



The Diastolic Shock Index in Septic shock

Key Articles

- *Ospina-tascón GA, Teboul JL, Hernandez G, et al. Diastolic shock index and clinical outcomes in patients with septic shock. Ann Intensive Care. 2020;10(1):41.*

Background

- Identification of shock often includes abnormal hemodynamics with associated impairments in tissue perfusion and cellular hypoxia
- There is a close relationship between blood [pressure] and [flow], especially early on in the patient's critical illness.
- Traditional clinical judgements about shock include changes in MAP and/or systolic blood pressure, but clinical interpretation is often more nuanced than an isolated or individual number.
- Shock indices have been proposed to incorporate a blood pressure and heart rate to determine patient's compensatory response to their critical illness, but each have limitations.
- This past month, the diastolic shock index was proposed as a tool to be used as an interventional trigger for vasopressors in septic shock.

The original "Shock Index" and Interpreting the BP during circulatory shock

- "Shock index" has historically defined as the [HR] / [Systolic BP]
 - SI > 0.8: Associated with a 95% sensitivity of predicting shock, post-intubation hypotension, and need for massive transfusion in trauma. (Rady et al. Ann Emerg Med 1994; Heffner et al. Resuscitation 2013; Schroll et al. Injury. 2018)
 - SI > 1.2 after fluid resuscitation associated with increased use of vasopressor use during the first 24 hours of resuscitation.
- Alterations in the pulse pressure could grossly mirror the underlying mechanism of acute circulatory failure
- Systolic blood pressure
 - SBP is particularly important in *hypovolemic* (hemorrhagic) and *cardiogenic* shock
 - In early stages, SBP will fall, while the diastolic arterial BP tends to stay the same
 - Left ventricular stroke volume decreases, causing the pulse pressure to narrow, and heart rate to increase to maintain cardiac output
- This is why the original shock index has been so helpful for predicting the severity of traumatic illness and peri-intubation risk.

Why is the systolic Shock Index less sensitive in sepsis?

- In healthy patients, DBP is determined by vascular tone and remains relatively constant from the proximal to distal peripheral blood vessels
- Hypotension in sepsis is the result of vasodilation, relative hypovolemia (with some absolute hypovolemia due to capillary leak), myocardial dysfunction, and altered blood flow distribution.
- Vascular smooth muscle fails to constrict to maintain systemic vascular resistance
- As a result, cardiac output increases but often cannot produce enough flow to adequately perfuse tissues
- In septic shock, diastolic arterial pressure better reflects vasodilation than systolic or mean arterial pressure
- It is critical to consider the severity of the patient's *diastolic* hypotension as it is a better reflection of the loss of vascular tone.
- There is some evidence for this
 - Diastolic BP < 40 classified as life threatening hypotension by the French Intensive Care Society (Pottecher T et al. Crit Care, 2006)
 - Using DBP < 40 as a vasopressor trigger, early norepi administration increased cardiac output (via increased cardiac preload and contractility) (Hamzaoui O et al. Crit Care, 2010)
- **Authors question: Could a tool that rapidly assesses the severity of vasodilation influence therapeutic decisions, such as the initiation of early vasopressors to restore tissue perfusion, and reduce unnecessary fluid resuscitation?**
- **Author's hypothesis: Very early Diastolic Shock Index values could promptly identify patients could trigger very early Norepi administration as well as identify those at high risk for poor outcome**

The Study

- Population: 761 patients with sepsis requiring vasopressor support in 29 South American Hospitals (424 patients were included from ANDROMEDA-SHOCK trial) between 2015 – 2017
- Methods
 - Sepsis defined by SEPSIS-2 and SEPSIS-3 criteria (changed during enrollment period)
 - Excluded patients with advanced liver failure, chronic atrial fibrillation, patients with a pacemaker, and DNR patients
 - **DSI definition:** HR/DAP registered immediately before the start of vasopressors, or at ANDROMEDA-SHOCK randomization then calculated 2, 4, and 8 hours after Norepi Initiation
 - **Note: Most of the BP measurements were non-invasive BP cuff measurements**
- Results
 - Progressive increases in pre-vasopressor DSI were related with progressive increases in the relative risk of death

- Unlike Diastolic Shock Indices, mean arterial pressure, isolated diastolic BP, and the systolic shock index had poor performance for 28-day, 90-day, and organ injury scores
- Very early start of norepinephrine (started within the first hour of the first fluid load for resuscitation) was related with lower mortality in the higher DSI Groups.
- Is there “a number” to be concerned about?
 - Answer:
 - DSI > 2 on presentation was common in survivors, > 2.5 in non-survivors
 - DSI often failed to improve between the start of pressors and subsequent measurements at 2-4-and 8 hours in non-survivors

Interpretation

- 4 important findings:
 - A progressively higher DSI value before or at the start of vasopressors are associated with an increase in risk of death for patients with septic shock.
 - Isolated low DBP or high HR values DO NOT clearly identify high risk patients
 - Non-survivors receive/require more vasopressors and fluids than survivors
 - Pre-vasopressor DSI showed similar performance to SOFA score and initial lactate levels to predict mortality, while mean arterial pressure and systolic shock index did not
- DSI depicted a similar AUC–ROC than SOFA score and initial lactate, but could add some practical and valuable information about how to intervene the initial hemodynamic condition in sepsis
- DSI should not be interpreted as “another index of death”. Instead, a higher DSI value at presentation of severe cases of sepsis could identify patients who might benefit from early vasopressors