Original Research Paper

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Emergency Medicine

HEMODYNAMIC CHANGES IN PATIENTS TREATED FOR HYPOVOLEMIA

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ABSTRACT **INTRODUCTION:** Traumatic causes can result from penetrating and blunt trauma. Common traumatic injuries that can result in hemorrhagic shock include the following: myocardial laceration and rupture, major vessel laceration, solid abdominal organ injury, pelvic and femoral fractures, and scalp lacerations. Every individual in the world is at risk for traumatic injury. **METHODOLOGY:**The study was conducted in 50 patients and 50 controls who visited the Emergency Department. This is a prospective comparative study conducted in the adult Emergency Department. **RESULTS:** Heart rate was more in the pre-fluid resuscitation with a mean of 114.40 compared to the 100.30 in the post-fluid resuscitation group **CONCLUSION:** Systolic blood pressure was lesser in the pre-fluid resuscitation with a mean of 76.84 compared to the 101.08 in the post-fluid resuscitation group

KEYWORDS : Hypovolemia, Hemodynamic Changes, Heart rate

INTRODUCTION:

Hypovolemia is a condition characterized by significant decrease in the blood volume. It occurs when there is excessive external or internal blood loss. A defined volume is difficult to measure in most situations, and the loss evaluated visually is often underestimated. Hypovolemia if extensive leads to hypovolemic shock or hemorrhagic shock.Shock occurs when there is hypo-perfusion of vital organs.Hypovolemic shockis a medical emergency that is frequently encountered by physicians in emergency rooms, operating rooms, and intensive care units.[1] Significant loss of intravascular volume may lead sequentially to hemodynamic instability, decreased tissue perfusion, cellular hypoxia, organ damage, and death.

Traumatic causes can result from penetrating and blunt trauma. Common traumatic injuries that can result in hemorrhagic shock include the following: myocardial laceration and rupture, major vessel laceration, solid abdominal organ injury, pelvic and femoral fractures, and scalp lacerations.Every individual in the world is at risk for traumatic injury. The etiologies of injury are as diverse as the lifestyles and socioeconomic backgrounds of its victims, ranging from interpersonal violence and terrorism to motor vehicle crashes and occupational accidents. Worldwide, an estimated 5 million people died as a result of injury in 2000, with a mortality rate of 83 per 100,000 of the population.[2] Injury represented 9% of worldwide deaths and 12% of the burden of disease. More than 90% of injury-related fatalities occur in low- and middle-income nations. The highest mortality rates from injury occur in the less wealthy nations in Eastern Europe, with the lowest rates in North America, Western Europe, China, Japan, and Australia. Globally, road traffic injuries result in 1.2 million deaths per year, with an additional 20–50 million injuries. They rank as the 11th leading cause of death overall, accounting for 2.1% of all deaths worldwide and 25% of injury-related deaths. The majority of traffic injuries and fatalities occur in low- to middle-income nations, with some of the highest fatality rates found in European nations.[3] Violence is a large contributor to injury-related fatality as well, with 1.6 million deaths worldwide in 2000, representing 16% of mortality from injury. It is by far the leading cause of death among those aged 15–44 years and is much more prevalent in low- to middle-income countries. Self-inflicted violence represents 16% of injury-related mortality worldwide, with falls and burns accounting for 6% and 5%, respectively.[4]

METHODOLOGY:

The study was conducted in 50 patients and 50 controls who visited the Emergency Department. This is a prospective comparative study conducted in the adult Emergency Department.

- **INCLUSION CRITERIA:**
- Adult patients more than 16 years of age
- Clinical features of hypovolemia
- dry mucosa, reduced skin elasticity, cool extremities
- lengthened capillary refill times
- tachycardia
- reduced urine output
- orthostatichypotension, and fatigue;
- patients in whom hypovolemia is anticipated (such as abnormal uterine bleeding, gastrointestinal bleeding,diarrhea, and vomiting)

- **EXCLUSION CRITERIA:**
- Measurements could not be performed because of technical and anatomical reasons
- obesity
- excessive abdominal gas
- Patients who had
- tricuspid failure
- right-sided heart disease
- portal hypertension
- obstructive lung disease
- Intubated patients

RESULTS:

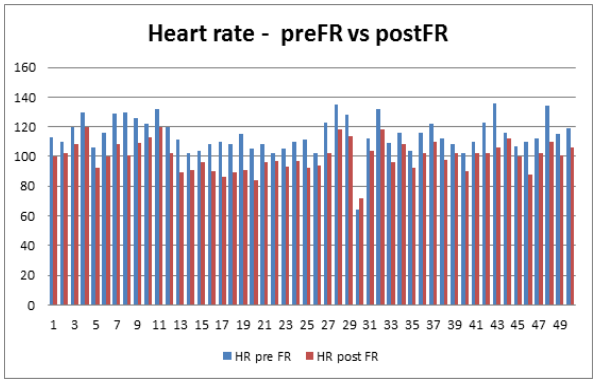

Chart 1 – Bar chart showing heart rate (beats per minute) in patients (n=50) before (HR preFR) and after (HR postFR) fluid resuscitation

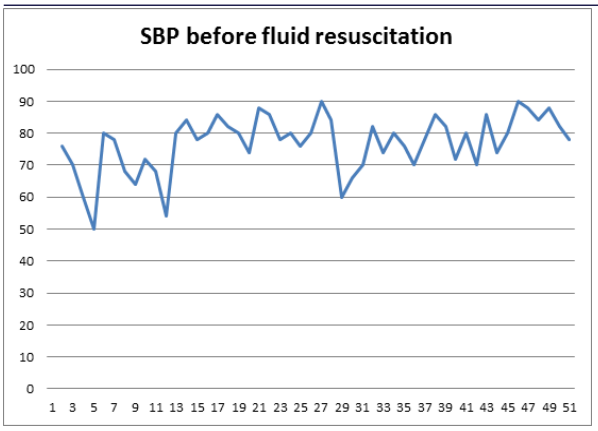

Chart 2 – Line chart showing (SBP) Systolic blood pressure (mm Hg) in patients (n=50) before fluid resuscitation

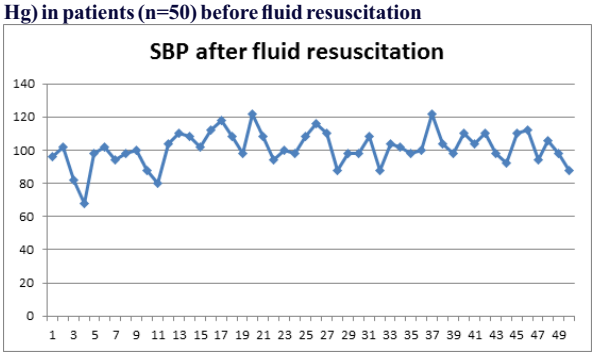

Chart 3 – Line chart showing (SBP) Systolic blood pressure (mm Hg) in patients (n=50) after fluid resuscitation

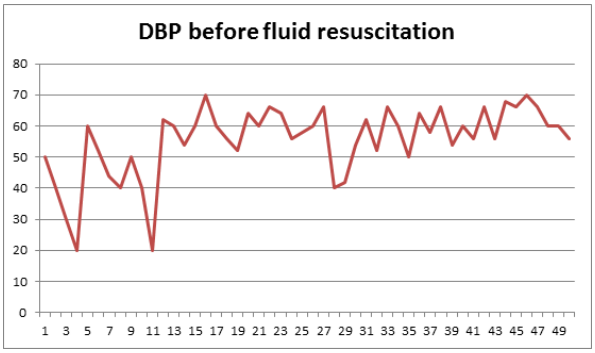

Chart 4 – Line chart showing (DBP) Diastolic blood pressure (mm Hg) in patients (n=50) before fluid resuscitation

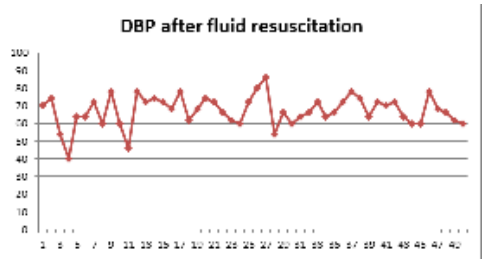

Chart 5 – Line chart showing (DBP) Diastolic blood pressure (mm Hg) in patients (n=50) after fluid resuscitation

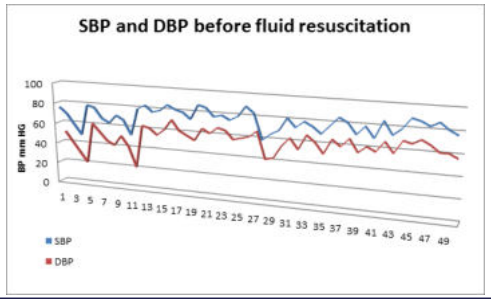

Chart 6 – Line chart showing (SBP) Systolic blood pressure (mm Hg) & (DBP) Diastolic blood pressure (mm Hg) in patients (n=50) before fluid resuscitation

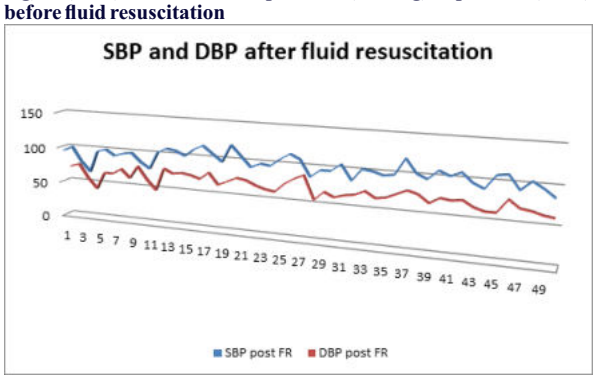

Chart 7 – Line chart showing (SBP) Systolic blood pressure (mm Hg) & (DBP) Diastolic blood pressure (mm Hg) in patients (n=50) after fluid resuscitation

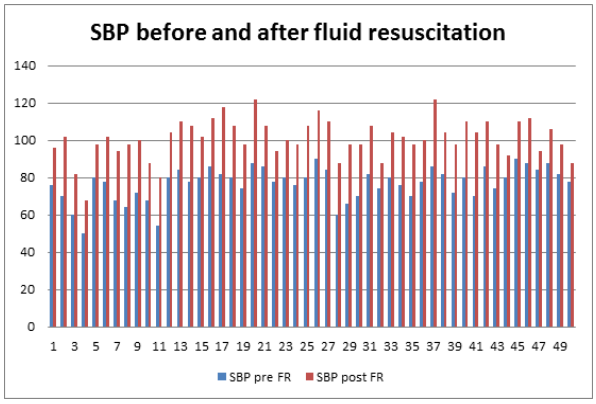

Chart 8– Column chart showing (SBP) Systolic blood pressure (mm Hg) in patients (n=50) (SBP preFR) before & after (SBP postFR) fluid resuscitation

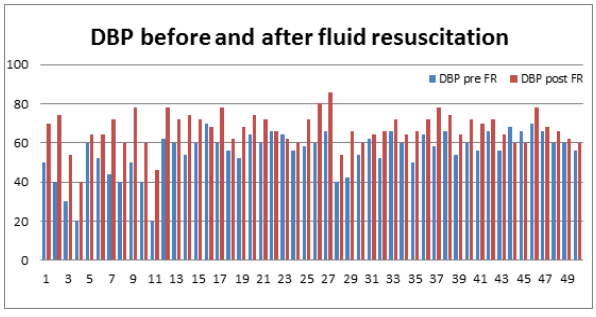

Chart 9 – Column chart showing (DBP) Diastolic blood pressure (mm Hg) in patients (n=50) (DBP preFR) before & after (DBP postFR) fluid resuscitation

Heart rate was more in the pre-fluid resuscitation with a mean of 114.40 compared to the 100.30 in the post-fluid resuscitation group. Systolic blood pressure was lesser in the pre-fluid resuscitation with a mean of 76.84 compared to the 101.08 in the post-fluid resuscitation group. Diastolic blood pressure was lesser in the pre-fluid resuscitation with a mean of 56.79 compared to the 68.17 in the post-fluid resuscitation group

DISCUSSION:

Hypovolemia and hypovolemic shock must be diagnosed and treated promptly in the EDs. In both conditions, it is important to identify intravascular volume status of the patient. In EDs, it is sometimes difficult to detect the intravascular volume status.[5] For this purpose, CVP measurement is a frequently used method. However, measurement of CVP is an invasive procedure. Its use is limited because of the invasive nature of the procedure and possible complications (arterial puncture, venous thrombosis, infection, etc) during or after the process. Recent publications indicate that CVP is not an ideal method, and clinical decisions regarding volume management should not be made based on CVP, because of its poor predictive role. [6]

Physical examination findings, vital signs, and laboratory results are other parameters used to estimate the intravascular volume status. These parameters are not reliable because they are influenced by various clinical conditions. Some of these parameters may be found normal as the compensatory mechanisms of the body initiate; thus, this may result in delays in the detection of volume loss.

For instance, in some patients, a 30% loss of total body liquid would be compensated by the body, and blood pressure may be held at normal levels, whereas this amount of loss is sufficient to initiate multiple-organ failure.[7]

The basal cardiac rate of many patients admitted to the ED is unknown. Although tachycardia is an indicator of acute liquid loss, it is not sufficiently specific and sensitive for a diagnosis or follow-up because it may be influenced by different inner and outer signals.

Serum lactate level is a biochemical parameter used as an indicator of tissue hypoperfusion; however, it is insufficient for the early diagnosis of hemodynamic instability and in the guidance of liquid resuscitation. [8]

To determine the status of intravascular volume, the dIVC ultrasonographic measurement is another method. The IVC is a highly collapsible major vein, and its diameter closely correlates with right-sided cardiac functions. The dIVC has not been found to be affected by the body's compensatory vasoconstrictor response to volume loss. Hence, it reflects volume status more closely than other parameters based on the arterial system, such as blood pressure, pulse rate, and others.

CONCLUSION:

Heart rate was more in the pre-fluid resuscitation with a mean of 114.40 compared to the 100.30 in the post-fluid resuscitation group

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