Anesthetic considerations for coronary artery surgery

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Anesthetic considerations for coronary artery surgery encompass a wide range of important details, all of which have as an end the reduction of perioperative myocardial injury.[1-6] We believe that with the advances made in the past decade every patient should leave the operating room with a better functioning heart, and the challenge to the anesthesiologist is to bring the patient safely to that point in the operation when the surgeon arrests and repairs the heart.

We consider the single greatest advance in this field to be the introduction of cold crystalloid potassium cardioplegia.[7] It has reduced our perioperative transmural infarction rate from 20.8% to 4% and it is clear to us that this reduction has been entirely due to the use of cardioplegic arrest of the heart.

Anyone discussing coronary artery disease inevitably analyzes those factors that affect the crucial balance between the oxygen supply and demands of the myocardium. I will not critically evaluate each factor that, in one way or another, can affect this balance as this will be discussed elsewhere. I do emphasize, however, that maintaining this critical oxygen balance is the single most important factor that the anesthesiologist may control and that will affect the successful outcome of surgery. The pur-

pose of this paper is to emphasize those factors that we believe are important if we are to provide the patient with a maximal opportunity for a successful operation.

Preoperative assessment

The factors that we consider important begin with the preoperative evaluation of the patient, where every effort must be made to determine the patient's myocardial reserve. It is essential that the anesthesiologist should know the anginal history, the number of infarcts and most recent infarct, the extent of the coronary lesions to be operated on, and the mechanical efficiency of the heart. The ejection fraction, end-diastolic pressure, and wall motion are among the laboratory investigations that will allow one to determine a patient's reserve.[8]

The patient should be brought to the operating room in optimal condition. When there is evidence of failure, the patient should be receiving digitalis. Five years ago, all patients on a regimen of propranolol to control angina would probably have had it discontinued preoperatively because of an exaggerated fear of the myocardial depression produced by this drug. It is now generally recognized that in patients whose angina is being controlled by propranolol, myocardial ischemia and ventricular arrhythmias may develop if the drug is suddenly stopped.[9] For this reason we maintain patients on that amount of the drug that will help control the angina; we like to see a resting heart rate of 55 to 60/min. It is continued to the time of surgery. All patients receive sublingual nitroglycerin to treat any anginal attacks that occur, and patients with unstable angina have nitroglycerin paste applied to their chests. Some of these patients will have associated diseases such as systemic hypertension, diabetes, and renal disease. We believe that all associated diseases should be appropriately investigated and treated prior to surgery.

These patients tend to be anxious and anxiety can precipitate anginal attacks. Therefore, we premedicate them rather heavily with 0.15 mg/kg diazepam orally and 0.15 mg/kg morphine intramuscularly, combined with 0.4 mg scopolamine.

Anesthetic management

Previous techniques. We have had experience anesthetizing these patients for myocardial revascularization procedures for more than 20 years. Our method of anesthetizing these patients has varied considerably over this period. In 1970, we used diazepam, 0.25 mg/kg intravenously, and if the patient did not fall asleep on this dose a small amount of pentothal was given. The amount required varied between 50 and 100 mg. Anesthesia was maintained with nitrous oxide 60% and oxygen 40%, supplemented by intravenous morphine to a total of 1 mg/kg throughout the operative procedure. Muscle relaxation was accomplished with d-methyl curare. With this technique, it was not uncommon for the patient to become hypertensive and develop tachycardia at the time of laryngoscopy and intubation. When this occurred, halothane was added until the patient's blood pressure and heart rate were controlled. This sometimes proved difficult as the hypertensive patient could suddenly become hypotensive and these episodes at times induced ischemic changes on the electrocardiogram. We were partially successful in obviating hypertension and tachycardia secondary to laryngoscopy and intubation by having the patient gargle with 2 to 4 cc of 4% Xylocaine prior to induction, and administering 3 to 4 cc of

4% Xylocaine transtracheally as the eyelid reflex was disappearing. When halothane was used to treat hypertensive episodes, myocardial ischemia was not a major problem because halothane, while reducing blood pressure through myocardial depression, also reduces myocardial oxygen requirements.[10] When we began to use propranolol for the control of angina preoperatively and maintained it to the time of surgery, we found that the patients had a smoother induction sequence. This observation was confirmed by Kopriva et al[11] who showed that, with a thiopental-succinylcholine-nitrous oxide induction technique, although hypertension was not obviated (*Fig. 1*), tachycardia at the time of intubation was significantly reduced in patients on propranolol (average, 140 mg/day) (*Fig. 2*). The product

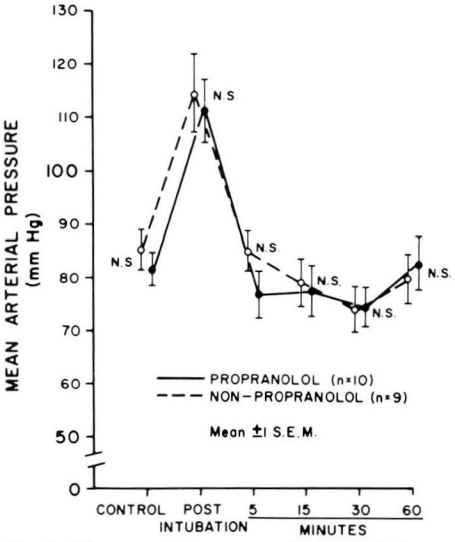

Fig. 1. Mean arterial pressure during halothane-N_2O-O_2-pancuronium anesthesia. During anesthesia, serial mean arterial pressures in patients receiving propranolol did not differ from corresponding values in patients who had not received the drug. (Reproduced with permission from Kopriva CJ, Brown AC, Pappas G. Hemodynamics during general anesthesia in patients receiving propranol. Anesthesiology 1978; **48:** 28–33.)

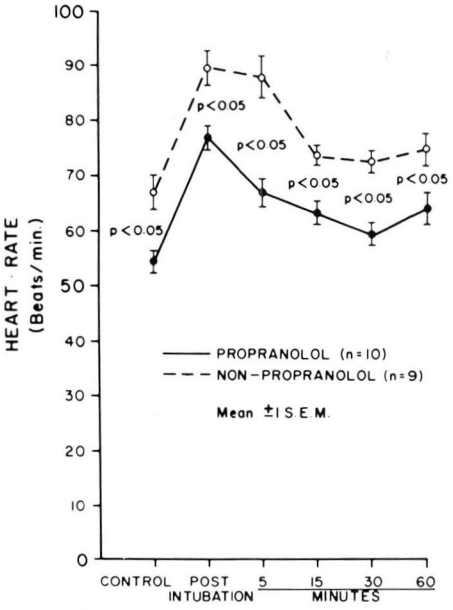

Fig. 2. Heart rate during halothane-N_2O-O_2-pancuronium anesthesia. Uniformly slower intra-anesthetic heart rates were apparent in patients who received propranolol compared with those who did not. (Reproduced with permission from Kopriva CJ, Brown AC, Pappas G. Hemodynamics during general anesthesia in patients receiving propranolol. Anesthesiology 1978; **48:** 28–33.)

of the heart rate and the systolic blood pressure correlates well with myocardial oxygen requirements,[12] and the lower product encountered at the time of intubation in patients on propranolol implies that the critical balance between oxygen supply and demand is better maintained.

Current techniques. Since mid-1979 we have changed our technique in an effort to provide a more stable anesthetic course until cardioplegia is started. We recognized that, although hypertension and tachycardia are most likely to be encountered at the time of intubation, they may also occur at the time of skin incision and sternotomy. As previously stated, the administration of halothane, while obviating these prob-

lems, makes the patients susceptible to hypotension due to the myocardial depressant action of the drug, which may be aggravated by blood loss. Lunn et al[13] have reported that oxygen and high-dose fentanyl provide a remarkably stable hemodynamic course in patients undergoing open heart surgery, and we are now using this technique to anesthetize patients for aortocoronary bypass surgery. All patients have a peripheral IV radial artery cannulation and Swan-Ganz catheter inserted prior to the induction of anesthesia. We had expected that the insertion of this catheter prior to the induction of anesthesia would be upsetting to these patients and that tachycardia and hypertension might develop. The resultant increase in rate pressure product may, of course, be detrimental to them, but the premedication that we have described and careful explanation to the patients preoperatively as to exactly what will be done has resulted in their tolerating these procedures and maintaining a remarkably stable rate pressure product. The highest heart rate and blood pressure encountered is usually that first taken when the patient is brought into the operating room where induction is carried out. During the insertion of the various lines, the patient is repeatedly assured that things are going well and, as a result, the rate pressure product often has fallen by the time the Swan-Ganz catheter is in place.

We use leads 2 and V5 to monitor the electrocardiogram. V5 is used to detect global ischemia and Roy et al[14] have verified the effectiveness of this lead for this purpose.

We studied 10 consecutive patients, seven men and three women (mean age, 59.3 ± 3). Anesthesia consisted of oxygen and 100 μg/kg fentanyl for aortocoronary bypass surgery, and hemodynamic studies were made during induction and initial surgical stimulation. All patients preoperatively were taking propranolol, isosorbide dinitrate, and nitroglycerin. Following insertion of the catheters, control measurements were taken of heart rate (HR, 58 ± 8 bpm), systolic blood pressure (SBP, 151 ± 37 torr), pulmonary capillary wedge pressure (PCWP, 10.4 ± 2.7 torr), cardiac index (CI, 2.73 ± 0.59 1/min), left ventricular stroke work index (LVSWI, 58.5 ± 17.7 g M.M), systemic vascular resistance (SVR, 1646 ± 611 dyne/sec/cm^5/m^2), and rate pressure product (RPP, 8842 ± 2889).

All patients were then given 100% oxygen to breathe followed by pancuronium, 1 mg, prior to the start of the fentanyl infusion. Fentanyl, 50 μg/kg, was infused over a 5- to 8-minute period. Patients no longer responded to verbal command after receiving about 10 μg/kg. Severe chest wall rigidity and contracture of the arms appeared as the patient lost consciousness. Full relaxation was achieved by the injection of pancuronium, 0.1 mg/kg, as soon as inappropriate responses were noted during the fentanyl infusion; this aborted any chest spasm that developed. The patients were subsequently ventilated with 100% oxygen. Observations were repeated after 50 μg/kg fentanyl, endotracheal intubation, a further 50 μg/kg fentanyl, skin incision, and sternal splitting.

The results are shown in *Figure 3*. No significant changes from control were seen in PCWP, SVR and RPP. After 50 μg/kg fentanyl, there was a significant rise (13%) in HR (66 ± 16 bpm). This increase in HR is probably due to the vagolyticlike action of the pancuronium and an increase in $PaCO_2$ to 45 to 50 torr. Fentanyl alone will usually produce a slight slowing or no change in

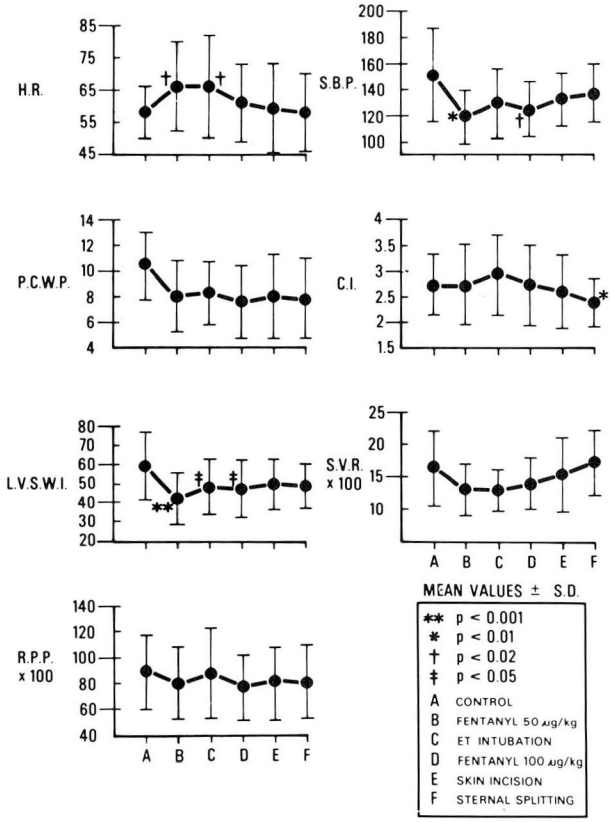

Fig. 3. Mean hemodynamic data for ten patients anesthetized with oxygen and high-dose fentanyl (100 µg/kg).

HR.[13] There was a 21% fall in SBP (119 ± 21 torr) and a 28% fall in LVSWI (41.7 ± 13.1 g M.M). Intubation did not produce hemodynamic changes. There was no significant change in the hemodynamic parameters measured following the infusion of a further 50 µg/kg fentanyl and skin incision. By the time of sternal splitting, the CI had fallen to 2.37 ± 0.46 1/min/m^2, which was 13% below the control value, and a 21% decline from the highest value that was obtained at the time of intubation.

The RPP is accepted as an index of myocardial oxygen consumption. Throughout the duration of the study, the mean RPP showed no significant change from control. The changes in HR and SBP associated with the induction of anesthesia may be considered unimportant insomuch as the corresponding RPP remained unchanged. Laryngoscopy and intubation classically produce an increase in HR, SBP, SVR, and CI. Such changes were minimal in the present study. One patient in the study came to the operating room with a blood pressure of 210/105 torr. By the time the Swan-Ganz catheter was in place, the blood pressure was 180/100 torr, and following the administration of 50 µg/kg fentanyl she became normotensive. This one patient in part accounts for the highly significant fall in SBP on induction with fentanyl.

Muscular rigidity has been a problem

in previous reports. It was not a problem in this study because paralysis was secured as soon as the response to verbal command was inappropriate. High concentrations of fentanyl given into peripheral venous catheters have, in our experience, produced severe thrombophlebitis. This was avoided by administering the drug through the side arm of the Swan-Ganz catheter introducer.

We no longer topicalize the airway and we have not had to resort to the use of nitrous oxide or halothane as in the past to control hypertension at the time of intubation. Following sternotomy and prior to the institution of cardiopulmonary bypass, some of these patients tend to develop hypertension slowly. We have been unable to abort it with further administration of fentanyl. It can, however, be controlled by administering 50% to 60% nitrous oxide with the oxygen and, on rare occasions, halothane has been used for this purpose.

We always prepare an intravenous infusion of nitroglycerin and use it in concentrations of 160 μg/ml. This infusion can be used if at any time during the course of anesthesia ischemic changes are detected on the electrocardiogram. An amount of drug is given to revert the electrocardiogram to normal, which is always accompanied by a reduction in PCWP and central venous pressure and produces a slight reduction in systemic pressure. A reflex tachycardia usually occurs with the drug, but the RPP is almost invariably within preoperatively determined desirable values. When the HR is undesirably rapid and the blood pressure at a value deemed necessary for adequate coronary perfusion, the HR can be lowered by the judicious use of intravenous propranolol.

Before 1979 we did not use a Swan-Ganz catheter. It had been our practice to insert a pulmonary artery line or left atrial line to monitor left ventricular function when the patient was off bypass. Prior to bypass, we relied on SBP, HR, central venous pressure, and the electrocardiogram as indices of myocardial performance and the balance between oxygen supply and demands of the myocardium. It has been shown that patients with an ejection fraction above 0.5 and normal lungs who undergo aortocoronary bypass surgery will have their central venous pressure correlate well with left atrial pressure.[15] It has also been shown that following bypass individualized filling pressures must be used for each ventricle irrespective of the presence or absence of lung disease.[16] At present, although we feel that most patients can be safely anesthetized through the critical period from induction of anesthesia to the beginning of bypass without monitoring data provided by a Swan-Ganz catheter, we recognize that there are some patients for whom these data are of great value in fine tuning pharmacologic interventions. It seems reasonable that these catheters be introduced prior to the induction of anesthesia as left- and right-sided filling pressures and essential for the proper monitoring of postpump myocardial function and these data can be obtained, in our experience, easily and safely in the preinduction phase by the insertion of a Swan-Ganz catheter. The information provided by this catheter is mandatory in patients who preoperatively have been shown to have myocardial dysfunction.

Summary

It is not within the scope of this paper to deal with all the problems that may be encountered during cardiopulmonary bypass or to discuss the pharma-

cologic and assist devices that may be required to wean a patient from bypass. However, this is not to imply that these phases in the total operative course are not as equally important as the prebypass phase that we have emphasized.

We believe that morbidity and mortality in aortocoronary bypass surgery will be minimal if three important goals are achieved. First, the patient should be anesthetized and brought to that stage of the operation where cardiopulmonary bypass is instituted without upsetting the delicate balance between the oxygen supply and demands of the myocardium. The oxygen high-dose fentanyl technique described accomplishes this remarkably well. Second, the myocardium is properly protected during cardiopulmonary bypass and this, in our experience, has been accomplished by the use of a cardioplegic technique. Third, the techniques and drugs used in the perioperative period should be kept to the minimum that will achieve a safe and successful operation, for each intervention may create more problems than it cures.

References

1. Hillis LD, Braunwald E. Myocardial ischemia (first of three parts). N Engl J Med 1977; **296:** 971–8.
2. Hillis LD, Braunwald E. Myocardial ischemia (second of three parts). N Engl J Med 1977; **296:** 1034–7.
3. Hillis LD, Braunwald E. Myocardial ischemia (third of three parts). N Engl J Med 1977; **296:** 1093–6.
4. Tarhan S, White RD, Moffitt EA. Anesthesia and postoperative care for cardiac operations. Ann Thorac Surg 1977; **23:** 173–93.
5. Lappas DG, Powell WM Jr, Daggett WM. Cardiac dysfunction in the perioperative period: pathophysiology, diagnosis, and treatment. Anesthesiology 1977; **47:** 117–37.
6. Wynands JE, Sheridan CA, Batra MS, Palmer WH, Shanks J. Coronary artery disease. Anesthesiology 1970; **33:** 260–81.
7. Kirklin JW, Conti VR, Blackstone EH. Prevention of myocardial damage during cardiac operations. N Engl J Med 1979; **301:** 135–41.
8. Braunwald E. Determinants and assessment of cardiac function. N Engl J Med 1977; **296:** 88–9.
9. Slogoff S, Keats AS, Ott E. Preoperative propranolol therapy and aortocoronary bypass operation. JAMA 1978; **240:** 1487–90.
10. Bland JH, Lowenstein E. Halothane-induced decrease in experimental myocardial ischemia in the non-failing canine heart. Anesthesiology 1976; **45:** 287–93.
11. Kopriva CJ, Brown AC, Pappas G. Hemodynamics during general anesthesia in patients receiving propranolol. Anesthesiology 1978; **48:** 28–33.
12. Robinson BF. Relation of heart rate and systolic blood pressure to the onset of pain in angina pectoris. Circulation 1967; **35:** 1073–83.
13. Lunn JK, Stanley TH, Eisele J, Webster J, Woodward A. High dose fentanyl anesthesia for coronary artery surgery: Plasma fentanyl concentrations and influence of nitrous oxide on cardiovascular responses. Anesth Analg 1979; **58:** 390–5.
14. Roy WL, Edelist G, Gilbert B. Myocardial ischemia during non-cardiac surgical procedures in patients with coronary-artery disease. Anesthesiology 1979; **51:** 393–7.
15. Mangano DT. Pulmonary artery monitoring during cardiac surgery. Anesthesiology 1979; **51:** S162.
16. Byrick RJ, Noble WH. Influence of elevated pulmonary vascular resistance on the relationship between central venous pressure and pulmonary artery occluded pressure following cardiopulmonary bypass. Can Anesth Soc J 1978; **25:** 106–12.