Anesthesia in the heart patient for noncardiac surgery

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There is a small hazard for every patient who is given anesthesia and undergoes an operation. If the patient also has cardiovascular disease, the surgical event may be likened to an uncontrolled stress test associated with significant morbidity and mortality. That most patients with severe heart disease survive even the most major surgical procedures is a credit to the modern management of anesthesia and supportive care.

Since approximately 5% of the adult population of the United States have ischemic heart disease, most practicing anesthesiologists must regularly manage the problem. To do this safely, we must recognize those at increased risk and be cognizant of the hazards related to anesthesia and operation. Preoperatively these patients must be assured that they are in the best state possible. In the preoperative and postoperative periods they need special care and attention.

Recognition and evaluation of ischemic disease

Recognition is often difficult, and a high index of suspicion must be maintained. Many patients with severe ischemic disease are asymptomatic with no positive history. Fifteen percent to 20% of myocardial infarctions may be painless[1] and undiagnosed at that time. An important aid to the

diagnosis of occult disease is the coronary risk profile, which includes obesity, hypertension, diabetes, familial hyperlipidemia, stress, and smoking. Ischemic heart disease is more common from the fifth decade onward, but a considerable incidence of coronary occlusion occurs in the 30- to 40-year-old group. A careful history, physical examination, and investigation are essential for accurate recognition and evaluation.

History. Angina pectoris may have many different presenting features, often being misdiagnosed as a variety of conditions, including reflux esophagitis, peptic ulcer, cholelithiasis, or musculoskeletal problems. Dyspnea on effort may be related to myocardial ischemia, frequently occurring without classic precordial pain. The degree of exercise tolerance must be ascertained, and if angina is present, it should be graded according to the classification of the New York Heart Association.[2] A history of intermittent claudication makes coronary artery disease likely, and transient cardiac ischemic episodes or syncope should point to the possibility of conduction defects or severe aortic stenosis. Nocturnal dyspnea and orthopnea are well-known symptoms of left ventricular failure. A nocturnal cough may be the first symptom in a young patient with mitral valve disease. It is due to bronchial mucosal congestion secondary to elevated bronchial-venous pressure.

Examination. A meticulous examination of systems should identify those patients with significant cardiovascular disease. Even if the results appear to be negative, one should assume that disease exists if high risk factors are present, such as obesity and chronic obstructive lung disease. An important predictor of underlying ischemic disease is the presence of a diagonal crease on the ear lobe.[3]

Investigation. Mass screening preoperatively for the detection of ischemic disease is a complex issue. Its value must be continually reappraised in terms of cost effectiveness and possible harm to the patient population.[4] There are advocates for a preoperative electrocardiogram in all patients over a certain age for comparison should complications occur later.[5] This must be balanced against the likelihood of postoperative infarction, which is less than two per thousand in one large series.[6] The inherent limitations of electrocardiography must be considered. Patients with a history of nontransmural myocardial infarction may have normal electrocardiograms.[1]

Most institutions follow the policy whereby all patients older than age 40 have a preoperative baseline electrocardiogram, unless they had one within 12 months, with no symptoms in the interim. A similar policy should exist regarding preoperative chest roentgenograms.

The above simple sequence of history, physical examination, and investigation forms the foundation of evaluation of the average patient. The investigation can be restricted to electrocardiography and chest roentgenograms unless there is uncertainty as to the existence and severity of cardiovascular disease.

Electrocardiographic exercise stress testing may be misleading due to a sensitivity of only 50% to 80% in identifying symptomatic patients with occlusion of large vessels.[7] The sensitivity in asymptomatic patients may be less than 50%.[8] Current research is attempting to improve the predictive value of stress testing. The pretest risk profile of coronary disease present in each patient is important in interpreting the stress test.[9]

Radionuclide techniques are more accurate than exercising to a stress level,

but are not yet widely available.[10] After intravenous injection, the radionuclide, e.g., potassium 43, distributes throughout the myocardium in proportion to regional blood flow. The pattern of distribution is detected by a scintillation camera with ischemic or infarcted areas showing as cold spots, i.e., patches of reduced radioactivity.

Echocardiography is another valuable noninvasive technique. Left ventricular function can be assessed, indicating the degree of myocardial ischemia. The presence and severity of valvular disease can also be investigated.

If the clinical findings, including those found in noninvasive investigations, are suggestive of critical coronary artery disease, then coronary arteriography and cardiac catheterization are necessary for more definitive evaluation.

The long-term prognosis in coronary disease depends on the extent of narrowing of vessels, ability to alter the risk profile, and the response to medical and surgical treatment. Surprisingly, the outlook is now improving. Since 1968 the death rate from coronary disease in the United States has declined 28%.[11] Both primary prevention through changes in risk factors and better medical care probably have been involved, but which of these is more important is not yet known. If the total number of heart attacks is decreasing, changes in risk factors must be largely responsible. If the total incidence is unchanged, better medical treatment must be reducing the mortality rate due to infarction.

Preoperative preparation

Unstable angina. In patients with intractable (Class IV) angina, elective, noncardiac operations should be postponed until after myocardial revascularization with bypass vein grafts. When noncardiac surgery is preceded by bypass grafting, the incidence of perioperative infarction is reduced.[12, 13] If the noncardiac operation is an emergency, two options are open: (1) perform aortocoronary bypass grafting and the noncardiac operation at the same session or (2) perform the noncardiac operation with an intraaortic balloon in place, thus reducing myocardial work and improving coronary perfusion.

Congestive failure. Preoperative therapy should be aggressive while avoiding digitalis toxicity. Digoxin is preferred to digitoxin, since its effects are of shorter duration and is faster acting. Renal function and fluid and electrolyte balance must be monitored frequently. Patients on long-term diuretic therapy often have a substantial potassium deficit. This may be unmasked by the fluid and electrolyte fluxes during and after operation, with potentially lethal digoxin-induced dysrhythmias. If digoxin toxicity is a real possibility, the drug should be withheld for 24 to 48 hours before operation.

Hypertension. There is a difference of opinion in what is considered a significant elevation of blood pressure. However, diastolic pressures above 110 mm Hg should be stabilized at a lower level with the patient on a suitable regimen chosen by a medical consultant. Patients with untreated severe hypertension show marked lability of pressure under anesthesia, whereas those receiving effective medications do not.[14] Thus in patients with well-controlled blood pressures, the drugs should be continued to the morning of operation. Similarly, antihypertensive drugs should be given early postoperatively, even intravenously, to prevent acute hypertension.[15]

Beta blockade. Blockade of beta- adrenergic nerve endings is widely used for control of angina and hypertension. Propranolol should be given also until

the morning of operation. The beneficial effect is that beta blockade reduces the sympathetic response to stress, avoiding a disastrous increase in myocardial oxygen need. There appears to be no valid reason even to taper the dose; indeed the anesthetist may give propranolol before induction to inhibit sympathetic response.

Heart block. A temporary pacemaker should be inserted preoperatively in patients with second- or third-degree block. Those with bifascicular block (i.e., left anterior hemiblock and right bundle-branch block) need a pacemaker only if they have had episodes of syncope.

Oxygen availability. Pulmonary function and oxygen-carrying capacity must be improved maximally. Smoking should be stopped at least 2 weeks preoperatively. Chest physiotherapy, antibiotics and bronchodilators may be beneficial. Pulmonary studies must be done if function is suspected to be compromised. Anemia should be corrected by the best means in the time available. In this respect, iron therapy is preferable to red cell transfusion.

Prophylaxis of thromboembolism. Thromboses and emboli occur more commonly in patients with ischemic heart disease than in normal patients.[16] Pelvic procedures and operations on the legs carry the highest risk. Prophylaxis consists of a regimen of heparin subcutaneously, which continues postoperatively, plus early mobilization and physiotherapy.

Preoperative sedation

Cardiac patients tend to be more apprehensive and anxious than usual. They should be given a more lengthy explanation of forthcoming events, with maximum reassurance and establishment of rapport. Sedation and relief of anxiety is the goal. Pentobarbitol (200 mg) administered the night before, plus diazepam orally (5 to 10 mg) followed by Pantopon or morphine (10 to 20 mg) and perphenazine (2 to 5 mg) preoperatively, usually achieve the desired effect.

Anesthetic management

To anesthetize a patient without occasionally provoking severe changes in hemodynamics requires infinite care. Merely inserting an oral airway at a light level of anesthesia can cause a profound sympathetic response. This is even more likely with laryngoscopy and endotracheal intubation.[17] The consequent hypertension increases left ventricular wall tension with a rise in circulating catecholamines, tachycardia, and increased myocardial contractility. The need for myocardial oxygen becomes greater due to a rise in major determinants of oxygen demand. With a compromised oxygen supply from narrowed arteries, ischemia and later infarction can rapidly supervene.

Management of patients and particularly those with ischemia must be aimed at maintaining the myocardial oxygen supply-demand ratio. Events that increase oxygen demand, tachycardia, and hypertension must be avoided.

One must not allow a decrease in the supply of oxygen, which may occur when the oxygen-carrying capacity of blood is reduced (hemodilution, decreased hemoglobin) and when coronary blood flow falls (severe hypotension).

General anesthesia

Monitoring. Patients with ischemic disease should have electrocardiographic monitoring with V4 (or V5) unipolar lead, which is best for noting ST-segment changes caused by is-

chemia. Direct arterial pressure and blood gas monitoring are essential in the high-risk case. Central venous pressure is an adequate guide for volume replacement when left ventricular function is normal. For patients with left ventricular dysfunction or failure or those undergoing extensive operations, only the flow-directed pulmonary arterial catheter can provide the necessary information.[18] Precise management of the circulation in the presence of impaired myocardial function can only be done with knowledge of left ventricular end-diastolic pressure. The wedge or pulmonary arterial occluded pressure correlates well, except in severe pulmonary hypertension.

Induction. Intravenous thiopental depresses the myocardium and dilates the peripheral bed, but a sleep dose (150 to 300 mg) given slowly seldom causes a major fall in blood pressure. Similarly, diazepam (5 to 20 mg) induces sleep rapidly with minimal circulatory change. Morphine (to 1 mg/kg) intravenously is another approach causing no cardiac depression, but it must be given over a 10- to 20-minute period to avoid hypotension from vasodilation. Fentanyl (10 μg/kg) is the effective inducing agent.[15] It minimizes the pressure response to intubation but may cause bradycardia.

Pancuronium (0.05 to 0.1 mg/kg) is the usual relaxant, particularly in the well beta-blocked patient with a rate below 60/min. It causes a significant rate increase in the patient not blocked with propranolol. In this context, *d*-tubocurare (0.3 mg/kg) is returning to favor. While not raising heart rate, it often produces severe hypotension, which will be reversed by intubation. Metocurine (0.05 to 0.1 mg/kg) is another effective competitive blocker that has minimal circulatory effects.[19]

A safe sequence for induction and intubation consists of thiopental followed by pancuronium; then assisted ventilation with nitrous oxide, oxygen, and a volatile agent (enflurane or halothane) for 3 to 4 minutes. The arterial pressure is closely watched and the mean pressure reduced by about 25% below the awake level. Anesthesia should be deepened with inhaled agent, as much as the blood pressure will allow to blunt the sympathetic response to intubation. With the latter, the pressure will rise slightly above normal without a large overshoot.

When rapid induction is necessary and particularly in hypertensive patients, intravenous propranolol (1 to 5 mg) is useful before induction until there is evidence of beta blockade. This dampens the sympathetic pressor response to intubation. Careful topical spraying of the larynx,[20] without laryngoscopy, or intravenous lidocaine (2 mg/kg) are also useful measures.

Dysrhythmias after intubation are usually ventricular and accompany severe hypertension. Rapid treatment is essential to reverse myocardial ischemia. Deepening the anesthetic is necessary. Intravenous propranolol, lidocaine, nitroglycerin (200 to 400 μg) to reduce the blood pressure will reduce oxygen consumption and abolish hyperirritability.

Maintenance. The aim is to produce and maintain a state of controlled myocardial depression. The heart rate should be kept slow and the blood pressure slightly below the awake reading. However, coronary perfusing pressure must be adequate (no severe hypotension) and the arterial oxygen tension kept from 100 to 150 mm Hg.

Halothane and enflurane have effects analogous to propranolol, a reduction of contractility directly related to the concentration of the agent in blood. But

since they are potent agents, sufficient anesthesia results from the low concentrations that may be required in cardiac patients. Another advantage is that anesthesia can be quickly deepened in anticipation of a strong stimulus to prevent increases in rate and pressure.

Neuroleptic anesthesia (fentanyl and droperidol) or increments of morphine, along with nitrous oxide, are other approaches. Nitrous oxide depresses cardiac output[21] and must be omitted in the most critically ill patients.

Intermittent analysis of blood gases is indicated when there is doubt relative to acid-base status or adequacy of oxygenation. With low cardiac output, dangerously low arterial oxygen tensions can be present with 50% inspired oxygen due to the widened alveolar-arterial differences in tension. When PaO_2 falls below 100 mm Hg, the nitrous oxide should be turned off. The $PaCO_2$ is best kept normal rather than low, because alkalosis will reduce serum potassium (arrhythmias) and cause hypotension.

Managing the hyperdynamic circulation

Frequent calculation of the rate-pressure product is the best way of avoiding myocardial ischemia.[22] Simply multiply the heart rate by the systolic pressure, e.g., 110 × 60 = 6600. Either rate or pressure increases, or usually both, produce a larger product. If it exceeds 15,000, or certainly at 20,000 (200 × 100), immediate intervention is necessary even if electrocardiographic signs of ischemia are not yet present. Again propranolol (to 3.5 mg in 0.5- to 1-mg doses) or boluses of nitroglycerin, 200 to 400 μg) or nitroprusside (100 to 400 μg) will rapidly reduce rate and blood pressure. It is likely that nitroglycerin or nitroprusside by drip will be needed to prevent recurrence. Ischemic electrocardiographic changes may occur before the rate-pressure product rises severely; the same treatment is indicated. If there is a possibility that anesthesia is too light, when the rate-pressure product rises significantly, further depth should be brought about.

In using the intravenous dilators by drip, aortic diastolic pressure must not be reduced too severely, since it is the major determinant of coronary perfusion. Nitroglycerin has its major effect on veins, so preload reduction may be a problem requiring increase of circulating volume, whereas nitroprusside primarily reduces afterload by arteriolar dilatation. In using these potent drugs, the Swan-Ganz catheter is essential to follow accurately left ventricular filling pressure.

Managing the hypodynamic circulation

Acute cardiac decompensation during anesthesia may be caused by many factors, all deleterious to cardiac performance. A major determinant is the degree of cardiac dysfunction present before anesthesia, such as scarred, noncontracting segments or a generally reduced contractility. Rate or rhythm problems can appear intraoperatively from electrolyte or acid-base abnormalities.

Inappropriate dosage of potent inhaled agents may severely compromise cardiac function. The opiates in large doses cause venous pooling and inadequate venous return, especially when chronic hypovolemia from diuretics is present.

The positive pressure of controlled ventilation reduces venous return and blood flow from the right to left heart, compounding the fall in output. In this situation, all inhalational agents should be discontinued, the inspired oxygen

concentration should be increased to 100% and the CO_2, if low, should be allowed to rise. If the resulting rise in endogenous catecholamines does not quickly improve cardiac output and blood pressure, positive inotropic agents are the next therapeutic step. Again, use of the pulmonary arterial catheter is the only way left ventricular dysfunction can be assessed and managed.

For acute and severe hypotension due to myocardial failure (wedge pressure >25 mm Hg), $CaCl_2$ (1 to 2 mg/kg increments to 1 to 2 g) often will improve cardiac output.[23] It is also a pulmonary and peripheral arteriolar constrictor, the latter action helping to raise the blood pressure.

Catecholamines by drip may also be needed to maintain satisfactory cardiac output. Dobutamine is a relatively new and effective one that appears to have less chronotropic effect than dopamine and isoproterenol.[24] Epinephrine is seldom used. Norepinephrine by itself produces too great an increase in afterload for a failing heart, but a combined solution of norepinephrine and phentolamine has its advocates. The phentolamine antagonizes the alpha-agonistic peripheral effects of norepinephrine leaving the positive inotropic action on the myocardium. In general, the aim with catecholamines is to increase cardiac output without increasing blood pressure or heart rate above normal.

The circulatory response to decreasing cardiac output is to increase peripheral resistance, so that the patient in heart failure is likely to have intense constriction, which will further reduce output. Based on this, another valuable method of treatment is to reduce afterload by nitroprusside or nitroglycerin by drip, or hydralazine in intermittent doses. Usually the blood volume must be increased to fill the larger bed and maintain venous return. Simultaneous administration of nitroprusside or nitroglycerin for peripheral dilation and a catecholamine for myocardial stimulation, seems to be the best current approach to treatment of cardiogenic shock.[24]

Rapid digitalization with digoxin may improve cardiac performance if the patient was not receiving it previously. Initially, 0.5 mg should be given and followed with 0.2 mg at 20- to 30-minute intervals until signs of effect are seen, usually at 1.5 mg.

Other measures for intraoperative acute myocardial depression include a high inspired oxygen tension, treatment of rate and rhythm problems, metabolic acidosis, and hypokalemia. In addition, the blood volume should be increased to the point that the pulmonary wedge pressure rises to 20 mm Hg.

References

1. Ross RS, Lesch M, Braunwald E. Acute myocardial infarction. In: Harrison's Principles of Internal Medicine. New York: McGraw-Hill, 1977: 1271–2.
2. New York Heart Association. Nomenclature and Criteria for Diagnosis of Diseases of the Heart and Great Vessels. 8th ed. Boston: Little, Brown and Co, 1979.
3. Sprague DH. Diagonal ear-lobe crease as an indicator of operative risk. Anesthesiology 1976; **45:** 362–4.
4. Hiatt HH. Protecting the medical commons; Who is responsible? N Engl J Med 1975; **293:** 235–45.
5. Goldman L, Caldera DL, Nussbaum SR, et al. Multifactorial index of cardiac risk in noncardiac surgical procedures. N Engl J Med 1977; **297:** 845.
6. Tarhan S, Moffitt EA, Taylor WF, Giuliani ER. Myocardial infarction after general anesthesia. JAMA 1972; **220:** 1451–4.
7. Borer JS, Brensike JF, Redwood DR, et al. Limitations of the electrocardiographic response to exercise in predicting coronary-artery disease. N Engl J Med 1975; **293:** 367–71.
8. Borer JS. Exercise testing; electrocardiogra-

phy and radionuclide cineangiography. Cleve Clin Q 1978: **45:** 9–10.
9. Rifkin RD, Hood WB Jr. Bayesian analysis of electrocardiographic exercise stress testing. N Engl J Med 1977; **297:** 681–6.
10. Hillis LD, Braunwald E. Myocardial ischemia. N Engl J Med 1977; **296:** 971–8.
11. Levy RI. Progress in the prevention of cardiovascular disease. Prev Med 1978; **7:** 464–75.
12. Mahar LJ, Steen PA, Tinker JH, Vlietstra RE, Smith HC, Pluth JR. Perioperative myocardial infarction in patients with coronary artery disease with and without aorta-coronary artery bypass grafts. J Thorac Cardiovasc Surg 1978; **76:** 533–7.
13. McCollum CH, Garcia-Rinaldi R, Graham JM, DeBakey ME. Myocardial revascularization prior to subsequent major surgery in patients with coronary artery disease. Surgery 1977; **81:** 302–4.
14. Prys-Roberts C, Meloche R, Foëx P. Studies of anaesthesia in relation to hypertension. I. Cardiovascular responses of treated and untreated patients. Br J Anaesth 1971; **43:** 122–37.
15. Prys-Roberts C. Anesthetic considerations in the hypertensive patients. Annual Refresher Course Lectures. American Society of Anesthesiologists. Course 139, 1979.
16. Dalen JE, Dexter L. Operation in the patient with heart disease. In: Cardiac and Vascular Disease. Philadelphia: Lea & Febiger, 1971: 1450–62.
17. Prys-Roberts C, Greene LT, Meloche R, Foëx P. Studies of anaesthesia in relation to hypertension. II. Haemodynamic consequences of induction and endotracheal intubation. Br J Anaesth 1971; **43:** 531–46.
18. Lappas DG, Powell WMJ Jr, Daggett WM. Cardiac dysfunction in the perioperative period; pathophysiology, diagnosis and treatment. Anesthesiology 1977; **47:** 117–37.
19. Stoelting RK. Hemodynamic effects of dimethyltubocurarine during nitrous oxide-halothane anesthesia. Anesth Analg 1974; **53:** 513–5.
20. Stoelting RK. Circulatory changes during direct laryngoscopy and tracheal intubation. Influence of duration of laryngoscopy with or without prior lidocaine. Anesthesiology 1977; **47:** 381–4.
21. Eisele JH, Smith NT. Cardiovascular effects of 40 percent nitrous oxide in man. Anesth Analg 1972; **51:** 956–63.
22. Wilkinson PL, Moyers JR, Ports T, Chatterjee K, Ullyot D, Hamilton WK. Rate-pressure product and myocardial oxygen consumption during surgery for coronary artery bypass. Circulation 1979; **60** (suppl 1): 170–3.
23. Stanley TH, Isern-Amaral J, Liu WS, Lunn JK, Gentry S. Peripheral vascular versus direct cardiac effects of calcium. Anesthesiology 1976; **45:** 46–58.
24. Chatterjee K. Low output syndrome in ischemic heart disease. Hemodynamic diagnosis and management. Annual Refresher Course Lectures, American Society of Anesthesiologists. Course 219, 1979.