the antero-lateral wall. Conservative management is usually successful, but surgical correction is sometimes indicated.

This presentation will show some practical aspects of ET physiology that are relevant to surgeons, methods for evaluating ET function and a systematic approach for diagnosing pathology. Accurate diagnosis of ET disorders will lead to successful management and when appropriate, surgical indications will be clear.

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Pathogenesis of Cholesteatoma (R636)

ID: 636.1

Pathogenesis of the cholesteatoma: changing old concepts

Presenting Author: **Leticia Rosito**

Leticia Rosito

Hospital de Clínicas de Porto Alegre

Learning Objectives: 1. To define and classify the cholesteatomas and the spreading routes followed by the disease.; 2. to understand a novel model of pathogenesis with special emphasis on the key role of tympanic membrane retractions; 3. to employ an algorithm to aid the decision making process to maximize surgical results.

Cholesteatoma is a very intriguing condition and still poses a challenge to the otologist. Since it was first described by Duverney in 1683 it has been extensively studied but there are still many pending questions about its development, natural history and prognosis. In 2015 our group proposed a new and embracing classification system for acquired cholesteatomas based on pathogenesis. Our recent studies have shown differences in cholesteatoma growth patterns between children and adults and demonstrated the effect of the disease in the inner ear in both groups. Our contralateral ear studies have also confirmed the essential role of tympanic membrane retractions in the pathogenesis of cholesteatoma.

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Pathogenesis of Cholesteatoma (R636)

ID: 636.2

Congenital Cholesteatoma: Clinical and ethiopatogenetic aspects

Presenting Author: **Dragoslava Djeric**

Dragoslava Djeric

Medical Faculty University of Belgrade

Learning Objectives: To present the features of congenital cholesteatoma.

Design: Case series

Patients and Methods: Ten patients were included in the study. The diagnosis of congenital cholesteatoma was based on previous history that excluded tympanic membrane perforation, otorreha, or otologic procedure, an intact tympanic membrane on otomicroscopic examination and a identified cholesteatoma at the time of surgical procedures (tympanotomy, atticotomy, tympanomastoidectomy).

Results: Six of the 10 patients had lesions isolated to the anterosuperior quadrant of the tympanum, the other had more extensive cholesteatoma that involve posterior part of the tympanic cavity and mastoid. Three of the patients underwent surgery for recidivism(none were from isolated anterior lesions). One of these patients was referred at the time of recurrence, one had known residual cholesteatoma, and one had recurrence.

Conclusion: Clinical and surgical findings suggest that congenital cholesteatoma showed various characteristics depending on the location and stage of development.

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Pathogenesis of Cholesteatoma (R636)

ID: 636.3

The Pathogenesis of Cholesteatoma - Experimental Evidence (R636) 6–6

Presenting Author: **Richard Chole**

Richard Chole

Washington University in St. Louis School of Medicine

Learning Objectives: The objective of this presentation is to understand the scientific basis for the etiology of aural cholesteatoma.

Over the last century, a number of theories have been proposed to explain the pathogenesis of acquired cholesteatomas. Several of these theories have experimental evidence in animal studies.

Support for the ***retraction pocket invagination*** theory is seen in Eustachian tube obstruction models in Mongolian gerbils. When Eustachian tubes of gerbils are ligated in middle ear (bulla) fills with fluid, then over time the pars flaccida retracts, accumulates keratin and forms cholesteatomas.

Support for the ***epithelial ingrowth*** theory had been documented in a number of animal models. When toxic materials are applied to the tympanic membrane, destruction of the tympanic membrane and ingrowth of keratinizing epithelium occurs. In infected gerbils cholesteatomas often rupture leading to epithelial ingrowth. Human temporal bone studies have also supported this theory.

The ***squamous metaplasia*** theory is not support by experimental evidence. The only demonstration of squamous metaplasia has been seen in vitamin A deficiency. When rats are deprived of dietary vitamin A, the middle ear mucosa changes to a multilayered squamous epithelium, but cholesteatomas have never been seen in this model.

Basal cell hyperplasia and ingrowth through the basal lamina has been observed in human temporal bones for many years. Ruedi first described this phenomenon. It has been observed in human temporal bone section and occurs in spontaneous in induced cholesteatomas and Mongolian gerbils.