Clinical Conference

Hypertension in a 74-Year-Old Man with Hydronephrosis and Coronary Disease

Principal Discussant
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Case Presentation

A 74-year-old man with bilateral iliac aneurysms had a 20- to 30-year history of hypertension, which was reasonably well controlled by medications. He had suffered two myocardial infarctions 12 and 20 years previously. Three years previously, he had undergone elective repair of an infrarenal abdominal aortic aneurysm with an aortobifemoral prosthetic graft. He had suffered several episodes of urinary tract infection in the 3 to 4 years preceding admission; 5 months before admission he was treated successfully for another urinary tract infection, but residual microscopic hematuria was noted. Results of a cystoscopy were normal, but an intravenous pyelogram demonstrated gross left hydronephrosis with bilateral iliac artery aneurysms. He was admitted for arteriography and operation.

The patient had no history of diabetes, hypercholesterolemia, cigarette smoking, or alcohol abuse. Medications included triamterene and hydrochlorothiazide (Dyazide), 1 capsule per day, α-methyldopa, 250 mg t.i.d. orally, and nadolol, 40 mg/day orally.

The patient had mild stable angina and 2- to 3-block dyspnea on exertion. He also complained of dysuria, hematuria, and twice-nightly nocturia.

Physical examination on admission revealed a well-developed man in no distress. His pulse rate was 64 beats/min, and his blood pressure was 140/86 mm Hg without postural change. Carotid pulses were normal, with a right carotid bruit. Examination of the lungs showed bilateral basilar rales. Point of maximal impulse was the sixth intercostal space at the anterior axillary line; S1 was constant, S2 was physiologically split, and S4 was present. A grade 2/6 apical systolic murmur radiating to the left sternal border was also present. The patient's abdomen was obese with a pulsatile left lower quadrant and no organomegaly. A small, firm prostate was noted, and the extremities showed no edema. Ankle pulses were present except for an absent right posterior tibial pulse.

Laboratory studies disclosed the following values: normal electrolyte levels; creatinine, 2.1 mg/dl; blood urea nitrogen, 33 mg/dl; hematocrit, 42%. Chest roentgenogram showed cardiomegaly and tortuous aorta. Electrocardiogram showed left axis deviation, left atrial abnormality, prominent lateral S waves, possible pulmonary disease, and nonspecific ST-T wave changes. Urinalysis demonstrated proteinuria (4+) and 25 to 30 red blood cells per high power field. Urine culture was negative.

Case Discussion

The patient is an elderly man with a history of longstanding hypertension well controlled on medications who also has symptomatic coronary artery disease characterized by two remote myocardial infarctions.
and stable angina pectoris. He had a series of urinary tract infections in the months before admission and on evaluation was found to have bilateral hydronephrosis secondary to iliac artery aneurysm. His case history illustrates the problems of the patient with hypertension, coronary artery disease, and peripheral vascular disease who undergoes noncardiac surgery. Further, the appearance of labile hypertension that was refractory to converting enzyme inhibition while the patient was hospitalized raises the question of the possible contribution to the patient’s hypertension of non-renin-dependent mechanisms related to his hydronephrosis.

**Etiology of the Patient’s Hypertension**

**Chronic**

Although we are given no family history, the long (20–30 yr) duration of his hypertension, the good response to empirical medical treatment, and the absence of clues to secondary etiologies provide strong evidence that this patient has underlying primary, or familial, hypertension. The status of his renal arteries at the time of admission for operation was unknown, as the angiograms that were obtained did not clearly visualize these vessels. Thus, he also may have had functionally significant renal artery stenosis. Superimposed on this problem are difficulties related to his hydronephrosis. It is possible that factors secondary to his ureteral obstruction and hydronephrosis, with concomitant renal dysfunction and activation of a variety of pressor mechanisms and/or inactivation of renal medullary depressor mechanisms, contributed to the acute exacerbations in his hypertension that were observed in the hospital following discontinuation of antihypertensive therapy.

**Acute**

The patient’s hypertension was well controlled on a modest (small doses of a thiazide diuretic and α-methyldopa plus a β-blocker) medical regimen at the time of admission, but he experienced a hypertensive episode accompanied by a fall in packed cell volume from 42% to 31% while in the hospital. The hypertension responded promptly to volume repletion and withdrawal of antihypertensive therapy and was attributed to an axillary hemorrhage following angiography. One day later angina at rest was noted with accompanying ST depression in the lateral leads; on the next day, severe hypertension developed, which was responsive to parenteral methyldopa and hydralazine, oral prazosin and nifedipine, and topical nitrates but not to captopril. Blood pressures ranged between 180/140 mm Hg and 80/50 mm Hg during the period of titration of antihypertensive therapy. There was no electrocardiographic evidence of myocardial infarction during this period. Urinalysis for vanillylmandelic acid, metanephrines, and catecholamines was performed during this period, but the results are unavailable to us.

Several possible etiologies for the acute labile hypertension present themselves, including abrupt withdrawal of antihypertensive drugs that have sympatholytic actions and a number of neurohumoral abnormalities that are secondary to ureteric obstruction. The latter include stimulation of the renin-angiotensin system, reflex activation of the sympathetic nervous system, altered production of renal prostaglandins, and deficiency of renomedullary vasodilator neutral lipids.

Rapid withdrawal of antihypertensive agents that have sympatholytic actions, including clonidine and α-methyldopa, particularly when administered in combination with β-adrenergic blocking drugs, has been shown to result in rebound hypertension associated with exaggerated sympathetic activity. The withdrawal syndrome is characterized by rapid (24–48 hr after discontinuing the drug) reversal of the antihypertensive effect with a return of blood pressure to or above pretreatment levels, frequently accompanied by symptoms and signs of catecholamine excess, including palpitations, tachycardia, sweating, anxiety, insomnia, nausea, and vomiting. The rebound hypertension is characteristically labile, and plasma norepinephrine levels and urinary catecholamine excretion are increased, mimicking pheochromocytoma. The withdrawal syndrome appears to be far more frequent (more than half of patients studied) following discontinuation of short-acting imidazoline drugs such as clonidine than after longer acting agents such as α-methyldopa. Further, rebound hypertension appears to be more common in patients treated with high doses of antihypertensive drugs for long periods and in those with higher pretreatment blood pressure levels. Nevertheless, accelerated hypertension has been reported following discontinuation of α-methyldopa in doses as small as 750 mg/day. Thus, the withdrawal syndrome may have contributed to the etiology of our patient’s acute hypertension in hospital.

The molecular mechanism of hypertension following withdrawal of centrally acting α-adrenergic agonists is uncertain, but down-regulation of adrenergic receptor density similar to that described during long-term administration of β-adrenergic agonists has been postulated. According to this hypothesis, active drug would be cleared from the circulation more rapidly than adrenergic receptor number would return to normal. Thus, in the early period after drug withdrawal, there would be a disequilibrium characterized by failure of central α-adrenergic receptors to inhibit sympathetic outflow and modulate baroreflex function normally. To date this hypothesis has not been tested with specific measurements of adrenergic receptor number and affinity following α-adrenergic agonist withdrawal.

Sudden withdrawal of β-adrenergic blocker therapy may also have contributed to the hypertension and unstable angina observed in this patient following discontinuation of his antihypertensive treatment. β-Adrenergic blocker therapy has been shown to exacerbate hypertension associated with clonidine withdrawal, presumably because blockade of peripheral vascular β-adrenergic receptors prevents catecholamine-induced vasodilatation. Further, abrupt with-
drawal of β-blocking drugs has been associated with exacerbations in severity of angina.12

Rebound hypertension and symptoms of increased sympathetic activity can be controlled by reintroducing the agent that was withdrawn or by administering the combination of a selective α1-adrenergic receptor antagonist such as prazosin, a selective β1-receptor antagonist such as atenolol, and a benzodiazepine such as chlordiazepoxide as a central anxiolytic.13 Alternatively, the combined α-adrenergic and β-adrenergic receptor antagonist labetalol can be used successfully in this situation.14 Peripheral vasodilators lower blood pressure in this syndrome but do not affect the symptoms of increased sympathetic activity. Accordingly, the antihypertensive agents that were used to treat this patient’s acute hypertensive syndrome were appropriate, although they required some time to restore his blood pressure to the normal range.

A causal relation between urinary tract obstruction and hypertension is suggested by the numerous reports of improvement in, or cure of, hypertension in patients with hydronephrosis after nephrectomy or restoration of urine flow.15–28 A variety of mechanisms have been postulated to explain this form of hypertension. Increased renin levels have been reported in the venous effluent of the obstructed kidney in patients with unilateral hydronephrosis and hypertension.15–19, 28–31 The enhancement in renin secretion during ureteral obstruction may be related to renal ischemia,22 activation of intrarenal baroreceptors secondary to compression of the renal vasculature,33 and/or activation of the macula densa.34 A functional consequence of enhanced renin production by hydronephrotic kidneys is hypertension,34–37 and there is evidence that removal of the hydronephrotic kidney or relief of the obstruction reduces renin production in patients in whom it ameliorates or cures hypertension. Weidmann et al.28 observed that when renin levels in the effluent from the hydronephrotic kidney exceeded those in the contralateral renal effluent by more than 50% (ratios of >1.5), nephrectomy or corrective surgery was followed by improvement in or cure of hypertension in 14 of 15 patients, while six patients who had renal venous renin ratios of less than 1.3 did not benefit from the operation. Elevated renal venous renin levels and favorable responses to operation occurred even in the presence of “normal” peripheral plasma renin levels.38 These “normal” circulating renin values fell to lower levels in association with a decline in blood pressure after operation. Thus, the “normal” preoperative plasma renin activity values may have been inappropriately elevated considering the sodium and volume status of the patient and the state of reactivity of the peripheral vascular bed.28–30

Further study is needed to determine the role of activation of the renin-angiotensin system in the pathogenesis of hypertension associated with unilateral urinary tract obstruction.40 Bailey et al.40 measured renal vein renins in 17 normotensive and 12 hypertensive patients with unilateral reflux nephropathy. Only three normotensive and two hypertensive patients had renal vein renin ratios greater than 1.5. Of the three normotensive patients, one had evidence from divided renal function studies to suggest functional renal ischemia. Since the surgical results were not described in this study, the usefulness of differential renal vein renin determinations as prognostic indices could not be assessed.

Further study is needed to determine the role of activation of the renin-angiotensin system in the development and maintenance of hypertension secondary to obstructive uropathy. It is likely that this varies from person to person depending on renal functional status and the contribution of secondary salt and volume overload. Failure of our patient’s hypertension to respond to captoril rules against a major role for angiotensin II in its pathogenesis.

Reflex activation of the sympathetic nervous system is another mechanism by which urinary tract obstruction could lead to hypertension. Elevated ureteral pressure enhances renal afferent nerve activity,41 with a resultant increase in efferent sympathetic nerve activity and secondary stimulation of tubular sodium reabsorption42 and vasoconstriction43 in the contralateral kidney. Denervation of either kidney abolishes the contralateral renal vasoconstriction, which confirms that it represents a renorenal reflex.44 These responses to ureteral occlusion may be mediated by mechanoreceptors in the parenchyma of the kidney44 and/or by renal chemoreceptors that are sensitive to ischemia45 or to the ionic composition of the pelvic urine.46, 47 Spinal cord transection at T-6 interrupts both afferent and efferent neural pathways to the kidneys, which indicates that the central connections for this renorenal reflex are above this level and that this response is not a local spinal reflex.43

Generalized systemic vasoconstriction with attendant hypertension has not been described following ureteral obstruction in the anesthetized experimental animal, the model from which most of the available data on renorenal reflexes were derived. In contrast, direct electrical stimulation of afferent renal nerves in the anesthetized cat consistently elicits increases in arterial pressure and heart rate48 along with enhanced sympathetic nervous system activity. Further, activation of renal chemoreceptors by infusion of adenosine into the renal artery or renal pelvis of the conscious dog has been shown to produce a potent systemic pressor response in association with an increase in afferent renal nerve activity (Figure 1).49 The pressor response to renal artery adenosine infusion was associated with increases in heart rate, pulse pressure cardiac output, and plasma norepinephrine levels50 and with a marked increase in vascular resistance in the contralateral kidney.51 Both efferent renal nerve activity and renal norepinephrine secretion were increased in the contralateral kidney during infusion of adenosine into the renal artery.49 and ganglionic blockade attenuated and renal denervation abolished the pressor response to renal artery adenosine infusion. Taken together, these data indicate that intrarenal infusion of adenosine produces a pressor response associated with increased activity of the sympathetic nervous system in conscious
Comparison of mean arterial pressure response to infusion of adenosine into the renal artery (upper panel; \( p < 0.001 \)), inferior vena cava (middle panel), and renal artery after renal denervation (lower panel) in eight conscious dogs. The horizontal bars indicate the periods of infusion. (Reproduced from Katholi et al. with permission of Gower Medical Publishing Limited.)

animals with intact renal nerves. Similarly, ureteral occlusion in conscious animals and human subjects could activate neural pathways that mediate pressor responses by increments in sympathetic outflow. Further study is needed to determine whether such neural mechanisms play an important role in the pathogenesis of the hypertension that follows ureteral occlusion in clinical situations. The lability of our patient’s acute hypertension, its failure to respond to captopril, and its apparent responsiveness to prazosin and \( \alpha \)-methyldopa favor a neurogenic mechanism, whether secondary to withdrawal of \( \alpha \)-methyldopa and \( \beta \)-blockade or to a renal reflex.

A third mechanism that has been postulated to explain hypertension secondary to urinary tract obstruction is altered production of renal prostaglandins. Enhanced prostaglandin synthesis has been demonstrated in the hydronephrotic kidney. Both prostaglandin \( \mathrm{E} \) and thromboxane \( \mathrm{A}_2 \) are released in large amounts from the isolated perfused ureteral-obstructed rabbit kidney, presumably as a consequence of increased synthesis in cortical blood vessels. The observation that imidazole (an inhibitor of thromboxane synthetase) but not indomethacin increases the glomerular filtration rate of the obstructed kidney suggests that thromboxane \( \mathrm{A}_2 \) contributes to the increased renovascular resistance seen in high-grade ureteral obstruction. In addition, renal vasodilator prostaglandins are important in maintaining the renal circulation under conditions in which renal pressor agents such as angiotensin II are released in excess. In contrast, there is little evidence that the renal prostaglandins play a major regulatory role in the extrarenal circulation and, therefore, that they contribute directly to the pathogenesis of hypertension secondary to urinary tract obstruction.

Finally, deficiency of renomedullary depressor substance(s), including the vasodilator neutral lipids, has been implicated in the pathogenesis of many forms of experimentally induced and naturally occurring hypertension, including that which appears in the setting of ureteral occlusion. The renal medulla has a depressor effect in many forms of hypertension, particularly those characterized by sodium and/or volume overloading. Transplants of renal papillae or of renomedullary interstitial cells derived from monolayer cell culture prevent or reverse many forms of experimental hypertension but do not alter the blood pressure of normal subjects. Several depressor substances isolated from the renal medulla have been implicated in this function. The best characterized of these are the antihypertensive polar and neutral renomedullary lipids (APRL and ANRL respectively), which have been extracted from fresh renal medulla by Muirhead and colleagues. While APRL is a conglomerate of 1–0 alkyl ethers of phosphatidyl choline, including mainly the C16:0, C16:1, and C18:1 ethers, ANRL has been identified as a nonpolar entity by chromatographic and solubility studies. In vitro, ANRL can be converted to APRL, while APRL causes a sudden, dose-dependent depressor response when injected as a bolus intravenously into either hypertensive or normotensive animals. In addition, APRL is a potent vasodilator when administered to either an artificially perfused resistance vascular bed or to an intact animal. The depressor response to APRL in the intact animal is accompanied by a reflex tachycardia and activation of the renal nerves. In contrast, ANRL causes a slower and more prolonged depressor response that is accompanied by slowing of the heart rate and reduced sympathetic activity. The vasodilatory effects of ANRL are evident only in the intact animal.

Both ANRL and APRL are found in high concentrations in the renal venous effluent of one-kidney, one clip and two-kidney, one clip hypertensive rats after removal of the clip. The renal venous effluent of the unclipped isolated kidney lowers blood pressure, heart rate, and sympathetic tone, a constellation of responses mimicked by intravenous infusion of purified ANRL derived from the renal papilla. Further, the renal interstitial cells of the clipped kidney have been observed to undergo degranulation during the reversal
of hypertension that follows unclipping.\textsuperscript{70} Thus, release of ANRL and APRL from the renomedullary interstitial cells has been adduced as the mechanism of the depressor, bradycardic, and sympatholytic response to unclipping of the renal artery, which suggests that these compounds are true antihypertensive hormones.\textsuperscript{66} Muirhead and Pitcock\textsuperscript{71} have proposed a scheme by which ANRL and APRL may interact with and modulate the endogenous pressor systems (Figure 2). The roles of these compounds in mediating cardiovascular homeostasis in the normotensive subject and their interactions with other cardiovascular control systems, such as the central and peripheral nervous systems, the renin-angiotensin-aldosterone system, and the excretory function of the kidney, in the pathogenesis of various forms of hypertension remain to be fully elucidated.

Ablation of the renal papillae, whether by surgical removal,\textsuperscript{5} \textsuperscript{72}-\textsuperscript{74} chemical injury induced by bromethylamine hydrobromide,\textsuperscript{75} \textsuperscript{76} genetically mediated nephropathies,\textsuperscript{77} or ischemic necrosis secondary to ureteral ligation,\textsuperscript{78} is associated with an elevation in blood pressure or an exacerbation of underlying hypertension, particularly when the subject is challenged by a sodium or volume load. Renomedullary transplantation has been shown to protect against salt-induced hypertension in rats with unilateral hereditary hydronephrosis and extensive destruction of the medulla of the affected kidney.\textsuperscript{82} In addition, ureteral ligation prevents the depressor response that is normally seen following unclipping of the renal artery in one-kidney, one clip renal hypertensive rats, which reaffirms that ureteral ligation interferes with the renopapillary antihypertensive function.\textsuperscript{80} Thus, loss of renal vasodilator lipid production secondary to his bilateral ureteral obstruction could have contributed to our patient’s acute hypertensive episode directly by removing vasodilator and sympatholytic substance(s) and indirectly by failing to oppose the enhanced vasoconstrictor function of the activated renin-angiotensin and sympathetic nervous systems.

Preoperative Evaluation

The patient had a history of chronic ischemic heart disease with two remote (12 yr and 20 yr before admission) myocardial infarctions, chronic stable angina, and an abnormal resting electrocardiogram, which showed left axis deviation, left atrial enlargement, and nonspecific ST-T wave abnormalities. In the days before operation he experienced, in conjunction with an exacerbation of his hypertension, a worsening of his angina and transient increases in ST-T wave depression in the lateral leads. He also had signs and symptoms of congestive heart failure, including a history of dyspnea on exertion, bilateral basal rales on physical examination, and cardiomegaly on both physical examination and chest roentgenogram. Evidence for extensive peripheral vascular disease includes a history of repair of an abdominal aortic aneurysm 3 years before admission, the presence of bilateral external iliac artery aneurysms, a right carotid bruit, and a tortuous aorta on chest roentgenogram, suggestive of a descending aortic aneurysm. He also had a grade 2/6 apical systolic murmur that radiated to the left sternal border and that was never formally evaluated. In addition, he had evidence of mild renal dysfunction.

This constellation of cardiac risk factors places this man at increased risk for cardiac death or serious cardiovascular complications in the postoperative period. The emergent nature of his medical problem left little choice but to move directly to operation; had the operation been elective, a more thorough evaluation of the extent of his coronary and cerebrovascular disease might have been indicated to more precisely assess the risk of iliac artery surgery and possibly to repair major coronary and extracranial cerebrovascular lesions before operation.

The advantages of aggressive and invasive evaluation of patients with clinical evidence of coronary or extracranial cerebrovascular disease, or both, before noncardiac surgery are illustrated in a recent report.\textsuperscript{79} Of 100 successive patients with atherosclerotic renovascular disease who underwent revascularization at the Cleveland Clinic, 39 had clinical evidence of coronary artery disease and 19 had evidence of extracranial cerebrovascular disease. These patients were studied angiographically, and coronary artery bypass grafting (14 patients) or carotid endarterectomy (11 patients) was done before renal revascularization when high grade lesions were found. There were only two (2%) postoperative deaths in this series, only one of which was attributed to a cardiovascular cause. This result contrasts with a much higher (9.3\%) in the Cooperative Study of Renovascular Hypertension\textsuperscript{80} surgical mortality rate in patients with atherosclerotic renal artery stenosis in most centers, where preoperative evaluation has been less rigorous and preoperative coronary and carotid revascularization procedures usually have not been undertaken.
Experience from other series also suggests that the risk of noncardiac surgery is reduced in coronary patients who have undergone successful bypass grafting. For example, in one series of 358 patients who underwent major noncardiac surgery following successful coronary artery bypass grafting, the surgical mortality rate was 1.1% (4 deaths), which is comparable to that in patients without coronary disease. This finding was true whether the operations were staged, in which case the noncardiac surgery was scheduled for 6 to 12 weeks after bypass, or unplanned and performed either emergently or electively because of the appearance of new lesions. In the subset of 49 patients who underwent resection of abdominal aortic aneurysms following coronary artery bypass grafting, all survived, in contrast to a predicted mortality rate of 12% for this operation in patients without coronary artery disease. These reports, though provocative, are uncontrolled retrospective studies in which no attempt was made to stratify the coronary disease patients into various levels of risk.

The most comprehensive and rigorous assessment of cardiac risk factors in noncardiac surgery yet undertaken is a prospective study carried out by a group of senior medical residents at the Massachusetts General Hospital. Between October 1975 and April 1976, 1001 consecutive patients over the age of 40 who were scheduled to undergo major surgery were entered into the study. Each patient was seen preoperatively (or, in the unusual instance of those undergoing emergency surgery, early in the postoperative period) by one of the collaborating investigators, and a history and physical examination oriented toward the cardiovascular system were obtained. Postoperatively, all patients were followed up by one of the collaborating investigators and the occurrence of cardiovascular events was carefully documented.

There were 19 (1.9% of all patients) deaths and 18 (1.8% of all patients) diagnosed postoperative myocardial infarctions, five of which were fatal, in this group. Fourteen patients died from cardiac causes without documented myocardial infarction: five had sudden cardiac deaths, probably due to arrhythmia, and nine died with refractory cardiogenic shock. Of the 19 patients with postoperative cardiac deaths, 11 had documented ischemic heart disease manifested by angina or a history of myocardial infarction, seven had documented congestive heart failure, and six had arrhythmias. The remaining three patients who died of cardiac causes were elderly and had other evidence of major heart disease. Pulmonary edema developed in 36 patients, and 12 had documented ventricular tachycardia postoperatively. Overall, 39 patients experienced one or more nonfatal but life-threatening postoperative cardiac complications (3.9% of the series).

Half of the postoperative infarctions were associated with pain; the other half were painless but were diagnosed on the basis of new or worsened cardiac signs and symptoms, such as congestive heart failure (5 patients), hypotension (3 patients), and supraventricular tachycardia (1 patient). Only two of the infarctions occurred intraoperatively; six more occurred within the first 3 postoperative days, and the remainder occurred by postoperative Day 6. In addition, transient postoperative ST-T wave changes suggestive of ischemia but unaccompanied by other evidence of infarction developed in 51 patients. Of all the signs and symptoms of heart disease, only five were significantly correlated with intraoperative or postoperative myocardial infarction: age greater than 70 years, symptoms of congestive heart failure (dyspnea, orthopnea, or edema), grade 2/6 or louder murmur of mitral regurgitation, more than 5 premature ventricular contractions per minute documented at any time preoperatively, and a tortuous or calcified aorta on chest roentgenogram.

In contrast, nearly every sign and symptom of ischemic heart disease had a significant correlation with postoperative cardiac death by univariate analyses. Table 1 summarizes the risk ratios for postoperative cardiac death (ratio between percent of patients who had the preoperative variable dying of cardiac causes to the percent of patients who did not have the preoperative variable who died of cardiac causes) for a large number of preoperative variables related to underlying cardiovascular disease.

Surprisingly, a number of cardiovascular abnormalities, including hypertension (past or present, treated or untreated), angina (not classified into stable or unstable, resting or exertional, or graded with respect to severity in this study), S4 gallops, systolic ejection...
murmurs without accompanying evidence of marked aortic stenosis, aortic abnormalities noted on roentgenogram, and evidence of atherosclerotic vascular disease were not associated with increased risk for postoperative cardiac death. Similarly, hyperlipidemia, diabetes mellitus, smoking, and obesity were not significant risk factors for postoperative cardiac death. Although preoperative hypertension was not an independent predictor of cardiac death or morbidity due to postoperative myocardial infarction, congestive heart failure, or arrhythmia, hypertensive patients were more likely to experience intraoperative or postoperative hypertension than were normotensive patients, whether or not they were treated and whether or not their hypertension was controlled at the time of operation. Most of the episodes of postoperative hypertension occurred in patients who underwent vascular surgery.

Multivariate regression analysis identified nine factors that have statistically significant independent correlations with postoperative cardiac death (Table 2). Evidence of uncompensated congestive heart failure (S3 or jugular venous distention) at the time of preoperative evaluation had the strongest predictive value for postoperative cardiac death. Accordingly, elective noncardiac surgery should be delayed in patients with these findings until intensive treatment for congestive heart failure can be administered and their condition stabilizes. Previous myocardial infarction had prognostic significance only if it occurred within 6 months before operation. In the Massachusetts General Hospital series, postoperative cardiac death occurred in 5 of 22 patients (22.7%) who had an infarction within 6 months preoperatively but in only 2 of 79 patients (2.5%) whose prior infarction was greater than 6 months before operation. In the latter group, the risk of cardiac death was no higher than in those who had never had an infarction (12 of 894 patients; 1.3%). Further, the risk of postoperative cardiac death at 1 to 6 months after an infarction was not significantly lower than at less than 4 weeks. An even greater (38%) risk of cardiac death or recurrent infarction following noncardiac surgery in patients operated on less than 6 months after a myocardial infarction was found by Goldman et al. on reviewing seven reported series that included 151 cases of postoperative cardiac death or recurrent infarction. Accordingly, it is recommended that, wherever possible, noncardiac surgery be deferred for at least 6 months following myocardial infarction.

Of the nine preoperative factors independently related to postoperative cardiac death or life-threatening cardiac complications, our patient had two: aortic (or near aortic) operations and age greater than 70 years. His renal failure was not quite severe enough to satisfy criteria for "poor general medical condition," and his operation, while not entirely elective, was not a true emergency. According to the cardiac risk index of Goldman et al. (Table 3), he would have 8 points, which would put him in class II and give him a 2% probability of postoperative cardiac death and a 5% probability of cardiac complications that are life-threatening.

Although a detailed discussion of his intraoperative management is beyond the scope of this article, it is worth commenting on the choice of anesthetics. Spinal anesthesia was selected for our patient, presumably because of his prior history of congestive heart failure, and turned out to be a good choice, as his operation was uncomplicated by either fluctuations in blood pressure or worsening of his heart failure. In the Massachusetts General Hospital experience general anesthesia was associated with a 4.3% incidence of development of congestive heart failure de novo and a 22% incidence of worsening of heart failure in patients who had a previous history; neither problem was seen with spinal anesthesia. The reason for this difference is not fully explained, but it may relate to the myocardial depressant effects of volatile anesthetics such as halothane, enflurane, methoxyflurane, and nitrous oxide and to the tendency of general anesthesia to be used in larger operations. Spinal anesthesia does not reduce the probability of developing intraoperative hypotension, intraoperative or postoperative arrhythmias, or postoperative cardiac death, however. Further, although spinal anesthesia does not depress the myocardium, it must be used with caution because of its tendency to cause hypotension secondary to venodilation and decreased afterload.

In summary, this elderly man had long-standing essential hypertension complicated by coronary, extracranial cerebrovascular, and peripheral vascular dis-
HYPERTENSION, HYDRONEPHROSIS, AND CORONARY DISEASE

TABLE 3. Computation of the Cardiac Risk Index

<table>
<thead>
<tr>
<th>Criteria</th>
<th>Multivariate discriminant-function coefficient</th>
<th>Points</th>
</tr>
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<tbody>
<tr>
<td>1. History</td>
<td></td>
<td></td>
</tr>
<tr>
<td>a. Age &gt; 70 yr</td>
<td>0.191</td>
<td>5</td>
</tr>
<tr>
<td>b. MI in previous 6 mo</td>
<td>0.384</td>
<td>10</td>
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<tr>
<td>2. Physical examination</td>
<td></td>
<td></td>
</tr>
<tr>
<td>a. S3 gallop or JVD</td>
<td>0.451</td>
<td>11</td>
</tr>
<tr>
<td>b. Important VAS</td>
<td>0.119</td>
<td>3</td>
</tr>
<tr>
<td>3. Electrocardiogram</td>
<td></td>
<td></td>
</tr>
<tr>
<td>a. Rhythm other than sinus or PACs on last preoperative ECG</td>
<td>0.283</td>
<td>7</td>
</tr>
<tr>
<td>b. PVCs &gt; 5/min documented at any time before operation</td>
<td>0.278</td>
<td>7</td>
</tr>
<tr>
<td>4. General status</td>
<td></td>
<td></td>
</tr>
<tr>
<td>PO2 &lt; 60 or PCO2 &gt; 50 mm Hg; K+ &lt; 3.0 mEq/L or HCO3 &lt; 20 mEq/L, BUN &gt; 50 mg/dl or Cr &gt; 3.0 mg/dl; abnormal SGOT; signs of chronic liver disease; or patient bedridden from noncardiac causes</td>
<td>0.132</td>
<td>3</td>
</tr>
<tr>
<td>5. Operation</td>
<td></td>
<td></td>
</tr>
<tr>
<td>a. Intrapertitoneal, intrathoracic, or aortic operation</td>
<td>0.123</td>
<td>3</td>
</tr>
<tr>
<td>b. Emergency operation</td>
<td>0.167</td>
<td>4</td>
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<tr>
<td>Total possible</td>
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Class | Point total |
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<tbody>
<tr>
<td>I (n = 537)</td>
<td>0–5</td>
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<tr>
<td>II (n = 316)</td>
<td>6–12</td>
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<tr>
<td>III (n = 130)</td>
<td>13–25</td>
</tr>
<tr>
<td>IV (n = 18)</td>
<td>&gt; 26</td>
</tr>
</tbody>
</table>

Numbers in parentheses indicate percentages.

MI = myocardial infarction; JVD = jugular venous distention; VAS = valvular aortic stenosis; PACs = premature atrial contractions; ECG = electrocardiogram; PVCs = premature ventricular contractions; Po2 = partial pressure of oxygen; PCO2 = partial pressure of carbon dioxide; BUN = blood urea nitrogen; Cr = creatinine; SGOT = serum glutamic-oxalacetic transaminase.

*Documented intraoperative or postoperative myocardial infarction, pulmonary edema, or ventricular tachycardia without progression to cardiac death.

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References

HYPERTENSION, HYDRONEPHROSIS, AND CORONARY DISEASE/Oparil


Hypertension in a 74-year-old man with hydronephrosis and coronary disease.
S Oparil

Hypertension. 1985;7:824-833
doi: 10.1161/01.HYP.7.5.824

Hypertension is published by the American Heart Association, 7272 Greenville Avenue, Dallas, TX 75231
Copyright © 1985 American Heart Association, Inc. All rights reserved.
Print ISSN: 0194-911X. Online ISSN: 1524-4563

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