

McCance: Pathophysiology, 6th Edition

Chapter 46: Shock, Multiple Organ Dysfunction Syndrome, and Burns in Adults

Key Points – Print

SUMMARY REVIEW

Shock

1. Shock is a widespread impairment of cellular metabolism involving positive-feedback loops that places an individual on a downward physiologic spiral that, if not reversed, can lead to MODS.
2. Types of shock are cardiogenic, hypovolemic, neurogenic, anaphylactic, and septic. A newly identified type of shock, traumatic shock, combines features of hypovolemic shock and septic shock.
3. The final common pathway in all types of shock is impaired cellular metabolism—cells switch from aerobic to anaerobic metabolism. Energy stores drop, and cellular mechanisms relative to membrane permeability, action potentials, and lysozyme release fail.
4. Anaerobic metabolism results in activation of the inflammatory response, decreased circulatory volume, and decreasing pH.
5. Impaired cellular metabolism results in cellular inability to use glucose because of impaired glucose delivery or impaired glucose intake, resulting in a shift of glycogenolysis, gluconeogenesis, and lipolysis for fuel generation.
6. Glycogenolysis is affected for up to 10 hours. Gluconeogenesis results in the use of proteins necessary for structure, function, repair, and replication that leads to more impaired cellular metabolism. Lipolysis is ineffective because of a lack of transport serum proteins.
7. Gluconeogenesis contributes to lactic acid, uric acid, and ammonia buildup; interstitial edema; and impairment of the immune system, as well as general muscle weakness leading to decreased respiratory function and cardiac output.
8. Cardiogenic shock is attributable to heart failure and is characterized by a decrease in cardiac output and impaired cellular metabolism.
9. Hypovolemic shock is caused by loss of blood or fluid in large amounts. The use of compensatory mechanisms may be vigorous, but tissue perfusion ultimately decreases and results in impaired cellular metabolism.
10. Neurogenic (vasogenic) shock results from massive vasodilation, causing a relative hypovolemia (even though cardiac output may be high), and results in impaired cellular metabolism.
11. Anaphylactic shock is caused by physiologic recognition of a foreign substance. The inflammatory response is triggered, and a massive vasodilation with fluid shift into the interstitium follows. The relative hypovolemia leads to impaired cellular metabolism.

12. Septic shock begins with impaired cellular metabolism caused by uncontrolled septicemia. The infecting agent triggers the inflammatory and immune responses. It is part of a continuum known as SIRS. Mortality for septic shock is very high.

Multiple Organ Dysfunction Syndrome

1. MODS is the progressive dysfunction of two or more organ systems resulting from a systemic inflammatory response after a severe illness or injury. The inflammatory response can be triggered by sepsis, necrotic tissue, trauma, burns, ARDS, acute pancreatitis, and other severe injuries.
2. Primary MODS is the immediate local or mild systemic response to the triggering event or illness. It primes the inflammatory system.
3. Secondary MODS is the uncontrollable, excessive systemic inflammatory response that develops after a latent period and results in organ dysfunction.
4. People at greatest risk for developing MODS are older adults, those with significant tissue injury or preexisting disease, and those in whom resuscitation from the initiating illness or injury has been delayed or inadequate.
5. Mortality from MODS is very high: 45% to 55% for failure of two organ systems, 80% for failure of three or more organ systems, and nearly 100% if the failure of three or more organs persists longer than 4 days.
6. Multiple organ dysfunction involves the stress response; release of complement, coagulation, and kinin proteins; changes in the vascular endothelium; and numerous inflammatory processes mediated by substances released by activated neutrophils and macrophages.
7. The consequences of the release of inflammatory mediators in MODS are vasodilation, increased vasopermeability, and selective vasoconstriction resulting in maldistribution of blood flow; hypermetabolism; myocardial depression; and hypoxic injury to cells. Cellular hypoxia and acidosis impair cellular metabolism, leading to organ dysfunction.
8. Clinical manifestations of the development of MODS are general during the first 24 hours: low-grade fever, tachycardia, tachypnea, dyspnea, and altered mental status. Over the next several days, beginning with the lungs, individual organ systems show signs of failure.
9. Because there is no specific therapy for MODS, early detection is extremely important so that supportive measures can be initiated as soon as possible.
10. At present the therapeutic management of MODS consists of prevention or removal of triggering mechanisms and support of individual organs. Recent scientific knowledge about inflammatory mediators has led to many promising future therapies for MODS.

Burns

1. Burns are classified according to depth and extent of injury.
2. First-degree burns involve the superficial skin without loss of protective function.

3. Second-degree burns are superficial (blister formation) or superficial involving partial skin thickness with a waxy white appearance and no involvement of dermal appendages.
4. Third-degree burns involve full skin thickness and often underlying tissues. They are painless and can be life threatening as a result of hypovolemic shock and metabolic and immunologic responses.
5. The TBSA burned is estimated using either the rule of nines or the Lund and Browder chart. Burns exceeding 20% TBSA are considered major burns.
6. Hypovolemia associated with burn shock is caused by increased capillary permeability with massive fluid losses from blood volume.
7. Altered cell membrane permeability and loss of electrolyte homeostasis contribute to burn shock.
8. Cardiac contractility is decreased during the first 24 hours with shunting of blood away from the liver, kidney, and gut.
9. Fluid resuscitation, such as with lactated Ringer solution, involves infusion of fluid at a rate faster than the loss of circulating volume.
10. The most reliable criterion for adequate resuscitation of burn shock is urine output.
11. Capillary seal is the term used to indicate the end of burn shock.
12. Transmembrane potentials are altered in cells not directly damaged by heat, with impairment of the sodium-potassium pump and loss of magnesium and phosphate.
13. The stress of a major burn activates the sympathetic nervous system with release of catecholamines, cortisol, glucagon, and insulin.
14. Burn injury produces a hypermetabolic state that persists until wound closure and is related to a higher thermal regulatory set point.
15. The local inflammatory response at the burn site releases cytokines, oxygen radicals, chemotactic factors, and eicosanoids, which leads to a systemic inflammatory response and contributes to hypermetabolism.
16. A posttraumatic hypermetabolic response is associated with increased visceral heat production.
17. Alterations in clotting factors produce a hypercoagulable state following major burns.
18. The immune response following a burn is immediate, prolonged, and severe.
19. Numerous alterations in inflammatory cytokines are evident in the immediate burn period, affecting cellular immunity, antibody production, and attraction of neutrophils and contributing to the vasodilation and increased capillary permeability associated with burn shock.
20. White blood cells are altered, and there is decreased opsonization and phagocytosis, contributing to the development of sepsis.
21. Changes in intestinal wall integrity lead to translocation of bacteria, endotoxemia, and septic shock.

22. Loss of intact skin with a major burn results in significant evaporative water loss contributing to hypovolemia.
23. Treatment of major burns involves meticulous wound management, adequate fluids and nutrition, early surgical excision and grafting, modulation of the hypermetabolic state, and pain management.