

***HYPOVOLEMIA:
Management of Hypovolemic Shock
in the Trauma Patient***

2.0 Contact Hours

Presented by:

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HYPOVOLEMIA: Management of Hypovolemic Shock in the Trauma Patient

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OBJECTIVES

After completing the course the health care provider should be able to:

- I. Assess a patient for the progression of shock based on the patient's clinical presentation.
- II. Appropriately, manage patient care in the initial/early stage of shock to prevent further patient injury and begin to correct fluid volume losses.
- III. Change clinical management techniques for patients with hypovolemic shock based on changes in patient status.
- IV. Demonstrate knowledge of the body's compensatory mechanisms such as: how/why they are initiated and how they work within the body.

Hypovolemic Shock

Hypovolemic shock is the most common type of shock experienced in patients who suffer from traumatic injury. It occurs as the result of "volume failure" when the body experiences a sudden and massive loss of circulating volume (fluid volume in the intravascular space). The decrease in circulating volume decreases tissue perfusion which prevents the metabolic needs of the tissues and cells from being met and eventually the cells die. As cellular death progresses, tissue death occurs and will eventually lead to death of the organism (or patient) if hypovolemia is not corrected.

There are two different types of hypovolemia, absolute hypovolemia and relative hypovolemia. Absolute hypovolemia occurs from a direct loss of whole blood or body fluid, as commonly occurs with traumatic injuries such as multiple gun shot wounds, stab injuries to an artery or the abdomen, and/or GI bleed. In contrast, relative hypovolemia occurs from fluid shifts within the body. In the case of relative hypovolemia fluid/blood has moved out of the intravascular space and begun pooling elsewhere. Relative hypovolemia can occur in patients with ascites, anasarca, and/or peritonitis.

Immediate Quick Review:

- Volume failure: decreased volume → decreased tissues perfusion → metabolic needs of cell cannot be met or maintained → cell cannot carry out normal processes → cellular death
 - Remember: cell → tissue → organ → organ system → organism.
- Absolute hypovolemia vs. relative hypovolemia
 - Absolute hypovolemia: Results from whole blood loss, loss of blood plasma, or direct loss of body fluid.

-Relative hypovolemia: Results from a pooling of blood somewhere outside of the vascular space

I. Trimodal Death Pattern

The severity of hypovolemia and patient status depends mostly on the amount of volume lost and the time frame of the loss. However, it is also important to consider the age of the patient and the patient's health status prior to the injury. The patient's prognosis is dependent upon these four factors, as hypovolemic shock can cause three different types of death (based on time): immediate death, early death, and late death.

Immediate death generally occurs at the scene of the injury or "in the field" and is the result of a severe injury to the patient. Immediate death can be related to: laceration of a major blood vessel or injury to a high level of the central nervous system. Early death occurs within several hours after injury and is related to fluid volume loss that is too great to be corrected despite aggressive treatment. Deaths that occur "early" are probably related to traumatic brain injury or acute hemorrhage. Late death occurs days to weeks after initial injury and is usually the result of complicating factors such as Multiple Organ Dysfunction Syndrome (MODS) or infection. In this case the patient has been treated for hypovolemia, and the hypovolemia may have been corrected, but the patient's body could not handle another insult and the patient begins to decompensate eventually leading to death.

Immediate Quick Review:

- Severity of hypovolemic shock is related to:
 - amount of volume loss
 - time frame of volume loss
 - age of patient
 - patient health status prior to injury

-Immediate Death:

-Occurs at the scene, generally, "in the field" where the initial injury occurred.

-THINK: SEVERE INJURY

-Early Death:

-Occurs within several hours after the initial injury

-Patient made it to the hospital/ED, but cannot be resuscitated

-Late Death:

-Occurs weeks after initial injury was inflicted

****FACT: 50% of trauma deaths occur in the immediate or early stage and the majority of these deaths are related to acute hemorrhage.****

II. General Clinical Presentation

When the patient arrives to the ED, their initial set of symptoms is generally related to the amount of volume lost and the time in which it was lost. The symptoms are a result of the body's effort to compensate for the loss.

Patients are likely to present with the following s/s:

-Tachycardia (heart rate 100 - > 140 bpm) : Occurs when the body is trying to compensate for the decrease in circulating volume by pumping faster to circulate the volume left as fast as possible. Remember, $\text{HEART RATE} \times \text{STROKE VOLUME} = \text{CARDIAC OUTPUT}$. Therefore, if you decrease stroke volume (as occurs in hypovolemia) then you have to increase heart rate to maintain cardiac output.

-Rapid and thready peripheral pulses: Rapid pulse is the result of tachycardia and thready pulse occurs because the blood is being shunted away from the peripheral vasculature to maintain perfusion of vital organs (brain, heart, lungs).

-Hypotension (begins @ > 1,500 mL of blood loss)

-Change in mental status: Can be the result of two different sets of clinical data: poor perfusion of the brain/CNS or hypoxia. Hypoxic patients are likely to present as agitated, whereas the patient will lose consciousness when greater than 2 L of blood is lost.

-Decreased stroke volume and cardiac output: Both are a direct result of hypovolemia, as the blood leaves the intravascular space there is less to pump (decreased stroke volume) and if less blood is going "into" the heart to be pumped then less blood is going to "come out" (decreased cardiac output).

-Decreased urinary output: As the body begins to shunt blood to the heart, lung, and brain, it decreases blood flow to the kidneys causing poor renal perfusion and decreased urinary output.

-Cool, clammy skin: This is also the result of blood shunting, blood is shunted away from the skin leading to poor perfusion and cool, clammy extremities.

Immediate Quick Review:

-Initial presenting s/s:

-tachycardia (heart rate 100 - > 140 bpm)

-peripheral pulses rapid and thready

-hypotension begins @ > 1,500 mL of blood loss

-change in mental status:

-slight agitation → complete loss of consciousness @ > 2,000 mLs of blood loss

-decreased stroke volume and cardiac output

- decreased urinary output as renal perfusion decreases
- cool, clammy skin

III. Stages of Shock and Associated Signs and Symptoms

-Initial/Early Shock: The signs and symptoms associated with the early stages of shock can be slow and subtle mostly because the body is easily able to compensate for the amount of fluid volume lost. However, the patient can quickly progress to the advanced stages of shock depending on the amount of fluid volume lost and the time frame in which it is lost. The patient may even “skip” the initial/early stage all together (Most likely, a patient with traumatic injury patient experienced this stage before arriving to the institution).

S/s include:

- BP decreased by 5-10 mmHg
- mild increase in heart rate
- change in metabolism from aerobic to anaerobic
 - increased production of lactate
- **FACT: Reference range for lactate: 0.3 to 2.3 mEq/L**

-Compensatory Stage: At this point the body is working harder to compensate for the for the volume loss causing a greater change in vital signs to show a clear reflection of fluid volume deficit. Initiating volume resuscitation and preventing further loss is now crucial to patient care.

S/s include:

- BP decreased by 10 -15 mmHg
- Patient becomes noticeably tachypnic and/or tachycardic
- Decreased urinary output
- Peripheral vasoconstriction
 - cold extremities, increased capillary refill time
 - blood being shunted to vital organs (brain, lungs, heart)

-Progressive Stage: During the progressive stage, the body’s compensatory mechanisms are no longer as effective and the patient is in a state of sustained hypotension causing decreased perfusion of vital organs. At this point, the patient is likely to have end organ damage. The patient’s cardiac, respiratory, and neurological status should be closely and continuously monitored for changes while aggressive fluid volume resuscitation is continued. (Do not use vasopressors for increasing blood pressure until the fluid volume deficit is corrected.)

S/s include:

- Pulse: tachycardic, weak, thready
- Blood shunting away from skin, abdomen, kidneys
 - Patient is/has: cold to touch, poor wound healing, decreased bowel sounds, irregular bowel movements, and decreased urinary output
- Decreased contractility of the heart r/t:
 - volume problem → pump problem

-Refractory Stage: At this point end organ damage has occurred along with multi-system organ failure. This stage is IRREVERSIBLE.

S/s include:

- Hypoxia
- Oliguria from renal shutdown
- Disseminated intravascular coagulation (DIC)

IV. Compensatory Mechanisms

- Decrease in blood pressure → Sympathetic Nervous System stimulation
 - Once the SNS has been stimulated, epinephrine and nor-epinephrine are released within the body causing: increased heart rate, increased contractility of the heart, and increase in diastolic blood pressure.

- Decreased renal perfusion → stimulation of the rennin – angiotension - aldosterone system
 - Renin release → release of angiotension I → conversion to angiotension II → adrenal release of aldosterone → increased absorption of sodium and water in the intravascular space → increased circulating volume (decreased urinary output) → increased cardiac output and increase in blood pressure

- Anti-diuretic Hormone (ADH)
 - release of ADH → re-absorption of sodium and water → increased intravascular volume and cardiac output (NO change in blood pressure) → decreased urinary output (patient will have increased urine specific gravity).

- Intracellular fluid shifts
 - Fluid moves from interstitial and intracellular space to intravascular space causing increased cardiac output and blood pressure. However, this will cause major cellular dehydration.

NOTE: Compensatory mechanisms begin before the body recognizes a decrease in cardiac output and tissue perfusion.

V. Clinical Management

-The main goals when managing a hypovolemic patient are preventing further loss and restoring previous losses. Controlling bleeding and initialing fluid volume resuscitation are the two most important initial interventions and should be initiated in the field whenever possible.

-When initiating fluid volume resuscitation:

- insert two or more large bore IVs (18 gauge or larger)
- give fluid at a 3:1 ratio
 - give 300 mLs for every 100 mLs lost
- IV solution should be Normal Saline or Lactated Ringers
- Give warmed fluid to prevent hypothermia
- Begin blood products after giving 3-4 liters of IVF
- Do NOT use vasopressors to increase BP until hypovolemia is corrected
- Monitor input and output
 - insert Foley Catheter

- After initiating therapy, continuously monitor:

- oxygen saturation
- core temperature
- hemodynamics
- check blood coagulation studies routinely (PT/PTT)

-Hypothermia is a common complication of fluid volume therapy. The body temperature is 98.6 F yet the patient is receiving large amounts of fluid at room temperature (about 70 F) causing a decrease in the patient's core temperature. Maintaining the core temp is critical to patient care because some of the body's natural processes/compensatory mechanisms (ex. coagulation) are ineffective when the body is hypothermic.

Warm the patient by:

- using warm IVF
- covering patient with blankets as much as possible while still allowing for continued assessment
- increasing the room temperature
- use warming lights if necessary

-Once the patient has been stabilized, prepare the patient for transport to diagnostic testing, the operating room, or the intensive care unit for further evaluation and treatment.

-Remember: Positive patient outcomes depend on early recognition and intervention

Crystalloids vs. Colloids (What type of IVF should I administer?)

Fluid Type	Examples	Notes
Crystalloids	Normal Saline (NS) Lactated Ringers (LR)	-Fluid will remain in the vascular space when given → increased intravascular volume. -Should be used for initial volume replacement. -Monitor the patient for fluid overload. -Do NOT use LR in a patient with liver failure.
Whole blood and/or Blood products	Packed Red Blood Cells (PRBCs) Fresh Frozen Plasma (FFP) Whole blood	-Used to replace whole blood loss. -Will increase body/blood oxygen carrying capacity → increased delivery of oxygen to the cells. -May be used if the patient has a low hemoglobin/hematocrit (check CBC) or does not respond to IVF therapy using crystalloids. -Administer according to hospital policy and using standard precautions for blood administration.
Colloids	Hetastarch (Hespan) Human Albumin Dextran	-Fluid volume expanders -Dextran is use should be limited because it can cause a change in the body's clotting factors as a side effect. -Hetastarch is cheaper than Albumin but should also be administered with caution because of its anticoagulant effect. -When administering Albumin, monitor the patient for side effects of: fever, chills, and urticaria.

VI. Potential Complications

-Hypothermia: Hypothermia occurs secondary to inadequate tissue perfusion and poor oxygenation. At this point, the patient can not control or maintain his/her own temperature. Patient factors that may increase the patient's risk of hypothermia include: patient age, severity of injury, patient alcohol use, amount/temperature of IVF use to resuscitate patient, blood product administration, and exposure of body cavities to the environment (as a result of injury or during surgical intervention). Hypothermia is associated with increased patient mortality rates and can lead to acidosis; therefore, it should be managed quickly and effectively.

-Metabolic Disturbances: Respiratory alkalosis often occurs in mild to moderate shock but will progress to metabolic acidosis as shock progresses. As shock progresses the body begins to maintain a state of anaerobic metabolism causing an increase in lactic acid progression and eventual metabolic acidosis. The cause of the disturbance should be treated and oxygen delivery to the cells should be supported.

- Disseminated intravascular coagulation (DIC): Clotting factors within the blood change in the presence of hypothermia and acidosis. Treatment includes the transfusion of platelets or fresh frozen plasma (FFP), as needed based on coagulation studies. DIC is a major cause of death in trauma patients.

-SIRS/MODS

-SIRS (Systemic Inflammatory Response Syndrome): Occurs when the body sustains insult or injury from more than one source such as: ischemia, infarct, injury, and/or infection.

-Signs and symptoms include two or more of the following:

-Hyperthermia (>100.4 F) or hypothermia (< 97.0 F)

-Tachycardia (> 90 bpm)

-Tachypnea (> 20 breaths per min) or PaCO₂ <32 mmHG

-Increased white blood cell count (> 12,000) or decreased white blood cell count (< 4,000)

-MODS (Multiple Organ Dysfunction Syndrome): At this point, the patient can no longer maintain homeostasis because of failure of more than one organ or organ system. There are two different types of MODS based on the onset of organ failure. Primary MODS has an early onset from a well defined cause. Secondary MODS, on the other hand, begins after SIRS has been diagnosed and uncontrolled for an extended length of time (later onset after the body has suffered multiple injuries). The transition within the body from SIRS to MODS is unclear. MODS presents as a worsening or progressive condition after SIRS has begun.

-Clinical Manifestations (MODS):

-Neurological: change in level of consciousness, altered mental status, seizure, fever

-Management: decrease cerebral oxygen demand (sedation, decreased patient stimulation), prevent cerebral ischemia/injury

-Respiratory: development of ARDS

-Management: mechanical ventilation as needed, oxygen therapy, continuous monitoring of oxygen saturations, consider serial ABGs

-Cardiovascular: tachycardia, decreased contractility, decreased MAP with systolic/diastolic dysfunction

-Management: continued volume management, continuous cardiac monitoring, vasopressors once fluid volume deficits are corrected, continuous hemodynamic monitoring, oxygen supply = oxygen demand

-Gastrointestinal: “dead gut” (ischemic bowel) related to hypoperfusion, intestinal bacteria “leaks” → bacteremia, GI bleed, formation of stress ulcer

-Management: medication administration for stress ulcer prophylaxis (Maalox, Tagamet, Protonix...), G-tube feedings, assess bowel sounds regularly – note any hypoactivity and continue to monitor closely

-Renal: decreased renal perfusion, decreased urinary output, increase in urine specific gravity, increased creatinine, renal tubular necrosis, eventual acute renal failure

-Management: promote diuresis, administer Dopamine (“renal dose”) as needed to promote renal perfusion, monitor input and output

-Hematologic: increased clotting times (difficulty clotting), change in clotting factors

-Management: replace clotting factors as needed, avoid invasive procedures whenever possible, monitor wound sites for increased bleeding or sanguineous exudates (apply pressure if needed to stop bleeding)

RESOURCES

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