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Anaesthetic Induction in Hypovolaemic Trauma Patients: Balancing Haemodynamic Stability and Urgency

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Abstract

Anaesthetic management of hypovolaemic adult trauma patients is challenging due to the need for urgent surgical intervention in the context of profound physiological instability. Standard induction agents may precipitate cardiovascular collapse, and intravenous access is often difficult to establish. In this narrative review, we synthesise current clinical guidelines, expert opinion, and peer-reviewed literature. Sources were identified through targeted searches of PubMed, EMBASE, and Cochrane databases from 2000 to 2025, using relevant trauma and anaesthetic terms.

In this review, we highlight that vascular access planning is critical, with peripheral and intraosseous routes often suitable initially. Ketamine and etomidate offer the most favourable haemodynamic profiles in this context, while agents such as propofol and high-dose opioids may precipitate cardiovascular collapse. Haemodynamic optimisation involves early blood product administration and minimal crystalloid use. In patients with traumatic brain injury, special considerations include the avoidance of hypotension, minimisation of positive end-expiratory pressure, and targeted ventilation and blood pressure goals.

A structured, tailored approach to induction may reduce the risk of peri-induction hypotension and improve outcomes. Core principles include agent selection, early resuscitation, airway planning, and vascular access strategy. Further research is required to standardise induction protocols and refine point-of-care resuscitation strategies in this high-risk population.

Key words: haemorrhagic shock, anaesthesia, general, resuscitation, infusions, intravenous, wounds and injuries

INTRODUCTION

Trauma remains a leading cause of mortality in individuals under 50 worldwide, placing major demands on emergency and anaesthetic services.[1] Anaesthetists are increasingly central to trauma care delivery, both in-hospital and in prehospital environments. Optimising anaesthetic management during the early phases of trauma care is critical to improving survival and outcomes.[2,3]

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Recent international consensus has shifted focus towards prioritising haemodynamic stabilisation before non-critical airway control in bleeding trauma patients.[4] However, induction practices vary widely, and evidence-based trauma-specific guidelines remain limited.[2,3] In this review, we summarise the current evidence on anaesthetic induction in adult trauma patients with hypovolaemia.

This narrative review is based on a synthesis of clinical guidelines, expert consensus, and peer-reviewed literature relevant to anaesthetic induction in adult trauma patients with hypovolaemia. A targeted literature search of PubMed, EMBASE, and Cochrane databases was conducted for articles published between 2000 and 2025 using keywords such as 'trauma', 'anaesthetic induction', 'hypovolaemia', 'haemorrhage', 'traumatic brain injury', 'permissive hypotension', and 'resuscitation'. Further sources were identified through reference tracking and manual review of recent trauma guidelines issued by major anaesthetic and surgical societies.

INITIAL ASSESSMENT

Anaesthetic management of hypovolaemic adult trauma patients is complex. Early care is guided by the principles

of damage control resuscitation and the timely initiation of lifesaving or damage control surgical interventions. Collectively, these strategies constitute a coordinated approach designed to enhance survival in severely injured patients by prioritising restoration of physiological stability before definitive anatomical reconstruction.[5,6]

On presentation, major trauma patients frequently exhibit signs of shock (tachycardia, hypotension, peripheral shutdown, altered mental status, and high lactate), reflecting dependence on compensatory vasoconstriction and sympathetic drive.[7] These physiological disturbances significantly reduce tolerance to standard induction agents. Assessment of injury mechanism, anticipated haemorrhage source, and degree of physiological derangement should occur immediately to guide resuscitation and induction planning.

Point-of-care diagnostics play a central role. Focused Assessment with Sonography for Trauma (FAST), lactate and base deficit measurement, and viscoelastic assays, where available, facilitate rapid identification of hypovolaemia and coagulopathy, enhancing early decision-making.[8,9]

Patients with confirmed or suspected traumatic brain injury (TBI) require a modified anaesthetic approach. Neurological assessment, including Glasgow Coma Scale and pupillary evaluation, should form part of the initial appraisal to identify those at heightened risk of secondary brain injury.[10]

OPTIMISATION AND RESUSCITATION

Vascular Access Strategies

Establishing vascular access is often the first critical step. Peripheral access using large-bore (16-18 G) cannulas is generally adequate initially.[2] If intravenous access is delayed, intraosseous access offers rapid temporary entry and can be lifesaving in unstable patients (see Table 1).[2]

Central venous access is rarely essential before induction and should not delay surgery.[2] When required, due to inaccessible peripheral sites or severe vasoconstriction, it must be performed by experienced clinicians, as the risk of inadvertent arterial puncture is high in patients with severe hypovolaemia.[7]

Choice of site depends on accessibility and speed of insertion. Internal jugular and subclavian veins allow high flow and are generally preferred.[11,12] Femoral lines can be challenging or relatively contraindicated in patients with large body habitus and in those undergoing surgery requiring femoral artery access or aortic cross clamping.

While subclavian lines carry a higher risk of pneumothorax than other central lines, some trauma patients may present with decompressive thoracostomies or intercostal drains, alleviating the risk of pneumothorax. Subclavian lines may also be inserted relatively quickly and easily under the landmark technique, by experienced operators. However, as this technique is becoming less popular due to risks of pneumothorax and because it is a poorly compressible bleeding site, many anaesthetists feel less confident with this technique.[11]

The choice of central line type in hypovolaemic trauma patients should prioritise ease of insertion and flow capacity, as venous access is often time critical. Preloaded dilator systems (e.g., trauma introducer sheaths) reduce insertion steps and enable faster placement. Short, wide-bore introducers (8.5-9 Fr) provide superior flow rates (>800 mL/min with rapid infusers) compared with standard multilumen lines.[13] However, user experience and familiarity are important, especially in stressful, time-critical circumstances.[14] Ultrasound guidance improves success and reduces complications and is recommended whenever available.[15,16]

Arterial Access

Arterial access is frequently required in major trauma surgery, providing continuous blood pressure measurement during severe hypotension and facilitating arterial blood gas analysis. Acidaemia should be recognised early, and lactate provides a useful surrogate marker.[2,17] However, arterial line placement should not delay lifesaving surgery. Securing arterial access can be technically challenging in agitated or profoundly hypotensive patients, potentially prolonging insertion at times when rapid progression to surgery is critical.[2] When arterial access is not essential and would delay surgery, postinduction arterial line insertion is a valid option and may allow for surgery to commence in the interim.[7]

Haemodynamic Management

Despite careful agent selection, peri-induction hypotension remains common.[18] A recent consensus statement from the World Society of Emergency Surgery emphasises the importance of achieving early haemodynamic stabilisation before induction in bleeding trauma

Table 1 – Comparison of vascular access options in hypovolaemic trauma patients

Access Type	Insertion Site	Advantages	Disadvantages	Use in Hypovolaemia
Peripheral IV	Forearm, antecubital, foot	Quick, easy to insert	Poor flow with vasoconstriction	Often first-line if accessible
Intraosseous	Tibia, humerus, sternum	Rapid access, useful in emergencies	Painful, temporary access	Used in unstable patients with no IV access
Internal jugular (central)	Neck (IJV)	Straight course, ultrasound guided	Risk of carotid puncture, pneumothorax	Preferred for large bore central access
Subclavian (central)	Infraclavicular	USS or landmark-guided, good flow	Risk of pneumothorax, bleeding	Useful when chest already decompressed
Femoral (central)	Groin	Compressible site, ultrasound guided	Deep in obese patients, infection risk	Used when upper body inaccessible

IJV, internal jugular vein; IV, intravenous; USS, ultrasound scan

patients, as premature airway intervention may precipitate cardiovascular collapse.[4] However, airway management must still take priority when airway obstruction, hypoxaemia, hypoventilation, or a markedly reduced level of consciousness is observed, and oxygen supplementation should be provided throughout resuscitation (see the Figure 1).[4]

Hypovolaemia is the primary driver of haemodynamic instability; therefore, damage control resuscitation should focus on blood product replacement.[7] Crystalloids and colloids should be minimised, as their use has been associated with increased mortality in hypovolaemic trauma patients.[7] Administration of large-volume crystalloids exacerbates the lethal triad of hypothermia, acidosis, and coagulopathy. These fluids do not treat ongoing haemorrhage or support coagulation; instead, they dilute clotting factors, contribute to tissue oedema, and may cause end-organ dysfunction when given in excessive volumes.[2,19,20] Commencing blood product transfusion before induction is often warranted due to the risk of postinduction haemodynamic instability.[18]

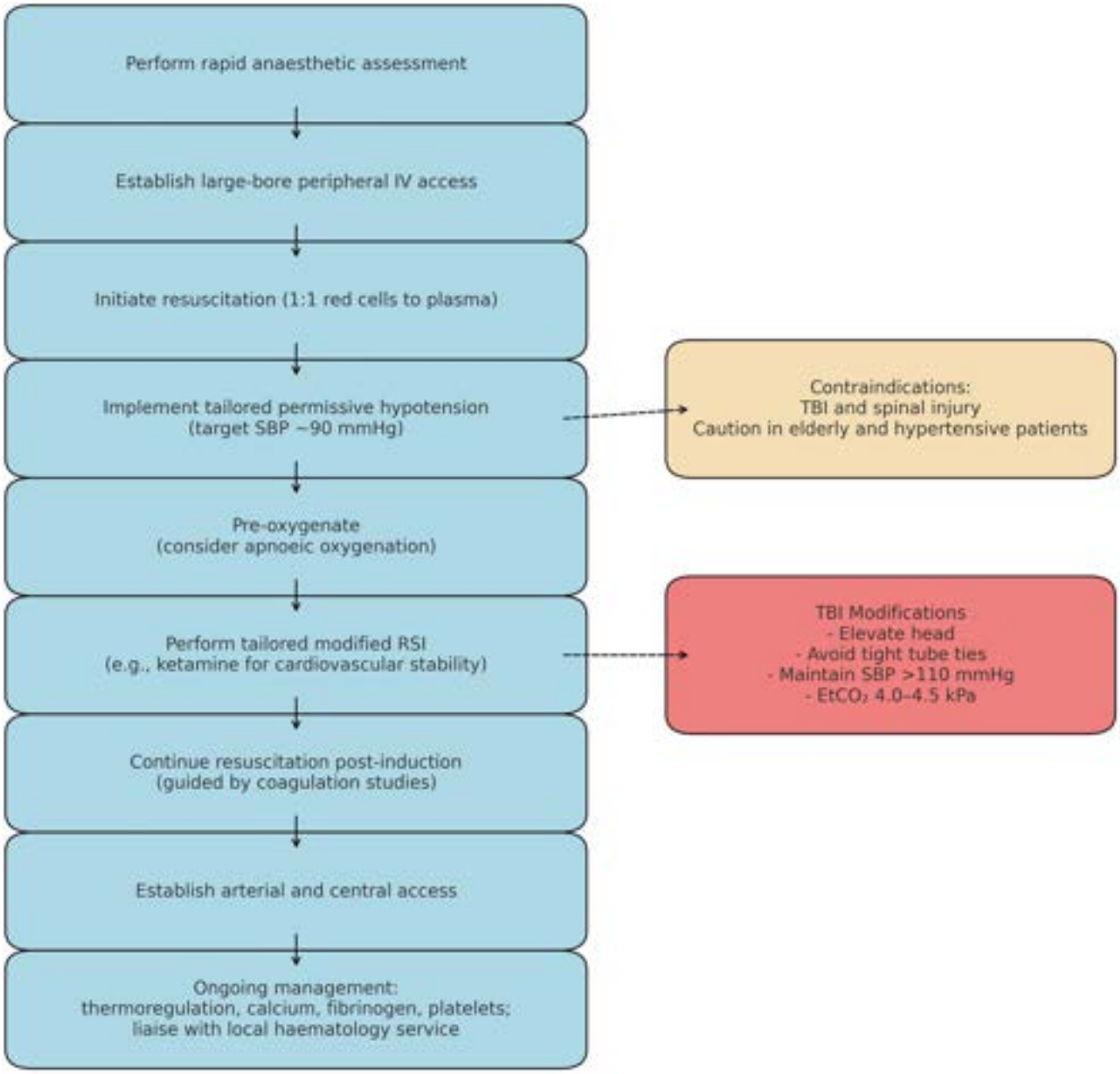

Figure 1 – Anaesthetic induction flowchart. Flowchart outlining a structured approach to anaesthetic induction in adult trauma patients with hypovolaemia. This algorithm highlights key priorities including rapid assessment, large-bore venous access, balanced transfusion, and use of cardiovascularly stable induction agents (e.g., ketamine). Tailored permissive hypotension (target arterial systolic blood pressure ~90 mmHg) is recommended, with contraindications shown in the adjacent panel. Modifications for patients with TBI are shown separately and include specific blood pressure and ventilation targets. Postinduction priorities include ongoing resuscitation guided by coagulation studies. Abbreviations: TBI, traumatic brain injury; SBP, systolic blood pressure; $EtCO_2$, end-tidal carbon dioxide

For major haemorrhage volume resuscitation, a 1:1 ratio of red blood cells to plasma should be targeted, with 1 pooled unit of platelets typically given for every 6 units of red blood cells and plasma.[4,14] Blood product replacement should be guided by clinical status and ongoing bleeding. Traditional markers such as arterial blood pressure are not reliable indicators of hypovolaemic shock, particularly in the awake patient prior to induction. Blood markers such as haemoglobin are similarly unreliable in the acute setting of haemorrhage and are often falsely reassuring in trauma patients.[8] Clinical signs such as tachycardia and bradycardia, agitation, and drowsiness may all be signs of significant haemorrhagic shock and impending decompensation.[7]

During massive blood volume transfusion, assigning a dedicated blood monitor is beneficial.[21] This individual should be tasked with monitoring and documenting blood product administration, liaising with blood bank, and advising on the immediate availability of blood products.[21]

Cell salvage or autotransfusion is an effective adjunct for red blood cell replacement during operative management of haemorrhage.[22] When used, the blood monitor should record autotransfused volumes in addition to exogenous product administration. If cell salvage is anticipated, theatre teams should be notified early to allow timely setup prior to incision.[23]

Local hospital blood bank and haematology service should be able to advise on transfusion ratios and platelet and coagulation factor replacement. As noted, it is generally advised for the patient to receive 1 pooled unit of platelets for every 6 units of red blood cells and plasma. Two to 4 grams of fibrinogen or cryoprecipitate should also be given for every 6 units of red cell concentrate and plasma.[14] Fibrinogen and platelet administration may be guided based on serum platelet count and Clauss fibrinogen assays,[22] although this can be an inappropriately slow method. Authors of clinical trials and observational studies have supported the use of viscoelastic testing such as rotational thromboelastometry or thromboelastography over conventional lab-based clotting tests to guide plasma, platelet, and fibrinogen administration. Not only are these methods faster than conventional lab-based tests, but they have also been shown to improve blood product use and survival.[22]

Tranexamic acid should be administered in the prehospital setting whenever possible, as early treatment has been shown to improve outcomes in bleeding trauma patients.[22] A total dose of up to 2 grams is recommended: the first gram should be given as soon as possible after injury, and the administration of the second gram should be commenced within 3 hours of injury.[6,22]

Another essential part of haemorrhage management is calcium replacement. Calcium should be maintained above 1.1 mmol/L.[24] In addition to its critical role in the coagulation cascade, calcium is also a positive inotrope and can be useful in the management of postinduction hypotension. Calcium replacement should be guided by regular blood gases, and overcorrection (>1.3 mmol/L) should be avoided, as this is also associated with increased mortality.[24]

Permissive hypotension involves aiming at arterial systolic blood pressures of 80-90 mmHg until definitive haemorrhage control is achieved.[22] However, recent advancements in prehospital care and damage control surgery along with emerging evidence are leading to a shift away from this as a general strategy and towards a more individualised approach.[2] While permissive hypotension continues to be endorsed in European trauma guidelines for non-TBI patients (target systolic blood pressure = 80-90 mmHg) and associated with reduced mortality and transfusion requirements, its blanket use is contested in elderly, hypertensive, TBI, and spinal injury cohorts.[25,26] Recent guidance has called for a tailored approach, adjusting resuscitation targets based on individual patient factors and urgency of haemorrhage control.[2]

Vasopressors and inotropes are not recommended as first-line treatment for hypotension in haemorrhagic shock. Authors of both observational studies and systematic reviews have associated early vasopressor use with worse outcomes.[27,28] If adequate arterial blood pressures cannot be achieved with blood product resuscitation, vasopressors and inotropes should only be considered second line for non-volume responders or in patients with suspected vasoplegia secondary to another cause, e.g., spinal shock.[26]

ANAESTHETIC MANAGEMENT

Induction Drugs

Ketamine is widely considered one of the most useful induction agents in hypovolaemic trauma patients. As an N-methyl-D-aspartate (NMDA) receptor antagonist, it causes rapid onset dissociative anaesthesia and analgesia. The cardiac stimulant effect may offset some of the sympathetic reduction encountered with other analgesics such as fentanyl.[29] Ketamine has a clinical half-life of 10-15 minutes due to the initial distribution. This permits slow introduction of maintenance anaesthetic agents during a period of significant haemodynamic instability (see Table 2).[29] However, in severely hypovolaemic and catecholamine-depleted patients, a dose reduction is necessary, as ketamine may exert direct negative inotropic effects in such states.[30]

Etomidate is an alternative cardiovascular stable induction agent that may be useful in hypovolaemic trauma patients. It has a very favourable profile in haemorrhagic shock with preservation of sympathetic tone and minimal cardiovascular depression.[3] Concerns remain regarding adrenal suppression; corticosteroid supplementation may be considered postoperatively. Despite this, etomidate remains a commonly used drug in major trauma patients.[3] Both ketamine and etomidate are considered safe induction agents in patients with TBI.[30]

Propofol remains the most widely used intravenous induction agent in anaesthesia but is generally unsuitable for hypovolaemic trauma patients. Propofol has potent vasodilatory and negative inotropic effects which can precipitate profound hypotension in this population, and therefore, it is recommended to avoid propofol for induction of unstable trauma patients.[3]

Fentanyl is often used as an adjunct to other agents during induction of general anaesthesia to reduce the sympathetic response to laryngoscopy. However, in unstable hypovolaemic patients, the reduction in sympathetic tone caused by fentanyl may lead to significant worsening of haemodynamic instability. Fentanyl should be titrated carefully or avoided completely in hypovolaemic trauma patients. Benzodiazepines such as midazolam should similarly be

Table 2 – Comparison of commonly used induction agents in hypovolaemic trauma, with key pharmacodynamic effects and considerations

Induction Drug	Mechanism	Haemodynamic Effects	Use in Hypovolaemia	Notes
Propofol	GABA-A agonist	↓ SVR, ↓ myocardial contractility	Generally avoided	Profound vasodilation and hypotension
Fentanyl	μ-opioid receptor agonist	↓ Sympathetic tone	Use cautiously or avoid	May blunt sympathetic compensation and cause collapse
Ketamine	NMDA antagonist	↑ HR, ↑ blood pressure	Preferred agent	Preserves airway reflexes, supportive cardiovascularly
Etomidate	GABA-A agonist	Neutral haemodynamic profile	Acceptable	Adrenal suppression risk

GABA, gamma-aminobutyric acid; HR, heart rate; NMDA, N-methyl-D-aspartate; SVR, systemic vascular resistance; ↓, decrease; ↑, increase

minimised or avoided in this cohort due to their negative cardiovascular depressant effects.[31]

As rapid sequence induction necessitates fast onset muscle relaxation, suxamethonium and rocuronium are neuromuscular blocking drugs of choice.[32] Both provide rapid onset of action; however, rocuronium will offer longer duration of action. Suxamethonium is contraindicated in hyperkalaemia and certain neuromuscular diseases.[32]

Awareness Risk

Hypovolaemic trauma patients are at increased risk of inadvertent awareness (recall of events experienced whilst under general anaesthesia) during anaesthetic induction and early maintenance. This cohort presents with several high-risk features for intraoperative awareness, including emergency surgery, haemodynamic instability, high American Society of Anesthesiologists (ASA) physical status, and critical illness.[33]

To preserve cardiovascular stability, anaesthetists frequently reduce or omit traditional doses of intravenous induction agents, particularly in the presence of hypovolaemia.[2] As discussed above, agents such as propofol and high-dose opioids are often contraindicated or given in smaller doses in this setting due to potent vasodilatory and myocardial depressant effects. Even with the use of ketamine or etomidate, reduced dosing strategies and a delayed transition to full anaesthetic maintenance, often deferred until the patient is surgically haemorrhage controlled, can result in a period of inadequate depth of anaesthesia.[34]

Mitigation strategies include the judicious use of agents like ketamine, which provides both haemodynamic support and a dissociative state, careful titration of volatile or intravenous maintenance agents once stability is achieved, and heightened vigilance for signs of awareness. Clear communication with the patient during recovery and formal follow-up in cases of suspected awareness are essential for supporting psychological outcomes and maintaining trust in future care.[33]

Intubation Technique

All acute trauma patients should be assumed to be unfasted and at risk of aspiration on induction. Gastroparesis due to pain, hypoperfusion, and opioid analgesia is common after trauma and normal fasting periods are not applicable.

Evidence for the type of laryngoscope used for rapid sequence inductions indicates favourable outcomes with video-assisted laryngoscopy as first line.[35,36] However, consideration should be given to user experience, availability, and clinical judgement. If blood or contaminated airways are anticipated, this may obstruct vision in video-assisted laryngoscopy, and a direct laryngoscopy approach may be advantageous.

Tracheal tube choice depends on surgical requirements (e.g., single-lung ventilation, prone positioning), underscoring the need for clear communication with the surgical team.

Cervical spine immobilisation is sometimes required in patients with actual or potentially unstable c-spine injury. Additional support with a dedicated staff member for performing in-line stabilisation is warranted, as a cervical collar should not be in place during induction and intubation.[37]

Preoxygenation should be performed where possible due to the shortened apnoea to desaturation time.[38] Preoxygenation may be difficult in patients who are agitated or non-cooperative and ensuring optimal positioning and environmental control is important. Apnoeic oxygenation may be advantageous in this cohort as it may reduce the risk of peri-intubation hypoxaemia. This increasingly used intervention is simple and low risk; however, its application should not delay the initiation of surgery.[39]

Temperature Management

Hypothermia (temperature <36°C) is present in up to 12.5% of trauma patients on arrival to hospital.[2] Prolonged on-scene time in cold environments and other factors such as prehospital intubation, immobilisation, and injury severity all increase the risk of hypothermia on arrival to hospital.[2] Factors such as patient exposure in cold operating theatres, anaesthetic vasodilation, surgical exposure, and cold intravenous fluid and blood product administration will exacerbate hypothermia.[2] Hypothermia not only worsens coagulopathy and acidosis; it also compounds haemodynamic instability due to worsening cardiac contractility and precipitation of dysrhythmias. Specific risks associated with hypothermia include higher vasopressor requirements, increased transfusion requirements, prolonged ICU admission, and death.[40] Early use of warmed blankets, overbody and underbody forced air warmers, and fluid warmers can all help prevent the deleterious effects of hypothermia.[2]

SPECIAL CONSIDERATIONS

Traumatic Brain Injury

Patients with confirmed or suspected TBI will require a modified approach to anaesthetic induction. Initial management should focus

on reducing intracerebral pressure (ICP) and improving cerebral perfusion (see Table 3).[10]

Patients should be positioned with 30° head elevation as soon as possible.[41] Many major trauma patients will be in spinal immobilisation, but this should not preclude a bed tilt to aid venous drainage and reduce ICP when indicated. Regarding airway choice and ventilation strategies, avoidance of tight tracheal tube ties is important, as these may impede central venous drainage and exacerbate high ICP. Once intubated, patients should be ventilated to achieve an end-tidal carbon dioxide of 4-4.5 kPa.[10] High positive end expiratory pressures (PEEPs) should be avoided, as these factors will also increase ICP.[10]

As previously stated, permissive hypotension is contraindicated, and in most cases, an arterial systolic blood pressure of 110 mmHg should be targeted to maintain cerebral perfusion pressure.[25] Large volumes of crystalloid should be avoided as for any major haemorrhage, with a focus on blood product replacement. Rapid reversal of coagulopathy and oral anticoagulants or antiplatelets is especially important in TBI, as even small bleeds can lead to rapid pathological increases in ICP.[2] Hypertonic saline or mannitol may be indicated prior to or during induction to temporise life-threatening raises in ICP.[25]

Regional Anaesthesia

Regional anaesthetic techniques have a limited role in actively bleeding, unstable trauma patients. Although neuraxial blockade can reduce surgical stress and blood loss in elective settings, it is generally contraindicated in trauma due to the risks of cardiovascular collapse and spinal haematoma in the presence of hypovolaemia or coagulopathy. Peripheral nerve blocks may provide effective analgesia and are alternatives in selected, haemodynamically stable patients with isolated injuries, but they do not offer adequate anaesthesia or airway protection for major trauma. Major trauma guidelines recommend general anaesthesia (typically with ketamine or etomidate) as the standard approach for induction in haemorrhaging patients, with regional techniques reserved only for carefully selected stable cases.[42]

HUMAN FACTORS

Major trauma cases are inherently high-stress situations, characterised by time-critical decision-making, significant cognitive load, and the coordination of large multidisciplinary teams. These pressures increase the risk of communication breakdown, task fixation, and omission of critical safety steps during anaesthetic induction. Applying human factors principles, including clearly defined leadership, closed-loop communication, and structured preinduction briefings, has been shown to enhance team performance and improve patient outcomes.[43] Clear role allocation within the anaesthetic team (e.g., team lead, primary and secondary intubators, drug administrator, vascular access lead, blood transfusion monitor) further supports task management and situational awareness.[43,44] Regular trauma team training, particularly through simulation, can reinforce non-technical skills, strengthen protocol adherence, and optimise performance in high-stakes environments.

Anaesthetic induction in hypovolaemic trauma patients thus, presents a uniquely high-risk combination of time-critical decision-making, haemodynamic instability, and limited physiological reserve. In states of profound shock, the choice of induction agents, timing, and resuscitation strategy can have a significant impact on outcome. Despite advances in trauma systems and early transfusion protocols, the induction phase remains a high-risk period with persistently high rates of peri-induction hypotension.

Ketamine and etomidate continue to be the most appropriate induction agents in haemorrhagic shock. Importantly, the induction process must not be isolated from resuscitation: preinduction blood product administration and minimisation of crystalloids is essential. The role of permissive hypotension is increasingly nuanced, and a tailored strategy based on injury pattern, particularly in cases of TBI, is imperative.

Airway management requires careful consideration. Preoxygenation, planning for difficult airways, and effective role allocation within the team can mitigate risk. Human factors, including leadership, closed-loop communication, and simulation training, can lead to improved outcomes.

Despite these principles, anaesthesia-specific, trauma-focused guidance on induction strategy in major haemorrhage is lacking. Future researchers should prioritise the development of standardised induction pathways and pragmatic, multicentre studies evaluating optimal agent selection, dosing, and timing in haemorrhagic shock.

Table 3 – Key differences in anaesthetic induction strategy in major trauma patients with and without TBI

Consideration	Non-TBI Trauma	TBI Trauma
Arterial blood pressure target	Tailored permissive hypotension	SBP ≥ 110 mmHg (avoid hypotension)
Vasopressors	Not first line for hypotension	In combination with blood products, especially with spinal shock
Ventilation target	Normal CO_2 (end tidal CO_2 4.5-5.0 kPa)	$EtCO_2$ 4.0-4.5 kPa; avoid hypercapnia
Head positioning	Neutral	30° head-up tilt if spinally safe, or whole bed tilt
Volume resuscitation	Balanced 1:1 transfusion	Early resuscitation to maintain CPP
PEEP	Use with caution as required	Minimise PEEP to avoid ↑ ICP
Tracheal tube tie	Standard	Avoid or avoid tight securing that obstructs venous drainage

CPP, cerebral perfusion pressure; $EtCO_2$, end-tidal carbon dioxide; ICP, intracranial pressure; PEEP, positive end expiratory pressure; SBP, systolic blood pressure; TBI, traumatic brain injury

Technological advancements, including real-time coagulation monitoring and continuous haemodynamic assessment, may enable more individualised, physiology-guided induction strategies. Interest in whole-blood resuscitation and its implications for anaesthetic management during the early phase of trauma care is also increasing. Enhancing human-factors performance through simulation, structured briefings, and cognitive aids represents another key area for improving safety during high-risk inductions. Together, these developments have the potential to significantly refine anaesthetic care for severely injured patients.

SUMMARY

Anaesthetic induction in hypovolaemic trauma patients demands meticulous timing, early haemodynamic optimisation, and the use of cardiovascularly stable agents. Prioritising balanced blood product resuscitation while minimising crystalloids is essential to reduce peri-induction deterioration. A structured, team-based approach to airway management, vascular access, and physiological targets improves safety and outcomes in this high-risk population.

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