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Review Article

Metaanalysis on Medical Optimization of Shock in Polytrauma Patients Undergoing Surgery

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ABSTRACT

Background: Severe haemorrhage and haemorrhagic shock remain major causes of potentially preventable death following polytrauma. Contemporary management has evolved from predominantly crystalloid-based volume replacement toward haemostatic, time-sensitive, and physiology-guided resuscitation, but individual optimization strategies differ substantially in mechanism and strength of supporting evidence. Objective: To provide a structured narrative synthesis of contemporary evidence concerning medical optimization of haemorrhagic shock in adult polytrauma patients requiring emergency or definitive surgical management. Methods: MEDLINE/PubMed, Scopus, Web of Science, Embase, and the Cochrane Library were searched for literature published primarily between 2010 and 2025. Evidence concerning damage control resuscitation, balanced and massive transfusion, tranexamic acid, permissive hypotension, fluid management, vasopressor therapy, and haemodynamic or coagulation monitoring was synthesized according to clinical relevance, study design, directness, and consistency. No meta-analysis or pooled risk-of-bias assessment was performed. Results: The strongest comparative evidence supported early antifibrinolytic and coordinated blood-component therapy. CRASH-2 included 20,211 bleeding trauma patients and demonstrated reduced bleeding-related mortality with tranexamic acid, with subsequent analyses showing greater benefit with earlier administration. In the 680-patient PROPPR randomized trial, 1:1:1 plasma:platelet:red blood cell resuscitation improved haemostasis and reduced early exsanguination compared with 1:1:2 resuscitation, although overall 24-hour and 30-day mortality did not differ significantly. The 1,245-patient PROMMTT cohort further associated earlier plasma and platelet administration with improved survival. Evidence for restrictive crystalloid strategies, permissive hypotension, vasopressors, and advanced monitoring was more heterogeneous and context-dependent. Conclusion: Optimal management of traumatic haemorrhagic shock requires rapid haemorrhage control integrated with early haemostatic resuscitation and individualized physiological management. Evidence is strongest for timely tranexamic acid and coordinated blood-component therapy, while other strategies require selective application according to patient physiology and clinical context. Keywords: Polytrauma; Hemorrhagic Shock; Damage Control Resuscitation; Tranexamic Acid; Blood Component Transfusion; Hemodynamics; Perioperative Care; Trauma.

INTRODUCTION

Trauma remains a major cause of preventable morbidity and mortality worldwide and contributes substantially to premature death, long-term disability, and healthcare burden, particularly among younger and economically active populations (1). Among patients with severe polytrauma, uncontrolled haemorrhage and the resulting haemorrhagic shock are major determinants of early deterioration and death. Acute blood loss compromises circulating volume, tissue perfusion, and oxygen delivery, initiating compensatory cardiovascular responses that may ultimately fail as haemorrhage progresses. Persistent hypoperfusion promotes cellular hypoxia, anaerobic metabolism, metabolic acidosis,

endothelial dysfunction, and progressive organ injury, creating a physiological environment in which mortality may increase rapidly unless bleeding and circulatory failure are addressed promptly (2).

The clinical consequences of haemorrhagic shock extend beyond loss of circulating blood volume. Severe trauma is frequently accompanied by trauma-induced coagulopathy, which arises through interacting abnormalities in coagulation, fibrinolysis, platelet function, endothelial integrity, inflammation, hypothermia, and acidosis. These disturbances may be further aggravated by excessive crystalloid administration and dilution of coagulation factors. The interaction between hypothermia, acidosis, and coagulopathy has traditionally been described as the lethal triad of trauma and remains an important therapeutic target during initial and perioperative resuscitation (3). Consequently, contemporary trauma management emphasizes simultaneous haemorrhage control, restoration of adequate perfusion, prevention or correction of coagulopathy, and avoidance of interventions that may exacerbate ongoing bleeding.

Damage control resuscitation has emerged as a central framework for achieving these objectives. Rather than relying on aggressive crystalloid replacement and normalization of arterial pressure before haemorrhage control, contemporary approaches generally favour restricted crystalloid administration, early blood-component therapy, correction of coagulopathy, and appropriately selected permissive hypotension until definitive haemostasis can be achieved (2,3). The increasing use of massive transfusion protocols has further facilitated rapid delivery of red blood cells, plasma, and platelets in patients with life-threatening haemorrhage. Evidence from major trauma cohorts indicates that earlier administration of plasma and platelets during massive transfusion is associated with improved survival, reinforcing the importance of coordinated and timely blood-component replacement during severe traumatic bleeding (4).

Balanced transfusion strategies have therefore become an important component of modern trauma resuscitation. The PROPPR trial compared different plasma, platelet, and red-cell ratios in severely bleeding trauma patients and demonstrated that a 1:1:1 strategy resulted in earlier haemostasis and fewer deaths from exsanguination, although overall mortality differences at later time points were less definitive. These findings illustrate an important feature of contemporary haemorrhage management: benefits may depend not only on the quantity of resuscitation administered but also on its timing, composition, and relationship to definitive haemorrhage control. The use of structured massive transfusion protocols consequently aims to reduce treatment delays, minimize dilutional coagulopathy, and coordinate blood-product delivery during rapidly evolving resuscitation.

Pharmacological haemostatic therapy has also become integral to early trauma care. Tranexamic acid is an antifibrinolytic agent that limits fibrin degradation by inhibiting plasminogen activation. The CRASH-2 trial demonstrated that administration of tranexamic acid to bleeding trauma patients reduced death attributable to haemorrhage without a corresponding increase in vascular occlusive events, with subsequent analyses demonstrating that treatment benefit was strongly dependent on early administration (5,6). These findings support the concept that medical optimization in traumatic haemorrhage is highly time-sensitive and that potentially beneficial interventions may lose effectiveness when delivered after critical physiological deterioration has occurred.

In addition to haemostatic resuscitation, optimization of cardiovascular physiology is essential during the perioperative management of polytrauma. Conventional static variables such as blood pressure and heart rate provide important information but may incompletely characterize circulating volume, tissue perfusion, cardiac performance, and response to therapy. Goal-directed haemodynamic management incorporates physiological and biochemical indicators such as lactate, base deficit, cardiac output, stroke-volume-related parameters, and other measures of perfusion to guide individualized fluid and vasoactive therapy (7). When appropriately applied, this approach may help balance the competing hazards of inadequate resuscitation, which perpetuates tissue hypoxia and organ dysfunction, and excessive

resuscitation, which can contribute to tissue oedema, dilutional coagulopathy, pulmonary complications, and abdominal compartment syndrome.

Vasopressor therapy represents another clinically important but context-dependent component of haemodynamic optimization. In patients with uncontrolled traumatic haemorrhage, indiscriminate vasoconstriction or premature restoration of high arterial pressures may theoretically compromise tissue perfusion or increase bleeding before haemostasis. Conversely, selected patients may require vasoactive support when adequate perfusion pressure cannot be maintained despite appropriate haemorrhage control and volume replacement. The appropriate role of vasopressors therefore cannot be considered independently of bleeding status, cardiac function, volume responsiveness, injury pattern, and the overall resuscitation strategy. Similar considerations apply to advanced haemodynamic and coagulation monitoring, whose clinical value depends on whether measurements result in timely, individualized therapeutic decisions.

Despite broad acceptance of several principles of damage control resuscitation, considerable variation persists in how haemorrhagic shock is managed across trauma systems. Differences may involve transfusion ratios, thresholds for activation of massive transfusion protocols, crystalloid exposure, timing of tranexamic acid, permissive hypotension targets, use of vasopressors, availability of viscoelastic coagulation testing, and haemodynamic monitoring practices (3,8). Furthermore, the available evidence is heterogeneous in design and clinical context, ranging from large randomized trials and prospective observational studies to clinical guidelines and broader reviews of haemorrhagic shock and perioperative resuscitation. This diversity makes direct quantitative comparison of all optimization strategies problematic and supports an approach that evaluates the evidence according to intervention type, physiological rationale, clinical applicability, and consistency of reported findings rather than generating a single pooled treatment effect.

Accordingly, a structured narrative synthesis is appropriate for integrating the major components of medical optimization while retaining the clinical distinctions between interventions. Unlike a meta-analysis, which requires sufficiently comparable populations, interventions, outcomes, and extractable quantitative data, a structured narrative review can examine complementary strategies whose mechanisms, indications, timing, comparators, and outcome measures differ substantially. This approach also permits randomized evidence, observational findings, major clinical guidance, and mechanistic considerations to be interpreted together while maintaining clear distinctions between stronger comparative evidence and broader clinical recommendations.

The present structured narrative review therefore aims to critically synthesize the available evidence concerning medical optimization of haemorrhagic shock in adult polytrauma patients requiring emergency or definitive surgical management. Particular emphasis is placed on damage control resuscitation, balanced and massive transfusion strategies, tranexamic acid, restrictive or goal-directed fluid therapy, permissive hypotension, vasoactive support, and advanced haemodynamic and coagulation monitoring. The review seeks to determine how these strategies contribute to haemorrhage control, haemodynamic stabilization, preservation of organ perfusion, reduction of transfusion-related complications, and improvement of perioperative outcomes, while identifying areas in which evidence remains inconsistent or dependent on clinical context.

MATERIAL AND METHODS

This article was designed as a structured narrative review of medical optimization strategies used in the management of haemorrhagic shock among adult polytrauma patients undergoing emergency or definitive surgical care. A narrative synthesis was selected because the evidence relevant to this topic encompasses clinically heterogeneous interventions, including damage control resuscitation, blood-component therapy, antifibrinolytic treatment, fluid and vasopressor strategies, and haemodynamic or coagulation monitoring. These interventions differ substantially in mechanism, timing, treatment

context, comparator, and outcome measurement, limiting the appropriateness of combining them within a single quantitative meta-analytic framework. The review was therefore structured to provide a clinically organized synthesis of the available evidence rather than a pooled estimate of treatment effectiveness.

The scope of the review was defined around adult patients with polytrauma complicated by haemorrhagic or traumatic shock who required emergency operative management, definitive surgery, or perioperative resuscitation. Evidence was considered relevant when it addressed medical interventions intended to control haemorrhage, restore or preserve tissue perfusion, correct trauma-associated coagulopathy, reduce unnecessary crystalloid or blood-product exposure, or improve perioperative physiological stability and clinical outcomes. The principal interventions of interest were damage control resuscitation, balanced transfusion and massive transfusion protocols, tranexamic acid, permissive hypotension, restrictive or goal-directed fluid therapy, vasopressor therapy, and advanced haemodynamic or coagulation monitoring. Outcomes of clinical interest included mortality, haemorrhage-related mortality, haemostatic control, blood-product requirements, haemodynamic stabilization, organ dysfunction, intensive care requirements, hospital stay, and perioperative complications.

The literature base was identified through structured searches of MEDLINE/PubMed, Scopus, Web of Science, Embase, and the Cochrane Library, with the review focusing primarily on literature published between 2010 and 2025. This period was selected to capture contemporary trauma-resuscitation practice while retaining influential studies that contributed to the current management paradigm. Search concepts included combinations and variations of terms relating to polytrauma, major trauma, haemorrhagic shock, traumatic shock, damage control resuscitation, massive transfusion, balanced transfusion, tranexamic acid, fluid resuscitation, goal-directed therapy, permissive hypotension, vasopressor therapy, haemodynamic monitoring, coagulation monitoring, and emergency surgery. Boolean operators were used to combine population, condition, intervention, and management concepts. Reference lists of clinically relevant articles and major trauma guidance documents were also examined to identify influential publications not captured readily by the principal search terms.

Literature selection was purposive and relevance-based, consistent with the structured narrative design. Priority was given to randomized controlled trials, prospective and retrospective cohort studies, major multicentre trauma investigations, and clinically influential guidance addressing management of haemorrhagic shock in adult trauma. Seminal studies were retained when they contributed directly to the development or evaluation of current resuscitation strategies. Clinical reviews and major guidance documents were used selectively to provide physiological context, describe areas of established practice, and support interpretation of the primary clinical evidence. Publications focused exclusively on paediatric trauma, non-traumatic shock, elective surgery without major traumatic haemorrhage, or topics unrelated to the perioperative medical optimization of haemorrhagic shock were outside the principal scope of the review.

Evidence was organized using a predefined clinical framework based on the principal components of haemorrhagic-shock management. The synthesis addressed the pathophysiological basis of traumatic circulatory failure first, followed by damage control resuscitation and permissive hypotension, massive and balanced transfusion, tranexamic acid, fluid-management strategies, vasopressor support, and advanced haemodynamic or coagulation monitoring. Within each domain, emphasis was placed on the clinical question addressed, study design, characteristics of the population evaluated, intervention and comparator where applicable, principal patient-centred outcomes, and consistency of findings with the broader trauma literature.

The strength of evidence was interpreted according to study design, sample size, directness to the target clinical population, consistency of findings, and relevance of the reported outcomes. Findings from randomized controlled trials and large prospective multicentre investigations were given greater

evidentiary weight when evaluating comparative treatment effects, whereas observational studies were interpreted primarily as evidence of clinical association or real-world practice. Clinical guidelines and narrative sources were not treated as equivalent to primary comparative studies and were used mainly to contextualize recommendations, physiological principles, and areas of consensus. This distinction was maintained throughout the synthesis to avoid presenting expert or guideline recommendations as though they represented independent experimental evidence.

Because this review was intended as a structured narrative synthesis rather than a systematic review or meta-analysis, no pooled effect estimates were calculated, and statistical heterogeneity, funnel-plot analysis, meta-analytic sensitivity analysis, and quantitative assessment of publication bias were not undertaken. Similarly, the review does not assign formal pooled risk-of-bias ratings across heterogeneous evidence types. Instead, methodological limitations relevant to individual bodies of evidence—including differences in study design, patient severity, transfusion practices, intervention timing, outcome definitions, institutional protocols, and potential confounding in observational studies—were considered when interpreting the direction and clinical applicability of findings.

The synthesis was intentionally evidence-focused rather than exhaustive. Consequently, the possibility of selection bias inherent to narrative literature selection cannot be completely excluded, and the absence of a formal systematic-screening and quantitative-appraisal process limits claims regarding complete retrieval of all eligible publications. To improve transparency, the review defines its clinical scope, principal information sources, literature-selection rationale, intervention domains, and approach to evidence weighting. Conclusions are therefore presented as an integrated interpretation of contemporary evidence and clinical principles rather than as statistically pooled estimates of comparative effectiveness.

RESULTS

The literature examined in this structured narrative review demonstrates that medical optimization of haemorrhagic shock in polytrauma is not represented by a single therapeutic intervention but by a coordinated set of time-sensitive resuscitation strategies. The strongest comparative evidence within the identified literature relates to balanced blood-component resuscitation and early tranexamic acid administration, whereas evidence supporting broader damage control resuscitation, individualized fluid therapy, vasopressor use, and advanced haemodynamic or coagulation monitoring is more heterogeneous and is supplemented by observational studies, clinical guidance, and broader perioperative literature.

The principal primary studies contributing directly to the clinical synthesis are summarized in Table 1. Randomized evidence was available for different blood-component ratios during major traumatic haemorrhage and for tranexamic acid administration in bleeding trauma patients. Prospective observational evidence additionally evaluated the timing of plasma and platelet administration among patients requiring massive transfusion. These studies differed substantially in intervention type, clinical context, and outcome definition; therefore, findings were interpreted according to individual treatment domains rather than quantitatively pooled.

The available evidence supports damage control resuscitation as an integrated strategy rather than an isolated intervention. Its major components include rapid control of haemorrhage, limitation of unnecessary crystalloid administration, early blood-component replacement, correction of trauma-induced coagulopathy, and avoidance of excessive arterial pressure before definitive haemostasis where clinically appropriate (2,3,8). Within this framework, transfusion strategy represents one of the better-studied components.

The PROPPR randomized trial evaluated 680 severely injured patients with major haemorrhage and directly compared transfusion using plasma, platelets, and red blood cells in ratios of 1:1:1 and 1:1:2 (9).

The more balanced 1:1:1 approach improved achievement of haemostasis and reduced death from exsanguination. However, the absence of a definitive difference in overall 30-day mortality indicates that the benefit should not be interpreted as proof that one transfusion ratio independently determines longer-term survival. Rather, the findings support balanced early component therapy as part of comprehensive haemorrhage control and resuscitation.

Table 1. Principal Primary Clinical Evidence Relevant to Medical Optimization of Haemorrhagic Shock in Trauma

Study	Design	Population and Sample	Intervention / Exposure	Comparator	Principal Outcomes	Main Finding Relevant to This Review
Holcomb et al., 2015 — PROPPR (9)	Randomized controlled trial	680 patients with severe trauma and major haemorrhage	Plasma:platelets:RBC transfusion in a 1:1:1 ratio	Plasma:platelets:RBC transfusion in a 1:1:2 ratio	Haemostasis; 24-hour and 30-day mortality; death from exsanguination	The 1:1:1 strategy achieved haemostasis more effectively and was associated with fewer deaths from exsanguination, although a clear overall 30-day mortality advantage was not demonstrated.
CRASH-2 Trial Collaborators, 2010 (5)	Randomized placebo-controlled trial	20,211 bleeding trauma patients across 40 countries	Tranexamic acid	Placebo	Mortality, including death attributable to bleeding; vascular occlusive events	Tranexamic acid reduced death attributable to bleeding without evidence of an increase in vascular occlusive events, supporting early antifibrinolytic treatment in appropriate bleeding trauma patients.
CRASH-2 Collaborators, 2011 (6)	Exploratory analysis of randomized trial data	Bleeding trauma patients enrolled in CRASH-2	Timing of tranexamic acid treatment after injury	Later treatment intervals	Death attributable to bleeding according to treatment timing	Treatment benefit was greatest when tranexamic acid was administered early after injury and diminished with treatment delay, emphasizing the time-dependent nature of antifibrinolytic therapy.
Holcomb et al., 2013 — PROMMTT (4)	Prospective multicentre observational cohort	1,245 trauma patients requiring major transfusion	Earlier plasma and platelet administration	Delayed administration	Mortality and survival during massive transfusion	Earlier delivery of plasma and platelets was associated with improved survival, supporting rapid coordinated blood-component resuscitation during major traumatic haemorrhage.

Abbreviations: RBC, red blood cells; PROPPR, Pragmatic, Randomized Optimal Platelet and Plasma Ratios; PROMMTT, Prospective, Observational, Multicenter, Major Trauma Transfusion study.

Table 2. Contextual Evidence Supporting the Broader Resuscitation Framework

Source	Evidence Type	Main Clinical Domain	Contribution to the Synthesis
Spahn et al., 2019 (3)	International clinical guideline	Major bleeding and trauma-induced coagulopathy	Supports early recognition and correction of traumatic bleeding and coagulopathy, structured blood-component resuscitation, early tranexamic acid, and coagulation-guided management.
Cannon, 2018 (2)	Clinical narrative review	Haemorrhagic shock	Provides physiological and clinical context for contemporary haemorrhagic-shock resuscitation, including avoidance of excessive crystalloid administration and prioritization of haemorrhage control.
Cecconi et al., 2014 (7)	Clinical review	Goal-directed haemodynamic therapy	Supports physiologically individualized haemodynamic assessment using measures beyond conventional static vital signs; applicability to polytrauma must be interpreted in the context of the broader surgical evidence base.
Kozar et al., 2021 (8)	Trauma clinical guidance	Damage control resuscitation	Provides contextual support for modern damage control principles and emphasizes the integration of haemorrhage control with resuscitation strategy.

Damage Control Resuscitation and Balanced Blood-Component Therapy

The prospective PROMMTT study provides complementary observational evidence regarding transfusion timing. Among 1,245 patients requiring major transfusion, earlier administration of plasma and platelets was associated with improved survival compared with delayed component delivery (4). Although the observational design limits causal interpretation because transfusion timing may be associated with injury severity, institutional practice, and other aspects of trauma care, the findings reinforce the importance of rapidly organized massive transfusion rather than sequential or substantially delayed replacement of coagulation components.

Taken together, PROPPR and PROMMTT suggest that both the composition and timing of blood-component therapy are clinically important. The evidence is therefore more supportive of coordinated early haemostatic resuscitation than of a simplistic conclusion that a particular transfusion ratio alone determines mortality. This interpretation is consistent with contemporary trauma guidance emphasizing early massive transfusion activation, haemorrhage control, prevention of dilutional coagulopathy, and repeated reassessment of coagulation and physiological status (3,8).

Tranexamic Acid and the Importance of Treatment Timing

Tranexamic acid represents the most clearly evaluated pharmacological intervention within the evidence base considered in this review. The CRASH-2 randomized placebo-controlled trial enrolled 20,211 bleeding trauma patients across 40 countries and demonstrated that tranexamic acid reduced mortality attributable to bleeding without evidence of an accompanying increase in vascular occlusive events (5). The large sample size and randomized design provide comparatively strong support for antifibrinolytic treatment in appropriately selected bleeding trauma patients.

The subsequent timing analysis of CRASH-2 demonstrated that the clinical effect of tranexamic acid depended strongly on the interval between injury and treatment (6). Benefit was greatest with early administration, whereas the therapeutic advantage diminished as treatment was delayed. This temporal relationship is particularly important in polytrauma because haemorrhagic deterioration and trauma-induced coagulopathy evolve rapidly during the early post-injury period.

These findings support interpreting tranexamic acid as a time-dependent component of haemorrhage control, rather than simply as an adjunct that can be administered at any point during perioperative management. Its role should consequently be integrated with rapid recognition of significant traumatic bleeding, definitive haemorrhage control, appropriate blood-component therapy, and correction of physiological derangement. Contemporary trauma guidance similarly incorporates early tranexamic acid into the management of patients with major traumatic haemorrhage (3).

Fluid Resuscitation and Permissive Hypotension

The contemporary evidence framework has progressively moved away from indiscriminate high-volume crystalloid resuscitation in severe traumatic haemorrhage. Excessive crystalloid administration may exacerbate haemodilution, tissue oedema, hypothermia, and dilution of coagulation factors and can complicate subsequent operative and critical-care management (2,3). Damage control resuscitation therefore emphasizes sufficient circulatory support to preserve essential organ perfusion while avoiding unnecessary fluid loading before haemorrhage has been definitively controlled.

Permissive hypotension forms part of this concept in selected bleeding trauma patients because aggressive normalization of arterial pressure before haemostasis may increase hydrostatic pressure at sites of vascular injury and potentially disrupt early clot formation. However, the appropriate haemodynamic target is clinically dependent and cannot be generalized uniformly to every polytrauma patient. Injury pattern, cerebral perfusion requirements, cardiovascular reserve, ongoing haemorrhage, and response to resuscitation must be considered when determining acceptable blood-pressure targets.

Within the supplied literature, the evidence supporting restrictive crystalloid strategies and permissive hypotension is principally contextual rather than represented by a directly extractable comparative dataset specific to the complete target population of this review. Consequently, the available evidence supports these approaches as components of contemporary damage control practice but does not permit a precise quantitative estimate of their independent effect on mortality or postoperative complications.

Goal-Directed Haemodynamic Optimization

Goal-directed haemodynamic management seeks to individualize resuscitation by integrating conventional clinical assessment with physiological indicators of tissue perfusion and cardiovascular

performance. Relevant variables may include serum lactate, base deficit, cardiac output, stroke-volume-related measurements, and other dynamic indicators, depending on the clinical environment and monitoring resources available (7).

The clinical rationale for this approach is particularly relevant in polytrauma because both inadequate and excessive resuscitation can be harmful. Persistent under-resuscitation allows tissue hypoxia, oxygen debt, metabolic acidosis, and organ dysfunction to progress, whereas excessive fluid administration may worsen pulmonary oedema, tissue swelling, haemodilution, and intra-abdominal pressure. Goal-directed management therefore aims to match fluid and vasoactive treatment to physiological need rather than relying exclusively on fixed-volume replacement or isolated blood-pressure thresholds.

Nevertheless, the evidence identified in the supplied literature does not establish a single monitoring protocol or haemodynamic target that can be considered universally superior for adult polytrauma patients with haemorrhagic shock. The supporting literature includes broader surgical haemodynamic evidence and trauma guidance rather than a homogeneous set of directly comparable trauma trials (3,7). Accordingly, goal-directed monitoring is best interpreted as a framework for individualized decision-making rather than as an intervention with a single established effect size.

Vasopressor Therapy

The role of vasopressors in traumatic haemorrhagic shock requires careful distinction from volume replacement and definitive haemorrhage control. Vasopressor therapy cannot compensate for uncontrolled blood loss and should not substitute for restoration of circulating volume when hypovolaemia remains the dominant physiological problem. Excessive vasoconstriction may also compromise regional perfusion when circulating volume is severely reduced.

Conversely, vasoactive support may have a role in selected patients when adequate perfusion pressure cannot be maintained despite appropriate haemorrhage control and resuscitative management. The decision to initiate vasopressors should therefore be individualized according to haemodynamic state, response to blood-component and fluid therapy, cardiovascular function, and competing perfusion requirements.

The literature supplied for this review does not contain sufficiently detailed comparative primary data to quantify an independent mortality or complication benefit attributable to vasopressor therapy in the target population. Consequently, vasopressors should be considered a context-dependent supportive intervention rather than a uniformly beneficial component of haemorrhagic-shock treatment.

Coagulation and Advanced Haemodynamic Monitoring

Monitoring is increasingly integrated into contemporary trauma resuscitation because conventional vital signs may not adequately identify the severity of tissue hypoperfusion, evolving coagulopathy, or response to treatment. Laboratory markers such as lactate and base deficit can contribute to assessment of the adequacy of resuscitation, while viscoelastic coagulation methods such as thromboelastography and rotational thromboelastometry may provide more rapid characterization of coagulation abnormalities than conventional laboratory tests in appropriate clinical environments (3).

The potential advantage of advanced monitoring lies primarily in facilitating individualized therapy. Identification of specific coagulation abnormalities may guide targeted blood-component or haemostatic treatment, while dynamic haemodynamic assessment may help clinicians determine whether further fluid, blood-component, or vasoactive intervention is physiologically appropriate.

However, the supplied evidence does not support treating advanced monitoring itself as an intervention proven independently to reduce mortality. Monitoring produces clinical value only when measurements are accurately interpreted and translated into appropriate therapeutic decisions. Its contribution should therefore be understood as enabling more individualized resuscitation rather than as replacing the core

priorities of haemorrhage control, blood-component replacement, correction of coagulopathy, and restoration of adequate tissue perfusion.

Overall Direction and Strength of Evidence

Across the available literature, the most consistent evidence supports rapid recognition and treatment of major traumatic haemorrhage, coordinated blood-component resuscitation, early antifibrinolytic treatment in eligible bleeding patients, minimization of unnecessary crystalloid exposure, and continuous reassessment of haemodynamic and coagulation status. The randomized evidence is strongest for tranexamic acid and for comparison of major transfusion strategies, while prospective observational evidence supports the importance of early plasma and platelet delivery.

Importantly, the evidence does not justify combining damage control resuscitation, transfusion ratios, tranexamic acid, fluid management, vasopressor therapy, and haemodynamic monitoring into a single numerical estimate of "medical optimization." These strategies operate through different mechanisms, are implemented at different stages of resuscitation, and have been evaluated using different study designs and outcomes. Their clinical effects are therefore more appropriately interpreted as complementary components of an integrated resuscitation pathway.

The evidence base also demonstrates several areas of uncertainty. The optimal implementation of fluid restriction, blood-pressure targets, vasoactive support, and advanced monitoring is influenced by injury phenotype, severity of haemorrhage, traumatic brain injury, cardiovascular reserve, institutional capability, timing of definitive haemorrhage control, and availability of blood products and monitoring technologies. The available evidence therefore supports protocolized principles combined with individualized clinical application rather than a uniform resuscitation algorithm for every patient.

The evidence synthesized in this review indicates that contemporary optimization of haemorrhagic shock in polytrauma has shifted from predominantly volume-based resuscitation toward early haemorrhage control, haemostatic blood-component replacement, timely antifibrinolytic therapy, restricted crystalloid exposure, and physiology-guided resuscitation. Among the treatment domains considered, balanced transfusion and tranexamic acid are supported by the most directly applicable comparative clinical evidence.

For blood-component therapy, the PROPPR randomized trial of 680 patients compared 1:1:1 with 1:1:2 plasma:platelet:RBC resuscitation and demonstrated better haemostatic control and fewer deaths due to exsanguination with the 1:1:1 strategy, although this did not translate into a definitive overall 30-day mortality difference (9). These findings are complemented by the prospective PROMMTT cohort of 1,245 patients, in which earlier administration of plasma and platelets during major transfusion was associated with improved survival (4). Together, these studies indicate that timely and coordinated blood-component delivery is clinically important, while also showing that survival after severe polytrauma depends on factors extending beyond transfusion ratio alone.

Tranexamic acid demonstrated a similarly strong time-dependent pattern. In the 20,211-patient CRASH-2 randomized trial, tranexamic acid reduced death attributable to bleeding without evidence of increased vascular occlusive complications (5). Subsequent analysis demonstrated that the benefit was greatest when treatment was initiated soon after injury and declined with treatment delay (6). The clinical implication is that antifibrinolytic therapy should form part of early haemorrhage management rather than being considered a late corrective intervention after advanced shock and coagulopathy have developed.

The evidence supporting damage control resuscitation more broadly is consistent with these findings but is methodologically more heterogeneous. Contemporary trauma guidance emphasizes rapid haemorrhage control, early blood-component replacement, restricted crystalloid administration, correction of trauma-induced coagulopathy, and physiologically appropriate blood-pressure

management (3,8). These strategies are mechanistically interconnected: reducing excessive crystalloid exposure may limit haemodilution and tissue oedema, balanced blood-component replacement supports haemostasis and oxygen delivery, and controlled resuscitation before definitive haemostasis may reduce the risk of exacerbating ongoing bleeding.

Goal-directed haemodynamic management and advanced monitoring provide an additional layer of individualization. Measures such as lactate, base deficit, cardiac output, stroke-volume-related parameters, and coagulation monitoring may identify persistent perfusion abnormalities or specific haemostatic deficits that are not fully reflected by conventional vital signs (3,7). However, the current evidence base does not establish one universally superior monitoring strategy for polytrauma, nor does it demonstrate that monitoring independently improves outcomes irrespective of the therapeutic responses it triggers.

Evidence regarding vasopressor therapy is comparatively less definitive within the literature available for this review. Vasopressors may support perfusion pressure in selected patients after appropriate correction of haemorrhage and intravascular volume loss, but they cannot substitute for haemorrhage control or adequate blood-volume replacement. Their use should therefore remain physiologically targeted and individualized rather than routinely applied to all patients with traumatic hypotension.

Overall, the direction of evidence favours an integrated and time-sensitive approach to traumatic haemorrhagic shock. The most persuasive evidence supports early tranexamic acid and coordinated blood-component resuscitation, while damage control principles, restricted crystalloid administration, individualized haemodynamic management, and coagulation-guided therapy provide the broader physiological framework in which these interventions are delivered. The heterogeneity of interventions and available evidence also confirms why a structured narrative synthesis is more appropriate for this topic than the previously presented simulated meta-analysis.

DISCUSSION

This structured narrative review indicates that contemporary management of haemorrhagic shock in adult polytrauma is best understood as an integrated, time-sensitive resuscitation strategy rather than as the application of a single therapeutic intervention. Across the evidence considered, the most consistently supported principles are rapid recognition and control of haemorrhage, early haemostatic blood-component resuscitation, timely administration of tranexamic acid in appropriate bleeding patients, avoidance of unnecessary crystalloid loading, correction of trauma-induced coagulopathy, and repeated physiological reassessment during operative and perioperative care (2,3). The strength of evidence, however, is not uniform across these components. Randomized evidence provides comparatively strong support for early tranexamic acid and informs the composition of massive transfusion, whereas the evidence supporting permissive hypotension, vasopressor therapy, individualized haemodynamic targets, and advanced monitoring is more heterogeneous and dependent on clinical context.

The transfusion literature illustrates the importance of interpreting individual resuscitation components within the broader process of haemorrhage control. In the PROPPR randomized clinical trial, a 1:1:1 plasma:platelet:red blood cell strategy did not significantly reduce overall mortality at 24 hours or 30 days compared with a 1:1:2 strategy, but it resulted in more frequent haemostasis and fewer deaths from exsanguination during the early period of active bleeding (8). This distinction is clinically important. It suggests that the principal advantage of balanced resuscitation may occur during the immediate haemorrhagic phase, when effective clot formation and rapid restoration of haemostatic capacity are particularly important, rather than translating automatically into a uniform longer-term mortality benefit. Consequently, balanced transfusion should not be interpreted as an isolated numerical ratio but as one component of coordinated damage control resuscitation.

The observational PROMMTT findings complement this interpretation by emphasizing the importance of timing. Earlier plasma and platelet administration among patients requiring major transfusion was associated with improved survival compared with delayed component administration (4). Because PROMMTT was observational, residual confounding by injury severity, treatment selection, institutional protocols, and survivorship effects limits causal interpretation. Nevertheless, when considered together with PROPPR, the evidence suggests that both timeliness and composition of blood-component therapy influence early haemorrhagic outcomes. This supports trauma systems capable of recognizing major haemorrhage promptly, activating structured transfusion pathways early, and delivering appropriate blood components without avoidable delays.

Tranexamic acid is supported by particularly influential randomized evidence. The CRASH-2 trial demonstrated a reduction in death attributable to bleeding among trauma patients receiving tranexamic acid without evidence of an associated increase in vascular occlusive events (5). The subsequent treatment-timing analysis provided an important refinement by showing that benefit was greatest when the drug was administered early and declined with increasing delay after injury (6). This finding reinforces a broader principle emerging across trauma resuscitation: effectiveness depends not only on which intervention is provided but also on when it is provided relative to haemorrhage, coagulopathy, physiological deterioration, and definitive haemostasis. Delayed correction after advanced shock has developed cannot necessarily reproduce the benefit of interventions delivered during the early haemorrhagic phase.

These findings also provide a physiological rationale for damage control resuscitation. Severe haemorrhage initiates a progressive interaction among tissue hypoperfusion, acidosis, hypothermia, endothelial disturbance, and trauma-induced coagulopathy. Excessive crystalloid administration may aggravate this process through haemodilution, reduced clotting-factor concentrations, tissue oedema, and worsening hypothermia (2,3,9). Contemporary resuscitation therefore seeks to maintain adequate organ perfusion while avoiding unnecessary fluid exposure and prioritizing haemostatic therapy and definitive control of bleeding. This represents a fundamental shift from earlier approaches based predominantly on rapid restoration of normal blood pressure through large-volume crystalloid administration.

Permissive hypotension should nevertheless be interpreted cautiously because the physiological objectives of resuscitation differ according to injury phenotype. Before haemorrhage control, excessive elevation of arterial pressure may theoretically increase ongoing blood loss or disrupt developing thrombi, providing the rationale for accepting lower pressure targets in selected patients. However, such an approach cannot be generalized indiscriminately. Patients with concomitant traumatic brain injury, impaired cerebral autoregulation, pre-existing cardiovascular disease, advanced age, prolonged shock, or other conditions requiring adequate perfusion pressure may have substantially different physiological requirements. The relevant clinical objective is therefore not hypotension itself but the achievement of sufficient organ perfusion without exacerbating uncontrolled haemorrhage. This distinction supports individualized targets rather than rigid application of a single blood-pressure threshold.

Goal-directed haemodynamic management extends the principle of individualization by integrating variables beyond conventional heart rate and arterial pressure. Lactate, base deficit, cardiac output, stroke-volume-related measures, urine output, peripheral perfusion, and other dynamic or biochemical indicators may contribute to determining whether tissue perfusion has been restored adequately. However, the supporting perioperative goal-directed therapy literature must be interpreted carefully when applied to polytrauma. The Cecconi et al. evidence concerned high-risk surgical populations rather than traumatic haemorrhagic shock specifically (7). Accordingly, it supports the physiological rationale for individualized haemodynamic management but should not be treated as direct evidence that a particular goal-directed protocol improves outcomes in polytrauma. Trauma-specific studies are needed

to determine which monitoring variables, thresholds, and treatment algorithms provide clinically meaningful benefit during active traumatic haemorrhage.

Similar caution is required when considering vasopressor therapy. Vasopressors can increase arterial pressure through peripheral vasoconstriction but cannot replace lost circulating blood volume, restore haemostatic components, or control the source of haemorrhage. Their routine use before adequate correction of severe hypovolaemia may therefore produce an apparently improved blood pressure without correcting the underlying deficit in circulating volume and tissue oxygen delivery. Conversely, selected patients may require vasoactive support when clinically important hypotension persists despite appropriate haemorrhage control and resuscitation or when cardiovascular dysfunction contributes to haemodynamic instability. The available evidence considered in this review is insufficient to define a universally optimal vasopressor strategy in traumatic haemorrhagic shock. Their use should consequently remain individualized and subordinate to haemorrhage control, blood-volume restoration, and assessment of tissue perfusion.

Advanced coagulation monitoring provides another mechanism for individualizing resuscitation. Conventional coagulation tests provide useful information but may not fully characterize the rapidly evolving haemostatic abnormalities occurring during severe traumatic bleeding. Viscoelastic techniques such as thromboelastography and rotational thromboelastometry can provide a more dynamic assessment of clot initiation, clot strength, and fibrinolytic abnormalities and may facilitate targeted haemostatic treatment within appropriately equipped trauma systems (3). Their potential benefit lies primarily in informing therapeutic decisions rather than in monitoring itself. Advanced technology cannot compensate for delayed haemorrhage control, inadequate blood-component availability, or failure to respond appropriately to physiological deterioration. Implementation should therefore be considered within an integrated resuscitation pathway rather than as an independent intervention.

An important finding of this review is the considerable difference in evidentiary strength among the various components commonly grouped under the broad term “medical optimization.” Early tranexamic acid is supported by a large international randomized trial, and transfusion-ratio strategies have been assessed through randomized comparative evidence (5,8). Timing of blood-component administration is additionally informed by prospective multicentre observational evidence (4). By contrast, broader recommendations concerning fluid restriction, permissive hypotension, vasoactive therapy, and individualized monitoring depend to a greater extent on physiological rationale, heterogeneous clinical studies, extrapolation from related surgical populations, and consensus-based trauma guidance (2,3,7,9). These differences should be preserved when translating evidence into practice rather than presenting every component as possessing an equivalent level of certainty.

The evidence also demonstrates why a single pooled estimate of “medical optimization” would be clinically difficult to interpret. Tranexamic acid modifies fibrinolysis; balanced transfusion restores circulating blood components and haemostatic capacity; fluid strategies influence circulating volume and haemodilution; vasopressors modify vascular tone; and advanced monitoring provides information used to guide other treatments. These interventions differ in mechanism, indication, timing, comparator, and outcome. Combining them statistically as though they represented a common intervention would risk generating a numerically precise but clinically ambiguous estimate. The structured narrative approach used in the present review therefore allows individual therapeutic domains to be evaluated according to their underlying evidence while also examining how they function together during haemorrhagic resuscitation.

From a clinical perspective, the available evidence favours resuscitation pathways organized around rapid decision-making and coordination between emergency, anaesthetic, surgical, transfusion, and critical-care teams. Early identification of patients at risk of major haemorrhage permits prompt activation of blood-product pathways, antifibrinolytic treatment when indicated, accelerated haemorrhage control, prevention of hypothermia, and repeated reassessment of perfusion and

coagulation. Standardized institutional protocols may reduce avoidable treatment delays, but protocols should remain sufficiently flexible to account for differences in injury pattern, traumatic brain injury, comorbidity, response to initial therapy, availability of blood components, and local monitoring capability.

The evidence base has several limitations that affect interpretation. First, the interventions reviewed are clinically and methodologically heterogeneous, preventing direct comparison of their relative effectiveness. Second, some evidence derives from observational investigations in which treatment allocation and timing may be influenced by injury severity and institutional practice. Third, evidence used to contextualize haemodynamic optimization is partly derived from non-trauma surgical populations and therefore has limited directness for patients with active traumatic haemorrhage (7). Fourth, recommendations derived from clinical guidelines and narrative sources synthesize broader evidence but should not be interpreted as independent comparative treatment effects. Finally, because the present article uses a structured narrative rather than systematic-review methodology, it does not claim exhaustive identification of all eligible studies and remains susceptible to literature-selection bias.

These limitations also define priorities for future investigation. Additional multicentre prospective studies should determine which haemodynamic targets are most appropriate across different trauma phenotypes and clarify the role, timing, and patient selection criteria for vasopressor therapy. Further comparative research is required to determine how viscoelastic-guided resuscitation, conventional laboratory-guided management, whole-blood strategies, component therapy, and contemporary massive transfusion protocols influence patient-centred outcomes. Studies should also account explicitly for treatment timing, severity and source of haemorrhage, concomitant traumatic brain injury, prehospital treatment, time to definitive haemostasis, and institutional resource availability. Rather than evaluating isolated treatments without accounting for surrounding trauma-system performance, future research should increasingly examine how individual components interact within coordinated haemorrhage-control pathways.

Overall, the evidence supports a transition from indiscriminate volume replacement toward haemostatic, physiology-informed, and time-critical resuscitation. The clearest evidence supports early tranexamic acid and coordinated blood-component therapy, while other elements of medical optimization should be applied according to individual physiological requirements and the strength of available evidence. The central clinical principle is therefore not aggressive normalization of individual haemodynamic variables but rapid correction of the causes and consequences of haemorrhagic shock while definitive haemorrhage control is achieved.

CONCLUSION

Medical optimization of haemorrhagic shock in adult polytrauma requires an integrated approach combining rapid haemorrhage control, early haemostatic resuscitation, appropriate blood-component therapy, timely tranexamic acid administration, avoidance of unnecessary crystalloid loading, correction of trauma-induced coagulopathy, and individualized assessment of tissue perfusion. Randomized evidence most clearly supports early antifibrinolytic therapy and informs balanced transfusion strategies, while evidence for permissive hypotension, vasopressor therapy, goal-directed haemodynamic management, and advanced monitoring remains more context-dependent. These interventions should therefore not be considered interchangeable or assumed to provide equivalent clinical benefit. Effective perioperative management depends on matching therapy to the patient's evolving physiological state while minimizing delays to definitive haemostasis. Future trauma research should prioritize multicentre evaluation of integrated resuscitation pathways and define patient-specific haemodynamic and coagulation targets that improve clinically meaningful outcomes.

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