BJCMR Brazilian Journal of Clinical Medicine and Review

PCAA PUBLICAÇÕES CIENTÍFICAS DE ACESSO ABERTO

Health Review

Physiology-Driven Decision-Making in Trauma-Induced Coagulopathy: Integrating Shock Index, FAST, and Hemostatic Resuscitation

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Abstract: Trauma-induced coagulopathy (TIC) is a major determinant of early mortality in severely injured patients and represents a complex, multifactorial physiological disorder that develops shortly after injury. It results from the interaction between tissue hypoperfusion, endothelial dysfunction, inflammatory activation, and dysregulation of coagulation and fibrinolysis pathways. Contemporary evidence demonstrates that tissue hypoperfusion may precede overt hypotension, requiring integrated diagnostic and therapeutic strategies. This narrative review discusses the role of the Shock Index, focused assessment with sonography for trauma (FAST/eFAST), and computed tomography in the early stratification of hemorrhagic shock, as well as their integration with damage control resuscitation principles, including early tranexamic acid administration. The coordinated application of these tools allows earlier surgical decision-making, reduces therapeutic delays, and improves clinical outcomes, particularly in emergency settings and public healthcare systems.

Keywords: Severe Trauma; Trauma-Induced Coagulopathy; Shock Index; FAST; Hemostatic Resuscitation.

Citation: Rocha LFR, Rosati APM, Rosati ALM, Rocha FMS. Physiology - Driven Decision-Making in Trauma - Induced Coagulopathy: Integrating Shock Index, FAST, and Hemostatic Resuscitation. Brazilian Journal of Clinical Medicine and Review. 2026: Jan-Dec;04(1):bjcmr54.

https://doi.org/10.52600/2763-583X.bjcmr.2026.4.1.bjcm54

Received: 20 January 2026
Accepted: 10 February 2026
Published: 13 February 2026

1. Introduction

Hemorrhage remains the leading cause of preventable death following severe trauma, particularly within the first hours after injury. Contemporary trauma care recognizes that traumatic bleeding is not merely the consequence of mechanical vascular disruption but reflects a complex and rapidly evolving systemic process known as trauma-induced coagulopathy (TIC). This condition emerges early after injury, often before overt hypotension develops, and is driven by tissue hypoperfusion, endothelial injury, inflammatory signaling, and dysregulation of coagulation pathways [1, 2].

Central to early pathophysiology is disruption of the endothelial glycocalyx, leading to increased vascular permeability and dysregulated hemostasis. In parallel, activation of the thrombomodulin–protein C pathway has been associated with systemic anticoagulation and hyperfibrinolysis, supporting the concept that TIC is a distinct biological entity rather than a late, purely iatrogenic phenomenon [3].

2. Occult Shock and Early Physiological Deterioration

A defining challenge in modern trauma resuscitation is the recognition of occult shock. Patients may initially present with preserved systolic blood pressure due to compensatory sympathetic responses, masking ongoing hemorrhage and microcirculatory failure. Reliance on isolated vital signs delays recognition of physiological collapse and contributes to delayed activation of life-saving interventions. The Shock Index (SI), calculated as heart rate divided by systolic blood pressure, captures the dynamic relationship between cardiovascular compensation and circulatory reserve. Early clinical work demonstrated that SI can identify critical illness more effectively than conventional vital-sign thresholds [4]. In trauma, higher SI values correlate with increasing injury severity, transfusion needs, and mortality [5]. Occult shock should be suspected in patients with SI ≥0.9, particularly when accompanied by elevated serum lactate or base deficit abnormalities, even when blood pressure appears "normal" [6].

3. Operational Shock Index Thresholds

From a clinical perspective, SI thresholds must be operational rather than conceptual. An SI ≥0.9 identifies patients at increased risk of significant hemorrhage and should trigger heightened surveillance, serial reassessment, early blood bank notification, and proactive preparation for hemorrhage control. Prehospital and early in-hospital data suggest that SI >0.9 can identify patients at risk for massive transfusion who might otherwise be considered relatively normotensive [7]. Patients with intermediate SI values (0.9–1.1) represent a dynamic risk group. In this scenario, serial SI trends, lactate/base deficit trajectories, and response to limited resuscitation become more informative than isolated values. Failure of SI normalization within the first 60–90 minutes, or a rising SI despite resuscitation, should prompt escalation of care. Importantly, a persistently elevated SI ≥1.1 after initial resuscitation should be interpreted as a physiological trigger for immediate hemorrhage control; further diagnostic steps should not delay operative or endovascular intervention in the appropriate clinical context [5, 8].

4. Limitations of Shock Index

Despite its strengths, the Shock Index has recognized limitations. Elderly patients and individuals receiving beta-blockers may exhibit blunted tachycardic responses, reducing SI sensitivity and increasing the risk of false reassurance. In these populations, trend analysis and multimodal assessment are crucial. A single SI value should be interpreted in context, integrating mechanism of injury, clinical examination, ultrasound findings, and metabolic markers rather than serving as an isolated binary trigger [9].

5. FAST, CT, and Physiological Integration

Focused Assessment with Sonography for Trauma (FAST) remains a cornerstone of early trauma evaluation. FAST offers rapid bedside detection of hemoperitoneum and pericardial tamponade but does not quantify bleeding severity or exclude hollow viscus injury. The diagnostic performance of FAST-based pathways has been questioned by systematic reviews, underscoring heterogeneity across studies and the need for contextual interpretation [10]. FAST, computed tomography (CT), and physiological assessment should not be viewed as competing strategies but rather as complementary tools. In unstable patients or those with worsening physiology (including rising SI), hemorrhage control must proceed regardless of FAST findings. Stable patients with a positive FAST often benefit from CT for injury characterization, while equivocal FAST results in stable patients warrant repeat ultrasound or early CT rather than immediate surgical exploration. Modern whole-body CT can be efficient in stable or transient responders and should not be discouraged when physiologic criteria for imaging are met [11].

6. Hemostatic Resuscitation and Viscoelastic Testing

Damage control resuscitation aims to interrupt the lethal triad of hypothermia, acidosis, and coagulopathy. Initial resuscitation frequently relies on empiric balanced transfusion strategies, commonly approximating a 1:1:1 ratio of plasma, platelets, and red blood cells. However, "balanced transfusion" should not be interpreted as a rigid recipe independent of physiology. As early as feasible, empiric strategies should transition toward goal-directed hemostatic therapy. Viscoelastic testing, including thromboelastography (TEG) and rotational thromboelastometry (ROTEM), provides near-real-time assessment of clot initiation, strength, and fibrinolysis, enabling individualized therapy. Incorporation of viscoelastic testing early in resuscitation, ideally within the first transfusion cycle, can refine product selection, reduce unnecessary transfusion, and match treatment to coagulopathy phenotype [12, 13].

7. Tranexamic Acid and Temporal Windows

The CRASH-3 trial and subsequent analyses reinforce the time-dependent benefit of tranexamic acid (TXA) in bleeding trauma patients. Treatment within three hours of injury is associated with reduced mortality, with the greatest benefit when administered as early as possible, particularly within the first hour [14]. Delayed administration beyond this window may confer no benefit and should prompt careful clinical judgment. In public healthcare systems, early TXA administration at the prehospital or emergency department entry point represents a low-cost, high-impact intervention that complements physiology-driven resuscitation.

8. Physiology-Based Strategies in Resource-Limited Systems

Global trauma care operates across heterogeneous resource environments. In low- and middle-income contexts, access to advanced endovascular platforms, continuous laboratory monitoring, or rapid CT may be limited. Physiology-first decision-making frameworks leveraging SI and bedside ultrasound provide scalable solutions that reduce dependence on subjective judgment alone. Importantly, the portability and relative affordability of ultrasound has supported expanding use in low-resource settings, although training, governance, and quality assurance remain essential for safe implementation [15]. By standardizing early recognition of hemorrhagic risk, physiology-driven triggers may reduce inter-provider variability and promote greater equity in trauma care.

9. Future Directions and Artificial Intelligence

The next evolution of trauma resuscitation is moving from isolated time-point measurements to continuous physiological surveillance capable of anticipating decompensation. Machine learning models that integrate admission hemodynamics, including SI, together with laboratory surrogates and response-to-treatment features have demonstrated improved prediction of massive transfusion needs and early mortality compared with traditional scoring approaches [16]. Nevertheless, AI-based decision-support systems must be implemented cautiously, ensuring transparency, clinician oversight, calibration to local populations, and validation in real-world workflows. In many public systems, the most pragmatic near-term pathway may involve embedding simple physiology-based triggers into digital checklists and electronic triage tools, progressively advancing toward more complex models as infrastructure and governance mature.

10. Conclusion

Throughout the manuscript, evidence supporting shock index thresholds, ultrasound-based decision-making, viscoelastic-guided resuscitation, endothelial dysfunction, antifibrinolytic therapy, and trauma system implementation has been discussed and supported by the literature [1, 2, 4- 8, 10-12, 14-17].

Trauma-induced coagulopathy represents an early, physiology-driven process that demands prompt recognition and decisive intervention. The Shock Index, when opera-

tionalized with clear thresholds and interpreted dynamically, helps identify occult shock and supports earlier activation of hemorrhage control and hemostatic resuscitation. Integration of SI with bedside ultrasound, modern CT workflows for appropriate candidates, viscoelastic testing, and time-sensitive adjuncts such as tranexamic acid enables a cohesive, physiology-first strategy. By aligning simple clinical metrics with contemporary hemostatic principles and scalable decision pathways, trauma systems can move toward earlier, more equitable, and physiology-driven intervention across diverse resource environments [1, 8, 11, 17].

Funding: None.

Research Ethics Committee Approval: None.

Acknowledgments: None.

Conflicts of Interest: The authors declare no conflict of interest.

Supplementary Materials: None.

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