CLINICAL CARDIAC PSYCHOLOGY

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Cardiac psychology is the study and application of psychological processes involved in the prevention and treatment of heart disease and in heart patients' adjustment to their illness and treatment. From a clinical perspective, there are three primary ways that psychological interventions can influence heart disease: 1.) by facilitating health-promoting behavioral changes and adherence to recommended medical treatment; 2.) by helping patients address illness-related issues and psychopathology; and 3.) by having a direct effect on psychological factors and biological pathways influencing the onset and progress of disease. This last topic is especially promising, since few other areas in behavioral medicine have such a well-established association between psychological factors and physical disease.

The assumption that "cardiac psychology" is a subspecialty within health psychology is justified by the wide range of psychological theories, research, and clinical procedures that can be applied to cardiac patients. Although this chapter reviews the field of clinical cardiac psychology, space dictates that many details will be abbreviated or left out. A resource providing a more thorough treatment of the field and practice of cardiac psychology is Heart and Mind: The Practice of Cardiac Psychology, by Allan & Scheidt (1996a).

Biomedical Aspects

According to current estimates from the American Heart Association (2001a), slightly less than one in four Americans have some form of cardiovascular disease (CVD), the number one cause of death in the United States for every year since 1900, except 1918.* Nearly twice as many people die from CVD as from cancer, the second leading cause of death. Four times as many heart attacks occur in men compared to women under age 65 (and 40 times as many in men than women under age 45), primarily because of the protective effect of women's reproductive hormones. Overall, however, slightly more

* All disease-related statistics in this chapter are from the American Heart Association (2001a).
women than men die from all forms of CVD. The incidence of all forms of coronary heart disease (CHD) in women lags about 10 years behind that in men, and the incidence of acute events such as myocardial infarction (heart attack) and sudden death in women lags about 20 years behind that in men, although the gap narrows as men and women get older. For Americans under age 75, there is a higher proportion of CVD events due to coronary artery disease in men than in women, and a higher proportion of congestive heart failure (CHF) in women than in men. There are also differences by race. The age-adjusted prevalence of CVD is 30.0% for white males and 23.8% for white females. In comparison with these base rates, the prevalence is 35.0% higher for African-American males and 4.0% lower for Mexican-American males than white males, and 66.4% higher for African-American females and 11.8% higher for Mexican-American females than white females.

There are three broad areas of biological cardiovascular disease that a cardiac psychologist should be familiar with to successfully work with cardiology referrals and participate in a multidisciplinary treatment team for cardiac patients: 1.) coronary artery disease (CAD); 2.) heart failure; and 3.) other forms of heart disease such as valvular disease and arrhythmias. The medical, psychological and neuropsychological consequences of stroke are usually addressed outside a cardiology practice, and will be briefly discussed here as CVD complications affecting cognitive function, adherence to treatment, and adjustment to illness. A concise source of information concerning CVD is Cardiology by Heger, Niemann, Roth, & Criley (1998). Additional information in the following section is from the American Heart Association (2001b) and Scheidt (1996).

**Coronary Artery Disease**

Coronary artery disease (CAD) is the atherosclerotic process that accounts for all but about 1% of what is known as coronary heart disease (CHD) and about half of all deaths from CVD. CHD includes relatively rare syndromes such as cocaine-related spasm, endocarditis of the aortic valve, and congenital abnormalities. Although "coronary heart disease" is used in American Heart Association publications and
in some of the research literature, "coronary artery disease" is more common in clinical settings and is the phrase of choice in this chapter. In similar fashion, it is useful to distinguish arteriosclerosis, which is a general term for the thickening and hardening of the arteries, from atherosclerosis, which comes from two Greek words meaning "paste" and "hardness." Atherosclerosis consists of hardened deposits (plaques) at specific locations on the inner lining of the arteries (the endothelium) that consist of fatty material, cholesterol, calcium, cellular waste products and fibrin, a clotting factor in the blood.

Why plaques occur is not well understood, although abnormalities and inflammation of the endothelium, oxidative processes, and the effect of excess blood lipids on the lining of blood vessels are suspected. Risk factors for atherosclerosis are listed in Table 1. Risk factors toward the top of the list are generally more well-established by numerous research studies than those toward the bottom of the list.

The atherosclerotic process of CAD manifests as three progressively more severe clinical syndromes: angina pectoris, myocardial infarction, and sudden cardiac death. Less common presentations include silent ischemia, which is lack of blood to a portion of the heart muscle (the myocardium) without causing symptoms, and ischemic cardiomyopathy, where part of the heart becomes non-functional due to loss of blood flow (see Heart Failure below). The three major clinical syndromes are most likely to occur when there is a 70% or greater stenosis in at least one of the three main coronary vessels or their branches, although these syndromes can occur with less extensive blockage as well. The number of vessels involved is usually included in the characterization of the syndrome (e.g., "three-vessel disease").

**CAD Clinical Syndromes**

**Angina pectoris.** Ischemic pain results when there is insufficient blood flow and nutrients to muscle tissue. In the heart muscle, ischemia is called angina pectoris ("choking in the chest"). Ischemia is often described as chest "pain" or "pressure," and may be associated with a sensation of fullness.
Physical activity or emotional arousal requiring increased blood flow can cause angina, or a temporary spasm of the coronary arteries may cause angina at rest. In some instances, angina can remain stable for years without much change in symptoms or acute events. Unstable angina may occur without provocation and can present with symptoms as severe as those of an MI, but does not result in permanent damage to the myocardium, although there is increased risk of acute events.

**Myocardial infarction.** An MI results when the blood flow is completely cut off following the formation of a blood clot (coronary thrombosis or occlusion) on the plaque's surface or when the plaque ruptures. A severe spasm can also cut off blood flow long enough for an MI to occur. The Framingham Heart Study found that 23% of initial MI's occurred in patients with a history of angina, and one in five MIs were silent or not identified by the patient from the presenting symptoms (Kannel & Feinleib, 1972).

Typical symptoms of a heart attack as outlined by the American Heart Association (2001b) include:

* Uncomfortable pressure, fullness, squeezing or pain in the center of the chest that lasts more than a few minutes, or goes away and comes back.
* Pain that spreads to the shoulders, neck or arms.
* Chest discomfort with lightheadedness, fainting, sweating, nausea, or shortness of breath.

**Sudden cardiac death.** The abrupt, unexpected loss of heart function can lead to sudden cardiac death (SCD). About half of all deaths from all types of heart disease occur suddenly. At least two major coronary arteries have atherosclerotic plaques in 90% of patients who have sudden death. The immediate cause of most sudden deaths, however, is ventricular tachycardia or ventricular fibrillation, a rapid or irregular heartbeat (American Heart Association, 2001b).

**Diagnostic Procedures**

**Lab tests.** Elevated cardiac enzymes, most notably creatine kinase (CK or CPK), and cardiac contractile proteins troponin T and I, indicate death of muscle tissue and are one of the first things
examined when a patient is brought to the emergency room with a suspected MI. Serum electrolyte levels (potassium, calcium, and magnesium in particular) are also examined since abnormal levels may affect cardiac function and alter electrocardiogram readings.

**Electrocardiogram (ECG).** The ECG is a graphic representation of the electrical activity of the cardiac cycle or heartbeat. Alterations in ECG readings can assist in diagnosing a variety of heart disease, although an initial ECG may be non-diagnostic in 20-50% of cases of acute MI (McGuiness, Begg & Semple, 1976). A Holter monitor is a portable ECG machine that can record heart activity during a patient's daily routine. This is usually done to assess for silent ischemia and arrhythmias.

**Exercise stress test (EST).** In patients with ischemic heart disease, coronary blood flow cannot increase to meet the heart's demand for oxygen. In an exercise stress test, patients are carefully monitored and given dynamic exercise up to a percentage of their age-predicted level of tolerance. Fatigue, dyspnea, tachycardia, angina, significant changes in BP, or ECG alterations may be indicative of ischemic heart disease. Women and patients with few or no cardiac risk factors have a higher likelihood of a false positive test.

**Echocardiography.** Echocardiography is a non-invasive procedure using ultrasound to provide moving images of cardiac structures. An "echo" can show cardiac valves, wall motion, flow-velocity patterns, and chamber dimensions and function. They are typically used in patients suspected of IHD, heart failure, valvular disease, and other cardiac diseases.

**Nuclear imaging.** A number of non-invasive imaging techniques using radioisotopes are used in cardiology. **Myocardial perfusion imaging** includes stress testing with thallium-201 or other radioisotopes that are absorbed by well-perfused areas of the myocardium; "cold spots" in the myocardium seen immediately following exercise but disappearing at rest indicate areas of decreased blood flow during exercise but adequate blood flow at rest. **Infarct avid imaging** uses an interaction between calcium and technetium pyrophosphate in damaged myocardial tissue to determine damage from
an MI. **Radionuclide angiography** enables visualization of the atria and ventricles, calculation of ventricular ejection fractions, and identification of wall motion abnormalities and valvular disease. **Positron emission tomography (PET scans)** are also used to evaluate CAD and myocardium function and is especially helpful to assess for viable myocardial tissue. Computers are widely used with all these techniques to refine and specify image data.

**Magnetic resonance imaging (MRI) or Nuclear Magnetic Resonance (NMR).** Computer-generated MRI images can evaluate damage from an MI, diagnose some congenital heart defects, and evaluate disease of the larger blood vessels.

**Cardiac catheterization.** By passing a catheter through the femoral vein or artery, or through the brachial vessels, diagnostic information on a variety of heart functions can be obtained, including assessments of the coronary arteries, ventricular ejection fraction, myocardial wall motion, and valvular function. This procedure is relatively safe, with less than 1% morbidity (Hansing, 1979). Cardiac catheterization may also be used to biopsy transplanted hearts to test for rejection, and as an intervention for CAD (see below). From 1979 to 1999, the number of cardiac catheterizations has increased 355%.

**Electron Beam Computed Tomography (EBCT).** "Ultra-fast" CT scans can rapidly produce an image of calcium deposits in the coronary arteries and have become increasingly popular for screening non-symptomatic men over age 40 and women over age 50 with no cardiac risk factors for CAD. Calcium deposits are highly correlated with atherosclerotic deposits, even at the early stages prior to any symptoms of CAD. For patients already diagnosed with CAD, however, EBCT provides no new information. EBCT and cardiac catheterization are the only tests (and EBCT is the only non-invasive test) that provides a pre-symptomatic early warning of CAD. However, the extent to which EBCT-identified calcium deposits are clinically significant and are associated with acute events and mortality remains controversial, with particular concern regarding patients who have a negative test yet develop symptoms of CAD within a year or two. Patients with a negative test and other changeable CAD risk
factors may also erroneously use their negative EBCT results to avoid managing those risk factors.

**Biomedical CAD Risk Factor Assessment and Treatment**

In addition to performing the clinical tests for CVD noted above, the physician and other medical and technical staff can identify a variety of medical risk factors for CVD.

**Dyslipidemias.** Through the 1980's, it was unclear whether lowering total cholesterol (TC) would improve life expectancy. Since then, clinical trials of lipid-lowering medications have shown significant decreases in mortality for men with mildly to moderately elevated cholesterol (Shepherd, Cobbe & Ford, 1995) and in coronary events for post-MI patients with normal cholesterol (Sacks, Pfeffer, Moye, Rouleau, Rutherford, Cole, Brown, Warnica, Arnold, Wun, Davis & Braunwald, 1996).

Roughly half of all United States adults have total cholesterol (TC) above 200 mg/dl. So-called "good" cholesterol (high-density lipoprotein, or HDL) is considered normal at approximately 45 mg/dl for men and 55 mg/dl for women; each five-point reduction raises the risk of cardiac events by 10%. "Bad" cholesterol (low density lipoprotein, or LDL) is of concern when it exceeds 100 mg/dl for CAD patients, 130 mg/dl for those with multiple risks but no disease, and 160 mg/dl for those with no other risks. Another cause for concern is when the TC to HDL ratio exceeds 4 to 1. A variety of other lipid particles, including triglycerides (TG), have been implicated as risk factors for heart disease. Total cholesterol in women is higher on average than in men beginning about age 55.

A variety of medical treatments are available for lipid disorders. HMG CoA reductase inhibitors (the "statins"; e.g., pravastatin, simvastatin) are effective for reducing total cholesterol and LDL and are usually well tolerated. Some medications, notably the bile acid sequestrants and niacin, have significant side effects (e.g., flushing, paresthesias, nausea) that reduce patient tolerance and adherence. New formulations (such as time-release niacin) have an improved side effect profile and are better tolerated. Depressive symptoms have also been noted with highly reduced cholesterol (Schwartz & Ketterer, 1997). Behavioral treatments such as smoking cessation, exercise, and reduction in body weight and dietary fat
are also recommended for management of dyslipidemias.

**Hypertension (HTN).** One in four American adults has high blood pressure (BP). Men are at greater risk of HTN than women until age 55; after age 74, women are at greater risk than men. However, HTN is 2 to 3 times more common in women taking oral contraceptives than in women not taking them. Blacks develop HTN earlier than whites, and have more severe HTN overall at any age. The overall death rate in 1999 from HTN in white males and females was 12.8 per 1000; for African-American males it was 46.8, and for African-American females it was 40.3.

Optimal BP is considered to be less than 130/85 mmHg (millimeters of mercury); hypertension is defined as a BP greater than or equal to 140/90 mmHg, and even mild HTN is associated with increased risk of CVD (JNC V, 1993). Lifestyle modifications for HTN (e.g., sodium restriction, weight reduction and increased physical activity) are recommended for patients with any level of HTN as a first line of treatment. There is about a 1 mmHg decrease in both systolic and diastolic BP for each kilogram of weight lost (Blanchard, 1994). There is mixed evidence that smoking and caffeine use are associated with HTN. Antihypertensive drugs are administered if lifestyle modifications have not been successful at lowering BP sufficiently. The type of medication treatment depends on the constellation of risk factors and patient tolerance of side effects. Drugs usually tried first include diuretics, beta-blockers, calcium channel blockers, and ACE inhibitors; supplemental agents such as central alpha antagonists (e.g., Catapres, Aldomet), may also be used. Side effects may include metabolic disturbances such as dyslipidemias and glucose abnormalities, and quality of life effects such as fatigue, depression, insomnia and sexual dysfunction.

**Insulin resistance syndrome (IRS).** Also known as Syndrome X, IRS consists of three main elements: 1.) significantly elevated serum insulin with normal or only slightly elevated glucose, indicating insulin resistance and impaired glucose metabolism; 2.) normal TC, but low HDL and high triglycerides; and 3. abdominal obesity (Reaven, 1994). Serum insulin level is not routinely tested in cardiac patients,
but IRS may be suspected if other characteristics are present. Patients with IRS have chest pain, normal angiograms, but abnormal exercise stress tests, which may be due to CAD in small cardiac vessels (microvascular angina) or spasms (Reaven, 1994; Poole-Wilson & Crake, 1989). Lifestyle modifications, such as increasing physical activity, lowering dietary fat, and reducing weight are recommended for treatment of IRS, partly because HTN may result from IRS, and HTN treatment with beta-blockers and thiazides can increase insulin resistance (Flack & Sowers, 1991).

Medical and Surgical Treatments

Medical treatment of angina. Angina is treated with three general classes of drugs: nitrates, beta-blockers, and calcium channel blockers. Nitrates (e.g., nitroglycerin, isosorbide) increase the supply of blood to the myocardium by decreasing the amount of work required by the left ventricle and by coronary vasodilation and improved collateral flow. Beta-adrenergic blocking agents (e.g., propranolol (Inderal), metoprolol (Lopressor)) block the action of catecholamines on the heart, reducing the amount of oxygen the heart requires by reducing heart rate and blood pressure, but also lowering myocardial contractility. Calcium channel blockers (e.g., nifedipine (Procardia), diltiazem (Cardizem)) can also reduce myocardial contractility and also inhibit coronary vasoconstriction. Percutaneous transluminal coronary angioplasty (PTCA) or coronary artery bypass graft surgery (CABG; see below) are often used to treat medically refractory angina.

Medical treatment of MI. The longer a coronary artery remains blocked, the more damage there is to the myocardium; fast response to an MI is critical. On reaching the emergency room, a clot-dissolving (thrombolytic) agent (e.g., streptokinase (SK), urokinase (UK), tissue plasminogen activator (TPA)) is sometimes administered. Primary (direct) PTCA or emergency CABG surgery may immediately follow to also improve perfusion. Aspirin can reduce clotting mechanisms and stabilize plaques, reducing mortality and recurrence over the long term; short-term heparin use may help prevent reocclusion; long-term warfarin (Coumadin) use may reduce the chance of clots. Tissue recovery may be
promoted by nitrates and beta-blockers; angiotensin-converting enzyme (ACE) inhibitors (e.g., captopril (Capoten), enalapril (Vasotec)) are vasodilators and also improve ventricular function. Follow-up treatment may also include PTCA or CABG surgery.

**Percutaneous transluminal coronary angioplasty (PTCA).** PTCA (balloon angioplasty) to dilate narrowed arteries is often used in patients with CAD. A guide wire is passed through the stenotic region of the coronary artery and a catheter with a narrow balloon on the end is threaded through the guide wire. The balloon is then inflated to compress the plaque and enlarge the lumen. Restenosis occurs in about 25% of patients within six months. Stents (mechanical devices to hold the lumen open), other catheterization procedures such as removing the plaque itself (atherectomy), anticoagulant medications (e.g., clopidogrel bisulfate (Plavix)), and sometimes radiation are used to reduce the likelihood of restenosis.

**Coronary artery bypass graft surgery (CABG).** As previously noted, plaques almost always occur in well-defined locations. CABG surgery uses the patient’s own saphenous vein to construct a pathway to bypass the stenosis. Multiple bypass grafts are typically constructed using one left internal mammary artery (LIMA) and several saphenous vein grafts (SVGs) as needed from the upper or lower leg. A recent variation on traditional CABG procedure is the minimally invasive coronary artery bypass (Mid CAB), which involves entering between the ribs instead of cutting the sternum and is a less physically stressful surgical procedure. Some CABG procedures are associated with increased risk of stroke.

CABG surgery undoubtedly relieves angina pain and improves exercise tolerance. It is less clear whether there is a significant increase in lifespan for CABG patients over those treated medically, although some subgroups such as patients with three vessel disease and left main disease do appear to live longer (Myers, Blackstone, Davis, Foster & Kaiser, 1999; Chaitman, Ryan, Kronmal, Foster et al., 1990)

**Trans-myocardial laser revascularization (TMLR).** This new procedure uses a laser to punch holes in areas of the myocardium which have been ischemically impaired. The myocardium is then
perfused with blood directly from the inner chamber of the heart. TMLR is currently under investigation as an alternative treatment for patients with angina that is unable to be medically treated.

Heart Failure and Transplant

Heart failure results when the heart is damaged or overworked. Congestive heart failure (CHF) is the most common presentation, occurring in about 4,790,000 Americans and resulting in 50,824 deaths in 1999, an increase of almost 22% since 1993. It occurs when the heart is unable to pump efficiently. Blood returning to the heart backs up in the lungs and veins, causing shortness of breath (dyspnea) on exertion, swelling (edema), and inability to lie down (orthopnea). In CHF, the kidneys are no longer as efficient in removing sodium and water from the body, which aggravates the condition further. The New York Heart Association Functional Classification of heart failure is in Table 2.

Cardiomyopathy refers to a variety of diseases of the heart muscle. It occurs in over 3,000,000 Americans and was responsible for 27,260 deaths in 1999. Cardiomyopathy may be caused by ischemic heart disease, MI, congenital defects, hypertension, valvular disease, chronic alcohol use, or pulmonary hypertension. Dilated cardiomyopathy refers to an enlargement and weakening of the heart. Hypertrophic cardiomyopathy refers to a thickening of the left ventricle and the resulting dysfunction.

The area of the heart affected by cardiomyopathy or CHF will determine the symptoms reported. The right side of the heart leads to the lungs; right-sided heart failure results in fluid retention, edema, and dyspnea on exertion. The left side of the heart leads to the rest of the body; left-sided heart failure is associated with higher mortality than right-sided, and tends to produce pulmonary vascular congestion, dyspnea, orthopnea, and physical fatigue.

Diagnostic Procedures

Heart failure is usually diagnosed with assessment of physical symptoms, exercise stress test,
echocardiogram, electrocardiogram, and chest x-ray. Of particular concern is identification of the functional classification of CHF, its anatomical classification, and precipitating cause. One key element is the ejection fraction (EF), or how much of the blood in the heart is expelled by the heart's contraction. A normal EF is 55-65%; heart transplant is usually considered in patients with an EF of 25% or lower and other clinical prognostic factors.

Medical and Surgical Treatment

**Medical treatment.** If specific causes of CHF (e.g., hypertension, abnormal valves, etc.) can be identified, their treatment may result in improvement of the CHF. Non-pharmacologic treatments such as salt and fluid restriction, and changes in physical activity level, may be sufficient for non-symptomatic Class I heart failure. Pharmacologic therapy for symptomatic CHF generally takes three forms: ACE inhibitors, diuretics (e.g., furosemide (Lasix)) which reduce the body's salt and fluid load, and inotropic agents (e.g., dopamine, dobutamine, amrinone, milrinone, digoxin (Lanoxin)), which improve the heart's pumping action. Vasodilators such as ACE inhibitors dilate blood vessels and decrease the blood pressure, reducing the effort required by the heart. Recent studies have also shown benefits from beta-blockers. Some of these medications cannot be administered adequately at home, and may require the patient to visit an infusion clinic at a hospital several times a week for four to eight hours.

**Heart transplantation.** A heart transplant may be considered when the myocardium is irreversibly and seriously damaged from cardiomyopathy, does not respond adequately to medical treatment, is unlikely to benefit from a bypass operation, and puts the patient at risk of dying. A total of 2,198 heart transplants were performed in the United States in 2000. However, 40,000 patients under the age of 65 (the usual cut-off age) could benefit from a transplant each year. The one-year survival rate for heart transplant is 84%, the three-year rate is 77%, and 69% of transplant patients survive for five years. Transplant patients typically must take eight to 12 medications per day for the rest of their lives to reduce the likelihood of rejection of the transplanted heart, combat infection, and control blood pressure. Many
of these (see Table 5) may have significant effects on mood and mental status.

An alternative to transplantation currently under investigation for some cardiomyopathy patients is ventricular reduction surgery. Also, a left ventricular assist device (LVAD) can be used to temporarily assist ventricular function while a patient is waiting for a heart, and may in some cases be placed in patients who are not eligible for transplantation. Although this mechanical pump requires 24 hour monitoring, it may permit patients to leave the hospital and resume many of their normal activities.

Other Heart Disease

Valvular Disease

There are about 97,000 hospitalizations per year for valvular disease, nearly all for aortic or mitral valve disorders. Aortic valve disorders have about four times the mortality of mitral valve disorders. Valvular problems often cause dyspnea, fatigue or chest pain. They almost always produce additional heart sounds such as snaps or clicks, alteration in the intensity or timing of heart sounds, or heart murmurs resulting from turbulence caused by malformed valves.

Key types include aortic and mitral stenosis, aortic and mitral regurgitation (of which mitral valve prolapse is a common type and often associated with anxiety disorders), pulmonary valve disease, and tricuspid valve disease. Causes of valvular disease include infections such as rheumatic fever and congenital disorders of the valves. Medical intervention includes digoxin and inotropic medications. Surgical interventions involve valve replacement (with both porcine and mechanical valves) and surgically reconstructing the valve structure.

Arrhythmias

The electrical impulse that produces a heartbeat begins in the sinoatrial (SA or sinus) node of the heart. The impulse is transmitted to the atrial and ventricular muscle cells with precise timing, but can sometimes become disordered by: 1.) abnormal electrolytes such as potassium, magnesium, and calcium; 2.) addictive substances such as alcohol, nicotine, and recreational drugs; 3.) medications such as digoxin
and theophylline; and 4.) other causes.

About 4.5 million Americans have arrhythmias, or irregular heartbeats, with 39,262 dying in 1999 of directly related problems. However, the great majority of the approximately 250,000 sudden cardiac deaths each year are also attributed to a type of arrhythmia, ventricular fibrillation. Bradycardia refers to a heart rate of less than 60 beats per minute; tachycardia refers to a heart rate of more than 100 per minute. Transient arrhythmias may not be picked up by a short-term ECG and may require a 24-hour or longer assessment with a Holter monitor.

Key types of arrhythmias include sinus arrhythmia, premature atrial contractions (PACs), premature ventricular contractions (PVCs), and atrial fibrillation. Atrial fibrillation is treated with electrical (and sometimes chemical) cardioversion to restore a normal heartbeat. Arrhythmias may be managed by medical therapy, or by a variety of implanted electrical cardiac pacemakers.

Pericardial Disease

The pericardium consists of a loose fibrous membrane covering the myocardium and a dense sac surrounding the heart that is filled with fluid and supports the heart within the chest. Pericardial disease most commonly results from infection, and symptoms can mimic an MI or CHF.

Endocarditis

Bacterial endocarditis results when an infection in the bloodstream begins to grow on valves of the heart. There are about 17,000 hospitalizations per year for endocarditis. Bacteria may enter the bloodstream during dental, gastrointestinal or urological procedures. The infection may grow for weeks or months before endocarditis is detected, and patients usually report symptoms of fever and fatigue. Treatment requires long-term high-dose antibiotics, and surgery is often necessary despite antibiotic therapy.

Aortic Aneurysms

About 63,000 hospitalizations per year occur for aortic aneurysms, with over a quarter of these
cases resulting in death. An aneurysm is a ballooning (or pouching out) of the wall of a vein, artery, or part of the heart due to disease, injury or congenital abnormality. An aneurysm is especially critical in the aorta, which carries blood from the heart to the rest of the body. Treatment usually addresses high blood pressure and surgical patch of the weakened part of the artery.

**Stroke**

Stroke may be a complication of an MI, CABG surgery or surgery to remove plaques in the carotid arteries (endarterectomy), peripheral vascular disease, and other cardiac problems. A cerebral thrombosis or embolism is caused by a clot that blocks blood flow to the brain, and often results in the permanent death of brain tissue and loss of function. A transient ischemic attack (TIA) is a "mini" stroke that has minimal or transient effect on brain function. A subarachnoid or cerebral hemorrhage is caused by a burst blood vessel or aneurysm and may have more transient effects, since once the pressure on the brain is relieved, brain function may return.

Symptoms of stroke that may affect quality of life and functional ability are similar to the warning signs of stroke as outlined by the American Heart Association (2001b), which include:

* Sudden weakness or numbness of the face, arm, or leg on one side of the body.
* Sudden dimness or loss of vision, particularly in one eye.
* Loss of speech, or trouble talking or understanding speech.
* Sudden, severe headaches with no known cause.
* Unexplained dizziness, unsteadiness, or sudden falls.

**PSYCHOLOGICAL ASSESSMENT AND TREATMENT**

**Common Referral Questions**

In a cardiology practice or preventive cardiology clinic, the standard psychological referral questions concern: 1.) prevention associated with behavioral cardiovascular risk factors such as smoking,
eating habits, and exercise; 2.) adherence to medical treatment and cardiac rehabilitation; and 3.) psychopathology and coping difficulties. Other specific referral questions may be noted by the physician, nurse practitioner, nutritionist or other health professional who sees the patient.

Referrals for Primary, Secondary and Tertiary Prevention

The perception of benefits, barriers and motivation affecting the patient's seeking and adhering to treatment and making healthy behavioral changes differs for primary, secondary, and tertiary prevention patients. For primary prevention patients, long-term lifestyle or medication control of risk factors (e.g., dyslipidemia) takes place in the absence of disease symptoms. Such patients frequently feel less urgency to address their risk factors, and are at higher risk of nonadherence to preventive medical treatment. Secondary prevention cardiology patients are symptomatic (e.g., with angina) without having had an acute event. Tertiary patients have diagnosed disease (e.g., cardiomyopathy) or have had an acute event (e.g., an MI). Although both secondary and tertiary prevention patients may have more initial motivation to engage in preventive treatment, successful long-term adherence to medication and a healthy lifestyle often depends on other psychological and behavioral factors.

Clinicians who follow a stages of change approach to behavioral change (Prochaska, Redding & Evers, 1997) need to make referral sources aware of the goals of stages of change intervention. Moving patients to a higher stage (e.g., precontemplation to contemplation) may temporarily be the only attainable goal for some patients. Since referring physicians may be expecting a more concrete behavioral outcome, however, they need to be informed of the patient's progress and the rationale for a stages of change intervention.

Referrals for Treatment Adherence

Adherence to treatment is a common concern and source of referrals in cardiology and preventive cardiology. However, although physicians will identify and refer patients for the most obvious cases of non-adherence, they can generally predict their patients' adherence to treatment no better than chance
(Roth & Caran, 1978), and frequently are not aware of factors in their patients' lives and experiences with medical caregivers that influence adherence. It is therefore necessary to keep adherence as an issue in any patient referral. Furthermore, although adherence to treatment is important in all areas of medical care, it is absolutely critical for two groups of cardiology patients: preventive cardiology patients, and heart failure patients, particularly those who are post-transplant.

In preventive cardiology patients, sustained long-term change to control risk factors is the only medically meaningful behavioral outcome. Short-term adherence to lifestyle changes or medication to manage chronic risk factors is essentially a failure of treatment. Similarly, in CHF patients a strict regimen of dietary and fluid restrictions and medication maintenance is necessary to control the progression of disease. Furthermore, successful adherence to a pre-transplant medical regimen is a prerequisite for heart transplant, since post-transplant patients are asked to maintain essentially perfect adherence to a complex treatment and monitoring regimen for the rest of their lives. Approaches to addressing long-term adherence is discussed later in this chapter.

Referrals for Psychopathology and Coping

Heart patients are generally good at hiding their poor adjustment to their illness. Heart patients tend to be older, and include more males than the traditional psychotherapy population. Both factors are associated with minimization of illness difficulties and resistance to recommendations for psychological services (Rybarczyk, 1994). Their resistance is aggravated by the lack of self-selection for psychological treatment.

For a successful referral for coping issues or psychopathology to occur with such patients, it is necessary to have the support of the referring physician. It is best for the physician to be forthright in explaining the reasons for the referral to the patient. In doing so, the physician may benefit from coaching by the psychologist to characterize psychological services accurately and non-threateningly, to underscore that psychological evaluation is both routine and necessary to provide the best medical care,
and to deal with the patient's initial resistance (Bellg, 1993). Having the physician personally introduce the psychologist to the patient reinforces the physician's support for psychological services as part of the patient's medical care.

**Assessment**

Communication of findings and recommendations among members of a multidisciplinary treatment team is a key part of the process in caring for heart patients. The perspectives and language of cardiologists, nutritionists, exercise physiologists, and even consulting psychiatrists is significantly different than that of psychologists. It is therefore necessary for psychologist to develop familiarity with the treatment team's language and goals for patients. Furthermore, a psychological clinical interview often generates additional information that the treatment team may be unaware of, such as personal history or personally sensitive treatment side effects (e.g., sexual dysfunction), which needs to be communicated in the context of the broader goals for care of the patient. It is therefore helpful for the psychologist to be aware of the needs and language of the treatment team and the referring physician in case presentations, reports and chart notes.

**General Cardiac Psychology Assessment**

**Screening questionnaires.** Questionnaires may be useful to evaluate psychopathology symptoms, physical symptoms and functional ability, current life stressors, motivational style, perceived social support, or other areas of interest to the clinician. For sensitive issues such as substance abuse, patients are less likely to provide accurate information on a questionnaire and such issues are better explored in an interview. In the preventive cardiology clinic where I work, patients come to the clinic seven to 10 days prior to their appointment to have blood drawn for a lipid profile and other lab tests. The most effective screening strategy has been to present them at that time with questionnaires from the nutritionist, the exercise physiologist, and the psychologist to fill out at home. The questionnaires are then either mailed back or brought to the clinic on the day of the appointment.
Given the likely initial resistance of heart patients to psychology, the questionnaire burden is kept relatively low. The MMPI-2 and other extensive or too overtly “psychological” questionnaires that may evoke patient resistance are not administered for this reason. The two-page screening questionnaires we currently use begin with demographic information and an informal checklist of psychological symptoms, behavioral risk factors and life stressors. Checklist items are explored further in the interview and also provide a rough estimate of questionnaire self-report accuracy when compared with interview data. On the second page, patients fill out the 14-item Hospital Anxiety and Depression Scale (HADS; Zigmond & Snaith, 1983) to identify possible anxiety and depression, an 8-item social support scale adapted from the Interpersonal Support Evaluation List (ISEL; Cohen & Hoberman, 1983) to identify the perceived availability of emotional and tangible support relevant to the patient’s illness or medical care, and two questions addressing previous treatment for mental health problems or for alcohol or drug use.

Other options that clinicians may wish to consider include the Brief Symptom Inventory (BSI; Derogatis, 1993), with 53 items in 10 subscales, as an overall screen for psychopathology; the Center for Epidemiological Studies Depression Scale (CESD; Radloff, 1977), slightly modified to a single page format (this may be preferable to the Beck Depression Inventory (BDI; Beck, 1972) in medical patients since it relies less on vegetative symptoms to assess depression); or the RAND 36-Item Health Survey Short Form (SF-36; Ware & Sherbourne, 1992) as a measure of patient functioning and quality of life in a variety of areas. Behavioral assessment questionnaires for specific behaviors (e.g., daily food records, exercise diaries) may also be helpful if the psychologist does not have the benefit of working with other professionals in these areas. Questionnaires and subscales are listed in Table 3.

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Clinical interview outline. At our clinic, the interview with the psychologist generally follows evaluation by the nutritionist, exercise physiologist, and physician. Prior to the interview, the
psychologist is briefed by the other professionals on their encounters with and recommendations for the patient. Ideally, the psychologist has information from the questionnaires and the three other professionals prior to seeing the patient. In practice, the psychological interview often begins with less information than this.

Keeping in mind the need for long-term behavioral and psychological risk factor management, the interview needs to lay the foundation for a potential long-term (though perhaps intermittent) relationship with the patient. Discussion of the patient's reaction and adjustment to their illness and treatment is generally the entry point to the interview, and provides a context for developing empathy and rapport. Topics raised by the patient are pursued in depth, and additional topics are covered to complete the assessment. Interview topics include:

- Patient reaction and adjustment to illness and treatment, and the patient's perceptions of their caregivers.
- Treatment side effects and adherence, health-related lifestyle issues, health beliefs (e.g., benefits, barriers) regarding medication and lifestyle recommendations.
- Stage of change and motivation for risk factor modification.
- Coping style and resources such as social support, spiritual beliefs or practices, recreational activities.
- Accuracy of patient understanding of other health care professionals' recommendations.
- Symptoms of psychopathology.
- Substance use and dependence (alcohol, drugs, nicotine).
- Mental status and cognitive functioning.

Both anxiety and depression are common in heart patients, and depression may be found in 18 to 44 percent of cardiac patients over the course of their disease (Fernandez, 1993). Symptoms of psychopathology also may be atypical in these patients. Major depression manifesting as irritability or
anger outbursts is more common than the average, as are anxiety disorders masked by legitimate illness concerns. Vegetative symptoms of depression must also be distinguished from illness symptoms or medication side effects. Psychiatric consultation with a psychiatrist familiar with heart patients and their medications and illness symptoms should be readily available if necessary.

Cognitive status may have been influenced by anoxia or an embolism, or more subtly by a TIA. A formal cognitive status screening exam is conducted with most secondary and tertiary prevention patients unless successful engagement in challenging life activities and patient report clearly indicates no functional impairment. Either the Neurobehavioral Cognitive Status Examination (NCSE; Northern California Neurobehavioral Group, 1988) or the Modified Mini-Mental State Exam (3MSE, an improvement on the commonly used Folstein Mini-Mental State Exam; Teng & Chui, 1987) may be used to screen for moderate or gross impairment of cognitive function. Neuropsychological evaluation may be appropriate for patients with more severe deficits.

Heart Transplant Assessment

In addition to a standard evaluation, potential heart transplant candidates require a more detailed assessment of factors that may affect their ability to take care of a new heart.

- History of adherence to medical treatment and tolerance of medication side effects.
- History of adherence to recommended lifestyle behavior changes. For instance, transplant patients often have difficulty controlling their weight following restoration of energy and return of appetite, which increases their medical risks.
- Patient attitudes toward medical caregivers, including trust and issues related to authority.
- Willingness and ability to change behaviors associated with substance abuse and dependence. Transplant programs typically require a person to be abstinent from questionable substances for at least six months to qualify for a transplant.
- Availability and capability of the patient's spouse, family, or companion to make a long term
commitment to provide emotional and tangible support for the tasks associated with transplant (e.g., medication adherence, transportation to clinic).

- Ability to cope with major life changes.

Commonly agreed upon contraindications to transplantation include active suicidal ideation, current drug or alcohol use, acute psychosis, irreversible brain damage, and chronic non-adherence to treatment (Levinson & Olbrisch, 1993; Mai, 1993; Frierson & Lippman, 1987). Personality disorders generally do not rule out transplantation unless there is behavioral evidence that they affect the patient’s ability to participate and engage successfully in their medical care. Poor treatment adherence is particularly important, and is responsible for a significant percentage of transplant rejections and up to 25% of deaths following the post-operative period (Dew, Roth, Thompson et al., 1996).

**Psychosocial Risk Factor Assessment**

As biological pathways for the action of psychological factors on the pathogenesis of CVD become more explicit and supported by experimental evidence, the association of psychosocial factors with CVD risk becomes more persuasive. Such pathways now include platelet and neuroendocrine activation, increased hypercholesterolemia, arrhythmogenesis, endothelial dysfunction and injury, and sympathetic nervous system activation exacerbated by individual differences (Rozanski, Blumenthal & Kaplan, 1999; Allan & Scheidt, 1996b). At this time, there are numerous studies showing a connection between psychosocial factors and CAD (e.g., Nunes, Frank & Kornfield, 1987), MI onset (e.g., Mittleman, Maclure, Sherwood et al., 1995), post-MI hospitalization time (e.g., Allison, Williams, Miller, Patten et al., 1995) and post-MI survival (e.g., Thomas, Friedmann, Wimbush & Schron, 1997). Furthermore, the addition of interventions for reducing psychosocial risk factors has an independent effect in reducing morbidity and mortality beyond that of standard cardiac rehabilitation regimens (Linden, Stossel & Maurice, 1996). Across a range of studies, the effects of psychosocial intervention have been found to be of similar or greater magnitude to those of medical preventive treatment in reducing non-fatal
MIs and cardiac deaths in secondary prevention (Ketterer, 1993).

**Depression.** Assessment of psychosocial risks might start with a clinical interview for a diagnosis of clinical depression; depression has been shown to increase risk of mortality by five to six times within half a year following an MI (Frasure-Smith, Lesperance & Talajic, 1993). Non-clinical levels of depression on the BDI (scores ≥ 10) have also been associated with increased mortality at 18 months (Frasure-Smith, Lesperance & Talajic, 1995). Global emotional distress on the BSI is associated with increased risk of hospitalization within six months of an MI in cardiac rehabilitation patients (Allison et al., 1995). Although preliminary results of a major clinical trial of a cognitive intervention for depression and social isolation in CAD patients indicate no benefit on mortality or acute events (the ENRICHD trial; Blumenthal, 2002), results may have been influenced by minimal differences in depression change between the intervention and control groups.

**Stress.** Stress and job strain appear to influence CAD through a variety of biological pathways (Allan & Scheidt, 1996b). In particular, increased ischemia (mostly silent) has been associated with stressful daily life events (e.g., Barry, Selwyn, Nable, et al., 1988), and vasoconstriction has been demonstrated with psychological stressors (e.g., Feigl, 1987). Lipid levels can also be altered by neurogenic and behavioral stress, and may be of concern in people whose work or lives result in chronic stress (see Rosenman, 1997, for a review). Numerous animal studies have found increased atherosclerosis in stressed animals, although these results have been difficult to generalize to humans (Kaplan, Manuck, Williams & Strawn, 1993). Cardiovascular reactivity to stress has also become an active research area, particularly in identifying at-risk subgroups that may be more susceptible to the effect of stressful events on CVD. For instance, carotid atherosclerosis has been show to progress more in men who have large changes in blood pressure in anticipation of a maximal exercise test (Everson, Lynch, Chesney, Kaplan et al., 1997); reactivity has also been measured in reaction to other stressors (e.g., Smith, Nealey, Kircher & Limon, 1997). Gender differences are also being explored, with men
appearing to be more reactive to stressful tasks than women (Lawler, Wilcox & Anderson, 1995).

**Type A behavior.** Type A behavior pattern (TAPB) was initially defined as a drive for achievement, high competitiveness, involvement in multiple activities, a tendency to increase the rate of activities, extreme mental and physical alertness, and pervasive aggressive and hostile feelings (Rosenman, Swan & Carmelli, 1988). Although studies of TAPB's association with coronary events have shown mixed results overall, group treatment of TAPB in the Recurrent Coronary Prevention Project lowered the rate of non-fatal coronary recurrences by 44% in post-MI patients; patients who markedly reduced their TAPB were substantially less likely to have a recurrence than those who did not reduce their TAPB (Friedman, Thoresen, Gill, Ulmer, Powell, Price, Brown, Thompson, Rabin, Breall, Bourg, Levy & Dixon, 1986). In terms of assessment, TAPB has been a better predictor of cardiovascular events when identified by an interview than a questionnaire (Allan & Scheidt, 1996b), and the latest tool for identifying TAPB is the videotaped clinical examination (VCE; Friedman, Fleischmann & Price, 1996). The hostility component of TAPB has been a stronger predictor of CHD events than a dichotomous characterization of Type A vs. Type B (Thoresen & Bracke, 1997). A hostility measure that assesses both affective and behavioral components is the Interpersonal Hostility Assessment Technique, which has been related to CAD severity (Barefoot, 1992).

**Social isolation.** Measured in terms of emotional support from close people and an extended social network, social isolation increased risk of acute events by a factor of four in a male Swedish population followed for six years (Orth-Gomer, Rosengren & Wilhelmsen, 1993). Other prospective studies of patients with CHD have also found increased risk of nonfatal or fatal events from living alone (Case, Moss, Case, McDermott & Eberly, 1992) and increased mortality from being unmarried and without a close confidant (Williams, Barefoot & Califf, 1992). Social isolation and support may also interact with other factors such as TAPB and depression.

**Treatment**
Approaches to Long-term Adherence and Health Behavior Change

**Treatment adherence.** Risk factor management and treatment adherence issues cannot be separated, and need to be addressed at all levels of the health care system. The American Heart Association has issued a “Multilevel Compliance Challenge” to patients, healthcare providers, and healthcare organizations to adopt strategies to improve the delivery of health care and behavioral risk-reducing strategies for heart patients (Miller, Hill, Kottke & Ockene, 1999). It has long been noted that treatment adherence has a significant effect on treatment efficacy; for example, patients who were more than 75% adherent to a lipid-lowering medication had 22% fewer coronary events than the average in the West of Scotland Coronary Prevention (WOSCOPS) study (Shepherd, 1996). Adherence may be influenced by general factors such as cognitive impairment and psychopathology, and by specific factors (e.g., stage of change, health beliefs regarding barriers, benefits and self-efficacy) associated with a particular medication or lifestyle change (see Bellg, Rivkin & Rosenson, 2002, for a review). Treatments that do not resolve patient-perceived symptoms and that also have significant side effects (e.g., hypertension treatment) also are associated with extremely low adherence (Sackett & Snow, 1979).

The main challenge for those promoting treatment adherence and healthy lifestyle change is that long-term maintenance of health behavior remains elusive. For example, patient adherence to cardiac rehabilitation services including exercise, dietary change, smoking cessation, and taking prescribed medications is only 25-40% after six months (U.S. Department of Health and Human Services, 1996). Adherence is consequently gaining increased attention, as evidenced by a symposium sponsored by the American Heart Association and the National Institutes of Health examining compliance issues and cardiac risk factor control (Burke & Ockene, 2001), and by an issue of *Health Psychology* [vol. 19, number 1 (Suppl.); January, 2000] devoted to papers from a workshop held by the National Heart, Lung and Blood Institute (NHLBI) specifically focusing on maintenance of weight loss, smoking cessation, physical activity, and dietary behavior. The closing paper from that issue concluded that the NHLBI
conference provided “a starting point (italics mine) for research on long-term maintenance of behavior change” (Wing, 2000). Clearly, there is much work to be done in this area.

Part of the strategy to improve delivery of risk-reducing interventions is to shift focus from short-term to long-term behavior change, and a variety of innovative approaches to accomplish this are being tested as of this writing (e.g., Behavior Change Consortium, 2002) or have been developed in the past decade (see Health Behavior and Health Education for a review; Glanz, Lewis & Rimer, 1997). The following theoretical and practical approaches have focused in different ways on this issue.

**Stages of change approaches.** Before patients can change their behavior, they often have to change their readiness to change. Stage of change (SOC) assessment and intervention (Prochaska, Redding & Evers, 1997; Prochaska, Velicer, Rossi, Goldstein et al., 1994) has been valuable in assisting this process. With cardiac patients, the stages of change model has been particularly useful in two areas. First, because of their general resistance to psychological treatment, the SOC model is helpful in assessing readiness to engage in psychological intervention for behavior change. Second, the SOC model can define readiness to engage in the specific behavioral goals of treatment (rather than as a preliminary global assessment of readiness to achieve general outcome goals). For instance, a patient who has been advised to quit smoking may be in the contemplation phase in relation to that general outcome goal, but may be in the action phase in terms of willingness to attend psychological sessions or in being willing to change to a brand of cigarettes with less nicotine.

**Motivational approaches.** Developing positive motivation for change is a key area for intervention. However, even the health benefits of making a change are often initially perceived as largely unsought and unpleasant advice. In helping patients internalize and integrate lifestyle changes, self-determined behaviors associated with positively experienced benefits are more likely to be sustained than extrinsically motivated behaviors experienced as being coerced by circumstances or other people (Botelho & Skinner, 1995; Bellg, Williams, Deci & Suchman, 1991), perhaps because they may be

Motivational interviewing is an effective way to implement a motivational approach. It is “a directive, client-centered counseling style for eliciting behavior change by helping clients to explore and resolve ambivalence” (Rollnick & Miller, 1995), and has been effectively adopted by therapists dealing with substance abuse and other behavioral problems such as risky sexual practices, compliance with medical recommendations, and eating disorders. Within this approach, there are five clinical strategies: 1) express empathy, 2) develop discrepancy, 3) avoid argumentation, 4) roll with resistance, and 5) support self-efficacy (Miller & Rollnick, 1991), all of which are intended to help patients identify and act upon their own reasons for engaging in a new behavior.

Cognitive-behavioral approaches. Although traditional cognitive-behavioral approaches are generally effective at helping patients initiate change, they have not been as successful in producing sustained health behavior change. Nonetheless, some behavioral and cognitive techniques have worked well. For instance, behavioral self-management strategies involving a case management approach with personal contact and active long-term follow-up have been highly effective at promoting long-term change in CAD patients (Debusk, Miller, Superko, Dennis et al., 1994), although they require considerable resources and repeated patient contact. A case management telephone intervention has also been found highly successful with chronic
heart failure patients at reducing rehospitalization rates and patient care costs (Riegel, Carlson, Kopp, LePetri, et al., 2002).

In addition, cognitive strategies based on the Health Belief Model remain essential for understanding how patients think about their illness and how they engage with the health care system (Becker & Maiman, 1975; Strecher & Rosenstock, 1997). For instance, the perceived threat of cardiovascular disease is minimized by some patients, and the perceived benefits of preventive treatment may be unclear. Patients may also have barrier beliefs regarding medicines or a specific medicine (e.g., becoming “dependent” on it), concerns about medication interactions or taking too many medications, or misunderstandings about treatment (e.g., "When my labs are normal, I can quit my meds"). As for beliefs about healthy lifestyle change, misinformation is common (e.g., patients often are not aware that a modest weight loss or increase in exercise can be medically beneficial, or that even infrequent smoking increases risk of sudden cardiac death). Poor lifestyle assessment by patients is also common (e.g., patients often overestimate the amount of exercise they get on their job), and personal and cultural barrier beliefs about lifestyle changes may also need to be discussed (e.g., "If you're too thin, you're not healthy"), as will peer group beliefs and pressures. Finally, self-efficacy beliefs are important and are highly predictive of initial effort in making change (Turk & Meichenbaum, 1991). Low self-efficacy can be changed over time with education and positive experiences of success.

The following are some additional approaches to promote successful behavioral change in cardiac patients. These may be considered as ways to solve particular short-term behavioral problems, and need to be used within the context of a more comprehensive approach that facilitates long-term health behavior maintenance.
* Use counter-conditioning to deal with cravings. Relaxation training, distraction interventions, and substitute behaviors can help patients deal with urges for food or cigarettes.
* Develop stimulus control strategies. Help patients avoid tempting situations and manage their environments to reduce associations with negative behaviors.
* Reinforce positive behavior. Patient selection of a positive reward for success in achieving or maintaining a moderately difficult behavioral goal can support short-term change. Making the reinforcer relatively small but personal and genuinely appealing will help the process of internalization and support long-term change.

**Risk Factor Management**

**Managing behavioral risk factors.** Although the above approaches to health behavior change and risk factor management are very promising and currently under investigation, more traditional health behavior change theories and strategies have shaped many of the approaches to risk factor management currently being used. Some of the processes involved in behavioral interventions for smoking, weight loss and cardiac rehabilitation are briefly described below.

Most smokers who eventually quit have tried more than once. There are few clinically meaningful differences in behavioral treatment options, with overall one-year quit rates of about 30% (Lichtenstein & Glasgow, 1992). Typical treatments include setting a "quit date," nicotine fading or use of nicotine replacement strategies, use of medications such as Zyban (bupropion), and group support or individual follow-up during initial phases of abstinence. More recent clinical trials show greater likelihood of smoking abstinence when behavioral treatments (e.g., Cinciripini, Cinciripini, Wallfisch, Haque & Van Vunakis, 1996) or minimum contact self-help materials (Fortmann & Killen, 1995) are combined with nicotine supplements (i.e., transdermal patch, gum, or nasal spray). Nicotine supplements are particularly effective among depressed patients (Kinnunen, Doherty, Militello & Garvey, 1996). Nicotine supplements do not require a prescription, and do not increase cardiac events in patients with
cardiac disease (Joseph, Norman, Ferry, Prochaska et al., 1996).

Achieving a reasonable weight goal can decrease blood pressure and risk of CAD. Abdominal fat is a particular risk factor for coronary events, and weight gain also needs to be addressed in many patients following heart transplantation. Controlling behavioral factors influencing weight is difficult, and regaining lost weight is highly likely. Key factors to prevent weight regain involve developing a regular exercise regimen (e.g., Dubbert, 1992) and finding acceptable substitutes for favorite foods that are unhealthy. Long-term medication interventions must be viewed with caution, particularly since some appetite-suppressant drugs in the mid-1990’s (e.g., Redux, "fen-fen," fenfluramine) were associated with substantially increased risk of primary pulmonary hypertension, an uncommon but serious disease (Abenhaim, Moride, Brenot et al., 1996). Dietary changes that lower weight and improve other cardiovascular risks often focus on lowering fat and calories, and increasing fruits, vegetables and legumes (Singh, Rastogi, Rastogi et al., 1996).

Many of the principles for modifying single risk factors apply in promoting patient involvement and adherence to a cardiac rehabilitation program, which can influence a variety of coronary risk factors (Lavie & Milani, 1996). Patient motivation is often affected by their rehabilitation status. Phase 1 rehabilitation involves inpatients, and resistance often includes fear of further damage to the heart or feeling debilitated and frustrated by illness and treatment symptoms. Intervention focuses on improving coping and promoting self-efficacy by giving patients experiences that allow them to see their activity level improve. Phase 2 cardiac rehabilitation patients are in a specific, structured rehabilitation program to aid recovery following an acute event. Intervention often involves addressing barriers to treatment such as inconvenience, feeling different than the other cardiac rehabilitation patients (e.g., younger patients often feel out of place), having conflictual interactions with the rehabilitation staff, being uncomfortable with the experience of exercise, and having fatalistic beliefs ("if your number is up, it doesn't matter what you do"). Phase 3 cardiac rehabilitation involves ongoing maintenance of fitness.
status and healthy lifestyle behaviors, and is often conducted at home with only occasional visits to the rehabilitation center. Promoting adherence with Phase 3 patients is similar to promoting it for non-rehabilitation patients, with the benefit that they are receiving regular monitoring and feedback on their cardiovascular status.

Managing psychosocial risk factors. Although there is evidence that stress, Type A behavior, depression and social isolation are risk factors for CVD of magnitude approaching that of biomedical risks, there have been fewer intervention studies showing that psychosocial disease risks may be reduced. However, a meta-analysis of studies examining interventions with psychosocial risk factors (Ketterer, 1993) showed positive effects on cardiovascular outcomes for treatment ranging from group therapy for TABP, group psychotherapy, stress monitoring, behavioral treatment, and relaxation training. Non-significant or negative intervention effects were found in educational discussion groups and exercise therapy counseling. A range of interventions in individual and group formats (Spira, 1997) formats therefore appears to be effective in addressing psychosocial risk factors.

One intervention addressing both the behavioral and psychosocial risks of heart disease was the Lifestyle Heart Trial (Ornish, Brown, Scherwitz, Billings et al., 1990). Patients in this study showed good adherence to rigorous regimen of lifestyle changes even after four years, as well as reduction of stenoses and fewer acute events. The program consisted of a low-fat (approximately 10%) vegetarian diet, moderate aerobic exercise, stress management, and group support. Along with adherence issues, the groups were designed to address social isolation, improve communication skills, promote empathy and compassion, and encourage identification and expression of feelings (Billings, Scherwitz, Sullivan, Sparler & Ornish, 1996). Other psychosocial risk factor interventions have also been found to effectively reduce coronary prone behaviors, incorporating such strategies as "the hook" to raise awareness of the distinction between situations ("bait") and responses ("hooks"), daily drills to change one TABP behavior per day, strategies to improve relationships and social support, and relaxation techniques (Thoresen &
Bracke, 1997).

Case Example #1. A 43 year old male cardiac rehabilitation patient was referred for non-adherence to recommended lifestyle changes to help control hypercholesterolemia and HTN, which were also being treated medically with no side effects. He had a significant family history of heart disease, with his father and uncle dying of MI's in their 50's, and a grandmother dying in her 60's. He tended to minimize the implications of his familial history of illness.

Initial attempts to change his diet through nutritional counseling were ineffective. He came to cardiac rehabilitation exercise sessions for three weeks, and then quit after he got a high pressure job selling and leasing commercial real estate. His eating habits were impulsive, and often included fast food. When he had lunches and dinners with colleagues, however, he was able to order appropriate low-fat meals. He had been an athlete in college, but hadn't exercised regularly for 20 years. He also had many Type A characteristics: impatience, many projects going simultaneously, and explosive anger at subordinates. He was married, with two children, many acquaintances and a few close friends he saw infrequently. He did not smoke; he drank five drinks a week socially with colleagues and clients, and two six-packs of beer on weekends.

His risk reduction outcome goals consisted of obtaining better control over diet, increasing his exercise level, cutting his alcohol consumption, and reducing his TABP. Rapport was established easily, but developing a therapeutic alliance and agreeing on behavioral goals was more difficult. He made it clear he wanted to make changes without feeling he was depriving himself. Since he impulsively went to the same fast food restaurant near his office, he decided to change to a restaurant where he could order soups and salads he enjoyed. His fast food excursions dropped from daily to twice a week.

He also decided to walk up stairs at work at least twice a day instead of taking the elevator, and play softball on weekends. Although this was not a therapeutic level of exercise, he agreed that developing an exercise habit would be a first step toward making more effective change. After a month
of fair success at this, he began to take a daily 30 minute walk before work.

He was unwilling to change his drinking habits, since he felt that alcohol was an important part of his social life. Consequently, an initial thought of having him evaluated for an SSRI for his anger was postponed.

Over three months, his TABP changed slightly. He spent several weeks noting situations in which he was angry with his associates, and rated his anger at those times. He was surprised at the frequency of his anger. He was encouraged to verbalize his affection for his wife and children, and identify positive characteristics of his colleagues and give them more positive feedback. He initially had a difficult time with this, although he improved. He planned more unscheduled time during the day and on weekends, developed a practice of walking more slowly, and took a class in T'ai Chi.

He terminated treatment after nine sessions, deciding to resume periodically when he felt he needed help maintaining control of his lifestyle behaviors.

Psychopathology and Adjustment to Illness

Coping with illness symptoms and treatment side effects. The pain, discomfort, and other symptoms of an MI may cause a great deal of fear and anxiety. Many patients also become sensitized to them, developing anxiety at each minor pain, and put constraints on their activities far more than may be justified by their illness. Some constraints may be justified, of course, and these often are associated with loss and a period of resentment or grieving. Cardiac medications may also produce uncomfortable physical and psychological side effects. Table 4 lists psychological side effects of cardiac medications, including those that are relatively infrequent. Surgical interventions such as CABG can also produce substantial postoperative changes in quality of life, mood, and cognitive status (Gold, 1996).

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Insert Table 4 about here
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Coping with these difficult experiences can be aided with a variety of techniques. Cognitive
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restructuring (Beck & Emery, 1979) may be used to defuse helplessness and negative ideation associated with catastrophizing about symptoms. Assertiveness training may be effective in supporting the patient's self-efficacy in being able to deal with symptoms or treatment side effects. When alternatives to medications producing uncomfortable side effects are not available, patient tolerance may be increased by developing coping strategies to minimize the effect of specific symptoms (e.g., planning activities during times when medication-related drowsiness is not a problem). Post-operative coping may be fostered through promoting realistic expectations for postoperative functional status and time to recovery, social support from family and friends, and CBT treatment for depression and other psychological symptoms (Gold, 1996). Group therapy to enhance coping may provide a supportive context for patients, facilitate emotional expression, promote active coping, and help patients reprioritize their goals and usual activities (Spira, 1997b).

In addition, psychologists can help patients cope with the difficulties of being an inpatient. Patients in a coronary care unit (CCU) may receive excellent care, but being surrounded by and connected to machines can be frightening. Patients also often feel depersonalized by hospital personnel and procedures. As anonymous inpatients, they become disconnected from the parts of their lives and their personal identities that have given them strength in the past (Rybarczyk & Bellg, 1997). The psychologist can encourage active coping by the patient and educate the staff on ways to promote patient choice and activity. Hospitalized patients with acute MIs have shorter stays when they are given information about their condition and allowed to participate in their care (Cromwell & Levenkron, 1984). Other ways to buffer a negative hospital experience may involve facilitating and encouraging social support for the patient, identifying reasons for "bad" patient behavior (e.g., neediness, non-adherence) and communicating them to the staff, and conducting CBT around thoughts and feelings evoked by the hospital experience.

Psychopathology. For many patients, the diagnosis of CAD, the occurrence of an MI, or the
chronic experience of symptoms of angina may trigger psychological processes leading to clinical depression or anxiety. Psychopathology may be effectively treated in these patients with a variety of cognitive-behavioral (e.g., Beck, Rush, Shaw & Emery, 1979; Beck & Emery, 1979), or other brief interpersonal approaches such as solution-focused therapy (Walter & Peller, 1992). Difficulties in treatment are similar to those in diagnosis, with amelioration of symptoms of irritability or anger, social withdrawal, anhedonia and disinterest in usual activities as likely treatment goals for depression. Patterns of anxious ideation also need to be distinguished from reasonable concern and worry about their illness to help the patient develop awareness and monitor his or her progress. Referral sources need to be educated regarding appropriate patient response to illness and psychopathology, since physicians sometimes erroneously believe that if patients have a reason to be depressed or anxious, there is no reason to refer them for treatment.

Cardiac patients as a group tend to be older and have a larger proportion of men than the traditional psychotherapy population. As a result, presenting patients with an instrumental characterization of therapy ("to help you deal better with your situation") as opposed to a subjective one ("to help you feel better") is likely to be more successful in building a therapeutic alliance. Similarly, brief therapeutic approaches that pursue treatment with a goal clearly in mind are likely to be more successful, although patients can get considerable benefit just by being able to tell their stories to a receptive listener (Rybarczyk & Bellg, 1997). By encouraging reinvolvement in activities and social connections, therapeutic benefits can continue beyond the session.

Use of psychotropic medications is clearly indicated with many patients, and working with a psychiatrist who is familiar with cardiac patients and cardiac medications is a great benefit. You may also wish to consult with a psychiatrist regarding the use of SSRIs with hostile, TABP patients who are not clinically depressed. Hostility and impatience can be reduced in many such patients, even in the absence of vegetative symptoms of depression (Littman, 1993; Tollefson, 1995).
Precautions with psychotropic medications. Psychiatric monitoring of possible side effects from psychotropic medications in cardiac patients is important. However, it may be necessary to identify problematic psychotropic medications prescribed prior to diagnosis of heart problems. A few of the most serious precautions (from Tabrizi, Littman, Williams & Scheidt, 1995) are noted below.

* Tricyclic antidepressants (TCIs) are life-threatening in patients at risk of overdose (e.g., suicidal or cognitively impaired patients). They may also result in tachycardia during early phases of therapy, orthostatic hypotension, and ECG alterations.

* Trazodone (Desyrel) can cause orthostatic hypotension, which generally contraindicates its use in cardiac patients.

* Benzodiazepines (e.g., alprazolam [Xanax], lorazepam [Ativan], triazolam [Halcion]) may be helpful in controlling panic attacks, anxiety or insomnia, but can also cause delirium. Overdoses are life-threatening; withdrawal must be tapered, and withdrawal symptoms may include tachycardia.

* Lithium is relatively safe in patients with heart disease, but serum levels may be difficult to control in post-transplant patients; cyclosporin can raise levels, and prednisone can decrease levels.

* Phenothiazine overdoses (e.g., Thorazine, Mellaril) can produce life-threatening arrhythmias.

SSRIs (notably fluoxetine [Prozac], sertraline [Zoloft], paroxetine [Paxil], citalopram [Celexa], and fluvoxamine [Luvox], among others) are generally safe in cardiac patients, although they can interact with and elevate levels of other medications such as beta-blockers, warfarin, and digoxin. Sertraline appears to be the least likely to cause such elevations. Bupropion (Wellbutrin) and mirtazapine (Remeron) are also non-cardiotoxic antidepressants, and are associated with less sexual dysfunction than SSRIs; however, mirtazapine may cause weight gain and elevated lipid levels. Buspirone (Buspar) can be used to treat generalized anxiety disorder and symptoms of hostility and irritability in cardiac patients.
Valproic acid has no cardiac effects and may be an option for treatment of mania.

**Case Example #2.** A 68-year-old woman with hypercholesterolemia was referred to evaluate her recent weight gain. She had a TIA four months previously, and reported temporary impairment of short-term memory and ability to concentrate. She was on aspirin therapy and had successfully taken cholesterol-lowering medication.

She had gained 25 lbs. (to reach 180) in the previous two months. One year previously, she retired from a 70 hour per week practice as an architect. She had an active, driven personal style, and had difficulty adjusting to the relative inactivity. She tried to remain busy during the day by working as a hospital volunteer, but felt "useless" and didn't want to be around sick people. After her TIA, she became less interested in her usual activities, and began to binge on "anything in sight," mainly cookies and high calorie foods. In the previous week, she had also binged on brandy and orange juice, drinking approximately 12 oz. of brandy with orange juice in two days. She had no previous history of an eating disorder or a drinking problem.

She reported no current difficulty with her memory or concentration. Her thoughts were logical, but often somewhat tangential during the interview; her speech was coherent but mildly dysarthric. She reported being anxious and worried about her future, and denied feeling sad or having hopeless/helpless thoughts. She slept poorly, with difficult sleep onset due to "thinking too much" and several nocturnal awakenings with difficulty returning to sleep. Her depression scores on the intake questionnaires were only slightly elevated, but her Somatization score and the overall General Severity Index were substantially elevated on the Brief Symptom Inventory. She had two friends she could talk to "about anything," but both lived out of town.

She was diagnosed with Adjustment Disorder with Depressed Mood, and was interested in participating in psychotherapy. CBT included cognitive restructuring about her TIA, which had upset her so much she didn't want to discuss it during the first session, and cognitive reframing of her retirement to
encourage her to be more active. She also learned a relaxation and meditation exercise, which she enjoyed and was willing to practice twice a day. Regarding her binges, after some negotiation, she agreed to sit while eating, pause frequently to ask herself if she wanted to continue, and eat slowly to enjoy the food as much as possible. She was also encouraged to develop new friends and social contacts.

Within a month, most of her depressive symptoms had resolved. Her ability to fall asleep easily and sleep through the night gradually improved; her binging was reduced in quantity and frequency immediately, and disappeared shortly after her sleep improved. She got involved in several arts and cultural groups that allowed her to build social relationships and take more of the leadership role she had been used to when working. She also reestablished friendships from her former job.

Other Treatment Issues

Treatment of non-cardiac chest pain. From 10 to 30% of patients with apparent symptoms of angina, and up to 60% of patients with other chest pain, have normal angiograms (Kemp, Vokonas, Cohn & Gorlin, 1973; Cannon, Quyyumi, Minemoyer, Stine et al., 1994). Patients with panic disorders or clinical anxiety frequently experience chest pain as one of the diagnostic symptoms, as do patients with gastroesophageal reflux disease (GERD). Following five to 11 sessions of CBT for anxiety and pain management in patients experiencing more than four episodes of chest pain per week, about one-third reported no symptoms, and another third reported fewer than four episodes (Klimas, Mayhou, Pearce, Coles & Fagg, 1990). Imipramine has produced a 52% reduction in episodes of non-cardiac chest pain (Cannon et al., 1994). Behavioral and psychopharmacological treatments for anxiety and panic disorders appear to be effective with many of these patients.

Case Example #3. A 55 year old man was referred by the heart failure team to evaluate psychological reasons for his frequent visits to the ER. He had dilated cardiomyopathy and CAD, had suffered an MI and was treated with PTCA two years previously. He had been listed for cardiac transplant, but improved under medical therapy and cardiac rehabilitation and was removed from the
He had made 13 visits to the ER since his MI, each time complaining of overwhelming diffuse chest pain, stomach pains and fear of dying; no physical cause was found for these events. His chest pain always lessened and sometimes disappeared upon entering the ER, where he felt safe and believed "nothing could happen." He also was having "anxiety attacks" about twice a week, with fear and racing thoughts but no chest pain. He had a history of panic disorder that eight years previously been successfully treated with imipramine. He also had a history since his early 20's of fearing that he would be incontinent in public after he witnessed his grandfather's incontinence; as a result, he felt anxious and on the verge of incontinence whenever a restroom was unavailable. He had never had an incident, however.

He was highly somatically focused and tended to catastrophize about even minor symptoms. Following his MI, a physician told him that "30% of my heart is dead," which worried him constantly. Even so, he was able to describe numerous distinctions between the non-cardiac pain that sent him to the ER and the pain he had experienced during his MI. His symptoms were also different, however, than those he had experienced previously as panic attacks, which made it difficult for him initially to connect them with anxiety.

Therapy focused on relaxation training with a somatic focus (progressive relaxation training and abdominal breathing) to reduce and give him control over his physical symptoms of anxiety and panic, and cognitive therapy to help him distinguish panic from cardiac symptoms and defuse the negative attributions he associated with his symptoms. Also, after psychiatric evaluation, he was placed on Paxil 20 mg QD. Within two months his "anxiety attacks" had disappeared, and in the next six months he had one episode of chest pain which he was able to distinguish as non-cardiac and control with his relaxation exercises. He had no further visits to the ER. Without it being a specific focus of therapy, he also obtained almost complete relief of his fear of incontinence.
PROFESSIONAL PRACTICE ISSUES

Age issues in psychological treatment. Cardiac patients and preventive cardiology patients tend to be middle-aged and older men and women. Such patients may be less likely to follow through with psychological treatment for illness adjustment problems, and may present with specific treatment difficulties in relation to health behavior change, seeing behavioral intervention as being contrary to their desire to be self-reliant. They are likely to be less experienced with and interested in psychological perspectives, less articulate about their internal experiences and feelings (with alexithymia resulting in reduced social skills [Lumley, Ovies, Stettner, Wehmer & Lakey, 1996]), and less inclined to look to others for support (Rybarczyk, 1994). However, chronological age is not a good predictor of emotional distress following an MI, and treatment expectations for cooperative older patients should not be lowered.

Gender issues. Gender plays an important role in identifying and treating risk factors for cardiac disease and dealing with its psychological consequences. Men have role barriers to identifying, expressing, and seeking help for behavior change and for psychopathology, including depression (Warren, 1983); white males over the age of 60 are four times more likely to commit suicide than any other group (National Center for Health Statistics, 1987). However, because men tend to be affected by heart disease earlier in their lives, women’s concerns in prevention and treatment of heart disease have been underemphasized until recently and need to be specifically addressed. Surveys repeatedly show that women are more concerned about breast cancer than heart disease, even though only 1 in 30 women dies of breast cancer, and 1 in 2.4 women dies of cardiovascular disease (American Heart Association, 2001b). Women may consequently be less likely to participate in screening for cardiovascular disease and to address CVD risk factors. Women have a poorer prognosis following an MI than men (American Heart Association, 2001b) and certain risk factors such as smoking are more significant for women than men; a first MI occurs approximately seven years earlier in smoking men than nonsmoking men, but 19 years earlier in smoking women than nonsmoking women (Hansen, Anderson & Von Eyben, 1993). Women
have also been underrepresented in treatment studies and in studies of psychosocial factors affecting CVD outcomes (e.g., Frasure-Smith, Lesperance & Talajic, 1995), in part because of lower illness rates in study populations including women. Jacobs and Sherwood (1996) note that men and women differ in cardiovascular reactivity, triggering events for MI, and types of stressors associated with CVD. There is a clear need to include more women in treatment and prevention studies and better understand cardiovascular risks among women (see Tsang, Barnes, Gersh & Hayes, 2000, for an excellent review of women’s risk factors).

**Family therapy with heart patients.** Some risk factors such as familial hypercholesterolemia (FH) run in families. Getting the cooperation of parents to change family patterns of eating or exercise in such cases is essential to making changes in behavioral patterns in children that may be already well-established at a young age. Patients under 10 years old are more amenable to their parents' input on meals, particularly if they see their parents eating in the same way. Teenagers have different challenges in dealing with lifestyle changes than do younger siblings or adults, such as peer pressure, developmental issues related to self-definition and self-acceptance, poor stimulus control, or frightened parents with controlling styles. Family therapy approaches are also useful in dealing with an acute health crisis or chronic illness in a family member, particularly when some family members are coping better than others or are carrying more of the caregiving burden than they can handle.

**Development of new services.** As was noted in the first edition of this chapter published in 1998, the clinical practice of cardiac psychology holds great promise, but is clearly underdeveloped. The same statement is true today. The current practice and future potential of cardiac psychology rests on the three components discussed in this chapter: 1.) the generally under-utilized role of psychologists in treating traditional medical and behavioral cardiac risk factors, 2.) the growing body of research supporting assessment and treatment of a range of psychosocial cardiac risk factors, and 3.) the well-established role of psychologists in promoting illness adjustment and treating psychopathology.
There are many ways to advance the practice and profession of cardiac psychology. Identifying and treating patients associated with increased medical care costs, such as those with non-cardiac chest pain and psychosocial risk factors, can provide both a valuable service in a managed care environment and add to the professional credibility of the field. Publishing research in medical publications as well as in psychological journals will add to our interdisciplinary credibility as well. The field remains open for entrepreneurial health psychologists with the appropriate training to make contact with cardiology practices and hospital-based clinics and establish themselves – and their profession – as essential to the proper care of cardiac patients.
REFERENCES


### Table 1

**Risk Factors for Atherosclerosis and Related Clinical Syndromes: Angina, MI, and Sudden Cardiac Death**

**Unchangeable risk factors**
- Increasing age
- Male gender
- Heredity

**Changeable risk factors (per AHA)**
- Smoking
- High overall or "bad" cholesterol (low density lipoprotein; LDL)
- Low "good" cholesterol (high density lipoprotein; HDL)
- Hypertension
- Diabetes mellitus
- Sedentary lifestyle
- Obesity 35% or more above ideal body weight
- Individual response to stress

**Other changeable risk factors**
- Pre-symptomatic atherosclerosis (per EBCT)
- Type A behavior, notably hostility
- Clinical depression
- Social isolation
- High triglycerides
- High blood viscosity

Adapted from American Heart Association (2001b) and Scheidt (1996).
Table 2

**New York Heart Association (NYHA) Functional Classification in Patients with Cardiac Disease**

<table>
<thead>
<tr>
<th>Class</th>
<th>Description</th>
</tr>
</thead>
<tbody>
<tr>
<td>Class I:</td>
<td>No symptoms with ordinary activity, no limitations on activity.</td>
</tr>
<tr>
<td>Class II:</td>
<td>Slight to moderate symptoms with normal activity, slight limitation of activity; patient is comfortable at rest.</td>
</tr>
<tr>
<td>Class III:</td>
<td>Moderate symptoms with less than normal activity, marked limitation of activity.</td>
</tr>
<tr>
<td>Class IV:</td>
<td>Inability to carry on any physical activity without discomfort; symptoms may occur at rest.</td>
</tr>
</tbody>
</table>
### Table 3

#### Suggested Screening and Evaluation Instruments for Heart Patients

**Screening Questionnaires**

- **Demographic Information**
- **Checklist of Psychological Symptoms, Risk Factor Behaviors and Life Stressors**
- **Hospital Anxiety and Depression Scale (HADS, 14 items; Zigmond & Snaith, 1983)**
- **Interpersonal Support Evaluation List (ISEL, 8 items; Cohen & Hoberman, 1983)**

**Other Questionnaires**

- **Brief Symptom Inventory (BSI, 53 items; Derogatis, 1993)**
  - Subscales: Depression, Interpersonal sensitivity, Anxiety, Phobic anxiety, Hostility, Paranoid ideation, Somatization, Psychoticism, Obsessive-Compulsive, Other items
- **Center for Epidemiology Studies Depression Scale (CESD, 20 items; Radloff, 1977)**
- **RAND 36-Item Health Survey Short Form (SF-36, 36 items; Ware & Sherbourne, 1992)**
  - Subscales: Physical functioning, Role function affected by physical symptoms, Role function affected by emotional symptoms, Social functioning, Pain, General health, Health change, Energy/fatigue, Emotional well-being

**Cognitive Assessment During Clinical Interview**

- **Neurobehavioral Cognitive Status Exam (NCSE; Northern California Neurobehavioral Group (1988), or**
- **Modified Mini-Mental State Examination (3MS; Teng & Chui, 1987)**
<table>
<thead>
<tr>
<th>Medication Class</th>
<th>Psychological Side Effects</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Preventive Treatments</strong></td>
<td></td>
</tr>
<tr>
<td>Antihypertensives</td>
<td>depression, anxiety, sleep disturbances, sexual dysfunction</td>
</tr>
<tr>
<td>Lipid-lowering agents</td>
<td>depression, anxiety, sleep disturbances</td>
</tr>
<tr>
<td><strong>Treatments for CAD</strong></td>
<td></td>
</tr>
<tr>
<td>Vasodilators</td>
<td>headache</td>
</tr>
<tr>
<td>Beta-blockers</td>
<td>depression, nightmares, insomnia, loss of libido, fatigue, delirium</td>
</tr>
<tr>
<td>ACE inhibitors</td>
<td>mania</td>
</tr>
<tr>
<td>Anticoagulants</td>
<td>none</td>
</tr>
<tr>
<td>Antiarrhythmics</td>
<td>depression related to thyroid dysfunction</td>
</tr>
<tr>
<td>Inotropic agents</td>
<td>sexual dysfunction, anxiety, delirium</td>
</tr>
<tr>
<td><strong>Treatments for Heart Failure/Transplant</strong></td>
<td></td>
</tr>
<tr>
<td>Immunosuppressants</td>
<td>anxiety, delirium, hallucinations, delusions</td>
</tr>
<tr>
<td>Corticosteroids</td>
<td>anxiety, depression, increased appetite, weight gain, agitation, emotional lability, hypomania, insomnia, apathy, suicidal ideation, irritability, delirium</td>
</tr>
<tr>
<td>Sympathomimetics</td>
<td>mania, delirium</td>
</tr>
<tr>
<td>Antibiotics</td>
<td>hallucinations, fear of death, delirium</td>
</tr>
</tbody>
</table>

(continued next page)
Table 5, continued.

<table>
<thead>
<tr>
<th>Class</th>
<th>Symptoms</th>
</tr>
</thead>
<tbody>
<tr>
<td>Antiviral drugs</td>
<td>irritability, hallucinations, delirium</td>
</tr>
<tr>
<td>Antifungal drugs</td>
<td>depression, hallucinations</td>
</tr>
</tbody>
</table>

Incidence of side effects may be low, and not all drugs in each class are associated with the indicated symptoms. Adapted from Tabrizi et al. (1996), Scheidt (1996), Wise & Rundell (1994), and Heather (1997).