Anesthetic Management of the Hypertensive Patient: Part II

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Hypertension is an important health challenge that affects millions of people across the world today and is a major risk factor for cardiovascular disease. It is critical that anesthesia providers have a working knowledge of the systemic implications of hypertension. This review article will discuss the medical definitions of hypertension, the physiology of maintaining blood pressure, outpatient treatment of hypertension, anesthetic implications, and the common medications used by anesthesia providers in the treatment of hypertension. Part I provided an overview of hypertension and blood pressure regulation. In addition, drugs predominantly affecting control of hypertension via renal mechanisms such as diuretics, angiotensin-converting enzyme inhibitors, angiotensin receptor blockers, and renin-inhibiting agents were discussed. In part II, the remaining major antihypertensive medications will be reviewed as well as anesthetic implications of managing patients with hypertension.

Key Words: Hypertension; Antihypertensives; General anesthesia; Sedation; Review.

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CALCIUM CHANNEL BLOCKERS

The currently available calcium channel blockers (CCBs) inhibit the opening of L-type voltage-gated calcium channels, and when inward flux of calcium is inhibited, the contraction of smooth muscle cells in peripheral arterial blood vessels decreases, and accordingly, blood pressure falls from decreased afterload. In addition, some CCBs decrease inotropy (contractility), chronotropy (heart rate), and dromotropy (conduction velocity). As a result, some CCBs decrease both systemic vascular resistance (particularly afterload) and myocardial oxygen demand.[1] There are 3 main classes of CCBs: (a) phenylalkylamines (verapamil) and (b) benzothiazepines (diltiazem), which inhibit activity at the atrioventricular node with a lesser degree of vasodilation than (c) dihydropyridines (eg, amlodipine, clevidipine, nicardipine), which are selective for arteriolar beds (see Table 1). The recent availability and advantageous pharmacologic properties of intravenous rapid-onset, rapid ester-metabolized clevidipine and short-acting nicardipine have largely replaced the use of nitrodilators such as sodium nitroprusside. Clevidipine and nicardipine can be beneficial when used as an infusion for intentional hypotension, as during orthognathic surgery, to decrease blood pressure without increasing the anesthetic depth or using long-acting vasodilators or β-blockers.

Anesthetic Implications

Inhalational agents decrease the availability of intracellular calcium, which in turn enhances the negative inotropic, chronotropic, and dromotropic effects of CCBs. The phenylalkylamines and benzothiazepines differ in their cardiovascular selectivity compared with dihydropyridines, and they exhibit cardiac depressive properties equal to the vasodilatory properties. The physiologic blunting of expected reflex tachycardia occurs with the reduced cardiac output provided by CCBs.[5] Furthermore, CCBs can potentiate all neuromuscular blocking agents, potentially impair hypoxic pulmonary vasoconstriction, and mildly increase intracranial pressure.[1] Another important consideration is the concern with CCBs in the treatment of malignant hyperthermia, which can be a potentially fatal disorder

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Table 1. Comparative Properties of Common Calcium Channel Blockers (CCBs)[2–4]

CCB	*Inotropy*	*Chronotropy*	*Peripheral Vasodilation*	*Coronary Vasodilation*	*Reflex Tachycardia*
Diltiazem	0/↓	↓	+	++	0
Verapamil	↓	↓	+	++	0
Nicardipine	0/↓	0	+++	+++	+
Nifedipine	↓	0	+++	+++	++
Clevidipine	0/↓	0	+++	+++	+

of calcium regulation in the sarcoplasmic reticulum of skeletal muscle. The intuitive idea of adding a CCB in an event where there is unregulated calcium release in the patient seems to make logical sense. However, CCBs are contraindicated during the treatment of a malignant hyperthermia crisis due to an increased potential for cardiac collapse after concomitant administration of both a CCB and dantrolene.[6] Except in cases in which malignant hyperthermia screening reveals susceptible patients, CCBs should generally be continued in the perioperative period.

α-ADRENERGIC RECEPTOR ANTAGONISTS (α-BLOCKERS)

α-Blocking medications act directly on α-adrenergic receptors and interfere with the ability of catecholamines or other sympathomimetics to provoke α responses at the peripheral vasculature and heart.[2] These medications include the nonselective α-adrenergic antagonists phentolamine, prazosin, and phenoxybenzamine. Phentolamine and prazosin reversibly bind to α-adrenergic receptors and competitively antagonize circulating catecholamine action.[7] Phenoxybenzamine is an irreversible α-adrenergic receptor antagonist, and despite the decrease in blood pressure, can cause an increase in cardiac output and greatly enhance orthostatic hypotension.[2] These drugs are seldom used as first-line therapies but are generally reserved for use in combination therapies. Their side effects, which include reflex tachycardia, marked orthostatic hypotension, and fluid retention, have made them less popular for controlling blood pressure since other medications have become available.[8] α-Blockers continue to be used for benign prostatic hypertrophy, but the currently used highly selective agents (eg, tamsulosin, an α-1A–receptor antagonist) have fewer adverse cardiovascular effects.

Phentolamine mesylate is an α adrenergic receptor antagonist used in dentistry for "reversal," or more accurately, limiting the duration of action of soft-tissue anesthesia after nonsurgical dental procedures by promoting local vasodilation that thereby accelerates the rate of systemic uptake of local anesthetic away from the site of action.[9] Phentolamine mesylate is also used to manage a hypertensive crisis associated with pheochromocytoma, commonly in combination with a β-blocking agent to attenuate the increase in heart rate. However, phentolamine mesylate, as administered by injection intraorally at the established dose of 0.4 mg for local anesthetic reversal, does not have clinically significant cardiovascular effects.

Anesthetic Implications

α-Blocking medications are not commonly used agents at this time, except for the management of benign prostatic hypertrophy, for which highly selective agents are now used. If prescribed, these medications should be continued preoperatively.[8]

β-ADRENERGIC RECEPTOR BLOCKING AGENTS (β-BLOCKERS)

β-Blockers have a variety of pharmacologic and physiologic properties (see Table 2) and comprise an effective group of antihypertensive medications that are used to treat not only hypertension but also tachyarrhythmias, ischemic heart disease, chronic congestive heart failure, and even migraine prophylaxis. With regard to cardiovascular effects, β-1 receptors predominate on the heart, causing increased cardiac contractility, heart rate, and atrioventricular conduction. In addition, these receptors are present in the kidney, causing increased renin secretion that activates the renin-angiotensin-aldosterone system. β-2 receptors predominantly regulate relaxation of smooth muscle in the vascular beds of skeletal muscle and the bronchi.[10] It should be noted that 25–30% of the β-receptors on the heart are β-2 that function like β-1 receptors. By blocking cardiac β-1 receptors, it is possible to decrease the inotropic, chronotropic, and dromotropic effects, as well as renal effects and, by doing so, decrease afterload and stress on the myocardium, thus reducing myocardial oxygen demand. In the case of nonselective β-blockers, the β-2 receptors are also inhibited, which can have the unwanted physiologic side effect of increased bronchial constriction.

Table 2. Comparative Properties of Common β-blockers[2–4]

Beta Blocker	*Heart Rate*	*Mean Arterial Pressure*	*Receptor Antagonism*	*Onset*	*Duration*
Esmolol	↓↓	↓	β-1	2 min	10–30 min
Labetalol	↓	↓↓	β-1, β-2, α-1	5–15 min	2–8 h
Metoprolol	↓	↓	β-1	1–5 min (peak 20 min)	5–8 h
Propanolol	↓	↓	β-1, β-2	2–10 min	6–10 h

Combined α- and β-blockers are a subclass of β-blockers that include medications such as carvedilol, labetalol, and dilevalol, all of which nonselectively block β-1 and β-2 receptors and selectively block α-1 receptors. The antihypertensive activity of these medications is characterized by a decrease in peripheral vascular resistance, resulting from the vasodilator activity of the compound, with no reflex tachycardia, as a result of β-adrenoceptor blockade. The α-1 adrenoceptor antagonist portion of these medications accounts for most, if not all, of the vasodilating response produced by the compound.[11] Labetalol deserves particular attention because of its common use in anesthesia practice. The α- to β-blocking ratio is 1:7 for intravenous labetalol and 1:3 for oral labetalol. The initial recommended dose of labetalol, 2.5–10 mg administered intravenously over 2 minutes, displays an elimination half-life of greater than 5 hours.[12] Labetalol's clinical onset is 5–15 minutes, which is significantly slower than the rapid onset of the short acting β-1–blocker, esmolol, with a clinical onset of 2 minutes (duration of action 10–30 minutes). The most common side effect of labetalol is orthostatic hypotension.

Anesthetic Implications

An important consideration regarding β-blockers is the strong evidence recommending that patients should continue their usual home dose prior to anesthesia and surgery. Abrupt discontinuation of β-blockers is associated with significant rebound hypertension and tachycardia, which can lead to myocardial ischemia or infarction.[1] These withdrawal symptoms are due to increased sympathetic activity, which is a probable reflection of adrenergic receptor upregulation during the period of sympathetic blockade.[13] This β-adrenergic receptor upregulation results in a hypersensitivity to circulating catecholamines and hence leads to the negative effects that include rebound hypertension and tachycardia on medication discontinuation. It is therefore recommended that patients take their β-blocker medication with a small sip of water on the morning of the surgery. If the patient forgot to take their β-blocker on the day of surgery, it is reasonable to give the patient a longer-acting β-blocker for a longer case or a short-acting β-blocker if the anticipated anesthesia duration is short. A typical dose of esmolol for immediate treatment of severe hypertension with tachycardia is 0.5 mg/kg over 60 seconds or simply titrating in boluses starting with 5–10 mg of esmolol intravenously.[14] Furthermore, β-blockers can be used in an attempt to use less opioids during the perioperative period, particularly for hypertensive patients.[15]

Because of possible bronchoconstrictive effects of blocking β-2 receptors with nonselective β-blockers, there is naturally an increased risk for bronchoconstriction during anesthesia when these drugs are used.[16] Further, β-2 agonists are used in the treatment of asthma, and the β-2 antagonistic effects of the β-blockers can be counterproductive in the acute treatment of bronchospasm. Therefore, nonselective β-blockers should be used with caution in patients with clinically significant chronic obstructive pulmonary disease and asthma. It should be noted that selective β-1 blockers likely still elicit some β-2 adrenergic blocking effects, although significantly less than nonselective β-blockers.

Adverse effects of β-blockers are generally an overextension of the pharmacologic effects, including bradycardia, orthostatic hypotension, atrioventricular conduction delays, and hypotension. Bronchospasm, particularly with agents that block β-2 receptors, can occur.[4] Because compensatory glycogenolysis is blunted by β-blockers and the warning signs of hypoglycemia (tachycardia and tremor) are masked, there is a relative contraindication to β-blockade in patients with poorly controlled diabetes mellitus.[6] Furthermore β-blockade during the preoperative period can also blunt the effects to surgical stimulation, which can be advantageous in some instances to use less opioids ("opioid-sparing") throughout a case, or it can have the negative effect of sometimes leading to subanalgesic or subanesthetic doses of pain medications and anesthetics.

A specific dental and anesthetic consideration regarding nonselective β-blockers is their interaction with epinephrine. Usually when small doses (10–20 μg intravenously given slowly) of epinephrine are given, small changes in mean arterial pressure occur since the α-adrenergic vasoconstriction effects are offset by the β-2 adrenergic peripheral vasodilation. Nonselective β-blocker antagonism at the peripheral vasodilatory β-receptors is potentially problematic and may prompt unopposed α-adrenergic effects when epinephrine is given. This acute vasoconstriction with resultant hyper-

tension can lead to reflex bradycardia. In the treatment of patients medicated with nonselective β-blockers in the outpatient, ambulatory dental setting, it is prudent to record blood pressure and heart rate before administering local anesthetic-vasopressor formulations and then reassess those parameters 3–5 minutes after each cartridge is administered to ensure unwanted hypertension has not occurred before administering more anesthetic.[17] Another option is to consider using non–vasoconstrictor-containing local anesthesia formulations, or formulations with a lower dose of 1:200,000 epinephrine concentrations, if appropriate.

α-2–ADRENERGIC AGONISTS (α-2 AGONISTS)

α-2 Agonists include a class of medications that are useful because of their sedative, anxiolytic properties and mild analgesic properties. These medications act on α-2 receptors with various subtypes that include α-2A, α-2B, and α-2C subtypes. The α-2A and α-2C subtypes are found within the central nervous system and are thought to play a role in sedation, analgesia, and sympatholytic effects, while the α-2B receptors are found peripherally on vascular smooth muscle and have been shown to mediate vasoconstrictive effects.[18] Clonidine and dexmedetomidine are 12 major α-2–adrenergic agonists with a high selectivity for activation of the α-2 receptors (clonidine 220:1 and dexmedetomidine 1620:1 α-2 to α-1 activity).[1] Tizanidine, an antispastic, is another α-2 agonist that has found utility in the dental anesthesia community as an anesthetic adjunct due to the sedative, anxiolytic, and analgesic properties similar to clonidine but with less fluctuation in blood pressure and heart rate.[19]

Anesthetic Implications

It is important to consider that an abrupt withdrawal of oral α-2 agonists, typically clonidine or guanabenz, can precipitate a rebound hypertensive crisis similar to β-blockers that can be relieved with administration of clonidine or labetalol. The onset of action of oral clonidine is 30–60 minutes and oral tizanidine is 60 minutes; thus, for severe episodes, acute treatment is with an intravenous β-blocker such as labetalol. In contrast, intravenous dexmedetomidine has a more rapid onset of action intravenously of approximately 30 seconds and a terminal elimination half-life of 2 hours.

One of the greatest benefits of α-2 agonists as sedative agents is that at low doses, the depression of the respiratory drive is minimal and not clinically significant. In addition, α-2 agonists are one of the few classes of drugs known to reduce anesthetic requirements of intravenous or inhaled agents during general anesthesia due to augmenting sedation and analgesia.[20] Dexmedetomidine has shown utility in awake fiber-optic intubation and in minimizing the need for large doses of opioids in obese patients or others with obstructive sleep apnea while providing adequate analgesia.[21] The α-2 agonists have also been effective in alleviating preoperative anxiety and emergence delirium in children.[1]

Overall, the most common adverse effects that occur in patients who receive α-2 agonists are clinically significant hypotension and bradycardia, particularly when used as an infusion for sedation or in large doses.

OTHER VASODILATORS

Vasodilators are used to control systemic hypertension; increase cardiac output by decreasing afterload, preload, or both; control pulmonary hypertension; and control cardiac shunting.[22] Commonly used agents include nitroglycerin and hydralazine, with sodium nitroprusside less commonly used today. Nitroglycerin and sodium nitroprusside generate intracellular nitric oxide, which then augments cGMP in vascular smooth muscle, specifically in both arteries and veins, leading to vasodilation.[2] Sodium nitroprusside also interacts with oxyhemoglobin and forms methemoglobin while releasing cyanide. Nitric oxide release is responsible for its vasodilating effects. Sodium nitroprusside acts primarily on the arterial vasculature, while nitroglycerin has its most prominent effect on venous capacitance vessels.[23] Hydralazine is a direct systemic arterial vasodilator that produces baroreceptor reflex stimulation with resulting increases in heart rate and myocardial contractility.[9]

Anesthetic Implications

Sodium nitroprusside can cause cyanide and thiocyanate poisoning, especially in those with renal failure or reduced renal perfusion.[22] Methemoglobinemia produced from sodium nitroprusside breakdown is unlikely; however, the nitrate metabolite of nitroglycerin is capable of oxidizing the ferrous ion in hemoglobin to the ferric state with the production of methemoglobin.[2] Hydralazine is an effective, although unpredictable, afterload-reducing agent because it relaxes the arterial smooth muscle more than it relaxes the veins. Because of this effect, hydralazine can cause a reflex tachycardia, which may counterproductively increase cardiac demand. Hydralazine has an onset of 5 to 20 minutes and

a clinical duration of between 2 and 6 hours. At low doses of 5 mg intravenously, reflex tachycardia is usually not clinically significant. In higher doses of 20–30 mg intravenously, reflex tachycardia can be clinically significant and in most cases is used for the purpose of increasing heart rate while lowering blood pressure. In addition, it is important to note that a synergistic effect results from the combination of phosphodiesterase inhibitors, such as sildenafil, and nitric oxide–releasing vasodilators, and they should be avoided in combination.[23] This synergistic effect can lead to profound hypotension and inadequate coronary perfusion.

HYPERTENSION AND NON-STEROIDAL ANTI-INFLAMMATORY DRUGS (NSAIDs)

NSAIDs are valuable postoperative pain medications that are commonly used in the perioperative period and can have some untoward side effects in the hypertensive patient. They have potent anti-inflammatory and analgesic properties, which are produced through a reduction of inflammatory mediators such as prostaglandins (PG) via the cyclo-oxygenase pathway.

Circulating PGs play a central role in the regulation of renal hemodynamics and renal hormone synthesis. They maintain the balance between hypertensive and antihypertensive mechanisms. The balance is manifest through thromboxane A2 and PGH2, both vasoconstrictive mediators, and through prostacyclin (PGI2) and PGE2, which are vasodilating mediators.[24] NSAIDs can impair and upset this compensatory ability and can potentially lead to a predominance of vasoconstrictors and a subsequent increase in blood pressure.[25]

The combination of NSAIDs and certain antihypertensive medications can also be problematic to varying degrees. The level of interaction between NSAIDs and antihypertensive drugs depends on how large a role PGs, particularly renal PGs, play in the mechanism of action of current hypertensive medications.[24] The efficacy of diuretics and β-blockers is usually moderately diminished with concomitant NSAID use, whereas angiotensin-converting enzyme inhibitors and angiotensin receptor blockers are more highly influenced by NSAID administration. These antihypertensive medications have been implicated in acute renal failure when coadministered with NSAIDs as well. CCBs are not dependent on the action of PGs and therefore do not significantly interact with NSAID use.[25]

It is therefore important to remember that concomitant use of NSAIDs and antihypertensives can lead to worsened blood pressure control. Both hypertension occurrence and NSAID use increase with age, and the older patient population is likely to be more predisposed to blood pressure elevation following NSAID use.[24] Potentially, this increase can be very serious, because even a relatively slight increase in blood pressure (5 mm Hg) can contribute to an increase in the occurrence of infarctions or the risk of heart failure.[26]

Anesthetic Implications

Because about 1 in 3 American adults has hypertension and approximately 50% of these have their condition under control,[27] this is a major medical condition afflicting many anesthetic patients. Proper preoperative evaluation of the hypertensive patient is essential. Preoperative hypertension is frequently a hypertensive urgency, not an emergency, as it typically does not involve overt end-organ damage or symptoms of chest pain, shortness of breath, headache, or visual changes, and there is usually adequate time to reduce the blood pressure.[28] Elevated blood pressures (eg, systolic $\geq$170 mm Hg, diastolic $\geq$110 mm Hg) have been associated with perioperative cardiac complications.[29] If antihypertensive medications were discontinued, intravenous antihypertensives can be administered or the patient rescheduled and instructed to take their antihypertensive medication before surgery. If significant hypertension is found in a nontreated patient, or a patient who had taken their usual antihypertensive medication(s), intravenous sedation may decrease anxiety-related sympathetic discharge. If this fails to bring blood pressure under control, a decision either to treat the hypertension or to reschedule the patient with physician follow-up is necessary. When emergent dental/oral surgery is necessary, excessive blood pressure elevations should be treated to prevent possible aggravation of bleeding and damage to vital organs.[30]

In long-term management of hypertension, Miller[22] recommended treatment based on 3 general beliefs: (a) the patient should be educated regarding the importance of lifelong treatment of hypertension, (b) perioperative hemodynamic fluctuations occur less frequently in treated than in untreated patients, and (c) hemodynamic fluctuations have some relation to morbidity. The more severe the hypertensive state, the greater the risk to the patient.

Vasculopathy and end-organ function are important considerations in the preoperative assessment, and in a patient in a more advanced stage of hypertension, appropriate lab work might include blood-urea nitrogen, creatinine, serum potassium, and a recent electrocardiogram.[31] Blood-urea nitrogen and creatinine levels are useful tools in assessing renal function in the hypertensive patient. Inadequate renal function can lead to a large number of clinical manifestations, including hypervolemia and hypertension. Furthermore, medications such as phenylephrine, which constrict the renal

vasculature, and other medications with active metabolites may accumulate when given in large doses or over prolonged periods of time for patients with significant renal dysfunction. Commonly used anesthetic agents with pharmacologically significant active metabolites include morphine, meperidine, and diazepam. The serum potassium levels and electrocardiogram readings are useful because many antihypertensive medications disrupt the potassium balance and can lead to significant clinical complications that range from nausea and vomiting to cardiac dysrhythmias. Initial electrocardiogram readings of hyperkalemia may show pathognomonic peaked T-waves and flattened P-waves, while hypokalemia will show the opposite findings with an increased amplitude of the P-waves and T-wave flattening or inversion. Long-standing mild hypokalemia rarely requires immediate treatment.

It is important to recognize that many chronically hypertensive patients have an upward autoregulation of their tissue perfusion pressures due to an increase in their arterial pressures.[32] During anesthesia, the cardiac depressant effect of many general anesthetic medications can cause significant reduction in systemic vascular resistance and, especially when combined with the decreased baroreflex response, can lead to large swings in blood pressure.[33] Hypertension is associated with a right shift in the physiologic autoregulation curve, particularly for cerebral blood flow. Caution is warranted as the risk for cerebral hypoperfusion and even ischemia can occur should perfusion pressures decrease during anesthesia administration.[8] In this setting, even an "optimal" blood pressure less than 120/80 may be inadequate for critical organ perfusion.

Induction of anesthesia should be carefully managed for hypertensive patients. The typical drugs used for induction such as propofol and fentanyl are acceptable, particularly if titrated slowly. Hemodynamic changes with induction most likely reflect unmasking of decreased intravascular fluid volume due to chronic hypertension combined with a stiffening of the arterial vasculature.[8] During direct laryngoscopy, there may be a strong sympathetic response that should be avoided with proper analgesic drugs, β-blockers, intravenous lidocaine, or other strategies. Some providers recommend administering topical laryngotracheal lidocaine to blunt the pressor response to airway and tracheal stimulation. Intraoperative blood pressure variations are common during surgery, and the anesthesia provider must be aware of fluctuations, particularly with the chronically hypertensive patient.[22] During anesthesia, the goal is to prevent extreme fluctuations in blood pressure, since hypertensive patients can exhibit exaggerated responses to anesthetic drugs and surgical stimulation.[34] These hemodynamic responses may be due to the cardiac depressant effect of many general anesthetic medications, which cause a large reduction in systemic vascular resistance coupled with a decreased baroreflex response.[4] Therefore, it is important to anticipate exaggerated blood pressure changes during induction and through the maintenance of sedation or general anesthesia. Caution is warranted with ketamine use because of the increase in heart rate and blood pressure that may accompany a ketamine induction.

There are no accepted definitions of proper intraoperative blood pressure levels despite a widespread belief that intraoperative management of blood pressure will positively affect postoperative outcomes. In one study by Monk and colleagues,[35] it was concluded that there is strong evidence that excessive intraoperative hypotension (systolic blood pressure <70 mm Hg, mean arterial pressure < 50 mm Hg, and diastolic blood pressure < 30 mm Hg), but not hypertension per se, is associated with increased mortality. This suggests that when a normotensive blood pressure is difficult to maintain during the intraoperative period, it may be safer to keep the patient's blood pressure somewhat higher rather than frankly hypotensive.[22]

Postoperatively, many hypertensive patients will return to their preoperative blood pressure levels. Some providers prophylactically treat for hypertension immediately after emergence. A good starting point for treatment after emergence may include returning to the oral regimen that the patient takes, particularly if they withheld medication for anesthesia and surgery. If a more immediate antihypertensive medication is needed in recovery, then an appropriate bridging medication might be an intravenous β-blocking agent, such as labetalol, or a vasodilator such as enalaprilat or hydralazine based on heart rate. When bridging antihypertensive medications from the anesthetic period to the regular home-dosing regimen, it is important to pay close attention to the pharmacokinetic profile of intravenously administered medications when recommending restarting of home antihypertensives. Orthostatic hypotension, especially when opioids are also used, should be explained. If a patient needs medications to control blood pressure and heart rate while at home, they should be encouraged to maintain their use postoperatively.[8]

CONCLUSION

The goal of long-term antihypertensive treatment is to reduce overall cardiovascular disease risk and thus its morbidity and mortality rates. The treatment is typically initiated with thiazide diuretics, aldosterone antagonists, angiotensin-converting enzyme inhibitors, angiotensin receptor blockers, or CCBs. When significant blood pressure modifications are needed, a combination of the

previous drugs are given and/or a β-blocker can be added. Fluctuations in blood pressure intraoperatively are common. Careful management of blood pressure, minimizing periods of significant hypertension or hypotension, is a cornerstone of anesthetic management.

The treatment of hypertension is highly variable, but common hypertensive medications should be well understood by the clinical anesthesia provider. β- and α-Blocking agents, α-2 agonists, and CCBs should be continued on the morning of surgery for sedation or general anesthesia. Rebound hypertension is a particular concern with a missed dose of β- and/or α-blocking agents and the α-2 agonists. Angiotensin-converting enzyme inhibitors, angiotensin receptor blockers, and direct renin inhibitors may be discontinued prior to general anesthesia because of the risks of encountering refractory hypotension during induction. Maintaining blood pressure within physiologic parameters for a given patient based on preoperative levels has always been a mainstay of anesthetic management. Avoiding significant hypotension is particularly important in preventing anesthetic complications.

With a proper understanding of hypertension and its anesthetic implications, the anesthesia provider is well equipped to give the hypertensive patient quality care while adhering to the current hypertensive medication recommendations.

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CONTINUING EDUCATION QUESTIONS

This continuing education (CE) program is designed for dentists who desire to advance their understanding of pain and anxiety control in clinical practice. After reading the designated article, the participant should be able to evaluate and utilize the information appropriately in providing patient care.

The American Dental Society of Anesthesiology (ADSA) is accredited by the American Dental Association and Academy of General Dentistry to sponsor CE for dentists and will award CE credit for each article completed. You must answer 3 of the 4 questions correctly to receive credit.

Submit your answers online at www.adsahome.org. Click on "On Demand CE."

CE questions must be completed within 3 months and prior to the next issue.

1. Why is a calcium channel blocker (CCB) medication that is used for hypertension treatment inappropriate for use during an malignant hyperthermia crisis?
   A. CCBs are ineffective at blocking calcium in skeletal muscle
   B. CCBs inhibit the thermoregulatory centers
   C. CCBs increase the potential for cardiac collapse after dantrolene administration
   D. CCBs accelerate the increase in $EtCO_2$
2. Which of the following antihypertensive medications does NOT increase the risk of renal complications with concomitant use with NSAIDS?
   A. CCBs
   B. β-blockers
   C. Angiotensin-converting enzyme inhibitors
   D. Angiotensin receptor blockers
3. Anesthesia providers should maintain mean arterial pressures of hypertensive patients at higher levels than patients with normal blood pressure. The reason for this is because hypertensive patients typically have a:
   A. Left shift of the autoregulation curve
   B. Right shift of the autoregulation curve
   C. Left shift of the oxygen-hemoglobin dissociation curve
   D. Right shift of the oxygen-hemoglobin dissociation curve
4. Which of the following medications may be held in the perioperative period prior to induction to general anesthesia to avoid possible refractory hypotension?
   A. β-blockers
   B. CCBs
   C. α-2 agonists
   D. Angiotensin receptor blockers