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Mini-Review

Shock Index, FAST, and Hemostatic Resuscitation in Trauma-Induced Coagulopathy: Physiological Thresholds, Occult Shock, and Contemporary Decision-Making

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Abstract: Trauma-induced coagulopathy (TIC) is increasingly recognized as an early, endogenous, and pathophysiologically complex response to hemorrhagic shock, frequently preceding overt hypotension and clinical deterioration. Despite substantial advances in trauma systems, the timely identification of patients who will require massive transfusion remains a major determinant of survival. The Shock Index (SI), defined as the ratio of heart rate to systolic blood pressure, has emerged as a practical and physiologically grounded marker capable of identifying occult hypoperfusion during the compensated phase of shock. This perspective provides an in-depth discussion of SI thresholds, integration with bedside imaging, contemporary hemostatic resuscitation strategies, and the evolving role of machine learning–based decision support. Particular emphasis is placed on real-world clinical implementation across heterogeneous healthcare systems, including low-resource environments.

Keywords: Shock Index; Massive Transfusion; Trauma.

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1. Introduction

The Shock Index (SI), defined as the ratio of heart rate to systolic blood pressure, has gained renewed interest as a simple yet integrative physiological marker capable of unmasking early circulatory compromise. Unlike isolated vital signs, SI reflects the dynamic interaction between cardiovascular compensation and circulatory failure. Large observational cohorts and meta-analyses have demonstrated that SI values ≥0.9 are consistently associated with increased mortality, need for massive transfusion, intensive care unit admission, and operative intervention [1-3]. Importantly, higher thresholds (≥1.1) appear to identify a subgroup at particularly high risk of rapid decompensation.

The concept of "occult shock" has emerged to describe a state in which global perfusion is already compromised despite apparently acceptable vital signs. Multiple studies have shown that patients may maintain normotension through intense sympathetic compensation while simultaneously progressing toward endothelial injury, microcirculatory collapse, and coagulopathy [4-5]. In this context, reliance on systolic blood pressure alone is insufficient and potentially dangerous. Early identification of this compen-

sated phase is therefore central to preventing progression to irreversible shock and established TIC.

Trauma-induced coagulopathy (TIC) is now recognized as an early, endogenous, and prognostically decisive phenomenon rather than a late consequence of hemorrhage or iatrogenic resuscitation. Contemporary trauma literature consistently demonstrates that TIC develops within minutes of injury and is independently associated with increased mortality, transfusion requirements, organ failure, and resource utilization [2,6-7]. Despite this recognition, many trauma systems continue to rely on late physiological markers, most notably sustained hypotension, to trigger aggressive hemorrhage control and massive transfusion protocols (MTPs). This delay represents a fundamental mismatch between modern pathophysiology and outdated clinical thresholds.

Parallel to advances in physiological monitoring, bedside imaging, particularly focused assessment with sonography in trauma (FAST) and extended FAST (eFAST), has become a cornerstone of early trauma evaluation. When interpreted in isolation, FAST findings may lack sensitivity in stable patients; however, when integrated with physiological markers such as SI, ultrasound gains decision-making power by contextualizing anatomical findings within real-time perfusion status [8-9]. This integrated approach aligns with damage control principles by prioritizing rapid hemorrhage control while avoiding unnecessary delays.

The present manuscript is intentionally framed as a perspective and narrative synthesis rather than a systematic review. Its objective is not to exhaustively catalog all available evidence, but to critically examine how the integration of SI, bedside imaging, and emerging data-driven tools can improve early decision-making for hemorrhagic shock and TIC, particularly within heterogeneous and resource-constrained trauma systems such as those found in Brazil and other middle-income countries.

2. Materials and Methods

This manuscript is structured as a narrative perspective informed by a comprehensive review of the literature published between 2010 and 2025. Electronic databases including PubMed, Scopus, and SciELO were searched using combinations of the terms 'Shock Index', 'trauma', 'massive transfusion', 'hemorrhagic shock', and 'trauma-induced coagulopathy'. Rather than pursuing a systematic synthesis, the objective was to contextualize high-quality evidence within the practical realities of trauma care. Priority was given to studies addressing physiological thresholds, bedside applicability, outcomes, and emerging predictive technologies. This approach is consistent with the intent of a Perspective article, which aims to integrate evidence with expert clinical insight rather than exhaustively catalog all available studies.

3. Discussion

Looking forward, the integration of SI into machine learning frameworks represents a logical evolution rather than a technological leap. Predictive models incorporating SI alongside lactate, hemoglobin trends, and ultrasound findings have demonstrated superior discrimination for massive transfusion and mortality compared with human assessment alone [10]. Importantly, these tools should be conceptualized as decision-support systems rather than autonomous decision-makers, particularly in settings with limited digital infrastructure.

Hemostatic resuscitation strategies have evolved beyond fixed transfusion ratios. While 1:1:1 plasma, platelets, and red blood cells remain a practical default in many settings, viscoelastic assays such as thromboelastography (TEG) and rotational thromboelastometry (ROTEM) offer goal-directed alternatives that may reduce product utilization and complications where available [11]. In resource-limited environments, SI and ultrasound serve as low-cost surrogates to guide early balanced resuscitation until advanced diagnostics are accessible.

Adjunctive therapies must also be time-sensitive. Evidence from the CRASH-3 trial and subsequent analyses supports administration of tranexamic acid within the first 3 hours after injury, with maximal benefit observed when given within the first hour [12]. For public healthcare systems, this reinforces the importance of protocolized early TXA use guided by physiological risk rather than confirmed hemorrhage alone.

At the molecular level, TIC is increasingly understood as a consequence of endothelial glycocalyx disruption and dysregulated activation of the protein C pathway. Hypoperfusion triggers shedding of the glycocalyx, loss of anticoagulant surface regulation, and systemic anticoagulation, fibrinolysis, and platelet dysfunction [6,13]. Recognition of this early biology reinforces the need for rapid physiological triggers rather than delayed laboratory confirmation.

The relationship between physiological instability and imaging strategy remains a frequent source of controversy. While concerns persist that computed tomography may delay life-saving intervention, modern whole-body CT (WBCT) protocols can often be completed within minutes and provide unparalleled diagnostic precision in hemodynamically stable or transient responder patients [14]. In this context, relative hemodynamic stability should be defined not by static blood pressure values but by sustained response to minimal resuscitation without escalating vasopressor or transfusion requirements. Patients with SI persistently ≥1.1 despite initial resuscitation should generally bypass CT in favor of operative or endovascular hemorrhage control.

Clinical decision-making based on SI must, however, account for important limitations. Elderly patients, individuals receiving beta-blockers, and those with autonomic dysfunction may exhibit blunted tachycardic responses, leading to falsely reassuring SI values [1]. In these populations, trend analysis, adjunctive markers such as lactate and base deficit, and close integration with imaging findings become essential. Rather than disqualifying SI, these limitations underscore the need for multimodal interpretation.

The early identification of patients at risk for trauma-induced coagulopathy hinges on recognizing physiological decompensation before the onset of overt hypotension. In this regard, the Shock Index has emerged as a pragmatic surrogate for global circulatory stress. Multiple studies suggest that an SI threshold ≥0.9 should be interpreted as a warning signal for occult shock, whereas values ≥1.1 correlate strongly with massive transfusion requirements, early mortality, and failure of nonoperative strategies [2-3]. These thresholds should not be viewed as rigid triggers, but as dynamic risk strata that inform escalating diagnostic and therapeutic intensity.

From an evidence synthesis standpoint, recent meta-analytical data reinforce the clinical utility of SI for predicting massive transfusion and mortality. In particular, contemporary systematic reviews report consistent discriminatory performance across heterogeneous trauma populations, supporting SI as a pragmatic component of early MTP activation rather than a standalone decision-maker [15]. More recent observational work has also evaluated SI in penetrating mechanisms and alternative transfusion thresholds such as the Critical Administration Threshold, highlighting that physiologic triggers must be interpreted alongside mechanism, injury pattern, and the trajectory of response to resuscitation [16]. These findings strengthen the rationale for using SI in a tiered pathway that integrates bedside ultrasound, rapid laboratory surrogates, and early hemorrhage control strategies.

4. Conclusion

Trauma-induced coagulopathy is an early, dynamic phenomenon driven by tissue injury and hypoperfusion, with clinically meaningful consequences well before overt hypotension. In practical terms, the priority is to identify occult shock early enough to initiate hemorrhage control and hemostatic resuscitation while the patient is still physiologically salvageable [6,17]. Within this time-critical window, the Shock Index provides a rapid, bedside-available signal of compensatory stress that can be operationalized in protocols and embedded into digital decision support. For implementation, we propose

interpreting SI as a graded risk marker. SI ≥ 0.9 should prompt early escalation, blood bank notification, preparation for balanced transfusion, and accelerated imaging, whereas persistent SI ≥ 1.1 despite initial resuscitation should favor immediate activation of massive transfusion protocols and expedited hemorrhage control, bypassing delays that add little diagnostic value [3,15]. This framework must explicitly recognize populations in which SI underperforms (older age, beta-blockade, autonomic dysfunction), using adjuncts such as lactate/base deficit and close integration with FAST/WBCT decision pathways.

Hemostatic resuscitation should be aligned with contemporary evidence: prompt tranexamic acid administration within the earliest feasible window, balanced blood product strategies, and, where available, viscoelastic testing to individualize therapy and reduce avoidable product exposure [12,18]. In systems with constrained resources, SI and point-of-care ultrasound remain low-cost anchors for standardization, helping reduce cognitive bias and inter-provider variability while preserving diagnostic precision through clear criteria for 'relative hemodynamic stability' and appropriate CT utilization.

Finally, the next step is not to repeatedly re-validate SI, but to refine how it is used: as a dynamic trend variable within integrated clinical pathways and machine learning–assisted decision-support tools. Recent work demonstrates that models combining admission hemodynamics (including SI) with readily available clinical features can improve early prediction of MTP activation and hemorrhage control needs, while comparative studies highlight the potential of ML to complement clinician judgment in high-tempo environments [19-20]. In this perspective, the Shock Index serves both as a timeless bedside metric and as a foundational feature for scalable, equitable, and data-informed trauma care.

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