• C.C. is a previously healthy 27-year-old man admitted to the critical care unit after an accident in which he was hit by a car and dragged along the pavement for nearly 100 feet. He suffered a frontal contusion, fractured clavicle and ribs, and extensive abrasions on his arms, legs, side, back, and buttocks. He was tachycardic, hypotensive, unresponsive, and ventilating poorly when admitted. He was placed on a mechanical ventilator and given IV fluids for shock. C.C. responded well to fluids, with an increase in blood pressure and an improvement in urine output.
Cardiac Output and Venous Return

- Shock – when tissues are not adequately perfused with blood

  - Circulatory Shock
    - Hypovolemic shock
      - 15 – 25% volume depletion
        » Hemorrhage
        » Shift of fluid into interstitial fluid (third spacing)
    - Distributive -
      - Neurogenic shock
        » Decreased sympathetic tone
          Brain injury
          drugs
          hypoxia
          hypoglycemia
          general anesthesia
Cardiac Output and Venous Return

- Anaphylactic shock
  - Immunological release of histamine, and other vasodilators
  - Also see
    - Bronchospams
    - Contraction of GI tract
    - Urticaria
    - Angioedema

- Septic shock
  - The result of systemic response to infection, usually bacterial
  - Bacteria release endotoxins which trigger immune response

- Obstructive
  - Inability of heart to fill properly (cardiac tamponade)
  - Obstruction to outflow from heart (pneumothorax)
Cardiac Output and Venous Return

– Cardiogenic Shock

  • > 80% of patients that develop this from MI will die

  • Result of loss of myocardial tissue (usually 40% or more is necessary) – cardiac output drops

  • As a result of drop in CO a vicious cycle develops, the result of increased sympathetic activity

– Neurogenic – typically the result of brain injury and lack of vessel tone
1. Based on his case history and responsiveness to fluid therapy, what type of shock was C.C. experiencing?

- Due to the extensive abrasions from the trauma, this patient most likely lost a significant amount of blood volume resulting in hypovolemic shock.

- The decreased intravascular blood volume leads to decreased venous return, then decreased cardiac output, decreased tissue perfusion, and then decreased oxygen delivery.

- The degree of hypovolemic shock has been divided into four classes based on factors such as amount of blood loss, mental status, urine output, heart rate, and capillary blanch test. (see table 20-2)
2. What other clinical findings would be helpful in confirming the type of shock?

- Hemodynamic monitoring is helpful in distinguishing hypovolemic shock from cardiogenic shock.
  
  • In cardiogenic shock, preload is high and cardiac output is low. In hypovolemic shock, preload and cardiac output are both low.

- Neurogenic shock can be the result of brain injury—another factor in this patient's case. Neurogenic shock typically responds better to vasoconstricting drugs whereas pharmacologic agents are not indicated in hypovolemic shock. Restoration of blood volume is most essential in hypovolemic shock.
3. Because of his many open wounds and invasive lines, C.C. is at risk for sepsis and septic shock. What clinical findings would suggest that this complication has developed?

- The presence of microorganisms in the blood stream can confirm sepsis. Normally the body’s mechanisms can fight off the bacteria and prevent widespread infection. But due to the trauma, this patient will be compromised and he may not be able to adequately fight infection.

- Obtaining blood cultures. By performing this test, the provider can figure out the exact organism that may be causing the infection and manage accordingly. Gram-negative bacteria are responsible for most cases of septic shock.

- Another indicator may be an increased white blood cell count, especially neutrophils. In order to fight infection, the body will produce more in an effort to eradicate the source of the infection. The clotting cascade, complement system, and kinin system may all be activated in the immune response.
Initially see high cardiac output (from immune-mediated vasodilation and sympathetic activation of the heart). The patient will be febrile, pink, and warm. Even though cardiac output is high, cellular hypoxia will be present because of the maldistribution of blood flow. This will be evidenced by a high SvO₂.

3. What is the link between sepsis and multiple organ dysfunction syndrome (MODS)?

MODS is associated with the systemic inflammatory response. The immune reactions of sepsis cause the prolonged/ongoing inflammatory response. The inflammatory response causes hypoxic injury to cells, leaky capillaries, damage from enzymes, and activation of the complement, kinin, and clotting system, which results in tissue destruction and organ dysfunction. When more than one organ dysfunction develops, MODS is the result.