Current Topics in Biophysics, 2007, 30 (suppl. B) 20-21

A MEMBRANE VIEW ON THE EXPLICATION OF PATHOGENESIS OF MÈNIÈRE'S DISEASE

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From the clinical standpoint, Mènière's disease is diagnosed on the basis of three simultaneously occurring symptoms, of paroxysmal nature. These symptoms are: vertigo, hearing impairment and tinnitus. The cause of the disease is the formation of the endolymphatic hydrops within the cochlea of the inner ear (Becker, Nauman & Pfaltz, 1999; Schuknecht, 1993). Its formation and growth are the result of disturbed balance between endolymph production, which occurs in the *stria vascularis*, and its absorption in the endolymphatic sac (*Saccus endolimfaticus*).

In our previous works (Kargol *et al*. 2007; Kargol, 2007) we have demonstrated, on the grounds of the Kargols mechanistic transport formalism (Kargol, 2007; Kargol & Kargol, 2003) that the stria vascularis may also be the place of endolymph absorption. This follows from the fact that the stria cell membranes are heterogeneous porous structures, i.e. membranes containing water-permeable pores with different sizes S of cross-section surfaces. Basing on this fact, we have also developed (Kargol *et al*., 2007; Kargol, 2007) a certain membrane model which emulates the border zone between endolymph and the stria vascularis cells. It has been assumed that the membrane M_n which constitutes this border is a heterogeneous porous membrane. With respect to this membrane, it is possible to propose the occurrence of such a solute (s) that the following relation will be satisfied:

$$S_w < S_1 < S_2 <...< S_s <...< S_N,$$

where S_w is the cross-section surface of water molecules, S_1 is the cross-section surface of the smallest pores, S_s is the cross-section surface of solute s particles, and S_N – the cross-section surface of the largest pores. This means that the given membrane will, with respect to the solute, comprise the number n_a of semi-permeable pores (i.e. pores permeable to water only) and the number $n_b = N - n_a$ of permeable pores, i.e. pores permeable to water and to the solute s (where N is the number of all the membrane pores). On this basis, we have demonstrated that, given the occurrence of the osmotic pressure difference $\Delta\Pi$ and the mechanical pressure difference ΔP on the membrane M_n, the components of endolymph (water included) may be transported from the stria vascularis cells to the endolymph of the cochlear duct. Moreover, simultaneous absorption of endolymph (water in particular) – *via* the membrane M_n – by the stria vascularis cells is also possible.

Basing on this membrane model, we have discussed certain new determinants pertaining to the formation and growth of the endolymphatic hydrops. These determinants may be conditioned by changes to active transport J_{vs} of the solute s across the membrane M_n, decrease in the number of pores n_b (through their closure) and increase in the number of pores n_a, as well as the cost of decrease in the number of pores n_b (due to incomplete closure of a certain number Δn_b of pores n_b).

In the present paper, we present further hypothetical possibilities of formation and growth of the endolymphatic hydrops. The starting point for these considerations is the extension of this hypothesis to include the entire system of labyrinth fluids, as seen in Fig. 1.

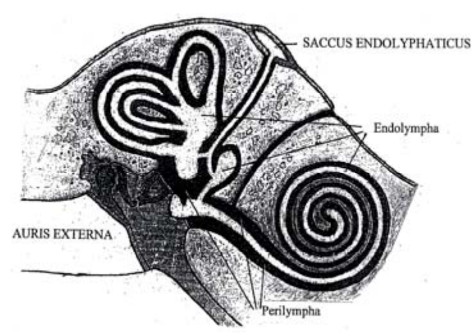

Fig. 1. Endolymphatic and perilymphatic spaces in the inner ear

Considering this extended scope of the investigation, it must be stressed that in the light of the former views, the place of endolymph absorption is the endolymphatic sac. Under the circumstances, the formation of the endolymphatic hydrops may be explained by impaired endolymph absorption in the sac or its increased production by the stria vascularis cells.

As already indicated, our membrane model proposed in the works (Kargol *et al*., 2007; Kargol, 2007), complements that view with the possibility of endolymph absorption by the stria vascularis.

To continue the analysis of the hypothetically plausible mechanisms contributing to the formation of the hydrops, we propose that mechanical interdependences between the perilymph and the endolymph be taken into account. It is not difficult to imagine, at this juncture, that the reason for the formation of the endolymphatic hydrops may also be a decrease in the pressure in the perilymphatic space (due to excessive perilymph absorption or its decreased production). To continue the considerations of this extended view of labyrinth fluid production and absorption, the effects of mechanical pressures on the fluids should be taken into account as well. In so doing, it is necessary to make allowances for the possibility of mutual transfer of these pressures between the endolymph and the perilymph across Reissner's membrane. This is possible due to the dynamic quality of its fine structure. It is reasonable to assume that the membrane is easily given to elastic strain at relatively very low mechanical pressure differences occurring thereon. This means that the mechanical pressure in the endolymphatic space may easily be decreased by decreasing the pressure of the perilymph within the vestibu-lar scala. As a consequence, the water flow across the membrane M_n will increase from the stria vascularis cells to the endolymphatic space of the cochlear duct.

This may lead to increased bulging of Reissner's membrane and its final split. Such a situation would be worth a detailed analysis, if experimental data concerning episodic blockage to the cochlear aqueduct (i.e. the connection between the perilymph and the cerebrospinal fluid) were available. Yet it seems that this possibility is considerably less likely in comparison to the former, which states that the endo-lymphatic hydrops is formed as a result of excessive endolymph production by the stria vascularis cells.

It should also be added here that in the case of allergic etiology of Mènière's disease, it may be assumed that the hydrops grows rapidly and violently, as observed in some allergic swellings.

Generally speaking, the pathogenesis of Mènière's disease appears to be highly complex in many respects. No wonder then that until the present it has been relatively little known. In view of the above considerations, the pathogenesis of this disease should be perceived as rather complex also in its biophysical aspect, including the mechanisms of substance transport across cell membranes.

REFERENCES

Becker W., Nauman H.H. & Pfaltz C.R. (1999) *Choroby Uszu, Nosa i Gardła*. BEL CORP Scientific Publ., Warszawa, 10–25.

Kargol M. & Kargol A. (2003) Mechanistic formalism for membrane transport generated by osmotic and mechanical pressure. *Gen. Physiol. Biophys*., 22, 51–68.

Kargol M. (2007) *Procesy Transportu Membranowego Substancji i Ich Biofizyczne Implikacje*. Wyd. WSTKiT Kielce, 167–174.

Kargol M., Kargol A., Mnich Z., Rogowski M. & Siedlecki J. (2007) Hipoteza membranowa patogenezy choroby Mènière'a. *Otorynolaryngologia*, 7, 16–20.

Schuknecht H.F. (1993) *Pathology of the Ear, Lea and Febiger*. Philadelphia, Baltimore, London, Tokyo, 80–153.