Perioperative Precision Medicine

Zentime

PERSPECTIVE

Sepsis-induced myocardial dysfunction: Hemodynamic stability as the cornerstone of anesthetic resuscitation

Qi Wang, Lingxuan Tang

Faculty of Anesthesiology, Changhai Hospital, Shanghai 200433, China.

Corresponding author: Lingxuan Tang.

Address correspondence to: Lingxuan Tang, Faculty of Anesthesiology, Changhai Hospital, No. 168 Changhai Road, Yangpu District, Shanghai 200433, China. E-mail: 19370620610@163.com.

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1 INTRODUCTION

Sepsis-associated cardiomyopathy (SCM), also known as sepsis-induced myocardial dysfunction (SIMD), is a significant determinant of poor outcomes in septic patients, and the associated cardiac dysfunction correlates closely with increased mortality [1]. Mechanistically, increases in inflammatory mediators and concurrent microcirculatory dysfunction suppress myocardial contractility, further impair right–left ventricular coupling, and make the heart susceptible to pump failure under abnormal loading conditions while diminishing tolerance to changes in intravascular volume [2]. During the initiation of anesthesia and resuscitation, patients are particularly susceptible to circulatory collapse owing to underlying hemodynamic instability and rapid cardiovascular changes during induction [3]. As the disease advances, myocardial dysfunction may progress to a stage where cardiac output becomes inadequate and systemic congestion occurs, culminating in the clinical manifestation of heart failure. Patients with sepsis and concomitant heart failure have higher mortality and are more prone to anesthesia-induced hypotension, yet specific perioperative guidance is lacking [4]. It is worth noting that sepsis-induced heart failure is not exclusively attributable to myocardial dysfunction associated with sepsis. It may also arise from the presence of pre-existing chronic heart failure in conjunction with sepsis, or from the simultaneous occurrence of both conditions during anesthesia, leading to hemodynamic instability. Thus, providing adequate organ perfusion while preventing fluid overload remains a major challenge in perioperative care. Here we describe a precision-based resuscitation strategy incorporating advanced imaging modalities, such as tissue Doppler and speckle-tracking echocardiography, and tailored volume management to preserve hemodynamic equilibrium and improve outcomes in this high-risk cohort.

2 PATHOPHYSIOLOGY OF SEPSIS AND MYOCARDIAL DYSFUNCTION

2.1 Systemic inflammatory response in sepsis

Sepsis is defined as life-threatening organ dysfunction caused by a dysregulated host response to infection. Inflammation-associated oxidative stress in sepsis promotes mitochondrial dysfunction, disrupts intracellular calcium handling, and ultimately diminishes myocardial contractility [5]. Endothelial barrier damage, leading to capillary leakage and diminished effective circulating volume, aggravates tissue hypoperfusion and microcirculatory dysfunction. The inflammation-mediated changes disrupt cardiovascular homeostasis and contribute to hemodynamic instability during anesthetic resuscitation, highlighting the need for targeted inflammatory and circulatory modulation.

2.2 Hemodynamic effects of myocardial dysfunction

The systemic hemodynamic effects of SCM are complex and potentially harmful. SCM is usually associated with compensatory left ventricular dilatation and impaired systolic function,

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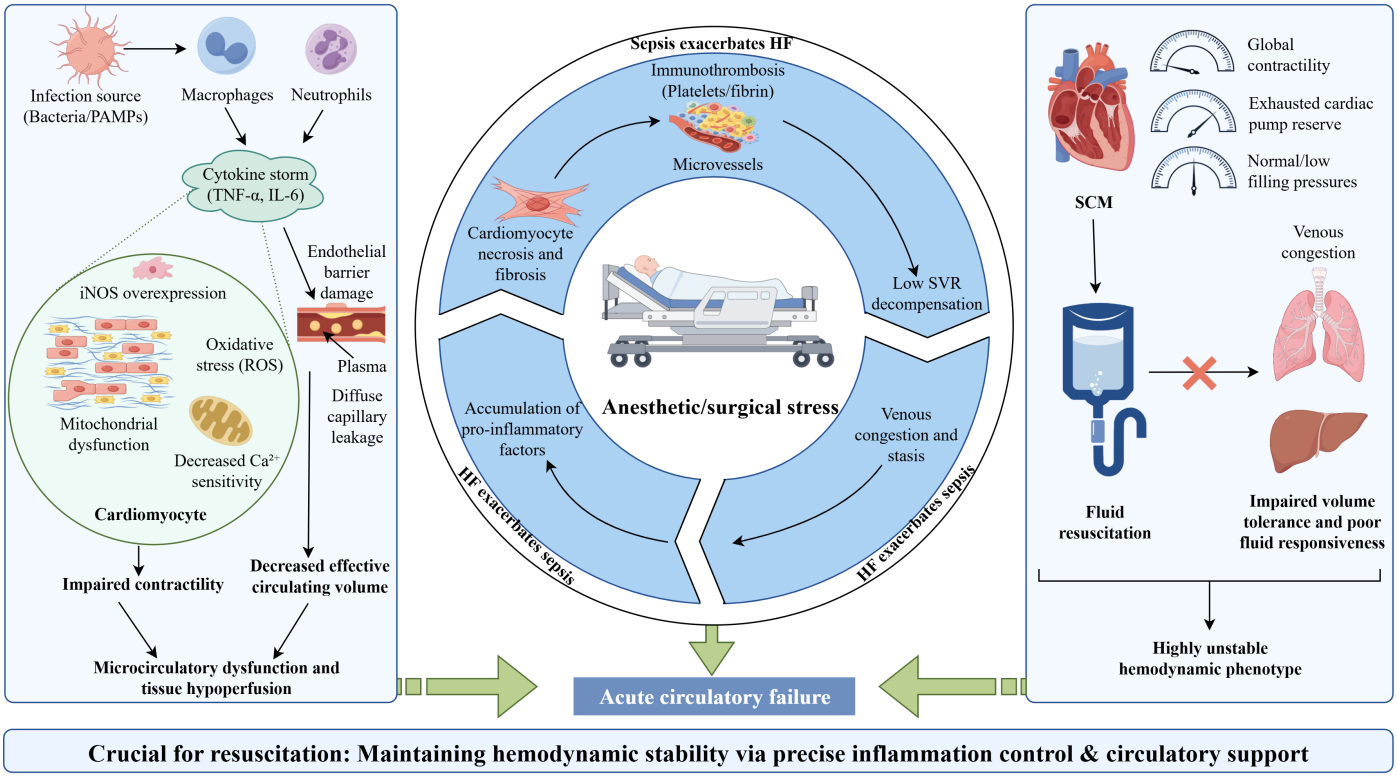


Figure 1. Pathophysiological mechanisms linking sepsis and myocardial dysfunction during anesthetic resuscitation. Acute inflammatory responses reflect the activation of immune cells, leading to increased production of pro-inflammatory cytokines such as TNF-α and IL-6. These cytokines can further activate iNOS, resulting in oxidative stress and exacerbation of mitochondrial dysfunction, ultimately leading to decreased myocardial contractility. Disruption of the vascular endothelial barrier results in capillary leakage and reduced effective circulating volume, which promotes microcirculatory dysfunction and impaired tissue perfusion. SCM leads to reduced cardiac contractility and diminished cardiac pump reserve, resulting in poor tolerance to fluid loading and an inadequate response to fluid resuscitation. Cross-activation of the inflammatory and coagulation pathways results in immunothrombosis and microvascular injury, whereas HF-related venous congestion further amplifies inflammatory mediator accumulation. These processes generate a vicious cycle that can culminate in acute circulatory failure. This figure was created with BioRender.com. Ca^{2+}, calcium ions; HF, heart failure; IL-6, interleukin-6; iNOS, inducible nitric oxide synthase; PAMPs, pathogen-associated molecular patterns; ROS, reactive oxygen species; SCM, sepsis-associated cardiomyopathy; SVR, systemic vascular resistance; TNF-α, tumor necrosis factor alpha.

but filling pressures are often normal or low at rest. The cardinal problem is depletion of cardiac pump reserve, which markedly reduces tolerance to volume loading and complicates fluid resuscitation. Patients with septic shock exhibit unstable hemodynamics, and anesthetic induction or pharmacological interventions may precipitate severe hypotension due to vasodilation and myocardial depression, potentially leading to circulatory failure [6]. It is therefore critical to understand heart failure–induced circulatory decompensation to enable tailored hemodynamic optimization.

2.3 Interaction between sepsis and myocardial dysfunction

The coexistence of sepsis and myocardial dysfunction involves complex systemic interactions rather than a simple unidirectional pathological process. The major pathophysiological interactions are illustrated in **Figure 1**. In sepsis, for instance, extracellular RNA released from injured tissues promotes inflammatory responses, leading to cardiomyocyte apoptosis and contributing to septic cardiomyopathy [7]. These structural alterations further impair cardiac performance and limit compensatory responses to sepsis-induced reductions in systemic vascular resistance. On the other hand, heart failure–related venous congestion promotes the accumulation of inflammatory mediators and microcirculatory dysfunction. This bidirectional interaction creates a vicious cycle that increases susceptibility to circulatory collapse during anesthetic resuscitation.

3 HEMODYNAMIC MANAGEMENT STRATEGIES DURING ANESTHETIC RESUSCITATION

3.1 Individualized fluid resuscitation

Fluid resuscitation in sepsis complicated by heart failure requires careful balance, as both inadequate resuscitation and fluid overload are associated with adverse outcomes [8]. Thus, the paradigm of resuscitation has shifted from empirical volume replacement to an increased focus on functional hemo-

dynamic assessment. International guidelines suggest using dynamic measures to guide fluid resuscitation rather than relying on physical examination or static parameters alone in patients with sepsis or septic shock [9]. Dynamic assessments such as stroke volume variation or the passive leg raising test should be preferred over static pressure indices alone to guide fluid titration. This individualized, fluid responsiveness–guided approach aims to balance tissue perfusion and cardiac preload status and constitutes a pillar of maintaining hemodynamic stability during anesthetic resuscitation in these high-risk subjects.

3.2 Emerging technologies for hemodynamic monitoring and assessment

As precision medicine has evolved, hemodynamic monitoring in anesthetic resuscitation has transitioned from measuring invasive pressures to performing functional and dynamic assessments. Transthoracic echocardiography can predict fluid responsiveness by measuring changes in left ventricular outflow tract velocity–time integral and stroke volume during the passive leg raising test. In patients with limited acoustic windows, the carotid artery velocity–time integral might be a practical alternative to provide rapid evaluation of circulatory status as it is simple to conduct and independent of cardiac imaging windows [10]. These bedside ultrasound modalities provide a means to monitor cardiac function and volume status, guiding hemodynamic optimization in real-time throughout anesthetic resuscitation.

3.3 Selection and application of pharmacological interventions

During the peri-anesthesia period and the resuscitation process, the judicious selection, combination, and titration of anesthetic induction agents are critically important in clinical anesthesia induction and the early stages of sepsis. For example, the administration of dobutamine, a β1-selective inotropic agent, improves cardiac output and splanchnic blood flow in patients with sepsis-associated myocardial dysfunction and low cardiac output. During induction, etomidate is preferable in patients with heart failure because of its favorable hemodynamic profile and has not been associated with increased in-hospital or 30-day mortality compared with other induction agents [11]. Upon the establishment of anesthesia, it is imperative that combination strategies addressing both vascular tone and myocardial contractility be tailored according to the patient's specific hemodynamic phenotype. For patients exhibiting preserved or hyperdynamic left ventricular function accompanied by significant vasodilation, the administration of norepinephrine as a first-line vasopressor is recommended as the first-line vasopressor. Conversely, in patients presenting with evident myocardial depression, characterized by reduced ejection fraction or low cardiac output despite adequate filling pressures, dobutamine, or a vasopressor-inotrope such as epinephrine, may be incorporated. The precise titration and withdrawal of vasoactive drugs are essential for achieving hemodynamic stability in patients with sepsis and heart failure. In patients with reduced myocardial reserve, the inappropriate use of vasopressor therapy can increase cardiac afterload, thereby elevating cardiac burden. Evidence regarding the optimal sequence of norepinephrine and vasopressin discontinuation remains inconclusive; in patients with septic shock and reduced left ventricular function, the discontinuation order was not associated with a significant difference in the incidence of clinically significant hypotension [12]. Consequently, therapeutic interventions should be guided by continuous physiological monitoring, with an emphasis on optimizing the equilibrium between tissue perfusion and myocardial oxygen consumption through incremental dose adjustments. A stepwise reduction strategy is advocated, accompanied by meticulous monitoring of hemodynamic alterations, to mitigate the risk of circulatory failure and ensure a seamless transition from the high-risk resuscitation phase.

4 FUTURE PERSPECTIVES AND INNOVATIONS IN CLINICAL PRACTICE

In patients with coexisting SIMD and chronic cardiac insufficiency, clinicians must balance the benefits of fluid resuscitation against the risk of cardiac decompensation within a very narrow therapeutic window. **Figure 2** illustrates the integrated future clinical framework. Emerging evidence suggests that institutionalized multidisciplinary implementation teams—comprised of surgeons, anesthesiologists, intensivists and quality-control specialists—are key for maintaining hemodynamic stability. Such teams transcend fragmented decision-making and enable data-driven, collaborative management. Within such collaborative frameworks, hemodynamic support can be continuously assessed and optimized using objective indicators. Vasoactive and inotropic requirements, as quantified by the vasoactive-inotropic score (VIS), have been shown to be strongly associated with postoperative outcomes, highlighting the importance of optimized hemodynamic management in the early postoperative intensive care unit (ICU) period [13]. This integrated model not only enables technical coordination but also holds potential to reduce perioperative mortality and length of intensive care unit stay, with possible benefits for long-term outcomes.

Standard scoring systems, such as the Sequential Organ Failure Assessment and Acute Physiology and Chronic Health Evaluation scores, suffer from intrinsic time lags in capturing real-time physiological dynamics. By incorporating large heterogeneous datasets and revealing complex nonlinear interactions, machine learning is revolutionizing perioperative risk assessment [14]. Concerning SIMD, machine learning algorithms iteratively evaluate the highly intricate interrelationship among volume status, vascular tone and myocardial contractility. Combined with closed-loop systems for vasoactive drug

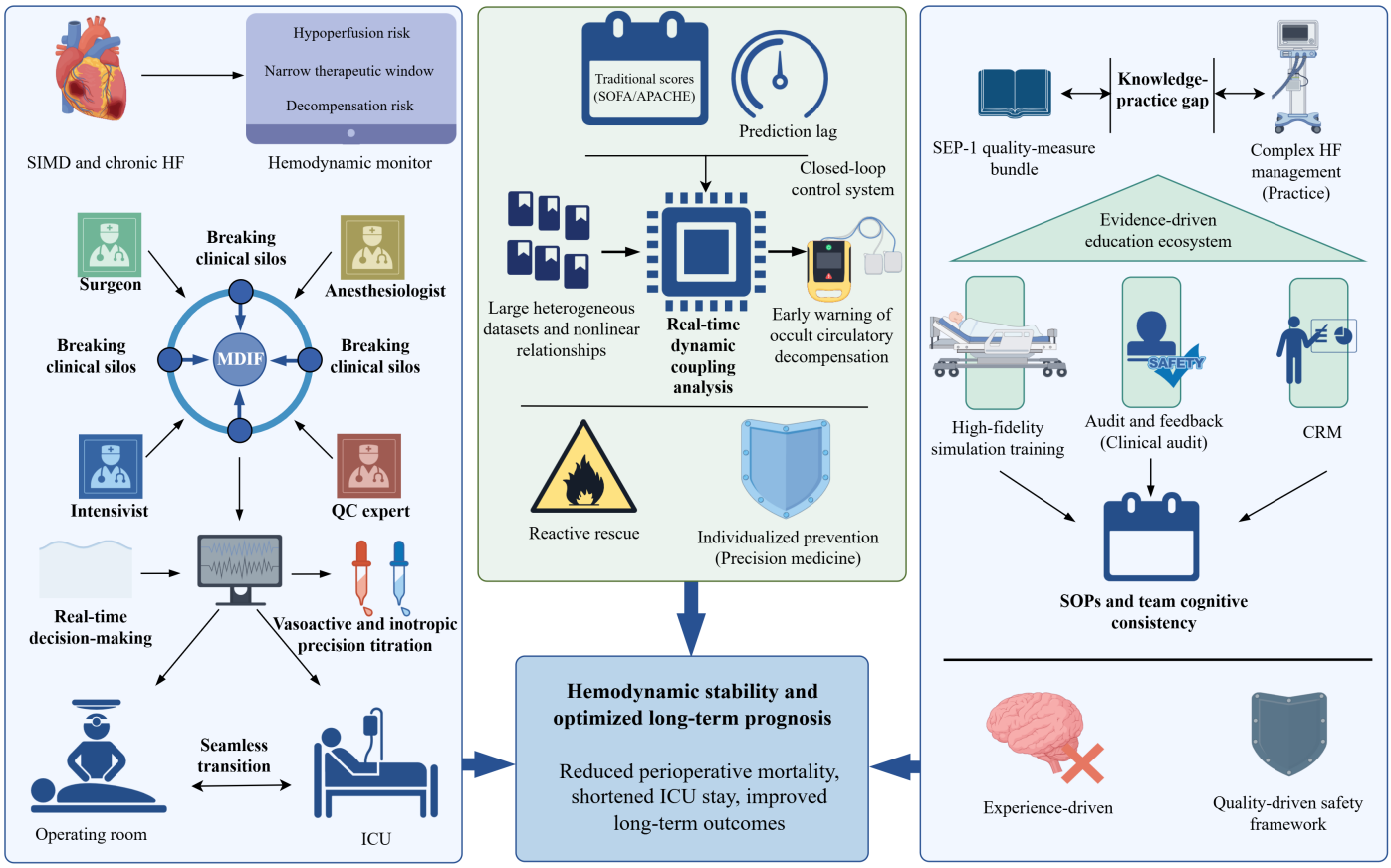


Figure 2. Integrated clinical innovation framework for perioperative management in SIMD with heart failure. The overlap between SIMD and chronic heart failure is common but is characterized by a narrow therapeutic window and a high risk of hemodynamic decompensation. Multidisciplinary collaboration among surgeons, anesthesiologists, intensivists, and quality-control teams can streamline perioperative decision-making, enabling precise titration of vasoactive and inotropic therapies across the perioperative continuum. Emerging technologies, such as machine learning–based dynamic monitoring and closed-loop control systems, may enable early identification of circulatory decompensation, shifting management from reactive rescue to individualized prevention. In parallel, evidence-based educational strategies—including simulation training, audit and feedback, and CRM—help bridge gaps between clinical practice and guideline recommendations. Collectively, these advances may contribute to improved hemodynamic stability, reduced perioperative mortality, shorter ICU stays, and improved long-term outcomes. This figure was created with BioRender.com. APACHE, Acute Physiology and Chronic Health Evaluation; CRM, crisis resource management; HF, heart failure; ICU, intensive care unit; QC, quality control; SIMD, sepsis-induced myocardial dysfunction; SOFA, Sequential Organ Failure Assessment; SOPs, standard operating procedures.

titration, these approaches allow early recognition of occult circulatory decompensation and facilitate a transition from reactive rescue to individualized preventative therapy. This integration of technology is an important step toward precision perioperative medicine.

Although the Sepsis One-Hour Bundle has been implemented worldwide, there remains a persistent gap between awareness of guidelines and clinical practice, even in patients with concomitant heart failure and complex hemodynamic instability. This population's high risk for myocardial decompensation not only requires clinical team expertise, but robust system-level support as well. It is therefore critical that, at the policy level, we invest in workforce development and structured training and provide adequate funding to improve outcomes by addressing these knowledge gaps [15]. Future practice can evolve toward an evidence-based framework for education and therapy. High-fidelity simulation can strengthen clinicians' responses to the development of hemodynamic instability, while regular audit and feedback help support the translation of guidelines into standardized practice. For example, anesthesiologists could undergo annual high-fidelity simulation training for the management of patients with sepsis and heart failure, combined with objective performance evaluation indicators and debriefing summaries. Implementation of multidisciplinary crisis resource management is also imperative to coordinate decision-making during resuscitation. Combined, these efforts enable a shift from experience-based care to quality-driven systems that enhance perioperative safety in high-risk patients.

DECLARATIONS

Author contributions

Lingxuan Tang and Qi Wang contributed to the manuscript writing and figure preparation, Lingxuan Tang designed the work and supervised the work. All authors reviewed the manuscript. All authors contributed to the article and approved the submitted version.

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Data availability

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Ethics approval and consent to participate

Not applicable. This manuscript does not contain any studies with human participants or animals performed by any of the authors.

Consent for publication

Not applicable. This manuscript does not include details, images, or videos relating to an individual person.

Competing interests

The authors declare that they have no competing interests.

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Not applicable.

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