DISCUSSION
DISCUSSION

Local burn wound management is still in the phase of trial. After many advances in medical science, mortality from burn has definitely reduced, but morbidity from contracture and keloid are still crippling the sufferer physically, mentally as well as socially.

Burn gives rise to a wide raw area of variable degree of depth. In full thickness burns, the vascular thrombosis is confluent and involves arterioles, venules and capillaries, extravasated erythrocytes and oedema are present in extravascular tissue to a variable degree. These changes are not necessarily confined to the dermis but may extend into the subcutaneous part or beyond, depending on the severity of injury. The devitalized tissue (eschar) eventually will slough spontaneously, mostly as a result of bacterial enzymatic proteolytic action. This pabulum of denatured protein and cellular debris, which constitutes the eschar, provides a substrate for proliferation of micro-organisms. The more efficient the wound bacteriostasis, the longer will be required for slough to occur (Order, S.E., Moncrief, J.A., 1965; and William, W. Monafo et al, 1987).
In partial thickness burns, thrombosis is incomplete and in superficial injury is limited to the upper or papillary dermis. The dermal circulation is restored gradually in partial thickness burns, but the process requires several days or more, so that significant interval of relative ischemia is present. Even in more superficial burns sloughing of partial thickness eschars will occur spontaneously as epithelial elements spread from viable remnants of the skin appendages to cover the denuded dermal surface (Order, S.E., Moncrief, J.A., 1965 and William, W. Monafo, et al, 1987).

It is evident from the foregoing that the delivery of systemically administered antimicrobial agents to the burn wound is limited. It is the zone of stasis, produced by ischemia and necrosis of tissue, which limit and to a large degree negate the efficacy of systemically administered antibiotic directed at burn wound sepsis by forming an effective barrier, preventing medication from reaching the vital area of sepsis, namely the subescharotic area. The systemically administered antibiotic can only reach the ischemic area from gradient diffusion from wound periphery which is inadequate to prevent colonization. (William, W.M., Bruce, F., 1987; Kock, D.M., 1985).

Many attempts have been made to limit the extent of the zone of stasis by such means as more effective
resuscitation, maximum dose of heparin and steroids, but
all have proved unsuccessful. Clearly, the topical
application of antimicrobials will be the best method to
ensure that they are present in adequate concentrations,
at least on the wound surface, where the risk of bacterial
contamination is the greatest.

Normal skin contains a sparse microflora consisting
mainly staphylococcus epidermidis, diphtheroids and other
bacterial species that are not ordinarily highly virulent.
Burn wound cultures taken within a few hours following
injury typically yield few or even none of the normal
flora unless there has been contamination, usually by
soil or fetid water during rescue and transport. The
rapidity with which subsequent colonization occurs and
the microbial density that is reached eventually, depends
on many factors, the most important of which are wound
extent and depth and prior state of the patient's health.
In untreated, extensive deep wounds, for example, dense
colonization, primarily by gram positive cocci, often
occurs within 24 hours. Within 3 to 7 days, aerobic gram
negative bacteria particularly pseudomonas aeruginosa
typically appear. Untreated this initial colonization
gives way to confluent, deeper spread via the ducts of
skin appendages. Ultimately active invasion of unburned
subjcet tissue may occur and spread systemically leading
to septicaemia. In burn of lesser extent, particularly
if they are relatively superficial, healing may occur
spontaneously, but the danger of conversion of superficial
burn to deep burn by infection and/or dissociation and
loss of body constituents remain a major problem.
Therefore, adequate topical therapy should be initiated
as rapidly as possible following injury, irrespective of
the depth of burn (W. Monafo, & Bruce Freedman, 1987).

With better understanding of pathophysiology of
burn wound, there is some consensus that wound infection
is the primary source of morbidity and mortality from
extensive burn injury. Therefore the main aim in the
treatment of burn is to re-establish the continuity of
skin. The raw area after burn should be covered to make
it a closed wound which subsequently reduces excessive
evaporative water loss and prevents wound infection
(Demling, R.H., 1983).

Autograft skin grafting is the most accepted
procedure for management of burn wounds. However, limited
available skin donor sites preclude the achievement of
prompt closure of burn wound (Bruce, J.F. et al, 1974).
Alternatively various temporary measures such as
allografts, heterografts or a variety of ingeniously
engineered skin substitutes to protect the open burn
wound have been and are being tested for this purpose
there is no evidence that mortality or morbidity - most of which is still because of infection - has been reduced in burns that exceed 40 percent of total body surface area, in addition these substitutes have limited availability and a very high cost. However, amniotic membrane has been used by various investigators and is still in use as a substitute for skin. The amniotic membrane fulfilled all the functions of an ideal biological dressing. In terms of their large size and readily availability at no cost to the patients. In addition, the membrane appears to have another property subjectively, the rapidity of ingrowth of epithelium from the borders of wound in full thickness defects and the rate of re-epithelization of partial thickness burns, appear to be increased by their use. Choao et al (1940) and Troensegard Hansen (1950) also have noted that amniotic membrane seemed to possess some specific healing power. They have reported a stimulation of both fibrous tissue growth and more rapid epithelial repair. Thus it is logical to compare the effectiveness of the combination of topical PVP solution + Neosporin powder and amniotic membrane as topical burn therapy.

Local polymyxin + Neomycin + Bacitracin (Neosporin) and Povidone iodine (PVP) combination forms an almost complete barrier against microbials. Polymyxin can protect the burn wound against colonization by Pseudomonas
pyocyanea, but not so against staphylococcus aureus and haemolytic streptococci. Povidone iodine on the other hand has wide antibacterial, antifungal, sporicidal and viricidal properties. Neomycin and Bacitracin supplement this action especially in relation to gram negative organisms.

Burn wound biopsy provides quantitative and qualitative bacteriology. Bacterial counts less than $10^5/cm^2$ of tissue exclude burn wound sepsis. If on the other hand the number of bacteria is more than $10^5/cm^2$ of tissue wound sepsis is generally present. There is correlation between death rate and presence of burn wound sepsis. No death occurred in patients, whose wounds biopsies revealed $\leq 10^5/cm^2$ of tissue, and patients died from burn wound sepsis, their wound biopsies showed $\geq 10^5$ bacteria/cm$^2$ of tissue (Artz, Moncrief & Pruitt, 1979; Berset & Chiolero, 1982; Krupp, 1982; Zamora, 1984 and Zelluer, 1980). Our study using PVP+N showed an appreciably better percentage of sterile cultures as compared with Amniotic membrane, both at seven days (52.6% vs 36.8%) and 18 days (68.4% vs 44.7%). Similarly, the numbers of cultures below $10^5/cm^2$ were significantly less for PVP+N both at 7th and 18th days (43.4% & 30.2% vs 47.3% & 42.1%). Even patients with a count of more than $10^5/cm^2$ were less in group treated with PVP+N (6.5% & 2.6% vs 18.4% & 13.2%). These figures agree with other studies. Thus Moncrief has shown 49% sterile
cultures and 84% less than the critical level of $10^5$/cm$^2$
in a study of more than 3200 bacterial cultures which compare well with our corresponding values of 68.4% and 30.2% on day 18, using PVP + N. Zellner and Bugi (1985) too have shown better results with PVP as compared to amniotic membrane. Our results which are markedly better than other studies with only PVP are due to the addition of neosporin.

The role of healing also showed a marked improvement on amniotic membrane in superficial, deep and mixed burn categories. The tanning effect of PVP is an added advantage for this keeps the surface dry, so holding colonization to a low level and also permitting early surgery. PVP + N combination forms a 'crust' which sets up a barrier to colonization and at the same time keeps the surface dry. In patients with superficial burns when epithelization was complete the crust separated itself and in clean cases no single incidence of infection was found.

In deep burn wounds, multiple injections of PVP subescharaly helped in two ways. In the first place, it kept the subescharal count to a low level. In fact this bacterial colonization and its inaccessibility to topical antimicrobials have been major factors in burn wound sepsis of deep burns. That subescharal injection of PVP was
beneficial and evident from the results, namely no single septicaemic mortality occurred in deep burn patients. The second beneficial effect is that it opens up sub-escharal plane thus helping in early escharolysis and decreasing bleeding on separation.

The burn wound in most of these patients could be grafted immediately after the eschar separation which was in marked contrast to the fact that topical agents are totally ineffective in subescharal colonization including superficially applied PVP cream and also that after escharectomy or lysis a considerable period of time is spent in limiting the infection at the burn site, before grafting can be taken up. Subescharal PVP injections were attempted basically because PVP has been shown to have beneficial antibacterial effects when used subcutaneously, intrapleurally or intraperitoneally without any serious iodine toxicity. The concentration of 0.25% PVP may seem to be too low for it to be effective but it has been mentioned that with this concentration there is an increase in free iodine and antibacterial activity. None of our patients showed any clinical evidence of iodine toxicity. The PVP injections in deep burns of more than 50% were limited to three in order to limit the total amount of PVP injected. We found that subescharal injections markedly reduce the incidence of septicaemia and mortality in these patients and at the same time keep
the surface healthy. Application of PVP + N was accompanied by minor pain. Once 'crust' has been formed, pain disappeared. Pain was not accompanied with application of Amniotic membrane, but submembranal suppuration causes great discomfort to the patients since it is a closed dressing. PVP was an open method and did not require removal of previously applied layer, and being an open method there was no question of suppuration being collected at any site. This in our view is an important psychological and clinical advantage and at the same time saves a lot of nursing personnel time. The iodine level are elevated after PVP application but this level creates no significant impairment of thyroid functions or manifestations of iodine toxicity. Further, the iodine levels returns to normal within a week after applications are stopped. Similarly the repeated serum creatinine compared with those of patients being treated with amniotic membrane showed that no toxicity because of drugs contained in neosporin mainly bacitracin and neomycin sulphate which are renally toxic. This was probably because after the three and two applications were limited to only those areas which were denuded, thus largely limiting the total amount of drugs used to a bare minimum. Even the PVP solution used, compared favourably with PVP ointment, commonly used, in terms of lesser amount of PVP used.