INTRODUCTION

Inflammation is a protective response of vascular tissue. Under normal circumstances, it functions as part of a surveillance system designed to quarantine and destroy harmful agents. The system is complex, with redundant cascades and amplification built into it. Because of this, inflammation is often exaggerated out of proportion to the inciting stimulus, and results in pathologic injury to the host. Certain types of clinical injury, such as severe trauma, burns, pancreatitis, and major surgery, can provoke such a profound response that it results in respiratory failure, coagulopathy, and multiorgan system dysfunction. These phenomena are often summarized under the term “systemic inflammatory response syndrome (SIRS).”

INFLAMMATORY CELLS

Neutrophils

Multiple cells are involved in the inflammatory response, but perhaps the best-characterized effector cell is the neutrophil. These
polymorphonuclear leukocytes (PMNs) are involved in the phagocytosis of pathogens and damaged tissue. They belong to the family of immune cells known as granulocytes, which are named for their abundant cytoplasmic granules. These granules contain proteolytic enzymes, precursors to oxygen radicals, and proinflammatory mediators. These toxic substances are released from the activated neutrophil in a process known, quite appropriately, as degranulation. The result is endothelial cell injury, production of cytolytic oxygen species, and further recruitment of leukocytes.

Although they circulate in the blood, neutrophils exert their effects by migrating into the interstitium. This is accomplished in a three-step process involving margination, adherence, and extravasation. First, the expression of adhesion molecules known as selectins on the neutrophil (L-selectin) and the endothelial cell (E- or P-selectin) slows the neutrophil and causes it to “roll” along the microvasculature. Next, the interaction of integrins located on the neutrophil with adhesion molecules on endothelial cells causes the leukocyte to adhere to the endothelium. The leukocyte integrins thought to play an important role are CD11a/18 and CD11b/18, whereas the adhesion molecules on the endothelium are ICAM-1 and VCAM-1 (1). Once fully arrested, extravasation of the neutrophil is mediated by chemoattractants from the interstitial space. After moving through the endothelial junctions, neutrophils degranulate, leading to the release of their toxic substances. However, because the attack is nonspecific, neutrophils have been implicated in producing the clinical manifestations of acute lung injury, reperfusion injury, and transfusion reactions (2,3).

**Basophils and Mast Cells**

Like neutrophils, basophils also have cytoplasmic granules that contain inflammatory mediators. Among them, histamine is perhaps the best characterized. Through interaction with H1 receptors, histamine causes an increase in vascular permeability, bronchoconstriction, vasodilatation, and chemotaxis of neutrophils. Although the basophil’s granules are chiefly released in response to the binding of immunoglobulin (Ig) E antibodies, complement represents another pathway to activation. This has an important implication in cardiac surgical patients, in that institution of cardiopulmonary bypass (CPB) is associated with massive complement activation (4).

Mast cells are another inflammatory cell important in the inflammatory response, and they share many similarities with basophils. Unlike basophils, however, they do not circulate in the blood. Instead, they are fixed in various perivascular spaces, including the heart, lungs, and