Anesthesia and heart reoperations

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The number of reoperations for heart disease in the Cleveland Clinic has been steadily increasing and currently comprises 4.5% of total myocardial revascularizations. Because of progressive disease and potential technical difficulties, reoperations usually have higher mortality and morbidity rates than the first operations (*Table*). This report summarizes our experience during the past decade in the management of anesthesia for patients undergoing these reoperations.

Reoperative revascularization is indicated for one or more of the following: progressive coronary atherosclerosis, graft failure, incomplete previous revascularization, Vineberg implants, and graft atherosclerosis.[1]

The incidence of left ventricular impairment and preoperative angina is also high. Loop et al[2] reviewed our experience in 500 patients; 25.6% had New York Heart Association Class II angina, 46.4% had Class III angina, and 26.8% had Class IV angina.[2] Ventricular hypokinesis or akinesis was detected in 29% of the patients, and 18% had poor ventricular contractility. Because of the severity of symptoms, an intraaortic balloon pump was required to support circulation in about 5% of the patients, either preoperatively or postoperatively.

Valve replacement is indicated when there is

Table. Complications and blood utilization following first operation/reoperative myocardial revascularization

	Reoperative revascularization		First operation
	Results		Results
	Pre-1978	1978	1978
Reoperations for bleeding	10.2%	4%	3%
Perioperative myocardial infarction	8.6%	6.6%	1.2%
Respiratory insufficiency	4.6%	3%	0.7%
Units of blood	7.3	3	2.1

deterioration of the valve after the initial aortic or mitral valve replacement, previous commissurotomy, endocarditis that is resistant to treatment, prosthetic dysfunction, perivalvular leak, and valve thrombosis. Not infrequently, these patients require emergency surgery, since sudden valve dysfunction or severe perivalvular leak can predispose to congestive heart failure or even acute pulmonary edema.[3]

The management of these patients necessitates every effort to maintain the myocardial oxygen demand ratio, starting in the preoperative period and continuing until the postoperative period. Beta blockers and antihypertensive therapy are continued until surgery to prevent preoperative ischemic and hypertensive crises.[4] Coronary vasodilators and beta blockers should be used deliberately to treat preoperative anginal pains and ischemia.

Our current anesthesia management for reoperations consists of strong premedication: scopolamine, 0.4 mg and morphine sulfate, 0.2 mg/kg. We also apply nitroglycerin paste, 2 square inches, to the back of the patient one hour before surgery to prevent anginal pain and to decrease afterload.

Comprehensive monitoring is started before induction of anesthesia. We monitor ECG leads II and V and introduce a pulmonary artery double lumen catheter (Swan-Ganz) to measure right and left ventricular filling pressures, cardiac output, and total peripheral resistance. These measurements are continued in the postoperative period and allow early detection and proper diagnoses for the causes of ischemia or myocardial dysfunction or both. Interpretation and coordination of the monitored parameters allow a specific, symptomatic treatment when it is indicated.

The final selection of anesthetic agents is usually determined at the time of induction according to the hemodynamic status of the patient at that moment.[5] In patients with coronary artery disease, the autoregulatory mechanisms may not function; therefore, adequate perfusion pressure is critical in maintaining the blood flow in the diseased vessels. We try to maintain the diastolic blood pressure above 65 mm Hg in patients with coronary artery disease.

If the heart rate is above 80 beats per minute before induction of anesthesia, we administer 1 to 2 mg propranalol intravenously to reduce the heart rate to about 65 beats per minute.

We usually adopt one of two anesthetic regimens. (1) In circulatory-stable patients, anesthesia is induced with a sleeping dose of sodium thiopental, + 50% O_2 + 50% N_2O, to which an in-

creasing concentration of halothane is added until the patient is probably anesthetized to minimize the reflex increase in heart rate and blood pressure that may complicate induction. (2) If the initial hemodynamics are unstable and hypotension is expected, we use a strong narcotic for induction (50 μg/kg fentanyl + 100% O_2). Pancuronium bromide is used as the muscle relaxant to ensure greater stability of the blood pressure. Maintenance of anesthesia is based upon hemodynamic changes that follow induction and is accomplished by either N_2O-O_2-halothane or intermittent doses of narcotics according to the hemodynamic status and changes following induction.

It is worth emphasizing that the time between induction of anesthesia and initiation of cardiopulmonary bypass is prolonged due to the difficulty in reentering the chest because of adhesions from previous surgery. We are constantly aware of possible trauma to the lungs, great vessels, and even to the heart itself during reentry, and a means for massive transfusion is always available.

Our surgeons routinely isolate the femoral vessels before opening the chest to initiate cardiopulmonary bypass immediately in case of major bleeding or myocardial dysfunction. They also mobilize the left ventricle on cardiopulmonary bypass to prevent hypotension and arrhythmias. At all stages of surgery, any signs of myocardial ischemia or myocardial dysfunction are treated promptly and specifically by coronary and peripheral vasodilators, inotropic agents, or vasopressor agents according to the needs of the patient.

During cardiopulmonary bypass, maximal myocardial preservation techniques are used including the administration of cold saline cardioplegic solutions, systemic cooling to 28 C, and occasional supplementation by local pericardial cooling.

Blood transfusions and excessive bleeding can cause complications in reoperations. Occasionally it is difficult to obtain enough properly matched blood because of the presence of antibodies resulting from blood transfusions given during the first operation. Some patients are receiving anticoagulant therapy preoperatively, which will aggravate the already high possibility of oozing due to adhesions. Also, massive blood transfusions can increase the incidence of postoperative hepatitis with its sequelae. For the past 5 years we have adopted a strict blood utilization and conservation program.[6] This includes withdrawal of 1 or 2 units of blood from the patient for retransfusion after heparin reversal, allowing hemodilution during bypass to a hematocrit of 20%, and the use of packed cells if the hematocrit falls below this value. The blood, which is recovered with suction bottles, is washed and retransfused. We ensure adequacy of heparization and its reversal by protamine in all patients by routine measurement of activated clotting time.

Currently at the Cleveland Clinic the incidence of postoperative reexploration for bleeding following reoperations has declined from 10.2% to 4%, average blood utilization dropped from 7.3 units to 3 units per patient, and the incidence of postoperative hepatitis has decreased to 1.6%.

Postoperatively, the incidence of respiratory insufficiency is higher in reoperations than in the first procedures (3% versus 0.7%, respectively). This higher incidence is attributed to unstable hemodynamics, prolonged cardiopulmonary bypass, the amount of blood trans-

fused, and both pleura being left open at the end of operation. Our routine respiratory care includes overnight ventilation utilizing preset volume ventilators, initial application of positive end-expiratory pressure, and gradual weaning from mechanical ventilation. Respiratory support is continued with incentive spirometry and chest physiotherapy until hospital discharge.[7]

Summary

Improved results of reoperations were dependent upon (1) a team of expert surgeons, anesthesiologists, and nurses who became familiar with technical problems, risks, and their management; (2) management that included optimal myocardial preservation before, during, and after surgery; (3) intelligent use of pharmacologic and mechanical means to support circulation; (4) optimal blood utilization and strict hemostasis; and (5) aggressive and extended postoperative hemodynamic and respiratory support.

References

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