Emotions have been connected to the heart throughout the ages, yet they have been largely discounted as playing an important role in heart disease. There is mounting evidence that anxiety, anger, depression, and stress play significant contributing roles in cardiac diseases. These emotional states, coronary artery disease, and heart failure have physiology consistent with the ongoing stress response characterized by parasympathetic withdrawal and sympathetic activation. Pharmacological therapies, vagal stimulation, and sympathetic ablation have shown efficacy in these diseases. Similar results can be obtained by biofeedback therapies.

Emotions and the Heart

Emotions have been connected to the heart in art, literature, and song. We speak of the heart as being core to issues of our lives. Expressions such as “heartfelt,” “heartbroken,” “heart throb,” “heart aches,” “heartburn,” “heart of the matter,” and “connecting heart to heart” reveal the connection between heart and emotion and the central place our heart plays in our lives. That place was displaced when medicine turned from a holistic to a mechanistic approach.

The heart became viewed as a piece of anatomic machinery that was necessary for the pumping of blood to the body. It could be repaired, transplanted, repiped, stented, and medicated, and its abnormal rhythms could be paced or shocked back to normalcy. With the proper concoction of medications to lower blood pressure and cholesterol, and create physiological homeostasis, all would be well. If patients would only be compliant, control their weight and diet, stop smoking, and control their diabetes, heart disease would not exist.

This view remained the dominant paradigm. Brief forays (which were later disputed) into the effect of behavior and psychological style on heart disease were presented in the form of research on Type “A” behavior (Friedman, 1996).

The most recent American Heart Association Guidelines (Smith et al., 2011) have extensive guidance on smoking, blood pressure, lipid management, exercise, weight control, diabetes management, antiplatelet agents, renin-angiotensin-aldosterone system blockers, beta adrenergic blockers, and cardiac rehabilitation. The only reference to psychosocial or emotional factors is that it would be reasonable to screen for depression in patients after myocardial infarction or coronary artery bypass graft, if the patient has access to case management. Little attention has been directed to the underlying causes of high blood pressure and high cholesterol, to the “lifestyle choices” aspects of smoking, eating, and exercise, or to the global physiological dysregulation often present in cardiovascular disease.

There is mounting evidence that there are psycho-social-emotional-physiological contributions to cardiovascular disease (Brosschot, 2010). In the Normative Aging Study, Kawachi and colleagues found that men without obvious heart disease who had noted two or more symptoms of anxiety over the ensuing 32 years had 3.2 times the risk of having fatal coronary heart disease and 4.5 times the risk of sudden death (Kawachi, Sparrow, Vokonas, & Weiss, 1994). Men with the highest levels of anger were more than three times more likely to have a nonfatal or fatal myocardial infarction than the men with the lowest level in 7 years of follow-up (Kawachi, Sparrow, Spiro, Vokonas, & Weiss, 1996). The Precursors study echoed these findings (Chang et al., 2002). Depression also portended early mortality from heart disease (Ford et al., 1998) and while there are a few negative studies, the vast majority of studies found that even if the known risk factors were accounted for, depression significantly increased the risk of having a significant cardiac event. Kubzansky summarized numerous prospective studies of the incidence of cardiac disease, finding only one study that didn’t show increased risk when either depression or anxiety was present (Kubzansky, 2007).
In the INTERHEART study with 30,000 participants from 52 nations, psychosocial factors conferred a 2.67 times increased risk of myocardial infarction (Rosengren et al., 2004). Not only do these negative emotional states confer an increased risk of coronary artery disease (CAD), but they also convey decreased life expectancy post myocardial infarction (MI) and post coronary artery bypass graft (CABG). Return to function was also impeded by negative emotion (Grace et al., 2005; Januzzi, Stern, Pasternak, & DeSanctis, 2000). Free-floating, unprovoked anger, and suppressed anger were equally damaging (Gallacher, Yarnell, Sweetnam, Elwood, & Stansfeld, 1999). PTSD and child abuse also increased the probability of later life coronary artery disease (Dong et al., 2004; Kubzansky, 2007). To date, two major trials, SADHART and ENRICH, have failed to show reduced mortality and cardiovascular morbidity in response to either antidepressant or cognitive behavioral therapy (Joyn & O’Connor, 2005).

Stress cardiomyopathy—also known as “broken heart syndrome”—occurs when an emotionally stressful event causes a massive release of catecholamines resulting in significant, in most cases reversible, cardiac dysfunction accompanied by ballooning of the left ventricle, usually in the absence of coronary artery disease. It also can cause coronary spasm and myocyte necrosis with mononuclear cell infiltration characteristic of inflammation (Wittstein, 2007; Wittstein et al., 2005). Voodoo death is thought to occur when the victim is totally incapacitated by fear causing catecholamine storm and death (Samuels, 2007). Severe external stressors such as earthquakes and internal stressors like angry episodes have markedly increased risk of MI and cardiac dysfunction (Dimsdale, 2008; Möller et al., 1999).

**The Human Stress Response**

We need to ask bigger questions as well as smaller questions. For instance, were those patients who were able to quit smoking either more resilient or less stressed than those who weren’t (Critchley & Capewell, 2003)? Are adult onset diabetes, hypertension, obesity, and high cholesterol manifestations of acute and chronic stress and not separate entities? Should they be looked at as additive to the risk factor or at least not subtracted? Are depression, anxiety, and anger actually signs of problematic stress coping and not separate entities? They are often found together and coexist with the traditional risk factors (Suls & Bunde, 2005). Chronic stress has been determined to be a cause of essential hypertension (Esler et al., 2008) associated with elevated cortisol and increased sympathetic nerve firing. Increased sympathetic firing has also been found in other conditions associated with increased cardiovascular morbidity (Lambert et al., 2008).

Briefly examining the stress response, we can see how these entities fit together. The stress response is activated by real or perceived threat or cognitive perseveration over a past threat or future worry (Brosschot, 2010). The stress response is complex, varies from individual to individual, and is situation specific. However, there are some generalities. Once activated in humans and mammals, it is characterized by parasympathetic (vagal) withdrawal and activation of the sympathetic nervous system (Gevirtz, 2007). At the same time, the hypothalamic-pituitary axis is activated. Corticotrophin releasing factor (CRF) and vasopressin are released from the hypothalamus and the posterior pituitary, respectively. CRF stimulates the release of adrenocorticotropic hormone (ACTH), which is released into the peripheral circulation and stimulates the release of cortisol and aldosterone from the adrenal gland. Sympathetic activation initiates the release of renin from the kidney, which triggers the activation of angiotensinogen to angiotensin I and conversion of angiotensin I to angiotensin II in the liver by angiotensin converting enzyme (ACE). Angiotensin II stimulates aldosterone secretion, fluid retention, and vasoconstriction (Saavedra et al., 2004). It also triggers the release of epinephrine and norepinephrine from the adrenal medulla. Both glucocorticoids and angiotensin II appear to be involved in the feedback and feed forward elements of the stress response. Blockade of angiotensin II receptors eliminates the stress response in rats exposed to restraint and social isolation stresses. The use of α and β sympathetic blockers, ACE inhibitors, and aldosterone blockers have been and continue to be mainstay therapies for hypertension and post myocardial infarction care (Smith et al., 2011). Successful treatment for refractory hypertension has been effected by radiofrequency catheter ablation of sympathetic nerves to the kidney and adrenals through the renal artery (Geisler et al., 2012; Kandzari et al., 2012). Vagal nerve stimulation has been shown to be efficacious in the treatment of congestive heart failure (De Ferrari & Schwartz, 2011). These methods demonstrate the efficacy of decreasing sympathetic stimulation and increasing parasympathetic tone.

**Conclusion: The Promise of Heart Rate Variability Biofeedback for Cardiovascular Disease and Other Stress-Related Conditions**

In coronary artery disease, heart failure, anxiety, depression, fear, and anger, there is altered heart rate variability with loss of the parasympathetic component and increase of...
the sympathetic component (Carney et al., 1995; Kawachi, Sparrow, Vokonas, & Weiss, 1995; Kiebigh, 2010; McCraty, Atkinson, Tiller, Rein, & Watkins, 1995). Heart rate variability (HRV) training and biofeedback have been shown to be effective in reversing these effects (Del Pozo, Gevirtz, Scher, & Guarneri, 2004; Moravec & McKee, 2011).

HRV biofeedback is far less invasive, less expensive and has fewer side effects than pharmacological and invasive measures. More research is clearly needed as well as more effort to bring these modalities into more widespread clinical practice.

References


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