Management of the Patient with Severe Angina Pectoris
An Internist's Point of View

By John J. Sampson, M.D., and Kenneth H. Hyatt, M.D.

SUMMARY
Severe angina pectoris may be reduced to tolerable levels by suppressing below the pain threshold those factors elevating systolic blood pressure and heart rate and prolonging systolic ejection period. Their product parallels myocardial oxygen consumption (MVO₂) as noted in several excellent reviews. The level of this product associated with angina, the "angina index," defines the individual threshold for onset of angina.

Prevention of angina-inducing physical and psychic stress is desirable, but often impractical, and the use of nitroglycerin preceding or following stress may lead to years of acceptable life. Prolonged, spontaneous, or readily induced angina, especially with a change in pattern, may be due to an acute coronary occlusive episode, whether defined as a prodromal period or an actual myocardial infarct. This form of attack requires close observation and appropriate care. The psychic stress-prone patient may repeatedly present a benign duplicate of such pain production, producing a diagnostic challenge. Routine protection of the angina patient, especially against arrhythmias, is discussed.

Different therapeutic modalities for control of intractable angina are presented, many with promise, but those currently giving the best results are: (1) elimination of precipitating conditions, i.e., (a) brady- and tachyarrhythmias, (b) hypertension, (c) hyperthyroidism, (d) mechanical obstruction of ventricular outflow, and (e) heart failure (2) beta-adrenergic blocking agents, i.e., propranolol, with or without nitrates or nitroglycerin; and (3) surgical revascularization preceded by selective coronary angiography.

The aortocoronary saphenous vein bypass has been effective in prompt relief of angina in patients with viable myocardium and potentially good flow in the distal artery, with acceptably low operative mortality from skilled teams. Mechanical efficiency and thus congestive failure may improve, and there is hope for increased longevity. Contraindications are poorly defined, especially as related to its performance in recent infarction of varying magnitude. With only 3 years' experience in determining long-term benefits, it seems rational to repeatedly redefine indications for surgery, but currently not to operate on every patient with "significant" coronary disease.

I T IS THE PURPOSE of this paper to present the management of patients with angina pectoris whose failure to respond to the routine modalities of therapy, as ably reviewed by Logue and Robinson,¹ results in an intolerable discomfort or restriction of their social and occupational life.

Patients with persistent intractable angina do not appear frequently in the population of an ambulatory clinical practice. Only seven of 150 successive patients with angina reported by one of us² could be classified as intractable. This does not imply that more sophisticated therapy than rest and nitroglycerin may not have been required for patients of marginal or irregularly severe anginal attacks. Persistent periods of severe angina lasting hours or days are often related to myocardial infarction or to acute coronary insufficiency without infarction resulting from transient imbalance.

From the Departments of Medicine, University of California, Mount Zion Hospital, and the U. S. Public Health Service Hospital, San Francisco, California.

Circulation, Volume XLVI, December 1972

1185
of myocardial oxygen supply to demand. They can be controlled by narcotics until their brief course terminates by compensatory mechanisms, e.g. expanding collateral circulation. Not always is angina related quantitatively to the degree of underlying occlusive coronary disease, as evidenced by absence of symptoms despite advanced "three-vessel" disease in an active 53-year-old woman until 2 months before her sudden death. Conversely, coronary arteriography, the best current method of quantitating the degree of occlusive coronary disease, too often fails to give the explanation for severe typical angina pectoris. Small-vessel disease, stagnant capillary flow, defective transmission of oxygen to the myocardium, or faulty cellular oxidative processes are some possible, but as yet unproven, causes for such faulty correlation.3, 4

In most instances the recommendation to expand therapy to such complex procedures as surgical revascularization is based on the subjective evidence of a patient's awareness and response to pain or its equivalent. This varies individually as illustrated by a 56-year-old man who maintained a 10-hour daily work program and normal social life for 14 years while requiring 6-12 sublingual doses of nitroglycerin daily for pain. Some angina was spontaneous and other attacks were precipitated by excitement, by walking 100 yards after a meal, or by undressing at night.

In contrast, a 74-year-old entertainment-business executive who had suffered three attacks of minor myocardial infarction in 2 years found life so unbearable because of 2-4 anginal attacks daily, generally relieved by nitroglycerin, as to demand the trial of possible relief by surgical revascularization. The psychic effect of nonspecific therapy in a condition sensitively influenced by nervous pathway impulses is illustrated by the favorable response to any of several agents in 35% of anginal patients during an initial 3-month rapport period.5

A more objective view of the influence of factors, including psychogenic ones, precipitating angina pectoris has come with the current recognition of the relatively fixed individual setting of an angina threshold at a certain level of measured determinants of myocardial oxygen consumption (MVO2).6 These determinants are clinically measurable as systolic blood pressure, heart rate, and systolic ejection time. Irrespective of the causes that alter them, the "triple product" of systolic blood pressure × heart rate × systolic ejection time at anginal induction appears reproducible and therefore constitutes an "angina index." Practical estimates of this angina index may generally be made from the systolic blood pressure and heart rate alone.7 Changes of the angina threshold seem to occur only with alteration of the myocardial integrity as generally influenced by modification of the coronary circulation and possibly by physical training.8

The action of nitroglycerin in dispelling or preventing angina is largely due to widespread vasodilation, both arterial and venous, resulting in a reduction of the systolic blood pressure and of venous inflow. The triple product and MVO2 will thus be reduced despite the increase of heart rate and rate of rise of left ventricular systolic pressure (dp/dt). Additionally, there is evidence for less myocardial anaerobiosis after stress when nitroglycerin has been used.9 Similarly beta-adrenergic blocking agents diminish the triple product by decreasing the MVO2 determinants of heart rate and systolic blood pressure.

Medical Management of Severe Angina
The occurrence of intractable angina falls largely into three categories: (1) persistent incapacitating angina, rapidly or slowly developing, without laboratory evidence of acute coronary occlusion or myocardial infarction; (2) the prodromal syndrome or impending myocardial infarction (preinfarction angina); and (3) angina of the immediate postmyocardial infarction period.

Persistent Incapacitating Angina:
Modalities of Treatment
Sublingual Nitroglycerin
In doses of 0.15, 0.30, 0.40, and 0.60 mg, sublingual nitroglycerin is considered the
standard medication. Amyl nitrite, inhaled, and oral sodium nitrite are short-acting nitrates which are rarely used now. Sustained-action nitrates include sublingual and oral erythritol tetranitrate (Cardilate), pentaerythritol tetranitrate (Peritrate), and isosorbide dinitrate (Isordil or Sorbitrate). These drugs have no striking advantage over nitroglycerin in immediate elimination of pain, but provide some prolongation of action.

It is not unusual for some patients to have severe anginal pain in irregularly occurring sustained episodes of ½-1 hour in duration, not relieved by rest or one or two doses of nitroglycerin. They may occur at intervals varying from days to weeks without apparent cause. These attacks can be termed acute coronary insufficiency without further definition or may prove to be either the prodromal syndrome or actual myocardial infarction on continued observation. Relief without sequelae by a second or third dose of nitroglycerin at 10-30-min intervals, or by increase of dosage from 0.15 to 0.6 mg, decreases the likelihood of a prodromal attack or infarction. Some of these patients who generally respond to nitroglycerin may be completely resistant at times, and only gain prompt and sustained relief from an opiate taken orally, e.g. meperidine hydrochloride (Demerol) 50 mg, morphine sulfate 10-15 mg, codeine sulfate 30 mg or oxycodone hydrochloride with homatropine terephthalate (Percodan).

Inconsistent nitroglycerin responses were reported in 22% of a series of 100 successive patients with angina,2 and 10% were uniformly nonresponsive to nitroglycerin. Development of tolerance to nitrates has not been a problem in our experience. Apparent nitroglycerin resistance may be due to deterioration of drug potency after 1-6 months in opened or plastic containers.10

Increase in the size or frequency of the nitroglycerin dosage in effect may be demonstrable for 1 hour after a single dose, and presents the hazard of a serious hypotensive reaction. It should be recognized that hypotensive reactions have been encountered in the presence of unrecognized blood volume decre-
occasional ability to accomplish physical feats, i.e. running up 25 stairs without pain, is belied by his consumption of 10–20 nitroglycerin tablets daily for both spontaneous and exertional angina. Many episodes last 15–30 min and often are undiminished by nitroglycerin or rest.

The physician is challenged repeatedly to decide whether or not the attack may represent prodromal pain of myocardial infarction (preinfarction angina), actual myocardial infarction, or prolonged benign pain. The more protective, fearful, and concerned attitude the attending physician displays, the more likely that the symptoms will be intensified. The personality of the pain-susceptible patient can be illustrated by the description of a group tending to have pain after myocardial infarction. A study of this group of patients revealed: emotional instability, depression, timidity, apprehension, conformity, conscientiousness with a sense of urgency, imaginativeness, and a high level of anxiety and neuroticism with a tendency toward hypochondriasis and hysteria. These qualities are similar to those described by Friedman and Rosenman in people defined as “personality A” who are prone to coronary disease. Psychotherapy by trained psychiatrists is sought but rarely alters the complex or its sequel.

The prognosis of the group of postinfarction angina-prone patients was found to be no different from that of postinfarction patients without angina. Thus, there may be little benefit from overrestriction of activity in the recovery period of myocardial infarction. The physician may permit the same light activity, i.e. walking, after 1–2 weeks of bed rest, as recommended to those without angina, but with due awareness of the possibility of extended or secondary attacks. Similar activity has seemed harmless for patients with prodromal syndromes dependent on their individual tolerances of physical effort.

**Alcohol**

Ingestion of alcohol does not produce coronary vasodilatation. Moderate-to-heavy ingestion may increase $\text{MVO}_2$ and some patients have shown ischemic electrocardiographic changes following moderate consumption. However, White and others, including the authors, have noted rapid relief of angina by 30–40 ml of spirituous liquor in some patients resistant to nitrites, possibly due to a tranquillizing effect.

**Beta-Adrenergic Blocking Agents**

Lowering both heart rate and blood pressure, beta-adrenergic-blocking agents are not often required in the treatment of patients with infrequent or readily controlled angina, but have proven to be prompt and highly effective in prevention of either fixed or recurrent cycles of severe incapacitating angina. Despite evidence that some of these agents (practolol, sotatol, oxyprenolol, and alprenolol) have less bronchospastic and negative inotropic effects, until broader clinical experience is acquired it seems prudent to avoid their use in patients with obstructive lung disease, overt congestive heart failure, and valvular insufficiencies with markedly dilated ventricular chambers.

It would be illogical to use these drugs in bradyarrhythmias, i.e. sinus bradycardia or A-V block. However, minor congestive failure has not been aggravated by cautious use, and the overt failure induced by tachyarrhythmias diminishes when the rate is slowed by a beta-adrenergic blocking agent as used when other measures are ineffective.

The daily dosage necessary to suppress angina has varied widely from patient to patient in our experience. Propranolol generally lowers resting heart rate to a range of 50–60 beats/min when angina has been decreased with a divided daily oral dose from 80 to 160 mg. In some cases as little as 60 mg daily in three divided doses has given relief with heart rates no less than 65–80 beats/min. In contrast, one patient required 480 mg daily to obtain a reduction in angina of approximately 50%. At this dose the heart rate at rest was not reduced below 76–90 beats/min. Dosage must be individualized starting at a level of 10 mg four
times daily to determine sensitivity or idiosyncrasy. In many cases doses of 40–60 mg four times daily are required for optimal improvement. In some, maximum benefit is not obtained below a dosage of 400-480 mg daily. In the authors’ experience, arrival at this dosage is frequently precluded by gastrointestinal symptoms.

Sowton and associates concluded that the effect of all these beta-blocking agents was related to dose and magnitude of effort within certain ranges. Alpenolol gave better results at 400 mg/day than 200 mg/day, but 800 mg/day had little, if any, additional benefit. As previously mentioned, the long-acting nitrates seemed synergistic in some cases and in others gave no demonstrable clinical improvement over the use of beta-blocking agents alone.

In patients with intractable angina, when the use of beta-blocking agents is contraindicated by conditions previously mentioned, the following measures are successively or jointly applied often as “holding” regimens while awaiting angiography preparatory to cardiac revascularization.

**Diuretics**

Personal experience supports that of others in the prevention of angina by use of diuretics in some patients with or without signs of overt heart failure. Reduction of blood volume or systemic vasodilation, accompanied by a decrease of MVO₂ could account for the effect. Oral furosemide, 80–160 mg, ethacrynic acid 50–150 mg, or large doses of thiazide diuretics, i.e. 1.0 g chlorothiazide, have terminated periods of spontaneous severe angina attacks. Aminophylline, 0.25 g given slowly by intravenous route in 10% aqueous solution to avoid hypotension, has relieved the immediate recurrent postinfarction pains.

**Digitals**

In adequate dosage digitalis has seemed to give relief from persistent pain attacks in patients with or without apparent heart failure, although its effect is not as sharply apparent as that of diuretics.

**Thyroid-Activity Suppression**

Coexistent hyperthyroidism routinely aggravates angina and requires correction. In euthyroid patients total thyroidectomy was employed for a short period for intractable angina but was abandoned because of intolerable and ill-controlled myxedema. Radioactive iodine gave the same benefits, but late myxedema was still troublesome, and the hazard of a hyperthyroid reaction required low individual doses (under 10 mCi) and a long delayed course of treatment. Antithyroid drugs whose effect may be terminated promptly by discontinuance can be a more practical method of suppressing troublesome angina. Moderately large doses, i.e. 300–800 mg propylthiouracil daily, are required. Side effects warranting withdrawal are late colloid goiter and more rarely agranulocytosis.

**Prodromal Syndrome of Impending Myocardial Infarction**

This entity represents an acute ischemic episode characterized by prolonged (over ½ hour) or frequently recurring shorter spontaneous anginal attacks and/or increased severity and altered distribution patterns of anginal pain. The episodes usually occur abruptly. In many cases they probably indicate an actual minor infarct as evidenced by small elevations of serum enzyme activity, e.g. a rise in CPK to 25 units in a patient with a 15-unit baseline, or casual level of 50–60 units, and electrocardiographic T-wave inversions. The quantitative nature of this syndrome is still not formally recognized. Widely different estimates of the risks and successes of specific surgical revascularization procedures are reported in patients diagnosed as prodromal by various workers, because the criteria for classifying patients in the broad spectrum of this syndrome are ill defined. The length of this unstable acute myocardial ischemic period varies with the opinions of the different clinicians, but would seem rationally to range from 1 to 21 days. In approximately half of the patients major infarcts terminate the syndrome, especially if there was a preceding history of signs and symptoms of severe coronary disease. In those
patients, especially female, who have exhibited little or no evidence of severe occlusive coronary disease previously, the prognosis is excellent for complete clinical and electrocardiographic recovery.\textsuperscript{31}

Nitroglycerin often fails and relief of severe or persistent pain is obtained only by narcotics, and thus addiction is a threat. Beta-adrenergic blockade may be initiated if no significant heart failure exists.\textsuperscript{32, 33} Anticoagulation therapy seemed to have decreased the development of a succeeding infarction and/or death according to some reports,\textsuperscript{34} but not in others, nor with certainty in our personal experience.

Hospitalization in a monitored unit may be the safest way to minimize the hazard of the prodrome progressing to a large infarct, but in mild and prolonged cases supervised home care may be reasonably safe and less extravagant. When the spontaneous pains of the syndrome fail to recede and seem to become a fixed pattern, a trial of a new modality of treatment or a more normal program of activity with appropriate supervision, including return to semisedentary work can be planned. Coronary arteriography and revascularization surgery are increasingly used in this prodromal state with much success.

**Angina during the Immediate Postinfarction Period**

Severe and frequent spontaneous anginal pain may follow within a day of the relief from the prolonged pain of the onset of myocardial infarction and may continue for days or weeks, generally in diminishing degree and frequency. The patient should be reassured that such pains are not an uncommon sequel to a heart attack and are generally benign. However, they may represent an extension of the infarct or a secondary attack as revealed by serial electrocardiograms and serum enzyme activity.

During the first 3 days after infarction when hypotension is frequent and may become severe, withholding nitrites seems advisable. Narcotics should be used for the initial 3 days or until the blood pressure is safely stabilized. (Long continued use presents the hazard of addiction.) After stability seems assured, nitroglycerin may be used, as reported by Harrison and others,\textsuperscript{35, 36} without fear of a serious shocklike reaction.

Papaverine hydrochloride im three to four times daily in doses of 30 mg at 4-hour intervals has seemed to break the pattern in some cases. It is somewhat painful. Propranolol should be used only if no heart failure is evident. However, heart failure is likely when persistent pains accompany these somewhat large infarcts.

**Surgical Procedures in the Management of Severe Angina**

Failure of medical therapy to produce consistent relief of angina or improvement of myocardial function has encouraged the development of many surgical methods of treatment. Most surgical procedures were ineffectual and did not justify the risk. Discarded procedures include surgical interruption of afferent nerve pathways, resection of the cardiac and thoracic sympathetic ganglia, dorsal rhizotomy, total thyroidec- tomy, attempted revascularization through pericardial adhesions or extracardiac grafts, and ligation of the internal mammary artery. Surviving procedures are aimed either at reducing MVO\textsubscript{2} or, more importantly, at increasing oxygen delivery.

**Carotid Sinus Stimulation**

This technic can relieve angina pectoris,\textsuperscript{37, 38} and recent reports have described the use of an implantable radiofrequency carotid sinus stimulator in the treatment of incapacitating angina pectoris.\textsuperscript{39, 40} Effective pain relief is provided, primarily by reduction of arterial systolic pressure and MVO\textsubscript{2}. Initial reports indicated somewhat high operative mortality with a number of undesirable complications, including myocardial infarction and temporary bilateral hypoglossal or cervical nerve palsy. At present, use of this technic should be highly selective. Future reports may be more favorable.

**Coronary Arteriography**

This technic is currently an essential preliminary to revascularization surgery and is not
an innocuous procedure. Ventricular fibrillation, myocardial infarction, and death may occur. Risk from coronary arteriography is variable and is largely dependent on the capability and breadth of experience of the arteriographer. The mortality rate varies from <0.5% in hospitals with reasonably large patient loads to 8% in small hospital services. Less critical complications include occlusion of the artery at catheter entry, periarterial hemorrhage, and systemic embolism, notably to brain or eye. Temporary derangement of myocardial function often follows the injection of the iodine-containing radiopaque media; e.g., LVEDP rose in one patient from 6 to 28 mm Hg after 76% diatrizoate meglumine was injected into the coronary arteries. Severe angina ensued. In others we have observed pulmonary edema. Thus, the indications for coronary arteriography and surgery should be well delineated, particularly in hospitals which have not reached peak technical accomplishment.

The major indication for undertaking coronary arteriography is the consideration for myocardial revascularization in the following situations: (1) progressive angina pectoris, persistent and unrelieved by medical management; (2) as a preventive measure in patients with extensive or repeated myocardial infarctions and presently favored in the patient under 60 years of age; (3) progressively disabling heart failure presumably due to coronary artery disease; (4) determining the degree of occlusive coronary disease in a young patient suspected of angina, particularly in the presence of a suggestive electrocardiographic abnormality, e.g., broad Q waves (The fallacy of such studies to establish a diagnosis is that coronary obstructive disease is often found in asymptomatic subjects and thus may be unrelated to the ill-defined pain.); and (5) in aortic stenosis, on the chance that coronary disease, in part or wholly, may cause the associated anginal pain rather than it being solely related to the left ventricular work load imposed by the restricted outflow tract. An additional indication is the determination of postoperative status of aortocoronary bypass grafts. The risk of coronary arteriography in most medical centers presently does not seem to justify surveys of the incidence of various grades of coronary disease when the subjects, not in the above categories, have little or no suggestive findings of coronary disease.42

Features revealed by coronary arteriography that currently are considered to require revascularization surgery include: 75% or greater obstruction of one main coronary artery and/or 50% or more in other branches; elements favoring adequacy of the introduced auxiliary blood supply include collateral arterial supply preferably visible distal to the obstruction; a runoff of distal blood; and functioning myocardium in the area of prospective revascularization.43 Demonstrable patent distal segments, although strongly desired, are no longer required in most centers as surgical exploration may reveal an angiographically unrevealed graftable segment44 or core, or gas endarterectomy may establish a “runoff” channel.45 High-grade stenosis of the main left coronary trunk has a very poor prognosis requiring urgent surgery.46 Operative mortality is high.

Myocardial Revascularization

The currently used revascularization procedures appear to offer great promise of success. These procedures include endarterectomy with or without venous patch graft,47, 48 implantation of one or more internal mammary or other arteries into viable but partially blood-deprived areas of myocardium49, 50 internal mammary artery-to-coronary artery bypass by direct anastomosis,51, 52 venous graft bypassing occluded segments, and aortocoronary bypass using saphenous vein autografts.53-56

Internal Mammary Artery Implant into the Myocardium

Until introduction of aortocoronary bypass, the implantation of one or more open internal mammary or other arteries into viable but partially ischemic areas of myocardium was popular.57 It was favored because of the relatively low operative mortality (5.4% in
single and 8.1% in double implants) and the fact that implanted arteries appeared to have frequently enhanced collateral communication and flow.\textsuperscript{57-59} The improved irrigation of the affected myocardium has not been measured. There was a marked postoperative decrease of angina reported in 62\% of cases although often delayed for 3–9 months.\textsuperscript{60} Angina threshold, as indicated by time-tension index, was shown to be increased after surgery.\textsuperscript{61} 

Despite favorable reports, our experience and that of personal contacts was one of many months of delays and inconsistency of angina prevention, irrespective of some favorable angiographic results. In one study, 60\% of surviving patients showed postoperative improvement despite a demonstrated implant patency of only 33\%.\textsuperscript{62} Postoperative physical and psychic rest as well as placebo effect may have decreased postoperative angina. Currently, internal mammary artery implantation, endarterectomy, or arteriotomy and patch graft are used chiefly as auxiliaries to aorto-coronary bypass.

\textbf{Aortocoronary Artery Venous Bypass} 

Bypass of an occluded coronary artery was accomplished in 1967 by Favaloro and early experience with the procedure was reported by Kerth, by Favaloro, and by Johnson and their associates.\textsuperscript{53-56} The apparent prompt high degree of relief from angina and the low operative and hospital mortality associated with this procedure has encouraged its wide adoption. Reported mortality has ranged from 2.3\% in low-risk patients to at least 20\% in patients with severe myocardial damage.\textsuperscript{63} Relief of angina in patients with patent grafts is usually evident within days or weeks and is reported to be as high as 95\%.\textsuperscript{64} As may be expected, improved ventricular function has been demonstrated as a sequel to the enhanced coronary circulation.\textsuperscript{46, 64} 

Loss of graft patency by thrombotic occlusion is the major cause of poor outcome, but it is unlikely with flow rates of 50 ml/min or more, measured at surgery. Flow rates as small as 20 ml/min may be effective. An early patency rate of 89\% was reported in one series,\textsuperscript{63} and in another study 89\% on the mean of the 14th postoperative day and 79\% on the mean of the 12th month, with 9\% local vein graft stenosis.\textsuperscript{65} 

The glowing reports from only 3 years of experience with aortocoronary bypass have resulted in a marked expansion of criteria for surgical intervention. Contraindications, such as age over 65 years and advanced degrees of myocardial disease, are no longer considered valid in certain centers where “no patient is denied surgical treatment because of the extent of his coronary disease.”\textsuperscript{64} Single or recurrent myocardial infarction, or evidence of diffuse myocardial disease, or localized dysfunction do not consistently exclude revascularization by some surgeons.\textsuperscript{56, 66} However, patients with extensive poor myocardial contractility are infrequently accepted for surgical treatment. The general enthusiasm has diminished the contraindications in some centers to that of a major myocardial infarction within 6 weeks of the planned date of surgery. Operations for revascularization have been performed during the acute phase of recognized myocardial infarction, even with shock.\textsuperscript{67} These procedures have been undertaken generally in desperate cases with the patient in heart failure, requiring assisted circulation. The risks are very great, but the small salvage compared to the near certain fatality when “medically” treated, seems worth the gamble.

Recent reports indicate the successful use of aortocoronary bypass during the period of the impending myocardial infarction syndrome. However, the prognosis in many patients with this syndrome is so good, when it is not preceded by a history or signs of severe coronary disease, i.e. major myocardial infarction, that there should be no urgency for surgical intervention.\textsuperscript{68} The main factor limiting aortocoronary bypass surgery in certain areas is the fact that the logistic demand inherent in undertaking surgery exceeds the capacity of cardiac catheterization laboratories and hospital bed and surgical schedule space.\textsuperscript{63} However, during recent discussions and presentations at the 1971 American Heart
Association Scientific Sessions, a trend could be sensed that a more controlled set of surgical indications was being developed on balance of risks and benefits with more precise knowledge and experience. However, the consensus is still not conservative.

The enthusiastic acceptance of aortocoronary bypass may prove to be well justified. However, the "natural" history of severe angina pectoris is unknown, and is unlikely to be forthcoming shortly as a baseline for evaluation of the procedure. Efficacy of new surgical procedures has rarely been evaluated by a parallel series of unoperated cases, nor does it seem likely to be. Patients are often unwilling to give informed consent for participation in a random allocation study involving the withdrawal of a possibly beneficial treatment. Nor will most physicians be likely to consent to change and cooperate with centers attempting to conduct such studies. W. W. L. Glenn reported in a presidential address at the American Heart Association 1971 Scientific Session that a prospective study of the effects of revascularization surgery is planned by the National Heart and Lung Institute of the U. S. Public Health Service. Preoperative and postoperative courses will be compared to provide a clue to the natural history of severe coronary disease and how it is altered. At present it seems futile to resist liberal indications for a procedure while awaiting acquisition of comprehensive statistical evidence of its values and risks. All new procedures must go through a period of development of technical skill and diagnostic methodology with variable hazards to the patient. With competent teams current evidence indicates improvement of these guidelines and concomitant lowering of risks.

Elements entering into the changing picture of risks and benefits are: (1) Postoperative reports of decreased angina seem valid but are difficult to evaluate because of bias by either the physician or patient and possibly by the favorable effect of enforced rest. (2) Functional improvement of the myocardium has been clearly demonstrated after successful venous bypass, especially if removal of ineffective scarred ventricular wall has been feasible. (3) Prolongation of life, which requires slowing or reversal of the occlusive process, has not been demonstrated by this, or any revascularization procedure to date. (4) Venous grafts rarely may be subject to aneurysmal dilatation and rupture. The augmented arterial flow, artificially created, may result in fibrosis or rarely, atherosclerosis of the venous graft. Experience with aortocoronary bypass is too new for accumulation of much meaningful data on subsequent venous disease. (5) Competition for flow by the high pressured graft channel with a partially occluded collateral artery may cause the latter to shut down with a resultant unfavorable total flow to the affected area. Only time will reveal the duration of consistent relief of angina, prevention of recurrent infarction, and possibly the effect on life-span.

The internist's decision for surgery must be based on unemotional and carefully weighed evidence which is continually changing. Premature or overly optimistic press releases may be responsible for patients' enthusiasm and desire to have the surgical procedure performed, which may sway the opinion of the internist toward recommending surgery. Conflicting with the broadest indication for surgery is the known prognosis of the many medically treated patients who survive with useful lives for over 20 years following myocardial infarction. If this type of patient can be identified, subjecting him to surgery with any attendant risk would be poor judgment.

Indications for surgery at present may be outlined, but it should be recognized that criteria may be altered by improvements of surgical technics, definition of surgical risk factors and factors related to predictable improvement, and delineation of long-term survival statistics. Surgery may be considered in patients preferably whose coronary arteriograms show suitable vessels for venous graft (vide supra) for the following reasons: (1) incapacitating angina in spite of adequate medical therapy; (2) evidence of progressive
worsening of the disease process; (3) prevention of recurrent myocardial infarction, especially in younger patients (under age 60 years); (4) prevention of infarction in the prodromal period (if it can be identified) in those patients with a dangerously high risk of developing a large infarction; (5) myocardial salvage in patients who develop infarction while awaiting aortocoronary bypass; and (6) life salvage in patients with uncontrollable ventricular arrhythmias, heart failure, or shock, since the mortality with or without surgery is extremely high in these cases, but surgery may be worth the risk.

A prophylactic bypass because of the statistical chance of a minor coronary occlusive lesion becoming life threatening would seem hardly justifiable at the present time.

Acknowledgment

The authors wish to acknowledge the faithful and capable secretarial services of Linda Jaillite.

References

1. LOGUE RB, ROBINSON PH: Medical management of angina pectoris. Circulation, 1972
5. BEECHER HK: Appraisal of drugs intended to alter subjective responses, symptoms. JAMA 158: 399, 1955
9. CHONG MA, WEST RO, PARKER JO: Influence of nitroglycerin on myocardial metabolism and hemodynamics during angina induced by atrial pacing. Circulation 45: 1044, 1972
14. FRIEDMAN M, ROSENMAN RH: Association of specific overt behavior patterns with blood and cardiovascular findings. JAMA 169: 1286, 1959
15. WEBB WR, DECERLI IU: Ethyl alcohol and the cardiovascular system. JAMA 191: 77, 1965
22. MARSHALL FA: Angina pectoris, its alleviation with chlorothiazide. Amer J Cardiol 3: 180, 1959
MANAGEMENT OF ACUTE MYOCARDIAL INFARCTION

37. Levine SA, Harvey WB: Temporary improvement of anginal pain by carotid sinus stimulation. Trans Ass Amer Physicians 60: 255, 1947
46. Cohen MV, Cohn PF, Herman MV, Gorlin R: Diagnosis and prognosis of main left coronary artery obstruction. (Abstr) Circulation 44: 102, 1971

Circulation, Volume XLVI, December 1972


64. Danielson GK, GaU GT, Davis CD: Early results of vein bypass grafts for coronary artery disease. Circulation 43 (suppl II): II 101, 1971


Management of the Patient with Severe Angina Pectoris: An Internist's Point of View

JOHN J. SAMPSON and KENNETH H. HYATT

Circulation. 1972;46:1185-1196
doi: 10.1161/01.CIR.46.6.1185
Circulation is published by the American Heart Association, 7272 Greenville Avenue, Dallas, TX 75231
Copyright © 1972 American Heart Association, Inc. All rights reserved.
Print ISSN: 0009-7322. Online ISSN: 1524-4539

The online version of this article, along with updated information and services, is located on the World Wide Web at:
http://circ.ahajournals.org/content/46/6/1185

Permissions: Requests for permissions to reproduce figures, tables, or portions of articles originally published in Circulation can be obtained via RightsLink, a service of the Copyright Clearance Center, not the Editorial Office. Once the online version of the published article for which permission is being requested is located, click Request Permissions in the middle column of the Web page under Services. Further information about this process is available in the Permissions and Rights Question and Answer document.

Reprints: Information about reprints can be found online at:
http://www.lww.com/reprints

Subscriptions: Information about subscribing to Circulation is online at:
http://circ.ahajournals.org//subscriptions/