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 circulation 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 μg/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. Hypertension 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 thromboembolism 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 pa-
tients became hypercoagulable within
the first 2 weeks postoperatively. Pre-
dominant abnormalities in this group
were increased thrombin generation
and increased platelet adhesiveness.
Twenty-four patients had normal co-
agulation studies. Comparison with
the control group revealed (1) de-
crease in the incidence of pulmonary
embolism from 8% to 0%, (2) increase
in vein graft patency from 70% to
89%, and (3) elimination of anticoag-
ulant therapy in 24%.

Based on these data the authors
recommend determination of anti-
thrombin-III activity, thrombin gen-
eration and platelet adhesiveness
preoperatively and on the third, sev-
enth and 14th days postoperatively.
Patients with preoperative abnor-
malities should be treated with Couma-
din indefinitely, and those develop-
ing abnormalities postoperatively for
only a limited period.

This study provides objective data
for selective use of anticoagulation in
patients undergoing myocardial re-
vascularization. If these data are sup-
ported by other studies, it would ap-
pear that a combination of clinical
judgment and a few tests for hyper-
coagulability 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, Ullyot DJ: Systemic
hypertension following myocardial revas-
cularization. J Thorac Cardiovasc Surg 71:

2. Estafanos FG, Tarazi RC, Viljoen JF, et
al: Systemic hypertension following my-
ocardial revascularization. Am Heart J 85:

3. Hanson EL, Kane PB, Askanazi J, et al:
Comparison of patients with coronary ar-
tery or valve disease; intraoperative differ-
ences in blood volume and observations of
vasomotor response. Ann Thorac Surg 22:

Comparative hemodynamic responses to
chlorpromazine, nitroprusside, nitroglyc-
erin, and trimethaphan immediately after
open-heart operations. Circulation 51, 52

Hypertension and the renin-angiotensin
system following open heart surgery.
American Association for Thoracic Sur-
gery, 57th Annual Meeting, April 1977,
Toronto, Ontario, Canada.

6. Roberts AJ, Herman SD, Abel RM, et al:
Prospective analysis and treatment of per-
ioperative hypertension related to coro-
nary artery surgery. American Association
for Thoracic Surgery, 57th Annual Meet-
ing, April 1977, Toronto, Ontario, Can-
da.

Anticoagulant therapy

1. Sagar S, Nairn D, Stamatakis JD, et al:
Efficacy of low-dose heparin in prevention
of extensive deep vein thrombosis in pa-
tients undergoing total-hip replacement.

2. Steele P, Battock D, Pappas G, et al: Cor-
relation of platelet survival time with occlu-
sion of saphenous vein aorto-coronary by-

Coagulation factors influencing thrombo-
sis of aortocoronary bypass grafts. J

4. Zajtchuk R, Collins GJ, Schuchmann GF,
et al: Coagulation abnormalities in patients
undergoing myocardial revascularization.
J Thorac Cardiovasc Surg 75: 168–170,
1978.