beta Blocker Heart Attack Trial. The subjects were questioned at the start of the study as to their self-assessment for ongoing stressful life situations and for relative degree of social isolation. At the end of three years, these investigators found a threefold to sixfold increase in mortality among subjects who had both high life stress and social isolation at the beginning of the study compared to men who rated low on these items regardless of level of education.

Coronary-Prone Behavior

An extended study that remains controversial is whether particular behavioral traits may predispose to premature coronary degeneration. An early reference to this motion was made by Sir William Osler in 1897:

> In the worry and strain of modern life, arterial degeneration is not only very common, but develops often at a relatively early age. For this, I believe that the high pressure at which men live in the habit of working the machine to the maximum capacity are responsible rather than excesses in eating and drinking (24).

Osler maintained that he could diagnose coronary artery disease by watching how the patient entered his office, the vulnerable person being "a keen and ambitious man, the indicator of whose engine is always at 'full speed ahead.'"

This style of behavior as a possible risk factor for coronary artery disease was revived as an area of intensive research by Friedman and Rosenman in the late 1950s (25). They and numerous collaborators have since published numerous papers relating to what they have come to call the "type A behavior pattern." This may be summed up as a description of a person who engages in a joyless, frenetic, life struggle to achieve more and more in a time-urgent fashion with an air of free-floating hostility. Most recently Friedman has coined this the AIAI complex, signifying anger, irritation, aggravation, and impatience (4). He believes that the presence of this type of behavior is correlated with at least double the incidence of coronary artery disease compared with people who do not show this behavioral style. This belief was confirmed in 1981 by a panel of scientists convened by the National Heart, Lung, and Blood Institute (26).

There have been some interesting investigations of physiological changes in persons described as possessing the type A behavior pattern, particularly under conditions of stress or challenge. Friedman's group has found significant associations between this behavioral pattern and increased circulating norepinephrine, ACTH, cholesterol, triglycerides, and increased blood pressure, especially in response to situations of perceived challenge (4). They have also described in these subjects a hyperinsulinemic response to glucose, decreased growth hormone, decreased rate of clearing plasma lipids following ingestion, and increased intraarteriolar red blood cell sludging in the presence of high blood fats.

Also studying type A subjects, Williams demonstrated a direct correlation between elevated hostility scale scores on psychological testing and degree of occlusion of coronary arteries (27). In follow-up research, Williams' group has also shown significant differences in cardiovascular and neuroendocrine responses in type A subjects exposed to mental challenges in the laboratory setting (28). Compared to normals, the type A subjects showed greatly elevated responses in forearm blood flow (with decreased forearm vascular resistance), plasma norepinephrine, epinephrine, cortisol (while doing mental arithmetic), and elevated testosterone (during reaction time tasks). McKinney has extensively reviewed the hemodynamic-biochemical responses as pertains to cardiovascular disease (29).

Along with investigations into the degree and significance of psychosocial factors in promoting coronary artery disease, the role of psychosocial and behavioral interventions in modifying the morbidity of coronary artery disease is an area of growing interest. There would probably be the least argument about behavioral interventions that would help patients stop smoking, lose weight, eat healthier diets, and engage in regular exercise as useful interventions in reducing the risk of first occurrence as

well as the recurrence rate of myocardial infarction. It would seem from Ruberman's findings (23) that psychosocial interventions, which generally reduce stress and alleviate isolation, may also reduce recurrence of myocardial infarction and possibly prevent first occurrence in vulnerable persons.

Direct interventions into type A behavior problems deserve consideration based on some encouraging findings reported to date. A four-year ongoing controlled experimental behavioral intervention program was conducted by Friedman's Recurrent Coronary Prevention Project with over 1,000 postmyocardial infarction subjects (30,31). It was found that the subjects with uncomplicated myocardial infarctions who participated in a behavior change group geared toward reducing type A behavior had about half the recurrence rate of myocardial infarction as subjects who participated in a cardiologist-led group counseling program that only addressed physical health interventions. Others have been exploring various psychotherapeutic and behavioral interventions as approaches to dealing with coronary artery disease. The results, though preliminary, deserve consideration for interested clinicians and researchers. These preliminary results include reports on group therapy (32,33), behavior therapy (34), cognitive therapy and stress management training (35), the relaxation response (36), and numerous other approaches that are described in reviews (37-39).

Summary

The interface between mind, emotion, and the heart, particularly disturbances of psyche as they may contribute to cardiac malfunction has been reviewed. The wealth of research and literature in this field could appropriately be referred to as "psychocardiology." This area of study should also include the emotional changes resulting from cardiac disease, a separate topic not addressed in this paper.

The evidence for acute emotional stresses immediately culminating in perturbations of cardiovascular function is much easier to demonstrate than links between chronic psychosocial stressors or behavioral styles and coronary degeneration. The former is so well known as to be folk wisdom. The latter are difficult to demonstrate in straightforward scientific research largely because of problems in objectifying complicated psychological experiences over time and the intrusion of many other variables. Nevertheless, the findings to date are intriguing, although controversial, and certainly stimulate the need for more research built upon the foundation that has been established.

As for interventions, it certainly cannot be claimed at this time that stress reduction measures will reduce coronary morbidity or mortality. However, it cannot help but improve the quality of life of these patients. The chance that psychological interventions may some day actually have a legitimately accepted and proven role in reducing coronary morbidity and mortality is definitely an idea which deserves testing until a consensus can be reached.

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