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Controlled Hypotension in Patients During Lumbar Fixation Surgery

Noha Mohamed Ali Mohamed Abdelkareem, Hala Abd-Elsadek Elattar, Heba Mohamed Fathi and Hatem Ahmed Nazmy

Anesthesia, Intensive care and pain management Department, Faculty of Medicine, Zagazig University, Egypt.

***Corresponding author: Noha Mohamed Ali Mohamed Abdelkareem**

E-mail: nohaabdelkareem414@gmail.com

Abstract:

Controlled hypotension is a deliberate and reversible reduction of arterial blood pressure induced during surgery to minimize intraoperative blood loss, improve visualization of the surgical field, and reduce the need for blood transfusion. It is commonly employed in procedures such as orthopedic, neurosurgical, and ENT surgeries where excessive bleeding may obscure the surgical site. Despite its benefits, controlled hypotension requires careful patient selection and monitoring due to the risks of hypoperfusion, tissue ischemia, and postoperative complications. This review aims to evaluate the efficacy, safety, and outcomes of controlled hypotension in surgical patients, focusing on hemodynamic stability, blood loss reduction, transfusion requirements, and recovery profiles.

Keywords: Controlled hypotension; deliberate hypotension; intraoperative blood loss; anesthesia management; surgical outcomes; patient safety; hemodynamic stability; intraoperative monitoring.

Introduction:

Deliberate hypotension is defined as the intentional reduction of the systemic perfusion pressure by reduction in systolic blood pressure (SBP) to 80-90mm Hg (30% decrease in the SBP from the baseline pressure) or a decrease in the mean arterial pressure (MAP) to 50-65 mm Hg in normotensive patients **(1)**.

Lumbosacral spine fixation surgeries are among the most common spinal surgical operations done by neurosurgeons and spine surgeons nowadays. These procedures are done due to different causes, which may be congenital, degenerative, neoplastic, or spine trauma. The underlying purpose of this surgery is to stabilize and fuse the lumbosacral spine **(2,3).**

Bleeding is known as an important intraoperative complication during spinal fusion that interferes with the success of the operation and increases the complications during and after the operation (4).

Controlled hypotension is one modality for reduction in bleeding during certain surgeries, as this technique must be gradual and within limits where mean arterial blood pressure is achieving patient safety. Hypotensive technique was achieved by reducing both the peripheral vascular resistance with maintaining or slight decrease of the heart rate within safety margin **(2)**.

By defining a reduction in blood pressure and heart rate, the goal of clearing the surgical field can be achieved **(3)**. Excessive bleeding during surgery, in addition to reducing the surgeon's view of the surgical field, causes more trauma to the surrounding tissues, and the longer the period of surgery. Controlled hypotension reduces bleeding from the surgical incision, thereby providing technical freedom and better vision for the surgeon in terms of operating more accurately **(4)**.

Many anesthetic agents and vasoactive drugs are used frequently to produce controlled hypotension, including inhalational anesthetics, direct-acting vasodilators, autonomic ganglion blockers, β-adrenergic blockers, and calcium channel blockers **(5)**.

History and evolution of controlled hypotension(6):

- Deliberate hypotension was first introduced in 1917 in order to provide a bloodless field for surgeries.
- In 1946, the concept of induced hypotension using arteriotomy to produce a bloodless field was introduced.
- In 1948, high spinal anesthesia was used to induce hypotension and create a dry field.
- In 1951 the high epidural block was introduced.
- In 1962, sodium nitroprusside was first used to induce hypotension during anesthesia

Physiology of blood pressure:

The cardiovascular system provides blood supply throughout the body. Response to various stimuli can control the velocity and amount of blood carried through the vessels. The cardiovascular system comprises the heart, arteries, veins, and capillaries. The heart and vessels work intricately to provide adequate blood flow to all body parts. The regulation of the cardiovascular system occurs via a lot of stimuli, including changing blood volume, hormones, electrolytes, osmolarity, medications, adrenal glands, kidneys, and much more. The parasympathetic and sympathetic nervous systems also play a key role in regulating the cardiovascular system **(7)**.

The cardiovascular system consists of 2 main loops: systemic circulation and pulmonary circulation. Its purpose is to provide adequate blood circulation through the body. Pulmonary circulation allows for the oxygenation of the blood, and systemic circulation allows oxygenated blood and nutrients to reach the rest of the body **(8)**.

To understand the physiology of the heart, it is important to understand the cardiac output, stroke volume, preload, Frank-Starling law, afterload, and ejection fraction **(9)**.

- **Cardiac Output:**

The cardiac output (CO) is the amount of blood ejected from the left ventricle; normally, it equals the venous return. The calculation is CO = stroke volume (SV) x heart rate (HR). It is usually expressed in litres per minute (L/min). Normal values for a resting healthy individual would be approximately 5-8L **(10)**.

- **Stroke Volume:**

The SV is the amount of blood pumped out of the heart after 1 contraction. It is the difference between end-diastolic (EDV) and end-systolic (ESV) volume. It increases with increased contractility, increased preload, and decreased afterload.

Normal values for a resting healthy individual would be approximately 60-100mL **(11)**.

- **Preload:**

The preload is the pressure on the ventricular muscle by the ventricular end diastolic volume (EDV) **(12)**.

- **Frank-Starling Law:**

Frank-Starling law describes the relationship between EDV and SV. This law states that the heart attempts to equalize CO with venous return. As venous return increases, a larger EDV in the left ventricle leads to further stretching of the ventricle, leading to a larger contraction force and a larger SV. A larger SV leads to a larger CO, thus equalizing CO with venous return **(12)**.

- **Afterload:**

Afterload is the pressure the left ventricular must exceed to push blood forward. Mean arterial pressure best estimates this. Also, afterload can be estimated by the minimum pressure needed to open the aortic valve, equivalent to the diastolic pressure. Thus, diastolic blood pressure is one of the better ways to index afterload **(11)**.

- **Diastolic Blood Pressure:**

Diastolic blood pressure (DP) is the lowest pressure in an artery at the beginning of the cardiac cycle while the ventricles are relaxing and filling. DP is directly proportional to total peripheral resistance (TPR). Also, the energy stored in the compliant aorta during systole is now released by the recoil of the aortic wall during diastole, thus increasing diastolic pressure .normally it should be less than 80 mmHg **(9)**.

- **Systolic Blood Pressure:**

Systolic blood pressure (SP) is the peak pressure in an artery at the end of the cardiac cycle while the ventricles contract. It is directly related to stroke volume; as stroke volume increases, SP also increases. SP is also affected by aortic compliance. Because the aorta is elastic, it stretches and stores the energy caused by ventricular contraction, decreasing the SP. In normal individuals it should be less than 120 mmHg **(9)**.

- **Pulse Pressure:**

Pulse pressure is the difference between systolic and diastolic blood pressure. Pulse pressure is proportional to SV and inversely proportional to arterial compliance. Thus, the stiffer the artery, the larger the pulse pressure. Normal pulse pressure is approximately 40 mmHg **(13)**.

- **Mean Arterial Pressure:**

Mean arterial pressure (MAP) is the average pressure in the arteries throughout the cardiac cycle. The MAP is always closer to DP. MAP is calculated by MAP= DP + 1/3 (pulse pressure). Also, MAP = CO x TPR. This value is significant because whenever CO decreases, the TPR increases to maintain the MAP, which is relevant in many pathophysiology problems **(14)**.

Regulation of blood pressure through Baroreceptors and Chemoreceptors:

The nervous system regulates the cardiovascular system with the help of baroreceptors and chemoreceptors. Both receptors are located in the carotid and aortic arch. Also, both have afferent signals through the vagus nerve from the aortic arch and afferent signals through the glossopharyngeal nerve from the carotids **(15)**.

Baroreceptors are more specifically located in the carotid sinus and aortic arch. They respond quickly to changes in blood pressure. A decrease in blood pressure or blood volume causes hypotension, which leads to a decrease in arterial pressure which decreases the baroreceptors' stretch and decreases afferent baroreceptor signaling. This decrease in afferent signaling from the baroreceptor causes an increase in efferent sympathetic activity and a reduction in parasympathetic activity, which leads to vasoconstriction, increased heart rate, increased contractility, and an increase in BP. The vasoconstriction increases TPR in the equation MAP=CO x TPR to increase pressure (MAP). An increase in blood pressure or blood volume causes hypertension, increasing the baroreceptors' stretch **(16)**.

Chemoreceptors come in 2 types: peripheral and central. Peripheral chemoreceptors are specifically located in the carotid body and aortic arch. They respond to oxygen levels, carbon dioxide levels, and the pH of the blood. They become stimulated when oxygen decreases, carbon dioxide increases and the pH decreases. Central chemoreceptors are located in the medulla oblongata and measure the cerebral spinal fluid's pH and carbon dioxide changes **(17)**.

Autoregulation(9):

Autoregulation is how an organ or tissue maintains blood flow despite a change in perfusion pressure. When blood flow decreases to an organ, arterioles dilate to reduce resistance. There are two theories of auto regulation**:**

- **Myogenic theory:** Myogenic regulation is intrinsic to the vascular smooth muscle. When there is an increase in perfusion, the vascular smooth muscle is stretched. This causes it to constrict the artery. If there is a decrease in perfusion to the arteriole, then there is decreased stretching of the smooth muscle. This leads to the smooth muscles' relaxation and arteriole dilation.

- **Metabolic theory:** Blood flow is closely related to metabolic activity. When there is an increase in metabolism to muscle or any tissue, there is an increase in blood flow to that location. Metabolic activity creates substances that are vasoactive and stimulate vasodilation. The increase or decrease in metabolism leads to increased or decreased metabolic byproducts that cause vasodilation. Increased adenosine, carbon dioxide, potassium, hydrogen ions, lactic acid levels, decreased oxygen levels, and increased oxygen demand all lead to vasodilation.

Techniques of controlled Hypotension:

1) Positioning:

The Anti-Trendelenburg position: by Placing the operated area higher than the heart [Figure 1]. This reduces blood pressure by orthostatic or postural hypotension and decreases venous pressure in operating area. Blood pressure return quickly to normal level after patient repositioning. However, there is a risk of air embolism with this technique **(18).**

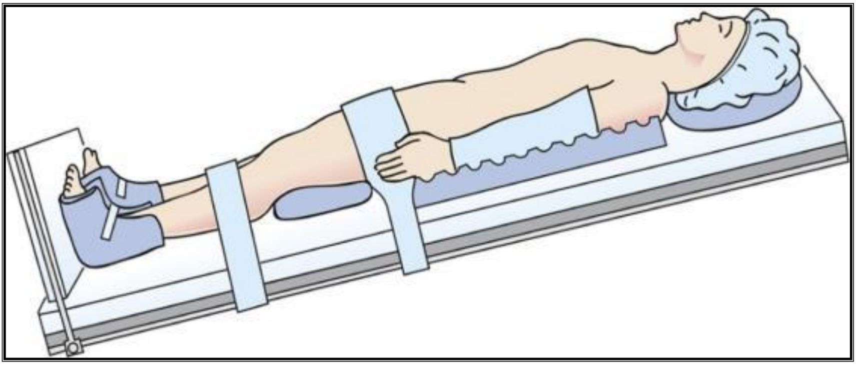

Figure (1): The Anti-Trendelenburg position **(19).**

2) Regional Anesthesia:

Epidural anesthesia and spinal anesthesia have been considered good controlled hypotensive techniques as they reduce blood pressure and bleeding **(5).**

Spinal anesthesia causes a sympathetic block, which results in fall of peripheral vascular resistance and cardiac output (CO) via venous return reduction and thus, systemic hypotension. The disadvantage of spinal anesthesia is that the relationship between drug dosage and the degree of hypotension remains unpredictable. Also, it's difficult to control the duration of action, as it's very variable **(20).**

It was showed that epidural anesthesia results in less intraoperative blood loss than total intravenous anesthesia with propofol plus remifentanil during primary total hip replacement surgery and concluded that it appears to be the most effective method for bleeding reduction in this type of surgery **(21).**

3) Anesthetic drugs:

There are two main strategies for achieving hypotensive anesthesia **(6).**

- **Increasing the level of anesthesia /analgesia.**
- **Standard anesthesia and hypotensive agents administration.**

By deepening the anesthesia and using high doses of analgesics, such as opioids, the recovery time may be prolonged. On the other hand, administering a hypotensive agent to a patient who is anesthetized using a standard anesthetic protocol may result in postoperative hypotension. In practice, the two strategies are used to achieve controlled hypotensive anesthesia.

1) Increasing the level of anesthesia /analgesia:

- **Anesthesia:**

Increasing the level of anesthesia either inhalational or intravenous as following:

Inhalational anesthesia:

The potent vasodilator action of volatile anesthetic agents, such as isoflurane and sevoflurane makes them used in induced hypotensive techniques. However, when volatile anesthetics are used alone, high concentrations are required to achieve a significant reduction in intraoperative bleeding, and these higher concentrations may lead to hepatic or renal injury. In addition, the volatile-mediated reduction in blood pressure is not meticulously controlled **(22).**

Studies have used isoflurane in combination with alprostadil, nitroglycerin, adenosine, morphine, labetalol, esmolol or dexmedetomidine, in order to reduce each agent concentration and their respective adverse effects**. Rossi et al. (22)** have compared sevoflurane with desflurane and showed that sevoflurane like isoflurane has systemic and coronary vasodilator effect at average or high concentrations, without hepatic toxicity but with an increased risk of renal impairment. These drawbacks recommend other hypotensive agents addition to reduce the concentration and toxicity of each agent.

On the evidence bases, inhalation anesthetics are valuable because of their major role as hypnotics in current adult anesthesia and increasingly in pediatric anesthesia. At safe clinical concentration, only slight hypotension happens, and adjunctive hypotensive agents are needed to induce a truly controlled hypotension without adverse effects **(23).**

Intravenous anesthesia:

Intravenous anesthesia as Propofol is a widely used intravenous anesthetic agent which has a potent hypotensive ability. It has the advantages of achieving hypotensive anesthesia when administered as part of total intravenous anesthesia and the rapid restoration of normal blood pressure when propofol infusion is discontinued. There is no problem with short-term propofol infusion, but a long-term propofol infusion can cause propofol infusion syndrome in children "The syndrome is characterized by progressive and refractory metabolic acidosis, lipemia, brady-arrhythmias, hepatomegaly, rhabdomyolysis, hemodynamic instability and culminating cardiovascular collapse" **(24).**

Propofol was compared with isoflurane for hypotensive anesthesia and found no significant difference in intraoperative blood loss and operative conditions **(25).**

- **Analgesia:**

Opioids:

Their analgesic effects make them widely used in anesthesia. They have been used as additives to other hypotensive agents in controlled hypotension. Alfentanil, Sufentanil, and Remifentanil are potent synthetic and short acting opioid drugs. When used for hypotensive anesthesia; each of these three drugs is effective in achieving hypotensive anesthesia for the required duration since the recovery times from this type of anesthesia are short. *Alfentanil*, a fentanyl derivative, has quicker onset and shorter duration of action than fentanyl and its vago-mimetic properties are more intense than those of fentanyl and sufentanil. *Sufentanil* is a more potent analgesic than fentanyl and seems better than the other opioid analgesics, such as morphine or meperidine, in maintaining hemodynamic stability during surgery. *Remifentanil* is a potent mu-opioid receptor agonist that is rapidly metabolized by non-specific blood and tissue esterases **(26).**

According to its unique pharmacokinetic properties, remifentanil- based hypotensive anesthesia provides intraoperative analgesia with a rapid and predictable postoperative awakening, which is independent of the duration of the infusion. It appears to offer a superior intraoperative hemodynamic stability during stressful surgical events and maintains intact cerebral blood flow reactivity. It reduces blood loss and better visibility of the surgical field when combined with propofol or inhalational anesthesia than other opioids **(27).**

2) Standard anesthesia and hypotensive agents administration:

The ideal agent that can induce controlled hypotension has certain criteria which does not exist in one agent **(6)**.

- Easy to administer.
- Has a short onset time.
- Its effect disappears quickly when administration ends.
- Fast elimination without toxic metabolites.
- Negligible effects on vital organs.
- A predictable and dose-dependent effect.

Agents used in controlled hypotension:

(1) Vasodilators:

A-Sodium Nitroprusside (SNP): It is the most used hypotensive agent since the 1950s and is always used as a reference agent due to:

1) Its direct peripheral vasodilator effect acting directly on the vascular smooth muscle causing generalized vasodilatation through releasing of nitric oxide “a naturally occurring potent vasodilator released by endothelial cells (endothelium- derived relaxing factor) (EDRF)” plays an essential role in regulating vascular tone throughout the body.

2) Its onset time is very fast (<30 seconds) and the hypotensive effect does not exceed 2 minutes after administration ceases **(28).**

B-Nitroglycerin: A non-specific, direct vasodilator for the veins and incidentally for the arteries; it has a short half-life and no clinically toxic metabolites. It decreases cardiac output proportionally by increasing capacity of venous blood vessels and reducing venous return. The anesthetic agents partially block the adrenergic response and that limit the arterial vasoconstriction resulting from this phenomenon **(29).**

C-Non-Nitrovasodilator Hypotensive Agents:

Hydralazine: increases cyclic guanosine monophosphate (cGMP) causing pre-capillary resistance vessels dilatation via arteriolar smooth muscles relaxation. An intravenous dose of 5–20 mg of hydralazine can control intraoperative hypertension. The onset of action is within 15 min and the anti-hypertensive effect usually lasts for 2-4 hours. Hydralazine undergoes acetylation and hydroxylation in the liver **(30).**

Fenoldopam: A direct vasodilator has a selective peripheral dopamine receptor agonist that affects the renal, coronary, cerebral, muscular skeletal and splanchnic circulations. It also shows a moderate affinity for α-2 adrenoceptors. Fenoldopam produces arterial vasodilatation, lowering blood pressure especially in malignant hypertensive patients to an extent comparable to nitroprusside, in children and in recovery from cardiac surgery without impairing coronary revascularization or causing coronary spasm **(31).**

Adenosine: A natural substance derived from purines, formed from the metabolism adenosine triphosphate after intravenous administration; further metabolism results in uric acid. Adenosine causes dose-dependent systemic and coronary arterial vasodilatation. Its administration increases plasma renin activity and catecholamine levels, in addition to increased cerebral blood flow and impairment of the auto-regulatory function of the cerebral micro-vascular circulation **(32).**

Alprostadil: A prostaglandin E1 synthetic analogue decreases arterial pressure by lowering vascular resistance, it has an intrinsic negative chronotropic effect causing limitation in the reflex tachycardia noted with other vasodilators **(33).**

Calcium Channel Blockers (CCBs):

Clevidipine: is a relatively new drug with a short half- life, which assists its rapid titration. Unlike verapamil and diltiazem, the dihydropyridine CCBs have minimal effects on cardiac conduction and ventricular contractility. Calcium channel blockers bind to L-type calcium channel and impair calcium entry into the vascular smooth muscle. These L-type receptors are more prevalent on arterial vessels than venous capacitance vessels. Consequently, cardiac filling and preload is less affected by these agents than nitrates. When preload is maintained, cardiac output often increases when vascular tone is reduced by use of dihydropyridine CCBs **(34).**

Diltiazem: A calcium blocker has been used successfully as an adjunct to reduce blood loss and to reduce sodium nitroprusside required dose by half during spinal surgery. It has negative chronotropic and dromotropic effects **(35).**

Nicardipine: unlike nitroglycerin and alprostadil, causes a decrease in cerebral microvascular autoregulation during anesthesia with propofol plus fentanyl. It has been used mainly in orthopedic surgery, where it causes prolonged hypotension reducing blood loss. Therefore, it avoids rebound hypertension, despite the increase in plasma renin activity and catecholamine levels. In spinal surgery, nicardipine reduced blood loss to the same extent as isoflurane. The utility and effectiveness of nicardipine have also been reported in pediatric surgery in association with isoflurane and sufentanil **(35).**

Mg sulphate: It functions as a calcium channel blocker, anesthetic, tocolytic, anti-arrhythmia, analgesic, anticonvulsant and cardiovascular medication. It consists of a metal sulphate, an organic magnesium salt, and a magnesium salt**(36)**.

(2) The Autonomic Nervous System Inhibitors:

a. Clonidine: A central α-2 adrenoceptor agonist, which diminishes sympathetic nerve impulses and so induces bradycardia and hypotension, indicating a use as an adjunct to other agents in controlled hypotension (isoflurane, labetalol, urapidil). It has only been successfully used as an oral premedication because it has a sustained and unpredictable effect **(37).**

b. Dexmedetomidine (Precedex): is a potent parenteral selective α-2 agonist with sedative properties. It appears to be more selective for the α-2 receptor than clonidine. Dexmedetomidine (DEX) causes dose-dependent sedation, anxiolysis and some analgesia and blunts the sympathetic response to surgery and stress. It has an opioid-sparing effect and does not significantly cause respiratory drive depression and excessive sedation **(38).**

c. Labetalol: A competitive antagonist at β-1, β-2 adrenoceptors, and α-1 adrenoceptors. It decreases myocardial contractility and heart rate via its β-adrenoceptor effects and causes vasodilatation via its α- adrenoceptors antagonistic effects. Its onset time is slow (5–10 minutes), with a peak effect from 1–3 hours **(39).**

D. Phentolamine: Reversible, non selective alpha blocker that induce vasodilatation **(40)**.

Patients selection and Limitations:

In spite of there were no specific complications seen in a large series of patients subjected to severe controlled hypotension (MAP < 50mmHg) over a prolonged duration, the risk of tissue hypoxia and the difficulty in evaluating this risk are very real **(26).**

Controlled hypotension could affect end-organ perfusion by suppressing the microcirculatory autoregulation of the vital organs and inhibiting the autonomic nervous system. The aim of controlled hypotension is to maintain a pressure sufficiently low to reduce bleeding with preserved microcirculatory autoregulation of vital organs (i.e. brain, heart or kidney) **(26).**

Controlled hypotension with a MAP between 50 and 65 mmHg is not risky in young healthy patients. The mortality due to controlled hypotension is almost the same rate of all anesthesia types.The morbidity resulting from it had a very low variable incidence as neurological complications (dizziness, cerebral, retinal, and cerebellar thrombosis) and anuria **(41).**

- **Induced hypotension is unsafe in some patients such as(6):**
- Renal, cerebral, and coronary artery diseases.
- Anemia, hemoglobinopathies, and polycythemia.
- Hypovolemia.
- Hepatic dysfunction.
- Respiratory insufficiency.
- Severe systemic hypertension.
- Patients with disseminated vascular disease, in whom atheromatous vessels which provide organ perfusion, may suffer from hypo-perfusion during hypotension.
- Patients with ischemic heart disease or carotid artery stenosis are candidates for normotensive anesthesia or, in selective cases, minor blood pressure reduction, this is called “modified hypotensive anesthesia”, adjusted to their condition.

Complications of controlled hypotension:

Main concern in deliberate hypotensive technique is the risk of end- organ hypo perfusion and tissue hypoxia and the risks are real and difficult to evaluate in each patient. The risks depend on individual patients physical and functional status, degree of hypotension, type, and duration of surgery, associated medical conditions, the knowledge and skill of the concerned anesthesiologist and facilities for monitoring etc. Induced hypotension can result in tissue hypoxia by reducing or suppressing the autoregulation of vital organs at the microcirculation level and by inhibiting the autonomic nervous system. Current goal of deliberate hypotension is to preserve this autoregulation by maintaining a pressure just low enough to allow reduction in bleeding without compromising the microcirculation of vital organs. Absolute contraindications are mainly unfamiliarity with the technique, inability to monitor the patient adequately and lack of understanding of the techniques. Whereas, patient related factors are presence of cardiac disease, ischemic cerebrovascular disease, hepatic or renal impairment, respiratory insufficiency, severe systemic hypertension, diabetes mellitus, hematological problems such as anemia, polycythemia, hemoglobinopathies, sickle cell anemia, and intolerance to drugs available to produce hypotension**(42)**.

These groups of patients are candidates for normotensive anesthesia or in selected cases, a modified hypotensive anesthesia in the form of mild to moderate reduction in blood pressure adjusted to their medical conditions. Well treated hypertension may not pose greater risk and is not considered as contraindication for controlled hypotension **(43).**

The risk of combining controlled hypotension with various techniques of autologous transfusion must be weighted since this combination with surgical stimuli can lead to marked decrease in central venous oxygen saturation (SvO2) indicating impairment in tissue oxygenation **(44).**

Monitoring:

- **Blood pressure**: Invasive blood pressure monitoring is recommended as it not only gives immediate and often more accurate blood pressure data than non- invasive blood pressure monitors but also permits sampling for arterial blood gases, hematocrit and other laboratory investigations when needed. Although not the ideal, hypotensive anesthesia may be carried out without invasive blood pressure monitoring especially in short procedures, and techniques used that are not likely to cause rapid blood pressure swings **(20).**

- **Fluid Therapy:** An experienced anesthesiologist can assess the patient's fluid volume status and vasoconstriction degree based on the integration of different data. First is the calculation of "ins and outs", the kind and amount of fluid administered, and the estimated fluid lost or consumed, including hemorrhage volume, metabolic maintenance and insensible losses to atmospheric evaporation. This is an important exercise to perform, particularly for long cases. Even when meticulously calculated, paper estimates of fluid requirements often underestimate the actual need **(26).**

- **Patient's anesthetic requirement:** the total dose of narcotic, volatile, and sedative medication maintaining the anesthetized state. It is easy for the inexperienced practitioner to incrementally reduce the anesthetic dose in response to decreases in blood pressure caused by hypovolemia, particularly in the presence of muscle relaxants that prevent patient movement. This leads to intraoperative patient awareness. Even more insidious, this may move the patient to a physiologic state of occult shock "severe hypovolemia is being compensated by catecholamine production and peripheral vasoconstriction" **(20).**

- **Five leads electrocardiogram**: Which is essential for ST-segment analysis, may give an indication of cardiac ischemia if it occurs and warn the anesthetist of compromised coronary blood supply **(26).**

- **Oxygen saturation:** As hypotension can cause ventilation/ perfusion mismatch as well as reduced blood flow to peripheral tissues, it is important to monitor oxygen saturation **(26).**

- **End-tidal carbon dioxide (Capnography):** Gives an indication of the PaCO2 tension and will help to prevent both hypo and hypercarbia. But it does not eliminate the need of arterial blood gas as the relationship between PaCO2 and End Tidal CO2 (EtCO2) is altered during hypotension **(20).**

- **Urine output (UOP):** Decreases during hypotensive anesthesia. However, some urine is formed. If the operation is to exceed 5 hours, a urinary catheter is a must. Measuring the urine output is the insurance that renal perfusion is maintained. Usually, once the hypotension is reversed, urine output reverts to normal **(26).**

- **Blood loss:** During hypotensive anesthesia, the physiological response to blood loss may be lost; Therefore, blood loss should be carefully estimated by measuring blood volume in suction bottles **(20).**

- **Core temperature monitoring:** Is important because body heat dissipates more rapidly from dilated vessels. Lowered temperature may decrease vasodilators effectiveness and increase the dose requirements if compensatory vasoconstriction occurs **(26).**

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