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Letters to the Editor

Dear Sir,

On the Pathogenesis of Glue Ear

I have been reading with great interest the authoritative review of Professor Jacob Sadé about the pathogenesis of Secretory Otitis Media of the Glue Ear syndrome (Sadé, 1994). I would like to underline some of the pathological features of this ailment, well known to the pioneering otologists but ignored by pathologists. A thorough knowledge of the normal morphology of the middle ear system is essential in any consideration of the pathological changes. With the exception of certain areas such as the proximity of the orifice of the Eustachian tube and the surface of the promontory, the middle ear system is lined by a thin layer of flat cells usually described as 'endothelial cell-like'. In fact these cells are derived from the respiratory system and undergo, in the inflamed (infected) middle ear, overt or latent changes, resulting in a reversal or transformation into a secretory type epithelium (Friedmann, 1963, 1974, 1977, 1987; and Arnold, 1993; Tos, 1980).

The term 'metaplasia' has also been applied to this mechanism; that may or may not be the correct interpretation of mucosal change (Sadé, 1994). The cause or causes of secretory otitis media may not be known but it has been shown that after experimental infection with various micro-organisms the epithelial lining of the middle ear of various animals employed is promptly transformed, the surface of the inflamed mucosa becoming covered with tall ciliated columnar cells interspersed with many goblet cells (Friedmann, 1955, 1974). Similarly in the human middle ear the increased goblet cell population in the middle ear mucosa, consequent on chronic otitis media, contributes greatly to the amount of mucoid secretion and forms the basic substrate of the glue ear syndrome or catarrhal otitis media (Friedmann and Arnold, 1993). The term 'glue ear' may appear to define a more or less clear-cut clinical picture, but it cannot be related readily to a separate pathological entity except as a phase in the course of chronic otitis media.

The exact cause is not known, but it may be assumed that the condition starts as a bacterial or viral otitis media; this is then modified by immunological or other factors. Attempts to isolate a virus have failed. The transformed mucosa of the middle ear may behave like that of the nose and nasal sinuses; consequently some of the aetiological factors that lend to nasal discharge, including allergy, may enhance the production of mucus secretion of high viscosity in the tympanic membrane.

The severity of the condition may be expressed as the correlation between the secretory activity of the mucosa and the ability of the Eustachian tube to transport the secretory product. It may be interesting to mention that atypical cilia have been observed impeding the transport of the secretion, as first described by Sade in 1967 (Kawabata and Paparella, 1969; Friedmann, unpublished). In conclusion, I consider it of great importance that agreement seems to have been reached among otologists in support of the inflammatory and infectious pathogenesis of Glue Ear.

I am,
Yours sincerely,
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Dear Sir,

High Frequency Jet Ventilation—A Review of Its Role in Laryngology

In their paper in the January 1994 issue of the Journal, Evans *et al.* drew attention to the benefits of using the jet ventilator in a number of different ENT situations. However, they made no mention of its place in the management of acute airway obstruction, as has been reported in this Journal in the past (Squires and Frampton, 1986). While not for one moment suggesting that jet ventilation is the answer in every such situation, it is an option that should be borne in mind.

Yours faithfully
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References

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