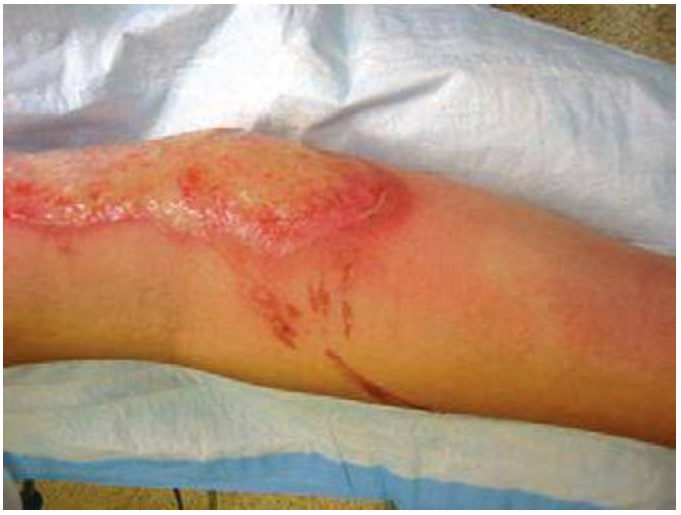

FIGURE 166e-1 Cellulitis complicating a burn wound of the arm, with extension of the infection to adjacent healthy tissue. *(Courtesy of Dr. Robert L. Sheridan, Massachusetts General Hospital, Boston; with permission.)*

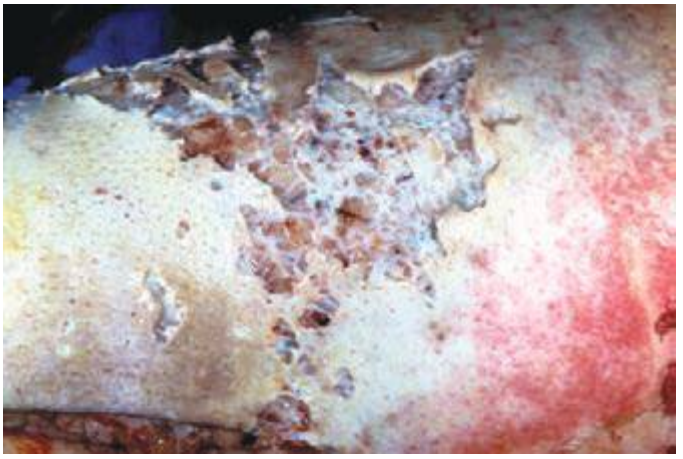

FIGURE 166e-3 A burn wound infected with Pseudomonas aeruginosa, with liquefaction of tissue. Note the green discoloration at the wound margins, which is suggestive of *Pseudomonas* infection. *(Courtesy of Dr. Robert L. Sheridan, Massachusetts General Hospital, Boston; with permission.)*

cultures); (3) burn-wound cellulitis (extension of infection to surrounding healthy tissue; Fig. 166e-1); and (4) invasive infection in unexcised burn wounds (infection that is secondary to a partial- or full-thickness burn wound and is manifested by separation of the eschar or by violaceous, dark brown, or black discoloration of the eschar; Fig. 166e-2). The appearance of a green discoloration of the wound or subcutaneous fat (Fig. 166e-3) or the development of ecthyma gangrenosum (see Fig. 25e-35) at a remote site points to a diagnosis of invasive *P. aeruginosa* infection.

Changes in body temperature, hypotension, tachycardia, altered mentation, neutropenia or neutrophilia, thrombocytopenia, and renal failure may result from invasive burn wounds and sepsis. However, because profound alterations in homeostasis occur as a consequence of burns per se and because inflammation without infection is a normal component of these injuries, the assessment of these changes is complicated. Alterations in body temperature, for example, are attributable to thermoregulatory dysfunction; tachycardia and hyperventilation accompany the metabolic changes induced by extensive burn injury and are not necessarily indicative of bacterial sepsis.

Given the difficulty of evaluating burn wounds solely on the basis of clinical observation and laboratory data, wound biopsies are necessary for definitive diagnosis of infection. The timing of these biopsies can be guided by clinical changes, but in some centers burn wounds are routinely biopsied at regular intervals. The biopsy specimen is examined for histologic evidence of bacterial invasion, and quantitative microbiologic cultures are performed. The presence of $>10^5$ viable bacteria per gram of tissue is highly suggestive of invasive infection and of a dramatically increased risk of sepsis. Histopathologic evidence of the invasion of viable tissue and the presence of microorganisms in unburned blood vessels and lymphatics is a more definitive indicator of infection. A blood culture positive for the same organism seen in large quantities in biopsied tissue is a reliable indicator of burn sepsis. Surface cultures may provide some indication of the microorganisms present in the hospital environment but are not indicative of the etiology of infection. This noninvasive technique may be of use in determining the flora present in excised burn areas or in areas where the skin is too thin for biopsy (e.g., over the ears, eyes, or digits). Rapid identification of organisms and institution of appropriate therapy are critical to the survival of patients with severe burn injury; polymerase chain reaction (PCR) is now being used for rapid identification of specific pathogens, sometimes in <6 h, to allow earlier treatment interventions.

In addition to infection of the burn wound itself, a number of other infections due to the immunosuppression caused by extensive burns and the manipulations necessary for clinical care put burn patients at risk. Pneumonia, now the most common infectious complication among hospitalized burn patients, is most often acquired nosocomially via the respiratory route. The incidence of ventilator-associated pneumonia among burn patients is 22–30 cases per 1000 ventilator-days—more than double that among surgical or medical ICU cohorts; this infection usually results from colonization of the lower respiratory tract and parenchyma because of sustained microaspiration. Among the risk factors associated with secondary pneumonia are inhalation injury, intubation, full-thickness chest wall burns, cutaneous thermal injuries, immobility, blood transfusions, and uncontrolled wound sepsis with hematogenous spread. Septic pulmonary emboli also may occur. Suppurative thrombophlebitis may complicate the vascular catheterization necessary for fluid and nutritional support in burns. Endocarditis, urinary tract infection, bacterial chondritis (particularly in patients with burned ears), and intraabdominal infection also complicate serious burn injury. Staphylococcal scalded skin syndrome due to burn-wound infection with *S. aureus* has been described as a rare complication. Finally, burn surgical-wound infections contribute to morbidity and have been found in up to 39% of patients; these infections often result in repeat skin grafting and prolonged hospitalization.

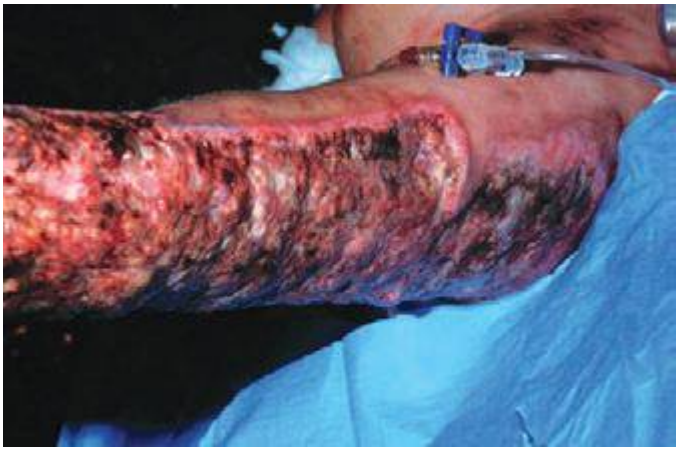

FIGURE 166e-2 A severe upper-extremity burn infected with *Pseudomonas aeruginosa*. The wound requires additional debridement. Note the dark brown to black discoloration of the eschar. *(Courtesy of Dr. Robert L. Sheridan, Massachusetts General Hospital, Boston; with permission.)*