

Acute Arterial Hypertension in Patients undergoing Neurosurgery

Hipertensão arterial aguda em pacientes submetidos a neurocirurgia

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Abstract

Introduction Between 20–50% of neurosurgical patients may develop early perioperative complications, and ~ 25% have more than one clinical complication. The most commons are high blood pressure (25%) and cardiovascular events (7%). Intraoperative hypertension is characterized by an increase of 20% in basal blood pressure.

Objectives The aim of this paper is to review and discuss the pathophysiology, diagnosis and treatment of perioperative hypertension in patients undergoing neurosurgery, and to propose one table with therapeutic options.

Methods A review using Scielo, PubMed, Ebsco and Artmed databases with inclusion and exclusion criteria. Articles published from 1957 to 2015 were selected.

Discussion Five factors were established as causes: arterial hypertension, clinical conditions, surgical procedures, and operative and anesthetic factors. Specific causes preoperative, intraoperative and posoperative. The pathophysiology may have some relationship with catecholamines and sympathetic nervous system stimulation.

Conclusion Perioperative hypertension in neurosurgery may have many causes, some of them recognizable and preventable. This increased pressure may be associated with intracranial hematomas in some cases. The recognition and treatment of this disease can be helpful in the management of the postoperative period.

Keywords

- postoperative period
- arterial hypertension
- neurosurgery
- emergence hypertension

Resumo

Introdução Entre 20–50% dos pacientes neurocirúrgicos podem desenvolver complicações perioperatórias precoces, e cerca de 25% têm mais de uma complicação clínica. A complicação mais comum é pressão arterial elevada (25%); eventos cardiovasculares (7%). Hipertensão Arterial intraoperatória é considerada quando se aumenta 20% o valor absoluto do seu valor da pressão de base.

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Palavras-chave

- período pós-operatório
- hipertensão arterial
- neurocirurgia
- emergência hipertensiva

Objetivos O objetivo deste artigo é discutir a fisiopatologia, diagnóstico e tratamento da hipertensão perioperatória em pacientes que passaram por neurocirurgia, e propor uma tabela com opções terapêuticas.

Métodos Revisão utilizando as bases de dados SciELO, PubMed, Ebsco e Artmed com os critérios de inclusão e exclusão. Artigos publicados de 1957 a 2015.

Discussão Cinco fatores foram estabelecidos como causas: hipertensão, condições clínicas, os procedimentos cirúrgicos, fatores operatórios e fatores anestésicos. Existem causas específicas pré-operatório, intraoperatório e pós-operatório estabelecidas. A fisiopatologia pode ter alguma relação com catecolaminas e com a estimulação simpático do sistema nervoso.

Conclusão A hipertensão perioperatória na neurocirurgia pode ter várias causas, algumas delas reconhecíveis. Este aumento da pressão arterial pode estar associado à incidência de hematomas intracranianos. Reconhecimento e manejo adequado da doença pode ajudar no controle da pressão durante o período pós-operatório.

Introduction

All patients who undergo neurosurgical procedures, even a well-performed operation, are in a potentially unstable cardiopulmonary state and at risk for secondary neuronal injury. Depending on the specific operation performed, these patients can also have fresh surgical incisions, delicate vascular anastomoses, friable resection beds, brittle patency of newly open vessels, and/or tenuous hemostasis. All of these factors leave these patients especially vulnerable to post-operative complications.^{1,2}

Perioperative hypertension can occur during the induction of anesthesia. Intraoperative hypertension is associated with acute pain-induced sympathetic stimulation that leads to vasoconstriction. In the post anesthesia period, hypertension can be associated with pain-induced sympathetic stimulation, hypothermia, and/or hypoxia. Hypertension may also be the result of intravascular volume overload from excessive intraoperative intravenous fluid therapy, and it may persist 24 to 48 hours until the fluid has been mobilized from the extravascular space. Blood pressure can also rise due to discontinuation of blood pressure medications postoperatively.^{3,4}

Between 20–50% of neurosurgical patients may develop early postoperative complications, and ~ 25% will have more than one complication. Many of these complications are “minor”, the most common being nausea/vomiting (30%), or shivering (18%). The incidence of other complications is difficult to determine, and in part depends on the procedure, as well as on how the complications are classified. These include: respiratory (3%), airway trauma (4%), cardiovascular (7%), and neurological (6%).⁵

The frequency of acute postoperative hypertension, by surgical is establish: carotid endarterectomy (9–65%), cardiac surgery (22–54%), abdominal aortic surgery (33–75%), radical neck dissection (10–20%), intracranial neurosurgery (57–91%), elective non-cardiac surgery (20%), elective general surgery (3–9%)^{5–13}

Systemic hypertension associated with emergence from anesthesia has long been believed to contribute to intracra-

nial hemorrhage and cerebral edema following craniotomy. Lewelt et al demonstrated that elevated postoperative blood pressure was a correlate of intracerebral bleeding after craniotomy.¹⁴ Forster et al observed that in anesthetized animals, sudden substantial increases in arterial pressure can result in a breach of the blood-brain barrier.^{15,16} The incidence of perioperative hypertension has been reported to be as wide as 54–91% in various studies. Basali et al report an incidence of 57% for post-craniotomy hypertension.¹⁷

With the presence of preoperative hypertension, it is more difficult to have hemodynamic control during anesthesia, there is an increased risk of intraoperative and postoperative cardiovascular events, and there are challenges with post-procedural blood pressure control. The presence of hypertension alone is a risk factor for other cardiovascular diseases that may contribute to perioperative adverse events. In fact, a National Veterans Administration Surgical Risk study of 83,000 patients found that hypertension was the second most common risk factor associated with surgical morbidity.^{6,7,18,19}

The aim of this paper is to discuss the pathophysiology, diagnosis and treatment of perioperative hypertension in patients undergoing neurosurgery, and to propose a table with therapeutic options.

Methods

We performed a literature review using PubMed, Medline, Science Direct, Embase, Clinical Trials, Ebsco, and Scielo databases. Articles from 1957 to 2016 were selected. The search resulted in total of 70 papers that fit the inclusion criteria.

Pathophysiology

The pathophysiologic mechanism underlying acute postoperative hypertension (APH) is uncertain, and it may vary with the surgical procedure and other factors; however, the final common pathway leading to hypertension appears to be an

activation of the sympathetic nervous system, as evidenced by elevated plasma catecholamine concentrations in patients with APH.^{20–23}

At the time of the development of postoperative hypertension, plasma catecholamine concentrations are significantly greater than in normotensive postoperative patients. The activation of the renin–angiotensin–aldosterone system may also contribute to APH; however, plasma renin, angiotensin II, and aldosterone activity are not significantly different between hypertensive and normotensive patients in all studies, suggesting that the predominant mechanism in APH is sympathetic activation. Wallach et al reported significant correlations between mean arterial pressure (MAP) and both plasma epinephrine ($r = 0.59, p < 0.01$) and norepinephrine ($r = 0.58, p < 0.01$) concentrations after coronary artery bypass grafting.¹⁹ The primary hemodynamic alteration observed in APH is an increase in afterload (systemic vascular resistance [SVR], systolic blood pressure [SBP], and diastolic blood pressure [DBP]), with or without tachycardia; there is no difference in cardiac index, left ventricular stroke volume, or left atrial pressure compared with normotensive patients.^{8,11,24–27}

These findings are consistent with a predominant sympathetic-mediated rise in MAP secondary to vasoconstriction. Many preoperative patient characteristics and operative factors may be associated with an increased risk of APH, and several postoperative factors may precipitate increased sympathetic activity, and therefore cause or aggravate APH.²⁸

Five factors were established as causes: 1) *hypertension* (especially if poorly controlled); 2) *clinical conditions* (diabetes, vascular disease, advanced age, kidney disease, pain, anxiety, hypothermia, post-anesthesia tremors, excitement, hypoxia, hypercapnia, antihypertensive withdrawal, hypervolemia, hypovolemia, myocardial ischemia, drug interactions, increased intracranial pressure, pulmonary embolism, vasopressor therapy, bronchodilators); 3) *operative factors* (surgical technique, duration of the procedure); 4) *surgical procedures* (vascular, cardiothoracic, neurosurgery, head and neck); and 5) *anesthetic factors* (pancuronium inhibitors, acetylcholinesterase, opioid antagonists, placement of the endotracheal tube, bladder distension).^{5,29,30}

Preoperative Hypertension

At least 25% of patients undergoing non-cardiac surgery will have hypertension before undergoing their procedure. This has important outcome implications, as high blood pressure is associated with complications such as myocardial ischemia. Preoperative hypertension is frequently a hypertensive urgency - not an emergency -, as it doesn't involve end organ damage and there is usually enough time to reduce the blood pressure. It has been suggested that a DBP of 110 mmHg or above is considered a preoperative marker of perioperative cardiac complications in patients with chronic hypertension. In a study by Forrest et al. Preoperative hypertension was associated with perioperative bradycardia, tachycardia, and hypertension, and Browner et al found a fold increase on

postoperative death when compared with normotensive patients.^{9,15,16,18,31–34}

When hypertension is detected, it is necessary chase for secondary causes of hypertension should be done. Even though pheochromocytoma is rare, if present, it can produce serious complications during a procedure. Long-term excess catecholamine stimulation can produce vasoconstriction and hypovolemia that can potentially complicate management.

Clonidine withdrawal syndrome can simulate the hypertensive crisis of pheochromocytoma. This can be misdiagnosed, as symptoms usually present 18 to 24 hours after sudden discontinuation of clonidine. When detected, clonidine withdrawal symptoms can be corrected simply by administering intramuscular clonidine or treating with labetalol and methyldopa.^{10,11,14–16,25,32,34–41}

Intraoperative Hypertension

Intraoperative acute blood pressure elevations of over 20% during surgery are considered a hypertensive emergency, and chronic hypertensive patients are more likely to have labile hemodynamics during a procedure. Even small blood pressure elevations during surgery can result in increased risk of postoperative mortality and renal failure, especially during cardiovascular procedures. Hypertensive events occur more commonly in patients undergoing surgery of the carotids followed by neurosurgery.⁴²

The causes of hypertension during recovery from anesthesia are: pain, hypothermia, hypo-osmolality, anemia, hypercarbia and hypoxia, emergence excitement, catecholamine release or sympathetic stimulation, brain manipulation, and epinephrine-containing local anesthetic administration. The final common pathway for all of these causes leading to elevations of blood pressure in patients undergoing craniotomy appears to be the activation of the sympathetic nervous system. Elevations of plasma catecholamine concentrations are common in patients after craniotomy.^{17,43,44}

Olsen et al²⁸ studied the effects of vasoactive modulators in the perioperative period in patients undergoing craniotomy and their relation to postoperative hypertension. They concluded that in addition to increased discharge of the sympathetic system, as evidenced by elevated levels of norepinephrine and epinephrine, the activation of the renin-angiotensin aldosterone system may also play an important role in the development of postoperative hypertension after craniotomy. However, norepinephrine administration has also been shown to be associated with reductions in cerebral blood flow. Thus, circulating catecholamines as such cannot account for the cerebral hyperemia seen during craniotomy. Other causes such as metabolic stress associated with cerebral activation due to surgery may also be involved.^{28,45,46}

Postoperative Hypertension

Postoperative Hypertension is more common in preoperative hypertensive patients, and in those undergoing vascular procedures. It has been defined as an SBP of above

190 mmHg and/or DBP of 100 mmHg on 2 consecutive readings after surgical intervention. Postoperative hypertension episodes occur in the first 20 minutes of the postoperative period, although their resolution can require up to 3 hours. If left untreated, postoperative hypertension increases the risk of myocardial ischemia, myocardial infarction, cerebrovascular accidents, and bleeding.⁴⁷⁻⁴⁹

Acute postoperative hypertension is characterized by peripheral vasoconstriction, catecholamine release and reduced baroreceptor sensitivity. Some of the complications associated with APH are myocardial ischemia, myocardial infarction, cardiac arrhythmia, congestive heart failure, pulmonary edema, cerebral ischemia, hemorrhagic stroke and encephalopathy; it also increases the risk of bleeding from the surgical site.⁵⁰

Myocardial ischemia most commonly occurs in the postoperative setting, and it may present hours to days after the surgical procedure. Several factors may increase the risk of myocardial ischemia, such as oxygenation problems, altered thrombotic potential, tachycardia and postoperative hypertension, which increase myocardial oxygen demand. Postoperative hypertension can also cause pulmonary edema in patients with preexisting left ventricular systolic cardiac dysfunction.^{30,51,52}

Rose et al found that patients that presented with intraoperative hypertension, excessive pain, and inadequate ventilation had a higher risk of developing APH; they also noted that these patients had more critical care admissions and a higher risk of mortality. Additionally, a unique cause of hypertension may occur after surgical repair of coarctation of the aorta in 2 ways: an early component during the first 36 hours of systolic hypertension, and a late component of systolic and/or diastolic hypertension that persists beyond postoperative day 2. If it persists beyond day two, there is an increased risk of developing postcoarctectomy syndrome, characterized by abdominal pain and associated mesenteric arteritis.⁵²⁻⁵⁴

Emergence Hypertension and Intracranial Hemorrhage: A Temporal Relationship

The occurrence of systemic hypertension during anesthetic recovery is quite common in neurosurgical patients undergoing craniotomies, as stated before. This occurrence has often been related to the development of intracranial hematoma postoperatively. However, this association between peri-extubation hypertension and the occurrence of intracranial hematomas has not been fully investigated.⁵⁵

Basali et al,¹⁷ in a retrospective case-control study, evaluated the relationship between surges of blood pressure in the emergence period and the occurrence of postoperative intracranial hematomas. Of the total number of patients who developed hematomas in the postoperative period, ~ 62% had hypertension; the incidence of hypertension in the control group was of 34%. However, the study did suffer from innate limitations, principally because of its retrospective design, and also because of the limited number of blood pressure readings, especially in the control group. Hence,

while the study did show an association, it did not demonstrate a cause-and-effect relationship between acute hypertension and intracranial hemorrhage.^{17,56}

Wintzen et al related systemic hypertension to the occurrence of intracerebral hemorrhage in patients on anticoagulants. Similarly, Fukumachi et al and Kalfas et al related hypertension to intracerebral hemorrhage in patients with normal coagulation. Kalfas et al reported a 16% incidence of intracranial hematomas in hypertension, but again failed to demonstrate a causal relationship between the two.⁵⁷⁻⁵⁹

It is reasonable to expect that an increased blood pressure in the perioperative period would result in cerebral hyperemia with a propensity for hemorrhaging intracranially. Significant hypertension might well overwhelm the reserves of cerebral autoregulation. Equally feasible is the argument that vasomotor paralysis might already have occurred at sites of surgical manipulation, either due to surgical retraction or residual tumors. With such tenuous cerebral autoregulation in the perioperative period, any hypertension might increase the blood flow to the brain and result in a tendency to develop postoperative hematoma.⁶⁰

Hence, additional studies are needed to elucidate the mechanisms of post-craniotomy intracranial hematomas and elucidate whether a better control of hemodynamics could improve overall neurological outcomes. It is possible that there are other mechanisms apart from hypertension that might have a more telling effect on intracranial hemorrhage after craniotomy. It is also possible that hypertension might be an indicator of intracranial phenomena that are not yet fully understood, but are more temporally related to intracranial hemorrhage. It is also possible that other factors like age; anticoagulation; location of intracranial space-occupying lesions and their histological type; invasion of venous sinuses; extent of tumor removal; surgical hemostasis; cerebral venous pressures; and genetic predisposition may play a more significant role in post-craniotomy hemorrhage. Tracheal intubation might be associated with elevations of cerebral venous pressures due to straining on the endotracheal tube, and it may predispose the development of intracranial hematomas. Significantly, pharmacotherapy in the form of vasodilators to control hypertension might actually result in cerebral vasodilation and hyperemia, and may be related to the causation of postoperative intracranial hemorrhage.⁶¹

Management of Emergence Hypertension in Neurosurgical Patients

Before starting the antihypertensive pharmacological treatment, other reversible causes of postoperative hypertension should be addressed.

While none of the studies to date have been able to establish a cause-and-effect relationship between emergence hypertension and the development of intracranial hemorrhage or demonstrate better neurological outcomes with better hemodynamic control, it would still seem worthwhile to strive for stable hemodynamics perioperatively and attain a 'smooth' emergence at the end of any neurosurgical procedure. Most anesthesiologists would use β -blockers, such as

Table 1 Agents commonly used to treat acute postoperative hypertension

Agent	Typical dosage	Time to onset of action	Duration of action	Potential adverse events	Comments
Intravenous agents: Sodium nitroprusside	0.5–5 µg/kg/min (maximum, 10 µg/kg/min)	< 1 min	1–3 min	Tachycardia, precipitous reductions in BP, myocardial ischemia, cyanide and thiocyanate toxicity, pulmonary V/Q mismatch, rebound hypertension, restlessness, nausea, vomiting	Requires continuous monitoring. Sodium thiosulfate prevents cyanide toxicity
Nitroglycerin	5–300 µg/min	< 1 min	5–10 min	Tachycardia, headache, hypotension, nausea, vomiting	Tolerance develops. Good choice with myocardial ischemia
Labetalol	Bolus dose: 10–20 mg followed by 10–40 mg q10 min. Infusion: 0.5–4 mg/min (maximum 300 mg)	< 5–10 min	3–5 h	Bradycardia, bronchospasm, left ventricular dysfunction, prolonged hypotensive effect	Usual precautions for nonselective β-blockers
Esmolol	Bolus: 500 µg/kg. Infusion: 25–200 µg/kg/min	< 6–10 min	< 20 min	Bradycardia, bronchospasm, left ventricular dysfunction	Usual precautions for nonselective β-blockers
Nicardipine	10 mg/h initially, increased by 2.5 mg/h q5–15 min to a maximum of 15 mg/h	10–15 min	15–20 min	Tachycardia, hypotension, nausea, vomiting	Use caution with loading infusion as opposed to maintenance infusion dose
Hydralazine	5–20 mg q6h	15–30 min	4–6 h	Tachycardia, headache, hypotension, nausea, vomiting, cardiac ischemia	Not routinely recommended
Fenoldopam	0.1 µg/kg/min initially, increased by 0.05–0.1 µg/kg/min q15–20 min (maximum, 1.6 µg/kg/min)	20–40 min	15–30 min	Hypotension, tachycardia, headache, flushing, dizziness, bradycardia, electroencephalographic changes, elevated intraocular pressure	Avoid in patients with glaucoma or high intraocular pressure
Enalaprilat	0.625–1.25mg (repeat if needed)	15–20 min	> 4 h	Hypotension, renal dysfunction, hyperkalemia, angioedema	Contraindicated in patients with bilateral renal artery stenosis
Oral agents: Clonidine	0.1–0.2 mg (repeat q1h to a total dose of 0.8 mg)	30–60 min	> 4 h	Central-nervous-system depression, bradycardia, hypotension	Not well studied in APH. Slow onset and long duration may limit value.
Captopril	12.5–25 mg, repeat after 30–60 min	30–60 min	> 4 h	Hypotension, renal dysfunction, hyperkalemia, angioedema	Contraindicated in patients with bilateral renal artery stenosis
Nifedipine	10 mg, repeat after 30–60 min	20–30 min	> 4 h	Precipitous reduction in BP, cerebral hypoperfusion, tachycardia, myocardial ischemia	No longer recommended. Never give sublingually

Abbreviations: BP, blood pressure; V/Q, ventilation/perfusion ratio; APH, acute postoperative hypertension.

Source: Mancuso CE, Paloucek FP. Understanding and preparing for pharmacy practice residency interviews. Am J Health-Syst Pharm. 2004;61(16):1686-9.

metoprolol, labetalol or esmolol to tide over acute elevations of systemic blood pressure often encountered during recovery from anesthesia. Labetalol, because of its low potency, slow onset of peak effect and unpredictability in dose requirements may not be the most ideal agent in these circumstances. Esmolol similarly is only mildly effective, and is associated with bradycardia and conduction defects. Nicardipine is more effective than both labetalol and esmolol in controlling peri-extubation hypertension. However, calcium channel blockers have been associated with dose-dependent cerebral vasodilation, inhibition of autoregulation and hypotension. Experience with hydralazine has not been encouraging, as it increases intracranial pressure significantly.⁶²⁻⁶⁵

Dexmedetomidine has also been evaluated by several authors as an adjuvant to anesthesia for neurosurgery, including intracranial surgery. Improved perioperative hemodynamic control has been reported in these studies when compared with placebo. Lignocaine is often employed during emergence to reduce airway responsiveness and decrease the incidence of coughing and straining. Doses of 1.5 mg/kg are often appropriate for this purpose.⁶⁵⁻⁶⁹

For most non-cardiac types of surgery, there is a lack of agreement about when and how to treat APH aggressively. The significance of transient postoperative increases in blood pressure (BP), the definition of APH, treatment goals, and the potential adverse effects of vasodilators are debated. Prospective studies showing the clinical benefits of aggressive BP control in the postoperative period are lacking. There is no consensus concerning the ideal treatment threshold for a clinical management of non-cardiac surgery patients with APH; treatment is frequently a bedside decision made by the anesthesiologist or surgeon that takes into consideration the patient's baseline BP, concomitant diseases, and the perceived risk of complications. In contrast, it is generally well accepted in cardiothoracic surgery that BP elevations may be associated with significant postoperative complications, and that aggressive treatment with intravenous vasodilators is indicated. The most commonly quoted thresholds for the treatment of hypertension in cardiac surgery are a BP of > 140/90 mmHg or a MAP of at least 105 mmHg, but there is no consensus. The first step in the management of APH is a careful assessment of the patient to identify postoperative hypertension.

Pain and anxiety are common contributors to BP elevations shortly after surgery, and should be ruled out or treated before administering antihypertensive therapy. ► **Table 1**

Other potentially reversible causes of APH include hypothermia with shivering, hypoxemia, inadequate ventilation leading to hypercarbia, and bladder distention. Emergence from anesthesia with excitement is another common cause of transient APH that is usually managed by eliminating potential causes, providing proper analgesia and sedation, and giving supportive care.

Both intravascular hypervolemia and hypovolemia can cause acute elevations in BP, and should be managed with proper loop diuretics or fluid administration. The endotracheal tube may cause discomfort, contribute to anxiety and agitation, and increase sympathetic activity; therefore, the

patient should be extubated as early as is deemed safe, or provided adequate analgesia and sedation. In order to minimize the risk of APH, preoperative antihypertensive therapy should be continued until surgery. If antihypertensive withdrawal syndrome is suspected as a cause of APH, the preferred treatment is reinstitution of preoperative antihypertensive medications if possible. Acute myocardial ischemia can elevate BP, as well as be a result of APH. If there is diagnostic or clinical evidence of myocardial ischemia, treatment should be directed primarily toward relieving the ischemia, preferably with agents that will also contribute to the management of the elevated BP. Some less common causes of postoperative hypertension should also be considered, such as increased intracranial pressure, pulmonary embolism, sympathomimetic drugs, anticholinergic agents, monoamine oxidase inhibitors, and, rarely, pheochromocytoma.¹⁷

Short-term administration of antihypertensive drugs is recommended when there is no identifiable, treatable cause of the hypertension. This recommendation is primarily intuitive, since there are no controlled, prospective trials demonstrating that the aggressive management of APH improves clinical outcomes or reduces the likelihood of postoperative complications. Intravenous agents are commonly recommended, since in most cases APH occurs shortly after the completion of surgery, while the patient remains intubated or otherwise unable to tolerate oral medications and is being monitored.⁷⁰ Oral agents may be considered for patients who are outside the postanesthesia care unit or intensive care unit, but the use of oral agents does not obviate the need for careful monitoring. The ideal agent should have a rapid but smooth onset of action and a short duration of action to allow careful adjustment of the dosage and easy termination of effect. In addition, the agent should have minimal effects on heart rate, cardiac function, and myocardial oxygen demand, and have an otherwise benign adverse-effect profile. No agent meets this profile; the choice of drug therapy depends on the clinical presentation, patient characteristics, the environment of care, the properties of the drug, and the clinician's experience.^{12,13,44}

Conclusion

Systemic hypertension during emergence from anesthesia is commonly encountered in patients undergoing craniotomies. Causes may be multiple, some identifiable, some not quite as well understood. While an association of such hypertension with the incidence of intracranial hematomas may be found, there is no scientific evidence to date that establishes a temporal relationship between the two. Further prospective randomized trials are required to identify a cause-and-effect relationship between them. Multiple techniques have been employed for quelling the blood pressure response, especially during emergence from anesthesia, with varying degrees of success, but there is no doubt that peri-extubation hypertension in this subset of patients remains a challenge for anesthesiologists.

Nitroprusside should only be used when no other agents are available. Enalaprilat and nitroglycerin are recommended for combined therapy, but not as single agents.

Labetalol, esmolol, hydralazine, and fenoldopam are recommended in very specific circumstances, and attention must be paid to their possible adverse effects. Nicardipine and clevidipine are currently the agents recommended for the majority of perioperative cases requiring hypertensive management, as their efficacy has been well documented and they are proven to be safe.

Acute postoperative hypertension is a potentially serious condition. When treatment is necessary, therapy should be individualized for the patient.

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