

Diastolic Shock Index to Predict Progression into Septic Shock

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Abstract

In septic shock, hypotension is primarily caused by the failure of vascular smooth muscle to constrict, leading to systemic vasodilation and eventual diastolic dysfunction. Therefore, the diastolic shock index (DSI) may be a more appropriate measure than mean arterial pressure (MAP) or systolic shock index (SI) for quantifying the severity of septic shock. A DSI of 2.0 or greater has been associated with increased mortality and demonstrates superior predictive value compared to heart rate (HR) and systolic SI alone. The DSI is calculated as: $DSI = HR / DBP$. Its early calculation can predict progression to septic shock before hemodynamic instability manifests, thereby enabling clinicians to initiate prompt treatment for sepsis and potentially avert overt shock. Furthermore, DSI may also serve as a valuable predictor of mortality in patients with sepsis.

I. HOW TO USE

When to Use

For patients with suspected sepsis, the DSI should be calculated using vital signs obtained at triage, early in the Emergency Department (ED) visit, or upon admission. This calculation is most valuable *prior to* the administration of vasopressors or fluids, as it aims to predict progression to septic shock before hemodynamic interventions. Additionally, DSI can be trended over time to evaluate the patient's response to therapy.

Pearls / Pitfalls

Currently, there are no large, prospective studies validating the use of DSI in the ED population. It is also important to understand that an elevated DSI does not necessarily correlate with an isolated low DAP or an elevated HR. In the original study by Ospina-Tascon et al., neither isolated low DAP nor elevated HR alone showed prognostic value for septic shock outcomes; however, DSI demonstrated similar performance to lactate levels and the SOFA score in predicting mortality. One should be aware that this study omitted pediatric patients, pregnant patients, and those with atrial fibrillation or liver cirrhosis.

Furthermore, while the Jeon et al. study indicated DSI as a predictor of progression to septic shock when derived from initial vital signs in the ED, it screened patients for sepsis using qSOFA criteria, which contradicts current guideline recommendations for sepsis screening.

Lee et al. demonstrated that elevated DSI values upon ICU admission were significantly associated with in-hospital mortality among patients with septic shock and normal left ventricular ejection fraction (LVEF); however, this association was not found in those with decreased LVEF. Consequently, this study raises important questions regarding the influence of cardiac function on the applicability and interpretation of DSI.

Therefore, while DSI shows promise in sepsis care, it should currently be used as an adjunct to other established measures, rather than as a replacement for them.

Why Use

Historically, severity of shock is quantified by decreased MAP and SI, both of which rely on systolic arterial pressure as the main measure for hypotension. However, in septic shock, hypotension is primarily caused by failure of vascular smooth muscle to constrict, causing systemic vasodilation and eventual diastolic dysfunction. Therefore, it may be more appropriate to use DSI rather than MAP or systolic SI. Early calculation of DSI may predict progression to septic shock before hemodynamic instability begins, allowing the clinician to immediately treat sepsis and potentially avoid shock.

II. NEXT STEPS

Advice

DSI is an increasingly recognized hemodynamic parameter gaining traction for its potential to offer early insights

into circulatory compromise. It is a quick and non-invasive parameter that focuses on the diastolic component of cardiovascular function. In addition to predicting septic shock, DSI can provide continuous assessment of hemodynamic status that can be trended over time to evaluate response to therapy.

Diastolic parameters are most accurate when the aortic valve is competent. Conditions such as severe aortic regurgitation can pathologically lower DBP, mimicking vasodilation and affecting DSI. Similarly, baseline heart rate variability, medications, and autonomic dysfunction can influence its interpretation.

In patients with sepsis, the DSI value is not a diagnostic test and can be abnormal in a variety of conditions. Based on the available evidence, it is unclear how DSI will be used in the sequence of events from screening, diagnosis, and triggering of sepsis-related interventions. At this time, it should be used as an adjunct to lactate level and clinical picture.

Management

Step 1: Calculate the DSI using the equation

Diastolic shock index = Heart rate / Diastolic blood pressure

Step 2: Interpret the DSI value

While a universally agreed-upon cutoff value for DSI is currently lacking, a measurement of 2.0 or greater is frequently noted as a significant threshold. Any elevation in DSI should, consequently, raise concern regarding the potential for progression to septic shock. Clinicians should promptly consider initiating early, aggressive resuscitation, such as vasopressor and fluid therapy, upon observing an elevated DSI.

The management of sepsis is continually evolving and is detailed in the [Surviving Sepsis Campaign: International Guidelines for Management of Sepsis and Septic Shock 2021](#).

Critical Actions

For patients with an elevated DSI, it would be prudent to ensure initial triage has not missed the presence of sepsis. Evaluation of [SIRS criteria](#) would be beneficial. An elevated DSI in a patient suspected to have sepsis should prompt clinicians to consider initiation of aggressive sepsis management including early initiation of vasopressors, and may help identify patients requiring higher levels of care and monitoring.

DSI alone should not replace clinical gestalt or other indicators of sepsis or septic shock such as persistent SBP < 90 mmHg or MAP < 65 after fluids, altered mental status, respiratory distress or need for airway support, delayed cap refill, or elevated lactate.

Maintain a high index of suspicion for mimics of sepsis such as hemorrhagic, cardiogenic, obstructive shock or toxicologic causes. DSI does not currently apply to these other causes of shock.

This tool may underperform in certain populations. Use caution in pregnant/postpartum, immunosuppressed, and pediatric patients. Patients on beta-blockers may not produce tachycardia in shock states which can affect your interpretation of DSI.

III. EVIDENCE

Abstract

The pathophysiology of septic shock is complex and multifaceted but well understood. Septic shock is caused primarily by a failure of vascular smooth muscle to constrict, but also by hypovolemia, myocardial dysfunction, and altered blood flow distribution. Owing to the key role of systemic vasodilation in the pathophysiology of septic shock, the use of the DSI is now emerging. Calculated as HR/DBP, the DSI attempts to predict the clinical outcomes of septic shock using DBP.

A DSI greater than or equal to 2.0 has been associated with greater mortality and has shown more predictive value than heart rate (HR) and systolic SI alone. In one study, DSI showed similar performance to lactate levels and the Sequential Organ Failure Assessment (SOFA) score in predicting mortality. Early calculation of DSI may predict progression to septic shock before hemodynamic instability begins, allowing the clinician to immediately treat sepsis and potentially avoid shock.

Evidence Appraisal

The original derivation of DSI (Ospina-Tascón et al., *Annals of Intensive Care*, 2020) is a retrospective cohort study utilizing two separate patient cohorts. The first, considered the “primary cohort”, included 337 patients with septic shock requiring vasopressor support in a single ICU from a hospital in Columbia, South America. The second cohort included 424 patients with septic shock included in the recent randomized control trial, the ANDROMEDA-SHOCK trial. This cohort included patients from 28 hospitals in 5 different South American countries. Patients in the primary cohort were identified using the Surviving Sepsis Campaign: International Guidelines for Management of Severe Sepsis and Septic Shock: 2012 while those included in the ANDROMEDA-SHOCK trial were identified using The Third International Consensus Definitions for Sepsis and Septic Shock (Sepsis 3.0)

- **Population:** 761 adults with septic shock.
 - **Preliminary cohort:** 337 ICU patients with septic shock in Colombia (single academic center, Jan 2015–Feb 2017).

- **ANDROMEDA-SHOCK cohort:** 424 patients with septic shock enrolled in a multicenter RCT across 28 hospitals in 5 Latin American countries (Mar 2017–Apr 2018).
- Inclusion was septic shock; definition differed slightly by era (2012 Surviving Sepsis Campaign definition in the early cohort vs Sepsis-3 in ANDROMEDA-SHOCK)
 - just **before** vasopressors (“pre-VP DSI”) in the preliminary cohort,
 - **at vasopressor start / randomization** in ANDROMEDA-SHOCK.
 They then recalculated DSI at 2, 4, and 8 hours into resuscitation.
- **Outcomes:** 28-day and 90-day mortality, plus organ failure, vasopressor dose trajectory, fluids, etc.

They found that DSI had a higher area under the curve (AUC) than MAP and systolic SI in predicting mortality and that just before or immediately after the use of vasoactive agents, higher DSI values were associated with increased mortality rates.

Kim et al. have demonstrated a scoring model that predicts vasopressor requirement by combining the DSI and lactate levels. Their results suggest that early vasopressor use might show more benefit for improving survival in high-risk patients (DSI ≥ 2.0 and lactate ≥ 2.5 mmol/L) than in low-risk patients (DSI < 2.0 and lactate < 2.5 mmol/L). Thus, a DSI ≥ 2.0 may be a clinically useful cut-off to consider applying to patients presenting with sepsis. Authors also found that DSI and a high lactate were predictive of need for vasopressor administration among hypotensive patients with presumed infection with an area under the receiver curve (AUC) of 0.741 (95% confidence interval).

Jeon et al found an AUC of 0.717 (95% confidence interval) for DSI as a predictor of progression to septic shock when utilized on initial vital signs in the ED. In two separate validation studies using AUC, higher DSI was associated with progression to septic shock. These studies are limited as all are observational in nature and thus cannot control for unknown confounding variables.

Lee et al. demonstrated that DSI values upon ICU admission were significantly associated with in-hospital mortality in patients with septic shock and normal left LVEF, an association not observed in patients with decreased LVEF. This study raises questions about the impact of cardiac function on the utility and interpretation of DSI.

Overall, these studies should prompt the reader to think critically about the clinical and physiologic plausibility of the DSI and its utility in sepsis management. Though studies suggest a correlation between elevated DSI values and mortality, they do not provide a practical cut off that can be universally applied to patients present-

ing with sepsis. Further large-scale, multicenter, prospective research must be done to validate its clinical use.

Original Validation/Derivation study

Citation	Ospina-Tascón GA, Teboul JL, Hernandez G, Alvarez I, Sánchez-Ortiz AI, Calderón-Tapia LE, Manzano-Núñez R, Quiñones E, Madriñán-Navia HJ, Ruiz JE, Aldana JL, Bakker J. Diastolic shock index and clinical outcomes in patients with septic shock. <i>Ann Intensive Care</i> . 2020 Apr 16;10(1):41. doi: 10.1186/s13613-020-00658-8. PMID: 32296976; PMCID: PMC7160223.
Type of study	Retrospective cohort study utilizing two separate patient cohorts
Number of patients	761 patients
Inclusion criteria	Adult patients in septic shock; definition differed slightly by era (2012 Surviving Sepsis Campaign definition in the early cohort vs Sepsis-3 in ANDROMEDA-SHOCK)
Exclusion criteria	Patients <18 years old, pregnant patients, patients with liver failure (prothrombin time >15 seconds or INR > 1.5 and hepatic encephalopathy), advanced liver cirrhosis, atrial fibrillation, presence of ventricular dysrhythmia, use of pacemaker, and those with do-not-resuscitate orders. ANDROMEDA-SHOCK had additional trial-specific exclusions reported in the original RCT.
Study endpoints and findings	In the primary cohort, the DSI was calculated prior to the initiation of vasopressor therapy whereas the DSI was calculated at the randomization point for those in the ANDROMEDA-SHOCK cohort. In both populations, DSI was again calculated at the 2-hour, 4-hour, and 8-hour post vasopressor initiation. DSI levels were then distributed into 5 different quintiles. Researchers conducted a variety of different statistical analyses to calculate the relative risk of mortality associated with a given DSI quintile, as well as broke the DSI calculation into its components and studied the relationship of DSI, HR, and DAP with death at 28 and 90 days. Researchers found that increases in DSI were associated with increased risk of mortality at 90 days in both study groups. When compared to other measures of mortality in sepsis including lactate, SOFA, and APACHE II scores, DSI performance did not differ significantly. Progressive DAP decreases or HR increases were associated with higher mortality risks only when DSI concomitantly increased. It was found that early initiation of vasopressors was associated with more favorable outcomes in those with high DSI values.
Strengths	Researchers attempted to control for a variety of variables, including volume of resuscitation prior to vasopressor initiation, volume of resuscitation from vasopressor initiation to 8 hours, initial arterial lactate, and a variety of additional variables
Criticisms	Retrospective study. Study lacks external validity and generalizability, as it took place on a single continent in a select few hospital settings.

Original/Additional Validation Study

Citation	Yumin Jeon, Sungjin Kim, Sejoong Ahn, Jong-Hak Park, Hanjin Cho, Sungwoo Moon, Sukyo Lee, Predicting septic shock in patients with sepsis at emergency department triage using systolic and diastolic shock index, <i>The American Journal of Emergency Medicine</i> , Volume 78, 2024, Pages 196-201, ISSN 0735-6757
Type of study	Retrospective observational study
Number	1267 patients

of patients	
Inclusion criteria	Adult patients (> 18) who screened positive for sepsis by quick SOFA (qSOFA) criteria and had confirmed presence of infection and organ dysfunction by physician.
Exclusion criteria	Patients <18 years old, Do Not Attempt Resuscitation (DNAR) orders or who refused life-sustaining treatments that did not comply with the SSC guidelines.
Study endpoints and findings	The primary outcome was progression to septic shock defined in this study as a diagnosis of septic shock according to the Sepsis-3 definition during the ED stay. Secondary outcomes were the time to vasopressor requirement, high dose vasopressor requirement, vasopressor dose, 7-, 14-, 30-day mortality and severity according to SI and DSI ranges. The AUC for predicting progression to septic shock for DSI was 0.717 (95% confidence interval [CI], 0.688–0.747), while that for SI was 0.707 (95% CI, 0.677–0.737). The AUC for DSI and SI did not show a statistically significant difference ($p = 0.14$; DeLong et al.). The AUC for predicting progression to septic shock for DSI and SI were significantly higher than those for NEWS, NEWS2, and MEWS. Based on the Youden index , the optimal cutoff for SI was 1.05, with a sensitivity of 69.7% and specificity of 61.7%, while that for the DSI was 1.99, with a sensitivity of 54.1% and specificity of 77.8%. SI and DSI at triage were statistically significant predictors of progression to septic shock in patients with sepsis compared to other early warning scores.
Strengths	DSI is evaluated at triage, which can have clinical impact in the emergency department without invasive testing. Study also utilized data from a prospectively collected sepsis registry.
Criticisms	Retrospective observational study. Study lacks external validity and generalizability, as it took place in two hospital settings at a single tertiary center in South Korea. Study included only patients with positive qSOFA scores upon ED admission. The Surviving Sepsis Campaign guidelines do not recommend screening for sepsis using the qSOFA alone.

Citation	Lee KJ, Kim YK, Jeon K, Ko R-E, Suh GY, Oh DK, et al. (2024) Shock indices are associated with in-hospital mortality among patients with septic shock and normal left ventricular ejection fraction. PLoS ONE 19(3): e0298617. https://doi.org/10.1371/journal.pone.0298617
Type of study	Prospective cohort study
Number of patients	392 patients
Inclusion criteria	Patients at least 19 years old with septic shock requiring vasopressors and with lactate levels > 2 mmol/L, admitted to the intensive care units, and that underwent transthoracic echocardiography within 24 hours of sepsis recognition.
Exclusion criteria	< 19 years old, patients with sepsis alone, time intervals from time zero to intensive care admission exceeding 24 hours, missing data on vital signs or lactate, and initial heart rates below 60 or above 180 beats/minute.
Study endpoints and findings	Primary outcomes were the association between the SI, DSI, modified shock index (MSI), and age shock index (Age-SI) (both at time zero and at ICU admission) and in-hospital mortality rate. Secondary

	outcomes were changes of the indices according to the completion of 3-h bundle components, and the association between the indices and ICU and 30-day mortality rates. The SI, DSI, MSI, Age-SI at ICU admission were significantly associated with in-hospital mortality in patients with septic shock and normal LVEF, which was not found in those with decreased LVEF. In multivariable analysis, each of the four indices at ICU admission was significantly associated with in-hospital mortality ($P < 0.05$) among patients with normal LVEF. This association was stronger for patients with lower lactate levels (< 4.0 mmol/L), compared to those with higher lactate levels. The results also favor that that evaluating shock indices after early sepsis resuscitation, rather than at time zero, may be more valuable in cases of septic shock.
Strengths	Prospective, multicenter cohort study, across 20 hospitals. First study to look at differences between normal and decreased EF.
Criticisms	Smaller sample size with initial exclusion of a large proportion of patients

Further research is needed into DSI to further determine its efficacy, predictive value, and to compare it to other standard measurements of sepsis.

Formula

Diastolic shock index = Heart rate / Diastolic blood pressure

Literature

Ospina-Tascón GA, Teboul JL, Hernandez G, Alvarez I, Sánchez-Ortiz AI, Calderón-Tapia LE, Manzano-Nunez R, Quiñones E, Madriñán-Navia HJ, Ruiz JE, Aldana JL, Bakker J. Diastolic shock index and clinical outcomes in patients with septic shock. *Ann Intensive Care*. 2020 Apr 16;10(1):41. doi: 10.1186/s13613-020-00658-8.

Jeon Y, Kim S, Ahn S, Park JH, Cho H, Moon S, Lee S. Predicting septic shock in patients with sepsis at emergency department triage using systolic and diastolic shock index. *Am J Emerg Med*. 2024;78:196-201. doi:10.1016/j.ajem.2024.01.028.

D.S. Kim, J.E. Park, S.Y. Hwang, D. Jeong, G.T. Lee, T. Kim, *et al.* Prediction of vasopressor requirement among hypotensive patients with suspected infection: usefulness of diastolic shock index and lactate. *Clin Exper Emerg Med*, 9 (2022), pp. 176-186, [10.15441/ceem.22.324](https://doi.org/10.15441/ceem.22.324)

Lee KJ, Kim YK, Jeon K, Ko R-E, Suh GY, Oh DK, et al. (2024) Shock indices are associated with in-hospital mortality among patients with septic shock and normal left ventricular ejection fraction. PLoS ONE 19(3): e0298617. <https://doi.org/10.1371/journal.pone.0298617>