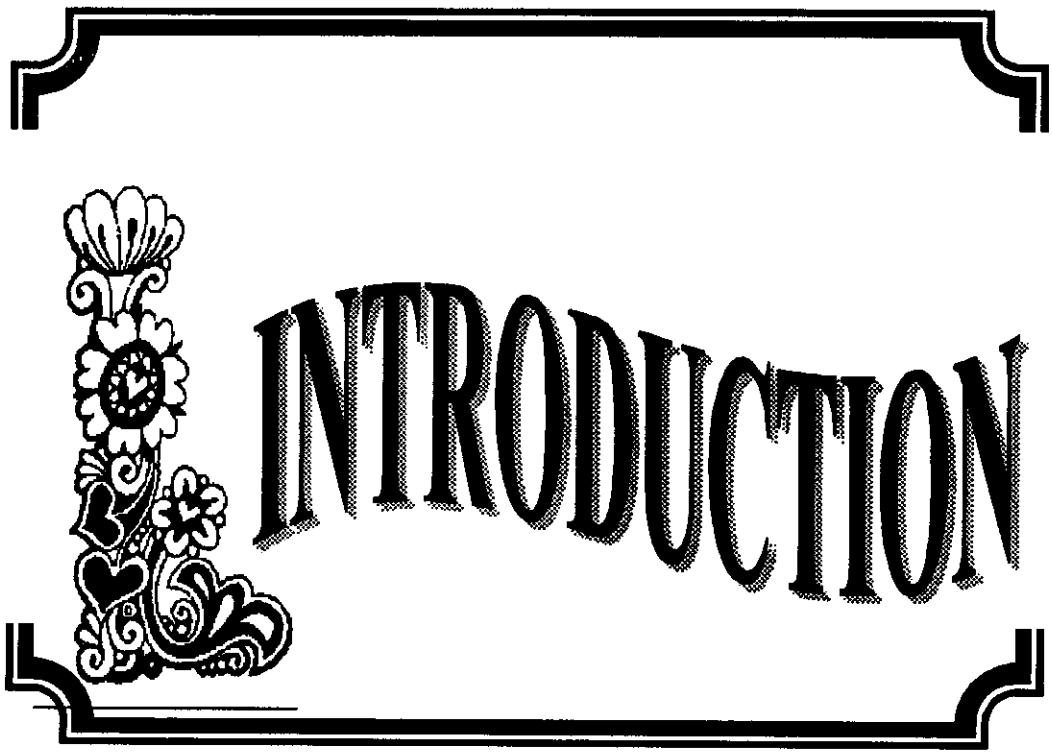
INTRODUCTION

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Nasal polyps are one of the most common lesions encountered in the nose. It is a common clinical problem and it affect between 1-4% of the population. There are still many unanswered questions concerning their incidence, pathogenesis and treatment ***(Marque and Ahmes, 1992).***

The aetiology of nasal polyps is still unknown. The most common theories put forward to explain their formation are infection and allergy ***(Dawes et al., 1989).***

Slavin (1988) concludes that, infection is the likely pathogenesis of nasal polyps on the basis that nasal polyposis in allergic rhinitis is uncommon. However, nasal polyp tissues have large amount of histamine and have eosinophil infiltration.

Barry et al. (1987) suggest that allergic mechanisms may contribute to the development of nasal polyps the contribution of nasal polyposis to the induction and exacerbation of bronchial asthma is still unresolved. Patients with atopic asthma and atopic rhinitis have hyperresponsive airway to both specific and non specific allergic stimulus.

Bronchial hyperreactivity (B.H.R.) is condition of airways causing them to narrow excessively in response to the provoking stimulus ***(Cerveri et al., 1988).***

The mechanisms of airway hyperresponsiveness are numerous and complex. The inflammatory process is one of the most important ***(Pin and Godard, 1994).*** Neural control of airways is more complex than originally apprciated, cholinergic, adrenergic and non adrenergic, non cholinergic (NANC) mechanisms appear to be important in controlling human airway function ***(Barnes, 1986).***

Nasal polyposis and asthma are both expression of airways inflammatory diseases. They also share what is thought to be a control feature in the pathogenesis of the disease ***(Manabu Nonaka et al., 1995).*** The association between nasal polyps and bronchial asthma may be explained by the fact that bacteria also play a role in the development of asthma ***(Hallen et al., 1994).***