Introduction

Right ventricular and diastolic volume index (RVEDVI) and brain natriuretic peptide (BNP) reflect cardiac preload and myocardial stretch, respectively. RVEDVI can be measured using a modified pulmonary artery catheter with an accuracy comparable to transesophageal echo. Compared to wedge pressure, RVEDVI better correlates with cardiac index in assessing preload in trauma, surgical, and shock patients. ICU patients with RVEDVI < 100 ml/m² have been shown to have a positive hemodynamic response to fluid resuscitation whereas those with values > 150 ml/m² do not. Trauma patients volume-resuscitated to RVEDVI of 120 ml/m² have increased visceral perfusion compared to patients supported on pressures to reach blood pressure goals.

Brain, or B-type, natriuretic peptide is produced in the ventricles in response to stretching of myocytes and results in diuresis. In the acutely dyspneic patient, a BNP level of less than 100 pg/ml is used to rule out congestive heart failure as a cause. Patients with BNP > 500 pg/ml have severe sepsis or septic shock, Five had ARDS, and three had cardiovascular collapse due to gastrointestinal bleeding, intrapleural myocardial infarction, and massive pulmonary embolus. Blood volume did not correlate with either RVEDVI (r=0.069, R²=0.048) or BNP (r=0.241, R²=0.052). BNP correlated with RVEDVI (r=0.678, R²=0.460, p<0.001). Sixteen of 62 measurements showed RVEDVI ≥ 120 ml/m² in conjunction with hypovolemia (20%). Eleven percent of measurement involved hypovolemia matching a BNP > 500 pg/ml.

Methods

Patients were enrolled into the study within twenty-four hours of pulmonary artery catheter placement. Admission criteria included sepsis or septic shock, surgical patients requiring pressor support, and patients with ARDS. Exclusion criteria included pregnancy, GCS < 12, age < 18, and non-survivable conditions. Plasma volume was measured using the BVA-100 (Daxor, New York, NY). Blood volume (plasma volume + red cell volume) was calculated from the hematocrit (red cell volume/blood volume). Results are reported as a percentage deviation from that expected based on the patient’s height and weight. RVEDVI was recorded at the time of the blood volume measurement and BNP was measured daily. Data were collected on days 1, 2, and 5 of enrollment plus an additional measurement on day 5 to 7. For those critically ill patients with vascular volume expansion, hypovolemia was defined as any value less than or equal to +8% deviation from ideal blood volume. The upper limit of fluid resuscitation was considered to be an RVEDVI of 130 ml/m² and a BNP of ≤ 500 pg/ml.

Results

Twenty-three patients contributed 82 data points. The average age (standard deviation) was 59±19 years. The mean apical score was 23±5. Fifteen patients have severe sepsis or septic shock, Five had ARDS, and three had cardiovascular collapse due to gastrointestinal bleeding, intrapleural myocardial infarction, and massive pulmonary embolus. Blood volume did not correlate with either RVEDVI (r=0.069, R²=0.048) or BNP (r=0.241, R²=0.052). BNP correlated with RVEDVI (r=0.678, R²=0.460, p<0.001). Sixteen of 62 measurements showed RVEDVI ≥ 120 ml/m² in conjunction with hypovolemia (20%). Eleven percent of measurement involved hypovolemia matching a BNP > 500 pg/ml.

Conclusions

There was no relationship between blood volume and RVEDVI or BNP. RVEDVI may be an accurate measurement of cardiac preload, but volume of the heart does not necessarily reflect that in the periphery. Other measures of pressure such as the central venous and pulmonary capillary wedge pressures have also shown poor correlation with blood volume. Resuscitation to a goal RVEDVI may not be adequate in a subset of patients. BNP has utility in evaluating the dyspneic patient in the ER, but high levels should not be interpreted as a need for diuresis. Factors other than fluid overload or CHF can cause BNP to be elevated. These include septic shock, ARDS, pulmonary embolism, renal failure, and subarachnoid hemorrhage—combining findings in the ICU patient.

References

Ong, SK, et al., Cardiac preload, splanchnic perfusion, and their relationship during resuscitation in trauma patients. J Trauma, 1997;43(3): p.537