The Diastolic Shock Index in Septic shock

Key Articles

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 > 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:
  - 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 who 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 is 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.