DISCUSSION
The problem of burn wound infection is now becoming increasingly important. In 1960's one could easily determine the location of the burn ward in a hospital by the smell of pseudomonas infections (Zelmer and Bugyi, 1985) i.e. bacterial infection has always been considered one of the most serious complication of burn injury and is probably one of the most common cause of death. Inspite of the local treatment, mortality and morbidity remained almost unchanged which focussed the attention on three basic concepts regarding problems of burn injury.

1. Burn injury provides a large raw area which causes a loss of large amount of water, electrolytes and plasma proteins. Studies showed that the denuded skin increases the insensible loss upto 75 times and this also increases the heat loss.

2. Large raw area with serum exudate provides a huge culture plate for micro-organisms and leads to wound sepsis which is the basic problem in the management of burn wound.

3. The problem of pain caused by irritation of exposed nerve endings by clothing dressing or even the mere contact of air.
The improvement in infusion therapy in burns patients had led to a clear reduction in mortality (Zenilner and Metzger, 1976).

This means that the problem of infection is now becoming increasingly important. The vascular nature of burn tissues as a result of thrombosis of vessels, limits the delivery of endogenous phagocytic cells and also decreases the efficacy of systemically administered antibiotics. In addition to infection wound maceration and pressure necrosis also favours microbial proliferation and impairs circulation. At deep burn sites due to prolonged ischaemia in the subescharal plane, systemically administered antibiotics reach only by diffusion gradient from the wound periphery (Yurf and McManus et al., 1984; Koch, 1985 and William and Bruce, 1987).

The large raw area produced by the burn wound with its exudate or serum works like a huge culture plate on which organisms can multiply uninhibitedly. The superficial burn can convert to a deep burn in the presence of infections. Thus the burn wound remains a constant potential source of systemic sepsis until eschar separation is completely despite the use of topical antimicrobial agents (Baxter et al., 1973).

It is still thought that burn wound can be virtually sterile at the time of injury. This concept is purely academic. Adequate sampling of burn surface will reveal bacteria in every instance, although in first few
hours the concentration may be very low (Robert B. Lindberg).

The nature of therapy and the circumstances of handling the burnt patient affects the initial seeding of burn wound and this flora is of little infections significance. The variety of bacterial species found in burn vastly greater than the limited number found on normal skin. The normal skin flora consists primarily of gram positive organisms including co-agulase negative Staphylococci, corny bacterium and micrococci. The only gram negative forms commonly found on skin are Mima and Hecellea groups. These organisms are confined to moist skin areas.

In large burn areas, dense colonization of pathogens can occur within 24 hours (William and Bruce, 1987 and Krupps et al, 1985). In untreated patients, immediately after injury few bacteria can be recovered and these are predominantly gram positive. The type and density of organisms present in the untreated burn wound change with time. By the fifth post burn day, pseudomonas can be recovered (William and Bruce, 1987). By the middle of the second post burn week, the burn wound organisms are predominantly gram negative, rods and fungi especially candida (Order & Moncrief, 1965; Arts et al, 1979 and William and Bruce, 1987). The organisms penetrate the eschar by migration and extend down to the viable-nonviable
tissue interface. At this site further microbial proliferation commonly occurs and promotes lysis of denatured collagen and spontaneous sloughing of the eschar (Order and Moncrief, 1965). In patients with inadequate host defense capacity or those in whom the topical therapy is ineffective, the subescharal organisms invade the underlying unburnt tissue and may spread systemically (Moncrief, Teplitz, 1964; William and Bruce, 1987).

Evaluation and treatment of the microflora in the burn wound to prevent septic complications are a challenging clinical problem. Bacterial colonization of the burn wound may reach a concentration of $10^6$ to $10^7$ per gram of burn tissue before changes are evident either in the appearance of the burn wound or in the detection of clinical signs or symptoms of systemic sepsis (Charles R Baxter et al, 1973). Surface culture techniques including swabs, contact plates and capillary guaze technique fail to predict accurately the presence or progression of burn wound sepsis due to poor correlation between the surface flora and the colonization of the eschar and subcutaneous tissue (Bretano and Gravens, 1967; Clarkson, Ward and Polk, 1967; Colebrooke, Lowbery and Hurst, 1960; Georgides et al, 1969). Blood culture is helpful in few life threatening sepsis complications but bacteremia is frequently absent. Multiple escharal biopsy obtained serially from representative area of the burn wound and culture quantitatively and qualitatively furnish valuable information.
about bacterial count of $10^5$ gm of tissue as the upper limit for minimal penetration to deeper tissue level (Moncrief, Teplitz, 1964; Krupps, Barchler, Bille, 1985).

If the concentration of bacteria is $7 \times 10^5$ bacteria/gram of tissue, burn wound sepsis is generally present. Under such conditions skin grafts are often autolyzed by infection. Therefore, prior to skin grafting, aggressive wound treatment must be instituted and continued until the bacterial concentration of wound biopsies falls $\leq 10^5$ bacteria/gram of tissue (Artz, Moncrief and Pruitt, 1979; Parks, Linares and Thompson, 1981; Robson, Krizek and Heggers, 1975; Steen, 1983; Teplitz, 1969; Teplitz, 1974).

There is also a correlation between death rate and presence of burn wound sepsis. No death was seen in group whose wound biopsies revealed $\leq 10^5$ bacteria/gram of tissue (Krupps, Barchler and Bille, 1985).

Considering all the above facts we use PVP + Neosporin powder topically in all patients. This combination forms an almost complete barrier against microbials (Sinha et al., 1987). Neosporin powder contains polymyxin, neomycin and bacitracin. Polymyxin can protect burn wound against colonization by pseudomonas pyocyanae (Georgiade and Harris, 1973) but not against staphylococcus aureus and haemolytic streptococci (Jackson, Lowbury and Topley, 1951). Povidone-Iodine on the other hand has a wide antibacterial, antifungal sporicidal and virucidal properties (Zellner and Bugyi, 1985; Robson, Schaefer, Krizek, 1974; Law & MacMillan, 1972).
William and Bruce, 1987; Georgiadis and Harris, 1973; Peter Zellner, 1985). The tanning effect of PVP has an added advantage on dead layers of skin, creating a demarkation between viable and nonviable areas. The tanned second or third degree burns were never transformed into sticky necrosis. In addition, the tanned skin is less likely to produce infected material that can be transported into lymphatic and blood vessels during surgical scraping (Peter Zellner, 1976), so by this effect PVP keeps the surface dry holding colonization to low level and also permitting early surgery (Zellner and Bugyi, 1985).

Daily spray of PVP lotion and neosporin powder on the burn wound forms a 'crust' which sets up a barrier to colonization of bacteria, helps in healing of superficial burn within 2-3 weeks and deep dermal ones heal over few more weeks with limitation of infection.

In treatment of deep burn injuries even though control of infection is important.

It would not be overlooked that even today quite a number of deaths after burn injuries are due to blood volume loss. Early excision of third degree burned skin may be recommended to control infection by many workers but the problem of blood loss that occurs in every surgical intervention should not be under-estimated on the post traumatic volume shift that has not yet been compensated.

In general, the shock phase lasts until there is volume compensation between the intravascular and extra-
vascular fluid. The possible pathways of volume loss from the intravascular space are fluid lost to the interstitial space or from the body surface.

In most patients, weight of the patient increases acutely during initial weight only about 10 days after injury (Peter Zellner, 1976). During this period, important parameters, such as clotting mechanism, normalize only gradually.

Early removal of third degree burned skin appears to be a logical step to eliminate the substrate for necrosis and thereby avert general infection. We should not overlook, however, that the physiologic equilibrium between extracellular and intravascular space has not yet been restored and, therefore, the rate of intraoperative and post operative complications will probably be higher than when the imbalance is corrected. Interestingly, even with early excision no statistically significant lowering of mortality in burn casualties has been generally demonstrated. With very extensive burns, the necroses cannot all be removed in one session. Thus, only a part of the third-degree burned skin can be removed in early excision. The remaining damaged areas must be await later surgical intervention.

The clinical estimation of a burn the differentiation between second and third degree burn is not always early even for experienced surgeons. The part of second degree burn area heals spontaneously within about 2 weeks. For this reason, in an early excision one will not uncommonly
also sacrifice viable areas.

By the above discussion, the possibility of infection should be a consideration in planning the optimal time for surgery. So, if there is no or minimum infection we can postpone the surgery and can grant time for removal of eschar in their due course and can prevent haemorrhage. Deep burn start to loose their eschar in 2 to 4 weeks. In presence of infection, eschar separation is early because of collagenase production, from bacteria, but because of poor presence of granulation tissue and $7 \times 10^5$ bacteria/gram of tissue skin graft cannot be applied to cover the wound.

Boxter et al (1973) first used antibacterial therapy in the subescharal plane by subescharaclysis. This method permits the delivery of high concentration of specific antibiotics into the avascular burn wound interface by multiple needle clysis. Later MacManus et al (1982) also used antibiotics in the subescharal plane. But one thing was common in all studies that antibiotic solution was used in the presence of infection i.e. bacterial count $7 \times 10^5$/gm i.e. when the patient was critically ill, none of them used them prophylactively or in the earlier course of wound infection. Sinha et al (1988) in his study used injection of PVP subescharally first time in all patients of deep burn. That time also a controversy was there as to whether such a procedure (routine PVP-I subescharal injection) was helpful or not? 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 (Zomora, 1984). 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 (Zomora, 1984). In our study we found that patients treated with multiple subescharal injections of PVP show markedly reduced subescharal bacterial colony count as compared to the control group. That subescharal injection of PVP was beneficial, was evident from the result, only one septicemic mortality occurred in the test group of patients. The second beneficial effect is that it opens up a subescharal plane by tanning of non-viable tissue and reduced sticky necrosis thus helping in early escharolysis and devascularing bleeding at separation. Additionally tanned skin produced least infected materials so that transport in lymphatic and blood vessels was minimum. The burn wounds in most of the patients were grafted immediately after the eschar separation because most of the patients showed a colony count far less than \(10^5/\text{gm}\) which is the upper limit below which grafting can easily be done without much fear of graft rejection. In the control group a considerable period of time was spent in limiting the infection at the burn site and infection and less granulation tissue was the cause of graft rejection.

However, reservations have been expressed by some
authors that the large quantity of PVP-I absorbed during treatment can have an effect on the thyroid gland in non-syndrome patients and can cause thyrotoxicosis and thyroid dysfunction. Law and McMillan (1974) reported thyroid toxicity in 2 patients after using PVP lotion (1% available iodine). The high serum iodine concentration can also trigger renal insufficiency but Zellner and Bugyi (1983) in the study of approximately 1500 patients never saw thyroid and renal dysfunction in patients treated with PVP lotion, who were examined several times over a long period of time. In our study none of patients showed any clinical evidence of iodine toxicity.

Zellner and Bugyi in their study with PVP-I in burn patients, measured T₃, T₄, TSH and the iodine level in blood and urine over a period of 21 days. The result showed an initial steep increase in the iodine level in blood and a parallel rise in urine. This increase reached their maximum on second or third day. The high level of iodine is an indicator of the good penetration of PVP-I through the burn wound. Thereafter, the serum iodine concentration falls and a little later the urine iodine concentration falls as well, despite continued administration. An explanation for this phenomenon was given that it could be that either the application of ointment was less or that the healing process was preventing penetration. Most probably penetration was less because of the tanning effect. The iodine levels returned to normal about 1 week after withdrawal.
of treatment. Initial decrease in $T_3$ and $T_4$ pointed to transitory hypothyroidism due to inhibition of iodine deposition because of the high serum iodine concentration. However, a gradual decrease in $T_3$ and $T_4$ was also observed in burn patients by Becker et al. (1980) who had never received topical treatment with iodine so one can say that this change can be brought about by different kinds of stress or disease (Burger et al., 1976). Furthermore, the thyroid hormones bound to proteins also become lost in large quantities because of the high protein loss in burn patients due to exudate and oedema.

The TSH rises because of compensatory hyperfunction on the part of the pituitary, but only temporarily and soon returns to normal values. This clearly show that the organism adapts very well, without irreversible disorders of function due to the increase supply of iodine.

Nakanos (Japan) in 1991 reported chemical burn with PVP-I but we did not see it in our study.

Some times PVP-I injection causes slight pain to patient when we inject either in wrong plane or too much amount is injected or the eschar is attached too tightly.

Hence it can safely be concluded that subescharal injection of PVP is effective in decreasing bacterial count in subescharal plane, early eschar separation and aiding possibility of early grafting and it's better acceptance.