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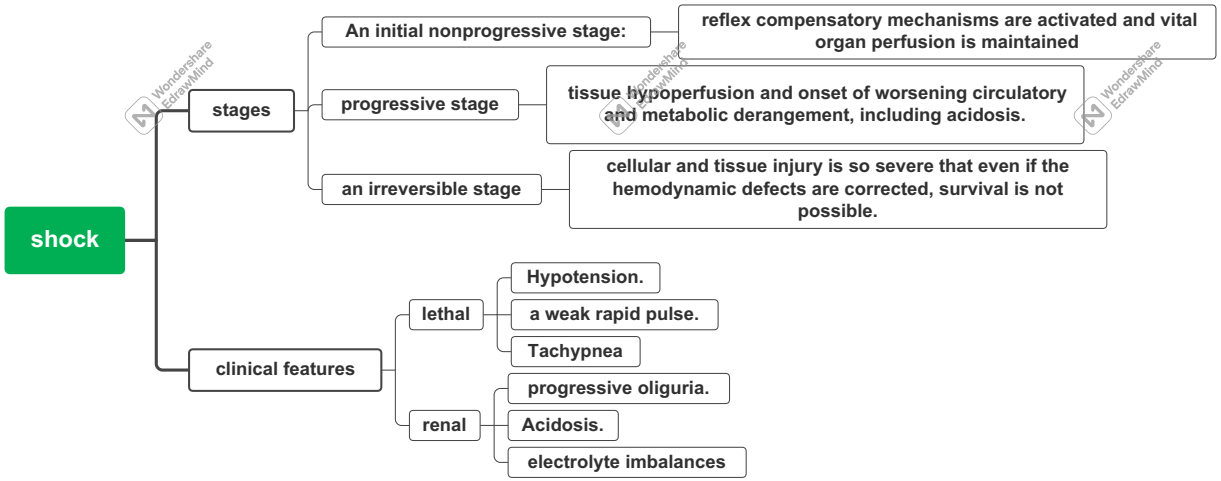
shock

- major types
  - cardiogenic shock
    - results from low cardiac output as a result of myocardial pump failure.
    - It may be caused by:
      - myocardial damage (infarction).
      - ventricular arrhythmias.
      - extrinsic compression (cardiac tamponade*).
      - outflow obstruction (e.g., pulmonary embolism
  - hypovelemic shock
    - results from low cardiac output due to loss of blood or plasma volume (e.g., resulting from hemorrhage or fluid loss from severe burns).
  - neurogenic shock
    - result from a loss of vascular tone.
  - anaphylatic shock
    - results from systemic vasodilation and increased vascular permeability that is triggered by an immunoglobulin E–mediated hypersensitivity reaction.
  - septic shock
    - Caused by massive outpouring of inflammatory mediators from innate and adaptive immune cells
    - it produces
      - arterial vasodilation.
      - vascular leakage.
      - venous blood pooling
    - leads to
      - tissue hypoperfusion*
      - cellular hypoxia
      - metabolic derangements
    - pathogenesis
      - Inflammatory response
        - innate immune cells produce numerous cytokines, including TNF, IL-1, IFN-γ
        - microbial products can
          - engage receptors , e.g: (TLRs)
          - activate complement cascade forming anaphylatoxins
          - activate coagulation directly through factor XII and indirectly through altered endothelial function.
      - counter inflammatory response
        - shift from proinflammatory (TH1) to anti-inflammatory (TH2).
        - production of anti-inflammatory mediators (e.g., soluble TNF receptor, IL-1 receptor antagonist, and IL-10).
        - lymphocyte apoptosis
      - Endothelial activation and injury
        - cytokines loosen endothelial tight junctions forming exudate
        - Activated endothelium also upregulates production of nitric oxide (NO) ,C3a, C5a, and PAF, which may contribute to vascular smooth muscle relaxation and systemic hypotension
      - Induction of a procoagulant state:
      - metabolic abnormalities
        - Septic patients exhibit insulin resistance and hyperglycemia.
        - TNF and IL-1, glucagon, growth hormone, glucocorticoids, and catecholamines all drive gluconeogenesis

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