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REVIEW
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Diastolic shock index: Its importance and application in critically ill patients: A narrative review

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ABSTRACT:

A recently developed method to measure vasodilatation is the diastolic shock index (DSI), which can be calculated by dividing heart rate by diastolic blood pressure. The DSI plays a significant role in many medical conditions. The focus of this review is to determine the evidence-based data of diastolic shock index in various conditions. Current trials recommend adding norepinephrine when diastolic arterial pressure is below 40 mmHg or diastolic shock index is more than 3. Besides, recent trials have studied the diastolic shock index in myocardial infarction, the peri-intubation period, the intraoperative period, and emergency department triage. Higher diastolic shock index value at presentation of severe cases of sepsis could identify patients who might benefit from early vasopressors and predict the progression of septic shock in emergency department triage. Moreover, it could help as a tool to identify a higher risk of death in myocardial infarction and peri-intubation period hypotension. However, the cut-off points for the diastolic shock index vary across different conditions.

Keywords: Diastolic shock index; Septic shock; Diastolic blood pressure

INTRODUCTION

Hemodynamic change depends on various conditions, including preload, cardiac function, and vascular tone. For assessment of vascular tone, it involves evaluating the constriction or dilation of blood vessels. A diverse range of methods, including dynamic arterial elastance (Eadyn), systemic vascular resistance (SVR), diastolic blood pressure (DBP), and the diastolic shock index (DSI), are available for assessing vascular tone.

The most straightforward parameter that is easy to measure vascular tone and can be done on a regular basis is DBP. The French Intensive Care Society recently defined life-threatening hypotension as a DBP of less than 40 mmHg [1]. It has been demonstrated that increasing preload and contractility by using DBP < 40 mmHg as a trigger for early norepinephrine delivery improves cardiac output [2]. These results emphasize how early management can be guided by using DBP for vascular tone assessment and management.

On the basis of physiology, however, DBP represents both the length of the cardiac cycle and vascular tone. When arterial pressure drops, the sympathetic nervous system is activated, leading to tachycardia. With an increased heart rate, diastolic time shortens, which can cause an elevation in DBP. Consequently, a patient with tachycardia may have a falsely elevated DBP if the severity of vasodilation is rapidly assessed using this measurement. A set DBP target of 40 mmHg

may not be the ideal choice because of its dependence on heart rate, highlighting the need for more dynamic and comprehensive measures.

DIASTOLIC SHOCK INDEX DEFINITION

Diastolic shock index (DSI) is calculated by dividing the heart rate by the diastolic blood pressure [3]. Under normal circumstances without aortic insufficiency, the DBP is mainly determined by vascular tone, and it remains nearly identical from the center to the periphery. However, DBP should not be evaluated separately from the heart rate (HR) because acute reduction in arterial pressure and vascular tone is often counterbalanced by increasing sympathetic activity. Normally, DBP is associated with HR. An increase in HR can result in higher DBP because of the shorter diastolic time. As a result, in pathological conditions, concurrent and divergent variations in the DBP and HR, such as a progressively declining DBP alongside increasingly severe tachycardia, may indicate more profound cardiovascular dysfunction, with shorter diastolic times unable to compensate for a DBP decrease as a consequence of the progressive loss of vascular tone. To address the drawbacks of DBP, a DBP-corrected HR index, such as DSI, was suggested. Higher DSI (high HR and low DBP) may be a sign of decreased vascular tone. Nevertheless, there was little information about the proper cut point utilizing DSI.

Ospina-Tascón and colleagues [3] were the first to introduce DSI. In a retrospective analysis of patients in septic shock, they demonstrated that DSI at pre-vasopressor and vasopressor start points may be a very early indicator of patients who are at a high risk of mortality, independent of solo DAP or HR measurements. They suggested that DSI > 2.2 might be used to start or direct therapeutic interventions in early septic shock resuscitation. Additional information on DSI in different populations has been gathered based on its physiology and published data on septic shock patients. This information will be covered in the following sections.

EVIDENCE OF DSI AND OUTCOMES

The use of the diastolic shock index is supported by the available data. The DSI cut-off point in a variety of populations, including sepsis, myocardial infarction, pre-intubation, intraoperative, and emergency department triage, is supported by a number of different pieces of data. Table 1 summarized the DSI and clinical outcome investigations.

Sepsis

Hypotension in sepsis is the result of vasodilatation, relative hypovolemia, myocardial dysfunction, and altered blood flow distribution. The initial resuscitation in septic shock includes three components: (1) assess fluid responsiveness; (2) check cardiac function; and (3) determine the necessity for a vasopressor after vascular tone has been assessed. Titrating an appropriate vasopressor during early resuscitation is crucial to reducing fluid overload complications and improving mortality. The use of

KEY MESSAGES:

- A new vasodilatation metric called the diastolic shock index (DSI) is derived by dividing the heart rate by diastolic blood pressure.
- When severe cases of sepsis are presented, a higher DSI value may help determine which patients might benefit from early vasopressor use and may also help in emergency department triage by predicting the course of septic shock.
- The cut-off points for the diastolic shock index vary across different conditions.

DSI for vascular tone assessment was initially presented by Ospina-Tascón GA, et al. in 2020. This retrospective cohort analysis demonstrated that mortality was linked to concurrently elevated DSI. Vasopressor dosages and resuscitation fluid were much greater in those with high DSI. The first DSI before initiating vasopressor was significantly higher in non-survivors than in survivors (2.2 vs. 2.0, p=0.001). Therefore, they came to the conclusion that DSI before or at the onset of vasopressors might identify patients in septic shock who were at a high risk of mortality and trigger early therapeutic interventions.

Kim et al. predicted the need for vasopressors in patients with suspected infections and hypotensive episodes using the DSI and lactate levels. The high DSI (≥2.0) and high lactate (≥2.5 mmol/L) criteria were given one point each in the study's score [4]. An early start of vasopressor administration (less than 30 minutes or between 30 and 60 minutes) was significantly associated with increased 28-day mortality (AOR 7.28, 95% CI [1.43–37.10], p=0.017 or AOR 4.76, 95% CI [1.07–21.14], p=0.040), but an hourly delay of vasopressor administration was not associated with 28-day mortality (a score of 0 points). On the contrary, with a score of 2 points, both an hourly delay of vasopressor administration (AOR 1.06, 95% CI [1.01–1.11], p=0.021) and an early start (<30 minutes) of vasopressor administration (AOR 0.37, 95% CI [0.14–0.94], p=0.038) were significantly associated with 28-day mortality.

Furthermore, Monnet et al. proposed a flowchart for deciding when to early administer norepinephrine [5]. Because it should be used in patients with very low arterial tone. Three important criteria for life-threatening hypotension were noted: (1) diastolic arterial pressure ≤40 mmHg; (2) diastolic shock index ≥3; and (3) high risk of fluid overload. Indeed, the decision to start vasopressors early, along with fluid therapy, seems logical to initiate vasopressors when DAP is very low, such as <45 mmHg or DSI>2 [6-7].

In addition, Lee et al. investigated the association between the in-hospital mortality rate at time zero and at ICU admission and the shock index (SI), diastolic shock index (DSI), modified shock index (MSI), and age shock index (Age-SI) [8]. Multivariable analysis revealed that

Table 1. Summary of trials that studied the association between diastolic shock index and clinical outcomes in different populations.

Author	N	Year	Population	Cut-off point of DSI	Primary outcome	Major Findings
Ospina-Tascón GA, et al.	761	2022	Adult patients with sepsis requiring vasopressor support	≥2.2	90-days mortality	There was an association between increased diastolic shock index score and mortality at 90 days (AUC of 0.690 (95% CI, 0.633 0.748)
Kim et al.	1,917	2018-2019	Adult patients with suspected infection and hypotension in the ED	high DSI (≥2.0) and high lactate (≥2.5 mmol/L)	28-day mortality	Early vasopressor therapy initiation was significantly associated with decreased 28-day mortality (adjusted odds ratio, 0.37; 95% CI, 0.14–0.94)
Kyu Jin Lee	246	2019-2021	Critically ill patients with septic shock requiring vasopressors and with lactate levels > 2 mmol/L and normal LVEF	≥2.2	In-hospital mortality rate	The SI, DSI, MSI, and Age-SI at ICU admission were significantly associated with in-hospital mortality in patients with septic shock and normal LVEF (AUC of 0.771 (95% CI, 0.656 0.885)
Ana Karen Del Socorro Arévalo-Coronado et al.	162	2022	STEMI Mexican population in the ED	> 1.2143	Mortality rate in acute myocardial infarction with ST segment elevation prior to admission to the Hemodynamics Room	Mortality rate after procedure sensitivity of 62.5% and a specificity of 77.4% with a p < 0.05 (ROC 0.67) PPV 28.84% NPV91.81%
Nathan J. et al.	151	2014-2017	Critically ill patients undergoing emergent endotracheal intubation	≥2.0	Peri-intubation hypotension and worse SOFA scores	Peri-intubation hypotension and worse SOFA scores
Indraratna	25	2024	Cardiac surgery patients	≥2.2	Relationship between the coupling ratio and the DSI	No relationship between the coupling ratio and the DSI
JeonY. et al.	1,267	2016-2022	Patients met the quick SOFA (qSOFA) criteria for ED admission	≥1.99	Progression to septic shock	DSI were significant predictors of progression to septic shock. (AUC of 0.717 (95% CI, 0.688 0.747), sensitivity of 54.1% and specificity of 77.8%

all four of the indices at ICU admission were significantly associated with in-hospital mortality ($p < 0.05$) for patients with normal LVEF; however, the indices for patients with lower lactate levels (≤4.0 mmol/L) had better discrimination power than those with higher lactate levels. Additionally, this study indicated that in septic shock, it would be more beneficial to assess shock indices following early sepsis resuscitation rather than at time zero [8].

ED TRIAGE for septic shock

Triage at an Emergency Department (ED) should be straightforward and effective. Jeon Y. et al. conducted a study aimed to assess systolic shock index (SI) and diastolic shock index (DSI) as predictors of progression to septic shock. SI was defined as HR/SBP, while DSI was defined as HR/DBP [9]. The results showed that the optimal cut-off 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%. The AUC for predicting progression to septic shock for DSI and SI was significantly higher than those for NEWS, NEWS2, and MEWS. Likewise, Zhang et al. study found that prevasopressor SI, MSI, and DSI could identify patients with septic shock requiring vasopressors who are at increased risk of early death [10].

PERI-INTUBATION PERIOD

Based on recent evidence, there appears to be an association between peri-intubation hypotension and patient morbidity and mortality [11-12]. Peri-intubation hypotension remains the most common complication during

airway management procedures. Thus, prevention strategies may improve patient outcomes.

In order to identify shock indices like the shock index (SI), modified shock index (MSI), and diastolic shock index (DSI) as potential pre-intubation risk factors for peri-intubation hypotension, Nathan J. et al. conducted a prospective, single center, multi-unit, randomized, parallel-group clinical study [13]. They comprised patients undergoing urgent endotracheal intubation in the medical, surgical, oncologic, and transplant intensive care units. This trial defined peri-intubation hypotension as a decrease at any point in mean arterial pressure less than 65 mmHg and/or a reduction in mean arterial pressure of 40% from baseline. The results showed that increasing SI, MSI, and DSI were significantly associated with worse sequential organ failure assessment (SOFA) scores with elevated SI ≥1.0, MSI ≥1.3, DSI ≥2 (Spearman rank correlation: $r = 0.30$, $r = 0.40$, and $r = 0.45$ for SI, MSI, and DSI, respectively, all $p < 0.001$) but not with other outcomes.

INTRA-OPERATIVE PERIOD

Typically, transesophageal echocardiography was used to measure the ventricular-arterial coupling ratio in order to assess vasodilatation and manage hemodynamics. Transesophageal echocardiograms, however, need skill and ongoing monitoring. In 2024, a trial of 25 measurements of cardiac surgery patients was taken to compare transesophageal echocardiogram and diastolic shock index as validity in cardiac surgery [14]. The results showed no relationship between the coupling ratio and the DSI ≥2.2. Therefore, the DSI cannot be used to guide therapy in the presence of low blood pressure during cardiac surgery.

Myocardial infarction

About 5 to 15% of people who have an acute ST-segment elevation myocardial infarction (STEMI) have cardiogenic shock. It is still the primary cause of death for patients with STEMI, even with advancements in treatment. The shock index at admission has been used as a predictor of major adverse cardiac events in patients with STEMI. According to a multicenter study conducted by Reinstadler et al., shock index was associated with more severe cardiac microvascular and myocardial damage as determined by cardiac magnetic resonance imaging [15].

The diastolic shock index data for this particular patient population was scarce. The severity of myocardial patients may be influenced by a number of mechanisms: (1) in patients who were previously hypotensive, the decrease in diastolic arterial pressure would lead to impaired coronary artery perfusion; (2) in patients who were previously hypotensive, the decrease in arterial pressure also reduces left ventricular afterload, which may be the ventricle's physiological response to a decreased load, involving decreased phosphorylation of the calcium channels between the transverse tubules and the sarcolemma, resulting in a subsided intrinsic contractility [16]. Only one study has examined the relationship between the DSI and MI, revealing an intriguing association and proposing a distinct cut-off value. Arévalo-Coronado et al. conducted a cross-sectional study involving 162 patients with acute ST-elevation myocardial infarction who underwent cardiac catheterization. Their findings indicated that a DSI >1.2143 was associated with increased mortality, with a sensitivity of 62.5%, a specificity of 77.4%, $p < 0.05$ (95% CI [0.59–0.74]), and an area under the curve (AUC) of 0.67 [17].

Aortic insufficiency

Aortic insufficiency (AI) involves diastolic blood reflux from the aorta into the left ventricle (LV). In acute AI, LV diastolic pressure rises rapidly, end-diastolic pressure becomes markedly elevated, and the mitral valve may close prematurely. DBP is often low with minimal pulse pressure widening, and severe cases can lead to reduced cardiac output and hypotension. In chronic AI, LV remodeling accommodates regurgitant flow, increasing stroke volume to sustain forward flow. This results in a dilated LV, widened pulse pressure, and low DBP, characteristic of chronic AI.

The hemodynamic shift in AI, specifically in DBP and DSI, limits the utility of DSI to predict vascular tone. Applying this metric to this specific case is challenging. AI was not excluded from the majority of previously published studies on DSI, and aortic valve evaluation was not mentioned either. Further studies are required to assess the application of DSI in AI and its appropriate cut-off point.

CONCLUSION

Hemodynamic changes depend on various factors, including preload, cardiac function, and vascular tone. One method to assess vascular tone is the diastolic shock index, calculated by dividing the heart rate by the diastolic blood pressure. DSI is a useful tool in various conditions: (1) A user-friendly bedside calculating index; (2) Helping the management team identify sepsis patients with high DSI who are more likely to require the administration of vasopressors; (3) Being a predictor for mortality in ST segment elevation myocardial infarction and hypotension during peri-intubation. However, there are some limitations we need to address: (1) In aortic insufficiency, the interpretation of DSI is limited; (2) DSI cannot be used to guide therapy in the presence of low blood pressure during cardiac surgery; (3) the cut-off points for DSI vary across different conditions. Further large-scale studies are needed to determine the appropriate cut-off points for DSI in various conditions.

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