        - Dry (a), wet (b)
        - Internal gangrene can occur
    - Gas necrosis: caused by bacteria
        - In muscles
        - Gas bubble forms
            - Dangerous when it gets into blood vessels

Inflammation Response: after injury, non-lethal cell injury

- **Acute inflammation**: first step in inflammation response
    - Response is immediate
    - Arterioles dilate: allows blood flow to the site
    - Edema and swelling
    - Blood becomes thicker: due to other things coming to the site
    - Leukocytes migrate to the area
    - Accumulate cells and proteins at site
    - Site gets cleaned: macrophages, prep for resolution
- Plasma Systems:
    - First: complement system
        - A series of proteins activated by endotoxins from bacteria
    - Second: clotting system
        - Formation of mesh work, prevent spread of injury, localizes problem
    - Third: Kinin-bradykinin system:
        - Bradykinin dilates blood vessels, induces pain, and increase permeability
    - Specific protein that controls the systems: C1 esterase inhibitor, dilation
        - Hereditary condition: angioneurotic edema
            - No C1 esterase inhibitor
                - Systems continue to function
- Inflammatory cells: leukocytes
    - Granulocytes and agranulocytes*
    - Granulocytes: contain material in granules that is released to attract other cells to site
    - Agranulocytes: do not release anything
        - Neutrophils: most common
        - Monocytes*: leave blood and invade tissue, can become macrophage
        - Lymphocytes*: B and T cells (antibody)
        - Eosinophils: parasitic defense
            - Throw up on parasite to digest it
        - Basophils: mediator release (histamine)
        - Mast cells: immune system cells that release mediators