

## Distributive Shock

Distributive shock is characterized by widespread vasodilation and increased capillary permeability, leading to a maldistribution of blood volume. There are several subtypes, with septic shock being the most common.

### Pathophysiology

In all subtypes, a significant release of inflammatory mediators (e.g., cytokines) causes widespread vasodilation, which drastically reduces systemic vascular resistance (SVR). Blood volume is effectively "misplaced" as it pools in the periphery, leading to inadequate blood return to the heart and decreased cardiac output. Increased capillary permeability also allows fluid to leak from the vessels into the interstitial space. The body's compensatory mechanisms may initially cause a high cardiac output (warm shock), but as the condition worsens, cardiac output can drop (cold shock).

The core problem in neurogenic shock is a loss of sympathetic tone, which leads to massive vasodilation (hypotension) and an unopposed parasympathetic response (bradycardia). In a high-level spinal cord injury, particularly injuries to the cervical spine (C1-C7) or high thoracic spine (T1-T6), the sympathetic nerve pathways that descend from the brain to the heart are severed. The vagus nerve is no longer balanced by sympathetic input, causing bradycardia.

### Causes

- Septic shock: Caused by a severe systemic infection. Common sources include pneumonia, urinary tract infections, and intra-abdominal infections.
- Anaphylactic shock: Exposure to a specific allergen (e.g., insect venom, food, medication) leads to a massive release of histamine and other inflammatory mediators.
- Neurogenic shock: Caused by spinal cord injury involving disruption of the sympathetic nervous system pathways, most commonly from injury above the T6 level.
- Less common causes include adrenal insufficiency and drug overdose or toxicity, particularly with potent vasodilators such as calcium channel blockers and hydralazine.

### Diagnosis

- Patient history:
  - Septic shock: Fever, chills, cough, pleuritic pain, dysuria, abdominal pain, diarrhea, recent surgical wounds, indwelling catheters or devices, headache and nuchal rigidity. Immunosuppression is a significant risk factor.
  - Anaphylactic shock: Known allergen exposure.
  - Neurogenic shock: Acute spinal cord injury.

- Physical exam findings: Warm, flushed skin in early septic shock (in contrast to the cool skin of other shock types), bounding peripheral pulses, purulent sputum, cellulitis or infected surgical wounds, erythema around intravascular device insertion sites, fever, or hypothermia. Anaphylactic shock presents with respiratory distress, stridor or wheezing, angioedema (swelling of lips, tongue), and hives. Neurogenic shock is characterized by hypotension and bradycardia as well as loss of motor and sensory function.
- Diagnostic evaluation by subtype:
  - **Septic shock:** Lab studies including comprehensive metabolic panel, complete blood count with differential, lactate, blood cultures, cultures from any suspected source of infection, inflammatory markers such as procalcitonin and C-reactive protein. X-ray, CT scan or ultrasound based on history and physical exam finding.
  - **Anaphylactic shock:** Anaphylaxis is primarily a clinical diagnosis based on a rapid-onset allergic reaction. Serum tryptase is a lab test that can support the diagnosis. Tryptase levels rise within minutes of an anaphylactic reaction and peak at 1-2 hours.
  - **Neurogenic Shock:** Spinal imaging including CT or MRI of the spine for visualizing a spinal cord injury, vertebral fractures, or other pathology.

## Treatment

### Septic shock:

- Fluid resuscitation, usually 30 ml/kg administered over 1-2 hours.
- Balanced crystalloids, like Lactated Ringer's (LR), are preferred over normal saline.
- After the initial bolus, fluid administration should be guided by a continuous assessment of the patient's fluid responsiveness to avoid fluid overload.
- Prompt (within 1 hour) administration of broad-spectrum antibiotics. Broad-spectrum antibiotics should cover the most likely pathogens for the suspected site of infection.
- Vasopressors are used to counteract vasodilation. Norepinephrine is the first line vasopressor. Vasopressin is often added as a second-line agent to norepinephrine if the MAP goal is not met.
- Identify and control the source of infection, which may require surgical intervention, removing an infected catheter, etc.
- Low-dose intravenous hydrocortisone (200 mg daily) may be beneficial in patients with refractory septic shock who remain hypotensive despite adequate fluid resuscitation and vasopressor therapy.

### **Anaphylactic shock:**

- Epinephrine is the cornerstone of anaphylaxis treatment. It is the only medication that can reverse the life-threatening syndrome of anaphylaxis. It must be administered immediately upon recognition of the signs and symptoms.
  - The preferred route is intramuscular (IM) injection into the anterolateral aspect of the thigh. The standard dose for adults and children over 30 kg (66 lbs) is 0.3 mg of 1:1000 epinephrine. For children under 30 kg, the dose is 0.15 mg.
  - For IV dosing, a 0.1 mg/mL solution of epinephrine may be administered. The typical initial dose is 0.1 mg (1 mL), given at a rate of 1 to 4 mcg/min.
  - Alternatively, an infusion may be initiated at 5 to 15 mcg/min, with adjustments based on clinical response.
  - Epinephrine auto-injectors (e.g., EpiPen, Auvi-Q) are designed for easy and rapid use by patients, family members, or bystanders.
  - If symptoms do not improve within 5-15 minutes, a second dose of epinephrine should be administered.
- Assess airway, breathing and circulation frequently. If the airway is compromised, bag the patient and prepare for intubation.
- Administer IV fluids to treat the hypovolemia associated with vasodilation.
- Adjunctive therapies such as H1 blockers (e.g., diphenhydramine) and H2 blockers (e.g., famotidine) can help alleviate cutaneous symptoms like hives, itching, and flushing.
- Corticosteroids such as methylprednisolone or hydrocortisone are given to prevent a delayed biphasic reaction.

### **Neurogenic shock:**

- Spine immobilization prevents further movement that might worsen the injury. This is achieved with a cervical collar, head blocks, and a rigid backboard. Halo bracing or surgical stabilization may be required.
- Reversal of the effects of the loss of sympathetic and unopposed parasympathetic response.
  - Fluid bolus with crystalloids, preferably LR. Assess response and give additional fluids as needed.
  - Vasopressors: Norepinephrine is the first line agent, as it has both alpha- and beta-adrenergic effects. The alpha effect causes vasoconstriction, which increases blood pressure, and the beta effect increases heart rate, which counteracts the bradycardia. The target mean arterial pressure is typically 85-90 mmHg for 5-7 days after injury. This is a higher target than most forms of shock

- to ensure perfusion to the damaged spinal cord and decrease the risk of secondary ischemic injury.
- Bradycardia management with atropine or pacing in cases of severe refractory bradycardia.
- Support temperature regulation due to loss of autonomic control.

### Nursing Considerations

- Monitor vital signs, mental status, and urine output frequently.
- Maintain 2 large bore IV lines for the administration of fluids and vasopressors.
- Administer antibiotics within the "golden hour" of septic shock diagnosis, after cultures have been sent.
- Monitor for signs of fluid overload during resuscitation, as some patients with distributive shock may require a balance between fluid administration and vasopressor use.
- For anaphylaxis, ensure the airway is protected and administer epinephrine as the first-line treatment.
- For neurogenic shock, maintain spinal immobilization, and use antipyretics and cooling blankets to normalize body temperature.

### References

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