Review article
Pathophysiological and therapeutic considerations of otitis media with effusion from viewpoint of middle ear ventilation

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Abstract

Using nitrous oxide, we examined the gas exchange function through the middle ear mucosa in ears with otitis media with effusion (OME) in children, and found the function to be impaired in 50% of them. The size of the mastoid was significantly smaller in ears with negative gas exchange function than those with positive function, and the presence or absence of the function was even more significantly correlated with the presence or absence of aeration in the middle ear. Furthermore, the presence or absence of aeration in ears with OME was found to be significantly correlated with their prognosis (response of OME to antibiotics treatment) and also with the presence or absence of eardrum mobility examined by a pneumatic otoscope. Finally, after myringotomy and removal of effusion, the gas exchange function recovered in most of the ears with impaired function. These results indicate that the eardrum mobility test may serve as an appropriate indicator for the surgical treatment for OME. © 1998 Elsevier Science Ireland Ltd. All rights reserved.

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1. Introduction

It is known widely that the pathogenesis of otitis media with effusion (OME) is closely related to the dysfunction of middle ear (ME) ventilation, and many studies on the Eustachian tube (ET) function have been reported to date [1,3,4,7,8]. However, several questions still remain unsolved when viewed in terms of the ET function. For instance, the ME pressure does not progressively decrease even when the ET fails to open in children [2], and spontaneous resolution of OME cannot be understood from the viewpoint of the ET pathophysiology, because the ET function usually remains poor even in the convalescent stage of OME as well as in the active stage [15].

The answers to these remaining questions regarding the pathophysiology and treatment of OME have become clearer ever since the gas exchange function through the ME, namely the mastoid mucosa, has come to be recognized as important as the ET function in terms of the ME ventilation and pressure regulation in recent decades [6,10].

This article introduces clinical observations we conducted on the pathophysiology of ventilation in OME which have provided us several clinical indices for the appropriate diagnosis and treatment of OME mainly from the viewpoint of the gas exchange function through the ME mucosa [9].

2. Pathophysiology of OME in children

For the ME to function as a sound conduction system, it is important that atmospheric pressure is constantly maintained by normal ventilation, which is provided by gas exchange through the ME mucosa as well as by the ET. The gas exchange is a passive movement of any gas within the body according to the partial pressure gradient of each gas in the blood, interstitial tissue and any air space within the body such as the ME cavity. Those passive movements of the gases always render the ME pressure approximate to the atmospheric pressure through the summation of all the partial pressures of those gases [6,10]. Therefore, in normal ears, both positive and negative ME pressures tend to be equalized by this function even when the ET is unable to open (Fig. 1).

A problem is that there exists only a few reports on the assessment of the ME gas exchange function, except for those related to the constitution of middle ear gases [11–14]. Recently, we developed and established a method for the assessment of the gas exchange function using nitrous oxide [16]. The presence or absence of the ME pressure increase by the application of nitrous oxide indicates positive or negative exchange function of any gas through the ME mucosa (Fig. 2).

Using this method, we examined the gas exchange function in 84 ears, 44 children with OME who underwent myringotomy or ventilation tube insertion under general anesthesia, and then, examined the relationship between the gas exchange function and the size and aeration (presence or absence of air space) of the mastoid of those ears [18].

Fig. 1. Chronological changes of applied positive and negative middle ear pressures without deglutition. Both positive and negative pressures tend to approach to the atmospheric pressure in most cases.

Fig. 2. Typical example of middle ear pressure increase by application of nitrous oxide (N\textsubscript{2}O) in a normal ear.
The gas exchange function was found to be impaired in 42 ears (50%), and the size of the mastoid examined on X-ray (Schuller’s view) was significantly larger in ears with positive gas exchange function than in those without the function (Fig. 3). More interesting was that the presence or absence of the function was even more significantly correlated with the presence or absence of an air space in the mastoid examined on the computed tomography (CT) (Fig. 4).

These results may indicate that an air space (an air-fluid interface) is necessary for the gas exchange to work, and that a larger mastoid is more advantageous to preserve the air space in the ME when suffering from OME. This may be one of the reasons why ears with larger mastoids have lower susceptibility and higher curabil-

**Fig. 3.** Relationship between gas exchange function and size of mastoid in ears with otitis media with effusion.

**Fig. 4.** Relationship between gas exchange function and air space (aeration) in the middle ear in ears with otitis media with effusion.
ity of OME. Furthermore, the presence or absence of an air space in the ME is considered one of the significant keys for the formation of a pathophysiological condition in the ME in OME. Since it is well known that the ET ventilatory function, the other ventilatory function for the ME, is impaired in most of ears with OME [1,3,4,7,8], the ME ventilation and pressure regulation may be managed only by the gas exchange function in ears with OME. When fluid accumulates in the ME, it may smoothly be excreted from the ME through the ET if there is an air space left in the ME, because negative ME pressure due to the
excretion of the fluid can constantly be compensated by the gas exchange (entrance) through the ME mucosa. On the other hand, if the ME is filled with fluid without any air space, the fluid may not be excreted completely because the gas exchange function or ET is not available in the ME; this may cause a dead-locked condition in the ME characterized by coexistence of the negative pressure and fluid retention (Fig. 5).

3. Diagnosis

Thus, knowing if an air space exists in the ME is considered important for understanding of the pathophysiology of the ME in OME, but the question is how to determine if the space is present or not. To perform CT on every child with OME is apparently unrealistic, and to our best knowledge there has been no literature describing another good method to examine it.

For this purpose, we focused on the eardrum mobility test, of which the diagnostic value for the ME effusions has recently been reevaluated due to its reasonable cost-effectiveness in comparison with tympanogram [5], and drew a hypothesis. When there exists an air space in the ME, the eardrum moves according to the pressure applied from the external ear and is transmitted to the ME through the eardrum because air easily changes its volume in response to the pressure. In contrast, OME ears full of fluid in the ME and consequently having no air space will have no eardrum mobility because fluid almost never changes its volume by pressure. To test this hypothesis, the eardrum mobility was examined using a pneumatic otoscope in 56 ears with OME, and the results were correlated with the presence or absence of an air space in the ME examined by CT [17].

The eardrum mobility was found to be less often impaired in ears with OME than we expected; 30 of the 56 ears (53.6%) had good mobility of the eardrum. Incidentally, the tympanogram detected ME effusions by type B or C2 in 21 of the 27 ears examined (77.8%); this indicates that the tympanogram has higher sensitivity in detecting the ME effusion than the eardrum mobility test. CT was performed in 42 of the 56 ears. Twenty-nine ears showed positive mobility of the eardrum, in which 25 ears (86.2%) had aeration in the ME, while only three of the 13 ears (23.1%) without mobility of the eardrum had aeration in the ME, indicating high correlation of eardrum mobility with aeration of the ME. The presence or absence of the aeration in the ME in ears with OME can consequently be detected by the eardrum mobility test by a pneumatic otoscope in most cases.

4. Treatment

Now that we know that ears filled with effusion in the whole ME space are in a dead-locked condition with inability to expel the effusion from the ME, and that the condition can be detected by eardrum mobility test by a pneumatic otoscope, the next question would then be how we should treat OME ears of which the whole
ME is filled with fluid and the gas exchange function is impaired? Again, to our best knowledge, there exists no literature examining the relationship between the condition of ME space (fluid-filled or positive air space) and the prognosis of OME.

What we first attempted was thus to see if antibiotics are effective to release such a dead-locked condition. We examined the response of 62 ears with OME in children to antibiotics treatment (30 mg/kg of amoxicillin for 7 days) by tympanogram, and the results correlated with those of the eardrum mobility [17]. Eleven of 32 ears (34.3%) with movable eardrums showed improvement of OME, while the improvement was only three of 30 ears (10.0%) with immovable drums. These results indicate that ears with movable eardrums show a significantly higher improvement rate than those with immovable eardrums, and that the medical treatment does not prove effective for OME ears with fluid-filled ME. Furthermore, it was also suggested that the eardrum mobility test by a pneumatic otoscope has a significant prognostic value rather than a diagnostic value for OME.

Next we focused on the surgical treatment. Our task was to examine the gas exchange function after myringotomy and removal of the ME effusions in 21 ears in which the gas exchange function had not been observed before myringotomy [18]. In most of the cases (16/21, 76%) the function recovered after myringotomy. These results suggest that the impairment of the gas exchange function is likely to be reversible, and that the surgical treatments for OME such as myringotomy and tympanostomy tube insertion creates an air space in the ME to reactivate the gas exchange function and the ME ventilation.

5. Discussion

From the results of our several clinical observations on the gas exchange function of the ME mucosa, several important aspects of the ME pathophysiology of ears with OME were found.
1. The gas exchange function is impaired in half of ears with OME, and the impairment is closely related to the absence of aeration in the ME.
2. The absence of aeration in the ME in ears with OME can be detected by the absence of eardrum mobility using a pneumatic otoscope.
3. Effects of medical treatment such as antibiotics cannot be expected for OME ears without aeration in the ME.
4. Surgical treatment proves justifiable to those OME ears without aeration in the ME in terms of reactivation of the gas exchange function, which is the only ventilation and pressure-regulation function for the ME in ears with OME.

Consequently it is relatively important to take the gas exchange function into consideration when discussing the pathophysiology of ventilation in the ME in ears with OME. For instance, we examined the ET function in ears in the convalescent stage of OME, and found that the most important ET function, the negative ME pressure equalizing function, has not recovered even at that stage. Assuming that the ET is the only ventilation system for the ME, a large number of spontaneous resolutions of OME in children cannot be explained. Now this can be explained by
the entrance of air through the ME mucosa as the inflammatory factors in the ME decrease with the age of children. The significance of the mastoid pneumatization was also clarified in some parts by these studies. A large mastoid should be difficult to be filled with fluid due to OME, and gas entrance to the ME through the mucosa may facilitate transtubal excretion of the fluid by constantly equalizing the ME pressure when fluid production subsides.

The significance of the eardrum mobility test was also clarified. It shows almost precisely the presence or absence of an air space in the ME, indicating positive or negative gas exchange function of the ear. This examination thus allows us to predict the prognosis of OME of whether the spontaneous resolution or effects of medical treatment can be expected, because an OME ear with impaired gas exchange function will not be ventilated through any route. Since there has been almost no easy and consistent method for the determination of surgical treatments for OME until now, the eardrum mobility test may serve as a good indicator for the surgical treatment for OME.

References