CONTROLLED HYPOTENSIVE ANAESTHESIA

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INTRODUCTION

Induced or controlled hypotension is a method by which the arterial blood pressure is decreased in a predictable and deliberate manner. The intent of deliberate hypotension is to reduce bleeding and thus facilitate surgery and to decrease the amount of blood transfused. In certain surgical procedures, notably middle ear surgery, the amount of bleeding does not have to be great to obscure the operative area and jeopardize success.

In other procedures, a dry field may result in a more definitive removal of a neoplasm as well as in less damage to vital structures. Finally, trauma and tissue infection are minimized because fewer sutures are required and less electrocoagulated, devitalized tissue remains in the wound.

DEFINITION

The definition of controlled hypotensive anaesthesia is taken from the very early studies done by ECKENHOFF AND RICH in 1966. It takes into account the level required to produce the effect but at the same time is limited by safety. The level of hypotension should not be determined beforehand but rather provided until the desired level (for a bloodless field) is obtained but within the safety limits of cerebral and coronary flow. This safety limit is individualised.

Generally, it is taken that a MAP as low as 50 mmHG or a 30% drop in MAP is safe for an ASA 1 subject. This might not be appropriate for a chronic hypertensive patient who may not tolerate a drop of more than 25% of the MAP. Again, this level might not be appropriate for a patient with cerebrovascular disease who may not tolerate any drop whatsoever in the MAP.
HISTORY AND EVOLUTION

History and Evolution of Controlled Hypotension

Deliberate hypotension was first introduced in 1917 in order to provide a bloodless field for neurosurgery.

In 1946, the concept of induced hypotension using arteriotomy to produce a bloodless field was introduced.

In 1948, high spinal anaesthesia was use to induce hypotension and create a dry field whilst in 1951 the high epidural block was introduced.

Subsequently, the technique of ganglion blockade using pentamethonium, hexamethonium and trimethaphan was used to induce hypotension during anaesthesia.

Thereafter, deep anaesthesia with volatile inhalational agents was favoured by many.

In 1962, sodium nitroprusside was first used to induce hypotension during anaesthesia.

Since then, nitroglycerine, calcium channel blockers, beta blockers, purine compounds and prostaglandin E1 have been used.

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 to the vital organs. Controlled hypotension rarely results in damage, however, because organ blood flow is normally well maintained.

Flow is a function of both MAP and autoregulation in the cerebral, myocardial and renal beds. Using the concept of MAP (rather than systolic blood pressure), the physiology of these 3 systems needs to be examined separately to determine which is the critical “weak link” i.e. the system that sets the minimal permissible pressure.
The mechanisms of autoregulation include:

1. stretch- myogenic mechanism: the smooth muscle in the vasculature responds to altered tension

2. passive mechanical: applies to encapsulated organs, where expansion of the organ with increasing pressure compresses thin walled vessels and leads to an increase in vascular resistance.

3. metabolic: changes in pressure produces vasoactive substances.

Those organs capable of autoregulation are able to maintain their perfusion over a wide range of pressure changes, and it is only when the pressure decreases to relatively low levels that adequate perfusion cannot be maintained. This critical pressure varies from vessel to vessel, organ to organ, and probably from individual to individual.

CEREBRAL CIRCULATION

Many feel that it is the perfusion of the cerebral circulation that is the critical factor that limits MAP reduction. This is probably because even minimal derangements in post operative function of this organ are unacceptable. Through autoregulation, normal cerebral blood flow is maintained at 45-50mls/100g/min, through a MAP range of 50-150mmHg (Fig 1).
The absolute value for cerebral blood flow below which cerebral ischaemic hypoxia develops is not known but several studies have allowed the estimate to be made. Sundt et al measured cerebral blood flow with xenon and simultaneously recorded EEG during carotid endarterectomy with halothane anaesthesia and normocapnia.

They found no changes indicative of ischaemia if cerebral blood flow was above 25mls/100g/min. If this flow rate is taken as the critical limit, it is possible to estimate a critical perfusion pressure below which ischaemia might occur (Fig 2).
However, several factors under the control of the anaesthesiologist modify the calculation:

1. **PaCO2**

   Within the range of autoregulation, the arterial PCO2 is the most important factor in determining cerebral blood flow. For every 1mmHg increase in PaCO2 there is an increase in cerebral blood flow in the order of 1mls/100g/min and vice versa (Fig 3)
These effects of carbon dioxide are obviously relevant in the clinical situation and should be remembered when hypocapnia is part is part of the induced hypotensive technique. The combination of hypotension and the cerebral vasoconstrictive effects of a low arterial PCO₂ could be detrimental to cerebral function.

2. PaO₂

Changes in arterial oxygen tension also affect cerebral blood flow. Since high oxygen tensions, particularly in the hyperbaric range, can produce toxic effects on cerebral function, the brain protects itself by vasoconstriction. For example, inhalation of 100% oxygen reduces the cerebral blood flow by about 1/5. If the arterial oxygen tension decreases below normal, cerebral blood flow increases because of vasodilation. It may therefore be that the practise of administering high concentrations of oxygen during induced hypotension is not beneficial to cerebral blood flow, although added oxygen may be necessary to offset the effects of hypotension on pulmonary gas exchange.
3. VOLATILES

Volatile anaesthetics attenuate or abolish the autoregulation of cerebral blood flow in a dose dependent manner in the following order: halothane > enflurane > isoflurane.

![Figure 4 EFFECT OF VOLATILES ON CEREBRAL BLOOD FLOW](BRITISH JOURNAL OF ANAESTHESIA, 1980)

4. TEMPERATURE

A linear relationship exists between cerebral blood flow and temperature. Hypothermia causes cerebral vasoconstriction whereas an increase in body temperature causes cerebral vasodilation. Cerebral blood flow changes 5-7% per degree celcius change in temperature.
5. VASODILATORS

Vasodilators such as nitroprusside and nitroglycerine attenuate the autoregulation of cerebral blood flow in a similar manner to that of volatile agents.

A final difficulty arising from the discussion of cerebral blood flow is when overall flow is contrasted to regional blood flow. In regions of marginal flow, local factors such as hypoxia or acidosis may predominate, which may cause maximal dilatation of vessels in that area.

Since the skull limits the volume of blood that can be accommodated, events that increase blood flow to areas of normal perfusion may displace flow that went to areas of marginal perfusion. Because of this uncertainty of regional blood flow distribution, a safety factor is inserted into the determination critical pressures. Assuming the use of volatiles with the attendant shift in the cerebral autoregulatory curve and an essentially normal PaCO2, it should be safe to lower the MAP to 50mmHg with no significant decrease in cerebral blood flow.

The hypertensive patient deserves special comment since it has been shown that the autoregulatory curve is shifted higher at both ends. Theoretical safe limits of hypotension may be calculated based on this shifted curve. However, these patients do pose an increased risk, even when the elevated MAP is accounted for. Therefore, hypertension poses a relative contraindication to controlled hypotension.

Elevation of the head during hypotensive anaesthesia can aggravate the decrease in cerebral perfusion pressure. The perfusion pressure decreases by 2mmHg for every 2.5cm the head is raised above the point of monitoring.

CORONARY CIRCULATION

Coronary blood flow is dependent upon the aortic diastolic blood pressure and the coronary vascular resistance. Control of coronary blood flow is autoregulated predominantly by means of alteration in coronary vascular resistance that are made to meet myocardial oxygen demand. Even at rest the myocardium extracts most of the oxygen delivered to it. Hence, any increase in myocardial oxygen demand requires a parallel increase in coronary artery blood flow.
Hypotensive anaesthesia may substantially decrease coronary blood flow. However, it simultaneously decreases myocardial oxygen demand due to the reduction in afterload or/and preload. Furthermore, coronary autoregulation ensures adequate myocardial blood flow. Studies have shown that during hypotensive anaesthesia there is a poor correlation between the lowest degree of hypotension achieved and the development of ischaemic ECG changes (Rollason et al, 1959).

Patients with coronary artery disease may have some areas of myocardium that are entirely dependent upon pressure to supply adequate blood flow. In addition, the use of vasodilators in these patients may induce a steal phenomenon. Hence, controlled hypotensive anaesthesia is accompanied by significant intraoperative risk of myocardial infarction.

In a study done by Rollason and Hough in 1959, there was no ecg evidence of myocardial damage following induced hypotension with ganglion blocking agents although about 40% of cases showed evidence of transient myocardial ischaemia. These changes were observed in elderly patients or patients with pre-existing hypertension when systolic arterial blood pressure fell below 60mmHg. In these patients a MAP of at least 80mmHg should be maintained at all times whenever induced hypotension is contemplated.

**RENAL CIRCULATION**

Renal blood flow is controlled in two ways: extrinsic autonomic and hormonal mechanisms and intrinsic autoregulation.

Miles, Venton and De Wardener (1954) showed that there was autoregulation of blood flow over the range 80-180mmHg. Autoregulation of renal blood flow is attenuated during general anaesthesia and decreased renal blood flow occurs with even moderate decreases in arterial blood pressure (systolic value of 80-90mmHg, Larson et al, 1974).

If arterial blood pressure drops below these values renal blood flow may decrease to a point where urine flow stops. Also, when the MAP drops below 75mmHg GFR falls (Fig 5).
In addition, opioids and most inhalational agents stimulate secretion of antidiuretic hormone. All these factors result in oliguria during hypotensive anaesthesia. As with the brain, there is likely some critical value of renal blood flow below which acute renal failure may ensue. This probably varies from individual to individual and it may be related to pre-existing renal damage. Provided therefore that induced hypotension does not reduce renal blood flow below the critical value for the kidney, it is unlikely that serious renal damage will ensue. In fact, it has been shown that following termination of hypotensive anaesthesia, urine formation rapidly recovers provided the patient was well hydrated.

HEPATIC CIRCULATION

Most of the liver blood flow (70%) is via the portal vein. The remainder of the liver blood flow is supplied from the hepatic artery. The splanchnic circulation is richly innervated by the sympathetic nervous system. In contrast to the brain and kidney, the liver is not an autoregulated organ. Therefore a decrease in arterial pressure will lead to a decrease in liver blood flow. In addition, an increase in PaCO2 or a decrease in PaO2 will
lead to a catecholamine response which causes splanchnic vasoconstriction and therefore a decrease in liver blood flow. Also, hypocapnia produced incidentally by hyperventilation during IPPV leads to a decrease in liver blood flow as a result of the mechanical effects.

Apart from the effects of hypotension itself, liver blood flow may be altered directly by the effects of anaesthetic agents on splanchnic blood flow.

Studies in animals have shown that all volatiles cause a reduction in MAP and cardiac output but a more pronounced reduction in total hepatic blood flow, portal blood flow and hepatic artery blood flow is seen with halothane (Fig 6).

![Figure 6 IMPACT OF VOLATILES ON TOTAL HEPATIC FLOW](image)

**Figure 6 IMPACT OF VOLATILES ON TOTAL HEPATIC FLOW**

( MILLER, 6th EDITION )

![Figure 7 IMPACT OF VOLATILES ON HEPATIC ARTERIAL OXYGEN DELIVERY](image)

**Figure 7 IMPACT OF VOLATILES ON HEPATIC ARTERIAL OXYGEN DELIVERY**

( MILLER, 6th EDITION)
Studies in animals also show that halothane causes the greatest reduction in hepatic oxygen delivery (Fig 7).

Based on limited clinical and experimental data, it appears that intravenous anaesthetic agents have only a modest impact on hepatic artery blood flow and no meaningful adverse influence on post operative liver function. In fact studies have shown that propofol actually increases blood flow in both the hepatic arterial and portal venous systems as a result of a direct vasodilator effect on the splanchnics.

Regardless of the above effects of volatiles on total hepatic blood flow, deliberate hypotension seems to be well tolerated by the liver and there are no reports showing morbidity or mortality from hepatic hypoperfusion during deliberate hypotension.

RESPIRATORY SYSTEM

During controlled hypotensive anaesthesia the following occurs:

- Pulmonary blood flow gravitates to the dependent areas of the lungs. Hence, the non dependent regions are ventilated but not adequately perfused thus increasing dead space. This scenario is aggravated by the head up position.
- The use of vasodilators to induce hypotension inhibits the hypoxic pulmonary vasoconstriction response thereby increasing intra-pulmonary shunt.

All these factors result in hypercarbia, an increase in arterial-end tidal CO2 gradient and hypoxaemia. Hence, regular PaCO2 measurements are necessary during controlled hypotension. In addition, a higher FiO2 may be necessary.

BLOOD PRESSURE GOAL

The aim of hypotensive anaesthesia is to reduce blood loss and provide a “dry” operating field. Hence, the degree of hypotension should be individualised. The hypotension should be considered satisfactory when bleeding appears to be minimal and organ perfusion adequate. Theoretically, as long as the MAP exceeds the sum of colloid osmotic pressure and venous pressure, the blood flow should be adequate to meet the tissue needs. Again theoretically, a MAP of 32mmHg should suffice but this may probably be below the safe limit due to specific organ
flow requirements and the possibility of disease and other causes of altered circulation.

Hypotensive anaesthesia 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 from it as long as organ perfusion is adequate.

- With reference to the discussion on physiology above, it is suggested that inducing hypotension to a MAP of 30% below a patient’s usual MAP, with a minimum of 50mmHg in ASA 1 patients and 80mmHg in the elderly is clinically acceptable.

CONTRAINDICATIONS

Contraindications to the use of Hypotensive Anaesthesia

ANAESTHETIST LIMITATIONS

- Lack of understanding of the technique
- Lack of technical expertise
- Inability to monitor the patient adequately

PATIENT LIMITATIONS

- Cardiac disease
- Diabetes mellitus
- Anaemia, haemoglobinopathies, polycythaemia
- Hepatic disease
- Ischaemic cerebrovascular disease
- Renal disease
- Respiratory insufficiency
- Severe systemic hypertension
- Intolerance to drugs available to produce hypotension
RISKS OF HYPOTENSIVE ANAESTHESIA

In a retrospective review of nearly 30000 cases done by Little et al in 1954, controlled hypotensive anaesthesia was associated with a morbidity on 1 in 31 and mortality of 1 in 291. By far, the commonest causes of mortality was compromise of the circulation to vital structures such as the kidney, brain and the heart. Other causes of mortality included reactionary haemorrhage, high spinal anaesthesia, over heprainization following arteriotomy, arterial air embolism, pulmonary infarcts and pulmonary oedema.

The vast majority of cases in this study (> 90%) were performed with a systolic blood pressure less than 80 mmHg which would probably account for the high morbidity and mortality rates. Subsequent studies (Eckenhoff 1966, Madsen 1987, Yamada 1988) have shown that controlled hypotension is safe provided the limits discussed previously are adhered to.

EVIDENCE

Evidence for the Successful Implementation of Hypotensive Anaesthesia

The early studies conducted by Little et al in 1954 found unacceptable mortality and morbidity rates of 1 in 291 and 1 in 31 cases respectively. However in this retrospective review, the target systolic blood pressure in majority of the cases were below 80mmHg. In fact, in 17% of the cases the systolic blood pressure was 60mmHg or less. Since then, the target MAP was set at 50mmHg or higher for ASA 1 patients.

The studies done, based on this new target, reveal advantages of controlled hypotension on both the amount of blood lost and hence transfusion requirements and the quality of the surgical field. There were also no significant differences in morbidity and mortality of hypotensive and the normotensive groups. Again here, majority of the studies were on ASA 1 patients.

The early study done by Gale Thompson et al in 1978 (Anesthesiology;48;91-96;1978) showed that hypotensive anaesthesia done on patients scheduled for total hip arthroplasty had significantly lower intraoperative blood loss and operating times compared to normotensive group (Fig 8). There were no significant differences in mortality and morbidity in terms of cerebral,
cardiovascular, renal and hepatic function between the hypotensive and normotensive groups.

Figure 8 ANESTHESIOLOGY;48;91-96;1978

A study done by Martin et al in 1989 (Anesth Analg 1989;69;379-383) on ASA 1 patients scheduled for maxillary and mandibular osteotomies showed a significant reduction in blood loss and transfusion requirements (Fig 9) and improvement in the quality of the surgical field (Fig 10) in the hypotensive group compared to the normotensive group. There was no significant differences in renal function post operatively between the two groups. The effects on cardiovascular, cerebral and hepatic function was not assessed post operatively.

Figure 9 ANEST ANALG;1989;69;379-383

<table>
<thead>
<tr>
<th></th>
<th>Control (N = 27)</th>
<th>Hypotension (N = 25)</th>
<th>P value</th>
</tr>
</thead>
<tbody>
<tr>
<td>Measured Blood Loss (mL)</td>
<td>755.3 ± 334.6</td>
<td>454.0 ± 211.3</td>
<td>&lt;0.001&lt;sup&gt;a&lt;/sup&gt;</td>
</tr>
<tr>
<td>(95% CI mean difference)</td>
<td></td>
<td>(144.0 - 458.6)</td>
<td></td>
</tr>
<tr>
<td>Blood loss &gt; ABL (no. of patients)</td>
<td>12</td>
<td>3</td>
<td>&lt;0.02&lt;sup&gt;b&lt;/sup&gt;</td>
</tr>
</tbody>
</table>
The quality of the surgical field was assessed using the surgeons’ scale for the quality of surgical field as follows:

0 - no bleeding, virtually bloodless field
1 - bleeding so mild it was not even a surgical nuisance
2 - moderate bleeding, a nuisance but without interference with accurate dissection
3 - moderate bleeding that moderately compromised surgical dissection
4 - bleeding, heavy but controllable, that significantly interfered with dissection
5 - massive, uncontrollable bleeding
TECHNIQUES

Techniques of Hypotensive Anaesthesia
The key equation in the provision of hypotensive anaesthesia is:

MAP = CARDIAC OUTPUT X SYSTEMIC VASCULAR RESISTANCE

Hence MAP can be manipulated by reducing either SVR or Cardiac output or both. Inducing hypotension purely by a reduction in cardiac output is not ideal because the maintenance of tissue blood flow is essential.

SVR can be reduced by peripheral vasodilation (of the resistance vessels) whilst cardiac output can be reduced by lowering venous return, heart rate, myocardial contractility or a combination of these.

METHODS TO REDUCE CARDIAC OUTPUT

1. Reduction in blood volume by arteriotomy
   This technique was initially described by Gardner in 1946 and involves removal of 500 mls aliquots of blood from the radial artery cannula until the systolic blood pressure was reduced to 80mmHg. The problem with this technique is obvious- the acute blood loss reduced oxygen delivery to the tissues because of the compensatory vasoconstriction and reduced haemoglobin levels. Metabolic acidosis developed rapidly. Hence, it is not surprising that this technique is no longer used.

2. Dilate the capacitance vessels using nitroglycerine to reduce preload.

3. Decrease in cardiac contractility using inhalational agents or Beta blockers.

4. Decrease in heart rate using inhalational agents or Beta blockers.

METHODS TO REDUCE PERIPHERAL VASCULAR RESISTANCE


2. Relaxation of vascular smooth muscle eg. Direct acting vasodilators (nitroprusside), calcium channel blockers, inhalational agents, purines (adenosine), prostaglandin E1.
MECHANICAL MANOEUVERS TO POTENTIATE THE ACTION OF HYPOTENSIVE AGENTS

1. Positioning

Position of the patient is critical to ensure success of the controlled hypotensive technique. Elevation of the site of operation allows easy venous drainage from the site of surgery. This is critical to ensure a bloodless field.

Remember that as a result of gravitational effects, the blood pressure increases or decreases as the vertical distance from the heart changes. This change in blood pressure is at a rate of 0.77mmHg per cm change in vertical height from the heart. This change in blood pressure must be factored in to ensure adequate blood flows to vital organs.

A hypotensive technique reduces the peripheral circulation. This is especially important in areas overlying weight-bearing and bony prominences. Hence, additional supportive pads should be placed beneath the patient with special attention paid to the occiput, scapulae, sacrum, elbows and heels. Also, pressure must be kept off the orbits (especially in the prone position) to avoid compromising retinal blood flow.

2. Positive airway pressure

An attractive adjunct to hypotensive anaesthesia is the use of positive pressure ventilation with high tidal volumes, prolonged inspiratory times and raising positive end expiratory pressure. This would certainly reduce venous return, thereby assisting with controlled hypotensive anaesthesia.

However, this increase in tidal ventilation would most likely also increase respiratory dead space in hypotensive anaesthesia and the increased intrathoracic pressure will decrease cerebral venous outflow thereby increasing cerebral blood volume and intracranial pressure. Hence, although attractive, respiratory manipulations to minimize venous return are usually not employed during hypotensive anaesthesia.
ANAESTHETIC MANAGEMENT

PREOPERATIVE MANAGEMENT

As a prerequisite, the anaethetist conducting the hypotensive anaesthesia must have a thorough knowledge of the technique.

Appropriate patient evaluation and selection is important.

Studies show that a minimum Hb of 10g/dL is safe.

Arterial blood gas sampling provides a baseline for intraoperative and post operative measurements.

Various premedication including anxiolytics, analgesics, alpha blockers, beta blockers and anti-hypertensives can assist with the induction of hypotension during anaesthesia.

INTRAOPERATIVE MANAGEMENT

A stress free induction is achieved by obtunding the intubation response. This would set the stage for a smooth hypotensive technique.

If using nasal intubation, ensure that the tube does not exert undue pressure on the tip of the nose.

If an intravenous hypotensive drug is to be used, a second intravenous line is necessary.

Care must be taken to protect the pressure points.

Meticulous **monitoring** is essential for patient safety during hypotensive anesthesia:

- Invasive blood pressure monitoring is recommended as it provides beat to beat monitoring of the blood pressure and it also permits sampling for arterial blood gases and Hb.
- ECG: monitoring of especially the V5 lead with ST segment analysis helps to detect cardiac ischaemia.
- Oxygen saturation must be monitored because of the risk of hypoxaemia due to the mismatch of ventilation and perfusion.
• End tidal CO2: prevention of hypercarbia and hypocapnia are essential. Remember that the relationship between EtCO2 and PaCO2 changes with hypotension, so that arterial blood gas determination should be carried out intermittently to make sure that the PaCO2 is within the desired range.
• Temperature: core temperature monitoring is important because body heat dissipates very quickly from dilated vessels. Hypothermia may decrease the effectiveness of vasodilators and increase the dose requirements if compensatory vasoconstriction occurs.
• Blood loss: the physiological response to blood loss may be lost during hypotensive anaesthesia. Therefore, blood loss should be carefully estimated using swab weighing and measuring blood volume in suction bottles.

Proper fluid therapy is essential during hypotensive anaesthesia. The aim of induced hypotension is to lower MAP while maintaining adequate perfusion to all vital organs. Thus, preoperative fluid status must be assessed and corrected. At the same time maintenance volumes need to be infused. Blood loss must be replaced with an equal amount of colloid or three to four times the amount of crystalloid. If the blood loss exceeds a predetermined level (eg. 20-25% of the patient’s total blood volume), a blood transfusion is warranted.

Induction of hypotension should only begin at the time that it is required. 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 using an infusion device. Hypotension should only be carried out 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.

POST OPERATIVE MANAGEMENT

Adequate post operative care with resuscitation facilities is necessary. Post operatively, attention should be given to airway maintenance, oxygenation, analgesia, monitoring, positioning, reactionary haemorrhage and fluid balance. Rebound hypertension, especially with inadequate analgesia is a concern during this period.
CONCLUSION

Controlled hypotensive anaesthesia has the advantage of minimising blood loss during surgery thereby reducing blood transfusion requirements.

In addition, an improved surgical field results thereby improving surgical technique and dissection and reducing the need for electrocauterization.

This potentially could reduce post operative pain and sepsis.

It is also a safe technique provided appropriate patient evaluation and selection, proper positioning and monitoring and adequate fluid therapy is adhered to.
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