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pH CHANGES ON THE SURFACE OF BURNS

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THE state of the burn surface is important for healing and the successful take of skin grafts. Little is known, however, of the pH changes which occur and a study was therefore undertaken of the surface pH and its relation to the arterialised capillary blood pH, pCO_2 and standard bicarbonate, and the bacterial flora of the burn.

MATERIALS AND METHODS

Readings of the pH of the burned surface at multiple sites were made on 13 patients with burns of up to 35 per cent of body surface and sufficiently severe to be admitted to hospital, with a portable pH meter and electrodes of the flat head type. Each reading was taken in triplicate, and the mean recorded. When eschar was present, a small window was cut to allow the electrode to record the pH of the surface beneath. At each site a record was kept of the depth of the burn, which was verified or corrected by later assessment.

Before pH recordings were taken, the ambient room temperature was standardised at 75°F (24°C).

pH, pCO_2 and standard bicarbonate were estimated from arterialised capillary blood samples (Anderson *et al.*, 1960).

Bacteriological samples from each site were taken on a 1-inch square gauze placed in contact with the burn surface for 2 minutes. The gauze was shaken out in isotonic saline by a mechanical shaker, serial dilutions were prepared and cultures plated on to blood agar and McConkey's medium. Colony counts were made and the organisms identified at 48 hours.

In vitro measurements of the pH produced in 0·1 per cent glucose-broth solutions by inoculations with different organisms were made immediately after inoculation and after incubation at 37°C for 48 hours.

The patients were nursed on a siliconised polyurethane foam mattress on a mesh frame. Initially the burns were exposed and, if the eschar became moist, dressed with Furacin gauze. Patients received intravenous Dextran 110 for 48 hours in the shock phase; no electrolyte fluids were administered.

RESULTS

Surface pH. The mean pH of unburned skin of 6 healthy volunteers was 7·3 (SEM, 0·1). There were no significant differences on different parts of the body.

Full thickness burns had a mean pH of 7·9 (SEM, 0·05), and partial thickness burns had a mean pH of 8·1 (SEM, 0·1). The difference in the pH between unburned skin and full thickness burns was significant ($P < 0·001$) as was also that between the pH of unburned skin and partial thickness burns ($P < 0·01$). The difference between the pH of full thickness and partial thickness burn was not significant ($P < 0·1$).

There was no significant correlation between the pH of the burn surface and the arterial pH, pCO_2 or bicarbonate data which remained within normal limits as the burns studied were insufficiently extensive to disturb them.

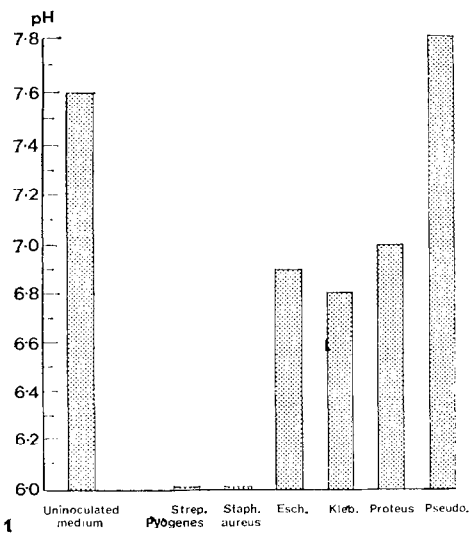
Becteriological flora. Figure 1 shows the burn surface pH related to the bacterial flora on the surface. The highest pH, 8·6 and 8·5, occurred when the surface was colonised by *Pseudomonas* and *Proteus* spp., the lowest (pH 7·0) with *Streptococcus pyogenes*. The pH of burned skin from which no organism was cultured was 8·3. No correlation could be demonstrated between the amount of growth, as measured by the colony counts, and the pH.

In vitro inoculation. The pH of 0·1 per cent uninoculated glucose-broth solution was 7·6. Figure 2 shows the pH of the broth solutions produced after the inoculations of different organisms. The highest pH was obtained with the Gram-negative bacilli; the lowest with *Streptococcus pyogenes* and *Staphyloccus aureus*. Comparison with Figure 1 shows that these figures of pH almost exactly parallel the results found by measurement of the pH of the burn surface in the presence of the same types of organisms.

Post-burn day. Figure 3 shows the relation of the pH of the burn surface to the number of days after the injury.

The pH on the first 3 days after the burn injury was highly alkaline (8·4 to 8·5) and then fell rapidly to a level which was then maintained. As healing occurred, the pH returned to that of normal unburned skin, pH 7·3.

Graft survival. A further series of pH readings of the surface was taken in the operating theatre immediately before skin grafting. The take of individual grafts were followed at subsequent dressings. As other workers have also found, it is in practice difficult to identify with accuracy individual grafts and for this reason this part of the investigation was incomplete. There was, however, a distinct impression that the higher the burn surface pH at the time of graft application, the greater the successful take.



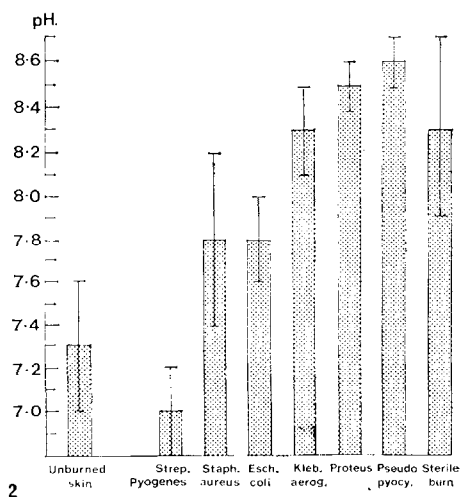


FIG. 1. pH of the burn surface colonised with different organisms.

FIG. 2. pH produced in 0·1 per cent glucose-broth solution by inoculated organisms.

Discussion

The pH of unburned skin closely approximates the normal arterial pH, 7·3. Following burning the surface pH rises while a metabolic acidosis occurs in the internal milieu. The subsequent slow fall of the surface pH towards that of unburned skin coincides with a rise in arterial pH from the initial acidosis during the shock phase.

The studies of Black *et al.* (1971) in children confirm our findings. The mean pH in their cases was higher than normal and continued to rise for the entire period of study up to the 28th post-burn day. This they attributed to increased adrenocortical activity. It is probably an over-simplification that the loss of base from the burn surface directly contributes to the acidosis, although the exudate which appears on the burn surface is derived from and therefore influences the intra- and extra-vascular spaces.

The initial high pH during the phase of rapid fluid loss on to the burn surface is followed by a plateau pH (Fig. 3). This period of stabilisation corresponds with a return of the arterial pH to normal levels. Finally, when epithelialisation is being completed there is a further fall as normal skin impermeability is achieved.

The observation that *Streptococci* on the burn and in culture significantly reduced the pH while the presence of *Pseudomonas* raised it, offers an explanation for the success of the older treatment of surface infection by the application of topical bicarbonate or weak acids. In the same way that mandelic acid is used to treat a *Pseudomonas* pyelonephritis, these topical acids or bases alter the surface pH to a level in which growth of the infecting organism is inhibited. The Furacin tulle which was used as a dressing had rather an inconsistent pH, varying between 7·3 and 7·8, but tulles may be applied having an acid or alkaline bias depending on the known flora, in order to alter the surface pH.

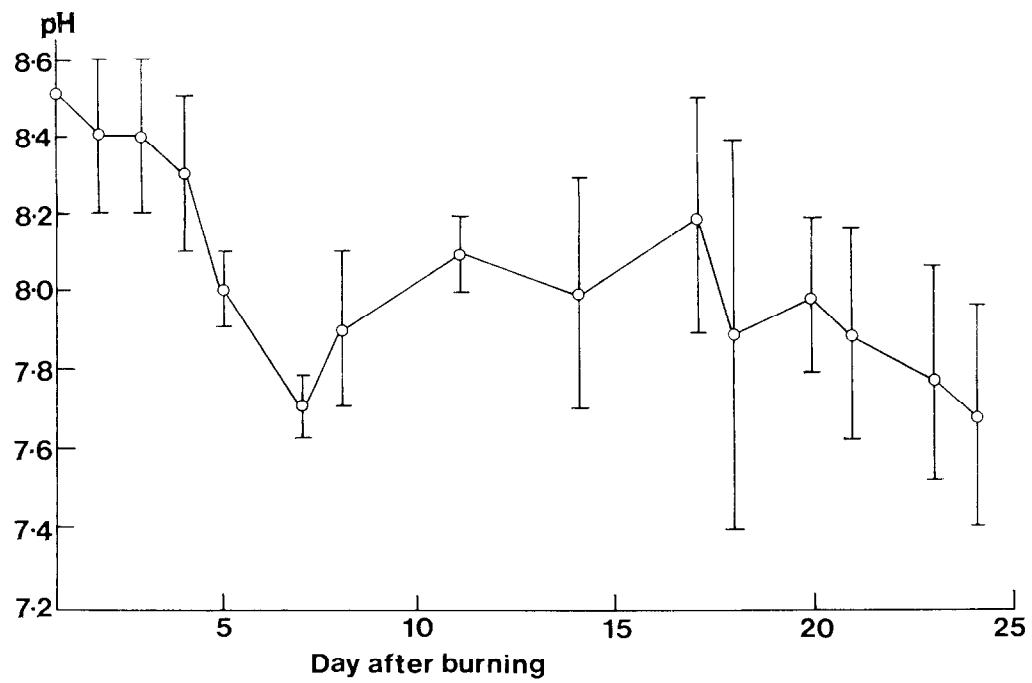


Fig. 3. pH of the burn surface related to the day after burn injury.

The probably greater success of grafts at the higher pHs may be due either to the absolute level of pH favouring graft take, or, because at the higher pH, fewer *Streptococci* were growing.

For any given species of bacterium, there is only a narrow pH range which allows strong growth. *Streptococci* ferment sugar, without using free oxygen, to obtain energy with the production of lactic acid, and growth is inhibited in more alkaline media. *Pseudomonas*, on the other hand, forms ammonia from amino-acids (Sherris *et al.*, 1957). The ammonia on the burn surface combines with free negative ions to form weak alkalis, and the pH is thereby raised.

Proteus spp. all produce acid from glucose (Rustigan, 1945) but also decompose urea and amino-acids to ammonia (Stumpf, 1944). The burn surface pH in the presence of *Proteus* is a resultant of the varying weak alkali and weak acid production.

Surface pH is determined both by the pH of the tissue fluid exudate and by the metabolic products of the surface organisms. Altering either will alter the surface pH, while altering the surface pH will affect the bacteriological flora and possibly also influence the survival of grafts.

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