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**The effect of antiseptics and topical antimicrobials on wound healing.**

In 1919 Alexander Fleming stated that ‘antiseptics will only exercise a beneficial effect in a septic wound if they possess the property of stimulating or conserving the natural defensive mechanism of the body against infection’. He also considered that in estimating the value of an antiseptic it was more important to study its effects on tissues than any effects on bacteria (Fleming, 1919).

Before and since Fleming, antiseptics have been used to treat chronic infected ulcers or open wounds healing by secondary intention but without much evidence of their value. Rigid nursing disciplines and procedures have consequently not been questioned or altered for many decades. Antiseptics have varied from cobwebs and faeces, milk and honey, and urine and resins in our ancient and medieval civilisations (Lister, 1867; Forrest, 1982a), through Lister’s phenolics (Forrest, 1982b) to the modern day use of halogen compounds, oxidizing agents, alcohols and aldehydes, phenols and bisbiguanides and metal-containing agents.

Invasive bacterial infection of a wound, cellulitis or abscess formation has long been recognized to delay healing (Carrell & Hartman, 1916) and it has been argued that the optimal wound healing rate cannot be achieved unless surface bacteria are eliminated from open wounds (Burke, 1971). The topical application of antimicrobial agents to irrigate wounds or to provide long term action by incorporation into ointments or dressings has become widespread. However, with the advent of occlusive dressings into this field it has become apparent that septic sloughing wounds may debride themselves without recourse to the use of antiseptics or topical antibiotics (Alper et al., 1983; Alvarez, Merz & Englestein, 1983; Skog et al., 1983). Occlusive dressings have been shown in experimental studies to provide the moist environment suitable for re-epithelialization (Winter, 1972) and encouragement of healthy granulation tissue provided that *Pseudomonas aeruginosa* does not colonize and infect the wound (Leaper et al., 1984). There is no question that antiseptics, such as the hypochlorite solutions, can adequately debride sloughing wounds, probably by causing necrosis of the superficial layers and allowing separation of superficial slough. The toxic effects on healthy healing tissues have not been measured, nor has the hypothetical need to sterilize an ulcer surface been assessed. Topical antibiotics have not been shown to have any direct effect on wound healing but risk allergic reactions and the chance of overgrowth of antibiotic-resistant pathogens.

There is some evidence that wound fluid contains antibacterial substances (Hohn et al., 1977; Buchan, Andrews & Lang, 1981) and may contain either stimulating wound hormones or debriding proteases which would be neutralized by over-zealous, frequent wound cleansing. Such substances in wound fluid are part of the natural defence mechanism and probably are the agents which provide auto-debridement in occluded wounds.

The evidence that antiseptics are toxic to healing tissues is scanty and available information suggests that conventional nursing procedures should be reviewed with a move from frequent dressing changes involving antiseptics, particularly hypochlorites or topical antibiotics, to less frequent changes of occlusive dressings without antiseptics. Surgical debridement of necrotic tissue will, of course, improve...
the chances for any ulcer to begin to heal. Commonly used antiseptics have a toxic effect on fibroblasts grown in tissue culture (Leaper & Brennan, 1986). Although this is a crude test of membrane-active antiseptics' toxicity in vivo, the fibroblast must be considered as the key cell for the laying down of collagenous scar tissue and probably is the initiator of wound contraction. In experimental ulcers hypochlorite-containing antiseptics significantly delay healing, re-epithelialization and production of collagen and prolong and increase the acute inflammatory response (Brennan, Foster & Leaper, 1986). In the rabbit ear chamber model the hypochlorite antiseptics irrecoverably damage the new vessels of healing which are vital for the nutrition of healthy granulation tissue (Brennan & Leaper, 1985). Aqueous chlorhexidine, however, was found to be innocuous in both of these experimental models. Adverse effects on the host defence system may also counteract any benefit afforded by the reduction in numbers of viable bacteria. Aqueous povidone iodine is toxic to phagocytic cells at very low concentration (Van den Broek, Buys & Van Furth, 1982) and suppression of lymphocyte response occurs in the blood of patients treated with povidone iodine (Ninneman & Stein, 1981). Again, in contrast, the bisbiguanide chlorhexidine exhibits few toxic effects (Butler & Iswaran, 1980). Chlorhexidine has not been shown to have any inhibitory effect on healing of experimental infected open wounds (Lindhe et al., 1979; Platt & Bucknall, 1984). How closely these experimental animal models resemble the healing process in clinical practice is debatable and no clinical trial of antiseptics in wounds to prevent post operative infection has shown any suggestion of delayed healing. The weight of evidence must surely be that, with the exception of hypochlorite, antiseptics, unlike disinfectants, have low toxicity.

So, what is the value of antiseptics and topical antibiotics in open-wound management? There is little question that in necrotic, sloughing wounds and ulcers their effective action in debridement is worth keeping in reserve but should not replace surgical removal of adherent necrotic material whenever this is appropriate. Once a wound is clean a change to occlusion may be desirable. Topical antimicrobial prophylaxis with silver sulphadiazine and silver nitrate-chlorhexidine has been shown to reduce morbidity and mortality in patients with severe burns (Platt et al., 1976). Earlier use of neomycin tulle gras was associated with a considerable delay in separation of slough, probably because of suppression by the antimicrobial action of bacteria that produce proteolytic enzymes (Lowbury et al., 1962). No burns practitioner would withhold antiseptics or systemic antibiotics for infected burns. For other epithelial defects, often with a complicated aetiology like the venous ulcer, a reappraisal of conventional nursing practices is probably indicated. Although antiseptics and topical antibiotics are relatively inexpensive their inappropriate and prolonged use may delay healing, particularly with the hypochlorite antiseptics, and thereby lengthen an in-patient stay or delay return to normal life. Further studies of the interactions between antiseptics and antibiotics, bacteria and the immune system are needed to determine those clinical situations where their use is beneficial to the patient.

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