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LETTER FROM THE EDITOR

Dear Readers:

In this edition of Re-flections, Dr. Oscar Cartaya presents the second part of his article exploring the application of disability evaluation concepts relating to the cardiovascular system and life underwriting evaluations; Jeanne Mariani provides us with valuable information about the potential perils and pitfalls of using herbal medications; and I provide some of my thoughts on C-reactive protein, a blood test that has recently gained interest in the lay and medical press.

I hope that you enjoy these articles and that they provide insight and assistance in your underwriting practices.

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C-REACTIVE PROTEIN

Considerable attention has been given to a study published in the New England Journal of Medicine on Nov. 14, 2002. A large group of healthy women were studied over an eight-year period to determine the prognostic value of measuring C-reactive protein (CRP) in assessing the risk of cardiovascular events. The study was designed to compare the value of C-reactive protein and LDL-cholesterol as biologic markers for cardiovascular events using population-based data, and concluded that "the C-reactive protein level is a stronger predictor of cardiovascular events than the LDL cholesterol level…"[1] These new findings are certain to generate debate on how best to use this laboratory test, both as a screening tool for cardiovascular disease, and perhaps as a way of evaluating the effects of treatment aimed at reducing inflammation of the coronary arteries. >>

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>> The idea of measuring CRP in conjunction with acute myocardial infarction dates back to the 1940s, but the test's lack of sensitivity limited its usefulness in detecting the lower degree of inflammation typically found in Coronary Artery Disease (CAD). This test of inflammatory activity is, unfortunately, also not specific to coronary artery inflammation; meaning that elevations of CRP may be due to many other types and locations of inflammation as well. More recently, the availability of highly sensitive assay systems have allowed the development of high sensitivity C-reactive protein (hsCRP) assays that can detect slight elevations in CRP consistent with the amount of inflammation found in coronary arteries or in other parts of the vascular system. CRP testing has a lower limit of sensitivity—about 3 mg/L, whereas hsCRP assays can measure as little as 0.175 mg/L. The problem of specificity has not been satisfactorily addressed, but the level of CRP elevation and correlation with clinical history can be helpful in determining why the CRP is elevated.

What does an elevated CRP mean? CRP is an acute-phase reactant. During severe infection or inflammation, blood concentrations of CRP may increase by a factor of 500 or more. CRP is a protein released into the bloodstream when there is active inflammation in the body. CRP appears to play a role in the process of inflammation by attracting the cells that fight infection. Like other acute-phase reactants, its role in the inflammatory process is probably complex, and it may also promote thrombosis or clotting. The inflammation leading to CRP elevations may be due to various causes, such as infection, injury or conditions like arthritis. Since an inflammatory process is now thought to play a significant role in CAD, it is reasonable to expect that CRP will also be elevated in this condition.

Coronary artery inflammation is thought to produce relatively modest rises in CRP. In general, CRP levels of greater than 15 mg/L should be investigated for non-CAD types of inflammation or infection.[2] Within the usual test range of hsCRP (<0.7mg/L->3.8mg/L), there is a direct linear relationship between the level of CRP and the risk of cardiovascular events. There may also be a relationship between hsCRP levels and the risk of rupture of soft or vulnerable plaques.[3] Post-mortem studies have correlated CRP levels with the appearance of plaque in the coronary arteries, suggesting that hsCRP may be a reasonable biological marker for plaque instability, and by extension, plaque rupture and acute thrombotic events.

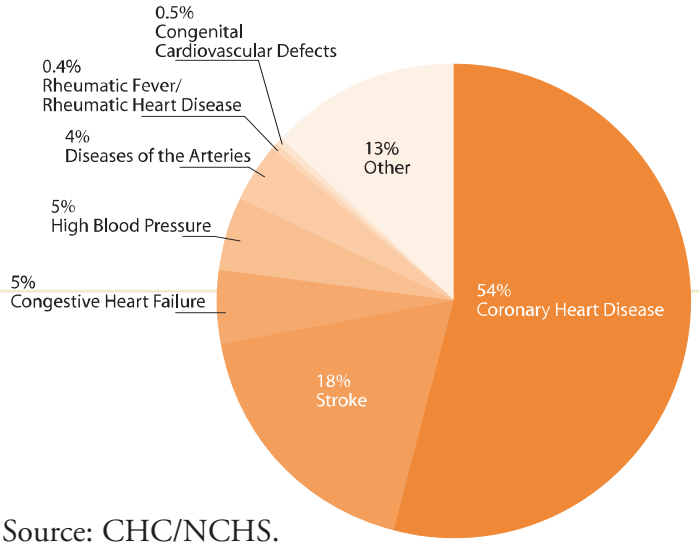
How will clinicians and the insurance industry utilize hsCRP testing? As yet, there is no clearly defined practice guideline for clinicians to routinely screen the general population. In spite of this, there is a reasonable expectation that we will begin to see more reports of elevated hsCRP in clinical records. For insurance purposes, the prognostic value of hsCRP, especially in combination with other more traditional lipid tests, may be useful in decision making.

At present, hsCRP is rarely used in insurance medicine. It is an independent marker for cardiovascular events. Current screening tests certainly identify some of the people at higher cardiovascular risk. Presumably, using hsCRP as a routine screen will increase the size of this group, some of whom are no doubt now being included in the preferred-risk group. If routine testing is accepted, it will likely increase the percent of the insurance population that has an above-average risk for CAD, and since CAD is so highly prevalent in our society, may well alter the relative proportion of preferred- and standard-risk pools.

A further consideration will be how to deal with applicants who are using treatments aimed at lowering serum CRP. Since CRP elevation is correlated to inflammation, it may be that anti-inflammatory drugs like aspirin will have an effect on CRP levels. Whether this will thereby reduce coronary risk is not certain. It is also suggested that the use of statins may inhibit the acute phase response and decrease CRP production by diminishing the deposition of LDL-cholesterol in smooth muscle cells of the arterial wall.[4] This may also reduce the risk of cardiovascular events, and may explain some of the mortality gains attributed to statin use.[5] This new information is certainly useful in our understanding of cardiovascular mortality, but unfortunately it also adds to the complexity of risk assessment. On one hand, elevated-CRP levels suggest increased risk; on the other, if simply taking low doses of aspirin reduces the risk to base-line levels, the value of this test as a risk-stratifying tool may be debatable.

Coronary artery disease (CAD) is a major cause of death in North America, and the evaluation of its presence and severity is a critical function of medical underwriting. It is responsible for 54 percent of cardiovascular mortality, which itself accounts for 30–50 percent of all deaths in developed nations (See Graph 1). The prevalence of cardiovascular disease also increases with age, although it can be difficult to fully determine its presence in otherwise healthy and asymptomatic people (See Graphs 1 and 2). While we know that cardiovascular disease is highly prevalent in older ages, it is present even at younger ages (See Graph 2). Although we know with a fair degree of accuracy the extent that CAD is directly responsible for deaths in the general population, it is not nearly so clear what percent of the insured population has occult coronary disease but dies of other non-related causes. The true extent and early age of onset of CAD can perhaps best be gauged after looking at some of the post-mortem studies of the coronary arteries of young men who died traumatically in the Korean and Vietnam wars.[6] Signs

Graph 1
Percent Breakdown of Deaths From Cardiovascular Diseases
United States: 2000



Source: CHC/NCHS.
Heart Disease and Stroke Statistics—2003 Update,
American Heart Association

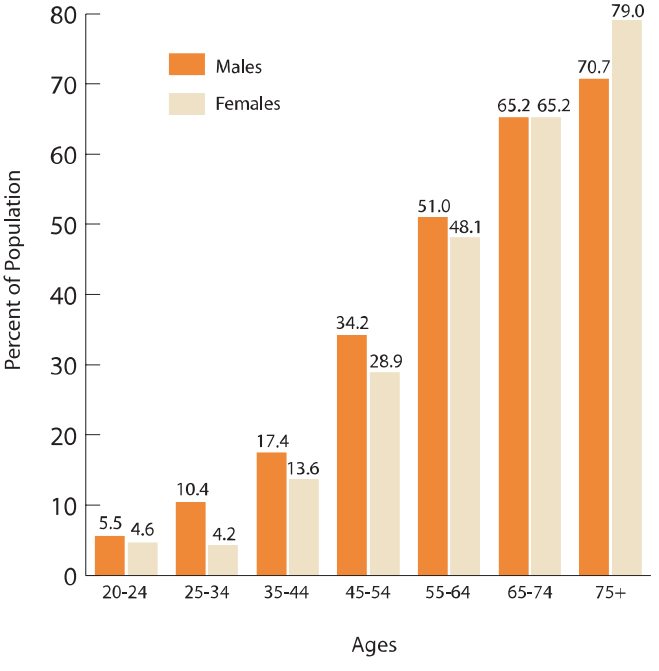
of coronary atherosclerosis were seen in 78.3 percent of the total study group. A further study of young trauma victims examined in the Pathobiological Determinants of Atherosclerosis in Youth Study (PDAYS)[7] showed intimal lesions in all of the aortas and more than half of the right coronary arteries of the youngest age group (15–19 yrs). It is clear from these and other studies that CAD and other forms of cardiovascular disease begin even in the teens. In our society, with such a high prevalence of obesity, hypertension, smoking and diabetes, it is perhaps surprising that CAD is not universally present. As more tests are developed to measure this complex disease process, perhaps some day the medical community will realize that all humans are at risk in one way or another.

A significant number of underwriting requirements exist primarily to detect this coronary risk. These include blood tests, EKGs, stress tests, paramedical examinations and attending physician statements. These clearly represent a significant cost to the insurance industry, but also provide some level of protection against premature claims experience. We have developed a certain degree of familiarity with these tests and how to use the information they provide to stratify risk.

Historically, as an industry we have applied extra premiums to the policies of people who have already developed clinically recognized CAD, whether this is established by abnormal EKGs, ECHOcardiograms, stress testing, perfusion studies, angiography, or medical reports of ischemic symptoms. The advent of lipid testing has changed the way CAD is evaluated. While it is accepted that hyperlipidemia plays a role in the pathogenesis of CAD, it appears that not all people with known hyperlipidemia have clinically recognized CAD or will even develop it. Hypercholesterolemia (specifically LDL-cholesterol) is recognized as a risk factor for the development of CAD, but its mere presence does not guarantee that the coronary arteries are already diseased, or indeed that they will ever be diseased enough to lead to angina, infarction or death. The same can be said for all the other known risk factors for the development of CAD such as low HDL-cholesterol, smoking, hypertension, or diabetes. This will likely also apply to CRP.

When evaluating risk factors, the real issue is whether medical underwriters should treat applicants with a propensity or pre-disposition for a disease in the same manner as those who have already demonstrated overt disease. This may very well become the most significant question to face underwriters and the legislators that govern us. We are slowly developing a fuller understanding of the pathogenesis of diseases such as CAD. Scientific publications like the New England

Graph 2
Prevalence of Cardiovascular Diseases in Americas, Age 20 and Older by Age and Sex
United States: 1988-94



Source: NHANES III (1988-94), CDC/NCHS.
Heart Disease and Stroke Statistics—2003 Update,
American Heart Association

Journal of Medicine have recently published articles suggesting that tests such as hsC-reactive protein have the same prognostic value for CAD as tests like LDL-cholesterol. Assuming we continue to probe into the pathogenesis of CAD, we are likely to find more information pointing to important environmental and/or genetic factors.

In many diseases, the interplay between host and environment determines the outcome. We are all exposed to a plethora of insults to our bodies, yet each of us is a unique individual and we react somewhat differently to these stressors. It is probably safe to say that this also applies to how our coronary arteries respond to the factors that trigger the inflammatory process. Ultimately, we may possess the genetic knowledge that allows us to determine a person's risk of developing serious diseases many years in the future. How we use these early predictors of potential extra mortality will become increasingly important as our knowledge grows.

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HERBAL SUPPLEMENTS

The use of herbal supplements in the United States is increasing. Many people feel that herbs are a safer and more natural way to treat ailments than traditional medicine. Although some herbal supplements are safe and effective, others can be dangerous, even life threatening, especially when mixed with other medications. Remember, many powerful traditional medicines such as digoxin and taxol are derived from plant extracts. The difference is that FDA-approved drugs are required to go through rigorous efficacy and safety testing before they are released to the U.S. market; herbal supplements are not. This article includes general information about herbal supplements, followed by information about some specific popular herbs.

Prevalence of Use

In studies completed in 1990 and 1997, surveys of the general population showed herbal supplement use rose from 2.5 percent to 12.1 percent. In a more recent study of adults in the Minneapolis/St. Paul metropolitan area in 2001, 61.2 percent of adults who responded indicated that they had used herbal supplements within the past year. Other studies show varying rates of use from 10 percent to 40 percent. It is obvious to anyone who shops at the mall or the local grocery store that herbal supplement use is becoming very popular. In addition, these surveys indicated that most people using herbs are receiving information about the treatments from family and friends, not from doctors. In fact, many do not even inform their doctor about herbal supplement use, which leads me to conclude that it is unlikely they are informing their insurance agent or paramedical examiner, either.

U.S. Food and Drug Administration Regulation

Before Congress passed the Dietary Supplement Health and Education Act (DSHEA) in 1994, dietary supplements (which include vitamins, minerals and herbs) were subject to the same FDA regulation as other foods. In 1993, the FDA sought to establish regulations requiring manufacturers of herbal supplements to prove the efficacy of products. However, due to the expense of the studies and the fact that long-used herbal remedies could not be patented, there was a backlash from manufacturers and users of these products. As a result, the DSHEA passed with no proof required from the manufacturer of the safety and efficacy of herbal supplements. In addition, herbal supplements are not subject to the same quality control as traditional medicines, meaning that they can contain contaminants, such as pesticides or heavy metals, and that the amount of active ingredient can vary from product-to-product. Manufacturers of herbal supplements can make claims about a product's action without scientific research to back up claims, which, according to the DSHEA, must be "truthful and not misleading". The FDA has the authority to remove any herbal supplements from the market that are found to be dangerous, but it does not have the authority to test the products.

SOME COMMON HERBAL SUPPLEMENTS

Ginkgo Biloba (gingko, maidenhair-tree)

Commonly used to improve memory, Ginkgo is actually an anticoagulant and, as such, is also used for circulation problems. Studies show that ginkgo provides some benefit

for patients with dementia and intermittent claudication (AmJMed 2000; 108: 276-281). However, it does not prevent the onset of dementia or Alzheimer's disease in healthy adults, nor does it enhance memory (JAMA 1997; 278: 1327-32; JAMA. 2002; 288: 835-840). Ginkgo should not be used in conjunction with other medicines that affect clotting time, including aspirin, plavix or coumadin. Side effects of ginkgo include muscle spasms, cramps, bleeding and digestive problems. Other anticoagulant herbal supplements include feverfew, garlic and ginger.

St. John's Wort (amber, goatweed, hardhay, klamath weed, tipton weed)

St. John's wort is used to relieve depression and is proven effective in the treatment of mild depression with fewer side effects than some prescription antidepressants. However, it is not effective against major depression. St John's wort can effect the etabolism of many other drugs, causing either a buildup or decrease of the medication in the bloodstream. Therefore, it should not be taken concurrently with any other drugs, especially chemotherapy, digoxin, theophylline, cyclosporine, or antidepressants.

Saw Palmetto (sabel)

Saw palmetto is used to treat symptoms of benign prostatic hypertrophy (BPH). Studies indicate that saw palmetto can improve urine flow and emptying of the bladder. Side effects include upset stomach.

Ephedra (ma-huang, herbal ecstasy)

Ephedra is found in a number of diet/appetite suppression preparations (such as Herbalife, Metabolife) and in herbal products that claim to enhance energy. Ephedra is a stimulant, and should not be mixed with caffeine, other stimulants, decongestants, cardiac drugs or antidepressants. Taking ephedra is proven to increase the risk of MIs, CVAs and seizures (NEJM 2000; 343(25): 1833-1839), and the risk is increased when ephedra is mixed with any of the substances listed above. Recently, the FDA proposed new warning labels on ephedra in light of reports of at least 100 deaths linked to its use. Consumer groups are pushing for a ban on the sale of ephedra.

Of note, other diet products that do not contain ephedra, such as dieter's tea, contain potent laxatives and diuretics. The weight loss effect, which is actually the result of large losses of fluid, can also cause dangerously low levels of potassium. This can lead to dehydration and cardiac arrhythmias.

Echinacea

Echinacea is used for preventing or shortening the duration of the common cold by stimulating the immune system. Studies regarding its effectiveness are inconclusive, possibly because there are different species of echinacea plant and different parts of the plant may be used to manufacture the final product. A recent study conducted on college students with early cold symptoms found no detectable difference in the length of illness in students given unrefined echinacea and those given a placebo (Ann Intern Med. 2002; 137: 939-946). Other studies showed that it may have some effectiveness. Echinacea can affect the liver, and should not be taken with any other medication that affects the liver, or in combination with immunosuppressants.

There are a number of other herbals that affect the liver including kava, comfrey, chaparral, black cohosh and celandine. It would stand to reason that some of the elevated liver-function results on insurance-blood testing with no known etiology may be the result of herbal supplement use.

In conclusion, herbal preparations can be potent and effective health supplements if they are taken cautiously and under a doctor's supervision. Although they can be effective in certain cases, using them the wrong way or in combination with the wrong medication can lead to serious side effects, or even death. As underwriters, we should be aware of the different popular herbal supplements available and the possible consequences of use by our applicants.

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DISABILITY CONCEPTS IN LIFE INSURANCE UNDERWRITING, II

Cardiovascular System

This is the second in a series of articles exploring the relationship between current disability claim practice and life underwriting practice. As in the prior article (*Reflections*, Volume 10, Nov. 2002), I will use adaptations of the functional-capacity concepts developed by the U.S. Social Security Administration (SSA). In effect, applicants who are so functionally limited as to be unable to perform standard job functions are not always good life underwriting risks and should be evaluated with extra care. This is particularly true when dealing with disorders of the heart.

Disorders of the heart do not provide easily determinable endpoints that can be used to establish physical limitations or life expectancy. The evaluation of applicants with these disorders is based upon careful review of prior medical history and current functional testing. For both life underwriting and disability claims, the applicant's cardiac history and test findings are evaluated in order to reach valid conclusions about current residual-function capacity as well as life expectancy.

Most of this is, of course, routine. However, there are specific methods used in disability-claim adjudication that are not well-known within the life-underwriting community. These methods are useful in identifying a subset of the applicant pool that is severely limited or disabled by cardiac problems and should be underwritten conservatively. Some of these severely impaired applicants may not be readily recognizable through regular life-underwriting methodology. This article deals with these disability-claim evaluation methods, most of which are derived from the SSA disability listings and SSA claims practice.

Cardiovascular

Limitations of cardiac function resulting in total incapacity to perform useful work result from five primary causes:

1. Heart failure
2. Angina
3. Syncope due to poor central nervous system perfusion
4. Cyanosis from congenital or pulmonary vascular problems
5. Peripheral vascular disease

In terms of functionality, these conditions affect applicants in similar ways. Symptoms appear at increasingly lower levels of stress as the applicant becomes progressively limited in what he can do. Using standard life underwriting techniques, it is somewhat difficult to establish levels of functional limitation consistently; therefore, functional limitations tend to be used as just another risk factor. In reality, the level of functional limitation resulting from cardiac disease can be evaluated and underwritten in a systematic fashion if the right methods are used.

In order to be useful for insurance purposes, measures of cardiac limitations must be consistent and replicable. The New York Heart Association (NYHA) Classification provides a systematic framework useful in evaluating the effects of cardiovascular disease on function. This is a broadly used therapeutic classification for heart function originally developed to evaluate residual function in patients with congestive heart failure, but in fact, it is applicable to many cardiovascular conditions. The NYHA Classification is based upon the level of activity that causes symptoms in a patient, as follows:

Class I	No activity limitations or symptoms.
Class II	Minor activity limitations, can tolerate some degree of exertion and has no symptoms at all at rest.
Class III	Marked limitation of activity, cannot tolerate any but the lightest exertion level, comfortable at rest.
Class IV	Extreme limitation of activity, symptoms occur at rest, cannot tolerate any exertion.

Under the NYHA Heart Classification (See Table 1), Class I is equivalent to no limitations; Class II is equivalent to light/medium work limitations (lift/carry 20 lbs. with occasional lift/carry of 50 lbs.); Class III is equivalent to sedentary work limitations (lift/carry of up to 10 lbs. on an occasional basis, limited standing walking requirements); and Class IV is

Table 1
Limitation Levels, Standard Classification

1.) No Limitation. No weight-based lifting or carrying limitations, no limitations of standing, walking or hand use.
2.) Medium Limitation. Limited to lift/carry of 50 lbs. on an occasional basis with constant lift/carry of up to 25 lbs. No limitations of standing, walking or hand use.
3.) Light Limitation. Limited to lift/carry of 20 lbs. on an occasional basis with constant lift/carry of up to 10 lbs. No limitations of standing, walking or hand use.
4.) Sedentary Limitation. Limited to lift/carry of 10 lbs. on an occasional basis with constant lift/carry of less than 10 lbs. Standing/walking limited to no more than two hours per working day. Hand use may be limited to a minor degree.
5.) Less Than Sedentary (totally disabled). Limited to lift/carry of less than 10 lbs. on an occasional basis, no significant lift/carry weight tolerated on a constant basis. Standing/walking limited to less than two hours per working day. Significant limitations of hand use may be present.

equivalent to less than sedentary work limitations (disabled). See Table 1 for definitions of the limitation levels. In actual use, these classes may be combined according to the symptoms experienced by the individual. For example, a man who is able to work loading trucks but occasionally gets chest pains while doing so would be a Class I to II. A man that can take occasional long walks but cannot tolerate lifting over 10 lbs. on a regular basis, and occasionally has chest pain while sitting watching television would be a Class III to IV. The NYHA Classification is not an objective tool, but it is widely used in disability-claim practice because it allows for quick placement of a claimant within a standard functional scale.

The NYHA Classification framework may appear easily applicable to disability evaluations but actually is not, due to variability in how patients experience symptoms, particularly chest pain and shortness of breath. This type of empirical, subjective evaluation of an individual's functionality based upon record and test reviews depends heavily upon the reviewer's level of expertise. A consistent and replicable application of the NYHA Classification to insurance cases requires a high level of expertise in evaluating cardiovascular symptoms. This is not a methodology that can be applied consistently by personnel with a low level of training or limited experience. However, this does not negate the validity of the NYHA Classification; applicants classified as NYHA Class IV cardiac patients after a valid review by a qualified examiner are disabled from gainful work. These applicants also have a very high cardiovascular mortality and cannot be considered acceptable life insurance risks.

In disability practice, it is desirable to avoid decisions that are based largely upon subjective evaluations. Therefore, sets of objective criteria have been developed around functional limitations resulting from cardiovascular disease. Of these, the most useful are the criteria developed to evaluate standard exercise tolerance tests. SSA has developed very specific, objective criteria for the interpretation of standard exercise stress tests (Bruce protocol) to evaluate cardiac-function capacity. The SSA criteria are used to evaluate inability to function due to an underlying cardiac condition. Under SSA listing rules, a claimant is considered totally disabled if the following general conditions are met:

Table 2
Criteria for Symptomatic Heart Disease

1. Cardiac enlargement by appropriate imaging techniques as follows:
   a) Cardiothoracic ratio of over 0.5 on a PA chest X-Ray.
   b) Echocardiography showing a left ventricular diastolic diameter of greater than 5.5 cm.
2. The presence of a S3 sound on physical examination, or a left ventricular ejection fraction of 30 percent or less.
3. Symptoms of shortness of breath or cardiac overload with exertion.
4. Chest pain with exertion while on a medical treatment regimen.

1. A claimant must have active symptomatic heart disease as defined in Table 2.

2. A standard Bruce stress test reveals the following:
   a) Inability to reach five METS* prior to termination of the test.
   b) Inability to increase the systolic blood pressure by 10 mm Hg. during exercise, or to decrease the systolic pressure below the usual resting level in the recovery phase.

3. A standard stress perfusion test (thallium test or similar) documenting reversible perfusion defects at a workload equivalent of 5.0 METS or less is indicative of total disability.

4. For applicants with a diagnosis of Congestive Heart Failure (CHF) a standard Bruce stress test reveals the following:
   a) Evidence of inadequate cerebral perfusion (ataxic gait or mental confusion, for example) during the exercise test.
   b) Runs of three or more Premature Ventricular Beats (PVB) during the test.

5. For applicants with a diagnosis of Coronary Artery Disease (CAD) a standard Bruce stress test reveals the following:
   a) A horizontal or downsloping 1.0 mm ST segment depression during the exercise phase at a workload equivalent of 5.0 METS or less, persistent for at least one minute into the recovery phase.
   b) An upsloping 2.0 mm ST segment or junctional depression during the exercise phase at a workload of 5.0 METS or less, persistent for at least one minute into the recovery phase.
   c) A 1.0 mm. ST segment elevation during the exercise phase at a workload of 5.0 METS or less, persistent for at least 3.0 minutes into the recovery phase.

6. An applicant's doctor concludes that the performance of a standard Bruce protocol stress test presents a significant risk to the applicant, and the applicant suffers with symptomatic heart disease as listed in Tables 1 and 2.

*Note: A MET or Metabolic Equivalent is defined as the level of oxygen consumption at rest, or 3.5 milliliters of oxygen per minute per kilogram of body weight. The level of oxygen consumption, or METS, is related to the level of exercise. Five METS in a standard Bruce protocol stress test is roughly equivalent to three minutes of exercise.

The criteria listed in Table 2 were developed to evaluate cardiac functional capacity, not to diagnose ischemia or coronary artery disease. Indeed, a pre-existing diagnosis of congestive heart failure or coronary artery disease is required for the application of these criteria. Meeting these criteria accurately identifies severe cardiac functional limitations and the inability to do gainful work because of cardiovascular problems.

The application of these cardiac functional limitations to life insurance underwriting evaluations is straightforward. It is generally recognized that applicants incapable of tolerating a standard level of stress have a higher mortality risk. However, there are no generally accepted life underwriting standards for rating applicants who have very poor exercise tolerance during standard stress tests, (Bruce protocol). In effect, the SSA criteria presented provide standardized measures to identify applicants with severe cardiac functional limitations. Applicants with impaired cardiac functional capacity during standard stress tests who meet the criteria listed above have an unacceptably high mortality risk and should be underwritten accordingly.

Other criteria developed by the SSA for cardiac-functional evaluations appear less useful in life underwriting practice. For example, Peripheral Vascular Disease (PVD) is evaluated by the use of Doppler studies. If the ankle/brachial systolic blood pressure ration is less than 0.50, the claimant is totally disabled by PVD.

The application of the SSA guidelines may help eliminate some of the subjectivity in rating applicants with cardiac disorders and poor exercise tolerance. It is important to realize that heart and lung disorders are often found together in the same individual, particularly where there is a smoking history. In cases of combined heart and lung disease, the end stage presents with significant functional limitations arising from common pathophysiological pathways and manifestations that are caused by both heart and lung disease acting together in a synergistic way. The shared manifestations of these disorders are mediated by inadequate oxygenation of the blood and inadequate blood flow patterns. These applicants have chest pain, shortness of breath, exercise intolerance and arrhythmias, all of which cause limitations and increase mortality risk. Applicants with significant cardiovascular and respiratory limitations should be evaluated carefully when applying for life insurance.

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