Induced Hypotension During Anesthesia, with Special Reference to Orthognathic Surgery

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Since Gardner first used arteriotomy during anesthesia to improve visibility in the surgical field, various techniques and pharmacological agents have been tried for the same purpose. With reports documenting the spread of acquired immune deficiency syndrome through blood transfusions, prevention of homologous blood transfusions during surgery has also become a major concern. Induced hypotension has been used to reduce blood loss and thereby address both issues. In orthognathic surgery, induced hypotension during anesthesia has been used for similar reasons. It is recommended that hypotensive anesthesia be adjusted in relation to the patient's preoperative blood pressure rather than to a specific target pressure and be limited to that level necessary to reduce bleeding in the surgical field and in duration to that part of the surgical procedure deemed to benefit by it. A mean arterial blood pressure (MAP) 30% below a patient's usual MAP, with a minimum MAP of 50 mm Hg in ASA Class I patients and a MAP not less than 80 mm Hg in the elderly, is suggested to be clinically acceptable. Various pharmacological agents have been used for induced hypotension during orthognathic surgery. In addition, there are many drugs that have been used in other types of surgery that could be used in orthognathic surgery to induce hypotension. Recent reports using control groups do not show significant differences in morbidity and mortality attributable to induced hypotension during anesthesia. Appropriate patient evaluation and selection, proper positioning and monitoring, and adequate fluid therapy are stressed as important considerations in patients undergoing induced hypotension during orthognathic surgery.

Orthognathic surgery involves the surgical correction of oral and facial deformities involving bones and soft tissues. There is significant bleeding from both incised soft tissues and bones during these procedures due to the rich blood supply to the areas involved. Considerable bleeding from the bone occurs because most of the vessels traversing the bones cannot be identified and isolated before or after oseous sectioning. In addition, severe bleeding may occur during maxillary osteotomies from damage to the descending palatine artery, maxillary artery, pterygoid venous plexus, or occasionally from damage to the second part of the maxillary artery. Severe bleeding can also occur during mandibular osteotomies from damage to the masseteric artery, retromandibular vein, pterygoid venous plexus, or occasionally from damage to the facial artery. Furthermore, due to limited access, poor visibility to isolate and ligate or cauterize vessels that otherwise would have been ligated or cauterized with ease adds to the problem of bleeding during orthognathic surgery, frequently obscuring the operative field.

In order to help the surgeon reduce bleeding and to improve visibility in the operative field, many anesthetists have employed induced hypotension during orthognathic surgery. Induced (or controlled) hypotension is a method by which the arterial pressure is decreased in a predictable and deliberate manner. Unlike in procedures such as aortic surgery, where hypotensive anesthesia is often employed and where the bleeding vessel can often be identified, in orthognathic surgery bleeding vessels may not be identified. Although it is similar to lumbar surgery in this respect, orthognathic surgery differs from the latter in that large areas of medullary bone are exposed during orthognathic surgery, which contributes to the blood loss. Patients who undergo aortic and lumbar surgery are often old and medically compromised, whereas patients who receive orthognathic surgery are usually young and healthy. Thus, the risks of hypotensive anesthesia in orthognathic surgery are far less than in aortic or lumbar surgery.

Several controlled1-4 and uncontrolled studies5 have demonstrated that induced hypotension improves the quality of the surgical field in orthognathic surgery. One study, by Fromme et al,6 disputes this finding. In this study, though the assessment was by surgeons who were
blind to the method of anesthesia, the comparison was of operations on both maxillae and mandibles. The specific operations were not stated. The degree of blood loss during different maxillary and mandibular osteotomies may differ. Furthermore, the frequency or the time of assessment, which may influence the outcome, was not stated. In a study conducted on patients undergoing specific operations of the maxilla and mandible, where assessments were carried out at frequent intervals by a similar blind method, there was a significant improvement of the quality of the surgical field. A newer technique of estimation of reduced blood loss during orthognathic surgery has been reported in which quantification of blood flow to oral tissues in humans is measured using the Doppler technique. In a controlled study using this technique of quantification of blood flow to the maxillary gingival mucosa, a significant reduction in blood flow following hypotensive anesthesia was found.

Initially, induced hypotension was also thought to decrease the surgical time due to improved visibility. Results of controlled studies indicate that this belief is not true.

In many controlled and uncontrolled studies where induced hypotension had been employed, blood loss has been less than without hypotensive anesthesia. Again, no statistically significant difference was observed by Fromme et al. However, their result may have arisen simply because their groups were small numerically. The data in the groups where hypotension was employed, in fact, showed a trend toward reduced blood loss.

The recent concern about hazards associated with blood transfusions, such as transmission of diseases like acquired immune deficiency syndrome and hepatitis, and the possibility of incompatibility, have increased the demand for techniques to prevent blood loss during surgery and thereby blood transfusions. In many studies in orthognathic surgery, where induced hypotension was employed there was either no necessity or a decreased requirement for blood replacement.

HISTORY

Deliberate hypotension to provide a bloodless field and better operative conditions for neurosurgery was first proposed by Harvey Cushing in 1917. In 1943, Kohlstaedt and Page experimenting with dogs described the use of arterial bleeding to produce hypotension resulting in a state of shock. In 1946, Gardner introduced the concept of induced hypotension during anesthesia using arteriotomy as the technique to induce hypotension and produce a relatively bloodless surgical field. Subsequently, pentamethonium was introduced to induce hypotension during anesthesia, soon to be followed by other ganglionic blocking drugs: hexamethonium, trimethaphan, and pentolinium. Enderby stressed the importance of posture to induce hypotension. Subsequently, deep anesthesia with volatile inhalational agents became favored by many. In 1962, Moraca described the use of sodium nitroprusside to induce hypotension during anesthesia. Use of cardiac pacemakers to effect hypotension was introduced by Dimant in 1967. Since then, nitroglycerin, calcium-channel blocking agents, short-acting β-adrenergic blockers, purine compounds, and prostaglandin E1 have also been used.

The first use of hypotensive anesthesia with ganglionic blocking drugs in maxillofacial surgery was described by Enderby. The first study on blood loss and hypotensive anesthesia in orofacial corrective surgery using sodium nitroprusside was reported by Schaberg in 1976. Since then, induced hypotension during orthognathic surgery using nitroglycerin, labetalol, isoflurane, and esmolol has been reported.

PHYSIOLOGY

In order to use induced hypotension for the optimal benefit of the patient, it is necessary to have an understanding of the regulation of blood flow of vital organs. Even relatively short periods of unwanted hypotension, such as those associated with "shock," may be followed by irreversible organ damage. In this situation, the severe decrease in organ blood flow is accompanied by acid-base and metabolic changes. Controlled hypotension rarely results in damage, however, because organ blood flow is normally well maintained.

Richly perfused tissues, such as the brain, heart, liver, and kidneys exhibit autoregulation of their own blood supply through mechanisms related to the intrinsic elasticity of the vascular smooth muscle and vasodilator substances produced in the metabolically active tissues. That is, they maintain their perfusion over a wide range of pressure changes, and it is only when the pressure decreases to relatively low values that adequate perfusion cannot be maintained. This critical pressure varies from vessel to vessel, organ to organ, and probably from individual to individual.

Central Nervous System

Normally, cerebral blood flow remains constant over a mean arterial pressure (MAP) range of 60 to 150 mm Hg. In chronically hypertensive patients, the autoregulatory curve is shifted to the right, and in whom the blood pressure has been controlled by antihypertensive treatment, the curve tends to shift back towards normal.
Volatile anesthetic drugs attenuate or abolish the autoregulation of cerebral blood flow in a dose-dependent manner in the following order: halothane > enflurane > isoflurane.35 The relative potency of these agents to decrease tissue metabolism is in the reverse order.36 Decreases in the arterial carbon dioxide tension (PaCO2) causes cerebral vasconstriction. For every millimeter decrease in PaCO2, the cerebral blood flow decreases by 2% in normotensive subjects.37 This effect is either attenuated38 or abolished39,40 during hypotension, depending on the hypotensive agent. With isoflurane, the decrease in cerebral blood flow appears to be less than or equal to decrements in cerebral oxygen demand regardless of whether the patient is hypocapnic.41

Vasodilators such as sodium nitroprusside and nitroglycerin attenuate the autoregulation of blood flow in a similar manner to that of volatile agents.36 Animal data suggest that cerebral metabolism and cerebral function are preserved during hypotension with hypocapnia in the following order: isoflurane > nitroprusside = nitroglycerin > trimethaphan.42-44

If the reduction in cerebral blood flow exceeds the reduction in the cerebral metabolic requirement for oxygen, cerebral ischemia may develop. When cerebral blood flow is reduced by 40% to 50% below control values, ischemic changes appear in the electroencephalogram (EEG), and below 60% the EEG becomes isoelectric.45 Elevation of the head during hypotensive anesthesia can aggravate the decrease in cerebral perfusion pressure. The perfusion pressure decreases by 2 mm Hg for every 2.5 cm the head is raised above the point of monitoring.46,47

**Respiratory System**

A head-up position in a patient decreases the apical perfusion of the lungs due to gravity, thus increasing dead space.48 Accordingly, the PaCO2 may rise. Induced hypotension may also decrease pulmonary artery pressure, increase blood flow through dependent regions of the lungs, and inhibit the hypoxic pulmonary vasconstrictive effect, thereby increasing the intrapulmonary shunt and the PaCO2.49 This inhibition occurs with nitroprusside, nitroglycerin, and calcium-channel blockers, and to a lesser extent with isoflurane.50-51

The end-tidal carbon dioxide tension (EtCO2) is commonly monitored during hypotensive anesthesia. It must be remembered that the gradient between the PaCO2 and the EtCO2 may increase significantly during hypotensive anesthesia due to the above factors.52 To maintain normocarbia, it may be helpful to perform frequent PaCO2 measurements.

The arterial oxygen tension (PaO2) may be reduced during hypotension for the same reasons described for the PaCO2. Thus, it is essential to maintain the oxygen saturation within normal limits, which may necessitate giving more than the normal amount of oxygen during hypotensive anesthesia. However, 100% oxygen is not preferable during hypotension, as this may cause vasconstriction, especially in the brain.

**Cardiovascular System**

Even at rest, cardiac muscle extracts most of the oxygen delivered to it. Consequently, any increase in myocardial oxygen demand requires a parallel increase in coronary artery blood flow. Hypotensive anesthesia may substantially decrease coronary blood flow. However, it simultaneously decreases myocardial oxygen demand, due to the reduction in afterload (usually) and/or preload (sometimes). Furthermore, the coronary arteries dilate in response to the accumulation of metabolic vasodilator substances in the myocardium, thus ensuring an adequate blood flow to the myocardium. Hypotension with a normal or slow pulse is more beneficial than one with tachycardia. Tachycardia will increase the oxygen demand and shorten diastole, thus decreasing blood flow to the left ventricular subendocardium. Coronary blood flow is rarely compromised in pediatric patients.45 Factors that increase myocardial oxygen demand, such as tachycardia, should rarely cause myocardial ischemia even when the child is hypotensive. In patients with coronary artery obstruction, vasodilators used for hypotension may induce unfavorable intramyocardial blood flow redistribution as a consequence of resistance vessel dilatation, resulting in regional poststenotic hypoperfusion (the "steal" phenomenon) and possibly myocardial ischemia.52

**Renal System**

Decreases in renal blood flow stimulate the renin-angiotensin system. When the MAP falls below 75 mm Hg, the glomerular filtration rate falls.53 Opioid analgesics and probably most inhalational agents stimulate secretion of antidiuretic hormone.54 All these factors result in oliguria during hypotensive anesthesia. However, following termination of hypotensive anesthesia, urine formation rapidly recovers, provided the patient is well hydrated.55

**Hepatic System**

In patients with normal liver function, there are no reports of reduced liver function attributable to hypotensive anesthesia. The technique has been used successfully in
selected patients to reduce blood loss during operations for lienorenal shunts.56

BLOOD PRESSURE GOAL

Since the aim of hypotensive anesthesia is to reduce blood loss and provide an easily visualized surgical field, the degree of reduction should depend on each individual patient and clinical situation. The hypotension should be considered satisfactory when bleeding appears to be minimal and organ perfusion adequate. In theory, as long as the MAP exceeds the sum of the colloid osmotic pressure and the venous pressure, the circulation should be adequate for tissue needs. Although a pressure of 32 mm Hg should be sufficient theoretically, in practice it is probably below the safe limit due to the specific blood flow requirements of different organs and the possibility of disease and other causes of altered circulation.57

In patients undergoing cerebrovascular surgery, somatosensory evoked potentials and the redox potential of cytochrome a, a3 in the mitochondria of cerebral cortical cells indicate that it is still possible to maintain cerebral metabolism at a functional level with a MAP between 40 and 60 mm Hg.58 However, below a MAP of 40 mm Hg, deterioration of cerebral metabolism occurs, which, if not corrected, results in irreversible brain damage.

In orthognathic surgery, a decrease of MAP to between 50 and 65 mm Hg significantly reduced blood loss.2,3,10,11 If the MAP was maintained at 70 mm Hg or above,1,2 blood loss was similar to that of patients not receiving hypotensive anesthesia. However, those patients whose blood pressure was reduced by 20% or more from baseline exhibited a significant decrease in blood loss irrespective of the absolute blood pressure.1,5

Hypotensive anesthesia should be induced in relation to the patient’s preoperative blood pressure rather than to a specific target pressure and should be limited to that level necessary to reduce bleeding in the surgical field and in duration to that part of the surgical procedure deemed to benefit by it. It is suggested that inducing hypotension to a MAP 30% below a patient’s usual MAP, with a minimum MAP of 50 mm Hg in ASA Class I patients and 80 mm Hg in the elderly, is clinically acceptable.52,58

CONTRAINDICATIONS

Table 1. Contraindications to the Use of Hypotensive Anesthesia

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<tr>
<th>Anesthetic limitations</th>
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<td>Lack of technical expertise</td>
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<td>Inability to monitor the patient adequately</td>
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<th>Patient limitations</th>
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<td>Diabetes mellitus</td>
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<td>Hepatic disease</td>
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<td>Intolerance to the drugs available to produce hypotension</td>
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<td>Ischemic cerebrovascular disease</td>
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<td>Renal disease</td>
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<td>Respiratory insufficiency</td>
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<td>Severe systemic hypertension</td>
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MORBIDITY AND MORTALITY

In a large postal survey conducted in 1955, 96 deaths were reported in 27,930 cases of induced hypotension during anesthesia.59 Over 50% of the deaths were related to decreased blood flow to the vital organs (eg, brain, heart, and kidneys). Other causes included reactionary hemorrhage (8), high-spinal anesthesia (5), and isolated cases of arterial air embolism, aortic-femoral thrombosis, pulmonary infarction, pulmonary edema, persistent hypotension, and excessive heparinization. In a separate study of 9,107 hypotensive anesthetics conducted between 1950 and 1960, nine deaths (0.1% mortality) were reported.59 Of these, six were probably attributable to the technique. A report in 195951 describes a death following middle cerebral artery thrombosis in a patient who received hypotensive anesthesia, and another in 197421 attributes a death due to extensive cerebral and cerebellar infarction. Finally, a significantly higher mortality after hypotensive anesthesia was noted in patients with a history of cerebrovascular disease (3 out of 40 cases) than in patients with no history of cerebrovascular disease (2 out of 960 cases).62

Cerebrovascular System

In the aforementioned report of 1955,59 29 patients suffered cerebral thrombosis, of which 18 died. One case of hemiplegia was recorded in the series performed between 1950 and 1960.60 Additional cases of middle cerebral artery thrombosis, hemiplegia, and severe cerebral and cerebellar infarction have also been reported.21,61 The survey in 196162 reported 3 out of 40 cases who developed cerebral thrombosis as an extension of their existing cerebrovascular disease.

Although mental changes are common in the elderly following hypotensive anesthesia, they also occur in the absence of hypotensive anesthesia.63
Cardiovascular System

Mild coronary ischemia may occur during induced systolic hypotension below 60 mm Hg. Patients with a previous history of hypertension are more susceptible to ischemia. Furthermore, rapid hypotension may be accompanied by an earlier onset of ischemic changes in the electrocardiogram (ECG). In the 1955 series, 18 cases of myocardial infarction were reported, of which eight died. Additionally, 22 cardiac arrests and 36 cardiovascular collapses were noted, with 14 dying. In a report in 1961 of 1,000 elderly patients who underwent hypotensive anesthesia, seven suffered myocardial infarction. Three cases of retinal artery thrombosis cases were also listed in the 1955 series.

Due to the long action or toxicity of some hypotensive agents, persistent hypotension following hypotensive anesthesia has been reported. Reactionary hemorrhage can also occur following hypotensive anesthesia as the blood pressure reverts to normal or overshoots to a higher level in the immediate postoperative period. Such hemorrhage occurred in 243 patients described in the 1955 study, of which eight died from this complication. In a subsequent prospective study, postoperative bleeding was reported in 10.8% of patients undergoing thoracotomy, but the incidence was similar in those who did not have hypotensive anesthesia.

Other Systems

Two deaths attributed to hypoventilation were reported in the 1950 to 1960 series. Anuria was recorded in 62 cases and oliguria in 54 cases of the 1955 review. Nine of these patients died.

Skin Necrosis

Necrosis of the skin with ulcer formation can occur after prolonged hypotensive anesthesia from pressure-induced hypoperfusion. Common sites are the tip of nose (due to the pressure of the endotracheal tube or nasogastric tube) and pressure areas in the sacral region with the patient in the reverse Trendelenberg position. Recently, there was a report of a finger injury from pressure of a finger probe (pulse oximeter) in a patient who had received hypotensive anesthesia. In a similar manner, thermal burns may appear in sacral areas when patients are placed on warming blankets during hypotensive anesthesia.

Cyanide Poisoning

Large doses of nitroprusside administered to effect hypotension can result in cyanide poisoning, which manifests as metabolic acidosis, persistent hypotension, and tachycardia. In some cases it has been fatal.

Overall Risk

The validity of conclusions drawn from early retrospective surveys is limited. More recent reports using control groups have been unable to show significant differences in morbidity and mortality attributable to induced hypotension during anesthesia. This is probably due to the more sophisticated modern monitoring techniques and better understanding of the physiology of respiratory, cardiovascular, and cerebrovascular systems and the pharmacology of anesthetic agents and hypotensive drugs.

METHODS OF HYPOTENSIVE ANESTHESIA

Since blood pressure is determined by the cardiac output and systemic vascular resistance, blood pressure can be lowered by decreasing the peripheral vascular resistance (afterload) and/or by lowering cardiac output (Table 2). A decrease in peripheral vascular resistance is obtained by causing vasodilatation of the resistance vessels. Cardiac

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<th>Table 2. Strategies for Inducing Controlled Hypotension</th>
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<td>Decrease cardiac output</td>
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<td>Decrease peripheral vascular resistance</td>
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output can be lowered by reducing venous return (preload), the heart rate, myocardial contractility or a combination of these factors. Inducing hypotension purely by a reduction in cardiac output is not ideal, however, because an adequate cardiac output is crucial for maintaining blood flow to the tissues. The circulation must be sufficient to supply oxygen and energy substrates to the tissues and to remove metabolic waste products before they can accumulate sufficiently to cause damage.

The final effect of hypotensive anesthesia on cardiac output will depend on the interactions among the afterload, preload, myocardial contractility, and heart rate. This balance is delicate and may be disrupted by intrinsic and extrinsic factors interacting with other homeostatic mechanisms. Such factors include the patient’s clinical state, the administration of additional drugs, and the ventilatory pattern. To achieve the desired level of hypotension, maneuvers such as altering the position of the patient, adjusting the airway pressure, or adding other hypotensive drugs to complement the activity of the primary hypotensive agent can be used.

Once hypotension is induced, the required blood pressure to minimize blood loss may be maintained by adjusting the amount of the hypotensive agent administered, either manually or automatically by a self-tuning adaptive control device. In the latter method, cardiovascular parameters are monitored electronically, with data being continuously transferred to a microcomputer. According to a preset algorithm based on the age and weight of the patient, the computer then controls the infusion of the hypotensive agent to maintain the blood pressure within the target range.

**Pharmacological Agents**

The ideal agent for inducing deliberate hypotension during orthognathic surgery would allow easy administration, a predictable effect, rapid onset and recovery, quick elimination with no toxic metabolites, and minimal alteration in blood flow to vital organs. At present, no perfect agent exists. Many pharmacological agents have been used to induce hypotension during orthognathic surgery. In addition, there are a number of drugs that have been used for induced hypotension in other types of surgery.

**Ganglionic Blocking Drugs.** These drugs block the ganglia of the autonomic nervous system, thus inhibiting both sympathetic and parasympathetic outflows. The hypotensive response results from blockade of sympathetic tone to both arteries and veins. Ganglionic blockade also affects the parasympathetic fibers to the heart, eye, gastrointestinal tract, bladder, and salivary glands, resulting in tachycardia, mydriasis, cycloplegia, decreased gastrointestinal mobility, urinary retention, and xerostomia. Many of these changes are undesirable and limit the use of these drugs as hypotensive agents.

**Methonium compounds.** Pentamethonium and hexamethonium have been used to induce hypotension. The latter has a more predictable action. However, both cause tachycardia, and fit young patients show tachyphylaxis.

**Pentolinium.** Pentolinium is five times as potent as hexamethonium and rarely leads to tachycardia because sympathetic fibers are more effectively blocked than parasympathetic fibers. The initial fall of blood pressure and the subsequent recovery of normal pressure after termination of the drug are slower than with hexamethonium.

**Trimethaphan.** The primary ganglionic blocking drug still used for hypotensive anesthesia is trimethaphan camyslate. In addition to its ganglionic blocking action, trimethaphan directly relaxes arterioles and causes the release of histamine, which further enhance the hypotensive effect. Trimethaphan may be given in intermittent doses of 10 mg of a 1% solution or more commonly by an infusion of a 0.1% solution in normal saline. The blood pressure begins to decrease after 4 min, and the peak effect occurs within 10 min. Hypotension depends on the speed of the drip; after the drip is stopped, recovery is moderately rapid. Trimethaphan has a short half-life (1 to 2 min) resulting from its rapid inactivation by plasma cholinesterase and subsequent renal excretion.

Trimethaphan can cause severe hypotension in arteriosclerotic patients, interferes with the heat-regulating mechanism, causing hypothermia after long operations, and reduces the need for anesthesia once a low pressure has been established. Younger robust patients may prove resistant to its hypotensive action. Halothane is often used as an adjuvant to reduce the development of tachycardia. The development of tachyphylaxis may be a problem, and the maximum recommended dose is 100 mg in the first 15 min and a total dose of 1 g. Other potential problems include histamine release and potentiation of neuromuscular blockade. Histamine release can theoretically dilate cerebral blood vessels and increase intracranial pressure. Direct cerebral toxic effects following trimethaphan have been reported. High brain lactate concentrations suggesting increased glycolytic activity, EEG burst suppression, and slowing of high voltage waves have been reported at a MAP of 50 mm Hg. In addition, animals receiving trimethaphan exhibited signs of cerebral ischemia, which did not occur with other agents at comparable levels of hypotension.

**α-Adrenergic Blocking Agents.** A number of agents possess the ability to block α-adrenergic receptors. Drugs used for this purpose range from agents classified
as α blockers to those in which α blockade is normally regarded as an undesired side effect.

**Phentolamine.** Phentolamine is a short-acting α-adrenergic receptor blocking agent. Its action is rapid, with peak effects occurring within 2 min. Its action lasts 10 to 15 min. Phentolamine has a powerful vasodilating effect. As a result of reflex sympathetic stimulation of the heart, tachycardia may become quite prominent. It is usually given in 2- to 5-mg intravenous boluses until adequate blood pressure control is obtained. Phentolamine can also be administered as an infusion.82

**Labetalol.** Labetalol is an α- and β-adrenergic receptor blocking agent. It is 4 to 8 times more potent at β than α adrenoceptors.87 Thus, it reduces the blood pressure by reducing the peripheral vascular resistance while preventing a reflex increase in heart rate. It is 6 to 10 times less potent than phentolamine at α-adrenergic receptors, 1.5 to 4 times less potent than propranolol at β1 receptors, and about 11 times less potent on β2 receptors.

Labetalol can be given in intermittent bolus doses or by infusion. Its action is prompt, with peak effect occurring within 5 min. Although its half-life is relatively long at 4 hr,11 the clinical effect of induced hypotension appears to last less than 30 min. The drug exhibits remarkable hypotensive synergy with inhalational agents, such as halothane88 and isoflurane.52 It appears to be less potent in reducing the blood pressure during neurolept anesthesia.89 Advantages appear to be readily controllable hypotension, avoidance of tachycardia and tachyphylaxis, and avoidance of increased intracranial pressure and re-bound hypertension.90 A disadvantage is that it will mask the response to acute blood loss even in the postoperative period because of its long half-life.91 Contraindications include heart failure, bradycardia, hypovolemia, and atioventricular block. In orthognathic surgery, labetalol has been used successfully to induce hypotension and significantly decrease blood loss and the need for blood transfusion.11

**Antipsychotic Agents.** Chlorpromazine and droperidol, antipsychotic agents with significant α-adrenergic blocking properties, have been used to control blood pressure during anesthesia. Due to their long half-lives, they are usually given for premedication in patients undergoing controlled hypotension, thus acting as adjuncts to other hypotensive agents.45

**Direct-Acting Vasodilators.** Drugs that relax smooth muscle directly include nitroprusside, nitroglycerine, and hydralazine.

**Nitroprusside.** Nitroprusside is one of the most widely used agents for inducing hypotension in a variety of procedures, including orthognathic surgery.1,5,6,82 It was first used in anesthesia for this purpose in 1962.24 It acts directly on the vessel walls by interacting with membrane-bound sulphydryl groups.92 Nitric oxide is released from the drug molecule, which then diffuses across the cell membrane to stimulate guanylate cyclase. The resultant increase in guanosine 3',5'–monophosphate inhibits the contractile mechanism. A blockade of the intracellular release of calcium facilitates the relaxation. Nitroprusside mainly acts on the arterioles, or resistance vessels, and less on the venules, or capacitance vessels.93 This differential effect reduces the arteriolar-venular pressure gradient. As a result, tissue oxygenation may fall during nitroprusside infusion even though the PaO2 remains normal.94 Sensitivity to vasodilation from nitroprusside increases with age, probably due at least partly to an age-related resistance of cardiac adrenergic receptors to catecholamine stimulation.

Nitroprusside is available in bottles, each containing 50 mg of the drug in powder form. The agent can be dissolved in 5% dextrose or normal saline. The solution should not be exposed to light because it breaks down, and the container is usually wrapped with aluminum foil. If the solution attains a blue color, indicative of the breakdown of nitroprusside, it should be discarded.95 It is given by intravenous infusion as its half-life is about 2 min.96 Its action is rapid, appearing within 1 min. The degree of hypotension can be titrated against the infusion rate. The usual dose range of nitroprusside is 0.5 to 10 μg/kg-min. If an adequate reduction in blood pressure is not obtained within 10 to 15 min at the highest recommended dose, the infusion should be stopped to prevent cyanide toxicity.91 Reversal is spontaneous with the termination of infusion.

Metabolism of nitroprusside occurs when it comes in contact with either tissue or red blood cell sulphydryl groups.97 Inside red blood cells, cyanomethemoglobin and an unstable tetracyano compound are formed. Cyanide released from this unstable compound combines with thiocyanate to form thiocyanate. This reaction is catalyzed by the liver and kidney enzyme rhodanase. Cyanide also combines with hydroxocobalamin producing cyanocobalamin. Both thiocyanate and cyanocobalamin are water soluble and are excreted in the urine. Some hydrogen cyanide is exhaled. Cyanide can also react with mitochondrial cytochrome oxidase to form a cytochrome oxidase-cyanide complex, which inhibits cellular respiration and results in cytotoxic hypoxia. A high level of this complex can lead to metabolic acidosis and death.84 Nitroprusside should not be given to patients with Leber's optic atrophy, tobacco amblyopia, vitamin B12 deficiency, or liver or renal failure, as these conditions affect normal cyanide metabolism.95

Adverse effects of nitroprusside include cyanide toxicity. Metabolic acidosis is an indirect indicator of cyanide toxicity.52 Additional signs of toxicity include tachycardia and persistent severe hypotension even after withdrawal.
of the drug. The blood cyanide concentration is related to the total dose of nitroprusside administered per unit time. Correlation of blood cyanide with subsequent metabolic acidosis has led to decreasing recommendations for safe limits of sodium nitroprusside, the latest recommended safe dose being 0.5 to 0.7 mg/kg in 2 to 3 hr.\textsuperscript{97} If cyanide toxicity is suspected, the infusion should be immediately stopped. Sodium nitrite, 5 mg/kg, can be injected slowly intravenously or amyl nitrite can be given by inhalation. Nitrates form methemoglobin, and cyanide combines preferentially with methemoglobin to produce cyanomethemoglobin, preventing the cytochrome oxidase-cytochrome tissue complex from forming. In case of severe toxicity, sodium thiosulfate 150 mg/kg can also be given intravenously. This acts as a sulfur donor and converts cyanide to thiocyanate through the rhodanese pathway.\textsuperscript{52} If no improvement occurs, 20 mL (300 mg) of cobalt edetate should be injected at the rate of 1 mL/sec.\textsuperscript{95} Although cobalamins normally present in the food can combine with cyanide to produce water-soluble cyanocobalamin, the efficacy of hydroxocobalamin in reversing actual sodium nitroprusside-induced cyanide toxicity remains to be demonstrated.\textsuperscript{91}

Tachyphylaxis or increasing dose requirements to maintain hypotension has been reported with sodium nitroprusside. In these patients, the blood cyanide concentrations are higher and may or may not be accompanied by metabolic acidosis. Acute tolerance may result from a decreased responsiveness of the guanylate cyclase system to nitric oxide. Resistance may also occur due to increased plasma renin, angiotensin II, and catecholamine activity. If tachyphylaxis occurs, the infusion should be immediately stopped, as it can lead to cyanide toxicity.\textsuperscript{91}

Rebound hypertension of 20 mm Hg or greater of systolic blood pressure lasting for 30 min following abrupt termination of nitroprusside infusion has been reported.\textsuperscript{98} Increased renin secretion associated with the hypotension results in production of angiotensin I and angiotensin II. The increase in blood pressure is due to angiotensin II. This response can be attenuated by tapering the infusion over 30 to 45 min, using agents such as propranolol to inhibit renin secretion, or giving angiotensin-converting enzyme inhibitors such as captopril\textsuperscript{99,100} or enalaprilat\textsuperscript{101} to inhibit production of angiotensin II.

Nitroprusside can also decrease the platelet concentration,\textsuperscript{102} inhibit platelet aggregation,\textsuperscript{103} and impair pulmonary hypoxic vasoconstriction.\textsuperscript{49}

In order to reduce the dose of nitroprusside used, thereby preventing cyanide toxicity, various agents can be used to augment the action of nitroprusside. Trimethaphan provides an excellent example. Both drugs are dissolved in 5% dextrose in the proportion to be infused. Nitroprusside/trimethaphan ratios from 1:10 to 1:2.5 have been employed with success.\textsuperscript{104–109} Nitroprusside and esmolol can also be used together to induce hypotension.\textsuperscript{110,111} Esmolol infusion causes dose-dependent reductions in the nitroprusside requirement by decreasing heart rate, myocardial contractility, and renin activity. The angiotensin-converting enzyme inhibitors captopril and enalaprilat provide a similar additive effect.\textsuperscript{99–102} They decrease the rise in endogenous vasoconstrictors due to stimulation of the renin-angiotensin system during nitroprusside infusion.

In orthognathic surgery when nitroprusside was used to reduce pressure to a predetermined MAP, blood loss was not significantly reduced compared to that in normotensive patients.\textsuperscript{1,3,6} However, when the MAP was reduced by 20% or more from the preoperative value, blood loss was significantly reduced.\textsuperscript{1,5}

Nitroglycerin. Intravenous nitroglycerin has been used successfully to induce hypotension during orthognathic surgery.\textsuperscript{10,112} It acts directly on the vascular smooth muscle\textsuperscript{92} in a similar manner to that of nitroprusside. However, its action is predominantly on capacitance vessels, resulting in decreased venous return to the heart, and therefore a reduced stroke volume and cardiac output.\textsuperscript{113}

Nitroglycerin is supplied in 20-mL glass ampoules containing 10 mg of drug. In this form it is stable in light. It may be adsorbed by plastic, hence the drug should be infused in glass bottles or high-density polyethylene syringes and tubing\textsuperscript{114} and is not diluted before injection.\textsuperscript{91} It is given as an intravenous infusion, since its half-life is about 2 min.\textsuperscript{52} The infusion is started at a rate of 1 to 2 \(\mu\)g/kg-min and increased to achieve the desired mean arterial pressure.\textsuperscript{91} Its action is rapid, appearing within 1 min. There is no upper limit to the rate of infusion as there are no toxic metabolites reported. Usually with 2 \(\mu\)g/kg-min, a good hypotensive response is obtained. However, especially in young patients, it may be impossible to achieve the desired degree of hypotension with nitroglycerin.\textsuperscript{52} Reversal is spontaneous with the ending of the infusion.

Metabolism of nitroglycerin occurs principally in the hepatic\textsuperscript{115} and pulmonary vascular beds.\textsuperscript{116} Unlike nitroprusside, no significant inactivation occurs in the peripheral blood and no toxic metabolites are produced.\textsuperscript{52}

Arterial and tissue oxygen tensions remain unchanged,\textsuperscript{94} probably because of the persistent pressure gradient between the arteriolar and the venular sides of the microcirculation and the lesser degree of diversion of blood through arteriovenous shunts during nitroglycerin-induced hypotension, as compared to that of nitroprusside. An additional advantage over nitroprusside is the absence of rebound hypertension.\textsuperscript{91}

Hydralazine. Hydralazine has been used intravenously to produce hypotension during anesthesia.\textsuperscript{117–119} It acts
directly on vascular smooth muscle, mainly on the arterioles or resistance vessels, at least in part through the release of nitric oxide. Reflex tachycardia commonly accompanies the hypotension. Onset of the hypotensive effect may take up to 20 min. As with nitroprusside and nitroglycerin, it should not be used in patients with low intracranial compliance.91

**Calcium-Channel Blockers.** Nicardipine,120–125 nifedipine,52, diltiazem,126 and verapamil27 have all been used to produce hypotensive anesthesia. Agents other than verapamil vasodilate resistance vessels but have no effect on the capacitance vessels. With clinical doses they maintain myocardial contractility and cardiac output with no increase in heart rate or only a modest tachycardia.28 However, verapamil has a significant negative inotropic effect, a lesser vasodilating effect, and should be reserved for its antiarrhythmic and antianginal actions.

Nicardipine is popular as it is sufficiently water soluble to be administered in intravenous solutions. When 0.5 mg of nicardipine is given intravenously, the maximal effect comes on in 2.5 min.52 Its distributional half-life is 14 min and terminal half-life is 4.75 hr.121 Clearance is mainly by hepatic metabolism to inactive metabolites. Compromised renal function is a potential complication of nicardipine.52 Despite normal left-heart filling pressures and cardiac output, blood is shunted away from the renal vascular bed, producing a clinical picture consistent with prerenal failure. Once the drug is withdrawn, patients recover renal function rapidly. Careful titration of the infusion to attain the desired level of hypotension should be carried out, as excessive decreases may not be quickly reversed.

**Purines.** Adenosine and adenosine triphosphate are potent vasodilators, and both have been used to induce hypotension during anesthesia.28,29,127–134 Adenosine triphosphate is rapidly decomposed to adenosine in the blood, and the vasodilatation it produces has been ascribed to adenosine.135 Adenosine stimulates specific receptors on the vessel walls of arterioles to produce vasodilatation.128 A lack of effect on capacitance vessels may be associated with the rapid tissue metabolism of adenosine (half-life: 10 to 20 sec)52 resulting in most of it being metabolized before reaching the veins. In contrast to nitroprusside and nitroglycerin, the decrease in peripheral vascular resistance is accompanied by an increase in cardiac output without an increase in the heart rate.128 As it has no positive inotropic action on the myocardium,136 this effect is probably due to its selective ability to reduce peripheral vascular resistance. In doses higher than used to induce hypotension, adenosine can produce bradycardia by a direct action on the sinus node137 and the conducting system of the heart, as well as by inhibition of cardiac sympathetic neurotransmission.138 Adenosine dilates the coronary blood vessels, increasing the coronary blood flow.130–132

Adenosine can be infused in an isotonic saline solution at a rate of 60 to 350 μg/kg-min.130–132 Hypotension occurs rapidly, and a stable blood pressure with easy control is obtained. Switching off the infusion brings back the blood pressure without rebound hypertension. Rebound hypertension and tachyphylaxis do not occur because adenosine inhibits renin secretion131 and norepinephrine release139 by a presynaptic effect.140

Adenosine is metabolized to inosine or is taken up by the cells. Inosine is converted to hypoxanthine, which is oxidized to uric acid.141

Because large doses of adenosine may have to be used to produce hypotension, uric acid may accumulate as a metabolite, a problem for patients with gout.52 Adenosine should also be used with caution in patients suffering from myocardial ischemia as it may cause the "steal phenomenon," shifting coronary blood flow away from ischemic areas.130 Adenosine can increase the cerebral blood volume and intracranial pressure.142 If adenosine triphosphatase is used, considerable phosphate is released, and the phosphate binding of calcium and magnesium can lead to dysrhythmias.143 Thus, it is better to use adenosine rather than adenosine triphosphate to induce hypotension.

In order to reduce the dose of adenosine required to produce hypotension and thereby reduce the amount of uric acid formed, dipyridamole, a drug that inhibits adenosine uptake by cells, can be administered concomitantly.144

**Prostaglandin E1.** Prostaglandin E1 is one of the more recently used agents for inducing hypotension during anesthesia.125,145–147 It is a potent vasodilator148 of systemic and pulmonary vascular beds. Its inotropic effect149,150 on the heart increases the cardiac output, an effect aided by the peripheral vasodilation and decreased afterload. Following infusion of PGE1, no significant increase in heart rate is observed despite a significant fall in arterial pressure,147,151 probably due to the attenuation of the baroreflex in response to the induced hypotension. However, the arterial baroreceptor response to acute hypovolemia during hypotensive anesthesia is better maintained than with nitroprusside and trimethaphan.32 Prostaglandin E1 also maintains the oxygen supply to the myocardium151 and is antiarrhythmic.152,153 In patients without decreased intracranial compliance, it maintains cerebral blood flow, autoregulation, and normal jugular venous oxygen tensions.154 At physiological and pharmacological concentrations, it does not affect clotting time, bleeding time, or adenosine diphosphate-induced platelet aggregation.151 Prostaglandin E1 increases renal
blood flow, urine formation, and sodium excretion during hypotension under general anesthesia.

**Inhalational Agents.** The inhalational agents halothane,1,9,21,155–158 enflurane,6,22,23 isoflurane,2,159–161 and desflurane162,163 have been used to induce hypotension during anesthesia.

Hypotension with halothane results primarily from myocardial depression. Although vasodilatation occurs in the skin, brain, and splanchnic vasculature, the peripheral resistance does not decrease significantly because of the increase in tone of skeletal muscle and renal arterioles.164 Halothane is used for hypotensive anesthesia because of its ability to produce hypotension with a lack of or even suppression of tachycardia. The disadvantages of halothane are its direct myocardial depressant effect without decrease of peripheral vascular resistance, sensitization of the myocardium to the arrhythmogenic effect of catecholamines, and the possibility of halothane hepatitis.112 With deep halothane anesthesia, significant oozing of blood despite hypotension has been reported.165

Hypotension with enflurane is caused by a combined decrease in peripheral vascular resistance and myocardial depression.164 Enflurane is also less arrhythmogenic when used in conjunction with catecholamines. However, it may precipitate epileptiform convulsions in those who are susceptible to it, especially in the presence of hypocarbia, and may produce renal damage in those with renal disease or in obese patients.165 Controlled hypotension under deep enflurane anesthesia has produced no decrease in blood flow to the maxilla, mandible, or tongue and has even produced an increase in blood flow to the suprathyroid and masseter muscles.166 Thus, deep enflurane anesthesia per se may not be beneficial to oral and maxillofacial surgery.

In equipotent doses, isoflurane produces rapid hypotension by decreasing peripheral vascular resistance without myocardial depression.23,112 It also provides better myocardial perfusion than halothane or enflurane.112 Although myocardial perfusion improves generally, in patients with coronary artery stenosis, coronary steal may occur.52 With isoflurane, baroreflex activity is better maintained during hypotensive anesthesia.21,112 This may result in tachycardia during controlled hypotension. Isoflurane has been used as the sole agent to induce hypotension during orthognathic surgery2 and has significantly reduced blood loss and improved the quality of the surgical field.

Desflurane is a new volatile anesthetic agent whose cardiovascular effects have been reported to be similar to those of isoflurane.159 It has been used for hypotensive anesthesia in dogs160 and has decreased the blood pressure by reducing peripheral vascular resistance and cardiac output. As yet, there are no reports of its use for induced hypotension in humans. It appears best to use inhalational agents as adjuvant drugs for hypotensive anesthesia rather than using them as sole agents.

**β-Adrenergic Blocking Agents.** β-Adrenergic blocking agents are either used as the sole hypotensive agent3,167 or as an adjunct9,110,111 to other hypotensive techniques. In orthognathic surgery, stimulation of branches of the trigeminal nerve in turn stimulates the sympathetic nerve centers in the medulla, resulting in tachycardia and increased blood pressure.168 β-Adrenergic antagonists attenuate this response. Furthermore, any drug that lowers the peripheral vascular resistance and decreases blood pressure will cause activation of baroreceptors and a reflex increase in the heart rate to compensate for the decrease. β-Adrenergic blocking agents help to block this response as well. They may also prevent tachycardia in response to hypoxia, hypocarbia, hypovolemia, and hypoglycemia. Caution should be exercised before administering them to patients with established right or left ventricular failure, hyperdynamic circulation in anemia, or heart block.168

Propranolol has enjoyed the longest use in hypotensive anesthesia.50,169,170 Its hypotensive effect is believed to occur by the blockade of cardiac β adrenoceptors, inhibition of renin release, and a central action. After a test dose of 0.25 to 0.5 mg to evaluate the degree of response or any side effects, the drug can be given in similar increments until the desired response is obtained. As it is not a cardioselective β blocker, propranolol may cause bronchospasm by blocking β2 receptors in susceptible patients.168

Metoprolol has also been used for hypotensive anesthesia.171,172 The primary advantage over propranolol is that it is a selective β, blocker and is less likely to produce bronchospasm in susceptible patients.

Esmolol is a relatively new cardioselective β blocker available in 10 mg/mL, 100 mg/mL, and 250 mg/mL ampoules. It has a quick onset and a short duration of action.173 It has a rapid distribution half-life (2 min) and an elimination half-life of only 9 min. Thus, it can be used in bolus doses or preferably as an infusion. Once the infusion is terminated, the blood pressure reverts to the original pressure within 30 min. It has successfully been used for controlled hypotension during anesthesia either alone3,166 or in conjunction with another hypotensive agent acting peripherally.110,111 It is metabolized primarily by esterases in the cytosol of red blood cells to an inactive metabolite and is safely eliminated in patients with hepatic or renal impairment.174 Esmolol can significantly decrease the cardiac index, an effect opposite that of nitroprusside.174
Adjunctive of Agents

A number of drugs that are not used specifically to lower blood pressure intraoperatively nevertheless promote drug-induced hypotensive anesthesia. Chief among these are the neuromuscular blocking drugs and the opioid analgesics.

Neuromuscular Blocking Agents. Tubocurarine was the first neuromuscular blocker to be used in patients undergoing controlled hypotension during anesthesia. It produces a fall of blood pressure in its own right due, in part, to ganglionic blockade and histamine release. Large doses of alcuronium and, to a lesser extent, atracurium, can also cause a drop in blood pressure through histamine release. Vecuronium is a primary example of a "pure" neuromuscular blocker, one that has no significant direct effect either on heart rate or blood pressure. Nevertheless, the blood pressure may decrease after vecuronium simply as a consequence of the neuromuscular blockade. The necessity for positive pressure ventilation tends to decrease venous return to the heart, an effect that is augmented by the loss of muscle tone in tissues surrounding the capacitance veins. Pancuronium, which produces tachycardia, is an exception to the general rule that neuromuscular blockers facilitate hypotensive anesthesia.

Opioid Analgesics. Opioids decrease the doses of agents necessary to produce anesthesia and hypotension. Certain opioids, such as meperidine and morphine can cause histamine release. More important, however, is the ability of opioids to attenuate sympathetic responses to stress.

Mechanical Maneuvers to Potentiate the Action of Hypotensive Agents

Positioning. The importance of correct positioning of the patient during controlled hypotension in anesthesia cannot be overemphasized. Elevation of the site of operation allows easy drainage of venous blood from the site of surgery. Inclining the patient (raising the head) 15° to 25° off the horizontal plane facilitates pooling of blood in the dependent regions of the body, thus reducing venous return to the heart and the cardiac preload. Blood pressure is also reduced gravimetrically in the operating field. For each 2.5-cm elevation of the site above the heart, there is a 2-mm Hg fall in blood pressure. The hypotensive effects of most of the hypotensive agents are potentiated by posture. Rapid changes in posture may be followed by rapid changes in blood pressure; thus, tilting the head down may be used to rapidly reverse excessive hypotension.

Positive Airway Pressure. Use of positive pressure ventilation decreases the venous return during the positive pressure phase. This effect can be enhanced by increasing the inspiratory volume, prolonging the inspiratory time, and raising the positive end-expiratory pressure. However, these manipulations not only cause a deleterious effect on the cardiac output and pulmonary perfusion, but also result in increased respiratory deadspace. Increasing intrathoracic pressure can also decrease venous outflow from the cranium sufficiently to increase the cerebral blood volume and intracranial pressure. Thus, respiratory manipulations to minimize venous return are usually not employed in controlled hypotension.

ANESTHETIC MANAGEMENT

Preoperative Management

The decision to induce hypotension is best taken preoperatively following examination of the patient. As a crucial prerequisite, the anesthetist conducting the hypotensive anesthesia must have a thorough knowledge of the technique that is to be performed. Appropriate patient evaluation and selection are also important for safe induced hypotensive anesthesia. Preoperative investigations including hemoglobin (Hb), urea, electrolytes, and ECG should be carried out. A minimum Hb of 10 g/dL should be safe in those undergoing hypotensive anesthesia. Either in the ward before transport or in the operating room before anesthesia, arterial blood gas sampling may help provide a baseline for intraoperative or postoperative measurements. Various premedications, including anxiolytics, analgesics, α blockers (eg, droperidol, clonidine, β blockers (eg, metoprolol), and antihypertensives (eg, clonidine), can assist the induction of hypotension during anesthesia. Vagolytic drugs are best avoided.

Intraoperative Management

Following preanesthetic monitoring, induction of anesthesia can be carried out in the usual manner. A stress-free induction and obtunding the increase in blood pressure and tachycardia to intubation will help set the stage for a smooth hypotensive technique. The endotracheal tube and any other tube placed through the nares should not exert undue pressure on the tip of the nose. A second intravenous line is preferable if the hypotensive drug is to be given as an intravenous infusion. During maintenance of anesthesia, a patient who is to undergo hypotensive...
anesthesia during orthognathic surgery is usually ventilated with the help of neuromuscular blocking agents.

**Posture.** As mentioned previously, the patient should be positioned with the head elevated about 20° above the heart. Having the legs slightly elevated by flexing the table at the hips of the patient prevents the patient slipping down the table and prevents pressure sores in the sacral region during hypotensive anesthesia. Soft cotton wool pads for the sacral region and the feet, the regions which act as pressure points, will help to prevent skin necrosis.

**Monitoring.** Meticulous monitoring is essential for patient safety during hypotensive anesthesia.

**Blood pressure.** Intraarterial blood pressure monitoring during hypotensive anesthesia is recommended by many as it not only helps in getting immediate and often more accurate blood pressure data than can be obtained with noninvasive blood pressure monitors but also permits sampling for arterial blood gases, hematocrit, and other laboratory investigations. If noninvasive blood pressure monitoring is also to be used, attaching a blood pressure cuff to the same arm that is used for arterial cannulation will help check the accuracy of blood pressure recordings from the indwelling catheter. Noninvasive monitoring will also be helpful during periods of problems with arterial blood pressure monitoring. Although not the ideal, hypotensive anesthesia may be carried out without invasive blood pressure monitoring, especially when the procedure is short and techniques are used that are not likely to cause rapid swings in blood pressure.

**Electrocardiogram.** Monitoring of the V5 lead of the ECG with an appropriate input filter, which is essential for ST-segment analysis, may give an indication of cardiac ischemia if it occurs and warn the anesthetist that the coronary blood supply is compromised.

**Oxygen saturation.** It is important to monitor oxygen saturation, as hypotension can cause a mismatch of ventilation and perfusion in the lungs as well as reduced blood flow to peripheral tissues. The pulse oximeter probe should not exert undue pressure on the finger tip.

**End-tidal carbon dioxide.** The measurement of the end-tidal carbon dioxide tension (EtCO2) will give an indication of the PaCO2 and will help prevent both hyperventilation and hypercarbia. However, the relationship between PaCO2 and EtCO2 changes with hypotension, so that arterial blood gas determinations also should be carried out intermittently to make sure that the PaCO2 is within the desired range.

**Temperature.** Core temperature monitoring is important during controlled hypotension, because body heat dissipates more rapidly from dilated vessels. Lowered temperature may decrease the effectiveness of vasodilators and increase the dose requirements if compensatory vasoconstriction occurs.

**Central venous pressure.** A greater hypotensive response will be exhibited by hypovolemic patients at the onset of controlled hypotension. Central venous pressure measurements will help in determining the adequacy of circulating blood volume at the start of hypotension and will help to maintain it during the period of controlled hypotension.

**Urine volume.** Urine output decreases during hypotensive anesthesia. However, some urine is formed. If the operation is to exceed 5 hr, a urinary catheter should be inserted. By measuring the urine output, it is possible to ensure that renal perfusion is maintained. Usually, once the hypotension is reversed, urine output reverts to normal.

**Blood loss.** The physiological response to blood loss may be lost during hypotensive anesthesia. Therefore, blood loss should be carefully estimated using swab weighing and measuring blood volume in suction bottles.

**Cardiac output.** The decrease in cardiac output during hypotensive anesthesia can be measured by the insertion of pulmonary artery catheters. The catheters can also help in monitoring the fluid status, left heart function, and mixed venous oxygen content.

**Fluid Therapy.** Proper fluid therapy is essential during hypotensive anesthesia. The aim of induced hypotension is to lower the MAP while maintaining adequate perfusion of all vital organs. Thus, preoperative fluid and electrolyte status must be assessed and corrected if necessary before anesthesia. During anesthesia, the fluid deficit acquired in the preoperative period because of fasting should be calculated and replaced during the first 1 or 2 hr. For maintenance, 5 to 6 mL/kg-hr of crystalloids should be infused. Blood loss must be carefully observed, and replacement carried out with either an equal amount of colloid or three times the amount in crystalloid. If the blood loss exceeds 20% to 25% of the patient's total blood volume, the loss should be replaced with blood. In operations where major blood loss is expected, it is preferable to insert a central venous catheter to ensure adequacy of the circulating volume.

Induction of hypotension should begin at the time of mucosal incision. That will give time to attain the required MAP before osteotomy. Once hypotension is induced, the required level of blood pressure to minimize blood loss may be maintained by adjusting the amount of hypotensive agent, either manually or automatically by self-tuning adaptive control.77,78

**Hypotension should be carried out only to that level needed to reduce bleeding and only for that time of the surgery where it is of benefit in reducing significant blood loss.**
Postoperative Management

After anesthesia with induced hypotension, adequate postoperative care with resuscitation facilities and experienced nursing staff is essential to prevent morbidity and/or mortality that can occur during this period. Postoperatively, attention should be given to airway maintenance, oxygenation, analgesia, monitoring, positioning, reactionary hemorrhage, and fluid balance. Persistent hypotension, which may be related to the positioning of the patient, a prolonged action of the hypotensive drugs, or both, may occur. Rebound hypertension, especially with inadequate analgesia, is also a concern during this period.

CONSIDERATIONS FOR FUTURE STUDIES

There are many deficiencies in the majority of the studies on hypotensive anesthesia carried out during orthognathic surgery. Results obtained from retrospective studies \(^5,9\) are unreliable compared to those of prospective studies \(^1,4,6,8,10,11\). Control of the age group \(^4,10\) and the physical status \(^2,4,10,11\) is important, as different age groups and patients of different physical status react differently to hypotensive agents. Furthermore, lack of sufficient number of patients \(^6\) results in inconclusive data. Orthognathic surgery consists of maxillary and mandibular surgery using different operative techniques. It is said that blood loss during maxillary osteotomies is greater than that of mandibular osteotomies and may vary with different techniques. A comparison should preferably be not of patients undergoing orthognathic surgery in general but should be specified to similar types of orthognathic surgery, \(^1-4\) such as Le Fort I operations. \(^8\) Many reported studies are uncontrolled \(^5,9,11\). To obtain valid results, controlled studies \(^1,4,6,8\) are necessary. The lowering of blood pressure to a percentage range of the preoperative blood pressure appears to be more rational than to a range of specific target blood pressures. \(^1,3,6,8-11\)

The technique of induced hypotension and the hypotensive drug(s) used should be the same for all patients in the hypotensive group during comparison of normotensive and hypotensive anesthesia. \(^1-3,5,6,9-11\) If more than one drug or technique is used, the results should provide comparisons of the different drugs and techniques. Assessment of the quality of the surgical field carried out blindly by the surgeons at frequent intervals is better \(^4\) than an overall assessment at the end of surgery. \(^6\) Preferably, the time of assessment should be stated. \(^4\) Estimation of blood loss may vary from the true blood loss. Preoperative and postoperative hemoglobin \(^7\) and hematocrit values \(^3,5,10\) have been used to supplement the calculation of blood loss and appear to be useful. Further studies designed with the aforementioned considerations, and if possible supplemented with the Doppler technique to quantify blood flow to the area of orthognathic surgery, \(^8\) will be able to confirm the benefits of hypotensive anesthesia during orthognathic surgery.

In conclusion, hypotensive anesthesia is undoubtedly of great value in improving the quality of the surgical field during orthognathic surgery. Although autologous blood transfusion and normovolemic hemodilution may be acceptable alternatives to homologous blood transfusion, they do not constitute a universal replacement for homologous blood transfusion. \(^190\) Reverse Trendelenberg position, sequential surgical site packing, keeping only the current site of operation exposed at any one time, injection of local anesthesia with epinephrine, and meticulous surgical technique with gentle tissue manipulation and use of cautery and vessel ties when appropriate are additional methods that help reduce blood loss. However, until satisfactory alternative methods are found to eliminate the need for homologous blood transfusion, it remains necessary to conduct hypotensive anesthesia during orthognathic surgery to prevent blood loss and transfusion therapy.

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