INTRODUCTION
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Ordinarily man lives comfortably in an environment that teems with microorganisms. Since most microbes are harmless commensals and some are actually beneficial, people therefore ordinarily enjoy a state of "peaceful co-existence."

However, when persons enter a hospital as patients the situation is remarkably different. There, they come into close association with a number of different people and with a many more and different microorganisms than they do normally. Many of the organisms that they encounter are pathogens. Furthermore, greater exposure to more and different microorganisms than persons are normally accustomed engenders the likelihood of developing an infection during hospitalization.

Infection is as old as mankind itself. The Arabs learned several years ago that cauterization of a wound with hot metal prevented infection. This was common procedure despite, the fact that the patient would be scarred for life. Even though cauterization was traumatic, it gave the victims, more fighting chance to overcome the effects of pathogen, - [Wistreich and Lechtman (1973)].
The concept of antisepsis was introduced largely by Semmelweiss and Lister - [Wistreich and Lechtman (1973)]. Before the relationship of microorganisms to infection was established, infection rates in hospital were so high that these institutions were often referred to as 'Pest houses.'

It was only a little more than a century ago that Ignatz Semmelweiss, a Hungarian physician working in Vienna observed that incidence of child bed fever, was very much higher in Obstetrical ward in Vienna in General Hospital operated by physicians, than similar ward run by midwives. Semmelweiss observed that the midwives washed their hands frequently where as physicians and medical students, after performing autopsies treated their patients without changing their blood splattered cloths or washing hands. In order to lower the occurrence of infection in his maternity ward he required the attendant to wash their hands with chlorinated lime (CaCl₂). The infection rate dropped from 12 % to 1 %.

In early 1800's microorganisms were considered biological nonentities. Infections were believed to be caused by some magic power in air or by imbalance of body fluids. It took incredible tenacity of Pasteur to show the world that microorganisms could not only ferment fruit juice to wine but also cause spoilage of wine. The evolution of germ
theory of infection by Louis Pasteur gave the dreaded complication of wounds after injuries a new significance, - [Altemeier (1980)].

Sir Joseph Lister in England undertook the challenge of preventing surgical infections. Lister reasoned that the very basis of surgical infection - sepsis, might be microbial in nature. Subsequently to prevent access of microorganisms to wounds he designed a system which came to be known as antiseptic surgery. He included heat sterilization of instruments and application of Carbolic acid to wounds by means of dressing - [Wistreich and Lechtman (1973)]. Robert Koch, the German bacteriologist published a monograph "The cause of infection in wounds" which showed for the first time the different types of bacteria for causing distinctive types of clinical pictures.

Near the beginning of twentieth century Van Bergmann developed the principle and practice of aseptic surgery introducing the aseptic era which offered the hope of surgical intervention completely free from infection, this compounded the significance of Lister's discoveries and a new day dawned in practice of surgery, - [Altemeier (1980)]. Another new era, the golden era of antibiotic therapy was introduced in 1940s when Penicillin became available and
about a decade later broad spectrum antibiotics were introduced, - [Agarwal (1972)].

The germ theory of infectious disease was accepted and asepsis was widely believed in and practiced; until antibacterial chemotherapeutic agents such as Sulfanamides and antibiotics such as penicillin came into use. These agents were so effective at the outset that emphasis on asepsis was decreased and reliance was placed upon the two new agents. However, it was soon learned that a selection process was going on whereby the susceptible organisms were eliminated and resistant organisms were retained in the hospital environment. Unfortunately, the resistant organisms appeared to retain their pathogenicity and much to the consternation of all concerned, the incidence of hospital acquired infections rebounded, - [American Hospital Association, (1979)].

Hospitals have a notorious reputation for infections. For centuries infections have been one of the greatest obstacles in advancement of surgery. It has only been since, Lister's time that deep inroads have been made into the revages of sepsis. Though we have come a long way from pre-Listerian era of 'Laudable pus', Hospital gangrene, 'St. Anthony's fire' still it is not long enough. We still have
not met the dictum Florence Nightingale made over a century ago that 'The very first requirement in a hospital is that it should do the sick no harm,' - [Cruickshank et al. (1974)].

Infection acquired in hospital Nosocomial infection is common and resists strenuous efforts to eliminate it. Nosocomial infections have been increasing for the past two decades. The incidence of hospital infection of all kinds in India varies from 8 percent to 33 percent as against 3 percent to 15 percent in U. S. A., and that is mostly due to staphylococci and Pseudomonas aeruginosa. The incidence of Pseudomonas aeruginosa infection is about 5 % to 30 %. In India, different authors recently have reported a higher incidence; Sengupta et al. (1977) at Aurangabad Medical College Hospital recorded 68 %; Agrawal et al. (1980) at Lucknow found the infection rate 44 %, Public Health Laboratory Service Report (1960) shows rates between 10 % to 37 %; Usha Udgaonkar and Bhavthankar (1985) found the infection rate 30.43 %.

It is seen that in Indian Series the infection rate varies from 5.1 % to 68 %. It is suggested that incidence of infection rate varies depending upon the case material, hospital environment. The greater incidence of infection in
recent years is giving rise to growing concern. The fact that they are on the increase can be attributed to the emergence of resistant strains of various organisms. Nosocomial infection is common. Many factors are involved in its spread, these are reflected in the great difficulties experienced in its control, [Cruickshank et al. (1974)].

NOSOCOMIAL INFECTION OF NEW BORN:

The normal foetus is sterile until shortly before birth as long as amniotic membrane remains intact. After birth the new born infant who has essentially no microbial flora is immediately exposed to the microbial and innoculated with millians of bacteria and other microbes; [Bhatia et al. (1988), Rotimi et al. (1981), Hurst V. (1965)].

After birth, the infant is at the mercy of his environment. He may acquire infection from the hands of his attendants, from apparatus used in his resuscitation or general care. Risk particularly from the humidifying units or such equipment is high. Air and feeds are obviously the common sources of bacteria; [Davies P. A. (1971)].

Bacterial colonization is not synonymous with infection. Higher the colonization rate with potential pathogens
greater is the risk of frank clinical infection; - [Barr (1974)].

When infants are colonized heavily at the sites other than rectum, they are more likely to have bacterial infections than if lightly or insignificantly colonized, - [Davies P. A. (1971)].

The host immunity is yet to develop and largely dependent on the maternal immunoglobulins, which are passively transferred across placenta. At this stage some commensals may become opportunistic pathogens particularly in compromised neonates who must remain in hospital for the treatment of congenital abnormalities, - [Rotimi and Duerden (1981)].

The most important neonatal factor predisposing to infection is prematurity or low-birth weight. There is 3 to 10 fold higher incidence of Sepsis, meningitis or urinary tract infections in these infants than in full term, normal birth weight-infants, - [Glasgow C. A.; Overall J. C. (1983)].

The full term infant is equipped with defense mechanism against infection and can bring them into action when necessary. If prematurely born, the baby is even less able to withstand infection and more liable to suffer serious

The premature infant's defences against invasion by microorganisms are limited in several respects. In term of humoral immune systems, the pre term neonate is compromised at birth by having lower concentrations of IgG than his counterpart, - [Conwax S. P., Dear P. R. F., Smith I (1985)].

Neonatal IgG being largely of maternal origin and transferred mostly as the foetus near term. - [Bridges R. A. et al. (1959); Salimonu L. S. et al. (1978)]. The premature infants because of early birth are deficient in IgG antibodies. New born infants also lacks IgM antibodies at birth. Because of their large molecule size IgM cannot cross the placenta, - [Singh M. (1978)]. It may be also deficient in endogenous immunoglobulin production; - [Ravi Varma K. R. et al. (1979)]. These factors may combine to produce a state of hypogammaglobulinaemia with a associated increased susceptibility to infection - [Hobbs and Davies (1967)].

NOSOCOMIAL INFECTION OF BURNS :

It is reasonable to assume that burns are among the oldest
injuries that afflict mankind. In the majority of cases death is due to septicaemia. If the septicaemia is to be avoided systemic efforts should be made to find out the causes of Septicaemia and proper measures for its control. For a long time the reason for infection in burns seemed obvious, the loss of protective skin covering allowed the entry of bacteria and the coagulated serum plus eschar on the burn surface provided an ideal pabulum for bacterial growth. In the past few years, however, clinical and experimental evidence has cast doubts on this interpretation. Some fatal infections occur with extreme rapidity even while the burn wound shows no evidence of colonisation by the offending pathogen. Experimentally increased susceptibility to infection can be demonstrated within a few minutes of burning; at a time when the burn wound is still sterile. Conventional therapy by suitable antibiotics does not solve this problem. This is because antibiotics can not eliminate infection without a concomitant host response. The administration of an appropriately selected systemic antibiotic to an experimental animal with infected burn does not prevent death, even if the organism in the inoculum is highly sensitive to the antibiotic in vitro, [Munster, (1972)].

Recent literature throws some light upon this problem. The
literature has been extensively reviewed by Munster (1972). Besides the loss of integument the inflammatory reaction is defective. Cellular mobilisation is improper due to hampered chemotactic activities, phagocytosis is depressed and so also the intracellular killing mechanism. Bacteriocidal activity is deranged. This is further enhanced by loss of opsonic capacities due to depletion of complement and immunoglobulins such as IgG in particular.

Cellular immunity is also at risk. Depletion of T cells in number and function both add to defective cellular functions. All these factors together probably permit virulent as well as opportunistic pathogens to break the host defence. However, the reviewed literature does not give a comprehensive idea about the state of immunological system according to extent of burns.

Multiple factors are involved in the spread of the pathogens in the hospitals e. g. their presence in the infected patients, and various carrier site of nurses and others; its viability and consequent density in dry and wet environments outside the body and presence of susceptible sites among the other patients.

The common bacteria that involved in bacterical infections of the new born infants are gram positive cocci like
Staphylococci, Streptococci, Pneumococci and gram negative bacilli like E. Coli, Klebsiella species, Proteus species and Pseudomonas aeruginosa. Since these bacteria are known to colonize remain a potential source for infection, especially, premature infants have immature defences to meet this unique challenge.

Among the gram negative bacilli producing nosocomial infection Pseudomonas aeruginosa is given special attention; because the organism is well known secondary invader and may cause systemic infections leading to septicaemia, specially in infants and old persons. The organism is worldwide in distribution and multiplies freely in the inanimate environment.

Nosocomial infection have been increasing for the past two decades. Five to fifteen per cent of the patients admitted in the hospital acquire these infections, and this is mainly due to presence of pathogenic and potentially pathogenic microorganisms in high concentration in hospital environment, instrumental procedures on the patients and lowered resistance in some cases.

These infections are frequently due to multiple drug resistant organisms which have emerged as a result of over
indulgent and injudicious use of antibacterial agents, in treating the patients.

Range of bacteria causing nosocomial infections has been changing over the years. During second world war Streptococci were the main organisms. Afterwards Staphylococci took over till around 1960, but in the last two decades gram negative rods have replaced gram positive cocci. This changing pattern is mainly due to, use of broad spectrum antibiotics; immunosuppressive drugs and prolonged surgical procedures.

A rise in the incidence of wound infection due to gram negative organisms has been reported; of which the most dreaded is Pseudomonas aeruginosa as it is resistant to most of the routinely used antimicrobial agents - [Barret et. al (1968)].

Congregating a large number of sick people under a single roof has many advantages but one serious draw back is that the infection from one is transmitted to another. Naturally many infections are so transmissible, but specific condition associated with the practice of surgery have caused by far the highest mortality in the distant past. The conditions in the hospital before the days of Semmelweiss and Lister
were so appeal that one wonders how any patient was persuade
to enter them.

Antiseptics and more particularlly aspetic methods removed
most of these risks and affected an enormous reduction in
the frequency of septic complications after surgery. But
sepsis even in epidemic form was still not entirely unknown.
Awareness of the importance of tracing the source of
infection of Pseudomonas aeruginosa and of studying the
distribution of this organism in the hospital wards to note
the routes by which it may spread in hospitals among the
patients remains obligatory. It is necessary to unveil the
true characters of the strain precisely. To determine the
identicalness between two isolates, one from the patients
and the other from the probable source, several typing
schemes have been developed. The aim is to trace and prove
the source confirmetorily.

Among the various organisms causing hospital infection
Staphylococci are posing a special problem because of their
resistance to various antimicrobial drugs. Since the
introduction of penicillin in 1941, the Staphylococci have
persistently displayed a potential to develope resistance to
virtually every new antistaphylococcal drug discovered.
Scientist have tried to tackle this problem of Staphylococcal multidrug resistance by employing semisynthetic penicillins and many new antimicrobials. But still Staphylococcus is one of the most commonly encountered organism in hospital personnel and wards.

Septic infections due to Staphylococcus aureus is world wide in distribution and is of particular importance among hospital patients. Staphylococcus aureus has been considered by some workers as the most important organisms responsible for hospital infection, - [Clarke, (1957); Browne et al. (1959)]. Although Staphylococcus aureus was also one of the commonest organism as regards wound infection in the pre-antibiotic era, since the advent of antibiotics Staph. aureus has become the most difficult organisms in wound infection, - [Wasek et al. (1965)]. Of the organisms that cause wound sepsis staphylococcu aureus is the chief and is the commonest cause of sepsis in accidental wounds, - [Williams and Miles (1949)]. In the Public Health Laboratory Service survey (1960). It was isolated from 60 per cent of septic wounds and in 45 per cent it was the only organism found.

Even though in recent years the gram negative organisms are recovered more frequently from cases of hospital infection,
Staphylococcal nosocomial infection still pose a major problem, - [Barber, (1961); Lowbury (1962); Sengupta et al. (1969)].

Staphylococcus aureus infections in burns are common but surprisingly delay healing less than they do in incised wounds, - [Williams et al. (1966)].

Staphylococcal pneumonia of infants tends to affect the premature, but epidemics may occur among groups of healthy newborn infants in hospital, [Guthic and Montogomery, (1947); Browning, (1955); Beavan and Burry, (1956); Disney et al. (1956)].

Generalized infection may take the form of septicaemia with evidence of non suppurative damage to the liver, Kidneys and Lungs, - [Powel, (1961 a)].

Staphylococcal Septicaemia or pyaemia often occurs in debilited hospital patients, - [Smith and Vickers, (1960); Powel (1961 b)].

Although Staphylococcus produces various types of infections, the nosocomial infections of staphylococci are more common and so the investigations for the sources of
Healthy carriage of staphylococci by hospital staff is harmful and it is a potential 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.

The nasal carriage rate of pathogenic staphylococci in the hospital staff in this country has been reported between 20-95 per cent by various workers, - [Chitale (1956); Sayed et al. (1959); Hardas et al. (1964); Verma et al. (1965); Udgaonkar and Bhavthankan (1985)].

Doctors and nurses are a special danger to their patients as there are several studies showing that the staff working in hospital have a higher carriage rate, - [Ghose-Ray and Walia (1962); Seth et al. (1973); Talib et al. (1973)].

Sources of Staphylococci in the environment are the beds, blankets of patients, dust from floors, ceilings, walls, clothing etc. Also the contamination of gaons and uniforms worn in burn unit and the transfer of patients Staphylococci by means of nurses uniforms was reported, - [Hamberaeus,
The problem of distinguishing pathogenic from non pathogenic Staphylococci is continuously being discussed and several criteria like source, haemolysis, chromogenesis and coagulase production have been put forwards. Of these properties the value of coagulase test was well established and it has been universally accepted, as a criterion for the identification of pathogenic staphylococci, - [Jayakar and Bhaskaran, (1969)].

Growth on glycerol monoacetate and resistance to low concentration of mercuric chloride. (Moore, 1960) can be used as markers of epidemic strains of staphylococci, however, other epidemiological markers such as serological typing, phage typing and antibiotic typing are more selective for epidemiological study to investigate the source of infection.

Strains of Staphylococcus aureus isolated from different patients and carriers differ in their degree of sensitivity to particular antibiotics. Most of the strains infecting patients and carriers outside the hospital are sensitive to various antibiotics but most of those 75%; infecting in hospitals are resistant to penicillin and many other anti
The majority of typical hospital Staphylococci are multiple drug resistant and actively produce penicillinase. The antibiotic resistant strains frequently isolated from the hospitals reported to belong predominantly to phage grp. III, phage Gr. I and II included a large majority of strains which are reported either sensitive to all antibiotics or resistant to penicillin alone.

Ability of coagulase negative Staphylococci (CONS) to become an opportunistic pathogen has long been recognised, - [Holt R, (1969)].

In the past two decades, serious infections have been increasingly seen in immunocompromised patients, - [Harris L. F. (1985)]. Because of their prevalence on the skin and use of prosthetic devices in patients during hospitalization, CONS are ideally suited to cause serious infections in such individuals, - [Parisi J. T. (1985)].

CONS constitute 4 % of all bacterial isolates from nosocomial infection of genito-urinary tract and surgical wounds, - [Jay, (1983)].

It is the nosocomial pathogen of prosthetic valves, CSF
shunts, joint prostheses, post operative wounds, septicaemia, abscesses and osteomyelitis, - [Archer et al. (1985)].

CONS are the common blood culture isolates in the neonatal ICU, - [Preeman et al, (1990)].

In last ten years, the rapidly increasing problem associated with CONS is, their resistance to various antimicrobial drugs. This is seen significantly in hospital environment, - [Jesson et al. (1969)]. All hospital strains are resistant to almost all antibiotics.

The change in the character of hospital cross infection is mainly due to the advent of the antibiotic era. In a study of bacteriological patterns of hospital infections, McNamara et al, (1967) found that gram negative bacilli accounted for almost two-thirds (64.5 percent) of hospital acquired diseases. The organisms most prevalent were E. Coli, Pseudomonas and Klebsiella pneumoniae in almost equal proportion, - [Weil et al, (1966); Steinhauer et al, (1966)]. Klebsiella species in particular have been responsible and the apparent ease with which these organisms can spread, especially to debilitated patients is a matter of concern, - [Price and Sleigh, (1970); Hill et al, (1974)].
It is well known from the available literature that Pseudomonas aeruginosa is naturally resistant to the common antibiotics in use, - [Garrod et al, (1954); Dube et al, (1965)].

The only agents to which strains are regularly sensitive are Gentamycin, Carbenicillin and polymaxin. Other agents to which some strains may be sensitive are streptomycin, neomycin and kanamycin but varying degrees of cross resistance between these agents have been reported - [Cruickshank, (1975)].

For some years, the only useful antibiotics against P. aeruginosa were colistin and gentamycin, both of which are more or less toxic. Carbanicillin is used in life threatening situations in combination with gentamycin. Recently introduced tobramycin and amikacin are highly useful against gentamycin resistant strains, both are relatively toxic.

Clinically significant infection with P. aeruginosa should not be treated with single drug therapy as the success rate is slow with such therapy and also because the bacteria can rapidly develope resistance when single drug is employed. The penicillins most useful against P. aeruginosa -
ticarcillin, mezlocillin and piperacillin. These antibiotics should be used in combination with an aminoglycoside - usually gentamycin, tobramycin or amikacin. The pattern of infecting organisms changes depending on the type of treatment employed, type of antibiotics used etc. Therefore, it is very important to know the factors responsible for infection and the spectrum of infecting organisms, as it exists today in our hospitals. This will be very useful in combating the nosocomial infections in wards/hospitals.

We may have to agree with Mary Barber (1961) in saying "However and whatever may be the changing pattern, hospital cross infection has remained a problem yesterday and today. If enough people take it seriously, there is no reason why it should continue tomorrow."