Main Articles

Functions of the mastoid cell system: auto-regulation of temperature and gas pressure

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Abstract

This article presents a new approach to understanding the physiological functions of the mastoid cell system. It is suggested that the cell system, in combination with the continuous blood flow through the adjacent large vessels, makes up a compound functional unit that serves to protect the sensitive vestibular part of the inner ear from inadequate stimulation by external temperature changes. By virtue of the large surface area of the cell system mucosa with respect to the enclosed gas volume, the mastoid cell system may also work as a pressure regulator. Variations of the bi-directional exchange of fluid over the capillary network in the mucosa will change the size of the lumen that is available for the gas in the cell system. Volumes of gas and fluid can thus be exchanged to keep the intratympanic pressure within physiological limits. The process is most effective in a cell system with a high area-to-volume ratio.

Key words: Ear, Middle; Mastoid; Physiological Processes

Introduction

In most land living mammals the middle-ear cavity is widened into a tympanic bulla, and in some species the cavity extends into the temporal bone as a complex system of cell tracts. The mastoid cell system is highly developed in humans, even though the size varies considerably between individuals. The volume of the healthy cell system is much larger than that of the mesotympanum, and it is remarkable that so little is known about its physiological functions.

According to a previous view, the cell system was seen as a passive container; an extra volume serving to buffer pressure changes.^{1–3} This view applies, for example when a small volume of gas is removed from the cavity, and the eustachian tube remains closed for a period of time. In this case the resulting pressure change will be less in the presence of a large cavity as compared to the situation when the same amount is removed from a small cavity. At the same time, the extra volume supplied by the cell system implies negative consequences. Once a certain pressure level is induced, as for example, a negative intratympanic pressure induced by sharp sniffing,⁴ or when landing in an aeroplane, the amount of gas that must ultimately be passed through the eustachian tube to eliminate this pressure change will be larger in the presence of a large cavity, as compared with a

small one. In this respect, a large cell system volume would by itself be of no advantage.

The compound cavity comprised of the middle ear and 'air' cell system is filled with a gas mixture that is quite different from atmospheric air. The major part is composed of inert gases, nitrogen (80.5 per cent), and argon (one per cent). The rest is a mixture of biologically active gases, carbon dioxide (seven per cent), water vapour (six per cent), and oxygen (5.5 per cent).⁵ The area of the cell system is greatly enhanced by bony septa by which the volume is subdivided into a large number of small cavities. The bony septa are lined by a thin one-layered cubical respiratory epithelium that for the most part lacks cilia and goblet cells. The cell system thus constitutes a porous material with a large surface area, and it is postulated here that this specialized anatomical structure is of functional importance in connection with internal temperature control and active pressure regulation.

External temperature and vestibular stimulation

Characteristically, a material that contains numerous gas-filled pores is a temperature isolating material. In the case of the mastoid cell system, this may be of importance for protecting the inner ear from the influence of environmental temperature changes. The vestibular part of the inner ear is highly sensitive

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Fig. 1

Leaf-beating nystagmus released by external cooling of the right mastoid process. Three segments from the investigation are shown: five, eight, and 12 minutes after onset of stimulation. Bars in the timeline indicate seconds.

to thermal stimulation, which is the basis for nystagmographic investigation of the balance function when the external ear canal is flushed with water a few degrees above or below body temperature. A general sensitivity to external temperature changes would be highly impractical, and thermal stimuli from the environment (hot sunshine or cool winds) are not known to cause caloric responses with nystagmus and vertigo.

The inner ear labyrinth is located deep in the temporal bone, enclosed by the cell system and suspended by thin bone trabeculae. Medially, the cell system has broad contact areas with the cerebral dura. Only millimetre-thin bone plates separate the cell system from the middle and posterior cranial fossae. In addition, there is close anatomical contact with the large blood vessels, the internal carotid artery, and the venous sinuses. By separating the inner ear from the body surface, the sponge-like insulating layer of gas-filled cells with the high flow of blood through the large vessels may prevent the build-up of steep temperature gradients and rapid temperature fluctuations, and thus protect the inner ear from inadequate stimulation by external temperature changes.

This does preclude the possibility that vestibular responses can be released by extreme thermal stimulation at the body surface. Experiments performed at our vestibular laboratory show that nystagmus and a feeling of rotation can be provoked by cooling the mastoid process. Such a response is shown in Figure 1. Left-beating nystagmus was observed after application of a small plastic bag containing ice cubes and water over the mastoid behind the ear for five minutes. The temperature front seemingly proceeds at a slow pace through the porous material of the cell system since the reaction showed up and peaked after a latency of several minutes.

External temperature and middle-ear pressure

The gas pressure in a closed cavity will change according to the local temperature, and a stable temperature is of importance for the pressure regulation of the middle ear. Despite the possible temperature stabilizing property of the cell system, external temperature changes can cause pressure



Fig. 2

Pressure recordings from right and left ears using serial tympanometry. The pressure in the right ear (squares) increased with heating, and decreased with cooling of the skin over the mastoid process. Specifically, switching the fan from cool to hot air caused a large and rapid increase of the intratympanic pressure. The left ear served as control. The pressure in the left ear (circles) showed a continuous increase with interruptions in connection with tubal opening.

changes in the ear. Experiments performed by the present author using a hot and cool air blower directed against the mastoid process show that the middle-ear pressure can both decrease as a result of cooling the skin, and increase by heating the skin over the mastoid process. In the experiment illustrated in Figure 2, the subject lay down to avoid tubal opening and pressure equalization. During alternating one-hour periods, hot air (45°C), or cool air (22°C), was fanned against the right mastoid. The external meatus and choncha auriculi were protected by a rich amount of cotton wool. The middle-ear pressure was measured with serial tympanometry. The pressure was increased by heating, and decreased to a level well below the atmospheric pressure when the skin over the mastoid was cooled.

Diurnal pressure variation in the middle ear

The pressure in the middle ear does not remain constant but, rather, there are both slow and rapid



Fig. 3

Horizontal CT frame showing the location of the inner ear deep in the pyramid of the temporal bone, and the close anatomical relations between the cell system and the posterior and middle cranial fossae, and the sigmoid sinus. The right part of the picture shows how individual cells were traced to determine perimeters and areas.

pressure changes, and a characteristic course over time is displayed. In the daytime there is usually a slow, continuous decrease of the pressure to a negative level slightly below the atmospheric pressure. This is interrupted by intermittent fast pressure changes in the positive direction when the eustachian tube opens. During sleep, the course of events is reversed like a mirror picture of the daytime pattern. There is a slow spontaneous increase of the pressure to a level above the atmospheric pressure, and this is interrupted by intermittent rapid pressure changes in the negative direction.⁶⁻⁸ The 'traffic' through the eustachian tube is thus generally reversed every morning and night. In part, this can be explained by shallow breathing during the sleeping hours with increase of the carbon dioxide content of the blood, and a subsequent diffusion of CO₂ to the ear.' A diurnal variation of the gas-containing volume of the mastoid cell system may be an additional explanation.

Pressure regulation by gas-to-fluid exchange

The continuous exchange of fluid over the capillary network of the cell system mucosa is based on an intricate balance between the capillary filtration pressure that promotes egress of fluid, and the colloid osmotic pressure difference that holds the fluid back. The effective filtration pressure depends on several factors. Two of these factors are of special interest in this context, and both will influence the fluid dynamics in the cell system mucosa: The body position, and the intraluminal gas pressure of the cell system. Because of the hydrostatic pressure the intravascular pressure is higher in the horizontal position as compared with the vertical position. The filtration and re-absorption rates in the cell system mucosa can be assumed to change accordingly, leading to a net production of fluid at night, followed by local re-absorption during the day. The exudate is primarily interstitial within the mucosa, and may later form a thin fluid film over its surface. The

mucosa and its capillary network are also exposed to the gas pressure in the cavity. The filtration rate increases with a negative pressure in the cell system, and decreases when the pressure is positive. As a consequence of the variable volume of fluid, the volume that is available for the enclosed gas will change, and so will the gas pressure in the middle ear-cell system compartment.

Gas-to-fluid exchange and area-to-volume ratio

It is the large surface area of the cell system that is of main interest here. The cell system is partitioned in a large number of cell tracts having a complex form with a highly curved 3-dimensional inner surface, and the real surface area is much larger than indicated by the lateral X-ray projection. The mucosal lining also has a depth consisting of the mucosa with a thin superposed fluid layer. A change in this dimension will change the size of the enclosed gas volume. The surface area and the enclosed gas volume can be measured with the aid of high resolution computerized tomography (CT), and values for 24 healthy ears have recently been published by Park, Yoo and Hoon.⁹ The inner area was measured by adding the perimeters of all cells in all tomographic frames throughout the cell system, and the total volume was achieved by taking the sum of all surface areas. Finally, the results were multiplied by the frame thickness (1.5 mm). In their investigation the mean surface area was 167 cm² and the volume was a mean of 10.4 cm.

From these values the ratio between the surface area and volume can be calculated $(k_{A/V})$. For the measured ears the mean value for $k_{A/V}$ was 16.1 (cm⁻¹). Using this value it is possible to calculate the change of mucosal thickness (d) that is needed to change the intraluminal gas pressure by a certain amount, for example, 1 kPa (approximately 100 mmH₂0), according to the formula: d = F/k_{A/V}; where F is the pressure change expressed as a fraction of the atmospheric pressure. For a negative



FIG. 4

Functional model of the cell system. Cell tracts are depicted as a number of narrow wing-shaped slits with a large surface area, representing the ear's interphase to the body interior for exchange of fluid and dissolved gases. Only a minimal change of mucosal thickness within the slits results in a substantial pressure change in the middle ear (ME). Intermittent access to the atmospheric air is provided by the eustachian tube (ET).

pressure change of 1 kPa (one percent of the atmospheric pressure), F = 0.01 and in this case d = 0.0006 cm. We thus arrive at the remarkable result that a microscopical change of the mucosal thickness amounting to 0.006 mm will change the intraluminal gas pressure by 100 mmH₂0!

The present author measured the area and volume of one cell system of a healthy ear, using a program for morphometry ('NIH Image', a public domain program developed at the U.S. National Institutes of Health). Very thin CT frames (0.5 mm) were used in this case with the aim to trace even finer structures (B. Magnuson, unpublished data). The value for $k_{A/V}$ was 16.4 cm⁻¹ in this ear; thus only marginally different from the value given above. One frame from this ear is seen in Figure 3. A previous theoretical estimate yielded a value of the same order of magnitude.¹⁰ The numerous mucosal folds that normally span the lumen in the cell system also contribute to the total area of the cell system, but are not visible in CT-frames. So, it is reasonable to assume that the real area-to-volume ratio is underestimated.

Comments on the formula $d = F/_{A/V}$

An extreme value for F can be used to test the formula. Setting F to unity means a pressure change amounting to the total atmospheric pressure. In this special case, when all gas is removed from the cavity the whole volume should be filled up with fluid to keep the pressure constant. Using the present value for k_{AVV} (16.1 cm⁻¹), the resulting d is 0.6 mm (0.06 cm). This result appears to be counterfactual because many of the round, oval, or irregularly formed cells in the mastoid are obviously much larger. However, the correctness can easily be checked by multiplying the known surface area (167 cm²) by 0.06 cm, which yields the known volume \exists 10 cm³. Thus, the calculated 'd' is a mean

value for all spaces, broad and narrow. A rectilinear model of the complex surface anatomy of the cell system makes this more easy to understand. In Figure 4 the surface area is stretched out to form a container bordered by flat surfaces.

Discussion

It is argued here that the gas-filled cavities of the cell system in combination with the continuous flow of blood through the large vessels constitute a composite functional unit that prevents disturbance of the temperature sensitive vestibular sensors of the inner ear. This temperature stabilizing function may appear to be self evident once the idea is brought up and communicated. However, the cell system seemingly does not protect the ear from pressure changes caused by external temperature changes. This is explained by the fact that the lateral aspect of the cell system is located close to the body surface. A local temperature change involving only the most superficial part of the cell system will immediately lead to a pressure change within all interconnected cell tracts, and including the middle-ear cavity. In the case of a sclerotic or fluid-filled cell system the characteristics may be expected to be different from, or even opposite, to those of the healthy and wellaerated cell system. Because of the different physical properties of solid bone and fluid compared to gas, the intratympanic pressure would be less influenced while, at the same time, such ears would be more susceptible to vestibular stimulation caused by external temperature changes.

That the large mucosal area of the cell system can be of importance for regulating the pressure may seem paradoxical. However, it is neither the volume, nor the surface area that is decisive but, rather, how



Scheme to illustrate author's conception of the pressureregulating system. The middle-ear pressure is a result of a dynamic interplay between the three different components.

the cell system area relates to the enclosed volume; the area-to-volume ratio (k_{AVV}) . The central point is that the volume available for the gas in the cell system is not constant. The volume variation of the gas-filled spaces caused by the mucosal gas-to-fluid exchange occurs relatively slowly. One aquatic mammal (the hooded seal) is known to make use of a special adaptation for pressure regulation during deep sea diving. In this species, cavernous sinuses located in the middle ear can change volume rapidly, thereby adjusting the middle-ear pressure according to the diving depth.¹¹

In terms of pressure regulation, the enlarged cell system mucosa can be viewed as the ear's interface to the blood circulation and the body interior, while the eustachian tube is the interface to the external world (Figure 4). The pressure-regulating system of the middle ear is comprised of the continual interplay between three components, each of which can change the pressure up and down: passage of gas to or from the ear through the eustachian tube and, via the mucosa, diffusion of gas to or from the ear, and production and elimination of fluid, see Figure 5. This 'transmucosal route' may work independently and continuously for periods of time, the 'tubal route' affording intermittent correction of the pressure.

Exudation and local re-absorption of fluid are seen here as important components in the physiological pressure regulation. This is actually a variation of the classical hydrops-ex-vacuo theory that deals with the aetiology of middle-ear effusion in pathological cases (secretory otitis media). The classical theory is focused on patho-aetiology but, in contrast, the bidirectional process of gas-to-fluid exchange in the cell system is emphasized as essential in normal physiology for the regulation of the middle-ear pressure. When a small or moderate pressure change occurs in the ear – irrespective of whether it is caused by a change of ambient pressure, by gas diffusion, or in some other way - a very small addition or subtraction of fluid is needed to balance this change; the fluid may be interstitial within the mucosa, or a thin film spread over its surface. In a cell system with a small area-to-volume ratio the fluid will be distributed over a smaller surface area, which implies a risk for effusion of fluid. Further, the closing pressure of the Eustachian tube must be high enough in order to keep up the physiological positive intratympanic pressure during the night. Otherwise, with a low closing pressure, and without the build-up of a positive counter-pressure, the transudation of fluid might continue until the cavity is flooded. The closing pressure of the tube is known to vary between individuals, and also varies over time.¹² Children with middle-ear effusion often have a low closing pressure, which may be one of several other causes for the development of this disturbance.

Conclusion

The central theme in this paper is that the mastoid cell system is not a mere gas container. The normal cell system ensures a stable temperature environment, and thus prevents excitation of the vestibular sensors by external temperature changes. The gasto-fluid exchange over the large convoluted mucosal area of the cell system provides a means for regulating the middle-ear pressure. The volume available for the gas enclosed in the cell system is not constant, and a volume of fluid can replace the same gas volume, and vice versa, while keeping the pressure within physiological limits. Inclusion of fluid dynamics in the theory of the pressure regulation in the middle ear and cell system yields a new perspective, and highlights the relationship between the normal physiological state and pathological conditions.

References

- 1 Diamant M, Rubensohn G, Walander A. Otosalpinghitis and mastoid pneumatization. *Acta Otolaryngol (Stockh)* 1958;**49**:381–8
- 2 Austin DF. On the function of the mastoid. *Otolaryngol Clin North Am* 1977;**10**:541–7
- 3 Sadé J. The correlation of middle ear aeration with mastoid pneumatization. The mastoid as a pressure buffer. *Eur Arch Otorhinolaryngol* 1992;**240**:301–4
- 4 Magnuson B. On the origin of the high negative pressure in the middle ear space. Am J Otolaryngol 1981;2:1–12
- 5 Hergils L, Magnuson B. Human middle ear gas composition studied by mass spectrometry. *Acta Otolaryngol* (*Stockh*) 1990;**110**:92–9
- 6 Hergils L, Magnuson B. Morning pressure in the middle ear. Arch Otolaryngol Head Neck Surg 1985;11:86–9
- 7 Hergils L, Magnuson B. Middle ear pressure under basal conditions. *Arch Otolaryngol Head Neck Surg* 1987;**113**:829–32
- 8 Tideholm B, Carlborg B, Jonsson S, Bylander-Groth A. Continuous long-term measurements of the middle ear pressure in subjects without a history of ear disease. *Acta Otolaryngol (Stockh)* 1998;**118**:369–74
- 9 Park MS, Yoo SH, Lee DH. Measurement of surface area in human mastoid air cell system. J Laryngol Otol 2000; 114:93–9
- 10 Magnuson B. Physiology of the eustachian tube and middle ear pressure regulation. In: Jahn AF, Santos-Sacci JR, eds. *Physiology of the Ear*. 2nd edn. San Diego: Singular Publishing Group, Thomson Learning, 2000;75–99
- 11 Stenfors LE, Sadé J, Hellström S, Anniko M. How can the hooded seal dive to a depth of 1000 m without rupturing its tympanic membrane? A morphological and functional study. Acta Otolaryngol (Stockh) 2001;121:689–95
- 12 Bunne M, Falk B, Hellström S, Magnuson B. Variability of eustachian tube function in children with secretory otitis media at tube insertion and at follow-up. *Int J Pediatr Otorhinolaryngol* 2000;**52**:131–41
- 13 Bunne M, Falk B, Magnuson B, Hellström S. Variability of eustachian tube function: Comparison of ears with retraction type disease and normal middle ears. *Laryngoscope* 2000;**110**:1389–95

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