INTRODUCTION
Lesions in the skeletal remains of primitive man, writings of Hippocrates, and Biblical allusions suggest that staphylococci and a man have lived together for many centuries.

During the past 20 years, certain events have altered this almost symbiotic relationship; staphylococcal disease has emerged as one of the most important causes of illness and of death in hospital practice. Certain far-reaching changes in the ecology of man and of staphylococci seem to have contributed to this situation.

The staphylococci have been a continuous source of human sufferings for centuries. The distressing adversities of the Biblical Patriarch, Jab, included an attack of boils while it is questionable whether this phase of his vexation was actually due to staphylococcal infection. There is little doubt of this wide distribution and their capacity to cause disease. Most individuals at sometime or the other have had the experience with the staphylococcal infection. Infected cuts or wounds, a bout of food poisoning, repeated attacks of boils, the distress of chronic osteomyelitis, the inconvenience of hospital acquired infection and other rare forms of manifestations are too well known to bear repetition. Then the question that comes up is "what are the properties which determine the potential qualities of the pathogenicity?" Pathogenic staphylococci may be analysed from this point of view.
Strains isolated from human infections commonly elaborate a number of extracellular factors of potent biological properties. Coagulase, a substance clotting certain animal and human plasmas; alpha haemolysin, a toxin with lethal, demonecrotic and leukocytic properties, leucocidin which damages polymorphonuclear leucocytes; and hyaluronidase, fibrinolysin and proteolytic and lipolytic enzymes have been described. Many attempts have been made to relate virulence to the elaboration of one or more of these factors.

Little is known of the factors which determine tissue invasion by a staphylococcus which has remained as a harmless parasite in the host. How far does the production of ferment like hyaluronidase or fibrinolysin affect the type of pathologic lesion or spread of infection through the tissues? Is mutation a major factor in infectivity?

Virulence of an organism is based chiefly upon its invasiveness and its toxigenicity. Staphylococcus provides an excellent example of an organism, which despite these properties, can remain as a harmless pathogen.

Infection does not result from the mere presence of cocci in the tissues. When one considers the wide distribution of staphylococci and the numerous opportunities for exposure, the incidence of staphylococcal disease is not correspondingly high. To produce an infection experimentally in the human skin or to infect a laboratory animal requires the inoculum of the order of one million or more cocci. Under natural conditions
it is very likely that only a relatively few cocci are involved at the initial stage of colonisation. Once they have gained access to the tissues, the cocci must find a favourable environment. The opportunity to grow and multiply is of immense importance and the emergence of the clinical disease depends as much on the host as on intrinsic properties of the organism.

It is important to identify exactly the factors that are responsible for infection. Some of these have been implicated, but the final proof of the isolation, purification and categorisation is still awaited.