Morphological and biochemical alterations of polymorphonuclear neutrophil (PMN) leukocytes from patients with inborn errors of phagocytic function: a comprehensive review
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The purpose of this review is:

(1) to describe the cellular alterations in blood PMN during infection leading to phagocytic killing of bacteria at the site of infection;

(2) to separate these in vivo cellular alterations into specific sequential phases and to link selective in vitro tests of PMN function to each phase;

(3) to relate alterations of these tests to disorders of PMN phagocytic function; and

(4) to present examples of inborn errors of phagocytic function including
chronic granulomatous disease, Chediak–Higashi syndrome, actin dysfunction, and humoral deficiencies of complement components and specific immunoglobulins.

The blood phagocytes include PMN, monocytes, and eosinophils; each cell type is capable of carrying out all phases of the phagocytic act. However, the chief phagocytic cell is the PMN and it predominates in the bloodstream and bone marrow and participates extensively in eradication of pyogenic bacteria and certain fungi from extravascular sites. As pointed out by Rebuck the PMN arrives at inflammatory sites rapidly after the inciting stimulus has been applied and continues to accumulate for 6–12 h before an influx of mononuclear cells occurs. This remarkable response of blood phagocytes to inciting stimuli such as tissue injury or infection has held the attention of investigators since the days of Eli Metchnikoff, the astute Russian biologist who injured a starfish larva with a thorn and observed the phagocytic process in its entirety.

Three related humoral systems are activated during bacterial infection; complement, kinin and plasmin play an integrative role in the inflammatory response. Before the tissue becomes hyperaemic by the action of bradykinin, the circulating PMN marginates to the endothelial surface and adheres to it until finally it emigrates through the vascular endothelium.

Figure 14.1 The phagocytic events include the following sequence of responses in the blood PMN: adherence to the endothelial surface, emigration out of the vascular tree, chemotaxis or directed movement to the site of the infection, opsonization of the bacteria and recognition of the opsonized bacteria by the PMN, ingestion, degranulation, and subsequent peroxidative killing of the bacteria by the PMN.