Coronary Artery Disease

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Coronary artery disease (CAD), a leading cause of mortality, is unpredictable in its course. Morbidity and mortality are significantly increased when patients with this disease are exposed to the stresses of operation.[4, 93, 183] The purpose of this article is to discuss the measures which alter operative morbidity and mortality in patients with CAD. The discussion is based on a review of current literature and extensive experience with such patients. Emphasis is placed on information gained from patients undergoing anesthesia for revascularization procedures of the heart because these patients have extensive coronary artery disease and are subjected to lengthy major operations.

History

In the early forties, Vineberg implanted the left internal mammary artery into a tunnel in the wall of the left ventricle of the dog and thereby increased the blood supply to the myocardium.[205] In 1950, he implanted the left internal mammary artery into the wall of the left ventricle of a human heart.[210] In 1961, Sones [188] demonstrated by coronary arteriography that an implant done by Vineberg in 1952 was patent, filling the anterior descending artery in a retrograde manner and revascularizing the surrounding myocardium.

The impetus given by Sones' demonstration and the need for effective therapy for CAD motivated many centers to use various techniques to bring extracardiac blood to the heart and to repair the diseased artery itself. Table 1 lists the revascularization procedures currently being done, either separately or in combination. There is considerable controversy regarding the effectiveness of the various operations, because of the uncertainty in establishing the site and extent of CAD and determining the most suitable surgical procedure.[46, 66, 75, 159, 207, 208, 211, 212] There is little doubt that the myocardium can be "revascularized," resulting in significant reduction of disability owing to coronary artery insufficiency.[75, 188, 207, 211, 212]

Anesthesia in patients with coronary artery insufficiency must interfere as little as possible with metabolism in myocardial cells, regardless of the operation performed. Reductions of morbidity and mortality can be achieved only by avoiding myocardial hypoxia, which occurs when the oxygen requirements of the myocardium exceed the amount of oxygen available.

Factors Determining the Availability of Oxygen

Oxygen Content of Arterial Blood

The oxygen content of arterial blood depends on hemoglobin content and oxygen saturation. Saturation of hemoglobin is determined by Pa_{O_2} and the configuration of the hemoglobin dissociation curve, which is influenced by *pH, temperature* and Pa_{CO_2}.[176] Pa_{O_2} depends on the tension of oxygen in the inspired gases, adequacy of ventilation, and a normal capillary interface; it is reduced by the amount of blood which is shunted without coming into contact with adequately ventilated alveoli.

Coronary Blood Flow

The blood flow to the region of the heart supplied by a coronary artery which is partially obstructed differs from the normal flow. Three factors, perfusion pressure, autoregulation, and effective viscosity, are discussed separately, although they are closely interrelated.

Perfusion Pressure. The coronary circulation is unique in that the heart must generate its own perfusion pressure. When systolic pressure is increased the work and oxygen re-

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quirements of the myocardium will increase for any given cardiac output; the intramyocardial pressure will also increase during systole, thus impeding coronary flow.[21] In the healthy heart this is not very important: coronary flow is adjusted by autoregulation.[59] In the ischemic heart, if systemic pressure is elevated with vasoconstrictor drugs, it is possible to increase the myocardial work and thereby increase the discrepancy between oxygen supply and oxygen demand. In considering the use of vasoconstrictor drugs it should be remembered that stroke volume is mainly determined by the filling pressure of the heart in diastole and cardiac output usually can be maintained by providing adequate circulating blood volume.

At rest, 70 per cent of the coronary flow occurs during diastole. Both aortic diastolic pressure and diastolic time become important in ischemic hearts. As heart rate increases, the duration of diastole is reduced and coronary flow decreases.[132] Pacemaker-induced tachycardia is one method of testing patients for angina pectoris.[249] All the changes of angina pectoris can be induced at rest when the atria are stimulated at rates of 120 to 140/min during catheterization. Hypovolemia is a frequent cause of tachycardia postoperatively, but ischemic hearts are also prone to a variety of tachycardias from other causes which may require treatment. Marked bradycardia also has a deleterious effect, because a larger stroke volume must be accommodated to maintain a given cardiac output. Large stroke volumes are associated with cardiac dilatation and increased tension in the myocardial wall. The maintenance of this increased tension also increases the oxygen requirement.[269] Therefore, it is important to maintain adequate, but not excessive, systolic and diastolic pressures, and to keep the heart rate in the normal range in a patient with minimal coronary reserve.

Autoregulation. In the normal heart the major site of the resistance of coronary flow occurs at the small arterioles, not in the larger superficial arteries.[121] These arterioles respond to the metabolic state of the myocardium. When they are fully dilated in the normal heart, an increase in coronary flow to as much as 400 per cent of the resting value may be seen.[50] In patients with coronary atherosclerosis the superficial arteries usually are the site of significant resistance. When the oxygen tension is reduced in part of the myocardium, the contraction of smooth muscle of the arterioles is released and the coronary flow is preferentially directed to this area, thus compensating for the ischemia.[21] In patients with angina at rest, this mechanism may be fully utilized and there will be no "coronary reserve." It is important to realize that if the arterial blood should become hypoxic, vasodilatation will occur in only the relatively healthy parts of the heart, tending to compromise further the blood flow to the ischemic areas. In the same way, potent coronary vasodilator drugs such as dipyridamole (Persantine), which increases overall coronary blood flow, produce overperfusion of the normal areas; this can be detrimental to the relatively ischemic areas.[191] Catecholamines such as epinephrine and isoproterenol are often considered coronary vasodilator drugs; however, their predominant effect is to increase oxidative metabolism; hence, the resultant increase in flow is mediated by the autoregulatory mechanism.

Where muscle mass is increased due to myocardial hypertrophy secondary to systemic or pulmonary hypertension, there is no appropriate increase in vascularity. This vascular insufficiency is aggravated by an increase in distance from the capillary to the center of the myofibril.[26, 214]

Blood Viscosity and Myocardial Perfusion. The concentration of erythrocytes has a marked effect on the viscosity of blood.[215] In dogs, if the blood volume is carefully controlled, there is an inverse relationship between cardiac output and hematocrit.[164] Several in-

TABLE 1. Revascularization Procedures of the Heart

1. Left internal mammary artery implant into left ventricular myocardium
2. Right internal mammary artery implant into right ventricular myocardium
3. Right gastroepiploic artery implant
4. Epicardiectomy
5. Free omental graft
6. Coronary artery graft operations—patch and bypass