INTRODUCTION
Injury to the body sometimes is followed by a complex series of degenerative changes leading finally to the disappearance of the entire structure of the affected tissue. "This degenerative change implies that there is an alteration in the tissue arrangements themselves and, if the damage is severe, irreversible changes may occur which are incompatible with cell function, and resulting death of the tissue, is called necrosis." (Cameron and Abraham, 1962).

At the end of 19th century Xaver Bichat evaluated systemically the physiology and the pathology of the connective tissue. But this was masked by the concept of Virchow till the early part of the 20th century. He believed the connective tissue as bradytrophic tissue without a typical architecture and it has only mechanical function. Virchow (1858) stressed the importance cellular components of tissues to the exclusion of the extracellular. Subsequently, but many years later, the hypothesis emerged that the noncellular components of the connective tissues might have important functions quite apart from their parent cells (Schade, 1921).

From this work it also appeared that the connective tissues in different parts of the body had common structural characteristics and common functions. In this way the concept of the connective tissue system emerged. Schade was alone in this concept till Klinge (1933) believed the 'fibrinoid degeneration,' a manifestation of degenerative collagen to be a pri-
lesion of the rheumatoid arthritis. This laid the foundation stone of the revolutionary concept of Paul Klemperer and his colleagues (1942), the collagen diseases. They recognised the connective tissue system may be the site for disseminated disease resulting in diffuse disturbances of the connective tissue ground substances and of collagen fibres characterised by two pathological lesions - fibrinoid necrosis and collagen sclerosis, in SLE, Scleroderma, rheumatic fever, rheumatic arthritis and polyarthritis nodosa.

Angévine (1959) stressed the importance of ground substance in addition to collagen fibrils and other components of connective tissue in any form of injury. In response to injury there is accumulation of excess of metabolites in the ground substance which may be one of mucopolysaccharides, water, serum, proteins polypeptides, aminoacids or nitrogenous derivatives of nucleic acids.

Metchnikov (1892) stressed the experimental research on the study of inflammation in the connective tissue from histopathological point of view. More recently, a biochemical approach to wound healing stimulated a great impetus to the biological behaviour of connective tissue to various injuries (Dunphy and Udupa, 1955). During repair as in wound healing,
host of biochemical alterations were revealed in addition to the histological
and histochemical changes. Forscher and Cecil (1957) were interested in the
chemistry of the inflammation and studied the chemical response to radiation.
Gedigk and Bontke (1957) and Kellgren (1952) studied the metabolism of
granulation tissue in detail.

Since the time of Cohnheim in 1873 morphological changes in the
various organs have been studied utilizing various methods. Houck and
Jacob (1958) initiated the study of necrosis of the connective tissue histo-
logically and chemically in response to the croton oil injury. Subsequently,
they extended their study to similar studies in response to the thermal and
the alkali injuries, but the latter studies were less informative in contrast
to that of the croton oil injury. Moreover, Houck (1963) in his detailed
study on the croton oil injury stated that "wound formed granulation
tissue 24 - 48 hours after injury." Thus, in their study the true period
of necrosis was very short. From a further study on the kinetics of this
croton oil injury, Houck and Jacob (1966) agreed that the chemistry of
necrosis has yet to be fully understood. They, however, did not deal with
the other classical and common injuries, like the cold, the ultraviolet and
the radiation, causing necrosis of the connective tissue. Hence, it is felt
that these could be studied in detail from the histobiochemical level. Moreover, a complex series of degenerative changes could occur in the connective tissue, as observed in the other organs, before the morphological manifestation of necrosis. This situation has yet to be characterised histologically and biochemically. Since the connective tissue is regarded as a system during the process of necrosis, it should evoke a systemic response. Thus, there are many gaps in our knowledge of the connective tissue necrosis from various classical and common injuries. These facets, therefore, need elucidation.