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REVIEW ARTICLE

Hypertension crisis

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Abstract

Hypertensive crises (76% urgencies, 24% emergencies) represented more than one fourth of all medical urgencies/emergencies. Hypertensive urgencies frequently present with headache (22%), epistaxis (17%), faintness, and psychomotor agitation (10%) and hypertensive emergencies frequently present with chest pain (27%), dyspnea (22%) and neurological deficit (21%). Types of end-organ damage associated with hypertensive emergencies include cerebral infarction (24%), acute pulmonary edema (23%) and hypertensive encephalopathy (16%), as well as cerebral hemorrhage (4.5%). The most important factor that limits morbidity and mortality from these disorders is prompt and carefully considered therapy. Unfortunately, hypertensive emergencies and urgencies are among the most misunderstood and mismanaged of acute medical problems seen today. The primary goal of intervention in a hypertensive crisis is to safely reduce BP. Immediate reduction in BP is required only in patients with acute end-organ damage (i.e. hypertensive emergency). This requires treatment with a titratable short-acting intravenous (IV) antihypertensive agent, while severe hypertension with no acute end-organ damage is usually treated with oral antihypertensive agents. Patients with hypertensive emergencies are best treated in an intensive care unit (ICU) with titratable IV hypotensive agents. The aim of this review is to summarize the details regarding the definition-impact, causes, clinical condition and management of hypertensive crises.

Key Words: *Crisis, emergencies, hypertension, urgencies*

Definition-impact

Hypertension is one of the most common chronic medical conditions affecting approximately 27% of the adult population in Europe (1). Worldwide, hypertension may affect as many as 1 billion people and be responsible for approximately 7.1 million deaths per year (2). It is estimated that approximately 1% of patients with hypertension will at some point, develop a hypertensive crisis, and it has been estimated that hypertensive emergencies account for 25% of all patient visits to the medical section of an emergency department (ED), with hypertensive emergencies detected in one-third of these cases (3). Before the advent of antihypertensive therapy, this complication occurred in up to 7% of the hypertensive population (3). Men are affected twice as frequently as women. Among specific situations such as postoperative hypertensive crisis, the incidence varies depending on the population being reported; however, such a crisis is reported more frequently with immediate postoperative bypass surgical graft patients (4). Also, preclampsia (pregnancy-induced hypertension with significant proteinuria 300 mg/l or 500 mg/24-h) occurs in approximately 7% of all pregnancies, with the majority of them being null-gravidas (4). Most patients who present with a hypertensive crisis have previously been diagnosed as hypertensive and many have been prescribed antihypertensive therapy with inadequate blood pressure (BP) control (4).

The syndrome of hypertensive emergency was first described by Volhard & Fahr in 1914 and was characterized by severe accelerated hypertension (also called malignant-accelerated hypertension), accompanied by evidence of renal disease and by signs of vascular injury to the heart, brain, retina and

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kidney, and by a rapidly fatal course ending in heart attack, renal failure or stroke.

Today a large number of different terms have been applied to define acute severe elevations in BP, and the current terminology is somewhat confusing. The 2003 Joint National Committee (JNC) on Prevention, Detection, Evaluation, and Treatment of High Blood Pressure (JNC 7) defines "hypertensive crisis" as a systolic BP (SBP) >179 mmHg or a diastolic BP (DBP) >109 mmHg (Table I) with or without acute target organ involvement, while it is important to define a true emergency from urgency (5,6).

Hypertensive emergencies represent severe elevations in BP that are complicated by evidence of progressive target organ dysfunction and require immediate BP reduction (not necessarily to normal levels) to prevent or limit target organ damage (TOD). Examples include hypertensive encephalopathy, sympathetic crisis, perioperative hypertension, acute aortic dissection, acute coronary event, subarachnoid hemorrhage or cerebrovascular accident, pre-eclampsia or eclampsia of pregnancy.

Table I. Causes of hypertensive emergencies.

Uncontrolled essential hypertension
Cerebrovascular conditions
Hypertensive encephalopathy
Ischemic stroke
Intracerebral hemorrhage
Eclampsia, pre-eclampsia
Renal diseases
Acute glomerulonephritis
Renovascular hypertension
Renal crises from systemic sclerosis
Post-renal transplantation
Renal malformation
Endocrine diseases
Pheochromocytoma
Cushing syndrome
Primary aldosteronism
Cardiac diseases
Acute aortic dissection
Acute left-ventricular failure
Myocardial infarction
Post-coronary artery bypass
Acute coronary syndrome
Aortic coarctation
Drug-induced hypertension
Cocaine
Amphetamine
SSRI
MAOI in combination with certain foods or drugs
Rebound hypertension
Abrupt withdrawal of clonidine, ACEI, or beta-blockers
Postoperative hypertension
Burns
Head injuries and CNS trauma
Autonomic hyperactivity (Guillain–Barré syndrome)
Vasculitis

SSRI, selective serotonin reuptake inhibitors; MAOI, monoamine; ACEI, angiotensin-converting enzyme inhibitors; CNS, central nervous system.

The hypertensive urgency is a less clearly defined condition in which severe uncontrolled hypertension is observed in a patient who may have evidence of previous end-organ damage related to hypertension, but in whom there exists no evidence of ongoing or imminent target organ dysfunction related to the current episode of hypertension. Most often this occurs in patients with previously diagnosed chronic hypertension (7). These patients do not require hospital admission or acute lowering of BP and can be effectively managed in the ED with oral agents and appropriate follow-up within 24 h to several days, depending upon individual patient characteristics (6,7).

Pathophysiology

The factors leading to the severe and rapid elevation of BP in patients with hypertensive crises are poorly understood. Hypertensive crisis is thought to be initiated by an abrupt increase in systemic vascular resistance likely related to humoral vasoconstrictors (8,9). The subsequent increase in BP generates mechanical stress and endothelial injury leading to increased permeability, activation of the coagulation cascade and platelets, and deposition of fibrin. With severe elevations of BP, endothelial injury and fibrinoid necrosis of the arterioles ensue (8,9). This process results in ischemia and the release of additional vasoactive mediators generating a vicious cycle of ongoing injury. The renin–angiotensin system is often activated, leading to further vasoconstriction and the production of proinflammatory cytokines (10). The volume depletion that results from pressure natriuresis further stimulates the release of vasoconstrictor substances from the kidney. These collective mechanisms can culminate in end-organ hypo-perfusion, ischemia and dysfunction that manifests as a hypertensive emergency.

Management of hypertensive crises

Evaluation

The medical history includes: (i) duration and previous levels of high blood pressure; (ii) administration of drugs or substances that can raise blood pressure (liquorice, nasal drops, cocaine, amphetamines, oral contraceptives, steroids, non-steroidal anti-inflammatory drugs, erythropoietin, cyclosporine); (iii) quantification of smoking and physical activity; (iv) weight gain; (v) dietary intake of fat, salt and alcohol; (vi) concomitant pathologies such as coronary artery disease, heart failure, cerebrovascular or peripheral vascular disease, renal disease, diabetes mellitus, gout, dyslipidemia, asthma or any other significant illnesses, and drugs used to treat those conditions; (vii) previous antihypertensive therapy, its results and adverse effects; (viii) symptoms of the sleep apnea syndrome should be explored (11,12).

Physical examination should include: (i) palpation of kidneys (polycystic kidney); (ii) auscultation of abdominal murmurs (renovascular hypertension); (iii) auscultation of heart sound and murmurs (aortic coarctation or aortic stenosis); (iv) murmurs over neck arteries; (v) motor or sensory neurological defects; (vi) abnormal cardiac rhythms, ventricular gallop; (vii) pulmonary rales; (viii) fundoscopic abnormalities in the retina; (ix) intravascular volume status with estimation of the jugular venous pressure and peripheral edema; (x) absence, reduction or asymmetry of pulses in lower extremities and ischemic skin lesions; (xi) body weight, waist circumference and body mass index. BP must be measured in both arms to detect any significant differences. In addition to blood pressure, heart rate should be carefully measured because the repeated finding of values above normal may be an indication of greater risk, increased sympathetic or decreased parasympathetic activity or of heart failure. Furthermore, vital signs monitoring (heart rate and rhythm, blood pressure, and oxygen saturation) is important in a patient with a hypertensive crisis. Careful assessment of the abdominal area may disclose the presence of a pulsatile mass consistent with an abdominal aortic aneurysm or the bruit of a renovascular lesion (11,12).

Initial laboratory studies should be limited and rapidly expedited. A urinalysis with microscopic examination of the urinary sediment, a chemistry panel and an electrocardiogram (ECG) should immediately be obtained. The urinalysis may reveal significant proteinuria, red blood cells and/or cellular casts. Cellular casts are suggestive of renal parenchymal disease. Electrolyte abnormalities, particularly hypokalemia or hypomagnesemia, increase the risk of cardiac arrhythmias, and the chemistry panel will also identify evidence of renal and/or hepatic dysfunction. The ECG should provide evidence of coronary ischemia and/or left ventricular hypertrophy, and the finding of pulse deficits may raise the question of aortic dissection. An X-ray of the thorax can be indispensable, particularly when dyspnea is the presenting complaint (evaluation of cardiomegaly or pulmonary edema) or information on large intrathoracic arteries or the pulmonary circulation is sought, but in general chest X-ray is an obsolete standard procedure during a hypertensive crisis. When the clinical examination suggests cerebrovascular ischemia or hemorrhage, or in a comatose patient, a brain computed tomographic scan or magnetic resonance study should be immediately obtained (10).

Patients with hypertensive emergency usually present for evaluation as a result of a new symptom complex related to their elevated BP. Patient triage and physician evaluation should proceed expeditiously to prevent ongoing end-organ damage. The distinctions between hypertensive emergencies and urgencies are often ambiguous. The first consideration in BP management in the setting of a life-threatening condition in the primary care is that BP level is not the most critical factor in determining the existence of a hypertensive emergency. The initial goal to therapy is not to achieve a normal BP but, more appropriately, a DBP of 100–110 mmHg to minimize the risk of a too low level of cerebral or cardiac perfusion. After BP is reduced to this level, then over several days a normal goal BP, as tolerated by the individual patient, can be achieved (7–13).

Pharmacological agents used in the treatment of hypertensive emergencies

The ideal pharmacological agent for the management of hypertensive crises would be fast-acting, rapidly reversible and titratable without significant side-effects. Although no single ideal agent exists, a growing number of drugs are available for the management of hypertensive crises. The agent of choice in any particular situation will depend upon the clinical presentation.

Oral/sublingual therapy with short-acting nifedipine has been widely used in the management of hypertensive emergencies. It is now understood that nifedipine is not absorbed through the buccal mucosa, but is rapidly absorbed from the gastrointestinal tract after the capsule is broken and dissolved. Nifedipine causes direct vasodilatation of arterioles, reducing peripheral vascular resistance. A significant decrease in BP is observed 5–10 min after nifedipine administration. However, when given as an oral peripheral vasodilator to treat hypertensive urgency and emergency, nifedipine has an erratic and unpredictable effect on BP. Numerous serious adverse events have been attributed to its use for rapid lowering of BP including stroke and death. Nifedipine immediate-release formulations must be abandoned as a treatment option in the management of hypertensive urgency and emergency (12).

Clonidine and angiotensin-converting enzyme (ACE) inhibitors are long acting and poorly titratable; however, these agents may be useful in the management of hypertensive urgencies. ACE inhibitors are contraindicated in pregnancy (14,15). The recommended IV antihypertensive agents are reviewed below.

Sodium nitroprusside, a first-choice agent for the majority of hypertensive emergencies, is an arterial and venous vasodilator that decreases both afterload and preload (16,17). Nitroprusside decreases cerebral blood flow while increasing intracranial pressure, effects that are particularly disadvantageous in patients with hypertensive encephalopathy or following a cerebrovascular accident (18). In patients with coronary artery disease, a significant reduction in regional blood flow (coronary steal) can occur (19,20). Because of its potency, rapidity of action and the development of tachyphylaxis, intra-arterial BP monitoring is recommended.

The most important adverse effect of sodium nitroprusside includes intoxication with thiocyanate (a metabolite of nitroprusside), which can occur when this agent is administered for more than 48–72 h. Considering the potential for severe toxicity with nitroprusside, this drug should only be used when other IV antihypertensive agents are not available and then only in specific clinical circumstances and in patients with normal renal and hepatic function. The duration of treatment should be as short as possible, and the infusion rate should not be >2 μg/kg/min (21).

Nitroglycerin is a powerful venodilator that reduces preload, increases coronary blood flow through collateral coronary vessels dilation, suppresses coronary vasospasm, and decreases cardiac oxygen demands. Higher doses are required to produce arteriolar vasodilatation. Nitroglycerin is the best agent in hypertensive crises that are complicated with ischemic heart disease and after coronary bypass. Tolerance to nitroglycerine develops if it is administered continuously for 24–48 h (22).

Nicardipine is a second-generation dihydropyridine-derivative calcium-channel blocker with high vascular selectivity and strong cerebral and coronary vasodilatory activity. The onset of action of IV nicardipine is from 5 to 15 min, with a duration of action of 4–6 h. A useful therapeutic benefit of nicardipine is that the agent has been demonstrated to increase both stroke volume and coronary blood flow with a favorable effect on myocardial oxygen balance. This property is useful in patients with coronary artery disease and systolic heart failure (23).

Labetalol is a combined selective α1-adrenergic and non-selective β-adrenergic receptor blocker with an α- to β-blocking ratio of 1:7 (24). Labetalol is metabolized by the liver to form an inactive glucuronide conjugate (25). The hypotensive effect of labetalol begins within 2–5 min after its IV administration, reaching a peak at 5–15 min following administration, and lasting for about 2–4 h (25). Because of its beta-blocking effects, the heart rate is either maintained or slightly reduced. Unlike pure β-adrenergic blocking agents that decrease cardiac output, labetalol maintains cardiac output (26).

Labetalol reduces systemic vascular resistance without reducing total peripheral blood flow. In addition, the cerebral, renal, and coronary blood flow are maintained (26,27). This agent has been used in the setting of pregnancy-induced hypertensive crisis because little placental transfer occurs mainly because of the negligible lipid solubility of the drug. Labetalol may be administered as loading dose of 20 mg, followed by repeated incremental doses of 20–80 mg at 10-min intervals until the desired BP is achieved. Alternatively, after the initial loading dose, an infusion commencing at 1–2 mg/min and titrated up until the desired hypotensive effect is achieved is particularly effective. Bolus injections of 1–2 mg/kg have been reported to produce precipitous falls in BP and should therefore be avoided (28).

Esmolol is an ultrashort-acting cardioselective, β-adrenergic blocking agent. The onset of action of this agent is within 60 s, with a duration of action of 10–20 min (29,30). The metabolism of esmolol is via rapid hydrolysis of ester linkages by RBC esterases and is not dependent on renal or hepatic function. Because of its pharmacokinetic properties, some authors consider it an "ideal β-adrenergic blocker" for use in critically ill patients. This agent is available for IV use both as a bolus and as an infusion. Esmolol is particularly useful in severe postoperative hypertension (31–33). Esmolol is a suitable agent in situations in which cardiac output, heart rate and BP are increased. Typically, the drug is administered as a 0.5–1-mg/kg loading dose over 1 min, followed by an infusion starting at 50 μg/kg/min and increasing up to 300 μg/kg/min as necessary.

Fenoldopam is unique among the parenteral BP agents, as it mediates peripheral vasodilation by acting on peripheral dopamine-1 receptors. After a starting dose of 0.1 μg/kg/min, the dose can be titrated every 15 min depending on the BP response. It has been demonstrated to have a dose-dependent decrease in BP in the infusion range of 0.03–0.3 μg/kg/min (34). No adverse effects have been noted with its use. In a prospective, randomized, open-label, multicenter clinical trial, fenoldopam was as effective as nitroprusside for the treatment of hypertensive emergency. Consequently, fenoldopam may be particularly beneficial in patients with impaired renal function with hypertensive emergency (35,36). Phentolamine is a pure alpha-adrenergic antagonist utilized for the management of catecholamine-induced hypertensive emergencies (e.g. pheochromocytoma or tyramine ingestion in a patient being treated with a monoamine oxidase inhibitor) (37,38). It is administered in 1–5-mg IV boluses with an immediate effect that can last up to 15 min. Continuous infusions can be used if necessary, but phentolamine may cause tachyarrhythmias or angina. Once initial catecholamine-induced hypertension is under control, oral phenoxybenzamine, a long-acting alpha-adrenergic antagonist, should be administered.

Diuretics are not generally first-choice agents in the treatment of hypertensive crises; however, intravenous administration of agents such as furosemide and bumetanide may be particularly useful in patients who present with pulmonary edema with concomitant hypertension. Loop diuretics in combination with nitroglycerin may be all that is required to control BP in this patient population. In managing pulmonary edema, these agents increase venous capacitance within 5–15 min and produce natriuresis in approximately 20 min, resulting in a decrease in preload. The relative potency of these agents varies. Bumetanide is 40 times more potent than furosemide. Furosemide can be initiated at 20–80 mg IV over 1–2

min. In the setting of heart failure and/or renal insufficiency, larger doses may be necessary to achieve diuresis and natriuresis. Adverse effects can include decreased sodium, potassium, magnesium and calcium plasma concentrations after long-term use. Ototoxicity may also occur with intravenous administration (37,38).

Special circumstances regarding management

Acute aortic dissection. Patients who present to the ED with the presumptive diagnosis of aortic dissection should be started on parenteral antihypertensives as soon as possible. It is important to recognize that the propagation of the dissection is dependent not only on the elevation of the BP itself but also on the velocity of left ventricular ejection.

A vasodilator alone is not ideal as this can promote reflex tachycardia, increase aortic ejection velocity and promote dissection propagation; therefore, the combination of a beta-adrenergic antagonist and a vasodilator is the standard approach to treatment. Esmolol is the beta-adrenergic antagonist of choice with metoprolol as a suitable alternative (39,40). Although nitroprusside has traditionally been used as the vasodilator of choice, nicardipine or fenoldopam are less toxic, equally effective alternatives (40,41).

All patients with aortic dissection require a consultation to determine if surgical management is necessary. Unless significant medical comorbidities are present, surgery is indicated for all patients with ascending aorta type A dissection. Patients with type B dissections and distal aortic dissections can be managed with aggressive BP control as outcomes have been shown to be the same with either medical or surgical treatment unless complications such as leak, rupture, or impaired flow to vital organs occur (42).

Sympathetic crises. The most commonly encountered sympathetic crises revolve around the recreational use of sympathomimetic drugs such as cocaine, amphetamine or phencyclidine. Rarely, these crises may be seen with pheochromocytoma, a patient taking a monoamine oxidase inhibitor who ingests a tyramine-containing food, or patients who abruptly stop antihypertensive medications such as clonidine or beta-adrenergic antagonists (43).

In the clinical situations characterized by sympathetic overstimulation, beta-adrenergic antagonists should be avoided to prevent vascular beta-receptor antagonism resulting in unopposed alpha-adrenergic activity and potential increase in BP. In fact, in cocaine-induced hypertensive emergency, the use of beta-adrenergic blockade can increase coronary vasoconstriction, fail to control heart rate, increase BP and decrease survival (43,44). Interestingly, although labetalol is traditionally considered the ideal agent because of its alpha- and beta-adrenergic antagonism, experimental studies do not support its use in this clinical setting (45,46). BP control is best achieved with nicardipine, fenoldopam or verapamil in combination with a benzodiazepine (44,47). Phentolamine is an alternative agent (Table II) (48).

Table II. Recommended antihypertensive agents for hypertensive crises.

Condition	Preferred antihypertensive agent
Hypertensive encephalopathy	Labetalol, nicardipine or fenoldopam
Acute aortic dissection	Labetalol or combination of nicardipine or fenoldopam and esmolol or combination of nitroprusside with either esmolol or IV metoprolol
Pre-eclampsia, eclampsia	Labetalol or nicardipine
Sympathetic crisis/ cocaine overdose	Verapamil, diltiazem, or nicardipine in combination with a benzodiazepine

IV, intravenous.

Pre-eclampsia and Eclampsia. The presentation of a patient with pregnancy-induced hypertension (Table II) may range from a mild to a life-threatening disease process. Most pre-eclamptic patients are vasoconstricted and hemoconcentrated. Initial therapy of pre-eclampsia includes volume expansion, magnesium sulfate ($MgSO_4$) for seizure prophylaxis and BP control (49–53). Delivery is the definitive treatment for pre-eclampsia and eclampsia.

$MgSO_4$ is usually given as a loading dose of 4–6 g/100 ml over 15–20 min followed by a constant infusion of 1–2 g/h of $MgSO_4$ depending on urine output and deep tendon reflexes, which are checked on an hourly basis.

The next step in the management of pre-eclampsia is to reduce the BP to a safe range being diligent to avoid significant hypotension. The objective of treating severe hypertension is to prevent intracerebral hemorrhage and cardiac failure without compromising cerebral perfusion or jeopardizing uteroplacental blood flow, which is already reduced in many women with eclampsia (48). Most authorities and the current guidelines from the American College of Obstetricians and Gynecologists recommend keeping the SBP between 140 and 160 mmHg and the DBP between 90 and 105 mmHg (48,53). This recommendation is supported by a recent study, which demonstrated that a SBP>160 mmHg was the most important factor associated with a cerebrovascular accident in patients with severe pre-eclampsia and eclampsia (51). This would suggest that a SBP between 155 and 160 mmHg should be the primary trigger to initiate antihypertensive therapy in a patient with severe pre-eclampsia or eclampsia (49).

Hydralazine has been recommended as the drug of choice to treat severe pre-eclampsia and eclampsia since the early 1970s (54). Based on the available

data (54), we suggest that hydralazine should not be used as first line treatment of severe hypertension in pregnancy. Similarly, sublingual or oral nifedipine should be avoided in this setting. Our preference is IV labetalol or nicardipine, which are easier to titrate and have a more predictable dose response than hydralazine. Both agents appear to be safe and effective in hypertensive pregnant patients (55–57).

Cerebrovascular accidents. In the setting of acute stroke, patients may have an ischemic penumbra of brain tissue, which may have impaired perfusion but may not be irreversibly damaged. In this situation, a local drop in cerebral blood flow occurs, and cerebral arterioles dilate in an attempt to compensate and maintain flow to the potentially salvageable ischemic tissue. The magnitude of the low perfusion and duration of the ischemia are important variables. It is intuitively attractive that reperfusion of this area by dilatation of leptomeningeal collaterals may be advantageous. Most patients who present with acute ischemic or hemorrhagic stroke will present with elevated BP likely as an adaptive mechanism to maintain blood flow to the affected area (58,59). In these clinical situations, the elevated systemic BP is not a manifestation of hypertensive emergency, but rather a protective physiological response to maintain cerebral perfusion pressure to the vascular territory affected by ischemia. Because of impaired cerebral autoregulation from chronic hypertension, rapid BP correction can reduce cerebral perfusion and extend the ischemic penumbra to the entire arterial territory with catastrophic consequences. There is no evidence that this elevated BP affects the outcome during the acute phase of an ischemic stroke (60,61).

The optimal management of blood pressure in patients experiencing acute ischemic neurological events remains controversial. Markedly elevated blood pressure after an acute ischemic event may increase the risk of conversion from an ischemic to a hemorrhagic lesion, with life-threatening implications. However, inducing hypertension to increase cerebral blood flow has been attractive on the basis of experimental data. Most experts agree that a 10–15%, but not greater than 20%, reduction in BP during the first 24 h is an acceptable goal in patients with severe elevations of BP (DBP>120 mmHg) following an acute ischemic stroke (60–62). Antihypertensive treatment is recommended for both prevention of recurrent stroke and prevention of other vascular events in persons who have had an ischemic stroke or TIA and are beyond the hyperacute period (Class I, Level of Evidence A). Because this benefit extends to persons with and without a history of hypertension, this recommendation should be considered for all ischemic stroke and TIA patients (Class IIa, Level of Evidence B). An absolute target BP level and reduction are uncertain and should be individualized (63).

In the setting of hemorrhagic stroke with intracerebral hematoma, BP control is recommended when SBP is greater than 200 mmHg and/or DBP is greater than 110 mmHg (60,61). However, a rapid decline in BP within 24 h of presentation has been demonstrated to be independently associated with increased mortality in patients with intracranial hemorrhage (64). Judicious restraint in controlling BP in the setting of ischemic or hemorrhagic stroke is therefore warranted.

Perioperative hypertension management

Acute hypertension is common after major surgery and may be associated with an increased risk of serious cardiac and neurologic, complications. Indeed perioperative hypertension often occurs in conjunction with one of the following events: during the induction of anesthesia; intraoperatively as associated with acute pain-induced sympathetic stimulation leading to vasoconstriction; in the early postanesthesia period, associated with pain induced sympathetic stimulation, hypothermia, hypoxia or intravascular volume overload from excessive intraoperative fluid therapy; and in the 24–48 h postoperatively, as fluid is mobilized from the extravascular space. In addition, BP elevation secondary to discontinuation of long-term antihypertensive medication may occur postoperatively (65).

Hypertensive events occur most commonly with carotid surgery, abdominal aortic surgery, peripheral vascular procedures, and intraperitoneal or intrathoracic surgery (66). At least 25% of patients undergoing non-cardiac surgery have hypertension prior to their surgical procedure; elevated BP (e.g. SBP≥170 mmHg, DBP≥110 mmHg) have been associated with complications such as myocardial ischemia. Hypertensive urgencies, and emergencies, occur in approximately 50% of patients during and immediately after cardiac surgery (66,67).

The goal of controlling perioperative hypertension is to protect organ function, and is currently recommended based on the assumption that the risk of complications will be reduced and outcomes improved. However, the treatment of acute elevations in BP (defined as an increase in SBP, DBP, or mean arterial pressure by >20% over baseline in the perioperative period) is without a uniform approach. In general, the treatment goal should be based on the preoperative BP. A conservative target would be approximately 10% above that baseline; however, a more aggressive approach to lowering BP may be warranted for patients at very high risk of bleeding or with severe heart failure who would benefit from afterload reduction. Careful monitoring of patient response to therapy, and adjustment of treatment, are paramount to safe and effective treatment of perioperative hypertension. A wide selection of

available IV antihypertensive agents has provided clinicians with the ability to optimize therapy based on the specific situation and patient. After surgery, the clinician can safely transition the patient to an effective oral antihypertensive regimen to manage the long-term risks of hypertension and cardiovascular diseases (68–70).

The ideal antihypertensive agent should provide immediate onset of action, a short to intermediate duration of action, be easy to titrate precisely, and have demonstrated safety and efficacy in the treatment of perioperative hypertension. Newer agents such as fenoldopam, nicardipine and clevidipine are valuable additions to the arena of effective pharmacological options such as enalaprilat, labetalol, nitroglycerin, esmolol and hydralazine. Sodium nitroprusside should only be used when other IV antihypertensive agents are not available and then, only in specific clinical situations (70–74).

Conclusions

Hypertensive emergencies have the potential for permanent end organ damage and significant morbidity and mortality. Patients with hypertensive crises may require immediate reduction in elevated BP to prevent and arrest progressive end-organ damage. The appropriate therapeutic approach in each patient will depend on the clinical presentation. The best clinical setting in which to achieve this BP control is in the ICU, with the use of titratable IV hypotensive agents. With the development of pharmacological agents in the last decade, the traditional agent, nitroprusside, should be utilized significantly less given that the other agents such as esmolol, nicardipine and fenoldopam are now available and are equally effective with fewer adverse effects. Agents such as nifedipine and hydralazine should be abandoned because these agents are associated with significant toxicities or side-effects and increased mortality.

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