Chapter 2
Psychological Challenges of Coping with Coronary Artery Disease

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Introduction

A clinically effective approach to psychological aspects of coronary disease follows an appreciation of, first, the psychological conditions associated with coronary disease and, second, the mechanisms that link them. Because the process of association often runs a circular course with causes leading to effects which further exacerbate the causes, effective treatments of psychological aspects of coronary disease must often aim for multiple targets over extended periods of time with careful monitoring and vigilant relapse prevention efforts.

This chapter begins with a brief summary of coronary disease followed by a consideration of the mechanisms that link depression and anxiety to coronary disease. An understanding of the various linking pathways often guides the behavioral approach to the psychological challenges of coronary disease. Then a brief review of the epidemiologic evidence for depression, anxiety, and stress as risks for coronary disease lays the rationale for focusing behavioral treatment efforts on these conditions. Finally, general principles for treating depression and anxiety point toward specific management tips.

Coronary Disease

Also called coronary artery disease or coronary heart disease, coronary disease is a chronic, progressive condition defined by the formation of plaques in the major coronary arteries. The eventual narrowing of the lumen or the development of a thrombus leads to reduced perfusion of the cardiac muscle, often chest pain (angina
pectoris), and ischemia or infarction of the cardiac muscle tissue. Acute coronary syndromes include unstable angina, myocardial infarction, and cardiac arrhythmias or arrest. Though the process of atherosclerosis begins in the second or third decade of life, the clinical disorder usually does not afflict men until their 40s or 50s and women about a decade later.

About a third of US adults die of cardiovascular diseases, most often complications of coronary disease. In a given year, about 13 million US adults have coronary disease (www.americanheart.org). Though mortality rates from coronary disease have dropped substantially over the past 40 years because of treatment advances and public health education campaigns, incidence rates for coronary disease remain unchanged, suggesting that we have done little to prevent the onset of coronary disease. Of the six major modifiable risk factors for coronary disease (age, male gender, and family history are the three unmodifiable risk factors), any progress with reducing smoking rates has been offset by the obesity and diabetes epidemics. Hypertension, hyperlipidemia, and physical inactivity (the other three major risk factors) remain substantial public health problems. All of the six major modifiable risk factors are exacerbated by depression, anxiety, or chronic stress (Wulsin, 2007).

Coronary disease results from atherosclerotic plaque formation in the walls of the coronary vessels, a process that is hastened by chronic low-grade vascular inflammation, injury to the endothelial lining of the arteries at sites of turbulent flow, and lipid deposits at these injury sites. Vasospasm and platelet aggregation, both of which may be triggered by stress, contribute to the episodic progression of coronary disease.

Diagnosis of coronary disease results from a history of angina, fatigue, or weakness in the context of risk factors and confirmed by electrocardiogram evidence for ischemia or cardiac enzyme elevations. On coronary angiogram, evidence of more than 50% narrowing of the lumen of at least one major coronary artery points establishes the diagnosis of coronary disease in patients with a typical history.

Prospects for people with coronary disease remain risky. One-third of those with a first MI die within an hour of the onset of symptoms. Among those who survive the first MI, one-fifth die within a year. Current treatments for acute coronary syndromes include urgent thrombolysis or revascularization by angioplasty, stent placement or coronary artery bypass graft (CABG) surgery to abort MI, β-adrenergic blockade, statin therapy to stabilize plaque, and aspirin or antiplatelet drugs. Maintenance and preventive measures include the use of aspirin as an antiplatelet drug, exercise, lipid-lowering therapy with statin drugs, abstinence from or cessation of smoking, blood pressure control, and maintenance of normoglycemia (Shapiro & Wulsin, 2009).

Pathways from Negative Affect to CVD

Prolonged distress or misery accelerates the development and progression of cardiovascular diseases through many pathways. The pathways from negative affect to coronary heart disease are marked by a set of behavioral and biological mechanisms associated with exposure to depression, anxiety, and acute or chronic stress on the one hand, and...
with the outcomes of coronary heart disease and related conditions on the other (Carney, Freedland, Miller, & Jaffe, 2002; Rozanski, Blumenthal, & Kaplan, 1999).

The behavioral pathways to coronary disease include physical inactivity, smoking, social isolation, high-fat and high-carbohydrate diets, and poor adherence to medication and self-management regimens. Each of these behavior patterns is associated with exposure to clinical depression and anxiety. The biological pathways to coronary disease include sustained autonomic imbalance (sympathetic overactivity, parasympathetic underactivity), stress response dysregulation of the hypothalamic pituitary adrenal (HPA) axis, vascular inflammation, endothelial dysfunction, and increased platelet aggregation. It is also possible that negative affect and coronary disease share a set of common genetic vulnerabilities with the phenotypical expression of negative affect expressing itself usually decades before the phenotypic expression of coronary disease (McCaffery et al., 2006).

The first point to note in this list of behavioral and biological precursors of coronary disease is the large number of possible mechanisms and combinations of mechanisms linking persistent exposure to negative affect with the later development of chronic pathology in the cardiovascular system. It is doubtful that any single mechanism alone could spur the onset of any chronic disease, and many people who have more than one cardiac risk factor will soon develop others. Clusters of risk factors are necessary for the development of a condition as complex and gradual in its onset as coronary disease. One such cluster, the metabolic syndrome, consisting of hypertension, dyslipidemia, hyperglycemia, and abdominal obesity, predicts increased risk of atrial fibrillation, acute coronary syndrome, sudden cardiac death, and overall mortality (Gehi et al., 2009).

Persistent conditions of negative affect exert their pressure on these risk factors and the pathological processes of cardiovascular disease through disruptions in (a) the autonomic, peripheral, and central branches of the nervous system; (b) the endocrine system; (c) the immune system; and (d) the coagulation system.

The behavioral and biological mechanisms can be arranged along a set of pathways that suggest some of the common sequences of events for a hypothetical patient over the decades preceding the first cardiac event. This useful oversimplification conveys some of the complexity of the process over time at all levels of the biopsychosocial model. Figure 2.1 shows three pathways by which chronic depression, anxiety, and stress may contribute to coronary disease. The first noxious effect of prolonged exposure to negative affect may take the form of behavior changes leading to high-risk patterns self-care, diet, exercise, and social contacts. Persistent patterns of high-risk behaviors then establish early biological precursors to the major risk factors for heart disease, such as hypertension, diabetes, and obesity.

A second pathway from depression or anxiety to heart disease bypasses behavioral risks and directly affects biological factors that contribute to heart disease, such as inflammation (Kop & Gottdiener, 2005), autonomic imbalance (Thayer & Lane, 2007), and endothelial dysfunction. The third pathway amplifies associated forms of chronic distress, such as anxiety and exhaustion (Kop, 1999), which also exert both direct biological influences on cardiac risk factors and indirect influence via exercise and smoking.
This schematic diagram lists at least 18 variables potentially intervening between the exposures of depression or anxiety and the outcome of coronary disease. The evidence supporting the roles for these intervening mechanisms varies widely in the number of variables measured, the methods of measurement, and the duration of measurement (Carney et al., 2002; Rudisch & Nemeroff, 2003). Since no single study can hope to assess all or even most of these variables over the relevant decades of a sample, it is unlikely that a comprehensive study of this relationship could ever be done. So it is important to interpret all studies in this area in light of their potential methodologic limitations, which may include some of the following: narrow time frames for follow-up, limited measurement of the exposure to depression, exhaustion, or anxiety, and adjustment for only a few of the many potentially confounding variables.

However, understanding these pathways points to several principles for behavioral treatment. First, a clear understanding of all the behavioral and biological factors contributing to cardiac risk ideally guides comprehensive treatment planning for coronary disease. Second, it is unlikely that any single treatment alone could broadly reduce all contributing factors, so collaborative care and complex treatment plans tend to be the rule for achieving effective outcomes. Third, some factors lend themselves more easily to treatment and should be the targets of early treatment efforts. Fourth, the process of developing coronary disease requires years and sometimes decades of exposure; consequently, the duration of behavioral interventions often requires many months and sometimes years to reverse these patterns of biology and behavior before consolidating new more adaptive behavior patterns.
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Depression

In the good studies of community and cardiovascular samples, clinical depression is defined as a cluster of depressive symptoms for at least a week, usually associated with impaired functioning. Some retrospective studies have used a documented clinical diagnosis of depression and/or extended treatment with antidepressants or psychotherapy for depression. According to DSM IV, minor depression involves two to four depressive symptoms for at least 2 weeks, whereas major depression involves five or more symptoms for at least 2 weeks (American Psychiatric Association, 1994).

Self-report measures of depression, observer ratings, and the mood modules of structured diagnostic interviews are the major methods of measuring depression. At least ten accepted self-report measures (e.g., Beck Depression Inventory, Center for Epidemiologic Studies-Depression Scale, Type D Personality), four observer ratings (e.g., Hamilton Depression Rating Scale, Montgomery-Asberg Depression Rating Scale), and five structured interviews (e.g., Structured Clinical Interview for Diagnosis, Diagnostic Interview Schedule) have been used in methodologically good studies of depression in heart disease.

In the study of the many possible psychosocial risk factors for cardiovascular disease, clinical depression, more than any other psychosocial risk factor, has proven to have the strongest relationship to coronary heart disease. And this relationship holds across the spectrum of severity of clinical depression, whether the exposure is assessed by self-report measures of symptoms or by structured diagnostic interviews. That is, the dose-response relationship between depression and coronary disease is measurable and clinically significant in the range of mild depressive symptoms and minor depression, and as the severity of depression increases the cardiovascular risk increases (Lesperance, Frasure-Smith, & Talajic, 2002; Wulsin, 2004).

Depression, though common in coronary disease patients, is frequently overlooked by their cardiologists and primary care physicians. Clinically significant depressive symptoms are found in 40–65% of patients following a myocardial infarction, and major depressive disorder is found in 15–25% of such patients (Rudisch & Nemeroff, 2003). Depression is often chronic: Three-fourths of the patients with major depression 2 weeks after a myocardial infarction remain depressed 3 months later (Lesperance, Frasure-Smith, & Theroux, 2000).

Denollet and colleagues have examined the effect on coronary disease outcomes of “Type D Personality,” a set of traits that combine a pattern of anxious and depressive feelings with a tendency toward social inhibition and isolation (Denollet, Sys, & Brutsaert, 1995; Denollet et al., 1996). Denollet has shown in several controlled studies that people who score high on the 14-item Type D questionnaire have higher early mortality rates from coronary disease. He argues that what damages the cardiovascular system is not just depressed mood or feeling bad but the persistent pattern of feeling bad alone over many years (Denollet, Pedersen, Vrints, & Conraads, 2006).

Several systematic reviews have summarized the evidence supporting the hypothesis that clinical depression increases the risk for both the development and
the progression of CAD (Barth, Schumacher, & Herrmann-Lingen, 2004; Rugulies, 2002; van Melle et al., 2004; Wulsin & Singal, 2003). A number of large-scale, prospective, epidemiological studies included in these reviews have estimated that depression independently increases the relative risks for both the development of coronary disease and cardiac death by approximately 1.5–2.0.

In the Frasure-Smith study with 6- and 18-month follow-up of MI patients, symptoms of depression and the diagnosis of major depressive disorder conferred a 3.5–6.6-fold increased adjusted relative risk of death (Frasure-Smith, Lesperance, & Talajic, 1995). The Heart and Soul Study of 1,002 stable outpatients with coronary disease showed that depression more strongly predicts impairment in quality of life and physical functioning than reduced ejection fraction or exercise-induced myocardial ischemia (Ruo, Rumsfeld, Hlatky, Browner, & Whooley, 2003).

Just as the risk of incident coronary disease rises with the severity of depression, a dose-response relationship appears to exist between the severity of depression symptoms after acute MI or unstable angina and the risk of death over 5-year follow-up, even after controlling for other prognostically significant factors. This dose-response relationship has been found in over ten methodologically sound studies and the prognostic effect size is comparable to some of the more common cardiac predictors of poor outcome, such as low ejection fraction, previous MI, and smoking (Wulsin, 2004).

Severe depression 6 months after CABG surgery, or persistence of even moderate depression symptoms at 6-month postoperative follow-up, predicts increased risk of death over 12-month follow-up (Connerney, Shapiro, McLaughlin, Bagiella, & Sloan, 2001).

Psychosocial Treatments

In one study of 435 post–myocardial infarction patients, a nursing-based psychosocial intervention reduced cardiac mortality at 1 year, and the incidence of recurrent myocardial infarction was significantly lower at 7-year follow-up. However, two subsequent, large, randomized trials of multimodal interventions delivered by nurses or health visitors failed to improve depression or cardiac outcomes (Taylor, Miller, Smith, & DeBusk, 1997). In the Montreal Heart Attack Readjustment Trial (M-HART), a supportive and educational home nursing intervention was provided to the most psychologically distressed post-MI patients. This rather limited intervention was compared to usual care. At 1-year follow-up, the intervention had no effect on psychological distress and no overall effect on cardiac mortality, while it was actually associated with a higher mortality rate among women (Frasure-Smith et al., 1997). However, a subgroup analysis revealed that those patients whose psychological distress did improve with treatment did have more favorable long-term cardiac outcomes.

In the Enhancing Recovery in Coronary Heart Disease (ENRICHD), 2,481 recent myocardial infarction patients with depression and/or low social support randomly
received either cognitive behavior therapy (and SSRI antidepressants if indicated) or care as usual. There was no benefit in terms of cardiac outcomes or mortality, and cardiac outcomes appeared worse for women (ENRICHD, 2003).

A meta-analysis of 23 randomized controlled trials evaluated the additional impact of psychosocial treatment on rehabilitation from documented coronary disease (Dusseldorp, van Elderen, Maes, Meulman, & Kraaij, 1999). Relaxation training, stress management, and group social support were the predominant modalities of psychosocial intervention. Anxiety, depression, biological risk factors, mortality, and recurrent cardiac events were the clinical endpoints studied. These 23 studies included a total of 2,024 patients in intervention groups and 1,156 control subjects. Psychosocial treatment patients had greater reductions in emotional distress, systolic blood pressure, heart rate, and blood cholesterol level than comparison subjects. Patients who did not receive psychosocial intervention had 70% greater mortality and 84% higher cardiac recurrent event rates during 2 years of follow-up.

Cardiac rehabilitation itself may reduce high levels of hostility, as well as anxiety and depression symptoms, in post-MI patients. A meta-analysis of psychoeducational programs for coronary disease patients concluded that they led to a substantial improvement in blood pressure, cholesterol, body weight, smoking behavior, physical exercise, and eating habits and to a 29% reduction in MI and 34% reduction in mortality, without achieving significant effects on mood and anxiety (Linden, Stossel, & Maurice, 1996). These programs included health education and stress management components.

**Pharmacotherapy**

If depression lasts more than several weeks and meets diagnostic criteria for major depressive disorder, as happens in one-third or more of patients in the year after myocardial infarction, antidepressant pharmacotherapy is recommended in addition to psychotherapy. In general, SSRIs are the best first choice for the treatment of depression, and within this class, the best research has focused on sertraline and citalopram.

The multicenter Canadian CREATE trial found that in 284 patients with major depression and CAD, citalopram plus clinical management was more effective for remission of depression than placebo or clinical management alone (Lesperance et al., 2007). Interpersonal therapy for depression conferred no advantage over clinical management alone.

The Sertraline Antidepressant Heart Attack Randomized Trial (SADHART) was a double-blind, randomized, placebo-controlled trial of a selective serotonin reuptake inhibitor (SSRI) for major depressive disorder in patients hospitalized for myocardial infarction or unstable angina (Glassman et al., 2002). At 6-month follow-up, when compared with placebo, the more severely depressed patients who received an active drug were less depressed, although the less severely depressed patients did not show a treatment effect. The trial showed that sertraline was safe and effective for the treatment of recurrent major depression in patients with recent MI or unstable angina.
In this study, there was a 20% reduction in life-threatening cardiac events (including nonfatal myocardial infarction and death) among those on active drug, but this difference in cardiac outcomes was not statistically significant due to the number of patients in the trial (Glassman et al., 2002). Depression is the strongest predictor of quality of life in post-MI patients, and in this study, treatment with sertraline was associated with clinically meaningful improvements in quality of life over 6 months for those with recurrent depression (Swenson, 2004).

In a case-controlled study of smokers hospitalized for myocardial infarction, SSRI administration was associated with a lowered risk of recurrent myocardial infarction, suggesting that treatment of depression may reduce its negative prognostic influence on cardiac outcomes (Sauer, Berlin, & Kimmel, 2001). Thus, though it remains to be definitively demonstrated that either the pharmacologic or psychological treatment of depression following myocardial infarction significantly improves cardiac outcomes, the rationale for treatment of depression remains strong: to reduce suffering and to improve quality of life.

**Anxiety**

Though often confused with each other, anxiety and stress are best operationalized differently to cover the spectrum of distress. Anxiety, when applied to an enduring condition, even a brief one of several weeks or months, implies a diagnosable disorder, usually deserving treatment. Anxiety disorders may be brief, as adjustment disorders, or lifelong, as in post-traumatic stress disorder or severe forms of obsessive compulsive disorder. The five major anxiety disorders (generalized anxiety disorder, panic disorder, phobias, obsessive compulsive disorder, post-traumatic stress disorder) differ from each other in their dominant psychological features, epidemiology, biology, and response to treatments.

On the other hand, stress is a more general term for the distress experienced in response to either an acute event, such as an argument or injury, or a chronic condition, such as poverty or marital conflict. The term stress has played an important role in cardiovascular research since the time of Hans Selye, and more recently in research on mental stress–induced ischemia.

As with depression, the options for measuring anxiety include self-report measures (e.g., Beck Anxiety Inventory, GAD 7), observer ratings (e.g., Yale Brown Obsessive Compulsive Scale), and the anxiety disorders modules of structured diagnostic interviews. Measures of stress vary widely with the operational definitions selected, ranging from salivary cortisol levels to heart rate recovery following a mental challenge to self-reports of subjective distress.

Chronic mental stress, such as job strain or marital strife, also contributes to the development and the progression of coronary disease (Albert, Chae, Rexrode, Manson, & Kawachi, 2005). In a study of over 900 men and women who returned to work after their first MI, job strain (high demand plus low decision latitude) independently doubled the risk for recurrent cardiac events in the next 6 years, in a model that adjusted for 26 confounders (Aboa-Eboule et al., 2007). In a longitudinal
study of 812 Finnish employees over a 25-year period, those with high demands from and low levels of control over work conditions had more than double the risk for cardiovascular disease mortality (Kivimaki et al., 2002). Marital stress has been found to exert a negative prognostic influence on coronary disease in women and may be even more important than job stress for women (Orth-Gomer et al., 2000).

And in the largest case control study of predictors of MI, the INTERHEART Study found that among 11,119 cases of MI in 52 countries, psychosocial factors, which included self-reports of stress and depression, ranked as the third highest predictor of MI, raising the risk for MI by an odds ratio of 2.67, similar to smoking and diabetes (Rosengren et al., 2004).

Several prospective studies of initially healthy men and women found that high anxiety at baseline increased the risk for subsequent development of arteriosclerotic plaques, carotid artery intimal thickening, nonfatal myocardial infarction, and cardiac death. In the Framingham Heart Study, high levels of tension predicted increased risk for new coronary disease and anxiety predicted increased risk for mortality from all causes (Eaker, Sullivan, Kelly-Hayes, D’Agostino, & Benjamin, 2005). Another large prospective study found that high levels of phobic anxiety are associated with an increased risk of fatal coronary disease, particularly from sudden cardiac death (Albert et al., 2005).

Anxiety disorders may also worsen the course of established coronary disease. In a recent meta-analysis of 12 studies, post-MI anxiety was associated with about a 40% increase in risk for impaired cardiovascular outcome (Roest, Martens, Denollet, & de Jonge, 2010). This effect, though significant, is not as large as the effect of clinical depression or Type D personality. It remains unclear whether the adverse effects of anxiety operate more through arrhythmias and sudden cardiac death than through arteriosclerosis and infarction, or both.

Possible mechanisms explaining these long-term associations include sustained sympathetic nervous system upregulation with increased catecholamine production and decreased vagal activity, chronic low-grade inflammatory states, and persistent dysregulation of the stress response system, particularly the HPA axis.

**Stress**

Acute mental stress induces arterial endothelial dysfunction, with impaired vasodilation. Paradoxically, in atherosclerotic arterial segments, stress can induce vasoconstriction or at times vasospasm. Consequently, acute mental stress may have a significant constricting effect on coronary artery blood flow in patients with preexisting coronary disease. States of fear, excitement, and acute anger reduce blood flow through atherosclerotic coronary segments, provoke coronary spasm, and are associated with abnormal left ventricular wall motion and ECG evidence of myocardial ischemia. Acute stress also increases myocardial oxygen demands as a result of its hemodynamic effects. The normal stress response increases circulating cortisol and catecholamines, which activate platelets and promote platelet aggregation and which increase cholesterol and decrease high-density lipoproteins. The net result of these
actions is to increase cardiac demand while decreasing coronary blood supply and promoting plaque rupture and thrombus formation.

Mental stress–induced ischemia is associated with increased risk of subsequent cardiac events in patients with known coronary disease and may occur even in patients who do not demonstrate evidence of ischemia during exercise stress testing. Triggering of sudden cardiac death by mental stress was demonstrated in a study of deaths in the aftermath of a major California earthquake (Leor, Poole, & Kloner, 1996). After a surge of sudden deaths on the day of the earthquake, there was a several-day period of reduced incidence of sudden cardiac death, suggesting that only those predisposed to sudden cardiac death due to underlying disease were affected by the acute stress. Deaths were primarily associated with emotional stress rather than physical exertion. Both sudden death without antecedent angina, suggesting cardiac arrhythmia, and sudden deaths preceded by chest pain, suggesting acute coronary occlusions, were observed (Leor et al., 1996). Similar observations have been reported recently about sudden cardiac deaths in the aftermath of the destruction of the World Trade Center in New York in 2001 (Steinberg et al., 2004). Other studies have led to estimates that between 20% and 40% of sudden cardiac deaths are precipitated by acute emotional, rather than physical, stressors.

Mental stress–induced ischemia occurs at lower heart rates and at lower levels of myocardial work than does exercise-induced ischemia, suggesting that decreases in myocardial perfusion may play a role in mental stress–induced ischemia. In a laboratory study of 58 patients with coronary disease and three levels of LV function (normal, mild to moderately reduced ejection fractions 30–50%, and severe or <30% ejection fractions), ischemia was induced more frequently with mental stress in those with severe LV dysfunction (50%) compared to 9% of those with normal LV function. Mental stress-induced ischemia may be most important clinically in coronary disease patients with LV dysfunction (Akinboboye et al., 2005).

Mental stress–induced ischemia is more likely to be “silent,” or asymptomatic, than is ischemia induced by exercise. In one study, 83% of mental stress–induced ischemic episodes were asymptomatic. And about a third of coronary disease patients without exercise-induced ischemia experience mental stress–induced ischemia, suggesting the two forms of stress induce ischemia by different but related sets of mechanisms (Ramachandruni et al., 2006).

Treatment

Laying the Groundwork

The first step for the patient in the development of comprehensive care for the psychosocial aspects of coronary disease is to establish a relationship with a good primary care doctor. Reducing psychosocial risks goes hand in hand with reducing
cardiovascular risks, which will often be directed by the primary care doctor. Most good primary care doctors will also manage the initial stages of pharmacologic trials for uncomplicated depression and anxiety. And they will often be the first to learn of relapses in either the coronary disease or the distress associated with it.

The second step toward better care involves helping your patient learn his or her risks for depression, anxiety, and heart disease. This means learning a few facts and a few numbers that describe the risks. This learning process, often a late response to unwelcome symptoms, should begin early in adulthood, before habits and arteries have hardened. Until our patients learn their risk factors, they cannot hope to manage their risks, and we cannot help them much.

The exercise of drawing a family tree marked with the relevant illnesses often raises useful questions for prevention and treatment planning, pointing to generational patterns related to depression, anxiety, or heart disease, like alcoholism, diabetes, high blood pressure, and obesity. After a careful review of the family history, it should become clear whether the family history contributes a low, medium, or high risk for depression, anxiety, or heart disease, or some combination.

Identifying the risks for depression and heart disease means knowing a few numbers. Men in their 40s and women in their 50s should begin learning not only about their family histories but also about their cholesterol levels, blood pressure, body mass index, glucose tolerance, physical activity patterns, numbers of episodes of major depression, and current severity of depressive symptoms. Knowing the numbers makes it harder to deny the risks.

**Targeted Behavior Change**

Once your patient has established a relationship with a primary care doctor and identified key risk factors, the next step is to pick the targets for prevention and treatment. Setting these priorities for treatment is often guided by access to treatment resources, such as cardiac rehabilitation, psychotherapy, and selected specialists. Pain or fear often lends urgency to one factor over another. People in distress may underestimate their resources and feel unnecessarily helpless when resources may be around the corner.

For people with multiple risk factors, begin with the one or two targets that your patient can most easily improve. Pick your targets and be patient. It took decades to build these processes that contributed to the vicious cycle of depression and heart disease, and it usually takes several years – not days or weeks – to reverse them.

One of the most important problems modern medicine has yet to solve for the management of all chronic illnesses is how to change high-risk health behaviors in large numbers of people. Sustained behavior change is tough for anyone, and, in general, doctors in the United States report low success rates in helping their patients quit smoking, lose weight, or exercise regularly.

However, the “stages of change” method, originally developed by James Prochaska and his colleagues and described in their inspiring self-help book,
Changing for Good (Prochaska, Norcross, & DiClemente, 1994), has earned the lead role among strategies for change. This model has been adopted by the Centers for Disease Control and Prevention, the National Cancer Institute, and the National Health Service of Great Britain in a wide range of programs for reducing risky behaviors. Ampel studies of the stages of change method combined with motivational interviewing techniques have shown that patients, on their own and with the help of clinicians, can make sustained behavior changes that improve their health outcomes. Motivational interviewing is the counseling approach for helping people translate their particular set of arguments for and against a behavior change into a treatment plan that works.

Losing weight ranks among the toughest behavior changes. For most of us losing weight is long, slow, and frustrating work. Since there is no quick fix for obesity (even bariatric or gastric bypass surgery takes about a year from start to finish), we rely on discipline, tenacity, and patience as much as any formal program or diet to achieve a stable lower weight. Losing weight is no job for the depressed mind; it is hard enough to do when we have all our wits and function at our best. Depression sabotages the best laid plans for weight loss, particularly if your patient tends to eat or drink more when depressed. The depressed mind often shifts moods and behaviors, gives up easily, grows impatient in the face of frustration, views itself as ugly, and expects failure. Before your patient launches into an ambitious diet or weight loss program, treat the depression as fully as possible and continue that regimen while working on losing weight.

Exercise

Next to losing weight, increasing physical activity may be the hardest health behavior to sustain. But for people with depression or anxiety and heart disease, exercise is one treatment for two problems. What is good for the heart is good for the brain. The equivalent of three and half hours of brisk walking each week is a good place to get started. Exercise sets the range of vascular tone throughout the system, from the largest (the heart) to the smallest vessels (the arterioles where the oxygen diffuses across arteriole walls and into the tissues). Pitch exercise as a tune-up for various levels of demand on the cardiovascular system, a drill that calls into action the coordination of hormones, nerves, and the muscles in the vessel walls. The drill also tunes up the immune system and the limbic system. Once finely tuned, the rises in adrenalin, cortisol, and endorphins account for the transient good feeling, the runner’s high, that comes with exercise, often overriding the aches and the fatigue.

Exercise also promotes neurogenesis or nerve cell regeneration in the hippocampus of the limbic system, the part of the brain that shrinks under prolonged stress or depression. Exercise restructures this part of the limbic system in a way that improves the functioning of the whole system. For many people, regular exercise works as an antidepressant both immediately (within minutes and lasting hours) and over the long run (lasting weeks to months).
Studies of exercise as a treatment for heart disease show a beneficial effect for both the prevention of coronary disease and for the improvement of existing coronary disease (Taylor et al., 2004). Studies of exercise as a treatment for depression have focused mostly on mild to moderate depression and suggest that sustained regular exercise reduces depressive symptoms as much as psychotherapy or antidepressant medication, and exercise reduces the risk of relapse (Babyak, Blumenthal, Herman, & Parinda, 2000; Blumenthal et al., 1999).

The levels of exercise required to reduce risk for heart disease are about the same as the levels required to reduce symptoms of depression. Thirty minutes of vigorous walking three times a week is a good goal to start with for people who usually do nothing that would pass for exercise (about half of the US population). Beyond that level the benefits increase with increasing duration and frequency up to a point. Most of the benefits can be achieved by 30–60 min of daily vigorous walking or light jogging. Any combination of exercise types and durations will do, such as several 15-min walks.

After Bypass Surgery, Angioplasty, and Stents

One in five people develop major depression within weeks after the surgical correction of narrowed coronary arteries. And depression increases the risk for needing a second corrective procedure within 5 years. That is, the same or a new narrowing of the coronary artery is more likely to happen in depressed people than in those who are not depressed. And for many people, depression soon after a heart attack or unstable angina doubles ortriples the risk for dying from coronary disease (Connerney et al., 2001; Sullivan, Simon, Spertus, & Russo, 2002). For these three reasons (if dodging the misery of depression is not compelling enough by itself), the first 6 months after a cardiac procedure represent a vulnerable period worthy of diligent preventive efforts. Monitoring of depressive symptoms, supportive psychotherapy, and a daily self-management plan to prevent relapse provide the best chances of avoiding complications. The pros and cons of preventive courses of antidepressants are complex and justify a psychiatric consultation.

References


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