single surgeon between 2012 to 2014. We collected following data: kind of surgery (canal wall up (CWU) or canal wall down with mastoid obliteration (CWD), FND and its location after exenteration of disease, labyrinthine fistula, dural exposure and preoperative and postoperative facial function.

Results: The incidence of FND was 13% (29/224 ears) for total surgical procedures, 0.1% for CWU tympanoplasty (23/208), 38% for CWD tympanoplasty (6/16). The most common site of dehiscence (90%, 26/29) was the tympanic segment, posterior to the cochleariform process in 18 cases. We find 11 patients with labyrinthine fistula (5%) and 3 with dural exposure (1%). All but one have normal preoperative FN function, all retained normal function postoperatively.

Conclusion: In our series, incidence of facial nerve dehiscence and labyrinthine fistula was similar to the data reported in the literature. All patient retained normal function postoperatively.

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ID: IP111

Eustachian tube opening measurement by sonotubometry using perfect sequences for patients with chronic secretory otitis

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Learning Objectives: Most testing methods for evaluation of the Eustachian tube function are subjective and non-specific, likewise objective methods are insufficiently standardised and they poorly correlate with the clinical picture or are non physiological, therefore employed only under certain pathological conditions. Among the many studies, there is no 'golden standard' which could be widely used and serve as a benchmark to all.

The aim of the study: To examine the relationship between ET function and chronic middle ear diseases by applying sonotubometry with perfect sequences (PSEQ).

Methods: In order to objectively assess ET function, PSEQ-based sonotubometry results were assessed in healthy persons and in patients with ET dysfucntion. All subjects were performed comprehensive examination which included collection of anamnestic data, otoscopy, rhinoscopy, tympanometry, Valsava test and sonotubometry using PSEQ stimuli, nasal and nasopharyngeal videoendoscopy.

Results: The testing was conducted on 43 OME aptients (28 females (65,1%) and 15 males (34,9%)) and 39 healthy individuals (21 females (53,8%) and 18 males (46,2%)). The openings were not detected for 43,9 % of the OME patients and for 6,4 % of healthy individuals ($p < 0{,}001$). The mean ET opening duration in OME patients was 261 ± 147 ms, the mean sound wave amplitude $7{,}41 \pm 4{,}77$ dB , for healthy- 274 ± 153 ms and $12{,}26 \pm 5{,}40$ dB.

Conclusions: Average of the wave sound amplitude was shorter comparing to healthy individuals($p < 0{,}001$). Factors, statistically significantly related with not detected openings using sonotubometry were severe hypertrophy of inferior turbinate's, B type tympanogram and the character of the tympanic membrane retraction. More frequent ET dysfunction was found for the patients with retraction of pars tensa of tympanic membrane (0,038).

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Extensive supporting cell proliferation and mitotic hair cell generation through genetic reprogramming process

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Learning Objectives:

The activation of cochlea progenitor cells is a promising approach for hair cell (HC) regeneration and hearing recovery. The mechanisms underlying the initiation of proliferation of postnatal cochlear progenitor cells and their transdifferentiation to HCs remain to be determined. We show that Notch inhibition initiates proliferation of Wnt-responsive Lgr5 + progenitor cells and mitotic regeneration of HCs in neonatal mouse cochlea *in vivo* and *in vitro*. We demonstrate that Notch inhibition removes the brakes on the canonical Wnt signaling and promotes Lgr5 + progenitor cells to mitotically generate new HCs. While, by down-regulating Notch signaling, the proliferated supporting cells (SCs) and mitotic generated HCs mainly located at the apex region of cochlea, which usually lose less hair cells compared to the base region of cochlea. For pursuing the extensive proliferation and hair cell generation needed for hearing recovery, we genetically reprogramed the SCs by activating the β-catenin to up-regulate Wnt signaling, deleting the Notch1 to down-regulate Notch signaling and overexpressing the Atoh1 in $Sox2^+$ SCs in neonatal mouse cochleae, as we show here that the extensive proliferation of SCs followed by mitotic HC generation is achieved. Our study reveals a new function of Notch signaling in limiting proliferation and regeneration potential of postnatal cochlear progenitor cells, and provides a new strategy to regenerate HCs from progenitor cells by genetically reprograming SCs with defined genes involved in HCs formation.

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Middle Ear Adenoma: rare entity, life-long surveillance