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Systemic inflammatory response syndrome (SIRS) is a systemic manifestation of sepsis (Table 5.1), although the syndrome may also be caused by multiple trauma, burns or pancreatitis without infection. Serious infection, such as secondary peritonitis, may lead to SIRS through the release of lipopolysaccharide endo - toxin from the walls of dying Gram-negative bacilli (mainly Escherichia coli) or other bacteria or fungi. This and other toxins stimulate the release of cytokines from macrophages (Figure 5.6). SIRS should not be confused with bacteraemia, although the two may coexist. /uni25CF /uni25CF β /uni25CF Septic manifestations and multiple organ dysfunction syn drome (MODS) in SIRS are mediated by the release of pro inflammatory cytokines such as interleukin-1 (IL-1) and tumour necrosis factor alpha (TNF α). These cytokines normally stim ulate neutrophil adhesion to endothelial surfaces adjacent to the source of infection and cause them to migrate through the blood vessel wall by chemotaxis , where they can a ttack the bacterial invasion. A respiratory burst occurs within such acti vated neutrophils, releasing lysosomal enzymes, oxidants and free radicals, which are involved in killing the invading bacte ria but which may also damage adjacent cells. Coagulation, complement and fibrinolytic pathways are also stimulated as part of the normal inflammatory response. This response is usually beneficial to the host and is an important aspect of normal tissue repair and wound healing. On occasions, this response may become harmful to the host if it occurs in excess, when it is known as the systemic inflammatory response syn drome or SIRS. There are high circulating levels of cytokines and activated neutrophils that stimulate fever, tachycardia and tachypnoea. The activa ted neutrophils adhere to vascular endothelium in key organs remote from the source of infec tion and damage it, leading to increased vascular permeability , which in turn leads to cellular damage within the organs, which become dysfunctional and give rise to the clinical picture of multiple organ dysfunction syndrome or MODS. In its most se vere form, MODS may progress into multiple system organ failure (MSOF). Respiratory , cardiac, intestinal, renal and liver failure ensue in combination with circulatory failure and shock. In this state, the body’s resistance to infection is reduced and a vicious cycle develops where the more organs that fail, the more likely it becomes that death will follow despite all that a modern ICU can do for organ support (Summary box 5.11 Summary box 5.11 Definitions of infected states /uni25CF /uni25CF /uni25CF /uni25CF Moritz Kaposi, 1837–1902, Professor of Dermatology , Vienna, Austria, described pigmented sarcoma of the skin in 1872. Sepsis Six The European Society of Intensive Care Medicine (ESICM) alongside the Society of Critical Care Medicine (SCCM) spearheaded the Surviving Sepsis Campaign (SSC) in 2002 with several aims, including the development of guidelines for the diagnosis, treatment and post-ICU care of sepsis and a reduction in mortality from sepsis. The Surviving Sepsis Campaign continually develops and updates resources and implementation tools to further its mission of reducing sepsis and septic shock. The sepsis bundle , also known as the resuscitation bun - dle, is a combination of

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