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INVITED REVIEW

Insights in the Physiology of the Human Mastoid: Message to the Surgeon

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Abstract

To reach the temporal bone, surgeons often consider making access through the mastoid. It is, however, imperative that the surgeon is aware of the mastoid's morphology, physiology, its functional parameters and the implications of damaging or altering this structure.

We review established findings on the developmental and functional morphology, and the delicate pressure balance variations in the middle ear cleft. Then, an overview is given of the possible surgical access methods for the temporal bone through the mastoid. We look for implications of each procedure, which can only be understood through the knowledge of the preceding outline, so that surgeons can justify a certain surgical approach.

Introduction

Ear surgeons should be aware of the embryological, anatomical and not to forget physiological aspects of the mastoid cavity when considering this structure as a surgical approach to the temporal bone. Different anatomical ways of access have been described that allow reaching the targeted structures (cf. section 5), trying to avoid fundamental constitutive elements susceptible to injury. In our opinion, an initial better understanding of the developmental and functional aspects of the mastoid gas cells system is essential for the surgeon to justify a certain surgical approach.

The combined experience of the authors' numerous years as practicing otosurgeon and/or academic researcher in the field (cf. reference list) together with many personal communication with colleagues, led us to selection of key insights on the developmental and functional aspects of the middle ear cleft. The middle ear cleft includes the tympanic cavity and the mastoid gas cell system, cf. Figure 1.

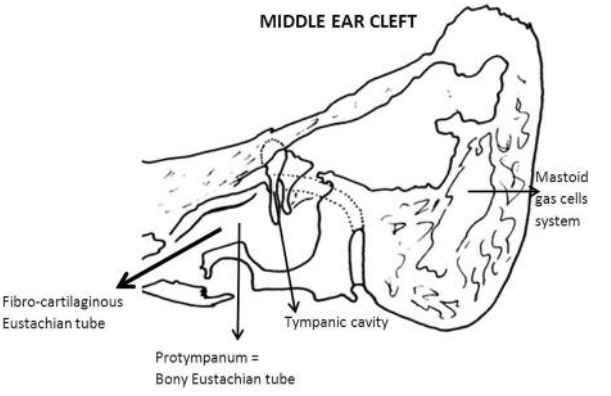

Figure 1. Illustration of a section through the temporal bone: Fibro-cartilaginous Eustachian tube and middle ear cleft: Tympanic cavity with ossicles and mastoid gas cells system.

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Our synthesis on the mastoid (or rather the entire middle ear cleft) is presented in sections 2-4, where we discuss three main questions:

(1) Does the developmental morphology of the middle ear cleft condition the physiology of the mastoid?

(2) Does functional morphology of the middle ear cleft influence the physiology of the mastoid?

(3) How does the balance of pressure variations in the pneumatic spaces of the temporal bone constitutes a basic requirement for healthy functioning?

Most of the information presented here reflects already established knowledge, which surgeons are already or should be aware of.

After addressing these topics on the mastoid morphology and physiology, an overview of the operation procedures using the mastoid as surgical approach for the tympanic cavity is given in section 5 with their implication, risks and/or merits, which is of interest and controversy to practicing ear surgeons since the 1950's [1,2].

With this review, we hope to fill the missing gaps in the surgeon's knowledge of the developmental and functional aspects of the mastoid, and hope to educate and help them in making a justified choice of surgical method.

Middle Ear Cleft Developmental Morphology

Because the mastoid gas cells system is an integral part of the middle ear cleft, cf. Figure 1, its development directly depends on the development of the whole middle ear cleft [3-6].

The tympanic cavity is endodermal. It originates at about four weeks of fetal life from the first pharyngeal pouch, which grows laterally and expands rapidly to pre-form two fundamental structures: the distal part forms the tubotympanic recess, which will become the primitive tympanic cavity, and the proximal part constricts to form the fibrocartilaginous Eustachian tube [7].

During the first twelve weeks of fetal life, the cartilaginous cells, precursors of the ossicles, are embedded in loose mesenchyme, which limits the expansion of the future tympanic cavity. This is invaded by the epithelium of the future fibrocartilaginous Eustachian tube. It divides into four sacci [8], which expand in four different directions, lining the tympanic cavity with epithelium and enveloping the ossicles:

- The saccus anticus extending in a cranio-frontal direction will form the anterior pouch of von Trôltsch;

- The saccus medius forms the attic, extending upward and usually breaking into three smaller sacculi;

- The saccus superior extending posteriorly and laterally between the malleus and the long crus of the incus, forming the posterior pouch of von Trôltsch;

- The saccus posterior extending along the hypotympanum, to form the round window niche, sinus tympani and oval window niche.

The antrum, a lateral extension of the epitympanum, starts to form at about twenty-two weeks. The mastoid gas cells develop as an outgrowth of the antrum. Epithelial buds from the tympanic cavity and antrum extend to adjacent areas of the temporal bone, after osteoclastic resorption of bone or differentiation of bone marrow into loose mesenchyme. Thus, mastoid buds from the antrum penetrate the temporal bone, giving rise to the mastoid gas cells. The degree of pneumatization of the mastoid varies greatly in normal temporal bones. The age at which the gas cells develop is subject to huge individual variation.

Expansion of the tympanic cavity is virtually complete by about thirty-three weeks of fetal life. The epitympanum follows approximately four weeks later. Because of the fusion of the periosteal layers of the otic capsule and the tympanic process of the squamous bone, the mastoid process begins to develop at about twenty-nine weeks. Formation of the antrum starts when the epitympanum becomes angled posteriorly and is well developed by the thirty-fifth week. As early as this, cavitation extends into the mastoid.

Pneumatization of the tympanic cavity or tympanum is followed by that of the associated gas spaces [9]. At 34 weeks, pneumatization of the mastoid may only just have started, and this progresses during infancy and childhood. As the mastoid grows, the antrum shrinks in size relatively, and assumes a more medial position. When the antrum is completely formed and in place, the ventro-lateral wall continues to grow until puberty or beyond, giving rise to the mastoid bone.

In contrast to the labyrinth and tympanic cavity, the postnatal growth of the mastoid can be seen in its length, width and depth [10,11]. It is governed by two principal forces: the first is external, caused by the traction of the muscles of the neck and mainly of the sterno-cleido-mastoidian muscle. The second is internal and consists in the progression of the buds of expansion of the tubo-tympanic epithelium following the resorption of the embryonic mesenchyme.

The postnatal development of the mastoid is neither uniform nor symmetrical, but it is obvious. The evolution of general morphology shows that after an accelerated growth during the first two years of the life, the development continues, relatively more progressive, in the child and the teenager. The internal configuration is even more prone to variations. Its development leads to the creation of a more or less high number of gas cells that vary greatly in size and which are grouped around a larger cell of constant anatomical localization, i.e. the antrum.

Number, size and volume of the mastoidian cells are individual characteristics. The external size of the mastoid never bodes the width of the contained gas cells system; so, very small mastoids can perfectly be well-pneumatized mastoids [8,9]. In fact, growth seems to be controlled by many intricate factors such as: heredity, environment, nutrition, gas exchanges and frequency of infections [12-14]. It is obvious that the infections during childhood, just like the other developmental incidents, carry a real inhibiting effect on the pneumatization. However, as soon as the end of the infection, or as soon as the lifting of the brake, pneumatization may start again. However, it is really quite difficult to specify if infections are the only ones able to act as a causal agent, stimulating the activity of the osteoblasts or, on the contrary, if this infection does not result from 'local morphological predisposing factors'.

Middle Ear Cleft Functional Morphology

The tympanic cavity is constricted in its superior third by the inter-attico-tympanic diaphragm, which is a bony membranous barrier perforated by two small permanent openings: the anterior tympanic isthmus, which is situated between the tensor tympani tendon and the stapes; and the posterior tympanic isthmus, which is between the double posterior ligament of the incus and the bony posterior tympanic wall.

This anatomic barrier divides the middle ear cleft into two separate compartments: an anterio-inferior one, principally devoted to the mucociliary clearance function, and a postero-superior one, more devoted to the gas exchange function. The barrier forms a diaphragm that is composed of two complementary types of structures: mucosal folds and bones with muscular and ligament structures: the head and neck of the malleus, the body and short process of the incus, the tensor tympani muscle, the anterior and lateral mallear, and the double posterior incudal ligaments. With this concept of partition, we are better able to understand the mechanisms involved in the pathogenesis of otitis media [6,15]. Clinical as well as surgical management will thereby be enhanced.

The antero-inferior compartment of the middle ear cleft, which is situated under the diaphragm, includes the pro, meso- and hypo-tympanum and is covered by secretory or non-secretory ciliated cells that enable mucociliary clearance. It consists of a less rigid chamber because of the presence of the eardrum. Because of the fibrocartilaginous Eustachian tube, it opens into an intermittently ventilated gas pocket. It communicates with the postero-superior compartment by both the anterior and posterior tympanic isthmi. It is often the site of secondary bacterial infections from the rhinopharynx. An inflammatory process involving the mucosa of the antero-inferior middle ear cleft compartment leads to dysfunction in mucociliary clearance and to the accumulation of mucus.

The postero-superior compartment of the middle ear cleft, which is situated above the diaphragm, includes the epi- and retrotympanum, aditus ad antrum, antrum, and mastoid gas cells system. It is covered by a richly vascularized cuboidal epithelium that is devoted primarily to gas exchange. It consists of a rigid chamber and an open non-ventilated gas pocket that communicates with the antero-inferior compartment via both openings. It may be the site of viral haematogenous infections. Inflammation of the mucosa of the postero-superior middle ear cleft compartment impairs the gas exchange, which in turn leads to the development of a 'gas deficit' in the middle

ear cleft, cf. section 3.2.2. The inter-attico-tympanic diaphragm conditions the topography of the tympanic membrane retraction pockets. When only one opening is blocked, the postero-superior quadrant of the pars tensa is attracted in the direction of the retrotympanum. When both openings are completely blocked, the pars flaccida is drawn in toward the epitympanum. In the same way, the diaphragm also influences the invasion of the middle ear cleft by a cholesteatoma [16-21].

Pressure Balance of the Temporal Bone Pneumatic Spaces

On the quantitative point of view, gas spaces of the middle ear cleft embody the most constitutive part of the pneumatic spaces of the temporal bone. The middle ear cleft consists of both the mastoid gas cells system and the tympanic cavity. The latter gathers the tympanum and four annexes: the epi- and hypo-tympanum, retro- and pro-tympanum that corresponds to the bony Eustachian tube. In fact, the middle ear cleft consists of a set of interconnected gas cells lined with the same respiratory mucosa. Because the gas exchange is actually performed through the mucosa of the cells, the total surface area of mucosa will influence the rate of gas exchange [22,23].

What is then the role of the mastoid? The physiology of the mastoid can only be understood in the process of balance of pressure variations in the middle ear cleft.

The middle ear cleft is essentially a non-collapsible poorly ventilated gas pocket through which sound wave energy is transported to reach the inner ear. Every amount of gas must be contained in a three-dimensional container or volume V. The pressure of a gas P is created by the molecules of gas striking the walls of the container. It is a force per unit of area. The physical characteristics of a gas are linked together by the ideal gas law: P.V = n.R.T, in which n is the mass expressed in molecule gram; R: the universal gas constant; T: the absolute temperature. The middle ear cleft is submitted to physiological variations in volume and pressure. It has the capacity to maintain a steady state in volume and pressure, which is achieved by different regulatory mechanisms, which neutralize or minimize the pressure and volume variations. This is performed by adjusting, either, the quantity of gas, its diffusion, and/or the volume of the middle ear cleft[24].

Gas exchanges

The gas content of the middle ear cleft is constituted and maintained thanks to gas exchanges between the middle ear cleft and the neighboring structures, including the environment across the tympanic membrane, the inner ear via the round window membrane, the blood compartment via the mucosa and the rhino-pharynx through the fibrocartilaginous Eustachian tube [25-32].

The gases that are present in the middle ear cleft are identical to those found in the blood and in the atmosphere: Oxygen (O_2), Carbon dioxide (CO_2), Nitrogen (N_2), Argon (Ar) and Water vapor (H_2O). Their relative amounts may be expressed by their partial pressure P(x). Nitrogen is neither produced nor consumed at the level of the middle ear cleft. The $P(O_2)$ in the middle ear cleft is slightly lower and the $P(CO_2)$ is slightly higher than in venous blood. This means that there exist a modest consumption of O_2 and production of CO_2 in the middle ear cleft. Oxygen and nitrogen are absorbed from the middle ear cleft via the mucosa into the blood compartment. Carbon dioxide and water vapor are diffused from the blood compartment, via the mucosa, into the middle ear cleft.

The steady exchange of gas through the mucosa depends on the functional properties of the cells of the mucosa, the specific diffusion rate of the gas, which is a constant value, characteristic for a particular gas, and the behavior of the vascular system. The primary purpose of the mucosa is to facilitate gas exchange between the middle ear cleft and the blood compartment through a constitutive tissular barrier.

To vibrate in an optimal manner, the tympano-ossicular system must remain in balance. This means that the intra-middle ear cleft pressure must be equivalent to the atmospheric pressure (760 mmHg). In ambient air, this equivalent value is obtained by calculating the sum of the partial pressures of the four main constitutive gases in the air: oxygen (158 mmHg), carbon dioxide (0.3 mmHg), nitrogen (596 mmHg), and water vapor (5.7 mmHg) [33].

However, in the middle ear cleft, the composition of gas varies for two main reasons. The middle ear cleft is a closed cavity that is connected with the nasal

cavities by the fibrocartilaginous Eustachian tube. The gas in the rhinopharynx, which enters the middle ear cleft via the tube, consists of exhaled gas that contains less oxygen and more carbon dioxide than ambient air. This gas composition also varies because gas diffusion occurs between the middle ear cleft and the arterial and venous blood, via the mucosa. In blood, the partial pressure of these gases differs. There is more oxygen in the arterial blood (93 mmHg) and more carbon dioxide in the venous blood (44 mmHg) because the gas exchange occurs at the level of the pulmonary air cells and the tissues throughout the body, respectively.

Thus, the composition of gas in the middle ear cleft differs from that in ambient air. The gradient 'out' from middle ear cleft to the capillaries is 57 mmHg for oxygen, and the gradient 'in' from the capillaries to the middle ear cleft is 39 mmHg for carbon dioxide. This should result in a negative pressure in the middle ear cleft. However, in spite of the difference in gas composition, the pressure in the middle ear cleft approximates the atmospheric pressure to enable optimal sound transmission.

The reason for this balance is threefold:

- Because the sum of the partial pressures of oxygen and carbon dioxide is lower in the middle ear cleft (90 mmHg) than in ambient air (150 mmHg), nitrogen exerts the higher partial pressure in the middle ear cleft (623 mm) because of its very slow diffusion towards the capillaries.

- The composition of the exhaled gas that enters the middle ear cleft via the tube contains less oxygen and more carbon dioxide than ambient air does. This reduces the passive diffusion of these gases through the mucosa.

- The blood flow through the middle ear cleft mucosa is probably low. This limits the importance of gas diffusion and enables the oxygen and carbon dioxide partial pressures to nearly equalize, because each has a high diffusion rate. The same mechanism applies to water vapor of which the diffusion rate is also very high. Transmucosal gas exchange results in gas absorption, cf. Figure 2.

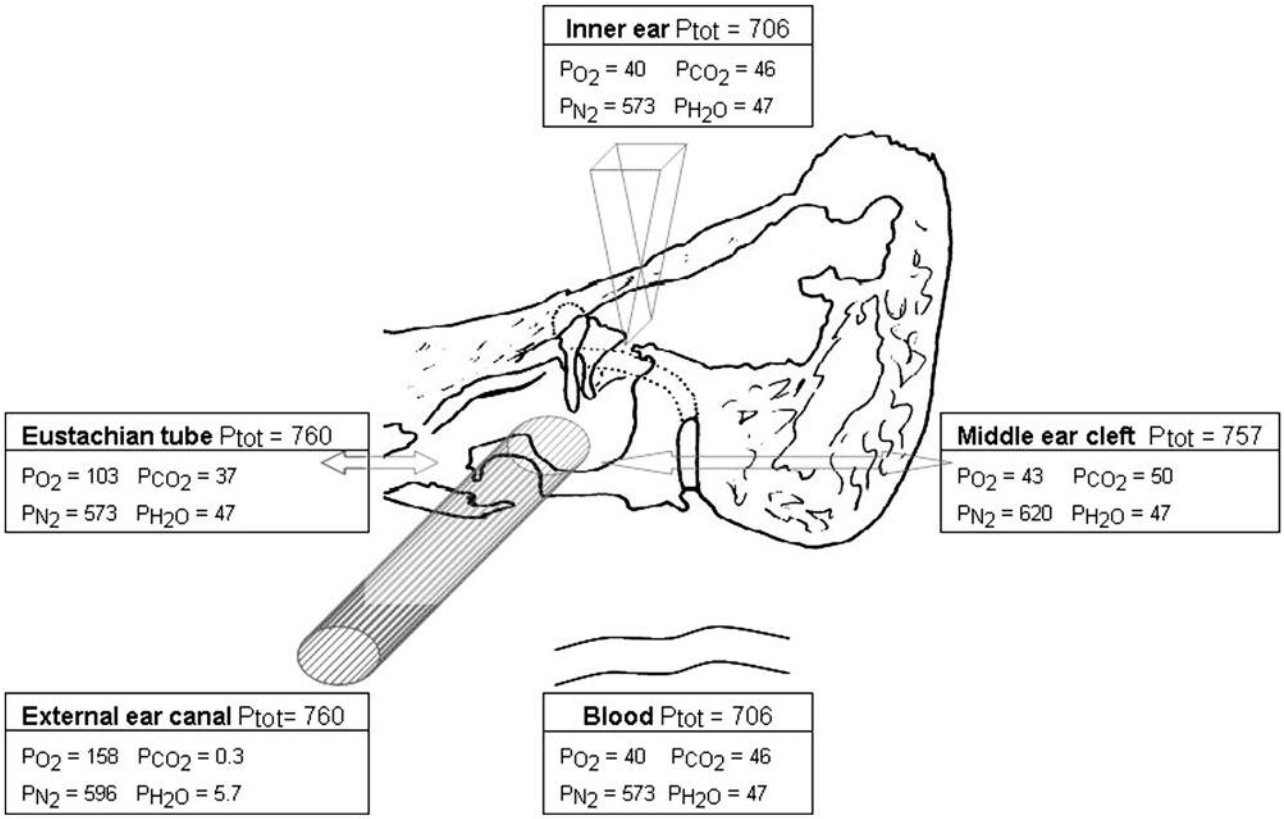

Figure 2. Partial pressures (mm Hg) of the physiological gases for the middle ear cleft and their neighboring structures. Values are taken Hergils et al [27] and from Felding et al [69].

Regulating systems

The steady state in volume and pressure into the middle ear cleft is preserved by the way of two types of regulating mechanisms: on the one hand, the in situ adaptation systems and, on the other hand, both central and peripheral nervous regulating system with retro-control between the middle ear cleft and the muscles of the fibrocartilaginous Eustachian tube.

The in-situ adaptation systems

The in situ adaptation systems consist mainly in the compliance of the tympanic membrane lamina propria, the fibrocartilaginous Eustachian tube function and the behavior of the vascular system of the middle ear cleft mucosa, especially that of the mastoid.

The compliance of the tympanic membrane lamina propria

The compliance of the tympanic membrane lamina propria is used in the case of sudden pressure changes (altitude, flight, diving, explosion, etc.) This occurs largely due to the viscoelastic properties of the lamina propria and to the flexibility of the incudo-mallear joint, which acts as a static pressure receptor for the tympanic membrane, ensuring three-dimensional movement into the malleus. Deformation of the eardrum causes a volume change of the semi-rigid middle ear cleft and can therefore compensate partially for fast pressure changes. The eardrum is an active pressure buffer, but also a passive pressure victim [34-38].

The fibrocartilaginous Eustachian tube function

The fibrocartilaginous Eustachian tube is closed most of the time, and it therefore certainly does not function as a ventilation hole, which keeps pressure inside the middle ear cleft equal to the outside. Only at large pressure differences, over 2kPa, the fibrocartilaginous Eustachian tube will open spontaneously.

Changes in position seem to affect the function of the fibrocartilaginous Eustachian tube. The mean volume of gas passing through the tube in upright position is reduced by one third when the body is elevated at 20 degrees to horizontal position. It is reduced by two thirds in horizontal position. This is the result of the increase in the venous tissular pressure around the fibrocartilaginous Eustachian tube [39].

The vascular system in combination with the mastoid gas cell system

The behavior of the vascular system plays an important regulatory role in the physiological balance of pressure variations in the middle ear cleft. Variations in the middle ear cleft blood flow, associated with variations in the permeability of the vessels, allow ample adaptation to normal gas pressure fluctuations [40,41]. The normal extensive variations observed in middle ear cleft pressure over a 24-hours period, which appear regularly, are related to the vascular adaptations required by body position and sleep [42]. The mastoid gas cell system constitutes the most important volume of the middle ear cleft and therefore represents the major part of the middle ear cleft mucosal area available for vascular gas exchange[43].

The amount of gas present in the mastoid is important in regulating middle ear cleft pressure for two reasons. First, the physical, anatomical, properties of mastoid volume affect compliance: the greater the volume, the more compliant the system. Second reason: the surface area affects mucosal gas exchanges. Both are important in the regulation of middle ear cleft pressure. The gas contained in the mastoid gas cells system acts as a physiological passive pressure buffer.

The size of the mastoid gas cells system varies between individuals to a great extent. However, whatever the cause, be it genetic determination of growth or environmental factors, there is unanimous agreement that the small mastoid gas cell system has a pathological association. The smaller the system, the faster are the deviation from normal pressures. However, nature is a matter of balance! It is not a question of determining the volume of the mastoid in order to predict its clinical potential. In normal conditions and provided with healthy mucosa, a small mastoid, in an adult, is normal in so far as it constitutes the complete outcome of normal and standard development, i.e. the individual simply possesses the morphological particularity of being ‘small’. A small

mastoid in an adult is normal, in that it is sufficient for the physiological balance of pressure variations in middle ear cleft. This small mastoid is no more likely to develop pathology than another is. However, when the mucosa undergoes inflammatory process, a small mastoid in an adult, e.g. as outcome of disturbed or failed development, is certainly at a disadvantage when it is exposed to excessive variations in pressure. This mastoid is 'too small' compared to the rest of the middle ear cleft. Fluctuations in middle ear cleft pressure in the presence of a small mastoid gas cells system will result in greater forces applied to the tympanic membrane, when compared with the same pressure changes in a larger mastoid gas cells system. If the expansion reservoir is too small, it cannot play its role adequately.

In normal conditions and healthy mucosa, a slight negative pressure is present in the middle ear cleft compared to the outside air, created by exchanges of gas between the cleft and the blood compartment. Gas exchanges through the mucosa of the middle ear cleft are induced by the gradient of partial pressure of gases between the cleft and the capillaries of the sub-mucosal connective tissue. Given the capacity of gas diffusion in the middle ear cleft, partial pressures of those gases aim at a balance on both sides of the mucosa barrier of the middle ear cleft. The progressive diffusion of the gases is capable of modifying the gas composition in the middle ear cleft as well as in the circulating blood. The gas, which penetrates into the middle ear cleft from the rhinopharynx, is not outside air. Its composition is rather close to exhaled air. Quantitatively, the main gas that enters the middle ear cleft is of course nitrogen. Nitrogen diffuses much more slowly in the blood than the other gases (35 times more slowly than CO_2 and 1.8 times less than the O_2) [44], cf. Figure 3.

Considering this nitrogen in steady state, there is a difference in partial pressure between the middle ear cleft and the blood flow around the middle ear cleft. This steady state corresponds more or less to the total amount of pressure difference (54 to 56 mmHg) between middle ear cleft and blood. In steady state, oxygen, carbon dioxide and water vapor have nearly the same partial pressures in the middle ear cleft as well as in the blood compartment. Consequently, an

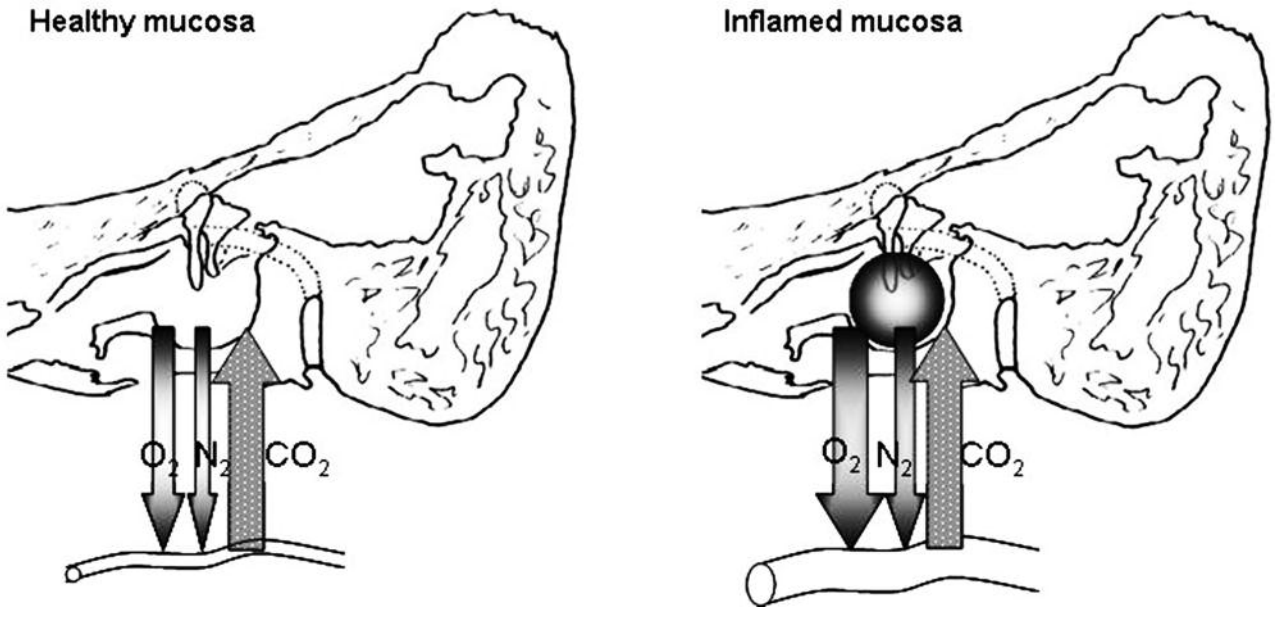

Figure 3. Gas exchanges between the middle ear cleft and the blood compartment through healthy (a) and inflamed mucosa (b).

increase in blood flow into the middle ear cleft will be of little effect on exchanges of these gases, except perhaps during the night, when carbon dioxide increases and oxygen becomes reduced in blood flow. On the other hand, nitrogen maintains always a higher partial pressure into the middle ear cleft as compared with the blood. Therefore, there is a clear and continuous elimination of nitrogen from the middle ear cleft in direction of the blood compartment. This leads to the negative pressure bias in the middle ear cleft. In normal conditions, this elimination is regularly compensated by the gas contribution originating from the rhinopharynx. A variation in blood flow could consequently explain variations in nitrogen absorption.

Inflammatory processes increase both the number and diameter of the blood vessels, thus increasing vascularization of the mucosa. In the middle ear cleft, the ratio ventilation / perfusion is respectively 4.75x10-3 and 3.98x10-3 in normal and inflammatory conditions. This means that the middle ear cleft is 210 times more perfused than ventilated under normal conditions; and 251 times more perfused than ventilated under inflammatory conditions. The result of this increased blood perfusion is a higher diffusion of nitrogen from the middle ear cleft in direction of the blood. This explains the apparent contradiction between the increase in thickness and barrier of the mucosa and the increase of the gas elimination in inflammatory conditions [45]. The normally slightly balanced middle ear cleft negative pressure thus increases. The higher nitrogen absorption would normally be sufficiently compensated through the fibrocartilaginous Eustachian tube, but could now generate pathological negative pressures into the middle ear cleft compared with the atmospheric pressure. This condition carries a "gas deficit" which could constitute a mutual physio-pathologic support to the development of chronic otitis media.

The central and peripheral nervous regulating system

In the middle ear cleft, the steady state in volume and pressure close to the atmospheric pressure is maintained not only by the way of the in-situ adaptation systems, but also by the way of a peripheral and central nervous regulating system with retro-control between the middle ear cleft and the muscles of the fibrocartilaginous Eustachian tube.

The mucosa of the middle ear cleft manages the quality of the gas contained into the middle ear cleft, especially its composition, mainly by means of chemo-sensitive sensors that act on the neuro-capillary system.

The fibrocartilaginous Eustachian tube manages the quantity (volume) of the gas contained into the middle ear cleft by means of baro-sensitive sensors that effect on the neuro-muscular system.

Sensors located in the middle ear cleft

Chemo-sensors

Within the middle ear cleft, the innervation of the mucosa originates from the autonomic nervous system, which gives and receives information's at the same time from sympathic and parasympathic networks [46,47].

Adrenergic nerve endings have been displayed; most of them being in the vicinity of vessels. They probably control their tonus. The peptidergic system non-adrenergic and non-cholinergic would also play a role. The presence of peptidic mediators such as P substance, vasoactive intestinal peptide (VIP) or calcitonin-gene related peptide (CGRP) has been demonstrated. It has been established that these mediators give rise to vasodilatation as well as an increase in the vascular permeability.

At the level of the middle ear cleft, chemo-sensors situated within the glomic tissue of the mucosa give answers to variations in the gas composition and give impulses to reflexes of gas transfer from the rhino-pharynx in the direction of the middle ear cleft.

Baro-sensors

The presence of baro-sensors has been reported at the level of the middle ear cleft as well as at the level of the tympanic membrane.

At the level of the tympanic membrane, stretching sensitive sensors have been described. They are corpuscles situated into the lamina propria. Their

sensitivity should fit the tympanic membrane movements. Moreover, they might play a role in the detection of pressure variations in the middle ear cleft[48-52].

The tensor tympani muscle does not appear to be involved in active dilatation of the fibrocartilaginous Eustachian tube. However, there may be stretch sensors in the tympanic membrane that could be related to modulation of the middle ear cleft pressure through the tensor tympani muscle, thereby affecting the tensor veli palatine muscle opening of the fibrocartilaginous Eustachian tube [53].

Sensors located at the pharyngeal orifice of the fibrocartilaginous Eustachian tube

Opening of the fibrocartilaginous Eustachian tube occurs when pressure imbalance takes place on both sides of the tympanic membrane [54].

On the one hand, baro-sensors are situated in the vicinity of the pharyngeal orifice of the fibrocartilaginous Eustachian tube [55], and on the other hand, within the middle ear cleft and most likely in the tympanic membrane. In this way, they suggest a neural connection between the sensory sensors in the tympanic membrane and the tubal muscles.

The opening of the fibrocartilaginous Eustachian tube would not be limited to a movement associated with or consecutive to yawning or swallowing, but could be controlled by a system whose retro-control could be initiated in the tympanic membrane.

Neural connection between sensors

The presence of a feedback system acting from the middle ear cleft to the tubal muscles as well as afferent nerves from the sensors located in the middle ear cleft to the salivary nucleus of the brain stem were described. This nucleus receives also the efferent fibers of both the sensitive and sensorial sensibilities from the V and IX. This nucleus also gives efferent fibers towards the swallowing muscles as well as fibers, which regulate the vasomotor tonus of the arteriole and pre-capillary sphincters from the active tissular areas, mainly the mastoidian ones. Therefore, this is consistent with the description of a complex system with a regulation loop with central nervous control [56-59].

Discussion

We summarize the above in section 5.1. In the following section 5.2, we consider most mastoidectomy procedures. This means surgery to remove hollow, gas-filled mastoid cells, either because of infection (removal of pus, necrotic bone, cholesteatoma, granulations therein) or to reach the tympanic cavity or inner ear (for corrective surgery, prosthesis or implants).

The implications of each operation procedure mentioned can only be fully understood by the knowledge of the previously accounted sections.

How the system operates

The gas exchange that governs the balance of middle ear cleft pressure variations is performed mainly into the mucosa of the middle ear cleft and through the fibrocartilaginous Eustachian tube.

In the middle ear cleft, the steady state in volume and pressure is maintained near the atmospheric pressure, on the one hand, by the working of local mechanisms of adaptability and on the other hand, by both central and peripheral nervous systems of regulation with retro-control between the middle ear cleft and the muscles of the fibrocartilaginous Eustachian tube.

The middle ear cleft is a poorly ventilated, but a highly perfused organ. Gas exchanges depend on variations in middle ear cleft blood flow. In a similar fashion to alveoli in the lung, the direction of gas exchange is predicted on the differences in partial pressure of the component gases in the middle ear cleft and in the blood compartment.

When the mucosa is healthy, there is a balance of gas exchanges because oxygen and nitrogen are absorbed by the mucosa on the same rate at which carbon dioxide is expelled. For a given mucosal blood flow, nitrogen is the exchange rate limiting factor of the middle ear gas loss, because it diffuses slower than carbon dioxide and oxygen which equilibrate relatively quickly with venous blood.

Inflammatory processes increase blood flow. The more inflamed, and thus vascularized, the mucosa, the greater is the rate of gas absorption, cf. Figure 3.

In case of inflammatory process in the mastoid gas cell system, what is the most accurate surgical procedure to restore the physiology? Because of the unhealthy status of the mastoid, opening is essential to permit drainage of the inflammatory secretion, removal of inflammatory granulated mucosa and creation of an enlarged gas reservoir. It is important for a surgeon to be aware of the physiological function of the mastoid gas cells, and the consequences of an operation upon it.

How to operate the system

When surgical approaches to the middle ear cleft are compared and considered, the decision of course depends on the specific pathological condition (the site, the extent of disease, the presence or absence of mucosal inflammation and Eustachian-tube dysfunction) and the availability of surgical and healthcare accommodations. The operation must thus be tailored to the patient and situation [60]. An overview of the different surgical options with some insights in their merits, risks or consequences will now be listed.

Two main mastoidectomy categories exist – canal wall up and canal wall down – with many subcategories. Mastoidectomy and access to the tympanic cavity can be initiated either by retroauricular (surgical incision behind the ear) or endaural approach (through the external ear canal), and each approach can continue through the bone by transmeatal or transcortical route[61].

Canal wall up

This procedure, where the postero-superior bony partition between the external ear canal and mastoid is kept intact, is commonly referred to as canal wall up (CWU). Subclassifications for this procedure are simple mastoidectomy, complete mastoidectomy, closed (cavity) mastoidectomy, cortical mastoidectomy, combined approach tympanoplasty and classic intact canal wall mastoidectomy. The two main subcategories are discussed.

Simple mastoidectomy

Simple mastoidectomy involves the removal of the gas cells of the mastoid without disturbing the contents of the tympanic cavity. The operation is performed retroauricular or endaural, and the surgeon opens the mastoid bone and removes the inflamed gas cells, without opening of the antrum or the attic (epitympanum).

Complete mastoidectomy

Some authors describe complete mastoidectomy as simple mastoidectomy with a facial recess approach. Removal of all of the mastoid gas cells along the tegmen, sigmoid sinus, presigmoid dural plate and posterior wall of the external auditory canal is then necessary; however, the external auditory canal wall still remains up. In this case, opening of the aditus ad antrum allows access to the epitympanum, and the incus and malleus may be removed for greater access.

Implications

These CWU methods have less post-operative care. Furthermore, after a canal wall up procedure, the mastoid can retain some of its native cuboidal nitrogen-absorbing epithelium.

Recurrence or persistence of the pathology is known to be common [61]. In case of an inflammatory process, again both the number and the diameter of the blood vessels increases, thereby increasing the diffusion of gas. Middle ear cleft negative pressure increases and is no longer fully compensated, and can thus become pathological again [15,62].

Canal wall down

When the postero-superior bony partition between the external auditory canal and mastoid is not preserved, one speaks of canal wall down (CWD). Synonyms or subclassifications for this procedure are open (cavity) mastoidectomy, atticotomy, classical radical operation, Bondy's operation, atticoantrotomy and retrograde mastoidectomy. We distinguish two categories:

Radical mastoidectomy

The procedure that converts the entire mastoid cavity, the external ear canal and the tympanic cavity with the epitympanum into one open(-air) cavity, is called radical mastoidectomy. The malleus, incus and chorda tympani are removed for better access. The innermost

ossicle (the stapes) is left behind to later offset the hearing loss. The method may sometimes be called open-mastoido-epitympanectomy (OME) [63]. Tympanic membrane and ossicles are not reconstructed, thus exteriorizing the middle ear cleft. The pro-tympanum can be obliterated with soft tissue to reduce the risk of a chronic otorrhea.

Modified radical mastoidectomy

This approach can be described as a radical mastoidectomy maintaining the tympanic cavity. Either some ossicles are left in place; or ossicular reconstruction is used to repair the bones (ossiculoplasty). Furthermore, the original eardrum is preserved or rebuilt through tympanic membrane grafting (tympanoplasty), with possible relocation deeper into the middle ear, to separate the mucosal lined middle ear space from the mastoid cavity and ear canal. The method may sometimes be called open-mastoido-epitympanectomy with tympanoplasty (OMET) [63].

Implications

The canal wall down technique enables the easy and full removal of the nitrogen-absorbing mucosa of the mastoid, and thus offers the best chance of full elimination of residual disease. The new epithelial lining of the mastoid bowl is a stratified keratinizing epithelium. Gas diffusion is no long occurring.

With radical mastoidectomy, the eardrum and middle ear structures may be completely removed, thus causing loss of hearing, and patients require life-long otologic care of their open middle ear cleft. Recurrent infection of this middle ear is possible.

Nowadays, modified radical mastoidectomy is therefore preferred by most surgeons over radical mastoidectomy [63], as it creates a more closed, dry and self-cleansing middle ear.

Canal wall reconstruction

First a CWD is performed, giving increased intraoperative exposure allowing full removal of the nitrogen absorbing mastoid epithelium, before the actual reconstruction of the postero-superior canal wall. The method is thus sometimes referred to as open-closed mastoidectomy.

Canal wall reconstruction is often combined with total or partial obliteration of the mastoid, using either local flaps (muscle, periosteum, fascia) or free grafts (bone chips, cartilage, fat, hydroxyapatite, bone pâté) [1,64].

Implications

The mastoid obliteration techniques reduce the transmucosal gas absorption in the middle ear cleft. In addition, the tympanum is isolated from the attic and mastoid. This design prevents recurrent postoperative retraction pockets if the fibro-cartilaginous Eustachian tube does not function properly [65]. Thus, the recurrence rates of pathologies that lead to a 'gas deficit' in the middle ear cleft are reduced.

The main setback of this surgical technique is the suppression of the buffer effect of this great gaseous area.

Even if regular controls are performed by accurate computed tomography imaging (M.R.I.) of the obliterated mastoid cavity, long-term complications of such a technique, including intracranial cholesteatoma and/or abscess, may occur.

Thoughts on antro-attico-mastoidectomy

The surface area of the mastoid cavity is greatly decreased after most mastoidectomy procedures as the fine maze of gas cells is reduced or removed. Because the passive pressure buffer function is partially governed by the mucosal surface area, it may not be desirable to remove all gas cells when the mastoid contains healthy cells lined with thin normal mucosa.

Taking this and our previous overview into account, these authors prefer CWU, where possible, but consider simple mastoidectomy to be insufficient. Optimally, an antro-attico-mastoidectomy, taking care to preserve the scutum (sharp bony spur formed by the lateral wall of the tympanic cavity and the superior wall of the external auditory canal) and the posterior bony auditory canal wall must be performed with the aim of restoring the function of gas exchange to the postero-superior part of the middle ear cleft. Antro-attico-mastoidectomy is a CWU procedure by retroauricular approach, which is then superiorly and anteriorly enlarged, obtaining access to the attic and

antrum. In this situation, the opening of the antero-superior compartment allows specific cleaning with restoration of the gas exchanges. If necessary, this could be combined with Jansen's posterior (attico) tympanotomy [66] for more inferior and posterior access.

So, in our opinion, the posterior wall of the external auditory bony canal must be preserved or at least reconstructed, overcoming the inconveniences derived from the open mastoid cavity, making superior hearing outcomes more likely [67], and as to constitute an impenetrable barrier giving the mastoid gas cell system the means of performing its function[68].

Conclusion: Message to the Surgeon

The mastoid gas cells system has a physiological passive pressure buffer role in the balance of pressure variations in the middle ear cleft, for two main reasons:

(1) The physical anatomical properties of the middle ear cleft volume affect compliance: the greater the volume, the more compliant the system;

(2) The surface area of the mastoid gas cells system greatly affects the mucosal gas exchange.

It is our understanding that most otosurgeons too often only think about the mechanical approach of middle ear cleft surgery, while they should be aware of all the functional parameters of the mastoid and middle ear cleft, so they can justify a certain surgical approach and its consequences. The operated middle ear cleft is often conceived as permeated by gaseous flows. However, one should always consider the physiologic aspect of an operation involving the mastoid. The operated middle ear cleft should, if circumstances allow, result in a cavity still covered by a mucosa through which gas diffusion occurs so that each gas – nitrogen N_2, oxygen O_2, carbon dioxide CO_2 and water vapor H_2O – has its own specific diffusion rate influencing the healthy balance of pressure variations in the middle ear cleft.

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