



Tympanic Membrane Perforations and Tympanoplasty: New Profiles, New Strategies

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Introduction

Tympanic membrane perforations are defined as a lack of integrity of the tympanic membrane (TM), which can result from different etiologies and require different treatments. They are part of a spectrum of diseases known as chronic otitis media (COM) [1, 2].

The COM, in turn, can be defined in several ways. From a clinical point of view, it is typically characterized as an inflammatory condition usually associated with otorrhea. Chronologically, it refers to an inflammatory process in the auditory cleft, which lasts no less than three months. Finally, histologically, perhaps the most comprehensive definition describes it as an inflammatory process of the middle ear associated with irreversible tissue alterations [2, 3].

For many decades, didactically, tympanic perforations have been classified as *central* or *marginal*. The *central* ones are those with tympanic membrane borders around 360° of the perforation, while *marginal* ones lack tympanic borders in any region around the perforation [4–6]. Such classification gained relevance in the literature due to the possibility of the formation of secondary cholesteatoma, which comes from the epithelial migration of the external acoustic meatus (EAC). The absence of protection from the tympanic annulus would be the determining factor in this case because, when present, it would act as an anatomical barrier to the migration of EAC epithelium toward the middle ear [3, 4].

It is imperative to remember that the currently most used COM classifications emerged between the 1950s and 1970s.

Back then, the instruments available for evaluation were flagrantly less developed and accurate. Otomicroscopy, endoscopy (straight or angled), computed tomography (CT), and nuclear magnetic resonance have revolutionized the evaluation of COM. As a result, the comprehension of the disease and its etiopathogenesis are being reformed, while the classifications for didactic and research purposes have not yet completed their renewal cycle.

Today, we can have a broader and deeper view of the disease. For instance, our group has been engaged, over the past 20 years, in the studies of COM. The accumulated knowledge and the instruments that arose over those years are the main factors contributing to this deeper view. Namely, the tools are:

- The high-resolution otoendoscopy.
- The capture devices that allow the recording and exhaustive reanalysis of each otoscopy.
- The computerized methods for measuring and locating perforations.

Among all the aspects that a disease as complex and still little understood as COM presents, we consider etiopathogenesis to be the most relevant. Looking at the present, that is, the established pathology, we seek to understand the past (etiology) and try to modify the future (prognosis).

We believe that the current classification of tympanic perforations does not allow for such a broad approach to the disease. In other words, the current categorization system only considers the established pathology at the time of its assessment. At the same time, an evolutionary view could be more productive in terms of allowing the understanding of the disease globally.

In this chapter, we will approach not only the traditional aspects of tympanic perforations, such as the anatomy and biology of the tympanic membrane, but also new concepts arising from studies by our group and recently published. Further in this chapter, we will address clinical treatment and general concepts related to the surgical treatment applied to pathogenesis. We remember that surgical techniques can be found in specific chapters in this book.

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Brief Anatomical and Physiological Summary Applied to Pathogenesis

Anatomy and Biology of the Tympanic Membrane

The middle ear is a three-dimensional, small, and complex compartment whose primary function is transmitting sound from the air environment to the liquid environment of the inner ear. It begins with the capture of air acoustic waves, followed by the vibratory stimulus of the tympanic-ossicular system. Finally, it is completed by transmitting the waves to the vestibular scale of the cochlea via the oval window. The TM defines the lateral limit of the tympanic cavity, having the capture and the beginning of the sound transmission process as the primary function. The mechanism is primarily based on the difference in area between the TM and the stapes footplate. The ossicular chain conducts the sound pressure captured in the tympanic membrane to the footplate of the stapes, which has a considerably smaller area, thus concentrating the energy [7].

The approximate dimensions of the TM are 8 mm wide, 9–10 mm high, and 0.1 mm thick. Shrapnell [8], considering structural properties and mobility, divided the TM into two portions: one named *pars flaccida* (PF), superior, and the other as *pars tensa* (PT), inferior. The anatomical landmarks that divide PF and PT are the anterior and posterior malleolar ligaments, which are the extension of the tympanic ring. The ring, at the level of the tympanic spines, extends toward the lateral process of the malleus. The PT, in turn, was subdivided into four quadrants: the anterosuperior (AS), the anteroinferior (AI), the posterosuperior (PS), and the posteroinferior (PI), which are illustrated in a normal TM in Fig. 39.1.

The TM consists of three layers: an outer layer of keratinized squamous epithelium, a fibrous middle layer of mesodermal origin, and an inner endodermal mucosal layer. The membrane is approximately 130 μm thick, with the squamous layer measuring about 30 μm , the lamina propria about 100 μm , and the mucous layer 1 μm or less [7, 8].

The **outer epidermal layer** of TM is formed by a stratum corneum, a granular layer, a spinous layer, and a basal layer. Basal cells undergo cell division and migrate upward to replace dead and desquamated horny cells. The exact control mechanism that regulates this growth needs to be clearly understood. Studies have shown the presence of epidermal growth factors and fibroblast growth factors that are believed to promote the healing of tympanic membrane perforations. The epidermal layer also has migratory properties responsible for the ear's self-cleaning ability [7].

The human TM epidermis migrates centrifugally to the periphery, preferably in a posterosuperior direction, at a rate

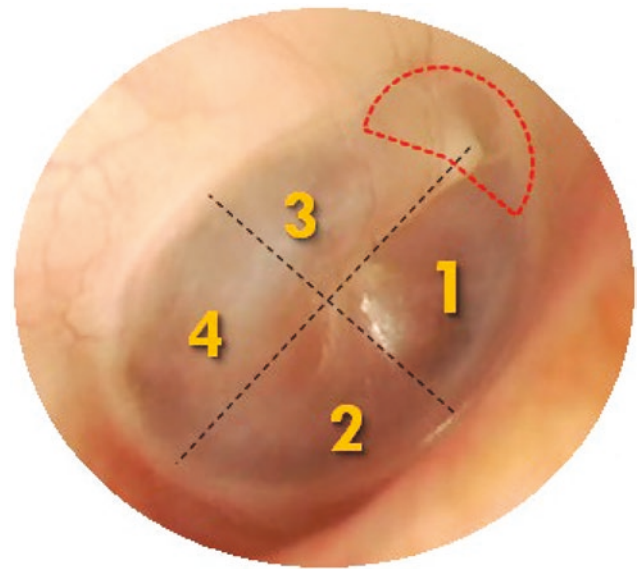


Fig. 39.1 TM considered normal—right ear. In red, the *pars flaccida* is delimited. The black dotted lines show the separation of the *pars tensa* into quadrants: (1) anterosuperior; (2) anteroinferior; (3) posterosuperior; (4) posteroinferior. Image from own collection

of about 131 $\mu\text{m}/\text{day}$, with a higher migratory speed in the umbo region. There is also a centripetal movement of the epithelium, which is speculated to be important in the healing process. Studies using cultures of fetal tympanic membranes have confirmed this unique feature. However, the exact cellular mechanism responsible for this migratory function is not clearly understood.

The epidermal layer also contains Langerhans cells, which are involved in the immune response as antigen-presenting cells, T lymphocytes, and mast cells. Thus, the tympanic membrane may present an immune response and, therefore, be part of the defense mechanism of the ear.

The **middle layer**, also called the lamina propria, has different composition depending on its location on the tympanic membrane. The lamina propria in Shrapnell's membrane, or *pars flaccida*, consists of a network of loose connective tissue, disorganized collagen and elastic fibers, and a network of blood vessels and nerves, internally and externally. The presence of elastic fibers explains the flaccidity of Shrapnell's membrane. There are also abundant histamine-filled mast cells in the *pars flaccida*. This finding, again, may be associated with primary or secondary hypersensitivity reactions with direct repercussions on the pathogenesis of otitis media.

In the *pars tensa*, the lamina propria consists of a subepidermal layer of loose connective tissue containing an internal network of blood vessels and nerve fibers. This layer, however, is composed of well-organized fibers, with radial fibers being the outermost and circular fibers being the inner-

most. This unique arrangement of fibers seems essential in the vibratory capacity of the tympanic membrane. It has critical reflections in understanding tympanic retractions and perforations, since the PS quadrant has particularities that differentiate it from the rest of PT and PF.

Specific Characteristics of the Posterosuperior Quadrant

This division mentioned before classically establishes the PF as the most sensitive area to retractions and, consequently, to the formation of primary cholesteatoma. However, recent studies indicate a very similar frequency in the formation of PF and PT cholesteatoma. With this information, and based on anatomical findings described below, we could begin to understand the PS quadrant as a transition between PT and PF since it has characteristics that sometimes resemble one region, sometimes the other.

The tympanic annulus consists of a thickening of the TM periphery, and it is inserted in the tympanic sulcus. In the PT, this combination between annulus and sulcus confers firmness and consistency to the region. However, as the tympanic annulus detaches from the sulcus, it goes toward the lateral process of the malleus, forming the anterior and posterior malleolar ligaments. Consequently, in the PF, there is no tympanic annulus. Thus, the TM is more malleable, filling the Rivinus notch and being attached directly to the scutum [9].

The tympanic sulcus, in its posterior region, is divided into two portions, separated, in most cases, by the emergence of the chorda tympani nerve. Inferior to the nerve, the sulcus maintains its characteristics identical to the inferior and anterior quadrants. It is well defined, with a depth between 0.5 and 0.9 mm, evident borders, and a stable surface. Above the nerve, the tympanic ring is no longer located within the sulcus but passes along the medial face of the posterior bone wall in 93% of the temporal bones studied by Paço et al. [9]. From that point on (the emergence of the chorda tympani nerve), the tympanic ring progressively becomes detached from the sulcus, which, in turn, progressively becomes shallower until it disappears. Topographically, the emergence of the chorda tympani nerve marks the limit of the posterosuperior quadrant. These characteristics confer less tension on the TM in the PS quadrant compared to the AS, AI, and PI quadrants. Figure 39.2 schematically shows such characteristics:

Another point we deem essential to highlight about the PS quadrant concerns the histology of the TM in this region about its middle layer (lamina propria). In PT, the lamina propria consists of collagen types II and IV and is connected to the malleus handle and the tympanic bone. It consists of two layers, one radially oriented and the other circular in shape. The radial fibers (stratum radiatum) are attached to the manubrium of the malleus and run radially to the annu-

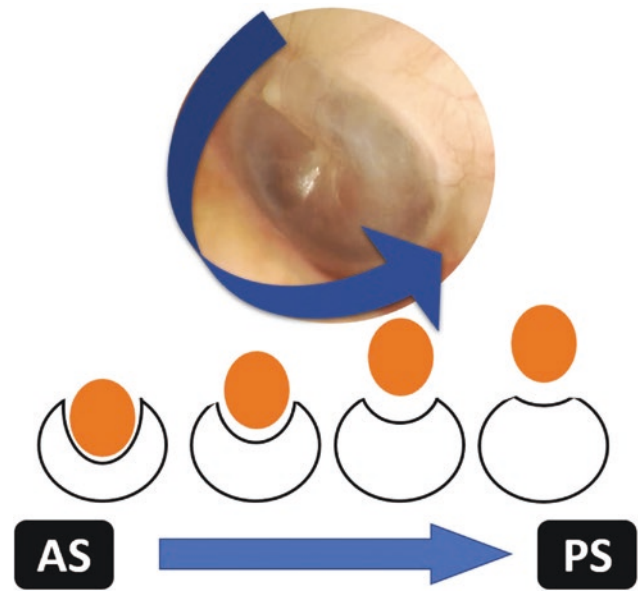


Fig. 39.2 Insertion of the ring (fibrous) in the tympanic sulcus (bone). In orange, the tympanic ring is represented. In black, the bone sulcus (groove). Progressively, when moving away from the anterior quadrants and reaching the PS, the sulcus will become shallower, and the annulus, in turn, more superficial and less adhered to the bone

lus. Meanwhile, the circular fibers (stratum circulare) are arranged concentrically with insertion into the manubrium. The latter are located medially in relation to the former.

In turn, the PS quadrant presents some peculiarities compared to the other portions of the PT, which would give it a greater chance of atrophy and consequent retraction in this region in case of negative pressure in the middle ear. First, the region does not have a developed circular fibrous layer, as mentioned in the previous paragraph (Fig. 39.3). In addition, its vascularization is more abundant, allowing greater penetration of collagenase-producing inflammatory cells, which have a more significant potential for destroying collagen fibers, which are already less dense by nature.

Furthermore, we must remember the anatomy of the PS quadrant, also characterized by peculiarities that increase susceptibility to the formation of retraction pockets in the region. Initially, a combination of the malleus handle, the subiculum of the promontory, and the tympanic annulus give the region anatomical accidents that culminate in reducing the distances and spaces for aeration in the region. The presence of the ossicular chain in this quadrant is yet another factor that differentiates it from the others because while the rest of the middle ear is filled only with air, the PS quadrant is full of structures competing for space. An occasional negative pressure in the middle ear could make the malleus medialize to the point of approaching, even more, the promontory, carrying the TM with it and, consequently, touching the subiculum of the promontory and the ossicular chain. Together with the greater flaccidity of the TM and the tym-

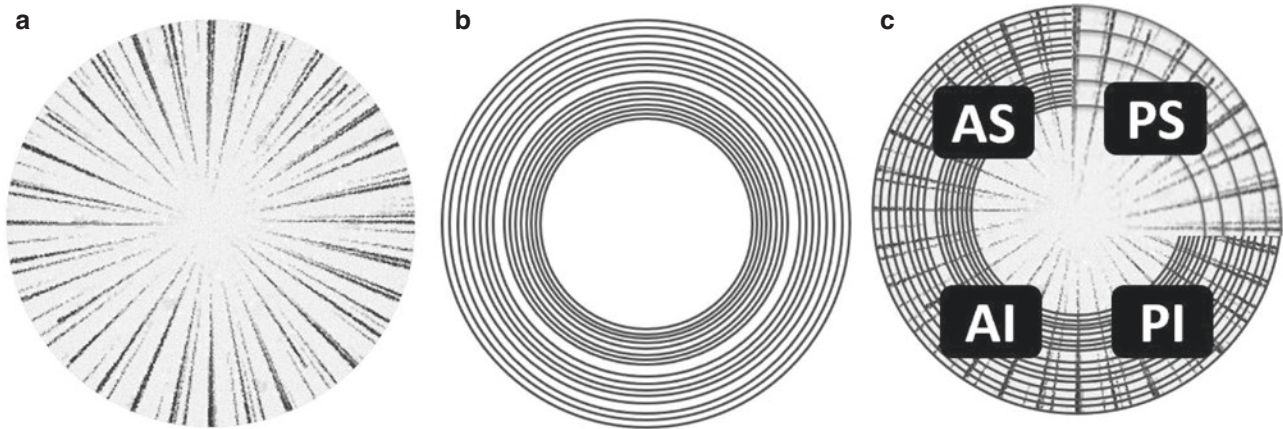


Fig. 39.3 Arrangement of collagen fibers in the TM. (a) Radial fibers, (b) concentric fibers, and (c) arrangement in the four quadrants of the *pars tensa*, where the PS quadrant has the least amount of concentric fibers

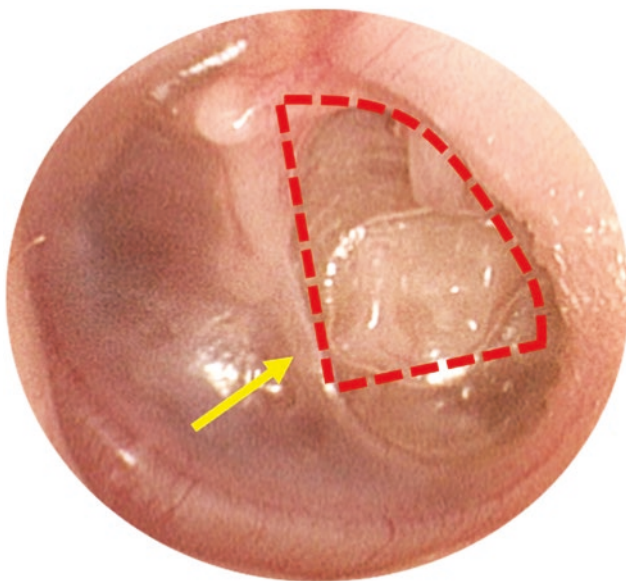


Fig. 39.4 Compartmentalization of the PS quadrant. The yellow arrow shows the point of major proximity between the TM (malleus umbo) and the promontory (subiculum). Negative pressure can make the malleus medialize, and, therefore, the TM will collapse over the promontory, isolating the PS quadrant from the ventilation routes

panic ring less inserted in the sulcus, there would be a compartmentalization of the region (Fig. 39.4), isolating it from the aeration routes originating from the mastoid via the aditus ad antrum, and the auditory tube, via the protympanum and mesotympanum.

Given all those peculiarities of the PS quadrant, we understand it as a transition between the *pars tensa* and the *pars flaccida*, as illustrated in Figs. 39.5 and 39.6.

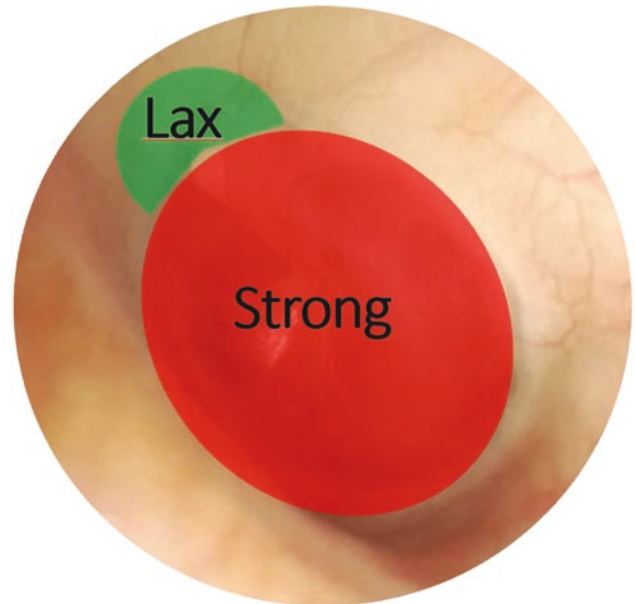


Fig. 39.5 Classic description of PT as robust and PF as weak (susceptible to retractions). We consider this division insufficient given the current knowledge of the anatomy and biology of TM

Healing of the Tympanic Membrane

For a perfect understanding of tympanic membrane regeneration processes, we also need to review some concepts about epithelial migration. Two types of epithelial migration are known. One is centrifugal movement from the umbo, which occurs at a rate of approximately 0.07 mm per day. In addition, studies have shown several areas of mitotic activity dispersed in the eardrum, which suggests the existence of

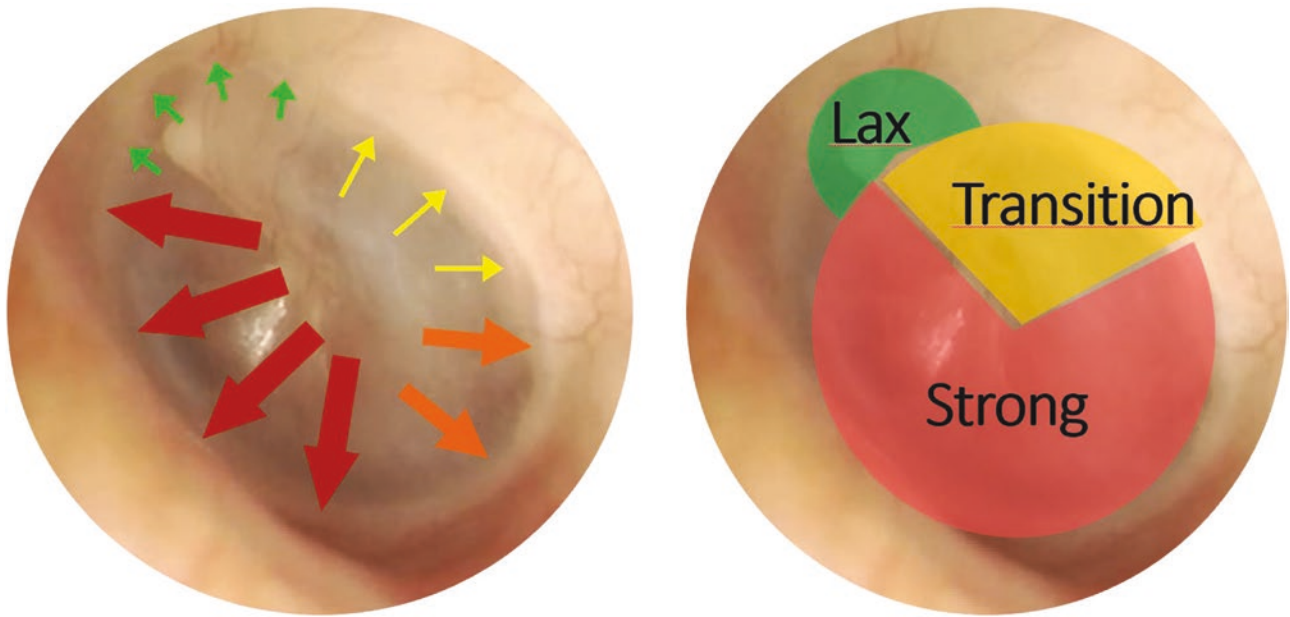


Fig. 39.6 TM firmness and tension in each quadrant, from the knowledge described in this section. Red arrows mark extreme tension, with a transition to green ones with less tension

multiple centers that generate this movement. This cell migration, called “keratin dispersion,” aims to remove keratin, cerumen, and foreign bodies in the external ear.

A second migratory pattern is a centripetal movement which, in turn, seems essential for tympanic perforations' healing. Unlike the wound healing in other tissues, mitotic activity is not confined to the wound edge only but rather the entire pars tensa of the tympanic membrane, with more significant activity around the tympanic ring and 2 mm from the edge of the lesion and, to a lesser extent, around the manubrium of the malleus. This distant migratory process is also suggested by the lack of proliferation of basal cells at the edge of the lesion, contrary to any other tissue where this cellular activity is intense.

Several studies have demonstrated the importance of F-actin microfilaments, a cytoskeletal contractile protein, in the migration of the squamous epithelium. Furthermore, this protein is abundant in the basal epithelial layer, suggesting that this layer plays an essential role in epithelial migration and consequent sealing of tympanic perforations.

The healing process traditionally involves the stages of hemostasis, inflammation, proliferation, remodeling, and contraction. First, platelets adhere and aggregate to injured cells in the wound, leading to vasoconstriction and thrombus formation. This event is followed by the formation of an inflammatory exudate composed of neutrophils, macrophages, and cytokines, with subsequent conversion of the fibrin matrices of the lesion into glycosaminoglycans and proteoglycans. At this point, when we talk about the healing of other tissues, fibroblasts proliferate, and collagen is deposited in the wound, with the consequent formation of granula-

tion tissue matrices, which allows the spread of epithelialization.

In the tympanic membrane, however, studies have shown that epithelial regeneration precedes fibrous proliferation. Immediately after perforation, there is retraction of the edges of the pars tensa, followed by hemostatic and inflammatory changes. Epithelial hypertrophy and advancement of the edges of the lesion are early events and can be seen only 48 hours after the insult. Layers of squamous epithelium migrate in an attempt to cover the perforation in the first place. Only at a second moment does the proliferation of the fibrous component of the lamina propria begin. The hypertrophy of the wound edges reduces the dimensions of the tympanic continuity solution, which is quickly followed by the proliferation of other layers of the epithelium. These events are analogous to grafts' "bridge" function in chronic otitis media surgery. In these situations, the graft acts as a substitute for the stratum corneum, over which currents of epithelialization slide in order to repair the perforation.

The lateral (epithelial) and medial (mucosal) tympanic layers regenerate during this process. On the contrary, the structure of the lamina propria remains fragile, consisting only of a few disorganized fibers and a few fibroblasts. Thus, in some situations, the process can lead to forming a practically dimeric tympanic membrane (lateral and medial layers only).

Efforts are being made to unravel the biomolecular secrets of tympanic membrane healing. Animal studies have demonstrated the presence of epidermal growth factor (EGF) and primary growth factors—basic fibroblast growth factor (bFGF) in the tympanic membrane as early as three days

after perforation. Growth factors are cytokines that regulate cell proliferation and differentiation. Receptors corresponding to these factors are also being discovered in animal models, suggesting that both may play relevant roles in TM perforation repair processes.

Symptoms

The cardinal symptoms of tympanic perforation are otorrhea and hearing loss. Other less frequent symptoms are otalgia, dizziness or vertigo, and, very rarely, facial paralysis. Right now, it is essential to point out that every patient with tympanic perforation will present two situations that may alternate with a relatively high frequency: infected and noninfected perforation. Such distinction is of fundamental importance to be recognized by the otorhinolaryngologist, since it may require clinical treatment and change planning, counseling, and surgical prognosis. Figure 39.7 illustrates the situation described above.

Hearing Loss: The patient with noninfected perforation will mainly report hearing loss symptoms. The main component is conductive due to the lack of area in the TM to capture the sound wave for consequent concentration in the footplate of the stapes and sound amplification. However, in some cases, the sensorineural component may also be present, configuring mixed hearing loss. One hypothesis for the existence of the sensorineural component would be cell death due to the migration of toxins to the inner ear, via the round window membrane, during infectious episodes. The intensity of the conductive loss is closely related to the degree of middle ear injury, and a conductive loss no higher

than 20 dB typically indicates the integrity of the ossicular chain. On the other hand, erosion of the ossicles, particularly of the long process of the incus, or its fixation, usually produces hearing losses of 30 dB or more.

Otorrhea: It is the most common manifestation of chronic otitis media, occurring primarily during perforation infection periods. Short-term, intermittent mucoid drainage usually denotes a simple perforation without major pathological findings. Once the clinical treatment is carried out, the infection will resolve, and, consequently, the otorrhea will stop. On the other hand, chronic, constant, purulent, bloody, and bad-smelling otorrhea, which continues despite treatment and clinical management, is usually associated with significant middle ear and mastoid pathologies.

Otalgia: Pain is not frequently associated with tympanic perforations but may be present in cases of infectious exacerbation. Still, when present, it requires a careful approach. Concomitant external otitis should be ruled out, sometimes due to self-sensitization of the EAC skin or suppurative complications such as abscesses, although rare when the cholesteatoma is absent.

Vertigo: Reports of vertigo episodes associated with middle ear pathology, with or without cholesteatoma, represent chemical irritation (serous labyrinthitis) or pyogenic invasion (purulent labyrinthitis) of the labyrinth. Concerning TM perforations, it can be present when there is very exuberant inflammation of the middle ear, with granulation tissue and pus generating an extension of the inflammatory process into the inner ear. In any of these circumstances, assessment must be carried out promptly and treatment instituted as soon as possible.

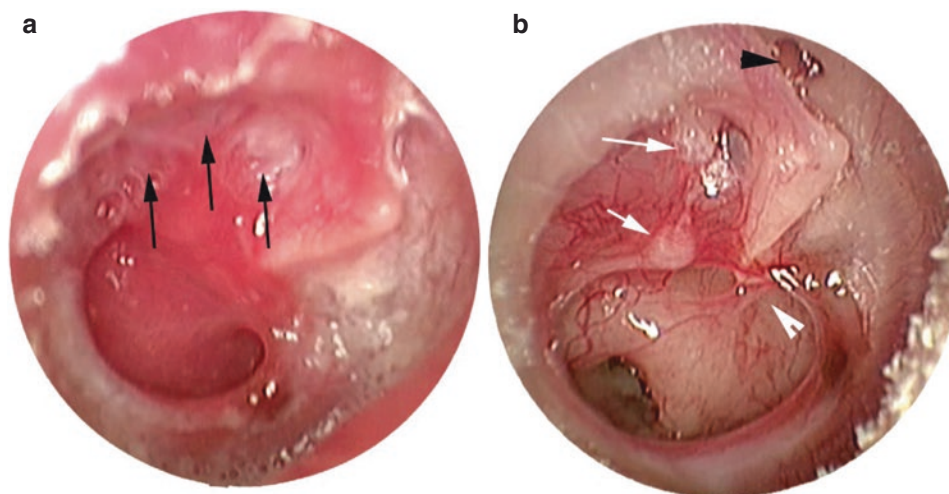


Fig. 39.7 Tympanic perforation in two different moments. (a) When infected, with an appearance that may suggest a cholesteatoma. Note the hyperplastic mucosa and the accumulation of epithelium in the posterosuperior quadrant (black arrows). (b) After clinical treatment and without infection. The absence of mucosal hyperplasia makes it possible

to visualize the ossicular chair, with an erosion of the long crus of the incus (long white arrow) and myringostapediopexy (short white arrow). It also allows for identifying signs of retraction, such as medialization of the malleus handle (white arrowhead) and attic retraction (black arrowhead)

Facial Nerve Paralysis or Paresis: The finding of progressive or complete peripheral facial paralysis associated with COM indicates the involvement of the nerve in its tympanic-mastoid segment until proven otherwise. It is infrequent to occur in perforations and other clinical forms without cholesteatoma. In these rare situations, surgery must be performed immediately. Thus, the pathological process can be completely evacuated, and the path of the seventh cranial nerve can be thoroughly explored from the geniculate ganglion to the stylomastoid foramen.

Clinical Assessment

Otoscopy

Otoscopy is the major step in evaluating tympanic perforations, and the otorhinolaryngologist must obtain as much information as possible during the TM inspection. Therefore, it is essential to use technologies such as otomicroscopy, otoscopy, and, of course, to adequately prepare the patient for the examination and subsequent recording.

If present, otorrhea must be entirely aspirated, and epithelial debris and desquamation of the external acoustic meatus must be completely removed. If present in TM or EAC, granulation tissue may require focal cauterizations, followed by antibiotics and topical corticosteroids. Granulation can sometimes hide underlying pathologies, especially cholesteatoma associated with perforation. Figure 39.8 shows a tympanic perforation in one ear with granulation in a risk area, as it could hide a possible posterior epitympanic cholesteatoma. In this case, the otorrhea could be perpetuated both by the granulation continuously secreting or by the possible cholesteatoma hidden by it. It undoubtedly needs to be treated to obtain the proper diagnosis.

Fungal co-infection (Fig. 39.9) should be treated whenever identified, especially before surgical procedures, as closing the EAC at the end of surgery and during the postoperative period could boost the proliferation of the fungus. Sometimes, the identification is quite obvious, with the presence of hyphae in the EAC before the aspiration (Fig. 39.9a) or also adhered to the TM and inside the middle ear through the perforation (Fig. 39.9b). At other times, the signs may be more subtle, such as lumpy otorrhea or refractory to antibacterial treatment.



Fig. 39.8 Granulation tissue on the edges of the tympanic perforation extends to the AS quadrant and the attic region. Therefore, the treatment of granulation is fundamental, not only as part of the clinical improvement but also for adequate preoperative evaluation

Fig. 39.9 Fungal infection in COM. (a) Hyphae filling the EAC before aspiration. (b) Hyphae adhered to the TM and infested the middle ear through the perforation

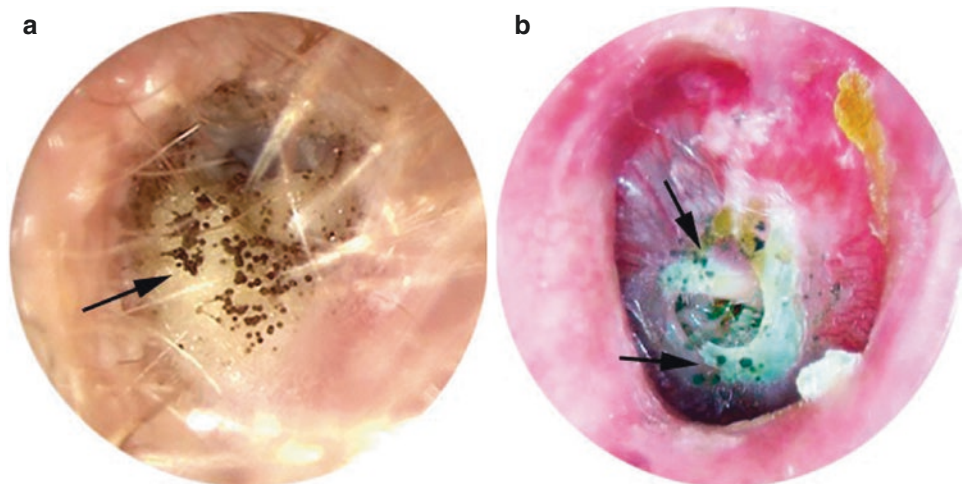
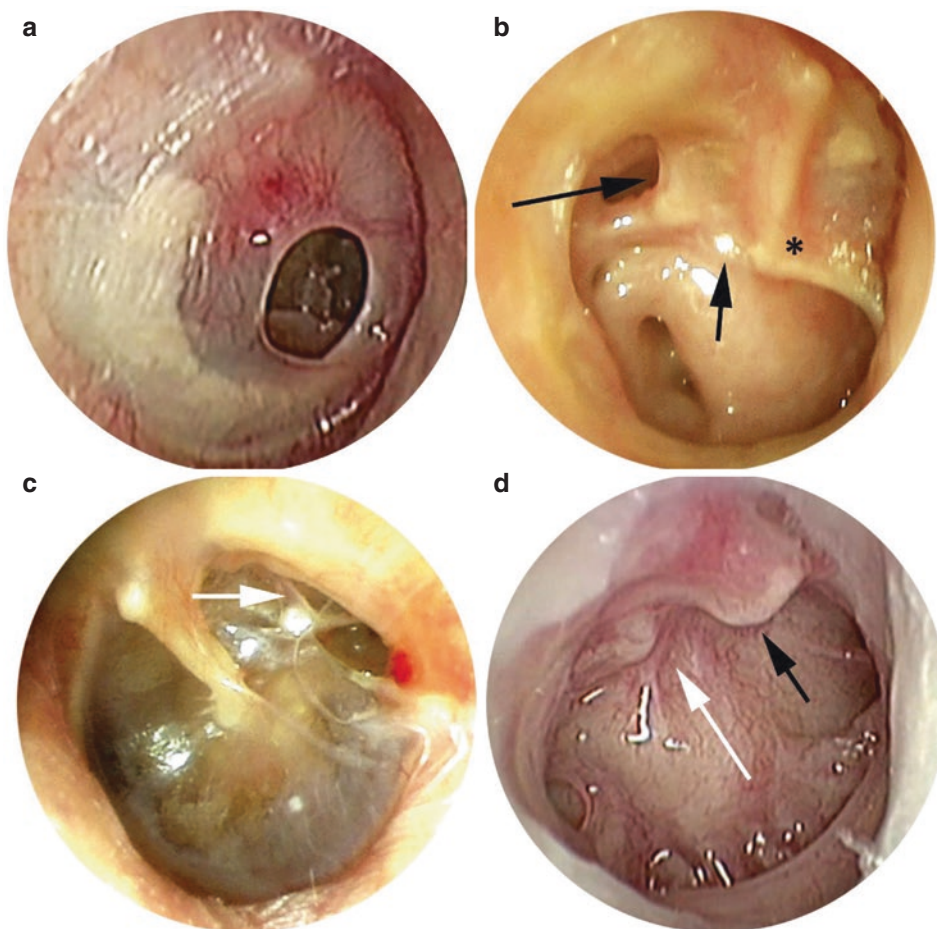


Fig. 39.10 Signs of retraction before perforation. (a) No signs of retraction. (b) Medialization of the malleus handle, including touching the promontory (asterisk); tympanic membrane touching the promontory (short black arrow), and tympanic membrane adhering to the ossicular chain (long black arrow). (c) Erosion of the long crus of the incus and tympanic membrane touching the ossicular chain, including myringostapediopexy (white arrow). (d) Medialization and amputation of the malleus handle (black arrow); tympanic membrane adhered to the promontory (white arrow) and ossicular chain



The presence of signs of retraction that occurred prior to tympanic perforation must also be evaluated. In this task, otoendoscopy is indispensable, with the objective of image magnification, better lighting, and consequent detailing of the characteristics pursued. The following signs are considered suggestive of the previous retraction: (1) medialization of the malleus handle; (2) tympanic remnants over the headland; (3) tympanic remnants over the ossicular chain, and (4) ossicular chain erosion. Figure 39.10 illustrates those signals, which will be better discussed later in this chapter.

We suggest the reader refer to the chapter Office Examination in Chronic Otitis Media to complement the steps of evaluation and registration of otoscopy.

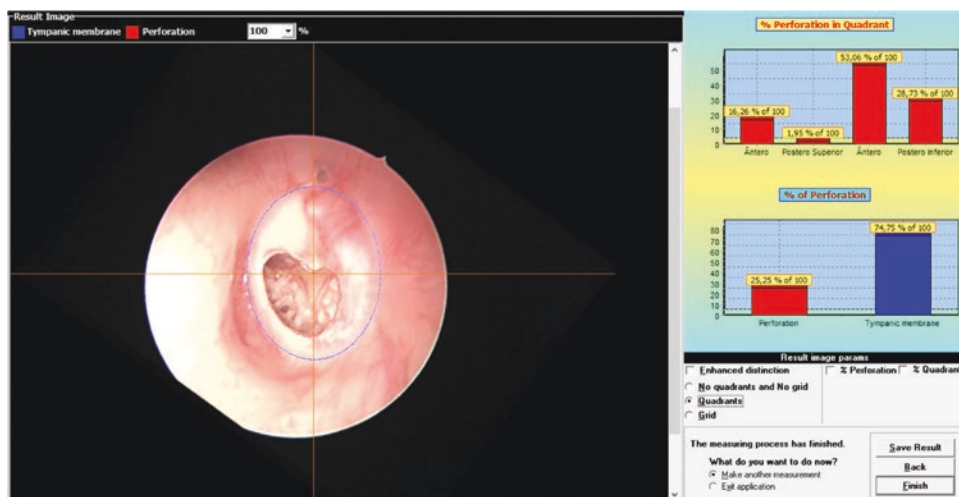
Size and Location

The analysis of the total affected area (TM perforation size) and the location according to the TM quadrants is also a relevant factor in the assessment. The posterior

quadrants of the TM are more subject to retractions and, consequently, to retractions-perforations. This fact is due to a set of anatomical and physiological factors of the PS quadrant, which have already been described and make the proper location of the disease in TM important, including for surgical planning.

Developed in partnership with the Federal University of Rio Grande do Sul and the Federal University of Santa Catarina, the Cyclops Auris Wizard software is used in our group to determine the size of tympanic perforation, as well as the location in each quadrant. The program is easy to use and intuitive, allowing its use by medical students until experienced otologists after brief training. Measurements are in relative size, initially taking into account the percentage of perforated area in a reason to the total area of the TM. Subsequently, each quadrant's relative percentages of perforation are provided in relation to the total perforated area. Figure 39.11 shows the result of a measurement.

Fig. 39.11 Result of a TM perforation measurement with the Cyclops Auris Wizard



Audiological Evaluation

Acumetry should be routinely performed with a tuning fork at 512 Hz. The otologist can never do without acumetry, especially during preoperative evaluations. A conductive loss pattern is expected, and sometimes mixed loss can be identified. If the otologist suspects deep sensorineural loss, it is essential to share this information with the audiologist.

Tonal and vocal audiometry is indicated in all patients with tympanic perforation. Similar to otoscopy, proper ear preparation is necessary before audiometry. The presence of accumulated debris or otorrhea in the EAC and granulations in the middle ear or mucosal hyperplasia may overestimate the conductive component of the patient in question.

Hearing loss will be characteristically conductive in patients with tympanic perforation. The degree of loss will be influenced by factors such as perforation size, perforation location, presence of tympanosclerosis, and ossicular chain involvement, whether due to mucosal adherence or ossicular erosion.

We can come across a mixed hearing loss with relative frequency, with the sensorineural component also in a variable degree. The etiology of sensorineural loss seems to be related to chronic inflammation, leading inflammatory mediators to the inner ear (via round window), and consequent cochlear cell destruction. It is an essential factor in patient counseling, as the conductive component can often be improved with surgery, whereas the sensorineural component cannot. Very pronounced bone pathway asymmetries should be investigated for possible retrocochlear etiologies.

Radiological Evaluation

We do not indicate ear computed tomography (CT) as a routine in evaluating patients with tympanic perforation. Central, dry perforations, without signs of associated retraction or other signs described below, may dispense with radiological evaluation. However, several situations may require CT imaging in COM without cholesteatoma.

It is essential to emphasize the gain in quality that CT has gained in recent years; as the resolution has increased considerably, the radiation used in the exam has also decreased. Today, an image of poor quality, with low resolution and coarse cuts, is unacceptable. In addition, the availability of coronal, axial, and sagittal oblique sequences is essential. Thus, the microscopic structures of the middle ear and mastoid are detailed. We also recommend reading the specific chapter.

Despite adequate clinical management and expected care by the patient, a perforation that keeps discharging requires tomographic evaluation to evaluate the mastoid cells. The filled mastoid cells can indicate the presence of accumulated secretion, granulation tissue, or cholesterol granuloma, which can act as a permanent source of bacteria and biofilms, perpetuating the mesotympanum's inflammation. Often, mastoidectomy associated with tympanoplasty will be indicated in these cases.

Conductive hearing losses greater than expected for the patient's otoscopy also require investigation through imaging examination. In a simplified way, an air–bone gap (ABG) above 25 dB will require investigation by high-resolution tomography. Not only can we find ossicular chain erosions

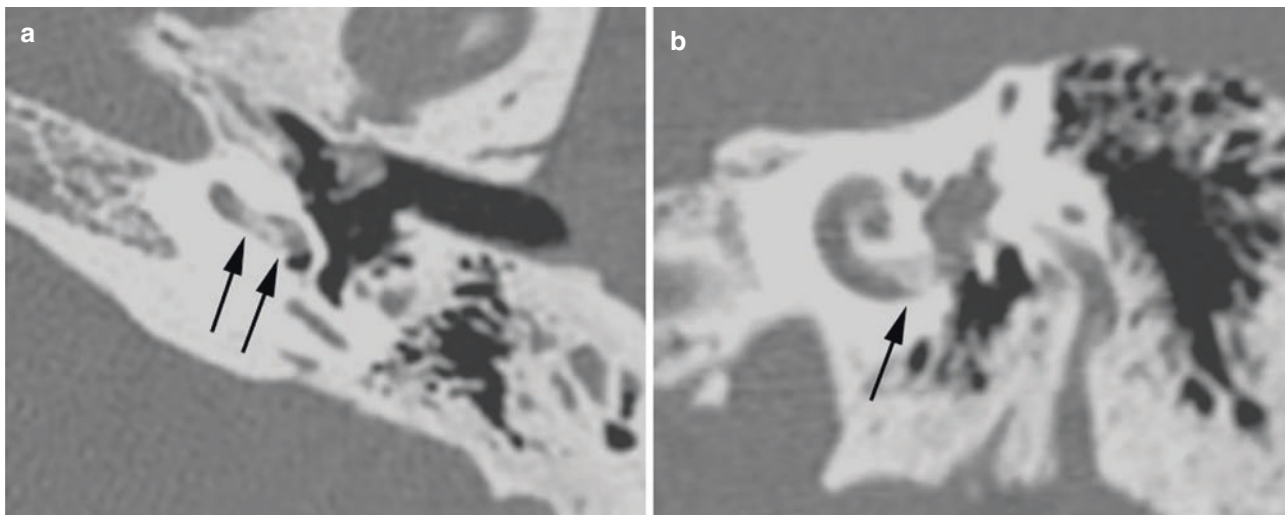


Fig. 39.12 Labyrinthitis ossificans. (a) Axial section shows calcification of the basal turn of the cochlea in a patient with COM without cholesteatoma (black arrows). (b) Oblique sagittal view from the same patient, showing better detail of the calcification (black arrow)

(sometimes not visualized in otoscopy), but also ossicular chain fixations and tympanosclerosis can be seen.

A sensorineural component is also a warning sign for investigation through imaging. In rare cases, chronic otitis media, even if not cholesteatomatous, can affect the inner ear, with intracochlear calcification and cell injury. This situation, known as labyrinthitis ossificans, can be seen in Fig. 39.12. In addition, asymmetric sensorineural hearing loss may require further investigation with nuclear magnetic resonance.

- Quick Tips: When to perform image in TM perforation?
 - A TM perforation that never gets dry
 - Conductive hearing loss greater than 25 dB
 - Sensorineural hearing loss
 - Asymmetric sensorineural hearing loss

Classification

Central Versus Marginal: The Traditional Classification

Historically, tympanic perforations are divided into **central**, that is, if there are TM borders in all 360° of the perforation, and **marginal**, if there is a lack of any of the perforation edges (Fig. 39.13). This classification gained relevance in the literature due to the potential effect of secondary cholesteatoma formation from a marginal perforation. According to this theory, there would be migration of epithelium from the EAC toward the interior of the middle ear, allowed by the

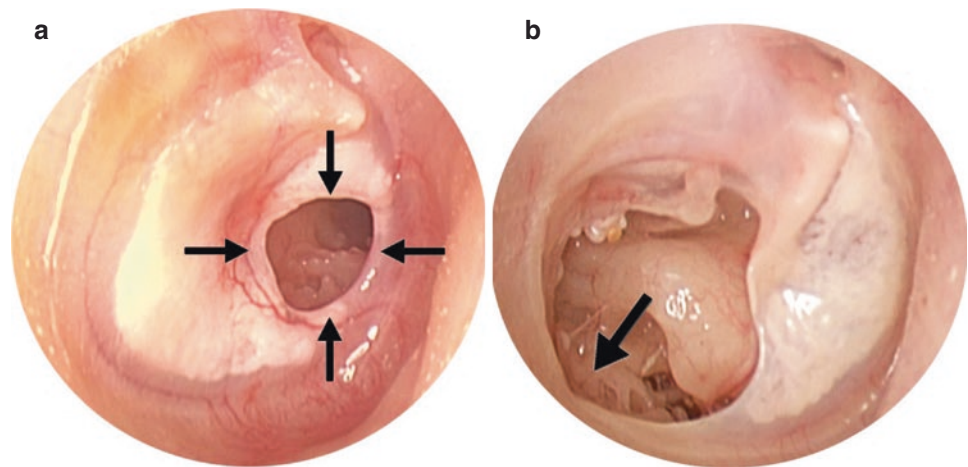
absence of anatomical protection provided by the tympanic annulus. However, such a theory is not consensual, being challenged daily in the daily practice of otologists and already criticized in the literature.

Still, the classification according to the borders of the perforation can be contested in other respects. Indeed, the most relevant would be the need for more practical applicability of the classification in therapeutic procedures. Even after decades of using the current division, we have still not been able to identify any indication for clinical or surgical treatment, which is modified if perforation is central or marginal.

Studies on the subject, primarily focused on surgical results, have not demonstrated relevant differences between a central and a marginal perforation. In addition to very heterogeneous and discordant results, no publication has yet been able to indicate a change in conduct according to the type of perforation. We can find publications comparing techniques and associating success rates to patient age, tympanoplasty graft material, graft position, use of preoperative medications, endoscopic or microscopic approach, as well as smoking, otorrhea, state of the mucosa, ossicular chain, and contralateral ear (CLE), among others. Studies looking for differences in the perforation closure rate according to the current classification—central or marginal—did not find significant differences.

Added to this is the fact that the current classification does not have any prognostic value, especially in the medium and long term, and, inevitably, we conclude that the classification used so far is quite deficient. At this point, it is worth remembering that the classification was developed many decades ago, even before the popularization of microscopes and, more recently, endoscopes in clinical practice.

Fig. 39.13 Traditional classification of tympanic perforations. **(a)** Central perforation, with 360° borders (black arrows). **(b)** Marginal perforation, with the absence of tympanic edges in part of the perforation (black arrow)



The view provided by the otoscope, without angulation, and with limited amplification, often blocks the view of essential points of the middle ear anatomy. The most evident and consensually perceived factor is difficulty visualizing the anterior tympanic borders in patients whose external acoustic meatus is tortuous. With the routine use of otoscopy, the perforation previously considered marginal can often be reclassified as central, since the borders, hidden by the tortuosity of the EAC, can now be visualized.

This technological evolution allowed many improvements while performing an otoscopy: angled vision, full HD amplification of the image, the possibility of proximity from the structures (often almost in direct contact with the tympanic membrane), and, finally, the recording of the otoscopy. This way, allowing the successive and exhaustive reanalysis of the otoscopies, new perspectives have emerged for assessing COM.

Today, we evaluate parameters that we consider undoubtedly more relevant than the mere presence, or absence, of tympanic borders around the perforation. The anatomy of the EAC, the size and location of the TM perforation, the presence of tympanosclerosis, and the presence or absence of signs of tympanic retraction before perforation are the main parameters that we must consider in the evaluation of the disease.

Therefore, we currently prefer to classify perforations as **outside-in** (or retraction-perforation) or **inside-out** (without associated retraction or explosive), as we will see below.

Inside-Out Versus Outside-In: A Look Over the Pathogenesis

The proposed pathogenesis for tympanic perforations, classically, involves a series of trigger situations in which an acute event would lead to a chronic condition (perforation). Acute suppurative otitis media, traumatic perforations, and

external otitis (with partial necrosis of the tympanic membrane), among others, are the etiologies usually described for the genesis of chronic otitis.

For this reason, the transition between acute and chronic conditions is explained by predominantly “explosive” mechanisms, that is, some sudden inflammatory or traumatic event affecting a previously healthy structure. Disruption of intratympanic homeostasis, caused by an acute mechanism, will modulate a series of histological transformations in the mucous lining of the middle ear, which would change permanently, characterizing COM. We can call this model the “direct line” between the acute (trigger situations) and the chronic (tympanic perforation).

However, clinical practice shows us that we receive many more patients with tympanic perforations than we can explain through this model. In other words, it seems that the mechanisms traditionally related to the origin of these perforations (the causes) are identified in everyday life in a much smaller proportion than the number of perforated TM that reach us (the consequences). It is evident in clinical practice that the otologist observes several patients with tympanic perforations. However, we rarely witness an acute event (recurrent acute otitis media, external otitis, and trauma) that could justify the chronic condition. Faced with this paradox, one can conclude that other mechanisms must be responsible for generating an expressive number of perforations.

Furthermore, the adoption of this “express” or “direct line” acute–chronic pathogenesis model disregards a daily situation witnessed by otorhinolaryngologists. Historically, events such as tympanic retractions, atelectasis, and adhesive otitis media, among others, have been inexplicably placed on the sidelines of this discussion. It is as if, in theory, tympanic membrane retractions were a chapter of chronic otitis media, while perforations would be in another (entirely separate) chapter.

From what has been exposed in this chapter so far, considering the technological evolution, the accumulated knowl-

edge acquired over the years, and the successive reflections around the paradoxes exposed above, we pursue other ways of classifying tympanic perforations. Thus, based on the detailed analysis of otoscopy, we began to value findings suggestive of tympanic retractions **associated** with perforations. Thus, we sought to understand a second model of pathogenesis for tympanic perforations, which associates and shows a critical intersection between the knowledge referring to retractions and the tympanic perforations.

The observed retraction characteristics associated with perforation are (a) medialization of the malleus handle, (b) tympanic remnants over the promontory, (c) tympanic remnants over the ossicular chain, and (d) erosion of the ossicular chain. Figure 39.14 illustrates these findings.

When we find zero or one of the characteristics mentioned above, we consider the perforation not associated with retraction, that is, an “explosive” perforation. This group of perforations, whose proposed etiology is the express model (acute leading directly to chronic), we convention to call “inside-out,” that is, from the middle ear to out, in an imagined explosive process. Of course, the nomenclature is

intended to translate a process (acute condition leading to chronic) and not a specific etiology. Etiologies may be not only acute otitis media or recurrent acute otitis media but also external ear processes such as fungal or bacterial infections that lead to necrosis of part of the TM.

In other words, they are previously healthy middle ears, which possibly have suffered aggression at a specific moment, be it traumatic, inflammatory, or infectious, generating tympanic perforation as a permanent sequel. The absence of signs of the previous retraction would be the mark of an “explosive” process or, in other words, punctual in time, characterizing the opposite of the subsequently mentioned group.

In the second group, there are the perforations with two or more retraction characteristics associated with the perforation. It represents an alternative pathogenesis model in which a progressive retraction would lead to perforation. The pressure self-generated by the retracted TM against the structures of the middle ear (ossicles and promontory) induces ischemia of the capillaries of the membrane and, consequently, the necrosis of a part of it. It is reaching the apex of this

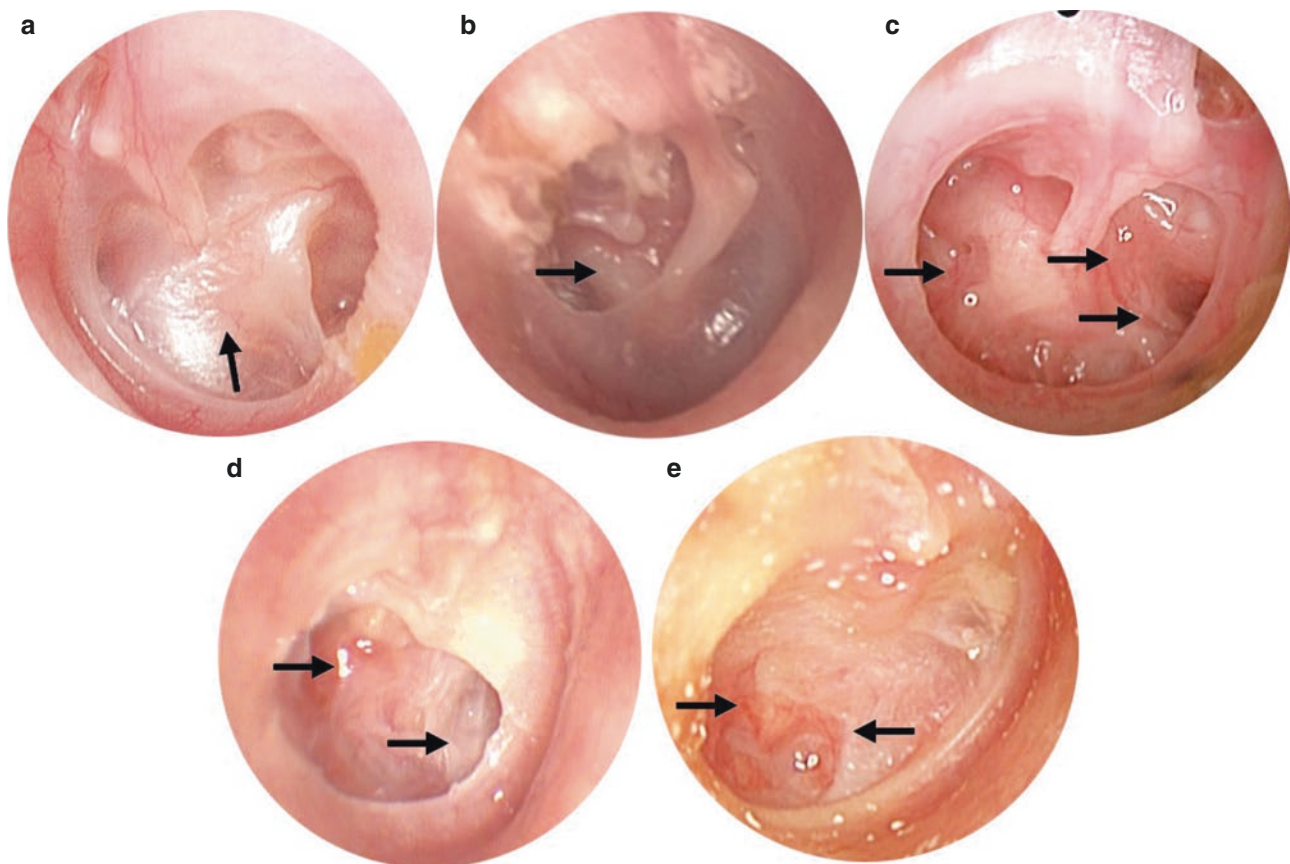


Fig. 39.14 Signs of retraction. (a) Bridge between the healthy TM and the one attached to the promontory. (b) More accentuated brightness over the promontory because the TM has adhered to it in the PS quadrant. (c) Brighter and smoother middle ear mucosa, suggesting atelecta-

sis with subsequent loss of the lateral epithelial layer of the TM. (d) Epithelium in the proliferation phase on the promontory. (e) Transition between MT adhered to the promontory with its epithelialized lateral layer and, more posteriorly, with a mucosal appearance

process that the retraction will turn into a perforation. Of course, a different type of perforation than mentioned in the previous paragraph. The illustrated signs (of the previous retraction) would be the marks left by this long process of progressive retraction, which we conventionally call outside-in. Thus, the pathogenesis proposed for this group of perforations is very similar to that of tympanic retractions, involving a negative pressure mechanism (probably from auditory tube dysfunction) and an entire altered middle ear homeostasis, which would be responsible for the progressive retraction until perforation.

In a study by Selaimen and collaborators (2020), the analysis of 1003 ears divided according to the above criteria into inside-out and outside-in showed very different parameters for each group. We will now present the main results, through which we will be able to understand the reasons that lead us to consider the proposed classification more representative of the pathogenesis.

Initially, observing the functional criterion, not only the tritonal mean thresholds on airway conduction (AC), but also the bone conduction (BC), and air–bone gap (ABG) were all higher in the outside-in group, with statistical significance, demonstrating that functionally they are the most affected ears. Also, the duration of symptoms was longer in the outside-in group, which included patients with a higher mean age.

When analyzing the contralateral ear (CLE), the inside-out group showed a significant majority of normal CLE. On the other hand, the outside-in group had a much higher rate of altered CLE. This difference occurred especially at this group's expense of more retractions and cholesteatomas. CLE has been studied for decades, with ample evidence that it is an excellent predictor of main ear behavior. Thus, understanding COM as a bilateral disease, such findings corroborate the theory that, while the inside-out group represents the

previously healthy ears, the outside-in represents chronically dysfunctional ones.

Finally, analyzing the differences between the groups in size and location of the perforation, the inside-out group had smaller and more anterior perforations. In comparison, the outside-in group included larger and more posterior perforations. Still, marginal perforations are found almost exclusively in the outside-in group, in agreement with a study by Rosito et al., which indicated that these perforations almost always result from the previous retraction.

In summary, based on these otoscopic signs of the previous retraction in tympanic perforation—or their absence—we believe we can differentiate a previously healthy ear from a chronically dysfunctional one. Furthermore, this classification of tympanic perforations seems to make more sense since it brings inferences about the etiopathogenesis of the disease. Table 39.1 summarizes the main differences between the types of perforations. Figure 39.15 illustrates three inside-out perforations, while Fig. 39.16 exemplifies three outside-in perforations.

Obviously, this distinction will lead to changes in treatment proposals, especially in surgical aspects. On the one

Table 39.1 Summary of characteristics according to the new classification of tympanic perforations

	Inside-out	Outside-in
Size	Smaller	Bigger
Location	More likely anterior quadrants	More likely posterior quadrants
Hearing (AC and BC)	Higher thresholds	Lower thresholds
Hearing (ABG)	Smaller gap	Bigger gap
Tympanic borders	Central (almost always)	About half marginal
Contralateral ear (CLE)	More normal ears	More abnormal ears

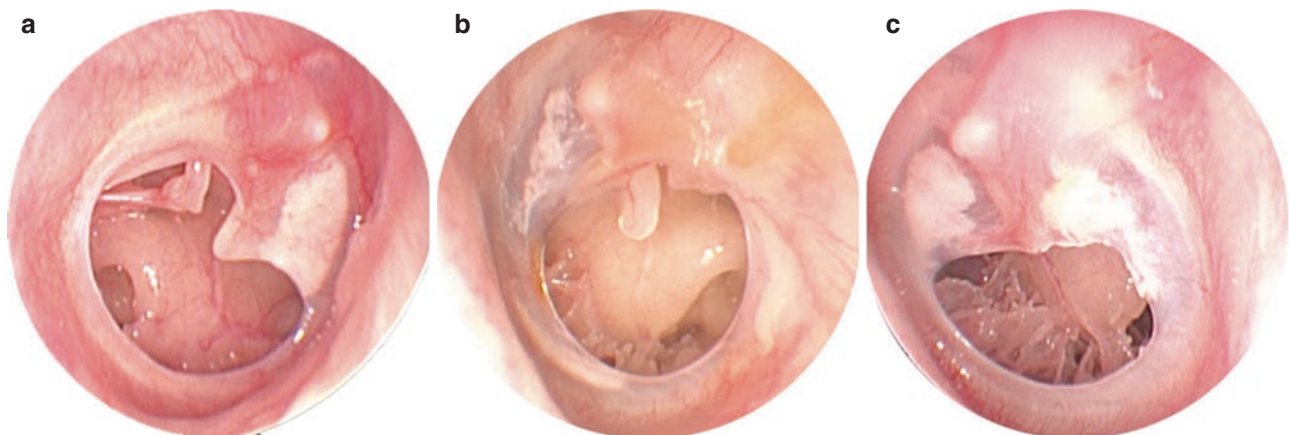


Fig. 39.15 Three examples of **inside-out**, or explosive, perforations (with no associated retraction). We did not see any of the described signs of retraction. (a) Perforation in quadrants AI, PI, and PS, with intact ossicular chain and without medialization of the malleus or tym-

panic membrane over the ossicular chain or promontory. (b) Perforation in all quadrants, sparing part of the AS, also with no signs of retraction. (c) Perforation in the AI and PI quadrants, without malleus medialization or other signs of the previous retraction

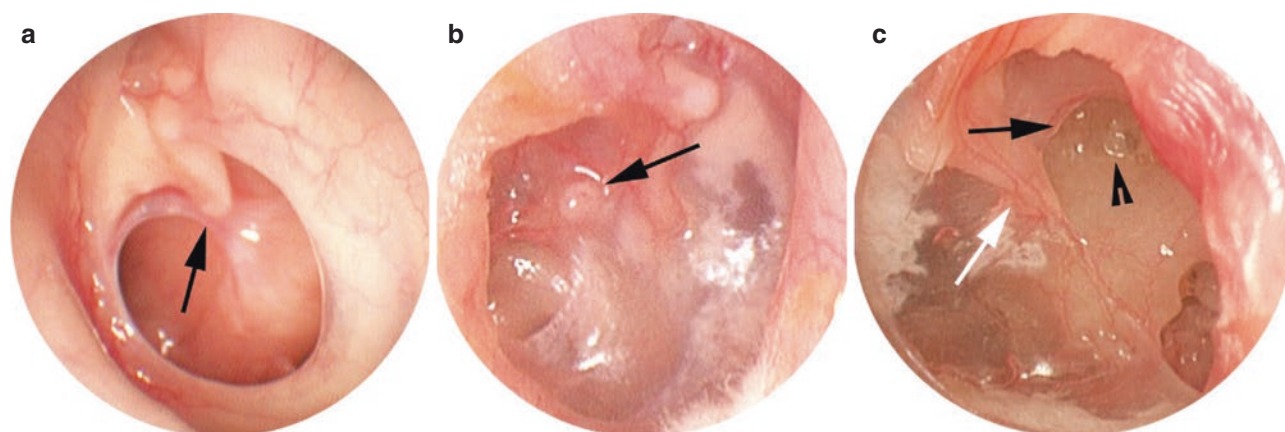


Fig. 39.16 Three examples of outside-in perforation, or perforation-retractions. (a) Medialization of the malleus manubrium, including touching the promontory with a TM bridge to the promontory (black arrow). (b) Erosion of the long incus crus with tympanostapedopexy (black arrow) and clear medialization of the TM in the posterior quad-

rants. (c) Medialization of the malleus manubrium (white arrow), tympanic membrane bridge between the malleus and the facial nerve (black arrow), and ossicular chain erosion—only the stapes footplate is present, without superstructure (black arrowhead)

hand, the inside-out perforations represent normal middle ears in terms of aeration and physiology, but which suffered an “accident on the way,” resulting in a tympanic perforation. On the other hand, outside-in perforations translate the end of a chronic process into a middle ear with altered physiology; they represent a chronic otitis media in its essence. Indeed, the processes to restore normality will need to be much more detailed in the second group of tympanic perforations.

Surgical Treatment

Principles of Treatment Applied to the Pathogenesis

The aim and physiological principle of tympanoplasties are to restore sound protection to the round window by obtaining an air-filled cavity, closed but ventilated, and by restoring the amplifier mechanism between the tympanic membrane and the footplate of the stapes. In other words, initially, obtaining a healthy, aerated, mucosa-lined middle ear is essential as an indispensable condition for subsequently proceeding with the ossicular restoration.

Regardless of the technique used, the main objectives of surgery for COM are as follows:

1. Removal of all diseased tissue from the middle ear, with complete elimination of inflammatory and infectious processes and the consequent obtaining of a dry and safe middle ear.
2. Preservation or reconstruction of the middle/outer ear complex.
3. Restoration of the mechanisms that conduct sound.

These objectives can be achieved in a single step or staged over two procedures. In the latter situation, the first surgery is performed to control the infection and recover tympanic integrity. The so-called “second stage” aims to reestablish the connection between the tympanic membrane and the endocochlear fluids, that is, the reconstruction of the ossicular chain.

The effort to differentiate the etiopathogenesis of each type of perforation has as its main objective the individualization of the treatment applied to each case. If the previous classification fails to present any practical applicability in the treatments, be it central or marginal perforation, the new parameters aim to improve the planning and execution of the treatment. The look at the past (etiology) helps us to understand the present (type of tympanic perforation) in order, fundamentally, to be able to plan the future (treatment and prognosis).

The handling of inside-out perforation seems to be much simpler. Since the middle ear was healthy before tympanic perforation, restoring normality can be a less complex process. The so-called “simple” tympanoplasty, with the interposition of fascia only, may be sufficient in most perforations from this group. **Later in this book, there’s a specific chapter for the surgical technique step-by-step.**

On the other hand, outside-in perforation, which represents the physiology of a previously dysfunctional middle ear, will certainly require more surgical steps to restore normality. If the negative pressure in the middle ear persists, it may lead to the recurrence of neotympanum retraction, with or without recidivism of the perforation. Therefore, additional steps may be necessary, which is the reason why we agreed to call this a “complex” tympanoplasty. **We suggest reading the specific chapter later in this book,** but in summary, outside-in perforations may require the following associated procedures:

- The use of more resistant grafts, such as cartilage associated with the temporal fascia (double graft), thus preventing future retraction.
- The insertion of a concomitant ventilation tube, thus ensuring adequate ventilation in the immediate and mid-term postoperative periods.
- Lateralization of the malleus (most likely including sectioning the tendon of the tensor tympani), thus allowing air circulation from the eustachian tube throughout the entire middle ear.
- Cleaning of epithelium adhered to the promontory (and often occupying posterior recesses), thus preventing the formation of a cholesteatoma in the future.
- The use of angled optics to inspect posterior recesses, hypotympanum, and supratubal recess.
- Ossicular chain reconstruction since ossicular erosion is more frequent in this group of perforations.
- Procedures aimed at improving eustachian tube function.

Preoperative Preparation

Almost all patients with tympanic perforation will, at some point, have an active infection with purulent discharge from the ear. As we mentioned earlier in the evaluation, the infection must also be controlled in the preoperative preparation to maximize the chances of success with graft vitality.

Counseling

We doctors are sometimes taken by an unjustified arrogance, and we judge the destiny of our patients without, many times, offering them a more comprehensive explanation of what we intend to accomplish. This conduct, which would be deplorable itself, constitutes an additional risk for the doctor, who faces a growing wave of medico-legal problems.

As soon as we indicate the surgery, it is our routine to meet with the patient and his family in a frank conversation and accessible language to make them aware of the characteristics of the disease, its natural history, and possible consequences. We take advantage of this moment to present our surgical vision plan, its possible complications, and a preview of the postoperative evolution and care. After explaining to the patient, we digitally sent the preoperative and postoperative instructions (including the care that will be taken in the 30 days after the surgery), as well as the informed consent form.

Thus, wholly informed about the nature of his problem, the patient can weigh the pros and cons of the proposed therapy and maturely decide on his own destiny. Also, after this consultation in which the procedure was indicated, a preoperative consultation will be carried out, in which the patient and their family members will be able to bring new questions

from the initial conversation and the reading of the terms sent to them.

Hospital Admission

The patient is admitted to the hospital on the day of the procedure. Routinely, we keep the patient hospitalized for one day after the procedure, especially those who come from other cities or states.

Surgical Center

In addition to the technical aspects (not discussed in this section), a series of small details must be observed in the operating room, and we will explain them below.

Patient: The patient must be positioned with the head at the foot of the table and tied firmly to it, as the surgeon may often request lateral decubitus for better visualization of structures in each surgical procedure. The patient's head should be brought as close as possible to the surgeon, taking care not to project the shoulder superiorly in such a way as to impede its movements. The head must be positioned in extension, resting on a roller that keeps it stable.

The operating table should be positioned with the patient's head slightly above the plane of the feet (anti-Trendelenburg position), as this measure may help to reduce bleeding in the operative field. The surgeon's chair should go under the surgical table, at the height of the patient's head, taking care that the base of the table does not impede the approximation of the chair and, consequently, cause discomfort to the surgeon (and a big backache!).

The surgeon must feel comfortable and at ease in the operating room. Therefore, the surgeon must maintain an upright posture throughout the surgery, sitting on an adjustable stool with support in the lumbar region and bringing the operating microscope closely.

Surgical instruments: The surgeon must be familiar with the instruments that will be used. The material used in otologic surgery is extremely delicate, especially compared to other surgical instruments. Thus, in the vast majority of surgical centers in general hospitals, breaking delicate materials (hooks, Rosens, needles) is, unfortunately, a routine.

Our group agreed to standardize instrument boxes for all otological surgeries. Thus, we could separate the cruder materials (Weitlaner, scalpel) from the delicate material (Rosens, needles, hooks), the latter being placed in custom-made boxes. Such boxes allow the material to be safely sterilized and not break during transportation and the sterilization process itself.

Often, for reasons beyond our control, we are forced to perform delicate surgeries outside our "natural habitat" and with a paramedical team that is also unknown to us. In these situations, arriving at the operating room well in advance is prudent, thus enabling a review of the instruments available and a complete check of the microscope, motors, pens, and drills.

Preparing the Surgical Field

It will be discussed in the surgical technique chapter, step-by-step.

Traditional Techniques and Access Approaches

Currently employed tympanoplasty techniques involve repairing the tympanic membrane defect by placing a graft over or under the edges of the perforation. The graft acts as a bridge, allowing regeneration of the squamous epithelium of the external ear, on its lateral side, and of the middle ear mucosa, on its medial side.

The two surgical techniques traditionally used are the underlay, which positions the graft below the tympanic remnant (medial technique, by Austin), and the overlay, which positions the graft above the remnant (lateral technique, by Sheehy). Furthermore, there is the possibility of performing the surgery with the inlay graft, that is, at the same level as the tympanic membrane. Trained otologists get nearly identical results. In our group, we have preferably used the Austin technique, reserving Sheehy's for exceptional situations.

Regarding the approaches, we can describe four techniques:

- Transcanal: surgeon works in the microscope with visualization through the ear specula, which can be fixed to the table with the holder, or by the surgeon's nondominant hand.
- Endaural: through the incisions of Lempert I, II, and III and positioning of the self-static retractors.
- Retroauricular: incision in the retroauricular sulcus and anterior bending of the pinna.
- Endoscopic: more modernly, through the use of endoscopes, transcanal tympanoplasty expanded its possibilities of use with this tool.

In our group, we prefer the retroauricular approach, as it provides wider exposure and, therefore, the greater possibility of handling possible trans-operative unforeseen findings. Below, we list the necessary criteria for the use of the transcanal approach:

- First surgery
- Favorable EAC
- COM without cholesteatoma
- Tympanic perforation in the posterior quadrants
- Air–bone gap less than 25 dB
- All perforation borders are visible preoperatively without the use of the endoscope.

When the patient does not meet all these criteria, which happens in most patients who come to our offices, the retroauricular access route will be preferred.

Postoperative Care

At the end of the skin suture, we insert gauze in the EAC and place a compressive dressing to avoid excessive edema in the external acoustic meatus and the retroauricular surgical wound.

Finally, the procedure must be described still in the operating room before the patient leaves. Then, the prescription and dressings must be performed by the surgeon, who must accompany the patient to the recovery room and remain there until making sure that the patient recovers well from the anesthesia and the functions of the seventh cranial nerve are maintained.

The surgeon's responsibility is to personally assess the patient and prescribe his medication at the bedside the morning after the procedure. At this moment, there is the opportunity to explain the surgical procedure details to the patient, who is already awake. The compressive dressing can be removed the day after surgery, and the patient is usually discharged. Instructing the patient to avoid wetting the operated ear until the healing process is complete is essential. He is also instructed that when he sneezes or coughs, he should do so with his mouth open and avoid any effort that increases pressure in the ear (Valsalva type). A common reaction after ear surgeries is the perception of clicking and the discreet drainage of a clear secretion. It must be informed to the patient as typical from the postoperative. In one week, the patient should be reevaluated. Subsequently, visits will be scheduled according to the postoperative evolution.

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