Social Security

Medical/Professional Relations

Disability Evaluation Under Social Security

(Blue Book- October 2008)

4.00 Cardiovascular System - Adult

Section

4.01
Category of Impairments, Cardiovascular System

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4.00 Cardiovascular System

A. General

1. *What do we mean by a cardiovascular impairment?*

a. We mean any disorder that affects the proper functioning of the heart or the circulatory system (that is, arteries, veins, capillaries, and the lymphatic drainage). The disorder can be congenital or acquired.

b. Cardiovascular impairment results from one or more of four consequences of heart disease:

(i) Chronic heart failure or ventricular dysfunction.

(ii) Discomfort or pain due to myocardial ischemia, with or without necrosis of heart muscle.

(iii) Syncope, or near syncope, due to inadequate cerebral perfusion from any cardiac cause, such as obstruction of flow or disturbance in rhythm or conduction resulting in inadequate cardiac output.

(iv) Central cyanosis due to right-to-left shunt, reduced oxygen concentration in the arterial blood, or pulmonary vascular disease.

c. Disorders of the veins or arteries (for example, obstruction, rupture, or aneurysm) may cause impairments of the lower extremities (peripheral vascular disease), the central nervous system, the eyes, the kidneys, and other organs. We will evaluate peripheral vascular disease under 4.11 or 4.12 and impairments of another body system(s) under the listings for that body system(s).

2. *What do we consider in evaluating cardiovascular impairments?* The listings in this section describe cardiovascular impairments based on symptoms, signs, laboratory findings, response to a regimen of prescribed treatment, and functional limitations.

3. *What do the following terms or phrases mean in these listings?*

a. *Medical consultant* is an individual defined in §§ 404.1616(a) and 416.1016(a). This term does not include medical sources who provide consultative examinations for us. We use the abbreviation "MC" throughout this section to designate a medical consultant.

b. *Persistent* means that the longitudinal clinical record shows that, with few exceptions, the required finding(s) has been present, or is expected to be present, for a continuous period of at least 12 months, such that a pattern of continuing severity is established.

c. *Recurrent* means that the longitudinal clinical record shows that, within a consecutive 12-month period, the finding(s) occurs at least three times, with intervening periods of improvement of sufficient duration that it is clear that separate events are involved.

d. *Appropriate medically acceptable imaging* means that the technique used is the proper one to evaluate and diagnose the impairment and is commonly recognized as accurate for assessing the cited finding.

e. *A consecutive 12-month period* means a period of 12 consecutive months, all or part of which must occur within the period we are considering in connection with an application or continuing disability review.

f. *Uncontrolled* means the impairment does not adequately respond to standard prescribed medical treatment.

B. *Documenting Cardiovascular Impairment*

1. *What basic documentation do we need?* We need sufficiently detailed reports of history, physical examinations, laboratory studies, and any prescribed treatment and response to allow us to assess the severity and duration of your cardiovascular impairment. A longitudinal clinical record covering a period of not less than 3 months of observations and treatment is usually necessary, unless we can make a determination or decision based on the current evidence.

2. *Why is a longitudinal clinical record important?* We will usually need a longitudinal clinical record to assess the severity and expected duration of your impairment(s). If you have a listing-level impairment, you probably will have received medically prescribed treatment. Whenever there is evidence of such treatment, your longitudinal clinical record should include a description of the ongoing management and evaluation provided by your treating or other medical source. It should also include your response to this medical management, as well as information about the nature and severity of your impairment. The record will provide us with information on your functional status over an extended period of time and show whether your ability to function is improving, worsening, or unchanging.

3. *What if you have not received ongoing medical treatment?*

a. You may not have received ongoing treatment or have an ongoing relationship with the medical

community despite the existence of a severe impairment(s). In this situation, we will base our evaluation on the current objective medical evidence and the other evidence we have. If you do not receive treatment, you cannot show an impairment that meets the criteria of most of these listings. However, we may find you disabled because you have another impairment(s) that in combination with your cardiovascular impairment medically equals the severity of a listed impairment or based on consideration of your residual functional capacity and age, education, and work experience.

b. Unless we can decide your claim favorably on the basis of the current evidence, a longitudinal record is still important. In rare instances where there is no or insufficient longitudinal evidence, we may purchase a consultative examination(s) to help us establish the severity and duration of your impairment.

4. *When will we wait before we ask for more evidence?*

a. We will wait when we have information showing that your impairment is not yet stable and the expected change in your impairment might affect our determination or decision. In these situations, we need to wait to properly evaluate the severity and duration of your impairment during a stable period. Examples of when we might wait are:

(i) If you have had a recent acute event; for example, a myocardial infarction (heart attack).

(ii) If you have recently had a corrective cardiac procedure; for example, coronary artery bypass grafting.

(iii) If you have started new drug therapy and your response to this treatment has not yet been established; for example, beta-blocker therapy for dilated congestive cardiomyopathy.

b. In these situations, we will obtain more evidence 3 months following the event before we evaluate your impairment. However, we will not wait if we have enough information to make a determination or decision based on all of the relevant evidence in your case.

5. *Will we purchase any studies?* In appropriate situations, we will purchase studies necessary to substantiate the diagnosis or to document the severity of your impairment, generally after we have evaluated the medical and other evidence we already have. We will not purchase studies involving exercise testing if there is significant risk involved or if there is another medical reason not to perform the test. We will follow sections 4.00C6, 4.00C7, and 4.00C8 when we decide whether to purchase exercise testing.

6. *What studies will we not purchase?* We will not purchase any studies involving cardiac catheterization, such as coronary angiography, arteriograms, or electrophysiological studies. However, if the results of catheterization are part of the existing evidence we have, we will consider them together with the other relevant evidence. See 4.00C15a.

C. *Using Cardiovascular Test Results*

1. *What is an ECG?*

a. *ECG* stands for *electrocardiograph* or *electrocardiogram*. An electrocardiograph is a machine that records electrical impulses of your heart on a strip of paper called an electrocardiogram or a *tracing*. To record the ECG, a technician positions a number of small contacts (or *leads*) on your arms, legs, and across your chest to connect them to the ECG machine. An ECG may be done while you are resting or exercising.

b. The ECG tracing may indicate that you have a heart abnormality. It may indicate that your heart muscle is not getting as much oxygen as it needs (ischemia), that your heart rhythm is abnormal (arrhythmia), or that there are other abnormalities of your heart, such as left ventricular enlargement.

2. *How do we evaluate ECG evidence?* We consider a number of factors when we evaluate ECG evidence:

a. An original or legible copy of the 12-lead ECG obtained at rest must be appropriately dated and labeled, with the standardization inscribed on the tracing. Alteration in standardization of specific leads (such as to accommodate large QRS amplitudes) must be identified on those leads.

(i) Detailed descriptions or computer-averaged signals without original or legible copies of the ECG as described in listing 4.00C2a are not acceptable.

(ii) The effects of drugs or electrolyte abnormalities must be considered as possible noncardiac causes of ECG abnormalities of ventricular repolarization; that is, those involving the ST segment and T wave. If available, the predrug (especially digitalis glycosides) ECG should be submitted.

b. ECGs obtained in conjunction with treadmill, bicycle, or arm exercise tests should meet the following specifications:

(i) ECG reports must include the original calibrated ECG tracings or a legible copy.

(ii) A 12-lead baseline ECG must be recorded in the upright position before exercise.

(iii) A 12-lead ECG should be recorded at the end of each minute of exercise.

(iv) If ECG documentation of the effects of hyperventilation is obtained, the exercise test should be deferred for at least 10 minutes because metabolic changes of hyperventilation may alter the physiologic and ECG-recorded response to exercise.

(v) Post-exercise ECGs should be recorded using a generally accepted protocol consistent with the prevailing state of medical knowledge and clinical practice.

(vi) All resting, exercise, and recovery ECG strips must have the standardization inscribed on the

tracing. The ECG strips should be labeled to indicate the date, the times recorded and the relationship to the stage of the exercise protocol. The speed and grade (treadmill test) or work rate (bicycle or arm ergometric test) should be recorded. The highest level of exercise achieved, heart rate and blood pressure levels during testing, and the reason(s) for terminating the test (including limiting signs or symptoms) must be recorded.

3. *What are exercise tests and what are they used for?*

a. Exercise tests have you perform physical activity and record how your cardiovascular system responds. Exercise tests usually involve walking on a treadmill, but other forms of exercise, such as an exercise bicycle or an arm exercise machine, may be used. Exercise testing may be done for various reasons; such as to evaluate the severity of your coronary artery disease or peripheral vascular disease or to evaluate your progress after a cardiac procedure or an acute event, like a myocardial infarction (heart attack). Exercise testing is the most widely used testing for identifying the presence of myocardial ischemia and for estimating maximal aerobic capacity (usually expressed in METs—metabolic equivalents) if you have heart disease.

b. We include exercise tolerance test (ETT) criteria in 4.02B3 (chronic heart failure) and 4.04A (ischemic heart disease). To meet the ETT criteria in these listings, the ETT must be a sign-or symptom-limited test in which you exercise while connected to an ECG until you develop a sign or symptom that indicates that you have exercised as much as is considered safe for you.

c. In 4.12B, we also refer to exercise testing for peripheral vascular disease. In this test, you walk on a treadmill, usually for a specified period of time, and the individual who administers the test measures the effect of exercise on the flow of blood in your legs, usually by using ultrasound. The test is also called an exercise Doppler test. Even though this test is intended to evaluate peripheral vascular disease, it will be stopped for your safety if you develop abnormal signs or symptoms because of heart disease.

d. Each type of test is done in a certain way following specific criteria, called a *protocol*. For our program, we also specify certain aspects of how any exercise test we purchase is to be done. See 4.00C10 and 4.00C17.

4. *Do ETTs have limitations?* An ETT provides an estimate of aerobic capacity for walking on a grade, bicycling, or moving one's arms in an environmentally controlled setting. Therefore, ETT results do not correlate with the ability to perform other types of exertional activities, such as lifting and carrying heavy loads, and do not provide an estimate of the ability to perform activities required for work in all possible work environments or throughout a workday. Also, certain medications (such as beta blockers) and conduction disorders (such as left or right bundle branch blocks) can cause false-negative or false-positive results. Therefore, we must consider the results of an ETT together with all the other relevant evidence in your case record.

5. *How does an ETT with measurement of maximal or peak oxygen uptake (VO2) differ from other*

ETTs? Occasionally, medical evidence will include the results of an ETT with VO2. While ETTs without measurement of VO2 provide only an estimate of aerobic capacity, measured maximal or peak oxygen uptake provides an accurate measurement of aerobic capacity, which is often expressed in METs (metabolic equivalents). The MET level may not be indicated in the report of attained maximal or peak VO2 testing, but can be calculated as follows: 1 MET = 3.5 milliliters (ml) of oxygen uptake per kilogram (kg) of body weight per minute. For example, a 70 kg (154 lb.) individual who achieves a maximal or peak VO2 of 1225 ml in 1 minute has attained 5 METs (1225 ml/70 kg/1 min = 17.5 ml/kg/min. 17.5/3.5 = 5 METs).

6. *When will we consider whether to purchase an exercise test?*

a. We will consider whether to purchase an exercise test when:

(i) There is a question whether your cardiovascular impairment meets or medically equals the severity of one of the listings, or there is no timely test in the evidence we have (see 4.00C9), and we cannot find you disabled on some other basis; or

(ii) We need to assess your residual functional capacity and there is insufficient evidence in the record to make a determination or decision.

b. We will not purchase an exercise test when we can make our determination or decision based on the evidence we already have.

7. *What must we do before purchasing an exercise test?*

a. Before we purchase an exercise test, an MC, preferably one with experience in the care of patients with cardiovascular disease, must review the pertinent history, physical examinations, and laboratory tests that we have to determine whether the test would present a significant risk to you or if there is some other medical reason not to purchase the test (see 4.00C8).

b. If you are under the care of a treating source (see §§ 404.1502 and 416.902) for a cardiovascular impairment, this source has not performed an exercise test, and there are no reported significant risks to testing, we will request a statement from that source explaining why it was not done or should not be done before we decide whether we will purchase the test.

c. The MC, in accordance with the regulations and other instructions on consultative examinations, will generally give great weight to the treating source's opinion about the risk of exercise testing to you and will generally not override it. In the rare situation in which the MC does override the treating source's opinion, the MC must prepare a written rationale documenting the reasons for overriding the opinion.

d. If you do not have a treating source or we cannot obtain a statement from your treating source, the MC is responsible for assessing the risk to exercise testing based on a review of the records we have before purchasing an exercise test for you.

e. We must also provide your records to the medical source who performs the exercise test for review prior to conducting the test if the source does not already have them. The medical source who performs the exercise test has the ultimate responsibility for deciding whether you would be at risk.

8. *When will we not purchase an exercise test or wait before we purchase an exercise test?*

a. We will not purchase an exercise test when an MC finds that you have one of the following significant risk factors:

(i) Unstable angina not previously stabilized by medical treatment.

(ii) Uncontrolled cardiac arrhythmias causing symptoms or hemodynamic compromise.

(iii) An implanted cardiac defibrillator.

(iv) Symptomatic severe aortic stenosis.

(v) Uncontrolled symptomatic heart failure.

(vi) Aortic dissection.

(vii) Severe pulmonary hypertension (pulmonary artery systolic pressure greater than 60 mm Hg).

(viii) Left main coronary stenosis of 50 percent or greater that has not been bypassed.

(ix) Moderate stenotic valvular disease with a systolic gradient across the aortic valve of 50 mm Hg or greater.

(x) Severe arterial hypertension (systolic greater than 200 mm Hg or diastolic greater than 110 mm Hg).

(xi) Hypertrophic cardiomyopathy with a systolic gradient of 50 mm Hg or greater.

b. We also will not purchase an exercise test when you are prevented from performing exercise testing due to another impairment affecting your ability to use your arms and legs.

c. We will not purchase an ETT to document the presence of a cardiac arrhythmia.

d. We will wait to purchase an exercise test until 3 months after you have had one of the following events. This will allow for maximal, attainable restoration of functional capacity.

(i) Acute myocardial infarction.

(ii) Surgical myocardial revascularization (bypass surgery).

(iii) Other open-heart surgical procedures.

(iv) Percutaneous transluminal coronary angioplasty with or without stenting.

e. If you are deconditioned after an extended period of bedrest or inactivity and could improve with activity, or if you are in acute heart failure and are expected to improve with treatment, we will wait an appropriate period of time for you to recuperate before we purchase an exercise test.

9. *What do we mean by a "timely" test?*

a. We consider exercise test results to be timely for 12 months after the date they are performed, provided there has been no change in your clinical status that may alter the severity of your cardiovascular impairment.

b. However, an exercise test that is older than 12 months, especially an abnormal one, can still provide information important to our adjudication. For example, a test that is more than 12 months old can provide evidence of ischemic heart disease or peripheral vascular disease, information on decreased aerobic capacity, or information about the duration or onset of your impairment. Such tests can be an important component of the longitudinal record.

c. When we evaluate a test that is more than 12 months old, we must consider the results in the context of all the relevant evidence, including why the test was performed and whether there has been an intervening event or improvement or worsening of your impairment.

d. We will purchase a new exercise test only if we cannot make a determination or decision based on the evidence we have.

10. *How must ETTs we purchase be performed?*

a. The ETT must be a sign- or symptom-limited test characterized by a progressive multistage regimen. It must be performed using a generally accepted protocol consistent with the prevailing state of medical knowledge and clinical practice. A description of the protocol that was followed must be provided, and the test must meet the requirements of 4.00C2b and this section. A radionuclide perfusion scan may be useful for detecting or confirming ischemia when resting ECG abnormalities, medications, or other factors may decrease the accuracy of ECG interpretation of ischemia. (The perfusion imaging is done at the termination of exercise, which may be at a higher MET level than that at which ischemia first occurs. If the imaging confirms the presence of reversible ischemia, the exercise ECG may be useful for detecting the MET level at which ischemia initially appeared.) Exercise tests may also be performed using echocardiography to detect stress-induced ischemia and left ventricular dysfunction (see 4.00C12 and 4.00C13).

b. The exercise test must be paced to your capabilities and be performed following the generally accepted standards for adult exercise test laboratories. With a treadmill test, the speed, grade (incline), and duration of exercise must be recorded for each exercise test stage performed. Other exercise test protocols or techniques should use similar workloads. The exercise protocol may need

to be modified in individual cases to allow for a lower initial workload with more slowly graded increments than the standard Bruce protocol.

c. Levels of exercise must be described in terms of workload and duration of each stage; for example, treadmill speed and grade, or bicycle ergometer work rate in kpm/min or watts.

d. The exercise laboratory's physical environment, staffing, and equipment must meet the generally accepted standards for adult exercise test laboratories.

11. *How do we evaluate ETT results?* We evaluate ETT results on the basis of the work level at which the test becomes abnormal, as documented by onset of signs or symptoms and any ECG or imaging abnormalities. The absence of an ischemic response on an ETT alone does not exclude the diagnosis of ischemic heart disease. We must consider the results of an ETT in the context of all of the other evidence in your case record.

12. *When are ETTs done with imaging?* When resting ECG abnormalities preclude interpretation of ETT tracings relative to ischemia, a radionuclide (for example, thallium-201 or technetium-99m) perfusion scan or echocardiography in conjunction with an ETT provides better results. You may have resting ECG abnormalities when you have a conduction defect—for example, Wolff-Parkinson-White syndrome, left bundle branch block, left ventricular hypertrophy—or when you are taking digitalis or other antiarrhythmic drugs, or when resting ST changes are present. Also, these techniques can provide a reliable estimate of ejection fraction.

13. *Will we purchase ETTs with imaging?* We may purchase an ETT with imaging in your case after an MC, preferably one with experience in the care of patients with cardiovascular disease, has reviewed your medical history and physical examination, any report(s) of appropriate medically acceptable imaging, ECGs, and other appropriate tests. We will consider purchasing an ETT with imaging when other information we have is not adequate for us to assess whether you have severe ventricular dysfunction or myocardial ischemia, there is no significant risk involved (see 4.00C8a), and we cannot make our determination or decision based on the evidence we already have.

14. *What are drug-induced stress tests?* These tests are designed primarily to provide evidence about myocardial ischemia or prior myocardial infarction, but do not require you to exercise. These tests are used when you cannot exercise or cannot exercise enough to achieve the desired cardiac stress. Drug-induced stress tests can also provide evidence about heart chamber dimensions and function; however, these tests do not provide information about your aerobic capacity and cannot be used to help us assess your ability to function. Some of these tests use agents, such as Persantine or adenosine, that dilate the coronary arteries and are used in combination with nuclear agents, such as thallium or technetium (for example, Cardiolyte or Myoview), and a myocardial scan. Other tests use agents, such as dobutamine, that stimulate the heart to contract more forcefully and faster to simulate exercise and are used in combination with a 2-dimensional echocardiogram. We may,

when appropriate, purchase a drug-induced stress test to confirm the presence of myocardial

ischemia after a review of the evidence in your file by an MC, preferably one with experience in the care of patients with cardiovascular disease.

15. *How do we evaluate cardiac catheterization evidence?*

a. We will not purchase cardiac catheterization; however, if you have had catheterization, we will make every reasonable effort to obtain the report and any ancillary studies. We will consider the quality and type of data provided and its relevance to the evaluation of your impairment. For adults, we generally see two types of catheterization reports: Coronary arteriography and left ventriculography.

b. For coronary arteriography, the report should provide information citing the method of assessing coronary arterial lumen diameter and the nature and location of obstructive lesions. Drug treatment at baseline and during the procedure should be reported. Some individuals with significant coronary atherosclerotic obstruction have collateral vessels that supply the myocardium distal to the arterial obstruction so that there is no evidence of myocardial damage or ischemia, even with exercise. When the results of quantitative computer measurements and analyses are included in your case record, we will consider them in interpreting the severity of stenotic lesions.

c. For left ventriculography, the report should describe the wall motion of the myocardium with regard to any areas of hypokinesis (abnormally decreased motion), akinesis (lack of motion), or dyskinesis (distortion of motion), and the overall contraction of the ventricle as measured by the ejection fraction. Measurement of chamber volumes and pressures may be useful. Quantitative computer analysis provides precise measurement of segmental left ventricular wall thickness and motion. There is often a poor correlation between left ventricular function at rest and functional capacity for physical activity.

16. *What details should exercise Doppler test reports contain?* The reports of exercise Doppler tests must describe the level of exercise; for example, the speed and grade of the treadmill settings, the duration of exercise, symptoms during exercise, and the reasons for stopping exercise if the expected level of exercise was not attained. They must also include the blood pressures at the ankle and other pertinent sites measured after exercise and the time required for the systolic blood pressure to return toward or to the pre-exercise level. The graphic tracings, if available, should also be included with the report. All tracings must be annotated with the standardization used by the testing facility.

17. *How must exercise Doppler tests we purchase be performed?* When we purchase an exercise Doppler test, you must exercise on a treadmill at 2 mph on a 12 percent grade for up to 5 minutes. The reports must include the information specified in 4.00C16. Because this is an exercise test, we must evaluate whether such testing would put you at significant risk, in accordance with the guidance found in 4.00C6, 4.00C7, and 4.00C8.

D. *Evaluating Chronic Heart Failure*

1. *What is chronic heart failure (CHF)?*

a. *CHF* is the inability of the heart to pump enough oxygenated blood to body tissues. This syndrome is characterized by symptoms and signs of pulmonary or systemic congestion (fluid retention) or limited cardiac output. Certain laboratory findings of cardiac functional and structural abnormality support the diagnosis of CHF. There are two main types of CHF:

(i) *Predominant systolic dysfunction* (the inability of the heart to contract normally and expel sufficient blood), which is characterized by a dilated, poorly contracting left ventricle and reduced ejection fraction (abbreviated EF, it represents the percentage of the blood in the ventricle actually pumped out with each contraction), and

(ii) *Predominant diastolic dysfunction* (the inability of the heart to relax and fill normally), which is characterized by a thickened ventricular muscle, poor ability of the left ventricle to distend, increased ventricular filling pressure, and a normal or increased EF.

b. CHF is considered in these listings as a single category whether due to atherosclerosis (narrowing of the arteries), cardiomyopathy, hypertension, or rheumatic, congenital, or other heart disease. However, if the CHF is the result of primary pulmonary hypertension secondary to disease of the lung (cor pulmonale), we will evaluate your impairment using 3.09, in the respiratory system listings.

2. *What evidence of CHF do we need?*

a. Cardiomegaly or ventricular dysfunction must be present and demonstrated by appropriate medically acceptable imaging, such as chest x-ray, echocardiography (M-Mode, 2-dimensional, and Doppler), radionuclide studies, or cardiac catheterization.

(i) Abnormal cardiac imaging showing increased left ventricular end diastolic diameter (LVEDD), decreased EF, increased left atrial chamber size, increased ventricular filling pressures measured at cardiac catheterization, or increased left ventricular wall or septum thickness, provides objective measures of both left ventricular function and structural abnormality in heart failure.

(ii) An LVEDD greater than 6.0 cm or an EF of 30 percent or less measured during a period of stability (that is, not during an episode of acute heart failure) may be associated clinically with systolic failure.

(iii) Left ventricular posterior wall thickness added to septal thickness totaling 2.5 cm or greater with left atrium enlarged to 4.5 cm or greater may be associated clinically with diastolic failure.

(iv) However, these measurements alone do not reflect your functional capacity, which we evaluate by considering all of the relevant evidence. In some situations, we may need to purchase an ETT to help us assess your functional capacity.

(v) Other findings on appropriate medically acceptable imaging may include increased pulmonary

vascular markings, pleural effusion, and pulmonary edema. These findings need not be present on each report, since CHF may be controlled by prescribed treatment.

b. To establish that you have *chronic* heart failure, your medical history and physical examination should describe characteristic symptoms and signs of pulmonary or systemic congestion or of limited cardiac output associated with the abnormal findings on appropriate medically acceptable imaging. When an acute episode of heart failure is triggered by a remediable factor, such as an arrhythmia, dietary sodium overload, or high altitude, cardiac function may be restored and a chronic impairment may not be present.

(i) Symptoms of congestion or of limited cardiac output include easy fatigue, weakness, shortness of breath (dyspnea), cough, or chest discomfort at rest or with activity. Individuals with CHF may also experience shortness of breath on lying flat (orthopnea) or episodes of shortness of breath that wake them from sleep (paroxysmal nocturnal dyspnea). They may also experience cardiac arrhythmias resulting in palpitations, lightheadedness, or fainting.

(ii) Signs of congestion may include hepatomegaly, ascites, increased jugular venous distention or pressure, rales, peripheral edema, or rapid weight gain. However, these signs need not be found on all examinations because fluid retention may be controlled by prescribed treatment.

3. *Is it safe for you to have an ETT, if you have CHF?* The presence of CHF is not necessarily a contraindication to an ETT, unless you are having an acute episode of heart failure. Measures of cardiac performance are valuable in helping us evaluate your ability to do work-related activities. Exercise testing has been safely used in individuals with CHF; therefore, we may purchase an ETT for evaluation under 4.02B3 if an MC, preferably one experienced in the care of patients with cardiovascular disease, determines that there is no significant risk to you. (See 4.00C6 for when we will consider the purchase of an ETT. See 4.00C7-4.00C8 for what we must do before we purchase an ETT and when we will not purchase one.) ST segment changes from digitalis use in the treatment of CHF do not preclude the purchase of an ETT.

4. *How do we evaluate CHF using 4.02?*

a. We must have objective evidence, as described in 4.00D2, that you have chronic heart failure.

b. To meet the required level of severity for this listing, your impairment must satisfy the requirements of one of the criteria in A and one of the criteria in B.

c. In 4.02B2, the phrase *periods of stabilization* means that, for at least 2 weeks between episodes of acute heart failure, there must be objective evidence of clearing of the pulmonary edema or pleural effusions and evidence that you returned to, or you were medically considered able to return to, your prior level of activity.

d. Listing 4.02B3c requires a decrease in systolic blood pressure below the baseline level (taken in

the standing position immediately prior to exercise) or below any systolic pressure reading recorded during exercise. This is because, normally, systolic blood pressure and heart rate increase gradually with exercise. Decreases in systolic blood pressure below the baseline level that occur during exercise are often associated with ischemia-induced left ventricular dysfunction resulting in decreased cardiac output. However, a blunted response (that is, failure of the systolic blood pressure to rise 10 mm Hg or more), particularly in the first 3 minutes of exercise, may be drug-related and is not necessarily associated with left ventricular dysfunction. Also, some individuals with increased sympathetic responses because of deconditioning or apprehension may increase their systolic blood pressure and heart rate above their baseline level just before and early into exercise. This can be associated with a drop in systolic pressure in early exercise that is not due to left ventricular dysfunction. Therefore, an early decrease in systolic blood pressure must be interpreted within the total context of the test; that is, the presence or absence of symptoms such as lightheadedness, ischemic changes, or arrhythmias on the ECG.

E. *Evaluating Ischemic Heart Disease*

1. *What is ischemic heart disease (IHD)? IHD* results when one or more of your coronary arteries is narrowed or obstructed or, in rare situations, constricted due to vasospasm, interfering with the normal flow of blood to your heart muscle (ischemia). The obstruction may be the result of an embolus, a thrombus, or plaque. When heart muscle tissue dies as a result of the reduced blood supply, it is called a myocardial infarction (heart attack).

2. *What causes chest discomfort of myocardial origin?*

a. Chest discomfort of myocardial ischemic origin, commonly known as angina pectoris, is usually caused by coronary artery disease (often abbreviated CAD). However, ischemic discomfort may be caused by a noncoronary artery impairment, such as aortic stenosis, hypertrophic cardiomyopathy, pulmonary hypertension, or anemia.

b. Instead of typical angina pectoris, some individuals with IHD experience atypical angina, anginal equivalent, variant angina, or silent ischemia, all of which we may evaluate using 4.04. We discuss the various manifestations of ischemia in 4.00E3-4.00E7.

3. *What are the characteristics of typical angina pectoris?* Discomfort of myocardial ischemic origin (angina pectoris) is discomfort that is precipitated by effort or emotion and promptly relieved by rest, sublingual nitroglycerin (that is, nitroglycerin tablets that are placed under the tongue), or other rapidly acting nitrates. Typically, the discomfort is located in the chest (usually substernal) and described as pressing, crushing, squeezing, burning, aching, or oppressive. Sharp, sticking, or cramping discomfort is less common. Discomfort occurring with activity or emotion should be described specifically as to timing and usual inciting factors (type and intensity), character, location, radiation, duration, and response to nitrate treatment or rest.

4. *What is atypical angina? Atypical angina* describes discomfort or pain from myocardial ischemia

that is felt in places other than the chest. The common sites of cardiac pain are the inner aspect of the left arm, neck, jaw(s), upper abdomen, and back, but the discomfort or pain can be elsewhere. When pain of cardiac ischemic origin presents in an atypical site in the absence of chest discomfort, the source of the pain may be difficult to diagnose. To represent atypical angina, your discomfort or pain should have precipitating and relieving factors similar to those of typical chest discomfort, and we must have objective medical evidence of myocardial ischemia; for example, ECG or ETT evidence or appropriate medically acceptable imaging.

5. *What is anginal equivalent?* Often, individuals with IHD will complain of shortness of breath (dyspnea) on exertion without chest pain or discomfort. In a minority of such situations, the shortness of breath is due to myocardial ischemia; this is called *anginal equivalent*. To represent anginal equivalent, your shortness of breath should have precipitating and relieving factors similar to those of typical chest discomfort, and we must have objective medical evidence of myocardial ischemia; for example, ECG or ETT evidence or appropriate medically acceptable imaging. In these situations, it is essential to establish objective evidence of myocardial ischemia to ensure that you do not have effort dyspnea due to non-ischemic or non-cardiac causes.

6. *What is variant angina?*

a. *Variant angina* (Prinzmetal's angina, vasospastic angina) refers to the occurrence of anginal episodes at rest, especially at night, accompanied by transitory ST segment elevation (or, at times, ST depression) on an ECG. It is due to severe spasm of a coronary artery, causing ischemia of the heart wall, and is often accompanied by major ventricular arrhythmias, such as ventricular tachycardia. We will consider variant angina under 4.04 only if you have spasm of a coronary artery in relation to an obstructive lesion of the vessel. If you have an arrhythmia as a result of variant angina, we may consider your impairment under 4.05.

b. Variant angina may also occur in the absence of obstructive coronary disease. In this situation, an ETT will not demonstrate ischemia. The diagnosis will be established by showing the typical transitory ST segment changes during attacks of pain, and the absence of obstructive lesions shown by catheterization. Treatment in cases where there is no obstructive coronary disease is limited to medications that reduce coronary vasospasm, such as calcium channel blockers and nitrates. In such situations, we will consider the frequency of anginal episodes despite prescribed treatment when evaluating your residual functional capacity.

c. Vasospasm that is catheter-induced during coronary angiography is not variant angina.

7. *What is silent ischemia?*

a. Myocardial ischemia, and even myocardial infarction, can occur without perception of pain or any other symptoms; when this happens, we call it *silent ischemia*. Pain sensitivity may be altered by a

variety of diseases, most notably diabetes mellitus and other neuropathic disorders. Individuals also

vary in their threshold for pain.

b. Silent ischemia occurs most often in:

(i) Individuals with documented past myocardial infarction or established angina without prior infarction who do not have chest pain on ETT, but have a positive test with ischemic abnormality on ECG, perfusion scan, or other appropriate medically acceptable imaging.

(ii) Individuals with documented past myocardial infarction or angina who have ST segment changes on ambulatory monitoring (Holter monitoring) that are similar to those that occur during episodes of angina. ST depression shown on the ambulatory recording should not be interpreted as positive for ischemia unless similar depression is also seen during chest pain episodes annotated in the diary that the individual keeps while wearing the Holter monitor.

c. ST depression can result from a variety of factors, such as postural changes and variations in cardiac sympathetic tone. In addition, there are differences in how different Holter monitors record the electrical responses. Therefore, we do not consider the Holter monitor reliable for the diagnosis of silent ischemia except in the situation described in 4.00E7b(ii).

8. *What other sources of chest discomfort are there?* Chest discomfort of nonischemic origin may result from other cardiac impairments, such as pericarditis. Noncardiac impairments may also produce symptoms mimicking that of myocardial ischemia. These impairments include acute anxiety or panic attacks, gastrointestinal tract disorders, such as esophageal spasm, esophagitis, hiatal hernia, biliary tract disease, gastritis, peptic ulcer, and pancreatitis, and musculoskeletal syndromes, such as chest wall muscle spasm, chest wall syndrome (especially after coronary bypass surgery), costochondritis, and cervical or dorsal spine arthritis. Hyperventilation may also mimic ischemic discomfort. Thus, in the absence of documented myocardial ischemia, such disorders should be considered as possible causes of chest discomfort.

9. *How do we evaluate IHD using 4.04?*

a. We must have objective evidence, as described under 4.00C, that your symptoms are due to myocardial ischemia.

b. Listing-level changes on the ECG in 4.04A1 are the classically accepted changes of horizontal or downsloping ST depression occurring both during exercise and recovery. Although we recognize that ischemic changes may at times occur only during exercise or recovery, and may at times be upsloping with only junctional ST depression, such changes can be false positive; that is, occur in the absence of ischemia. Diagnosis of ischemia in this situation requires radionuclide or echocardiogram confirmation. See 4.00C12 and 4.00C13.

c. Also in 4.04A1, we require that the depression of the ST segment last for at least 1 minute of

recovery because ST depression that occurs during exercise but that rapidly normalizes in recovery is a common false-positive response.

d. In 4.04A2, we specify that the ST elevation must be in non-infarct leads during both exercise and recovery. This is because, in the absence of ECG signs of prior infarction, ST elevation during exercise denotes ischemia, usually severe, requiring immediate termination of exercise. However, if there is baseline ST elevation in association with a prior infarction or ventricular aneurysm, further ST elevation during exercise does not necessarily denote ischemia and could be a false-positive ECG response. Diagnosis of ischemia in this situation requires radionuclide or echocardiogram confirmation. See 4.00C12 and 4.00C13.

e. Listing 4.04A3 requires a decrease in systolic blood pressure below the baseline level (taken in the standing position immediately prior to exercise) or below any systolic pressure reading recorded during exercise. This is the same finding required in 4.02B3c. See 4.00D4d for full details.

f. In 4.04B, each of the three ischemic episodes must require revascularization or be not amenable to treatment. *Revascularization* means angioplasty (with or without stent placement) or bypass surgery. However, reocclusion that occurs after a revascularization procedure but during the same hospitalization and that requires a second procedure during the same hospitalization will not be counted as another ischemic episode. Not amenable means that the revascularization procedure could not be done because of another medical impairment or because the vessel was not suitable for revascularization.

g. We will use 4.04C only when you have symptoms due to myocardial ischemia as described in

4.00E3-4.00E7 while on a regimen of prescribed treatment, you are at risk for exercise testing (see 4.00C8), and we do not have a timely ETT or a timely normal drug-induced stress test for you. See 4.00C9 for what we mean by a timely test.

h. In 4.04C1 the term *nonbypassed* means that the blockage is in a vessel that is potentially bypassable; that is, large enough to be bypassed and considered to be a cause of your ischemia. These vessels are usually major arteries or one of a major artery's major branches. A vessel that has become obstructed again after angioplasty or stent placement and has remained obstructed or is not amenable to another revascularization is considered a nonbypassed vessel for purposes of this listing. When you have had revascularization, we will not use the pre-operative findings to assess the current severity of your coronary artery disease under 4.04C, although we will consider the severity and duration of your impairment prior to your surgery in making our determination or decision.

F. *Evaluating Arrhythmias*

1. *What is an arrhythmia?* An *arrhythmia* is a change in the regular beat of the heart. Your heart may

seem to skip a beat or beat irregularly, very quickly (tachycardia), or very slowly (bradycardia).

2. *What are the different types of arrhythmias?*

a. There are many types of arrhythmias. Arrhythmias are identified by where they occur in the heart (atria or ventricles) and by what happens to the heart's rhythm when they occur.

b. Arrhythmias arising in the cardiac atria (upper chambers of the heart) are called atrial or supraventricular arrhythmias. Ventricular arrhythmias begin in the ventricles (lower chambers). In general, ventricular arrhythmias caused by heart disease are the most serious.

3. *How do we evaluate arrhythmias using 4.05?*

a. We will use 4.05 when you have arrhythmias that are not fully controlled by medication, an implanted pacemaker, or an implanted cardiac defibrillator and you have uncontrolled recurrent episodes of syncope or near syncope. If your arrhythmias are controlled, we will evaluate your underlying heart disease using the appropriate listing. For other considerations when we evaluate arrhythmias in the presence of an implanted cardiac defibrillator, see 4.00F4.

b. We consider *near syncope* to be a period of altered consciousness, since syncope is a loss of consciousness or a faint. It is not merely a feeling of light-headedness, momentary weakness, or dizziness.

c. For purposes of 4.05, there must be a documented association between the syncope or near syncope and the recurrent arrhythmia. The recurrent arrhythmia, not some other cardiac or non-cardiac disorder, must be established as the cause of the associated symptom. This documentation of the association between the symptoms and the arrhythmia may come from the usual diagnostic methods, including Holter monitoring (also called ambulatory electrocardiography) and tilt-table testing with a concurrent ECG. Although an arrhythmia may be a coincidental finding on an ETT, we will not purchase an ETT to document the presence of a cardiac arrhythmia.

4. *What will we consider when you have an implanted cardiac defibrillator and you do not have arrhythmias that meet the requirements of 4.05?*

a. Implanted cardiac defibrillators are used to prevent sudden cardiac death in individuals who have had, or are at high risk for, cardiac arrest from life-threatening ventricular arrhythmias. The largest group at risk for sudden cardiac death consists of individuals with cardiomyopathy (ischemic or non-ischemic) and reduced ventricular function. However, life-threatening ventricular arrhythmias can also occur in individuals with little or no ventricular dysfunction. The shock from the implanted cardiac defibrillator is a unique form of treatment; it rescues an individual from what may have been cardiac arrest. However, as a consequence of the shock(s), individuals may experience psychological distress, which we may evaluate under the mental disorders listings in 12.00ff.

b. Most implantable cardiac defibrillators have rhythm-correcting and pacemaker capabilities. In

some individuals, these functions may result in the termination of ventricular arrhythmias without an otherwise painful shock. (The shock is like being kicked in the chest.) Implanted cardiac defibrillators may deliver inappropriate shocks, often repeatedly, in response to benign arrhythmias or electrical malfunction. Also, exposure to strong electrical or magnetic fields, such as from MRI (magnetic resonance imaging), can trigger or reprogram an implanted cardiac defibrillator, resulting in inappropriate shocks. We must consider the frequency of, and the reason(s) for, the shocks when evaluating the severity and duration of your impairment.

c. In general, the exercise limitations imposed on individuals with an implanted cardiac defibrillator are those dictated by the underlying heart impairment. However, the exercise limitations may be greater when the implanted cardiac defibrillator delivers an inappropriate shock in response to the increase in heart rate with exercise, or when there is exercise-induced ventricular arrhythmia.

G. *Evaluating Peripheral Vascular Disease*

1. *What is peripheral vascular disease (PVD)?* Generally, *PVD* is any impairment that affects either the arteries (peripheral arterial disease) or the veins (venous insufficiency) in the extremities, particularly the lower extremities. The usual effect is blockage of the flow of blood either from the heart (arterial) or back to the heart (venous). If you have peripheral arterial disease, you may have pain in your calf after walking a distance that goes away when you rest (intermittent claudication); at more advanced stages, you may have pain in your calf at rest or you may develop ulceration or gangrene. If you have venous insufficiency, you may have swelling, varicose veins, skin pigmentation changes, or skin ulceration.

2. *How do we assess limitations resulting from PVD?* We will assess your limitations based on your symptoms together with physical findings, Doppler studies, other appropriate non-invasive studies, or angiographic findings. However, if the PVD has resulted in amputation, we will evaluate any limitations related to the amputation under the musculoskeletal listings, 1.00ff.

3. *What is brawny edema? Brawny edema* (4.11A) is swelling that is usually dense and feels firm due to the presence of increased connective tissue; it is also associated with characteristic skin pigmentation changes. It is not the same thing as pitting edema. Brawny edema generally does not pit (indent on pressure), and the terms are not interchangeable. Pitting edema does not satisfy the requirements of 4.11A.

4. *What is lymphedema and how will we evaluate it?*

a. *Lymphedema* is edema of the extremities due to a disorder of the lymphatic circulation; at its worst, it is called elephantiasis. Primary lymphedema is caused by abnormal development of lymph vessels and may be present at birth (congenital lymphedema), but more often develops during the teens (lymphedema praecox). It may also appear later, usually after age 35 (lymphedema tarda). Secondary lymphedema is due to obstruction or destruction of normal lymphatic channels due to tumor, surgery, repeated infections, or parasitic infection such as filariasis. Lymphedema most

commonly affects one extremity.

b. Lymphedema does not meet the requirements of 4.11, although it may medically equal the severity of that listing. We will evaluate lymphedema by considering whether the underlying cause meets or medically equals any listing or whether the lymphedema medically equals a cardiovascular listing, such as 4.11, or a musculoskeletal listing, such as 1.02A or 1.03. If no listing is met or medically equaled, we will evaluate any functional limitations imposed by your lymphedema when we assess your residual functional capacity.

5. *When will we purchase exercise Doppler studies for evaluating peripheral arterial disease (PAD)?* If we need additional evidence of your PAD, we will generally purchase exercise Doppler studies (see 4.00C16 and 4.00C17) when your resting ankle/brachial systolic blood pressure ratio is at least 0.50 but less than 0.80, and only rarely when it is 0.80 or above. We will not purchase exercise Doppler testing if you have a disease that results in abnormal arterial calcification or small vessel disease, but will use your resting toe systolic blood pressure or resting toe/brachial systolic blood pressure ratio. (See 4.00G7c and 4.00G8.) There are no current medical standards for evaluating exercise toe pressures. Because any exercise test stresses your entire cardiovascular system, we will purchase exercise Doppler studies only after an MC, preferably one with experience in the care of patients with cardiovascular disease, has determined that the test would not present a significant risk to you and that there is no other medical reason not to purchase the test (see 4.00C6, 4.00C7, and 4.00C8).

6. *Are there any other studies that are helpful in evaluating PAD?* Doppler studies done using a recording ultrasonic Doppler unit and strain-gauge plethysmography are other useful tools for evaluating PAD. A recording Doppler, which prints a tracing of the arterial pulse wave in the femoral, popliteal, dorsalis pedis, and posterior tibial arteries, is an excellent evaluation tool to compare wave forms in normal and compromised peripheral blood flow. Qualitative analysis of the pulse wave is very helpful in the overall assessment of the severity of the occlusive disease. Tracings are especially helpful in assessing severity if you have small vessel disease related to diabetes mellitus or other diseases with similar vascular changes, or diseases causing medial calcifications when ankle pressure is either normal or falsely high.

7. *How do we evaluate PAD under 4.12?*

a. The ankle blood pressure referred to in 4.12A and B is the higher of the pressures recorded from the posterior tibial and dorsalis pedis arteries in the affected leg. The higher pressure recorded from the two sites is the more significant measurement in assessing the extent of arterial insufficiency. Techniques for obtaining ankle systolic blood pressures include Doppler (See 4.00C16 and 4.00C17), plethysmographic studies, or other techniques. We will request any available tracings generated by these studies so that we can review them.

b. In 4.12A, the ankle/brachial systolic blood pressure ratio is the ratio of the systolic blood pressure

at the ankle to the systolic blood pressure at the brachial artery; both taken at the same time while you are lying on your back. We do not require that the ankle and brachial pressures be taken on the same side of your body. This is because, as with the ankle pressure, we will use the higher brachial systolic pressure measured. Listing 4.12A is met when your resting ankle/brachial systolic blood pressure ratio is less than 0.50. If your resting ankle/brachial systolic blood pressure ratio is 0.50 or above, we will use 4.12B to evaluate the severity of your PAD, unless you also have a disease causing abnormal arterial calcification or small vessel disease, such as diabetes mellitus. See 4.00G7c and 4.00G8.

c. We will use resting toe systolic blood pressures or resting toe/brachial systolic blood pressure ratios (determined the same way as ankle/brachial ratios, see 4.00G7b) when you have intermittent claudication and a disease that results in abnormal arterial calcification (for example, Monckeberg's sclerosis or diabetes mellitus) or small vessel disease (for example, diabetes mellitus). These diseases may result in misleadingly high blood pressure readings at the ankle. However, high blood pressures due to vascular changes related to these diseases seldom occur at the toe level. While the criteria in 4.12C and 4.12D are intended primarily for individuals who have a disease causing abnormal arterial calcification or small vessel disease, we may also use them for evaluating anyone with PAD.

8. *How are toe pressures measured?* Toe pressures are measured routinely in most vascular laboratories through one of three methods: most frequently, photoplethysmography; less frequently, plethysmography using strain gauge cuffs; and Doppler ultrasound. Toe pressure can also be measured by using any blood pressure cuff that fits snugly around the big toe and is neither too tight nor too loose. A neonatal cuff or a cuff designed for use on fingers or toes can be used in the measurement of toe pressure.

9. *How do we use listing 4.12 if you have had a peripheral graft?* Peripheral grafting serves the same purpose as coronary grafting; that is, to bypass a narrow or obstructed arterial segment. If intermittent claudication recurs or persists after peripheral grafting, we may purchase Doppler studies to assess the flow of blood through the bypassed vessel and to establish the current severity of the peripheral arterial impairment. However, if you have had peripheral grafting done for your PAD, we will not use the findings from before the surgery to assess the current severity of your impairment, although we will consider the severity and duration of your impairment prior to your surgery in making our determination or decision.

H. *Evaluating Other Cardiovascular Impairments*

1. *How will we evaluate hypertension?* Because *hypertension* (high blood pressure) generally causes disability through its effects on other body systems, we will evaluate it by reference to the specific body system(s) affected (heart, brain, kidneys, or eyes) when we consider its effects under the

listings. We will also consider any limitations imposed by your hypertension when we assess your

residual functional capacity.

2. *How will we evaluate symptomatic congenital heart disease? Congenital heart disease* is any abnormality of the heart or the major blood vessels that is present at birth. Because of improved treatment methods, more children with congenital heart disease are living to adulthood. Although some types of congenital heart disease may be corrected by surgery, many individuals with treated congenital heart disease continue to have problems throughout their lives (symptomatic congenital heart disease). If you have congenital heart disease that results in chronic heart failure with evidence of ventricular dysfunction or in recurrent arrhythmias, we will evaluate your impairment under 4.02 or 4.05. Otherwise, we will evaluate your impairment under 4.06.

3. *What is cardiomyopathy and how will we evaluate it? Cardiomyopathy* is a disease of the heart muscle. The heart loses its ability to pump blood (heart failure), and in some instances, heart rhythm is disturbed, leading to irregular heartbeats (arrhythmias). Usually, the exact cause of the muscle damage is never found (idiopathic cardiomyopathy). There are various types of cardiomyopathy, which fall into two major categories: *Ischemic* and *nonischemic* cardiomyopathy. Ischemic cardiomyopathy typically refers to heart muscle damage that results from coronary artery disease, including heart attacks. Nonischemic cardiomyopathy includes several types: Dilated, hypertrophic, and restrictive. We will evaluate cardiomyopathy under 4.02, 4.04, 4.05, or 11.04, depending on its effects on you.

4. *How will we evaluate valvular heart disease?* We will evaluate valvular heart disease under the listing appropriate for its effect on you. Thus, we may use 4.02, 4.04, 4.05, 4.06, or an appropriate neurological listing in 11.00ff.

5. *What do we consider when we evaluate heart transplant recipients?*

a. After your heart transplant, we will consider you disabled for 1 year following the surgery because there is a greater likelihood of rejection of the organ and infection during the first year.

b. However, heart transplant patients generally meet our definition of disability before they undergo transplantation. We will determine the onset of your disability based on the facts in your case.

c. We will not assume that you became disabled when your name was placed on a transplant waiting list. This is because you may be placed on a waiting list soon after diagnosis of the cardiac disorder that may eventually require a transplant. Physicians recognize that candidates for transplantation often have to wait months or even years before a suitable donor heart is found, so they place their patients on the list as soon as permitted.

d. When we do a continuing disability review to determine whether you are still disabled, we will evaluate your residual impairment(s), as shown by symptoms, signs, and laboratory findings, including any side effects of medication. We will consider any remaining symptoms, signs, and laboratory findings indicative of cardiac dysfunction in deciding whether medical improvement (as

defined in §§ 404.1594 and 416.994) has occurred.

6. *When does an aneurysm have "dissection not controlled by prescribed treatment," as required under 4.10?* An aneurysm (or bulge in the aorta or one of its major branches) is *dissecting* when the inner lining of the artery begins to separate from the arterial wall. We consider the dissection not controlled when you have persistence of chest pain due to progression of the dissection, an increase in the size of the aneurysm, or compression of one or more branches of the aorta supplying the heart, kidneys, brain, or other organs. An aneurysm with dissection can cause heart failure, renal (kidney) failure, or neurological complications. If you have an aneurysm that does not meet the requirements of 4.10 and you have one or more of these associated conditions, we will evaluate the condition(s) using the appropriate listing.

7. *What is hyperlipidemia and how will we evaluate it? Hyperlipidemia* is the general term for an elevation of any or all of the lipids (fats or cholesterol) in the blood; for example, hypertriglyceridemia, hypercholesterolemia, and hyperlipoproteinemia. These disorders of lipoprotein metabolism and transport can cause defects throughout the body. The effects most likely to interfere with function are those produced by atherosclerosis (narrowing of the arteries) and coronary artery disease. We will evaluate your lipoprotein disorder by considering its effects on you.

8. *What is Marfan syndrome and how will we evaluate it?*

a. Marfan syndrome is a genetic connective tissue disorder that affects multiple body systems, including the skeleton, eyes, heart, blood vessels, nervous system, skin, and lungs. There is no specific laboratory test to diagnose Marfan syndrome. The diagnosis is generally made by medical history, including family history, physical examination, including an evaluation of the ratio of arm/leg size to trunk size, a slit lamp eye examination, and a heart test(s), such as an echocardiogram. In some cases, a genetic analysis may be useful, but such analyses may not provide any additional helpful information.

b. The effects of Marfan syndrome can range from mild to severe. In most cases, the disorder progresses as you age. Most individuals with Marfan syndrome have abnormalities associated with the heart and blood vessels. Your heart's mitral valve may leak, causing a heart murmur. Small leaks may not cause symptoms, but larger ones may cause shortness of breath, fatigue, and palpitations. Another effect is that the wall of the aorta may be weakened and abnormally stretch (aortic dilation). This aortic dilation may tear, dissect, or rupture, causing serious heart problems or sometimes sudden death. We will evaluate the manifestations of your Marfan syndrome under the appropriate body system criteria, such as 4.10, or if necessary, consider the functional limitations imposed by your impairment.

I. *Other Evaluation Issues*

1. *What effect does obesity have on the cardiovascular system and how will we evaluate it?* Obesity is

a medically determinable impairment that is often associated with disorders of the cardiovascular system. Disturbance of this system can be a major cause of disability if you have obesity. Obesity may affect the cardiovascular system because of the increased workload the additional body mass places on the heart. Obesity may make it harder for the chest and lungs to expand. This can mean that the respiratory system must work harder to provide needed oxygen. This in turn would make the heart work harder to pump blood to carry oxygen to the body. Because the body would be working harder at rest, its ability to perform additional work would be less than would otherwise be expected. Thus, the combined effects of obesity with cardiovascular impairments can be greater than the effects of each of the impairments considered separately. We must consider any additional and cumulative effects of obesity when we determine whether you have a severe cardiovascular impairment or a listing-level cardiovascular impairment (or a combination of impairments that medically equals the severity of a listed impairment), and when we assess your residual functional capacity.

2. *How do we relate treatment to functional status?* In general, conclusions about the severity of a cardiovascular impairment cannot be made on the basis of type of treatment rendered or anticipated. The amount of function restored and the time required for improvement after treatment (medical, surgical, or a prescribed program of progressive physical activity) vary with the nature and extent of the disorder, the type of treatment, and other factors. Depending upon the timing of this treatment in relation to the alleged onset date of disability, we may need to defer evaluation of the impairment for a period of up to 3 months from the date treatment began to permit consideration of treatment effects, unless we can make a determination or decision using the evidence we have. See 4.00B4.

3. *How do we evaluate impairments that do not meet one of the cardiovascular listings?*

a. These listings are only examples of common cardiovascular impairments that we consider severe enough to prevent you from doing any gainful activity. If your severe impairment(s) does not meet the criteria of any of these listings, we must also consider whether you have an impairment(s) that satisfies the criteria of a listing in another body system.

b. If you have a severe medically determinable impairment(s) that does not meet a listing, we will determine whether your impairments(s) medically equals a listing. (See §§ 404.1526 and 416.926.) If you have a severe impairment(s) that does not meet or medically equal the criteria of a listing, you may or may not have the residual functional capacity to engage in substantial gainful activity. Therefore, we proceed to the fourth and, if necessary, the fifth steps of the sequential evaluation process in §§ 404.1520 and 416.920. If you are an adult, we use the rules in §§ 404.1594 or 416.994, as appropriate, when we decide whether you continue to be disabled.

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4.01 Category of Impairments, Cardiovascular System

4.02 *Chronic heart failure* while on a regimen of prescribed treatment, with symptoms and signs described in 4.00D2. The required level of severity for this impairment is met when the requirements in *both A and B* are satisfied.

A. Medically documented presence of one of the following:

1. Systolic failure (see 4.00D1a(i)), with left ventricular end diastolic dimensions greater than 6.0 cm or ejection fraction of 30 percent or less during a period of stability (not during an episode of acute heart failure); or

2. Diastolic failure (see 4.00D1a(ii)), with left ventricular posterior wall plus septal thickness totaling 2.5 cm or greater on imaging, with an enlarged left atrium greater than or equal to 4.5 cm, with normal or elevated ejection fraction during a period of stability (not during an episode of acute heart failure);

AND

B. Resulting in one of the following:

1. Persistent symptoms of heart failure which very seriously limit the ability to independently initiate, sustain, or complete activities of daily living in an individual for whom an MC, preferably one experienced in the care of patients with cardiovascular disease, has concluded that the performance of an exercise test would present a significant risk to the individual; or

2. Three or more separate episodes of acute congestive heart failure within a consecutive 12-month period (see 4.00A3e), with evidence of fluid retention (see 4.00D2b(ii)) from clinical and imaging assessments at the time of the episodes, requiring acute extended physician intervention such as hospitalization or emergency room treatment for 12 hours or more, separated by periods of stabilization (see 4.00D4c); or

3. Inability to perform on an exercise tolerance test at a workload equivalent to 5 METs or less due to:

a. Dyspnea, fatigue, palpitations, or chest discomfort; or

b. Three or more consecutive premature ventricular contractions (ventricular tachycardia), or increasing frequency of ventricular ectopy with at least 6 premature ventricular contractions per minute; or

c. Decrease of 10 mm Hg or more in systolic pressure below the baseline systolic blood pressure or the preceding systolic pressure measured during exercise (see 4.00D4d) due to left ventricular dysfunction, despite an increase in workload; or

d. Signs attributable to inadequate cerebral perfusion, such as ataxic gait or mental confusion.

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4.04 Ischemic heart disease, with symptoms due to myocardial ischemia, as described in 4.00E3-4.00E7, while on a regimen of prescribed treatment (see 4.00B3 if there is no regimen of prescribed treatment), with one of the following:

A. Sign-or symptom-limited exercise tolerance test demonstrating at least one of the following manifestations at a workload equivalent to 5 METs or less:

1. Horizontal or downsloping depression, in the absence of digitalis glycoside treatment or hypokalemia, of the ST segment of at least −0.10 millivolts (−1.0 mm) in at least 3 consecutive complexes that are on a level baseline in any lead other than a VR, and depression of at least −0.10 millivolts lasting for at least 1 minute of recovery; or

2. At least 0.1 millivolt (1 mm) ST elevation above resting baseline in non-infarct leads during both exercise and 1 or more minutes of recovery; or

3. Decrease of 10 mm Hg or more in systolic pressure below the baseline blood pressure or the preceding systolic pressure measured during exercise (see 4.00E9e) due to left ventricular dysfunction, despite an increase in workload; or

4. Documented ischemia at an exercise level equivalent to 5 METs or less on appropriate medically acceptable imaging, such as radionuclide perfusion scans or stress echocardiography.

OR

B. Three separate ischemic episodes, each requiring revascularization or not amenable to revascularization (see 4.00E9f), within a consecutive 12-month period (see 4.00A3e).

OR

C. Coronary artery disease, demonstrated by angiography (obtained independent of Social Security disability evaluation) or other appropriate medically acceptable imaging, and in the absence of a timely exercise tolerance test or a timely normal drug-induced stress test, an MC, preferably one experienced in the care of patients with cardiovascular disease, has concluded that performance of exercise tolerance testing would present a significant risk to the individual, with both 1 and 2:

1. Angiographic evidence showing:

a. 50 percent or more narrowing of a nonbypassed left main coronary artery; or

b. 70 percent or more narrowing of another nonbypassed coronary artery; or

c. 50 percent or more narrowing involving a long (greater than 1 cm) segment of a nonbypassed

coronary artery; or

d. 50 percent or more narrowing of at least two nonbypassed coronary arteries; or

e. 70 percent or more narrowing of a bypass graft vessel; and

2. Resulting in very serious limitations in the ability to independently initiate, sustain, or complete activities of daily living.

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4.05 Recurrent arrhythmias, not related to reversible causes, such as electrolyte abnormalities or digitalis glycoside or antiarrhythmic drug toxicity, resulting in uncontrolled (see 4.00A3f), recurrent (see 4.00A3c) episodes of cardiac syncope or near syncope (see 4.00F3b), despite prescribed treatment (see 4.00B3 if there is no prescribed treatment), and documented by resting or ambulatory (Holter) electrocardiography, or by other appropriate medically acceptable testing, coincident with the occurrence of syncope or near syncope (see 4.00F3c).

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4.06 Symptomatic congenital heart disease (cyanotic or acyanotic), documented by appropriate medically acceptable imaging (see 4.00A3d) or cardiac catheterization, with one of the following:

A. Cyanosis at rest, and:

1. Hematocrit of 55 percent or greater; or

2. Arterial O2 saturation of less than 90 percent in room air, or resting arterial PO2 of 60 Torr or less.

OR

B. Intermittent right-to-left shunting resulting in cyanosis on exertion (e.g., Eisenmenger's physiology) and with arterial PO2 of 60 Torr or less at a workload equivalent to 5 METs or less.

OR

C. Secondary pulmonary vascular obstructive disease with pulmonary arterial systolic pressure elevated to at least 70 percent of the systemic arterial systolic pressure.

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4.09 Heart transplant. Consider under a disability for 1 year following surgery; thereafter, evaluate residual impairment under the appropriate listing.

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4.10 Aneurysm of aorta or major branches, due to any cause (e.g., atherosclerosis, cystic medial

necrosis, Marfan syndrome, trauma), demonstrated by appropriate medically acceptable imaging, with dissection not controlled by prescribed treatment (see 4.00H6).

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4.11 Chronic venous insufficiency of a lower extremity with incompetency or obstruction of the deep venous system and one of the following:

A. Extensive brawny edema (see 4.00G3) involving at least two-thirds of the leg between the ankle and knee or the distal one-third of the lower extremity between the ankle and hip.

OR

B. Superficial varicosities, stasis dermatitis, and either recurrent ulceration or persistent ulceration that has not healed following at least 3 months of prescribed treatment.

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4.12 Peripheral arterial disease, as determined by appropriate medically acceptable imaging (see 4.00A3d, 4.00G2, 4.00G5, and 4.00G6), causing intermittent claudication (see 4.00G1) and one of the following:

A. Resting ankle/brachial systolic blood pressure ratio of less than 0.50.

OR

B. Decrease in systolic blood pressure at the ankle on exercise (see 4.00G7a and 4.00C16-4.00C17) of 50 percent or more of pre-exercise level and requiring 10 minutes or more to return to pre-exercise level.

OR

C. Resting toe systolic pressure of less than 30 mm Hg (see 4.00G7c and 4.00G8).

OR

D. Resting toe/brachial systolic blood pressure ratio of less than 0.40 (see 4.00G7c).

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