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Endoscopic evaluation of middle ear ventilation route blockage☆,☆☆

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Abstract

Objectives: To describe middle ear ventilation route blockage, relieved during middle ear endoscopic surgery, and to analyze its association with mastoid hypopneumatization/sclerotization.
Study design: Prospective case series with intraoperative analyses, and with a case-control computed tomographic scan comparison.
Methods: Intraoperative findings during endoscopic middle ear surgery are described. Patients with middle ear ventilation route blockage were included in the study group (22 patients), while patients without middle ear ventilation route blockage were included in the control group (16 patients). An intra-patient and inter-group comparison of evaluated mastoid pneumatization was performed from the preoperative computed tomographic scans.
Results: Middle ear ventilation route blockage was classified into three types (A, B, C) according to intraoperative findings. Intrapatient and intergroup comparisons showed that the presence of blockages of middle ear ventilation trajectories is associated with a statistically significantly higher prevalence of hypopneumatization/sclerotization of the mastoid in the study group, a typical sign of middle ear dysventilation pathologies.
Conclusions: Intraoperative evaluation of the middle ear anatomy during endoscopic surgery for inflammatory pathology allows us to clearly visualize the presence of anatomic blockages of the middle ear ventilation trajectories. These blockages might provoke a sectorial dysventilation of the middle ear, with consequent reduction of pneumatization of the mastoid. Further studies will be able to clarify to what extent selective dysventilation phenomena could be a principal factor in influencing middle ear pressure homeostasis.

1. Introduction

The study of tympanic compartments and their aeration pattern began more than one century ago with Prussak's original work on the anatomy and aeration of Prussak's space published in 1867 [1].

Then, in 1897, Sibenmann included observations of the epitympanum and tympanic folds in his book of human anatomy, according to the knowledge at the end of the 19th century [2]. A further important study regarding the actual principles of tympanic compartments and folds is an histological study by Hammar in 1902, who described the embryological development of the middle ear pouch and folds [3]. Many other authors have described the anatomy and development of tympanic compartments and folds because this knowledge is crucial in the understanding and treatment of middle ear disease. For the first time, Chatellier and Lemoine [4] introduced the concept of the "epitympanic diaphragm" in 1945, upon which the modern theories of tympanic ventilation have been developed. The epitympanic diaphragm was described as the floor of the epitympanum

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and consisted of the incus, malleus and their folds. Those authors thought that attic and mastoid aeration would occur through a tympanic isthmus located between the anterior crus of the stapes and the tensor tympani tendon. Later on, Proctor described a posterior isthmus medially to what he called the medial incudal fold [5].

More recently, tympanic isthmus and middle ear ventilation patterns have been described by Palva and colleagues in several articles [6,7]. They described a tympanic isthmus that arises from the tensor tympani tendon to the pyramidal process as the major pathway for mastoid cell and epitympanic ventilation [6]. Palva and colleagues [7] stated that the posterior isthmus described by Proctor is inconsistent because "the medial incudal fold" is in reality the incus intercrural fold, which was found to be atrophic at birth by Hammar [3] and found only once in 37 temporal bones by Palva and Ramsay. They also suggested that the posterior tympanic isthmus is located behind the tip of the short process of the incus and that it is often small and closed by the posterior incudal fold. When open, it can have a role in the aeration of the epitympanum and the mastoid through the incudal fossa, especially when the anterior tympanic isthmus is blocked by inflammatory material. Palva and colleagues also revised Chatellier's concept of the epitympanic diaphragm to include two other important folds: the tensor fold and the lateral incudomalleal fold [7,8]. The role of these 2 folds in the physiopathology of middle ear disease is now well-known, and this knowledge is crucial in the treatment of chronic otitis, as stated by Palva et al [9,10] in 2000. Fig. 1 summarizes the anatomy of middle ear folds and the 2 main ventilation routes. The same authors underlined the importance of tensor fold evaluation during middle ear surgery for chronic disease [9]. Although exploration of the tensor fold region during middle ear surgery for chronic disease has already been established in the international literature, it is not easy to reach this region in otomicroscopy. Several approaches have been described in the international literature, but we suggest an endoscopic approach to the tensor fold in patients with attic disease, which could be exclusive or combined with the traditional microscopic approach [11].

In the case of an isthmus blockage caused by chronic inflammatory disease or a tympanic membrane retraction pocket and a complete tensor fold, this condition leads to inadequate ventilation of the mastoid cells and epitympanic recess. Middle ear pressure seems related not only to a functioning Eustachian tube but also to transmucosal gas exchange through the mastoid mucosa. The mucosal gas exchange is related to the degree of mastoid pneumatization [12], which begins on the 33rd gestational week, continuing up to 8–9 years of age [13]. Because of these 2 gas pressure regulation systems, even if the Eustachian tube is functioning, an isthmus blockage could impair ventilation of the mastoid cells causing sclerotization of the mastoid. It is not clear whether chronic middle ear disease leads to inadequate mastoid pneumatization or conversely a sclerotic mastoid leads to chronic middle ear disease [14].

The aim of this study is to describe the possible kinds of anatomic blockage of the middle ear ventilation trajectories identified during endoscopic surgery procedures and to understand if those alterations could be associated with anomalous mastoid pneumatization, a classic sign of middle ear dysventilation problems.

2. Materials and methods

From March 2007 to February 2008, 57 patients affected by cholesteatoma underwent a surgical procedure at our Otolaryngology Department. The setting was a tertiary referral university hospital. All patients underwent tympanomastoid surgery with intraoperative use of the endoscope, and all procedures were recorded and stored digitally. The anatomic condition of the middle ear, with possible blockages and the integrity of the folds were accurately described in the operative report, and noted in a database soon afterward the operations. We also described the condition of the attical mucosa and the condition of the mucosa of the mesotympanic and protympanic spaces. In the case of missing data, the surgical procedures were reviewed using the recorded files stored digitally.

2.1. Surgical instrumentation and surgical procedure

The instrumentation consisted of 3-mm-diameter, wide-angle (20-cm length), 0° and 45° sinuscopes (Karl Storz, Tuttlingen, Germany). The video equipment consisted of a 3-chip video camera (Karl Storz) and 20-in high-definition monitor; all procedures were recorded digitally on a hard disk (Karl Storz). During the surgical approach to the middle ear, we introduced the endoscope into the middle ear and examined the tympanic isthmus and the tensor fold area to understand the epitympanic diaphragm of the upper unit of all our patients. This transcanal lateral approach permitted a good exposure of the tympanic isthmus. Endoscopic examination of the tympanic isthmus was possible using the 3-mm 0° and 45° endoscopes inserted into the tympanic cavity; we could explore all of the large tympanic isthmus between the medial part of the posterior incudal ligament posteriorly and the tensor tendon anteriorly. The 0° endoscope allowed us to magnify the space between the incudostapedial joint and cochleariform process with the tensor tendon (Proctor's anterior isthmus), whereas, after posterior atticotomy, the 45° endoscope permitted us to magnify the space between the pyramidal process and the short process of the incus (Proctor's posterior isthmus). When mastoidectomy was required, we used the 45° endoscope through the mastoid cavity to obtain a posterior view of the tympanic isthmus. Endoscopic examination of the tensor fold area was possible by

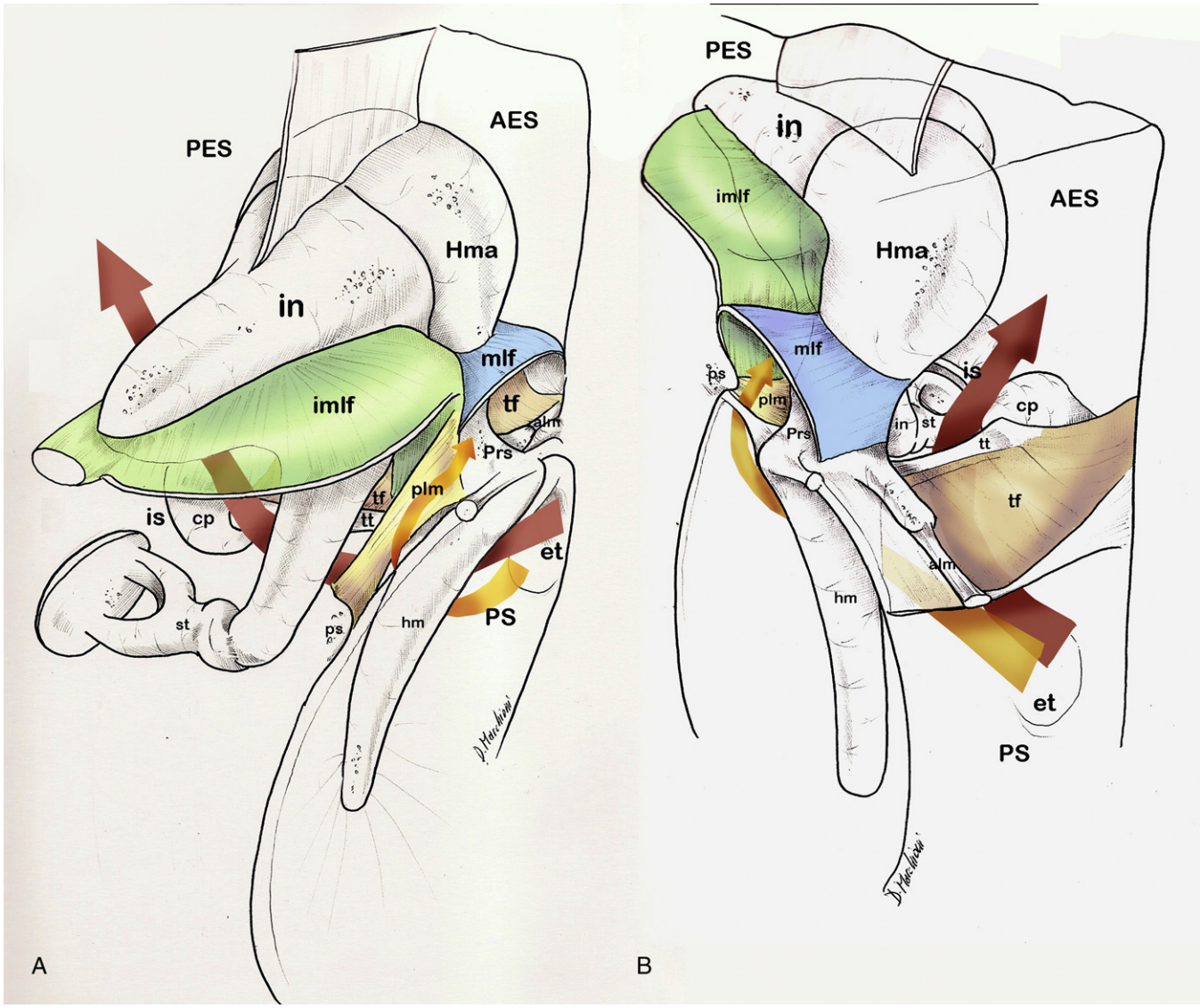


Fig. 1. (Right ear) A, posterior view; B, anterior view. A schematic drawing representing the two independent aeration routes of the epitympanum. The major aeration route (red arrow) passing through the isthmus for the large upper unit (epitympanic compartments, antrum and mastoid cells); the second independent aeration route (yellow arrow) for the smaller lower unit (Prussak's space) passing through the posterior pouch between the tympanum and the posterior malleolar ligamental fold. PES, posterior epitympanic space; AES, anterior epitympanic space; in, incus; Hma, malleus head; imlf, incudo malleolar lateral fold; mlf, malleolar lateral fold; is, tympanic isthmus; st, stapes; cp, cochleariform process; et, eustachian tube; pml, posterior malleolar ligament; aml, anterior malleolar ligament; tf, tensor fold; PS, protympanic space; tt, tensor tympani; Prs, Prussak space; hm, malleus handle; ps, posterior spine.

two kinds of approach using a 3-mm 45° endoscope as follows:

- *Inferior approach:* the endoscope was inserted into the protympanic region; we identified the Eustachian tube and supratubal recess; this position allowed us to obtain a good view of the inferior edge of the tensor fold;
- *Superior approach:* we performed an anterior atticotomy exposing the anterior epitympanic space; this procedure allowed us to obtain a good view of the superior edge of the tensor fold.

The study and control groups were selected prospectively as follows.

2.2. Study group

Patients affected by an anatomic blockage of the middle ear ventilation trajectories, visible at the endoscopic evaluation during the surgical approach, were included in the study group. On the other hand, patients affected by a disease involving the protympanic, the mesotympanic, and the retrotympanic regions, or patients who had already undergone middle ear surgery and with bilateral pathology

of the middle ear, were excluded from the study group. Twenty-two patients of 57 were finally included in the study group.

2.3. Control group

Subjects affected by middle ear disease who underwent a surgical procedure, with the absence of anatomic blockage of the middle ear ventilation trajectories visible at the endoscopic evaluation during surgery, were included in the control group; patients who had already undergone middle ear surgery in the past and patients with bilateral pathology of the middle ear were excluded from the control group. Sixteen patients of 57 operated for middle ear disease were finally included in the control group.

All 38 patients recruited in our study had a preoperative high-resolution computed tomographic scan of the temporal bone with axial projections obtained with sequential 1.0-mm slices performed from the arcuate eminence to the jugular fossa. The radiological mastoid pneumatization degree was reviewed in all 38 patients, and classified into 3 types, according to a modified CT scan classification by Görür et al [14]:

Type 1 (normal pneumatization): the mastoid pneumatization reaches the mastoid tip, zygomatic, perisigmoid and periantral regions; aditus ad antrum is open/present;

Type 2 (hypopneumatization): the mastoid pneumatization is present only in antral, and periantral regions; aditus ad antrum is present/open;

Type 3 (sclerotic): the mastoid pneumatization is totally absent; aditus ad antrum is present/open.

This classification has been applied to both ears (the pathologic and the healthy ear), both in the study group and the control group to assess possible differences intra-patients and intergroups.

Mastoid pneumatization data were compared using Fisher exact test (χ^2 analyses was not applicable because of insufficient data). $P < .05$ was considered statistically significant.

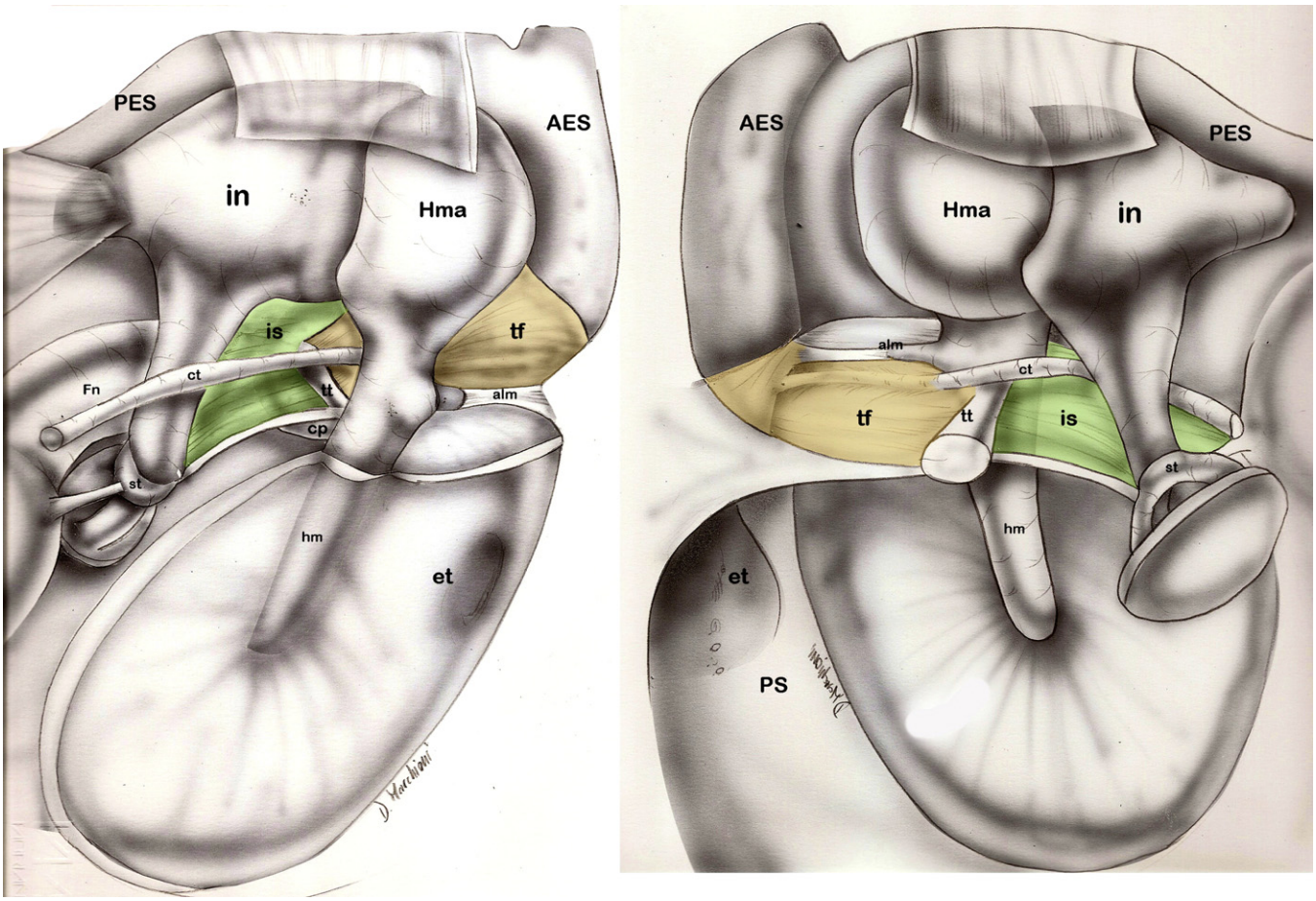


Fig. 2. (Right ear) A schematic drawing representing the attical aeration pattern Type A, mucosal fold or inflammatory tissue causing a blockage of the isthmus associated with a complete tensor fold. PES, posterior epitympanic space; AES, anterior epitympanic space; in, incus; Hma, malleus head; is, timpanic istmus; st, stapes; cp, cochleariform process; alm, anterior malleolar ligament; tf, tensor fold; PS, protympanic space; tt, tensor tympani; hm, malleus handle; fn, facial nerve; ct, chorda tympani.

3. Results

3.1. Study group

The study group consisted of 22 patients of which 6 patients were female and 16 were male; the mean age of the group was 37 (+/− 14.2 SD).

3.1.1. Pathology and symptoms

In 22 patients affected by isthmus block, we found the following pathology:

- 9/22 subjects were affected by attic cholesteatoma; in all these patients, the cholesteatoma sac was present in the attical-antral area without mesotympanic, protympanic and hypotympanic involvement;
- 8/22 subjects were affected by retraction pocket of the pars flaccida; in these patients, 6 presented an epitympanic compartment without disease while in 2 patients, we found a cholesterol granuloma in the anterior epitympanic space;
- 5/22 subjects were affected by chronic inflammation of the epitympanic compartment; these patients presented granulation tissue in the attical space with or without involvement of the incudomallear joint; in 2 patients, this inflammatory tissue was associated with attical-mastoid mucocele.

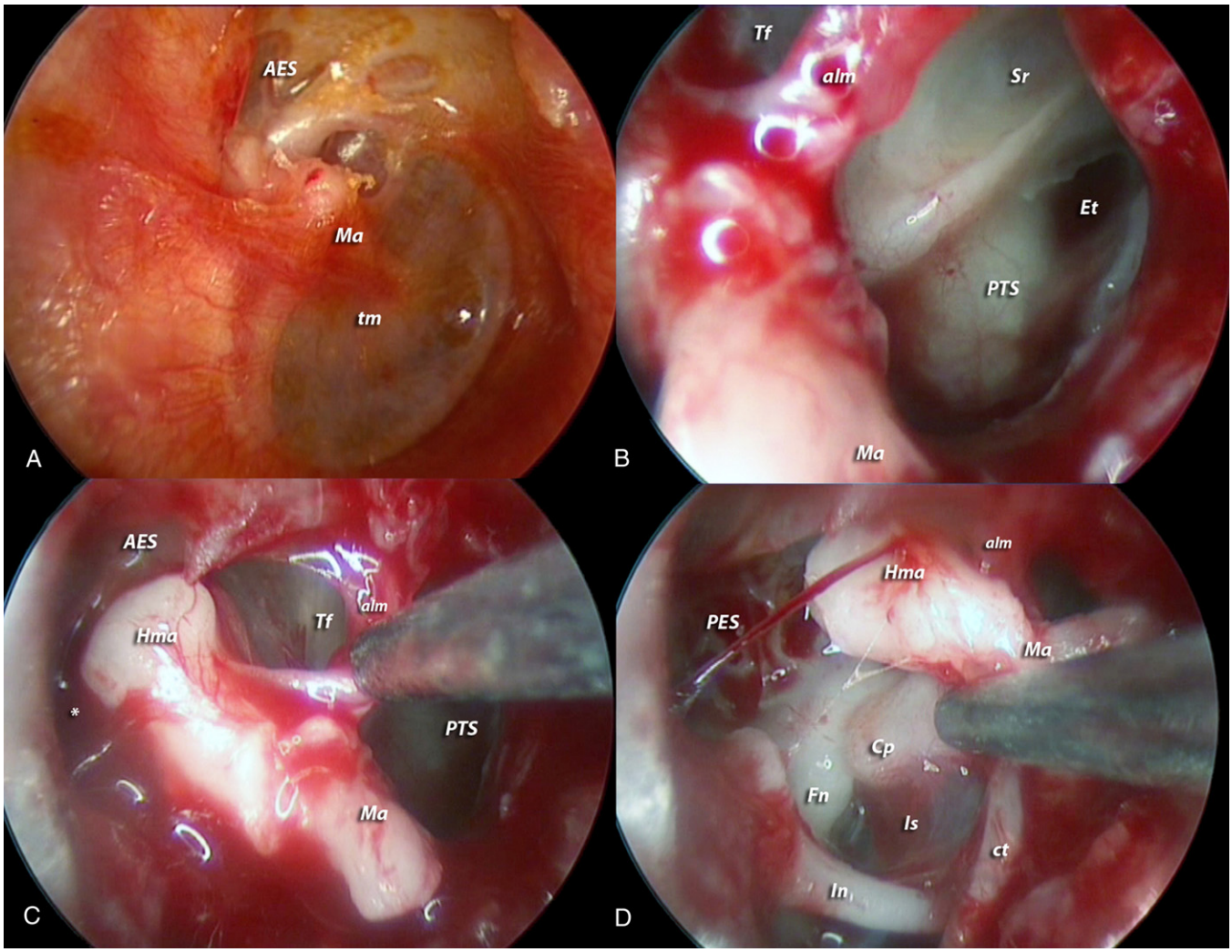


Fig. 3. (Right ear) Study group patient affected by attical aeration pattern Type A. (A) The 0° endoscopic exam of the drum showed a selective dysventilation of the anterior epitympanic space; the anterior portion of the pars flaccida presented a retraction pocket, and the scutum presents an erosion. (B) after creating a tympanomeatal flap entering inside the middle ear, the protympanic space was visible, the mucosa was normal, no granulation tissue or inflammatory web was present, and the lumen of the Eustachian tube was open. In this case, a wide supratubal recess was present. (C) after anterior atticotomy, the anterior epitympanic space and the tensor fold were visible just over the suction. In this case, the tensor fold is a complete fold dividing the anterior epitympanic space from the protympanic space. (D) the endoscopic approach allowed us to see a complete blockage of the isthmus by a mucosal fold involving posteriorly the incudostapedial joint and anteriorly the cochleariform process, tensor tendon and the handle of the malleus separating the mesotympanic space from the posterior epitympanic space. PES, posterior epitympanic space; AES, anterior epitympanic space; in, incus; Hma, malleus head; Ma, malleus handle; is, timpanic istmus; cp, choclearifom process; et, eustachian tube; aml, anterior malleolar ligament; tf, tensor fold; PTS, protympanic space; fn, facial nerve; ct, chorda tympani; sr, sovratubal recess; tm, tympanic membrane.

In all 22 subjects, the protympanic, mesotympanic and hypotympanic mucosa was normal, and the endotympanic lumen of the Eustachian tube was without disease, as the selection criteria required. Eighteen of the 22 patients presented chronic otorrhea before surgery.

3.1.2. Intraoperative endoscopic findings

Analyzing the anatomical structure that separates the epitympanic space from the mesotympanum (tympanic isthmus and tensor fold area), in all 22 subjects, a tree-type epitympanic diaphragm or attical aeration pattern was found:

- Type A (Figs. 2 and 3): 14/22 patients had a blockage of the isthmus associated with a complete tensor fold. This kind of blockage was present in 6 patients who showed selective retraction pockets without pathologic tissue in the epitympanic space, 3 patients presented epitympanic inflammatory tissue in the attic area, and 5 patients presented a limited attic cholesteatoma sac.
- Type B (Figs. 4 and 5): 6/22 patients had a blockage of the isthmus associated with an attical vertical blockage, consisting of a mucosal fold or granulation tissue involving the incudomallear joint creating an inflammatory web and exudate, and separating the anterior epitympanic space from the posterior epitympanic space with or without a complete tensor fold; this kind of blockage was found in 2 subjects who were affected by attical granulation tissue, 1 patient who presented an attical retraction pocket, and 3 patients who presented an attic cholesteatoma sac. In 6 patients with dysventilation type B, 3 patients presented an incomplete tensor fold. Two of these subjects presented an incomplete tensor fold associated with an attical vertical mucosal

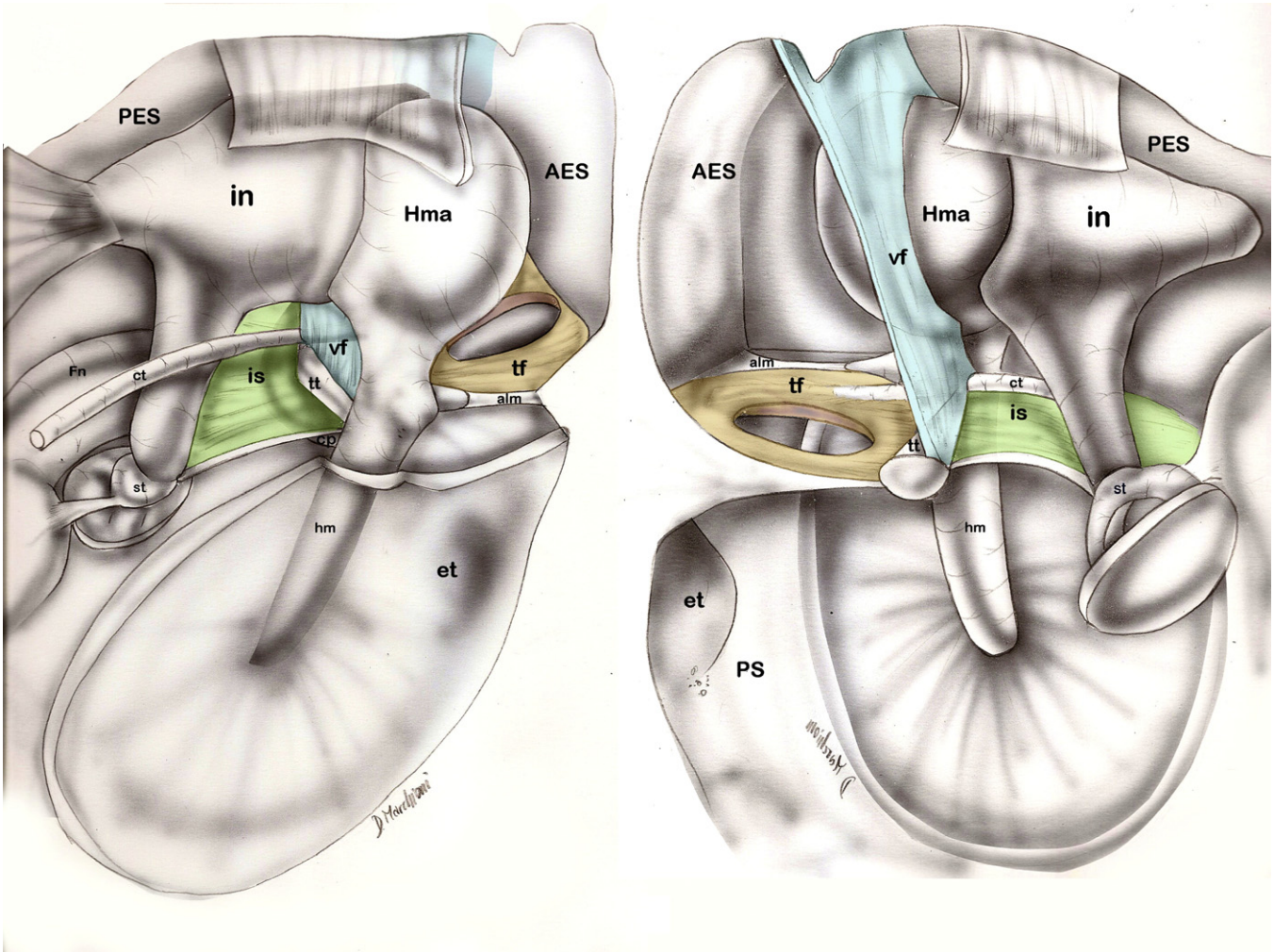


Fig. 4. (Right ear) A schematic drawing representing the attical aeration pattern Type B, a mucosal fold or inflammatory tissue causing a blockage of the isthmus is associated with a mucosal fold, or inflammatory tissue separating the posterior epitympanic space from the anterior epitympanic space, associated with an incomplete or complete tensor fold. PES, posterior epitympanic space; AES, anterior epitympanic space; in, incus; Hma, malleus head; is, timpanic istmus; st, stapes; cp, choclearifom process; et, eustachian tube; aml, anterior malleolar ligament; tf, tensor fold; PS, protympanic space; tt, tensor tympani; hm, malleus handle; vf, vertical fold; ct, chorda tympani.

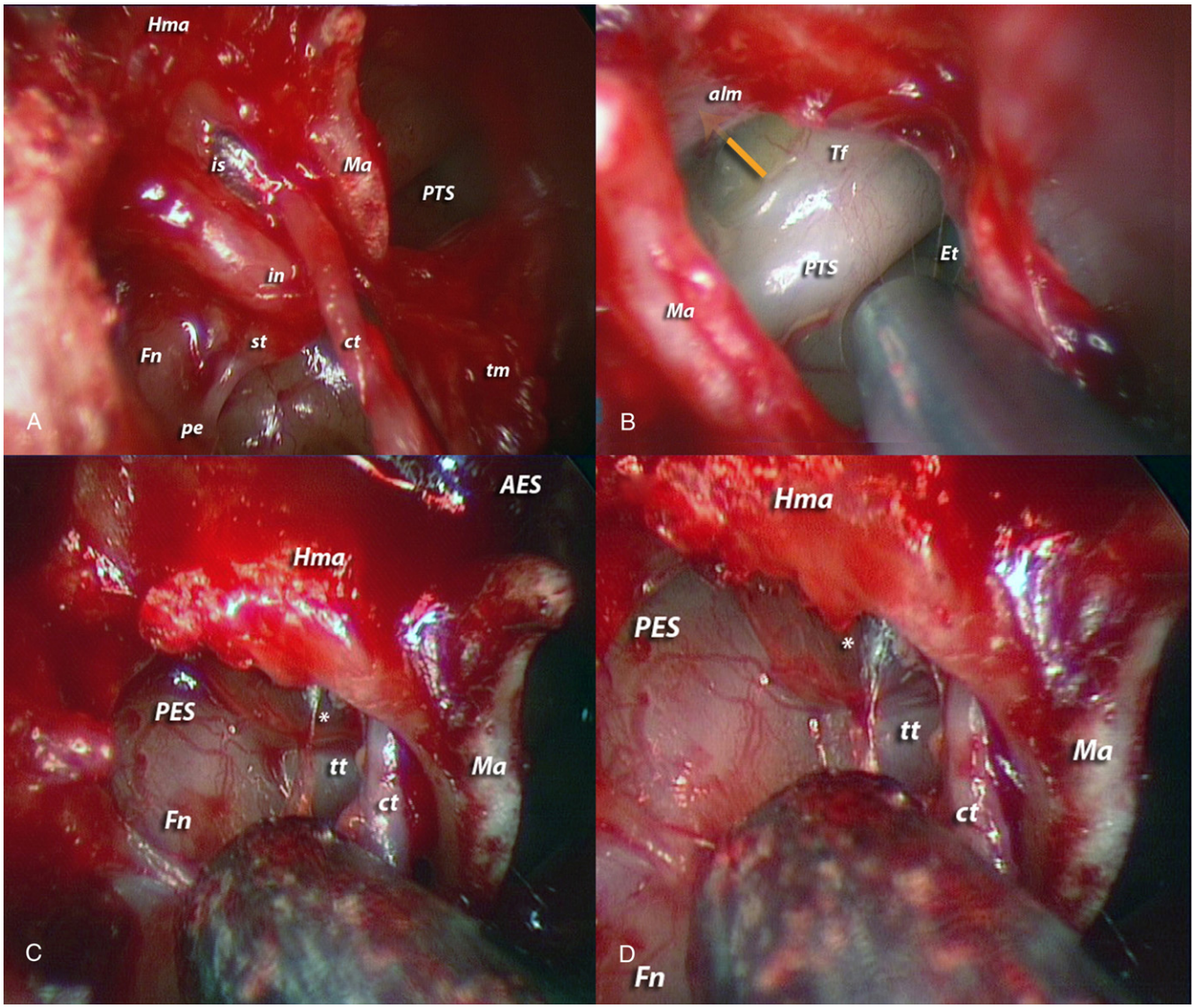


Fig. 5. (Right ear) Study group patient affected by attical aeration pattern Type B. (A) the 45° endoscopic approach showed a medialization of the handle of the malleus. This structure is close to the incudostapedial joint reducing the anterior isthmus space, a mucosal fold involving the tympanic isthmus between the handle of the malleus and the incus causing a blockage. (B) the protympanic space presented a normal mucosa, and the Eustachian tube lumen was open. In this case, the tensor fold was incomplete with a direct communication from the protympanum to the anterior epitympanic space (orange arrow). The suction helps to better visualize this area. (C) after incus removal, a vertical mucosal fold was visible over the cochleariform process separating the anterior epitympanic space from the posterior epitympanic space causing a selective dysventilation of the posterior epitympanic space and the antrum-mastoid area; (D) a 45° endoscopic view of the vertical fold and blockage. PES, posterior epitympanic space; AES, anterior epitympanic space; alm, anterior malleolar ligament; in, incus; Hma, malleus head; is, timpanic istmus; st, stapes; et, eustachian tube; tf, tensor fold; PTS, protympanic space; tt, tensor tympani; hm, malleus handle; pe, piramidal eminence; et, Eustachian tube; ct, chorda tympani; fn, facial nerve. *Isthmus blockage.

fold separating the posterior epitympanic space from the anterior epitympanic space. This anomalous vertical mucosal fold was inserted into the cochleariform process inferiorly, on the tegmen tympani superiorly and involved the head of the malleus anteriorly. One subject presented the same condition with granulation tissue involving the incudomallear joint and occupying the epitympanic space; an inflammatory web was present separating the posterior epitympanic space from the anterior epitympanic space. All 3 patients with incomplete tensor folds presented a selective dysventilation of the posterior epitympanic space and mastoid with healthy mucosa appearance.

- Type C (Figs. 6 and 7): 2/22 patients had a complete epidermization of the attic space causing a blockage of the isthmus, a complete antrum blockage and epidermization of the tensor fold area excluding the mesotympanic space from the epitympanic and mastoid spaces. This kind of blockage was found in 1 subject affected by attic cholesteatoma, and 1 subject affected by selective

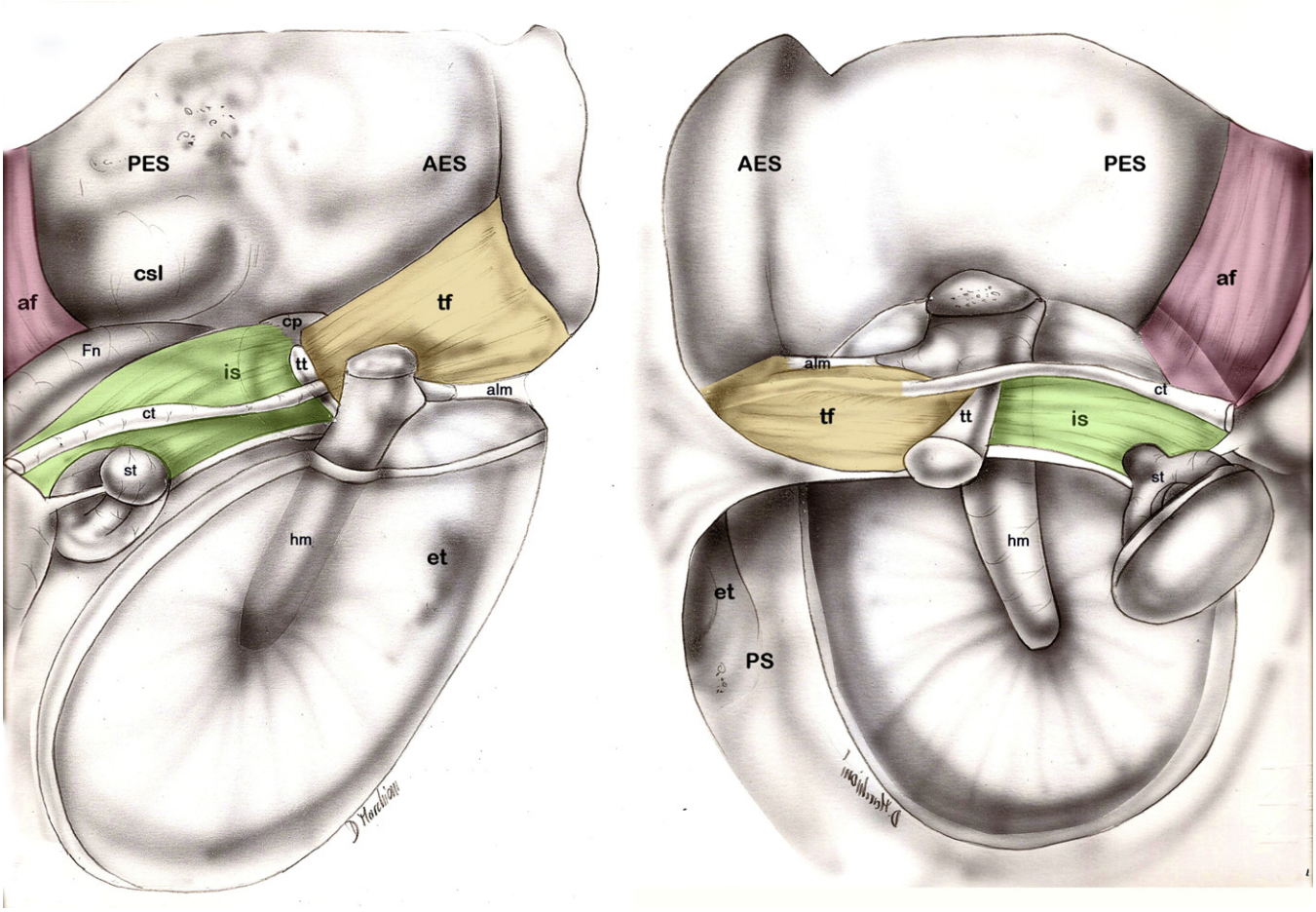


Fig. 6. (Right ear) A schematic drawing representing the attical aeration pattern Type C: a mucosal fold or inflammatory tissue causing a blockage of the isthmus associated with a complete tensor fold and an inflammatory tissue causing a blockage of the antrum. In both cases, this attical pattern was associated with erosion of the head of the malleus and the incus was not present. PES, posterior epitympanic space; AES, anterior epitympanic space; Hm, malleus head; is, timpanic istmus; st, stapes; cp, cochleariform process; cls, lateral semicircular canal; et, eustachian tube; aml, anterior malleolar ligament; tf, tensor fold; PS, protympanic space; tt, tensor tympani; hm, malleus handle; ct, chorda tympani.

retraction pockets. In all these cases, the head of the malleus was eroded and the incus was absent.

In 22 subjects, we found the following characteristics reducing the dimensions of the isthmus:

- In 8/22 subjects, the malleus presented a medialization with stapes adherences; this inflammatory web caused a reduction of the anterior isthmus spaces.
- In 8/22 subjects, an anomalous mucosal fold was visible causing an obstruction of the isthmus (involving the pyramidal process and stapes posteriorly and the cochleariform process and malleus anteriorly).
- In 5/22 subjects, the isthmus was blocked by granulation tissue with an inflammatory web involving the cochleariform process anteriorly, and the stapes and pyramidal process posteriorly.
- In only 1/22 patients, the obstruction of the isthmus was present as a tympanic retraction pocket between the malleus and the eroded long process of the incus.
- In 5 patients, we found an erosion of the long process of the incus, no erosion of the stapes and malleus was found.

3.1.3. Mastoid pneumatization

Of 22 subjects affected by anatomic blockage of the middle ear ventilation trajectories, 18 of 22 patients presented anomalies of the mastoid cells (Fig. 8) (hypopneumatized or sclerotic mastoid); of the patients who presented anomalous development of the mastoid cells, 7 of 18 presented a sclerotic mastoid, and 11 of 18 presented a hypopneumatized mastoid. On the other hand, only 4/22 patients presented a normal pneumatization of the mastoid spaces in the pathologic ears. Comparing the pathologic ear with the contralateral one intrapatient, the pathologic side of the study group had a higher prevalence of hypopneumatized or sclerotic mastoids (18/22) compared with the contralateral side (1/22), and this was statistically significant ($P < .001$).

The behavior of the mastoid pneumatization with respect to the type of anatomic blockage of the middle ear ventilation trajectories was also observed (Scheme 1):

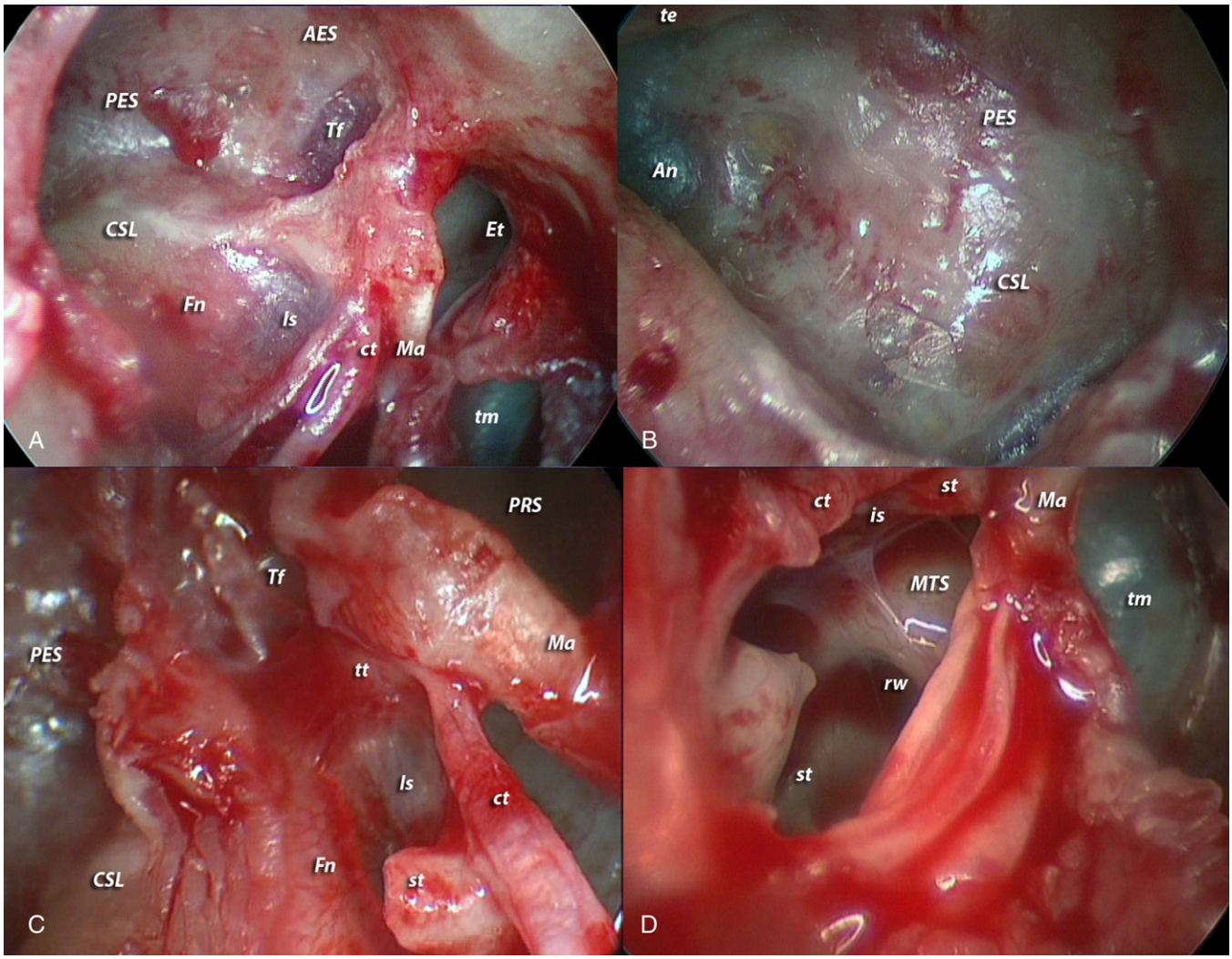


Fig. 7. (Right ear) Study group patient affected by attical aeration pattern Type C. (A) after creating a tympanomeatal flap entering inside the middle ear, the 45° endoscopic approach showed a complete epidermization of the epitympanic compartments, a total erosion of the head of the malleus was visible, and the incus was not present. A mucosal fold caused an isthmus blockage, and a complete tensor fold was present. The epidermization covered the isthmus fold and the tensor fold separating the mesotympanic space from the epitympanic space. The protympanic space was visible, and in this space, the mucosa was normal and the Eustachian tube lumen was open. (B) 45° endoscopic view of the posterior epitympanic space and antrum region. It is possible to see the lateral semicircular canal and the antrum; the skin formed a fold covering the antrum and separating the mastoid cells from the posterior epitympanic compartment. (C) after removal of epidermization from the tympanic isthmus and from the tensor fold, it was possible to see the superstructure of the stapes and a mucosal fold involving the isthmus and the corda tympani causing a blockage. This fold was inserted between the pyramidal process and the stapes posteriorly and the cochleariform process and tensor tendon anteriorly separating the protympanic space from the epitympanic compartments. (D) an endoscopic view of the mesotympanic space. In the anatomical spaces under the tympanic isthmus blockage, we observed a normal mucosa, and no inflammatory tissue was present. PES, posterior epitympanic space; AES, anterior epitympanic space; in, incus; Hm, malleus head; is, timpanic istmus; st, stapes; cls, lateral semicircular canal; et, eustachian tube; aml, anterior malleolar ligament; tf, tensor fold; PRS, protympanic space; tt, tensor tympani; tm, tympanic membrane; hm, malleus handle; ct, chorda tympani; rw, round window.

- Of 14 subjects affected by attical aeration pattern type A, 3 of 14 presented a normal mastoid pneumatization; 7 of 14 presented a hypo-pneumatized mastoid; 4 of 14 presented a sclerotic mastoid.
- Out of 6 subjects affected by attical aeration pattern type B, 1 of 6 presented a normal mastoid pneumatization; 4 of 6 presented a hypo-pneumatized mastoid; 1 of 6 presented a sclerotic mastoid.
- Out of 2 subjects affected by attical aeration pattern type C, 2 of 2 presented a sclerotic mastoid.

As readers can observe, patients affected by attical aeration pattern type C present the worst degree of pneumatization, while aeration of the mastoid seems to increase in type B and type A patterns.

3.2. Control group

The control group consisted of 16 patients, of which 7 patients were female and 9 were male; the mean age of the group was 39.8 years (+/− 15.2 SD).

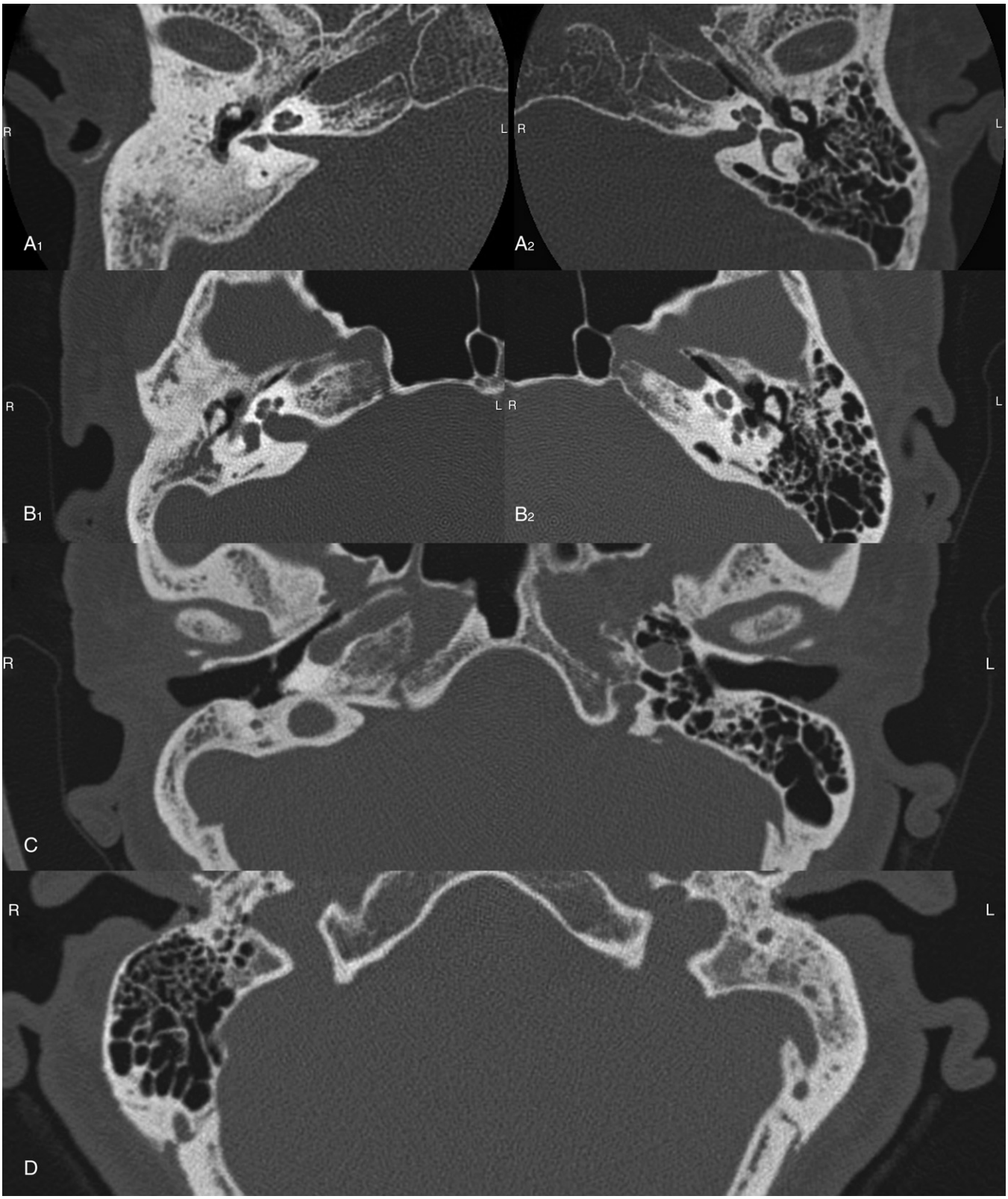
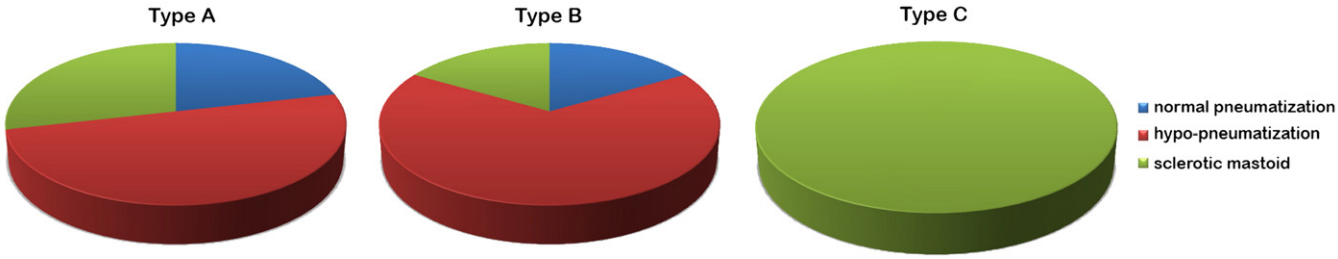


Fig. 8. A CT scan study of the mastoid in patients affected by a blockage of the isthmus (study group). (A1) sclerotic mastoid in a patient with attical aeration pattern Type C; (A2) the healthy side in the same patient showed normal pneumatization. (B1) patient affected by an attical aeration pattern Type B presenting a hypopneumatized mastoid on the affected side; in (B2), the healthy side in the same patient showed normal pneumatization. (C) hypopneumatization of the tip of the mastoid in the affected ear (left side), and normal pneumatization in the healthy ear (right side). (D), hypopneumatization of the tip of the mastoid in the affected ear (right side), and normal pneumatization in the healthy ear (left side).



Scheme 1. Distribution of mastoid pneumatization in the study group on the basis of the block classification.

3.2.1. Pathology and symptoms

Out of 16 patients affected by middle ear pathology without anatomic blockage of the middle ear ventilation trajectories, we found the following pathologies:

- 10 of 22 subjects presented perforation of the drum without inflammatory tissue in the middle ear.
- 6 of 22 subjects presented granulation tissue in the mesotympanic space without involvement of the tympanic isthmus and epitympanic compartments.

In all 16 patients, no inflammatory tissue was present in the attical areas, and the mucosa of the protympanic space was normal; 5 of 16 subjects presented a medical history of chronic otorrhea.

3.2.2. Intraoperative endoscopic findings

We analyzed the anatomical structure that separates the epitympanic space from the mesotympanum (tympanic isthmus and tensor fold area) in all 16 subjects without anatomic blockage of the middle ear ventilation trajectories. All 16 patients presented an opening between the pyramidal process posteriorly and the cochleariform process with the tensor tendon anteriorly. The space between the incudostapedial joint and the malleus was wide. No inflammatory web or mucosal fold was present (Fig. 9). Fourteen out of 16 patients presented a normal position of the malleus, 2 of 16 subjects presented a malleus medialization. Fourteen out of 16 patients presented a complete tensor fold, and the tensor fold presented a transversal position dividing the inferior supratubal recess from the anterior epitympanic space superiorly. In 2 of 16 patients, the tensor fold was incomplete. The tensor fold presented a posterior insertion on the tensor tendon and was inserting anteriorly on the bony roof of the anterior epitympanum.

3.2.3. Mastoid pneumatization

Out of 16 subjects without anatomic blockage of the middle ear ventilation trajectories, 14 patients presented a normal pneumatization of the mastoid spaces, and 2 patients presented a hypo-pneumatization of the mastoid cells; no sclerotic mastoid was observed in this group. Comparing the pathologic ear with the contralateral one intrapatient, in all 16 patients, there was symmetric pneumatization in both mastoid cells; 2 subjects presented a hypopneumatized

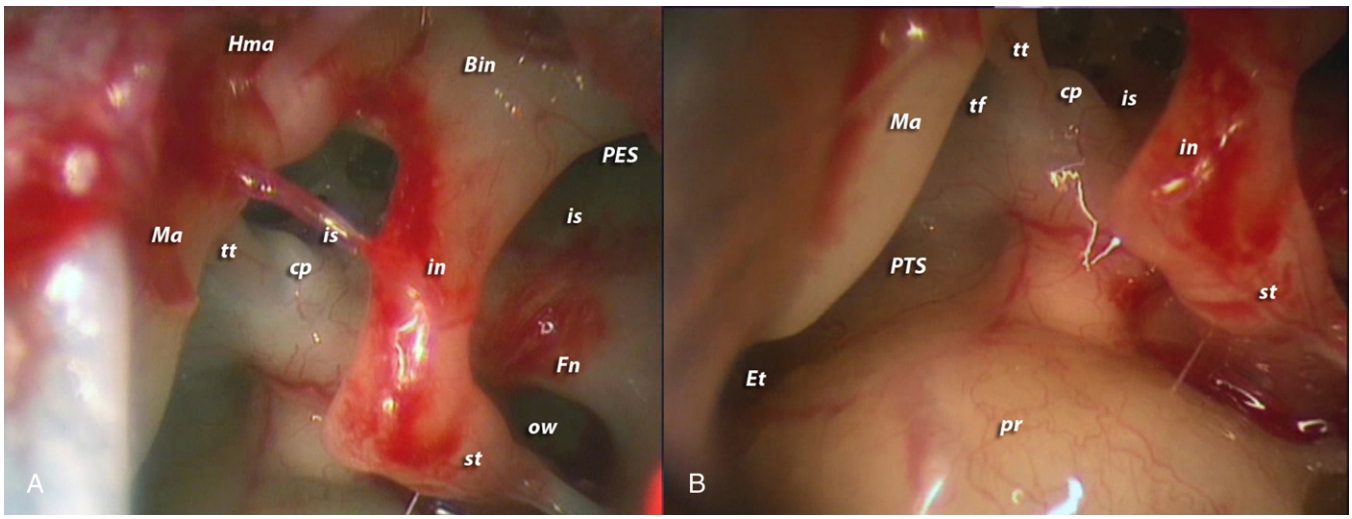
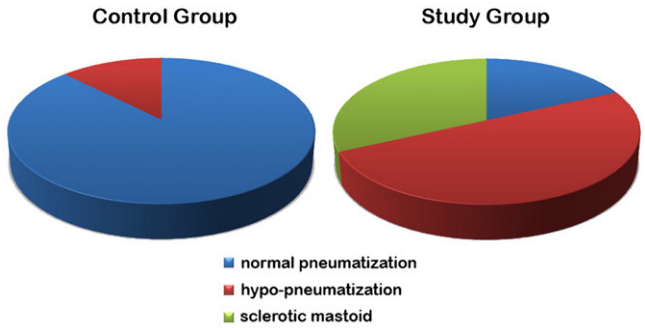


Fig. 9. (Left ear) Control group patient with a normal isthmus. The space between the cochleariform process and the incudostapedial joint was wide; the anterior epitympanic space and the posterior epitympanic space were visible, and the mucosa was normal. (A) endoscopic view with a 0° optical instrument. (B) endoscopic view with a 45° optical instrument. PES, posterior epitympanic space; in, incus; Hma, malleus head; is, timpanic istmus; st, stapes; cp, cochleariform process; PTS, protympanic space; et, eustachian tube; pr, promontory; tf, tensor fold; tt, tensor tympani; ma, malleus handle; ct, chorda tympani; ow, oval window.



Scheme 2. Inter-group comparison: distribution of mastoid pneumatization in the control group and in the study group.

mastoid on both mastoid sides, so there was no statistical significant difference between the pathologic ear and the controlateral ear in the control group, in terms of mastoid pneumatization (2 of 16 versus 2 of 16 with hypopneumatized mastoids, respectively) ($P = 1$).

3.3. Inter-group comparison

The prevalence of hypopneumatized/sclerotic mastoids in pathologic ears was compared between the study group (18/22) and control group (2/16). The higher prevalence of hypopneumatized/sclerotic mastoids in the study group was found to be statistically significant ($P < .001$) (Scheme 2).

4. Discussion

In 1946, Chatellier and Lemoine [4] formulated the concept of "the epitympanic diaphragm." The authors described different ligament and membranous folds, which, together with the malleus and incus, form the floor of a large epitympanic compartment. This space represents the upper unit and is aerated from the protympanic space through the tympanic isthmus.

Proctor [3] first described the tympanic isthmus in 1962. He considered this anatomic area as a small opening between the middle ear and the epitympanic space with the purpose of aerating the middle ear cleft. In his work based on fresh temporal bone dissections, Aimi [15] described the tympanic isthmus as a narrow passage between the tubotympanic cavity and the atticomastoid air space. He observed that obstruction of the tympanic isthmus is common in various types of middle ear disease and causes significant air-diffusion disturbance within the temporal bone pneumatic system.

Aimi [15] also noted that the factors that caused an obstruction of the tympanic isthmus were mucosal fold variations, inflammatory webs and exudate, retracted tympanic membrane, diseased attic mucosa and cholesteatoma. In our recent work on attic cholesteatoma, we found that obstruction of the tympanic isthmus is a consistent finding in patients affected by limited attic cholesteatoma [11]. Tympanic isthmus obstruction causes complete or incomplete separation of the tubotympanic cavity from the atticomastoid airspace.

Recently, the anatomy of the epitympanic diaphragm was studied by Palva and coworkers [6-10]. The authors described the "epitympanic diaphragm" which consists of three malleal ligamental folds (the anterior, lateral, and posterior), the posterior incudal ligamental fold and two purely membranous folds (the tensor fold and the lateral incudomalleal fold) together with the malleus and incus. All epitympanic compartments receive their aeration via the large tympanic isthmus between the medial part of the posterior incudal ligament and the tensor tendon. The authors observed that the aeration pathway from the Eustachian tube leads directly to the mesotympanic and hypotympanic spaces, whereas the epitympanum is away from the direct air stream and is only aerated through the tympanic isthmus, not including any possible auxiliary pathways.

Palva and colleagues [6,7] studied the anatomy of the tensor fold during temporal bone dissection. They observed that, in the majority of patients, the tensor fold was a complete fold separating the epitympanic compartment from the protympanum. In these patients, the isthmus was the only aeration pathway, however, in rare cases, it is possible to observe the presence of an incomplete tensor fold; in these cases, the anterior epitympanic space received aeration directly from the protympanum through the communication in the tensor fold area.

Palva et al [9] studied children affected by an isthmus blockage with a complete tensor fold and who had undergone a tympanostomy tube placement. They observed the persistence of inflammatory materials and cholesterol granuloma in the superior attic despite the tympanostomy tube. This condition may be the basis of attic cholesteatoma developing from a pocket retraction. Children with incomplete or absence of a tensor fold, presented good ventilation of the anterior attic.

Different authors have focused their attention on mastoid pneumatization and middle ear pressure variations [12,13,16]. Recently Sadé [16,17] noted that the functions of the atticomastoid spaces are important as an air reservoir to prevent rapid pressure change in the middle ear. In patients with blockage of the isthmus, this atticomastoid functional air reservoir is lost and the upper unit is under the constant strain of negative pressure across the tympanic diaphragm.

Sadé and colleagues [16-18] studied 39 adult patients affected by retraction of the tympanic membrane; they found a direct correlation between the degree of retraction and the middle ear volume displaced by the atelectasis. All 39 patients examined had a hypopneumatized or sclerotic mastoid. They observed that the mastoid is a natural gas reservoir that shares middle ear pressure aberrations. Well-pneumatized mastoids are rarely involved in chronic otitis media; instead, patients with retraction of the tympanic membrane usually have a hypopneumatized mastoid.

The authors concluded that the negative middle ear pressure is dependent on the degree of mastoid pneumatization; consequently, the pressure changes depend on the mastoid size; the sclerotic mastoid has a negative pressure [16,17]. The retraction pocket of the pars flaccida could be the first reaction to middle ear negative pressure in adults. This event acts as a buffering mechanism which counteracts middle ear negative pressure.

In our previous study [19] focused on the epitympanic size in patients affected by a limited attic cholesteatoma, we observed that the anterior epitympanic recess (AER) in an affected ear is smaller than in a nonaffected one. We hypothesized that the presence of a tympanic isthmus blockage associated with a complete tensor fold could exclude the AER from the posterior epitympanic space and from the protympanum. The blockage of the tympanic isthmus could create a selective negative pressure in the atticomastoid spaces; this chronic lack of aeration could provoke a hypodevelopment of the AER with a reduction of pressure level and consequently, an attic retraction and cholesteatoma sac development. This process is also possible in patients with a normal functioning Eustachian tube.

In the present study, we observed that patients affected by a blockage of the isthmus had a hypopneumatized mastoid. The blockage of the isthmus could be the basis for an epitympanic selective dysventilation with subsequent hypodevelopment of the mastoid size and volume. In these cases and also in patients with a normal functioning Eustachian tube, a selective negative pressure could be possible in the attic and the mastoid due to a pars flaccida retraction.

Although Eustachian tube dysfunction is the final common pathway for several types of pathologic changes in the actual tubal lumen resulting in negative middle ear pressure and subsequent retraction pocket formation, Palva and colleagues [6-10] demonstrated that an attic retraction pocket could occur through normal tubaric function; therefore, there should be other factors in the pathogenesis of attic cholesteatoma.

In fact, in these patients, endoscopic exploration of the protympanic and mesotympanic spaces showed a normal mucosa without inflammatory tissue in contrast to the epitympanic spaces where inflammatory tissue, cholesterinic granuloma and cholesteatoma were often present. In addition, patients with selective attic cholesteatoma presented a hypopneumatized mastoid associated with a blockage of the isthmus. It is possible to hypothesize that a selective negative pressure in the epitympanic space due to a progressive attical retraction pocket gives rise to a cholesteatoma sac.

No previous studies have been carried out during the surgical approach on patients affected by a middle ear chronic disease with blockage of the isthmus. Intraoperative evaluation of middle ear anatomy during endoscopic surgery allowed us to clearly visualize the presence of anatomic blockage of the middle ear ventilation trajectories. We have classified these anatomic blockage patterns into three types:

- Type A: blockage of the isthmus associated with a complete tensor fold (the majority of these patients presented a selective retraction pocket without pathologic tissue in the epitympanic space).
- Type B: blockage of the isthmus associated with an attical vertical blockage (consisting of a fold or granulation tissue involving the incudomalleal fold) separating the anterior epitympanic space from the posterior epitympanic space with or without a complete tensor fold.
- Type C: a complete epidermization of the attic space causing a blockage of the isthmus and a complete antral blockage excluding the mesotympanic space from the epitympanic and mastoid spaces.

All of these cases could present a normal functioning of the Eustachian tube associated with a selective dysventilation of the attical and mastoid spaces. This selective dysventilation patterns, excluding the attic and mastoid spaces from the mesotympanic space, are due to negative pressure and a hypodeveloped mastoid cell.

The long handle of the incus is located in the center of the isthmus dividing this anatomic space into two portions, the anterior isthmus, generally wider and the inconstant posterior isthmus. In our work, in patients with a blockage of the isthmus, we observed the presence of a long process of the malleus medialization reducing the space between the malleus and the incudostapedial joint. If the patient had a complete tensor fold, this condition could create a small anterior isthmus space due to selective chronic attical dysventilation.

Palva et al [8] suggested the removal of the tensor fold, to create a large new attic aeration pathway, in patients affected by a blockage of the isthmus, to restore normal ventilation of the attic space. In traditional microscopic middle ear surgery, some authors [20,21] have proposed different surgical approaches to visualize the tensor fold; otherwise, it is very difficult to observe this particular structure with a microscope. We have proposed [11] an exclusive endoscopic approach to the tensor fold.

In our personal series, tensor fold removal prevented postoperative retraction or cholesteatoma recurrence 1 year after the primary surgery. The use of the endoscope during surgery also permitted a good view of the tensor fold area and the isthmus timpani and, consequently, to enable us to understand the type of dysventilation pattern.

The goal of surgery in this kind of pathology could be restoration of normal ventilation of the attical-mastoid area. This solution is possible by removing the tensor fold and restoring the functionality of the isthmus.

5. Conclusion

Intraoperative evaluation of middle ear anatomy during endoscopic surgery for inflammatory pathology allows us to clearly visualize the presence of an anatomic blockage of

the middle ear ventilation trajectories. These blockages can be classified into three major types according to the intraoperative findings. Intrapatient and intergroup comparisons show that the presence of these blockages of the middle ear ventilation trajectories are associated with hypopneumatization/sclerotization of the mastoid, a typical sign of middle ear dysventilation pathologies. Further studies will be able to clarify to what extent selective dysventilation phenomena could be a principal factor in influencing middle ear pressure homeostasis.

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