SYMPOSIUM
Angina Pectoris

Introduction

Some Comments and Reflections on Changing Interests
and New Developments in Angina Pectoris

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RATHER SUDDENLY angina pectoris has become a glamor subject, intellectually and clinically exciting in almost every aspect—pathology, pathophysiology, diagnosis, treatment. Not long ago it was difficult to spark a bright glow of interest in angina pectoris, except possibly by reading Heberden’s sharp delineation of the clinical picture: “There is a disorder of the breast . . . They who are afflicted with it, are seized, while they are walking (more especially if it be uphill and soon after eating) with a painful and most disagreeable sensation in the breast, which seems as if it would extinguish life, if it were to increase or continue, but the moment they stand still, all this uneasiness vanishes . . .” Any further discussion was regarded as dull and drab.

The physical examination was thought to be unrevealing in uncomplicated cases. The electrocardiogram at rest was often or usually normal and noncontributory. To the less knowledgeable physician this sometimes led to diagnostic confusion—for how could the patient have so serious a disease as angina pectoris with a normal electrocardiogram? With the history often regarded as subjective and equivocal, and the physical examination and resting electrocardiogram noncontributory, the two-step test became the final arbiter, and no diagnosis of angina pectoris was genuine without the 14-carat imprimatur of a positive two-step test.

Treatment was simplistic. Sublingual nitroglycerin was consistently effective in relieving the acute attack provided it was taken soon enough to dissolve before the attack subsided spontaneously. Bottles of 100 were to be renewed frequently, even if only a few tablets had been used, or else they were said to be ineffective. The mechanism by which nitroglycerin relieved the pain was obvious and unquestioned: the drug promoted coronary blood flow by dilating the stenotic atherosclerotic vessels. Then long-acting nitrates were developed for prolonged vasodilation of the coronary arteries and prolonged protection against angina pectoris. Patients were convinced that certain well-advertised brand-name products were effective, whereas those with generic or little known brand names, of identical chemical structure, were not. Aside from occasional severe headaches and the cost of the drugs, their use led to no important unfavorable consequences unless the physician discontinued effective nitroglycerin as no longer necessary because of the alleged all-day protection of the long-acting nitrates. A host

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other drugs, designed to promote coronary blood flow, have come and gone. Had they been successful we would not now be viewing a spectacular pullulation of coronary surgical procedures.

Current Status

Pathology

Angina pectoris has entered a new era of study and knowledge. Even the pathology of angina pectoris has provoked renewed interest, not because of new discoveries but because of the increased importance of familiarity with old observations, and because of the possibility of visualizing coronary pathology in vivo. Although Vlodaver, Neufeld, and Edwards discuss numerous forms of coronary artery disease, practical considerations call for emphasis on occlusive atherosclerosis and aortic valvular disease as the essential causes of angina pectoris. Coronary atherosclerosis progresses for 20–40 years or longer in a preclinical form and may be asymptomatic even when the pathologic disease is extensive and moderately severe. When angina pectoris occurs, even if it is relatively mild and stable, it represents not early and moderate, but advanced and very severe occlusive atherosclerosis of the coronary arteries. Hemodynamic disturbance and consequent myocardial ischemia probably do not occur until the lumen of a major coronary artery is reduced in cross-sectional area by at least 75%.1 If allowance is made for collateral circulation, it is more likely that angina pectoris occurs only when there is 90–100% occlusion. These conclusions are amply supported by the classic studies of Schlesinger, Blumgart, and their associates.2 Blumgart, Schlesinger, and Davis3 demonstrated that angina pectoris occurred as a rule after two, and exceptionally after one, major coronary artery was occluded and the remaining major branches were severely narrowed.

These considerations are important in relation to current interpretations of coronary angiographic findings as indications for coronary artery surgery. Prior to coronary arteriography physicians treated most of their patients with angina pectoris without serious thought of surgery, reassured by the knowledge that the angina was of only moderate severity and frequency, was responsive to rest and nitroglycerin, and did not seriously interfere with the patient's work. Furthermore, the vast majority of such patients continued in this manner for many years.

At present after such patients undergo coronary arteriography, the physician is shocked when he sees that one major branch is completely and another, half or three quarters occluded. Coronary surgery is then suddenly regarded almost as acutely mandatory, lest the occlusion become completed or another vessel occluded. Yet that which the coronary angiogram displayed is what should have been anticipated as almost minimal findings once the diagnosis of angina pectoris was made. Blumgart, Schlesinger, and Zoll4 found that when persons with a history of angina pectoris came to autopsy there was an average of 3.5 occlusions per heart. The prognosis for the patient with 1% occlusions in his coronary arteriogram is no different from the prognosis of the patient with angina pectoris without a coronary arteriogram. Subsets of occlusive coronary disease as visualized on arteriograms and of patients with angina pectoris, and correlations between the two groups will have to be studied extensively and for years before arteriograms can be used as prognostic indicators.

Other aspects of coronary artery pathology have stimulated new or renewed interest. Normal coronary arteriograms have been reported in 5–10% of patients alleged to have typical angina pectoris. It remains to be determined whether the coronary angiograms in such cases have truly represented the actual pathology as revealed at autopsy. Preoccupation with small-vessel disease to explain these cases seems poorly founded. Angina pectoris is not a feature of a number of generalized diseases which may involve the small vessels of the heart but not the main coronary artery branches.

Angina pectoris has long been regarded as a harbinger of sudden death. As a rule sudden
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Death of a patient with a history of angina pectoris is not associated with an acute thrombosis (occlusion) or acute myocardial infarction. If the mechanism responsible for sudden death, e.g., ventricular fibrillation, is sparked by an acute ischemic episode, it would be informative if one could demonstrate early severe ischemic changes at autopsy when death occurs too rapidly to permit the development of myocardial necrosis. The recently reported nonenzymatic hematoxylin-basic fuchsin-picric acid stain, which stains ischemic tissue but neither normal nor necrotic tissue, may provide such an anatomic basis for sudden death in angina pectoris.

Coronary Blood Flow

It has always seemed fairly obvious that there is a reduction in coronary flow in patients with angina pectoris due to atherosclerotic occlusive coronary disease. Nevertheless a variety of methods introduced over the years failed to disclose such diminution in coronary perfusion. Recent studies have clarified this apparent discrepancy. In severe, occlusive coronary disease, blood flow is nonhomogeneous and regional hypoperfusion is obscured by normal or increased flow in the relatively normal coronary branches. The determined flow is then an average coronary blood flow. There is great need for a method to determine regional myocardial perfusion in angina pectoris, since regional ischemia is the essential physiologic basis of the disease. Combined with cinecoronary angiography it would provide a greatly improved evaluation of the extent, distribution, and severity of the underlying disturbance. Equally or more important, it would enable us to assess more objectively the effects of therapy, notably drugs and coronary surgery. If regional myocardial ischemia is the basis of angina pectoris, any effective treatment should relieve or abolish this deficiency.

A variety of methods for determining regional myocardial blood flow is now being investigated. They suffer from the need for an intracoronary or intramyocardial injection, deficiencies in available equipment, and also the relatively high cost of equipment and isotopes. Promising methods include the use of a scintillation camera with multiple crystals, each individually collimated, as well as a computer to calculate rates of myocardial blood flow. The Anger scintillation camera, which has a single crystal with multihole collimation, is also being used to disclose regional deficits in coronary perfusion. The various methods of measuring coronary blood flow and their shortcomings as well as recent developments are discussed in the article by Bing and Hellberg.

Pathophysiology and Hemodynamics

That angina pectoris is due to a disparity between myocardial oxygen consumption \( (\text{MVO}_2) \) and oxygen supply has long been surmised. Recent interest has centered on the various factors which determine \( \text{MVO}_2 \) and their role in the production of angina pectoris—heart rate, arterial pressure, preload, ventricular volume and mass, and contractility. The mechanism by which exercise (especially after a meal and in cold windy weather) and emotion precipitate angina pectoris is now reasonably understandable in terms of their enhancement of \( \text{MVO}_2 \) chiefly by increasing heart rate and blood pressure. On the other hand Prinzmetal's angina cannot be explained in terms of an increased \( \text{MVO}_2 \) since it is not preceded by a rise either in heart rate or arterial pressure. Consequently it is assumed that the angina results from a reduction in coronary perfusion, perhaps due to coronary artery spasm.

The action of drugs in angina pectoris may also be interpreted in terms of their influence on coronary blood flow (oxygen supply) and the factors which control myocardial oxygen consumption. Propranolol and other beta-adrenergic blocking agents reduce \( \text{MVO}_2 \) by diminishing three major determinants: heart rate, arterial pressure, and contractility. The action of nitroglycerin is less clear. That its benefit is due to dilation of coronary arteries was doubted when determinations of coronary blood flow showed no increase, but this may have been due to deficiencies in...
methodology. Newer methods of determining regional blood flow may show that nitroglycerin does indeed increase perfusion of ischemic areas. Nitroglycerin may also reduce MVO₂ by causing extracardiac (peripheral) arteriolar and venous dilation and consequent fall in arterial pressure as well as in left ventricular volume and pressure. Presumably this factor outweighs any effect of nitroglycerin-induced tachycardia. However, reports of the effect of nitroglycerin on MVO₂ have been contradictory.

A variety of metabolic, hemodynamic, and functional disturbances, recently recognized as consequences of myocardial ischemia, are discussed in the article by Cohn and Gorlin. Production of lactic acid by the myocardium instead of the normal myocardial extraction, indicative of anaerobic metabolism, is now being used as an index of myocardial ischemia, but may be demonstrable only when the myocardium is stressed. An elevated left ventricular end-diastolic pressure, indicative of diminished compliance, may be present constantly or only during an anginal attack. It has been related to the abnormal pulsation of the chest wall and an atrial gallop sound sometimes present in patients with angina pectoris. Segmental disturbances in ventricular contraction have also been observed and attributed to myocardial ischemia. There is an apparent dissociation between these hemodynamic and functional abnormalities and the onset of electrocardiographic S-T depressions and the chest pain which appear somewhat later. The presence or absence of left ventricular hemodynamic and functional abnormalities and their severity have been used to identify subgroups of patients with angina pectoris for purposes of prognosis and operative indication. Assessment of these abnormalities before and after coronary surgery has provided an objective evaluation of the effectiveness of coronary bypass surgery.

Clinical Aspects of Angina Pectoris and its Diagnosis

There has been little new in our knowledge of the symptomatology of angina pectoris, but coronary angiography has served to validate the high degree of accuracy of the diagnosis based on symptomatology. When a typical history of angina pectoris is obtained in a patient without aortic valvular disease or left ventricular outflow tract obstruction, severe obstructive coronary disease is present in about 90% of patients; conversely, such abnormality is observed in only 5% of patients with chest pain which is clearly nonischemic. There is need for careful studies of a third group of patients with "atypical angina pectoris" in whom severe obstructive coronary lesions may be found in approximately 40%. Discriminant analysis may reveal features in the history which could distinguish "atypical" cases which should be classified as typical angina pectoris from those which are definitely nonanginal.

It would be of interest to determine which of the host of symptomatic features of angina pectoris should be programmed and what relative weights assigned, if we were to attempt a computer diagnosis. It is doubtful that the numerous descriptive terms indicating the quality of the pain are necessary for diagnosis, or indeed specific enough by themselves to be reliable. Neither is the exact location of the pain or its radiation absolutely essential for diagnosis. Major characteristics provide a minimal basis for accurate diagnosis:

1. Pain, for which other terms such as pressure, tightness, or heaviness may be substituted, or even breathlessness if this is associated with any discomfort in the chest. Its location, though generally in the anterior or posterior chest, is less important provided it is not confined to the lower extremities or head. Thus pain may represent angina pectoris even if it is confined to or located predominantly in the upper extremities or abdomen, provided it is induced by walking rapidly or by emotion. Radiation is likewise not essential for diagnosis. Radiation to the upper extremity or extremities, though helpful in excluding certain possible causes of chest pain, is not specific for angina pectoris since pain in the anterior chest and arm may be due.
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to cervical radiculitis or to injury or strain of the pectoralis muscle. Radiation of chest pain to jaws, teeth, or face provides some diagnostic specificity but its sensitivity is low because of the relative infrequency of such radiation.

2. Precipitation of pain or pressure etc. by walking outdoors (especially when hurrying, carrying a package, or walking in cold weather or in a cold wind). That a patient can walk or exercise indoors without pain or that usually he can walk even long distances without discomfort should not be interpreted to exclude angina pectoris. Not what the patient can usually do without pain, but the circumstances at the moment when he does have pain are diagnostically decisive. If the pain occurs only occasionally, but specifically when walking to or from the subway, bus, or car, and forces him to stop or slow down to obtain relief, then he has angina pectoris, whether or not he can walk a mile or more at other times. Often the patient walks so little or rarely that he is unaware of this relationship and he must be urged to use appropriate outdoor walking as a diagnostic test.

3. Precipitation of pain by emotion, especially when associated with acrimonious discussion or prolonged conversation on the telephone. The pain must develop in close time relationship to the precipitating emotional experience, not hours later.

The effect of sublingual nitroglycerin is often diagnostically helpful but it may be necessary to make numerous trials, with careful timing of the duration of pain, with and without the use of nitroglycerin.

Sometimes a single observation is diagnostic and may be as valuable as any information obtained from a long, detailed description of the pain. For example, during the course of a searching and presumably stressful history, the patient obviously develops distressing discomfort in the anterior chest and left arm.

Physical Diagnosis

Whereas the physical examination was formerly regarded as noncontributory to the diagnosis of uncomplicated angina pectoris, more recently increasing attention has been paid to the study of objective signs which can be elicited at the bedside in an effort to substantiate or exclude the diagnosis.24

A number of hemodynamic and functional abnormalities discovered by cardiac catheterization and left ventriculography are being used for classification of subsets of patients with angina, for determining indications for coronary bypass surgery, and for objective evaluation of this surgical intervention. Efforts have been made to obtain by noninvasive methods essentially similar information to that provided by studies requiring cardiac catheterization. Accordingly, findings obtained at the bedside have been correlated with hemodynamic and angiographic findings.

Drs. Martin, Shaver, and Leonard deal with physical signs, apexcardiography, phonocardiography, and systolic time intervals in angina pectoris not only as lists of abnormalities that may occur in this condition but also in terms of their pathophysiologic mechanism and their relation to abnormalities which can be determined only by invasive means. The most important findings in angina pectoris, or at least those found with significant frequency, are an atrial gallop sound,25 a prominent apical pulsation corresponding to the atrial gallop, a prominent A wave in the apexcardiogram,26, 27 a late systolic bulge due to abnormal contraction of the left ventricular wall, a late systolic or holosystolic apical murmur of mitral regurgitation, a prolongation of the pre ejection period (PEP), and occasionally an abbreviation of left ventricular ejection time.28-30 Many of these abnormalities are discovered only during an attack of angina pectoris or are induced only with exercise or other stress. Paradoxic splitting of the second sound has also been reported, but its occurrence in uncomplicated angina pectoris must be extremely uncommon.

Use of these findings for diagnosis in classification of patients with angina pectoris must be tempered by awareness of their lack of sensitivity and specificity as well as uncertainty as to the reliability with which these signs can be discovered by most
physicians. A large minority, if not a majority, of patients with angina pectoris do not present with most of these physical and graphic signs. On the other hand the abnormal findings listed are quite infrequent in normal persons. Various diseases, besides angina pectoris, may be associated with these abnormal signs, such as hypertension especially with heart failure, cardiomyopathy, and acute or healed myocardial infarction, but usually these present no diagnostic difficulties. A great problem at present is the lack of adequate experience and skill in consistently detecting these abnormal findings. There is need for objective assessment of the physician’s ability to interpret abnormal heart sounds correctly. There are shortcomings in the equipment employed to obtain tracings of heart sounds and pulsations, and considerable experience is required before the practicing internist or cardiologist can regularly obtain good records and interpret them correctly. Nevertheless these noninvasive methods are sufficiently promising to justify continued effort to overcome present shortcomings. From the viewpoint of the office or bedside diagnosis of angina pectoris at the present time they cannot be regarded as useful determinants. The absence of gallop sounds or murmurs, or of abnormal pulsations, and the presence of normal systolic intervals, like the presence of a normal electrocardiogram, do not exclude angina pectoris.

Exercise and other Stress Tests

After clinical symptomatology the electrocardiogram after an exercise test, usually the Master’s two-step test, has been regarded as the most useful aid in the office diagnosis of angina pectoris. Although there was a fair statistical correlation between a positive two-step test, the presence of coronary heart disease, and a higher than expected mortality in studies of large groups of persons,31 there appeared to be an unwarranted confidence in the accuracy of the test as a diagnostic criterion for angina pectoris in the individual patient.32 Recent application of coronary arteriography has provided an objective means of assessing the two-step and other exercise tests in relation to the presence or absence of severe occlusive coronary atherosclerosis. As indicated in the reviews by Redwood and Epstein and by Friesinger and Smith, these studies have revealed the diagnostic inadequacies of the two-step test, with unsatisfactory sensitivity and specificity, which varied with the degree of S-T depression required for a positive test.33,34 For 1-mm S-T depression or greater, specificity was fairly high (low incidence of false positives) but sensitivity was poor (high incidence of false negatives) and the converse was true of lesser degrees of S-T depression such as 0.5 mm. The lowest error ratio (number of incorrect diagnoses/number of correct diagnoses) in the studies listed by Friesinger and Smith was 42%.

A major difficulty with the two-step test is the failure to provide an adequate stress in many persons. Their heart rates are only slightly elevated by the exercise and blood pressure little altered, and therefore myocardial ischemia is not induced. To increase stress intensity, more vigorous graded exercise tests with bicycle ergometer or treadmill have been devised. Heart rates up to 85 or 90% of anticipated maximum have been induced, resulting in an improved correlation between positive electrocardiographic tests and severe coronary disease in the angiogram.35 These tests are too cumbersome and time consuming for office diagnosis, and the two-step test must be interpreted with caution, i.e., with awareness of its diagnostic limitations in the individual patient. The more strenuous exercise tests are being used for comparative pre- and postoperative studies to provide an objective evaluation of the effectiveness of coronary bypass procedures. Atrial pacing which induces myocardial ischemia in patients with severe coronary disease by controlled increase of heart rate possesses several advantages as a stress test and has permitted many valuable studies.36,37 Since it is an invasive procedure it is not of practical value for clinical diagnosis.
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Coronary Arteriography

More than any other development, selective cinecoronary angiography is responsible for most of the exciting advances in our knowledge and management of angina pectoris. Sones and Baltaxe and Levin review coronary arteriography, differing somewhat from each other in emphasis and viewpoints. When coronary arteriography is performed left ventriculography is also carried out to study contraction of all segments of the ventricular wall. From the clinical viewpoint there are two common problems: (1) indications for coronary arteriography and (2) coronary arteriographic findings in relation to indications for coronary bypass surgery.

That the indications for coronary arteriography are not identical for different experts is apparent by a comparison of those listed by Sones and by Baltaxe and Levin. Undoubtedly the risk of the procedure will be an important consideration in determining how readily coronary arteriography is recommended. Whereas a mortality of 1 per 1000 has been reported in a couple of clinics with a large experience, 1–2% appears to be more frequent in many other experienced centers. The possible induction of acute myocardial infarction as well as other less serious complications must also be considered.

If coronary bypass surgery is contemplated, coronary angiography is essential to confirm the diagnosis of occlusive coronary disease, to depict clearly the site and extent of obstruction, the vascular runoff, and the origin and distribution of collateral circulation. Accordingly the indication for coronary arteriography will depend in large measure on the indication for coronary bypass surgery. Repeat coronary angiography is indicated for postoperative studies and when it is necessary to determine potency of the graft. Coronary arteriography is indicated when aortic valve replacement is considered in patients with severe angina pectoris. Sometimes it is important to perform coronary arteriography to prove the absence of severe coronary atherosclerosis. Many other indications may be listed, but they are essentially for research.

The value of coronary arteriography in patients who are to undergo coronary bypass surgery because of the severity of clinical symptoms and physical disability is generally accepted. However coronary angiography is frequently performed in patients with relatively moderate stable angina. If extensive, severe occlusive disease is discovered, is this an adequate indication for coronary bypass surgery even if symptoms and dysfunction are moderate and tolerable? More needs to be known of coronary arteriographic findings as prognostic indicators of the subsequent natural history before they can be properly used as an independent indication for coronary surgery.

Treatment of Angina Pectoris

The medical treatment of angina pectoris is fully discussed in this symposium. Logue and Robinson deal essentially with the patient who has mild or moderately severe, stable angina pectoris. Perhaps the most important new development is the use of the beta-adrenergic blocking agents, notably propranolol (Inderal). Whether other beta-blocking agents such as practolol or alprenolol possess any significant clinical advantages is uncertain. There is uncertainty also as to the indication for propranolol in patients with infrequent pain, or even with pain two or three times daily, which subsides rapidly with rest or sublingual nitroglycerin. Whereas some patients may be taking propranolol without requiring it, others with more severe and more frequent pain are receiving inadequate dosage. The dose should be progressively increased as tolerated, given if possible in relation to the time of most frequent attacks, and combined often with sodium restriction, digitalization, and diuretics to avoid induction of heart failure. Whatever prolonged benefit resides in the combination of isosorbide dinitrate (Isordil) with propranolol is due essentially to the propranolol. Isordil is clinically effective only when given sublingually but does not act as rapidly as nitroglycerin.
When given sublingually Isordil is not "long acting." Sublingual nitroglycerin remains the mainstay for rapid relief of attacks of angina pectoris. It should not be discontinued for this purpose when oral "long-acting" nitrates or beta-blocking agents are prescribed.

An enormous challenge is posed to medical treatment when angina increases in severity and frequency, occurs without apparent cause, awakens the patient at night, or is only briefly relieved by sublingual nitroglycerin. Sampson and Hyatt discuss the management of the patient with severe angina pectoris. In many respects the treatment is similar to that for the patient with milder, stable angina pectoris. However it may be possible to find and control an extracoronary factor (e.g. arrhythmia, anemia) or environmental cause which was responsible for the exacerbation. A period of bed rest, preferably in a hospital, protected from environmental stresses (often from certain members of the immediate family) may serve both to alleviate symptoms and disclose whether the intensification of symptoms is due to progressive coronary disease or extracoronary factors. Except on rare occasion, and then for not more than a few days, opiates should be strenuously denied. Opiate addiction in the patient with angina pectoris may be manifested as intractable angina. Propranolol should be given in increasing doses until relief is obtained or important side effects occur. Sedatives may be necessary. Most important, however, is the physician's ability to convey his confidence to the patient that the period of increased, unstable, severe angina pectoris will eventually terminate and that the discomfort will again reach a tolerable, stable state which will permit the conduct of the patient's usual activities. The use of radioiodine has been virtually abandoned. The insertion of a carotid sinus stimulator had considerable theoretic appeal, and benefit was reported in relatively small series of cases, but the procedure has attracted no widespread application.

The heart of the problem of management of angina pectoris is now to do or not to do coronary bypass surgery. Internal mammary implantation, like a host of myocardial revascularization procedures before it, was ill advised, ineffective, and fortunately now is going the way of the others. On the other hand, coronary bypass surgery is something else again. This is theoretically rational, is supported by effectiveness of similar procedures in other vascular beds, has apparently provided dramatic relief to many patients, and must be regarded as very promising. Favaloro and Manley and Johnson present their experiences with the procedure. Unfortunately they continue to complicate the operation in some or many patients by combining internal mammary implantation. Attention should be directed also to the procedure used by Green in which bypass is accomplished, not by venous graft but by anastomosis of the internal mammary artery to the coronary artery.

If coronary bypass surgery is now the heart of the problem of management of angina pectoris, then the appropriate surgical indication is the heart of the problem of coronary surgery. Appropriate indications cannot yet be formulated because of the inadequacy of data. Decisions must still be made on an individual basis with due regard for (1) anticipated natural history of the disease in a particular patient, (2) his satisfaction with his quality of life (tolerability of symptoms, capacity for useful work, and other activity), (3) the risk of the surgical procedure (in relation to the risk without operation), and (4) his willingness to assume this risk. It cannot be assumed that the risk is the same with all experienced cardiovascular surgeons or with any given surgeon performing his first 50 coronary bypass procedures or his five hundredth. Such decisions are greatly hampered by lack of knowledge of the natural history of angina pectoris and more particularly of various subsets of angina pectoris. The prognosis for medically treated patients with angina pectoris is far better than is generally stated. There is also uncertainty as to the surgical risk, although remarkably low mortality percentages have been reported from some centers with extensive experience. Evaluation
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is difficult because of an absence of exact characterization of the patient material and other deficiencies in data. The quality of critique does not always measure up to the high level of technic. It is important to determine whether there are patients with high and low surgical risk and to be able to identify and distinguish them. Careful attention should be paid to discovering the incidence of postoperative myocardial infarction since there are some disquieting reports as to its occurrence.44, 45 The relative outlook with medical and surgical treatment should be determined by prospective studies and by randomization of control and operated patients, but this is difficult because of enthusiastic prejudgment of the value of coronary bypass surgery.

The indication for coronary surgery seems most acceptable for patients with severe disabling and intolerable pain despite an adequate period of optimal medical care. In the absence of evidence that coronary bypass definitely reduces the incidence of subsequent myocardial infarction or enhances longevity the procedure does not now seem justified on the basis of severe occlusive disease discovered on coronary angiography in a patient with tolerable, stable angina pectoris with little incapacity. Decisions as to surgical treatment should not be fixed but reconsidered at regular intervals and revised if warranted by an alteration in the patient’s clinical state or more definite evidence of the value and improved outlook with surgery.

References


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