Management of anesthesia for coronary artery surgery

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Ten years ago anesthesiologists managed patients with coronary artery disease with a great deal of apprehension because of the high incidence of repeated myocardial infarction.[1] However, with the increased number of operations for myocardial revascularization, cardiac anesthesiologists contributed much to our knowledge of the physiology of circulation, hemodynamic changes during myocardial ischemia, the use of inotropics, vasodilator drugs, and mechanical support of the circulation.

The appropriate management of patients receiving anesthesia for coronary artery surgery depends upon (1) the variable clinical picture and the unpredictable hemodynamic status of the patient; (2) the effect of anesthetic agents and techniques on myocardial oxygen consumption and availability; (3) the individualized choice of anesthetic agents and techniques; and (4) prevention, early detection, and treatment of myocardial ischemia and dysfunction during surgery.

Clinical picture

Coronary artery disease may affect young, aggressive adults in their 30s, or its symptoms may not be apparent until age 60. It is more

common in smokers, diabetics, the obese, and hypertensives.[2] Frequently the patient has chronic obstructive airway disease, or may even have chronic renal failure. Because of the pathologic changes, the carotid arteries are frequently atherosclerotic and this adds to the risk. Usually the patient is receiving drugs such as digitalis, beta blockers, antihypertensives, antiarrhythmics, and tranquilizers. More important is the degree of coronary artery disease and how it affects the hemodynamic status. Bradycardia and conduction defects are common in patients with right coronary artery disease; in patients with left coronary artery disease, varying degrees of myocardial impairment and areas of dyskinesia or aneurysm are expected.[3] Inferior myocardium occasionally involves the papillary muscles and causes mitral valve insufficiency; the patient may be in congestive heart failure. At surgery, patients with main trunk lesions often have severe anginal pain, hypertension and hypotension, and may be in cardiogenic shock.

Oxygen consumption

Myocardial oxygen consumption is determined by myocardial wall tension, contractility, and heart rate,[4] and to a lesser degree by myocardial basal metabolism and activation energy. Myocardial oxygen consumption can be quantitated as the product of the heart rate and afterload. However, a simple clinical guide to myocardial oxygen consumption is the heart rate and systolic blood pressure product. This parameter is shown to correlate the onset of anginal symptoms in patients who are awake[5] and have S-T segment changes due to intraoperative myocardial ischemia.[6] Therefore, it is apparent that myocardial oxygen consumption in a hypertensive, tachycardiac patient with dilated left ventricle can far exceed the oxygen supply.

Myocardial O_2 supply

This is mainly determined by the O_2 carrying capacity of the blood and coronary blood flow. O_2 carrying capacity is seldom a problem in patients with adequate hemoglobin levels. However, we observed that in a large percentage of patients with coronary artery disease, preoperative PaO_2 levels are low, and any episode of mild hypoxia may be deleterious to the patient.

Coronary blood flow is controlled by its own autoregulatory mechanisms, blood pressure, diastolic interval and left ventricular end-diastolic pressure, sympathetic and parasympathetic nervous activity.[7] Changes in heart rate affect coronary blood flow as it changes diastolic time and myocardial oxygen consumption. Coronary blood flow is decreased when the left ventricular end-diastolic pressure is elevated, due to hypertension during endotracheal intubation, myocardial dysfunction, and injudicious use of vasopressors.

Because of the variability of the clinical findings and the unpredictability of coronary circulation, techniques and anesthetic agents should be individualized according to the hemodynamic status at the time of surgery. Also, they should be directed to decrease myocardial oxygen consumption to maintain coronary blood flow and the availability of O_2 to the heart.[8]

In general, adequate anesthesia decreases oxygen demand. Anesthe-

siologists can raise PaO_2 levels by increasing the percentage of oxygen administered and controlling ventilation. However, the blood flow in a diseased coronary artery is limited by the degree of narrowing. Consequently, high PaO_2 levels are beneficial only within limits.

Deep anesthesia should be avoided because it causes myocardial depression, peripheral vasodilation, decreased cardiac output, and perfusion pressure, all of which are deleterious to any patient with coronary artery disease.

Under light planes of anesthesia, surgical stimulation will increase sympathetic activity and catecholamine release. Tachycardia, increased vascular tone, and the rise in blood pressure will increase myocardial oxygen consumption, which may not be met in a patient with coronary artery disease. Nevertheless, amnesia and analgesia may be all that can be tolerated in a severely ill patient with congestive heart failure who is already living on compensatory increased sympathetic activity. We prefer to anesthetize the patient to the level that diminishes sympathetic reflexes during surgery rather than use adjuvant drugs to control tachycardia or hypertension. In other words, the preferred technique is that which minimizes the hemodynamic changes during induction and surgical stimulation, and more preferably it can add to the stability of the circulation and decrease the workload of the heart.

Management of anesthesia

Our management of anesthesia consists of strong premedication with narcotics such as morphine or Pantopon and strong sedatives, promethazine hydrochloride (Phenergan). We have started to use nitroglycerin paste 1 to 2 hours preoperatively if the circulation is stable. This premedication helps prevent anginal pain and decreases the afterload during induction. If anginal pain still occurs following premedication an acute episode of ischemia should be suspected.

Induction of anesthesia

In the patient with stable circulation, anesthesia is induced with a sleeping dose of Pentothal sodium followed by curare to facilitate intubation. Curare has both inotropic and antiarrhythmic effects. In clinical doses it causes a mild drop in blood pressure due to mild histamine release and sympathetic ganglion blockage, but also decreases myocardial oxygen consumption.[9] In the presence of bradycardia or tendency to hypotension, pancuronium bromide can be substituted for curare.[10] Pancuronium bromide has both inotropic and chronotropic effects; it increases the blood pressure and pulse rate without increasing the systemic vascular resistance. It is preferable to administer either relaxant in divided doses while monitoring the effect on blood pressure and heart rate. If necessary, half the relaxing dose of each drug may be used. In hypotensive and critically ill patients, a strong analgesic (morphine or Pantopon) is administered to minimize or substitute the dose of Pentothal sodium. For these patients, pancuronium bromide is the muscle relaxant of choice.

Maintenance of anesthesia

In patients with stable circulation, anesthesia is maintained with 50% O_2, plus 50% N_2O, together with a

choice of inhalation anesthetic agent (halothane, ethane, or methoxyflurane). We have used methoxyflurane since 1970. It has a minimal effect on heart rate and rhythm. It causes a mild decrease of blood pressure, cardiac output, cardiac index, and systemic vascular resistance, and its effect on both myocardial oxygen consumption and myocardial O_2 supply is well balanced. When it was used at analgesic concentrations there were no incidences of high output renal failure.

Halothane decreases myocardial contractility and, to a lesser extent, heart rate and systemic vascular resistance. However, recent studies indicate that it has protective effects on the myocardium in animals with coronary artery occlusion.

In general, the concentration of inhalational agents is easily adjusted to provide stable and adequate levels of anesthesia. This will minimize endogenous excretion of catecholamines and decrease the incidence of intraoperative hypertension. However, these potent volatile anesthetic agents may be deleterious for patients with severe left ventricular impairment, as their cardiac function might be critically dependent upon adrenergic drive. In this situation a strong narcotic (morphine or Pantopon) is used. Narcotics tend to decrease the heart rate, preload, and total peripheral resistance and decrease both myocardial oxygen consumption and O_2 availability to the heart. Narcotics do not block the response to sympathetic stimulation; hypertension and increased total peripheral resistance may become above normal on surgical stimulation.[11] Also, hypotension frequently occurs with the use of narcotics.

Adjuvant cardiovascular drugs (peripheral vasodilators, alpha adrenergic stimulators, beta blockers) may be used during anesthesia and surgery to keep the balance between preload, afterload, and filling pressures. They should be used only if necessary and while guided by the monitoring parameters.

Monitoring

To normalize the physiologic parameters to achieve stable hemodynamic status and to diagnose myocardial ischemia and dysfunction early, the following parameters should be monitored.

1. PaO_2, $PaCO_2$, pH, serum potassium, and sodium levels, temperature and urine output as often as necessary.
2. Electrocardiogram including chest lead at V5 to detect early signs of intraoperative ischemia.
3. Systemic systolic and diastolic pressures.
4. Left ventricular filling pressure; either pulmonary capillary wedge pressure or left atrial pressure will indicate the left ventricular end-diastolic pressure, left ventricular contractility and performance.[12]
5. Central venous pressure or right ventricular filling pressure. The value of central venous pressure measurement is sometimes questioned; however, it cannot be overemphasized in patients with coronary artery disease. Although rare, dominant intraoperative right side failure does occur.
6. Cardiac output, cardiac index, systemic vascular resistance. In critical situations the measurement of these parameters will be necessary to determine and regulate the use of alpha and beta sympathetic stimula-

tors and blockers and to decide about mechanical support of the circulation.

Causes and management of intraoperative myocardial dysfunction

Decreased myocardial contractility due to the following. *Metabolic factors.* Hypothermia, hypoxia, hypercarbia, pH, and electrolyte changes (these values are usually normalized by the anesthesiologist).

Intraoperative ischemia. Coronary artery spasm is minimized by controlling the sympathetic reflexes and routinely using intravenous coronary vasodilators such as nitroglycerin. We try to maintain perfusion pressure above 60 mm Hg and minimize periods of anoxic arrest. We resort to hypothermia and the use of cardioplegic solutions to arrest and preserve the myocardium in borderline patients, and in patients with hypertrophied left ventricle. Rarely, signs of ischemia can be due to air embolus or due to a kink of the graft used.

Changes in heart rate and rhythm. To maintain the balance between myocardial oxygen supply and myocardial oxygen consumption we try to maintain heart rate and rhythm within normal range. To achieve this, we resort to the use of electrical pacing, or administration of antiarrhythmic drugs or beta blockers.

Abnormalities in preload and afterload. Two important factors must always be kept in mind. Right and left sides of the heart function as two pumps in series and their function depends upon loading and unloading each other.[13] The relation between the initial fiber length and intraventricular pressure may vary considerably because of changes in ventricular compliance.

Accordingly, each ventricle has an optimal filling pressure; when it is achieved, the optimal cardiac output is achieved. Higher filling pressures will not increase the carbon monoxide but will cause cardiac dilatation, increase myocardial oxygen consumption, and eventually lead to cardiac failure.

Hypovolemia is manifested by decreased right and left ventricular filling pressures, reduced cardiac output, and falling blood pressure and tachycardia. Hypovolemia may be absolute or relative, since patients who have had congestive heart failure usually require larger volume to maintain the filling pressures. Hypovolemia may be necessary to control the filling pressures while supporting the circulation by other means.

Hypervolemia. To maintain the blood pressure relative, hypervolemia is the cheapest work for the heart. However, it should be allowed to the extent that it does not raise either the left or right filling pressure above the optimal levels. Overtransfusion does not necessarily cause hypervolemia, as many transfused fluids diffuse very rapidly to the extravascular compartment. In general, the amount of transfusion should be controlled according to changes in the filling pressures, most important the filling pressure of the impaired side whether it be right or left ventricle.

Hypertension is frequently noted during endotracheal intubation, while opening the chest, during cardiopulmonary bypass, and most commonly in the immediate postoperative period. Hypertension is deleterious to the patient with coronary artery dis-

ease since it increases left ventricular end-diastolic pressure and myocardial oxygen consumption. It is rarely due to hypervolemia, but mainly due to increased circulating catecholamines, or it is of neurogenic origin. We proved that cardiac reflexes play an important role in the pathogenesis of the immediate postoperative hypertension.[14] The role of these reflexes during surgery needs further study and investigation.

Hypotension. As hypotension decreases coronary perfusion it aggravates myocardial ischemia and initiates a viscious circle of myocardial dysfunction.

Cardiac decompensation. *Dominant right ventricular failure*[15] is not an uncommon situation in patients with right coronary artery disease. Although rare, it is a serious complication and the mortality rate is high. It is caused by severe intraoperative ischemia or infarction of the right side of the heart.

For this particular problem the following treatment is used: (1) insure maximum possible revascularization; (2) control heart rate by clinical pacing; (3) maintain balance between right ventricular preload and afterload.

The preload may be decreased by relative hypovolemia and the use of peripheral vasodilators. The right ventricular filling pressure should not be allowed to rise to the levels at which the right ventricle starts to distend and fail and its output becomes minimal. Therefore, lower left ventricular filling pressure and systemic blood pressure can be acceptable. Isoproterenol is a valuable drug in this situation because of its inotropic effect, and because it lowers the pulmonary vascular resistance and decreases the right ventricular afterload. In some instances mechanical assist of the circulation did improve right ventricular function, possibly secondary to increased coronary perfusion and improvement of left ventricular function.

Left ventricular failure and combination of varying degrees of both right and left side failure. In this situation cardiac output and systemic pressure can be improved by maintaining the balance and achieving optimal filling pressure for both sides of the heart. One or more of the following cardiovascular drugs can be used according to hemodynamic parameters.

1. Inotropic agents increase myocardial contractility and decrease heart size and ventricular pressure and decrease myocardial oxygen consumption. However, when used in large doses and in patients with hypertrophied ventricle, coronary blood flow is decreased causing subendocardial ischemia. Digitalis is often beneficial in these patients and is more frequently needed for patients who have been digitalized preoperatively.

2. Peripheral vasodilators are effective both on veins and arterioles;[16] nitroprusside, nitroglycerin, and phentolamine (Regitine) are preferred to arteriolar vasodilators. They decrease both afterload and preload. They do not stimulate the heart, nor do they cause pulmonary hypertension. As vasodilators decrease filling pressures of both the left and right sides of the heart, they reduce myocardial O_2 consumption.[17] However, during their use, systemic blood pressure, particularly the diastolic pressure, should not be allowed to decrease to the extent that it seri-

ously affects coronary perfusion.

3. Vasopressors elevate systemic pressure and increase coronary perfusion. This is usually done at the expense of increased left ventricular work and O_2 consumption.

4. Combinations of vasodilators and inotropic vasopressors achieve reduced cardiac work by vasodilators together with maintaining adequate systemic blood pressure.

5. The use of mechanical support of circulation. Return to partial or total cardiopulmonary bypass during surgery allows better perfusion of the myocardium while the myocardial oxygen consumption is enormously reduced. Also, it allows time for correction of any metabolic abnormality and for cardiotonic agents to achieve its effects.

The use of the intra-aortic balloon pump to assist the circulation mechanically has proved to be effective and valuable in many situations. The use of left ventricular assist devices and more recently right ventricular assist devices may play a more significant role in the assist of circulation in the future.

Summary

The clinical picture of the patient with coronary artery disease varies greatly from patient to patient. The hemodynamic status of these patients is unpredictable, since it can change and deteriorate in a few minutes. Therefore, the anesthetic management can only be planned for the particular patient according to his hemodynamic status at surgery. In general, anesthetic techniques and agents should be selected to minimize myocardial oxygen consumption and maintain or increase O_2 supply to the heart. This necessitates controlling the reflexes which cause changes in the blood pressure, heart rate, and rhythm. Every effort should be made to preserve the myocardium and to prevent intraoperative ischemia and dysfunction. These patients must be carefully monitored to normalize or stabilize any changes in the physiologic and hemodynamic parameters. Volume replacement and the use of cardiovascular agents should be directed toward the balance of preload and afterload to achieve optimal filling pressure in both the right and left sides of the heart, and to maintain adequate systemic and coronary perfusion.

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