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Vascular Medicine

Periprocedural Hypertension: Current Concepts in Management for the Vascular Surgeon

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Periprocedural hypertension is a common finding in patients undergoing vascular surgery or endovascular procedures, and this may pose a risk for subsequent cardiovascular morbidity or mortality. Accordingly, the vascular surgeon who wishes to improve outcomes needs to be proficient not only in surgical technique but also in the medical management of the patient's associated conditions, especially hypertension. Vascular procedures need not be cancelled unless the blood pressure (BP) is more than 180 mm Hg systolic or 110 mm Hg diastolic, but attention should also be paid to evidence of end organ damage in making this decision. In most cases preoperative antihypertensive medications should be continued up till the procedure. Postoperative hypertension may require 1 of a number of intravenous medications, which are listed. Oral nifedipine should generally be avoided for fear of inducing an uncontrolled hypotensive response and cardiac ischemia.

Introduction

Periprocedural hypertension is unfortunately common and may be a significant risk factor for postprocedural mortality, especially in the vascular patient who very often has significant cardiovascular pathology. In 1 study of 76 patients who

died of a cardiovascular cause within 30 days of elective surgery, a preoperative history of hypertension was 4 times more likely than among 76 matched controls.¹ However, this increase in mortality rate seems to occur only when the diastolic blood pressure is > 110 mm Hg² or when hypertension has caused end-organ damage.³ This morbid effect was confirmed by Towne et al,⁴ who demonstrated that systolic hypertension > 200 mm Hg before carotid endarterectomy was associated with an increase in postoperative cerebrovascular complications. Furthermore, blood pressure (BP) and heart rate can increase significantly with surgery, and this is even more prone to occur in hypertensive patients. Also, labile hypertension with periods of hypotension is also more commonly seen in these patients.⁵ Accordingly, the vascular surgeon who wishes to im-

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prove outcomes needs to be proficient not only in surgical technique but also in the medical management of the patients' associated conditions, especially hypertension.

Preprocedural Management

Preprocedural antihypertensive medications should be maintained until and including the day of surgery. Since many patients will be on diuretics, preprocedural volume blood urea nitrogen, creatinine, and potassium levels should be checked. There are some theoretical reasons to possibly withhold angiotensin-converting enzyme inhibitors (ACE), angiotensin receptor blockers (ARBs), and calcium channel blockers the day before surgery, but this has not been generally accepted. The former can blunt the compensatory activation of the renin-angiotensin system during surgery resulting in prolonged hypotension. This has been supported by a study of 150 patients undergoing vascular surgery that showed that hypotension during induction was noted less frequently when these medications were stopped the evening before surgery.⁶ These data would support discontinuing those medications if the indication for their use is congestive heart failure and not hypertension. Calcium channel blockers can increase postoperative bleeding⁷ owing to platelet inhibition. However, abrupt withdrawal can cause coronary vasospasm, and so it is not recommended that they be discontinued.

Acute withdrawal is also especially dangerous for beta-blockers and the centrally acting sympatholytic drugs.^{8,9} Further, beta-blockers in the perioperative period decrease mortality in high-risk cardiac patients¹⁰ and should be considered for all patients undergoing major vascular surgery.

Postoperative and Intraoperative Hypertension and Its Management

Hypertension is most likely to develop during induction of anesthesia and in the immediate recovery period. Laryngoscopy and intubation can cause a blood pressure elevation of 30 mm Hg in normotensive patients and 90 mm Hg in hypertensive patients.⁴

Postoperative hypertension is more common in patients who are hypertensive preoperatively

and is especially common in patients undergoing vascular procedures. Goldman et al³ reported that postoperative hypertension occurred in 57% of abdominal aortic aneurysm resections and 29% of other vascular procedures compared to 8% of other nonvascular procedures. Hypertension is also a potentially serious complication following carotid endarterectomy.⁴ Paradoxically, some patients may experience normalization of their blood pressure for many months after major surgery, although this seldom persists.¹¹

Before starting a patient on antihypertensive medications, other causes of hypertension specific to the postoperative period (pain, agitation, hypoxia and hypercarbia, bladder distension and hypervolemia) should all be addressed. Often adequate analgesia may be all that is necessary. In many patients blood pressure elevation will be transient and accordingly they will not require aggressive antihypertensive regimens that may cause adverse side effects. If the patient remains hypertensive and unable to take their customary oral agents, they should be started on a parenteral regimen described below (sublingual nifedipine should never be used since it can cause severe hypotension and cardiac ischemia¹²).

There are, however, major management issues that should be considered no matter what drugs are ultimately selected; and these relate to prior therapies, preexisting conditions and specific ailments. For example, although reducing blood pressure is advantageous in preventing stroke, hypertension following a thromboembolic stroke, eg, after carotid endarterectomy, is thought to be cerebro-protective by maintaining flow to hypoperfused areas.¹³ In fact, elevation in blood pressure is often noted for the first 10 days after such a stroke. Because of these findings, it is usually recommended that antihypertensive therapy be stopped after a thrombotic stroke unless the patient has cardiac failure, aortic dissection or diastolic blood pressure of > 120 mm Hg or systolic pressure of > 220 mm Hg¹³⁻¹⁵ Under such circumstances, labetalol may be preferred since it can be adjusted readily. Nitroprusside should be carefully used since it can increase intracranial pressure. Also, patients with chronic hypertension often have autoregulation of blood pressure shifted to a higher range, so reductions to "normal" blood pressure may result in hypoperfusion. This is most commonly seen in the elderly. Further, patients on longer-acting medications may have blockade of their compensatory mechanisms that would normally offset excessive decreases in pressure.

Medications for the Control of Intraoperative and Postoperative Hypertension

In the immediate postoperative period, especially in patients who cannot tolerate oral intake, intravenous antihypertensive medications will often be required. These may also be required during endovascular procedures. Further, if malignant hypertension is uncontrolled, hypertensive encephalopathy and hypertensive nephrosclerosis can result. As blood pressure rises, autoregulation in the arteriolar bed prevents transmission of the elevated blood pressure into the capillary bed, thus preventing endothelial destruction. Patients with chronic hypertension usually develop arteriolar hypertrophy, which minimizes pressure transmission into the capillaries. Accordingly, malignant hypertension is seen only when diastolic BP goes above 130 mm Hg. However, encephalopathy could occur in previously normotensive patients if diastolic blood pressure were to rise suddenly above 100 mm Hg, or in patients in whom autoregulation may be disordered, such as diabetics.

Slow onset of action and an inability to control the degree of BP reduction have limited the use of oral antihypertensive agents in the therapy of hypertensive crises, in postoperative patients, or during endovascular procedures. They may, however, be useful when there is no rapid access to the parenteral medications described below. Both sublingual nifedipine (10 mg) and sublingual captopril (25 mg) can substantially lower the BP within 10 to 30 minutes in many patients.¹⁶ As mentioned, the major risk with these drugs is ischemic symptoms (eg, angina pectoris, myocardial infarction, or stroke) due to an excessive and uncontrolled hypotensive response.¹² Thus, their use should generally be avoided in the treatment of hypertension unless more suitable and controllable agents are not available.

Because of these considerations, parenteral medications listed below will often be necessary. For example, patients on: diuretics can receive intravenous furosemide or bumetanide; beta-blockers: propranolol, labetalol or esmolol; ACE inhibitors: enalaprilat; and calcium channel blockers nifedipine. All have a rapid onset, although nitroprusside is the most immediate. There is currently no scientific data to recommend resuming the same class of drug except in the case of beta-blockers and clonidine, which should not be stopped acutely. Accordingly, if another class of drugs is more suitable or if rapid action is re-

quired, another class of drugs satisfying these requirements can be used.

- Nitroprusside is an arteriolar and venous dilator, given as an intravenous infusion with an initial dose of 0.25 to 0.5 mg/kg per minute titrated every 1–2 minutes to a maximum dose of 8 to 10 mg/kg per minute. The maximal dose of nitroprusside should not be used for more than 10 minutes. Nitroprusside acts within seconds and has a duration of action of only 2 to 5 minutes. Accordingly, hypotension can be easily reversed by temporarily discontinuing the infusion, providing an advantage over the drugs listed below. However, prolonged use can lead to cyanide poisoning, particularly in patients with renal insufficiency. This is usually manifested as deterioration in mental condition, altered mental status, and lactic acidosis. Another disadvantage is that the medication is light sensitive. It can be used in most hypertensive emergencies and is especially suited to the postoperative patient. However, although during anesthesia it does not affect cerebral perfusion, in the unanesthetized postoperative patient it can increase intracranial pressure, causing a drop in cerebral perfusion. This could be a disadvantage in patients who have undergone carotid endarterectomy (CEA). It should also not be used without a concomitant beta-blocker in patients with aortic dissection because of its reflex stimulation of sympathetic tone, which will increase myocardial contractility.¹⁷
- Nitroglycerin is a coronary vasodilator and direct venodilator with variable arterial effects given as an infusion of 5–200 µg/minute. The usual starting dose is 5 µg/kg/minute titrated at 5 µg/kg/minute increments every 3–5 minutes. Once the dose exceeds 20 µg/kg/minute, increments can increase by > 20 µg/kg/minute. There is no absolute dosing limit, but the risk of hypotension increases with doses above 200 µg/kg/minute.¹⁷ Like Nitroprusside, it has a short duration of action (2–5 minutes) and its duration of action is 3–5 minutes. Unlike Nitroprusside, prolonged use is associated with minimal toxicity, but it can lead to methemoglobinemia and tolerance. It is especially indicated in patients with coronary ischemia and as such is a valuable medication in vascular patients who often have coexistent cardiac atherosclerosis. Headache and tachycardia are not infrequent side effects.
- Nicardipine is a short acting dihydropyridine

calcium channel blocker and acts as an arteriolar dilator. It is given as an intravenous infusion with an initial dose of 5 mg/hr and a maximum dose of 15 mg/hr with duration of action of 1–4 hours. Titration should be in 2-mg/hr increments adjusted every 15 minutes. It should be avoided in patients with acute heart failure. It also can raise intracranial pressure, so the same precautions exist for the post-CEA patient. It is light stable but has the slowest onset of the vasodilators (5–10 minutes).

- Fenoldopam is a peripheral dopamine-1 receptor agonist given as an intravenous infusion with an initial dose of 0.1 µg/kg per minute. The dose is then titrated depending upon the blood pressure response in 0.1 µg/kg/minute increments at 15 minute intervals up to a maximal dose of 1.6 µg/kg/minute.¹⁷ Its onset usually occurs within 5–15 minutes and has a short half life of 5 minutes. Like nitroprusside it can cause reflex tachycardia and so should be used with caution in patients with myocardial ischemia. It can be used in most emergencies but should be used with caution in patients with glaucoma. It has the added advantage of being somewhat renal protective and therefore may be well suited to patients who have borderline renal function, in patients undergoing renal angioplasty and/or stent placement, or in whom aortic cross clamp was above the renal arteries. Currently, it is one of the more expensive agents.
- Hydralazine is a direct arteriolar vasodilator that causes a rapid drop in blood pressure affecting diastolic more than systolic pressures. It can cause a profound increase in reflex tachycardia and therefore is not a good choice for the patient with cardiac disease or dissecting aneurysm. It also raises intracranial pressure. However, it can increase renal blood flow and so may be advantageous in patients undergoing renal angioplasty, stents or those who may have undergone aortic cross clamp. It is given as a slow intravenous bolus of 10–20 mg or intramuscular dose of 10–50 mg. Onset is the slowest of all these agents, occurring at 10–20 minutes with the intravenous route or 20–30 minutes with the intramuscular route. Duration of action is 3–8 hours making infusion difficult to manage, and so it is usually given as an original bolus and then 2–3 mg boluses as necessary.¹⁷ It is one of the least expensive agents.
- Enalaprilat is an angiotensin-converting enzyme (ACE) inhibitor given intravenously at a dose of 1.25 mg every 6 hours. There are no studies showing that higher doses are more effective, but regimens as high as 5 mg every 6 hr have been used safely. It should also be noted that ACE inhibitors are less effective in blacks and also are associated with an increased incidence of angioedema. It also has a slow onset of action at 15–30 minutes and can cause a precipitous fall in BP in high-renin states. It should be avoided in patients with an acute myocardial infarction (MI) but may be advantageous in patients with acute left ventricular failure.¹⁸ It does not cause reflex tachycardia and does not result in increased intracranial pressure.¹⁷
- Labetalol is an alpha- and beta-adrenergic blocker given as an intravenous bolus or infusion with a bolus of 20 mg initially, followed by 20 to 80 mg every 10 minutes to a total dose of 300 mg. Infusion is then maintained at 0.5 to 2 mg/minute. Onset of action occurs in 5 minutes but duration of effect can vary excessively from 20 minutes to 23 hours making it difficult to titrate as a continuous infusion. When given intravenously, the beta- to alpha-blocking ratio is 7:1.¹⁷ It should be avoided in patients with acute heart failure. This beta-blocker should be considered in patients who were on beta-blockers preoperatively but cannot take oral medications in the immediate postoperative period. It should generally be avoided, however, in patients with significant history of asthma, congestive heart failure or greater than first-degree heart block. It has a minimal effect on cerebral perfusion. Labetalol is the first line drug for the prophylaxis of surgical hypertension.
- Esmolol is an ultra-short acting (2 minutes) beta-blocker given first as a loading dose of 250–500 µg/kg/minute and then at a rate of 50 µg/kg/minute for 4 minutes. It has a short half life of 9 minutes; so if blood pressure is not adequately improved, the loading dose needs to be repeated prior to increasing the infusion dose to 10 µg/kg/minute. The maximal dose should not exceed 300 µg/kg/minute. It has been recommended for use in the perioperative setting and is the first line drug for patients with aortic dissection.¹⁸ It has a short half-life of about 9 minutes. Like the other beta-blockers, it should be used with caution in patients with asthma. It also can interact with warfarin.
- Trimethaphan is a ganglion blocker, which has been recommended for aortic dissections asso-

ciated with hypertension. It is given at a dose of 0.5–5 mg/minute.

- Phentolamine, an alpha-adrenergic inhibitor, is given in an intravenous dose of 5–15 mg IV. Its primary use is in patients with pheochromocytoma.
- Loop diuretics should also be considered in patients who are not volume depleted and may add to the efficacy of the above-listed medications.

Management of Hypertension in the Immediate Preoperative Period

Despite the fact that it is now well recognized that preoperative blood pressure control and the use of beta-blockers reduce mortality, many patients still enter the operating or angiography suite without the benefit of these medications or with BP inadequately controlled. In a recent study of 2,500 patients having carotid duplex scans in this vascular laboratory, only 7% were normotensive (< 120/80 mm Hg).¹⁹ Certainly, blood pressure elevation in many of these patients may have been due to “white coat syndrome,” whereby patients are usually normotensive but exhibit elevated blood pressure only at times of stress such as when being examined by their physician. However, so too will many patients exhibit such elevations before surgery or an endovascular procedure. The question then arises as to how to manage patients who present the morning of their procedure with elevated blood pressure? Fleisher,²⁰ in a review published in 2002, suggested that elective vascular procedures be postponed if the blood pressure is > 170/110 mm Hg, or if the procedure is emergent, then a parenteral medication should be chosen to reduce the blood pressure preoperatively. However, recently Howell et al²¹ performed a systematic review and meta-analysis of 30 observational studies and demonstrated an odds ratio for the association between hypertensive disease and perioperative cardiac outcomes of 1.35 (1.17–1.56). This association was statistically but not clinically significant. There was little evidence for an association between admission arterial pressures of less than 180 mm Hg systolic or 110 mm Hg diastolic and perioperative complications. The position was less clear in patients with admission arterial pressures above this level. Such patients are more prone to

perioperative ischemia, arrhythmias, and cardiovascular lability, but those authors claim that there is no clear evidence that deferring anesthesia and surgery in such patients reduces perioperative risk. They recommend that anesthesia and surgery should not be cancelled on the grounds of elevated preoperative arterial pressure.

Based on these two seminal papers, it is suggested that rather than cancel surgery solely on the basis of preoperative blood pressure, the vascular surgeon and anesthesiologist should also take into account evidence of existing target organ damage, such as coronary artery disease. However, caution should be exercised when the systolic blood pressure exceeds 180 mm Hg or the diastolic blood pressure is greater than 110 mm Hg.

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