The utility of the shock index in patients with acute respiratory failure

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Clinicians use vital signs to make diagnoses, to identify abnormal pathophysiologic states, and to monitor responses to treatment. Numerical records of respiratory rates, pulse rates, and blood pressures provide immediate information about the severity of various acute presentations and often directs attention to therapeutic interventions. In addition, vital signs are frequently used in risk scoring tools, including tools for pneumonia, gastrointestinal bleeding, acute myocardial infarction, and congestive heart failure.[1] The shock index (heart rate divided by systolic blood pressure) provides an overall integrated index of cardiovascular status.[2] It has been used in outpatient triage of trauma patients, in emergency departments to assist in decisions regarding patient disposition, and in prognostic estimates of both short and long-term outcomes.[3-5] The modified shock index is the heart rate divided by the mean blood pressure and includes information based on both systolic and diastolic blood pressures; the age-adjusted shock index incorporates age into this calculation (age times the shock index) to adjust for possible change in the cardiovascular responses associated with age.[3] This editorial will briefly consider the use of the shock index in patients requiring emergent intubation in intensive care units and emergency departments.

The shock index has normal values which range from 0.5-0.7 beats per minute per mmHg. Some investigators have defined an abnormal shock index as one that is ≥ 0.7; other investigators have defined an abnormal index as ≥1.0. Trivedi et al retrospectively studied the pre-intubation shock index in patients requiring emergent intubation who were apparently hemodynamically stable (systolic blood pressure>90mmHg, mean arterial blood pressure >65mmHg, and no vasopressors support within 60 minutes before the intubation).[6] This study included 140 adult patients in an intensive care unit. A pre-intubation shock index ≥ 0.90 had a significant association with post-intubation hypotension defined by systolic blood pressure of less than 90 mmHg within 60 minutes in univariate analysis (odds ratio: 2.13, 95% CI: 1.07-4.35) and multivariate analysis (odds ratio: 3.17, 95%CI: 1.36-7.73). It was also associated with higher ICU mortality rates. However, there was no association between pre-intubation shock index and ICU length of stay or 30 day mortality. There was no association found between the modified shock index and these outcomes. Heffner reported a retrospective study of 300 patients who underwent emergent intubation in a large emergency department over a 1 year period.[7] Sixty-six patients (22%) developed post-intubation hypotension defined as a systolic blood pressure ≤ 90 mmHg within 60 minutes of intubation. Multiple logistic regression analysis of variables in this study demonstrated that the pre-intubation shock index (≥ 0.8), a lower mean systolic blood pressure immediately prior to intubation, chronic renal disease, intubation for acute respiratory failure, age, and chronic use of β-blockers were independently associated with post-intubation hypotension. An elevated shock index had a sensitivity of 67% and a specificity of 80% in identifying these patients.

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Green et al studied the incidence, risk factors, and outcomes in patients with post-intubation hemodynamic instability in an emergency department.[8] They determined that age, chronic obstructive pulmonary disease, and pre-emergent endotracheal intubation hemodynamic instability were associated with the development of post-intubation hemodynamic instability. Patients with post-intubation hemodynamic instability had an increased mortality rates in the emergency department and in the hospital and had longer lengths of stay in the hospital. Heffner did a similar study and also reported that post-intubation hypotension was associated with increased mortality and length of stay.[9]

These studies suggest the pre-intubation shock index is an easy and simple tool to help clinicians predict post-intubation cardiovascular instability and to take action to minimize this outcome in emergent situations. Additionally, these studies demonstrate an association between post-intubation hypotension and in-hospital mortality. This could be explained by the fact that these patients are already critically ill and at increased risk for death and that hypotension induced during intubation causes more organ dysfunction and increases the likelihood of poor outcomes. These patients may benefit from reduced doses of sedative medications during intubation, pre-intubation volume expansion, or plans for rapid introduction of vasopressors during the post-intubation period. Clinicians should limit the intrathoracic pressures in these patients during the setup of mechanical ventilation with attention to tidal volume, peak pressures, and plateau pressures. In particular, the possibility of auto PEEP in these patients represents an important concern. In summary, clinician should make regular use of readily available vital sign information when evaluating patients during intubation and mechanical ventilation. Prospective studies are needed to determine the frequency of post intubation hypotension, risk factors for this complication, pre-emptive treatment strategies, and outcomes.

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