Some New Concepts in Coronary Artery Disease Which May Influence Insurance Medicine

by
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In the general population of the United States the mortality for the leading cause of death - namely, heart disease - has been decreasing somewhat in recent years. The death rate for the first six months of 1981 as compared to the average mortality during the corresponding months of 1976-1980 was somewhat lower for major cardiovascular diseases (1). Coronary artery disease is, of course, the largest contributor to this mortality.

Fortunately, during the past five to ten years an increasing amount of basic information has been obtained concerning disease. The information has altered the management of this widespread illness in many important areas, and therefore has real impact upon insurance medicine. A few of these new concepts will now be reviewed.

DIAGNOSTIC TESTS

The fundamental concept that myocardial ischemia is the major evidence for coronary artery disease remains the keystone of diagnosis and management. The details of the location of this ischemia, and what is more difficult, - the mechanism of production of the decreased myocardial blood flow - are being uncovered. Exercise testing, utilized as a form of stress to demonstrate a deficit in myocardial oxygen delivery when myocardial oxygen demand exceeds supply, has long been a tool utilized clinically in evaluating coronary disease. As a diagnostic maneuver it is utilized extensively in insurance medicine, - especially in the hope of revealing cryptic disease. The end point, or prime diagnostic feature, has for many years been one deviation alone - ST depression on the electrocardiogram. The simplicity of such a criterion is attractive but, we now know, is deceptive and other features found in treadmill and other exercise tests have recently been accepted as offering a more quantitative aspect for the disclosed ischemia. With quantitation and more precise localization of the ischemia identification of bad risks becomes easier. Correlations between angiograms and treadmill testing accompanied by myocardial scanning done during the treadmill exercise, have shown, for example, that depressions of 3 mm or more in ST segments are indicative of three-vessel disease, a higher risk group than those with fewer involved arteries. Another important point noted in a recent review (2) stresses that in symptomatic men the presence of an abnormal ST response during a stress test almost always predicts the presence of significant coronary disease, but that in asymptomatic men (often insurance applicants) and in women the correlation between a positive stress test and coronary disease is much weaker. Thus further criteria are needed and this group at the Evans Memorial in Boston (2) makes a plea that the interpretation of exercise testing should include other parameters than simple ST depression. These are, for example, the amount, time of onset and duration of the ST depressions, the duration of time the subject was able to exercise, the number of leads showing ST depressions, exertional hypotension, poor heart rate response to exercise and exercise-induced chest pain. Obviously this is an attempt to diagnose severity, as well as presence, of coronary disease. As an example of risk identification they studied (2) 436 patients to identify, if possible, exercise variables that could serve as predictors of left main or three-vessel coronary disease. The coronary vessel anatomy was identified by coronary arteriography. A pattern of 2 mm or greater downsloping ST segment depression that starts in Stage I of the treadmill test (Bruce protocol) lasts at least six minutes into the recovery period and is displayed in at least five leads was highly predictive (74 percent) and reasonably sensitive (49 percent) for the detection of these two forms of high risk coronary disease. Clearly the more sophisticated analyses of treadmill tests will be useful in insurance medicine. If allied to treadmill tests one can find material from myocardial scanning with thallium, measurement of ejection fraction by radionuclide cineangiography and ventricular wall motion by echocardiography in the ap-
In some cases, especially combined with other anti-anginal drugs. The uses of calcium blockers in acute myocardial infarction are still being studied. One can summarize the present therapeutic plan for angina as follows: nitrates remain the initial drugs of choice. If the angina is more than moderate and frequency of pain unacceptable other drugs are needed. In effort angina betablockers can then be added, and, in turn, if angina is still poorly controlled a trial of a calcium blocker may be effective. If rest angina is present and coronary spasm can be identified as the major mechanism (e.g., there is a variable threshold for the pain, there is ST elevation during pain and no heart rate or blood pressure change) a favorable effect can be found with a calcium blocker along.

The long-term effects of coronary spasm and/or its successful treatment with drugs is, of course, as yet unknown although of special interest for evaluation of medical risks.

NEW MECHANICAL MANEUVERS IN TREATING CORONARY DISEASE

Reduction of Infarct Size

Considerable interest has been developing over the past ten years (6) in means to modify the area of myocardial necrosis after a myocardial infarction. Depending upon the basic pathophysiology, i.e., the extent of coronary obstruction of a chronic anatomic type plus the contribution of thrombosis and vessel spasm, a number of centers are using immediate infusions of drugs (usually via intracoronary artery injection) as soon as an infarction is proven. Nitroglycerine may relieve the vessel spasm if present and is often the first approach if occlusion is noted by angiogram. If no response occurs, streptokinase (to dissolve intracoronary thrombi) is then given directly into the occluded artery. The streptokinase infusion has had substantial success in 70-90 percent of trials. However, a reclosure rate of 33 percent (in the data from the Massachusetts General Hospital (6)) remains a problem. The infusion must be started within 3-4 hours of the occlusion to be effective in limiting the myocardial injury. These approaches will deserve careful follow-up at present but offer real hope of salvaging heart muscle during an infarction.

Percutaneous Transluminal Angioplasty in Coronary Occlusion

A second new approach to relief of occluded coronary vessels is the method started by Gruntzig (7) in Zurich and now available in several other countries. This entails dilatation of partially occluded coronary arteries by an intracoronary balloon threaded into the vessel by a catheter. The technique must be mastered over some time but the operator can achieve opening or some real dilatation in 80 percent of cases attempted. Single vessel disease is more suitable for this than multivessel involvement (7). However, 20 percent of initially successful patients will restenose in a few months and the long-term prognosis of these subjects
is as yet unknown. The National Heart, Lung and Blood Institute has compiled a registry of the first 1,000 cases done here and its results are awaited to help evaluate this new procedure. A preliminary note indicates a success rate of dilatation of 60 percent.

**Surgical Treatment of Arrhythmias**

Sudden cardiac death is probably the major challenge to current cardiology. It is said that 600,000 persons die yearly from cardiac disorders in the U.S.A. and these are predominantly deaths due to coronary disease. It has been estimated that two-thirds of these deaths occur outside the hospital and result from arrhythmias. These data emphasize the need for continued extensive studies of these life-threatening rhythms. The surgeon has now joined the cardiologist and electrophysiologist in helping to identify the abnormal physiology and, more recently, to assist in treating the dangerous arrhythmias (8). Precise electrophysiologic mapping is necessary pre-operatively. Drug therapy, of course, must have failed. The types of arrhythmias now being treated surgically are those seen in a small subgroup of Pre-excitation of the WPW type, recurrent ventricular tachycardia insensitive to drugs, rare cases of atrial tachycardia with serious consequences and excessive ventricular rates during atrial flutter or fibrillation resistant to drug blockage of the AV node. The surgeon either severs a bypass tract involved in the circuit allowing an arrhythmia to develop, ablates an ectopic focus in atria or ventricles or cuts the AV connection at the node or bundle of His.

Some real success has been achieved, and again, this is an area to watch, especially with the latest probe - the laser beam - allowing for better surgical techniques and less risk.

**REFERENCES**


**Roster of Emeritus Members for Insurance Examinations**

In the last issue Dr. Francis Bicknell was mentioned as having indicated an interest in performing physical examinations in and around South Yarmouth, which was correct. But, we located the city in the wrong state. The correct state is Massachusetts. For this we apologize to Dr. Bicknell and our readers.

To repeat the doctor's full address, it is 69 Keel Cape Drive, South Yarmouth, Massachusetts 02664.

Another Emeritus member, Dr. George A. Simpson of 147 Norwood Avenue, Asheville, North Carolina 28804 has indicated an interest to be an examiner in the Asheville area. He can be reached by telephone at (704) 252-2600.