Hypertension in the surgical patient: management of blood pressure and anesthesia

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■ Although hypertension has always been a high risk factor during anesthesia and surgery, risk can be reduced by preoperative control of blood pressure, evaluation of the patient to determine risk factors that can exacerbate blood pressure rises, and continuation of preoperative antihypertensive therapy. Patient hemodynamics must be monitored to manage blood pressure fluctuations and signs of ischemia as early as possible. Selection of anesthetic and adjuvant agents must be tailored to the patients, the agents must be administered carefully and in a timely fashion, and the anesthesiologist must be aware of the relevant variables. Increased knowledge of the pathophysiology of hypertension, antihypertensive therapy, and the development of new anesthetics and muscle relaxants with minimum hemodynamic effects has helped minimize complications related to perioperative hypertension.

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THE INCIDENCE of clinical hypertension has decreased significantly in the last decade, probably due to increased awareness of food habits and other factors that predispose to hypertension. During the same time, new and more effective antihypertensive agents with fewer side effects have also been developed.

Hypertension has always been a high risk factor during anesthesia and surgery. As early as 1929, Sprague[1] reported a mortality of 30% in hypertensive patients undergoing surgery, and several later reports also emphasized the high risk of anesthesia and surgery in these patients.[2,3] More recently, Prys-Roberts and Meloche[4] emphasized the differences in risk between patients with controlled and uncontrolled hypertension. Also, in the last decade, new anesthetic agents, particularly opioids and muscle relaxants, have been developed. Many have minimal undesirable hemodynamic effects. These agents, together with the advanced experience of anesthesiologists in managing patients with heart disease, have reduced morbidity related to anesthesia and surgery.

However, the risks of anesthesia and surgery in hypertensive patients can't be overemphasized. Among the multiple risk factors are:

1. The effect of hypertension on the heart as a target organ,
2. The association between hypertension, coronary artery disease, peripheral vascular diseases, and diabetes,
3. The effect of hypertension on cerebral and renal circulation,
4. Preoperative antihypertensive drugs and their interactions with anesthetic agents,
5. Undiagnosed diseases that cause hypertension, and

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TABLE 1
POSSIBLE CAUSES OF HYPERTENSION

Essential hypertension (80% to 85%). No definite cause is identified, however, these factors have some correlations to the incidence of essential hypertension:
Heredity
Salt intake
Disturbance of renin-angiotensin system
Disturbance of sympathetic nervous system controls
Secondary hypertension (15% to 20%). Possible causes can be identified:
Kidney disease
Acute and chronic infection
Ischemia of renal tissue (renal artery stenosis, trauma)
Congenital abnormalities (polycystic kidney
Connective and collagen disease (polyarteritis nodosa, disseminated lupus, etc.
Endocrine disease
Hyperaldosteronism (Conn's syndrome)
Pheochromocytoma
Toxemia of pregnancy
Neurogenic causes
Sympatho-adrenal hyperactivity (tetanus)
Stimulation of sympathetic cardiovascular reflexes (postmyocardial revascularization hypertension)
Coarctation of the aorta

6. A need for life-saving surgery in patients with uncontrolled hypertension or patients undergoing hypertensive crises.

HYPERTENSION AND THE HEART AS A TARGET ORGAN

In cardiogenic hypertension the heart itself can be the cause of increased blood pressure; however, more commonly, the heart is a target organ during hypertension. As hypertension causes left ventricular hypertrophy, the heart becomes more vulnerable to subendocardial ischemia during hypotensive and hypertensive episodes. Congestive heart failure occurs more frequently in patients with arterial hypertension and causes a higher mortality.

HYPERTENSION AND CORONARY ARTERY DISEASE

Hypertension is a leading cause of ischemic heart disease. Levels of hypertension correlate directly with atherosclerotic changes in both coronary and systemic circulation.[5] The frequency of angina and myocardial infarction is twice as high in hypertensive patients as in normotensives. The progress of coronary artery disease is worse in hypertensive patients. Recent controlled trials failed to show that treating hypertension reduces mortality due to coronary artery disease.[5] In turn, acute coronary artery insufficiency and early stages of acute myocardial infarction are frequently complicated by acute and severe hypertension. Also, early postoperative hypertension has been reported in more than 35% of patients undergoing myocardial revascularization surgery.

The close association between coronary artery disease, hypertension, and myocardial hypertrophy explains the high risk of perioperative myocardial ischemia and myocardial infarction in these patients. In all hypertensive patients, anesthesia should be managed in a manner similar to that for patients with coronary artery disease.

Patients with peripheral vascular disease also have a very high incidence of coronary artery disease and diabetes. As many as 55% of patients who undergo surgery for peripheral vascular disease are hypertensive and up to 61% have definite coronary artery disease, frequently severe enough to require myocardial revascularization surgery.[6]

HEMODYNAMIC CHARACTERISTICS OF HYPERTENSION IN RELATION TO ANESTHESIA AND SURGERY

Traditionally, hypertension is classified as either primary hypertension or secondary hypertension with a known etiology (*Table 1*). These classifications do not take into consideration the hemodynamic characteristics of hypertension while successful antihypertensive therapy is directed towards these hemodynamic characteristics.

Almost all anesthetic agents, premedications, and muscle relaxants have hemodynamic effects that can alter the blood pressure significantly. Moreover, hypertensive patients are usually treated with more than one drug, which in itself can cause variable interactions and significant hemodynamic changes. Therefore, anesthesia that is managed according to the hemodynamic characteristics of hypertension will be safer and accompanied by minimal complications.

Blood volume and hypertension

In hypertensive patients, blood pressure fluctuations can be very closely related to blood volume changes. In patients with chronic renal failure, the blood pressure rises as the blood volume increases due to water retention. The blood pressure of these patients drops during dialysis as the blood volume is reduced. A minority of patients with renal disease have malignant hypertension that is due to the activation of the renal pressor system (renin dependent), and this type of hypertension is not controlled by dialysis. Hypertension secondary to primary aldosteronism is frequently associated with hypervolemia[7] and responds to diuretic therapy to reduce plasma volume.

When sympathetic blocking agents (other than beta blockers) are used to reduce the arterial pressure, the latter becomes directly related to the intravascular and plasma volumes. The degree of sympathetic blockade is inversely related to the plasma volume; the higher the degree of sympathetic blockade the more the arterial pressure becomes volume dependent. This explains why hypertensive patients receiving sympathetic blockers are sensitive to acute blood volume changes such as those due to blood loss or excessive transfusion during anesthesia and surgery. In patients with hypervolemic essential hypertension, diuretics are frequently used as one component of antihypertensive therapy. Patients receiving diuretics can have hypovolemia and hemoconcentration when they are admitted to surgery. Meanwhile, both induction of anesthesia and administration of muscle relaxants, which reduce both smooth and skeletal muscle tones, decrease the venous return and cardiac output in a hypovolemic patient.

Hyperkinetic hypertension

This type of hypertension is characterized by increased cardiac activity in the form of increased heart rate and myocardial contractility,[8] as evidenced by increased rate and rise of pressure. In these patients the cardiac output and the mean rate of left ventricular ejection are not necessarily increased. These hemodynamic changes are frequent in early stages of essential hypertension, patients who have renal artery disease, patients with primary aldosteronism, and patients receiving vasodilator therapy. In the latter group, the therapy may increase the heart rate, which in turn increases the cardiac output and decreases the effectiveness of antihypertensive therapy. In general, hyperkinetic hypertension responds better to beta blocker therapy.

Patients with hyperkinetic hypertension are expected to be more vulnerable to tachycardia and further blood pressure rises. Therefore, anesthetic agents and relaxants that are known to increase the heart rate (such as pancuronium bromide) should be avoided. Extra effort must be made to minimize the hypertensive responses to intubation and sympathetic stimulation. Opioid anesthetics minimize these stress responses. Inhalational anesthetics such as halothane or enflurane can be used in appropriate concentrations during maintenance to control blood pressure rises.

Decreased aortic distensibility and hypertension

In elderly hypertensive patients, aortic distensibility is reduced, and this causes a wide pulse pressure and reduced stroke volume. As the elasticity of the aorta is lost, there will be wide swings of pressure with each cardiac cycle.[9] If the diastolic pressure increases in these patients, the aortic distensibility is further reduced and the systolic blood pressure will be much higher for similar levels of diastolic pressure.

In this type of hypertension, marked blood pressure fluctuations occur in response to small doses of vasoactive drugs, anesthetic agents, and changes in muscle tone. Therefore, anesthetic agents, vasoactive agents, dilators, and pressors should be administered slowly, and patience is required to minimize blood pressure fluctuations.

Increased systemic vascular resistance

Increased systemic vascular resistance (SVR) is the main hemodynamic characteristic of established essential hypertension. In the early stages, the increased SVR is due to the increased neurogenic activity. Later, arteriolar hypertrophy is the main cause of the high SVR.[10] Large numbers of patients with chronic hypertension may be receiving diuretics, and therefore may be relatively dehydrated and have reduced blood volume. This, together with the increased SVR, explains the low cardiac output in patients with chronic essential hypertension. Sudden vasodilation, as induced by barbiturates or large concentrations of inhalational anesthetics, can cause severe hypotension with all its consequences.

PREOPERATIVE ANTIHYPERTENSIVE THERAPY AND BLOOD PRESSURE CONTROL

In the last two decades, we have learned more about the advantages and side effects of continuing and discontinuing antihypertensive therapy, the interactions between antihypertensive agents and anesthetics, and the direct relationship between blood pressure control and decreased mortality and morbidity. This increased knowledge has confirmed the need to maintain antihypertensive therapy until time for anesthesia and surgery.

Sympatholytic antihypertensive agents

Sympatholytic antihypertensive agents are the most effective agents (secondary to diuretics) for treatment of high blood pressure. They include autonomic ganglion blockers (eg, decamethonium), central adrenergic inhibitors (eg, clonidine), specific adrenergic nerve inhibitors (eg, guanethidine), mixed central and peripheral adrenergic nerve inhibitors (eg, reserpine), inhibitors of catecholamine synthesis (eg, metyrosine), and adrenergic receptor blockers (alpha blockers [eg, phentolamine], beta blockers [eg, propranolol], and alpha and beta blockers [eg, labetalol]).

Currently, adrenergic receptor blockers and central adrenergic inhibitors are the most commonly used of these drugs.

Reserpine and guanethidine. They are centrally active, reduce the requirement for analgesics, depressants, and tranquilizers, and sensitize the patient to the effects of alcohol.[11] These agents are known to cause exaggerated reaction to inhalational anesthetic agents (such as halothane), leading to peripheral vasodilation, hypotension, and myocardial depression.

Blood pressure of patients receiving reserpine or guanethidine is very sensitive to blood volume fluctuations and intravascular volume changes. Such fluctuations are very common during surgery because of blood loss or transfusion. Also, anesthetic agents produce relative blood volume changes by producing vasodilation and muscle relaxation, which explains why hypotension during anesthesia and surgery is very common in patients receiving these agents. The use of sympathomimetic vasopressors to treat hypotension in patients receiving reserpine and guanethidine can cause severe hypertension because both cause marked sensitivity to catecholamines.[11,12] The severe blood volume fluctuations during anesthesia and surgery in patients receiving these agents were the main reason for discontinuing them preoperatively. In turn, this led to the added risk of uncontrolled hypertension.

The use of this group of drugs has decreased significantly in the last decade because of their symptomatic side effects.

Clonidine. Clonidine's actions resemble those of methyl dopamine. It is a central antihypertensive agent that stimulates alpha$_2$-adrenergic actions. It also has partial peripheral adrenergic inhibitor properties. It reduces the heart rate and cardiac output as it reduces the sympathetic discharge from both cardiac and splanchnic nerves. It also reduces neurovascular resistance.

Abrupt withdrawal of clonidine leads to severe undesirable hypertensive reactions,[13] which may occur intraoperatively and/or postoperatively. Therefore, physicians at the Cleveland Clinic recommend continuation of clonidine until the day of surgery.

Beta adrenergic blockers. Soon after the development of beta adrenergic blockers, a few cases of severe myocardial depression were reported in patients receiving beta blockers and undergoing cardiac surgery,[14] and this led to a recommendation to discontinue these agents two weeks prior to anesthesia and surgery.

Thereafter, it became clear that discontinuation of beta blockers leads to a hypersympathetic state, rebound hypertension, as well as acute myocardial ischemia and myocardial infarction. As the agents were used with restrictions in patients with weakened myocardia and anesthesiologists became more knowledgeable in managing patients receiving beta blockers, it became clear that there is no need to discontinue these drugs. On the contrary, the current practice in our institution is to continue beta-blocker therapy until the day of surgery and allow the patient to have the therapy the morning of surgery. These drugs are also used intraoperatively to minimize and treat hypertensive episodes accompanied by tachycardia and to treat dysrhythmias and ECG signs of ischemia. Short-acting beta blockers such as esmolol can be used to control postoperative hypertension by themselves or to augment the use of sodium nitroprusside.

Calcium channel blockers

Calcium channel blockers are myogenic vasodilators. They prevent vasoconstriction by interfering with calcium ion movements in the muscle fibers and by interfering with the common pathway of vasoconstriction, and they can block the response to sympathetic stimulation. Calcium channel blockers are also myocardial depressants. Currently, a large number of hypertensive patients, particularly those who have coronary artery disease, are receiving both calcium channel and beta blocking drugs. These two drugs have additive effects on both the heart rate and myocardial contractility. Calcium channel blockers potentiate the myocardial depressant effects of inhalational anesthetic agents such as halothane. They also potentiate the bradycardiac effects of the potent opioids fentanyl and sufentanil, which are frequently used as anesthetic agents.

Therefore, patients receiving both calcium channel and beta blockers may be more vulnerable to episodes of bradycardia and hypotension. Bradycardia due to the interaction between calcium channel blockers and opioids is reversible by atropine.[15] However, hypotension related to calcium channel blockers responds to pure alpha$_1$ pressors such as methoxamine and does not respond to the same extent to alpha$_2$ pressors.[16] Therefore, when hypotension occurs in patients receiving calcium channel blockers, larger doses than usual of a drug such as norepinephrine (alpha$_1$ and alpha$_2$ pressor) are required.

We recommend the continuation of calcium channel blocker therapy to the day of surgery, but we do not recommend its frequent use intraoperatively because of the potential hypotension. As with all patients receiving antihypertensive therapy, patients receiving calcium channel blockers are more vulnerable to blood pressure fluctuations during anesthesia and surgery.

Converting enzyme inhibitors

Captopril decreases blood pressure by decreasing SVR with minimal effect on cardiac output. Unlike calcium channel and beta blockers, discontinuation of converting enzyme inhibitors does not cause rebound hypertension. However, we recommend that patients receive the evening dose of the drug and then continue therapy after surgery.

Diuretic agents

Chronic diuretic therapy causes reduction in serum potassium level as well as reducing blood volume.[17] Direct and indirect blood and fluid loss are more common during anesthesia and surgery. The work of the kidneys is much higher in dehydrated than overhydrated patients, which explains the greater possibility of renal dysfunction in dehydrated patients. Therefore, intraoperative hydration is desirable for patients receiving diuretics. Attempts to hydrate the patient with fluids that contain low levels of potassium or attempts to enhance diuresis by further use of diuretics during surgery can significantly decrease serum potassium levels. Diuretics are the only antihypertensive drug for which we recommend discontinuation a day or two prior to surgery when possible. This will lead to rebound fluid retention and minimize blood volume and serum potassium level changes.

Preoperative evaluation

Extensive preoperative evaluation is mandatory prior to anesthesia and surgery, especially in patients with preoperative hypertension. Special attention should be paid to the following:

1. The severity of hypertension, since preoperative hypertension levels are directly related to perioperative mortality and morbidity,[18]
2. The primary cause of hypertension, eg, coarctation of the aorta, pheochromocytoma, or other catecholamine-secreting tumors,
3. The extent of involvement of the heart as a target organ (signs of hypertrophy on ECG, chest radiograph, echocardiogram),
4. Signs and symptoms of accompanying coronary artery disease,
5. The adequacy of cerebral circulation and history of stroke,
6. The peripheral and renal circulation and renal function,
7. Preoperative antihypertensive treatment, assessment of possible side effects, and interaction with anesthetic agents (*Table 2*), and

TABLE 2
PREOPERATIVE ANTIHYPERTENSIVE THERAPY AND ANESTHESIA

Rauwolfia	
	Interaction with vasopressors
	Vulnerability to hypertension
Beta blockers	
	Extent of beta sympathetic blockade
	Heart rate, hand clasp test, isuprel test
Calcium channel blockers	
	Effect of heart rate
	Calcium channel blockers as long-acting arteriolodilators
Diuretic agents	
	Contracted blood volume
	Serum potassium levels
Clonidine	
	Potentiation of anesthetic agents

8. The hemodynamic profile of hypertension and its relevance to anesthesia (*Table 1*).

UNDIAGNOSED CAUSES OF HYPERTENSION AND ACCOMPANYING DISEASE

Other than latent and extrarenal pheochromocytomas, several rare chromaffin-tissue, catecholamine-secreting tumors, such as tumors of the aortic and ciliary bodies or glomus jugular, can also cause hypertension. These tumors can be asymptomatic and pass undiagnosed for some time. The stress of anesthesia and surgery stimulates these tumors, releasing large amounts of catecholamines suddenly and causing unexpected hypertensive crises with all their sequelae.[19] A recent history of paroxysmal hypertension, frequent headaches, palpitations, tachycardia, tremors, or a general history of hypermetabolism in the absence of hyperthyroidism and the presence of hypertensive retinopathy should signal the possibility of a latent catecholamine-secreting tumor.

REQUIRED SURGERY

The risks of anesthesia and surgery to patients experiencing hypertensive urgencies and emergencies (as defined by the Joint National Committee [JNC] on Detection, Evaluation, and Treatment of High Blood Pressure[20] [*Table 3*]), as well as to patients with uncontrolled hypertension, are serious, and include higher incidence of myocardial ischemia, infarction, and cerebral hemorrhage. Therefore, it is logical to recommend postponement of elective surgery on these patients as long as needed to control the blood pressure. However, emergency lifesaving surgery may be required. Such surgery may even be part of the treatment of hypertensive emer-

TABLE 3
SOME CAUSES OF HYPERTENSIVE EMERGENCIES AND URGENCIES

Severe uncontrolled hypertension
Malignant hypertension
Acute aortic dissection
Pheochromocytoma and other catecholamine-secreting tumors
Severe hypertension in kidney transplant patients
Eclampsia
Drug interactions (eg, MAO inhibitors)
Increased intercranial pressure, trauma, hemorrhage
Early stages of acute myocardial ischemic infarction

gencies, as in cases of increased intracranial pressure, eclampsia, iatrogenic renal ischemia, and even acute myocardial ischemia. For those who must undergo surgery, the following protocol may provide a practical answer.

Management of hypertensive rises in a surgical setup

In emergencies, short-acting potent antihypertensive agents are preferable because changes in SVR and circulating blood volumes are fast and abrupt. Short-acting agents have an immediate effect and can be reduced, withdrawn, or increased in response to the magnitude of blood pressure changes. There is no definite agreement as to how much the blood pressure should be reduced. A diastolic pressure of 100–110 mmHg has been recommended as a lower limit to ensure adequate cerebral and renal circulation.[21] In patients with dissecting aortic aneurysms, it is advisable not to lower the diastolic blood pressure below 100 mmHg,[21] but there is no agreement as to how much the blood pressure should be lowered in patients with subarachnoid hemorrhage.[18] Antihypertensive therapy should not be continued solely to achieve an arbitrary level of blood pressure control, as this level may not be feasible and continuation of excessive drug therapy may produce undesirable side effects. The most worrisome complication of acute reductions in blood pressure is central nervous system complication,[21,22] mainly in the form of cerebral ischemia.

Severe rises in blood pressure are most often accompanied by increased SVR with normal or contracted blood volume. Therefore, intravenous peripheral vasodilators are used most frequently.

Peripheral vasodilators. Sodium nitroprusside is the most commonly used drug for control of hypertensive crises. It is a peripheral vascular dilator that acts on the smooth muscles of both veins and arteries, and it helps to decrease both the preload and afterload.[23,24] It produces a drop in blood pressure within seconds; however, continuous monitoring is necessary. It is usually administered by a continuous infusion pump (5 μg/kg/min) with the doses adjusted according to the response of the blood pressure. Recently, computerized controllers of blood pressure have been developed to administer sodium nitroprusside automatically. Sodium nitroprusside causes preferential redistribution of small-vessel coronary blood flow; therefore, it is not the ideal agent during myocardial ischemia.[25] Sodium nitroprusside should not be administered for a prolonged period because of the possibility of cyanide toxicity.

Although nitroglycerin is not as potent an arteriodilator as sodium nitroprusside, it is a more potent venodilator. It is a rapid-acting vasodilator that produces its effect within a few seconds. Usually, large concentrations of nitroglycerin are required to lower the blood pressure (10–20 μg/min).[26] It is the preferred vasodilator in the presence of myocardial ischemia because of its specific vasodilator effect.[25]

Labetalol is both an alpha- and beta-adrenergic blocking agent. It lowers the SVR as well as slowing the heart rate with minimum changes in the cardiac output.[27] In patients with coronary artery disease, it has the advantage of not causing tachycardia or selective redistribution of coronary blood flow, and it decreases myocardial oxygen consumption. During intravenous use of labetalol, the beta-blocking activity dominates the alpha-blocking activity,[28] and it can reduce the cardiac output and the cardiac index. It is not the drug of choice in patients with congestive heart failure or atrioventricular block.[29,30] Other side effects of labetalol include nausea and vomiting. Labetalol can be injected intermittently or by continuous infusion, similar to sodium nitroprusside and nitroglycerin, at a rate of 0.5–2 mg/min. Total doses of labetalol should not exceed 300 mg.

Peripheral vasodilators combined with beta blockers. These two groups can be combined to get a significant reduction in SVR by the peripheral vasodilators while preventing or minimizing the reflex tachycardia by the beta blockers. This combination allows the use of smaller doses of beta blockers to minimize their effect on myocardial contractility. It is particularly valuable in patients suffering acute dissection of aortic aneurysms, because lowering the heart rate and blood pressure decreases the aortic wall stress. Such combinations are being used successfully in hypotensive anesthesia techniques.

After immediate blood pressure control is achieved, continuous IV infusion should be discontinued gradually while long-term antihypertensive treatment is initiated.

Calcium channel blockers. Nifedipine is the most

potent peripheral vasodilator among calcium channel blockers. However, it causes a compensatory increase in the heart rate.[31] It is an effective treatment for hypertensive crises because of its rapid onset of action (20–30 minutes) after oral administration.[32] Because of the prolonged half-life of nifedipine, one cannot predict its effect on the blood pressure changes that occur during anesthesia and surgery. Also, since it is administered orally, it is not a drug of choice to lower the blood pressure preoperatively.

Intraoperative monitoring of the hypertensive patient

Preoperative hypertension is an indication for careful monitoring, since blood pressure fluctuations (hypertension and hypotension) are common. The minimum monitoring requirements are reliable frequent blood pressure monitoring to detect blood pressure changes immediately, and continuous ECG monitoring to detect early signs of ischemia because of the high incidence of associated coronary artery disease. For patients undergoing major and prolonged surgery, it is preferable to monitor the blood pressure through an arterial line and ECG lead five, which detects ischemia in about 80% of the instances.

In extensive surgical procedures, hypertensive emergencies, and in the presence of severe myocardial involvement, it is useful to have a PA inserted to monitor the cardiac output changes in peripheral vascular resistance and the filling pressures of both sides of the heart.[33]

Management of anesthesia

The risks of anesthesia are high in hypertensive patients because these patients are more vulnerable to blood pressure fluctuations in response to anesthetic agents, vasopressors, and vasodilators. These fluctuations can lead to cerebral and myocardial ischemia. The baroreceptor reflex may be obtunded, particularly in older patients. Anesthetic agents and muscle relaxants have variable hemodynamic properties and are usually administered in significant amounts over a short period of time to induce anesthesia. Laryngoscopy and endotracheal intubation produce significant sympathetic stimulation and can lead to rises in both heart rate and blood pressure. Thereafter, initiating positive pressure ventilation decreases the venous return and can cause a drop in blood pressure in a patient who may have contracted blood volume. Hypocapnia can further decrease serum potassium levels, which may already be reduced due to diuretic therapy.[17] Principles of anesthesia management for hypertensive patients are:

1. Select the anesthetic agents and muscle relaxants whose hemodynamic effects produce minimal blood pressure fluctuations when administered to a hypertensive patient with a specific hemodynamic characteristic.
2. Be aware of preoperative drug therapy and its interactions with anesthesia management. The effect of all anesthetics used is usually exaggerated in the hypertensive patient, producing either rise or drop in the blood pressure.
3. Minimize direct and indirect blood volume changes.
4. Monitor the patient to detect significant blood pressure changes and signs of ischemia early enough to treat such changes immediately.

Agents. Barbiturates cause dose-related myocardial depression and decrease the cardiac output and blood pressure. The compensatory increase in heart rate and SVR that usually occurs may well be obtunded because of the drop in baroreflex and the preoperative drug therapy (beta and calcium channel blockade).[34]

Benzodiazepines are used more frequently as premedicants to provide sedation and amnesia, and occasionally as a component of inducing agents. When administered alone in small doses, this group of drugs causes minimal changes in myocardial contractility, heart rate, and blood pressure.[35] Using them as strong sedatives and amnesics minimizes the neurologic element of blood pressure rise due to patient apprehension. When benzodiazepines are combined with barbiturates, they can cause hypotension and myocardial depression.[36,37]

New potent opioids such as fentanyl and sufentanil do not depress the myocardium, and cause minimal changes in blood pressure.[33,38] Large doses of opioids suppress the release of catecholamines in response to sympathetic stimulation.[39] Large doses of opioids together with the appropriate muscle relaxant can be the best combination to minimize hemodynamic changes in the hypertensive patient. Large doses of opioids do not produce prolonged postoperative respiratory depression that requires artificial ventilation. Newer potent short-acting opioids such as alfentanil still have to be tested in hypertensive patients.

Large concentrations of inhalational agents cause hypotension and lability of blood pressure.[40] They do not suppress sympathetic responses (hypertension and tachycardia) to stimulation or endotracheal intubation. These agents can be used in low concentrations during surgery and are effective in lowering high blood pressure and decreasing myocardial O_2 consumption.

Muscle relaxants decrease the muscle tone, reduce venous return, and can cause a drop in blood pressure in

a hypovolemic patient. Preoperative ganglion blockers potentiate the effects of nondepolarizing muscle relaxants.[41] Various muscle relaxants have different effects on the heart rate and blood pressure. While pancuronium increases heart rate and blood pressure, d-tubocurarine increases the heart rate and reduces SVR and blood pressure. Relaxants such as vecuronium and atracurium alone have minimum cardiovascular effects. However, large doses of atracurium cause histamine release.

It is clear from this summary of the hemodynamic effects of the drugs used in anesthesia that there is no ideal agent that stabilizes hemodynamics or at least does not interfere with patient hemodynamics and interact with other drugs.

Techniques. Local and regional techniques are valuable because they are accompanied by lower risk when they are used for minor surgical procedures, particularly those on the extremities.

Spinal and epidural techniques provide a good alternative to general anesthesia for lower abdominal, pelvic, or lower limb surgery. The hemodynamic effect of spinal and epidural anesthesia varies greatly between controlled and uncontrolled hypertension and is related to the circulating blood volume prior to surgery. Patients with controlled hypertension tolerate lumbar and lower thoracic blocks well, with a moderate drop in blood pressure (18%–24%).[42] Patients with uncontrolled hypertension have a greater decrease in blood pressure and more frequent blood pressure fluctuations in response to stimulation and blood loss.

Patients receiving spinal and epidural anesthesia require appropriate replacement of lost blood and fluid. Fluid should be used to ensure adequate circulating volume and perfusion pressure when vasodilation is induced by spinal or epidural blocks.

It is evident from the above discussion that vasoactive agents are frequently needed to manage undesirable changes in heart rate, rhythm, and blood pressure promptly. Vasoactive drugs must be administered with special care to hypertensive patients, who react in an exaggerated manner to most of these agents. Therefore, the selection of anesthetic and adjuvant agents must be tailored to the patients, the agents must be administered carefully and in a timely fashion, and the anesthesiologist must be aware of all the variables discussed.

CONCLUSION

Perioperative hypertension is a definite risk during anesthesia and surgery and postoperatively. This risk can be reduced by preoperative control of the blood pressure, evaluation of the patient to determine the risk factors that can exacerbate blood pressure rises, and continuation of preoperative antihypertensive therapy, which minimizes blood pressure rises. The choice of anesthetic agents should be considered in relation to the hemodynamics of hypertension, interactions with preoperative therapy, and blood volume changes. Anesthesia management should also be directed toward preserving myocardial and cerebral circulation. Surgical procedures or disease that may increase the incidence of intraoperative and postoperative hypertension must be identified preoperatively and the anesthesiologist must be aware of the possible timing of blood rises to try to prevent them or handle them in a timely fashion. Patient hemodynamics must be monitored to manage blood pressure fluctuations and signs of ischemia as early as possible.

Increased knowledge of the pathophysiology of hypertension, antihypertensive therapy, and the development of new anesthetics and muscle relaxants with minimum hemodynamic effects has definitely helped anesthesiologists to manage anesthesia better and minimize the complications related to perioperative hypertension.

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