Hypovolemic shock refers to a medical or surgical condition in which rapid fluid loss results in multiple organ failure due to inadequate perfusion. Two examples of hypovolemic shock secondary to fluid loss include refractory gastroenteritis and extensive burns.

**Pathophysiology:** The human body responds to acute hemorrhage by activating 4 major physiologic systems: the hematologic, cardiovascular, renal, and neuroendocrine systems. The cardiovascular system initially responds to hypovolemic shock by increasing the heart rate, increasing myocardial contractility, and constricting peripheral blood vessels. This response occurs secondary to an increased release of norepinephrine and decreased baseline vagal tone (regulated by the baroreceptors in the carotid arch, aortic arch, left atrium, and pulmonary vessels). The cardiovascular system also responds by redistributing blood to the brain, heart, and kidneys and away from skin, muscle, and GI tract.

The renal system responds to hemorrhagic shock by stimulating an increase in renin secretion from the juxtaglomerular apparatus. Renin converts angiotensinogen to angiotensin I, which subsequently is converted to angiotensin II by the lungs and liver. Angiotensin II has 2 main effects, both of which help to reverse hemorrhagic shock, vasoconstriction of arteriolar smooth muscle, and stimulation of aldosterone secretion by the adrenal cortex. Aldosterone is responsible for active sodium reabsorption and subsequent water conservation.

The neuroendocrine system responds to hemorrhagic shock by causing an increase in circulating antidiuretic hormone (ADH). ADH is released from the posterior pituitary gland in response to a decrease in BP (as detected by baroreceptors) and a decrease in the sodium concentration (as detected by osmoreceptors). ADH indirectly leads to an increased reabsorption of water and salt (NaCl) by the distal tubule, the collecting ducts, and the loop of Henle.

The hematologic system responds to an acute severe blood loss by activating the coagulation cascade and contracting the bleeding vessels (by means of local thromboxane A₂ release).

- In a patient with possible shock secondary to hypovolemia, the history is vital in determining the possible causes and in directing the workup.
- The chronology of vomiting and hematemesis should be determined.
- The physical examination should always begin with an assessment of the airway, breathing, and circulation. Once these have been evaluated and stabilized, the circulatory system should be evaluated for signs and symptoms of shock.
- Do not to rely on systolic BP as the main indicator of shock; this practice results in delayed diagnosis. Compensatory mechanisms prevent a significant decrease in systolic BP until the patient has lost 30% of the
blood volume. More attention should be paid to the pulse, respiratory rate, and skin perfusion.

- Initial laboratory studies should include analysis of the CBC, electrolyte levels (e.g., Na, K, Cl, HCO₃, BUN, creatinine, glucose levels), ABGs, and urinalysis (in patients with trauma). Blood should be typed and cross-matched.
- Patients with marked hypotension and/or unstable conditions must first be resuscitated adequately.

**TREATMENT**

Three goals exist in the treatment of the patient with hypovolemic shock as follows: 1) maximize oxygen delivery - completed by ensuring adequacy of ventilation, increasing oxygen saturation of the blood, and restoring blood flow, (2) control further loss, and (3) fluid resuscitation.

1. Calculate H₂O needs (from Nelsons) A. Maintenance – Use well weight if dehydrated. (see below) Weight in Kg. H₂O fluid needs 1 - 10 100cc/Kg/day 11 - 20 1000cc + 50cc/Kg/day > 20 1500cc + 20cc/Kg/day B. Use 2/3 maintenance if pt. Has meningitis. C. Add 8% for every F₀ of temperature over 100°F.

2. Correction of fluid deficits. A. Assess H₂O loss by weight weight loss (Kg) = water loss (L) (within 24 hrs.) H₂O loss (100) /total body wt. = % dehydration B. or estimate H₂O loss by physical exam:

<table>
<thead>
<tr>
<th>Signs &amp; Symptoms</th>
<th>Mild Dehydration</th>
<th>Moderate</th>
<th>Severe</th>
</tr>
</thead>
<tbody>
<tr>
<td>gen. appearance</td>
<td>thirsty, alert</td>
<td>thirsty,</td>
<td>drowsy-</td>
</tr>
</tbody>
</table>

C. Calculate well weight. well weight = sick wt./ (1 - %dehydration as decimal) D. Give 1/2 deficit over 8 hrs. and 2nd1/2 over 16 hrs. If Hypertonic dehydration, correct over 48hours. E. If mod. to severe dehydration present, emergency rehydration may be needed (10 - 20cc/Kg Normal Saline - may repeat x 1). 3.
Calculate Na+ and K+ needs

A. Maintenance is Na = 3 - 4 meq/Kg/day K = 2 meq/Kg/day

B. Correction of Na deficit or excess Vd Na (well wt. Kg)(desired Na - actual Na) = 0.6 (wt.)(Na) = meq Na needed

4. HCO3 deficit = (normal HCO3) - (measured HCO3)(.4)(Well Wt.)

A. Give 1/2 deficit over 4 - 8 hrs then recheck.

B. If TCO2 >= 15meq/L then let kidneys do the rest.

C. Empiric if severe dehydration likely: Add 3cc of 7.5% HCO3 to a litre D5.5NS to make emergency rehydration fluid.

5. Calculate actual fluids [(meq Na needed)/total fluid needed] / meq Na in normal saline = % normal saline = (meq Na needed)/154

A. Usually use D5?NS as IV fluids (ie. D5.2NS)

Sepsis was defined as the systemic host response to infection with SIRS plus a documented infection. Severe sepsis was defined as sepsis plus end-organ dysfunction or hypoperfusion. Septic shock was defined as sepsis with hypotension, despite fluid resuscitation, and evidence of inadequate tissue perfusion.

In septic shock due to bacterial infection, circulatory insufficiency occurs when bacterial products interact with host cells and serum proteins to initiate a series of reactions that ultimately may lead to cell injury and death.

Septic shock develops in fewer than half of patients with bacteremia. It occurs in about 40% of patients with gram-negative bacteremia and in about 20% of patients with Staphylococcus aureus bacteremia.

- The physical examination should first involve assessment of the general condition.
  - Ask, “Does the patient appear healthy without obvious effects, or does the patient appear acutely ill?”

- Hypotension and shock may be caused by other mechanisms or causes
  - Hypovolemic shock: This may be due to loss of whole blood (eg, from hemorrhage) or saline (eg, from gastroenteritis).
  - Cardiogenic shock: This may be due to an inadequate heart rate (eg, profound bradycardia or tachycardia) or myocardial contractile failure (eg, cardiogenic shock from an acute infarction).
  - Distributive shock: This may be due to loss of vascular tone with profound vascular pooling (eg, neurogenic shock) or arteriovenous shunting with inadequate capillary perfusion (eg, anaphylaxis). The initial process of septic shock involves maldistribution of blood flow, which leads to end-organ hypoperfusion. As noted previously,
progression often will lead to some elements of relative hypovolemia and myocardial impairment.

- Obstructive shock: The flow of blood is obstructed at the level of the heart (pericardial tamponade), pulmonary artery (massive pulmonary emboli), or aorta (aortic dissection).

- CBC with differential
  - An adequate hemoglobin concentration is necessary to ensure oxygen delivery in shock. The hematocrit level should be more than 30%, and the hemoglobin level should be more than 10 g/dL.
  - Platelets are an acute-phase reactant and usually increase at the onset of a serious stress. The platelet count decreases with DIC.
  - The WBC count and the white cell differential are modestly helpful in the prediction a bacterial infection. In febrile adults without localizing symptoms or findings, a WBC count greater than 15,000/mm$^3$ or a neutrophil band count greater than 1500/mm$^3$ is associated with an rate of bacterial infection of about 50%. In a febrile elderly patient, a WBC greater than 14,000/mm$^3$ or a neutrophil band percentage is associated with an rate of bacterial infection of greater than 50%.

Patients should be monitored for signs of volume overload; these include dyspnea, pulmonary rales, and pulmonary edema on the chest radiograph. Improvement, stabilization, and normalization in the patient's mental status, heart rate, BP, capillary refill, and urinary output indicate adequate volume resuscitation

Antibiotics must have a broad spectrum and cover gram-positive, gram-negative, and anaerobic bacteria, because all classes of these organisms produce identical clinical pictures. Antibiotics must be given parenterally in doses adequate to achieve bactericidal serum levels.

Dopamine is the agent most commonly used for not responding to the iv infusion. Treatment usually begins at a rate of 5.0-10 mcg/kg/min with intravenous administration, and the infusion is adjusted according to BP and other hemodynamic parameters. Often, patients may require high doses of dopamine, as much as 20 mcg/kg/min. If the patient remains hypotensive despite volume infusion and high-dose dopamine, a direct vasoconstrictor (eg, norepinephrine) should be started at a dose of 1 mcg/min and then titrated to support a systolic BP of 90 mm Hg.