STIMULI FOR ACUTE INFLAMMATION

Acute inflammatory reactions are triggered by a variety of stimuli:

- Infections (bacterial, viral, parasitic) and microbial toxins
- Trauma (blunt and penetrating)
- Physical and chemical agents (thermal injury, e.g., burns or frostbite; irradiation; some environmental chemicals)
- Tissue necrosis (from any cause)
- Foreign bodies (splinters, dirt, sutures)
- Immune reactions (also called hypersensitivity reactions)

Each of these stimuli may induce reactions with some distinctive features, but all inflammatory reactions share the same basic features. We first describe the characteristic reactions of acute inflammation, and then the chemical mediators responsible for these reactions.

Changes during acute inflammation

There are 3 phases in inflammation:

- Alteration (vascular changes)
- Exudation
- Proliferation

1. Alteration (vascular changes)

Since the two major mechanisms of host defense against microbes (antibodies and leukocytes) are normally carried in the bloodstream, it is not surprising that vascular phenomena play a major role in acute inflammation. Normally, plasma proteins and circulating cells are sequestered inside the vessels and move in the direction of flow. In inflammation, blood vessels undergo a series of changes that are designed to maximize the movement of plasma proteins and circulating cells out of the circulation and into the site of injury or infection.