In these patients, adequate preoperative control of blood pressure represents a subgroup with significantly increased perioperative risk. Disease, congestive heart failure, or chronic renal insufficiency, increasing complications such as ischemic heart disease, cerebrovascular accident, myocardial ischemia, heart failure, or renal insufficiency. However, patients with mild to moderate hypertension and preexisting conditions such as ischemic heart disease, cerebrovascular accident, myocardial ischemia, heart failure, or renal insufficiency, do not appear to increase the risk of surgery significantly and therefore is not an absolute indication to postpone elective surgery. In normotensive persons and patients with adequately treated hypertension, anesthesia is not associated with a decrease in systemic vascular resistance. Therefore, the decrease in mean arterial pressure (MAP) is modest (25–30%). However, in patients with inadequate preoperative blood pressure control, anesthesia is associated with a concomitant decrease in systemic vascular resistance (SVR) of approximately 27%. The combined decrease in cardiac output and SVR leads to a profound decrease in MAP (45%) during anesthesia [33]. This intraoperative hypotension predisposes to myocardial ischemia, cerebrovascular accidents, and acute renal failure. Therefore, in patients with diastolic blood pressure over 110 mm Hg or those other high-risk groups, elective surgery should be postponed and blood pressure brought under control for a few weeks before surgery, if possible. Ideally, sustained adequate preoperative blood pressure control should be the goal in all hypertensive patients [34]. In patients with adequately treated hypertension, oral antihypertensive, and antianginal medications should be continued up to and including the morning of surgery, administered with small sips of water. Because hypovolemia increases the risk of intraoperative hypotension and postoperative acute renal failure, diuretics should be withheld for 1 to 2 days preoperatively except in patients with overt heart failure or fluid overload. Adequate potassium repletion should be given to correct hypokalemia well in advance of surgery. Continuation of β-blockers to within a few hours of surgery does not impair cardiac function and has been shown to decrease the risks of dysrhythmia and myocardial ischemia during surgery. In patients with complications and a history of cardiovascular disease or heart failure, or after coronary artery bypass surgery, postoperative hypertension should be managed with short-acting agents such as nitroglycerin or nitroprusside. In patients without complications, intermittent intravenous infusions of labetalol may be useful for management of mild to moderate postoperative hypertension until the preoperative oral antihypertensive agents can be resumed. Many patients with long-standing hypertension, even if severe, require much smaller doses of antihypertensive medications in the early postoperative course. Thus, the preoperative regimen should not be restarted automatically. Measurement of orthostatic blood pressures should be used as a guide to dosage adjustment during the postoperative recovery period. In most instances, the need for antihypertensive medications will gradually increase over a few days to weeks to eventually equal the preoperative requirement.
Hypertension and the Kidney

FIGURE 8-27
Hypertensive crises after coronary artery bypass surgery. Paroxysmal hypertension in the immediate postoperative period is a frequent and serious complication of cardiac surgery [35,36]. Paroxysmal hypertension is the most frequent complication of coronary artery bypass surgery, occurring in 30% to 50% of patients. It occurs just as often in normotensive patients as it does in those with a history of chronic hypertension. The increase in blood pressure usually occurs during the first 4 hours after surgery. The hypertension results from a dramatic increase in systemic vascular resistance (SVR) without a change in the cardiac output and is most commonly mediated by an increase in sympathetic tone owing to activation of pressor reflexes from the heart, great vessels, or coronary arteries. Hypervolemia, although often cited as a potential mechanism of postoperative hypertension, is a rare cause of postbypass hypertension except in patients with renal failure. In fact, increased SVR owing to marked sympathetic overreaction to volume depletion is a common, often unrecognized, cause of severe postoperative hypertension [37]. Patients with this paradoxical hypertensive response to hypovolemia are exquisitely sensitive to vasodilator therapy and may develop precipitous hypotension with even low-dose infusions of nitroglycerin or nitroprusside. Hypertension in this setting should be treated using careful volume expansion with crystalloid solutions or blood if required. Post-coronary artery bypass hypertension represents a hypertensive crisis because the heightened SVR increases the impedance to left ventricular (LV) ejection (afterload) that can result in an acute decrease in ventricular compliance with elevation of LV end-diastolic pressure (LVEDP) and acute hypertensive heart failure with pulmonary edema (Figs. 8-23 and 8-24). The increase in LVEDP also impairs subendocardial perfusion and can cause myocardial ischemia. Moreover, the elevated blood pressure increases the risk of mediastinal bleeding in these recently heparinized patients. The initial management of postbypass hypertension should focus on attempts to ameliorate reversible causes of sympathetic activation, including patient agitation on emergence from anesthesia, tracheal or nasopharyngeal irritation from the endotracheal tube, pain, hypothermia with shivering, ventilator asynchrony, hypoxia, hypercarbia, and volume depletion. If these general measures fail to control the blood pressure, further therapy should be guided by measurement of systemic hemodynamics. Intravenous nitroglycerin or nitroprusside is the drug of choice to provide a controlled decrease in SVR and blood pressure. Nitroglycerin may be the preferred drug because it dilates intracoronary collateral arteries [35,36]. Therapy with β-blockers is not indicated in this setting and may be detrimental because these drugs impair cardiac output and cause a further increase in SVR. Labetalol also has been shown to cause a significant reduction in cardiac output in postbypass hypertension. Postbypass hypertension is usually transient and resolves by 6 to 12 hours postoperatively, so that the vasodilatory therapy can be weaned. The hypertension usually does not recur after the initial episode in the immediate postoperative period.
Hypertensive Crises

Sudden increase in perfusion pressure in arteriocapillary bed that was previously protected from hypertension

Failure of autoregulation of cerebral blood flow (breakthrough of autoregulation)

Overperfusion of cerebral circulation

Vessel rupture (hemorrhage and infarction)

Hypertensive crises after carotid endarterectomy. Hypertension in the immediate postoperative period occurs in up to 60% of patients after carotid endarterectomy [38]. A history of chronic hypertension, especially if the blood pressure is poorly controlled preoperatively, dramatically increases the risk of postoperative hypertension. The mechanism of post-endarterectomy hypertension is unknown. The incidence of hypertension is the same whether or not the carotid sinus nerve is preserved. Hypertension after endarterectomy is a hypertensive crisis because it is associated with increased risk of intracerebral hemorrhage and increases the postoperative mortality rate [39]. A mechanism for the development of post-carotid endarterectomy cerebral hemorrhage owing to postoperative hypertension has been proposed. In patients with high-grade carotid artery stenosis, the distal cerebral circulation has been relatively protected from systemic hypertension. In this regard, the autoregulatory curve may be shifted to a lower threshold to compensate for reduced perfusion pressure. After repair of the obstructing lesion, a relative increase in perfusion pressure occurs in the cerebral arteriocapillary bed. In the setting of systemic hypertension the increased blood flow and perfusion pressure may overwhelm the autoregulatory mechanisms. Overperfusion and rupture may then occur, resulting in hemorrhagic infarction. Because poor preoperative blood pressure control increases the risk of postoperative hypertension, strict blood pressure control is essential before elective carotid endarterectomy. Furthermore, intra-arterial pressure should be monitored in the operating room and in the immediate postoperative period. Ideally, the patient should be awake and extubated before reaching the recovery room so that serial neurologic examinations can be performed to assess for the development of focal deficits. When the systolic blood pressure exceeds 200 mm Hg, an intravenous infusion of sodium nitroprusside should be initiated to maintain the systolic blood pressure between 160 and 200 mm Hg. The use of a short-acting parenteral agent is imperative to avoid overshoot hypotension and cerebral hypoperfusion.