INTRODUCTION

INTRODUCTION AND AIM OF THE WORK

Otitis media with effusion (OME) is one of the most common childhood diseases. In spite of numerous investigations its pathogensis still remains obscure. Even the terminology (Sterile otitis, middle ear effusion, catarrhal otitis, secretory otitis, serous otitis, tubotympanitis and glue ear) is diverse and confusing. However it is generally agreed that the Eustachian tubal dysfunction is associated with this condition. In an attempt to clarify the pathogenesis of OME, several investigators like Holmgren (1940) - Lim and Mussl, (1970) - Paparella etal., (1970) - Sade'etal., (1959) and Santuria etal.,(1961) and Santuria (1970), attempted to induce the conditions in laboratory animals by blocking or disturbing the Eustachian tube through various means. These procedures resulted in middle ear effusion in a large number of cases. Holmgren (1940) felt that the negative pressure created in the cavum tympani caused serous effusion. Santuria etal. (1961) and Suehs (1952) suggested that inflammation is responsible for such a condition. On the other hand, Robinson (1942), Reiner and Pulec (1969) were of the opinion that lymphostasis was its cause.

The source (s) of the liquid found in the middle ear has been studied by many investigators like Paparella etal., (1970), Carlson etal.,(1955) and Tonder and Gundersen (1971) seemed to indicate that the fluid is a transudate similar to

blood serum. On the basis of histological findings Friedmann (1963) and Sade' (1966), suggested that the proliferated secretory epithelium and glands are the major sources in cases where the fluid is mucoid. It has been further suggested by Sade'(1966) and by Lim and Hussl (1970) that some parts of serous effusion could have been secreted by secretory cells.

A proper understanding of etiology and pathology of secretory otitis media can contribute to the success of treatment of that disease and prevention of its sequelae. The ultrastructural studies of the middle ear mucosa appear to be of significant value in better understanding the pathology of OME. The middle ear mucosa normally is flat, thin posteriorly and thicken anteriorly with some goblet cells and mucous glands (Tos 1985). Under the stress of inflammation this mucosa may undergo various pathological changes such as oedema, hypertrophy, hyperplasia,metaplasia, glandular formation and increase of goblet cells (Tos 1981) with spontaneous resolution or treatment, some of these changes revert back to normal but some may progress to an irreversible state. It is the aim of modern management to prevent pathological changes from reaching the stage of irreversible damage.

This study aims to examination of the ultrastructurel of middle ear mucosa in 50 cases of OME proved clinically and audiologically.