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Infection and the burn patient

Can ... the leopard change his spots?[1]

Burn patient survival has significantly increased during the past four decades as hypovolaemic shock, acute renal failure, invasive bacterial burn wound infection, Curling's ulcer, and metabolic wasting have been controlled by timely adequate resuscitation, topical chemotherapy, gastric acid control, and effective nutritional support. Even though infection of the burn wound is rare (only 8 per cent of infections in 1989), infection remains the major predator and most common cause of death in burn patients in whom the respiratory tract has now become the most frequent portal of infection[2]. The success of current burn wound care is further indexed by the change in the predominant form of pneumonia, haematogenous pneumonia that commonly arose from an infected burn wound, having been supplanted by bronchopneumonia. Pneumonia and bronchitis comprised 53 per cent of all infections in burn patients treated at the US Army Institute of Surgical Research during 1989. The importance of pneumonia as a co-morbid factor in critically ill patients is documented by its recognition as a cause of death in 50 per cent of fatal burns treated during the period 1983–1987[3].

The presence of *Pseudomonas* infections in burn patients in the late 1950s and early 1960s, considered by many to be a burn specific aberration, was validated by subsequent experience to have been an epidemiological premonition of infections in other critically ill patients. Since the 1960s the predominant infection-causing organisms in burn patients have continuously changed as evidenced by sequential mini-epidemics of infections caused by *Providencia stuartii*, *Enterobacter cloacae*, a recrudescent *Pseudomonas* species, and *Staphylococcus aureus* (the most frequently recovered pathogen in the past 6 years)[4]. During the past 13 years recovery of *Pseudomonas aeruginosa* from the blood and burn wounds has strikingly decreased, while recovery of staphylococci, Candida sp. and true fungi from those sources has increased[5]. The incidence of candidaemia has increased more than tenfold and filamentous fungi have become the most common cause of invasive burn wound infections, i.e. 18 of the 25 cases documented histologically during the period 1983–1987.

The increase in survival time of those burn patients who die, 17 days in 1970–1971 *versus* 24 days in 1986–1987, coincides with improvements in early post-injury care and a later onset of the currently common infections. The prevalence of pulmonary infections attests to both the success of initial ventilatory support in patients with inhalation injury and the microbial ecological hazard of prolonged airway intubation that permits conversion of the airway flora to that of the burn wound. The effectiveness of current burn care techniques is limited by both the extent of burn and the duration of treatment, as evidenced by the average time of diagnosis of candidal burn wound colonization (the 31st postburn day), and the average time of diagnosis of candidal infections of the urinary tract and other sites (even later in the second postburn month). The late occurrence of these non-bacterial opportunistic infections is consistent with their occupancy of the ecological niche that exists in extensively burned patients who have received one or more courses of broad spectrum antibiotics as treatment for earlier occurring bacterial infections and whose wounds remain open.

The ever changing character of this predator's spots answers Mr Kipling's question[1] in the affirmative and reinforces the importance of institutional infection control programs, including scheduled surveillance cultures of the wound, sputum, urine and stool, antibiotic sensitivity testing of index organisms, and the use of burn wound biopsies to assess the microbial status of the wound and guide both local and systemic therapy[6]. Studies demonstrating that the effects of infections are organism specific suggest that the recent changes in the epidemiology of infection in burn patients have contributed to the improvement in survival and survival time. Gram-positive bacteraemia or candidaemia exert no demonstrable effect on burn patient survival, while the previously prevalent Gram-negative bacteraemia significantly increased the mortality of burn patients above that predicted on the

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basis of age and burn size[7]. Cohort nursing can be used to interrupt the perpetuation of recrudescent nosocomial epidemics of those mortality enhancing organisms[8].

The persistence of injury induced immunosuppression until the burn wound is completely closed provides impetus for the development of techniques and technologies that will permit early permanent closure of even the largest burn and limit the time during which those patients are at risk to infection. The alterations in the microbial ecology of the severely burned patient induced by such improvements in care and the effects of antimicrobial agents directed against newly emerging pathogens may only induce further change in the leopard's spots. Infection as the predominant cause of morbidity and mortality in burn patients will persist and frustratingly qualify the success of new treatment techniques until effective regimens of immunological support are developed for those patients in whom the extent of burn, associated injuries or supervening complications delay wound closure.

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