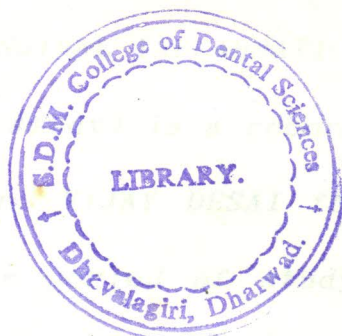


**EVALUATION OF GINGIVAL  
INFLAMMATION USING 'ORATEST'  
(A CLINICOPATHOLOGICAL STUDY)**



**DR. VIJAY DESAI**  
DEPT. OF PERIODONTICS  
S.D.M. COLLEGE OF  
DENTAL SCIENCES AND HOSPITAL  
DHARWAD.

DR. SHIVARAJ SHANKAR

M.D.S.

ASSOCIATE PROFESSOR

Dept. of Periodontics

S.D.M. College of

Dental Sciences & Hospital

Dharwad.

DR. ARCHIRANJEEVI

M.D.S. (Specialty)

PROFESSOR

Dept. of Periodontics

S.D.M. College of

Dental Sciences & Hospital

Dharwad.

**DISSERTATION SUBMITTED TO  
THE KARNATAKA UNIVERSITY  
IN PARTIAL FULFILMENT OF THE REQUIREMENTS  
FOR THE DEGREE OF MASTER OF DENTAL  
SURGERY IN THE SPECIALITY OF PERIODONTICS.  
FEBRUARY 1994**

The gingival sulcus is under continuous challenge by myriads of living bacteria and their toxic antigenic products. The classic experiments by Loe, et, al. Theilade and other investigators<sup>38,12,66</sup> have established and the recent studies have confirmed the impact of microbial plaque as the primary etiological factor for the initiation and progression of periodontal disease.<sup>19,61</sup>

There is evidence to support the hypothesis that chronic destructive periodontitis is episodic in nature with exacerbation of active destruction alternating with periods of quiescence. These different phases could possibly be reflected from the virulence of microorganisms. In the healthy periodontium of both humans and experimental animals, polymorphonuclear leukocyte (PMN) have been demonstrated to migrate towards or reside within the junctional and sulcular epithelium. With plaque accumulation and development of clinical inflammation, initially there is an increase in the number of PMN'S, with a predominance of lymphocytes and monocytes in the early lesion and that of plasma cells in a established lesion.

Inflammatory periodontal disease is an almost ubiquitous disorder in the adult population. This does not imply however that in a given individual all parts of the