

Postoperative care

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The general principles of postoperative care applicable to patients having coronary artery bypass are identical to those for any major surgical procedure and will not be reviewed here. The special considerations regarding circulatory support using intra-aortic balloon counterpulsation and those regarding mechanical ventilation are reviewed elsewhere in this symposium. Dr. Richard O. Russell, Jr. will emphasize hemodynamic monitoring and principles of cardiovascular management in this section of the program.

I shall review two topics of controversy in postoperative management: (1) the use of vasodilator therapy (problem of postoperative hypertension), and (2) the use of anticoagulants.

Vasodilator therapy

Hypertension occurring early in the postoperative period has been reported in approximately one third of patients following coronary artery bypass. This incidence is significantly higher than that in patients undergoing cardiac valve replacement or major noncardiac surgery. Hypertension is usually observed during the first 6 hours postoperatively and is defined as blood pressures exceeding 90 to 100 mm Hg diastolic and/or 140 to 160 mm Hg systolic.

The cause of this phenomenon is unknown. We showed that hypertension in this context is due to increased peripheral vascular resistance and not to increased cardiac output. Our preliminary studies indicate no consistent elevation of serum catecholamines during the hypertensive period compared to resting levels. Others have suggested increased plasma catecholamines or angiotensin II as contributory factors. Hypertension in the early postoperative period does *not* appear to be related to (1) preoperative blood pressure, (2) preoperative circulatory indices measured during cardiac catheterization, such as blood pressure, pulse rate, cardiac output, or left ventricular ejection fraction, (3) anesthetic agent (may occur with either inhalation or intravenous agent), (4) hypervolemia, or (5) obvious factors, such as shivering, hypoventilation, inadequate analgesia, convulsions, consciousness of the patient, use of hypertensive agents, thyrotoxicosis, or latent pheochromocytoma.

It is tempting to speculate that the high incidence of early postoperative hypertension is due to an exaggerated peripheral vasomotor response in patients with coronary artery disease. This is suggested by the observation that patients having Vineberg procedures manifest early postoperative hypertension with similar frequency. Also the fact that patients undergoing other types of cardiac surgery and who are exposed to median sternotomy, pericardiotomy, cardiac manipulation, hemodilution, and hypothermia fail to sustain a similarly high incidence of hypertension suggests that direct stimulation of cardiac reflexes and manipulations associated with extracorporeal circu-

lation are not primary causative factors.

The practical significance for postoperative management is that hypertension occurring early in the postoperative period may lead to (1) excessive hemorrhage, (2) myocardial ischemia, or (3) low cardiac output. Excessive bleeding and even suture line disruption have been reported coincident with hypertension. Subendocardial ischemia or ischemia in regions not able to be revascularized may result during hypertensive periods. Our studies showed the ratio diastolic pressure-time index/systemic pressure-time index (DPTI/SPTI), an indirect measurement of subendocardial oxygenation, was depressed in hypertensive patients and rose to acceptable levels after treatment. Numerous studies have shown the utility of vasodilator therapy in reducing afterload and thereby improving left ventricular performance with improvement in stroke work index and cardiac index.

Stinson and coworkers found sodium nitroprusside to have the best hemodynamic action used as a vasodilator to reduce afterload in patients after cardiopulmonary bypass compared to trimethaphan, nitroglycerin, and chlorpromazine.

We recommend short-acting, intravenously administered vasodilator therapy for early postoperative hypertension. We give sodium nitroprusside, 30 to 50 $\mu\text{g}/\text{min}$, as an average beginning dose in adult patients. In order to give this therapy safely, close observation is required with continuous intraarterial and preferably left atrial or pulmonary capillary wedge monitoring. Hypotension may occur, requiring immediate cessation of vasodilator ther-

apy, fluid administration, and even inotropic agents. If hypertension persists beyond 12 to 24 hours postoperatively, long-acting, orally administered antihypertensive medication is begun.

Anticoagulants

At present, there is no agreement about whether patients undergoing coronary artery bypass should receive anticoagulants postoperatively. There are two theoretical reasons for giving anticoagulants postoperatively: (1) to prevent thromboembolic complications, and (2) to improve graft patency.

Clinically detectable pulmonary embolization is uncommon following coronary artery bypass. Selected patients, such as those who are sedentary or obese, those with a history of thrombophlebitis or pulmonary embolization, those immobilized in the coronary care unit prior to surgery, and those shown to be hypercoagulable, may benefit from anticoagulant therapy. Several studies appear to confirm the efficacy of small-dose heparin in reducing the incidence of postoperative thrombophlebitis and pulmonary embolism without major bleeding complications. Heparin, 5000 to 6000 units given subcutaneously at 8-hour intervals beginning at least 2 hours preoperatively, is recommended.

Factors contributing to graft closure are reviewed elsewhere in this symposium. Whereas early graft patency is determined by technical factors and distal runoff in most cases, some patients appear to sustain graft closure because of hypercoagulable states. Retrospective studies have documented a higher incidence of hypercoagulability in patients with

one or more grafts occluded compared to that in patients with patent grafts. For example, Steele and coworkers found 95% (19/20) of patients with one or more grafts closed had shortened platelet survival time compared to 30% (5/15) in patients with all grafts open. Similarly, Zajtchuk and coworkers found hypercoagulability in 77% (23/30) of patients with all grafts closed compared to 0% (0/11) in patients with all grafts open.

These studies led to a prospective study by Zajtchuk et al, who have kindly allowed us their data for this presentation. One hundred patients were screened for hypercoagulability preoperatively and on the 3rd, 7th, 10th, 14th and 21st days postoperatively. One hundred patients not screened for hypercoagulability and operated on within this period comprised the control group. The coagulation parameters tested included hematocrit, prothrombin time, activated partial thromboplastin time, fibrinogen, platelet count, platelet adhesiveness, factor VIII, thrombin generation index, and antithrombin-III activity. Patients with values of three standard deviations from control were considered hypercoagulable. Patients found to be hypercoagulable were treated with heparin, aspirin, and Coumadin. When the prothrombin time became twice control values, heparin and aspirin were discontinued. If the abnormality was present preoperatively, treatment was continued for the duration of the patient's life. Those patients who developed abnormalities postoperatively were given anticoagulants until cardiac catheterization 6 months following operation.

Fifteen patients were found to be

hypercoagulable preoperatively. Their abnormality was low anti-thrombin-III activity. Sixty-one patients became hypercoagulable within the first 2 weeks postoperatively. Predominant abnormalities in this group were increased thrombin generation and increased platelet adhesiveness. Twenty-four patients had normal coagulation studies. Comparison with the control group revealed (1) decrease in the incidence of pulmonary embolism from 8% to 0%, (2) increase in vein graft patency from 70% to 89%, and (3) elimination of anticoagulant therapy in 24%.

Based on these data the authors recommend determination of anti-thrombin-III activity, thrombin generation and platelet adhesiveness preoperatively and on the third, seventh and 14th days postoperatively. Patients with preoperative abnormalities should be treated with Coumadin indefinitely, and those developing abnormalities postoperatively for only a limited period.

This study provides objective data for selective use of anticoagulation in patients undergoing myocardial revascularization. If these data are supported by other studies, it would appear that a combination of clinical judgment and a few tests for hypercoagulability will allow the clinician to prescribe anticoagulants selectively with reduction in thromboembolic complications and with improved graft patency.

Bibliography

Vasodilator therapy

1. Hoar PF, Hickey RF, Ulliyot DJ: Systemic hypertension following myocardial revascularization. *J Thorac Cardiovasc Surg* **71**: 859-864, 1976.

2. Estafanous FG, Tarazi RC, Viljoen JF, et al: Systemic hypertension following myocardial revascularization. *Am Heart J* **85**: 732-738, 1973.
3. Hanson EL, Kane PB, Askanazi J, et al: Comparison of patients with coronary artery or valve disease; intraoperative differences in blood volume and observations of vasomotor response. *Ann Thorac Surg* **22**: 343-346, 1976.
4. Stinson EB, Holloway EL, Derby G, et al: Comparative hemodynamic responses to chlorpromazine, nitroprusside, nitroglycerin, and trimethaphan immediately after open-heart operations. *Circulation* **51**, **52** (Suppl 1): 26-33, 1975.
5. Taylor KM, Morton I, Brown JJ, et al: Hypertension and the renin-angiotensin system following open heart surgery. American Association for Thoracic Surgery, 57th Annual Meeting, April 1977, Toronto, Ontario, Canada.
6. Roberts AJ, Herman SD, Abel RM, et al: Prospective analysis and treatment of perioperative hypertension related to coronary artery surgery. American Association for Thoracic Surgery, 57th Annual Meeting, April 1977, Toronto, Ontario, Canada.

Anticoagulant therapy

1. Sagar S, Nairn D, Stamatakis JD, et al: Efficacy of low-dose heparin in prevention of extensive deep vein thrombosis in patients undergoing total-hip replacement. *Lancet* **1**: 1151-1154, 1976.
2. Steele P, Battock D, Pappas G, et al: Correlation of platelet survival time with occlusion of saphenous vein aorto-coronary bypass grafts. *Circulation* **53**: 685-687, 1976.
3. Zajtchuk R, Collins GJ, Holley PW, et al: Coagulation factors influencing thrombosis of aortocoronary bypass grafts. *J Thorac Cardiovasc Surg* **73**: 309-311, 1977.
4. Zajtchuk R, Collins GJ, Schuchmann GF, et al: Coagulation abnormalities in patients undergoing myocardial revascularization. *J Thorac Cardiovasc Surg* **75**: 168-170, 1978.