DISCUSSION

NOSOCOMIAL INFECTIONS OF NEWBORN:

Newborn babies are not only vulnerable to becoming infected but also it is unfortunate that they frequently succumb to it. Septicaemia is the leading cause of neonatal mortality in the country. Out of 308 neonatal deaths amongst 7,969 babies born during 1973-77 at the All India Institute of Medical Sciences Hospital, New Delhi. Over one fourth (27.3 per cent) were accounted by sepsis alone, – [Singh, (1978)].

Infections are a frequent and important cause of morbidity and mortality in the neonatal period. As many as 2% of foetuses are infected in utero and up to 10% infants are infected during delivery or the 1st month of life.

Several general factors contribute to the frequency and severity of neonatal infections.

1) A variety of organisms including bacteria, viruses, fungi, protozoa, chlamydia and mycoplasma are etiological agents.
2) The host resistance mechanisms present in the newborn infants are immature and easily overcome by invading microorganisms, infections, therefore, may become fulminant and cause death within few hours or days, despite appropriate and intensive antimicrobial therapy. Many of
bacterial infections are caused by organisms relatively resistant to antibiotics, particularly the Gram negative enteric bacilli. These infections are difficult to treat and the dose of antibiotics that can safely be used is limited by adverse side effects during neonatal period. The presenting clinical features, in the neonate with infection may be subtle and may mimic the features of other common diseases during this period. As a result, the diagnosis of infection is often missed or delayed until the process has become widespread.

A variety of maternal and neonatal factors are associated with increased frequency and severity of infections. An important variable in the increase risk of neonatal sepsis in infants born of mothers with prolonged rupture of membranes is the development of ascending infection of the amniotic fluid called amniotic infection syndrome, in the foetus and subsequent neonatal sepsis. However, amniotic and foetal infection can occur with rupture of membranes for less than 24 hrs. Maternal genital tract may be colonized with wide variety of organisms that do not necessarily cause disease in mother, but may result in a heavy inoculum for the neonate at the time of birth and cause significant illness during the newborn period. These organisms include group B Streptococci, E. coli, Gonococcus, Candida, Listeria, Chlamydia, Herpes simplex virus and Cytomegalo
virus. Difficult or traumatic delivery is associated with an increase frequency of infections during the neonatal period.

The most important neonatal factor predisposing to infection is prematurity, [Glasgow et. al, (1983)]. There is a 3-10 fold higher incidence of Sepsis, meningitis or urinary tract infections in premature infants than in fullterm newborns. Males have an approximately 2 fold higher incidence of Sepsis, meningitis and urinary tract infection than females, suggesting the possibility of a sex-linked factor in host susceptibility, [Nelson, (1992)].

Resuscitation at birth, particularly if it involves endotracheal intubation, insertion of an umbilical vessel catheter, or both, is associated with increased risk of bacterial infection. The majority of infants cared for in a neonatal intensive care unit are exposed to a variety of diagnostic and therapeutic procedures, e.g. umbilical vessel catheters, endotracheal tubes, E. K. G. monitor leads, fetal scalp electrodes, intravenous catheters and so on; that may also compromise host defences and provide a portal of entry for organisms, e.g. Pseudomonas aeruginosa, Coagulase negative Staphylococci, Staphylococcus aureus etc. In addition, these infants may be exposed to antibiotic resistant organisms carried on the hands of personnel or
Outbreaks of infectious illness have occurred in nurseries and neonatal intensive care units due to variety of bacterial and viral agents. Although the most common nosocomial infections in newborn intensive care units are surface infections (EKG lead abscesses, omphalitis, conjunctivitis, pyoderma). More serious infections such as Pneumonia, bacteremia, Surgical wound infections, Urinary tract infection and meningitis account for almost half of those occurring. Staphylococcus aureus has been a major cause of hospital acquired infections and has resulted in out-breaks of pustules and cellulitis, pneumonia, Septicaemia and staphylococcal scalded skin syndrome. A number of Gram negative enteric bacteria including E. coli, Klebsiella pneumoniae, Pseudomonas aeruginosa, Proteus mirabilis, Serratia marcescens and Flavobacterium meningosepticum have resulted in epidemics of pneumonia, sepsis and meningitis.

Basically two factors contribute to a relatively high incidence and increased severity of infections amongst neonates.
i) Poor host resistance:

Both humoral and cell mediated immune response is deficient in the newborn especially amongst those born early and infants with severe intrauterine growth retardation, [Manerikar et al, (1976)].

The materno-foetal transfer of IgG antibodies occurs mostly during the third trimester of pregnancy, Preterm infants because of early birth, are therefore deficient in IgG antibodies. The normal infant lacks IgM antibodies at birth. Because of their large molecular size they cannot permeate the placenta. The newborn baby fails to localize infection due to poor phagocytic ability of the leucocyte and deficient inflammatory response.

ii) Greater exposure to infection:

There are greater opportunities, especially for low-birth weight babies, to become exposed to infection because they demand greater handling and come in contact with a variety of gadgets viz, resuscitators, Incubators, ventilators, catheters, infusion sets, tracheal tubes, masks etc. Warmth and excessive humidity in the nursery is also conductive to bacterial proliferation.
Infections occurring in neonates are acquired from

1. Some infections are transmitted from the mother during labour and delivery.

2. Most infections are produced by organisms acquired in the hospital, although it is often difficult to determine whether the organism was originally acquired from maternal or hospital sources.

Soon after birth of the infant, the skin and mucous membranes are exposed to external environment and becomes populated by the organisms, which are suited to grow at these sites. These organisms constitute the "normal flora". The bacteria of normal flora do not cause disease, but in the neonatal period, the dividing line between normal and invasive flora may be narrow, - [Davies P. A. (1971)].

According to the study conducted by Davies (1971) it was found that when infants are colonized heavily at sites other than rectum, they are likely to have bacterial infections than if lightly or insignificantly colonized. He also asserted that Gram negative bacterial infections are more likely to occur in moderately or heavily colonized, than gram positive or mixed flora.

The common bacteria involved in bacterial infections of the
newborn infants are Gram positive cocci like Staphylococci, Streptococci, Pneumococci and Gram negative bacilli like Esch. coli, Klebsiella spp. Proteus species and Pseudomonas aeruginosa. Since these bacteria are known to colonise, remain a potential source for infections, especially premature infants have immature defences to meet this unique challenge. While full-term infant is equipped with defence mechanisms against infection and can bring them into action when necessary.

NASAL FLORA:

Generally nose is sterile just after birth and becomes colonized after 12 hours when infants come into close contact with their mothers, [Torrey et al., (1945); Evans et al. (1970); Bhatia et al. (1988)] found sterile nasal swabs in 90% of the newborns at birth and in 11% of newborns after 72 hours. Evans et al. (1970), found sterile nasal swabs in 80 to 85% of full-term and premature newborns on first day and in 25 to 39% day 3. While in present study 85% of full-term and 75% premature newborns showed sterile nasal cultures on zero day (just after birth) and 7% premature and 32.5% full-term showed sterile nasal swabs on day 3. (TABLE 1)

Cunliffe (1949) observed a rise in the incidence of
Staphylococcus aureus in the nose of infants from 8.6% on the first day to 76% on the fourth day and about 100% at the end of first week. Forfar et al. (1968) and Dugdale et. al. (1967), have reported 60 and 22% colonization with S. aureus respectively. Bhatia et. al. (1988) found 1% of infants colonized with S. aureus just after birth and 40% of infants after 72 hours of age. However, Evans et al. (1973) and McCallister et. al. (1974), found low incidence of colonization with pathogenic Staphylococci, which was attributed to routine use of hexachlorophene and other anti infective measures; on the other hand Hurst et. al. (1960) reported a colonisation rate as high as 99% with pathogenic Staphylococci at discharge and half of these babies continued to carry these organisms even at the end of first year of life.

In present study 3.12% of premature newborns were colonised with S. aureus just after birth and 15.62% of premature on day 3. While 2.5% of full term newborns were colonised with staph. aureus just after birth and 7.5% on day 3.

In present study the incidence of Coagulase negative Staphylococci (CONS), was found to be higher than S. aureus. On 3rd day 53.12% of premature and 22.5% of full-term newborns were colonised with CONS. (TABLE No. 1), Evans et.
al. (1970), also found *S. epidermidis* was common than *S. aureus* in nares of newborns.

Besides *Staphylococci* 40.27% of premature and full-term showed colonization with Gram negative bacilli, especially with *Pseudomonas aeruginosa* spp. which was found in 23.61% of premature and full-term newborns, followed by *Klebsiella* spp. which was found in 15.25% of premature and full-term newborns.

The 40.27% isolation rate of Gram negative bacilli in the present study from the nose was high as compared to the reported incidence of 6% to 22% in the literature, - [Dugdale et. al. (1967); Evans et. al. (1970); Evans et. al. (1973); Eriksson et. al. (1982); Bhatia et. al. (1988)].

In relation to premature and full-term newborns, there was no significant difference in the yield of nasal swabs.

**THROAT FLORA:**

According to Torrey and Reese (1945), throat is usually free from aerobic organisms till after the infants have come in contact with their mother for feeding as a rate at about 12 hours after birth, Bhatia et. al. (1988) found 91% of newborns showed sterile throat swab culture just after birth.
and 14 %, after 72 hours of life. In present study, 87.5 % of premature newborn showed sterile throat swab culture just after birth and 6.2 % on day 3; while 95 % of full-term showed sterile throat swab cultures just after birth and 52.5 % on day 3.

Mathur et. al. (1967), McCallister et. al. (1974); found Streptococcus viridans as major isolate. Bhatia et. al. (1988), found amongst pathogenic organism, the most common yield was that of E. coli, followed by Klebsiella and S. aureus. McCallister et. al. (1974) on day 3, reported most significant growth of E. coli, Klebsiella and S. faecalis. However, in the study of Mathur et. al. (1967), S. aureus was most common organism followed by E. coli and Klebsiella. In the present study on day 3, the major isolate was coagulase negative Staphylococci, followed by Staph. aureus, E. coli, Pseudomonas aeruginosa, betahaemolytic Streptococci. (TABLE No. 2).

UMBILICAL FLORA :

Torrey and Reese (1945), Hurst (1960), Davies et. al. (1970) have reported that the umbilicus becomes colonised more quickly than the nose, throat or other areas of skin. Martimer et. al. (1966) have reported that the colonization of skin surface starts at the umbilical stump.
McCallister et. al. (1974) found 90% of the newborns showed bacterial colonization on day 3. Bhatia et. al. (1988) found 98% of newborn showed bacterial growth after 72 hours. In present study 93.75% of premature newborns and 57.5% of full-term newborns showed umbilical colonization (TABLE No. 3).

Conventry and Isbister (1951) found 54% of babies to be carrying Staphylococcus aureus on the stump by the third to fourth day. Payne et. al. (1965) and Johnson et. al. (1976) have also reported S. aureus as the predominant organism found at umbilicus. Rotimi and Duerden (1981) also reported S. aureus as a major isolate. Bhatia et. al. (1988) found E. coli and Klebsiella were more common followed by S. aureus and S. epidermidis. They reported 60% of newborns were colonized with E. coli and 47% with Klebsiella. McCallister et. al. (1974) also found Gram negative growths were more prominent, however, S. aureus was isolated in only 2% of the offsprings which was attributed to use of hexachlorophene. Dugdale et. al. (1967) has reported that the S. aureus was colonised even within 24 hours inspite of the use of anti-staphylococcal lotions.

In the present study on 3rd day, 73.6% of premature and full-term newborns were colonized with Coagulase negative Staphylococci, Staphylococcus aureus was found in 31.94%,
E. coli in 23.61 %, Klebsiella in 16.16 % and Pseudomonas aeruginosa in 15.25 %. Citrobacter 9.27 % and Alpha haemolytic Streptococci 2.77 %, (TABLE No. 3).

RECTAL FLORA :

The acquisition of aerobic Gram negative bacilli after birth is rapid in the rectum. E. coli, Klebsiella Spp., Enterococci and Proteus spp. are the most common aerobic organisms.

Bhatia et. al. (1988), reported 96 % of rectal swabs were sterile at birth as against 1 % after 72 hours of life. In present study 87.5 % of newborns showed sterile rectal cultures just after birth and on 3rd day, 30 % of newborns showed sterile rectal cultures (TABLE No. 4).

McCallister et. al. (1974) reported predominant growth of E. coli, Klebsiella Enterobacter cloacae and S. faecalis. Dugdale et. al. (1967), besides E. coli found, high isolation rate of Proteus (38 %) from the rectum on 3rd day. Erikson et. al. (1982), besides E. coli and Klebsiella, found S. aureus in 32 % of faecal specimens, in 7 days old neonates. Rotimi and Durden (1981), found S. faecalis as predominant species followed by E. coli and S. albus. Bhatia et. al. (1988) found 89 % of newborns showed
colonisation with E. coli and 38 % with Klebsiella.

In present study, predominant bacterial species isolated on 3rd day, were E. coli (50.0 %), Klebsiella p. (40.2 %), Coagulase negative Staphylococci (40.27 %), Staphylococci aureus (12.5 %), Pseudomonas aeruginosa (18.06 %). Isolation rate of Klebsiella and E. coli was more in premature than in full-term newborn (TABLE No. 4).

It was observed in the present study that isolation rates of bacterial flora from all the above stated body sites of newborns, were high. These high isolation rates may be due to -

1 - Poor hygenic conditions where the babies were born and transferred to the neonatal intensive care unit.

2 - Lack of complete asepsis in the neonatal care unit itself.

3 - Abscence of the liberal use of antibacterial agents.

The organisms commonly found in nearly all sites of the majority of the newborn were Coagulase negative Staphylococci, Klebsiella and Staphylococcus aureus; Pseudomonas aeruginosa. These organisms have also emerged as significant nosocomial pathogens in many neonatal
When infants are colonised heavily at sites other than rectum, they are likely to have bacterial infections than if lightly or insignificantly colonized (Davies, 1971). This holds true whether such colonization is Staphylococcal - Gillespie et. al. (1958), or Gram negative, - Davies et. al. (1970). Bacterial infections are more likely to occur in those moderatly or heavily colonized with gram negative bacilli, than Gram positive or mixed flora, Davies et. al. (1970).

Goldman et. al. (1978) stated that, the broad spectrum antibiotic therapy administered to most NICU patients on admission may suppress colonization, but other factors, such as separation from mother and altered feeding patterns contribute. When colonization finally occurs Gram negative bacilli often found in the nose, throat and umbilicus. Abnormal Gram negative colonization of neonates has been attributed to use of antibiotics and lack of breast feeding. Colonization with Gram negative bacilli predisposes NICU patients to nosocomial infection.

During recent years remarkable advances have been made in the medical care of sick newborn infants. Regional
intensive care units have been established throughout the world; and accumulating evidence suggests that such care has reduced morbidity and mortality in this high risk population; - [Alden et. al. (1972); Wagman et. al. (1974)]. Such improvement in medical care, however, is not without its risk. Recent surveys in general and pediatric hospitals indicate that patients who have severe underlying disease and who are receiving intensive care have a greater risk that a hospital acquired infection will develop; - [Thoburn et. al. (1968); Finegold DS, (1970); Gardner P, Carles DG, (1972)]. Nosocomial infections may not only prolong hospital stay but also contribute to mortality, - [Thoburn et. al. (1968); Finegold DS, (1970)].

During the study 72 infants with suspected nosocomial infection were studied. In the present study commonest symptoms signs noted were ‘‘not doing well’’ (73.6 %), lethargy (62.5 %), Jaundice (31.9 %), hypothermia (56.9 %), poor sucking (52.7 %). Other symptoms noted were abdominal distension (43.0 %), fever (20.8 %), vomiting (16.6 %) irritability (13.88 %) diarrhoea (9.7 %) and convulsions (2.7 %). Buetow et. al. (1965) reported the presenting symptoms in neonatal septicaemia were jaundice (37%), decreased respiratory exchange (33%), lethargy (31%), apnea (20 %), poor feeding (19 %), pustules cellulitis or other skin infections (18 %), retractions (17 %), vomiting (14 %)
abdominal distension (13 %) and diarrhoea (10 %).

Similar observations were made by Somu et. al. (1976), Mishra et. al. (1985), Mondal et. al. (1991), Sharma et. al. (1993), Piyush Gupta et. al. (1993).

Sinha (1986) analysed clinical features with causative organisms, that tachypnea and fever were mostly seen in septicaemia due to organisms other than Pseudomonas aeruginosa and diarrhoea was significant feature of Pseudomonas aeruginosa Septicaemia.

Khatua et. al. (1986) noted refusal to Suck (92.3 %), lethargy (74 %), diarrhoea (74 %), hypothermia (46.8 %), abdominal distension (60 %) and Jaundice (38 %) as major presenting symptoms.

TOTAL NOSOCOMIAL INFECTIONS :

A total of 72 infants (48 %) showed at least one infection. The site of infection in order of frequency were -

- Septicaemia 59.72 %
- Omphalitis 18.06 %
- Diarrhoea 9.72 %
- (with thrush, conjunctivitis)
- Meningitis 5.55 %

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Out of 72 neonates with clinically suspected nosocomial infection - 45 (62.5 %) cases were male and 27 (37.5 %) cases were females.

8 (11.12 %) cases were of 3 days old, 26 (36.1 %) were between 4 to 7 days and 38 (52.78 %) more than 7 days old as shown in (TABLE No. V).

Sex distribution shows predominance of male, with male : female ratio 1.67 : 1.

Total 60 (83.33 %) neonates were having low birth weight (Weight < 2500 gm) while 12 (16.67 %) cases were having birth weight more than 2500 gm (TABLE No. VI).

The percentage of low birth weight babies of the premature group were found to be the most susceptible, because the infection rates were higher.

The 48 % total nosocomial infection rate in the smallar babies with birth weight less than 2500 gm was significantly higher than 16.67 % rate in the larger babies over 2500 gm at birth. When examined according to body site of infection a significant association was found between lower birth
weight (less than 2500 gm) and higher rate of septicaemia and surface infections.

Hemming et. al. (1976) found the infection rate of 56.4 % in the smaller babies with a birth weight less than 1500 gm.

Sinha (1986) observed the incidence of 64.9 % in low birth weight babies. Similar findings were reported by Buetow et. al. (1965); Philips et. al. (1980); Sharma et. al. (1993).

TABLE No. VII shows that 55.55 % cases were full-term and 44.45 % were preterm neonates. It was noted from Table Nos. V, VI and VII that there is a male predominance in cases of nosocomial infections of neonates with 62.5 % being males and 37.5 % being females.

NEONATAL SEPTICAEMIA:

In the neonatal period the infant may suffer from a general blood-stream bacterial infection. Which has been acquired antenatally or postnatally. Septicaemia is an important and relatively frequent cause of morbidity and mortality of newborn infants - [Yippo A, (1990)]. There is a general agreement that septicaemia is disproportionately high in premature infants and is higher in males than in females, - [Silverman, (1961)].
Moncrieff (1953), in a series in Britain, found evidence that suggested that sepsis neonatorum was responsible for 15.4% of deaths in the first 30 days of life. He stated that the incidence must be very much higher in the poor hygienic conditions in most of the subtropics and tropics and it is very possible that it is one of the main cause of neonatal mortality.

In the present study, 43 (59.72%) cases were diagnosed and also bacteriologically proven to have septicaemia, out of 43 cases 33 (76.75%) patients were male and 10 (23.25%) patients were females. Buetow et. al. (1965) reported male incidence rate as 6.5% as compared with 4.5 per cent for female. Gluck (1966) reported high incidence of male. The male : female ratio 1.7 : 1.

Somu et. al. (1976) stated increase in incidence of septicaemia in male as compared to female accounting for 54.6 per cent in male and 45.9 per cent in female. Krishna Das et. al. (1978) stated similar findings of increase in incidence of septicaemia in male.

Kahtua et. al. (1986) stated increase in incidence of septicaemia in male is due to genetic constitution.

Female’s resistance to infection stems from her
heterozygocity for genes of chromosomes controlling immunoglobulin synthesis which results in a greater heterogeneity of antibody response, - [Washburn et. al. (1965)].

Females have a greater genetic diversity resulting from random inactivation of one of the two chromosomes, so that in some body cells the paternally derived chromosome is active and in others maternally derived chromosomes is active; - [Schilegel and Bellanti, (1969)].

Maya Raghvan et. al. (1992) stated that the consanguinity among parents, birth order and sex of the baby did not increase the risk of septicaemia. There was significant increase in the risk for septicaemia when the duration of labour was more than 24 hours; time interval between rupture of membrane and delivery of baby was more than 12 hours; liquor was muconium stained or foul smelling and delivery was operative. The neonatal factors identified with risk of septicaemia were pre-term delivery, low birth weight, birth asphyxia, assisted ventilation and intravenous alimentation.

Khatua et. al. (1986) reported high incidence of septicaemia in males (70.2 %) and in prematures (63 %). Vanita Kulkarni (1989) reported that out of 68 cases of septicaemia 37 were male and 31 were female accounting for 54.41 % and 45.59 % respectively.
Piyush Gupta et. al. (1993) reported high incidence of Klebsiella septicaemia in male (64.7 %) and stated that preterm and low birth weight babies accounted for 85.5 % cases of Klebsiella septicaemia and majority of newborns acquired this within first 10 days of life.

In the present study out of 72 samples, blood culture was positive in 43 (59.73 %) and culture was negative in 20 (27.77 %) cases and contaminants were grown in 9 (12.5 %) cases.

Negative cultures do not exclude sepsis. Somu et. al. (1976) reported negative blood cultures in 25 per cent cases. Squire et. al. (1979) reported 20 per cent negative blood culture and found seven cases with negative blood cultures, fatal illness and postmortem evidence of infection.

Monga et. al. (1986) found negative blood culture in 32 to 33.5 per cent cases.

Vanita kulkarni (1989) found negative blood culture in 18 samples (26.47 per cent).

Khatua et. al. (1986) reported the incidence of positive blood culture of 59.8 %. Namdeo et. al. (1985) showed
incidence of positive blood culture in 50% of neonatal septicaemia. Vanita Kulkarni (1989) reported the incidence of positive blood culture of 60.29 per cent.

However, Chaudhary et. al. (1975) have reported the incidence of positive blood culture in 30.6%. While Mittal et. al. (1980) have reported it is of 74.2% of neonatal septicaemia. A substantial number of patients who appear clinically to be bacteraemic do not have positive blood culture. Some of these are due to prior administration of antibiotics [Gould and Duerden, (1983)].

Contamination rate is 12.5 per cent in present study. The contamination rate varies from place to place. Buetow et. al. (1965) reported contamination rate as 4 per cent. Todd and Roe (1975) stated contamination rate was 1.8 - 2.8 per cent. MacGregor and Beaty (1972) reported in their study the contamination rate varies from 4 per cent to 12 per cent. Craint et. al. (1976) while studying a decade from 1966-1975 reported increasing rate of contamination from 1.6 percent to 11.6 per cent.

In this study findings of contamination are based on following criteria - 1) Isolation of organisms from single culture bottles, 2) Isolation of two different organisms from two different bottles, 3) Isolation of organisms
normally considered as contaminants like B. Subtalis.

The contamination rate in our study is on the higher side, because the contamination can occur at any stage of processing of the sample. Improper cleaning of the skin leads to contamination and media can get contaminated very easily. So chances of contamination are more. Though we have checked every batch of medium for contamination before using, possibilities of contamination could not be ruled out.

GRAM NEGATIVE SEPTICAEMIA :

TABLE No. VIII - shows organisms isolated repeatedly in blood, in 1st and 2nd week of hospital stay of newborns.

Changing pattern of isolates from blood of septicaemic infants is seen from early days Streptococcus which was predominant pathogen during 1930 - 1943 replaced by E. coli in 1944 and Pseudomonas aeruginosa emerged as a second most common pathogen (Nyhan et. al. (1958)]. From this started the era of Gram negative organisms. Gluck from 1957 to 1965 noted high incidence of E. coli as etiologic agent followed by Klebsiella. Gotoff and Behram (1970) from 1963 - 1968 and Freedman et. al. (1981) from 1966 - 1978 noted similar findings. Choudhary et. al. (1975) showed high incidence of
Klebsiella pneumoniae followed by Staphylococci. Piyush Gupta et al. (1993) noted high incidence of Klebsiella in about 40% cases. Mandira Banerajee (1993) reported high incidence of (70.2%) Klebsiella.

Similar findings are obtained in this study. We have detected Klebsiella pneumoniae (51.16%) followed by Coagulase negative Staphylococci (18.6%). Pseudomonas aeruginosa (16.28%), Staph. aureus (6.98%), E. coli (4.65%), Beta Strept. (2.33%) (TABLE No. VIII).

Yardi et al. (1984), Pandit et al. (1985), Khatua et al. (1986), Monga et al. (1986) showed dominance of Klebsiella pneumoniae in majority of cases. In 1987 Sharma et al. reported E. coli as predominant pathogen in septicaemia of infants and Klebsiella pneumoniae as next common pathogen.

In present study the Gram negative Septicaemia is more predominant i.e. 72.09 per cent. The high incidence of Gram negative septicaemia as compared to Gram positive septicaemia has been reported by various workers. MacCracken and Shinefield (1966) stated increase in incidence of Gram negative septicaemia over five years. Similar findings were stated by Buetow and Klein (1965), Bhakoo et al. (1968), Bhakoo et al. (1973) and Choudhary P. et al. (1975).
In 1976 Sornu et. al. while analysing septicaemia in infancy found coliform groups of organisms were the commonest cause of septicaemia giving incidence of 64.3 per cent. Guha et. al. (1978) while studying 400 newborn with septicaemia found 72.2 per cent gram negative septicaemia.

In 1984, Yardi et. al. studied 326 cases of septicaemia in paediatric age group and showed 59.9 per cent to be positive for growth with increase in incidence of Gram negative septicaemia.

Finn (1986) stated Gram negative septicaemia is becoming more common among hospital patients. This may be due to growing number of hospital patients at high risk, who have severe debilitating disease or undergoing major surgery transplantation or immunosuppressive therapy. Indiscriminate use of antibiotics is partly responsible for it.

Sharma et. al. (1987) stated Gram negative septicaemia in neonates accounted for 85 per cent. In 1993 Sharma reported the Klebsiella pneumoniae in 30 % followed by Staphylococcus aureus in (20 %) and Pseudomonas in (10 %) of cases of neonatal septicaemia. However, Sinha in 1986 found predominantly Pseudomonas aeruginosa (40.7 %) followed by Klebsiella (22 %), E. coli (18.3 %), Staph. pyogens (11 %).
Namdeo et. al. (1985) observed E. coli (29 %), Klebsiella (21 %), Pseudomonas aeruginosa (25 %) and Staph. aureus (21 %) in positive cases of neonatal septicaemia.

From last two decades, it shows that there is an increase in incidence of Gram negative septicaemia in infants. It represents an intensification of factors contributing to gradual increase in Gram negative septicaemia. This may occur as a complication of a number of localized Gram negative infections such as meningitis, pneumonia and urinary tract infections. Gram negative septicaemia occurring in association with any such localized infection implies a severe dissemination of infection, - [Forfar and Arneil, (1978)].

Another possible contributing factor may be increase in intrapartum infections lead to increase in Gram negative septicaemia, - [MacCracken and Shinefield, (1966)]. Kishore et. al. (1987) studied vertical transmission of aerobic microorganisms in mother and neonates and concluded that Gram negative pathogens predominated both in maternal vagina and neonatal colonization sites.
GRAM POSITIVE SEPTICAEMIA:

In present study Gram positive septicaemia accounted for 27.91%. The Gram positive Septicaemia which was common during 1927-32 gradually replaced by Gram negative septicaemia.

Dunham, 1927-1932 reported 66.7 per cent Septicaemia caused by Gram positive organisms which was dominated over Gram negative organisms Nyham 1933-43 reported Gram positive organisms responsible for septicaemia equally as Gram negative organisms.

After introduction of antibiotics the incidence of Gram positive septicaemia decreased considerably. The use of hexachlorophene cleansers decrease Staphylococcal septicaemia to a considerable degree [Gluck, Wood (1964)]. Indiscriminate use of antibiotics and resistance to antibiotics again contributed for rise in Gram positive septicaemia. Somu et. al. (1974) and Choudhary et. al. (1975) noted the incidence 35.7 per cent and 31 per cent respectively.

The isolation of Coagulase negative Staphylococci from the blood is much discussed. It is often refered as universal saprophyte of a skin and mucus membranes prolonged coagulase
negative staphylococcal bacteraemia and clinical sepsis suggests that these organisms are significant pathogens even found in single blood culture, - [Munson et. al. (1982)].

Choudhary et. al. (1975) noted 22.9 per cent incidence of coagulase positive Staphylococci as second most common pathogens. Battisti and Mitchison (1981) stated that Staphylococcus epidermidis was the organism most often isolated from both in early and in later life.

In 1976, Somu et. al. in their study from 1973-74 noted Staphylococcus both coagulase negative and coagulase positive were responsible for septicaemia.

In present study, 32 premature newborns (74.42 %) and 1 full terms newborns (25.58 %) all with low birth weight (birth weight between 1.6 to 2.2 kg) were diagnosed and also bacteriologically proven to have septicaemia. The causative organisms in these cases were Klebsiella pneumoniae (51.16%), followed by coagulase negative Staphylococcus (.18.6%), Pseudomonas aeruginosa (16.28 %), Staph. aureus (6.98 %) E. coli (4.65 %) and beta-haemolytic Streptococci (2.33 %). (TABLE No. VIII)

These causative organisms were also isolated from other body sites of infected newborns. These pathogenic organisms also showed multiple drug resistance. It means that these
organisms were acquired by neonates from hospital environment and it was also clear that the organisms colonizing are also responsible to cause disease. Bhatia et. al. (1989) and D'Angio et. al. (1989) also found that the organisms colonizing at various sites remains a potential source of infection in newborns.

Klebsiella and Coagulase negative Staphylococci are the major blood culture pathogens in our study patients. The other workers also reported that these two organisms are major blood culture isolates in many nursery units, - [Battisti et. al. (1981); Monga et. al. (1986); Singha et. al. (1986); Sharma et. al. (1987); D'Angio et. al. (1989)].

Out of the various individual tests for rapid diagnosis of neonatal septicaemia in proved sepsis group, C. R. P. emerged with maximum sensitivity of 83.72 % and with specificity of 90.0 per cent (TABLE No. IX).

In 1987 Singh et. al. found that C. R. P. was the most sensitive (80 %) test with specificity of 91.0 per cent. Squire et. al. found C. R. P. as the most sensitive (86 %) in distinguishing infected from non infected babies. Similar observations were made by Mishra (1989) and Kite et. al. (1988). However, Philips (1980) found sensitivity of 47 % and specificity of 86 % while Khatua et. al. (1986) reported the sensitivity 47 percent, specificity 86 % for C. R. P.
Chandana et. al. (1988) observed the sensitivity of 83 % but only 42 % specificity of C. R. P.

Superficial infection of newborn:

Out of 72 cases of nosocomial infection 29 cases showed surfaces infections and other serious conditions i.e. Meningitis, Diarrhoea, Jaundice (TABLE No. X) Omphalitis - umbilical sepsis was the predominant (18.06 %), followed by Diarrhoea with conjunctivities and Thrush - 9.72 %, Meningitis - 5.55 %, Jaundice - 4.17 %, Pustules - 1.39 % and Staphylococcus Scalded Syndrome - 1.39 %.

The total surface infection accounted for 20.83 %. Coagulase negative Staphylococci was predominant organism responsible for umbilical sepsis. Klebsiella pneumonia was predominant causative organisms for meningitis, while E. coli and Coagulase negative Staphylococci were responsible for diarrhoea.

Hemming et. al. (1976) detected a 24.6 per cent nosocomial infection rate among 904 infants hospitalized for 48 hours during 41 month of surveillance. In their study surface infections accounted for 40.1 per cent of the total, pneumonia for 29.3 per cent, bacteraemia for 14.0 per cent, surgical wound infection for 8.1 percent, urinary tract
infection for 4.5 percent and meningitis 4.0 per cent. Staphylococcus aureus (47.3 per cent) and gram negative enteric bacilli (45.1 per cent) were the most common organism recovered. Nosocomial infection rates were highest in infants with a birth weight less than 1500 gm.

Meharban Singh in 1978 reported that during 1973-77; 7.8 percent neonate developed superficial infection, conjunctivitis accounted for almost two third of cases, followed by pyoderma (15 %) Thrush (10 %) and umbilical sepsis (5 %), this low incidence of umbilical sepsis was due to routine prophylactic use of triple dye over the cord. Infective diarrhoea was seen in 3.5 % infants. Septicaemia was developed in 1.9 % neonates. Similar incidence of blood culture positive septicaemia among newborn babies has been reported by Bhakoo et. al. (1974), Pohowall et. al. (1960) reported 18.2 % incidence of superficial infections.

In present study, the overall incidence of superficial infection was 20.83 % and septicaemia, meningitis and Diarrhoeae together accounted for 79.17 %.

Antibiotic Sensitivity :

Antibiotic sensitivity : Pattern of Staphylococci isolated from different clinical sources of new borns are shown in
TABLE No. XI.

It was seen that 3.22 percent of Coagulase positive Staphylococci were sensitive to penicillin. In series of Wasek et. al. (1965) it was found to be 3 %. Bhargava et. al. (1966) found 2.8 % sensitivity. Srivastava et. al. (1969) reported 5 % sensitivity. Vinodkumar et. al. (1979) found 9 % sensitivity. Yardi et. al. (1984) found 23.6 % sensitivity. Usha Udgaonkar et. al. (1985) found 15 % sensitivity. Our series is comparable with Wasek et. al. (1965) and Bhargava et. al. (1966).

Streptomycin sensitivity of Staphylococcus aureus was 24.19% in our series. (TABLE No. XI) Wasek et. al. (1965) found 10% sensitivity. Bhargava et. al. (1966) found 57.14 % sensitivity. Srivastava et. al. (1969) found 13 % sensitivity. Vinodkumar et. al. (1979) and Prabhakar and Arora (1979) recorded 38 % and 47.5 % sensitivity. Yardi et. al. (1984) found 21.0 %. Usha Udgaonkar et. al. (1985) found 26.6 % sensitivity.

Tetracycline sensitivity of Staphylococcus aureus in our series was 53.22 % (TABLE No. XI). In series of Wasek et. al. (1965) it was 26 %. Bhargava et. al. (1966) reported 42.85 %. Srivastava et. al. (1969) found it to be 75 %. Vinodkumar et. al. (1979) and Prabhakar and Arora et. al.
Yardi et al. (1979) found it to be 52.38 % and 27 %. Yardi et al. (1984) found 10.5 % sensitivity. Usha Udgaonkar et al. (1985) reported 15 % sensitivity. Our series compared with that of Vinodkumar et al. (1979).

The sensitivity of Staphylococcus aureus to Kanamycin in our series was 50.0 %. Vinodkumar et al. (1979) and Prabhakar and Arora (1979) found it 66.66 % and 81.1 % respectively. Yardi et al. (1984) found it to be 39.4 % sensitivity. Usha Udgaonkar et al. (1985) found it to be 39.56 %.

The sensitivity of Staphylococcus aureus to chloromycetin in our series was 50 % and that in the series of Wasek et al. (1965) it was 88 %. Bhargava et al. (1966) it was 57.14 %. In series of Srivastava et al. (1969) it was 37 %. Vinodkumar et al. (1979) found it to be 71.42 % and Prabhakar and Arora (1979) found it to be 52.9 %. Yardi et al. (1984) found 21 %. Usha Udgaonkar et al. (1985) reported 35.97 %. Our series is comparable with Bhargava et al. (1966) and Prabhakar and Arora (1979).

The sensitivity of Staphylococcus aureus to Erythromycin in our series was 37.09 %. In series of Wasek et al. (1965) it was 93 %. Bhargava et al. (1966) reported 34.28 % sensitivity. Srivastava et al. (1969) it was 87 %. Vinodkumar et al. (1979) reported 66 %. Yardi et al. (1979) found it to be 52.38 % and 27 %.
(1984) found it to be 26.3 %. Usha Udgaonkar et. al. (1985) found it 39.56 %. Our series is more or less comparable with that of Bhargava et. al. (1966) and Usha Udgaonkar et. al. (1985).

Our series showed maximum sensitivity of Staphylococcus aureus to Gentamycin 72.58 % while Vinodkumar et. al. (1979) and Prabhakar and Arora (1979) found cent per cent sensitivity. Yardi et. al. (1984) found it 44.7 %. Usha Udgaonkar et. al. (1985) found it to be 62.58 %.

Our series showed sensitivity of Staphylococcus aureus to Tarivid (cefloxin) 53.22 %, Netromycin - 43.54 %, Bactrim - 24.19 % and Omnatax - 22.58 % and Methicillin - 16.12 %.

Coagulase negative Staphylococci:

It is now wellknown that coagulase negative Staphylococci isolated from hospital patients are often resistant to antibiotics than our patients cases.

Antibiotic sensitivity of CONS to penicillin in our series was 35.67 %. Usha Udgaonkar et. al. (1985) reported 13.63 % sensitivity. Jayanti Phatak (1991) reported 77.4 % resistance to penicillin.
Tetracycline sensitivity of Coagulase negative Staphylococci in our series was 49.24%. Usha Udgaonkar et al. (1985) found 18.18%.

Streptomycin sensitivity of Coagulase negative Staphylococci in our series was 41.20%. Usha Udgaonkar et al. (1985) found 25.45% sensitivity.

Kanamycin sensitivity of Coagulase negative Staphylococci in our series was 17.58%. Usha Udgaonkar et al. (1985) reported 55.45% sensitivity.

Chloromycetin sensitivity of Coagulase negative Staphylococci in our series was 34.17%. Usha Udgaonkar et al. (1985) reported 55.45% sensitivity.

Erythromycin sensitivity of Coagulase negative Staphylococci in our series was 35.67%. Usha Udgaonkar et al. (1985) reported 37.72% sensitivity.

Ampicillin sensitivity of Coagulase negative Staphylococci in our series was 17.58%. Usha Udgaonkar et al. (1985) found 53.63% sensitivity. Jayanti Phatak (1991) reported 72.6% resistance to Ampicillin.

Gentamycin sensitivity of Coagulase negative Staphylococci
in our series was 71.35 %. Usha Udgaonkar et. al. (1985) reported it to be 76.36 %. Jayanti Phatak (1991) observed minimum resistance to Gentamycin 21.6 %.

Antibiotic sensitivity of coagulase negative Staphylococci to Tarivid was 74.41 % to Bactrim 53.48 %.

**BETahaemolytic STREPTOCOCCI**

Betahaemolytic streptococcus infection is of trivial importance now a days; a change no doubt brought about by antibiotics, (Williams et. al. 1966). Thoburn et. al. (1968) found it to be 10.6 %. In Indian series Bhargava et. al. (1966) recorded 2 % incidence Prabhakar and Arora (1979) reported 1.8 % and Vinodkumar et. al. (1979) reported 2.3 % incidence. Usha Udgaonkar et. al. (1985) found 1.01 % incidence. In our series it was 2.77 %.

Sensitivity of betahaemolytic Streptococci to penicillin was 0 %. Bhargava et. al. (1966) reported 0 % sensitivity. Vinodkumar et. al. (1979) reported 100 % sensitivity. Yardi et. al. (1984) found 100 % sensitivity. Usha Udgaonkar et. al. (1985) found 0 % sensitivity. Our series compare with Bhargava et. al. (1966) and Usha Udgaonkar et. al. (1985).

Streptomycin sensitivity of betahaemolytic Streptococci in
our series was 0 %. Bhargava et. al. (1966) found 50 %
sensitivity. Vinodkumar et. al. (1979) reported 100 %
sensitivity while Prabhakar and Arora (1979) showed 77.7 %.
Yardi et. al. (1984) observed 100 % sensitivity. Usha
Udgaonkar et. al. (1985) found 100 % sensitivity.

Tetracycline sensitivity of betahaemolytic Streptococci in
our series was 0 %. Vinodkumar et. al. (1979) found 100 %
sensitivity. Prabhakar and Arora (1979) found 55.5 %
sensitivity. Yardi et. al. (1984) found it to be 25 %.
Usha Udgaonkar et. al. (1985) reported 0 % sensitivity.

Chloromycetin sensitivity of betahaemolytic Streptococci in
our series was 75 %. Bhargava et. al. (1966) reported 50% 
sensitivity while Vinod Kumar et. al. (1979) reported 100 %
sensitivity. Prabhakar and Arora (1979) found 66.6 %
sensitivity. Yardi et. al. (1984) found 75 % sensitivity.
Usha Udgaonkar et. al. (1985) reported 100 % sensitivity.

Erythromycin sensitivity to betahaemolytic Streptococci in
our series was 100 %, and in series of Bhargava et. al.
(1966), Vinod Kumar et. al. (1979) and Usha Udgaonkar et.
al. (1985). Yardi et. al. (1984) found it to be 75 %.

Ampicillin sensitivity of betahaemolytic Streptococci in our
series was 50.0 %. Prabhakar and Arora (1979) found it
Yardi et al. (1984) reported 75% sensitivity. Usha Usgaonkar et al. (1985) found it to be 100%. Our series compares more or less that with Prabhakar and Arora (1979).

Gentamycin sensitivity of beta-haemolytic Streptococci in our series was 100%. Prabhakar and Arora (1979), Yardi et al. (1984) and Usha Udgaonkar (1985) reported the same.

In our series Tarivid sensitivity of beta-haemolytic Streptococci was 50% and Netromycin sensitivity 50% and Bactrium 50%.

The antibiotic sensitivity of alpha-haemolytic Streptococci to penicillin was 73.33%, Erythromycin, Gentamycin and Tarivid (cefloxcin) sensitivity was 93.33%. Chloromycetin sensitivity was 86.66%. Ampicillin sensitivity was 86.66%. Netromycin sensitivity was 86.66% and to Streptomycin 86.66% sensitivity.

Antibiotic sensitivity of Gram negative organisms E. coli, Klebsiella pneumoniae, Citrobacter, Pseudomonas aeruginosa, Proteus has been shown in TABLE No. XI.

Gram negative organisms appear relatively more resistant to the routinely used antibiotics.
While considering individual gram negative organisms we found E. coli was 100% resistant to penicillin. Wasek et. al. (1965), Bhargava et. al. (1966), Srivastava et. al. (1969), Vinod Kumar et. al. (1979) and Yardi et. al. (1984) had reported 100% resistance, while Usha Udgaonkar et. al. (1985) found 9% sensitivity.

Streptomycin sensitivity of E. coli in our series was 42.66%. Wasek et. al. (1965) reported 7.8% sensitivity; Bhargava et. al. (1966) reported 40%. Srivastava et. al. (1969) have recorded 16.6%. Vinodkumar et. al. (1979) found 27.7% sensitivity, where as Prabhakar and Arora (1979) reported 40% sensitivity. Manorama Deb et. al. (1982) reported 34% sensitivity, Yardi et. al. (1984) found it to be 9.1%. Usha Udgaonkar et. al. (1985) find it to be 62.85%. Our series compared with that of Bhargava et. al. (1966) and Prabhakar and Arora (1979).

Tetracyclin sensitivity of E. coli in our series was 48.0%. In those of Wasek et. al. (1965) it was 47%, Bhargava et. al. (1966) reported 20% sensitivity. Srivastava et. al. (1969) recorded 66.6%, Prabhakar and Arora (1979) recorded 6% sensitivity while Vinod Kumar et. al. (1979) reported 22%. Manorama Deb et. al. (1982) reported 23.7% sensitivity. Usha Udgaonkar et. al. (1985) reported 14% sensitivity. Our series compared with Wasek et. al. (1965).
Kanamycin sensitivity of E. coli in our series was 53.33 %, while that in series of Vinod Kumar et. al. (1979) it was 83 % and in series of Prabhakar and Arora (1979) it was 64 %. Manorama Deb et. al. (1982) found 45 % sensitivity, Yardi et. al. (1984) found it 36.3 %. Usha Udgaonkar et. al. (1985) found it to be 55 %. Our series compared with Usha Udgaonkar et. al. (1985).

Chloromycetin sensitivity of E. coli in our series was 42.66 %, while Wasek et. al. (1965) found it to 89 %, Bhargava et. al. (1966) found 60 %, Srivastava et. al. (1969) found 83 % sensitivity, Prabhakar and Arora (1979) found 45 % and Vinod Kumar et. al. (1979) found 55 % sensitivity. Manorama Deb et. al. (1982) found 64.2 % sensitivity. Yardi et. al. (1984) reported 9.1 % sensitivity. Usha Udgaonkar et. al. (1985) found 55 % sensitivity. Our sensitivity of chloromycetin to E. coli was nearer to that of Prabhakar and Arora et. al. (1979).

Ampicillin sensitivity of E. coli in our series was 53.33 %, while in series of Prabhakar and Arora (1979) it was 11 %. Manorama Deb et. al. (1982) found it to be 9.5 %. Yardi et. al. (1984) found 27.3 % sensitivity. Usha Udgaonkar et. al. (1985) found 30 %.

Erythromycin sensitivity of E. coli in our series was 0 %.

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Bhargava et. al. (1966) reported it to be 20 %. Wasek et. al. (1965) and Srivastava et. al. (1969) recorded 0 %. Vinod Kumar et. al. (1979) have reported 11 % sensitivity. Usha Udgaonkar et. al. (1985) found it to be 11 %. Our series compared with that of Wasek et. al. (1965), Srivastava et. al. (1969).

Gentamycin sensitivity of E. coli in our series was 93.33 %. Vinodkumar et. al. (1979) and Prabhakar and Arora (1979) recorded 100 % and 98 % respectively. Manorama Deb et. al. (1982) found 86 % sensitivity. Yardi et. al. (1984) found 54.5 %. Usha Udgaonkar et. al. (1985) found it to be 82 %.

It was observed that sensitivity of E. coli to Tarivid was 86.66 %, to Netromycin it was 84 % and to Omnatax it was 80% in our series. It was observed that sensitivity of E. coli to higher and newer antibiotics is more as compared to old routinely used antibiotics.

Proteus Species:

Streptomycin sensitivity of Proteus in our series was 25 %. Wasek et. al. (1965) found it to be 25 %. Bhargava et. al. (1966) found 50 %. Srivastava et. al. (1969) found 10 %, Prabhakar and Arora (1979) found 36 % and Vinod Kumar et. al. (1979) found it to be 50 %. Manorama Deb et. al. (1982)
found 44.5% sensitivity. Usha Udgaonkar et. al. (1985) found 35% sensitivity. Our series compared with that of Wasek et. al. (1965).

Tetracyclin sensitivity of Proteus in our series was 25%. Wasek et. al. (1965) found it to be 16%, Bhargava et. al. (1966) 70%, Srivastava et. al. (1969) found 20%. Vinod Kumar et. al. (1979) and Prabhakar and Arora (1979) found it to be 14% and 10% respectively. Manorama Deb et. al. (1982) found 44.5%. Usha Udgaonkar et. al. (1985) found it to be 8%. Our series compared more or less with Srivastava et. al. (1969).

Kanamycin sensitivity of Proteus in our series was 50%, while that in series of Vinod Kumar et. al. (1979) and Prabhakar and Arora (1979) it was 71% and 36% respectively. Manorama Deb et. al. (1982) recorded 72%. Yardi et. al. (1984) found 50% sensitivity. Usha Udgaonkar et. al. (1985) recorded 26% sensitivity.

Ampicillin sensitivity of Proteus in our series was 25%, while Prabhakar and Arora (1979) found it to be 12%. Manorama Deb et. al. (1982) found 44% sensitivity. Yardi et. al. (1984) found 25%. Usha Udgaonkar et. al. (1985) found it to be 34%.

Erythromycin sensitivity of Proteus in our series was 0%.
Same findings were shown by Wasek et. al. (1965) Srivastava et. al. (1969). Vinod Kumar et. al. (1979), Prabhakar and Arora (1979) and Usha Udgaonkar et. al. (1985). However, Bhargava et. al. (1966) found it to be 40%.

Chloromycetin sensitivity of Proteus in our series was 25%. Wasek et. al. (1965) found 75% sensitivity. Bhargava et. al. (1966) reported 70% sensitivity. Srivastava et. al. (1969) found 50%, Prabhakar and Arora (1979) found 28%, Vinod Kumar et. al. (1979) reported 64% sensitivity. Manorama Deb et. al. (1982) reported 65% sensitivity. Usha Udgaonkar et. al. (1985) reported 15% sensitivity. Our series compared more or less with Prabhakar and Arora (1979).

Gentamycin sensitivity of Proteus in our series was 75%, while those in series of Prabhakar and Arora (1979) and Vinod Kumar et. al. (1979) it was 99% and 100% respectively. Manorama Deb et. al. (1982) found 93.5%, Yardi et. al. (1984) found 50% sensitivity. Usha Udgaonkar et. al. (1985) found it to be 48%.

Antibiotic sensitivity of Proteus to Tarivid and Netromycin in our series was 75%.
Klebsiella Species:

Sensitivity of Klebsiella to penicillin in our series was 0%. It was 0% in series of Bhargava et al. (1966) and Vinod Kumar et al. (1979). Nalwade (1982) reported 6.4% sensitivity. Yardi et al. (1984) found it to be 5.33%. Usha Udgaonkar et al. (1985) found it to be 4%.

Streptomycin sensitivity of Klebsiella in our series was 24.41%. In Bhargava et al. (1966) it was 46% and in Vinod Kumar et al. (1979) found 33.33%. Manorama Deb et al. (1982) found it to be 11.0%, Nalwade (1982) found 38.0%, Yardi et al. (1984) found 6.6% sensitivity. Usha Udgaonkar et al. (1985) reported 14% sensitivity.

Tetracycline sensitivity of Klebsiella in our series was 27.90%. Vinod Kumar et al. (1979) found it to be 33%. Manorama Deb et al. (1982) found 26.6% sensitivity. Yardi et al. (1984) found 4% sensitivity. Usha Udgonkar et al. (1985) found 8% sensitivity.

Kanamycin sensitivity of Klebsiella in our series was 87.2%, while Vinod Kumar et al. (1979) recorded it to be 83%. Manorama Deb et al. (1982) recorded 60% sensitivity. Nalwade (1982) found 40%. Yardi et al. (1984) found 28% sensitivity. Usha Udgaonkar et al. (1985) recorded it to be 41%.
Chloromycetin sensitivity of Klebsiella in our series was 13.95%. It was 69% in the series of Bhargava et. al. (1966) and 50% in series of Vinod Kumar et. al. (1979). Manorama Deb et. al. (1982) reported 25.5%. Nalwade (1982) found 16.8% sensitivity. Yardi et. al. (1984) found 6.6% sensitivity. Usha Udgaonkar et. al. (1985) found 10% sensitivity. Our series compared more or less with Nalwade (1982), Usha Udgaonkar et. al. (1985).

Erythromycin sensitivity of Klebsiella in our series was 24.41%. In Bhargava et. al. (1966) it was 23%. In Vinod Kumar et. al. (1979) it was 16%. Nalwade (1982) found it 6.8%. Yardi et. al. (1984) recorded 13.3%. Usha Udgaonkar et. al. (1985) recorded it to be 7%. Our series compared with Bhargava et. al. (1966).

Ampicillin sensitivity of Klebsiella in our series was 26.74%. Manorama Deb et. al. (1982) reported 3% sensitivity. Nalwade (1982) found 3.2%. Yardi et. al. (1984) found 28% sensitivity. Usha Udgaonkar et. al. (1985) reported it to be 30%.

Klebsiella was 83.72% sensitive to Gentamycin in our series. It was 100% sensitive in series of Vinod Kumar et. al. (1979) and 64% in series of Nalwade (1982). Manorama Deb et. al. (1982) reported 92.0% sensitivity. Yardi et.
al. (1984) found 48 % sensitivity. Usha Udgaonkar et. al. (1985) reported 60 % sensitivity.

Tarivid sensitivity of Klebsiella in our series was 50.0 %.

Mandira Banerjee et. al. (1993) reported Klebsiella pneumoniae were resistant to all drugs except third generation cephalosporins (ceftriazone sodium, ceftazidime, cefotaxime and ciprofloxacin to a variable extent), while Piyush Gupta et. al. (1993) observed 14 % resistant to all antibiotics, Kenneth et. al. (1993) observed nosocomial outbreaks of Klebsiella infection resistant to late generation cephalosporins.

**Pseudomonas aeruginosa**

Ampicillin sensitivity of Pseudomonas aeruginosa was 19.64 % in our series. Prabhakar and Arora (1979) found it to be 14 %. Manorama Deb et. al. (1982) found 0 %. Sumitra Jain et. al. (1983) found it to be 6.6 %. Yardi et. al. (1984) found it to be 20.6 %. Usha Udgaonkar et. al. (1985) found 4.5 % sensitivity. Bal and Bhalla (1988) found 0 % sensitivity.

Sensitivity of Pseudomonas to penicillin in our series was 0 % and in series of Wasek et. al. (1965) Bhargava et. al.
(1966), Srivastava et. al. (1969) and Vinod Kumar et. al. (1979), Usha Udgaonkar et. al. (1985). Yardi et. al. (1984) found it to be 6.5 \%.

Streptomycin sensitivity of Pseudomonas in our series was 37.5 \%. It was 0 \% in series of Wasek et. al. (1965), Bhargava et. al. (1966) found 20 \%, 0 \% in Srivastava et. al. (1969). Prabhakar and Arora (1979) found 18 \% and in Vinodkumar et. al. (1979) it was 4.5 \%. Manorama Deb et. al. (1982) found it to be 14 \%, Sumitra Jain et. al. (1983) found it 5 \% sensitivity. Yardi et. al. (1984) found 3.44 \% sensitivity. Usha Udgaonkar et. al. (1985) found it to be 12 \%. Bal & Bhalla (1988) found 37.5 \% sensitivity. Our series compared with that of Bal and Bhalla (1988).

The sensitivity of Pseudomonas aeruginosa to Kanamycin in our series was 19.64 \% Prabhakar and Arora (1979) found it 21 \% sensitivity. Vinodkumar et. al. (1979) reported 45 \% sensitivity. Manorama Deb et. al. (1982) found it 11.8 \%, Sumitra Jain et. al. (1983) reported 20.8 \% sensitivity. Yardi et. al. (1984) found 20.6 \% sensitivity. Usha Udgaonkar et. al. (1985) reported 9 \% sensitivity.

Tetracycline sensitivity of Pseudomonas in our series was 0\%. Same was observed by Wasek et. al. (1965). In series of Bhargava et. al. (1966) it was 13.3 \%. Srivastava et.
al. (1969) found it 10 %. Prabhakar and Arora (1979) found it 5 % and Vinodkumar et. al. (1979) found 4 % sensitivity. Manorama Deb et. al. (1982) found it to be 11.5 %. Sumitra Jain et. al. (1983) found it 21.5 %. Yardi et. al. (1984) found 6.8 %, Usha Udgaonkar et. al. (1985) reported 0 % sensitivity Bal and Bhall (1988) reported 0 % sensitivity.

Sensitivity of Pseudomonas to Erythromycin in our series was 0 % as well as in the series of Wasek et. al. (1965), Srivastava et. al. (1969), Vinodkumar et. al. (1979), Usha Udgaonkar et. al. (1985). Yardi et. al. (1984) found it 10 %.

Sensitivity of Pseudomonas to Chloromycetin was 10.71 % in our series. It was 0 % in series of Wasek et. al. (1965), Srivastava et. al. (1969) found it 20 %. Prabhakar and Arora (1979) found 22 % sensitivity. Vinodkumar et. al. (1979) found 9 %. Manorama Deb et. al. (1982) found it to be 4.5 %. Sumitra Jain et. al. (1983) found it 10 %, Yardi et. al. (1984) found 3.44 % sensitivity. Usha Udgaonkar et. al. (1985) found it to be 0 %.

Our series compare with that of Vinodkumar et. al. (1979) and Sumitra Jain et. al. (1983).

Gentamycin sensitivity of Pseudomonas was 87.27 % in our
series. Prabhakar and Arora (1979) and Vinodkumar et. al. (1979) found it to be 100 %. Manorama Deb et. al. (1982) found 93 %. Narang et. al. (1982) found 86.3 % sensitivity. Sumitra Jain et. al. (1983) found it to be 72 %. Yardi et. al. (1984) found 20.6 %. Usha Udgaonkar et. al. (1985) found 50 % sensitivity. Bal and Bhalla (1988) found it to be 75 % sensitivity.

Polymyxin-B sensitivity to Pseudomonas aeruginosa was 92.72% in our series. Usha Udgaonkar et. al. (1985) found it to be 74 %.

Sensitivity of Pseudomonas aeruginosa to Tarivid was 90.90%, to Netromycin 87.27 %.

Citrobacter Species:

The antibiotic sensitivity of Citrobacter to penicillin was 0 % on our series. Usha Udgaonkar et. al. (1985) found it to be 0 %.

Sensitivity of Citrobacter to Tetracycline was 87.09 % in our series. Usha Udgaonkar et. al. (1985) found 62.5 % sensitivity.

Streptomycin sensitivity of citrobacter was 67.74 % in our...
series. Usha Udgaonkar et. al. (1985) found 62.5% sensitivity.

Kanamycin sensitivity of Citrobacter in our series was 67.74%. Usha Udgaonkar et. al. (1985) found it to be 62.5%.

Chloramycetin sensitivity of Citrobacter in our series was 41.81%. While Usha Udgaonkar et. al. (1985) reported 62.5% sensitivity.

Gentamycin sensitivity of Citrobacter in our series was 93.54%, Usha Udgaonkar et. al. (1985) found it to be 62.5% sensitivity.

Sensitivity of Citrobacter to Tarivid was 87.09% and to Netromycin 83.87% in our series.

Various workers have reported that there has been a shift in the pattern of hospital infection from Staphylococci to gram negative organisms. [Barber (1961). Finland et. al. (1959)]. Finland et. al. (1959) dealing with causes of bacteraemia in Boston city Hospital found that the number of cases of bacteraemia in 1935 due to gram negative bacilli were less than 40 and about three-fourth of them were due to E. coli. In 1947, the total number was about 180, of which
70 were due to E. coli, 45 due to klebsiella aeogenes and 40 due to proteus. In all subsequent years of study these three species were each responsible for 45 to 60 cases per year. Bacteraemia due to Pseudomonas pyocyanea was rare in 1935, but the number rose steadily to 20 cases in 1957.

In contrast to E. coli, Pseudomonas aeruginosa are not usually present in the bowel, nose or skin. They are usually present in the environment. The usual place of infection is ward but some could be infected in operation theatre, by air borne contamination especially when the ventilation is of exhaust type. The air in ward may be contaminated from infected bedding and blankets of the patients. Indiscriminate use of antibiotics may be a factor in increase sepsis of wounds due to Pseudomonas aeruginosa. Taylor (1960) has clearly shown that the indiscriminate use of majority of systemically administered antibiotics was followed by a sharp rise in local population of bacteria.

The drug sensitivity pattern showed Gram positive bacteria were more sensitive to antibiotics in comparison to gram negative organisms which showed high degree of resistance in vitro (TABLE No. XI).

The most effective drug for Gram positive organisms were Gentamycin 72.58 % followed by Tetracycline - 53.22 % and
Kanamycin and Chloromycetin 50.0 % each, then Tetracycline 49.24 % and Erythromycin 37.09 %. In vitro sensitivity of Penicillin was low 3.22 %.

The gram negative organisms showed most sensitivity to Gentamycin 93.54 % followed by Kanamycin - 87.2 % then Tetracycline - 87.09 %, Streptomycin - 67.74 %. Pseudomonas aeruginosa were resistant to most of the drugs except Gentamycin to which it was 87.27 % sensitive and polymyxin - B to which it was 92.72 % sensitive. Therefore it can be concluded that antibiotic sensitivity is essential for controlling the nosocomial infection.

HOSPITAL EPIDEMIOLOGY:

In hospital epidemiology nasal carriage of hospital staff and patient has an important bearing. A carriage of Staphylococci specially in nose from theatre personnel and its bearing with wound infection is shown by Shooter et. al. (1956), Brownee et. al. (1959), Rajkumar and Mittal (1976), Usha Udgaonkar et. al. (1985).

Healthy carriage of Staphylococci is harmful and it is a potent source of infection. (William, 1963). Miles et. al. (1944) and Barber et. al. (1949) showed that the carriage rate amongst hospital staff was much higher than in the
general population. Doctors and nurses are a special danger to their patients as there are several studies showing that the staff working in hospital have higher carriage rate [Ghosh - Ray and Wadia, (1962); Seth et. al. (1973); Talib et. al. (1973)].

A previous study on the carriage of hospital staff by Ghosh Ray and Walia, (1962) revealed that 44 % of nurses, 33.3 % of doctors and 30 % of other workers were nasal carriers. Wasek et. al. (1965) reported 44 % carriage rate, Srivastava et. al. (1969) found 32.2 % carriage rate, while Usha Udgaonkar et. al. (1985) found 43.4 % nasal carriage, Sengupta et. al. (1982) found 30.6 % carriage rate. The carriage rate was 78.05 % in the present study [Table No. XIX]. The carriage rate was higher among the medical staff than the para medical staff and it was higher among doctors than nurses. These findings are consistent with those of Ghosh-Ray (1962). Seth et. al. (1973) also reported higher carriage rate among doctors than nurses. The high carriage rate in the present study among doctors probably being that doctors come into more contact with the ailing population.

Datta et. al. (1976) reported that the nose was the site most frequently found to yield Staphylococcus aureus. The other sites in decreasing frequency were throat and skin (Dorsum of hand). Similar findings have been reported by
various other workers [Williams (1946); Ridley (1959)]. However, in Williams series rate for back of wrist was equally high (46 %). The W.H.O. expert committee on Staphylococci and Streptococci (1968) reported the Staphylococci skin carriage about 5-10 %. In series of Sengupta et. al. (1982) the nasal carriage rate was higher (30.6 %) than throat (11.4 %), nail (17.6 %) and skin (13.2%). The lower skin carriage rate observed in their series was due to repeated washing of hands by the staff members. In the present study the nasal carriage rate was higher (78.05 %) than throat (23.36 %) and skin (Dorsum of hand) 37.5 % (TABLE No. XIX). The higher skin carriage observed in this study are consistent with those of Williams (1946).

Staphylococcus Phage Types :

(Epidemiological marker)

Different phage patterns were observed among the strains isolated from carriers. Most of them (TABLE No. XII AND TABLE No. XIII), were also found among the strains isolated from clinical specimens. So it can be said that these infections may have occured from the hospital staff members. Percentage of untypable strains was high (30.0 %) among the strains isolated from carriers strains and it was 26.92 % among the strains isolated from various environmental
sources. So it can be explained that % of non-typable strains found among carriers and environmental sources is more than the strains found in patients.

The phage pattern (TABLE No. XII AND TABLE No. XIII) showed that 100 % of Staphylococcus strains isolated from patients were typable.

In newborn nursery staff carriers 70.0 % strains were typable. Of the typable strains phage 29 of Group I was found to be relatively more common in patients, nose of newborn nursery staff and newborn nursery environment. While phage 81, 6 and 53 of Group III were relatively more common in patients, nose of nursery staff and environment. Phage 3A of Group II was seen in patients blood and wounds and nose of staff.

Out of 50 coagulase positive Staphylococci strains 37 (74.0%) were typable, while 13 (26.0 %) were untypable. In the study of Rajvanshi et. al. (1967) percentage of typable strains was 60 %, Bhujwala and Mahapatra 70.6 %. Greavis (1977) reported it 68 % and Shayegani et. al. (1978) 88 %, Aggyagiri et. al. (1979) reported the percentage of typable strains as 75 %. Sengupta et. al. (1982) reported 64.5 % strains phage typable. Usha Udgaonkar et. al. (1985) reported that in theatre staff 73.3 % were typable and in patient carriers 42.8 % were typable.
Out of 37 typable strains 5 (10.0 %) belonged to phage Group I, 2 (4.0 %) to phage Group II and 13 (26.0 %) to phage Group III and 17 (34 %) strains in mixed group and 13 (26.0%) were untypable.

In series of Kulkarni (1980) out of 142 typable strains 12 (8.4 %) belonged to phage Group I, 7 (4.9 %) to phage group II and 25 (17.4 %) to Group III, 22 (15.5 %) to group not allocated while 76 (54 %) showed the mixed group phage patterns.

Williams et. al. (1966) observed that the strains from the hospital environment belonged mostly to phage Gr. III. Several others, [Ghosh - Ray and Paul 1961, Chatterjee and Aikat, (1964); Chatterjee et. al. (1968)] also reported the predominance of Gr. III. Blair and Carr, (1960) and Mayer and Rische, reported predominance of the strains belonging to Group I. Wall mark and Finland (1961); Rosendal and Bulow (1967); Parker et. al. (1974); reported the predominance of Gr I and III. Agarwal et. al. (1963) reported the strains belonging to the mixed group to be commonest isolates from clinical sources. In series of Sengupta et. al. (1982) large number of strains (54 %) belonged to mixed group. In present study a large number of strains 34 % belonged to mixed group.
Besides the phage group the predominant phage patterns amongst the individual group are found to be different in several studies. Rountree and Freeman (1955) discovered a strain of Staph. aureus phage type 80 from an epidemic of unusual severity. Barber and Dulton (1968) and Gray et. al. (1962) reported small out breaks of infection by identical phage type 53/75/77. Chatterjee and Aikat (1964) in their study also isolated 53/75/77 as the predominant phage type. In study of Rajavanshi et. al. (1967) no predominant phage type was isolated in any group.

In present study 29, 3A, 81, 84, 85 phage types were found more often, otherwise no predominant phage type was observed in any of the group.

**Pseudomonas aeruginosa Pyocine Typing**

*(Epidemiological marker)*

In the past years the common Gram negative bacilli have become the leading cause of infections seen in large hospitals. As a group these organisms have replaced Staph. aureus as the most important agent in nosocomial infection. Of the gram negative bacilli, Pseudomonas aeruginosa has been of particular interest.

We observed that, Pseudomonas aeruginosa constituted 24.29 %
of gram negative infections in our study, Shriniwas (1977) noted that the incidence of hospital infection due to Pseudomonas aeruginosa varies from 5% to 30% in India. The importance of Pseudomonas aeruginosa infection was also noted by many workers. [Lowbury (1968); Pierson and Feller, (1970); Sengupta et. al. (1972)].

In present study 177 strains of Pseudomonas aeruginosa were isolated from all types of clinical samples and nursery staff from environmental sources of newborn nursery. (TABLE No. XIV).

From these strains - 16.27% were from blood culture
23.61% were from nasal swab
15.27% were from Throat swab
9.72% were from umbilical swabs
18.05% were from rectal swabs and remaining
17.08% from other sources.

Pseudomonas aeruginosa was also isolated from pus and conjunctival swabs.

Epidemiological investigation of newborn nursery included study of skin swabs, nasal swabs and throat swabs of hospital personnel, inanimate objects such as incubators, floor, walls, beds, humidifying appartus etc. Air sampling
was also done to find out aerial contamination.

A total of 117 strains (40.34%) of Pseudomonas aeruginosa were isolated out of 290 samples collected from newborn nursery. Thus in present study a total of 177 strains of Ps. aeruginosa were isolated from both clinical samples, staff as well as epidemiological materials.

Identification of Ps. aeruginosa:

Identification of Ps. aeruginosa is essential for epidemiological survey. One must be able to differentiate it from other gram negative organisms and from other species of the same genus. For diagnostic purpose simple, reliable and rapid tests are found to be useful.

In present study identification was made on the basis of colony characters, staining and motility and biochemical characters.

Majority (98%) of the strains showed colonies which were irregularly round, effuse and smooth. Few strains (2%) showed smooth-rough colonies. Phillips (1969) used colonial characteristic for identification of Ps. aeruginosa. The blue-green pigmentation of the colonies was found to be important criteria for identification of colonies. In the
present study this pigment pyocyanin was produced by 98.3% strains on nutrient agar while 100%. Strains showed pigmentation on Wahba and Darrells modified Sierra medium Brown and Lowbury (1965).

In the staining and motility procedures, all strains (100%) were Gram negative and actively motile.

Biochemical characterization of these strains was done using the key produced by Phillips (1969). Non-fermentation of sugars was found in all (100%) strains. Oxidase test was found very useful in identification of Ps. aeruginosa in the present study. The test was found useful in initial stages to eliminate other bacteria that did not belong to genus Pseudomonas. Balazevic et. al. (1976) also felt the oxidase test as the only test more useful for screening Ps. aeruginosa.

Kovacs (1956) used the oxidase test to distinguish Pseudomonas from enteric bacteria. When due precautions were taken to avoid oxidation of the reagent, the test was found sensitive and useful in classification and identification of Ps. aeruginosa.

For confirmation of Ps. aeruginosa, various other tests were used. The growth was examined for oxidative metabolism of
glucose in Hugh and Leifson’s medium, gelation, liquification, denitrification, arginine dihydrolysis, growth at 42°C and utilization of citrate as sole carbon source.

In 1953, Hugh and Leifson devised an oxidative and fermentative medium. In the present study, all the strains were found to metabolise glucose in oxidative manner. This test is very useful in differentiating most of the oxidase positive colonies that do not belong to genus Pseudomonas. [Philips (1969)].

All the strains (100%) were able to liquefy gelatin within 3 days of incubation. Arginine dihydrolysis was shown by all strains. All strains employed in this study grew at 42°C, whereas none grew at 4°C. Among the fluorescent Pseudomonas only Ps. aeruginosa grows at 42°C, whereas Ps. fluorescence and Ps. putida grow at 4°C and not 42°C (Blazevic, 1976). Therefore this test was found to be important from the point of view of differentiation.

Thus all the 177 strains were identified as Ps. aeruginosa. The key was found very useful for identification.

It was observed that Ps. aeruginosa poses a special problem in this hospital environment and it was found essential to
trace source of infection so as to take preventive measures. Aeruginocine typing method using 10 indicator strains, based on Darrell and Whaba's (1964) technique and modified by Shrinivas (1974) was used to study the strains isolated from the Clinical and Epidemiological materials. Pyocines are naturally occurring antibiotic substances produced by many strains of Ps. aeruginosa and active mainly against other strains of same species. Pyocine typing was developed in an effort to find out a system that would reliably identify different strains of Ps. aeruginosa and would be sensitive enough to differentiate among apparently similar organisms. The ideal method should require neither the sophisticated equipment nor the involved procedures of serologic or bacteriophagic typing. Holloway in (1960), suggested that the pyocines of Pseudomonas might be used as an epidemiologic marker. Darrell and Wahba in (1964) used the property of pyocine production for the typing of more than 1000 strains of Ps. aeruginosa. Gilles and Goven in (1966) proposed a standardized technique of pyocine typing using only 8 indicator strains and a simplified media. Thus aeruginosa typing has been recommended as an useful epidemiological marker in tracing the sources and routes of hospital infection with Ps. aeruginosa.

In the present study pyocine typing of 177 strain isolated from different clinical samples as well as epidemiological
materials was carried out using a set of 10 indicator strains. First eight indicator strains corresponded with Wahba's indicator strains No. 1 to No. 8 and two additional indicator strains No. 9 and No. 10 were those isolated by Shriniwas at AIIMS, New Delhi.

In the present study it was found that, of the total strains, 79.1 % strains were typable showing that the addition of 2 more indicator strains was advantageous which showed better discrimination and increase in the typability than those who used 8 indicator strains. Hackman et. al. (1972) used 8 indicator strains of Gillies and Goven and found a significant variation in the incidences of the various pyocine types. Shriniwas (1975) used 8 indicator strains and found 58.9 % strains typable.

The addition of indicator strains was found useful and appreciated by many workers. Goven and Gillies (1969) were able to recognise 8 subtypes of their type 1 by the use of 5 additional indicator strains. Tagg and Mushin (1973) used 2 additional indicator strains which enabled them to subdivide their common types and also reduced the frequency of apparently untypable strains. Similarly, Gadde (1978) found that the non-typability was reduced by 4 % by additional indicator strains. In contrast to these results, results obtained by Kumari et. al. (1974) were not encouraging
because they used 12 indicator strains and 54 % strains were typable. Also, addition of two indicator strains were not found useful by Jain et. al. (1977) as it did not reduce their untypability.

In order to find out different pyocine types prevalent in Iraq, AL-SHIBIB (1984) used 12 indicator strains of Darrell and Wahba and found that 73.7 % strains were typable. Non typability was observed ranging fro, 7.6 % (Wahba 1965) to 26.3 % A. AL SHIBIB (1984). On an average 14 %.

The difficulty also arises in grouping non-typable strains into a single aeruginocine type as these strains belong to various phage and serotypes. To avoid this Shriniwas (1977) suggested that search for additional indicator strains has to continue and by addition of one or two indicator stains, it may be possible to overcome non typability and clustering of the strains. Shriniwas (1975) also found that the aeruginocine typing method described by him was more suitable than the pyocine typing of Gillies and Goven (1966) for epidemiological typing of Ps. aeruginosa isolated in this country. AL-SHIBIB (1984) stated that whatever be the method of typing employed, a certain proportion of non-typable strains are always encountered and their number depends on various factors like the source of the strain.
When analysis of the results obtained by these 177 strains was made in the present study, it was observed that in all 21 different patterns of inhibition were produced by 140 strains and 37 strains did not show any inhibition and remained untypable.

The commonest type 1, in the present study, corresponds to type 1 of Agashe, type A of Wahba and type 1 of Shriniwas et. al. (1971) and again type 1 of Joshi and Nene (1975). The type 2 also represents type 2 of Agashe, type A of Wahba and type 1 of other workers except for the non inhibition of the two strains W9 and W10. Thus types 1 and 2 together formed about 28.58 % of the total strains. This finding supports the view of Shriniwas (1974).

Type 6, which accounts for 14.29 % of the total corresponds with type 3 of Agashe, type 22 of Joshi and Nene (1975), who have reported work done at the same place. They have included 18 % of their strains in type 22. Type 10 and Type 3 respectively correspond with type 6 and type 7 of Agashe, type 38 and type 33 of Joshi and Nene (1975) and account for about 6.43 % and 2.86 % of the total as compared to 4.7 % and 3.9 % of Agashe and to 8.3 and 8 % respectively of Joshi and Nene (1975).

Type 7, corresponds with type 4 of Agashe showed no
inhibition of any of the Wahba's indicator strains but showed inhibition of one of the strains added by shriniwas. 7.15 % of the total strains are included in this type. Thus it can be concluded that the introduction of these two strains has reduced the number of untypable strains.

Type 8, corresponds with type 5 of Agashe, type B of Wahba and type 4 of Shriniwas (1971) and accounts for about 10.0 % of the total.

About 60.02 % of the strains could be grouped in the four commonly occurring types described by Joshi and Nene (1975) who have studied 300 strains using same set of indicator strains. They could place 46.3 % of their strains in these 4 types. Agashe (1977) could place 40.6 % of the strains in the four commonly occurring types described by Joshi and Nene (1975). These findings are comparable and are important as the same method and the indicator strains were used and the study was carried out in the same institution. Thus it is suggested that these 4 types are prevalent in this part of the country.

In present study about 20.9 % of the strains were found to untypable and these figures are more or less comparable with those reported by other workers viz. 20 % of Niphadkar (1969) and A. AL-SHIBIB (1984) who reported 26.3 % untypable
strains.

As mentioned earlier use of the two indicator strains added by Shriniwas was found to be profitable, mostly because, it not only reduced the number of untypable strains but also helped in sub-division of Wahba's type A in types 1 and 2 of the present study.

Majority of the strains isolated from clinical samples and epidemiological materials fell into 7 commonly occurring types. Out of these strains about 60.02% belonged to the types 1 and 2, 6, 7, 8 which correspond with the types 1 and 2, 3, 6 and 7 of Agashe and types 1, 22, 38 and 33 of Joshi and Nene (1975) respectively. About 63.4% of Agashe's strains and 62.2% of Joshi and Nene (1975) strains isolated from burn cases and wound swabs were included in these 4 types. These results are comparable with the present work and are significant as the same set of indicator strains and the test strains isolated from the same hospital were used by these workers.

It was found that aeruginocine typing is a simple and reliable method giving a high degree of discrimination and is suitable for use in routine hospital laboratories. The method is satisfactory to a great extent if adopted suitably to the local situation and easy to do without requiring
complicated equipment and sophisticated procedures.

As highest incidence of Ps. aeruginosa was noted in newborn nursery unit, it became necessary to study the usefulness of aeruginocine typing as an 'epidemiological marker'. Material was obtained from different sources for bacteriological examination. The sample collection included swabs from hospital personnel, patients and swabs from inanimate objects such as furniture, equipments, sink, incubators etc.

A total of 117 strains of Ps. aeruginosa were isolated from newborn nursery. Majority of the strains isolated were from inanimate objects such as tray, incubators, floor, water, disinfectants etc. From the staff 4 persons showed presence of Pseudomonas aeruginosa on the skin of hands.

In newborn nursery types 1 and 2, 5, 6 and 8 were common in epidemiological studies while type 1 and 2 was predominant in clinical materials, such as blood cultures, faeces, umbilical swabs. From the environmental samples of newborn nursery, type 1 and 2 were isolated from skin and inanimate objects such as incubators, cots, water, disinfectants etc. Type 1 was isolated from floor, window, catheters, cannulas. It was found that skin of a hospital person from this unit was infected with type 1 and the same strain was isolated
from blood of a premature infant.

This suggest that the person might have acted as source of infection. But many inanimate objects also showed type 1 indicating that the contamination might be due to these objects. Aerial contamination of newborn nursery with Ps. aeruginosa was observed. Though all sorts of material showed presence of Ps. aeruginosa from this wards. Predominance was observed with blood, faeces and inanimate objects.

In the present study it was noted that the skin flora of Ps. aeruginosa also played an important role in causation of infection. The strains isolated from skin were mainly from the hospital personnel. Other sources of infection were mainly environmental.

It is an accepted fact that finding Pseudomonas in environment, even in areas with potential for dissemination is not sufficient to identify the source of contamination. The environmental contaminants must be shown to be identical to the pathogenic organism. Thus, in the present study attempts were made to find the source of infection in newborn nursery. But there was no direct correlation observed in the strains except in 3 cases of newborn nursery where identical strains was isolated from a hospital person
and the premature infant. This may indicate that the infection via hospital personnel is most likely to occur. Shriniwas (1977) stated that the presence of organisms on hands of the staff is usually transient and it is difficult to evaluate the significance of it detecting occasionally.

Aerial contamination also does not appear to play any significant role in transmission of Ps. aeruginosa infection in the present study.

Favero and his colleagues (1971) had obtained Ps. aeruginosa from hospital distilled water. It was also isolated from commonly used disinfectant like Dettol Cetavilon etc. (Lancet, Leading article 1961). In the present study identical Ps. aeruginosa strains were isolated from water sample, taps of newborn nursery and also in two samples of disinfectants but their role in the spread of infection appeared insignificant. Ayliffe et. al. (1966) isolated strains of Ps. aeruginosa from hospital sinks which did not correlate with infecting strains. In the present study it was important to note that two strains were isolated from sinks, and identical strains were isolated from premature babies indicates that contaminated sinks do play a part in cross-infection. Whythy and Rampling (1972) found that Pseudomonas aeruginosa was isolated very frequently from hospital sinks and wash basins and these often remained
contaminated with strains of *P. aeruginosa* for long periods. According to them contaminated sinks do play a part in cross-infection.

It thus indicates that cross-infection due to *Pseudomonas aeruginosa* is a common problem in our newborn nursery unit.

**IMMUNOLOGICAL STATUS OF NEWBORN:**

After birth, the neonates rapidly acquire commensal bacteria that colonize the skin and mucus membranes. The host defence mechanism in the form of inflammatory responses, immunoglobulins, complement, lysozyme, C-reactive proteins and phagocytosis are not well developed at this stage and some commensals may become opportunist pathogens, - [Rotimi and Duerden (1981)].

Neonatal polymorphonuclear leucocytes are deficient in both chemotaxis and phagocytosis. Intraleucocyte bacterial killing activity in normal in healthy infants but depressed in newborn infants subjected to stress or illness. Complement levels are low and this may contribute to the poor opsonic activity of neonatal serum.

The placenta effectively excludes maternal IgA, IgM and IgE
immunoglobulins. The exclusion of IgM antibody to gram negative enteric bacilli may explain in part the enhanced susceptibility of new born infants to infection with these organisms. IgG immunoglobulin concentration usually are lower in premature infants comparable to full term infants and higher in post term infants than in the mother.

T lymphocyte function, cell mediated immunity is intact in normal infant but is depressed in infants born with chronic intrauterine viral infection.

Breast milk contains secretary IgA antibody. Lactoferrin, lysozyme, complement large numbers of monocytes and both B and T lymphocytes. It also promotes the growth of lactobacilli and the resulting low stool pH inhibits the growth of E. coli and Shigella.

Prematurity is also important predisposing factor to infection. The premature baby is more prone to infection than full-term baby because of incomplete placental transferance of immune bodies; and impaired capacity to manufacture them. The low serum globulin level may contribute to the poor response of premature babies to infection.

The observations of the present study (TABLE No. XVI)

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clearly demonstrate that Cord serum IgG levels were significantly reduced in the premature infants (701.4 mg/100 ml) as compared to full term (1265 mg/100 ml). The low cord serum IgG levels in preterm babies have also been reported by other workers, Young and Hobbs (1968), Chandra R.K. (1968), Chandra R. K. and Ghai (1972), Tondon et. al. (1984).

However, Prasad et.al. (1971) did not find any change in full term and preterm babies probably because of least number of samples (6 cases only).

The maternal serum IgG levels (1071.5 mg/100 ml) were almost higher than the cord serum IgG levels in premature infants, because the transplacental transfer of IgG occurs during later months of pregnancy, - [Gotoff (1974), Janeway (1966)].

The cord serum levels in full term infants were correlated to maternal levels. Similar observations have also been reported by other workers, - [Chandra R. K. (1968); Chandra R. K. (1970)].

In present study IgA was not detected from cord sera, levels of IgM were detected from 5 cases of neonatal septicaemia (TABLE No. XVII), the mean IgM levels were 23.4 mg/100 ml
indicating intrauterine infection in these infants. The majority of newborns (74.42%) having septicaemia were of 28 to 29 weeks of gestation age and having birth weight of 1.6 to 2.2 kg.

The IgM and IgA concentration may be raised in cord blood, in the presence of antigenic stimulus during pregnancy, (Davies, 1971). Kelkar and Kirthy (1979), detected IgM levels in 20 out of 30 samples of Cord blood, and also reported absence of IgA in all cord sera in their study. Prasad et. al. (1971) also reported absence of IgA in Cord sera.

Gupta et.al. (1980), have reported that they could detect IgA levels in 66% Cord blood samples. The reason for it was intraterine infection in these newborns.

Prasad et. al. (1971) studied IgG, IgM and IgA levels in normal neonates, in prematures, in neonates with acute infection and in neonates with congenital malformations. They found that IgA fraction was absent in Cord blood but present in detectable amounts within the first week. Compared to normal neonates, premature infants appeared immunologically inferior but not immunologically incompetant. They also found there was generally appreciable rise in IgA and IgM fractions in response to
infection but no change was found in cases with congenital malformations.

Gupta et. al. (1980) and Malik et. al. (1980) have studied the levels of IgG, IgM and IgA in cord blood of newborns and have reported that the levels of IgA and IgM in cord blood were higher than those reported by Western workers and they stated that this could be explained due to the high endemicity of parasitic and bacterial infections in our country. Tandon et. al. (1984) studied maternal and cord serum IgG levels in relation to gestation and intrauterine growth. They found that the IgG levels in Cord serum of preterm babies were significantly lower than the cord serum IgG levels in full term babies. They stated there was no correlation between maternal and Cord serum IgG levels.

Haworth et. al. (1965) estimated levels of immunoglobulins in premature infants. They stated that there was no significant correlation between the concentration of immunoglobulins in the plasma of premature infants and the incidence of infection in them.

Steihm et. al. (1966) found that IgG concentration in Cord blood is possibly related to gestation age where as IgM showed an insignificant rise over the weight range studies. IgA is present in very low amount in both normal and
premature infants. Hobbs and Davies (1967) confirmed the above findings and stated that there was a linear relation between the logarithm of IgG in Cord blood and gestational age. They also concluded that, levels of IgG were likely to fall below 100 mg/100 ml in infants of 32 weeks gestation or less and these levels were below safety limits.

Young and Hobbs (1968) and Evans et. al. (1971) further confirmed these findings. They also stated that there is significant low levels of IgG in small for date newborn.

In the present study the C3 levels were also at lower level (<42 IU/ml) especially in premature and low birth weight neonates (as compared with 101 IU/ml in control grp.) and this may be one of the important factor predisposing infection in premature and low birth weight neonates.

In the present study we found that only premature infants and infants with low birth weight and having low levels of IgG are at greater risk of infection. The premature infants are deficient in IgG levels as the IgG transfer occurs during last months of pregnancy. The premature are also deficient in endogenous production of IgG. These factors may combine to increased susceptibility to nosocomial infection.
DISCUSSION

NOSOCOMIAL INFECTIONS OF BURN WARD:

Nosocomial Infection of Burns:

Burn injuries are a major problem in developing countries; accounting for 5 per cent or more of the total number of hospital inpatients. Burns occur commonly in the lower socioeconomic group where there are less safe means of cooking, heating, fewer safety restraints upon small children and where burns are inflicted as means of assault.

In India sixty per cent of the population lives in villages and for daily cooking they use fire wood and splinters. The illiterate people are unaware and careless leading to the major bulk which comes to the hospital as accidental burns. Apart from domestic causes, industrial and agricultural causes are also responsible for different types of burns like chemical burns, electrical burns, scalding etc. The immediate mortality in burns of severe degree is mostly due to shock and loss of fluids. But with advent of recent methods of resuscitation the immediate mortality has been effectively reduced to a great extent. This had led to the prolonged stay of burn patients in the hospital with emergence of a few more important problems. Out of these
the infection is a major problem of burns in patients surviving the initial period of shock.

Infection in burns has been recognised since ancient times. First clinical account of infection of burns can be recognised as early as in 1607 A.D. Various clinical accounts have been cited from ancient literature. Alexander (1971) stated that, "It is doubtful that a sterile burn wound ever exists.' If carefully examined it will show some microbes. It is clearly evident that burn patients are easily susceptible to infection. Burnt areas are directly exposed to the atmosphere. The resultant necrotic tissue forms a good culture medium for the growth of the organisms. Even if proper precautions are taken, it still does not prevent the colonization of the wound by patients' own gastrointestinal tract, respiratory tract and genitourinary tract microbial flora [Hummel et. al. (1970)]. All burn wounds are considered to be infected to some extent. Changes that allow superficial bacteria to colonize and invade a burn wound are not fully understood. It appears that the burn wound passes through three successive stages, sterile, contaminated and infected. The later two stages are sometimes difficult to distinguish from bacteriological point of view - [Sepetjian et. al. (1974)].

Different research workers have studied the problem from
various angles. The aim has been to find out the various factors which are responsible for the high rate of infection leading to considerable mortality and morbidity.

With respect to the socioeconomic background of the patients more than 90 percent were considered to belong to the low or lower socioeconomic strata of society. About 60 per cent of the adult patients were illiterate and another 30 per cent had received only primary school education. Most of the patients came from relatively large families which lived in one or two room with cooking facilities at ground level on either an open fire or a kerosene pressure stove. The relatively young female housewives wear flowing garments made of inflammable fabric. The overcrowded conditions cause young children to play in the vicinity of the cooking area leading to scalds from spilled hot fluids or foods.

The commonest age group involved is between 18-30 years. [Kinogova, (1976)]. In our hospital the age and sex distribution (TABLE No. 1) showed the same pattern. The cases of children below 15 years were not considerable in number. The largest number of cases were in the age group between 15 to 30 years. The maximum number was that of females (58.46 %). The male in the same age group were only 27.90 % of the total.
Although, as stated above, slightly more females than male sustained thermal injury, there is considerable variation in sex ratio for differing age groups. TABLE No. 1 for males and females respectively shows that in the 0-15 year age group the percentage of males out numbered the females. Whereas in the 15-30 and 30-50 years age group the percentage of females out numbered the males.

Information from other general hospitals in different parts of the subcontinent (India) shows very similar results. Rohtak and Choithram Hospital showed very similar results with peak incidence of injury in the age group 21-30 years and peak death rates in group 11-30 years (103/210 patients, 49 per cent - Rohtak), (21/51 patients, 41 per cent, Choithram); both hospitals only saved a life of one patient each when the burned area exceed 60 per cent BSA. When the burns covered half the body surface area four and five patients respectively survived.

Madras Hospital also showed a peak incidence of burning injury in the 21-30 years age group. Seventy eight per cent (150/192) of their patients were between 11 and 40 years of age and 82 of the 150 patient died. No patient with a burn covering more than 30 percent BSA survived.

Lokmanya Hospital reported that out of 1001 adults (over 12
year old) 71 per cent were female and 29 percent were male. Most of the patients were between 13 and 40 years old (913/1001). Burns covering more than 30 per cent BSA were present in 543 patients and 446 (82 per cent) died.

Masina Hospital reported most of their patients were between 11 and 40 years of age with a marked peak between 21 and 30 years. There were no survivors when the burned area covered more than 60 per cent BSA and only 3 survivors when the burned area covered more than half the body surface area.

The observations in respect of distribution of percent area burnt, extent of burns in respect to degree of burns showed regular pattern. Most of the cases were distributed in first three groups. From 0 to 15 percent, 15 to 30 percent and 30 to 45 percent burnt area. All the cases of burns in the present study in first three groups had burns of second degree, which were included in the group of 15 to 30 percent and 30 to 45 percent. The percent area burnt and degree of burns had been important factors when infection was considered.

The mortality rate from six hospitals in India ranged between 37 % and 49 % for adults, [Davies, (1990)].

In present study majority (96.62 %) patients were in adult
group. The mortality rate of 60% in the present study is high. It was observed that majority patients had extensive burns with BSA covering more than 45%. This may be one of the major factor contributing for high mortality rate and also because of heavy infection of the open wounds probably cross infection either from medical personnel and from environmental sources. The infecting organisms are also responsible for high mortality rate, especially Pseudomonas aeruginosa and Staphylococcus aureus which showed multiple drug resistance to antibiotics used in treatment for burns.

When etiology of burns was considered it showed the maximum incidence of dry thermal injuries. Females were most frequently affected in this group.

Blood culture has been accepted as a good indication of the systemic involvement in cases of burns. Serial blood cultures along with wound cultures may yield positive information regarding the factors influencing infection and invasion.

The first week blood cultures (TABLE No. II) showed that 30.61 per cent cases had systemic involvement. Out of 15 positive cases 11 had a single organisms in blood while 4 cases showed presence of more than one organism. The systemic invasion of bacteria went on decreasing with
subsequent weeks of stay in most of the cases. Second week samples showed 23.6 per cent positive cultures with 18 cases having single organism and 3 cases had more than one organism.

Invasion of blood stream was seen to be more in the 2nd week than 1st week.

Tumbusch et. al. (1961), Teplitz (1965) also reported the occurrence of generalized septicaemia in cases of burn. MacMillan et. al. (1972) reported positive blood cultures in 14 patients out of 65, giving a incidence of 21.5 per cent. Smith (1975) reported that 17 out of 20 patients who died from various complications of burn injuries had positive blood culture. Carswell et. al. (1976) reported 23 patients with positive blood culture out of 71 cases admitted. Davies (1990) reported that a relatively high incidence of positive blood cultures are not unexpected considering the very high frequency with which the burned tissues contaminated with bacteria. This contamination is virtually 100 per cent by the end of the first week after injury and is common within a day or two.

The organisms isolated repeatedly in the 1st and 2nd week from blood culture in our series were (TABLE No. III) Pseudomonas aeruginosa (21.21%), Coagulase negative Staphylococci (18.18%) Staphylococcus aureus (15.15%)
closely followed by Klebsiella pneumoniae (12.12%) beta-
haemolytic Streptococci (9.1%), E. coli (6.06%) and alpha-
haemolytic Streptococci were other organisms isolated from
blood culture.

Pseudomonas aeruginosa and Staphylococcus aureus have been
dominating in other series also. Cason et. al. (1968) reported that 9 cases out of 46 died due to septicaemia only in one case Pseudomonas aeruginosa was causative agent, in others Proteus Klebsiella and E. coli were isolated. Foley (1969) reported that Pseudomonas aeruginosa was the leading cause of septicaemia and death. Staphylococcus pyogenes and fungi dominated in later period. Munster et. al. (1971) stated that gram positive organisms were closely following with Candida associating with equal frequency. Smith et.al. (1975) reported the predominant organism was Pseudomonas aeruginosa followed by Staphylococcus pyogenes. Yamul et. al. (1981) reported that in first week Staphylococci were most common organism isolated. But this decreased with subsequent weeks, being replaced by gram negative organisms predominantly by Pseudomonas aeruginosa. Davies (1990) reported that strains of Staph. aureus were most commonly isolated during the first week after injury, followed by Gram negative bacilli, - (particularly Pseudomonas aeruginosa, E. coli, Proteus spp. Klebsiella aerogenes) during the second and third weeks, - [Keswani et. al.
Candida was isolated only in one case of 50 per cent burnt area.

The sequential study in burn wounds showed sterile wounds in first week only (36.84 %) (TABLE No. IV). Majority showed presence of single organism (37.22 %) and certain (25.94 %) cases showed presence of multiple organisms.

The percentage of organisms isolated in the 1st week of hospital stay is less as compared to the percentage of organisms isolated in 2nd and 3rd week of hospital stay. (TABLE No. IV); while the percentage of organisms isolated in 2nd week were almost equal as that of the percentage of organisms isolated in 3rd week of hospital stay.

The incidence of infection in second week was 80.66 percent and in the third week 74.35 per cent, which means that there is no significant difference between the incidence of infection in two weeks. It clearly indicates that if contamination has to be decreased, it is very important to maintain the wound sterile from beginning only. Initial contamination gave place to infection which was resistant to the most available treatment.
The predominant organisms in the first week were *Pseudomonas aeruginosa*. (TABLE No. V) 132 strains of *Pseudomonas aeruginosa* were isolated (40.87%), followed by *Staphylococcus aureus* (26.94 %) and *Esch. coli* (11.64%). In second and third week, pattern shows definite change. There was a distinct fall in the incidence of *Pseudomonas aeruginosa* and its place was being taken up by *Staphylococcus aureus*. Davies (1990) reported that the organisms most frequently isolated from wound infection in Indian series were *Staphylococcal aureus*, *Pseudomonas aeruginosa*, *Esch. coli*, *Proteus spp.*, *Klebsiella* and other gram positive organism (*Streptococci* and *aerobic bacilli*) in descending order of frequency; [Hooda et. al. (1977); Yamul et. al. (1981); Menon et. al. (1984); Ramkrishna et. al. (1985), Gore et. al. (1988)].

*Staphylococcus aureus* was the most common pathogen in burn wounds in all the surveys reviewed, 68 percent (Ramkrishna et. al. 1985) in a series in India, 60 per cent in a series in Denmark (Thomson, 1970), 21 per cent in two different series in Negeria (Sowemimo 1983; Mahogunje et. al. 1987) and 44.08 % in our series. Nearly 84.62 % were methicillin resistant *Staphylococcus aureus*.

We, in our series observed that, *Staphylococcus* was the first invader and was controlled, but if the patients were
too debilitated the place of Staphylococci was taken up by gram negative bacteria. Pseudomonas aeruginosa especially was a serious invader in such cases.

In our series the most dominating organisms from wound infection was Pseudomonas aeruginosa (53.76 %) followed by Staphylococcus aureus (44.08%), E. coli (20.16 %), Coagulase negative Staphylococci (13.97 %), Proteus spp. (11.29%), Klebsiella Pneumoniae (10.75%), Beta-haemolytic Streptococci (7.25 %), Citrobacter (2.4 %), alpha-haemolytic Streptococci (1.07%) and Candida albicans (1.61%). Gram negative bacteria were predominant in 2nd and 3rd week of hospital stay.

It was interesting to note that, in all the cases of burns extending from 15 % to 93 % anaerobic infections were not observed. This may be probably due to the aerobic conditions of burn wounds which may have not supported the growth of anaerobic organisms.

Finland et. al. (1959) and Barber (1961) have reported increase in infection due to gram negative bacteria in adult population. Lowbury, (1970) observed that in first 2-3 weeks period gram negative bacilli like Pseudomonas aeruginosa, Proteus Spp. were common. Alexander (1971) observed that the bacterial flora constantly changes
depending on various factors, Staphylococcus aureus is seen in initial stages while Pseudomonas aeruginosa and Candida are last to invade and are least affected by drugs.

MacMillian (1975) in a 10 years experience with burn wounds at Cincinnati Unit of Shriners Burns Institute found that 60% of bacteria in acute burn were Staphylococcus aureus. In subsequent years gram negative bacilli showed increased incidence up to 70 percent, Pseudomonas aeruginosa and Klebsiella accounted for 54.5 percent, remaining were E. coli.

Hooda et. al. (1977) observed increased incidence of Gram negative bacilli. A total of 79 percent was due to Pseudomonas aeruginosa. A definite pattern of change from Staphylococcus aureus to gram negative bacilli was seen.

Yamul et. al. (1981) reported that the predominant organism in first week was Staphylococcus aureus which was replaced by Gram negative bacilli, E.coli, Pseudomonas aeruginosa in subsequent weeks.

Apart from Staphylococcus aureus and gram negative bacilli, coagulase negative Staphylococci, beta-haemolytic Strpetococci, alpha-haemolytic Streptococci and Candida were also isolated in wound cultures.
Candida infection is not very significant in our series. It has gained importance nowadays. Several authors have documented the rising incidence of fungi in burn wounds - [Munster et. al. (1971); MacMillian et. al. (1972); Smith (1975); Linares et. al. (1977).

There is a good correlation between the percent of burns and the infecting organisms in many series. In our study the percentage ranged from 15 to 93 percent. But majority were second degree, which were included in the group of 15-30 percent and 30 to 45 percent burns. Staphylococcus aureus was most common organism in less percent of burns; followed by Coagulase negative Staphylococci and beta-haemolytic Streptococci (TABLE No. VI). In higher percentage of burns i. e. 15-30 percent, the incidence of Gram negative bacteria, especially E. coli, Proteus Klebsiella and Pseudomonas aeruginosa was higher than Staphylococcus aureus. However, in burns extending more than 30 percentage the incidence of infection by Gram negative bacteria and Gram positive bacteria was almost equal. The presence of Staphylococcus aureus however did not interfere with healing process where the area of burn was up to 15 percent. But with increasing percent area of burns the presence of Staphylococcus aureus along with other gram negative organisms led to frank infection and pus formation; as it was observed in cases with 30 to 45 percent of area of
burns.

The highest incidence of gram-negative bacteria was seen in two groups; extending from 15 to 30 and 30 to 45 percent. The predominant organism were Klebsiella pneumoniae, E. coli, Pseudomonas aeruginosa. It was observed that these organisms along with Staphylococcus aureus are responsible for conversion of contaminated wound to infected wound. Unless the infection of Gram negative bacilli was controlled no improvement in the condition of the wound was observed.

We had 6 cases of Candida infection which were in the group of 30-45 percent of burns. The incidence of fungal infections is high in cases with deep burns with high percent of surface area involved. In long term hospitalization of the patients the dominance of Pseudomonas aeruginosa is seen in almost all cases over taking E. coli infections.

Thomson and Shore (1971) found that the infection rate was more in cases of higher percent of burns. Sengupta (1972) stated that many poorly understood alterations occur in severely burnt patients resulting in a general decreased resistance to infection. Due to the same factors seemingly non-pathogenic bacteria like Staphylococcus albus, Micrococi and Klebsiella can result in infection. There are various
factors which are responsible for high rate of infection in hospitalized patients.

TABLE No. VII shows the isolation of organisms from different sites of the body. Upper ¼ of the body showed the predominance of Gram positive organisms, Staphylococcus aureus, Coagulase negative Staphylococci, beta-haemolytic Streptococci and alpha-haemolytic Streptococci. E. coli and Pseudomonas aeruginosa were less in number. The lower ¼ of the body showed predominance of gram negative bacilli - klebsiella p., Pseudomonas aeruginosa in predominance.

Other workers also have documented same type of finding. Hooda et. al. (1977) found the predominance of Staphylococcus pyogenes in upper part of the body while lower half of the body showed the predominance of gram negative bacilli. Yamul et. al. (1981) reported that upper one third of body showed the predominance of Staphylococcus pyogenes, the middle one third and lower one third of the body showed less number of Staphylococcus pyogenes and predominance of gram negative bacilli.

This distribution serves useful purpose in tracing the source of infection.

The organisms observed from other parts of the body and
other samples from patients showed that hospital strains of Staphylococcus are present in the environment and patients acquire them by cross-infection. - [Thomson, (1971); MacMillian, (1975); Sengupta, (1976); Yamul et. al. (1981)].

In addition patients own flora is an important source of infection. E. coli and Pseudomonas aeruginosa were seen especially in lower ¼ of the body and these organisms were also present in other samples collected from the patients, (TABLE No. IX) and in patients who had burns in lower part of the body. It appears that infection of E. coli and Streptococcus faecalis is autogenous and other patients acquired it by cross-infection.

The samples from hands, nose and throat from the staff of the burns ward showed presence of organisms (56.06 %) [TABLE No. X]. Staphylococcus aureus was predominant organism present in nose (83.82 %) (TABLE No. XI); hands (50.98 per cent), Throat (46.47 per cent). These organisms were present round the year. Other organisms isolated were E. coli, Pseudomonas aeruginosa, Coagulase negative Staphylococci. These organisms were present for a transient period. Especially after doing a dressing of a case infected by these organisms. It was observed that many times that the doctors, nurses had contaminated their nails and fingers from these cases.
It was found that due to cross-infection through these persons other patients also acquire these organisms. Pseudomonas aeruginosa was acquired by many cases by autoinfection and these cases were responsible for spread of these organisms to other cases and nurses many times transferred these organisms through their contaminated fingers.

Pyocine typing showed the identical pattern in strains isolated from wound of such cases and their gastrointestinal flora and also from strain isolated from staff.

Throat samples of doctors, nurses and other paramedical personnel showed the presence of Staphylococcus aureus (46.47%), beta-haemolytic Streptococci (19.19%), Klebsiella pneumoniae (15.15%), alpha-haemolytic Streptococci (13.13%), Coagulase negative Staphylococci (5.05%) and Pseudomonas aeruginosa (1.01%) (TABLE No. XI).

ANTIBIOTIC SENSITIVITY:

Antibiotic sensitivity pattern of Staphylococci isolated from different clinical specimen of burn patients are shown in TABLE No. XIV.

It was seen that 5.64 per cent of Coagulase positive
Staphylococci were sensitive to penicillin. In series of Wasek et. al. (1965) 3% were found to be sensitive to penicillin. Bhargava et. al. (1966) found 2.8% sensitivity. Srivastava et. al. (1969) reported 5% sensitivity. Vinodkumar et. al. (1979) found 9% sensitivity. Usha Udgaonkar et. al. (1985) found 15% sensitivity. Yardi et. al. (1984) found 23.6% sensitivity.

Our series is comparable with Srivastava et. al. (1969).

Tetracycline sensitivity of Staphylococcus aureus in our series was 15.38%. In series of Wasek et. al. (1965) it was 26%, Bhargava et. al. (1966) reported 42.85%, Srivastava et. al. (1969) found it to be 75%, Vinodkumar et. al. (1979) and Prabhakar and Arora (1979) found it to be 52.38% and 27%. Yardi et. al. (1984) found 10.5% sensitivity. Usha Udgaonkar et. al. (1985) found 15%. Our series is comparable with that of Usha Udgaonkar et. al. (1985).

Streptomycin sensitivity of Staphylococcus aureus in our series 21.02%. (TABLE No. XIV) Wasek et. al. (1965) found 10% sensitivity, Bhargava et. al. (1966) found 57.14%, Srivastava et. al. (1969) found 13% sensitivity, Vinodkumar et. al. (1979) and Prabhakar and Arora (1979) recorded a 38% and 47.5% sensitivity. Yardi et. al. (1984) found 21% sensitivity. Usha Udgaonkar et. al. (1985) found 26.6%.
Our series is comparable with Yardi et. al. (1984).

The sensitivity of Staphylococcus aureus to kanamycin in our series was 36.4 %. In series of Vinodkumar (1979) and Prabhakar and Arora (1979) was 66.66 % and 81.1 % respectively. Yardi et. al. (1984) found 39.4 % sensitivity. Usha Udgaonkar et. al. (1985) found it to be 56 %. Our series is more or less comparable with Yardi et. al. (1984).

The sensitivity of Staphylococcus aureus to Chloromycetin was 31.28% and that in series of Wasek et. al. (1965) was 88%, Bhargava et. al. (1966) reported 57.14%. In series of Srivastava et. al. (1969) it was 37%. Vinodkumar et. al. (1979) found it to be 71.42%. Prabhakar and Arora (1979) found it to be 52.9%. Yardi et. al. (1984) found 21.0% sensitivity. Usha Udgaonkar et. al. (1985) found it to be 35.97%. Our series compared more or less with that of Srivastava et. al. (1969) and Usha Udgaonkar et. al. (1985).

The sensitivity of Staphylococcus aureus to Erythromycin in our series was 37.43 %. In series of Wasek et. al. (1965) it was 93 %, Bhargava et. al. (1966) reported 34.28 % sensitivity. Srivastava et. al. (1969) reported 87 %. Vinodkumar et. al. (1979) reported 66 %. Yardi et. al. (1984) found it to be 26.3 %. Usha Udgaonkar et. al. (1985)
it was 39.56 %. Our series is more or less comparable with that of Bhargava et. al. (1966) and Usha Udgaonkar (1985).

Our series showed maximum sensitivity of Staphylococcus aureus to Gentamycin (66.15 %). While Vinodkumar et. al. (1979) and Prabhakar and Arora (1979) found a 100 % sensitivity. Yardi et. al. (1984) found it 44.7 % and Usha Udgaonkar et. al. (1985) found it to be 62.58 %. Our series is comparable with that of Usha Udgaonkar et. al. (1985).

Our series showed the sensitivity of Staphylococcus aureus to Tarivid (cefloxacin) 56.41 %, Netromycin - 47.69 %, Omnatax - 21.02 %, Bactrium - 5.64 %, Lincomycin - 26.15 % and methicillin - 15.38 %.

Coagulase negative Staphylococci:

It is now well known that Coagulase negative Staphylococci isolated from hospital patients are often resistant to antibiotics than out patients cases.

Antibiotic sensitivity of Coagulase negative Staphylococci to penicillin in our series was 13.63 %. Usha Udgaonkar et. al. (1985) reported 13.63 % sensitivity. Jayanti Phatak (1991) found 77.4 % resistance to penicillin.

Tetracycline sensitivity of Coagulase negative Staphylococci
in our series was 18.18 %. Usha Udgaonkar et. al. (1985) found 18.18 % sensitivity.

Streptomycin sensitivity of Coagulase negative Staphylococci in our series was 27.27 %. Usha Udgaonkar et. al. (1985) reported it to be 25.45 %.

Kanamycin sensitivity of Coagulase negative Staphylococci in our series was 54.54 %. Usha Udgaonkar et. al. (1985) reported 55.45 %.

Chloromycetin sensitivity of Coagulase negative Staphylococci in our series was 60.60 %. Usha Udgaonkar et. al. (1985) reported 55.45 % sensitivity.

Erythromycin sensitivity of Coagulase negative Staphylococci in our series was 31.81 %. Usha Udgaonkar et. al. (1985) found it to be 37.72 %.

Ampicillin sensitivity of Coagulase negative Staphylococci in our series was 25.75 %. Usha Udgaonkar et. al. (1985) found it to be 53.63 %. Jayanti Phatak (1991) found 72.6 % resistance to ampicillin.

Antibiotic sensitivity of Coagulase negative Staphylococci to Tarivid was 72.86 %, to Netromycin 75.37 %, to Omnatax
71.35 %, to Methicillin 8.04 %, to Bactrim 32.66 %.

Beta-haemolytic Streptococci:

The antibiotic sensitivity of beta-haemolytic Streptococci to penicillin was 0 % i.e. 100 % resistance. Bhargava et al. (1966) have also showed 100 % resistance. Vinodkumar et al. (1979) found 100 % sensitivity. Yardi et al. (1984) found 100 % sensitivity. Usha Udgaonkar et al. (1985) found 100 % resistance. Our series is comparable with that of Bhargava et al. (1966) and Usha Udgaonkar et al. (1985).

Streptomycin sensitivity of beta-haemolytic Streptococci in our series 90.69 %. Bhargava et al. (1966) have showed 50% sensitivity. Vinodkumar et al. (1979) have reported 100 % sensitivity while Prabhakar and Arora et al. (1979) showed 77.7 % sensitivity. Yardi et al. (1984) observed 100 % sensitivity. Usha Udgaonkar et al. (1985) 0 % sensitivity.

Tetracyclin sensitivity in our series of beta-haemolytic Streptococcus was 0%. Vinodkumar et al. (1979) found 100%. Prabhkar and Arora (1979) reported 55.5% sensitivity. Yardi et al. (1984) found it to be 25%. Usha Udgaonkar et al. (1985) reported 0% sensitivity.
Chloromycetin sensitivity of beta-haemolytic Streptococcus in our series was 81.39%. Bhargava (1966) reported 50%, Prabhakar and Arora (1979) reported 66.6%. Yardi et al. (1984) found 75% sensitivity. Usha Udgaonkar et al. (1985) reported 100% sensitivity.

Erythromycin sensitivity to beta-haemolytic Streptococci in our series was 74.41%, and in series of Bhargava et al. (1966), Vinodkumar et al. (1979) and Usha Udgaonkar (1985). Yardi et al. (1984) found it to be 75%.

Ampicillin sensitivity of beta-haemolytic Streptococcus in our series was 86.04%. Prabhakar and Arora (1979) reported 66.6% sensitivity. Usha Udgaonkar et al. (1985) found it to be 100%. Yardi et al. (1984) found 75% sensitivity.

Gentamycin sensitivity of beta-haemolytic Streptococci was 90.69% in our series. Prabhakar and Arora (1979), Yardi et al. (1984), Usha Udgaonkar et al. (1985) showed 100% sensitivity.

In our series Tarivid (cefoxacin) sensitivity of Beta haemolytic Streptococci was 74.41% and Bactrim sensitivity was 53.48%.

The antibiotic sensitivity of alpha-haemolytic Streptococci to penicillin was 80.95%. Streptomycin sensitivity was
71.42 %. Kanamycin sensitivity was 90.47 %, Chloromycetin sensitivity of alpha-haemolytic Streptococci was 76.19 %. Erythromycin showed 85.71 % sensitivity. Ampicillin sensitivity to alpha-haemolytic Streptococci was 95.23 %, Gentamycin showed 100 % sensitivity. Bactrium sensitivity to alpha-haemolytic Streptococci was 61.90 % and Tarivid (cefloxcin) showed 100 % sensitivity.

Antibiotic sensitivity of non haemolytic Streptococci to penicillin and Ampicillin was 85.71 %. It was 71.42 % to streptomycin Kanamycin and Erythromycin. It was 42.85 % to Chloromycetin. It showed 57.14 % sensitivity to Bactrim. It was 100 % to Gentamycin and Tarivid.

Antibiotic sensitivity of Gram negative rods - E. coli, Klebsiella pneumoniae, Citrobacter, Pseudomonas aeruginosa, Proteus has been shown in TABLE No. XIV. Gram negative organisms appear relatively more resistant to the routinely used antibiotics.

While considering individual Gram negative organisms, we found Pseudomonas aeruginosa is the most resistant organisms in burn wards.
Pseudomonas aeruginosa:

Ampicillin sensitivity of Pseudomonas was 4.08% in our series. Prabhakar and Arora (1979) found it to be 14%. Manorama Deb et al. (1982) found it to be 0%. Sumitra Jain (1983) found it to be 6.6%. Yardi et al. (1984) found it to be 20.6%, Usha Udgaonkar (1985) found 4.5% sensitivity. Bal and Bhalla (1988) found 0% sensitivity. Our series is comparable to that of Usha Udgaonkar et al. (1985).

Sensitivity of Pseudomonas aeruginosa to penicillin in our series was 0% and also in series of Wasek et al. (1965), Bhargava et al. (1966), Srivastava et al. (1969), Vinodkumar et al. (1979), Usha Udgaonkar et al. (1985). Yardi et al. (1984) found it to be 6.5%.

Streptomycin sensitivity of Pseudomonas aeruginosa in our series was 19.70%. It was 0% in series of Wasek et al. (1965), in series of Bhargava et al. (1966) it was 20%, Srivastava et al. (1969) reported 0%. Prabhakar and Arora (1979) reported 18% sensitivity and Vinodkumar et al. (1979) 4.5%. Manorama Deb et al. (1982) reported 14%, Sumitra Jain (1983) found it to be 5%, Yardi et al. (1984) found 3.44%, Usha Udgaonkar et al. (1985) found it to be 12%. Our series is comparable to Bhargava et al. (1966)
and Prabhakar and Arora (1979).

The sensitivity of Pseudomonas aeruginosa to Kanamycin in our series was 15.98 %. Prabhakar and Arora et. al. (1979) found it to be 21 %, while Vinodkumar et. al. (1979) reported 45 % sensitivity. Manorama Deb et. al. (1982) found it to be 11.8 %, Sumitra Jain et. al. (1983) reported 20.8 %. Yardi et. al. (1984) found 20.6 %, Usha Udgaonkar et. al. (1985) reported 9 % sensitivity.

Tetracyclin sensitivity of Pseudomonas aeruginosa in our series was 3.34 %. Wasek et. al. (1965) reported 0 %. In series of Bhargava et. al. (1966) it was 13.3 %, Srivastava et. al. (1969) reported 10 %. Prabhakar and Arora (1979) reported it to be 5 %, Vinodkumar (1979) found it to be 4%, Manorama Deb et. al. (1982) found it to be 11.5 %, Sumitra Jain et. al. (1983) found it 21.5 %. Yardi et. al. (1984) found it to be 6.8 %, Usha Udgaonkar et. al. (1985) recorded 0 % sensitivity, Bal and Bhalla (1988) reported 0 % sensitivity. Our series is comparable to that of Prabhakar and Arora (1979) and Vinodkumar et. al. (1979).

Sensitivity of Pseudomonas aeruginosa to Erythromycin in our series was 0 %, as well as in series of Wasek et. al. (1965), Srivastava et. al. (1969), Vinodkumar et. al. (1979) and Usha Udgaonkar et. al. (1985). Yardi et. al. (1984)
reported 10.0 % sensitivity.

Sensitivity of Pseudomonas aeruginosa to Chloromycetin was 1.85 % in our series. Wasek et. al. (1965) reported 0 % sensitivity. Srivastava et. al. (1969) found it to be 20 %, Prabhakar and Arora (1979) found 22 % sensitivity. Vinodkumar et. al. (1979) reported 9 %, Manorama Deb et. al. (1982) found 4.5 %, Sumitra Jain et. al. (1983) found it to be 10 %, Yardi et. al. (1984) found 3.44 % sensitivity. Usha Udgaonkar et. al. (1985) reported 0 % sensitivity.

Gentamycin sensitivity of Pseudomonas aeruginosa was 45.72 % in our series. Prabhakar and Arora (1979) and Vinodkumar et. al. (1979) found it to be 100 % sensitive. Manorama Deb et. al. (1982) found 93 % sensitivity. Narang et. al. (1982) found 86.3 %, Sumitra Jain et. al. (1983) found it to be 72 %, Yardi et. al. (1984) found 20.6 %. Usha Udgaonkar et. al. (1985) found it to be 50 %. Our series compare with that of Usha Udgaonkar et. al. (1985). Polymaxin B sensitivity of Pseudomonas aeruginosa was 66.91 %, Usha Udgaonkar found it to be 74 %.

Sensitivity of Pseudomonas aeruginosa to Tarivid was 68.02 %, Nitromycin sensitivity was 32.34 %, Omnatax sensitivity of Pseudomonas aeruginosa 64.31 %, Lincomycin - 15.98 %.
Sensitivity of Esch. coli to penicillin in our series was 9.8%. Wasek et. al. (1965), Bhargava et. al. (1966), Srivastava et. al. (1969), Vinodkumar et. al. (1979) had reported 100% resistance. Yardi et. al. (1984) found 100% resistance, while Usha Udgaonkar et. al. (1985) found it to be 9% sensitive. Our series compare to that of Usha Udgaonkar et. al. (1985).

Streptomycin sensitivity of E. coli in our series was 59.84%. Wasek et. al. (1965) reported only 7.8% sensitivity. Bhargava et. al. (1966) reported 40% sensitivity. Srivastava et. al. (1969) have recorded 16.6% sensitivity. Vinodkumar et. al. (1979) found 27.7% sensitivity while Prabhakar and Arora et. al. (1979) found 40% sensitivity, Manorama et. al. (1982) reported 34% sensitivity, Yardi et. al. (1984) found it to be 9.1%. Usha Udgaonkar et. al. (1985) reported 62.85% sensitivity. Our series compare with that of Usha Udgaonkar et. al. (1985).

Tetracycline sensitivity of E. coli in our series was 13.63%. In those of Wasek et. al. (1965) it was 47%, Bhargava et. al. (1966) reported a 20% sensitivity. Srivastava et. al. (1969) recorded 66.6% sensitivity. Prabhakar and Arora (1979) recorded 6% sensitivity; While
Vinodkumar et. al. (1979) reported 22 % sensitivity.
Manorama Deb et. al. (1982) reported 23.7 % sensitivity.
Usha Udgaonkar (1985) reported 14 % sensitivity. Our series compared with Usha Udgaonkar et. al. (1985).

Kanamycin sensitivity of E. coli in our series was 62.87 % while that in series of Vinodkumar et. al. (1979) it was 83 % and in series of Prabhakar and Arora (1979) it was 64 %. Manorama Deb et. al. (1982) found it to be 45 %. Yardi et. al. (1984) found it 36.3 %. Usha Udgaonkar et. al. (1985) found 55 % sensitivity. Our series compared with that of Prabhakar and Arora (1979).

Chloromycetin sensitivity of E. coli in our series was 13.68 %. Wasek et. al. (1965) found it to be 89 %, Bhargava et. al. (1966) found 60 %, Srivastava et. al. (1969) found 83 %, Prabhakar and Arora (1979) found 45 % and Vinodkumar et. al. (1979) found 55 % sensitivity, Manorama et. al. (1982) found 64.2 % sensitivity. Yardi et. al. (1984) found 9.1 % sensitivity. Usha Udgaonkar et. al. (1985) found 55 % sensitivity.

Ampicillin sensitivity of E. coli in our series was 17.42 %; while that in series of Prabhakar and Arora (1979) it was 11%. Manorama Deb et. al. (1982) found Ampicillin sensitivity to be 9.5 %. Yardi et. al. (1984) found 27.3 %
sensitivity. Usha Udgaonkar et. al. (1985) found it to be 30 %.

Erythromycin sensitivity of E. coli in our series is 11.36%. Bhargava et. al. (1966) reported it to be 20 %. Wasek et. al. (1965) found it 0 %. Srivastava et. al. (1969) recorded 0 % sensitivity. Vinodkumar et. al. (1979) have reported 11% sensitivity. Usha Udgaonkar et. al. (1985) found it to be 11 %. Our series compared with Vinodkumar et. al. (1979) and Usha Udgaonkar et. al. (1985).

Gentamycin sensitivity of E. coli in our series was 65.15 %. Vinodkumar et. al. (1979) and Prabhakar and Arora (1979) reported 100 % and 98 % sensitivity respectively. Manorama Deb et. al. (1982) found 86 % sensitivity. Yardi et. al. (1984) found 54.5 % sensitivity. Usha Udgaonkar et. al. (1985) found it to be 82 %.

Antibiotic sensitivity of E. coli to Tarivid was 65.15 %, to Netromycin it was 61.36 %, to Omnatax it was 55.30 % and Bactrim it was 28.03 %. It was observed that the sensitivity to higher and newer antibiotics is more as compared to old routinely used antibiotics.
Proteus Species:

The penicillin sensitivity of Proteus in our series was 0 %. The same was seen in the series of Wasek et. al. (1965), Bhargava et. al. (1966), Vinodkumar et. al. (1979), Usha Udgaonkar et. al. (1985).

Streptomycin sensitivity of proteus in our series was 21.73 %, Wasek et. al. (1965) found it 25 %, Bhargava et. al. (1966) found it 50 %, Srivastava et. al. (1979) found it 10 %. Prabhakar and Arora (1979) 36 %, and Vinodkumar et. al. (1979) found it to be 50 %. Manorama Deb et. al. (1982) reported 44.5 %, Usha Udgaonkar et. al. (1985) found it to be 35 %. Our series is comparable with that of Wasek et. al. (1965).

Tetracycline sensitivity of proteus in our series was 8.6 %. Wasek et. al. (1965) found it to be 16 %, Bhargava et. al. (1966) found 70 %. Srivastava et. al. (1969) found it 20 %. Vinodkumar et. al. (1979) and Prabhakar and Arora (1979) found it 14 % and 10 % respectively. Manorama Deb et. al. (1982) found 44.5 %. Usha Udgaonkar et. al. (1985) found 8 % sensitivity. Our series compared with Usha Udgaonkar et. al. (1985).

Kanamycin sensitivity of Proteus in our series was 28.26 %,
while that in series of Vinodkumar et. al. (1979) and Prabhakar and Arora (1979) it was 71 % and 36 % respectively. Manorama Deb et. al. (1982) recorded 72 % sensitivity. Usha Udgaonkar (1985) recorded 26 % sensitivity. Our series compared with that of Usha Udgaonkar et. al. (1985).

Ampicillin sensitivity of Proteus in our series was 21.73 %, while Prabhakar and Arora et. al. (1979) found it to be 12%. Manorama Deb et. al. (1982) found 44 % sensitivity. Yardi et. al. (1984) found 25 %. Usha Udgaonkar et. al. (1985) found it to be 34 %. Our series is comparable to that of Yardi et. al. (1984).

Erythromycin sensitivity of Proteus in our series was 0 %. Same findings were shown by Wasek et. al. (1965), Srivastava et. al. (1969), Vinodkumar et. al. (1979) Prabhakar and Arora (1979) and Usha Udgaonkar (1985) have reported the same reports like our series. Bhargava et. al. (1966) found it to be 40 %.

Chloromycetin sensitivity of Proteus in our series was 19.56%. Wasek et. al. (1965) found 75 % sensitivity. Bhargava et. al. (1966) reported 70 % sensitivity. Srivastava et. al. (1969) found it to be 50 %. Prabhakar and Arora (1979) found 28 %, Vinodkumar et. al. (1979)
reported 64 % sensitivity. Manorama Deb et. al. (1982) found 65 %. Usha Udgaonkar et. al. (1985) reported 15 % sensitivity.

Gentamycin sensitivity of proteus in our series was 56.52 %. While those in series of Prabhakar and Arora (1979) and Vinodkumar et. al. (1979) it was 99 % and 100 % respectively. Manorama Deb et. al. (1982) found 93.5 %, Yardi et. al. (1984) found 50 % sensitivity. Usha Udgaonkar et. al. (1985) found 48 %. Our series compared with Yardi et. al. (1984).

Antibiotic sensitivity of Proteus to Tarivid was 63.0 %, to Netromycin 56.52 %, to Omnatax 50 % to Bactrim 23.9 %.

Klebsiella Species:

Sensitivity of Klebsiella to penicillin in our series was 5.88 %. It was 0 % in series of Bhargava et. al. (1966) and Vinodkumar et. al. (1979). Nalwade (1982) reported 6.4 % sensitivity. Yardi et. al. (1984) found it to be 5.33 %. Usha Udgaonkar et. al. (1985) found it to be 4 %. Our series is comparable with that of Yardi et. al. (1984).

Streptomycin sensitivity of Klebsiella in our series was 21.56 %. In series of Bhargava et. al. (1966) it was 46 %,
and in Vinodkumar et. al. (1979) it was 33.33 %. Manorama Deb et. al. (1982) found it to be 11.0 %. Nalwade (1982) found 38 %. Yardi et. al. (1984) found 6.6 % sensitivity. Usha Udgaonkar et. al. (1985) reported 14 % sensitivity.

Tetracycline sensitivity of Klebsiella in our series was 9.8 %. Vinodkumar et. al. (1979) found it to be 33 %. Manorama Deb et. al. (1982) found 26.6 % sensitivity. Yardi et. al. (1984) found 4 % sensitivity. Usha Udgaonkar et. al. (1985) found 8 % sensitivity. Our series compared that with Usha Udgaonkar et. al. (1985).

Kanamycin sensitivity Klebsiella in our series was 45.09%. While Vinodkumar et. al. (1979) recorded 83 %. Manorama Deb et. al. (1982) found it to be 60 %. Nalwade (1982) found 40% sensitivity. Yardi et. al. (1984) found 28 %. Usha Udgaonkar et. al. (1985) found it to be 41 %. Our series is comparable with that of Nalwade (1982) and Usha Udgaonkar et. al. (1985).

Chloromycetin sensitivity of Klebsiella in our series was 17.64 %. It was 69 % in series of Bhargava et. al. (1966) and 50 % in series of Vinod Kumar et. al. (1979). Manorama Deb et. al. (1982) reported 25.5 %, Nalwade (1982) found 16.8 % sensitivity. Yardi et. al. (1984) found 6.6 % and Usha Udgaonkar et. al. (1985) found 10.0 %. Our series
Erythromycin sensitivity of Klebsiella in our series was 21.56%. In series of Bhargava et al. (1966) it was 23%. In series of Vinodkumar et al. (1979) it was 16%. Nalwade (1982) found 6.8%, Yardi et al. (1984) found 13.3% sensitivity. Usha Udgaonkar et al. (1985) found it to be 7%. Our series is comparable with that of Bhargava et al. (1966).


Gentamycin sensitivity of Klebsiella in our series was 60.78%. It was 100% sensitive in series of Vinodkumar et al. (1979). Manorama Deb et al. (1982) reported 92.0%. Nalwade (1982) found 64.0% sensitivity. Yardi et al. (1984) found 48% sensitivity. Usha Udgaonkar et al. (1985) found 60% sensitivity. Our series is comparable with that of Nalwade (1982) and Usha Udgaonkar et al. (1985).

Tarivid sensitivity of Klebsiella in our series was 56.86%,
Mandira Banerjee et. al. (1993) reported Klebsiella pneumoniae were resistant to all drugs except third generation cephalosporins (ceftriazone, sodium ceftazidime, cefotaxime and ciprofloxacin to a variable extent), while Piyush Gupta et. al. (1993) observed 14% resistant to all antibiotics. Kenneth et. al. (1993) observed nosocomial out-break of Klebsiella infection resistant to late-generation cephalosporins.

**Citrobacter Species:**

Antibiotic sensitivity of Citrobacter to penicillin in our series was 0%. Usha Udgaonkar et. al. (1985) reported 0% sensitivity.

Sensitivity of Citrobacter to tetracycline in our series was 50%. Usha Udgaonkar et. al. (1985) found it to be 62.5% sensitivity.

Kanamycin sensitivity of Citrobacter was 50% in our series while Usha Udgaonkar et. al. (1985) reported 62.5% sensitivity.

Chloromycetin sensitivity of Citrobacter in our series was
60 % while Usha Udgaonkar et. al. (1985) reported it to be 62.5 %.

Gentamycin sensitivity of Citrobacter in our series was 60%.
Usha Udgaonkar et. al. (1985) reported 62.5 %.

Sensitivity of Citrobacter to newer antibiotic Tarivid was 50.0 %, to Nitromycin 60 %, to Omnatax 80 %, to Bactrim 40%.
Epidemiological study

The examination of hospital environment for bacterial contamination of the air, walls, floor, bathtub, cots, bedpan, tables, windows, I.V. sets, disinfectants, wash basins, tapwater showed presence of specific type of organisms. (TABLE No. XII).

Mean colony count of 65 air settle plates was found to be 820 colonies/hour/plate. The range of number of colonies varied from 324 colonies/hour/plate to 1310 colonies/hour/plate.

The maximum colony count (1040) was seen in the rooms where dressings were done. The colony count was less (360) before the dressing were done in the room and the count increased (970) after the dressings were done. It was also observed that the colony count was less in these rooms where ventilation was good.

The organisms isolated from the environment was predominantly Staphylococcus aureus (38.90 per cent). Floor, cots, tables, bedpans and other articles also showed very high number of pyogenic Staphylococci; other organisms included Pseudomonas aeruginosa, E. coli, Klebsiella pneumoniae, Coagulase negative Staphylococci and fungi.
especially Candida albicans. (TABLE No. XIII).

PYOCINE TYPING OF PSEUDOMONAS AERUGINOSA

Pseudomonas aeruginosa isolated from various sources in hospital, nurses and patients. It was observed that all types of cross-infections were probably active in these cases. Pyocine typing in our laboratory showed that one single conclusion could not be drawn as identical pyocine type pattern was observed in strains isolated from wound, blood and other samples from patients, gastro intestinal flora of patients, in strain isolated from burn ward staff and in strains isolated from environmental sources. Autoinfection was seen in patients especially with burns of lower half of the body, mostly due to faecal contamination. It was also observed that the skin flora of Pseudomonas aeruginosa play a role in causation of infection. Identical pyocine types were observed on hands of burn ward staff and in wound and faeces of two patients.

Urine cultures from patients yielded Pseudomonas aeruginosa. The strains isolated in urine and from wound swabs were of same pyocine types. However causal relationship cannot be established between these strains because same pyocine types were also present in burn ward environment. The source of infection may be either patient himself or it may be
inanimate environmental source. Water samples and savlon, dettol also showed presence of Pseudomonas aeruginosa. In only five cases where identical strain were isolated from savlon and burn wounds. Study of hands of nursing and medical staff showed correlation in thirteen cases where identical strains were isolated from skin of hands and wound swabs, - (TABLE No. XVIII).

It was observed that Pseudomonas aeruginosa poses a special problem in this hospital environment and it was found essential to trace source of infection so as to take preventive measures. Pyocine typing method using 10 indicator strains based on Darrell and Wahba’s technique (1964) and modified by Shriniwas (1974) was used to study the strains isolated from the clinical and epidemiological materials.

In the present study pyocine typing of 659 strains isolated from different clinical samples as well as epidemiological materials was carried out using a set of 10 indicator strains. First eight indicator strains corresponded with Wahba’s indicator strains No. 1 to No. 8 and two additional indicator strains No. 9 and No. 10 were those isolated by Shriniwas at AIIMS, New Delhi.

In the present study it was found that of the total strains
81.56 % strains were typable showing that the addition of 2 more indicator strains was advantageous which showed better discrimination and increase in the typability than those who used 8 indicator strains. Togg and Mushin (1973) used 2 additional indicator strains which enabled them to subdivide their common types and also reduced the frequency of apparently untypable strains. AL-SHIBIB (1984) used 12 indicator stains of Darrell and Wahba and found that 73.7 % strains were typable.

When analysis of the results obtained by these 659 strains was made in the present study it was observed that in all 21 different patterns of inhibition were produced by 555 strains (84.22 %) and 104 strains (15.88 %) did not show any inhibition and remained untypable. (TABLE XVIII AND XVII).

The commonest pyocine type 1 in the present study, corresponds to type 1 of Agashe, type A of Wahba and type 1 of Shriniwas et. al. (1971) and again type 1 of Joshi and Nene (1975). The pyocine type 2 also represents type 2 of Agashe, type A of Wahba and type 1 of other workers except for the non-inhibition of the two strains W9 and W10. Thus type 1 and type 2 together formed about 35.68 % of the total strains.

Pyocine type 6, which accounts for (8.47 %) of the total,
corresponds with type 3 of Agashe, and type 22 of Joshi and Nene (1975), who have reported work done at the same place. They have included 18 % of their strains in type 22.

Pyocine type 7, corresponds to type 4 of Agashe showed no inhibition of any of the Wahba's indicator strains but showed inhibition of one of the strains added by Shriniwas. 9.91 % of the total strains are included in this type. Thus it can be concluded that the introduction of these two strains has reduced the number of untypable strains.

Pyocine type 8, corresponds with type 5 of Agashe, type B of Wahba and type 4 of Shriniwas (1971) and accounted for 3.6 % of the total.

Pyocine type 10 and type 3 respectively correspond with type 6 and type 7 of Agashe, type 38 and type 33 of Joshi and Nene (1975) and accounted for 5.95 % and 3.78 % of the total as compared to 4.7 % and 3.9 % of Agashe and to 8.3 % and 8.0 % respectively of Joshi and Nene (1975).

About 56.94% of the strains could be grouped in these four commonly occurring types described by Joshi and Nene (1975). They could place 46.3 % of their strains in these 4 types. Agashe (1977) could place 40.6 % of the strains in the four commonly occurring types described by Joshi and Nene (1975).
Thus it was observed that these four types are prevalent in this hospital.

In present study about 15.78% of the strains were found untypable and these are more or less comparable with those reported by other workers viz. 20% by Niphadkar (1969) and AL-SHIBIB (1984) who reported 26.3% untypable strains.

As mentioned earlier, use of the two indicator strains added by Shriniwas was found to be profitable, mostly because it not only reduced the number of untypable strains but also helped in sub-division of Wahba's type A in type 1 and 2 in the present study.

Majority of (66.67%) of the strains isolated from clinical samples and epidemiological material fell into 7 commonly occurring types in this hospital. Out of these strains 56.94% of the strains belonged to the types 1 and 2, 6, 7, 8 which correspond with the type 1 and 2, 3, 6 and 7 of Agashe and type 1, 22, 38 and 33 of Joshi and Nene (1975) respectively.

About 63.4% of Agashe's strains and 62.2% of Joshi and Nene (1975) strains isolated from burn cases and wound swabs were included in these four types.
It was found that pyocine typing is a simple and reliable method giving a high degree of discrimination and is suitable for use in routine hospital laboratory.

As highest incidence of Pseudomonas aeruginosa was recorded in burn ward, it became necessary to study pyocine type prevalent in this hospital. Material was obtained from hospital personnel, different sources from burn ward environment, inanimate objects such as furniture, equipments, sinks, bath tub etc. and also from patients for bacteriological examination.

A total of 374 strains of Pseudomonas aeruginosa were isolated from burn ward. Majority of the strains isolated were from inanimate objects such as bathtub, cots, tables, floor, disinfectants, sinks etc.

From the staff of burn ward 7 personnel showed presence of Pseudomonas aeruginosa on the skin of hands.

In burn ward pyocine types 1 and 2, 6, 7 and 10 were common in epidemiological studies, while pyocine types 1 and 2, were predominant in clinical material, such as wound swabs, urine and faeces and also in nursing staff.

Pyocine type 1 and 2 were isolated from environmental
samples, bath tub, cots, water, disinfectants and also from nursing staff of burn ward. It was found that skin-dorsum of hands of 7 nursing staff were infected with pyocine type 1 and pyocine type 2 and the same strains were isolated from wound swabs of 13 patients and also in urine samples of two patients.

This suggest that these nursing staff might have acquired these pyocine types during dressing as transient flora and might have acted as source of infection. But many inanimate objects i.e. bath tub, sinks and disinfectants also showed pyocine type 1 and pyocine type 2 indicating that the contamination might be due to these objects also. Air samples also showed heavy aerial contamination especially of dressing room with Pseudomonas aeruginosa. All though all sorts of material showed presence of different pyocine types, predominance of type 1 and type 2 was observed in wound swabs, urine, faeces and blood cultures.

In the present study it was noted that the skin flora of Pseudomonas aeruginosa plays important role in causation of infection. In present study attempts were made to find out the sources of infection in burn ward, but there was no direct correlation observed in the strains except in 13 cases of burns, where identical strains were isolated from nursing personnel and burn patients. This may indicate that
the infection via hospital personnel is most likely to occur.

In present study identical pyocine types of Pseudomonas aeruginosa strains were isolated from water samples, sinks, and also in five samples of savlon. In present study it was important to note that identical strains of Pseudomonas aeruginosa were isolated from sinks and identical strains were isolated from wound swabs of burn patients, indicating that contaminated sinks play a part in cross-infection. It was also interesting to note that identical strains of Pseudomonas aeruginosa were isolated from bathtub and identical strains were isolated from wound swabs of burn patients. This indicates that sinks and bath tub play a part in cross-infection.

It thus indicates that cross-infection due to Pseudomonas aeruginosa is a common problem in our hospital.
Healthy carriage of Staphylococci is harmful and it is a potent source of infection, (William 1963). Doctors and nurses are a special danger to their patients. Several study showed that the staff working in hospital have a higher carriage rate [Ghosh - Ray and Walia (1962); Seth et. al. (1973); Talib et. al. (1973)].

Healthy carriage rate in the present study was higher in medical staff (83.82 %). The carriage rate was higher among the medical staff than the paramedical staff and it was higher among doctors than nurses. The high carriage rate in the present study probably being that doctors come into more contact with the ailing population.

Ghosh-Ray and Walia (1962) showed that 44 % of nurses, 33.3% of doctors and 30 % of other workers were nasal carriers. Sengupta et. al. (1982) found 30.6 % carriage rate while Usha Udgaonkar et. al. (1985) found 43.4 % nasal carriage.

Different phage patterns were observed among strains isolated from carrier. Most of them (TABLE No. XIX AND XX) were also found among the strains isolated from clinical specimen. So it can be said that these infections may have
transmitted from hospital personnel.

Percentage of untypable strains was high (42.5%) among the strains isolated from patients as well as strains isolated from carrier (38.46%) among staff member; and it was 27.12% among the strains isolated from environmental sources (TABLE No. XX).

Of the 125 coagulase positive Staphylococci strains 82 strains (65.6%) were typable and 43 strains (34.4%) were untypable. In the study of Rajvanshi et. al. (1967) percentage of typable strains was 60%. Greavis (1977) reported it 68%, Sengupta et. al. (1982) reported 64.5% strains phage typable. Usha Udgaonkar et. al. (1985) reported that in theatre carriers 73.3% were typable and in patients carrier 42.8% were typable.

Out of 82 typable strains 12 (14.63%) belonged to phage group I, 6 (7.31%) to phage group II and 26 (31.70%) to phage group III and 38 (46.34%) strains in mixed group.

In series of Kulkarni (1980) out of 142 typable strains 12 (8.4%) belonged to phage Group I, 7 (4.9%) to phage Group II and 25 (17.4%) to Group III, 22 (15.5%) to group not allocated while 76 (54%) showed the mixed group phage patterns.
Williams et. al. (1966) observed that the strains from the hospital environment belonged mostly to phage Group III. In series of Sengupta et. al. (1982) large number of strains (54 %) belonged to mixed group. In present study a large number of strains (46.34 %) belonged to mixed group.

Besides the phage group, the predominant phage patterns amongst the individual group are found to be different in several studies.

Of typable strains type 29 of group I was relatively more common in patients wound and nose of staff. Phage type 3A from group II was seen in patients wound swabs and nose of burn ward nursing staff and also in environmental sources while phage 81, 53 and 47 of group III were relatively more common in wound swabs from patients and environment; (TABLE No. XIX). Phage types in mixed group 53, 54, 84, 85, 47 were common in patient, nursing staff, doctors and environment.

Rountree and Freeman (1955) discovered a strain of Staphylococcus aureus phage type 80 from an epidemic of unusual severity. Chatterjee and Aikat (1964) in their study isolated 53/75/77 as the predominant phage type. In study of Rajavanshi et. al. (1967) no predominant phage type was isolated in any group.

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In the present study, we found high nasal carriage among hospital staff 83.82%. The phage typing of wound sepsis correlated to that of carriers. The commonest belonged to group III. In the carriers 54, 53, 81 were common and it correlated with those found in the environment, hospital staff and lesions.

Staphylococci causing infection differs from place to place and therefore studies carried in a particular hospital has its own significance in that hospital only.

IMMUNITY STATUS IN BURNS

Infection continues to be the leading cause of death in thermally injured patients. Increased susceptibility to infection stems from the open contaminated wound, increased metabolic requirements, decreased nutritional intake, loss of plasma protein into the burn area and suppression of host defence mechanisms following thermal injury is a complex interaction of nutrition, hypermetabolism, and immunological alterations resulting in a complicated and to date, an unsolved problem.

Alexander (1966) for the first time observed the impairment of the ability to produce specific antibodies to a primary antigenic stimulus and felt it to be a significant
contributory factor to the development of septic complication in severely burned patients. He could not locate the site of the immunological defect produced by thermal injury. He thought it to be some where from the process of phagocytosis to the coding process for antibody synthesis.

Later on humoral immunity in burned patients was studied by many other Workers. They aimed at pin pointing the defect in immunological system and its contribution to septicaemic process and mortality. Some of them used it as an index for prognosis of the patient.

The present study revealed that there is decrease in the levels of immunoglobulins studied. The level of decrease depended on the extent of burns and duration of burns and also on type of immunoglobulin (TABLE No. XXI).

Artuson et. al. (1969) measured immunoglobulin levels in serum and blister fluid. They found that changes in serum concentration of the immunoglobulins were correlating with the severity of the Trauma and differing from the changes in total protein, albumin and acute phase reactions. In blister fluid the immunoglobulin concentration was lower than in serum, this difference being smaller for IgG and larger for IgM indicating depletion of IgG in blister fluids.
at a higher proportion. Kohn et. al. (1969) confirmed the findings of Artuson et. al. (1969). They had also found fall in serum values of IgG and less so of IgA and IgM. They further concluded that administration of immunoglobulin and/or plasma does not help in any phase. In early phase loss is very severe and in reabsorption phase immunoglobulin values are either already normal or super normal. So rationale for immuno therapy was questioned by them.

Munster (1970) carried out immunoglobulin estimation in adults. He noticed sharp fall in IgM levels after invasion of burnt tissue by fungi and concluded that mechanical leakage of protein from the burn wound does not entirely account for the observed pattern. He found no difference in the patterns of Immunoglobulins in burns over or under 40 % involvement.

Kohn et. al. (1972) found that immune response in burns is influenced by severity of burn, age, presence of infection and type of treatment. Georges et. al. (1978) measured immunoglobulin levels and extent of burned surface. They used both the findings together as indices of prognosis. Both values being low simultaneously augur a bad prognosis.

It appears that most of previous workers had more or less similar findings. The present study however exhibit certain
differences.

IgG levels which varies in inverse proportion to extent of burns shows initial fall due to mechanical depletion in blister fluid followed by reabsorption and reaching normal values on fifth day and secondary fall approximating the period of setting of infection.

In present study, maximum depletion of IgA was recorded, (TABLE No. XXI) a finding in contrast to that by previous workers. Depletion started from very first day and approched normal limits by end of second week. This may be due to loss of IgA on burned surface for opsonic activities in preference to IgG.

For IgM most of the values clustered at lower side of normal limits during initial period and in less extent of burns especially on first day. This is probably due to initial transient bacteraemia which deplete intravascular opsonic activities. This followed by more or less normal values throughout the period and again secondary fall in later half of second week. This probably singifies secondary bacteraemia or fungal invasion [Munster, (1969)].

The present study, revealed that there is decrease in the levels of complement C$_3$ (< 24 IU/ml). The level of decrease
depended on the extent of burns. It was observed that fall of C₃ level in serum correlates with extent of burn injury.

Farrel et. al. (1973) have studied the levels of total haemolytic complements and individual components of complement in hourly records. They found that fall of all components of complement correspond to fall of total serum proteins. But the return to normal or supernormal values of individual complement component is dissociated from each other and from total serum protein levels. It was within few hours for C₉ whereas other components took 60 to 100 hours for return to normal levels. It was much late for serum protein levels. They thought that complement behaved as an acute phase reactant.

Bjornson et. al. (1977) worked out the serum levels of immunoglobulins, complement and properdin. Serum opsonic activity was reduced significantly only during sepsis, suggesting that this abnormality occurred as a result rather than a cause of infection. Consumption of components of the classical or alternative pathways of complement activation may be an important mechanism by which infection is perpetuated in the burned patients.

Bjornson et. al. (1978) worked out for the second time the levels of complement for classical and alternative pathways
and concluded that classical complement pathway was activated during septicaemia in burned patients and that activation of this pathway occurred predominantly due to inhibition of the alternative pathway. In addition the data show that complement consumption may reduce opsonic capacity of a patient’s serum for certain organisms and not for others.

Bjornson et. al. (1979) have indicated the measurement of components of classical pathway of complement activity in burned patients as a diagnostic tool for predicting the severity of septic episodes and for monitoring recovery. In addition, the observation that complement consumption did not reduce the opsonic capacity of the patient’s sera for their infecting microorganisms suggests that current concepts regarding the role of immunoglobulins and complement in opsonisation of opportunistic microorganisms require re-evaluation.

Albin et. al. (1979) evaluated the role of antiepithelial antibodies in host defence. He reached a hypothesis that anti epithelial antibodies may block or neutralise a serum inhibitor, present following thermal injury, which may interfere with activation of $C_3$ proactivator to its enzymatically active form $C_3$ activator in alternative pathway of complement activation and subsequent generation
of histologically active components. e. g. anaphylotoxins and chemotactic factors, vital to host defences. Thus anti epithelial antibodies play a helpful role in host defence.

For augmenting humoral immunity vaccination by polyvalent bacterial antigen would be useful.

Alexander et. al. (1969) used heptavalent purified soluble Pseudomonas antigen for vaccination purpose. Antibody titres against Pseudomonas were measured. Direct relationship between antibody titre and resistance to infection was established. Failure to the vaccine may occur in those instances when Pseudomonas infection has become well established before an antibody response can be expected in patients with heavily colonized wounds who develop severe abnormalities of neutrophil function and in patients to whom initial immunizing injection is given after 6th post burn day. For optimal immunization combination of intradermal and intramuscular routes and boostering at weekly intervals was advocated.

Sach’s (1970) used a Wellcome vaccine combining two Staphylococcus pyogen strains (Methicillin resistant), six Pseudomonas aeruginosa strains and Staphylococcus toxid. Immunoprophylaxis decreased death rate and increased survival time of fatal cases of burns. Satisfactory
antibody response was observed inspite of cellular immunodeficiency. Active immunoprophylaxis may have a role in the prevention of systemic infection in any immunodeficiency state including the artificial suppression associated with organ transplantation.

It is obvious that humoral immunity is at a definite risk during burns. However, observations made by various workers are not un-equivocal. Majority of them have recorded low levels of IgG (240 mg/100 ml of serum), IgA (140 mg/100 ml) and IgM remaining normal throughout burn period (above 80 mg/100 ml of serum). Tissue immunoglobulin studies further supported the mechanism of depletion. A protective role of autoantibodies was suggested. Complement components were found to be deranged in various proportion for each of them and recovered at variable periods. Role of vaccination was assessed by some and more controlled clinical trails were indicated. Recently effect of Immunoglobulin depletion and loss of complement component activity in opsonisation was questioned.

More rigorous clinical trials, however, will have to be undertaken to evaluate the various ways to aid the specific immuno system so as to restore the bacteriocidal action and thus correcting the compromise of host defence in burned patients.
This seems to be the urgent need of close future. This will reduce the high mortality rate and also limit the aftermaths of burns in form of disfigerment and disability.